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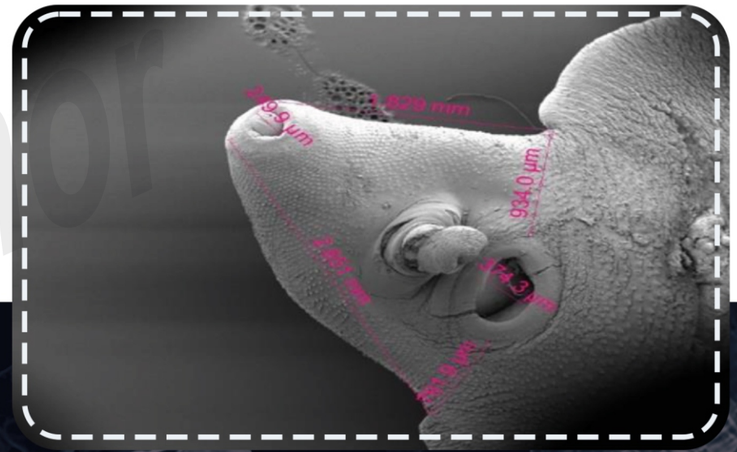
VETERINARY PARASITOLOGY PART I

Platyhelminthes and Nemathelminthes



THIRD EDITION
2025

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Veterinary Parasitology Part I

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PREFACE

This is a third edition of the textbook on veterinary parasitology has been updated and enhanced with new references materials. It has been developed not only to embrace educational classes of parasitology for veterinarian students but also more advance for clinical practitioners and university researchers. The first edition was only written as an overview concerning nematodes and trematodes parasites. The second edition was written with more details. Therefore, it is a privilege to expand more information of parasites in a new textbook and inspire veterinarian students to comprehend parasitology.

The first chapter (Chapter One) include the introduction and concept of the parasitology. Also, it deals with several important topics includes the relationship between hosts and parasites species, parasites terminologies, routes of parasite transmission, internal and external life cycle, general characteristics of parasites. Two important texts including the epidemiology of parasites and history of parasites is annotated as further section in the chapter one. The chapter on nematodes and flukes (Chapter two) also have been revitalized. In this book, a new section describing cestodes (tapeworms) is added, with the critical information of tapeworm species in livestock, humans, poultry, and canids.

Nematodes are the subject of chapter three, which contains a plethora of details on their macroscopic and microscopic forms, host immunity, treatment, and control or preventative measures. Interestingly, we provided the readers with many colorful mages especially about morphology and life cycle of the parasites. As we hope, the new edition would be praised by the students and staff members, help them

advance their scientific careers, and make the topic of parasites more understandable to readers.

At the end of this book, you can complete the test as homework using various questions that have been randomly extracted from this book.

CONTENTS

CHAPTER ONE

What is Parasitology: is the study of parasites species and a disease that caused by infection with parasites itself. This not confined either by organism or restricted by environment spaces but also connected to the life cycle of parasites and development in a host or as infective stage. Diagnostic techniques of the parasites have been applied along with other integrative disciplines involved the molecular biology, biochemistry and immunology.

A species is binomial

Shoveling history records, Linnaeus indicated a basic rule that a species is always binomial. For those who interested in the parasitology field, comprehension this concept did not succeeded. The Swedish scientist Carolus von Linnaeus (1707-1778) developed the binomial system of nomenclature and to-date remains immanent. Narratively, *Homo sapiens*is a group of extant species (humans therefore evolved). *Homo* was grouped as the genus and *sapiens*is a trivial name (*nomentriviale* or specific epithet). The epithet *sapiens* is not the species, although few old texts wrongfully referred to it. The word species is binomial (consists of two names). However, the taxonomically of parasites was classified based on the scheme below (figure 1):

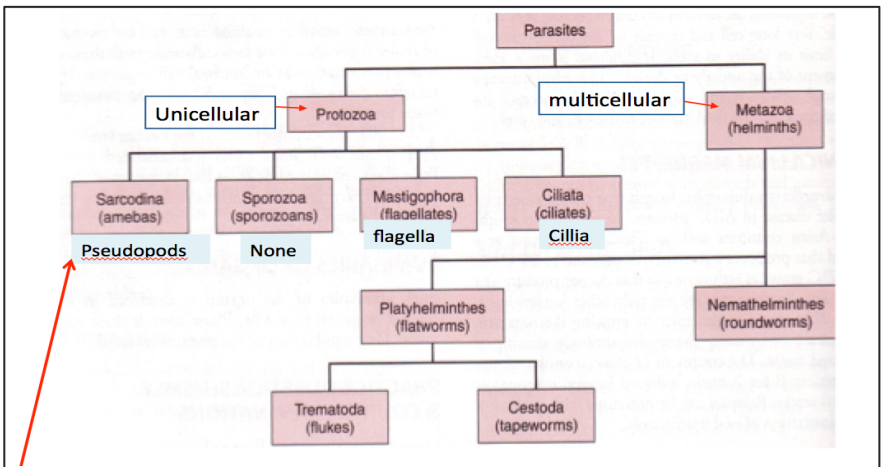


Figure 1. parasite classification hierarchy

Introduction to the Parasitology

Three main groups of animal parasites are medically important including protozoa, Helminthes (worms), and arthropods. It can directly or indirectly transmit and seriously cause a disease. The relationship, however, between organisms is illustrated in the following figure (2):

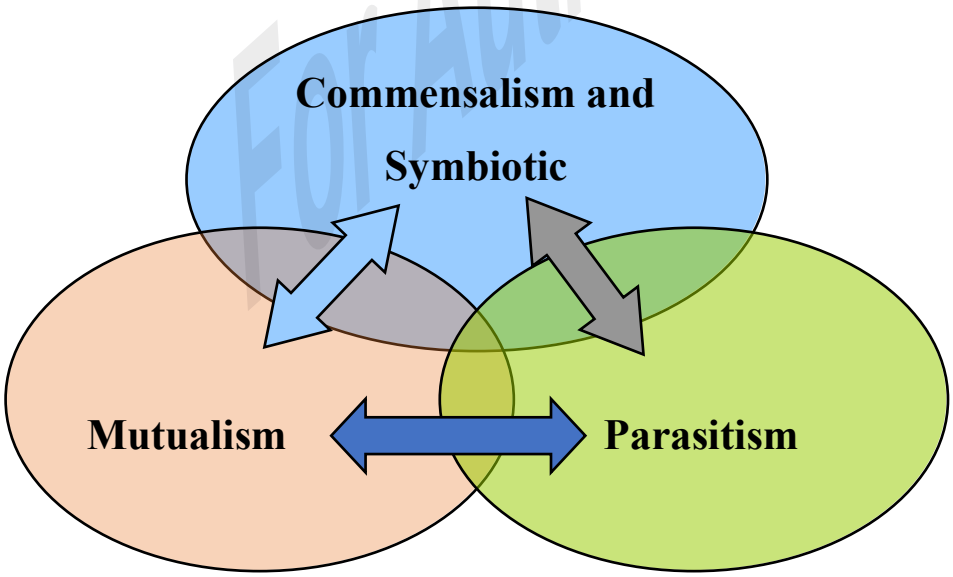


Figure 2. Type of parasite- host relationship

Parasitology is a type of SYMBIOSIS that living together in plant, animal, or protest, which is closely associated with another organism of a different species and each member is termed a SYMBIONT. There is various type of symbiosis includes:

A. PHORESIS is a traveling together or to carry, that is mean a smaller organism is termed a PHORONT that is carried mechanically by a HOST for instance, bacteria, fungus, cysts, or eggs on insect legs or even passively within an arthropod gut.

B. COMMENSALISM means when one symbiont is named a COMMENSAL, benefits and the other animal is neither helped nor harmed and true commensalism is difficult to find, and may not even actually exist. Close inspections usually reveal either a mutualistic or parasitic association. For example, *Entamoeba gingivalis* in mouth to some degree; some pilot fish and remoras associated with sharks.

C. MUTUALISM means each member is named MUTUALIST, depends upon the other and obligatory or facultative in nature for instance, flagellates produce cellulose in gut of termites, ciliates in ruminants, algae and fungus forming a lichen; crocodiles and Egyptian teeth cleaning plovers.

D. PREDATION means where one member is named a PREDATOR, benefits and a smaller organism is called a PREY that is harmed and usually eaten. This association is not usually considered a type of symbiosis, but it technically falls under the definition. Examples include coyotes and rabbits, cats and mice.

E. PARASITISM means where one member is named a PARASITE, lives in or on another organism that is called a HOST.

However, PARASITOLOGY is the study of the relationship between a parasite and its host. This method of existence is the single most successful way of making a living, and it has been estimated that no less than 80% of all species of organisms are parasites. Parasitic relationships may be temporary, facultative, or obligatory.

The Basic types of parasitism and terminology

- ECTOPARASITE: lives on surface of the host, appropriate terminology includes the terms "infected" and "infested" [i.e. ticks, lice, fleas].
- ENDOPARASITE: lives within the host, appropriate terminology is "infected;" infested is inappropriate terminology [i.e. roundworms in gut, tapeworms in gut].
- HYPERPARASITE: parasite lives within a parasite [i.e. malaria in mosquitoes; tapeworm larvae in fleas].
- VECTORS: transmits parasites from host to host, and this is divided into:
    - BIOLOGICAL VECTOR: essential in life-cycle of parasite.
    - MECHANICAL VECTOR: unessential in life-cycle of parasite phonetic.

Class of Parasites

1. Temporary Parasite: spend a short period of time in a host.
2. Permanent Parasite: a parasitic life throughout the whole period of its life.
3. Facultative Parasite: a parasite does not rely on a host with opportunity to survive in environment.
4. Obligatory Parasite: Cannot exist without a parasitic life.
5. Occasional or Accidental Parasite: Attacks unfamiliar host.
6. Wandering or Aberrant Parasite: does not have a specific place to settle in.

Types of Hosts:

- Definitive or Final Host: Host in which parasite reaches sexual maturity and reproduction.
- Intermediate Host: Some development in host, but does not reach sexual maturity, often asexual stages.

- Paratenic or Transport Host: parasite not develop but continues to live and being infective to next host, for instance pseudophyllidean tapeworm larvae in fish.
- Reservoir Host: Non-human animals that serve as sources of infection to humans.

Modes of the parasite dynamic infection and transmission

Various ways were identified as a mode of the parasite infection and an etiological agent can pass from a host to another. The transmission can be taken placed either direct or indirect (figure 3):

1- Direct transmission: the parasite transmits from animal to a person through direct contact such as touching with contaminated hands. Echinococcus eggs can be transmitted directly from a companion dog to humans or children play on ground may be contaminated with the eggs of live parasite.

2-Indirect transmission: this type of transmission occurs indirectly through a mediate object such as airborne, waterborne, vegetables and grass contamination, dust, vector, and fomites). For example, *Leishmania* spp. is transmitted to humans by phlebotomine sand fly bites. Another example *Trypanosoma* is transmitted to humans by tsetse fly (*Glossina*). Several animal hosts can be acting as a reservoir for parasites infection and the transmission is substantially occurred by:

1. **Vector hosts** area living organism involves in the life cycle of the parasite. Most of these vectors are bloodsucking insects talking a blood meal from animal or human and transferred to another host. A typical example of vector borne diseases including Schistosomiasis, human African Trypanosomiasis, Leishmaniasis, and Onchocerciasis.
2. **Reservoir hosts** are humans, animals, crustaceans, and inanimate organic (faeces or food) where parasites are able to live, grow and multiply under optimum condition. For example, *Diphylobothirum latum*

(**fish or broad tapeworm**), the infections are acquired by eating raw or undercooked fish.

3. **Carrier hosts** is referred to the host harboring a pathogen without showing clinical symptoms or being less severally ill. Thus, animals serve as a source of infection transmission parasites from animal to another or to humans while remains in apparently diseased sometimes called “asymptomatic carriers” example, nematodes parasites.

There are two or more host species and parasite exploits food chain relationships of host increases chances of locating hosts and reduces chances of super-infecting host.

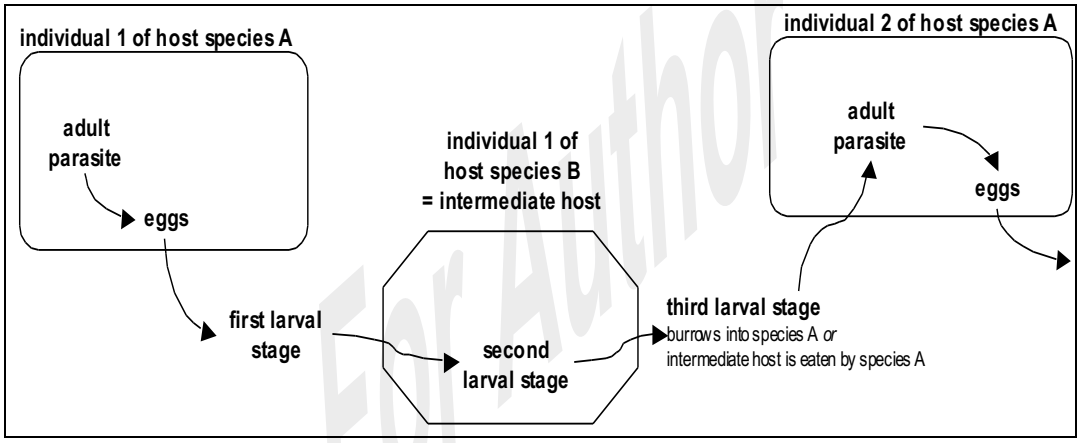


Figure 3. Scheme of Complex Life Cycle

Nomenclature of Parasites

Each parasite possesses two names, a generic and a specific that the former begins with an initial capital and the latter with an initial small letter, after which comes the designator's name followed by punctuation and finally the year. The generic and specific names are in italics but not the designator's name. For example, the common name of intestinal roundworm of horse was named *Parascaris eqourium*, Linnaeus, 1758. When the name assigned to the parasite is later transferred the correct name is written as usual followed by the original name with the year of parenthesis. The describing animal parasites certain rules of

zoological nomenclature are followed and each phylum may be further subdivided:

SUPERPHYLUM----------PHYLUM--------SUBPHYLUM

SUPERCLASS----------CLASS---------- SUBCLASS

SUPERFAMILY--------FAMILY----------SUBFAMILY

SUPERORDER----------ORDER---------- SUBORDER

GENUS

SPECIES

IMPORTANT CONCEPTS OF PARASITIC INFECTIONS

- Infection = presence of an agent and has ability to cause disease
- Disease = the occurrence of dysfunction
- Infectious = capable of causing infection
- Infection --- Disease --- Infectious

EXAMPLES:

A. The dog showed no adverse symptoms to the 2 females *Dirofilaria immitis* in its right ventricle (Infection).

B. *Haemonchus contortus* produce 1,000 juveniles and cause acute anemia in the lamb (Infection + Disease).

C. The cats suffer from large bowel diarrhea due to *Tritrichomonas foetus* which pass active trophs in their feces (Infection+ Disease+ Infectious).

D. The cat passed several active proglottids of the flea tapeworm, *Dipylidium caninum* (Infection).

Parasitism

There are different ways or characteristic of parasitism:

1. High reproductive potential, i.e. multiple fission in Apicomplexa; hermaphrodism of trematodes; parthenogenesis in *Strongyloides* spp.; i.e. strobilation of tapeworms for high ova output; and overall high ova/larval output of many worms.
2. Often unique morphological or physiological specializations, loss of structures, like:
    a. loss of digestive tract of tapeworms
    b. loss of wings of fleas and lice
    c. loss of many sensory structures of nematodes
    d. development and refinement of a tegument; a living external layer of digenea, cestodes and acanthocephalan that allows digestion and other functions across body surface
    e. development of special holdfast organs, including hooks, suckers, teeth, clamps, cutting plates, spines
    f. production of anti-coagulants in leeches and hookworms
3. Often special site specificity.
4. Usually, but not always, non-lethal to host.
5. Generally more numerous than hosts.
6. Generally much smaller than host if larger, then termed a predator.
7. Often have evolved methods of evading host immune system, like:
    a. Antigenic variation of trypanosomes.
    b. Tough tegument of acanthocephalans.
    c. Intracellular habitat of coccidian and *Trichinella* larvae.
    d. Antigen acquisition of Schistosoma.
    e. Suppression eosinophiles or neutrophil migration to the site of the parasite.

f. Encystment.

g. Ability to cleave antibodies or consume complement.

h. Ability to trigger certain arms of the immune response, which may in turn damage host tissue enough to facilitate parasite invasion.

8. Level of pathology due to the parasitism, like:

a. Physical trauma: Cells-tissue destruction because of the migration of nematodes through tissues, ulceration of intestinal wall and liver by cysteine proteases of *Entamoeba histolytica*, displacement of tissue or structures by hydatids, protease digestion of epithelial cells by *Trichomonas virginals*, ulceration due to insertion of hooks and spines directly into intestinal wall.

b. Nutritional diversion: such as giardiasis results in diarrhea and mal absorption and *Diphylobothirum* absorbs vitamin B12.

c. Toxins/Excretory products/Immune complexes: African trypanosomes slough antigen/Ab complexes that are absorbed by RBC, complement activated, massive RBC lyses, as an excretory product of some trematodes and cestodes which ultimately leads to anaphylaxis as a fibrosis and inflammation around *Schistosoma* eggs in the granulomas, while fibrosis and edema formed against filarids.

9. Blood loss: hookworms which causing anemia.

Transmission ways of parasites

INGESTION from food or water / inhalation included VECTORS or/ and DIRECT PENETRATION of skin from environment.

Some additional terminology to be used in class of **Anthroponoses** in the human diseases that can be transmitted to animals.

Epidemiology and Parasite Diseases in terrestrial animals

The study of defining causes, determinants of illness incidence and distribution, disease control, and prevention is referred to as “epidemiology”. In veterinary medicine the epidemiology has become important field used to investigate on a source of the disease and to assess economic losses that resulted from a disease morbidity and mortality. The epidemiology also involves addressing different categories including age, sex, religion, education, time, place, grazing system, sanitation level, anti-worm administration, animal transportation and trades, and vaccination. In addition, epidemiology is used to establish a hypothesis regarding the causation of illness, and statistical tools are utilized to illustrate the link between variables and disease incidence. The main goals of the epidemiology are recapitulated as following:

1. Identifying the etiological agent of a disease;
2. Assessing the risk factors are associated with the disease spreading;
3. Indicate valuable information on the environment and history of the disease;
4. Highlighting the significant impacts caused by the disease.
5. Designing effective control programme to reduce the disease and it is extent;
6. Monitoring and surveillance to prevent reinfection of the disease;
7. Incorporating biosecurity measurements to shield the community from other diseases impingement.

Terminologies are related to epidemiology and disease occurrence

Endemic: is a constant persist of a disease level within a given population or certain geographical region or zone. For example, cystic echinococcosis is endemic disease in sheep in Basrah province and other regions in Iraq because of absence control measures which dogs and other carnivores spread the infection agent (*eggs of Echinococcus*) in the environment, contaminated water and grass.

Hyperendemic: (Hyper means above) is a continuity level of a disease beyond or above the expected prevalence. When a parasite is evaluated with highly prevalence rate reach to more than 30%, for example the prevalence of *Toxocara canis* was evaluated to be high proportion in free-raoming dogs in Iraq.

Epidemic: is referred as outbreak take place by a highly contagious disease or more transmissible to humans or animals. For example, *Trypanosoma* in Africa swiftly annually infect indigenous people and has been escalated due to environment condition influence of vector transmission. The species *T. b. gambiense* and *T. b. rhodesiense* are widely distributed among local families and in a large extent between villagers, in western and eastern Africa. The disease is common in cattle being reservoir and rare transmitted by wild animals.

Epizootic: (Epi: upon, zoon: animal) similar to epidemic but the disease event restrictively occurs as an outbreak within animal. Highly number of new cases occur over period of time and probably responsible for animal death.

Pandemic: (Pan mean across): is an epidemic that is widespread a country or continent or become worldwide spreading. The outbreak usually has largely affected population with unstoppable reporting new cases.

Epidemiological glossaries

1-Aberrant host: is similar to accidental host and does not seem to play an important role in epidemiology or persist parasite life cycle.

2-Accidental host: has similar to aberrant host in the definition.

3-Incidence: is defined as a number of new cases of a parasite disease that present in a population (animals or humans) in a certain geographic region within a defined period, for example, 20 new cases of Leishmaniasis per 100,000 persons every year.

4-Infection: transmission of a disease or agent from a potential carrier host to a new host.

5-Monitoring: ongoing collection of information on diseases in a population over period of time

6-Surveillance: is a systematic ongoing collection, collation and analysis information related to animal health with timely disseminate information to the health authority to take action for the community for take warning.

7-Morbibdity: number of animals or humans have a disease.

8-Mortality: Number of deaths in population due to particular reason often diseases occurrence.

9-Screening: undergone test to diagnose a disease for those who being asymptomatic to infection.

10-Survey: Collect information from samples on a disease based on a specific design study.

The epidemiology triad

Three interrelated pillars that briefly constitutes empirical concept of epidemiology and diseases existence encompassing agent, host, environment (figure 4).

Agent: is any microorganism that cause a disease from the entering a host like bacteria, virus, parasite.

Host: animal or human or even a vector when the agent entering the target host or a susceptible host. Parasite like worms (Macroparasite) can be found mainly in the small intestine of humans and animals. Microparasites like schizonts can only found inhabitant and developed in tissue or blood such as *Theileria* and *Eimeria*.

Environment: is favorable surrounding and outside condition that allow a disease to transmit to human or animal. Environmental factors fundamentally include ecological factors (temperature & humidity), in addition to social and cultures factors. Disease occurrence is often related to a specific point of time in the year because of the agent activation or propagation after dormant period. For example: ticks in sheep are more common during in the hot season. Some parasite diseases are restricted to certain geographical area because of rainfall and relevant temperature influence on existence or surviving an infection agent.

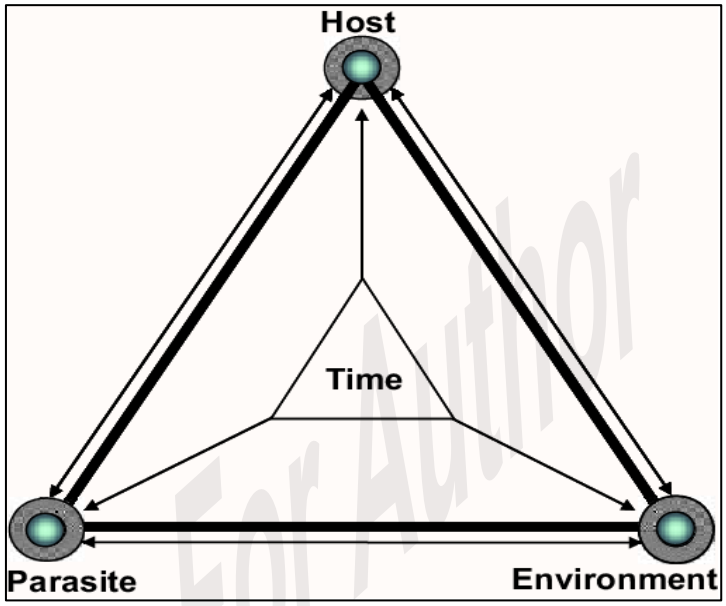


Figure 3. the triangle of epidemiology

Life cycle of parasites (figure 5)

- Domestic cycle: this type of cycle involves only domestic animals (eg: cattle, buffalo, sheep, goats, camel).
- Intermediate cycle: cycle in which involves both domestic and wild animals. (eg: Echinococcus found in feline species)
- Sylvatic cycle: this involves only wild animals. (eg: *Echinococcus granulosus* was found in a wide range in cervid animals [moose (*Alces alces*)] in the indigenous areas of Canada.
- Synanthropic cycle: Cycle associated with human habitats (eg: *Trypanosoma cruzi*).

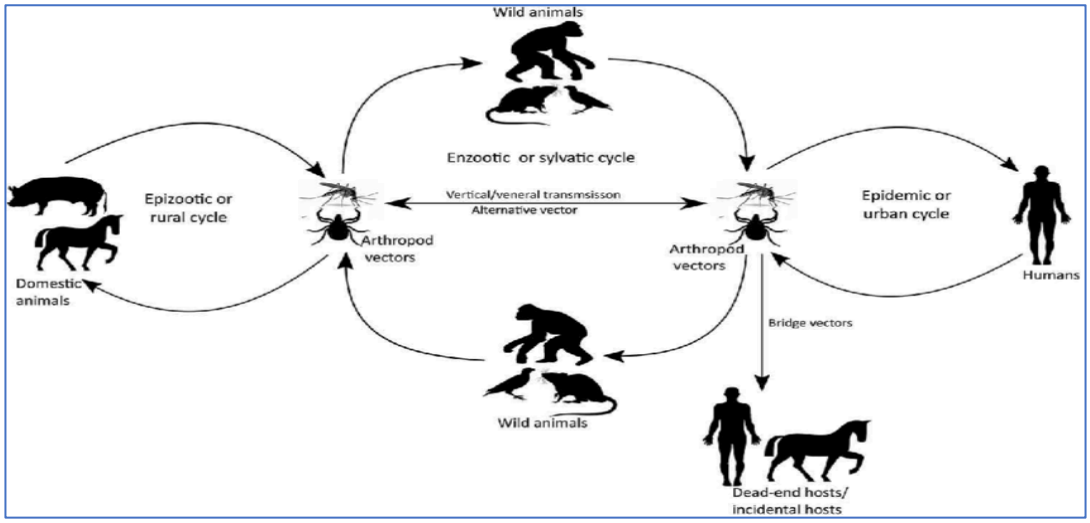


Figure 4. Sylvatic and domestic of the biological life cycle of an etiological agent

Parasitic disease behaviors and patterns

Many diseases included parasite infection does not occur randomly in a group or animal or in herd or in country or an area. There is always driving factors that manipulate the diseases existence in a population could be associated with the agent, host, ecological variables. These factors cause persisting the agent in the population as long as would never be controlled thereafter, including:

1. Individual characterization
2. Population characterization
3. Incubation period
4. Pathogenicity and virulence
5. Herd immunity and vaccination approval
6. Parasite diseases by time
7. Parasite disease by place

History of parasite discoveries (hydatid cysts, Leishmania, Babesia)

1. Leishmaniasis

Leishmaniasis was known for thousands of years dates back to the rule of Ashurbanipal in the 7th century, and according to some evidence discovered on a clay tablet. Because of the reflection of the skin lesion form, the disease was given the labels white leprosy and valley sickness in the 15th and 16th centuries. Until the mid-seventeenth century, Alexander Leishman outlined the signs of illness of a patient. Subsequently, the diseases were given the name Leishman after Willian Leishman, a British army doctor who served for a certain time in India and examined several patients afflicted with Leishmaniasis. In 1903, the first publication describing the disease was published. Charles Donovan studied a patient tissue specimen infected with Leishmaniasis and called the disease *L. donovani*. Both physician Lionel Napier and Ernest Struthers at the School of Tropical Medicine in the United Kingdom formulated a theory of *Leishmania* transmission by sand fly. According to epidemiological studies, several numbers of countries in tropical and subtropical regions are excruciated the disease, with around 12 million people were reported having the diseased interpreting to 1.5–2.0 million new cases each year, particularly foci in America western Asia and the Middle East. Obviously, the disease beside it flavors high temperature can also be seen in a poor area with lack of hygienic condition. Cutaneous disease is often recorded in Afghanistan, Algeria, Brazil, Colombia, and Iran, while mucocutaneous disease is most common in Bolivia, Brazil, and Peru, and visceral disease is most common in Bangladesh, Brazil, Ethiopia, India, and Sudan.

2. Hydatid cysts

Hippocrates (∽460-377 BC) was the first person to document information about hydatid cysts and indicated “In those whose water stuffed liver opens into the omentum, the belly is filled with water, and they die”. Al-Rhazes, a Persian physician, described hydatid cysts in the liver as watery balloons in *circa* AD

900. At that time, it was believed that the cysts were eggs or embryos of an insect or a cystic tumor. Aelius Galenus 1821-1833 (cited in Grove, 1990) reported that "the liver is very much inclined to generate hydatid cysts in the surrounding fascia". Hydatid cysts were diagnosed in several patients at that time, but the disease or its cause were not understood, and it was believed the cysts were due to dysfunction of the lymphatic glands. In the 17th century many researchers described the features of hydatid cysts. For example, Peter Pallas divided hydatid cysts into adherent and non-adherent forms and named the cyst *Taenia hydatigenia*. The protoscolices in the hepatic cysts were described in detail by Goeze, and subsequently Batsch (1786) described the hydatid cysts in sheep and named them as *Hydatigenia granulose*. During the 18th century advancements were made on the understanding of the life cycle of the parasite in both the definitive and intermediate hosts. Several experimental studies were performed by Carl Theodor von Siebold, Breslau, and Friedrich Küchenmeister to further the understanding of the life cycle of Echinococcosis. They fed a group of dogs metacestodes of *E. veterinorum* isolated from slaughtered sheep, which led to the discovery of the strobilar stage. Subsequently Von Siebold named the adult worms *Taenia* (*T.*) *echinococcus*. However, a series of further experiments were undertaken to confirm the life cycle of the parasite. The faeces of infected dogs were fed to a group of sheep resulting in the production of fertile liver cysts. Rudolf Leuckart fed a group of piglets with eggs of *Taenia echinococcus*, obtained from a dog, and four weeks later detected the presence of small vesicles (0.25 to 0.35 mm) with a laminated layer in the liver of these animals. Finally, Rudolphi in 1801 suggested the genus name of *Echinococcus*, being derived from the Greek word meaning "spine and berry". Subsequently German researchers, including Leuckart and Virchow, undertook studies to determine the morphology of the cysts and their contents. This resulted in the discovery that the single cyst lesions were caused by *Echinococcus granulosus* and multiple cysts in the infected organs were caused by *Echinococcus multilocularis.*

3. Babesia

In 1888, victor babes who was Romanian microbiologist first identified the etiology and named haematococcus bovis. He described the disease in cattle and sheep causing lyses in the blood cells. Both Theobald smith and Fred kilborne identified the parasite which responsible for Texas cattle fever and the disease is transmitted by ticks, serve as disease vector. In 1957, the first cases of babesiosis in human was reported in Europe. Several human cases were also documented in 1969 with individuals diagnosed with splenomegaly. Epidemiological investigations were identified hundreds of human cases clustered in Massachusetts, Rhode Island, and New York, and the *Babesia microti* was the dominated species infected humans.

General view

Many parasites dynamically cause changing host behavior. Growing number of scientists believe that many ecological studies need to include Parasitology as component as much animal behavior can be explained by level of parasitism. Especial behavior of some insects harboring larval stages of parasites, bird behavior in response to both ectoparasites and densities of some intestinal worms.

Host Specificity

Some parasites have specific host to infect and often are sensitive to their body temperature or environmental conditions as well as might attack other available hosts such as fleas.

Immunity against parasites

Parasites possess three major characteristics that make them difficult for a host to control immunologically: their size, their elaborate life cycles and their antigenic complexity. The Protozoa are most have complexed life cycles and the various stages of either antigenically distinct, as in the malaria parasites, or variable as in the African trypanosomes. Protozoa inhabit the gut, blood or other tissues,

including macrophages and the immune responses elicited are more appropriate to the site of infection than to the nature of the parasites themselves.

In the helminthes infestation, the nature of the surface of the worm, which is the part available for immune stimulation and attack is important. The tegument of the worm, for instance Digeneans and Cestodes is delicate while the outer of the nematodes surface is a protective cuticle, the antigenic nature of which may vary during the life cycle. Like protozoa, different Helminthes occupy different sites usually the gut, but sometimes the blood or other tissues and the immune response is again more appropriate to the site of infection than to the actual parasites.

A further complication in helminthes infections is that during its life cycle worm may not only change its features but may also change a site of infection over times. In *Ascaris lumbricoides* infections, larvae pass through various internal organs before become mature in the host gut, the result of these variations cause the expression of antigens and elicit immunity against foreign antigens at the site of infection.

Parasitism Aspects

- Each animal can be a host of many parasites; thus, there are far more parasitic organisms on earth than there are non-parasitic organisms.
- It has been estimated that more than 50% of all living plants and animals are parasitic at some stage during their life cycle.

Veterinary importance of parasites

- A poultry farmer can be wiped out by Coccidia.
- Cattle, pigs, and sheep infected with parasites fail to gain weight and may not reproduce.
- Dogs may become infected with heartworm and die if untreated.
- Cats are infected by many species of protozoans and helminthes.

• In Africa, cattle cannot be raised in an area equal to that of the U.S. due to trypanosomes.

Why do we study Parasitology?

Generally, parasites provide as a unique example of biological phenomena not found in free-living organisms and distribution in all the world and found even in plants, so, a different importance can be found, like:

• Medical importance: when infect or infest human and causing severe disease some time leading to the death.

• Veterinary Importance: when infect or infest different animals such as wild, domestic, birds and different mammals and causing economical losses or sever disease or complicated disease like viral, bacterial and rektticial leading to the death.

• Economic Importance: when infect or infest different animals of domestic and birds that causes economical losses of meat and milk product, egg product, or other products.

CHAPTER TWO

SYSTEMATIC PLATYHELMINTHES

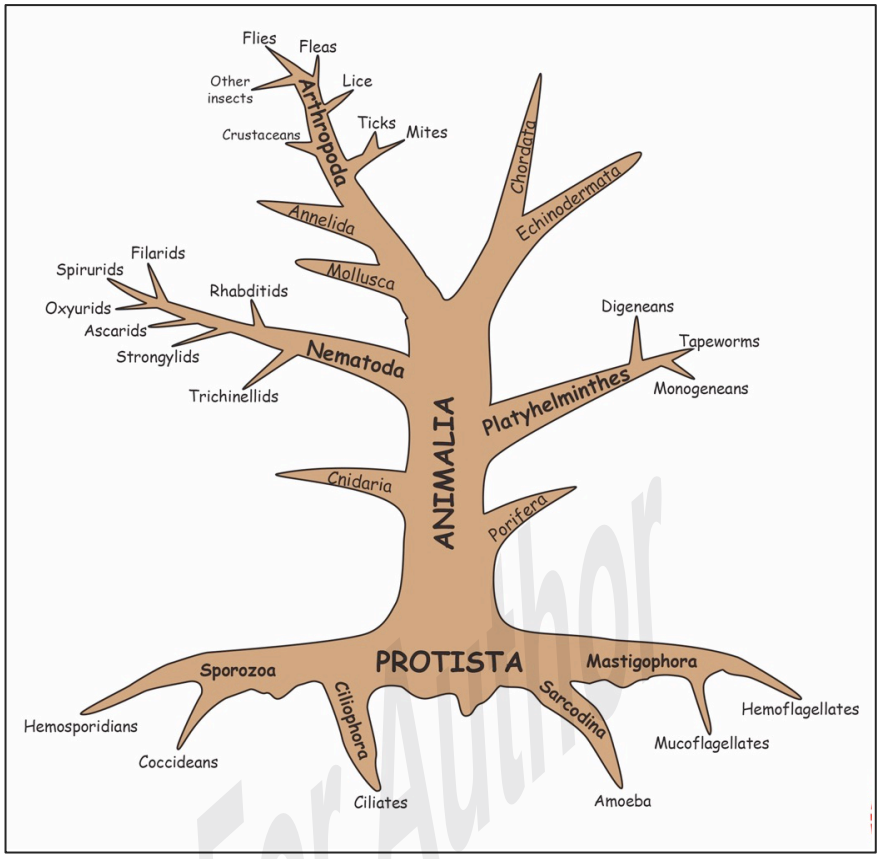


Figure 5. systematic tree of parasite classification

General characteristics of the Platyhelminthes:

Platyhelminthes is one of the most important phylum with numerous species were identified worldwide (figure 6) and their general characteristics are briefly as following:

1. bilaterally symmetrical; dorso-ventrally flattened.
2. A celomate.
3. cannot synthesize fatty acids.
4. tegument (living external layer).
5. digestive tract incomplete or absent.
6. excretory system protonephridia.

The phylum Platyhelminthes consists of four classes

1. Turbellaria

Free-living flatworms and some of their species are associated with echinoderms, molluscs, fish, cnidarians, as symbionts. A large number are commensals even though few inhabit the invertebrates. Example of the free-living parasite is *Planaria* that naturally lives in aquatic environments, while the giant terrestrial land planaria in the genus *Bipalium*. The commensal/parasitic forms include *Syndesmis* spp. resides in the intestines of sea urchins, *Bdelloura* spp. on the gills of horseshoe crabs, and *Stylochusfrontalis* in the valves of oysters.

2. Monogenoidea (Monogenea)

All parasitic, with the majority of species preferring to live on fish gills or skin; however, some internal species have been found to inhabit the urinary bladder, nasal passages, and cloaca. Biologically, this class appears to be more closely related to cestodes than trematodes, with two main "traditional" subclasses:

- Subclass: Monopisthocotylea
- Subclass: Polyopisthocotylea

3- Trematoda

Live in the digestive tract, provided with suckers, and have two hosts for their life cycles, and divided into three subclasses**:**

1.Subclass: Digenea (typical flukes)

2.Subclass: Aspidogastrea

3.Subclass: Didymozoidea

4- Cestoidea

Note: All parasitic; tapeworms, most have 2-more host life cycles, no digestive tract, with segmented body.

Monogenea

Introduction: Monogenea occur at low levels on fish and has no serious harm. Infested fish farms with monogenea causes significant mortality

General characteristics of Monogenea (figure 7)

1. hermaphrodites
2. normally ectoparasites on aquatic vertebrates
3. generally, site specific on host and host specific
4. live a few days - years, depending on species
5. morphologically: anterior end bear adhesive or feeding organs. Eyespots sometime present, photoreceptors near two anterior ganglia. The tegument is the external layer.

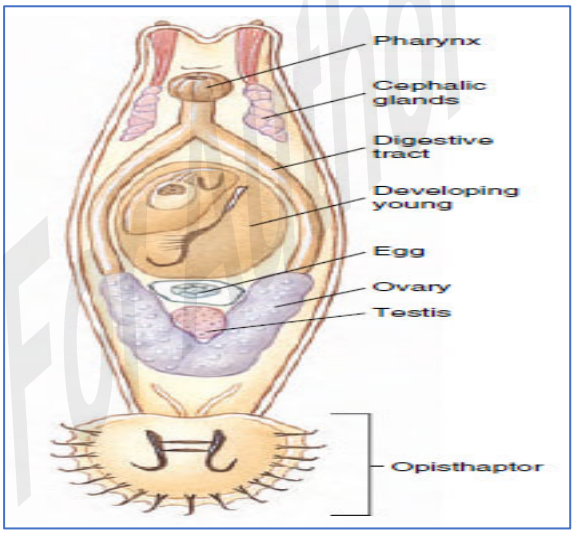


Figure 6. Structure of Monogenea

Alimentary tract consists of:

1. mouth anterior, usually with Prohaptor.
2. esophagus with muscular pharynx
3. intestine branches into cecae, often with diverticula
4. monopisthocotylea feed mainly on epidermis and mucus; Polyopisthocotylea mainly on blood, host cells, and mucus
5. blind-ended gut, regurgitate waste

Protonephridia [excretion] consists of:

1. have two main lateral ducts, extend posteriorly; then curve and extend anteriorly.
2. contractile bladders laterally.
3. flame cells drive fluid within ducts.

Reproductive systems consist of:

- **Male:** 1-200 testes (1-2 most common), testes - vas efferent - vas deferens - seminal vesicle - cirrus - gonopore; sometimes prostate
- **Female:** one ovary, normally anterior to testes, oviduct from ovary – ootype, Mehlis gland lubricates uterus, forms egg shell capsule, genito-intestinal canal, only in Polyopisthocotylea; connects oviduct with right intestinal caecum for excess secretions discharge, 0-2 vaginas - sperm transfer; if none, use gonopore, sometimes a seminal receptacle, vitellarial secretions add to egg-shell formation; ducts fuse near oviduct, eggs pass through uterus, out gonopore; sometime a muscular metraterm

Development

- eggs shed, normally with filaments, and filaments stick to host.
- oncomiracidium ciliated, hooked posteriorly, about a 24 hours life, if eyespots, phototactic, some attracted to fish mucus, grow to adult directly.

Opisthaptor which may be:

- suckers (=suckerlets)
- anchors (large hooks, sometimes called hamuli or central hooks)
- hooklets (left over larval hooks, sometimes called marginal hooks)
- bars (often called accessory sclerites; they support anchors)
- clamps (complex, muscular structures more advanced than suckers)

Digenea

Digenia has two suckers and attached organ (figure 8).

A. oral (usually anterior) sucker. Surrounds mouth.

B. acetabulum (ventral sucker).

Two or more hosts involve in the life cycle, but few is monecious (self-fertilized)

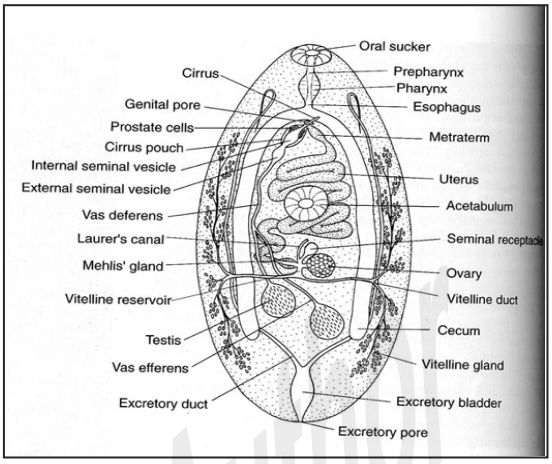


Figure 8. Typical structure of Digenia

Types of adult flukes according to the sucker as name and position:

1- distome (most common; oral sucker and ventral acetabulum).

