Animal Health

Level-III

Based on March 2022, Version 4 Occupational standard



Module Title: - Identifying and Handling Parasitic Diseases of Animals

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MINISTRY OF LABOR AND SKILLS

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Introduction to the Module

This module covers the knowledge and skills and attitude required to develop basic understanding on biology, characteristic feature and, clinical/economic/public health impacts of infectious microorganisms. It also requires the trainees' ability to classify, characterize and identify microorganisms which are threat of animal health through the use clinical examination and microbiological laboratory examination. In addition this competency covers trainees' skill to treat and prevent/control important and more prevalent microbial diseases animals.

LG #5	LO #1- Develop a general knowledge on parasitology and parasites

Instruction sheet 1

This learning guide is developed to provide you the necessary information regarding the following content coverage and topics:

- Clinical and economic impacts of parasitic diseases
- Statistical information on distribution and implication of parasitic disease
- Public health significance of zoonotic parasitic agents
- Classification of parasites
- Hosts and life cycles of common parasites

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, you will be able to:

- Introduce the clinical and economic impacts of parasitic diseases
- Explain statistical information on the current disease distribution and its implication in Ethiopian livestock sector
- Mention Public health significance of zoonotic parasites
- Classify Parasites are categorized on the basis of Taxonomic
- Explain hosts and lifecycles of commonly prevalent parasites

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.

3. Read the information written in the information Sheets
4. Accomplish the Self-checks

”

Information Sheet 1:

1. Introduction to veterinary parasitology

Veterinary Parasitology is the study of animal parasites, especially relationships between parasites and animal hosts. Parasites of domestic animals, (livestock and pet animals), as well as wildlife animals or A scientific study of parasites, their hosts, and the relationship between the parasite and the host. Veterinary Parasitology explains the diagnosis, treatment and control of major endo and ecto parasites of domesticated animals. Topics covered includes: Coccidia, Arthropods, Nematodes, Trematodes, Cestode, Arthropode and Protozoas. Parasitology is a branch of science that is concerned with parasites and parasitism. Parasitism is A form of symbiosis in which one organism (called parasite) benefits at the expense of another organism usually of different species (called host). The association may also lead to the injury of the host. An example of parasitism is the association between the parasitic tapeworms and the vertebrate hosts.

A parasite is an organism that lives in or on another and takes its nourishment from that other organism, or “host.” Parasites of animals and humans come in many forms, including helminths (worms), arthropods (lice, ticks, mosquitoes, etc.), and protozoa. There are over 1,000 species of parasites affecting domesticated animals throughout the world. They can be broadly classified as external or internal, depending on where they live on their host.

4.1. Clinical and economic impacts of parasitic diseases

1.1.1.Terminilogies

- **Definitive host**- is host which harbor adult/sexually matured parasites
- **Intermediate host**- is host which harbor larval stages of parasites / an organism that supports the immature or non-reproductive forms of a parasite.

- **Transport hosts (parathenic hosts)**- an organism that harbors the sexually immature parasite but is not necessary for the parasite's development cycle to progress.
- **Reservoir hosts/waiting host** - an organism that harbors a pathogen but suffers no ill effect and serves as a source of infection to other species
- **Prevention**- action taken to decrease the chance of getting a disease or condition/ avoiding risk factors and increasing protective factors
- **Control-** to reduce the incidence or severity after occurance of unwanted conditions

1.1.2. Clinical Impacts of parasitic diseases

Both external and internal parasites may weaken an animal's immune system and create conditions favorable to bacterial disease. In severe cases, these diseases can also be deadly.they also cause the following conditions like Bleeding and blood sacking, Competition for food/ fluid, Mechanical Obstruction of GIT/ blood vessel/lymphatic vessel, Tissue damage/perforation, Cyst formation, Skin/hide damage and Production loss (milk, meat, egg,..).

External parasites often annoy their hosts by biting, embedding, or otherwise irritating the skin. They can cause serious diseases, such as mange and scabies, which affect animals' health and growth while **Internal parasites** live in the blood or tissues inside an animal's body. Some organisms enter an animal when it swallows contaminated food or water. Others burrow through the skin, reach the blood stream, and settle in a preferred location to mature and reproduce. Internal parasites often interfere with digestion and assimilation of food, causing poor growth, temporary or permanent injuries, or death.

Parasites have been responsible for economic losses ever since humans first undertook the domestication of animals. Farmers and ranchers whose herds are infected with parasites pay higher costs to raise sick animals and earn less because of lower production. Economic losses occur not only when animals die, but also when they are unable to perform their regular work, or when they produce inferior meat, milk, wool, hides, or eggs.

1.1.3. Economic Impacts of parasitic diseases

One of the major challenges facing the U.S. Department of Agriculture in the mid- to late-1800s was the need to identify, understand, and develop effective controls against the parasites plaguing

American livestock. Advances in transportation and both domestic and international trade had driven explosive growth in the country's commercial livestock production. Farmers and ranchers needed to minimize parasite damage in order to make their products marketable, and they began to demand that their government be involved in finding a solution.

High animal mortality, Loss of production performance, Cost of treatment and control and International trade ban. For more information about some of these projects see the following: **p**arasites are a major cause of disease and production loss in livestock, frequently causing significant economic loss and impacting on animal welfare. In addition to the impact on animal health and production, control measures are costly and often time-consuming. Parasites are a major cause of disease and production loss in livestock, frequently causing significant economic loss and impacting on animal welfare. In addition to the impact on animal health and production, control measures are costly and often time-consuming. A major concern is the development of resistance by worms, lice and blowflies to many of the chemicals used to control them.

Planned preventative programs are necessary to minimise the risks of parasitic disease outbreaks and sub-clinical (invisible) losses of animal production, and to ensure the most efficient use of control chemicals. Integrated parasite management programs aim to provide optimal parasite control for the minimal use of chemicals by integrating pre-emptive treatments, parasite monitoring schedules and non-chemical strategies such as nutrition, genetics and pasture management. The Department of Primary Industries and Regional Development provides recommendations for the most effective approaches to the control of the major parasites of livestock.

1.2. Statistical information on distribution and implication of parasitic disease

Zoonotic parasitic infections are a major global public and veterinary health problem and widespread among stray dogs. The objective of this study was to establish the prevalence of intestinal parasites in stray dogs in the urban, rural and coastal areas of Mexicali County in northwest Mexico. In 2014, from January to December, 380 stray dogs were captured. The entire small intestine, cecum and faeces samples were collected and examined by using simple zinc sulfate flotation and Lugol's solution staining. Data were statistically analysed. Overall, about

21.5% of examined dogs were found positive for intestinal parasites. Toxocara canis was the most frequent detected parasite, with a prevalence of 7.1%, followed by Toxascaris leonina (5.5%), Cystoisospora spp. (5.0%), Taenia spp. (3.9%) and Dipylidium caninum (2.8%). Dogs were more frequently found to be infected with a single genus of intestinal parasite (18.7%) than co-infected (2.8%). Intestinal parasites were more prevalent in samples from the coastal area (25%) than in those from the rural (24.4%) and urban (20.6%) areas, however, only statistical association was found between capture area and specific intestinal parasitic infection. There were significant differences in the prevalence of taeniasis among two age groups . In previous studies conducted in the municipality of Mexicali in northwest Mexico, the overall prevalence of parasitic diseases in stray dogs was 66% (Luna et al 1981). In 2007, another report showed a 56.1% serologic prevalence of toxocariasis in domestic dogs (TinocoGracia et al 2007a) and contamination with Toxocara 106 TRASVIÑA-MUÑOZ ET AL canis eggs in 62.5% of soil samples collected from public parks and playgrounds where the presence of domestic and stray dogs was common and frequent (Tinoco-Gracia et al 2007b).

1.3. Public health significance of zoonotic parasitic agents

Intestinal parasitic infections are distributed virtually throughout the world, with high prevalence rates in many regions. Amoebiasis, ascariasis, hookworm infection and trichuriasis are among the ten most common infections in the world. Other parasitic infections such as abdominal angiostrongyliasis, intestinal capillariasis, and strongyloidiasis are of local or regional public health concern. The prevention and control of these infections are now more feasible than ever before owing to the discovery of safe and efficacious drugs, the improvement and simplification of some diagnostic procedures, and advances in parasite population biology.

The amount of harm caused by intestinal parasitic infections to the health and welfare of individuals and communities depends on: (a) the parasite species; (b) the intensity and course of the infection; (c) the nature of the interactions between the parasite species and concurrent infections; (d) the nutritional and immunological status of the population; and (e) numerous socioeconomic factors. All the above factors may in turn be modulated by seasonal and climatic conditions. Thus, while it is generally extremely difficult to measure the suffering caused by

infectious diseases, in the case of intestinal parasitic infections this is even more true because so many cases of the diseases are asymptomatic and therefore remain undetected. The public health significance of intestinal parasitic disease would be best assured if the available quantitative techniques could be adopted to evaluate morbidity.

The willingness of societies to pay for the elimination or the control of disease might be readily harnessed if government policy-makers and health planners could be given the chance to compare cost-benefit or cost-effectiveness analyses of the various courses of action with estimates of the predicted economic losses due to morbidity. For a given intestinal parasitic infection quantitative information might be sought about years of potential life lost, number of healthy days lost, incidence rates of the disease, and prevalence and case fatality rates by using quantitative methods now being used for other diseases (e.g., cancer and cardiovascular diseases).

The Food and Agriculture Organization's (FAO) interest in emerging diseases caused by foodborne pathogens derives from its role as the leading United Nations agency with a mandate for food quality and safety matters. The Food Quality and Standards Service of FAO's Food and Nutrition Division is active in all areas related to food safety and implements the FAO/World Health Organization Food Standards Program. Its activities include providing assistance to FAO's member nations in addressing problems,strengthening infrastructure, promoting standardization as a means of facilitating trade, and safeguarding the interests of consumers. This paper considers the importance of emerging foodborne diseases from the perspectives of the consumer, international trade in food, producers and processors, and developing countries and addresses prevention and control measures.(FAO,)

1.4. Classification of parasites

Parasites can be divided in to two broadly, Endoparasites(Nematodes/Round worms, Trematodes/flukes & Cestodes/tape worms) and ecto pararsites(Ticks, Mites, lices, fleas, flies & bedbugs). Nematodes (Roundworms) adult and larval roundworms are bisexual, cylindrical worms. They inhabit intestinal and extraintestinal sites.

1.4.1. Nematodes (Round worms)

The **nematodes** (Latin: *Nematoda*- **roundworms)** constitute the phylum **Nematoda** (also called Nemathelminthes) with plant-parasitic nematodes also known as **eelworms**. They are a diverse animal phylum inhabiting a broad range of environments.

They have tubular digestive systems with openings at both ends and nematode species can be difficult to distinguish from one another. Consequently, estimates of the number of nematode species described to date vary by author and may change rapidly over time.

A widely referenced article published in 1993 estimated there may be over 1 million species of nematodes A subsequent publication challenged this claim, estimating the figure to be at least 40,000 species . The life cycle of nematode has six stages. The egg state, J1 or first stage larva, J2 or second stage larva, J3 or third stage larva, J4 or fourth stage larva and the adult stage. The first four stages are the immature stages and are known as juvenile stages. Fertilized eggs are are rounded and have a thick shell with an external mammillated layer that is often stained brown by bile. In some cases, the outer layer is absent (known as decorticated eggs). Fertile eggs range from 45 to 75 µm in leng

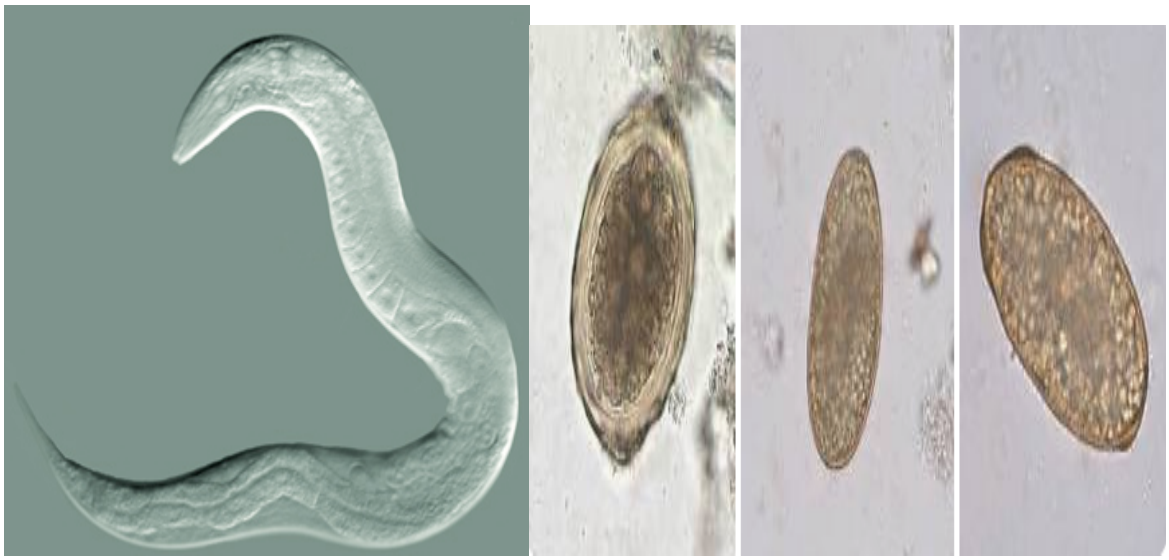


Figure 1.1. Model species of round worms and their eggs

Nematodes are dioecious, both sexes look similar. Reproductive system is tubular. Generally reproduction is sexual. Usually males are smaller than females or hermaphrodites (often much smaller) and have a characteristically bent or fan--shaped tail.Copulation between a female and a male nematode is necessary for fertilization except in the genus *Strongyloides,* in which parthenogenetic development occurs (i.e., the development of an unfertilized egg into a new individual).

In females the reproductive organs are used as traits for identification because the number of ovaries and the position of the vulva in the female nematode's body are easily seen under the light microscope.

Male nematodes have one or two testes and they are easily identified by the presence of spicules. Some evidence indicates that sex attractants (pheromones) play a role in heterosexual mating. During copulation, sperm is transferred into the vulva of the female. The sperm enters the ovum and a fertilization membrane is secreted by the zygote.

1.4.2. Trematode(Flukes)

flukes are flattened and generally leaf-shaped, dorsoventrally flattened body, bilateral symmetry, and a definite anterior end are features of platyhelminths in general and of trematodes specifically. Flukes are leaf-shaped, ranging in length from a few millimeters to 7 to 8 cm. The tegument is morphologically and physiologically complex. Flukes possess an oral sucker around the mouth and a ventral sucker or acetabulum that can be used to adhere to host tissues. A body cavity is lacking. Organs are embedded in specialized connective tissue or parenchyma. Layers of somatic muscle permeate the parenchyma and attach to the tegument.

Flukes have a well-developed alimentary canal with a muscular pharynx and esophagus. The intestine is usually a branched tube (secondary and tertiary branches may be present) consisting of

a single layer of epithelial cells. The main branches may end blindly or open into an excretory vesicle. The excretory vesicle also accepts the two main lateral collecting ducts of the excretory system, which is of a protonephridial type with flame cells. A flame cell is a hollow, terminal excretory cell that contains a beating (flamelike) group of cilia. These cells, anchored in the parenchyma, direct tissue filtrate through canals into the two main collecting ducts.

Except for the blood flukes, trematodes are hermaphroditic, having both male and female reproductive organs in the same individual. The male organ consists usually of two testes with accessory glands and ducts leading to a cirrus, or penis equivalent, that extends into the common genital atrium. The female gonad consists of a single ovary with a seminal receptacle and vitellaria, or yolk glands, that connect with the oviduct as it expands into an ootype. The tubular uterus extends from the ootype and opens into the genital atrium. Both self- and cross-fertilization occur.

The components of the egg are assembled in the ootype. Eggs pass through the uterus into the genital atrium and exit ventrally through the genital pore. Fluke eggs, except for those of schistosomes, are operculated (have a lid).

The blood flukes or schistosomes are the only bisexual flukes that infect humans. Although the sexes are separate, the general body structure is the same as that of hermaphroditic flukes. Within the definitive host, the male and female worms inhabit the lumen of blood vessels and are found in close physical association. The female lies within a tegumental fold, the gynecophoral canal, on the ventral surface of the male.



Figure1. 2a. Adilt trematode species(faciola, Schistosoma and paraphistomum respectively)

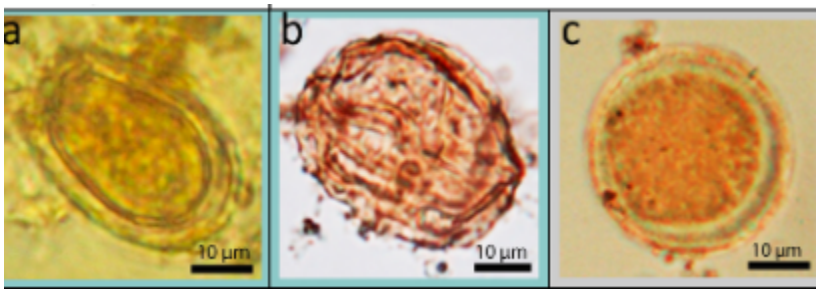


Figure 1.2b. Fasciola eggs

1.4.3. Cestode(Tape worms)

As members of the platyhelminths, the cestodes, or tapeworms, possess many basic structural characteristics of flukes, but also show striking differences. adult tapeworms are flattened, elongated, and consist of segments called proglottids. Tapeworms vary in length from 2 to 3 mm to 10 m, and may have three to several thousand segments.