2- amphistome (oral sucker; posterior sucker, usually posterior to testes).

3-monostome (oral sucker only).

4-gasterostome (mouth in center of oral, ventral sucker).

5-holostome (forebody and hindbody; tribocytic organ posterior to acetabulum).

6-echinostome (collar of spines around anterior sucker).

Tegument consist of two zones:

1. outer cytoplasmic syncytium provided with microvilli [mitochondria, ER, vacuoles, lipids, etc.] [pinocytosis; spines]

2. inner area of nucleated cell bodies – cytons.

3. zones separated by basal lamina.

4. circular & longitudinal muscle under basal lamina.

Numerous chemicals in the tegument:

- mucopolysaccharides inhibit host digestive enzymes.
- acid & alkaline phosphatases, esterases, and aminopeptidases for digestion.

Alimentary tract consists of:

- incomplete gut.
- pharynx, when present, masticates food.
- esophagus leads to 2 blind cecae.
- entire gut secretion by cells along gut; proteases, lipases.
- in cecae, absorption process.

*** Belongings species usually feed on blood, mucus, and epithelium cells.**

Reproductive system consists of:

• Male reproductive system consists of: usually two testes; taxonomic importance, vas efferens - vas deferens - cirrus pouch, cirrus pouch encloses seminal vesicle, prostate glands, and cirrus; some with external seminal vesicle outside of pouch, sperm stored in seminal vesicle and prostate secretes fluid to keep sperm alive.

• Female reproductive system (figure 9) consists of: usually single ovary, 2ndry oocytes released, through short oviduct, into ootype, 3 organs enter into ootype: Mehlis gland, cluster of unicellular glands, enhance egg tanning by maintaining correct pH. Different cell types in gland, secretions cause release of shell globules from vitelline glands, secretes first membrane around egg into lubricates uterus and activates sperm, which are passed down ootype. Common vitelline duct (passage of vitelline gland secretions for eggshell formation), Duct from seminal receptacle (absent in some species), Occasionally a fourth duct, vitelline reservoir, as diverticulum of vitelline duct.

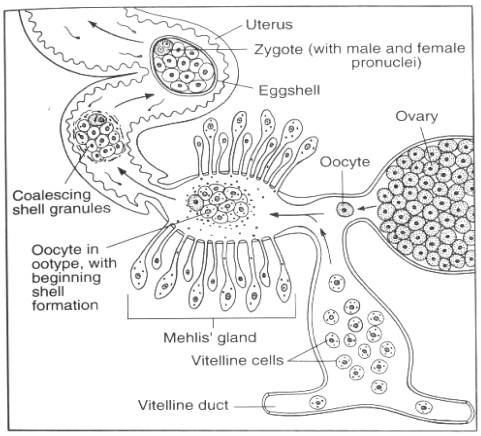


Figure 7. Structure of female reproductive system

Excretory system consists of:

- protonephridia (flame cells).
- excretory bladder in posterior, with excretory pore.

Development:

• Egg: usually operculate, lid pops off during hatching (in Schistosomes, no lid; shell splits).

• Miricidium: hatches from egg and then penetrates mollusc (rarely annelids), and develop as asexual stages from miricidium, with morphologic characteristics: apical stylet, apical papilla [where ducts from glands open; also nerve endings for chemoreception], apical gland [histolytic enzymes], cephalic glands [lytic enzymes], photoreceptors [eyespots], germinal mass [initiate asexual stages], cilia [locomotion], excretory pore, actively swim, chemoreception to snail mucus, attaches to snail with apical papillae; lytic enzymes dissolve tissues, and takes 30 minutes for penetration (figure 10).

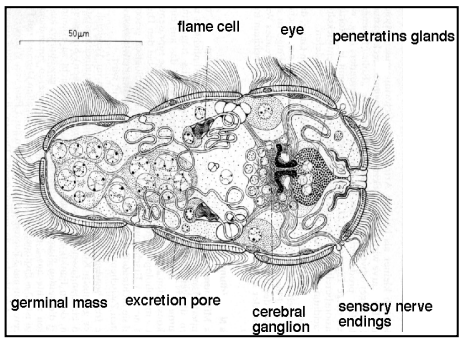


Figure 8. Typical structure of miracidium in Digenia

- Sporocyst: it is asexual stage with various shapes but does not have a mouth or digestive system, absorbs nutrients through tegument, may produce rediae, daughter sporocysts, or cercaria (figure 11).

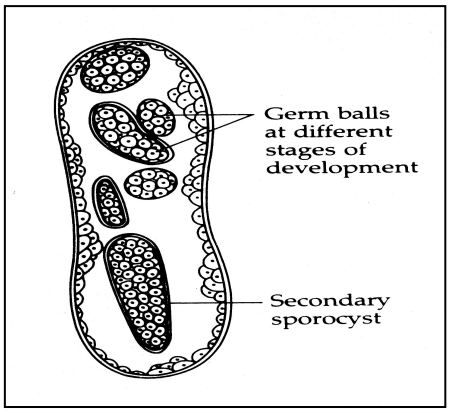


Figure 9. Typical structure of sporocyst in Digenia

- Redia: can develop directly from miricidium, or from embryos generated from sporocysts, elongate; crawl actively, muscular pharynx, mouth, blind-ended sac caecum, 1-more ambulatory buds for movement often, because ingestion, many species cause extensive damage to host, will form daughter rediae or cercaria (figure 12).

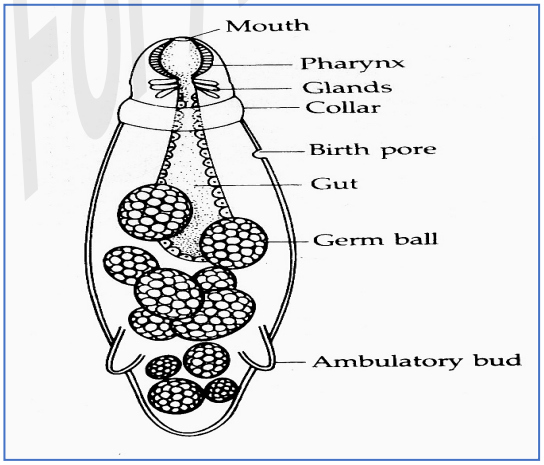


Figure 10. Redia stage of Digenia (structure)

- Cercaria: emerged from redia or sporocyst, free-swimming, leaves snail and seeks host, miniature immature fluke with tail, penetrate skin of definitive host, encyst on vegetation as metacercaria, encyst as metacercaria in intermediate host

or eaten by intermediate or definitive host or mesocercaria (encysted juvenile) in tissues (figure 13).

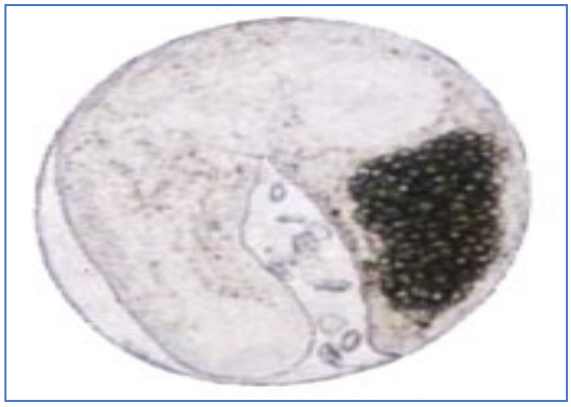


Figure 11. Morphology of Metacercaria of Digenia

Classification based on sucker placement of cercaria

- **monostome**

a. 1 sucker only, anterior
b. 2 eyespots
c. long, simple tails
d. develop from redia
e. give rise to monostome adults

- **amphistome**

f. posterior sucker, often anterior too
g. eyespots
h. develop from redia
i. give rise to amphistome adults
j. all in superfamily: Paramphistomoidea

- **gasterostome**

k. mouth ventral
l. develop into gasterostome adults
m. all in family: Bucephalidae

- **distome**

n. 2 suckers, one oral and one ventral

o. most common type

Additional classification, based on cercarial tail shape and other structures:

- pleurolophocercous
- furcocercous
- echinostome
- xiphidiocercariae
- ophthalmocercariae (with eyespots)

Trematoda is divided according to the site of infection in the definitive host:

1- Liver Flukes

2- Intestine Flukes

3- Lung Flukes

4- Blood Flukes

Liver Flukes

Family: Fasciolidae, with two important species ***Fasciola hepatica*** and ***Fasciola gigentica***

Phylum: Platyhelminthes

Class: Trematodes

Subclass: Digenea

Order: Echinostomatiformes

Family: Fasciolida

Genus: *Fasciola*

Fasciola hepatica

Characteristics

1. large, leaf-shaped, with cephalic cone.
2. present mainly in herbivores.

3. resides in the intestinal cecae, testes, and ovary dendritic.

Intermediate host: fresh water snails

Final host: Cattle

Site of Infection: hepatic and portal vessels

Life cycle: The adult worms have a proclivity to infect the bile ducts, eggs expelled with feces, and the gall bladder (figure 14). Unembryonated can develop over the course of two weeks, hatch into the miricidia stage, and then puncture a snail. A week after infection, cercaria appear, and encyst as metacercariae underwater vegetation. The sporocyst has two redial generations. When consumed by different host, they pass through the gut and enter the liver (bile's glycocholic acid serves as a trigger for migration), feeding there for about two months before moving to the bile ducts, where they develop and begin to lay eggs after another month.

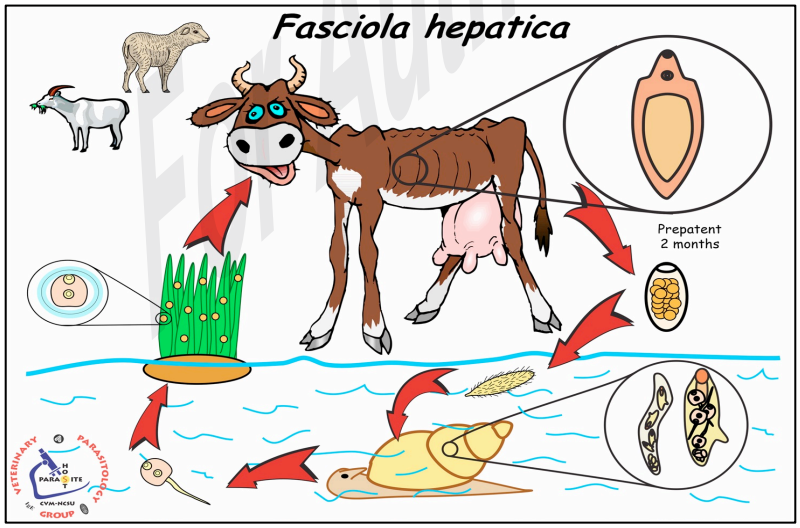


Figure 12. Life cycle of *Fasciola hepatica*

Pathogenesis

A. inflammation and erosion of the bile ducts

B. destruction tissue of the liver

C. fibrosis of liver and bile duct walls

D. cirrhosis and jaundice

E. blockage of bile ducts

F. abscesses in liver

G. migrating juveniles may cause ectopic abscesses in lungs, brain, skin, eye

H. halzoun - adults attach in nasopharynx after eating raw liver.

Symptoms:

- **Acute**
  - More common in sheep
  - 10,000+ Metacercariea consumed at one time
  - Dramatic Liver Inflammation, Frequently Resulting in Death
- **Chronic**
  - More Common and Rarely Fatal
  - Nonspecific Symptoms
- **Halzoun**
  - Eating raw, infected liver
  - Infects pharynx
  - Causes swelling and obstructs breathing

Fasciola gigantica

widely distributed in Africa, India, portions of Europe, Indonesia, Asia, and Hawaii. The most common infections occur in cattle, sheep, and goats. Patent infections occurs humans). Hybrids between this species and *Fasciola hepatica* has been often reported (figure 15,16,17).

Other Fasciola species include:

Fasciola jacksoni (nasty pathology in Asian elephants)

Fasciolopsis buski (swine and humans in Asia).

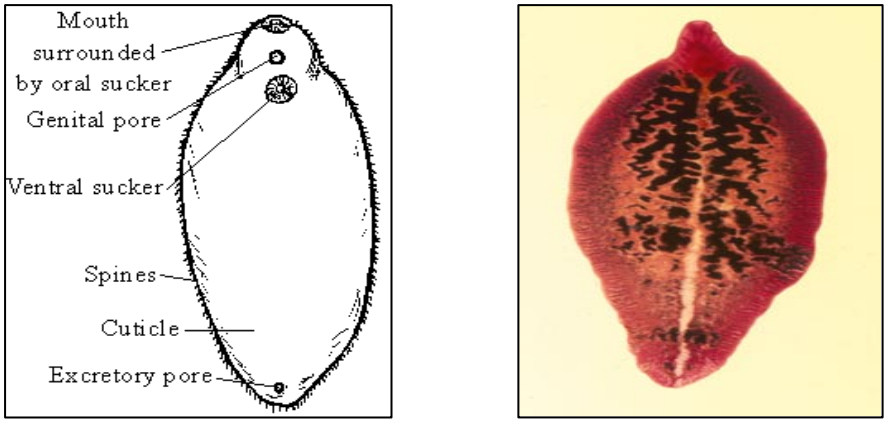


Figure 13. *Fasciola gigantica* (unstained and stain) worms.

Both left & right

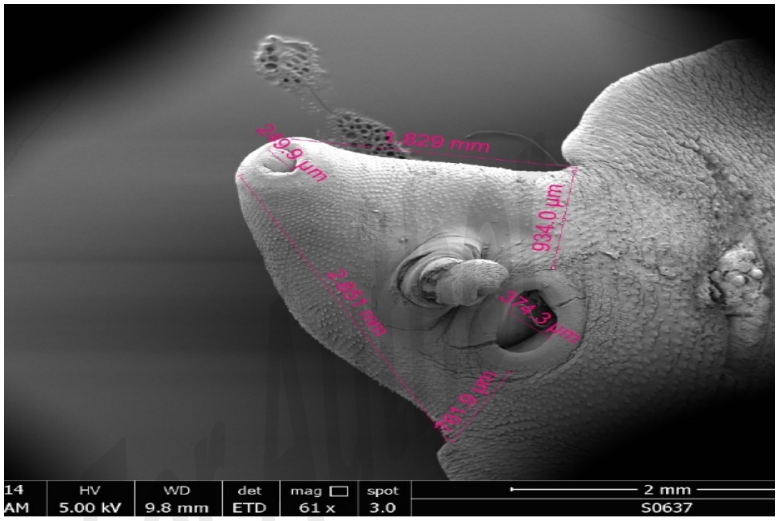


Figure 14. The apical zone of *Fasciola gigantica* with oral and ventral suckers and genital pore (61 X). By scanning electron Microscope.

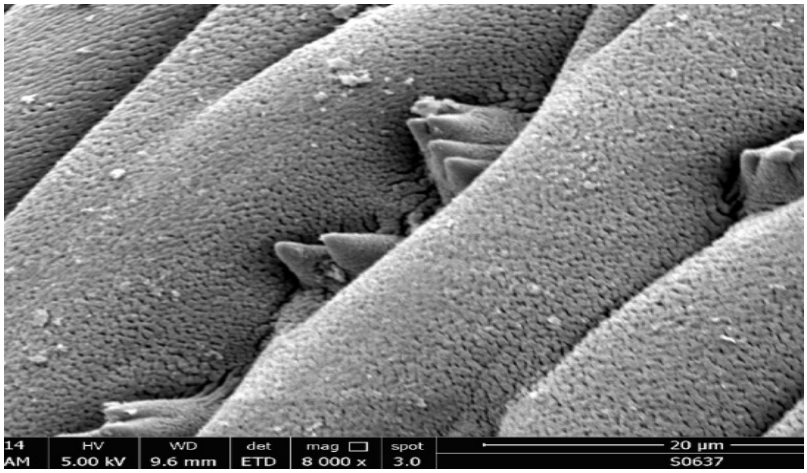


Figure 15. Spiny tegument of the cirrus genital organ of *Fasciola gigantica* (8000 X). By scanning electron Microscope.

Treatment options

- **Bithional**
  - Highly Effective
  - Large Dose
  - High Cost
  - Long Treatment Period
- **Triclabendazole**
  - Easier to Use
  - 1-2 Oral Doses in 24 hours
  - Virtually 100% Effective
- **Surgery option**

Order: Plagiorchiformes

Adults are quite diverse and many do not resemble one another. Larvae and juveniles share more similarities comparing with adults. Wall of excretory bladder epithelial. Cercaria with simple tail and dorsal fin fold. Oral stylet usually present (xiphidiocercariae). Most species have small eggs and eaten by snail to hatch. It is tended to be medium to small worms.

Phylum: Platyhelminthes

Class: Trematodes

Subclass: Digenea

Order: Plagiorchiforms

Family: Dicrocoeliidae

Genus: *Dicrocoelium*

Species: *D. dendriticum*

Dicrocoelium dendriticum

The disease caused by *Dicrocoelium dendriticum* **called "Dicrocoeliasis"**

Intermediate host: First: land snails, Second: Ants

Final host: Cattle

Site of infection: portal and hepatic vessels

Synonyms are referred as *Dicrocolium lancelatum* and *Distoma dendraticum*. Medium sized, elongate, and flattened worms ("lancet fluke") (ca 6-10 x 1.5-2.5 mm), body pointed at both ends, and cecae simple; ovary post-testicular (figure 18). Common existence in the Europe, Asia and it was thought to be introduced into Australia and North America.

Figure 16. *Dicrocoelium dendriticum*
(Adult worm)

Life cycle of *Dicrocoelium dendriticum*

The adult worms infect the bile ducts, gall bladder, and pancreatic ducts of ruminants, rodents, and rarely humans. Embryonated eggs passed out with feces, and eaten by non-aquatic snails (figure 19). Around 55 different species of snails are a fit host. The eggs hatch into two sporocyst generations and subsequently to xiphidiocercaria that accumulate in pulmonary chamber of snail, where cercaria aggregate as masses and secrete thin cyst wall. Snail then coats cercaria with mucus, and deposits slime balls which containing hundreds of cercaria in slime trails, this slime balls eaten by ants. Several genera of ants were identified as the intermediate hosts (*Formica fusca* in America). Ants feed on slime balls and for their larvae. Most of the metacercaria encyst in

hemocoel and are infective to final host; however, 1-2 encyst in depression between roots of subesophageal nerves leading to mouthparts (never become infective), as temperature decreases in the evening, ants climb up grass and clamp down with mandibles. Paralyzed ants found only at temperatures under 20 C^0, mandibles release when ants warm up day, so, ruminants graze in evenings and mornings, ingesting these exposed ants, metacercaria excyst in duodenum, migrates up common bile duct and development to adult.

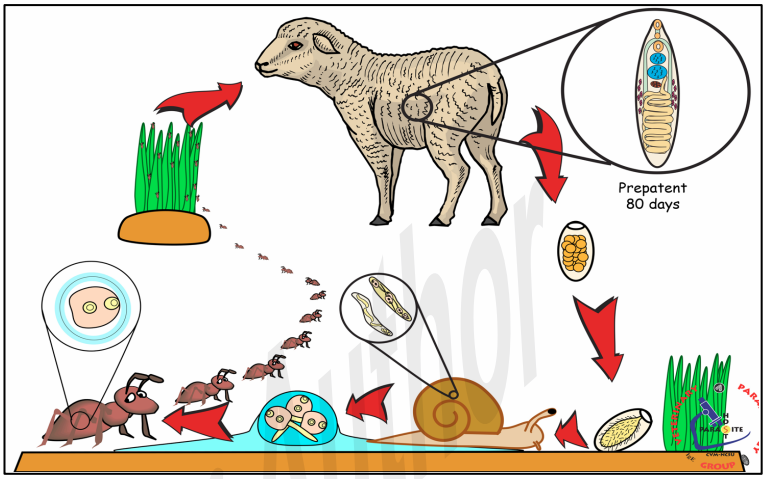


Figure 17. Life cycle of *Dicrocoelium dendriticum*

Treatment:

1- Hexachloroethan and Fouadein, which cutoff ova production from adult worms.

2- Hetholein (19-22) mg/Kg. body weight

3- Thibendazol (200-300) mg/kg body weight which can kill 96%from adult worms.

Order: Opisthorchiformes

Typically: worm has medium to small size, testes usually posterior, cirrus absent and seminal receptacle present. Eggs passed fully embryonated, while, metacercariae encysted in the tissue of fish.

Phylum: Platyhelminthes

Class: Trematodes

Subclass: Digenea

Order: Opisthorchiforms

Family: Opisthorichidae

Genus: *Opisthorichis*

***Clonorchis sinensis* (syn. *Opisthorichis sinensis*)**

The disease caused by this species called "Opisthorchiosis"

Intermediate host: First: snails, Second: fishes

Final host: Human

Adults of the Chinese liver fluke live in bile ducts, are elongate, 8-20 x 1.5-5 mm. Asiatic in distribution, infecting cats, humans, dogs, badgers, mink, etc. Large, dendritic testes posteriorly that are tandem.

Life cycle

Adults infect in bile ducts, produce up to 4000 eggs per day; live about 6 months, eggs passed in feces fully embryonated, eaten by snails (most common, *Parafossarulusmanchouricus*) one sporocyst and one redial generation, cercaria with eyespots; when contacts solid object swims upward, attaches to fish epithelium; over 100 species of cyprinids suitable, enters through skin, encysts under scales or in muscle as metacercaria. Some crustacea will also support metacercaria after that fish eaten, so, metacercaria excyst; migrate to bile duct. Pathological changes, includes erosion of the biliary epithelium. There is also evidence to suggest that this parasite is probably carcinogenic factor (figure 20).

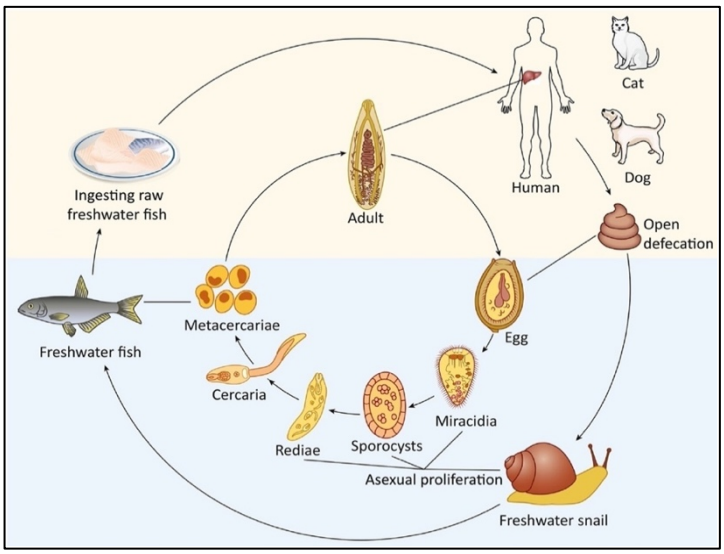


Figure 18. Life cycle of *Clonorchis sinensis*

***Opisthorchis felineus* (syn. *Opisthorchis tenuicollis*)**

It is found in Europe and Asia and a variety of mammals serve as hosts especially felids, other carnivores, and humans.

Opisthorchis viverrini

Found in southeast Asia was reported to infect up to 10 million humans. Some evidence suggests that this parasite can induce cholangiocarcinoma in humans.

Intestinal Flukes

1- Family: Echinostomatidae: *Echinostoma* spp.

Phylum: Platyhelminthes

Class: Trematodes

Subclass: Digenea

Order: Echinostomatiformes

Family: Echinostomatidae

Genus: *Echinostoma*

Echinostoma revolotum

Intermediate host: First: snails, Second: Fishes, tadpoles, and Planaria

Final host: Birds

Site of Infection: Intestine

Tend to be relatively non-host specific in semi-aquatic vertebrates. Many of their species elongate; anterior sucker and large acetabulum anterior, 27-51 circumoral collar of spines, depending upon species (figure 21)

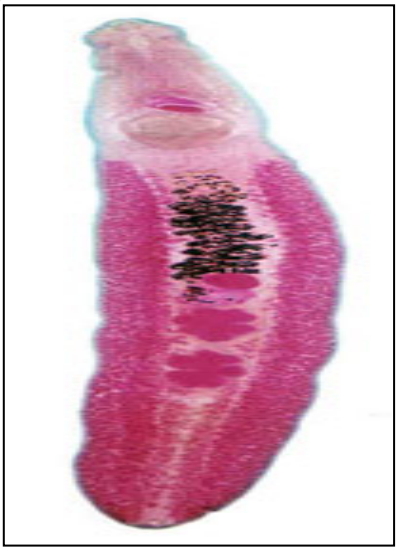


Figure 19. Adult worm
Echinostoma spp.

Life cycle

Adults reside in the gut, eggs passed in feces, hatch; miricidia penetrate snails, for instance, sporocyst; two redia, cercaria, metacercaria in molluscs, planaria, fish, tadpoles, etc. later one is eaten by definitive host.

Typical species

A. ***Echinostoma caproni*** (mammalian and avian hosts; Africa)

B. ***Echinostoma trivolvus*** (mammalian and avian hosts; North America)

C. ***Echinostoma revolutum*** (mammals and birds; Europe and Asia)

Order: Paramphistomiformes

This order often placed as a superfamily of the order Echinostomatiformes. It composed of amphistomes (acetabulum at or near posterior end). The worm is thick and fleshy, and ovary is post-testicular. Moreover, this species is cosmopolitan in distribution. Adults reside in the rumen of cattle, sheep and goats.

2-Family: Paramphistomidae

Phylum: Platyhelminthes

Class: Trematodes

Subclass: Digenea

Order: Paramphistomatiforms

Family: Paramphistomidae

Genus: *Paramphistomum* sp.

Paramphistomum cervi

Life cycle

Adults dominate in the rumen (figure 22); eggs are expelled with faeces and develop into miracidium after hatching in water. One sporocyst with two redial generations, cercaria encyst on aquatic vegetation, consumed by herbivores, excyst in duodenum, penetrate stomach, migrate through tissues to abomasum,

enter lumen, move anteriorly to rumen, and mature in 2-4 months. It penetrates multiple genera of snail (figure 23).

Pathogenesis: present in great numbers causes intestinal ulceration and probably death when it is combined with bacterial infections.

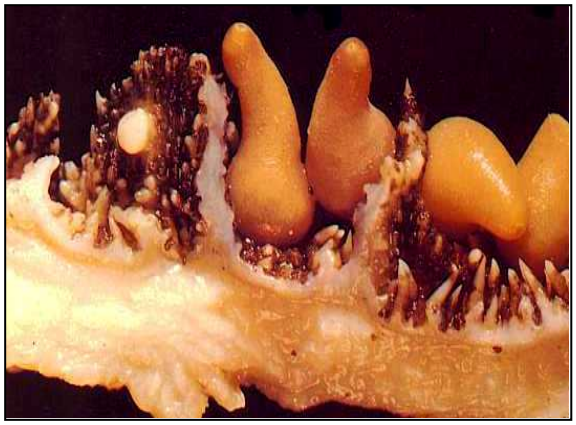


Figure 20. Adult worm *Paramphistomum cervi* attach in ruminant of the host

Figure 21. Intermediate host (snail) in the life cycle of *Paramphistomum cervi*

Treatment

Hexachloroethan or Pentonine can be used for eliminate the infected animals giving 180 gram per Kg of body weight. Mollescicide is used as a chemical to control snail populations.

Phylum: Platyhelminthes

Class: Trematodes

Subclass: Digenea

Order: Opisthorchiforms

Family: Heterophyidae

Genus: *Heterophyes*

Heterophyes heterophyes

Description: The diagnostic features of the adult worm have three suckers (interior, posterior and genital), with rough spiny tegument. Furthermore, adult

worm is the smallest trematodes in size (2 mm in length). While eggs are oval, yellowish, has a conical operculum, and thick shell. Further information displayed in the table 1.

Geographical distribution: The parasite occurs in fish and eaten by mammals (figure 24). It more commonly distributed in Asia and North Africa; utilize brackish water fish such as mullet for second intermediate hosts.

Pathogenesis: Presence of flukes in human intestine initiate intestinal mucosa inflammation, most of cases are asymptomatic, if symptoms appear, abdominal pain and watery diarrhoea occurs.

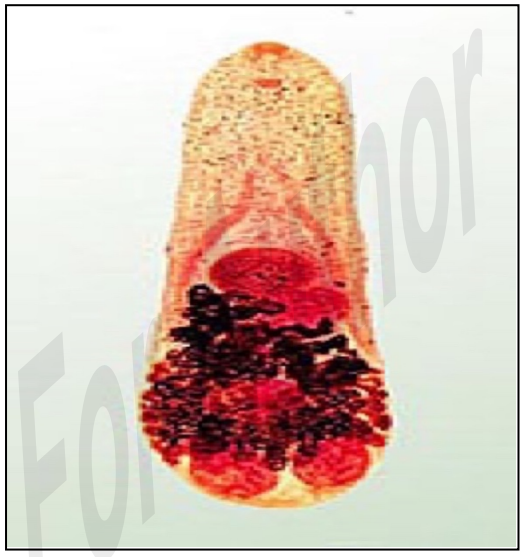


Figure 22. *Heterophyes heterophyes*

Table 1. Characteristics, host and distribution of *Heterophyes heterophyes*.

Name	***Heterophyes heterophyes***
Definition	Fish tape worm (intestinal flukes)
Distribution	Egypt, Far East, Middle East & Africa. (For Egypt it found near to Port said & Abu Rawash)
Disease	Heterophiasis
Diagnostic Stage	Eggs in stool (yellowish oval eggs containing mature miracidium)
Infective Stage	Encysted metacercaria in fish muscles.

Mode of infection	Eating raw or undercooked fish, mainly boulty & bory containing encysted metacercaria in their muscles
Treatment	Praziquantel (Distocide, Biltricide) & Niclosamide (Niclosan, Yomesan)
Prevention	Proper disposal of human sewage

Metagonimus yokogawii

Present in fish eating mammals in Asia; utilizes freshwater trout, other salmonids, and cyprinids, for second intermediate hosts (figure 25). The adult which measured 1-2.5 X 0.4-0.7 millimeter, the whole body is covered with spiny cuticle, ventral sucker at right side of the body. Egg has an operculum and miracidum inside egg shell (measured 27-30 X 15-17 µm). In the figure 26, the samples were collected from dog's intestine from Basrah city/ southern Iraq in 2004 by Prof. Suzan Al-Azizz, which represented a first record of this parasite in Iraq and Basrah city.

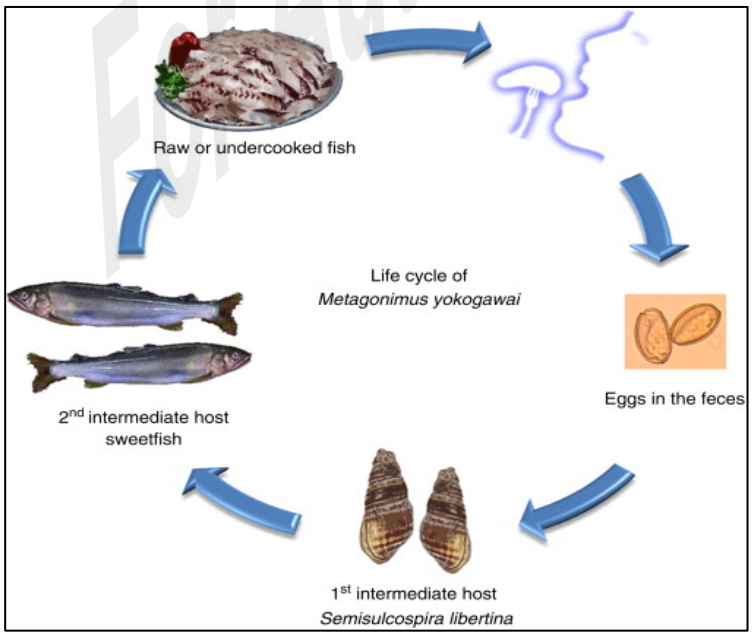
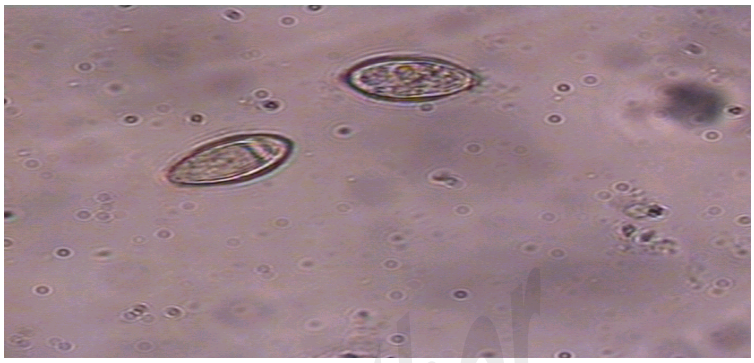


Figure 23. Life cycle of *Metagonimus yokogawei*

A



B

Figure 24. A-Adult Metagonimus *yokogawei* isolated from dog's intestine, B- Ova with miricidium inside

Lung Flukes

Family: Troglotrematidae

Paragonimus westermani

It is a human lung fluke and was found in India and Philippines. Nearly 50 known species in the genus; this particular species is commonly found in the North America. The adult worm is large and fleshy, occurs in the lungs. Canines, pigs, felids, raccoons, goats, muskrats, opossum are hosts for this parasite (figure 27).

Definitive host becomes infected by eating uncooked crustacean. Adult infections become established in the lungs but larval forms may wander into brain, pleura, mesentery or other organs (ectopic infection). Reservoir hosts include dogs, cats, pigs, rodents.

Man becomes infected by eating improperly cooked crabs, ingestion of metacercaria from cutting boards where salads are fixed, medicinal use of crab juices).

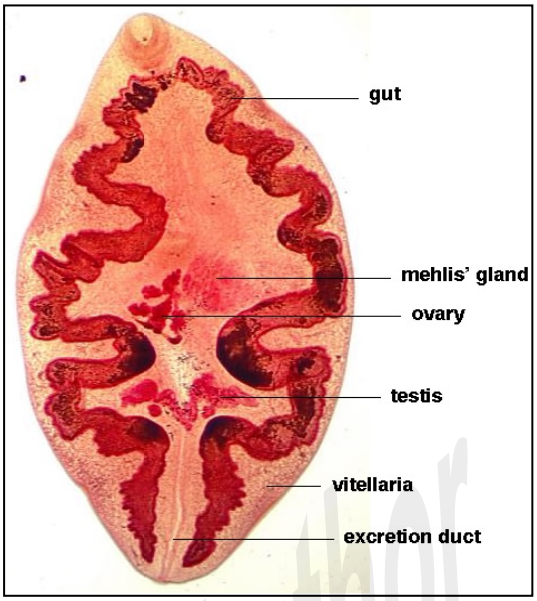


Figure 25. *Paragonimus westerman*

Pathogenesis

• Early invasive stages usually asymptomatic.

• In the lung or ectopic site, connective tissue forms pseudo-tubercules. In the CNS, they can cause paralysis and in rare cases can be fatal. In the heart they can cause severe damage and can be fatal.

• Lung infections cause chronic cough, bloody sputum, and pneumonia

Prevention and control

– Cooking of crabs, crayfish

– Care when eating salads, no crab juice.

– Proper disposal of feces and sputum.

Life cycle: Adults encysted as pairs in lungs, eggs up trachea; out with feces, then, mature in environment in several weeks after that hatch to miricidium which penetrates special intermediate host snail (*Pomatiopsislapidaria*), then sporocyst

and two redial are generated. Later, cercariae either emerge and penetrate crayfish (i.e. *Cambarus* spp.), or snail with cercariae are eaten by crayfish. Other *Paragonimus* species may use freshwater crabs or other crayfish species. The metacercaria in gills, muscle swallowed by a definitive host which bores through gut wall; through diaphragm and penetrates lung and directly maturation into adults (figure 28).

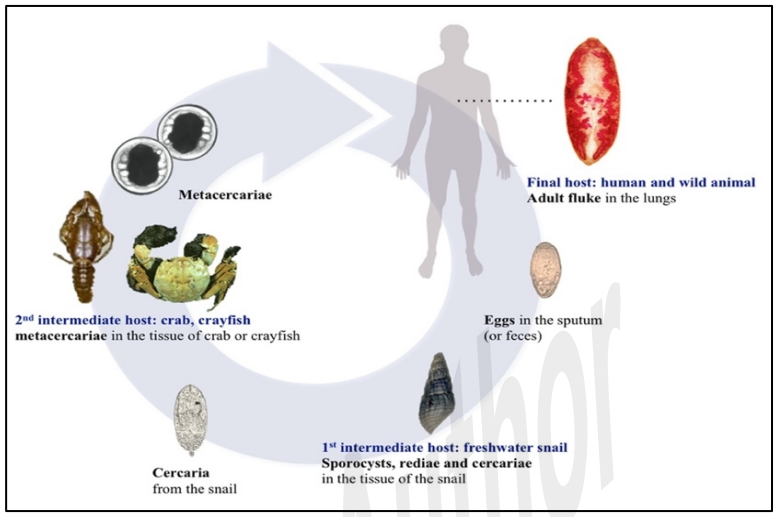


Figure 26. Life cycle of *Paragonimus westermani*

Blood Flukes

Family: Schistosomatidae:

***Schistosoma* spp.**

Elongate bodies without pharynx, in birds (many genera); mammals (3 genera), eggs non-operculate, live in blood vessels, especially mesenteric blood vessels. *Schistosoma* spp. (in mammals; 4 groups)

A. *Schistosoma haematobium* group

- 7 species.
- most use *Bulinus* snails as intermediate host.
- indigenous to Africa and adjacent regions.
- most with posterior spine on egg.

- *S. haematobium, S. intercalatum, S. mattheei* in primates. Available evidence suggests that *S. haematobium* can cause urinary bladder carcinoma.
- *S. mattheei, S. bovis, S. curassoni, S. margrebowiei, S. leiperi* in artiodactyla.

B. *Schistosoma mansoni* group

- 4 species
- most used *Biophalaria* snails as intermediate host.
- indigenous to Africa; introduced to the Caribbean and South America
- most with large, sublateral spine of egg

Species:

1. *S. mansoni* in primates and rodents
2. *S. rodhaini* in carnivores and rodents
3. *S. edwardiense, S. hippopotami* in artiodactyla

C. *Schistosoma indicum* group

- 4 species
- most species use *Indoplanorbis* snails as intermediate hosts.
- indigenous to Asian countries
- Most species have egg with terminal spine

1. *S. indicum, S. spindale, S. nasale* in artiodactyla
2. *S. incognitum* in rodents, carnivores, and artiodactyla

D. *Schistosoma japonicum* group

- 4 species
- variety of snails as intermediate hosts.
- indigenous to Asian countries
- most eggs spherical or subspherical, with small spine
- *S. japonicum* in primates, rodents, and carnivores. Evidence suggests that this parasite may cause hepatic carcinoma.

1. *S. mekongi* in primates and carnivores

2. *S. sinensium, S. malayensis* in rodents

Life cycle of *Schistosoma* spp.

Adults occur in the veins of the visceral region; female inch down into venioles to release eggs, eggs trapped in capillaries; like granuloma; passed out with feces or urine or remain trapped, next embryonate eggs pushed out and then hatch to miracidia. The miricidium larvae penetrates a snail and two sporocyst released. Subsequently, furcocercouscercariae excreted and penetrate skin of the definitive host resulting from schistosomule which can migrates through blood vessels to heart and liver. Maturation to adult takes three weeks.

Pathogenesis and immunity response

- adults evade immune system by coating themselves with host proteins
- adults cause little damage
- most pathology associated with eggs; many carried to exotic sites
- delayed type hypersensitivity around egg granulomas; leaking antigens; eosinophilia; neutrophilia
- blood vessel occlusion; fibrosis; bloody diarrhea; bloody urine; edema; ascites; cirrhosis

Other Genera and Species

- ***Schistosoma tiumdouthitti***

  1-rodents and lagomorphs in far North America

  2- hepatic portal system

- ***Heterobilharzia americanum***

1. medium sized in mammals; carnivores; in North America
2. found in raccoons

- ***Gigantobilharzia, Bilharziella, Trichobilharzia, Microbilharzia*, etc.**

  Found in birds

- ***Ornethobilharzia turcestanicum***

Characterizations (figure 29)

• Schistosome: A parasitic trematode worm contracted from infested water that is capable of causing liver, gastrointestinal tract and bladder diseases.

• Three main species of these trematode worms (flukes) - *Schistosoma mansoni*, *S. haematobium*, and *S. japonicum* that produce disease in humans.

• Schistosomiasis or bilharzia after the German physician Theodor Bilharz (1825-1862). Nickname "Bill Harris" by British soldiers serving in Europe during World War I

• Ova of *S. mansoni* with lateral spin (figure 30); Ova of *S. haematobium* with terminal spin (figure 31).

• Ova of *S. japonicum* with minute spin or knob (figure 32).