Anatomically, cestodes are divided into a scolex, or head, which bears the organs of attachment, a neck that is the region of segment proliferation, and a chain of proglottids called the strobila. The strobila elongates as new proglottids form in the neck region. The segments nearest the neck are immature (sex organs not fully developed) and those more posterior are mature.

The terminal segments are gravid, with the egg-filled uterus as the most prominent feature. The scolex contains the cephalic ganglion, or "brain," of the tapeworm nervous system. Externally, the scolex is characterized by holdfast organs. Depending on the species, these organs consist of a rostellum, bothria, or acetabula. A rostellum is a retractable, conelike structure that is located on the anterior end of the scolex, and in some species is armed with hooks. Bothria are long, narrow, weakly muscular grooves that are characteristic of the pseudophyllidean tapeworms. Acetabula (suckers like those of digenetic trematodes) are characteristic of cyclophyllidean tapeworms.

Cestodes are hermaphroditic, each proglottid possessing male and female reproductive systems similar to those of digenetic flukes. However, tapeworms differ from flukes in the mechanism of egg deposition .*Hymenolepsis nana, Hdiminuta, Taenia saginata, T solium, Diphyllobothrium latum, Dipylidium craninum*. Note hexacanth embryos. Cysticercus larva in cow and pig

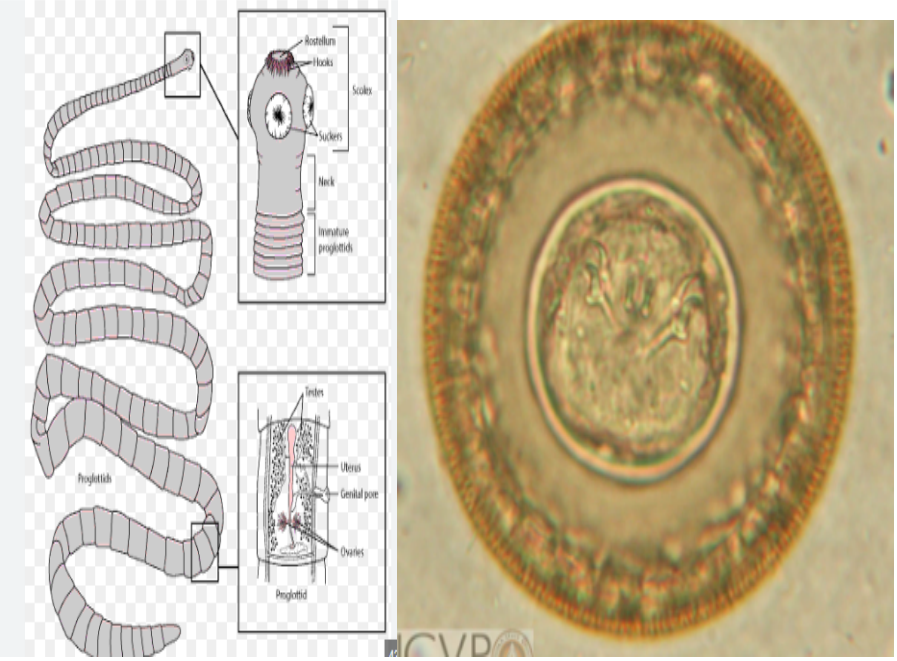


Figure 1.3- Adult Cestode parasite and its egg

1.5. Hosts and life cycles of common parasites

1.5.1. Taenia Saginata

Disease: Taeniasis **Definitive host**: Man **Adult parasite:** *Taenia saginata*

Intermediate Host: Cattle

Larval stage: *Cysticercus bovis / " beef measles"*

Life cycle

- Adult parasite resides in small intestine of man.
- Gravid proglottid & eggs are passed with human feces.
- Eggs are ingested by cattle.

- Onchospheres are liberated and penetrate the intestinal wall of cattle and enters the blood circulation.
- *Cysticercus* bovis develop in muscles of cattle.
- Larvae are ingested by humans along with raw or undercooked beef.
- Larvae evaginates and attaches to the human intestinal wall.
- The adult parasite develops to sexual maturity in 8-10 weeks

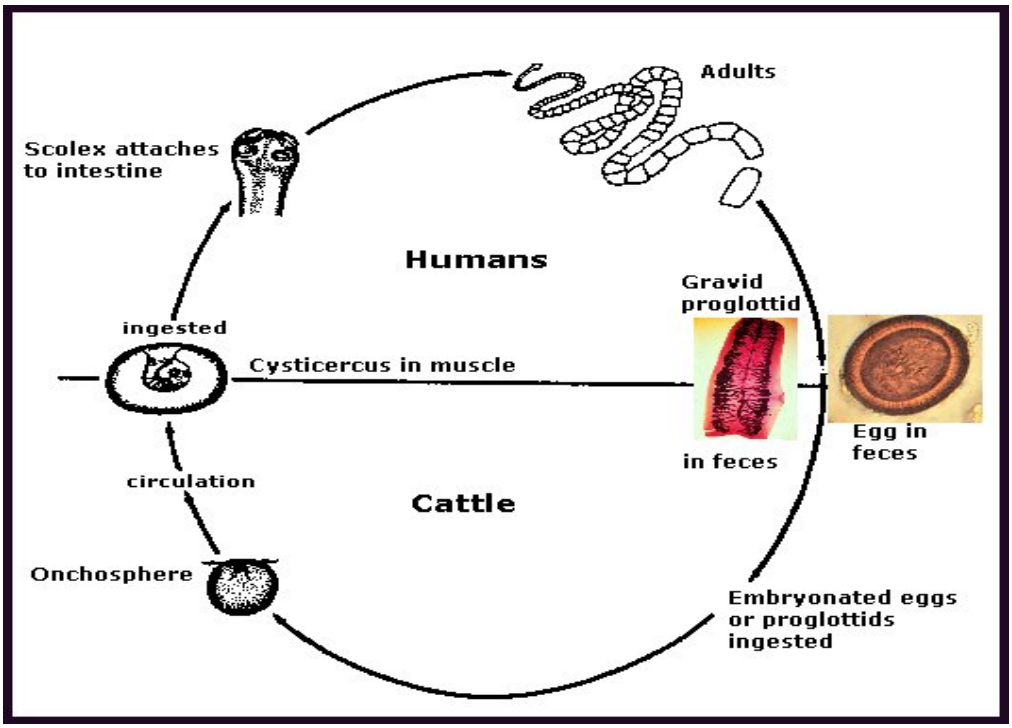


Figure1. 4. Teania Seginata life cycle

1.5.2. Echinoccocosis/Hydatidosis

Disease: Echinococcosis/ hydatidosis **Definitive host:** dogs and other canines

Adult parasite : *Echinococcus granulosus*

Intermediate host: Cattle, sheep, goats, pigs, camels including man.

Larval stage: hydatid cyst

Life cycle

- The adult parasite, *Echinococcus granulosus* resides in the small intestines the definitive hosts i.e dogs.
- Gravid proglottids and eggs are passed with feces.
- After ingestion by a suitable intermediate host (sheep, goat, swine, cattle, 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.
- The definitive host becomes infected by ingesting the cyst-containing organs of the infected intermediate host (Lung, liver, spleen, heart etc).

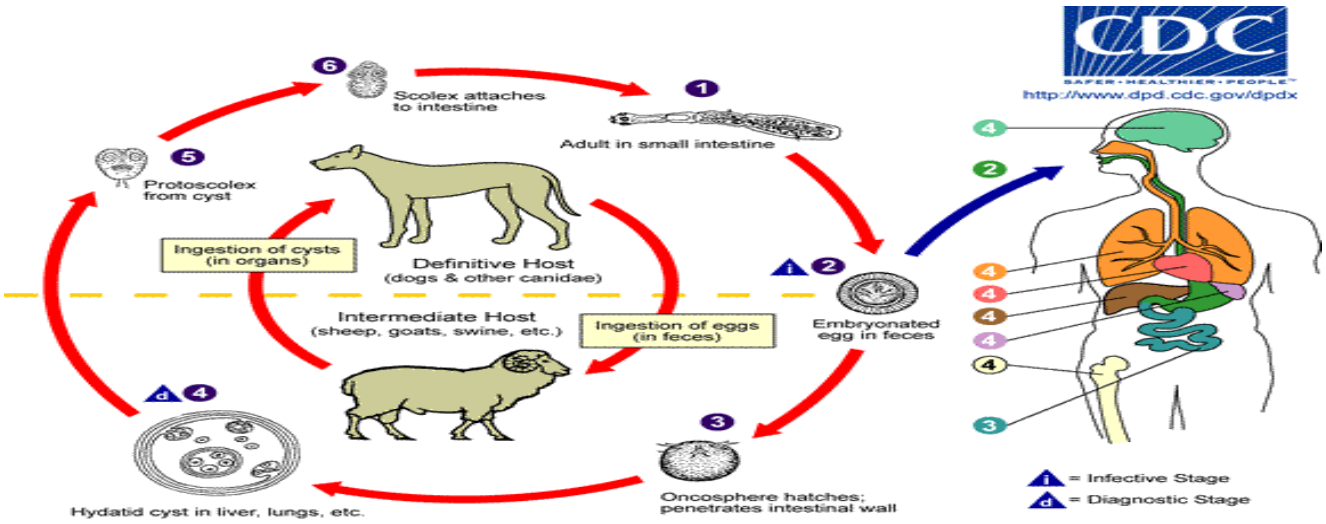


Figure 1.5. Hydatid cyct life cycle

Clinical Features in man

- No clinical signs are observed in animals.
- Hydatid cyst infections in man remain silent for years before the enlarging cysts cause symptoms in the affected organs.
- Hepatic involvement can result in abdominal pain, a mass in the hepatic area, and biliary duct obstruction.
- Pulmonary involvement can produce chest pain, cough, and hemoptysis.
- Rupture of the cysts can produce fever, urticaria, eosinophilia, and anaphylactic shock, as well as cyst dissemination.

1.5.3. Schistosoma/Bilharzia

Diseases- Schistosomiasis/Bilharziasis

Etiology- S. *Japonicum and S. mansoni* are considered to be true zoonotic disease.

Definitieve hosts: man, cattle, sheep, goats, and wild animals and birds.

Intermediate Hosts: different species of snails (amphibious snail)

Reservoir hosts: Cattle, buffaloe, pig, dog

Endemic to central and southwestern China, the Philippines and Indonesia.

In China and Phillipines 1.5–2.0 million people estimated to be infected

Host immune reaction to egg in liver may be fatal

Transmission: Cutaneous, ingestion, congenital

Risk groups

- Farmers working in irrigation (rice and sugar cane fields).
- Fishermen working in fish culture ponds.
- Children swimming in body of water contaminated with snails.
- women washing clothes and utensils at river banks

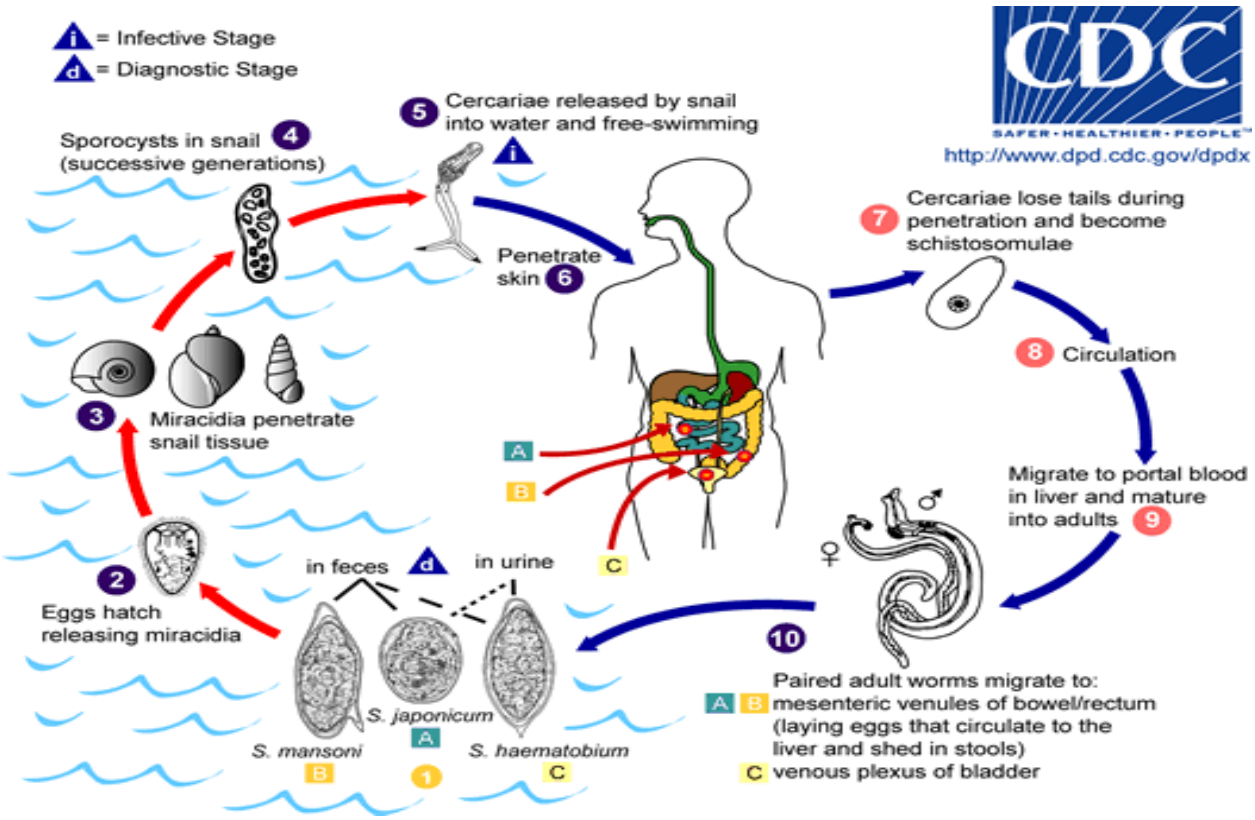


Figure1. 6. Schistosoma species life cycle

1.5.3. Toxoplasma

Foodborne: ingestion of undercooked or raw meat (lamb, pork) containing tissue cyst-bradyzoite, ingestion contaminated water, unwashed fruit and vegetables (sporulated oocysts) Animal-to-human (zoonotic) contact with cat feces- contamination while cleaning litter box, gardening (poor hygiene), Congenital- mother to fetus

- People at higher risk

- Veterinarians and para veterinary personnel
- Lab personnel
- Pet attendants
- Meat handler
- Pregnant women

Life cycle

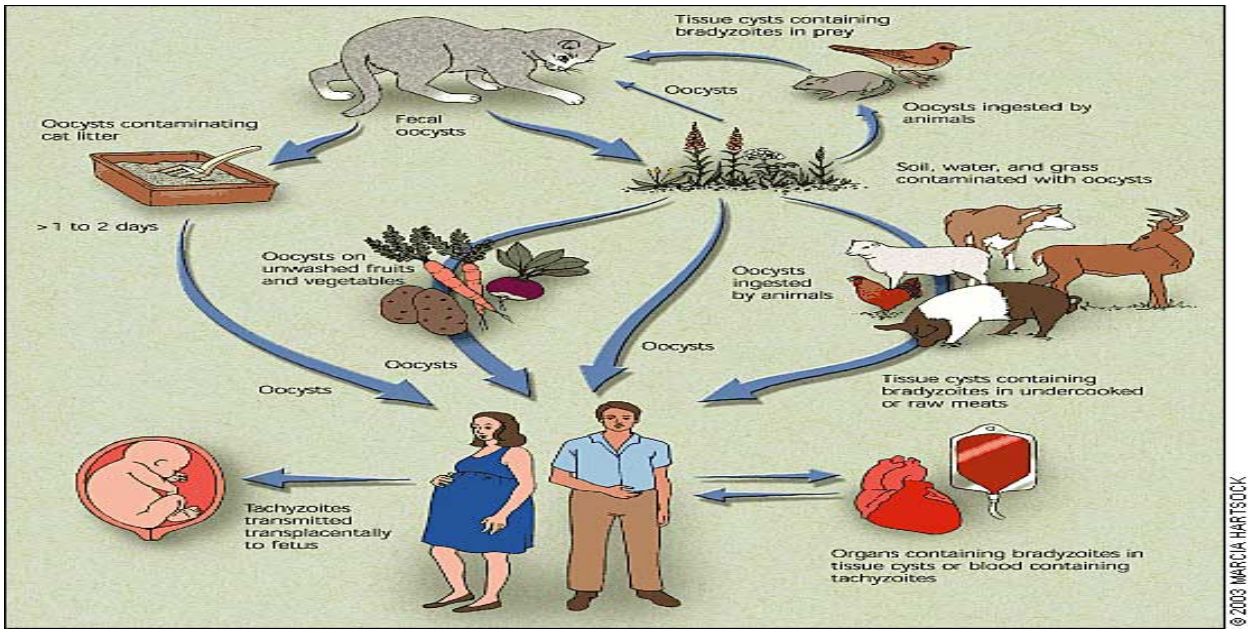


Figure1. 7. Life cycle of Toxoplasma species

1.5.4. Styrongyloides

The *Strongyloides stercoralis* life cycle is complex, alternating between free-living and parasitic cycles and involving autoinfection. In the **free-living cycle:** Rhabditiform larvae are passed in the stool of an infected definitive host, develop into either infective filariform larvae (direct development) or free-living adult males and females that mate and produce eggs , from which rhabditiform larvae hatch and eventually become infective filariform (L3) slarvae .The filariform larvae penetrate the human host skin to initiate the parasitic cycle (see below) This second generation of filariform larvae cannot mature into free-living adults and must find a new host to continue the life cycle.