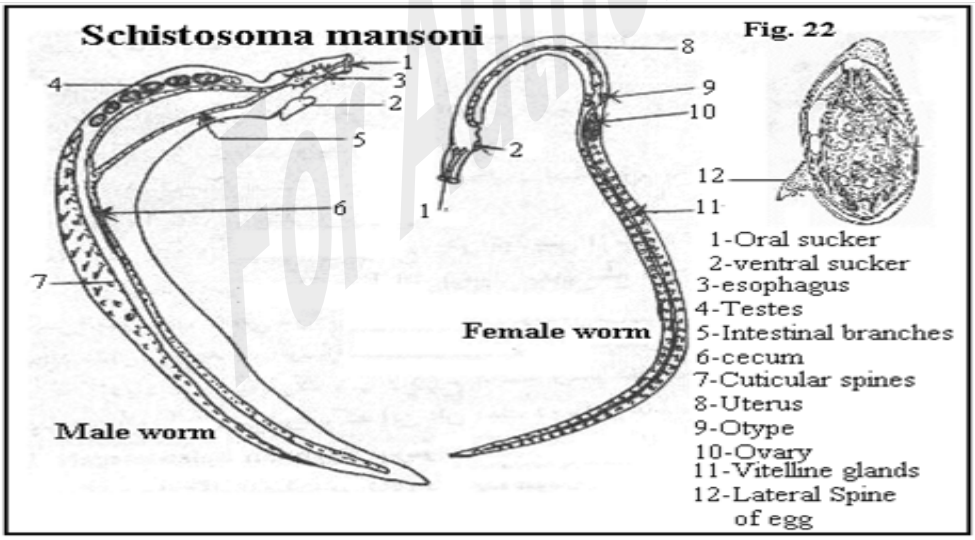


Figure 27. Typical structure of male, female and ovum of *Schistosoma mansoni*

Figure 30. ovum of *Schistosoma mansoni*

Figure 31. Ovum of *Schistosoma haematobium*

Figure 32. Ovum of *Schistosoma japonicum*

Definitive Host/ Intermediate Host

- Definitive host: Human
- Intermediate host: Snail

  Biomphalaria (S. mansoni)

  Bulinus (S. haematobium)

  Oncomelania (S.japonicum)
- Reservoirs: monkeys, rodents, cats, dogs, cattle, horses, swine, wild mammals.
- causes swimmer's itch, where cercaria penetrate skin, die, and cause inflammation in abnormal hosts (figure 33).

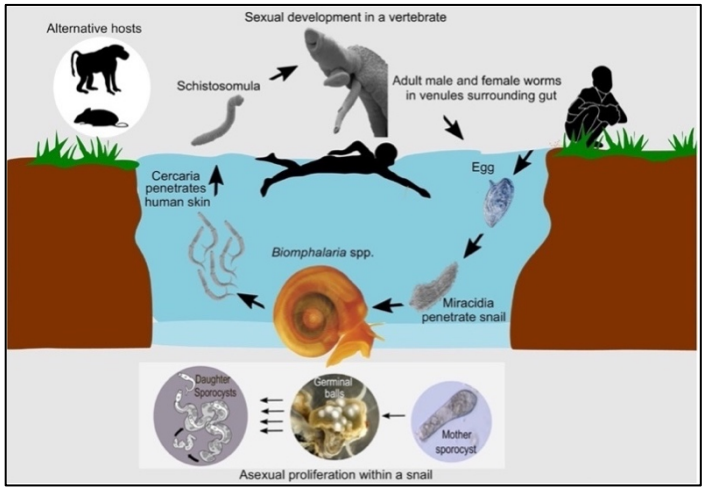


Figure 33. Life cycle of *Schistosoma* spp.

Pathogenesis

1-S. mansoni

- most pathogenic
- site of infection: veins of large intestine

2-S. haematobium

- bloody urine
- site of infection: veins of bladder

3-S. japonicum

- high morbidity
- site of infection: veins of small intestine

Clinical Symptoms

Migratory phase

- without symptoms

Acute phase (Katayama fever)

- Chills, fever, fatigue, headache, muscle aches, cough, abdominal pain, diarrhea, and eosinophilia.

Chronic phase

- Bloody diarrhea, enlargement of liver and spleen, ascites, bloody urine, bladder cancer, kidney damage, CNS lesions.

Control & Prevention

- Education of the public
- Sanitation of drinking water
- Diagnosis and treatment
- Management of the environment
- Control of the intermediate hosts (freshwater snails).

Diagnosis

- Microscopic identification of eggs in stool or urine (most practical)
- Tissue biopsy- rectal or bladder biopsy may demonstrate eggs when stool or urine examinations are negative.
- Antibody detection
- Morphologic comparison with other intestinal parasites

Treatment

- praziquantel -drug of choice, effective in the treatment of all forms of Schistosomiasis.
- Oxamniquine -used exclusively to treat intestinal schistosomiasis
- Metrifonate - effective for the treatment of urinary schistosomiasis

Figure 34. *Schistosoma* spp in the mesenteric

Ornithobilharzia turkestanicum

Orienthobilharzia spp. is a Schistosomes infection that cause Orientobilharziasis in livestock and other mammals, where they settle in the portal or intestinal veins of the infected animals causing emaciation, anemia and diarrhea. The sever infection may lead to acyesis or abortion in females and reduce thriving. The parasite is a wide spread in many countries including Russia, Mongolia, Turkey, India, Iran, China and Iraq. The figures (35, 36, 37, 38) illustrate *Ornithobilharzia turkestanicum* were diagnosed by the researchers (Ismael, Al-Azizz and Abdullah, in 2017), from the college of Veterinary Medicine/University of Basrah.

Figure 35. Oral and ventral sucker of male *Ornithobilharzia turkestanicum*

Figure 36. Gynecophoric canal of male *Ornithobilharzia turkestanicum*

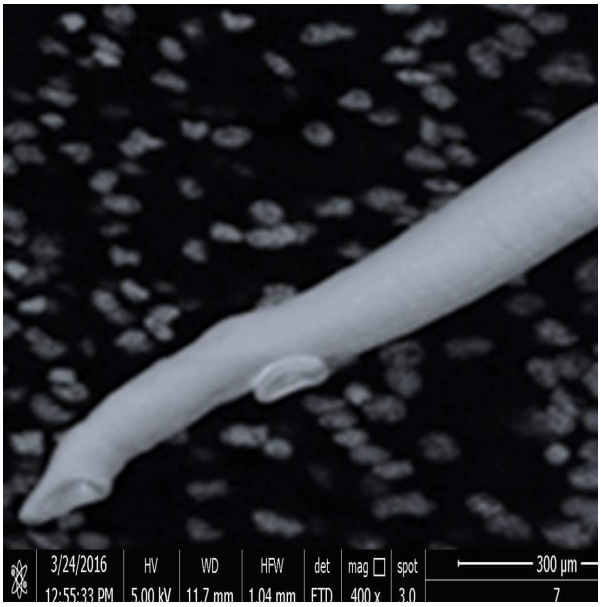


Figure 37. Adult female of *Ornithobilharzia turkestanicum*

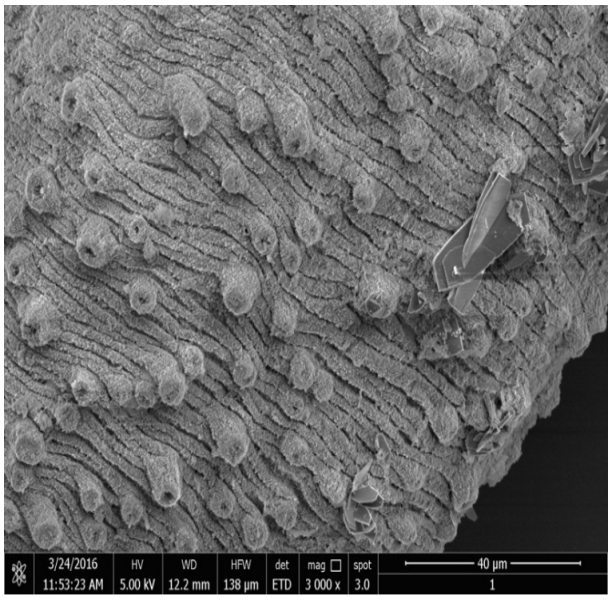


Figure 38. Spiny tegument of *Ornithobilharzia turkestanicum*

Schistosoma cercarial dermatitis or swimmer's itch (Skin rash)

Schistosomes infect both animals or humans by penetrating skin, but they are unable to establish themselves in the blood vessel system of humans, leading to sores in various parts of their bodies and the condition known as "swimmer's itch" or "digger's itch," where cercaria penetrate skin and die. This condition frequently results in dermatitis, which can be acute and, in some cases is a life-threatening due to systematic inflammation (figure 39).

Figure 39. Swimmers itch (skin sores)

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoidea

Subclass: Cestoda

Cestodes: it is known as tapeworms and mainly occupy the digestive tract of vertebrates as adults, and establish in the organs as a juvenile (figure 40).

Main features

* Tapeworms do not have a mouth like the fluke.

* Do not have a head or a digestive tract with digestive enzymes, food is absorbed through the surface tegument whose surface is enlarged by microvilli.

* The ends differ, but neither has any organs or sensors that could be associated with what is commonly thought of being a "head." However, through a segment called a scolex, they are able to absorb predigested food.

* The scolex attaches to the intestinal wall by hooks or suckers.

* The body contains hundreds of segments (proglottids), and each is a sexually complete unit that can reproduce.

* Some tapeworms have reached lengths of more than ten meters (thirty feet) with a lifespan, inside a host, of thirty years or may be more.

* Cestodaria is the unsegmented subclass of tapeworm affecting various fish and some reptiles.

* Tapeworms are dependent on two hosts for their development stages, one human and animals. Larvae stage exists in animal hosts, while the adult worm is found in the intestine of humans. However, there are two species where this development is reversed. *Echinococcus granulosis* and *E. multilocularis* differ from other tapeworm species. The adult worm infects animal hosts and the larvae form produces slow growing cysts in the viscera of humans. This condition is known as Echinococcosis or Hydatid disease, which need prolong treatment or a surgical intervention to remove the cysts from infected organs.

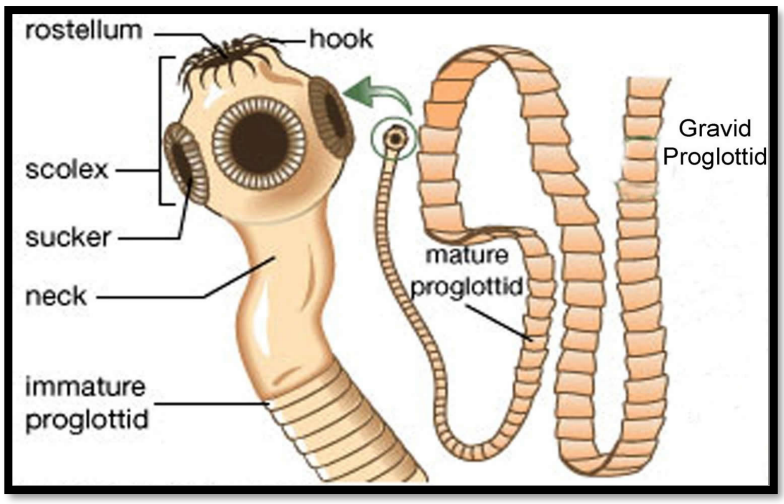


Figure 40. Morphology of cestode

The hosts

The life cycle of the tapeworm involves a definitive and intermediate host. ***Taenia solium*** and ***Taenia saginata*** are pork and beef tapeworms, respectively, whereas ***Echinococcus granulosus*** and ***E. multilocularis*** are dog tapeworms.

Canines and sheep are the definitive and intermediate hosts of *Echinococcus granulosus*. *E. multilocularis* is found in other carnivores such as dogs, wolves, and cats, their larval occur in vermin.

Diphyllobothrium latum is a fish tapeworm with two or more intermediate hosts, a copepod, fish and mammals.

Hymenolepis nana and ***H. diminuta*** are rodent tapeworms that use fleas and beetles as intermediate hosts.

Life Cycle (Basic Themes)

1- **Embryogenesis** within the egg → embryo (**oncosphere** or **hexacanth**).

2- Hatching of the **oncosphere** before or after being eaten by the next host → penetrates to extraintestinal site.

3- Metamorphosis of larva into a juvenile **metacestode** – usually with a scolex.

4- Ingestion of metacestode → development of the adult worm from the metacestode in the intestine of another host.

The adult tapeworms are found in the intestinal tracts of their definitive. Each adult tapeworm consists of a head (scolex), which attaches the tapeworm to the

intestinal wall, neck, and various numbers of segments, developing from the neck region. As new segments are formed at the neck and older segments are pushed back. Tapeworms are hermaphroditic; each segment has two sets of male and female reproductive organs, which will fill the segment with fertile eggs as the segment is pushed back from the neck. When the segment is full of eggs, it detaches itself from the adult tapeworm and passed in the feces.

Each species of tapeworm has at least one intermediate host, which ingests the tapeworm eggs. After the eggs hatch, the immature tapeworms migrate out of the intestine and move to various tissues, depending on the genus of tapeworm. The immature tapeworm enters the tissue of the intermediate host and enclosed as a cyst. The definitive hosts are infected through eating the cystic tissues of intermediate hosts contains an immature tapeworm.

Body structures

Scolex

The worm's scolex "head" attaches to the intestine of the definitive host. In some species, the scolex is dominated by bothria (tentacles), which are sometimes called "sucking grooves", and function like suction cups. Other species have hooks and suckers that aid in attachment. Cyclophyllid cestodes can be identified by the presence of four suckers on their scolex.

Proglottids

The body is composed of segments (proglottids). The sum of the proglottids is called a **Strobila**, which is thin, and resembles a strip of tape. Each proglottid contains the male and female reproductive structures.

Like some other flatworms, cestodes use flame cells (protonephridia), located in the proglottids, for excretion. Older segments are pushed toward the tip of the tail as new segments are produced by the neckpiece. By the time a segment has reached the end of the tail, only the reproductive tract is left. It then drops off, carrying the tapeworm eggs to the next host.

Cestode systems

The main nerve center of the cestode is a cerebral ganglion in the scolex. Motor and sensory innervation depends on the number and complexity of the scolex. Smaller nerves emanate from the commissures to supply the general body muscular and sensory ending. The cirrus and vagina are innervated, and sensory endings around the genital pore. Sensory function basically includes Tactoreception and Chemoreception.

Tegument

1-Syncitium: Cytons connected by trabeculae (cytoplasmic bridges) to distal cytoplasm.

2- Microtriches: Singular microtrix, are fine hair-like filaments highly specialized microvilli covering the entire surface of the tegument distributed throughout the body of cestodes.

All cestodes lack digestive and excretory systems, therefore, the tegument with microtriches constitute the principal site of absorption of nutrients and elimination of waste materials. Moreover, microtriches are the primary structures for host-parasite interface and are metabolically active performing all the vital activities e.g., sensory, absorptive and secretory functions. Thus, microtriches amplify the total surface area of the tegument.

The microtriches increase the surface area of the tegument for enhanced absorption of nutrients. They act as sensory organs for detecting the surrounding environmental cues and primary target site of anthelmintic drugs. The capacity of the tegument to absorb exogenous materials is proportional to the number and extent of pits or microtriches and the number of mitochondria in the distal cytoplasm (figure 41).

3-Glycocalyx: The surface carbohydrate complex is responsible for inhibition the host digestive enzymes, absorption of cations and bile salts, and enhancement of

the host amylase activity. The acidic glycosaminoglycans of glycocalyx are specific for inhibiting a number of host digestive enzymes.

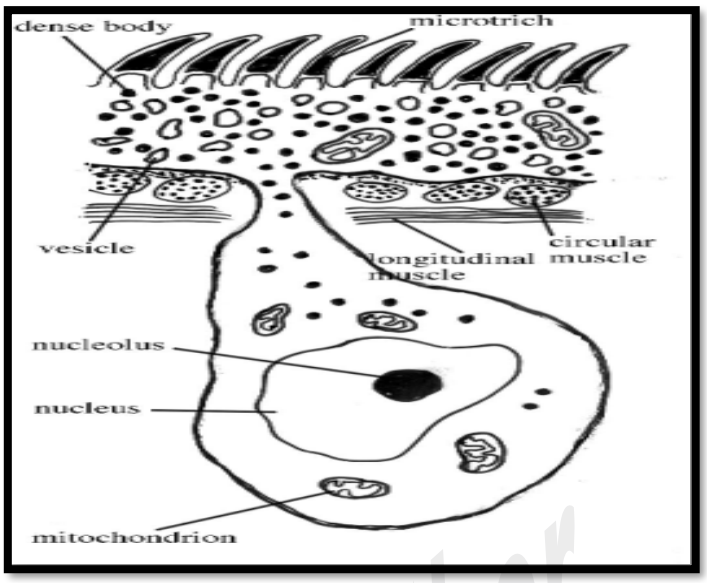


Figure 41. Section in cestode tegument

Parenchyma

The space enclosed by the tegument - except for the portion which occupied by reproductive organs, osmoregulatory structures, muscle fibers and nervous tissue- is filled with a spongy tissue known as parenchyma. Fluid fills the spaces between the parenchyma cells. Parenchyma cells are the primary sites for synthesis and storage of glycogen. There is speculation that a single population of cells, the myoblasts, gives rise to both the parenchyma and the musculature of most tapeworms.

Nervous System

The scolex contains the primary nerve center for the tapeworm (figure 42). The nerve centers consist of a complex of ganglia, commissures, motor and sensory innervations. The simpler holdfast structures associated with the scolex, the less complex the nerve center and vice versa. Longitudinal nerves extend posteriorly from the anterior ganglia. Small nerves arise from the longitudinal nerves to supply the body muscles and sensory endings. The vagina and cirrus are well-

supplied with nerves and the genital pore has more sensory endings than other parts of the strobila. Sensory endings seem to include tactoreceptors and chemoreceptors.

The cords are connected in each proglottid by cross connectives and the small motor nerves emanating from the cords and cross-connectives innervate the reproductive organs and musculature, while small sensory nerves supplying the tegument merge with the cords and connectives.

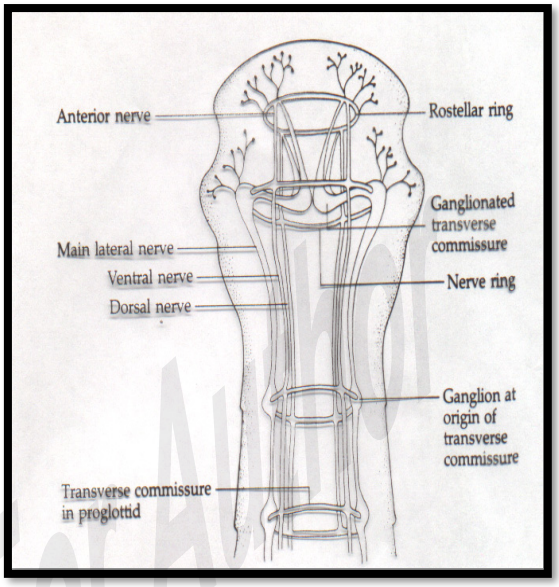


Figure 42. Nervous system of cestodes

Excretion and Osmoregulation

The cestodes have the protonephridial flame bulb system typical of the flatworms. A flame cell protonephridium and these are embedded throughout the parenchyma. The ductules of the flame cells appear to be syncytial as opposed to being formed by a single cell.

The excretory ducts are lined with microvilli suggesting where they are involved in transport, such as active transport of excretory wastes and help to regulate ionic concentrations of the excretory fluid. The excretory fluid contains glucose, soluble proteins, urea, ammonia, and lactic acid. The products of cestode are organic acids excreted through the tegument.

Reproduction of tapeworm

True tapeworms are exclusively hermaphrodites; they have both male and female reproductive systems in their bodies (figure 43). The reproductive systems include one or many testes, cirrus, vas deferens and seminal vesicle as male organs, and a single lobed or unlobed ovary with the connecting oviduct and uterus as female organs. There is a genital pore as part of reproductive systems in males and females, located at the surface opening of the cup-shaped atrium.

Even though they are sexually hermaphroditic, self-fertilization is a rare phenomenon. During copulation, the cirrus of one individual connects with that of the other through the genital pore and exchange their spermatozoa.

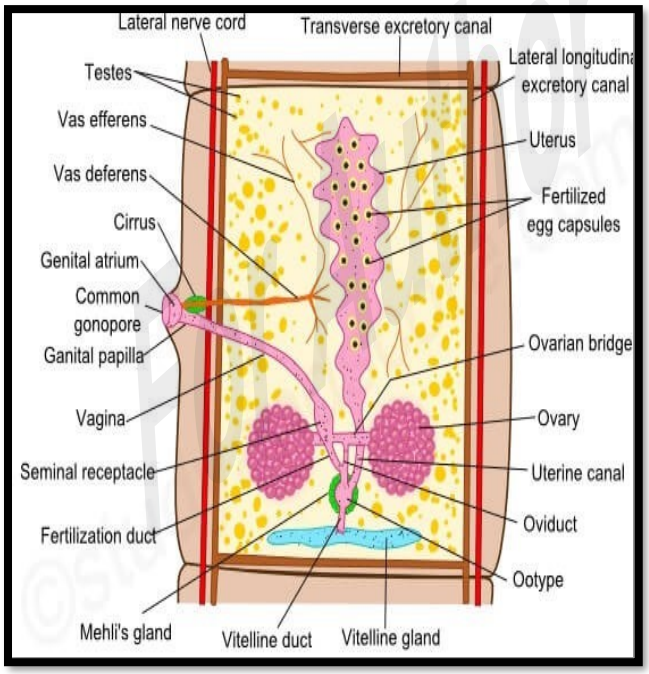


Figure 43. Reproductive system of cestodes

Female Reproductive System

Ova is produced in a single, sometimes bi-lobed ovary. Following fertilization, the resulting zygote passes into a region of the oviduct, the **ootype**, equipped with structures involved in eggshell formation.

Mehlis' gland surrounds the ootype and secretes into it material essential to the formation for eggshells. A single common **vitelline duct** enters the oviduct near to ootype. Vitelline duct is formed by the union of many **primary vitelline ducts** arising from vitelline glands.

Vitelline glands (vitellaria) may form a compact body or consist of numerous follicles scattered throughout the medullary parenchyma. The **vagina** carries sperm from the genital atrium to the oviduct, and fertilization occurs in the region where the vagina and oviduct join. Sperm is stored in the **seminal receptacle** (figure 44).

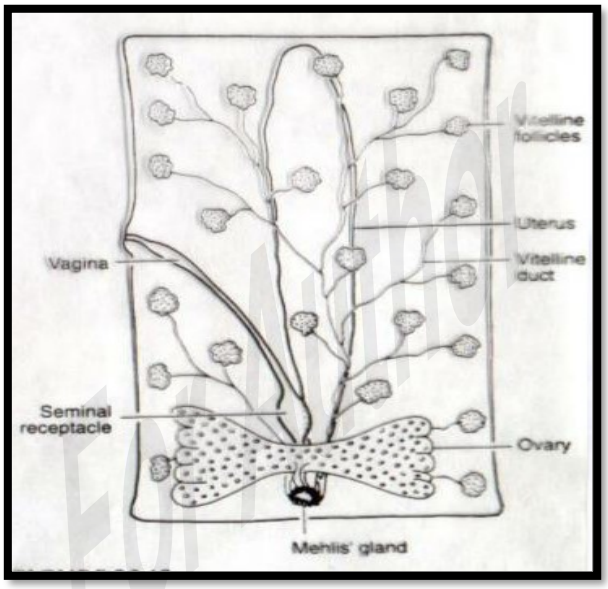


Figure 44. Female Reproductive System

Male Reproductive System

Consists of one-to-many testes embedded in the medullary parenchyma of each proglottid and emanating from each testis is a single vas efferens. Presence of multiple testes, the vas efference unite to form a common vas deferens, which is usually coiled. The distal portion of the vas deferens is modified as a muscular **cirrus**, usually enclosed within a **cirrus sac**.

In some species, the cirrus is equipped with spines assist during copulation. The cirrus everts through the male genital pore, which in turn, opens into the common

genital atrium. In most species there is an enlarged area of the vas deferens, the **seminal vesicle**, for the storage of sperm. When located within the cirrus sac, it is designated an **internal seminal vesicle**; when outside the sac, it is termed an **external seminal vesicle** (figure 45).

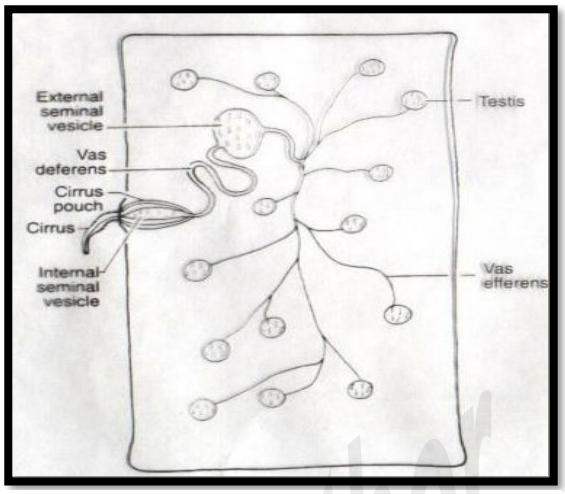


Figure 45. Male reproductive systems

The Egg: oncosphere (larvae within the egg), containing 3 pair of hooks, is encased in the envelope that is surrounded by extra membranous structure, the embryophore. A cellular zone known as the outer envelope lies between the embryophore and the shell (figure 46).

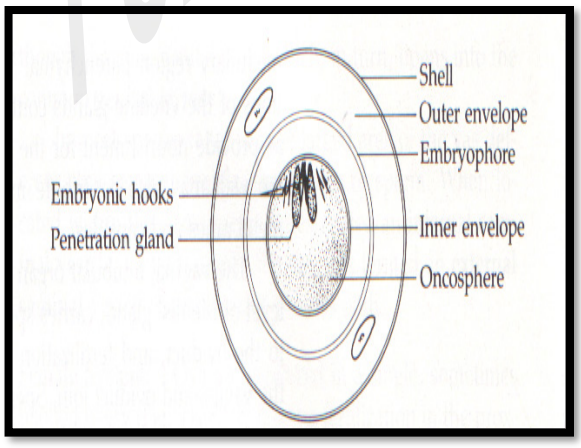


Figure 46. Egg of cestode

Clinical signs of tapeworm infection

Tapeworms cause disease in humans and animals either of the two stages of their life cycle: the adult stage develops in the intestines, where it causes some clinical manifestations. The larval stage causes signs and symptoms, presenting in various tissues of a mammalian host. Adult tapeworm is well-adapted to live in the digestive tract and cause minimal disorders.

For example, heavy infections with *Hymenolepis nana* can cause abdominal discomfort, diarrhea, and weight loss. Members of the Diphyllobothriidae cause vitamin B12 deficiency and megaloblastic anemia. In contrast to adult tapeworms, larvae can cause severe and even lethal disease when they develop in the brain, liver, lungs, eyes, muscles, and subcutaneous tissues.

In humans, *T. solium* causes cysticercosis, and *Echinococcus granulosus* and *E. multilocularis* cause cystic hydatid disease and alveolar disease, respectively. The larvae of *Spirometra* species, *Sparganum proliferum*, *Taenia multiceps, T. serialis*, *T. brauni*, and *T. glomeratus* also infect humans with forming lesions in skin or muscle, and less commonly, brain or eye depending on the species.

Treatment

The most common treatment for tapeworm infection includes oral medications that are toxic to the adult tapeworm, including:

- **Praziquantel (Biltricide)**
- **Albendazole (Albenza)**
- **Nitazoxanide (Alinia)**

The medication depends on the species of tapeworm involved and the site of the infection. These drugs target the adult tapeworm but not the eggs.

- **Surgery.** cysts can be removed surgically depends on their location and symptoms. Cysts are diagnosed in the liver, lungs and eyes should be removed, as long as threaten the function of organs.

Larvae stages description:

Embryophore: A membrane or wall around the hexacanth embryo of tapeworms, forming the inner portion of the eggshell. In the genus *Taenia*, the embryophore is exceptionally thick; in the genus *Diphyllobothrium*, the embryophore is ciliated and enhances the aquatic life cycle of this and other pseudophyllid cestodes.

Coracidium: is Ciliated first-stage aquatic embryo of pseudophyllid and other cestodes with aquatic cycles; within the ciliated embryophore is a hooked larva, the hexacanth, that develops in the intermediate host, usually an aquatic crustacean into the next larval stage, the **Procercoid** (figure 47).

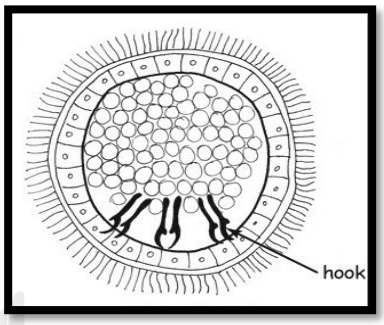


Figure 47. Coracidium

Cysticercus: The larva of certain tapeworms, parasitic found in an intermediate host, in which the head and neck are partly enclosed in a bladderlike cyst; **Bladderworm** (figure 48).

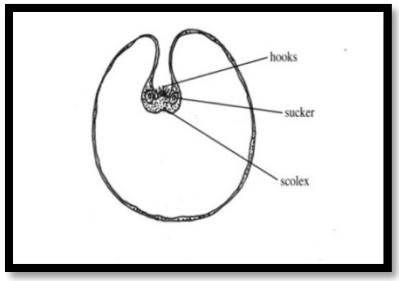


Figure 48. Cysticercus

Cysticercoid: is the larval stage of certain tapeworms, similar in appearance to a cysticercus, but having the scolex filling completely the enclosing cyst. In tapeworm infestations, cysticercoids can be seen in free form as well as enclosed by cysts in the intestinal mucosa. Also referred to as a **Metacestode** (figure 49).

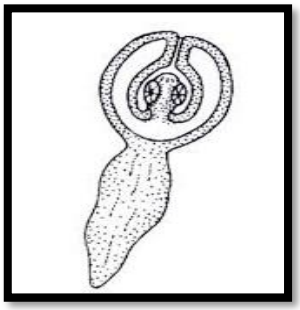


Figure 49. Cysticercoid

Coenurus:

The larval stage of tapeworms of the genus *Multiceps,* a semitransparent, fluid-filled, bladderlike organism that contains multiple scoleces attached to the inner surface of its wall and that does not form brood capsules. It develops in various parts of the host body, especially in the central nervous system (figure 50)

Figure 50. Coenurus

Tetrathyridium: A modified cysticercus resembling an elongated plerocercoid with a scolex invaginated at one end (51).

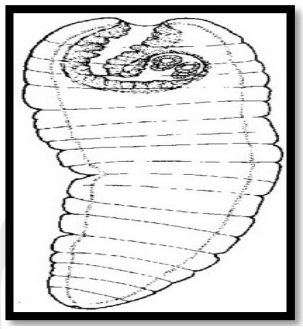


Figure 51. Tetrathyridium

***Echinococcus* and hydatids**

Any of a genus (*Echinococcus*) of tapeworms that alternate a minute adult living as a commensal in the intestine of carnivores with a hydatid larva invading tissues especially of the liver of cattle, sheep, swine, and humans and acting as a dangerous pathogen (figure 52).

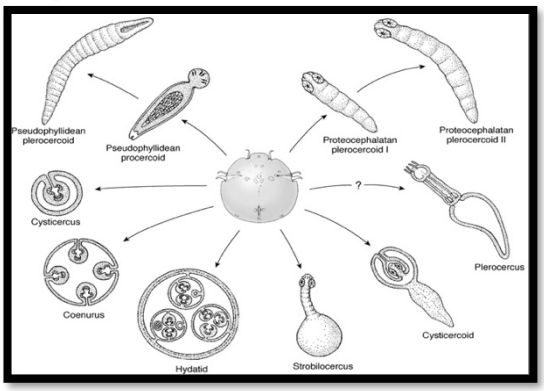


Figure 52. Different larval stages of cestodes

Eucestoda

Oncosphere has 6 hooks and tegument covered with microtriches. It consists of 13 orders. There are two human tapeworms medically important.

Order: Cyclophyllidea

Characteristics:

-Large or small worms consisting of chains of segments.

-Scolex is quadrate with four cup-like round suckers.

-An apical rostellum with hooklets may be present.

-Vitelline glands concentrated in a single mass.

-Common genital pore is marginal (on lateral side of segment).

-No uterine opening for the exit of eggs from the gravid uterus.

-Eggs only escape from the rupture or disintegration of ripe segments.

-Eggs are not operculated and can develop only in the intermediate host, fully embryonated.

-Oncosphere is never a ciliated embryo.

-Larval development proceeds in one intermediate host.

Order: Pseudophyllidea

Characteristics:

- Large worms consisting of a long chain of segments.

- "Head" has two slit-like sucking grooves called bothria instead of suckers.

-Uterine glands are widely scattered in the parenchyma and is composed of many acini.

-Genital pores are on the ventral surface of the segment and are not marginal.

-Uterus opens to the exterior through which eggs come out.

-Eggs are operculated and can develop only in water; immature when oviposited and oncosphere gives rise to ciliated embryo.

-Larval development proceeds in two intermediate hosts.

-First larval stage is called procercoid, and the Second is called plerocercoid.

Table 2. Differences between Pseudophyllidean and Cyclophyllidean.

Characteristic	Pseudophyllidean	Cyclophyllidean
Head or scolex	Bears 2 slit-like grooves	Bears 4cup-like suckers
Uterus	No branching, convoluted uterine tubes assume the form of rosettes	Branching, may or may not be present
Uterine pore	Present	Absent
Common genital pore	Ventral; in the midline	lateral
Eggs	Operculated; gives rise to ciliated larvae	Not operculated; larvae do not ciliated

Phylum: Platyhelminths

Class: Cestoda

Order: Cyclophyllidea

Family: Taeniidae

Genus: *Taenia*

- Rostellum, if present, is non-retractable.
- Mature proglottids have lateral genital pore, numerous testes, bilobed ovary, compact vitellarium.
- Diagnosis is presence of egg with thick embryophore.

***Taenia* species**

They are the most common cestode parasites of human. Both human and cattle or pigs are necessary to the complete life cycle of *Taenia* species. Eggs ingested by the intermediate hosts that contain oncospheres. The oncospheres then hatch out in the duodenum, pass into the intestine where they penetrate the intestinal wall and are then carried by the circulation to be deposited in tissues

(muscle). There they develop into cysticerci larva which are white and ovoid. Human become infected by ingesting inadequately cooked beef or pork with cysticerci, containing an invaginated protoscolex. The protoscolexes evaginate and pass into the small intestine where they attach themselves to mucosa and develop into adult worms. Both eggs and proglottids are passed out in the feces, and after that eaten by the intermediate host.

Microscopical description: Ova of *Taenia* species are spherical, yellowish brown (figure 53). The shell is thick and radially striated. Within the shell, the oncosphere has 3 pairs of hooklets. However, the microscopic appearance of the ova of *T. saginata* and *T. solium* are identical. The length of the adult *T. saginata* is 4-8 meters long and that of *T. solium* is 3-5 meters long. The proglottids of *Taenia* species can be identified by the number of uterine branches; 7-13 for *T. solium* and 15-20 for *T. saginata*. If the scolex is recovered, the four suckers and rostellum of hooklets of *T. solium* will distinguish it from *T. saginata,* which has four suckers but no hooklets. Mode of the human Infection with *T. saginata:* eating beef containing larval stage (**Cysticercus bovis**). While the mode of human infection with *T. solium*: eating measly pork containing larval stage (**Cysticercus cellulosae).**

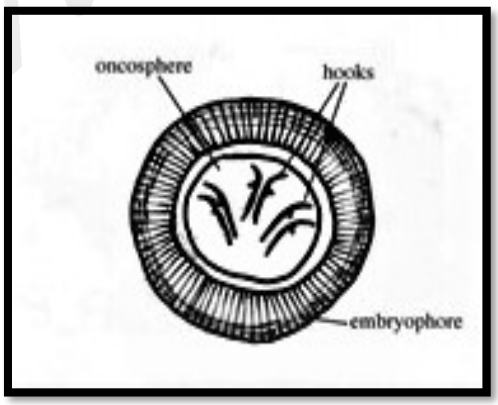


Figure 53. egg of *Taenia* spp.

Taenia saginata (Beef tapeworm)

Definitive hosts: Human

Intermediate host: Cows

Geographical distribution: Most common tapeworm found worldwide and human acquired this parasite from eating uncooked beef.

Biology: *Taenia saginata* is the beef tapeworm that is transmitted to human after the ingestion of undercooked or raw beef (figure 54). The worm can grow to a strand of 1,000 to 2,000 segments. Eggs are released individually in human feces or still within tapeworm proglottids. They are ingested by cows and hatch; the resultant larvae invade the gut and develop as small 1 cm cysts in muscle. When humans consume these cysts in raw beef, the cyst thereafter grows into an adult tapeworm and clasp in the intestine mucosa with suckers of scolex.

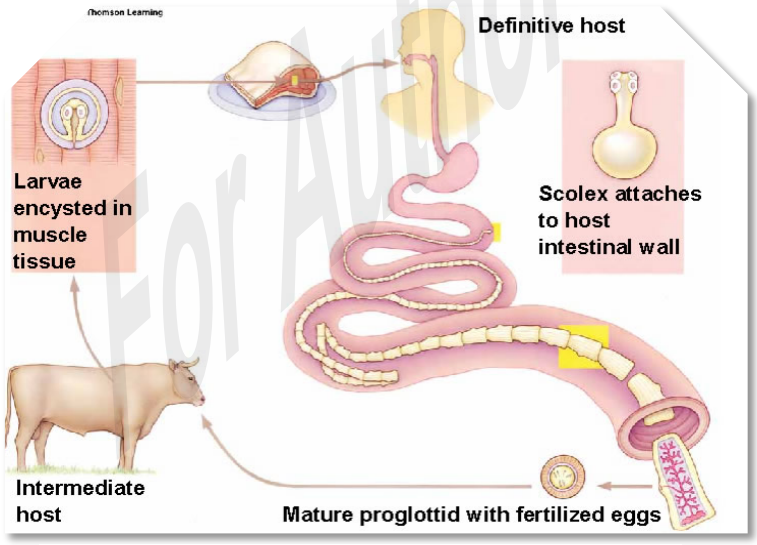


Figure 54. Life cycle of *Taenia saginta*

Clinical signs: the infection combining symptoms include diarrhea, abdominal cramping, nervousness, nausea, and loss of appetite. (proglottids) or segment chains are seen in the stools.

Diagnosis: Stool examination for eggs or segments. Segments differentiated from pork tapeworm by having large number of uterine branches examined by trans-illuminating light source.

Prevention: the transmission can be preventable by cooking or freezing beef meat.

***Taenia solium* (Cysticercosis)**

Definitive host: Human

Intermediate host: Pig

Geographical distribution: Not as widely disseminated as *T. saginata.* The infection is acquired by eating uncooked pork. The animals ingested the eggs of this tapeworm when contaminated with human stool. This occurs most frequently where human stool is used as fertilizer. This occurs most frequently in developing countries where pigs are reared intensively.

Biology: *T. solium* eggs, on ingestion, hatch into larvae which penetrate small intestine mucosa and are carried throughout the body where they are deposited and grow in muscle, subcutaneous, brain, eye, or heart (figure 55). It is small (1-3 cm cysts with an invaginated scolex) and can survive up to 7 years.

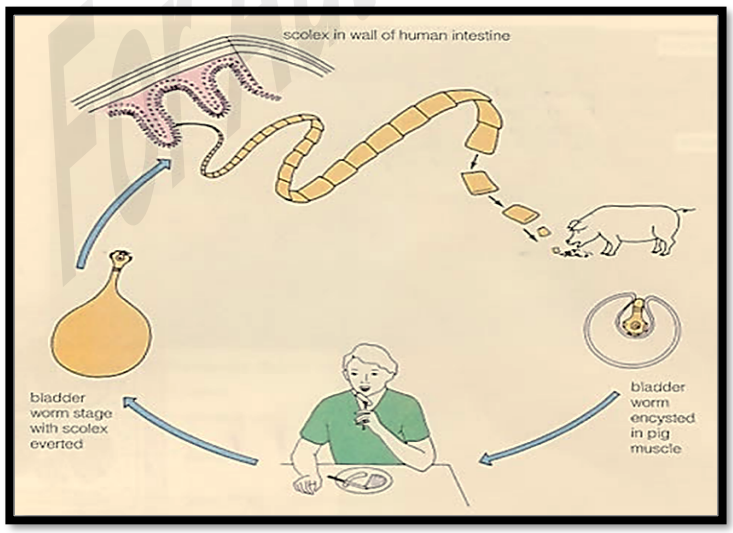


Figure 55. Life cycle of *Taenia solium*

Pathogenesis: the cysts is produced in especially brain. Foci due to inflammatory response when the cyst dies or killed with anti-helminthics. It is similar to the beef tapeworm, but shorter, having fewer than 1,000 proglottids (figure 56). When egg is ingested, the shell around the egg is dissolved in the stomach, and a

living embryo called an "oncosphere" is released. After about sixty to seventy days, these oncospheres become mature bladder worms called cysticerci or "cysts" that attach to the intestinal, using a head composed of four suckers and eight hooks. The waste of worms has toxic effects and cause intestinal obstruction. This worm can remain in a human for twenty-five to thirty years, reaching lengths of two and one-half to three meters (eight to ten feet). Infection with the adult stage of the pork tapeworm is called taeniasis, which is not a serious health threat. Eggs will appear in the stool eight to twelve weeks after eating infected pork, but eggs from a carrier can take several days to ten years to develop in another person. The infection of the brain leads to sever damage or may be lethal.