Parasitic life cycle: Filariform larvae in contaminated soil penetrate human skin when skin contacts soil , and migrate to the small intestine . It has been thought that the L3 larvae migrate via the bloodstream and lymphatics to the lungs, where they are eventually coughed up and swallowed. However, L3 larvae appear capable of migrating to the intestine via alternate routes (e.g. through abdominal viscera or connective tissue). In the small intestine, the larvae molt twice and become adult female worms .

The females live embedded in the submucosa of the small intestine and produce eggs via parthenogenesis (parasitic males do not exist) , which yield rhabditiform larvae.

The rhabditiform larvae can either be passed in the stool (see “Free-living cycle” above), or can cause autoinfection habditiform larvae in the gut become infective filariform larvae that can penetrate either the intestinal mucosa or the skin of the perianal area, resulting in autoinfection. Once the filariform larvae reinfect the host, they are carried to the lungs, pharynx and small intestine as described above, or disseminate throughout the body. The significance of autoinfection in *Strongyloides* is that untreated cases can result in persistent infection, even after many decades of residence in a non-endemic area, and may contribute to the development of hyperinfection syndrome. The three most important definitive hosts for Strongyloides stercoralis are humans, dogs, and cats. They frequently use other mammals as hosts as well, though with much less frequency. Parasitic females feed on the tissue of the host's internal organs which includes the intestines as well as the lungs.

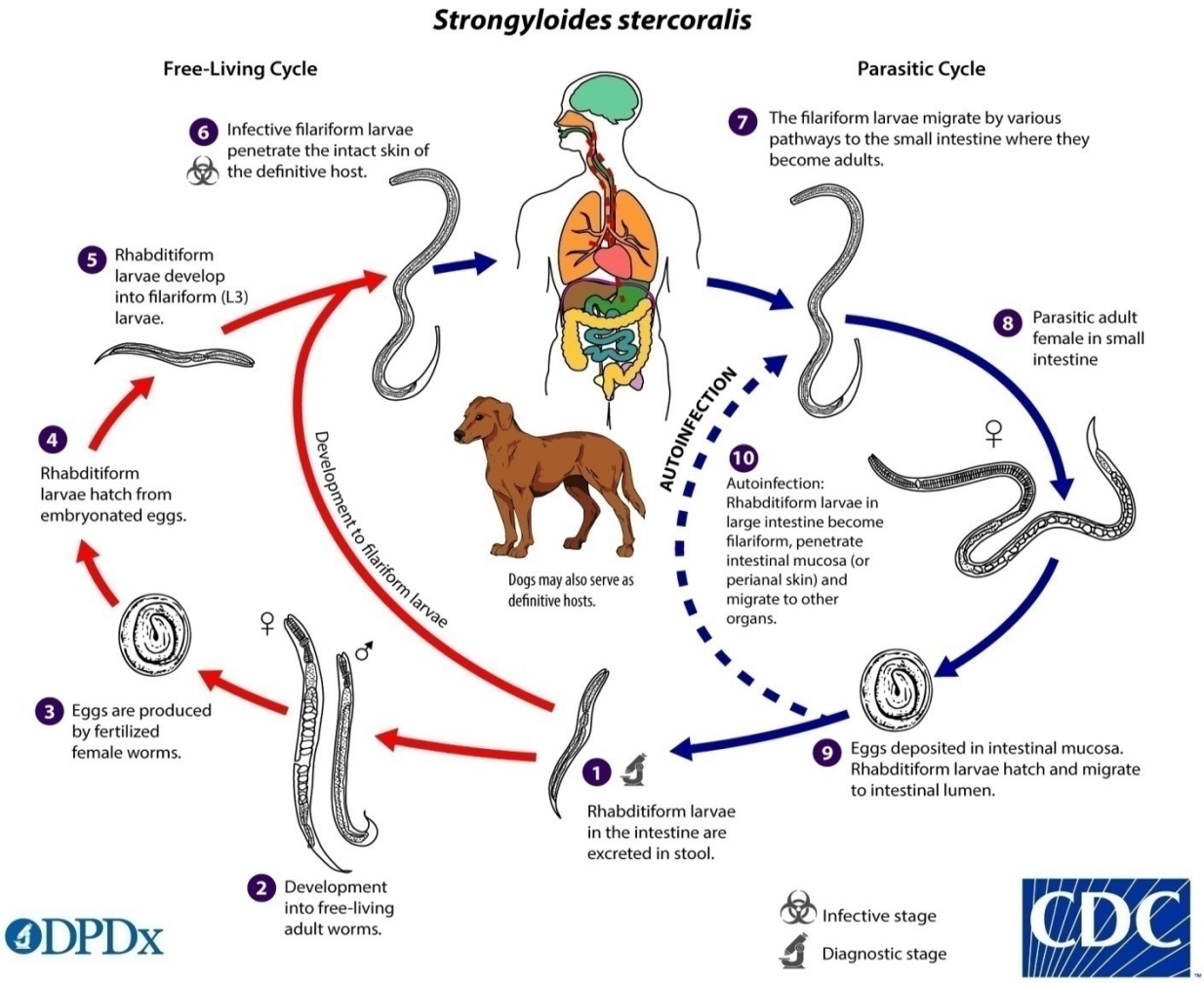

Strongyloides stercoralis
Free-Living Cycle
Parasitic Cycle
6 Infective filariform larvae penetrate the intact skin of the definitive host.
7 The filariform larvae migrate by various pathways to the small intestine where they become adults.
5 Rhabditiform larvae develop into filariform (L3) larvae.
8 Parasitic adult female in small intestine
AUTOINFECTION
10 Autoinfection: Rhabditiform larvae in large intestine become filariform, penetrate intestinal mucosa (or perianal skin) and migrate to other organs.
4 Rhabditiform larvae hatch from embryonated eggs.
Development to filariform larvae
Dogs may also serve as definitive hosts.
3 Eggs are produced by fertilized female worms.
9 Eggs deposited in intestinal mucosa. Rhabditiform larvae hatch and migrate to intestinal lumen.
1 Rhabditiform larvae in the intestine are excreted in stool.
2 Development into free-living adult worms.
DPDx
Infective stage
Diagnostic stage
CDC

Figure 1.8. life cycle of Strongyliodes stercoralis

Self-check 1	

Name…… ID…… Date…….

Directions: Answer the following questions below.

Part I: Choose the best answer among the given alternatives (2pts each)

1. From the following diseases which is zoonotic ?

   A, Teanasis B, Toxoplasmosis C, Trichostrongyloidosis D, all

2. Identify Economic impacts of parasitic diseases

   A, Death B, Trade ban C. anemia D. all

3. Identify clinic impacts of parasitic diseases

   A, Death B, decreased appetites C. anemia D. none

4. Among the followings ---------is/are trematodes?

   A. Fasciola B. Dictyocaulus C. Teania D. Ticks

5. Among the followings --------is/-are trematodes?

   A. Fasciola B. Dictyocaulus C. Teania D. Ticks

Prat II: Short Answer Questions (2pts each)

1. Write species of Teania that have zoonotic importance
2. Describe different types of hosts
3. What is intermediate hosts?
4. List clinical impacts of parasitic diseases
5. Write other name of Trematodes

Note: **Satisfactory rating - 20 points** **Unsatisfactory - below 20 points**

LG #6	LO #2- Identify and handle internal parasitic diseases of animals

Instruction sheet 2

This learning guide is developed to provide you the necessary information regarding the following content coverage and topics:

- Identify materials and equipments for diagnose and treatment of parasites
- Host, transmission, lifecycle, clinical sign, diagnosis, treatment and management of nematode parasites
- Host, transmission, lifecycle, clinical sign, diagnosis, treatment and management of trematode parasites
- Host, transmission, lifecycle, clinical sign, diagnosis, treatment and management of cestode parasites

- Host, transmission, lifecycle, clinical sign, diagnosis, treatment and management of protozoan parasites

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, you will be able to:

- Identify and implement Etiology, transmission, ecology, clinical sign, diagnosis, treatment and prevention/control measures of economically important and prevalent nematode parasites diseases of farm animals'
- Identify and implement Etiology, transmission, ecology, clinical sign, diagnosis, treatment and prevention/control measures of economically important and prevalent trematodes parasites diseases of farm animals'
- Identify and implement Etiology, transmission, ecology, clinical sign, diagnosis, treatment and prevention/control measures of economically important and prevalent cestodes parasites diseases of farm animals'
- Identify and implement Etiology, transmission, ecology, clinical sign, diagnosis, treatment and prevention/control measures of economically important and prevalent protozoan parasites diseases of farm animals.

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the information Sheets
4. Accomplish the Self-checks
5. Perform Operation Sheets
6. Do the "LAP test"

Information Sheet 2

2.1. Identify materials and equipments for diagnose and treatment of parasites .

Centrifuge, Microscopy, Slides, cover slides, test tubes, outoclave, oven, incubator, pippetes, Petridish, gloves, gown, thermometer, stetescopy syringe, needles, eyegoggle, respirator, mask, cottan, guaz, flask, rack, sieve, hemocytometer, beambalance, pistil and mortal

2.2. Host, transmission, lifecycle, clinical sign, diagnosis, treatment and management of nematode parasites

2.2.1. Hamonchus

Haemonchus contortus, better known as the barber pole worm or wire worm, is an intestinal worm that uses sheep and goats as a host . Adult worms live in the abomasum (also known as the true stomach) of an animal and feeds on their blood by puncturing the stomach. This causes haemonchosis, an infection characterized by severe anemia from blood loss. Collectively, worms can consume up to 1/10th of an animal's blood volume in 24 hours8. Haemonchus spp. has a direct life cycle. It is transmitted horizontally through grazing on natural pasturelands by third stage (L_3) larvae. It has a two-phase life cycle, a free-living and a parasitic within the abomasum of the host. Four host species (dromedary, zebu cattle, sheep and goats) share the same pastures for several months per year

Clinical Signs of Haemonchosis Signs of a Haemonchus contortus infestation include: anemia, dehydration, "bottle jaw" (accumulation of fluid in the lower jaw due to anemia), poor appetite, weight loss, and significantly reduced growth3,5,7,10. Due to anemia, the conjunctival mucous membranes around the eyes appear pale pink to while color

Life cycle of Haemonchus contortus.

Haemonchus is most commonly seen in the UK from late spring until autumn; the eggs don't survive well over winter but in warm and wet conditions the eggs hatch rapidly and pastures

become highly infective, resulting in very rapid and severe disease. The disease usually presents in two forms, depending upon the number of larvae on the pasture.

High numbers ingested cause severe anaemia, weakness and death. Low levels may result in a chronic infection; causing emaciation, moderate anaemia and fluid swelling under the chin (bottle jaw).

Haemonchosis can be easily confused with liver fluke infections, so it's important to consult us if you are concerned. Faecal egg counts will help us to diagnose cases, as very high counts (often over 10,000 eggs per gram!) indicate Haemonchus infections. The worm is best confirmed by post mortem, your vet will inspect the abomasum (one of the stomach compartments), where infected sheep will have large numbers of visible Barber's Pole worms. This is why affected sheep don't get diarrhoea, as the adults live in the stomach not the intestines.

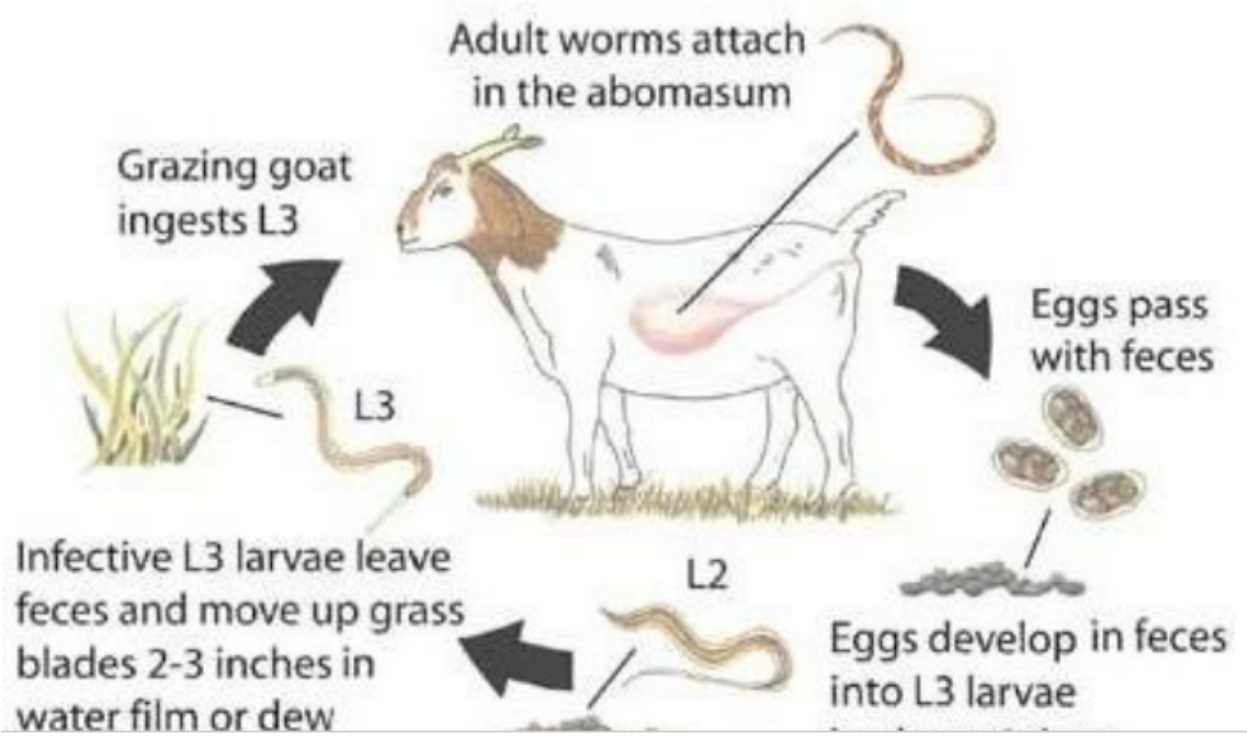


Figure1. 9. Hemonchus life cycle

Treatment/Prevention

Haemonchosis is a fatal disease of small ruminants caused by the parasite Haemonchus contortus (H. contortus).The most common drugs used in the treatment of H. contortus include albendazole, oxfendazole, and ivermectin.

However, as previously reported in the treatment of haemonchosis, these medicines have acquired drug resistance problems over time. Interestingly, natural plant compounds have demonstrated promising effects in the treatment of *H. contortus.* Therefore, the current study evaluated the

effects of plant extract, Ferula asafetida, against common drugs such as albendazole, oxfendazole, ivermectin, and closantel for the treatment of haemonchosis in small ruminants.

2.2.2. Trichostrongylus

The occurrence and impact of threadworms, Strongyloides spp., in companion animals and large livestock, the potential zoonotic implications and future research. Strongyloides spp. infect a range of domestic animal species worldwide and clinical disease is most often encountered in young animals.

Dogs are infected with Strongyloides stercoralis while cats are infected with different species according to geographical location (Strongyloides felis, Strongyloides tumefaciens, Strongyloides planiceps and perhaps *S. stercoralis.*

In contrast to the other species, lactogenic transmission is not a primary means of infection in dogs, and S. stercoralis is the only species considered zoonotic. Strongyloides papillosus in calves has been linked to heavy fatalities under conditions of high stocking density. Strongyloides westeri and Strongyloides ransomi of horses and pigs, respectively, cause only sporadic clinical disease.

These infections are generally of low relative importance in livestock and equines, most likely due to extensive use of macrocyclic lactone anthelmintics and/or improved hygiene. Future prevalence studies need to include molecular typing of Strongyloides species in relation to different hosts. More research is urgently needed on the potential zoonotic capacity of Strongyloides from dogs and cats based on molecular typing, information on risk factors and mapping of transmission routes. Threadworms, Strongyloides spp. within the Rhabditoidea superfamily are small (3–8 mm), slender nematodes of the small intestine (and caeca in some species). Only the female worms are parasitic and adult worms produce eggs by the process of parthenogenesis.

Life cycle

Larvated eggs or first stage larvae (L1), in the case of Strongyloides stercoralis, are excreted in the hosts' feces and undergo either rapid development through L1 and second stage larvae (L2) to infective third stage larvae (L3, homogonic cycle), or develop through the larval stages into

free-living adult worms that may reproduce in the external environment to generate infective L3 (heterogonic cycle). Under certain conditions, some Strongyloides species may have further generations externally, but at present it is unknown whether this takes place under natural conditions (Streit, 2008). The life cycle for Strongyloides ransomi of pigs, as representative of the species of veterinary importance.

Percutaneous or oral (by penetration of oral mucosa) infection by L3, after the free-living phase, is followed by migration; for example via the lymphatic or haemato-tracheal routes after which adult female worms develop in the proximal small intestine. The pre-patent period varies with nematode species and route of transmission. Lactogenic, mainly colostral, transmission from the dam to offspring following reactivation of inhibited somatic larvae is common in almost all species.

Strongyloides spp. infect a range of domestic animal species worldwide. In general, there is host specificity for each Strongyloides spp. However, this has been based primarily on morphology and not genetic confirmation. In studies on the prevalence and impact of infection, it is generally assumed that the species present is the one that has been previously identified as belonging to that host. Despite being capable of infecting different age groups, clinical disease is most often encountered in the young animals.

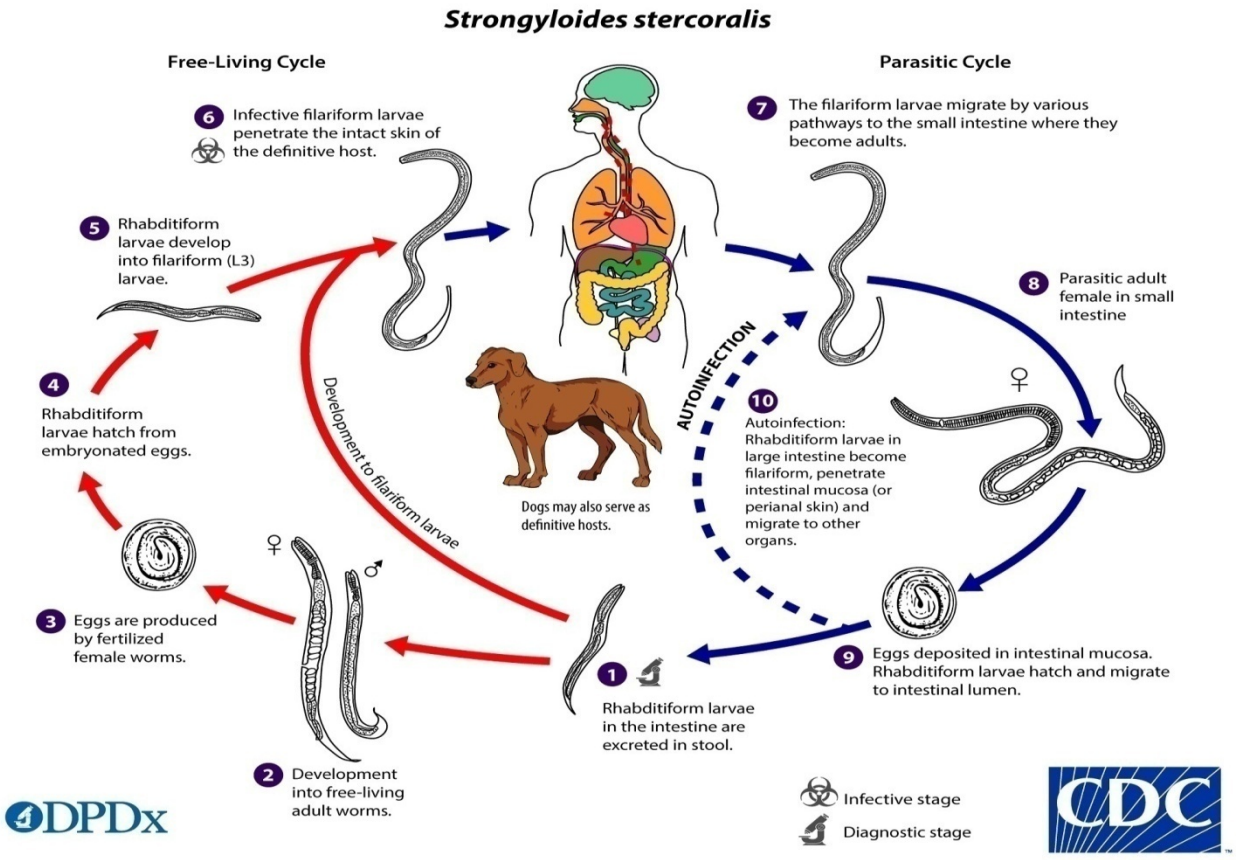


Figure1. 10. Styronyloides stercoralis

Clinical signs

Most often, *Strongyloides* occurs in neonatal puppies and kittens. The presence of clinical signs indicates that a heavy infection has been building up for some weeks. A blood-streaked, mucoid diarrhea, usually present in young animals during hot, humid weather, is characteristic. Emaciation

is often prominent, and decreased growth rate may be one of the first clinical signs. Appetite usually is good, and the dog or cat is normally active in the earlier stages of the disease.

In the absence of concurrent secondary infections, there is little or no fever. Usually in advanced stages, there is shallow, rapid breathing and fever, and the prognosis is grave. Autoinfection and systemic infections may be induced by the use of corticosteroids or other factors that affect immunocompetence. There may be larvae in tissues, and these animals are more likely to die. At necropsy, there can be evidence of verminous pneumonia with large areas of consolidation in the lungs as well as marked enteritis with hemorrhage, mucosal exfoliation, and much secretion of mucus.

Dignosis

- Baermann examination of feces
- Histopathology of small intestine at postmortem

First-stage *Strongyloides* larvae (~380 mcm long) may be identified by direct microscopic evaluation of a small quantity of feces. Usually, the Baermann technique is used to separate larvae from fecal material and increase the diagnostic sensitivity. Fecal PCR assay is also offered by some diagnostic laboratories. However, both Baermann and PCR assay examination have low sensitivity (20%–30%) due to inconsistent shedding of larvae; examination of multiple fecal samples increases the diagnostic sensitivity. It is important to use fresh fecal material obtained from an infected dog so the larvae can be easily differentiated from hookworm larvae or free-living soil nematodes. Occasionally, eggs (50–60 × 30–35 mcm) may be identified by flotation of fresh feces. Adult female worms can be identified by scraping the mucosa of the small intestine.

They are only ~2 mm long; however, the presence of eggs in the uterus easily differentiates them from larvae of other nematodes. Histopathology findings in the small intestine wall are characteristic.

Treatment

- Ivermectin or fenbendazole
- Supportive care

- Environmental management

Poor sanitation and mixing of susceptible and infected dogs can lead to a rapid buildup of the infection in all dogs in a kennel or pen. Dogs with diarrhea should be promptly isolated from dogs that appear healthy. Direct sunlight, increased soil or surface temperatures, and desiccation are deleterious to all free larval stages. Thorough washing of wooden and impervious surfaces with steam or concentrated salt or lime solutions, followed by rinsing with hot water, effectively destroys the parasite. Because the disease in humans can be serious, caution should be exercised when handling infected dogs. The disease in humans (as in dogs) is much more likely to be severe if the patient is immunosuppressed.

Infections in dogs can be treated with ivermectin (0.2 mg/kg, PO, daily, for two consecutive days) or fenbendazole (50 mg/kg/day, PO, for 5 days, repeated 4 weeks later). In cats, fenbendazole (50 mg/kg/day, PO, for 3 days) can be administered. These are not approved regimens in either cats or dogs. In all animals, feces should be examined regularly for at least 6 months after treatment to confirm efficacy.

2.2.3. Dictyocaulus

Dictyocaulus is a genus of nematode parasites of the bronchial tree of horses, sheep, goats, deer, and cattle. Dictyocaulus arnfieldi is the lungworm of horses, and Dictyocaulus viviparus is the lungworm affecting ruminants.

Dictyocaulus viviparous(Cattle lung worm)

Dictyocaulus viviparus is the most common lungworm of cattle; the infection is also known as husk or parasitic bronchitis. The parasite has a simple but interesting life cycle, with dispersal facilitated by a fungus.

Life Cycle

Adult D. viviparus worms reside in the bronchial tree of the animal's lungs. They lay eggs into the airways (bronchi). These eggs are coughed up and subsequently swallowed by the host. The eggs hatch into Stage 1 larvae (L1) in the gastrointestinal tract of the ruminant host. These L1 larvae are shed in the feces or manure. In the feces, larvae mature through two stages and become

infective as L3 stage larvae. Cattle feces supports the growth of Pilobolus sp. fungi. The L3 larvae of D. viviparus invade the inside of these fungi, and wait in the fungal sporangium. When the fungus sporulates, the larvae are dispersed as much as 10 feet in a pasture. Cattle grazing then ingest the L3 larvae. These larvae go through the intestinal system and penetrate the intestinal wall. They use the lymphatic system to reach the mesenteric lymph nodes, where they mature once again into L4 stage larvae. The L4 larvae use the blood supply and the lymphatic system to reach the lungs, where they become adults. The life cycle is completed.

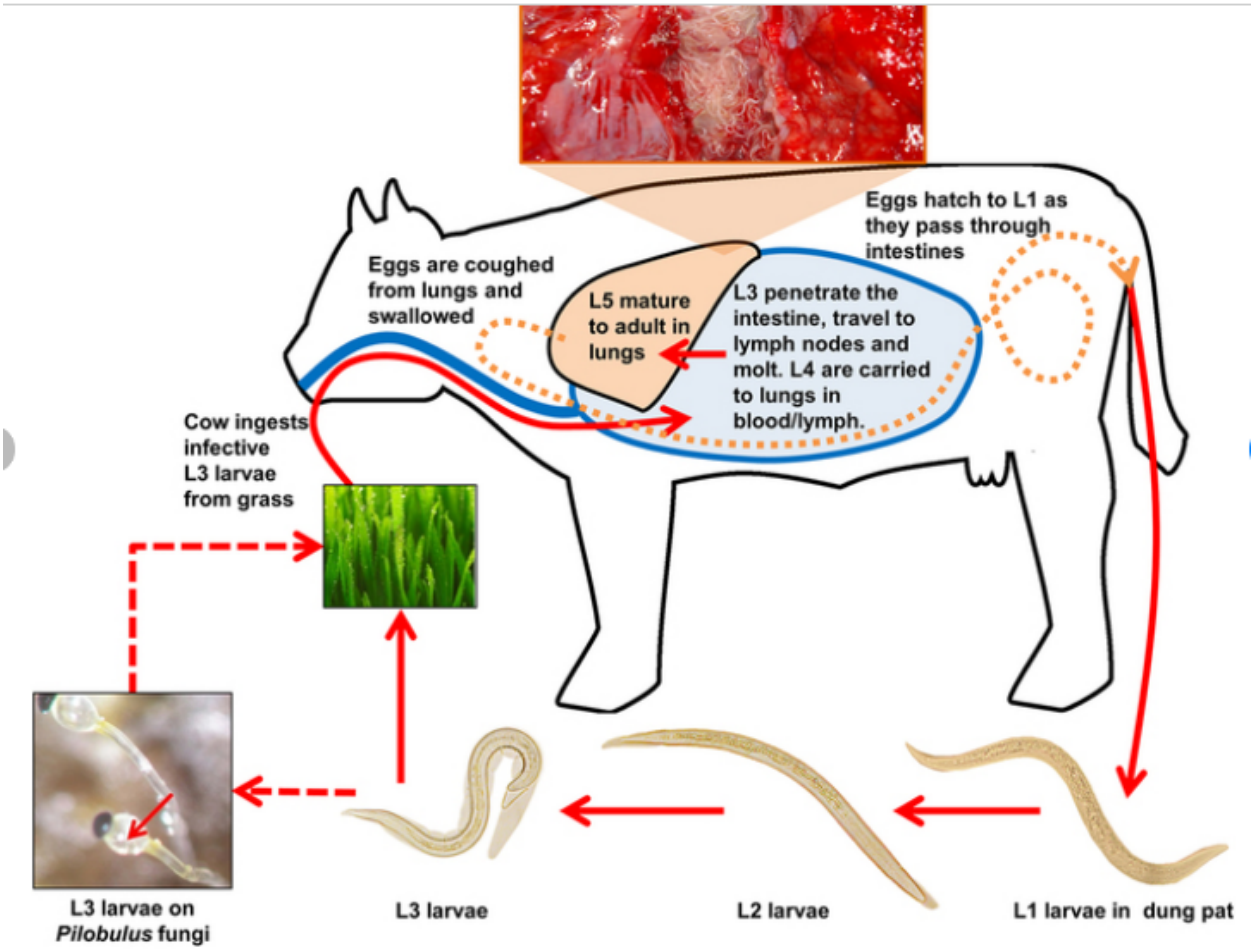


Figure1. 11. Dictyocaulus viviparous life cycle

Clinical sign

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

Diagnosis

- Microscope detection of L1 larvae in feces
- Microscope detection of eggs or larvae in the coughed up sputum or bronchoalveolar lavage fluid from affected animal
- Blood serology test for worm antigens (available in some countries / states)

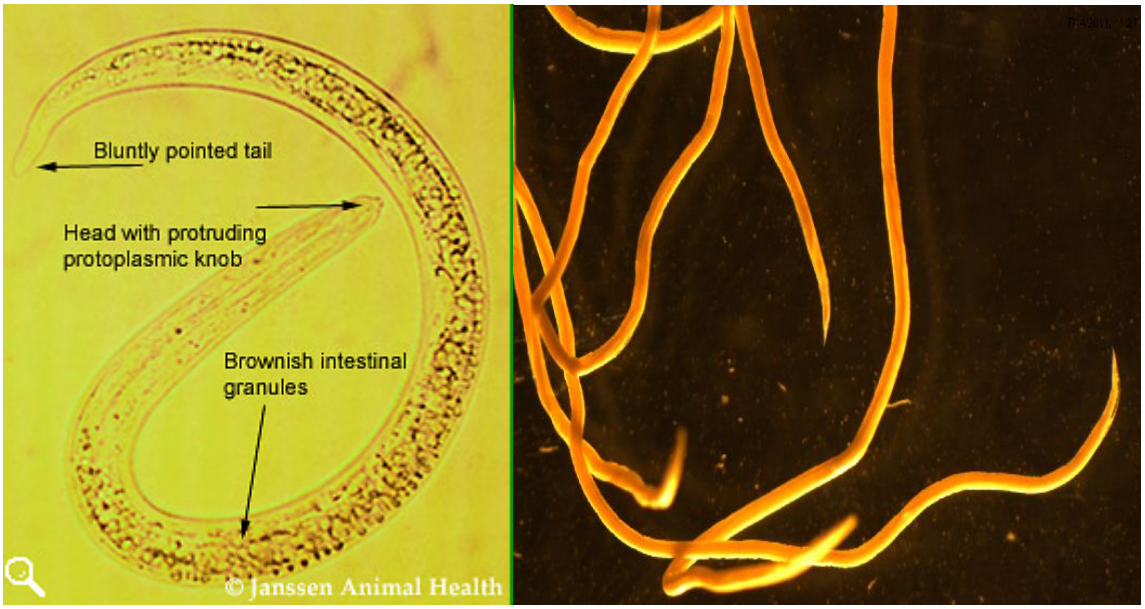


Figura 1.13.a , Dictyocaulus filaria(sheep/goat) **b,** Dictyocaulus viviparus(Lungworm of cattle)

https://youtu.be/FNH6Fejjvag and https://youtu.be/rKWAATIUnfE

Treatment(Rx)

Treatment of D. viviparus infection should be considered if chances of reinfection are high. This would include situations with high stocking rates, longer grazing seasons, and grazing on pastures where infected cattle have grazed. If treatment is

decided, ivermectin, albendazole, fenbendazole, febantel and other anthelmintics will target the lungworm. Pasture management should take into consideration the infectiveness of these organisms since the larvae can survive in the soil for extended periods of time.

Control of Dictyocaulus in animals

Lungworm infections in herds or flocks are controlled primarily by vaccination or anthelmintics. Oral vaccines are available in Europe for *D viviparus*. Two doses of irradiated infective larvae are administered 4 weeks apart, with the second dose given at least 2 weeks before the start of grazing or exposure to probable infection. Used properly, they prevent clinical disease; however, some vaccinated animals may become mildly infected to the extent that larvae are excreted, adding to pasture contamination and further infections.

Anthelmintic prophylaxis has become the mainstay of bovine lungworm control and has the added benefit of controlling GI nematodes as well in areas where anthelmintic resistance of GI nematodes has not become problematic.

With persistent anthelmintics (eg, ivermectin, doramectin, moxidectin, eprinomectin), two or three treatments during the grazing season, the timing of which depends on local grazing practice and epidemiology, are effective and may, by disrupting developing infections, stimulate immunity to the parasite. The use of multiple treatments, however, may delay immunity to *D viviparus* until the animal is an adult, when infection (albeit usually less severe) can occur.Other, more sporadic, infections can be controlled more easily by management, eg, not grazing horses with donkeys, not grazing sheep with goats, and indoor husbandry of pig cattle lungworm, Dictyocaulus viviparus , is a parasitic nematode that can cause severe pulmonary disease. Repeated natural infections result in protective immunity, and a live vaccine consisting of attenuated larvae was developed in the 1950's. However, development of immunity is sometimes reduced when the live vaccine is used together with prophylactic anthelmintic treatment against other nematodes, due to lack of boosting by natural infections.

As a consequence there is a need for alternative control methods. Development of a vaccine based on recombinant antigens and modern vaccine technology might provide such an alternative. To

enable the design of such a vaccine, identification of the immune mechanisms behind resistance to D. viviparus is fundamental. However, despite the long history of immunoprophylaxis, the understanding of the underlying immune reactions is poor and the studies published so far have mainly focused on the humoral immune responses.

2.2.4. Strongylus

Strongylus vulgaris (large strongyles) commonly known as the blood worm is a common horse parasite in the phylum Nematoda. It looks like a long worm with a large biting mouth. They are usually reddish in color because of all the blood they take from the equine host. This nematode is considered to be one of the "most pathogenic" of the large strongyles subphylum and is distributed worldwide, wherever there are grassland and temperate environments.