Diagnosis: diagnosis of intestinal taeniasis can be conducted by isolation ova from fecal samples. However, the ova of *T. solium* and *T. saginata* are identical and diagnosis is made by the recovery of the segments or its scolex.

The diagnosis of cysticercosis depends upon serology. MRI scans (**Magnetic resonance imaging)** may reveal the presence of lesions in the brain. Calcified cysticerci are less often seen in the brain: in about one-third of cases, 10 years or more after infection. Sometimes, the diagnosis is made histologically from tissue specimens. Calcification in muscles usually appears three to five years after initial infection and typically seen as spindle-shaped calcifications, most numerous in the thighs.

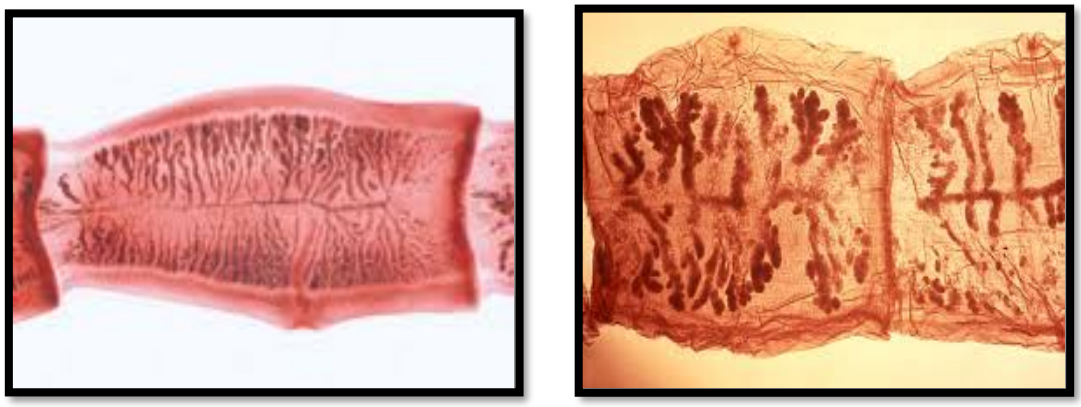


Figure 56. Proglottids of *Taenia saginata* and *Taenia solium*

Table 3. Differences between *Taenia saginata* and *Taenia solium*:

Characteristic	***T. saginata***	***T. solium***
Intermediate host	Cattle, reindeer	Pig, wild boar
Site of development	Muscle, viscera	Brain, skin, muscle
Scolex: adult worm	No hooks	Hooks
Scolex: cysticercus	No rostellum	Rostellum & hooks
Larval stage	*Cysticercus bovis*	*Cysticercus cellulosae*
Uterine branches	23 (14 - 32)	8 (7 -11)
Proglottids passing	Single, spontaneous	In groups, passively
Ovary	Two lobes	Three lobes
Vagina: Sphincter muscle	Present	Absent

Taenia ovis

Definitive hosts: Dogs, foxes, and other canids

Intermediate host: Sheep and goats

Geographical distribution: The adult stage of the parasite found in the intestines of dogs while the intermediate or larval stage (the cysticercus) can be found in the muscles of sheep. The intermediate stage in sheep is characterized by small cysts in the muscle tissue. Over time the cysts in the muscle degenerate and are no longer infective. They calcify and form a small nodule with a 'gritty' texture. However, this is the stage that is commonly known as sheep measles.

Biology: The sheep ingests the eggs of *T. ovis* (figure 57). The egg hatches in the small intestine and the larval tapeworm burrows through the intestinal wall and travel to the heart and muscles via the blood. Cysticercus can develop in the cardiac and skeletal muscles, reaching the infective stage in about 46 days. When the dog prey on infected tissue with cysticercus, the protoscolex attaches to the small intestinal wall and the worm begins to form proglottids. Gravid proglottids

that contain the eggs can detach from the end of the worm and pass out in the feces. The prepatent period is about two months.

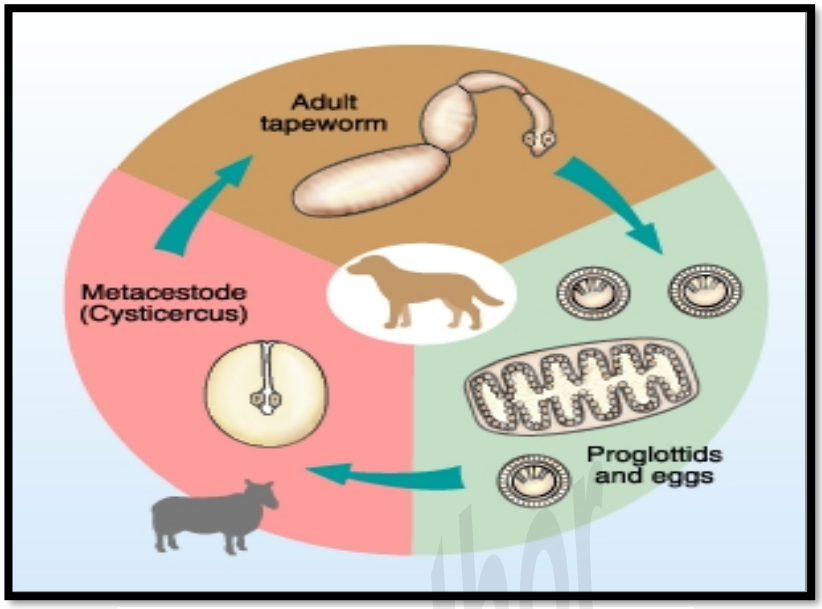


Figure 57. Life cycle of *Taenia ovis*

Pathogenicity: *T. ovis* does not present a public health risk, however these calcified cysts are unpleasant to eat and can result in carcasses being downgraded or even condemned at the abattoir.

Site in host where adult parasite is found: The small intestine.

Diagnostic Stage: Proglottid or eggs, eggs are 33 - 38 um in size.

Taenia hydatigena

Definitive host: Dog, foxes, wolves, raccoons, bears and cats.

Intermediate host: Sheep, other domestic and wild ruminants.

Geographical distribution: *Taenia hydatigena* is a cosmopolitan tapeworm in dogs and wild canids. The development of *T. hydatigena* requires two distinct hosts to complete its lifecycle. The adult parasites reside in the intestine of the definitive hosts such as dogs and other carnivores such as foxes, wolves, jackals, lynx, raccoons, bears and cats. The intermediate hosts, generally small ruminants

and, less frequently pigs, cattle, deer and other wild species, acquire the infection by ingesting eggs from contaminated pasture, often during grazing seasons.

Biology: The sheep ingests the eggs of *T. hydatigena* (figure 58). The egg hatches in the small intestine and the larval tapeworm burrows through the intestinal wall and travel to the liver via the blood. The cysticercus migrates to the liver for 18 to 30 days then burrows out into the peritoneal cavity where it attaches to the viscera. When the dog eats the sheep viscera and ingests the cysticercus, the protoscolex attaches to the small intestinal wall, and the worm begins to form proglottids. Gravid proglottids, containing the eggs, detach from the end of the worm and pass out in the feces. The prepatent period is about 51 days.

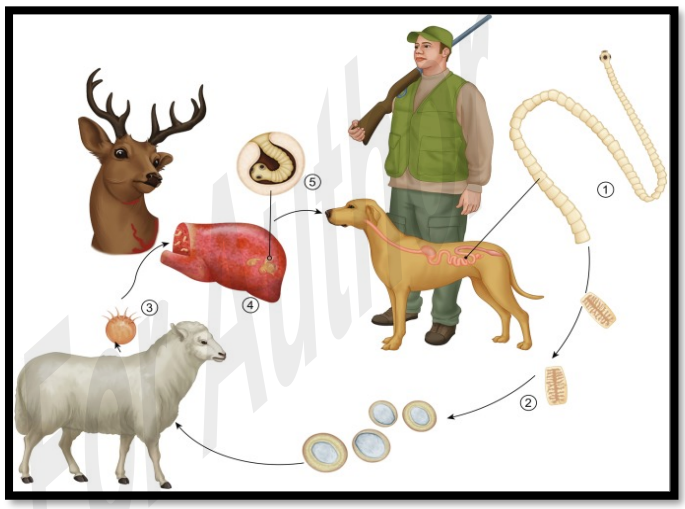


Figure 58. *Taenia hydatigena* life cycle

Pathogenesis: Acute disease occurs only with large numbers of cysticerci and is characterized by depression and weakness. Acute infection causes a liver damage. The chronic cystic stage usually is asymptomatic.

***T. hydatigena* cysticerci (Cysticercus tenuicollis):**

Usually found in the abdominal cavity. They are generally attached to the omentum, mesentery and occasionally the surface of the liver.

Site in host where adult parasite: small intestine.

Diagnostic Stage:1-Proglottid, 2- eggs are 32 - 38 um.

Taenia pisiformis

Cysticercus pisiformis (figure 59)

Definitive host: Carnivores like dogs, foxes, and sometimes of cats.

Intermediate host: Herbivorous host; rabbit or other lagomorph species may ingest the eggs while grazing contaminated grass.

Figure 59. Cysticercus pisiformis in liver of a rabbit

Geographical distribution: has been found all over the world, predominantly in rural regions. The development of the parasite occurs in two stages:

Dog is the definitive host for adult worms. The parasite lives in the small intestine (duodenum jejunum and ileum) of the dog and may reach a length of 2 meters. Mature segments of the tapeworm (protoglottid) containing mature eggs are shed along with the feces.

Biology: Transmission occurs via ingestion of feed or bedding contaminated with dog fecal material. Rabbits ingest feed or bedding that has been contaminated with dog feces (figure 60). The eggs develop into cysticerci (intermediate form) within the liver or peritoneal cavity. The life cycle is completed when the definitive host (dog) ingests an infected rabbit.

Clinical signs: not usually observed.

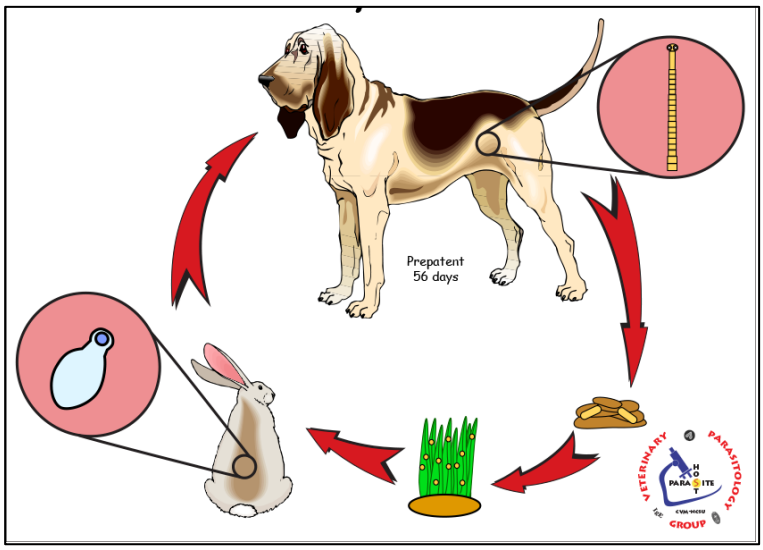


Figure 60. Life cycle of *Taenia pisiformis*

Clinical signs:

Taeniasis

Taeniasis is an infection with the adult tapeworm which usually remains confined in the small intestine. Most often, the infection causes minor gastrointestinal irritation and is frequently accompanied by nausea, diarrhea, constipation, hunger pains, chronic indigestion and passage of proglottids in the feces. Although these symptoms are usually milder when the infection is caused by *T. solium*, the risk of developing cysticercosis remains high.

Cysticercosis

Cysticercosis refers to the tissue infection caused by the metacestode, or larval stage, of *Taenia solium* and is acquired by the accidental ingestion of eggs (figure 61). The clinical manifestations associated with cysticercosis are a direct result of the inflammatory response induced to control parasite growth and may occur months to years after initial infection. Manifestations of disease are dependent upon a variety of factors including the site of infection and the number of cysticerci present within the tissues, which most often localize to sites within the eyes, skeletal muscles and brain. Cysticercosis is the most common intra-orbital parasitic infection and is observed in 13-46% of infected individuals. Infection

may involve the sub-retinal space (intra-ocular) or the extraocular muscles, eyelid and/or lachrymal glands (extra-ocular) surrounding the eye(s). Patients suffering from ocular infection frequently experience pain in the eyes accompanied by blurriness and partial or complete loss of vision. In extreme cases, infection may cause complete detachment of the retina.

Patients infected with cysticerci in the skeletal muscles and/or subcutaneous tissues are usually asymptomatic. In most cases, multiple cysts are present within the tissues, although solitary cysts may also be detected. Cysts range from 10-15 mm in length and arrange themselves in the same orientation as the muscle fibers. Leakage of fluid into the tissues, or death of the parasite, can trigger a strong inflammatory response, resulting in sterile abscess formation accompanied by localized pain and swelling.

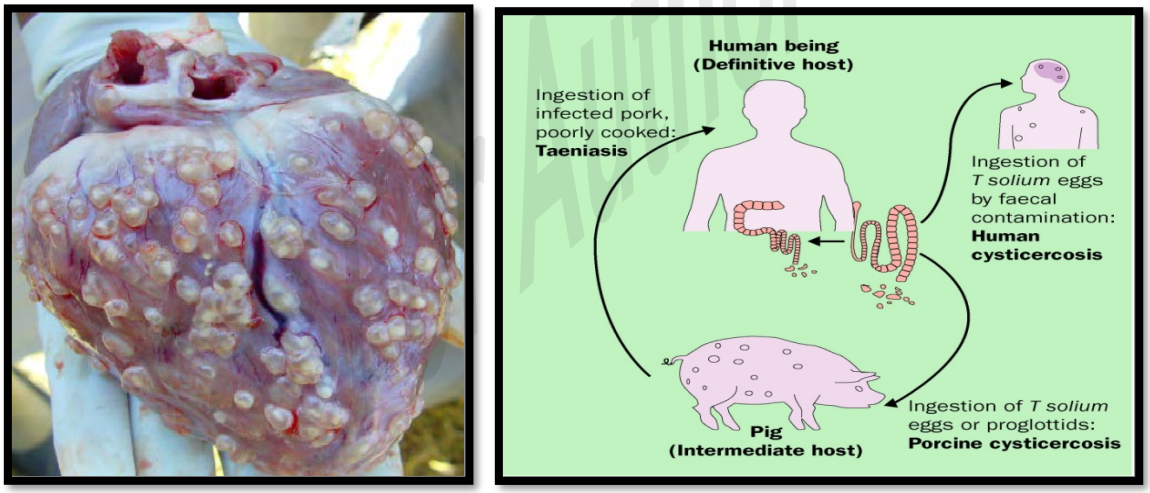


Figure 61. left side, cysticercosis lesion; right side life cycle of cysticercosis

Neurocysticercosis

Neurocysticercosis is the most common parasitic infection of the human central nervous system and observed in 60-90% of infected patients (figure 62). Cysts localized in the brain may range anywhere from 4-20 mm in length, but most commonly average between 8-10 mm. The cysts also can infect skeletal muscles and subcutaneous tissues, and an inflammatory response is induced which may

result in a subacute encephalitis. Seizures are the most common symptom reported in patients with neurocysticercosis and occur in 70-90% of infected patients. Other commonly clinical manifestations include headache, dizziness, involuntary muscle movement, intercranial hypertension and dementia. Not all patients with neurocysticercosis are symptomatic; a certain percentage of patients with neurocysticercosis never develop any symptoms and these infections are often self-resolving. In the figure 63, shows the life cycle of *Taenia solium* in the pigs and humans.

Figure 62. Neurocysticercosis

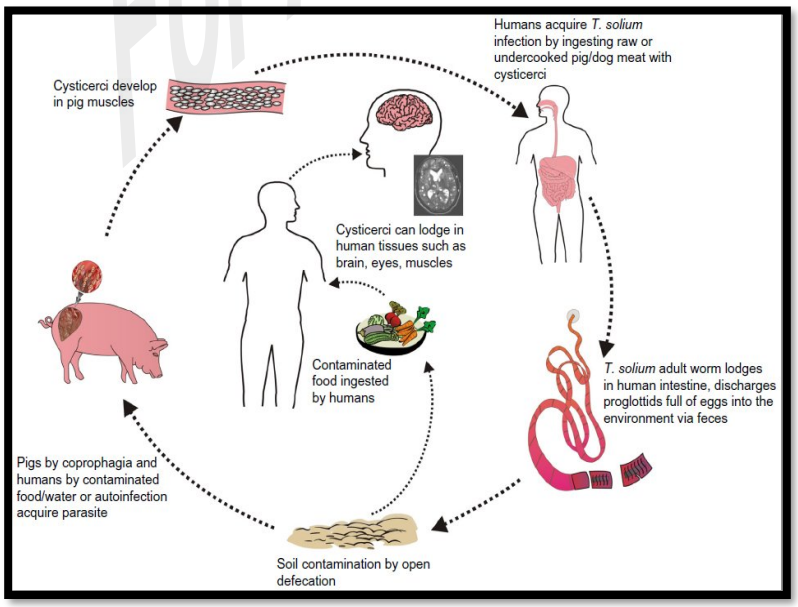


Figure 63. *Taenia solium* life cycle

Echinococcus granulosis

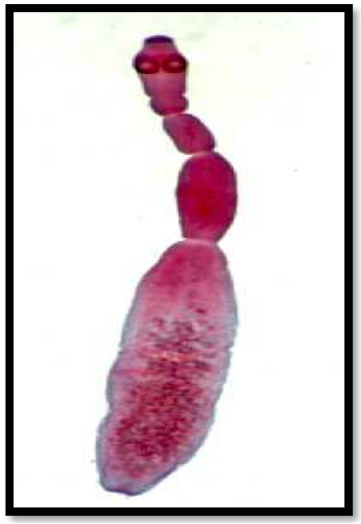


Figure 64. adult *Echinococcus granulosis*

Definitive host: Dogs or other canids

Intermediate host: Sheep, goat, swine, cattle, horses, camel

-Adult worms are small (3-6 mm in length)

-It is composed of: Scolex - Neck - Strobila –

segments: Immature, Mature and Gravid (figure 64).

Hydatid cyst features:

-Round & cystic

- Cyst wall: laminated layer, germinal layer.

- Contents: cystic fluid, brood capsules, protoscolex,

daughter & granddaughter cyst, and hydatic sand.

Hydatid sand: The protoscoleces generally settle down at the bottom of the cyst.

Geographical distribution: This is the smallest tapeworm, the larval (cyst) stage of which infects humans who eat food contaminated with dog feces containing embryonated eggs. The infection occurs most frequently in the regions where dogs and other domesticated hosts close to each other.

Biology: The adult *Echinococcus granulosus* resides in the small intestine of the definitive hosts, dogs or other canids. Gravid proglottids release eggs that are passed in the feces. After ingestion by a suitable intermediate host (under natural conditions: sheep, goat, swine, cattle, horses, camel), the egg hatches in the small intestine and releases an oncosphere that penetrates the intestinal wall and migrates through the circulatory system into various organs, especially the liver and lungs. In these organs, the oncosphere develops into a cyst that enlarges gradually, producing protoscolices and daughter cysts that fill the cyst interior (figure 65). The definitive host becomes infected by ingesting the cyst-containing organs of the infected intermediate host. After ingestion, the protoscolices

evaginate, attach to the intestinal mucosa, and develop into adult stages in 32 to 80 days (figure 66). Humans become infected by ingesting eggs, with resulting release of oncospheres in the intestine and the development of cysts, in various organs.

Clinical signs: The enlarging cysts can produce symptoms and signs related to their space occupation or can rupture with resultant anaphylactic shock. Leakage of daughter cysts produce hydatid cysts in new locations.

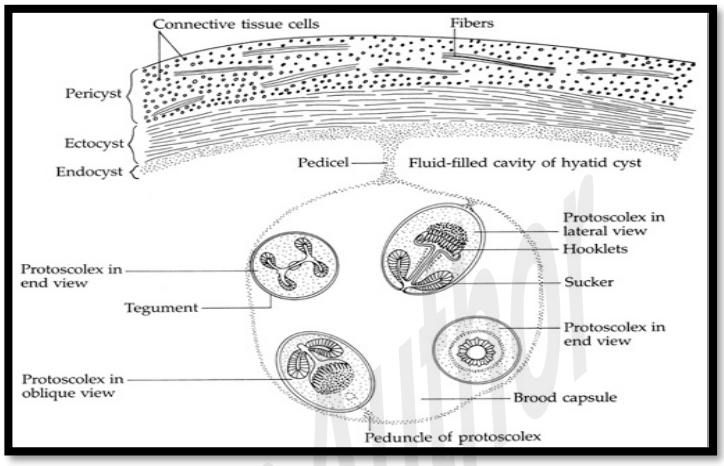


Figure 65. Brood capsule of hydatid cysts

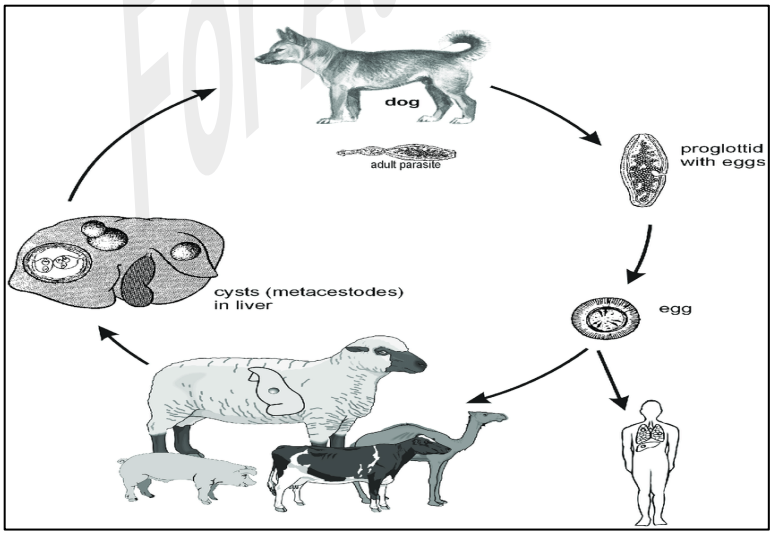


Figure 66. Life cycle of *Echinococcus granulosis*

Diagnosis and Treatment: hydatids can be diagnosed using X-radiography and ultrasonography. Several immunodiagnostic techniques are available, but these

are less sensitive than imagery. The cysts in the lung sometimes are not easy to diagnosis due to absence of protoscolices.

Order: Cyclophyllidea

Family: Anoplocephalidae

Genus: *Moniezia*

Monieza expansa

Definitive host: Ruminants

Intermediate host: Oribatid mites

Geographical distribution: *Monieza expansa* is a large tapeworm resides in the small intestines of livestock. The scolex is unarmed (i.e., hooks and rostellum are absent), presence of two sets of reproductive systems in each proglottid, and each proglottid is short but broad.

Morphology: *M. expansa* consists of the anterior scolex, neck and a highly extended body proper, the strobilus. The adult reachs up to 6–10 m in length. The scolex bears four large suckers, which are the holdfast organs to the host. There are no rostellum and rostellar hooks, and the suckers are devoid of spines. The boundary between the proglottids is studded with a row of interproglottid glands (figure 67). Tapeworm contains both male and female reproductive organs, and each proglottid is a complete reproductive set. Moreover, one defining feature of the genus is that there are two sets of reproductive organs situated at lateral sides associated with the cirrus pouches and genital pores in each proglottid. The testes are numerous.

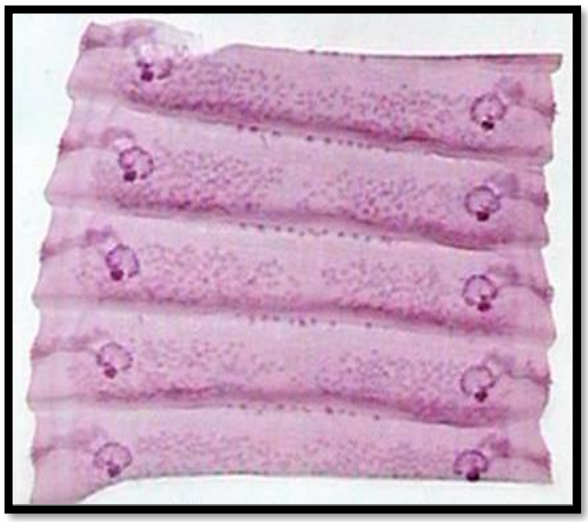


Figure 67. *Moniezia expansa* proglottids

Biology: the life cycle requires two hosts, ruminants as definitive hosts, and oribatid mites is an intermediate hosts. Eggs are passed out from the intestine along the gravid proglottids in the feces (figure 68). The eggs are eaten by soil mites. Eggs must reach the gut of mite hosts within 1 day of release, otherwise they are desiccated. Once inside the intestine of mites, the eggs hatch and the oncospheres penetrate into the haemocoel and develops to the cysticercoid stage. This stage may take up to 4 months. When the infected mite is swallowed by the grazing ruminants, mature cysticercoids are digested out of the mite, and develop to tapeworms, probably takes 5–6 weeks (figure 69).

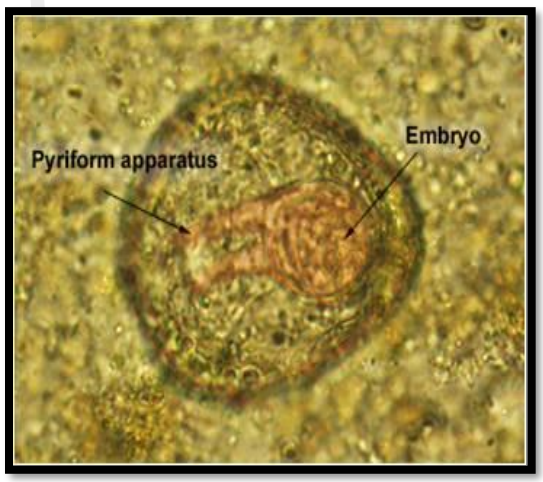


Figure 68. Egg of *Moniezia expansa*

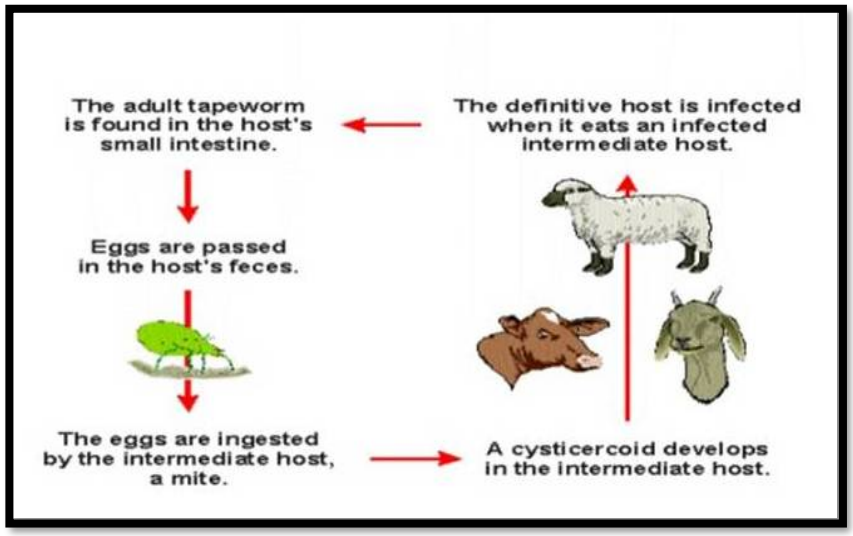


Figure 69. *Moniezia expansa* life cycle

Pathogenesis: *M. expansa* infections are harmless tapeworm and asymptomatic. In the cases of heavy infection causes intestinal obstruction, diarrhea and lost weight.

Diagnosis: examining stool samples in which eggs can be detected with pyriform apparatus (a chitinous ring with two projections), or often observation of the gravid proglottids in fecal samples.

Genus: Anoplocephala

Anoplocephala magna

Definitive host: Horses and donkeys

Intermediate host: Oribatid mite

Geographical distribution: it is equine tapeworms found in the small intestine, and occasionally in the large intestine or cecum.

Description: *A. magna* is similar to *Anoplocephala perfoliata* but is much longer. The scolex is large, 4–6 mm wide, with suckers opening anteriorly. The neck is short, as are the segments, being much wider than they are long. The genital organs are single and the pores are unilateral. Eggs are similar to those of *A. perfoliata* but slightly smaller and rounder, measuring 50–60 µm.

Biology: the larvae develop to infectivity within the oribatid mite over 2 to 4 months. After the infected mites are eaten accidently by the horse, develop to adult tapeworms in about 6 weeks (figure 70).

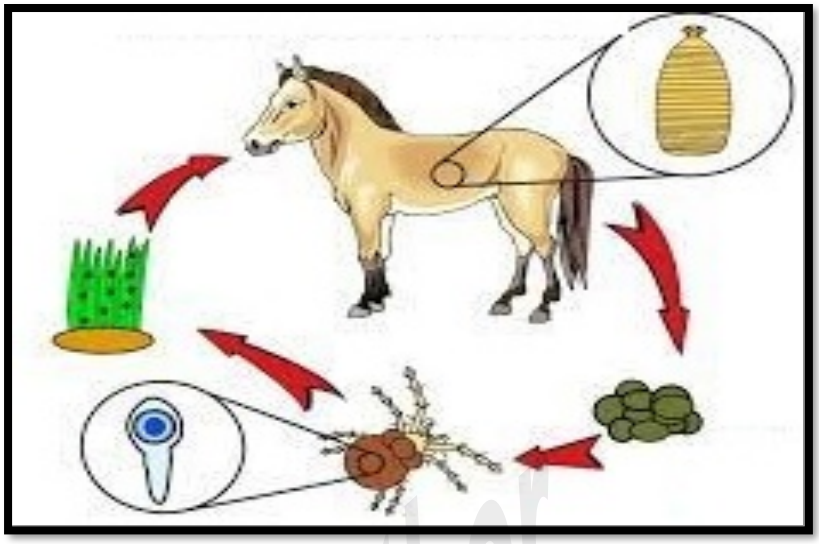


Figure 70. *Anoplocephala magna* life cycle

Pathogenesis: Large numbers of tapeworms cause intestine irritation, haemorrhagic or ulcerative enteritis. The intestinal blockage or intestinal inflammation may be seen due to the infection.

Diagnosis: examining animal fecal matters by a light microscope through identification eggs that are square in shape or the segments of worm in the feces (figure 71).

Control: adapting a treatment course would reduce egg counts as well as the number of infective mites on the pastures, thereby lessen reinfection.

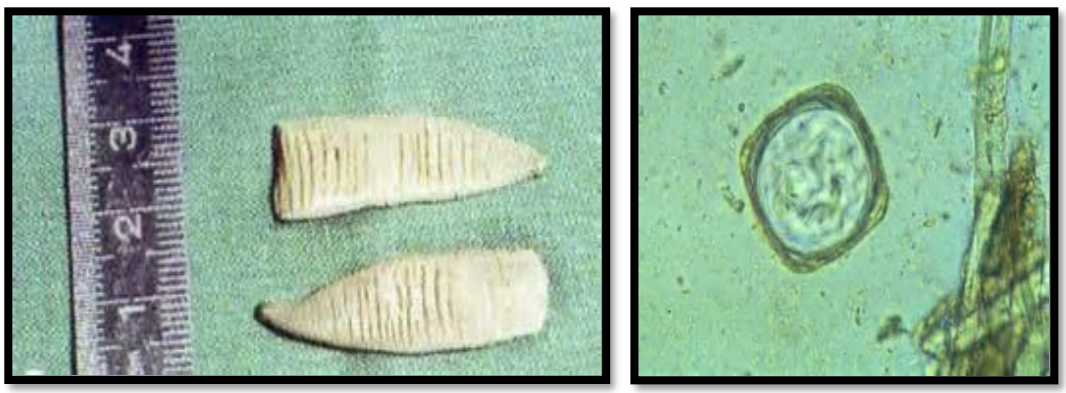


Figure 71. Adult & Egg of *Anoplocephala magna*

Family: Thysanomidae

Genus: *Avitellina*

Avitellina centripunctata

Definitive host: Sheep and goats

Intermediate host: Oribatid mites

Geographical distribution: Avitelliniasis or Avitellinosis is the infections with *Avitellina*. A tapeworm parasite that has mainly infected sheep, goats and less likely in cattle, camel and wild animals. It's do not affect dogs, cats, horses, swine, or poultry. This parasite is common in Africa, Asia, and Europe.

Morphology: Filiform, tapered at both ends. The anterior extremity exhibits an apical cone, its length 3m or more, width: 3mm, with very short proglottids and unspecified. The worms appear unsegmented (figure 72).

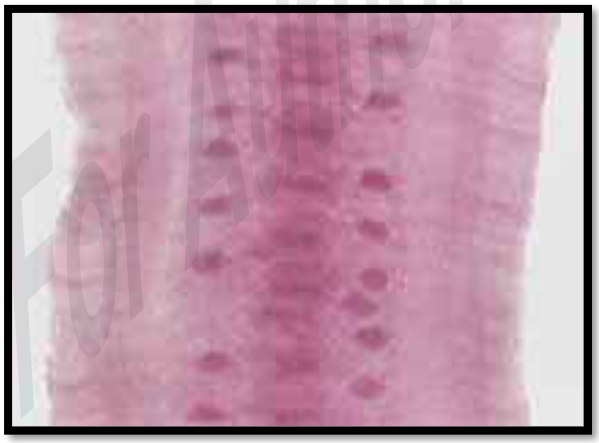


Figure 72. *Avitellina centripunctata*
Proglottids (Mature segment)

Biology: *A. centripunctata* has an indirect life cycle with ruminants as a definitive host, but the intermediate hosts still unknown. It seems that certain oribatid mites (also called "moss mites" and "beetle mites") and barklice (Psocoptera) are involved as suspicious intermediate host.

The adult worms produce eggs that are expelled in the feces of the host, either already free or included gravid segments. Depending on the species and the region they can survive for several weeks in the environment and survive

throughout cold winters, but remain sensitive to desiccation. The intermediate hosts ingest the eggs, which develop to infective cysticercoids in their body cavity.

The final host becomes infected after ingesting the contaminated intermediate hosts while grazing. After digestion the released cysticercoids attach to the gut's wall and develop to adult tapeworms within several weeks.

Clinical signs: Most infections with *A. centripunctata* are benign with no clinical signs. Even massive infections can remain asymptomatic. They compete for nutrients with the host and impacts productivity.

Diagnosis: based on fecal examination for the presence of gravid segments (proglottids, may look lice rice grains) or their eggs considering morphological feature. After necropsy the large tapeworms are easily seen inside the gut.

Genus: *Stilesia*

Stilesia globipunctata

Definitive hosts: Sheep, cattle and goat

Intermediate host: Oribatid mite

Biology: Adults are 45 - 60 cm in length, width: 2.5 mm, located in the small intestine. Each segment contains single set of genital organs, Have 4-7 testes on each side (figure 73). Genital pore opens irregularly alternate. Uterus dump bell shaped. Eggs are passed into two par-uterine organ and no pyriform apparatus.

Pathogenesis: worms are mainly attached at the junction of duodenum and jejunum. The immature worm penetrates the mucous membrane and forms the nodule in the intestine. Scolex and anterior part embedded in the nodule and rest of the posterior portion is free in intestine. Heavy infection usually causes death.

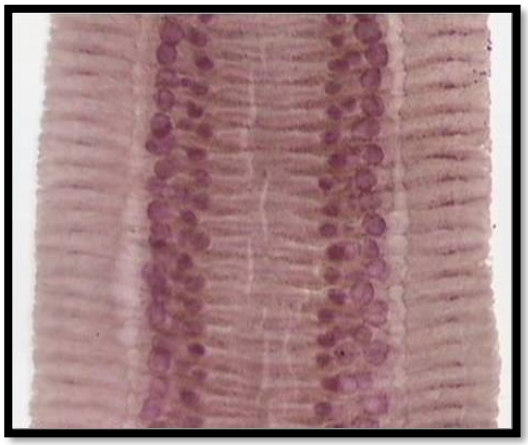


Figure 73. *Avitellina globipunctata* proglottids

Genus: *Thysoniezia*

Thysoniezia giardi

Definitive host: Cattle, buffalo, sheep and goats

Intermediate host: Oribatid mites.

Morphology: Length, 2m, and 12 mm in width, are similar to *M. expansa* except that the inter-proglottidal glands are linear, band like and restricted to the middle region (figure 74). Worm edges irregular because the proglottids have prominent cirrus on edges.

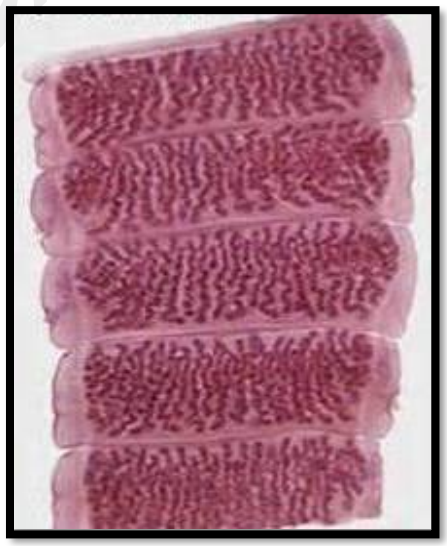


Figure 74. *Thysoniezia giardi* proglottids

Family: Davainedae

Genus: *Davainea*

Davainea proglottina

Definitive host: Chicken, turkey, other birds and pigeons.

Intermediate host: Terrestrial slugs and snails

Geographical distribution: It is found worldwide. It is quite common in free-ranging poultry. This species does not affect domesticated animals. The parasite causes a disease called davaineosis**.**

Morphological descriptions: *Davainea proglottina***,** is also called the minute tapeworm or the small chicken tapeworm. It is a small tapeworm, usually not longer than 4 mm with only 4 to 7 segments (proglottids). The head (scolex) has numerous hammer-shaped hooks (n=80 -94) and suckers armed with spines, and numerous hooks. The segments are whitish to translucent. The gravid segment is filled with eggs.

Biology: *D. proglottina* has indirect life cycle with certain birds (chicken, turkey, guineafowl, pigeons) as final hosts and terrestrial slugs and snail genus (*argriolimax*, *Arion*, *Cepaea*, *Deroceras*, *Limax*) as intermediate hosts. The gravid segments of adult tapeworms are shed with the birds' feces (usually one gravid segment per tapeworm per day). The gravid segments are motile and capable of creeping upwards on the vegetation. By mild and humid weather, the eggs can remain infective for slugs and snails during several days. The slugs and snails ingest the gravid segments, which release the eggs after digestion.

The eggs then develop to cysticercoids in the body cavity of the intermediate hosts. The birds ingest such contaminated snails or slugs. After digestion, the cysticercoids release the young tapeworms that attach to the gut's wall. The time between infection and shedding of the first eggs (**prepatent period**) is between 2 to 3 weeks (figure 75).

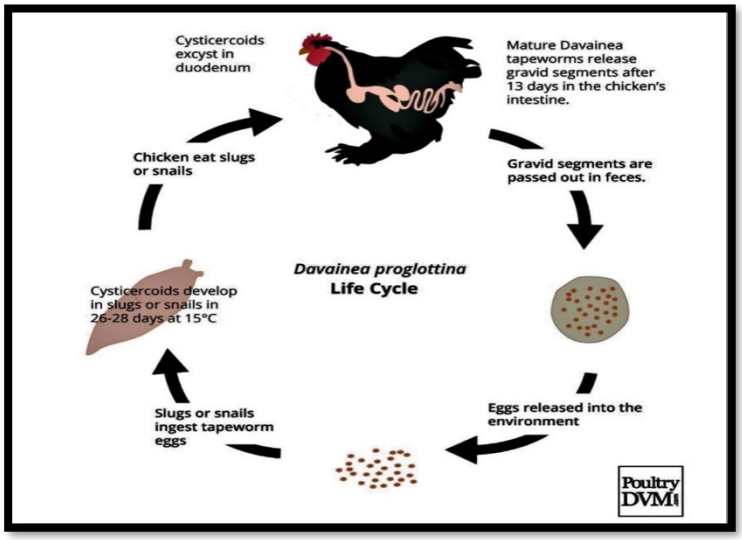


Figure 75. *Davainea proglottina* life cycle

Symptoms: *D. proglottina* is highly pathogenic for young birds. Free-ranging poultry are more at risk if they have free access to humid environments with abundant slugs and snails. *D. proglottina* can attach deeply into intestinal villi. Sudden massive infections can cause hemorrhagic enteritis and intestinal necrosis that can be fatal for the young birds. Chronic infections lead to reduced weight gains, diminished egg production, malnutrition, general weakness and even paralysis.

Diagnosis: usually done only after necropsy. It is important to examine a representative sample of the flock and to analyze intestinal smears under the microscope. Gravid segments can be found in the birds' feces, but not always, i.e., there can be false negatives.

Prevention and control: Frequent change of the birds' bedding and keeping it dry can help to avoid infections because it shortens the survival of the eggs and is unattractive for slugs and snails. Free-ranging birds should be kept off humid environments which are supportive of slugs and snails.