Life cycle

During the infective stage of the *S. vulgaris* life cycle, the larvae that have matured in the intestinal tract of the horse will migrate into the surrounding blood vessels. Once in the blood vessels, the larvae will continue their migration throughout the body to various organs causing damage to the blood vessels along the way. This can cause anemia or blockage of blood flow caused by the detachment of blood clots from the vessel wall resulting in tissue death. *S. vulgaris* are commonly found in pastures and stalls where feces are present. The larvae crawl up on the grass which is where they are then eaten by the horse. Harsh environments, such as freezing, do not kill *S. vulgaris*.

The adults of *S. vulgaris* are primarily found in the cecum and colon of infected equids and it is here that the females will lay their eggs. These eggs will pass out of the horse's body in the feces. Once outside the body the eggs will hatch and develop into the infective larval stage L3. The larvae enter the host when the horse grazes a patch of grass containing the larval stage. Back inside the host the infective larval stage migrates to the small intestine where it can enter the intestinal mucosa and further develop into the L4 stage. It is this stage that can enter the blood vessels and migrate throughout the body for a period of up to six months. During this time the L4 stage matures into the L5 or immature adult stage before returning to the intestinal wall. The L5

stage resides primarily in the cecum and colon where the males and females copulate thus starting the cycle over again.

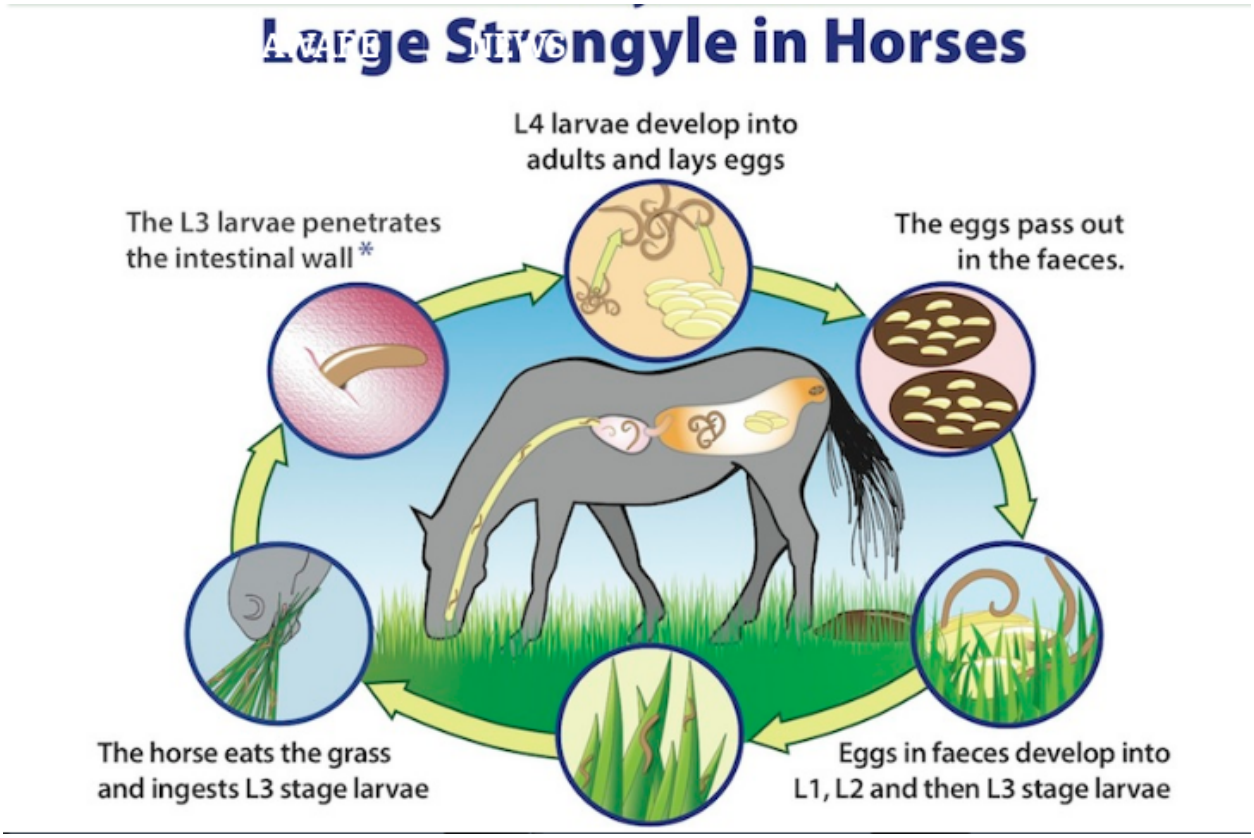


Fig 1.13. Strongyle life cycle



Figure 14. Strongylus vulguris in the intestine

Clinical Signs

Symptoms can range depending on how severe the infection is. In mild cases the most common clinical signs are weightloss, compromised performance, and a dull hair coat. Other clinical signs can range from diarrhea, weakness, anorexia, anemia if there are significant blood loss and abdominal discomfort. Severe cases can show signs of having severe colic, rupture of the intestines, and death. These signs may also be observed with other equine-related infections or illnesses and their presence alone should not be used to diagnose an infection of *S. vulgaris*. A veterinarian is required for further diagnosis when infection with *S. vulgaris* is suspected.

Diagnosis

Large strongyle infections are commonly diagnosed through the observations of fecal samples. These fecal samples can be viewed under a microscope to identify the eggs of *S. vulgaris*. Diagnosis via egg identification is not enough to determine if the horse has a *S. vulgaris* infection as the fecal samples often contain a mixture of large and small strongyle eggs that are very similar in appearance. The best diagnostic method to determine if a horse has a *S. vulgaris* infection is through a fecal culture. Here the eggs grow and develop into the infective larval stage that is specific to the large strongyle parasite.

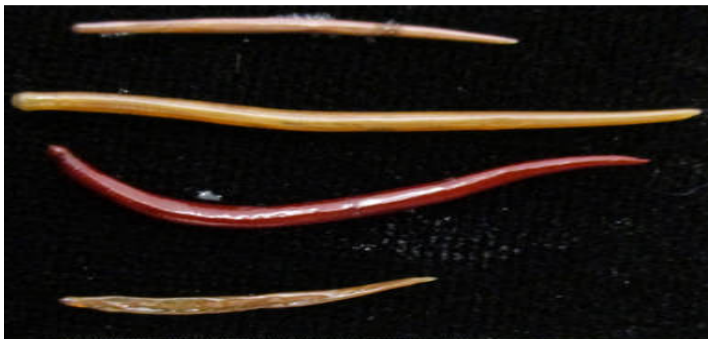
Treatment

Dewormers that have ivermectin or moxidectin are the most effective against these parasites. Treating a horse with regular dewormer when they have the worms inside of them may cause the worms to migrate all at once, causing rupture of the intestines and lead to death. Recommendations for deworming horses include performing fecal egg counts to determine what kind of protection each individual horse requires and alternating products in the fall and spring. An example would be using ivermectin based products (Equell or Zimectrin) in the spring and moxidectin based products in combination with praziquantel (Quest Plus) in the fall or vice versa. A deworming schedule should be discussed with a veterinarian.

Prevention

Horses are able to pick up this parasite from food and water contaminated by feces. This can be avoided by cleaning water tanks and picking out pastures often, avoidance of feeding on the ground, and avoidance of overstocking pastures. As foals can be extremely susceptible to this parasite, it is best to treat broodmares before they give birth and move them to uninfected pastures to avoid transfer. The three Strongylus species (S. vulgaris, S. equinus and S. edentatus) have migratory life cycles, with the larvae spending extended periods of time in various

predilection sites before returning to mature in the large intestine. Strongyles, also known as blood worms, are parasites ingested by the horse that can cause colic, anemia, diarrhea, weight loss, poor performance, and other problems.



a, Strongylus edentates



b,**Strongylus equines(Large Strongyle)**



C, Strongylus vulgaris



Figure1. 15. Strongylus species of (a.b c)

2.2.5. Bunostomum

Bunostomum is a genus of nematodes of the small intestine of ruminants and camelids. Important species include: *B. phlebotomum* in calves and *B. trigonocephalum* in lambs. The worms are stout and measure 1–3 centimetres (0.4–1.2 in) in length. Young animals are most commonly affected and only several hundred worms are necessary to cause morbidity. Adults often carry worms without showing clinical signs.

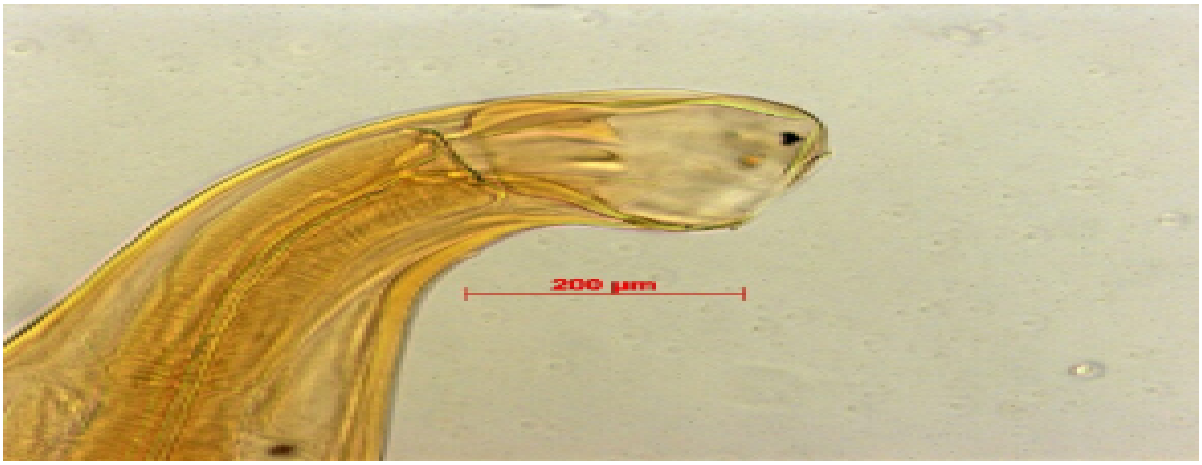


Figure16. Bunostomum

Life Cycle

Hookworm occurs in the duodenum and anterior part of the small intestine. The life cycle is direct, with a prepatent period of 30-56 days. Eggs hatch on the ground and develop into infective larvae in several weeks. Larvae penetrate through the skin or are ingested and then migrate to the respiratory system, are coughed up and swallowed and finally reach the small intestine. Eggs are then shed in the feces of the infected host.

Clinical Sign and Diagnosis

Diarrhoea is the primary sign, along with systemic signs of anorexia, lethargy and weight loss if the worm burden is severe. Hypoproteinaemia, anaemia and dehydration often occur.

Skin involvement due to larvae penetration appears as lesions especially on the feet and limbs. Analysis of faecal samples enables eggs to be found, or adults are visualised in the small intestine at necropsy

Treatment(Rx)

All available anthelmintics are efficient at treating the infection. Prophylactic anthelmintic treatme nt may help prevent the disease, as well as pasture management to avoid egg and larvae accumulation.This parasite is mostly a problem in dairy calves, 4-12 months of age and reared in the summer rainfall regions of coastal and subcoastal Australia. Disease occurs where heavy overstocking on permanently moist pastures occurs, or in calves managed on wet muddy yards or crowded onto boggy areas around water troughs or other watering points. Young cattle quickly develop a strong resistance to infection by about 12 months. Modern anthelmintics have reduced the incidence of this parasite.Further ecological information on worms and their control.

2.2.6. Ancylostoma

Ancylostoma braziliense is a species of hookworm belonging to the genus *Ancylostoma*. It is an intestinal parasite of domestic cats and dogs. Severe infection is often fatal to these pets, especially in puppies and kittens. The infection is particularly endemic in the southern United States. It is most often confused with the zoonotic hookworm species *Ancylostoma ceylanicum* because of their uncanny resemblance. *Ancyclostoma braziliense* larvae can cause accidental infection in humans called cutaneous larval migration or creeping eruption, which produces severe itching in the skin. It is the most common skin infection in tropical region, particularly along the beaches of the Caribbean. Like many other worms, the female worm is larger than the male. Also, the females are more difficult to distinguish because the teeth sizes appear very similar among the various species of *Ancylostoma*. Male worms, however, have two broad lateral lobes and a smaller dorsal lobe with rays on the copulatory bursa. These bursal rays' aids in distinguishing the different species of *Ancylostoma*

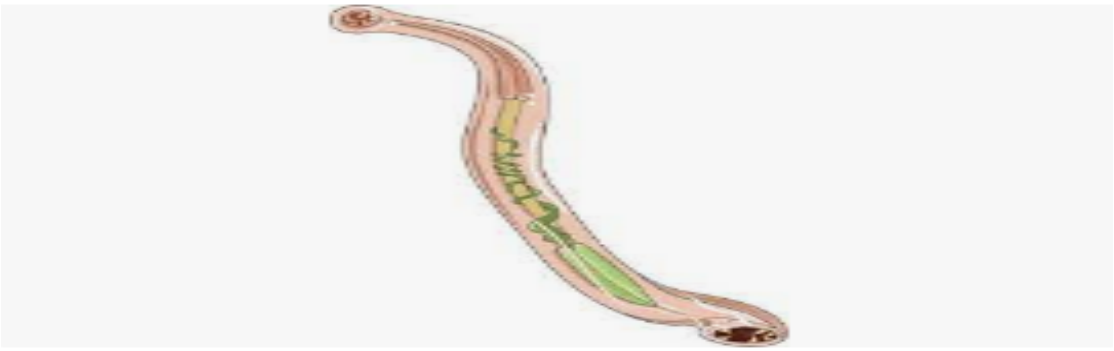


Figure1. 17. Ancyclostoma

Life Cycle

Ancylostoma braziliense eggs are passed into the environment through the faeces of cats and dogs. The eggs incubate on warm, moist soil, where they hatch into larvae. The infective juvenile penetrate the skin of the host. At this stage, the larvae are present in the epidermis, hair follicles, and glands of the skin, sometimes extending to sebaceous glands where they form coils. Then they migrate to the heart and lung by moving along the blood circulation. In the lungs, the juveniles enter the alveoli and are propelled by cilia up the respiratory tract. From this, the host swallow them and deposit them in the small intestine. They get attached to the intestinal mucosa via the buccal capsule. Here they undergo two successive molts to become sexually mature individuals.

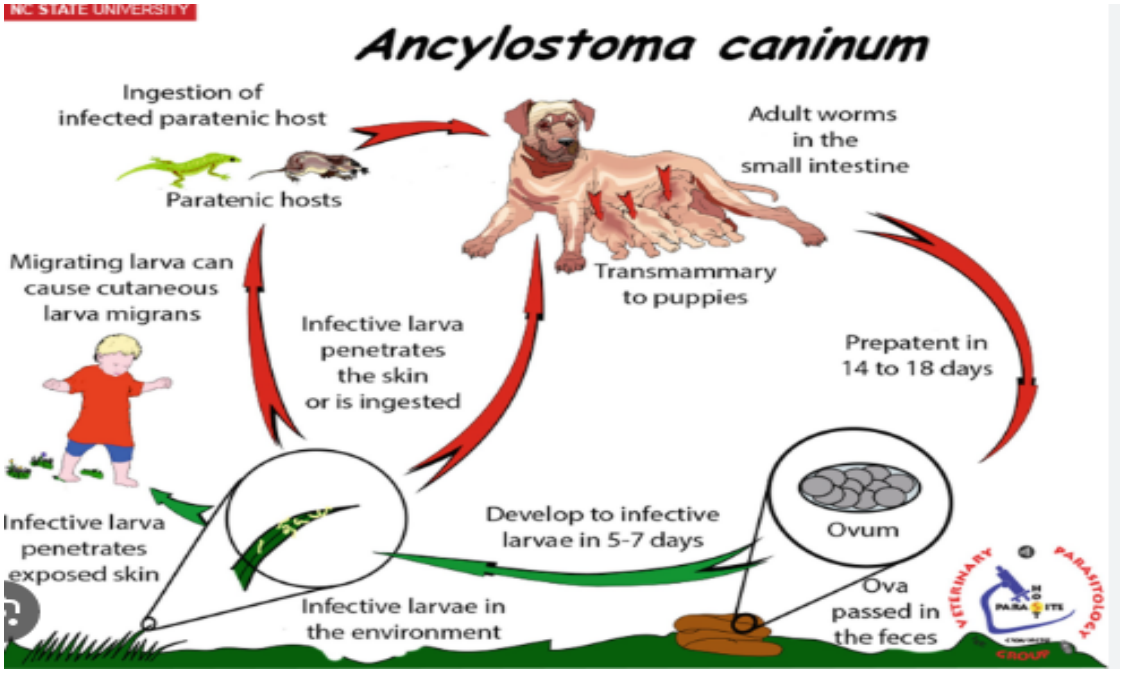


Figure1. 18. Ancyclostoma Life cycle

Clinical Sign

Cutaneous larva migrans

It can cause "creeping eruption" or "ground itch" It occurs when the larvae from the faeces of cats and dogs accidentally penetrate the human skin, causing severe itching and skin eruption. It is the most common ailment of tropically acquired dermatitis, particularly along the beaches of the Caribbean. However, humans are not the natural definitive hosts, and are therefore a deadend for the parasites. In humans, the larvae die after few months without further development.

Ancylostoma braziliense is a parasite that infects cats and dogs and occasionally humans. And,

they survive by eating blood from the intestine of cats and dogs, causing anemia. Pale gums and weakness are common signs of anemia. The infected animals also experience significant weight loss, bloody diarrhea, and are not able to grow up to their right size. Other symptoms like, skin irritation and itching is also caused by the larvae burrowing into the skin.

Diagnosis and Treatment

A. braziliense is difficult to differentiate from other hookworms. Microscopic examination of scatological samples can identify the eggs as they are generally smaller than those of other species. Most benzimidazoles are effective. Mebendazole, triclabendazole and fenbendazole are commonly used. Ivermectin and pyrantel pamoate are also effective. The combination of ivermectin 6 µg/kg and pyrantel pamoate at 5.0 mg/kg is 100% efficacious against adult worms in dogs. Human infection in case of cutaneous larva migrans is treated with oral thiabendazole at 500 mg in 4 daily dose Ancylostoma represents one of a group of helminth parasites (the hookworms) that gain access to the host via the skin and then progress on a complex migratory path.

2.2.7. Ascarididae

Ascaridia spp. (approximately 40 species have been reported in birds) are one of the most common nematodes in birds. They are large (16 to 120 mm), thick yellowish-white worms parasitizing the small intestine and sometimes the ceca of susceptible hosts. Females are often larger than males. Ascarids have a simple, direct life cycle. Nonembryonated, elliptical thick-walled single-cell eggs with fine granular brownish contents and colorless shell (approximately 80 × 50 µm) are shed in feces and embryonate in the environment with appropriate humidity and temperature in 2 to 3 weeks. Infective eggs hatch in the proventriculus or duodenum of the susceptible host releasing second-stage larvae that will invade small intestine mucosa where they molt to L3.

Depending on the species and host, larvae either return to the intestinal lumen where they molt and mature into adult worms or they may arrest their development at certain stage (i.e., L3) to become the predominant form of the parasite and become its reservoir throughout the life of the

host. Ascarids may use transport or paratenic hosts, such as earthworms or grasshoppers, but they do not develop or accumulate in them.

Ascaridia is a genus of parasitic nematodes. Members of the genus are primarily intestinal parasites of birds. Three species are well known, namely, *A. galli* found mostly in chickens, *A. dissimilis* in turkeys, and *A. columbae* in pigeons. Lesser known species such as *A. hermaphrodita*, *A. sergiomeirai*, *A. ornata*, *A. nicobarensis*, and *A. platyceri* are found in parrots.

Among these, *A. galli* is the most important and most pathogenic species, responsible for ascaridiasis of poultry.[3] The eggs of these nematodes are characterized by a thick shell, smooth and ellipsoidal, and composed of three distinct layers.

The eggs of *A. dissimilis* are marginally bigger than *A. galli*, and *A. columbae* is marginally smaller. All these ascarid eggs are similar in appearance to *Heterakis gallinarum*. All three species exhibit a direct lifecycle involving the release of eggs into the soil and the subsequent reingestion of them in food. *A. columbae* may also migrate through the liver.

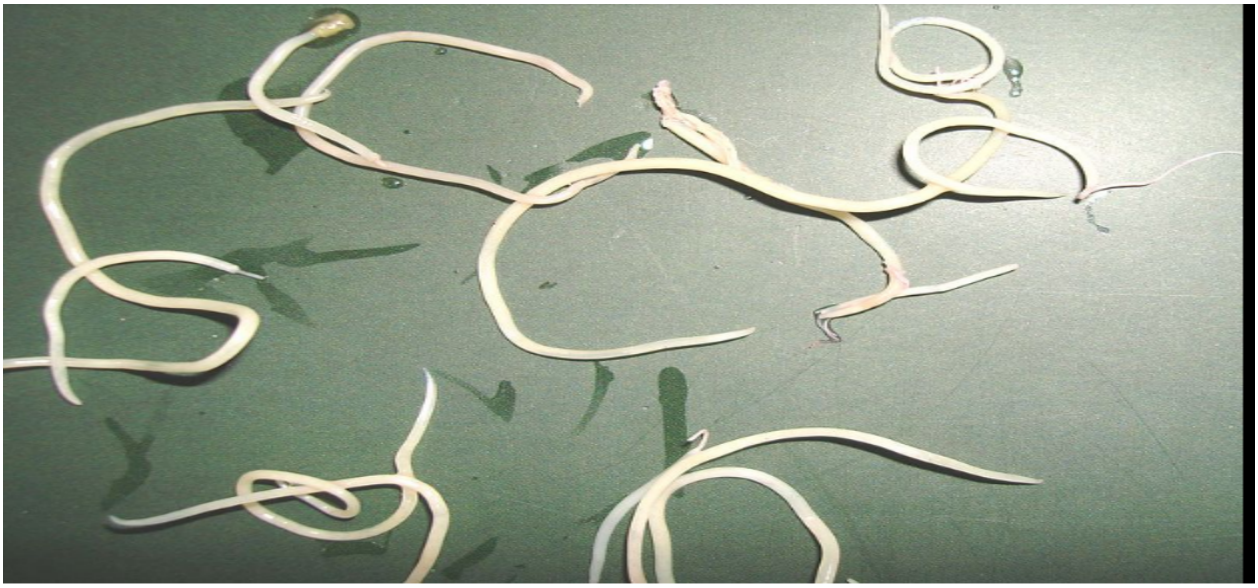


Figure1. 19-Ascarid galli

Life cycle

Ascaridia spp. (approximately 40 species have been reported in birds) are one of the most common nematodes in birds. They are large (16 to 120 mm), thick yellowish-white worms parasitizing the small intestine and sometimes the ceca of susceptible hosts. Females are often larger than males. Ascarids have a simple, direct life cycle. Nonembryonated, elliptical thick-walled single-cell eggs with fine granular brownish contents and colorless shell (approximately 80 × 50 µm) are shed in feces and embryonate in the environment with appropriate humidity and temperature in 2 to 3 weeks. Infective eggs hatch in the proventriculus or duodenum of the susceptible host releasing second-stage larvae that will invade small intestine mucosa where they molt to L3. Depending on the species and host, larvae either return to the intestinal lumen where they molt and mature into adult worms or they may arrest their development at certain stage (i.e., L3) to become the predominant form of the parasite

and become its reservoir throughout the life of the host. Ascarids may use transport or paratenic hosts, such as earthworms or grasshoppers, but they do not develop or accumulate in them.

Clinical Sign and Diagnosis

Symptoms of heavy infection include **anorexia, diarrhea, stunted growth, listlessness, a change in behavior, and enteritis**. Heavy infections can cause a partial or total obstruction of the gastrointestinal tract. Laying hens may produce soft eggs with thin and misshapened shells. Clinical signs are more common in young chickens less than three months old, as older chicken develop some resistance to infection. Infection may be diagnosed on clinical signs and fecal examination. Alternatively, *post mortem* examination should demonstrate enteritis - caused by the emergence of larvae from the mucosa. The gastrointestinal tract may also be distended in chronic cases.

Treatment

Piperazine salts, levamisole, and benzimidazoles are all reported treatments. Ascarid eggs are resistant to desiccation, persist for a long time in the environment, and remain directly infective. Therefore, control of infection involves the prevention of contamination of feeders and drinkers with feces (by raising them off the ground), pasture rotation, and regular dosing with the above mentioned treatments, especially in young birds.

Prevention and Control

Pasture Rotation, Disinfectants, and Nutritional Management- In ruminants, pasture rotation is considered as a nonchemical method that can be adopted to reduce environmental contamination with free-living stages of parasites. In laying hen flocks, pasture rotation did not reduce Ascarid infections significantly. Recently, it has been reported that about 2 to 3% of A. galli eggs remained viable and infective for up to 2 yr in the pasture. This indicates that used pastures or areas can harbor a large number of residual eggs from the previous flock thus reducing the effectiveness of pasture rotation management in A. galli. Disinfecting the empty barns with adequate active components before hen's placement can help in preventing the

transmission of A. galli infections to some extent. Use of chlorocresol disinfection solution of 1 to 2% on the shed was found to be ovicidal.

Selective Breeding for Disease Resistance

Lohmann Brown laying hens are considered more resistant to parasites than other breeds. A study conducted in 2 brown genotypes (Lohmann Brown plus and Lohmann Brown classic) showed the heritability for worm burden to be 0.55 and 0.56, respectively . This indicates the possibility of selecting hens based on superior genetics for parasite resistance. Further research could investigate the genetic background of resistance mechanisms at the molecular level. Similarly, another study was conducted to compare resistance to A. galli infection in Lohmann Brown and Danish Landrace laying hens. In this experiment, significantly lower worm burden and nematode eggs shed were observed in Lohmann Brown hens, indicating that breeding for resistance to A. galli is possible. A self-cure mechanism was observed in both of these breeds when infected at a week old. These hens could expel infections when they were challenged secondarily.

Biosecurity

Adherence to strict biosecurity measures can be helpful in preventing transmission of parasitic infections. Infective eggs which are present in the soil/pasture can be easily transmitted between farms or between areas within farms because these eggs can adhere to people, machinery, or other materials. There are some husbandry-based control measures that can reduce the level of parasitic infections such as dead bird disposal, preventing rodents and wild bird entrance, supplying clean feed and water, and restrictions of entry for personnel and vehicles

Anthelmintics and Immunization

In Australia, chemicals, anthelmintics, and pesticides are registered for the control of internal or external parasites in avian species. Levamisole and piperazine are currently the registered drugs for treatment of nematode infections in commercial layers in Australia with a nil withholding period for eggs. Commonly used anthelmintics from the benzimidazole group in Australia include mebendazole and fenbendazole which are mostly applied during rearing as they have an egg

withholding period. There is widespread anthelmintic drug resistance in many parts of the world for some parasites of livestock. No data for drug resistance in A. galli populations is available. The fact that this registered product has been commonly used for decades may indicate that selection for resistance has already occurred.

Therefore, it is important that methods for early infection detection are available to monitor the efficacy of anthelmintics in the production setting. The reliance on one active compound, levamisole to control A. galli in laying hens is a concern. Should resistance arise, control will become very difficult, so the registration of additional products or chemicals is desirable for the future of the free-range egg industry. Benzimidazole (flubendazoles and fenbendazole) is commonly used in Europe by applying in drinking water to control nematode parasites in egg laying birds. It was found effective in killing adult worms and also interrupted the worm's egg output temporarily. Some of the parasites may survive benzimidazole treatment and can later mature and reappear to cause damage .

However, no evidence of resistance of benzimidazole in A. galli infections has yet been documented. Vaccine development for nematode parasites like A. galli can be challenging due to the presence of the parasite using several life stages within the host and secondly due to their location in tissues with less accessibility to immune effector cells such as the intestinal lumen and mucosa. It has been reported that there is no protective maternal immunity against A. galli parasite. Chickens immunized with a crude extract of A. galli following an oral or intramuscular route of immunization developed a humoral and cell-mediated immune response.

However, to date the immune response initiated using A. galli vaccines failed to protect against A. galli infection.A. galli infection at the time of vaccination influenced the development of both humoral and cell-mediated immune responses induced by a live attenuated commercial Newcastle Disease Virus vaccine. In contrast, another field study showed no effects of helminth infection on the vaccine induced immune response in chickens.

These findings highlight the critical need for more studies to confirm the influence of gastrointestinal nematode infection on the magnitude and type of responses achieved through vaccination.

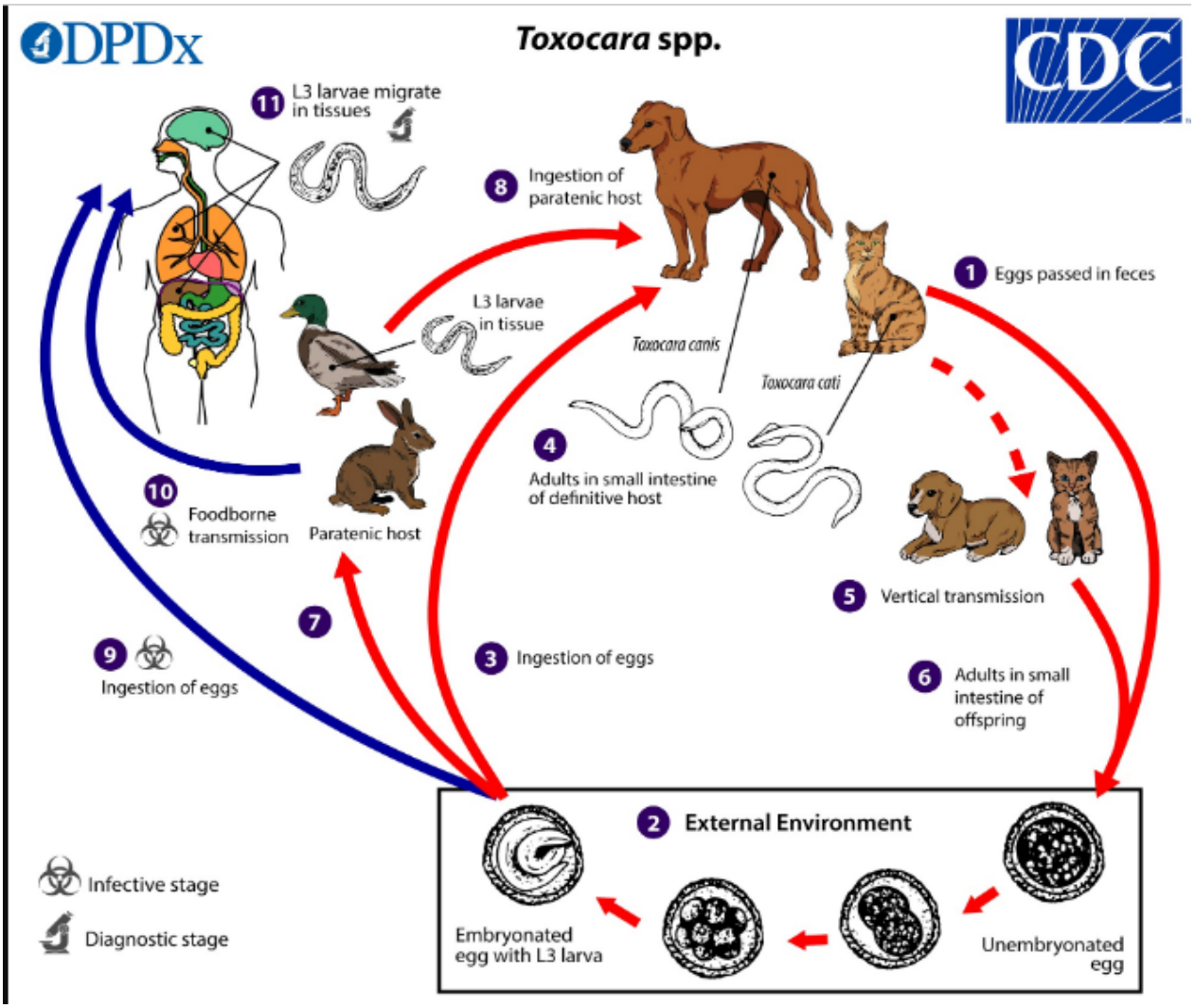


Figure1. 19- life cycle of Toxocara species

Toxocariasis in humans is caused by infection with larvae of *Toxocara* spp., which are common ascarid roundworms of mammals. Confirmed zoonotic species include the dog roundworm *T. canis* (presumed most common) and the cat roundworm *T. cati* (frequency not known). It is not known whether other closely-related *Toxocara* species can infect humans (e.g. *T. malaysiensis* of cats).

Toxocara spp. can follow a direct (one host) or indirect (multiple host) life cycle. Unembryonated eggs are shed in the feces of the definitive host (canids: ***T. canis;*** felids: ***T. cati***) . Eggs embryonate over a period of 1 to 4 weeks in the environment and become infective, containing third-stage (L3) larvae Following ingestion by a definitive host , the infective eggs hatch and larvae penetrate the gut wall. In younger dogs (***T. canis***) and in cats (***T. cati***), the larvae migrate through the lungs, bronchial tree, and esophagus, where they are coughed up swallowed into the gastrointestinal tract; adult worms develop and oviposit in the smallintestine . In older dogs, patent (egg-producing) infections can also occur, but larvae more commonly become arrested in tissues.

Arrested larvae are reactivated in female dogs during late gestation and may infect pups by the transplacental (major) and transmammary (minor) routes in whose small intestine adult worms become established. In cats, ***T. cati*** larvae can be transmitted via the transmammary route to kittens if the dam is infected during gestation, but somatic larval arrest and reactivation does not appear to be important as in ***T. canis***. ***Toxocara*** spp. can also be transmitted indirectly through ingestion of paratenic hosts. Eggs ingested by suitable paratenic hosts hatch and larvae penetrate the gut wall and migrate into various tissues where they encyst . The life cycle is completed when definitive hosts consume larvae within paratenic host tissue , and the larvae develop into adult worms in the small intestine.

Humans are accidental hosts who become infected by ingesting infective eggs or undercooked meat/viscera of infected paratenic hosts . After ingestion, the eggs hatch and larvae penetrate the intestinal wall and are carried by the circulation to a variety of tissues (liver, heart, lungs, brain, muscle, eyes) . While the larvae do not undergo any further development in these sites, they can

cause local reactions and mechanical damage that causes clinicaltoxocariasis.

Hosts

Toxocara canis infects essentially all wild and domestic canids; patent infections are more prevalent among puppies than older dogs. ***T. cati*** is found in wild and domestic felids of all ages, but patent infections are slightly more common in kittens.

Paratenic host ranges for both species encompass numerous species of mammals and birds. Livestock are important paratenic hosts; some human cases have been linked to consumption of undercooked beef, lamb, chicken, and duck meat (particularly liver).

Cockroaches and earthworms have been experimentally infected, and could possibly serve as paratenic or transport hosts.