Genus: *Raillietina*

Raillietina cesticillus

Definitive host: Chicken, guinea fowl and turkey

Intermediate host: Beetles

Geographical distribution: *R. cesticillus* sometimes called 'broad-headed tapeworm', it infects the small intestine of birds, which are generally in close proximity to backyard poultry. It is a relatively harmless species among intestinal cestodes in spite of a high prevalence. In fact, is the most common parasitic platyhelminth in modern poultry facilities throughout the world.

Morphological descriptions: It can be distinguished from the other species of *Raillietina* (figure 76). The body size is small, scolex is disproportionately large and uniquely shaped. Rostellum is wide. It employs beetles as intermediate host to complete a lifecycle. A significant diagnostic character is an unusually numerous hook (more than 500 hooks). The suckers are poorly developed, and completely devoid of special devices or spines.

Figure 76. *Raillietina cesticillus*

Biology: The tapeworm completes its life cycle within two different hosts, the definitive host is mainly poultry, and the intermediate hosts are beetles. More than 100 species of beetles are known to act as intermediate host (flour beetle *Tribolium* are particularly important as intermediate host. Other avian such as guinea fowl and turkey are also often infected when they ingest beetles. A

complete life cycle requires 2–4 weeks. One defining feature of the species during developmental stage is the occurrence of a single egg in each egg capsule. The development of an egg embryo to a mature cysticercoid in its intermediate host requires 28 days after infection, and the mature cysticercoid takes around 31–34 days. Adults were found in chicken after 15 days of infection with mature cysticercoid, and the gravid segments can be obtained in the feces from 27 to 112 days (figure 77).

Pathogenesis: the infection is asymptomatic. It is considered as the least pathogenic species of *Raillietina*. The most pathological symptoms include degeneration of epithelial cells, enteritis and aggregation of macrophage.

Diagnosis: Infection is diagnosed by identifying proglottids in the faeces, or adult worms in the intestine upon autopsy.

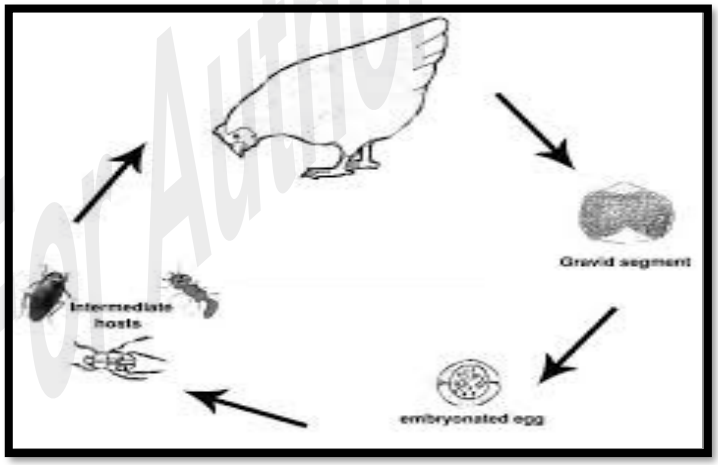


Figure 77. *Raillietina cesticillus* life cycle

Family: Dipylidiidae

Genus: *Dipylidium*

Diplidium caninum

Definitive host: Canids and felids

Intermediate host: Dog or cat flea (Larval stages) and occasionally the dog louse.

Morphological descriptions: bilateral genital pores present in each segment and have 2 sets of male and female reproductive organs. The scolex is small and globular with 4 deeply cupped elliptical suckers. Rostellum Protractible /retractile with 1-7 rows of rose thorn shaped hooklets (figure 78).

Geographical distribution: worldwide distribution; this tapeworm is ubiquitous and common among dogs and cats. Human infection is rare. Canids and felids are the normal hosts for *Dipylidium caninum.* The intermediate host is usually the larval stages of the dog or cat flea (*Ctenocephalides* spp.) and occasionally *Trichodectes canis* (dog louse).

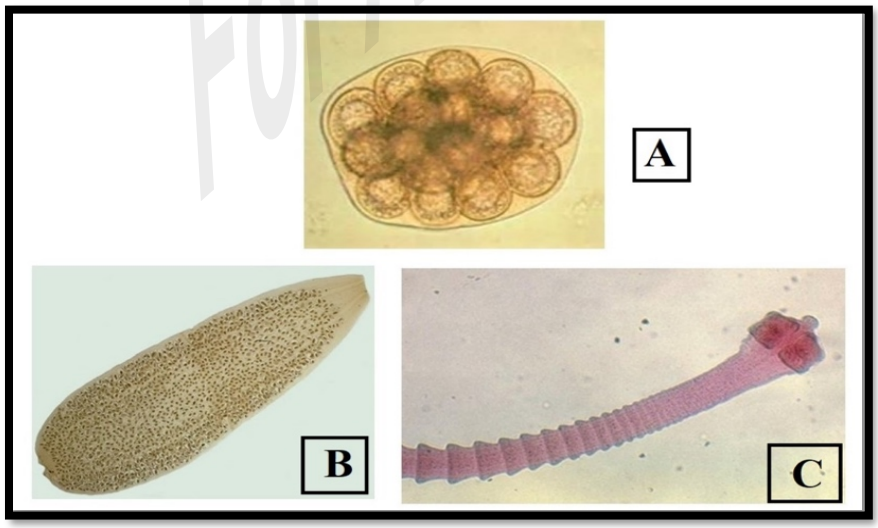


Figure 78. *Dipylidium caninum,* A: Egg mass, B: Gravid segment, C: Scolex with neck and segments

Biology: the worm measures 50 cm in length. It is transmitted to human by infected dog fleas. Also called the cucumber tapeworm or the double-pore

tapeworm, is a cyclophyllid cestode. As in all members of family Dipylidiidae, proglottids of the adult have genital pores on both sides (hence the name double-pore tapeworm). Each side has a set of male and female reproductive organs. The scolex has a rostellum with four rows of hooks, along with the four suckers that all cyclophyllid cestodes have. Children are the most frequently affected. Through an intimate relationship with dogs like kissing having it lick the face, an infected dog flea can easily be swallowed. Called the "pumpkin seed" tapeworm, the first hint of infection may be finding seed-like particles in the stool or undergarments. These particles are actually the egg-bearing segments of the tapeworm. After flea is swallowed, the larvae are liberated and reaches maturity for about 3 weeks (figure 79).

Symptoms: are vague, but include restlessness and persistent diarrhea.

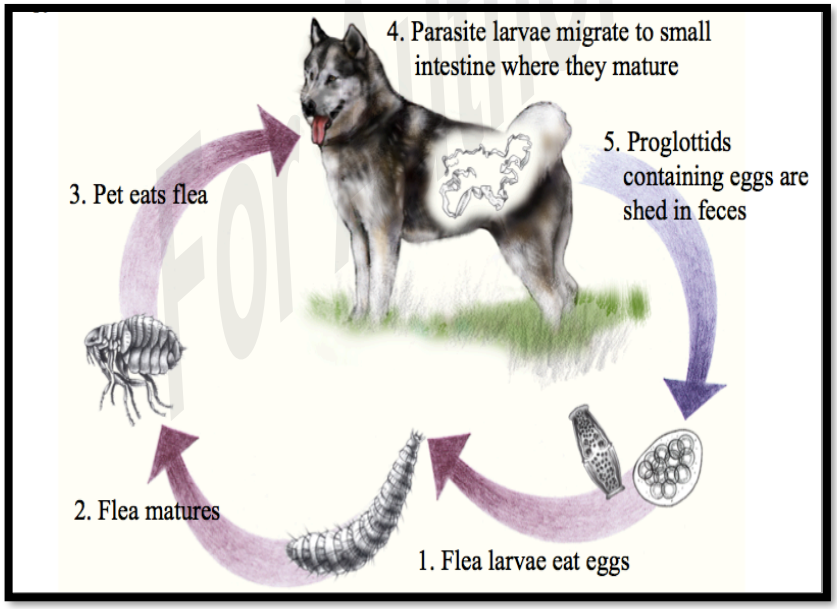


Figure 79. Life Cycle of *Dipylidium caninum*

Family: Hymenolepididae

Genus: *Hymenolepis*

Hymenolepis nana

Two important species: *Hymenolepis nana*, *H. diminuta*

Definitive host: Rats, mice and in human (accidentally)

Intermediate host: flea

Morphological description: it is a dwarf worm. The head is small with a ring of hooks and four sucker cups (figure 80, 81). The tapeworm usually infects human when the eggs are ingested accidently. The eggs can be transmitted by infected food handlers, grain beetles and other insects that infest grains, as well as rodent contaminated foods.

Figure 80. anterior region, *Hymenolepis nana*

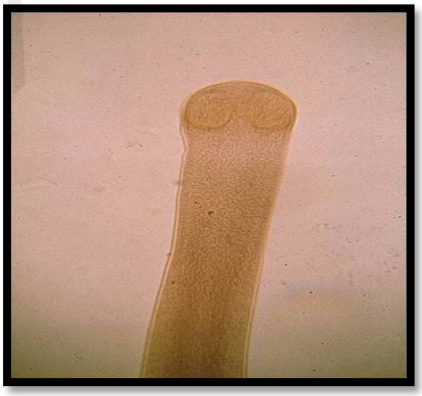


Figure 81. anterior region, *Hymenolepis diminuta*

Geographical distribution: occur worldwide, requiring an intermediate host.

Biology: The larval stage called cysticercoid in fleas, while, adult stage found in rats and mice, and in human especially children when inadvertently swallow fleas (figure 82). Also, the genus is derived from the membranous character of the egg shell “hymen”, with three testes in each mature segment and uterus is sac-like and transverse. The Egg has two membranes’ the outer membrane is thin and transparent. The larval stage was small bladder containing the invaginated head proximally and a solid, elongated portion as a caudal appendage.

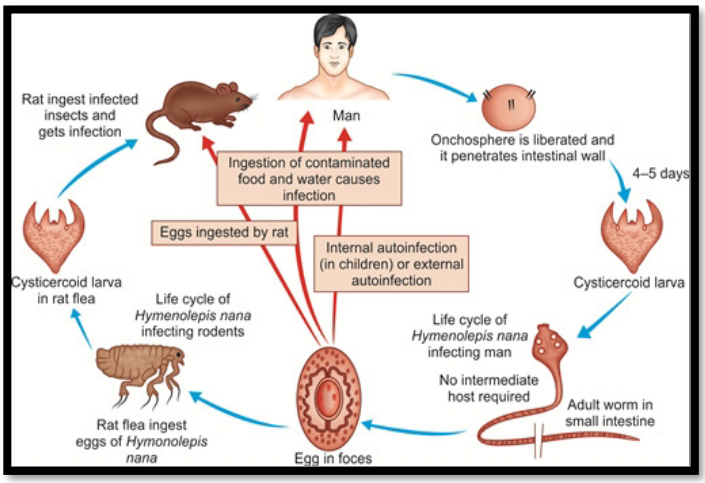


Figure 82. *Hymenolepis nana* life cycle

Pathogenesis: the infection is usually without manifested clinical signs, but a heavy infection leads to diarrhea, itching, abdominal pain, headaches, body weakness, weight and appetite loss.

Diagnosis: The eggs in fecal matter can be identified through presence an embryo with six hooklets.

Family: Mesocestoididae

Mesocestoididae is unarmed cyclophyllidean tapeworm in mammals, birds, reptiles, and carnivores, as well as humans Their larva is a slender threadlike contractile and always free in cavities or encysted in tissues. It causes inflammation in the intestine of humans, but are not harmful in animals. The life cycle of the worm involves a stage of tissue invasion which cause peritonitis.

Genus: *Mesocestoides* spp.

Definitive host: Cats**,** dogs and other wild carnivores
1ST intermediate host: Ants, beetles, oribatid mites
2ND intermediate host: Reptiles, amphibians, birds and small mammals.

Geographical distribution: A genus of non-human tapeworms that may rarely afflict humans who eat poorly-cooked meat with encysted muscle. This parasite occurs in wild and domestic animals. *M. lineatus* is an Old-World species

reported from Asia, Europe, and Africa. *M. variabilis* is endemic to North America. Species implicated in zoonotic infections include *M. lineatus* and *M. variabilis,* though the relationships between species in this genus remains under investigation (figure 83).

Figure 83. Segments of *Mesocestoides corti, Mesocestoides lineatus, Mesocestoides variabilis*

Biology*:* has a three-host and its life cycle involves cats**,** dogs and other wild carnivores as final hosts, and two intermediate hosts including:

1- The first **intermediate hosts** are **arthropods** such as ants, beetles, oribatid mites.
2- The second **intermediate hosts** are **small vertebrates** (reptiles, amphibians, birds and small mammals).

The eggs of adult worms in the intestine of the final host are shed with the feces inside the gravid segments. Once the gravid segments release the eggs, are ingested by arthropods. The eggs hatch in the intestine of these intermediate hosts and the young tapeworm larvae penetrate into their body cavity (hemocoel), where they develop to next stage, cysticercoids.

Small vertebrates (snakes, lizards, frogs, birds, rats, mice, etc.) ingest the infected arthropods and the cysticercoids are released in their gut. They migrate through the gut's wall and into various organs (mainly the lungs and the liver) and develop further to an infective larval stage, in this particular case called “tetrathyridium”.

The final hosts (cats, dogs, etc.) ingest the small vertebrates infected with

tetrathyridia. After digestion the young tapeworms are released, attach to the gut's wall and start producing segments.

The prepatent period (time between infection of the final hosts and first eggs shed with the feces) within 21 days.

Pathogenesis: the infection with *Mesocestoides* spp. reported in humans and typically involves low numbers of worms. Non-specific, recurrent gastrointestinal signs/symptoms have been also documented. To date, no larval-stage has been identified in humans.

Order: Pseudophyllidea

Family: Diphyllobothriidae

Genus: *Spriometra*

Spriometra mansonoides

Definitive Host: Dogs, raccoons, cats, bobcat.

1st intermediate host: Copepod.

2nd intermediate or paratenic host: Amphibians, reptiles, birds, and mammals.

Geographical distribution: Tapeworms is known as *Spirometra* develop in human after eating raw snake (figure 84). *Spirometra* causes a bizarre eye disease called "Sparganosis". Tapeworm larvae then migrate into the tissue around the eye. This is not solely a condition possible only in underdeveloped countries. For example, raw meat is often used in the western world for bruises and black eyes.

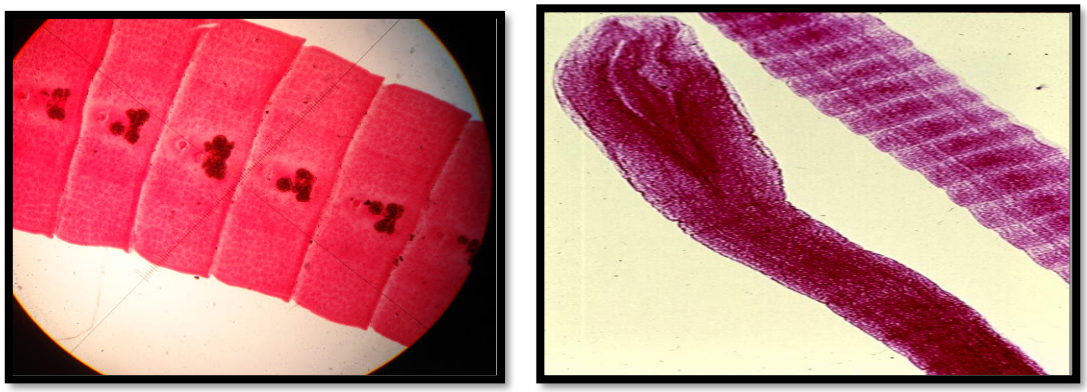


Figure 84. Proglottids and Scolex of *Spirometra mansonoides*

Biology: Adult members of the genus *Spirometra* live in the intestines of dogs and cats. Eggs are shed in feces, hatch in water and release coracidia, which are ingested by copepods. The coracidia develop into procercoid larvae in the copepod intermediate host. Second intermediate hosts, including fish, reptiles and amphibians, ingest infected copepods and acquire procercoid larvae. The procercoid larvae develop into plerocercoid larvae. The cycle is completed when a predator (dog or cat) eats an infected second intermediate host. Humans cannot serve as definitive hosts for *Spirometra* spp., but serve as paratenic or second intermediate hosts and develop sparganosis. Humans acquire sparganosis by either drinking water contaminated with infected copepods or consuming the flesh of an under-cooked second intermediate or paratenic host. Spargana can live up to 20 years in the human host.

Diagnosis: Egg (~ 39 X 57 µm), or examination of the proglottid if they passed in the feces (figure 85).

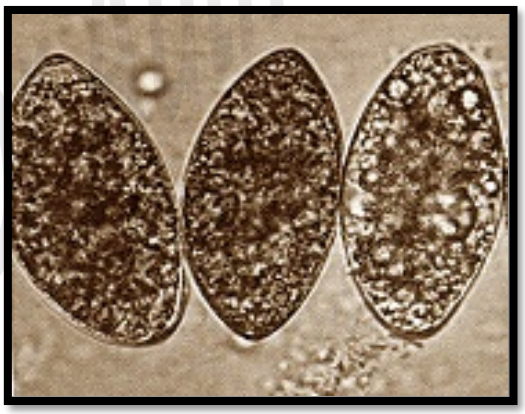


Figure 85. Eggs of *Spirometra*

Pathogenesis: Migrating spargana can result multiple symptoms which depends on a location in the host tissue (figure 86). Spargana may found in the subcutaneous tissue, breast, orbit, urinary tract, pleural cavity, lungs, abdominal viscera and the central nervous system. The migration in subcutaneous tissues is usually painless, but when spargana settle in the brain or spine and consequently neurological symptoms may occur, including weakness, headache, seizure, and abnormal skin sensations, such as numbness or tingling. If the inner ear is

involved, the patient may experience vertigo or deafness. Multiple plerocercoids of *Sparganum proliferum* cause proliferative lesions in the infected tissue.

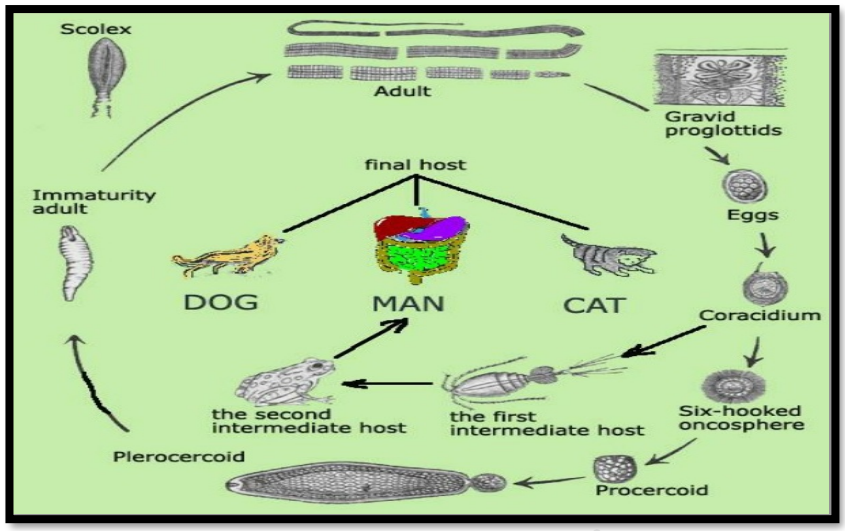


Figure 86. Life cycle of *Spirometra mansonoides*

Genus: *Diphyllobothrium*

Diphyllobothrium latum

Definitive host: Man, dog, cat

Intermediate host: Passed first in water.

1st stage larva: Coracidium, ciliated oncosphere that develops from egg in water.

2ndstage larva: **Procercoid**: Spindle-like solid body with cephalic invagination, found inside the cyclops (the first intermediate host).

3rd stage larva: **Plerocercoid**: Head is invaginated in the neck, found in the fresh water fish, the second intermediate host.

Mode of Infection: ingestion of imperfectly cooked infected fish or raw containing plerocercoid larvae.

Geographical distribution: Biotypes are mostly shallow freshwater littorals with vegetation favoring the development of copepods and fish. Infection with this parasite is most prevalent in areas of the North Temperate and sub-Arctic zones, where freshwater fish are commonly consumed.

Biology: Immature eggs are passed in feces of the mammal host (figure 87). After ingestion by a suitable freshwater crustacean such as a copepod (the first intermediate host), the coracidia develop into procercoid larvae. After ingestion of the copepod by a second intermediate host, typically small freshwater fish, the procercoid larvae are freed from the crustacean and migrate into the fish's flesh where they develop into a plerocercoid larvae (sparganum). The plerocercoid larvae are the infective stage for the definitive host (including humans). The sparganum can migrate to the musculature of the larger predator fish and mammals can acquire the disease by eating these later intermediate infected host fish raw or undercooked. Following ingestion of the infected fish, the plerocercoids develop into immature adults and then into mature adult tapeworms where reside in the small intestine. The adults attach to the intestinal mucosa by means of the two bilateral grooves (bothria) of their scolex. The adults can reach more than 10 m (up to 30 ft) in length with more than 3,000 proglottids. One or several of the tape-like proglottid segments regularly detach from the main body of the worm and release immature eggs in fresh water to start the cycle over again. Immature eggs are discharged from the proglottids (up to 1,000,000 eggs per day per worm) and are passed in the feces. The incubation period in humans, after which eggs start to appear in the feces is typically 4–6 weeks, but can vary from as short as 2 weeks to as long as 2 years. The tapeworm can remain active up to 20 years.

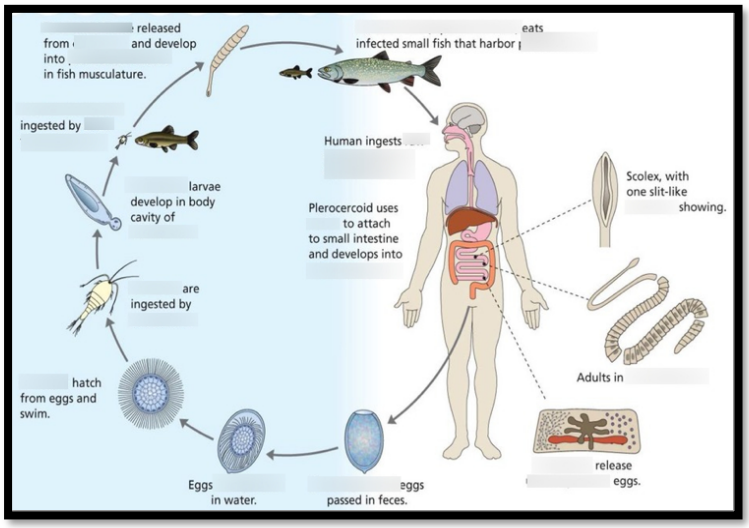


Figure 87. Life cycle of *Diphyllobothrium latum*

Pathogenesis: The fish tapeworm is the largest parasite found in human, with its length reaching 4,000 proglottids. The infection acquired after eating raw or uncooked fish. This parasite is competitive with nutritious by absorbing vitamin B12, leads to anemia with debilitating effects. Pain, nausea, and anorexia are common manifested symptoms due to gut disorders.

Diagnosis: Microscopic examination of feces can be performed for detection an operculated egg. Laboratory diagnosis depends on the recovery of characteristic eggs using formol ether. Proglottids may also be observed in fecal samples usually in a chain of segments from a few centimeters to about half of a meter in length (figure 88).

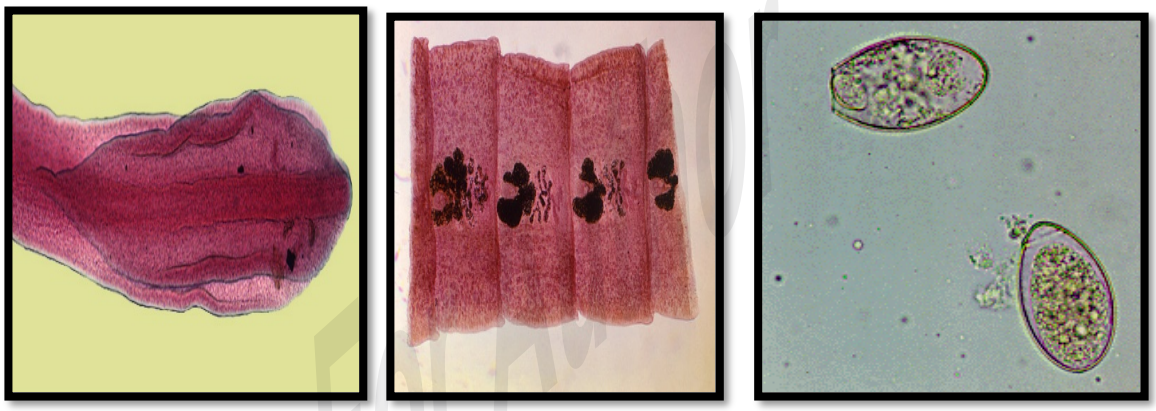


Figure 88. Scolex, mature segment and operculated eggs of *Diphyllobothrium latum*

CHAPTER THREE

Phylum: Nemathelminthes

Introduction to Nematodes

Nematodes are a wide spreading parasite that has been inhabited in a variety of domestic and non-domestic animals. The majority of their species develop inside their animal hosts and feed on mucus and food detritus. Others species like rhabditids (Strongyloides) are free-living parasites and feed on decaying materials. The biological interaction with the infected host has sever impacts, causing physical depletion and death occurring due to severity of infection. Clinical symptoms are associated with nematodes leads to loss wools and weight. The nematode superfamily is classified into two groups: bursate and non-bursate. The transmission from one host to another occurs mostly indirectly through contaminated water and food, as well as through intermediate hosts such as insects or flies carry juveniles.

Morphological Characteristics of Nematodes

The structure of nematodes adult worm is microscopically well defined and briefly as outlined:

1. Nematode parasites have a thick exterior cuticle structure and white to slightly yellowish (figure 89).
2. They have elongated cylindrical body.
3. Both sexes are dimorphic adults (separate-male and female)
4. Their mouth is small opening and provides with tree lips.
5. The anterior region is broaden called cervical alae.
6. The posterior is blunt for non-developed bursate except for trichostrongylid the males have well developed bursa (figure 90).
7. The digestive system consist of three parts includes stomodeum (mouth, esophagus), mesenteron (intestine), and proctodaeum (rectum and anus).

8. The male genital organ is provided with spicules (gubernaculum) while female reproductive system consists of ovaries and uterus.

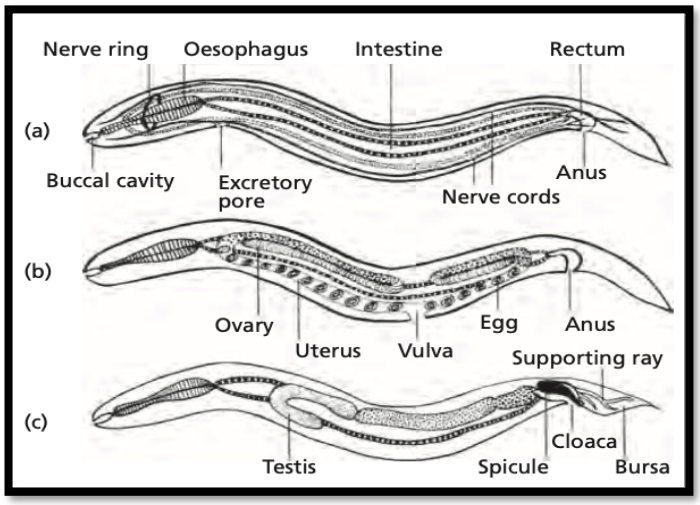


Figure 89. Digestive and reproductive system of the nematodes

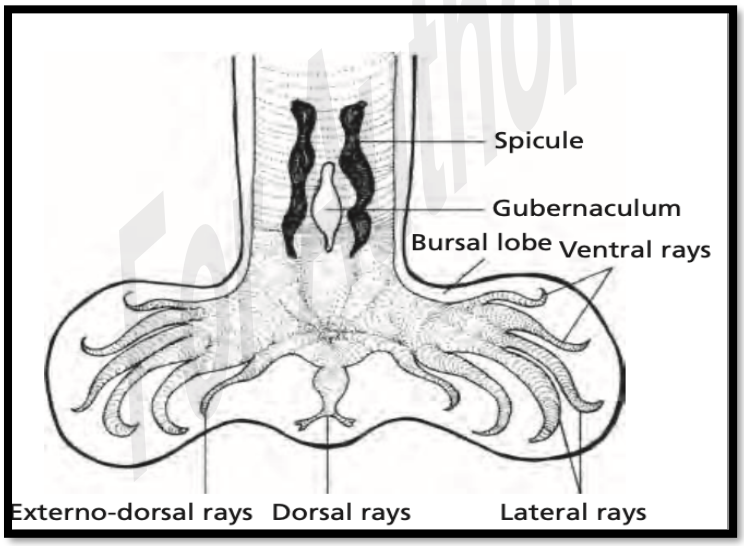


Figure 90. Posterior region of male trichostrongylid (Bursa with spicules)

Eggs of Nematodes

Nematode eggs have different size and shape, and the shell is somewhat thick and comprise from three layers (figure 91). The inner membrane has lipid properties which is impermeable, while the middle layer is chitinous and solid. The third outer layer consists of protein. Nematode species has an operculum (lid) or plug at one or both ends. In some species the eggshell is very thin and may be present

as a sheath around the larva. Because the thick shell of nematodes eggs, it can survive in soil and resist drought.

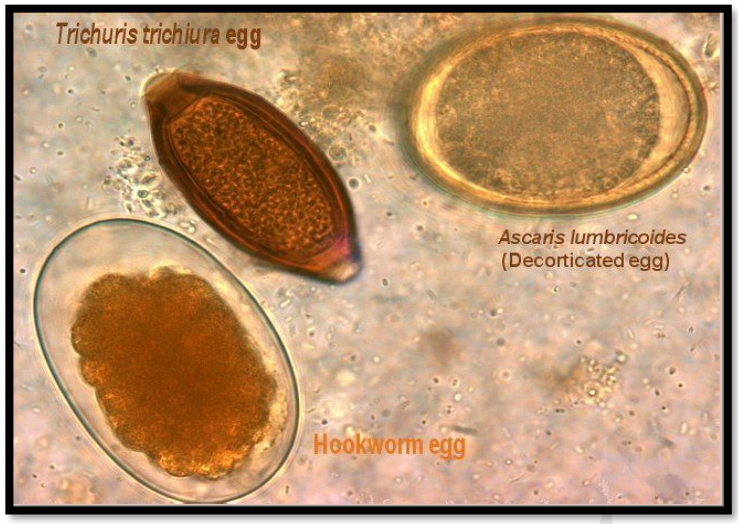


Figure 91. Eggs of nematodes from different species

The Life Cycle of Nematodes

The males of nematodes are smaller in length than females in all type of their species. In the alimentary tract of a definitive host, the adult worm shed more than 10,000 eggs in the faces. The first larvae develop with the eggs (infective stage) and hatching usually is influenced by temperature and humidity. When the larvated eggs are ingested by host and the hatching is stimulated gut enzymes. The subsequent larval stages are designatedL_2, L_3, L_4, and eventually protruding to L_5, which is a premature adult. The life cycle of nematodes might be diverged by inoculation of the infective stage by an intermediate host, and the life cycle continues when it is consumed by the final host. After that, the mature worm copulates in the gut lumen. The hepatic–tracheal migratory cycle is characterized in nematodes. The larvae can transmit from the gut to the liver via the portal system, then to the heart via the hepatic vein, and finally to the lungs via the pulmonary artery (figure 92).

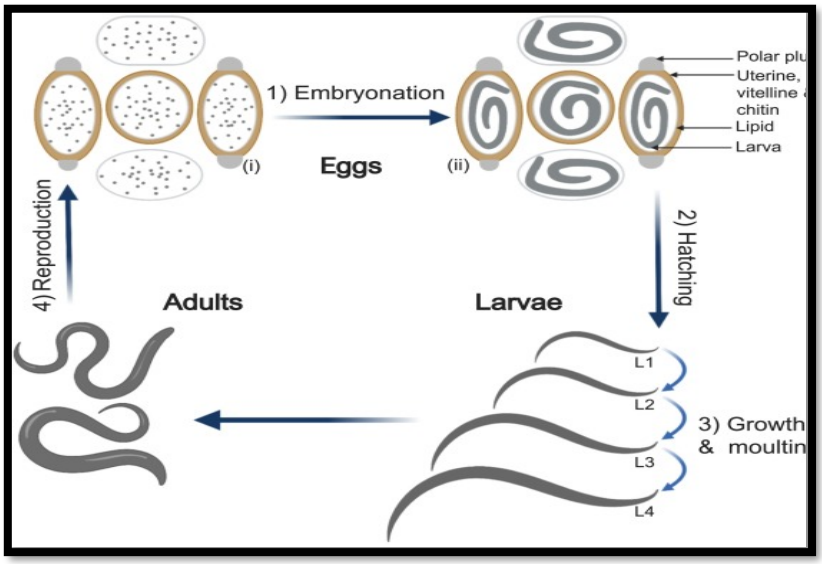


Figure 92. The life cycle of nematodes

Pathogenesis of Nematodes in Animals

The nematodes worm causes substantial damage to the host tissues while also affecting the host's immune system. The impacts of damage or destruction depends on the site of infection, including the nematode's migratory cycle, which reaches far into organs such as the liver and lungs. The capillaries of the abomasa mucosa appear to be overflowing with blood, and the pepsin enzyme is a contributory factor. *Ostratagia* infection in sheep contribute to hypergastrinemia and induce change in PH. The endoparasite invasion even individual can alter gastro-intestinal function and then protein metabolisms. The absorption is the most impairment may be induced which reflect normal appetite. Pepsinogen is the enzymes that secrets from intestinal and help in break down proteins is noticeably halt by the endoparasite. The lesion left by gastro-intestinal nematodes are correlated with heavy infection of worm generated and a number of eggs produced in the intestinal lumen.

Immunity Against Nematodes Infection

Once nematodes enter the gastrointestinal tract, the host immune system can react promptly and activate diverse immune cells. Macrophages, eosinophils, neutrophils, B-cells, T-helper cells, and cytokines were all involved in the immune response to infection, which included both innate and adaptive immunity. Interleukins mediators such as IL3, IL4, IL5, IL9, IL10, and IL13 can indeed be released by Th2 and B cells. As a result, eosinophils and mast cells are induced, culminating in a hypersensitive reaction at the infection site. L3 in the intestinal lumen has been noticed to be eliminated by eosinophils. A mechanical response could be triggered by the secretion of a high amount of galectin, a mucous form of galectin that can restrict parasite movement. IFN is released during a critical phase of the immune response, eliciting CD8 and macrophage. When sheep are infected with *Haemonchus contortus*, IgE is greatly activated. The infection of calves with *Ostertagia ostertagi* resulted in the generation of IFN- and Th2 cytokines. Heart worm (*Dirofilaria immiti*) causes blockages in the heart and pulmonary vessels, and IgG and neutrophils are the immune system's response to the parasite infection. In the infected lung and bronchus, IgA, IgE, CD8, and CD4 are all heavily reacted to *Dictyocaulus viviparus* infection.

Class: Secernentea
Subclass: Chromadoria
Suborder: Spiruria
Superfamily: Subuluroidea

Description: have weakly formed lips with sensilla and a thick-walled stoma, as well as three teeth. They are similar to *Heterakis*, although the tail is not as pointed. The males measure around 8–10 mm and the females up to 14–18 mm in length (*Subulura brumpti).*

***Subulura* spp.**

Hosts: *Subulura* is important genus in veterinary field that is commonly reported in poultry (figure 93).

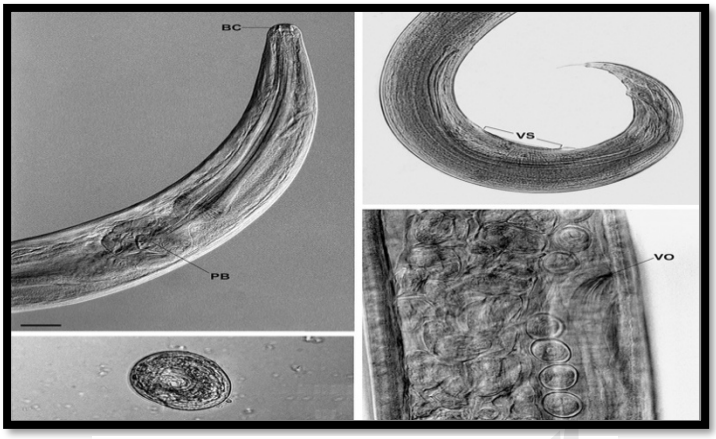


Figure 93. *Subulura brumpti*

Geographical distribution: The species of these parasite widely distributed in eastern Asia and Brazil.

Biology: Eggs discharged in fecal matter and then swallowed by the intermediate host (insects), where they grow into the infective L3 stage for approximately two weeks. The larvae travel to the lumen of the caeca after being consumed by the final host. The prepatent period between 6 to 8 weeks.

Table 4. the definitive and intermediate host of *Subulura* spp.

***Subulura* Species**	**Site of infection**	**Definitive host**	**Intermediate host**
S. *suctoria*	Caeca	Chicken, turkey, duck, fowl, quail, pheasant,	Beetles and cockroaches
S. tanjinensis	Intestine	Siberian chipmunks	Beetles
S. brumpti	Cecum	Quail	Grasshoppers
S. *chinensis*	Cecum	Owl	Beetles

Order: Ascaridida

Family: Heterakidae

Genus: ***Heterakis***

Species: *H. gallinarum*, *H. dispar* and *H. isoloche.*

Heterakis gallinarum

Host: inhabit the large intestine (cecum) of poultry

Definitive Hosts: Chicken, turkey, pigeon, pheasant, quail, guinea fowl, duck, goose and a number of wild Galli form birds.

Paratenic hosts: Earth- worms

Macroscopic description: the worm is white in color and 1.5 cm long, with elongated pointed tails. The male is 7–13 mm long and the female 10–15 mm.

Microscopic description: The esophagus has a large bulb. Generic identity may be confirmed by the presence of a large circular pre-cloacal sucker in the male and prominent caudal alae supported by 12 pairs of caudal papillae. The spicules are unequal in length, the left has broad alae and the right is slender (about 2 mm). their egg is ovoid in shape and has a smooth-shelled. The eggs measure 65–80 by 35–46 μm and are unsegmented when laid. Differentiation between the three species of *Heterakis* is based on the shape of the esophagus and the length and shape of the spicules (figure 94).

Geographical distribution: *H. gallinarum* is distributed worldwide and often present in chickens, domesticated turkeys, and other species of fowl, primarily of poultry. Their eggs survive for years in soil making it difficult to eliminate. Earthworms may ingest the eggs of *H. gallinarum* and contribute to the cause of infections in poultry. Although the eggs are themselves infective, they can develop into a second infective larval stage. This development occurs around 27 °C and takes 2–4 weeks.

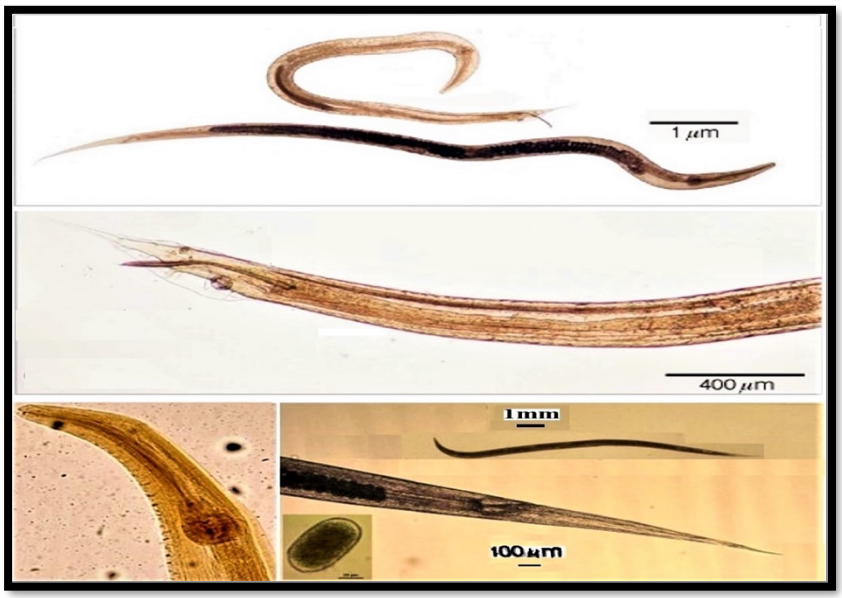


Figure 94. *Heterakis gallinarum*

Biology: Eggs of *H. gallinarum* are passed in fecal matter. At optimal temperature (22 °C), they become infective in 12–14 days and remain infective for years in soil. Upon ingestion by a host, the embryonated eggs hatch into second-stage juveniles in the gizzard or duodenum, and are passed to the cecum. Their development is completed in the lumen, but some may enter the mucosa and remain for years without further development. The prepatent time is 24–30 days. Earthworm and houseflies are considered paratenic hosts, as they can ingest the egg in feces and a juvenile may hatch in tissues, which stays dormant until eaten by birds (figure 95).