Geographic Distribution

Toxocara canis and ***T. cati*** are cosmopolitan parasites of domestic dogs and cats. While common globally, prevalence in both animals and people is highest in developing countries. In developed countries, more infections are detected among persons in lower socioeconomic strata.

Clinical Presentation

The main clinical presentations of toxocariasis are visceral larva migrans (VLM) and ocular larva migrans (OLM), although most infections are asymptomatic. In VLM, which occurs mostly in preschool children, the larvae invade multiple tissues (commonly liver, lung, skeletal muscle, occasionally heart) and cause various nonspecific symptoms (e.g. fever, myalgia, weight loss, cough, rashes, hepatosplenomegaly) usually accompanied by hypereosinophilia. Migration to the central nervous system (neurotoxocariasis or neural larva migrans (NLM)) is uncommon and can cause eosinophilic meningoencephalitis. Death can occur in instances of severe cardiac, pulmonary, or neurologic involvement.

In OLM, the larvae produce various ophthalmologic lesions, and may cause diffuse unilateral subacute neuroretinitis (DUSN). Involvement is typically unilateral (affecting one eye) and associated visual impairment usually presents with uveitis, retinitis, or endophthalmitis; permanent

visual damage or blindness can occur. Associated larval granulomas have in some cases have been misdiagnosed as retinoblastoma. OLM most often occurs in older children or young adults, who uncommonly have visceral manifestations.

2.2.8. Oxyuris

The equine pinworm**,** Oxyuris equi, has a worldwide distribution The pinworm *Oxyuris equi* occurs in horses and other equids throughout the world,. The parasite is reported to be far more common in young horses but can occur at any age. Although the larval stages cause inflammatory reactions in the mucosal walls of the cecum and ventral colon, no associated clinical manifestations have been described. Egg masses may cause local irritation and pruritus, which may progress to tail-rubbing. Cellophane tape tests or microscopy of perianal deposits may allow identification of the eggs. Treatments include washing of the perianal area along with oral administration benzimidazoles.

Recently, O equi has been reported resistant to ivermectin on several different continents. Equine pinworms are commonly found across the world. Two species exist, *Oxyuris equi* and *Probstmayria vivipara*, but the latter has not been found to be clinically relevant and is not discussed further.

For *O equi*, the infective stage is the embryonated egg. Upon ingestion, L3 larvae are released and invade mucosal walls,primarily in the ventral colon and to a lesser extent the cecum. They enter the intestinal lumen as L4 larvae and move to the dorsal colon, which is the predilection site for the adult stage. Gravid females migrate through the descending colon and rectum to deposit their eggs in packets onto the perianal skin (8,000–60,000 eggs in one deposit) and subsequently die in the environment. This process takes ~5 months to complete. Females can sometimes be observed in the feces, and due to their stout appearance (7–15 cm length), they can be mistaken for ascarids. However, their tapered posterior ends can be used to confirm identification.

Life cycle

Adult *O. equi* live in the large intestine. After mating, the males die and the females move to the rectum. With (or without) defecation, the females extend their long tails beyond the anus and lay

eggs in clumps on the peri-anal skin. These clumps are held in place with an adhesive produced by the female nematode. An infective larva develops within each egg over a minimum of a few days, during which period the adhesive loses its stickiness, and the egg drops to the ground. Infection of the horse is by ingestion of infective, larvated eggs. The eggs hatch within the small intestine and the larvae released undergo a mucosal migration in the large intestine. The pre-patent period for *O. equi* is approximately five months.

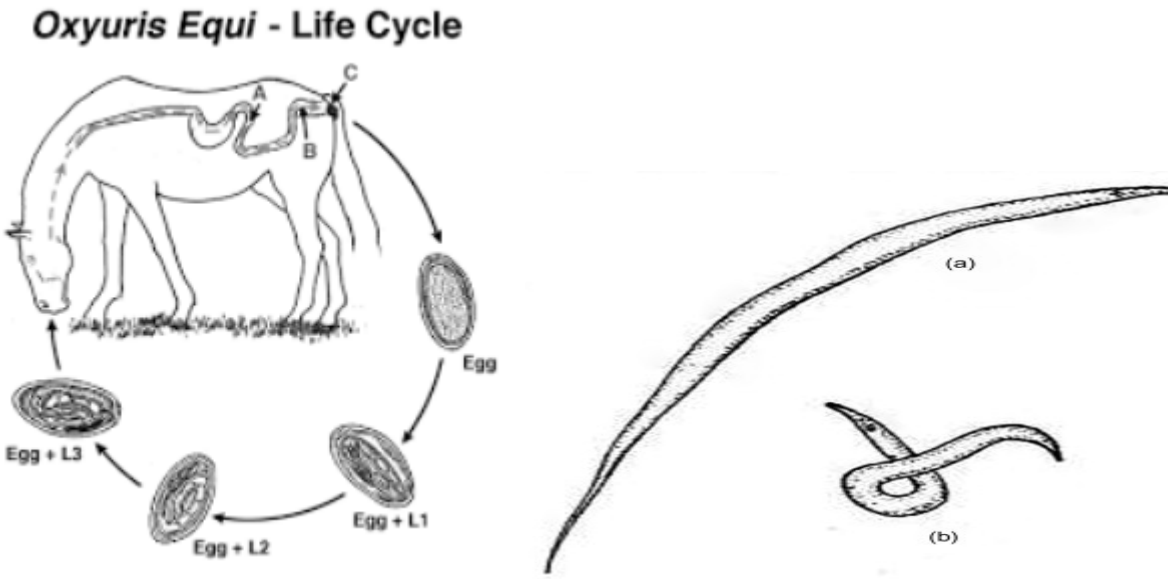


Figure 1.21. Oxyuri life cycle

Clical sign

Horses with patent *O. equi* infection may present with tailrubbing and automutilation of variable degrees as primary manifestations. **Clinical laboratory findings** are typically unremarkable.

Diagnosis of Oxyuris equi Infection in Horses

Direct microscopy of egg mass material from the perianal area reveals numerous *O equi* eggs, which are morphologically distinguishable from other equine helminth egg types. This can be done

by pressing the sticky side of a cellophane tape onto the perianal skin and then applying the tape to a glass slide for microscopy. Alternatively, a lubricated tongue depressor can be used to gently scrape the surface of the skin for subsequent microscopy. Fecal flotation and egg counts are not useful because the eggs are generally not released in the fecal matter. Female worms can sometimes be seen protruding from the anus as they deposit eggs or can be found in feces or attached to rectal sleeves after rectal examination.Tail-rubbing can be caused by multiple factors, so due consideration of differential diagnoses is warranted.

Treatment of Oxyuris equi Infection in Horses

Treatment includes thorough washing of the perianal area and treatment of skin lesions and reactions as appropriate.

Benzimidazoles are the recommended anthelmintics due to several documented cases of ivermectin resistance among *O equi* reported from different continents. Pyrantel efficacy has always been variable against *O equi*. Attempts to administer anthelmintic products by rectal lavage are not useful, given that the adult females are only transiently found in the rectum and that the predilection site of the parasite is the dorsal colon.

Prevention of Oxyuris equi Infection in Horses

Little is known about measures effective for prevention of *O equi* infection, but stable and stall hygiene could be considered, because eggs may be deposited onto various objects as a result of the tail-rubbing behavior.

Treatment and control

There are several products approved in Canada for treatment of *Oxyuris equi*. There are a few isolated reports of ***O. equi*** that are resistant to the macrocyclic lactones, but the distribution and severity of this resistance have not been fully elucidated. There are no reports from Canada.

Table 1. different anthelmintics used for the treatment of Oxyuris

Drug(s)	Product(Trade name)
Fenbendazole	Panacur, safe-guard

Ivermectin	Eqvalan, , bimectin, equell, panomec
Ivermectin with Praziquantel	Eqvalan gold, equimax
Moxidectin	Quest
Moxidectin with Praziquantel	Quest plus
Oxibendazole	Anthelcide
Piperazine	Various
Pyrantel pamoate	Strongid t, strongid p, exodus

2.2.9. Thelazia

Thelazia is a genus of nematode worms which parasitize the eyes and associated tissues of various bird and mammal hosts, including humans. They are often called "eyeworms", and infestation with Thelazia species is referred to as "thelaziasis" (occasionally spelled "thelaziosis"). Adults are usually found in the eyelids, tear glands, tear ducts, or the so-called "third eyelid" (nictitating membrane). Occasionally, they are found in the eyeball itself, either under the conjunctiva (the membrane that covers the white part of the eye) or in the vitreous cavity of the eyeball.[4] All species of Thelazia for which the life cycle has been studied are transmitted by species of Diptera (flies) which do not bite, but which feed on tears.

The genus *Thelazia* comprises several parasites, commonly named eyeworms, which infect the eyes and associated tissues of mammals, including humans. Transmission of eyeworms occurs via non-biting diptera that feed on the ocular secretions, tears and conjunctiva of animals. The disease, thelaziosis, is characterized by a range of subclinical to clinical signs, such as epiphora, conjunctivitis, keratitis, corneal opacity and ulcers. Human thelaziosis is common in poor socio-economic settings in many Asian countries. The relationship between eyeworms and

their hosts are discussed here, together with recent molecular insights that are instrumental in investigating the biology of ***Thelazia*** in their definitive and intermediate hosts.

Life cycle

Adults reside in the conjunctival sac of the definitive host where the ovoviviparous females release first-stage (L1) larvae ensheathed in a shell membrane. L1 larvae are ingested by the face fly intermediate host during feeding on tears and lacrimal secretions . In the digestive tract of the intermediate host, L1 larvae become exsheathed and invade various host tissues, including the hemocoel, fat body, testis, and egg follicles where they develop in capsules. The encapsulated larvae molt twice to become infective L3 larvae The fully developed L3 larvae break out of the capsules and migrate to the fly's mouthparts, where they remain until the fly feeds on the tears of the definitive host. The larvae invade the conjunctival sac of the definitive host upon the fly intermediate host's feeding and become adults after about a month and two additional molts . Humans may also serve as aberrant definitive hosts following exposure to an infected fly intermediate host in the same manner .

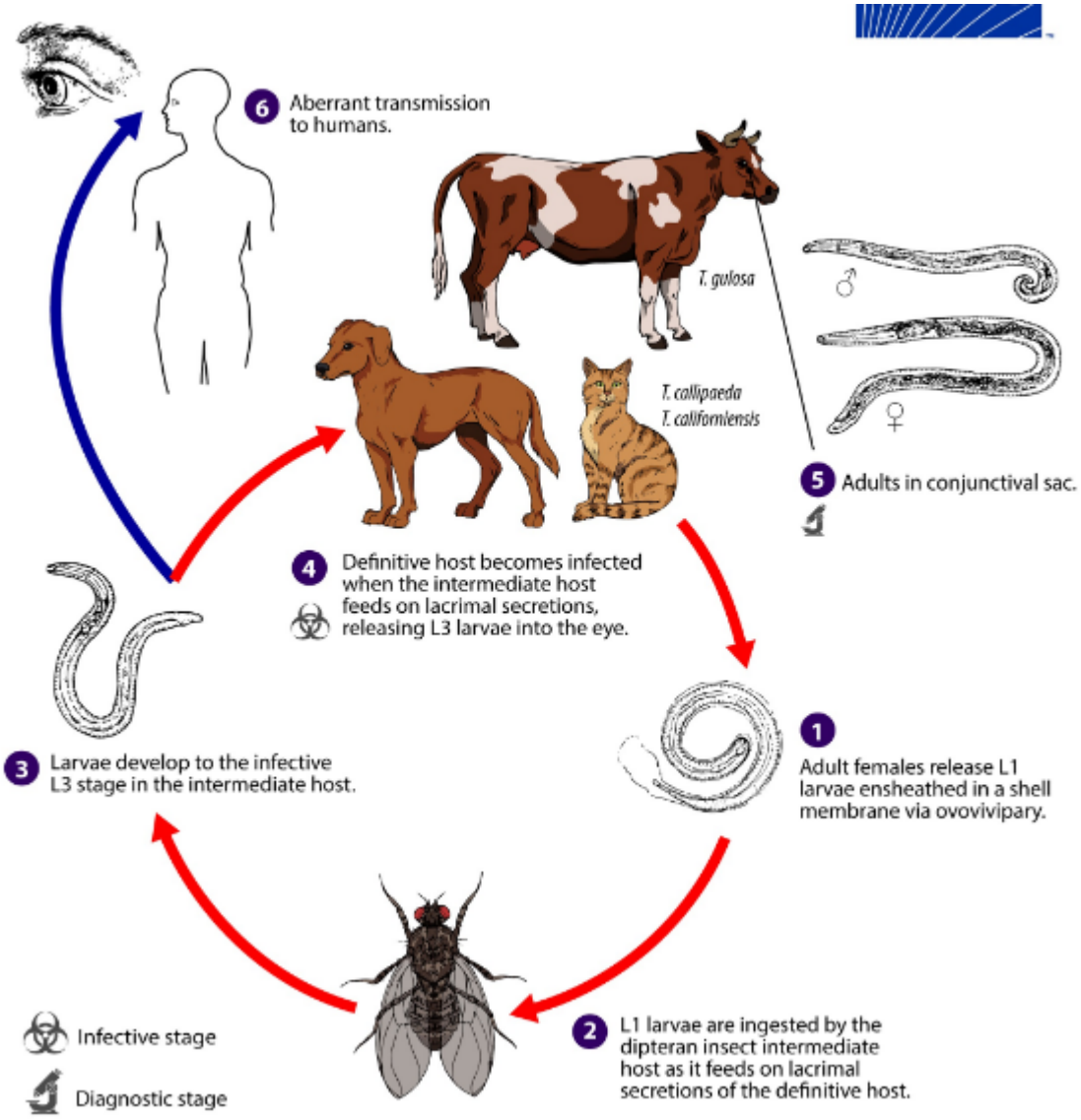
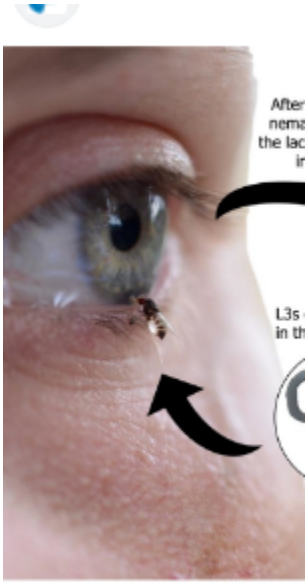

6 Aberrant transmission to humans.
T. gulosa
T. callipaeda
T. californiensis
5 Adults in conjunctival sac.
4 Definitive host becomes infected when the intermediate host feeds on lacrimal secretions, releasing L3 larvae into the eye.
3 Larvae develop to the infective L3 stage in the intermediate host.
1 Adult females release L1 larvae ensheathed in a shell membrane via ovovivipary.
2 L1 larvae are ingested by the dipteran insect intermediate host as it feeds on lacrimal secretions of the definitive host.
Infective stage
Diagnostic stage

Figure 1.21. Thelazia lifecycle

Relationship between Thelazia and their definitive hosts

The biology and epidemiology of Thelazia eyeworms are influenced by the susceptibility of their definitive hosts, and mainly by the presence, biology and seasonality of their vectors. Bovine thelaziosis is the most commonly reported animal thelaziosis, and has been broadly studied in North America and Europe probably because of the susceptibility of cows to Thelazia and that free-grazing animals are more likely to be exposed than other animals (e.g. carnivores) to fly vectors of the eyeworm.





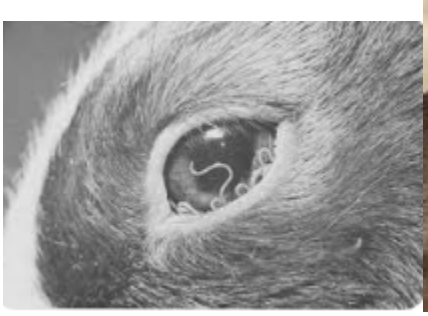




Figure 22. Thelazia infestation in different sppecies

Clinical sign

helaziases are caused by spirurid nematodes (also known as eyeworms), which cause infestation of the orbital cavities and associated tissues of several animal species. These whitish nematodes are transmitted by nonbiting dipteran muscid flies or drosophila fruit flies (intermediate hosts) while feeding on the lacrimal secretions of infected animals. Adults of Thelazia spp localize under the eyelids and the nictitating membrane, as well as in nasolacrimal ducts, conjunctival sacs, and excretory ducts of lacrimal glands, depending on the species targeted. Thelaziases may be responsible for eye disease, with signs and symptoms of varying severity (eg, lacrimation, ocular discharge, epiphora, conjunctivitis, keratitis, and sometimes corneal opacity or ulcers).

Levamisole and ivermectins (eg, doramectin) administered either subcutaneously or intramuscularly, are useful for the treatment of thelaziasis in cattle and horses. Some Thelazia spp may also infect humans.

Diagnosis of Eyeworms in Large Animals

- From asymptomatic to conjunctivitis, excessive lacrimation, keratitis, corneal clouding, and subconjunctival cysts
- Ophthalmic examination to identify the presence of nematodes
- Removal of nematodes by means of swabbing with cotton or saline lavage

Asymptomatic infections in horses and cattle appear to be typical of thelaziasis in North America. Infection may be encountered incidentally during surgery or at necropsy examination. However, *Thelazia* infections in cattle in North America may not always be innocuous.