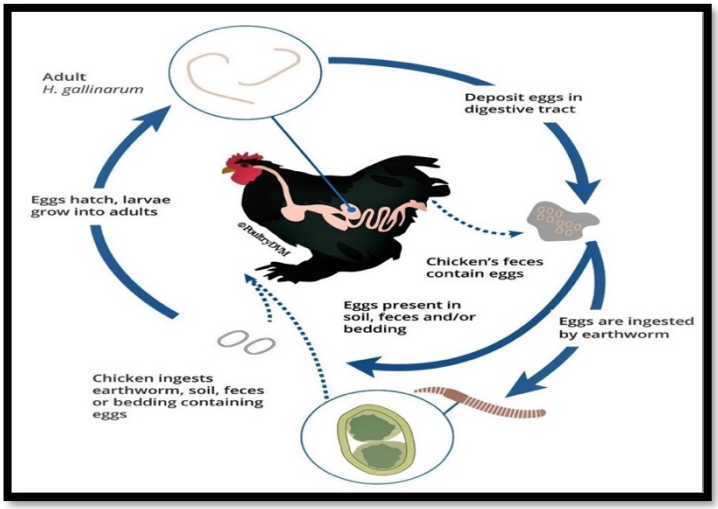


Figure 95. Life cycle of *Heterakis gallinarum*

Pathogenesis: *H. gallinarum* is recognized as an economically important parasite because its ovum serves as the vector for the protozoal parasite transmit *Histomonas meleagridis*, which causing histomonosis in poultry.

Diagnosis: Typically relies on fecal egg counts, which are prone to false negative diagnoses. Molecular techniques are used to confirm diagnosis of the parasite.

Control: Control of *H. gallinarum* is necessary based on hygiene the backyard flocks. It must isolate the turkeys from domestic poultry, and disposal of waste materials from the ground. Treatment of suspected chickens is important to prevent other from getting infection.

Treatment: Can be done with piperazine salts, levamisole or a benzimidazole is effective. Flubendazole, mebendazole or fenbendazole can be administered in the feed.

Family: Trichuridae

There are several species of veterinary importance:

- Dogs: *Trichuris vulpis*, *Trichuris campanula*
- Cats: *Trichuris serrata, Trichuris campanula*
- Cattle, Sheep, Goats and other Ruminants: *Trichuris discolor, Trichuris globulosa, Trichuris ovis*
- Pigs: *Trichuris suis*

Are animals infected with *Trichuris* worms contagious for humans?

YES, but not all the species, and usually not through direct contact with the animals, but mostly through ingestion of infective eggs that contaminate the environment of infected animals (pastures, gardens, soil, sandboxes, yards, etc.). Eggs shed with the feces of infected animals are not immediately infective, but need to mature in the environment during 10 to 25 days.

There are other worms belonging to the *Trichuridae* that infest pets and livestock, notably those of the genus ***Capillaria***.

Trichuris trichiura

Introduction:

The genus *Trichuris trichiura*, is known as the human whipworm that causes trichuriasis. It is referred to as the whipworm because it looks like a whip with wide handles at the posterior end. The whipworm has a narrow anterior esophagus and a thick posterior anus. The worms are usually pink and attach to the host via the slender anterior end. The female is usually larger than the male.

Host: Human

No intermediate host is required

Geographical distribution: The egg of the whipworm is the infective stage, and favorable conditions for its maturation are warm and humid weather. This is why most of the disease burden is seen in tropical climates, it is also found in rural areas. It is estimated between 450 million to 1 billion active cases, mostly diagnosed in children. It is thought there is partial protective immunity that develops with age.

Biology: The unembryonated eggs are passed in feces, **2-** In the soil, the eggs develop into a 2-cell stage, **3-** The cells continue to divide (advanced cleavage stage), **4-** Then the eggs embryonate and become infective in 15 to 30 days. They may be ingested when hands or food are contaminated with feces, **5-** The eggs hatch in the small intestine and release larvae, **6-** Larvae mature and establish themselves as adults in the cecum and ascending colon.

The female worm lay between 2,000 to 10,000 eggs per day. The eggs are deposited in soil from human feces. After 14 to 21 days, the eggs mature and enter an infective stage. If humans ingest the embryonated eggs, the eggs start to hatch in the human small intestine and utilize the intestinal microflora and nutrients to multiply and grow. The majority of larvae move to the cecum, penetrate the mucosa, and mature into adulthood. Infections involving a high-worm burden will typically involve distal parts of the large intestine. Children

appear to be vulnerable to the parasite and poor sanitation is associated with a heavy disease burden.

Pathogenesis: The adult worm invades the intestinal mucosa by its thin, thread like anterior end and feeds on tissue secretions but not on blood.

It causes petechial hemorrhage, inflammation, oedema and mucosal bleeding in the intestinal mucosa at their site of attachment. The lumen of appendix can be blocked in case of severe worm infection. Presence of worms in the mucus membrane irritates the nervous plexus of mucosa causing diarrhea and cramps. Occasional eosinophilia can be present. Approximately 0.005 ml of blood per worm per day is lost in the infected man.

Trichuriasis

Pathological complications are seen in cases of heavy infection with more than 800 worms, particularly in children. It results in stomach pain, tenesmus weight loss, bloody diarrhea with excessive mucus, cachexia, and severe anemia.

The rectum may prolapse if there are many worms spread throughout the colon and rectum. Appendicitis can occasionally be brought on by migrating worms (figure 96).

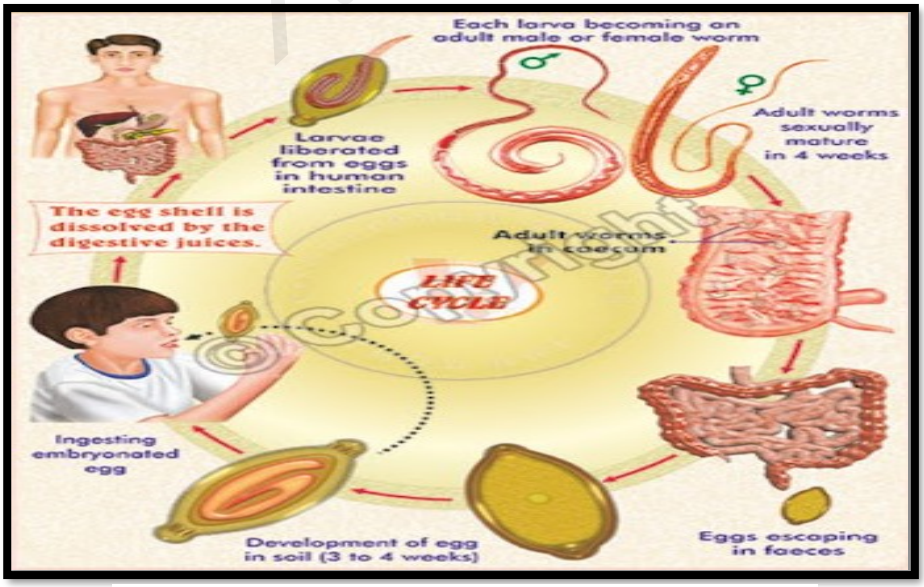


Figure 96. Life cycle of *Trichuris trichiura*

Diagnosis: Laboratory diagnosis of Trichuriasis is based on the microscopic examination of stool samples to determine the presence of eggs. During a heavy infestation, the eggs may be seen on a stool saline smear, but this has a low sensitivity, or by using the Kato-Katz method for counting eggs per unit weight of feces.

Stool samples may also demonstrate red blood cells (RBCs), and white blood cells (WBCs), specifically eosinophils. A complete blood count may show anemia.

Polymerase chain reaction (PCR) assays are currently being developed and used in research settings. This has improved the specificity and sensitivity of detecting the whipworm

Treatment and management: The treatment of trichuriasis is with mebendazole or albendazole. The suggested dose of mebendazole is 100 mg twice a day for 3 days or albendazole is 200 to 400 mg twice a day for 3 days. Mebendazole has been shown to be more effective comparing with other drugs used. Ivermectin 200 mcg/kg daily can be used.

Trichuris Ovis

Host: cattle, sheep, goats

Location: wide distributed in the North and South America, Australia, Europe, and Asia.

Morphology: have a long and thin neck. The male ranges from 53.04mm-75.08mm in length, while female is 32.03-70.19mm in length.

Symptoms and treatment: feces usually watery (diarrhea) and contain a mixture of mucus, water, and blood

Anthelminthic drug can be used for treatment infected animals, such as albendazole and mebendazole.

Family: Trichinillidae

Trichinellosis (trichinosis) is caused by nematodes (roundworms) of the genus *Trichinella*.

Geographical distribution: It is worldwide in many carnivorous and omnivorous animals, other species of *Trichinella* are now recognized, including *T. pseudospiralis* infect mammals and birds worldwide. Outbreaks occur in settings where multiple people consume the same *Trichinella*-infected meat.

Biology: Trichinellosis is caused by the ingestion of undercooked meat containing encysted larvae of *Trichinella* species (figure 97). After exposure to gastric acid and pepsin, the larvae are released from the cysts and invade the small bowel mucosa where they develop into adult worms. Females are 2.2 mm in length; males 1.2 mm. The life span in the small bowel is about four weeks. After 1 week, the females release larvae that migrate to striated muscles where they encyst. Diagnosis is based on clinical symptoms and is confirmed by serology or identification of encysted or non-encysted larvae in biopsy or autopsy specimens (figure 98).

Figure 97. Encysted larvae of *Trichinella* in muscle

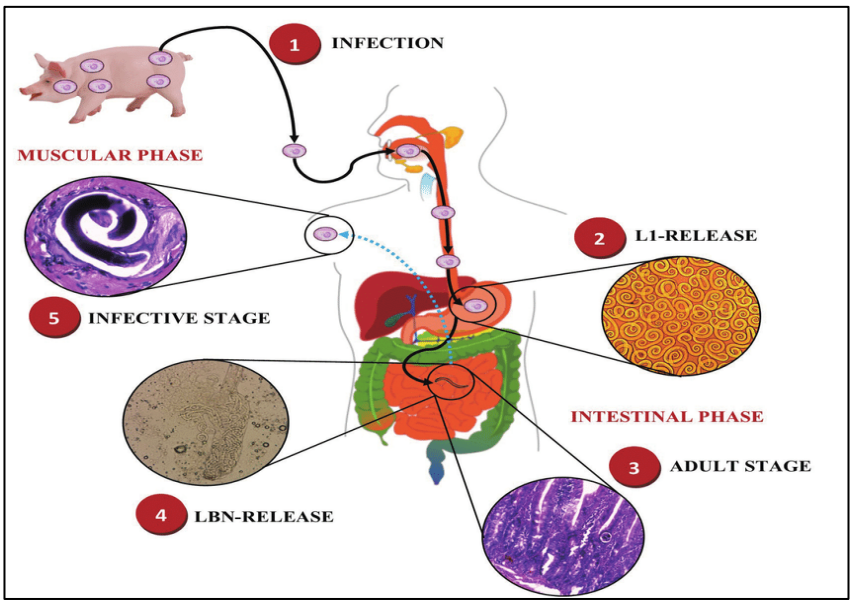


Figure 98. Life cycle of *Trichinella* spp.

Diagnosis: Laboratory diagnosis of Trichinellosis is most often made by a *Trichinella* antibody test. In some cases, a muscle biopsy may be performed.

Pathogenesis: The first symptoms of trichinellosis are gastrointestinal, usually occurring 1–2 days after consuming raw or undercooked meat from a *Trichinella*-infected animal. These symptoms include nausea, diarrhea, vomiting and abdominal pain.

The following classic symptoms of Trichinellosis often occur within 2 weeks after eating contaminated meat and can last up to 8 weeks: Muscle pain, fever, swelling of the face (particularly the eyes), weakness or fatigue, headache, chills, itchy skin or rash, cough, diarrhea and constipation.

Symptoms may range from very mild to severe and relate to the number of infectious worms consumed in the meat. Many mild cases of trichinellosis are never specifically diagnosed because they are assumed to be the flu or other common illnesses. Furthermore, many people with trichinellosis do not experience any symptoms at all.

Treatment:

Albendazole: 400 mg twice a day by mouth for 8 to 14 days

Mebendazole: 200 to 400 mg three times a day by mouth for 3 days, then 400 to 500 mg three times a day by mouth for 10 days

Prevention and Control: The best way to prevent trichinellosis is to cook meat. In addition to:

- Wash hands with warm water and soap after handling raw meat.
- Curing (salting), drying, smoking, or microwaving meat alone does not consistently kill infective worms; homemade jerky and sausage were the cause of many cases of trichinellosis in recent years.
- Freeze pork less than 6 inches thick for 20 days at 5°F (-15°C) to kill any worms.
- Freezing wild game meats, unlike freezing pork products, may not effectively kill all worms because some worm species that infect wild game animals are freeze-resistant.
- Clean meat grinders thoroughly after each use.

To help prevent *Trichinella* infection in animal populations, do not allow pigs or wild animals to eat uncooked meat, scraps, or carcasses of any animals, including rats, which may be infected with *Trichinella*.

Genus: *Capillaria*

Capillaria plica

Introduction: *Capillaria plica* infect the urinary bladder, and occasionally the ureters and renal pelvises, of dogs and cats. Quite similar but less common organism, *C. felis cati*, is also found in cats. Dogs and cats become infected by eating earthworms that contain the first-stage larvae. Mature *Capillaria* are threadlike, yellowish, and 13–60 mm long. The eggs are colorless, operculated, have a slightly pitted shell, and are 63–68 × 24–27 μm in size. Most dogs and cats are asymptomatic.

Geographical distribution: worldwide spreading parasite, and wild animals act as the intermediate hosts.

Biology: In dogs and cats, eggs of *Capillaria plica* are released in the urine of the mammalian definitive host. First stage larvae (L1) develop within the eggshell in 30–36 days.

When eaten by the intermediate host (Earthworm) the L1 larvae hatch in the earthworm's intestine. The larvae burrow through the intestinal wall and become embedded in connective tissue throughout the worm's body.

If the earthworm is eaten by a suitable mammalian host, the larvae molt into second stage larvae (L2), burrow through the intestinal wall, and molt again into third stage larvae (L3).

The L3 are carried through the circulatory system to the glomeruli of the kidneys. From there, they travel down the ureter to the urinary bladder.

By 33 days post-infection, third (L3) and fourth-stage larvae (L4) are found in the urinary bladder. Here they mature into adults and reproduce sexually, shedding fertilized eggs into the urine of the host within about 60 days of infection.

Pathogenesis: In many cases, dogs infected with *Capillaria* show no signs of illness.

When signs do occur, they relate to the specific *Capillaria* species and its site of infection. *P. plica* affects the walls of the bladder and the kidneys; therefore, infection may lead to blood in the urine, urinary accidents, and straining to urinate.

The eggs are shed in the urine and may be found in the urine sediment.

Diagnosis: Diagnosis can be difficult, because the eggs of *Capillaria* are shed only on an intermittent basis (figure 99). *C. plica* which affects the urinary tract, may be diagnosed based on urinalysis. When the urine is examined under a microscope, and may see signs of inflammation, including blood and/or inflammatory cells. Further examination may reveal the microscopic eggs of the parasite, which are shed into the urine.

Microscopic hematuria and increased numbers of epithelial cells may also be present.

Figure 99. egg of *Capillaria Plica*

Treatment: Reported treatments include levamisole, fenbendazole, albendazole, and ivermectin. The treatment of choice is unknown, but a single dose of ivermectin at 0.2 mg/kg, SC, is likely to be effective. It is not FDA approved for this use and is contraindicated in Collie breeds. The parasite may be self-limiting in the absence of reinfection.

Capillaria philippinensis

Definitive host: Human.

Intermediate host: Many species of freshwater fish appear susceptible to infection.

Reservoir host: Piscivorous birds (herons, egrets, and bitterns), suggested as a wildlife reservoir.

Geographical distribution: As the name suggests, *Capillaria philippinensis* is endemic in the Philippines and epidemics have occurred in the Northern Luzon region. The parasite is also endemic in Thailand, and sporadic cases have been reported from other East and Southeast Asian countries. More recently, a number of cases have been identified in northern Egypt.

Biology:

1-Typically, unembryonated, thick-shelled eggs are passed in the human stool and become embryonated in the external environment in 5—10 days.

2- After ingestion by freshwater fish, larvae hatch, penetrate the intestine, and migrate to the tissues.

3- Ingestion of raw or undercooked fish results in infection of the human host. The adults of *Capillaria philippinensis* are very small (males: 2.3 to 3.2mm; females: 2.5 to 4.3 mm) and reside in the human small intestine, where they burrow in the mucosa.

In addition to the unembryonated, shelled eggs which pass into the environment, the females can also produce eggs lacking shells (possessing only a vitelline membrane), which become embryonated within the female's uterus or in the intestine (figure 100).

Pathogenesis: Capillariasis manifests as abdominal/gastrointestinal disease, which can become serious if not treated because of autoinfection. A protein-losing enteropathy can develop which may result in complications such as cardiomyopathy, severe emaciation, cachexia, and death.

Symptoms: Severe diarrhea of 1-7 months duration associated with vomiting and central abdominal colics. Stools were yellowish or greenish and voluminous. Anorexia was profound with loss of weight. Bilateral painless ankle oedema developed.

Diagnoses: presence the eggs, larvae and adult worms of *C. philip*pinensis in stool.

Treatment: Mebendazole 400 mg/day in two divided doses for 28 days in addition to high protein diet and correction of electrolyte disturbance.

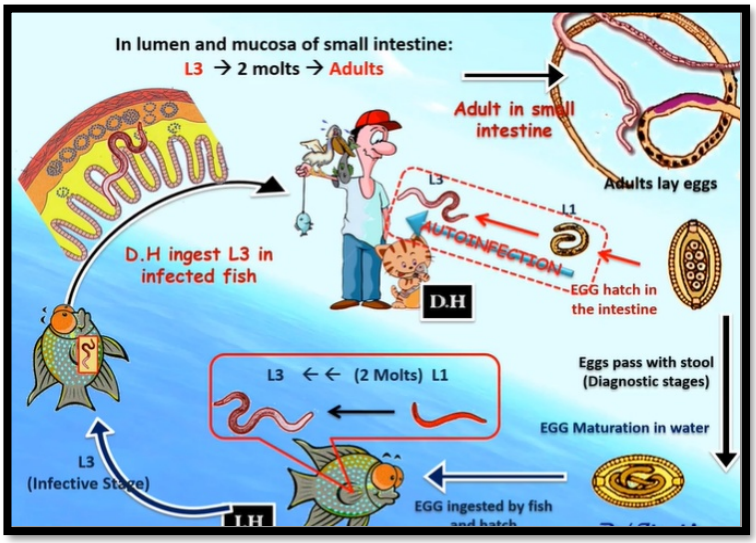


Figure 100. Life cycle of *Capillaria philippinensis*

Family: Spiruridae

Family of nematode worms that have the adults parasitic in vertebrates, that have the larval stages parasitic in insects, and that with related forms constitute a distinct superfamily of the order Spirurida.

The genus related to the family: *Spirura talpae, Viguiera adsimilisai, Gongylonema aequispicularis* , *Mastophorus muris, Spirura rytipleurites*

***Spirura rytipleurites* (*Filaria gastrophila*)**

Introduction: *Spirura* from the name of the group (round) and *ryti* = wrinkled cuticle and *pleurites* for the location of the cysts in the leural cavity of the intermediate host.

These worms were originally described from larvae recovered from the body cavity of the oriental cockroach or beetles and sometimes frogs (intermediate host). while the cat and accidentally sometimes the rat, Mongoose, Fox, hedgehog, and striped polecat regarded as definitive hosts.

Geographical distribution: These worms are common in North Africa and probably into parts of southern Europe. Reported on infections in cats, one from Lyon and one from Madagascar. Also reported from a cat in Egypt.

Site of infection: The adults live in the wall of the esophagus and stomach.

Biology: The adults of this genus are recognized by the presence of one or two ventral cuticular bosses (inflations) in the cervical region. The eggs are ovoid with a smooth thick shell, and contain a first-stage larvae with a cephalic hook when passed in the feces.

The larvae were capable of developing in cockroaches that become infected when they ingest the eggs. The larvae grow within capsules in the abdominal cavities of the cockroaches where they become quite long, over a cm in length. The third-stage larvae within the abdominal cavity of the insects have well developed reproductive systems. Cats become infected through the ingestion of infected cockroaches.

Diagnosis: Can described and illustrated by light and scanning electron microscopy from specimens collected from the stomach of final hosts.

Pathogenesis: The nematode was identified as belonging to the genus *Spirura*. The number of parasites in this animal was considered significant and contributed to the death of the animal.

Control/Prevention: As with many of the parasites, it is important to keep cats from hunting. At the same time, the large numbers of oriental cockroaches that can be present in houses would mean that in many parts of the world, infections could be very hard to control.

Family: Filariidae

The family causes diseases with economic importance by parasitic worms that are transmitted by the bite of blood-feeding insects. This debilitating group of diseases includes river blindness and lymphatic filariasis (commonly known as elephantiasis) in human as well as to animals (granulomatous nodule, pulmonary dirofilariasis).

Hosts: Dogs, foxes, wolves, coyotes, and cats.

***Dirofilaria immitis (*Dog heartworms)**

Introduction: *Dirofilaria immitis* is a parasitic nematode responsible of canine and feline cardiopulmonary dirofilariasis in both domestic and wild hosts, and the causal agent of human pulmonary dirofilariasis. It is a zoonotic parasitic disease mainly located in temperate, tropical, and subtropical areas of the world.

Transmission: Dirofilariasis is transmitted neither person-to-person nor person-to-mosquito-to-person. The transmission of dirofilariasis requires mosquitoes as the intermediate host as well as the production of microfilariae, which does not take place in humans. Different species of mosquitoes (*Culex* spp., *Aedes* spp., *Anopheles* spp.) act as an intermediate stage in order to complete their life cycle. Heartworm infection is a severe and life-threatening disease. Initially the pulmonary vasculature is affected, and the lung itself and,

finally, the right chambers of the heart. However, the development of the parasite in cats takes longer compared to dogs and most infections are amicrofilaraemic. Additionally, many cats tolerate the infection without any noticeable clinical signs or with signs manifested only transiently and sometimes sudden death may arise without warning.

Table 5. species, hosts, and prepatent period of the family Filariidae.

Species	**Final host/Habitat**	**Intermediate host**	**Prepatent period in final host (weeks)**
Onchocerca volvulus	Humans/Subcutaneous tissues	*Simulium* spp.	32–52
Onchocerca gutturosa	Cattle/Subcutaneous tissues	*Odagmia* spp.	28
Wuchereria bancrofti	Humans/Lymph nodes	*Aedes* spp., *Culex* spp.	52
Brugia malayi	Humans/Lymph nodes	*Mansonia* spp., *Anopheles* spp.	12
Loa loa	Humans/Subcutaneous tissues, eye	*Chrysops* spp.	52
Litomosoides carinii	Rats/Pleural cavity	Mites (Bdellonyssus)	10–11
Dirofilaria immitis	Cats, Dogs/pulmonary or nodules	Mosquitoes	6-7 months

Geographical distribution: *Dirofilaria immitis* is cosmopolitan in dogs particularly prevalent in warmer areas.

Biology: *Dirofilaria immitis* has an indirect life cycle. The adult parasite sexually reproduces in its vertebrate host, and the offspring are transferred to the intermediate host, which is usually a mosquito or a flea (figure 101).

The life cycle described as following:

1. The life cycle starts with introduction of L3 larvae from an infected mosquito into the canid host.
2. In the dog, the larvae mature into L4 larvae and then into adult worms that localize in the pulmonary arteries and occasionally in the right cardiac ventricle.

3. Following a prepatent period of 6 to 7 months, microfilariae are produced via sexual reproduction and are released into the dog's bloodstream, where they may remain viable for months to years.

4. Mosquitoes feeding on the infected host ingest the microfilaria, which then migrate to the Malpighian tubules where they mature into first-stage larvae.

5. Ultimately, the larvae mature to the L3 stage in the salivary glands of the mosquito and become infective, thus completing the heartworm life cycle.

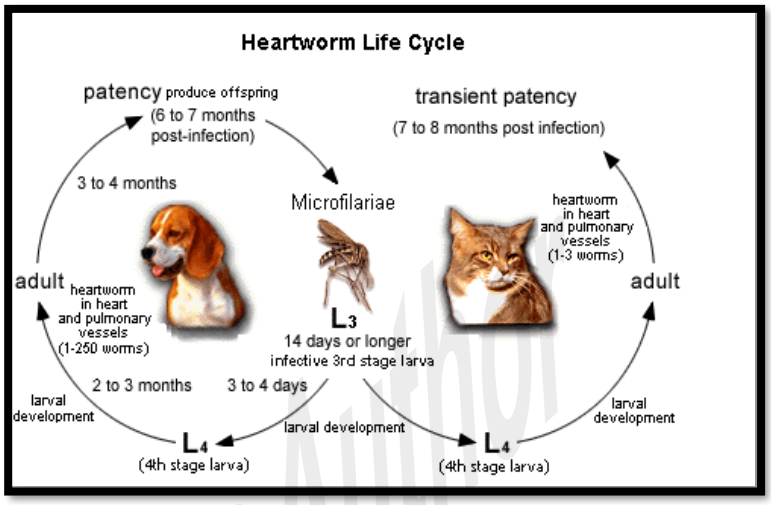


Figure 101. Life cycle of *Dirofilaria immitis*

Pathogenesis: the infection of cats occurs asymptomatically or chronic respiratory signs and acute death. In cases of pulmonary larval dirofilariasis, infected cats can have a Heartworm Associated Respiratory Disease (**HARD**), that is associated with asthma-like clinical signs.

Cats are inherently resistant to heartworm infection. This is reflected by relatively low adult worm burden, the lack of microfilaraemia and their short life span, which complicates the diagnosis of this disease in the feline patient.

Diagnosis: Dirofilariasis is diagnosed by detection of the microfilariae in the peripheral blood, immunological tests and molecular approaches also can be use. *Dirofilaria immitis* is usually diagnosed by the finding of the distinctive coin lesions on chest X-rays. The species that produce subcutaneous nodules are diagnosed by the finding of adult worms in biopsy specimens of these nodules.

Treatment: The definitive treatment of *Dirofilaria* infection in humans is surgical removal of lung granulomas and nodules under the skin; this treatment is also curative. In many cases, no treatment with medicines is necessary.

Control and prevention: Dirofilariasis can be prevented by avoiding mosquito bites in areas where mosquitoes may be infected with *Dirofilaria* larvae. The risk of such mosquito bites can be reduced by leaving as little skin exposed as possible, by the use of insect repellent when exposed to mosquitoes, and by sleeping under an insecticide-treated bed net in areas where *Dirofilaria*-infected mosquitoes bite at night and have access to sleeping areas.

Dirofilariasis in Human

Humans become infected with *Dirofilaria* through mosquito bites. In persons infected with *D. immitis*, dying worms in pulmonary artery branches can produce granulomas (small nodules formed by an inflammatory reaction), a condition called "pulmonary dirofilariasis." The granulomas appear as coin lesions (small, round abnormalities) on x-rays. Most persons with pulmonary dirofilariasis have no symptoms. People with symptoms may experience cough (including coughing up blood), chest pain, fever, and pleural effusion (excess fluid between the tissues that line the lungs and the chest cavity). Coin lesions on chest x-rays are not diagnostically specific for pulmonary dirofilariasis. Therefore, discovery of these lesions has led to invasive diagnostic procedures to exclude other, more serious causes, including cancer. Rarely, *D. immitis* worms have been found in humans in brain, eye, and testicle (figure 102).

Humans acquire the infection in the same manner as dogs, by the bite of a mosquito, but it is probable that most of the infective larvae die shortly after, with the infection resolving unrecognized and without causing any specific symptom. No predisposing factors are known to explain why in some cases larvae may develop further. After the bite of an infective mosquito, a stronger reaction with erythema, swelling and pruritus lasting 5–8 days. In most of the cases a single

worm develops, probably because the stimulation of the immune system prevents the development of others.

In rare cases the worm may develop to a mature adult and even fertilized worms releasing microfilariae have been described, especially in immunosuppressed patients, which in very rare cases may even reach the bloodstream.

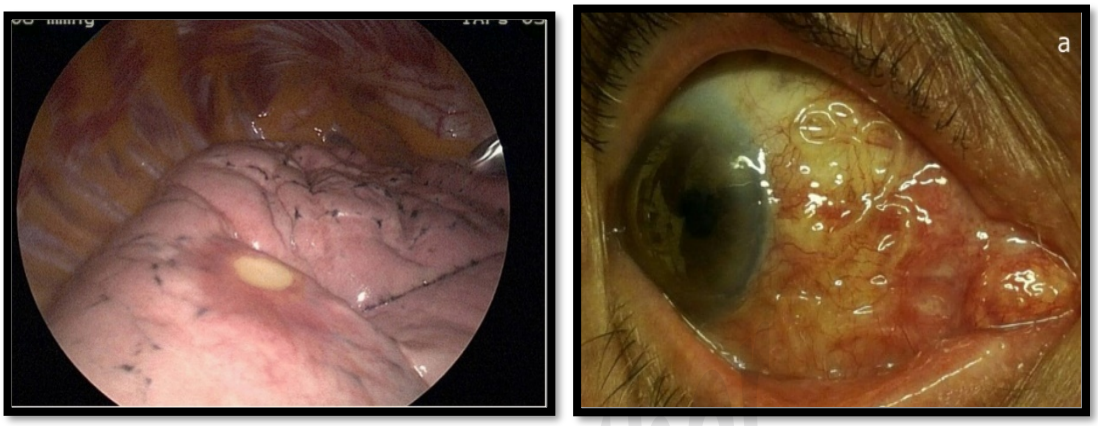


Figure 102. Human pulmonary and dirofilariasis (*Dirofilaria immitis*)

***Dirofilaria repens*:** the developing stages of *D. repens* migrate subcutaneously for weeks up to several months in several parts of the body, usually with mild and unrecognized symptoms and sometimes causing irritation and erythema due to larva migration. The larva may reach the eyes and becoming visible through the subconjunctiva (figure 103). Local surgery can be performed to remove a long whitish worm from the site of infection.

Other sites of infection: After weeks to several months from the infection, *D. repens* may stop to migrate and form a nodule of about one centimeter. In most cases, the nodules develop subcutaneously. Nodules have been reported in various human body areas and tissues, mostly in the superficial tissues of the facial regions, as perioral and periorbital tissues, forehead, skin of the lower leg, soft tissues of the hand or finger, subcutaneous tissue of the hypogastrium and of the neck.

Scrotum and testicles are other predilection site infested with larvae and, to a lesser extent, the breasts of women. Various reasons have been hypothesized for

these preferences, such as lower body temperature of these areas, higher awareness of patients for these body parts or a tropism of *D. repens* due to abundance concentrations of sexual hormones. The nematodes could reach lymph nodes, the abdominal cavity, muscles and even the dura. However, *D. repens* if left untreated may survive for up to one year.

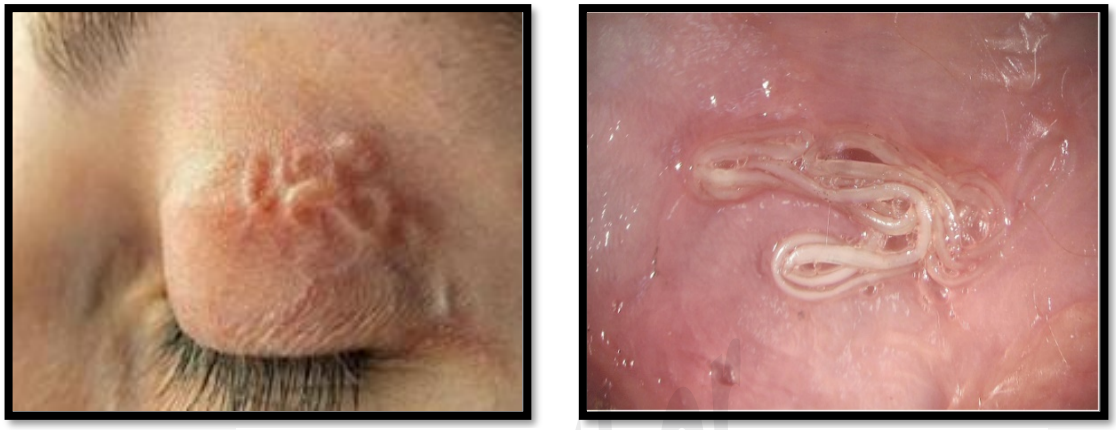


Figure 103. Eyes and subcutaneous tissues of human infected with *Dirofilaria repens*

Family: Ascaridae

Introduction: Ascariasis is the infestation of pigs by the roundworm, *Ascaris lumbricoides* or *A. suum*, which can cause pneumonia, hepatitis, and ill thrift. Mature ascarids are 15-40 cm long, thick bodied, round worms. A mature ascarid (prepatent period of seven to eight weeks) can lay almost two million eggs per day. Adults live in a host for about six months before spontaneous expulsion begins but may survive a year or longer. Heavy infections can result in hundreds of ascarids in the intestine. Ascarid eggs are thick shelled, yellowish brown, almost spherical, 50-80 by 40-60 µm, and are coated with a sticky proteinaceous cover. The eggs are resistant to environmental degradation and disinfectants but can be destroyed by prolonged exposure to full sunlight.

Hosts: Humans and swine are the major hosts for *Ascaris*. Natural infections with *A. lumbricoides* sometimes occur in monkeys and apes (figure 104). Occasionally, *Ascaris* sp. eggs may be found in dog feces. This does not indicate true infection but instead spurious passage of eggs following coprophagy.

Ascaris Species of Animals

There are several ascarids in animals, which live together or close to humans:

1- *Ascaris suum* (pigs) – ♀ up to 40 cm

2- *Parascaris equorum* (horses, equids) – ♀ up to 40 cm

3- *Toxascaris leonina* (dogs, cats) – ♀ up to 12 cm (

4- *Baylisascaris procyonis* (raccoons, dogs) – ♀ up to 12 cm

Ascaris lumbricoides

Geographical distribution: Infection is ubiquitous but is most common in the tropics, in areas of poor sanitation, and wherever human feces are used as fertilizer.

Morphology: The adult is cylindrical in shape, creamy-white or pinkish in color. The female averages 20-35cm in length, the largest 49cm. While the male is smaller, averaging 15-31cm in length and distinctly slenderer than the female. The typical curled tail with a pair sickle like copulatory spines.

On the tip of the head there are three lips, arranged as a Chinese word "品". They have a complete digestive tract.

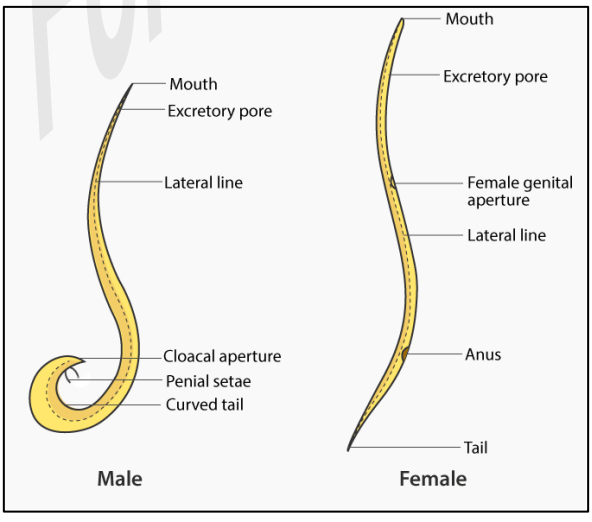


Figure 104. *Ascaris lumbricoides*

Reproductive organs are tubular. Male has a single reproductive tubule. The female has two reproductive tubule and the vulva is ventrally located at the posterior part of the anterior 1/3 of the body.

Biology: Adult worms live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the feces. Unfertilized eggs may be ingested but are not infective. Larvae develop to infectivity within fertile eggs after 18 days to several weeks, depending on the environmental conditions (optimum: moist, warm, shaded soil). After infective eggs are swallowed, the larvae hatch and invade the intestinal mucosa, which carried via the portal and subsequently to the lungs. The larvae mature penetrate the alveolar walls and ascend the bronchial tree to the throat and swallowed. Upon reaching the small intestine, they develop into adult worms. Between 2 and 3 months are required from ingestion of the infective eggs to oviposition by the adult female. Adult worms remain a live up to more one year (figure 105).

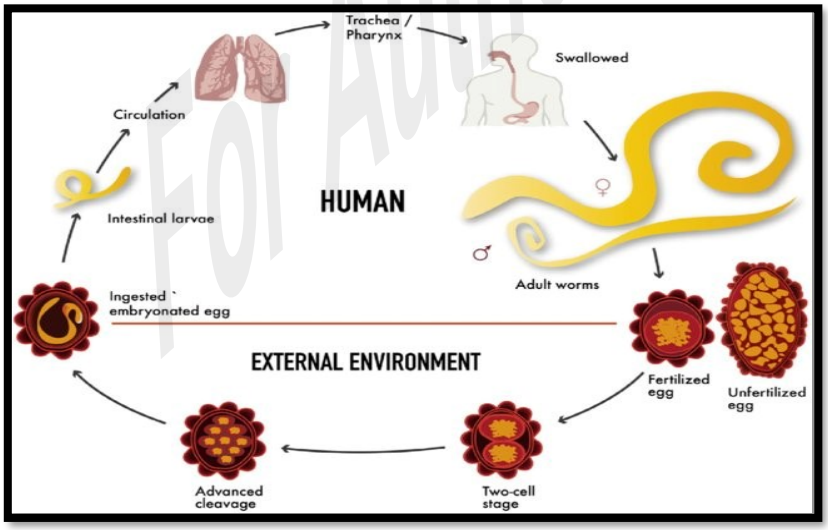


Figure 105. Life cycle of *Ascaris lumbricoides*

Eggs: Described according five aspects: size, color, shape, shell and content. Three types of the eggs: fertilized eggs, unfertilized eggs and decorticated eggs:

1. Fertilized eggs: broad oval in shape, brown in color, range from 45 to 75 µm in length. The shell is thicker and consists of Ascaroside (glycolipids), chitinous layer, fertilizing membrane and mamillated albuminous coat-stained brown by

bile. The content is a fertilized ovum. There is a new-moon (crescent) shaped clear space at each end inside the shell (figure 108).

2. Unfertilized egg: Longer and slender than a fertilized egg (up to 90 µm in length). The chitinous layer and albuminous coat are thinner than those of the fertilized eggs without ascaroside and fertilizing membrane. The content comprised from numerous refractile granules (figure 106).

3. Decorticated eggs: Both fertilized and unfertilized eggs sometimes may lack their outer albuminous coats and are colorless (figure 107-111).

The eggs develop and hatch to larvae after few days (figure 112, 113)

Figure 106. Unfertilized egg

Figure 107. Unfertilized, decorticated egg

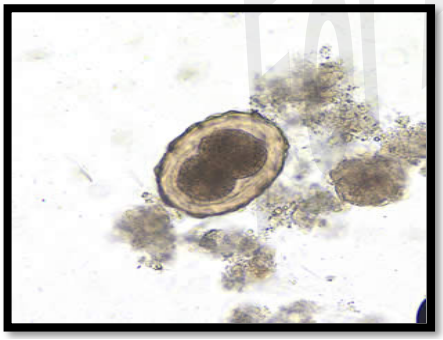


Figure 108. Fertilized egg early embryo in early stage of development

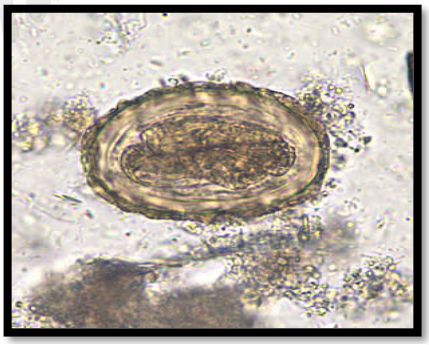


Figure 109. Fertilized eggs in a wet mount. A larva is visible in the egg

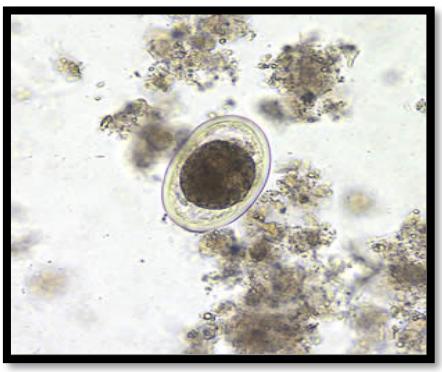


Figure 110. Decorticated, fertile egg

Figure 111. Fertilized egg

Figure 112. Egg with larva inside

Figure 113. Larva hatching from an egg

Clinical signs: heavy infection in children cause stunted growth due to a malnutrition. High numbers of worms lead to abdominal pain and intestinal obstruction and potentially perforation when intensity infections increased. Migrating adult worms may cause symptomatic occlusion of the biliary tract, appendicitis, or nasopharyngeal expulsion, particularly in infections involving a single female worm.

Symptoms: Individuals cannot detect the signs of this parasite in the beginning. However, when the infestation within the small intestine develops, patients can notice:

1. Loss of appetite, abdominal pain, vomiting, presence of worms in stool, weight loss, diarrhea, nausea and irregular bowel movement.
2. in advance stages where these parasites travel to the lungs, an individual may notice the following symptoms: Fever, gagging cough, bloody mucus, discomfort in chest, wheezing, and breathing problems.

Diagnosis: The primary process to diagnose the presence of *Ascaris suum* life cycle in a human is by collecting a stool sample. However, this process may not work during early infestation stages.

On the other hand, imaging tests are always helpful to determine the number of eggs inside a person. MRI, endoscopy, CT scan, ultrasound, etc. are used for this purpose.

Treatment: Albendazole and Mebendazole are the drugs of choice for treatment of *Ascaris* infections, regardless of the species of worm. The course of treatment takes 1–3 days only.

Prevention: maintain good hygiene is the best way to avoid the infection with the *Ascaris*. Washing hands before eating and cleaning fruits and vegetables can protect from this disease.

Parascaris equorum

Parascaris equorum has a direct life cycle without intermediate hosts. Adult females lay eggs in the small intestine of the host that are shed with the feces. A single female worm can shed more than 150.000 eggs daily.

Geographical distribution: The epidemiology of Parascarosis is the extreme persistence of the infective stage in the environment and the predominance of infection in juvenile horses. Larvated *Parascaris* eggs remain viable for around 10 years, so one patent infection on a premise can affect several future generations of foals. Animals acquire infection by receiving larvated eggs from the environment.

Morphological description: the worm is quite large, cylindrical in shape, and has a cuticle composed from three layers made of collagen and other compounds that protect the worm from the acids in the digestive tracts. The adult male ranges from 15-28 cm in length. The females are much larger and can grow up to 50 cm. It has three very large lips. Each of these lips has a transverse groove or labial sinus on the lateral margins, which divide the lip into apical and basal regions

(figure 114). The eggs are almost spherical and brownish in color. The eggs contain a zygote.

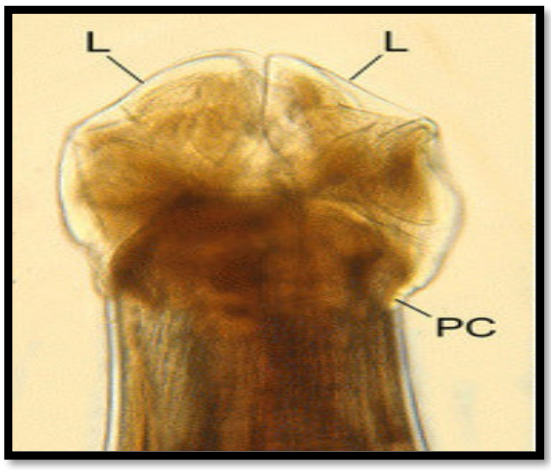


Figure 114. anterior part of *Parascaris equorum* (**L**) lips set off from the rest of the body by a deep post-labial constriction (**PC**)

Biology:

A- Hatch to 3rd larval stage in the stomach or small intestine and penetrate intestinal veins.

B- Larvae reach liver via portal vein, migration through liver tissue and penetration of liver veins.

C- Larvae reach lung via vena cava and right heart, penetration into lung alveoli and migration via trachea and pharynx to small intestine, molting to L4 and L5 before becoming adult worm. (Figure 115).

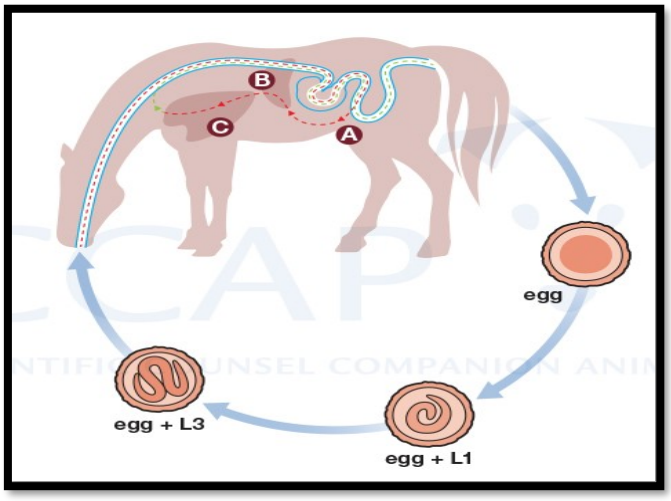


Figure 115. Life cycle of *Parascaris equorum*

Symptoms: increase heart rate, dehydration, hyper- or hypoperistalsis, positive gastric reflux, hyperemic mucous membranes, incredible pain, and limited response to analgesics.

Diagnosis: microscopic examination of the faecal samples indicate presence of the Ascarid eggs. Another technique is ultrasonography to visualize eggs in the digestive system, with some pathological changes also seen such as thickness of the mucosal walls and dilated loops of small intestine.

Treatment: Benzimidazoles group are advised to treat equine ascarids. fluid therapy must be given as a supportive treatment. Other treatment include:

- Pain medication
- Correction of acidosis/alkalosis
- Anti-inflammatory therapy
- Antibiotics

Control and Prevention: maintaining stables free of manure can decrease possibility of parasite transmission in horses. Rotation of pastures, particularly by putting animals of other species into the rotation, can also reduce the amount of parasite infestation.

Family: Oxyuridae

Members of Oxyuridomorpha are called pinworms. It is a slender with having a sharp-pointed tail. Adult oxyurids has a greater taxonomic range of hosts in compare to other nematode species. Their definitive hosts are invertebrate and vertebrate animals. Pinworms of the superfamily Oxyuroidea are common in mammals, birds, reptiles, and amphibians but rare in fish. One species, *Enterobius vermicularis,* is the most common nematodes of humans.

Others, Oxyuridomorpha is a member of a larger clade of entirely parasitic taxa (clade III) that includes Ascaridomorpha, Spiruromorpha, and Rhigonematomorpha. Six members of Oxyuridomorpha were originally grouped

together because they parasitize the posterior gut of animals and have a posterior pharyngeal bulb.

Enterobius vermicularis

Geographical distribution: Pinworms well-adapted for transmission among groups of individuals living in close proximity, such as families, schoolchildren, and in certain institutional settings including day care centers and the prevalence appears to be greater in temperate regions.

Transmission: Eggs remain viable in cold weather, moist conditions for several days. The most common objects of infection are by insertion of soiled fingers or other objects in the mouth or through use of contaminated bedding, towels, and other such objects (fomites). Humans can inhale and subsequently swallow airborne eggs, or eggs may remain in the nose until they hatch.

Contrary to popular belief, pinworms cannot be transmitted by dogs and cats because these animals are free of pinworms. But their fur can become contaminated with *E. vermicularis* eggs from their environment, and thereby serve as another potential source of infection. *Enterobius vermicularis* and *E. gregorii* have been reported from captive chimpanzees.

Morphology: In addition to *Enterobius vermicularis,* another species, *E. gregorii,* has been described from humans, but *E. gregorii* is likely only a morphological variant or developmental phase of *E. vermicularis*. Both sexes of *E. vermicularis* have three lips surrounding the mouth, followed by a cuticular inflation of the head. Males have a single spicule, which is 100 μm to 141 μm long.

Males are 1 mm to 4 mm long and have their posterior ends strongly curved ventrally. Conspicuous caudal alae are supported by papillae. Females measure 8 mm to 13 mm long and have the posterior end extended into a long, slender point, giving pinworms their name. The vulva opens between the first and second thirds

of the body. Gravid uteri contain thousands of eggs, that are elongated-oval and flattened on one side with a thin shell
elongated-oval measuring 50 μm to 60 μm by 20 μm to 30 μm.

Diagnosis: Positive diagnosis can be made only by finding eggs or worms on or in the patient. Ordinary fecal examinations often give false negatives because relatively few eggs are deposited within the intestine and passed in feces. Heavy infections can be revealed by examining the perianus closely under bright light, during the night or early morning.

When adults cannot be found, eggs often can be, as they are left behind on the perianal skin. A short piece of cellophane tape, held against a flat, wooden applicator or similar instrument, sticky side out, is pressed against the junction of the anal canal and the perianal region. The tape is then reversed and stuck to a microscope slide for observation.

Biology: Adult worms congregate mainly in the ileocecal region of the intestine but developing juveniles and young adults may be found throughout the small intestine. They attach themselves to the mucosa where they presumably feed on epithelial cells. Gravid females migrate within the lumen of the intestine, commonly passing out of the anus onto perianal skin.

One worm may deposit between 4600 to 16,000 eggs. Females die soon after oviposition, and males die soon after copulation. Consequently, many more females than males are recovered from hosts. When laid, each egg contains a partially developed juvenile, which can develop to infectivity within six hours at body temperature. Most oxyurids have been reported to have two molts during development of juveniles within eggs.

Most often the eggs, are swallowed, and they hatch in the duodenum. They slowly move down the small intestine, molting twice to become adults by the time they arrive at the ileocecal junction. Attached eggs may hatch and juveniles may enter the anus and hence to the intestine in a process known as **retrofection** (or

retroinfection). However, with respect to maintaining an infection in a single host. In the figure 116 illustrate the sketch of the life cycle of *Enterobius vermicularis* in humans.

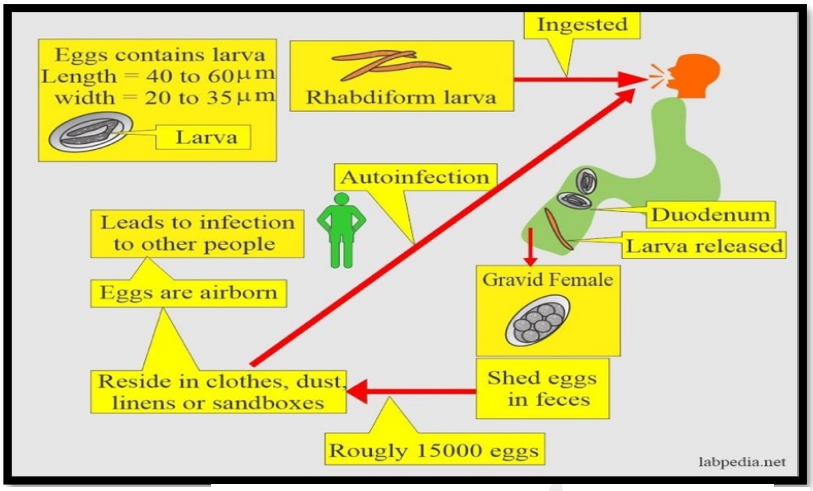


Figure 116. Life cycle of *Enterobius vermicularis*

Pathogenesis: due to the infection damage caused by extraintestinal migration of adult worms, and damage resulting from egg deposition around the anus. Ulcerations of intestinal mucosa from attachment of adults may lead to mild inflammation and probably bacterial infection. Movements of females out of the anus to deposit eggs, especially when the patient is asleep, lead to a tickling sensation of the perianus, causing the patient to scratch. Eggs present on the perianal skin cause dermatitis and itching.

A variety of other symptoms have been ascribed to heavy pinworm infection in children. These symptoms include nervousness, restlessness, irritability, loss of appetite, bed wetting, grinding of the teeth, perianal pain, nausea, and vomiting.

Treatment and control: Mebendazole or Albendazole are effectively used for treatment. Treatment should be repeated after two weeks. Personal hygiene is most important, all bed linens, towels, and undergarments should be washed with hot water to minimize the possible infection

Strongyloididae

Geographical distribution: *Strongyloides* species of this family are among the smallest nematode parasites of humans, males are significantly smaller than

Trichinella spp. More than 50 *Strongyloides* species have been described in mammals. *Strongyloides stercoralis* is the most common parasite infecting humans. The species *S. fuelleborni* also infected humans, found in Africa.

Transmission: *Strongyloides stercoralis* infects dogs, cats, and some other mammals, and strains isolated from humans vary in infectivity among multiple hosts. Other species include *S. ratti* and *S. venezuelensis* in rats, *S. ransomi* in swine, and *S. papillosus* in sheep. Fewer species have been described in birds, amphibians, and reptiles.

The parasitic generation consists only of parthenogenetic females according to most authorities. In contrast, *Parastrongyloides,* the sister group of *Strongyloides* has both parasitic males and females. The free-living generation of both *Strongyloides* spp. and *Parastrongyloides* spp. consists of both males and females.

Morphology: Parthenogenetic females reach a length of 2.0 mm to 2.5 mm. The buccal capsule of both sexes is small, and the animals possess a long, cylindrical esophagus that lacks a basal bulb. The vulva is in the posterior third of the body; uteri contain only a few eggs at a time. Both sexes of free-living adults have a rhabditiform esophagus (figure 117).

Males are up to 0.9 mm long and 40 µm to 50 µm wide. Males have two simple spicules and gubernaculum; their pointed tail is curved ventrally. Females are stout and have a vulva that is located at the midbody; uteri generally contain several eggs.

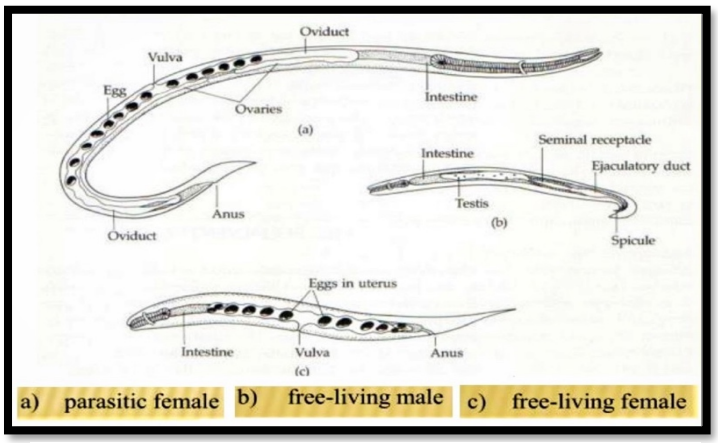


Figure 117. *Strongyloides stercoralis* forms

Biology: Parasitic females burrow their anterior ends into submucosa of the small intestine (figure 118). They are found occasionally in the respiratory, biliary, or pancreatic system. They produce several embryonated eggs a day and release them into gut lumen. They hatch during passage through the gut, and juveniles escape to the lumen. These J1s are 300 μm to 380 μm long and are usually passed with feces. Juveniles either develop into free-living adults or become infective, filariform J3s with a developmental arrest; it measures 490 μm to 630 μm long. Filariform juveniles develop no further unless they gain access to a new host by skin penetration or ingestion. If infection is by skin penetration, then juveniles must undergo a tissue migration to reach the small intestine. Juveniles are carried by blood circulation to the lungs and then to the trachea.

In dogs *S. stercoralis* juveniles seem to "scramble" from infection site to intestine. After a period of time, they move through olfactory bulbs and nasal mucosa, reaching the small intestine by 40 to 50 hours after infection. Given the lung symptoms sometimes seen in humans infected with *S. stercoralis,* which can be quite severe, significant numbers of juveniles must migrate through lung tissue.

Free-living adults can produce successive generations of free-living adults. Both parasitic and free-living females can produce juveniles that become filariform, infective J3s and juveniles that mature into free-living adults. The mechanism that determines whether a given embryo will become a free-living female or a parasitic female is a warm temperature (34°C) in which juveniles develop. If the ambient temperature is less than 34°C, they tend to molt to J4 and become free-living females. When juveniles have time to molt twice during movement in the digestive tract, they may penetrate lower gut mucosa or perianal skin, complete migration, and mature. This process is called **autoinfection.**

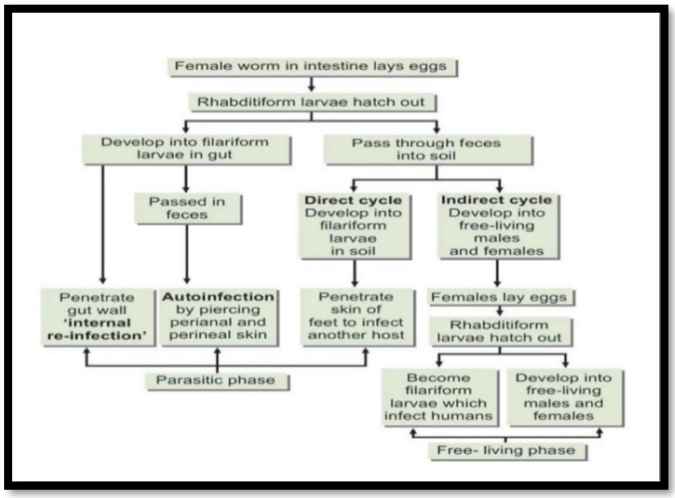


Figure 118. Life cycle of *Strongyloides* spp.

Geographical distribution: People typically become infected with *S. stercoralis* by contacting juveniles in contaminated soil or water. Transmammary infection can occur in dogs. This infection primarily occurs in humans within tropical regions but extends well into temperate zones on several continents. Strongyloidiasis is the most prevalent under conditions of low sanitation.

Pathogenesis: the infection with *S. stercoralis* is most commonly asymptomatic, but chronic strongyloidiasis can result in colitis. Effects of Strongyloidiasis may be described in three stages: **invasive**, **pulmonary**, and **intestinal**. Penetration of skin by filariform juveniles results in slight hemorrhage, swelling, and itching at the site of entry. If pathogenic bacteria are introduced with juveniles, inflammation may result as well. Migration of larvae through the lungs may be manifested wheezing, migratory pneumonia or even cough. Involvement of the intestines may be associated with abdominal pain, diarrhea, weight loss, malabsorption, nausea, GI bleeding or vomiting.

Diagnosis and:

1-A direct fecal smear is often effective in cases of massive infections.

2-Special isolation or concentration techniques for juveniles.

3-Technique of culturing fecal samples on nutrient agar is most effective.

4-Serodiagnosis by ELISA (detection of *S. stercoralis* antigens in patient serum).

5-Detection of *Strongyloides* DNA in feces by real-time PCR.

Treatment: Treatment includes: Ivermectin, and Albendazole. All patients with Strongyloidiasis should be treated, and the cure rate is higher with ivermectin than albendazole.

Ancylostomatidae

Members of this family are commonly known as **hookworms**. They inhabited the small intestine, attaching to the mucosa and feeding on blood or tissue fluids.

Morphological characterizations: most of their species are stout, and the anterior end is curved dorsally, giving the worm a hooklike appearance. The buccal capsule is large and heavily sclerotized and usually is armed with cutting plates, teeth, lancets, or a dorsal cone. Lips are reduced (figure 119). The esophagus is robust, with a swollen posterior end, giving it a club shape. It is mainly muscular, corresponding to its action as a powerful pump. Esophageal glands are extremely large and are mainly outside the esophagus, extending posteriorly into the body cavity. Cervical papillae are present near the rear level of the nerve ring.

Males have a conspicuous copulatory bursa, consisting of two broad lateral lobes and a smaller dorsal lobe, all supported by fleshy rays. The number and general pattern of rays is also common to other male nematodes in Rhabditomorpha, including free-living species. Spicules are simple, needlelike, and similar. A gubernaculum is present. Females have a simple, conical tail. The vulva is post equatorial, and two ovaries are present. The total production of eggs is several thousand per day.

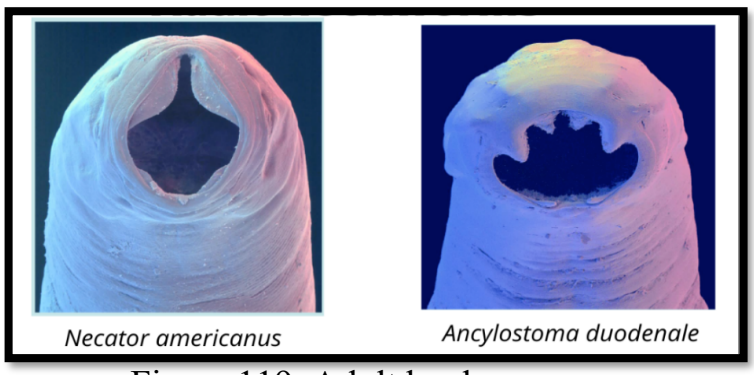


Figure 119. Adult hook worms

Life cycle summarized as below:

1-Eggs are passed in the stool

2-Under favorable conditions (moisture, warmth, shade), larvae hatch in 1 to 2 days and become free-living in contaminated soil. These released larvae grow in the feces and/or the soil.

3-After 5 to 10 days (and two molts) they become filariform (3rd stage) larvae that are infective.

4-The infective larvae can survive 3 to 4 weeks in favorable environmental conditions. Once contact with the human, typically bare feet, the larvae penetrate the skin and carried through the blood vessels to the heart and then to the lungs. They penetrate into the pulmonary alveoli, ascend the bronchial tree to the pharynx, and are swallowed.

5-The larvae reach the jejunum of the small intestine, where they finally reside and mature into adults. Adult worms live in the lumen of the small intestine, where they attach to the intestinal wall with resultant blood loss by the host (figure 120).

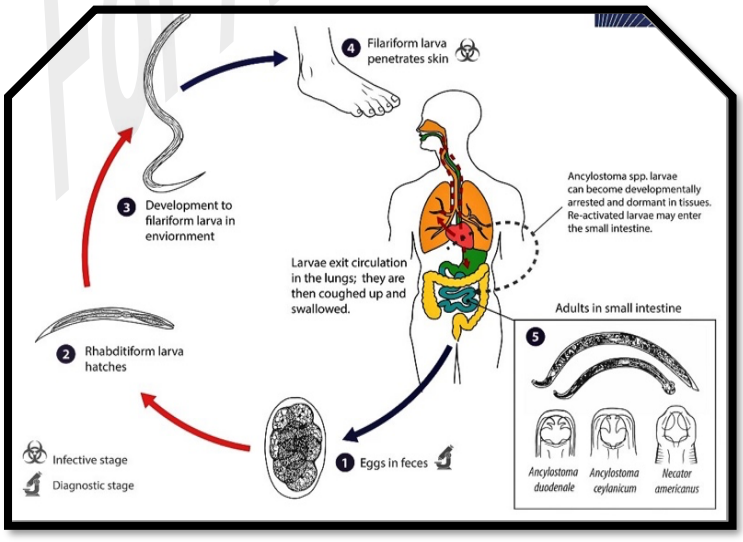


Figure 120. Life cycle of intestinal hookworm

Some *A. duodenale* larvae, following penetration of the skin, can become dormant (inactive). These larvae are capable of re-activating and establishing

patent, intestinal infections. The infection by *A. duodenale* probably occur by the oral and the transmammary route. *A. ceylanicum* and *A. caninum* infections may also be acquired by oral ingestion. *A. caninum*-associated eosinophilic enteritis is believed to result following oral ingestion of larvae, not percutaneous infection. *N. americanus* does not appear to be infective via the oral or transmammary route.

Other species of hookworms that infect both humans and animals including A. *braziliense*, *A.caninum*, *Uncinaria stenocephala,* causing a form of larva cutaneous migration.

Necator americanus

The worm causes a considerable economic loss. *Necator americanus* has a pair of dorsal and a pair of ventral cutting plates surrounding the anterior margin of the buccal capsule. A pair of subdorsal and a pair of subventral teeth are near the rear of the buccal capsule. The duct of the dorsal esophageal gland opens on a conspicuous cone that projects into the buccal cavity (figure 121).

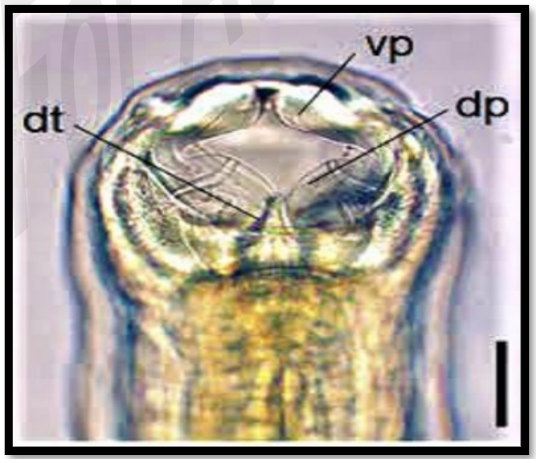


Figure 121. Anterior end of *Necator americanus*
(dt)dorsal tooth (vp) ventral cutting plate
(dp)dorsal cutting plate

Characteristic of male and female

Males are 5 mm to 9 mm long and have a bursa important diagnostic feature for the genus. The needlelike spicules have minute barbs at their tips fused distally.

Females are 9 mm to11 mm long and their vulva is located in about the middle of their body. They produce about 5000 to 10,000 eggs per day, and the normal life span is three to five years (figure 122).

Geographical distribution: high dominated in the tropical and subtropical regions, *N. americanus* is the most common species in humans in most of the world, accounting for about 85% of infections.

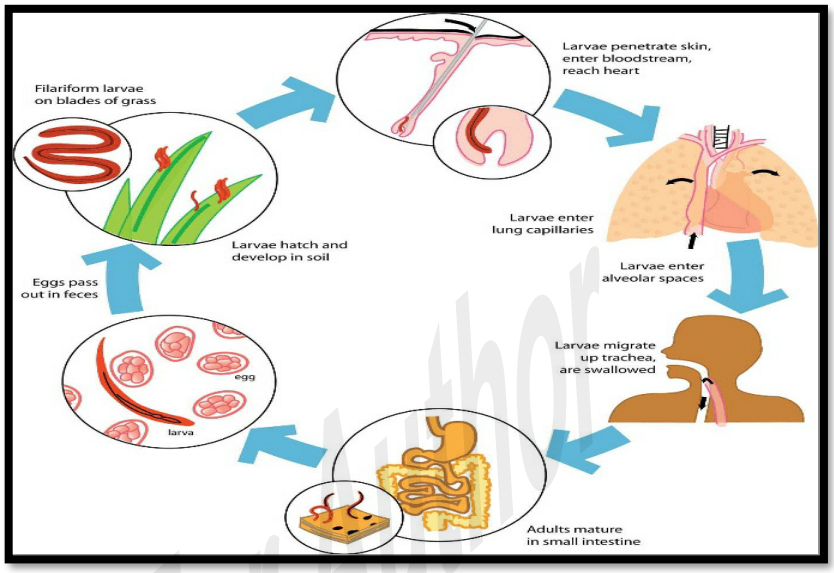


Figure 122. Life cycle of *Necator americanus*

Ancylostoma duodenale

Ancylostoma duodenale is the human hookworm and the most common parasitic infection in countries with poor access to adequate and clean water. *A. duodenale* along with other soil-transmitted helminths (STH) are transmitted through contact with contaminated soil.

Geographical distribution: It is estimated that more than 1.5 billion people worldwide are at risk for infection with *Ancylostoma* and other STH. Most of the infection have been reported in Asia and the Pacific where tropical climate, overcrowded population, poor hygiene, and poor sanitation are associated factors. *Ancylostoma,* along with the other hookworms, cause debilitation and sometimes death in a chronic infection. The highest at-risk population to

contract *Ancylostoma* infections are the pre-school and school-aged children and tourists visited tropical countries. Additionally, people in close contact with dogs and cats are highly risk to acquire the infection with *Ancylostoma*. The occurrences of *Ancylostoma* infection are inextricably linked to seasonal distribution, during the summer-autumn period.

Morphological characterizations: The anterior margin of the buccal capsule has two ventral plates, each with two large teeth that are fused at their bases (figure 123). A pair of small teeth is found in the depths of the capsule. The duct of the dorsal esophageal gland runs in a ridge in the dorsal wall of the buccal capsule and opens at the vertex of a deep notch on the dorsal margin of the capsule.

Adult males are 8 mm to 11 mm long and have a bursa characteristic for the species. The needlelike spicules have simple tips and are never fused distally.

Females are 10 mm to 13 mm long, with the vulva located about a third of the body length from the posterior end. A single female can lay from 10,000 to 30,000 eggs per day, and the normal life span is one year.

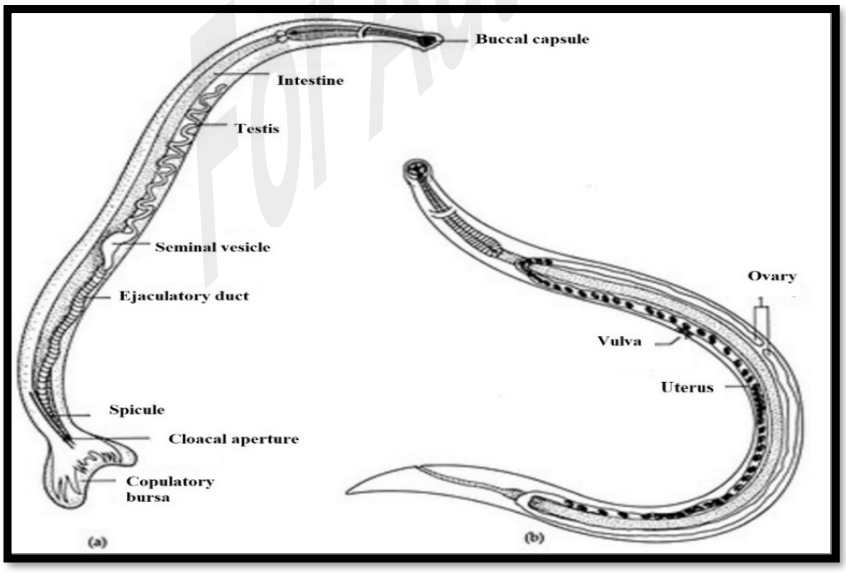


Figure 123. Male and Female of *Ancylostoma duodenale*

Life cycle outlined as below:

1- After the eggs of *Ancylostoma* pass from the stool of their host, the eggs hatch into larvae. In favorable conditions, this occurs in 1 to 2 days (figure 124).

2- The hatched rhabditiform (non-infective) larvae grow in the feces or soil for 5 to 10 days and mature into filariform (infective) larvae.

3- The filariform larvae can penetrate the human skin via hair follicles, enter the lymphatic system, and migrate to the heart and lungs.

4- In the lungs, the larvae penetrate the pulmonary alveoli, where they ascend from the bronchial tree to the pharynx, can be coughed up, swallowed, and reach the small intestine where they mature into male or female blood-feeding-adults.

5- The mating adult worms can produce thousands of eggs, and the cycle starts over.

6- The filariform larvae can spread via oral ingestion and trans-mammary route where the larvae directly mature in the small intestine or stay dormant in human skeletal musculature.

7- The dormant larvae have been proven responsible for vertical transmission during breastfeeding and are possibly the cause of transplacental infection.

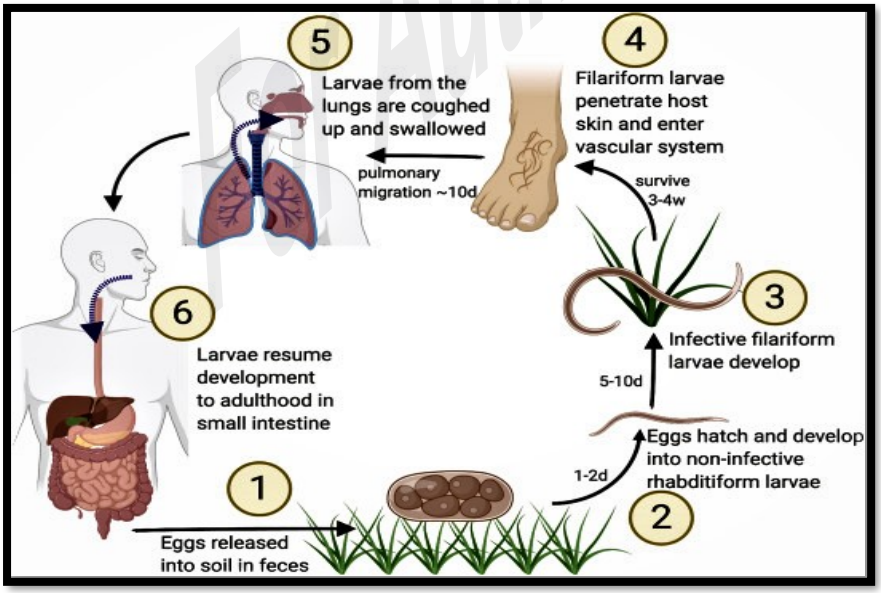


Figure 124. Life cycle of *Ancylostoma duodenale*

Diagnosis:

- People can turn out to be infected when walking barefoot since hookworm larvae reside in the earth and can enter the skin. At earliest, people may have

prickly itchiness where the larvae enter the skin, then high chills, coughing, and abdominal soreness, loss of hunger, and dehydration problems.

- Persistent infections can bring loss of blood and anemia that is occasionally severe enough to cause exhaustion and heart failure and pervasive swelling.
- The detect of infection by identifying hookworm eggs in a stool test.

Pathogenesis: Itching and a localized rash are often the first signs of infection. These symptoms occur when the larvae penetrate the skin. A person with a light infection may have no symptoms. A person with a heavy infection may experience abdominal pain, diarrhea, loss of appetite, weight loss, fatigue and anemia. The physical and cognitive growth of children can be affected.

Treatment: *A. duodenale* can be treated with three treatment options: mebendazole, albendazole, and benzimidazoles. Pyrantel pamoate is an option. In severe cases of anemia, blood transfusion may be required.

Control:1. Education. 2. Better sanitation practices. 3. Controlled disposal of human feces is essential. 4. Wearing shoes in prevalent areas can trim down the prevalence of infection. 5. Clean neighboring is of utmost importance. Risk of developing cutaneous larva migrants can be reduced by avoiding direct skin contact with potentially infested beach sand where dogs or cats have defecated and administration anti-worm for pets.

Other Hookworms Reported in Humans

Zoonotic hookworms are hookworms that live in animals and can also transmit to humans. Dogs and cats can become infected with several hookworm species, including *Ancylostoma brazilense*, *A. caninum*, *A. ceylanicum*, and *Uncinaria stenocephala.* The eggs of these parasites are shed in the feces of infected animals and can end up in the environment, contaminating the ground where the animal defecated. People become infected when the zoonotic hookworm larvae penetrate unprotected skin, especially when walking barefoot or sitting on contaminated soil or sand. This can result in a disease called cutaneous larva migrants (CLM),

when the larvae migrate through the skin and cause inflammation (figure 125). The CLM is impossible to survive more than 5 – 6 weeks in the human host.

Hookworm Disease the distinction between hookworm infection and hookworm disease is important. Far more people are infected with hookworms than exhibit disease symptoms. The presence and severity of disease depend strongly on three factors:

1-Number of worms present.

2-Species of hookworm

3-Nutritional condition of the infected person.

In general, fewer than *N. americanus* in a person will cause no symptoms, 25 to 100 worms lead to mild symptoms, 100 to 500 produce considerable damage and moderate symptoms, 500 to 1000 result in severe symptoms and grave damage, and more than 1000 worms causes very grave damage that may be accompanied by drastic and often fatal consequences.

Because *Ancylostoma* spp. suck more blood than *N. americanus,* fewer worms cause greater disease; for example, 100 worms may cause severe symptoms. The clinical disease is intensified by a nutritional condition and lower host immunity response.

Figure 125. Cutaneous larval migrant (CLM)
in a person's foot

Treatment: Mebendazole or Albendazole are drug choice used for treatment. Dietary supplementation is recommended.

How can prevent hookworm infection?

Protective measures through wearing shoes to avoid skin contact with sand or soil will prevent infection with zoonotic hookworms, especially for travelers. Deworming cats and dogs by anthelminthics should be considered. Disposal off animal feces can prevent eggs from hatching and contaminating soil.

Trichostrongylidae

Their species inhabit the digestive system of livestock causing great economic losses, and rare cases causing disease in humans.

General characterization: Trichostrongylid parasites are small, very slender worms, and have a rudimentary buccal cavity. Lips are reduced or absent, and teeth rarely are present. The cuticle of the head may be inflated. Males have a well-developed bursa, and spicules vary depending on species. Females are considerably larger than males. The vulva is located anywhere from preequatorial to near anus.

The mature worm release thin-shelled eggs that are in the morula stage. No intermediate host is required; eggs hatch in soil or water and develop directly into infective J3s. Some infections may occur through skin, but as a rule juveniles must be swallowed with contaminated food or water. Many trichostrongylids undergo exsheathment. Large numbers of juveniles may accumulate on heavily grazed pastures, causing serious or even fatal infections in ruminants. A host usually is infected with more than species and severe pathogenesis results from the cumulative effects of all the worms. Drugs choice to eliminate trichostrongyles is albendazole, mebendazole, and thiabendazole.

Haemonchus contortus

Barber's pole worm *H. contortus* is a potentially harmful roundworm of sheep which cause Haemonchosis. In some cases, large worm burdens can develop very rapidly and cause sheep deaths without warning.

Geographical distribution: Climatic conditions determine where barber's pole worms occur and when they are most prevalent throughout the year. The development of eggs and larvae is limited to areas and seasons where pastures are moist during the warm months. The larvae can survive on pasture, particularly during cool conditions and occasionally affect sheep at other times of the year.

Morphological characteristics: The worms are up to 2.5 (cm) long and occur in the abomasum or fourth stomach of sheep and goats. Prominent cervical papillae are found near the anterior end. It is cuticle consist of three layers, which has a principal function to protect the worm against enzymes of the digestive system. The female is longer (18 to 30 mm) than the male (10 to 20 mm). Have a red and white striped appearance, hence the name ‘barber's pole’. It is cylindrical and has a striking reddish appearance due to its blood-feeding habit. The vulva of the female is usually covered by a linguiform process (vulva flap) which is usually large and very prominent (figure 126).

Figure 126. Female Flap Vulvar of *Haemonchus contortus*

The males possess developed copulatory bursa, with an asymmetric dorsal lobe and a Y-shape dorsal ray. The spicules are 460-506 µm long, each provided with a small barb near its extremity (127).

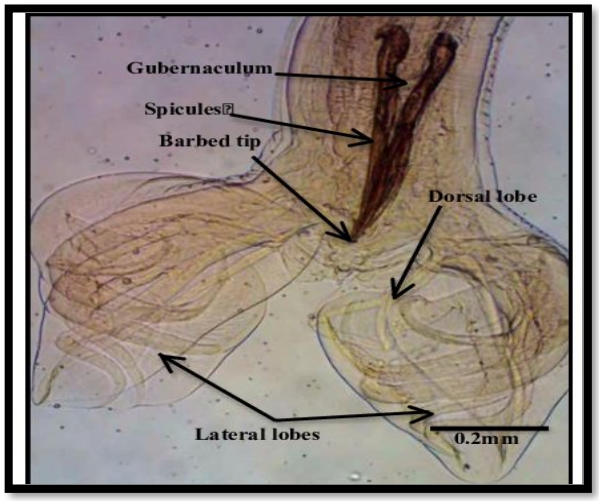


Figure 127. Male Copulatory Bursa of *Haemonchus contortus*

Biology: The adult female worm can release between 5,000 and 10,000 eggs, which are passed out in the faeces (figure 128). Eggs then develop in moist conditions in the faeces and continue to develop into the L1 (rhabditiform), and L2 juvenile stages by feeding on bacteria in the dung. The L1 stage usually occurs within four to six days under the optimal conditions of 24–29 °C (75–84 °F). The L2 rhabditform sheds its cuticle and then develops into the L3 filariform infective larvae. The L3 form has a protective cuticle, but under dry, hot conditions, survival is reduced. Small ruminants become infected when they graze and ingest the L3 infective larvae. The infective larvae pass through the first three stomach chambers to reach the abomasum. There, the L3 shed their cuticles and burrow into the internal layer of the abomasum, where they develop into L4, usually within 48 hours, or preadult larvae. The L4 larvae then molt and develop into the L5 adult form. The male and female adult copulates and live in the abomasum, where they feed on blood.

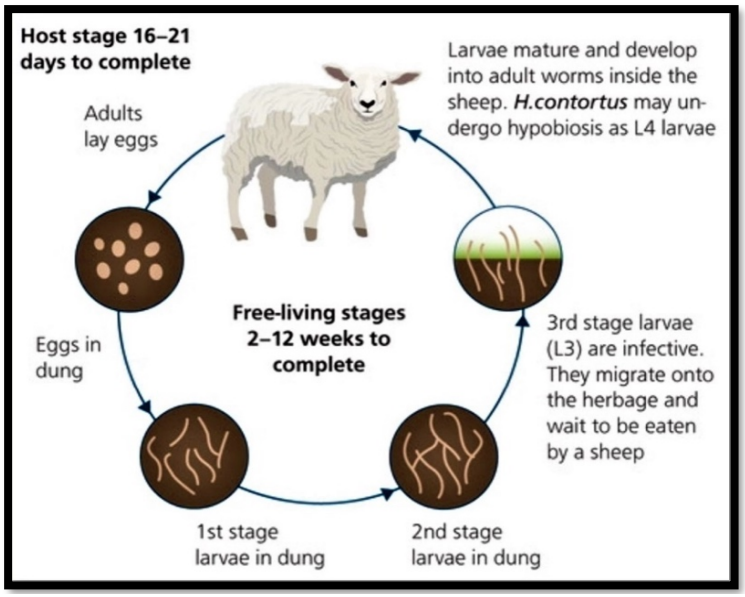


Figure 128. Life cycle of *Haemonchus contortus*

Pathogenesis: animal loss significant weight due to worm feed mainly on blood. Sudden death may be the only observation in acute infection, while other common clinical signs include pallor, anemia, oedema, ill thrift, lethargy, and depression. The accumulation of fluid in the submandibular tissue, a phenomenon commonly called "bottle jaw", may be seen. Growth and production are significantly reduced.

Diagnosis:

- Cannot be diagnosed on worm egg counts but a high worm egg count can cause suspicion – counts of over 10,000epg.
- *Haemonchus* eggs can be recognized from other Trichostrongylid in faecal samples by application of a fluorescent stain.
- Blood test to look at the hematology/biochemistry of an animal.
- *Haemonchus* can be seen in the abomasum on post-mortem examination by veterinarians at the abattoir.

Ostertagia spp.

Ostertagia spp. are small bursate nematodes that parasitize the gastrointestinal tract of mammals and birds and cause **Ostertagiosis**. These species are

responsible for mortality and morbidity for a number of ruminants, especially the species *O. ostertagi* which is widely considered the most important parasite of cattle.

Host: Direct life cycle. Common species of *Ostertagia* are:

O. circumcincta in sheep.

O. ostertagi in cattle and sheep.

O. trifurcata in sheep and goats.

Ostertagia spp. are similar to *H. contortus* in host and location, but they differ in color, being a dirty brown—hence, their common name, Brown Stomach worm.

- The buccal capsule is rudimentary and lacks a tooth.
- Cervical papillae are present.
- The male bursa is symmetrical.
- The vulva has a large anterior flap, and the tip of the female's tail bears several cuticular rings.
- Their life cycle is similar to that of *H. contortus* except that J3s invade gastric glands and elicit nodules. J3s molt before returning to the lumen, where they feed, molt, and begin producing eggs about 17 days after infection. *Ostertagia* spp. suck blood but not as much as *Haemonchus contortus.*
- Species of *Ostertagia* often undergo developmental arrest as J4.
- Economic losses in the cattle industry due to *O. ostertagi* and other nematodes probably exceed $600 million per year in the United States alone.

Geographical distribution: Predominately a parasite of cattle and buffalos. Occasional transmission to goats and wild ruminants although these species are rarely involved in transmission. Parasite survival within the environment; L3 parasites can survive winter periods on the pasture, and these are the main source of infection to stock when they are turned out in spring.

Clinical signs: The most common symptoms associated with *Ostertagia* infection in cattle are loss anorexia and watery diarrhea.

Diagnosis: Clinical signs are associated with increased serum pepsinogen.

Life cycle:

• Eggs passed in the faeces develop into first-stage larvae (L,), which hatch develop, and molt to become second-stage larvae (L2), which in turn develop and molt to the third infective stage (L3).

• The L3 then migrates under moist conditions onto the herbage. The L3 retains the outer sheath which considered is the most resistant the free-living stages.

• After ingestion, the parasitic cycle involves development the L3 to L4 stages in the gastric glands. This usually takes 21 days, by which time the adult parasite emerges from the glands onto the surface of the abomasa mucosa.

• The adult mate and the female worm will then produce thousands of fertilized eggs which pass out of the animal in the faeces (figure 129).

• **Treatment**: antiworm administration includes one of following: Albendazole, Doramectin, Eprinomectin, Febantel, Fenbendazole, Ivermectin, levamisole, Moxidectin, Netobimin, or Oxfendazole.

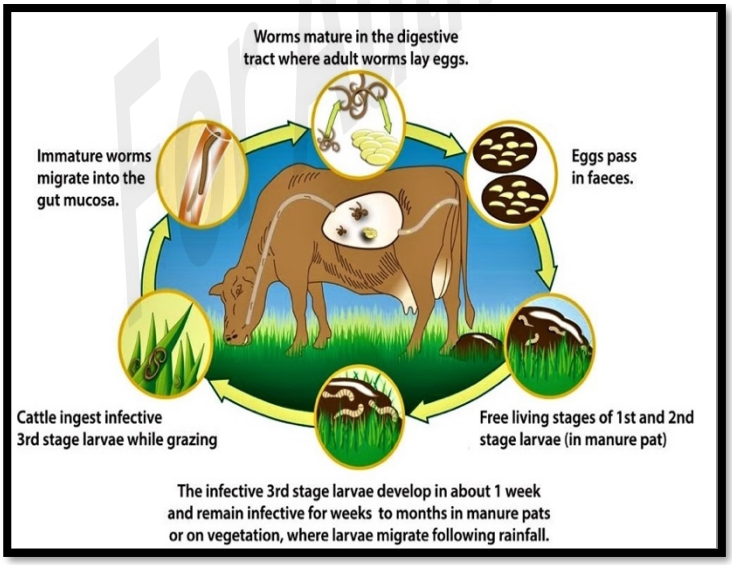


Figure 129. Life cycle of *Ostertagia* spp.

***Trichostrongylus* spp.**

Characteristics:

o *Trichostrongylus* spp. are the smallest members of the family ***Trichostrongylidae*** (figure 130). They are colorless and 7 mm in length. Many

species inhabit the small intestine of ruminants, rodents, pigs, horses, birds, and humans.

- Lack cervical papillae, and have a rudimentary, unarmed buccal cavity.
- The male's bursa is symmetrical, with a poorly developed dorsal lobe.
- Spicules are brown and distinctive in size and shape in each species.
- The vulva lacks an anterior flap.

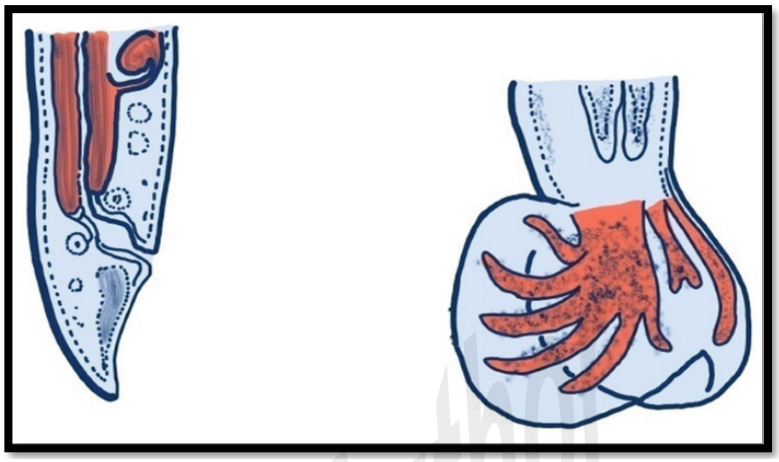


Figure 130. Female and Male posterior end of *Trichostrongylus* spp.

Common species of *Trichostrongylus*:

- *T. colubriformis* in sheep, goats,cattle, and deer.
- *T. tenuis* in galliform birds (grouse, pheasant,chickens, and turkeys).
- *T. capricola, T. falcatus,* and *T. rugatus* inruminants.
- *T. retortaeformis* and *T. calcaratus* in rabbits.
- *T. axei* ina wide variety of mammals.

Geographical distribution: *Trichostrongylus* causes a disease called Trichostrongyliasis in livestock. Humans are incidental hosts. The infection is acquired via consumption of contaminated vegetables with the larvae. The human cases have been reported worldwide with estimated prevalence of 5–6 million. The prevalence is high among farmers in the developing countries because of low hygienic conditions.

Diagnosis: Larvae can be diagnosed by fecal culture. Juveniles are very similar to those of hookworms and *Strongyloides* spp; therefore, differential diagnosis is

important. Molecular methods are available for the trichostrongylid species isolated from ruminants.

Biology: life cycle resembles to *Haemonchus* spp. except that J3s burrow into mucosa of the anterior small intestine, where they molt. After returning to the lumen, they bury their heads in mucosa and feed, grow, and molt for the last time. Egg production takes two weeks (figure 131).

1- Eggs are passed in the stool of the definitive host (usually an herbivorous mammal).

2- Under favorable conditions (moisture, warmth, shade), larvae hatch within few weeks. The released rhabditiform larvae that normally develop in the soil or on plants.

3- After 5 to 10 days (and two molts) they become filariform (third-stage) larvae that are infective.

4- Infection of the human occurs upon ingestion of these filariform larvae.

5- The larvae reach the small intestine, where they reside and mature into adults. The adult worm resides the digestive tract of their definitive hosts and may occur as incidental infections in humans (figure 132).

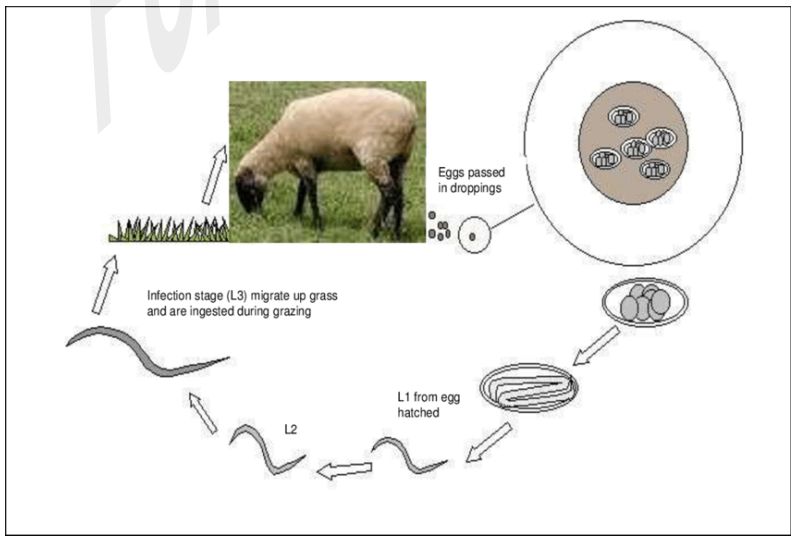


Figure 131. Life cycle of *Trichostrongylus* spp.

Pathogenesis: Most infections without manifested symptoms. Heavy infections cause gastrointestinal disorders, the juveniles and adults cause damage to intestinal epithelium. Clinical signs due to the infection include abdominal pain, diarrhea, anorexia, headache, fatigue, anemia and eosinophilia. Systemic poisoning by metabolic wastes of the parasites and hemorrhage, emaciation, with mild anemia may develop in severe infections.

Treatment: using anthelminthics such as Thiabendazole or Pyrantel.

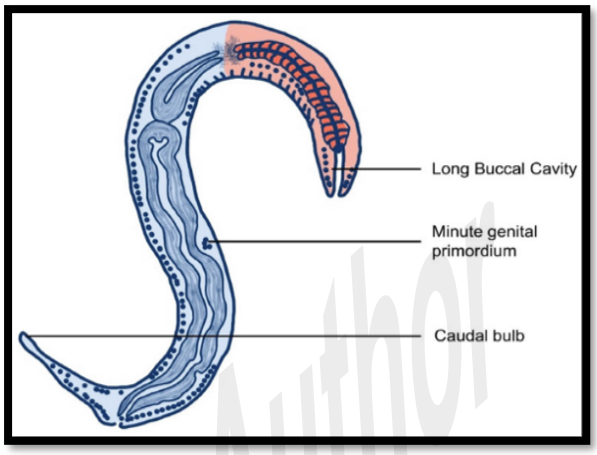


Figure 132. typical form of *Trichostrongylus* spp.

Family: Dictyocaulidae

***Dictyocaulus* spp.**

Species belongs to this genus are medium in size and parasitize the bronchi and trachea. *Dictyocaulus filaria* is an important parasite of sheep and goats, as well as wild antelope and deer (figure 133).

Other *Dictyocaulus* species

1- *Dictyocaulus viviparus* (cattle and deer)

2- *D. arnfieldi* (donkeys and horses).

3- *D. filaria* (sheep and goats).

Characteristics:

- They commonly cause death of their host.

- Fully grown adults are slender and long, with males reaching 80 mm and females 100 mm.
- The bursa is small and symmetrical; spicules are short, and boot shaped in lateral view.
- The uterus is near the middle of the body.
- Adults live in bronchi and bronchioles, where females produce embryonated eggs.

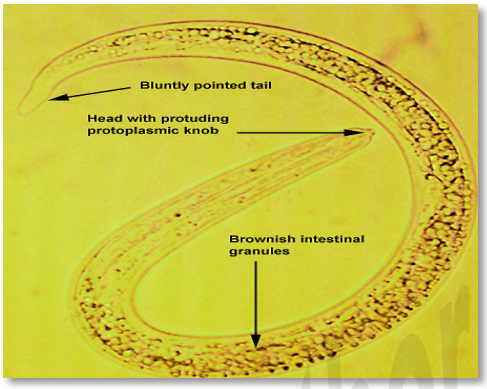


Figure 133. *Dictyocaulus filaria*

Geographical distribution: predominant in temperate areas and requires humid and cold environment, to survive longer.

Symptoms: Symptoms of *Dictyocaulus* can include coughing, rapid shallow breathing, expiratory dyspnea, tactile fremitus, serous nasal discharge, pyrexia, increased pulse rate, weight loss, and diarrhea.

Diagnosis:

1- Microscope detection of L1 larvae in feces.

2- Microscope detection of eggs or larvae in the sputum or bronchoalveolar lavage fluid from affected animal.

3- Serology test for detection the antigen of the parasites.

Pathogenesis: pulmonary emphysema due to heavy infestations of the cattle. Clinical signs range from bronchitis to severe consolidating pneumonia as eggs are inhaled to all areas of the lungs and inflammation occurred.

Biology: eggs hatch and carried toward the trachea by ciliary action. First-stage juveniles passed out with feces and develop to juvenile stage 3. The cuticles of the first and second stages are retained by the third stage until the worm is

ingested by a definitive host; then cuticles of all these stages are shed together. J3s penetrate the mucosa of the small intestine and enter mesenteric lymph nodes. There they undergo two molts to become small adults (about 500 m long), enter the circulation via thoracic duct (figure 134).

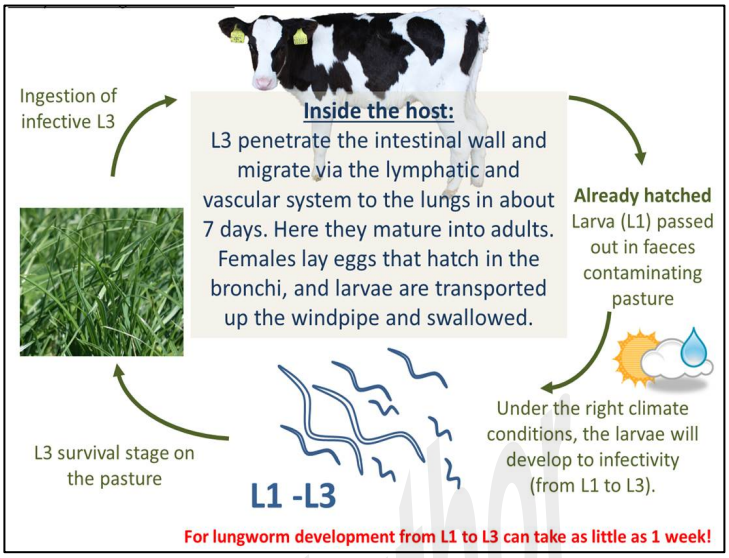


Figure 134. Life cycle of lung worm in livestock

Treatment: Ivermectin, albendazole, and levamisole are the recommended treatments for the Dictyocaulus infection in livestock. In equine the most effective treatment is ivermectin and moxidectin.

Further species of *Trichostrongyles*

- *Cooperia curticei* (Trichostrongylidae),
- *Nematodirus spathiger*
- *N. filicollis* (Molineidae)
- *Hyostrongylus rubidus* (Trichostrongylidae)
- *Heligmosomoides polygyrus, Nematospiroides dubius*) in mice
- *Nippostrongylus brasiliensis* (Heligmonellidae) in rats

Metastrongylus

Metastrongyles are worms in several families formerly placed in superfamily Metastrongyloidea, but recently reclassified as a family. Metastrongyles, along

with Dictyocaulids, are both nematode groups commonly referred to as "lungworms". Metastrongyles and Dictyocaulids are each monophyletic groups related as sister clades, according to molecular phylogenies.

The common name "lungworm" is really a misnomer for metastrongyles because adults of different species occupy a variety of other vertebrate tissue sites, including skeletal muscles, central nervous system, circulatory system, and frontal sinuses. Most species for which life cycles are known use gastropod intermediate hosts, although earthworms and marine fish serve this role for certain species. Some species also employ a vertebrate or invertebrate transport host. Most species mature in live in terrestrial mammals.

Superfamily Strongyloidea

Family Syngamidae

Genus: *Syngamus*

Syngamus trachea is the gapeworm of poultry that cause **Syngamosis** disease, because the adult residue in the respiratory tract of various bird species, causing gasping and gaping. The parasite appears to be harmful for a wide variety of avian orders, occasionally leading to a fatal outcome, particularly in young birds.

Host: Galliform birds (Chickens, turkeys, pigeons and game birds).

Paratenic host: Terrestrial mollusks, earthworms, and arthropods.

Geographical distribution: Worldwide distribution. Gapeworm infection is more likely to occur in chicken and turkey, especially if they are less than 3 months in age. The old chickens are carrier to this parasite. Disease is seen most frequently in breeding and rearing establishments where outdoor pens, such as those used for breeding pheasants, are in use. Eggs, passed by wild birds such as rooks and blackbirds, may initiate infection; these may also infect earthworms. Infection is usually highest during the hot season when earthworms are active.

Morphology: The worms have large shallow cup-shaped buccal capsules, which have up to 10 teeth at their base and there are no leaf crowns. The large reddish

female (≈ 1-3 cm long) and the small whitish male (≈ 0.5 cm long) are permanently in copula forming a 'Y' shape (figure 135). Eggs (≈ 78-100 µm) are thick- shelled with two plugs at their ends and contain a morula when laid (figure 136).

Clinical sign: The parasite causes dyspnoea, and gaping; producing a hissing noise as they do so. Severe infestation may obstruct the tracheal lumen resulting in suffocation. Other signs include coughing, weakness, emaciation and shaking of the head.

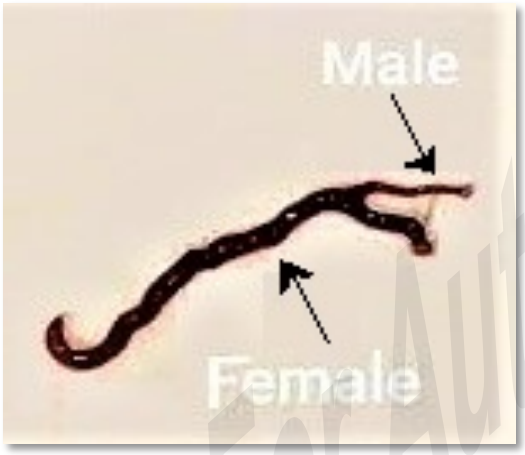


Figure 135. Adults *Syngamus trachea*

Figure 136. Egg of *Syngamus trachea*

Diagnosis: post-mortem can reveal on presence of small nodules and often adult worms may found in the trachea. Faecal smears can also be performed, which may reveal characteristic bioperculate eggs.

Biology:

1-The fowl coughs up eggs, swallows them, and then passes them in feces (figure 137).

2- Juveniles molt twice in the egg to become infective J3s.

3- Definitive hosts become infected through swallow embryonated eggs or juveniles.

4- Infective juveniles penetrate the gut wall, are carried by blood to the lungs where they break out into alveoli, and then proceed up to the trachea.

5-Males remain attached to a female via their copulatory bursa.

6-Young birds are most severely affected and may die with a heavy infection.

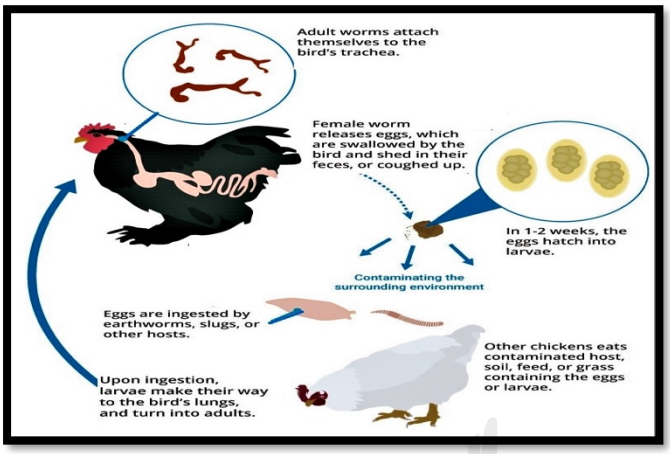


Figure 137. Life cycle of *Syngamus trachea*

Treatment: benzimidazoles are effective, ad- ministered usually over a period of several days. Birds need to be monitored, as severely affected ones may not ingest adequate anthelmintic. Nitroxynil and levamisole are also very efficacious when given in the water.

Strongylidae

Members of Strongylidae occur in the intestine of mammals, especially in equine. They are commonly recognized as large strongyles (several species of *Strongylus,* of which *S. vulgaris* is the most important species) and small genus of strongyles is known as *Cyathostomum*.

Strongylus vulgaris* and *Cyathostomum

Site of infection: large intestine of equines.

Morphology: Adults: robust dark-red worms (≈ 1.5-2.5 cm). The well-developed deep buccal capsule is oval in outline and contains two ear shaped rounded teeth at its base. The anterior margin of the buccal capsule bears two leaf crowns (figure 138). The copulatory bursa of the male is well developed. Eggs are of *Strongyloides* type (≈ 83-93 by 48-52 µm) with barrel-shaped side walls, a

smooth thin shell and contain a morula with several large blastomeres when laid**.** L3 is long-tailed.

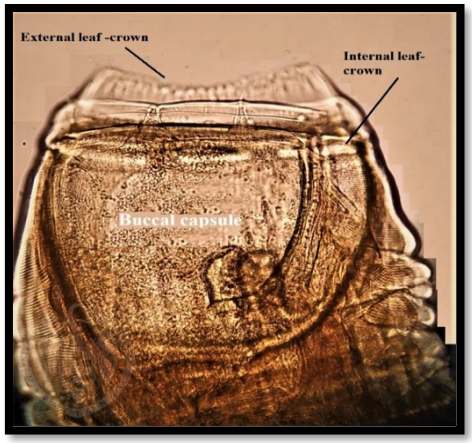


Figure 138. Buccal capsule of *Strongylus vulgaris*

Geographical distribution: The 1st and 2nd stages of larvae are very resistant to harsh environmental conditions because of a protective sheath. *Strongyles* can survive in the coldest environment, but a hot and dry weather can kill them. The infective larvae survive up to 31 weeks at winter temperatures, compared to up to seven weeks at summer temperatures

Clinical signs: Strongylosis is characterized by nonspecific signs such as weight loss and poor growth, rough hair coat, and loss performance. Larval *S. vulgaris* infections are associated with colic. In the case of larval cyathostominosis syndrome can occur. This syndrome is caused by emergence or large numbers of encysted cyathostomins and it is characterized by diarrhea, rapid weight loss and hypoproteinemia.

Diagnoses

- Coprological examination for visualization of the adult parasites' eggs.
- Hematological (anemia, leukocytosis and eosinophilia) and biochemical (hypoalbuminemia) findings.
- Serological techniques (ELISA).

- Molecular techniques (PCR and RLB), with a real-time PCR assay can be for the detection and semi-quantification of *S. vulgaris* DNA in faecal samples.

Biology: Adult strongyles produce eggs that are passed out in the feces of horses.

- These eggs then develop into infective larvae that exist on the pasture vegetation or in stalls.
- The horse is infected when it consumes grass, feed, or water contaminated with infective larvae.
- The larvae of *S. vulgaris* migrate through various parts of the body.
- The bloodworm, will burrow into and migrate in the walls of the arteries that are the primary blood supplier to the small and large intestines.
- This migration can result in the formation of blood clots, which can disrupt the blood flow to the intestines and cause scar tissue formation in affected arteries.
- After approximately four months, the larvae move to the lumen of the large intestine, where maturation is completed. As adults, these parasites will lay several thousand eggs each day, completing the life cycle (figure 139). The entire life cycle takes six to seven months.

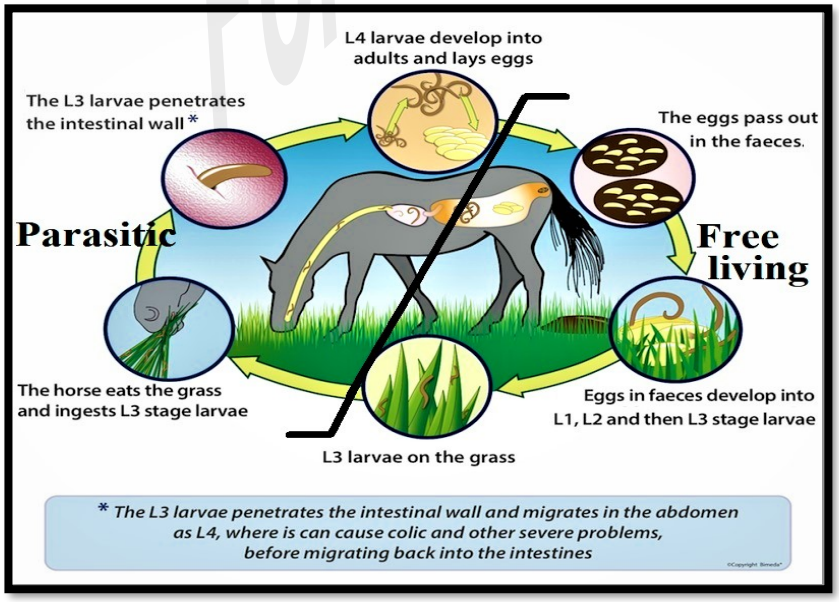


Figure 139. Life cycle of *Strongylus vulgaris*

Note: The life cycle of the small strongyle (***Cyathostomes***) is very similar to large strongyles except the larvae do not migrate beyond the wall of the intestines. The larvae burrow in or encyst in the wall of the large colon.

Treatment: Benzimidazoles, Macrocyclic lactones (Ivermectin and Moxidectin) and Tetrahydro-pyrimidines (Pyrantel) are effective against adult strongyles.

Only Fenbendazole and Moxidectin would be effective to encysted larvae of small *strongyles*. In addition to these two anthelmintics, Ivermectin could be used against migration of the larvae of the large strongyles.

Oesophagostomum spp.

Oesophagostomum bifurcum

Occurrence*: Oesophagostomum* spp. are called nodular worms and is the common parasites of livestock and other animals like goats, pig and non-human primates (figure 140). The parasite is reported frequently in Africa, eastern Asia, and Brazil.

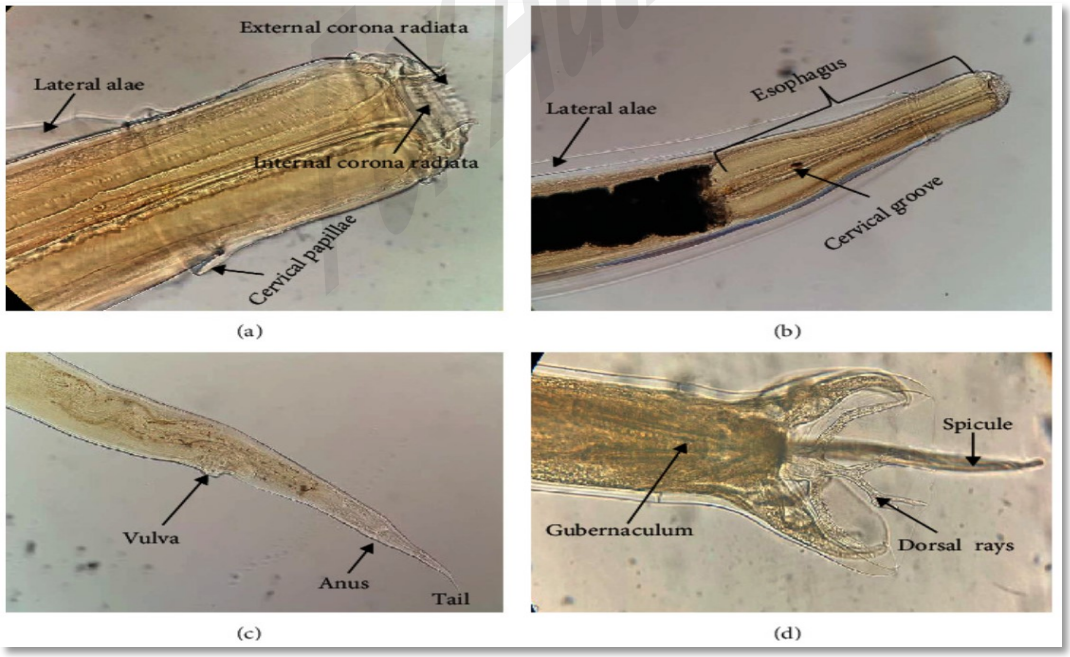


Figure 140. *Oesophagostomum* spp.

Life cycle: Adults live in the large intestine and juveniles form nodules in walls of the small and large intestine (figure 141, 142). The infections are acquired by ingestion of J3. The infections in humans occasionally happened.

O. bifurcum is highly prevalent in humans and nonhuman primates and the individuals with hookworm infection have a higher likelihood of being infected with *O. bifurcum*. Human infection typically presents as a painful abdominal mass that sometimes requires surgical intervention. Eggs of *O. bifurcum* are indistinguishable morphologically from hookworms, but J3s obtained after fecal culture show clear differences.

Diagnosis: Ultrasound and DNA techniques can also be utilized to identified *O. bifurcum*. *O. bifurcum* detected in humans and other primate hosts demonstrated various genetic differentiation.

Figure 141. *Oesophagostomum bifurcum*

Figure 142. *Nodular* worms

Appendix

Questions from book contents

Q1: Fill in the Blanks with a suitable word.

1- ---------- is the study of parasites, their hosts, and the relationship between them.
2- ---------- is a traveling together or to carry, that is mean a smaller organism is termed a ---------- that is carried mechanically by ------- for instance, bacteria, fungus, cysts, or eggs on insect legs or even passively within an arthropod gut.
3- ---------- leads a parasitic life throughout the whole period of its life.
4- Each parasite possesses ---------- names, ----------and ---------- that the former begins with an initial capital and the latter with an initial small letter.
5- ---------- is capable of causing infection.
6- ---------- number of deaths in population due to particular reason often diseases occurrence.
7- ---------- this involves only wild animals. (eg: *Echinococcus granulosus* was found in a wide range in cervid animals (moose (*Alces alces*)) in the indigenous areas of Canada.
8- Parasites possess three major characteristics that make them difficult for a host to control immunologically: ----------, ---------- and ----------.
9- ---------- When infected human and causing sever disease some time leading to the death.
10- The metacercaria eaten by cattle in ----------.
11- Parasitology is type of ----------.
12- Ectoparasites mean parasite ----------.
13- ---------- cannot exist without a parasitic life.

14- ---------- non-human animals that serve as sources of infection to humans.

15- Each animal can be a host of many ----------.

16- ---------- Intermediate host: First: land snails, Second: Ants Final host: Cattle.

17- *Dicrocoelium dendriticum* causes ----------.

18- ----------found in human when infected with cercaria of Birds Schistosoma.

19- The Swedish scientist ---------- (1707-1778) developed the binomial system of nomenclature that is still in use today.

20- Parasitology is a type of ----------.

21- The indirect transmission ----------.

22- Epidemic is ----------.

23- ---------- is an example of high reproductive potential.

24- Trematoda is ---------- symmetrical; ---------- flattened.

25- ---------- is normally ectoparasites on aquatic vertebrates.

26- ---------- is ciliated larval stage found outside the snail.

27- The cat passed several active proglottids of the flea tapeworm, *Dipylidium caninum*----------.

28- Aberrant host is ----------.

29- ----------is the life cycle that involves only wild animals.

30- During----------several researchers described the features of hydatid cysts.

31- ----------are free-living parasites, they feed on ----------

32- The digestive system of nematodes consists of ----------,----------,----------.

33- The male genital organ of nematodes is provided with spicules called --------

34- Nematode species has ----------at one or both sides.

35- Nematodes parasite can survive in soil because of ----------

36- The eggs of *Subulura brumpti* after released in faeces is swallowed by ----------.

37- The prepatent period of the parasite *H. gallinarum* is ----------.

38- The major features of the epidemiology of **----------** are the extreme persistence of the infective stage in the environment and the predominance of infection in juvenile horses.

39- When both fertilized and unfertilized eggs in Ascaris lack their outer albuminous coats and are colorless called----------.

40- ----------is the host of *Toxascaris leonina*.

41- *Dirofilaria immitis* is a parasitic nematode responsible of ---------- cardiopulmonary dirofilariasis.

42- Trichinellosis is caused by nematodes (roundworms) of the genus ---------- in addition to the classical agent ----------

43- *Capillaria plica* may infect the ----------, and occasionally the ureters and renal pelvises, of ----------and ----------.

44- *Trichuris trichiura*, also known as the human ----------.

45- *Trichinella pseudospiralis* infect mammals and ----------worldwide.

46- During migration *Dirofilaria repens* may reach the ----------, becoming visible through the ----------.

47- *Parascaris equorum* has ---------- life cycle.

48- ----------infections are associated with colic.

49- *Enterobius vermicularis* can survive in ---------- for several days

50- *Capillaria plica* can be treated using ----------, ----------.

Q2: Choose the correct answer from the bracket as following:

1- ---------- found in intestine of birds. (*Fasciola gigentica, Planaria sp., Metagonimus yokogawii*)**.**

2- ---------- with spiny collar. (*Echinostoma* sp., *Heterophyes hetreophyes*, *Paragonimus westermani*)

3- ---------- found at the surface of the host. (Ectoparasites, Endoparasites, Monogenia)

4- ---------- can diagnose their cercaria with forked till. (*Schistosoma haematobium, Clonorchis sinensis, Paragonimus waterman*)

5- ---------- with flame cells in excretory system. (Cestoda, Digenia, Mongenia)

6- ---------- can treat the infection by Metrifonate. (Tuberculosis, Planaria infection, *Schistosoma haematobium*)

7- ---------- separated sex trematoda. (*Fasciola gigentica, Schistosoma sp., Metagonimus yokogawii*)

8- ---------- in the life cycle the second intermediate host ants. (*Heterophyes heterophyes, Dicrocolium dendraticum, Ornithobliharizia turkestanicum*)

9- ---------- found at the surface of the host. (Ectoparasites, Endoparasites, Monogenea)

10- ---------- like a coffee beans shape (*Schistosoma haematobium*, *Clonorchis sinensis*, *Paragonimus waterman*)

Q3: Choose the correct answers:

1- Larval stage of *Taenia hydatigena* is called:

- **a.** Cysticercus pisiformis
- **b.** Cysticercus ovis
- **c.** *Cysticercus tenuicollis*

2- Pyriform apparatus found in the egg of:

- **a.** *Anoplocephala magna*
- **b.** *Moniezia expansa*
- **c.** *Dipylidium caninum*

3- --------- also called the dwarf tapeworm

a. *Davainea proglottina*

b. *Hymenolepis nana*

c. *Avitellina centripunctata*

4- The larval stages of Echinococcus *multilocularis* found in*:*

a. Sheep

b. Rodents

c. Copepods

5- ……are the primary sites for synthesis and storage of glycogen

a. Glycocalyx

b. Microtriches

c. Parenchyma

6- Coracidium is:

a. The larva which the head and neck are partly enclosed in a bladder like cyst.

b. The ciliated first stage aquatic embryo.

c. Spindle-like solid body with cephalic invagination

7- Sparganosis is the:

a. Bizarre eye disease

b. Double-pore tapeworm

c. Pernicious anemia.

8- The larval stage tetrathyridium related to the genus:

a. *Hymenolepis nana*

b. *Mesocestoides*

c. *Raillietina cesticillus*

9- The diagnosis of *Anoplocephala magna*:

a. Square, rough embryonated eggs

b. Thick radially striated egg shell

c. Operculated egg.

10- In terproglottid glands can be seen in:

a. Davainea proglottina

b. *Moniezia expansa*

c. *Hymenolepis diminuta*

11- The intermediate host of *Stilesia globipunctata:*

a. Sheep

b. Beetles and flea

c. Oribatid mite

12- The infection with the adult stage of the pork tapeworm is called

a. Cysticercosis

b. Taeniasis

c. Neurocysticercosis

13- --------- also called the minute tapeworm

a. Davaineaproglottina

b. *Hymenolepis nana*

c. *Avitellina centripunctata*

14- Coenurus is the larval stage of the genus:

a. *Taenia solium*

b. *Taenia pisiformis*

c. Multiceps

15- A modified cysticercus resembling an elongated plerocercoid with a scolex invaginated at one end called:

a. Cysticercoid

b. Embryophore

c. Tetrathyridium

16- Cestodaria is the unsegmented subclass of tapeworm affecting various:

a. Fish and some reptiles

b. Amphibians

c. Mammals

17- The egg of the genus *Spirometra:*

a. Outer membrane is thin and transparent

b. Operculated

c. Its shell is thick and radially striated

18- Larval stage of *Taenia hydatigena* called:

a. Cysticercuspisiformis

b. Cysticercusovis

c. *Cysticercus tenuicollis*

19- Cucumber tapeworm is:

a. *Hymenolepis diminuta*

b. *Dipylidium caninum*

c. *Mesocestoides* genus

20- The second larval stage of *Diphyllobothrium latum* called:

a. Procercoid

b. Plerocercoid

c. Cysticercoid

21- The Microtriches in cestodes:

a. Connected by cytoplasmic bridges to distal cytoplasm

b. Increase the surface area of the teguments for enhanced absorption

c. The primary sites for synthesis and storage of glycogen

22- Pyriform apparatus found in the egg of:

a. *Anoplocephala magna*

b. *Moniezia expansa*

c. *Dipylidium caninum*

Q4: Write False or True for the following sentences:

1. Parasites have never transmitted through water supplies.
2. Parasite can survive for more than ten years inside human organs.
3. A carrier host is the host that mostly harbors a parasite with clear symptoms of the infection.
4. Human cannot be involved as an intermediate host for any type of parasite life cycle.
5. A high mortality rate in ungulated animals reported due to a heavy infection with internal parasites.
6. Host, agent and environment do not relate to each other at the time of infection with parasites.
7. The disease caused by parasite almost always occurs randomly in animals.
8. Time is not important factor in distribution and infection of the parasite.
9. Leishmaniasis has not been frequently reported in a hot region.
10. Echinococcus is a genus name comes originally from an English word“Spine and Berry”.
11. Babesia had been reported in the middle of 20th century in many parts of Europe.

12. Bursa may found only in the male of Trichostrongyloides.
13. The eggs of Nematodes are impossible to be recognized from other parasite eggs species.
14. The life cycle of nematodes evolves through different larva stages.
15. The infection with *Ostertagia* combines with pathological changes and effects on lumen absorption.
16. Cellular immunities are stimulated at the time of nematodes invades a host.
17. Subulura spp infect the caecum of poultry.
18. The male of *H. gallinarum* is shorter than its female.
19. It ss necessary to maintain the hygienic environment of the cages and backyards of poultry to prevent the infection by *H. gallinarum.*
20. The intermediate hosts of *Fasciola gigentica* are snail and Ant.
21. *Ornithobilharzia turkestanicum* causes bottle jaw in animals.
22. Diagnose of the infection with Schistosomiasis is carried out by urine examination.
23. All Diagenia found in lung of their hosts.
24. *Paramphistomum cervi* could be found in dogs and cats.
25. Ectoparasites can bear endoparasites.
26. The intermediate host of *Dicrocolium dendraticum* is snail and Ant.
27. *Schistosoma bovis* causes bottle jaw in animals.
28. *Fasciola* sp. infection can be identified in the fecal matter through immunologically tools.
29. Parasitic infection causes eosinophilia.
30. *Metagonimus* is found often in dogs and cats.

Q5: What are the differences between the following:

1- Mutualism & Parasitism.

2- Ectoparasites & Endoparasites.

3- Vector hosts & Reservoir hosts.

4- Ectoparasites & Endoparasites.

5- Parasitemia & Parasitism.

6- Monogenea & Digenia.

7- Epizootic & Epidemic.

8- *Taenia hydatigena* & *Raillietina* spp.

9- *Avitellina centripunctata* & *Dipylidium caninum*

10- *Hymenolepsis nana* & *Hymenolepis diminuta*

11- Cysticercus tenuicollis & Cysticercus pisiformis.

12- Eggs of *Anoplocephala magna* & *Moniezia expansa*

13- *Taenia saginata* & *Taenia solium*

14- *Echinococcus granulosus* & *E. multilocularis*

15- Coenurus and *Echinococcus*

Q6: Talk in details with drawing the life cycle of:

1- *Dicrocolium dendraticum*

2- *Heterophyes heterophyes*

Q7: Define the following:

1- Tegument of cestode
2- double-pore tapeworm
3- Coracidium
4- Hydatid sand
5- Taeniasis
6- *Cysticercus bovis*
7- Cysticercoid
8- Coracidium.

Q8: What are the main features of the following:

a- Eggs of *Diphyllobothrium latum*.

b- Eggs of *Hymenolepis nana*.

c- Scolex of *Spriometra mansonoides*.

d- Proglottid of *Taenia solium*.

e- Egg of *Moniezia expansa*.

Q9: Mentioned the life cycle and diagnostic stages of *Anoplocephala magna*.

Q10: Draw section of cestode tegument.

Q11: Compare between Pseudophyllidean and Cyclophyllidean Cestodes.

Q12: Mention the mode of parasite transmissions in animals and humans

Q13: What are the objectives in studying the epidemiology of parasites?

Q14: Give five general morphological features of Nematodes.

Q15: Explain briefly the types of *Ascaris lumbricoides* eggs.

Q16: How can diagnose the Trichuriasis?

Q17: Refer to the Prevention & Control to Trichinellosis.

Q18: What is happening to a man infected with *Dirofilaria repens* when he left untreated?

Q19: What are the morphological characterizations of the following parasites?

1- *Dictyocaulus*

2- *Spirura*

3- Intestinal flukes

4- Ascaridae

Q20: What are the immunity responses against nematodes?

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