They may produce mild conjunctivitis, excessive lacrimation, localized edema, keratitis, corneal clouding, and occasionally, subconjunctival cysts. In Europe and Asia, thelaziasis is commonly associated with severe clinical signs, including conjunctivitis, photophobia, and keratitis. Characteristically, there is chronic conjunctivitis, with lymphoid hyperplasia and a seromucoid exudate. A clinically feasible technique for reliable detection of adult eyeworms is lacking. Gross inspection of the eyes may reveal the worms and is generally recommended for *T rhodesii*, commonly found in the conjunctival sac. However, *T gulosa* and *T skrjabini* in cattle, and *T lacrymalis* in horses, tend to be more invasive and are less apt to occur. Topical anesthetics allow for tissue manipulation and are useful for detection and recovery of worms. Microscopic examination of lacrimal fluids for embryonated eggs or larvae may be attempted.

Treatment and Control of Eyeworms in Large Animals

- Environmental measures to control flies
- Administration of ivermectins and levamisole
- Good hygiene in stables
- Mechanical removal with forceps after instillation of a local anesthetic is useful to treat T rhodesii in cattle.

This also may be feasible for the more invasive T gulosa or T skrjabini in cattle or for T lacrymalis in horses. Irrigation of the eyes with 50–75 mL aqueous solution of 0.5% iodine and 0.75% potassium iodide has been recommended for T gulosa and T skrjabini. This also may be effective for T lacrymalis in horses.

Topical application of 0.03% echothiophate iodide or 0.025% isoflurophate (both organophosphates) has been successful for T lacrymalis in horses. Concurrent administration of antimicrobial-steroid ointment for the inflammation and secondary invaders is recommended. These topical agents should also be useful for T gulosa and T skrjabini in cattle. Certain systemic anthelmintics have exhibited activity against eyeworms. In cattle, administration of levamisole at 5 mg/kg, SC, and ivermectin and doramectin, both at 0.2 mg/kg, SC or IM, have shown activity against Thelazia spp. Pour-on formulations of ivermectin or doramectin, delivered to achieve a dosage of 0.5 mg/kg, are also effective.

Doramectin has been approved in the US for treatment of adult eyeworms in cattle. For T lacrymalis in horses, single doses of the commonly used anthelmintics, including ivermectin, administered via stomach tube at 0.2 mg/kg, have had limited, if any, effect on eyeworms. In contrast, the multidose regimen of fenbendazole (10 mg/kg per day for 5 days) is efficacious against T lacrymalis. Fly control measures, directed especially against the face fly, aid in the control of thelaziasis in cattle and horses. Cattle on dry, open pastures have fewer face flies than those on pastures where shade and water are present

Key PPoints

Thelaziases (ie, eyeworm infestations) are caused by spirurid nematodes, which live at the adult stage in the orbital cavities (under the lids, in nasolacrimal ducts, in conjunctival sacs) of large and small animal species and may also infest humans. Nematodes are transmitted by nonbiting dipteran muscid flies (zoophilic drosophilas for T callipaeda in small animals), while the flies feed on the lacrimal secretions of infected animals. Clinical signs may be of varying severity (eg, lacrimation, ocular discharge, epiphora, conjunctivitis, keratitis, and even corneal ulcers). Administration of levamisole and ivermectins (eg, doramectin, either subcutaneously or intramuscularly) are useful for the treatment of thelaziasis in cattle and horses

2.2.10. Filaridea

The Filarioidea are a superfamily of highly specialised parasitic nematodes. Species within this superfamily are known as filarial worms or filariae (singular filaria). Infections with parasitic filarial worms cause disease conditions generically known as filariasis.

Drugs against these worms are known as filaricides. Latin: filum = filament. This term describes a family of the phylum nematodes, the members of which use mosquitoes as vectors. These mosquitoes are true intermediate hosts, since inside their body occurs the development from larva 1 (microfilaria) to larva 3 (infectious stage for vertebrate hosts).

Importance Parafilariasis, a nematode infestation of cattle, is characterized by focal hemorrhages on the skin and damage to the subcutaneous tissues that resembles bruising. The main impact of this disease is economic. Losses are caused by damage to the hide and trimming of the carcass during meat inspection. In animals with extensive lesions, the entire carcass may be condemned. The cost of controlling the parasite and its vector, together with economic losses in the slaughterhouse, can be significant.

Etiology- Parafilariasis is caused by infestation with the nematode parasite *Parafilaria bovicola,* a member of the family Filariidae. Other species of Parafilaria can cause similar lesions in other hosts. Species Affected- P. bovicola is known to affect cattle, water buffalo (Bubalus bubalis) and yaks (Poephagus grunniens). Other species, especially those closely related to these animals, might be susceptible. Zoonotic potential - P. bovicola does not normally affect humans, but at

least one parasite has been found in an aberrant location (conjunctiva). Geographic Distribution -P. bovicola has become established in Africa, parts of Asia and some countries in Europe.

Transmission P. bovicola is transmitted by flies in the genus Musca. Species reported to be vectors include M. xanthomelas, M. lusoria and M. nevilli in Africa, M. autumnalis in Europe and M. vitripennis in Asia. P. bovicola larvae are thought to be transmitted to susceptible animals when the fly feeds on wounds or ocular secretions. The larvae migrate under the skin, developing into adults in 5 to 7 months. Approximately 2 months later, the gravid female worms break the skin to deposit their eggs, and the eggs and free (L1) larvae are shed in the discharge of blood or serohemorrhagic fluid. Flies become infected when they feed on these lesions.

The parasite develops to the infective third stage larva inside the fly in approximately 10- 20 days, depending on the climate. Mature parasites do not appear to survive in lesions from year to year; infestations are newly acquired each year.

Incubation Period- Cases usually become apparent in 7 to 10 months, when gravid female nematodes break the skin and begin to lay eggs.

Life Cycle

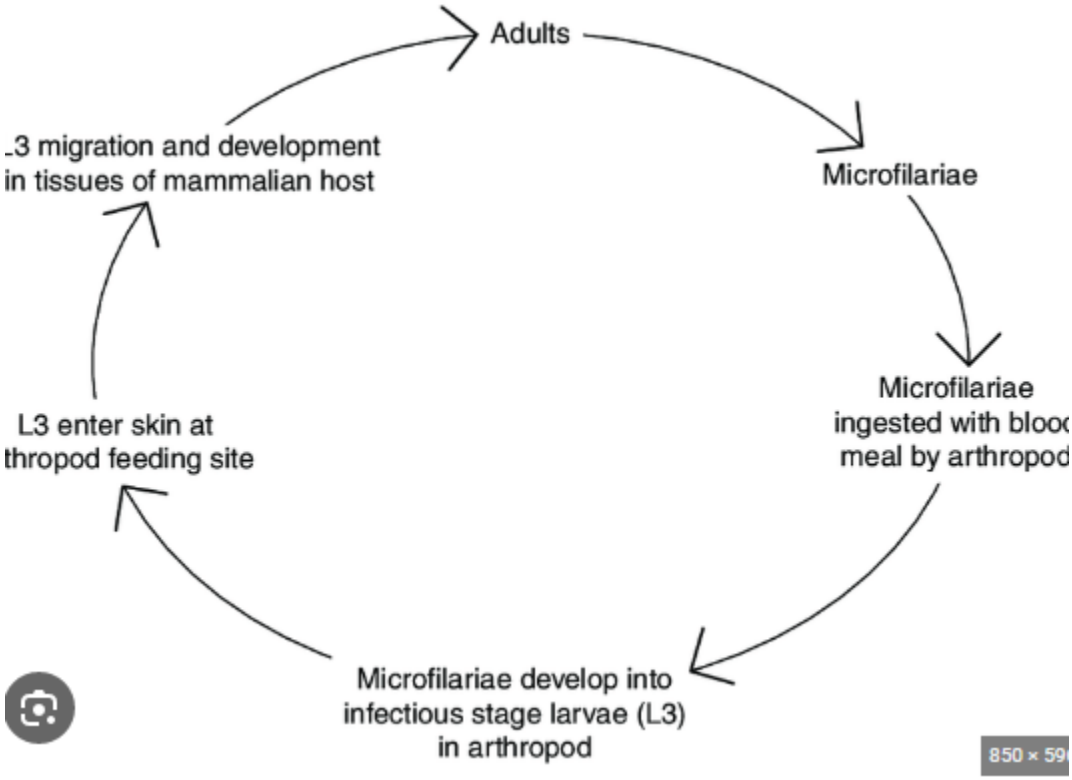


Figure1. 23. Life cycle of Filaria species

Clinical Signs- The clinical signs of parafilariasis are focal hemorrhages from small punctures (bleeding points) in the skin, and areas of edema or nodules in the subcutaneous tissues. The lesions are painful on palpation, and are most common on the skin of the back, neck, shoulders and withers. Blood or serohemorrhagic fluid may trickle from the small wound for minutes to hours. Later, another bleeding point may develop nearby. Although the lesions usually heal without complications, secondary issues can include myiasis, bacterial infections including

abscesses, and the development of cutaneous ulcers or necrosis. Discomfort from extensive lesions may result in weight loss or reduced milk yield.



Figure1. 24- parafilariabovicola

Post Mortem Lesions- In affected animals, there is evidence of bleeding points on the skin, and the subcutaneous tissues and fascia contain irregular, edematous lesions that resemble bruises. Most lesions are superficial; but extensive involvement of the muscles can be seen in more severe cases. Occasionally, there may be a few localized lesions in internal organs and tissues of the abdominal cavity, thoracic cavity or other sites. Acute lesions are usually opaque, yellowish–green, edematous or gelatinous, and occasionally hemorrhagic, with congested blood vessels. Older lesions are typically less edematous and greenish brown, yellow-brown or brown. As the lesions age, they also develop a characteristic metallic, unpleasant smell. Live or dead worms may be found in the subcutaneous lesions; however, parasites are not always present, as they tend to move after the eosinophilic reaction develops.

Diagnostic Tests- In endemic regions, the diagnosis is usually based on clinical signs, though it may be confirmed by finding eggs or microfilaria in bleeding points. However, it is important to identify P. bovicola in regions where this parasite has not been previously diagnosed. Parasite eggs and microfilaria can be detected in a sample of either fresh or dried blood/ exudate collected from a bleeding point (generally into 0.85% saline), which is then centrifuged and the sediment

examined under the microscope. Samples may contain P. bovicola eggs (approximately 45 x 30 µm) containing microfilaria, free microfilaria or both. A PCR test that can be used with exudates was recently published.

Lesions in carcasses or biopsies can be distinguished from bruises by the presence of numerous eosinophils in Giemsa-stained impression smears, and in older lesions, by the characteristic smell. Parasites may be observed in some cases; however, affected carcasses usually contain only small numbers of adult worms, which may be difficult to find. P. bovicola is a slender white worm, with adult females 50-65 mm in length, and males 30-35 mm. Incubation of tissues in warm saline can improve their recovery. Adult worms are not necessarily detected in lesions from live animals. Serology has been used occasionally, mostly in surveillance. Significant antibody titers to P. bovicola appear approximately three months after the animal has been exposed. An enzyme–linked immunosorbent assay (ELISA) was described, but the scarcity of reagents made this test impractical and it is unlikely to be in use.

Treatment- Parafilariasis has been treated with various anthelmintic agents, particularly injectable macrocyclic lactones (e.g., ivermectin, moxidectin), nitroxynil and injectable or topical levamisole. Oral or pour-on formulations of macrocyclic lactones seem to be less effective, with oral ivermectin and pour-on moxidectin having little or no effect in some reports. Delaying slaughter of the animal for 70-90 days after treatment can reduce economic losses by allowing the lesions to resolve. Anthelmintics are reported to be ineffective against immature larvae.

Control-

Disease reporting- Veterinarians who encounter or suspect parafilariasis should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention- Parafilariasis is not directly contagious between animals; development to the infectious form occurs inside the fly vector. However, it is critical to prevent P. bovicola from being transmitted to these flies in areas where it is not endemic. In endemic regions, topical

insecticides and insecticide–impregnated ear tags have been used to control the vectors. Keeping cattle indoors during the fly season may also help limit their exposure.

Public Health- There is only one report of a human infection with P. bovicola, in an elderly man in Thailand who developed conjunctivitis.

A single adult worm, which was described as a threadlike, curly object moving under the conjunctiva, was removed with the tip of a surgical blade. The patient was subsequently treated with ophthalmic antibiotics and recovered without complications*)*

2.3. Host, transmission, lifecycle, clinical sign, diagnosis, treatment and management of trematode parasites

2.3.1. Fasciola

flukes are flattened and generally leaf-shaped, dorsoventrally flattened body, bilateral symmetry, and a definite anterior end are features of platyhelminths in general and of trematodes specifically. Flukes are leaf-shaped, ranging in length from a few millimeters to 7 to 8 cm. The tegument is morphologically and physiologically complex. Flukes possess an oral sucker around the mouth and a ventral sucker or acetabulum that can be used to adhere to host tissues. A body cavity is lacking. Organs are embedded in specialized connective tissue or parenchyma. Layers of somatic muscle permeate the parenchyma and attach to the tegument.

Flukes have a well-developed alimentary canal with a muscular pharynx and esophagus. The intestine is usually a branched tube (secondary and tertiary branches may be present) consisting of a single layer of epithelial cells. The main branches may end blindly or open into an excretory vesicle. The excretory vesicle also accepts the two main lateral collecting ducts of the excretory system, which is of a protonephridial type with flame cells. A flame cell is a hollow, terminal excretory cell that contains a beating (flamelike) group of cilia. These cells, anchored in the parenchyma, direct tissue filtrate through canals into the two main collecting ducts.

Except for the blood flukes, trematodes are hermaphroditic, having both male and female reproductive organs in the same individual. The male organ consists usually of two testes with

accessory glands and ducts leading to a cirrus, or penis equivalent, that extends into the common genital atrium. The female gonad consists of a single ovary with a seminal receptacle and vitellaria, or yolk glands, that connect with the oviduct as it expands into an ootype. The tubular uterus extends from the ootype and opens into the genital atrium. Both self- and cross-fertilization occur. The components of the egg are assembled in the ootype. Eggs pass through the uterus into the genital atrium and exit ventrally through the genital pore. Fluke eggs, except for those of schistosomes, are operculated (have a lid).

Clinical Sign

During both phases of the infection, clinical features can include

- fever, malaise, abdominal pain, eosinophilia, hepatomegaly (an enlarged liver),
- abnormal liver tests. death, anemia, ill-thrift and loss of body condition

Diagnosis

- Fecal sedimentation test
- Commercial ELISA test

Treatment

Praziquantel remains the drug of choice for all trematode infections except fascioliasis, for which triclabendazole is the drug of choice.

Prevention and Control

- Anthelmintics
- Pasture management

2.3.2. Paramphistoma

flukes are flattened and generally leaf-shaped, dorsoventrally flattened body, bilateral symmetry, and a definite anterior end are features of platyhelminths in general and of trematodes specifically. Flukes are leaf-shaped, ranging in length from a few millimeters to 7 to 8 cm. The tegument is morphologically and physiologically complex. Flukes possess an oral sucker around the mouth and a ventral sucker or acetabulum that can be used to adhere to host tissues. A body cavity is

lacking. Organs are embedded in specialized connective tissue or parenchyma. Layers of somatic muscle permeate the parenchyma and attach to the tegument.

There are numerous species of paramphistomes (Paramphistomum spp, Calicophoron spp, Cotylo phoron spp) in ruminants worldwide. The adult parasites are pear-shaped, pink or red, up to 15 mm long, and attach to the lining of the rumen. Immature forms are found in the duodenum and are 1–3 mm long. Paramphistome eggs are passed in the feces, and miracidia hatch in the water and infect planorbid or bulinid snails.

Development in the snail is similar to that in the life cycle of *Fasciola hepatica*, with the snail shedding cercariae that encyst on the herbage. In the ruminant host, the young flukes excyst and remain in the small intestine for 3–6 weeks before migrating forward through the reticulum to the rumen. Eggs are produced 7–14 weeks after infection.

Adult paramphistomes do not cause overt disease, and large numbers may be encountered. Immature paramphistomes attach to the duodenal and, at times, the ileal mucosa via a large posterior sucker. The severity of disease is associated with how deep the immature paramphistomes burrow into the mucosa and submucosal layers. Severe enteritis, possibly necrosis, and hemorrhage result. Affected animals exhibit anorexia, polydipsia, unthriftiness, and severe diarrhea. Extensive mortality may occur. Clinical signs are usually confined to young cattle, whereas goats and sheep remain susceptible throughout their lives.
The large, clear, operculated eggs are readily recognized; however, in acute paramphistomiasis there may be no eggs in the feces.

Life Cycle

Their life cycle is indirect, requiring a definitive host such as ruminants, an intermediate host such as snail, and a free-living of external phases in water and plants. The sexually mature monoecious self-fertilises in the mammalian rumen, and release the eggs along with faeces. Eggs hatch in water into ciliated miracidia. The miracidia then enters the body of an intermediate host, which are snails belonging to the genera *Bulinus*, *Planorbis*, *Physa Stagnicola*

and *Pseudosuccinea*. Then the miracidia lost their cilia to become sporocysts. After a few days they develop up to 8 rediae, which are rapidly liberated. Each redia contains about 15-30 cercariae. Mature cercariae are possess by two eyespots and a long slender tail, by which they find aquatic plants or other suitable substrata, to which they get attached and encyst to become metacercariae. The mammalian hosts ingest the infective larvae. Once inside the duodenum and jejunum, their cysts are removed. They penetrate the intestinal wall by actively destroying the mucosa, and then migrate to the rumen, where the grow into adult.

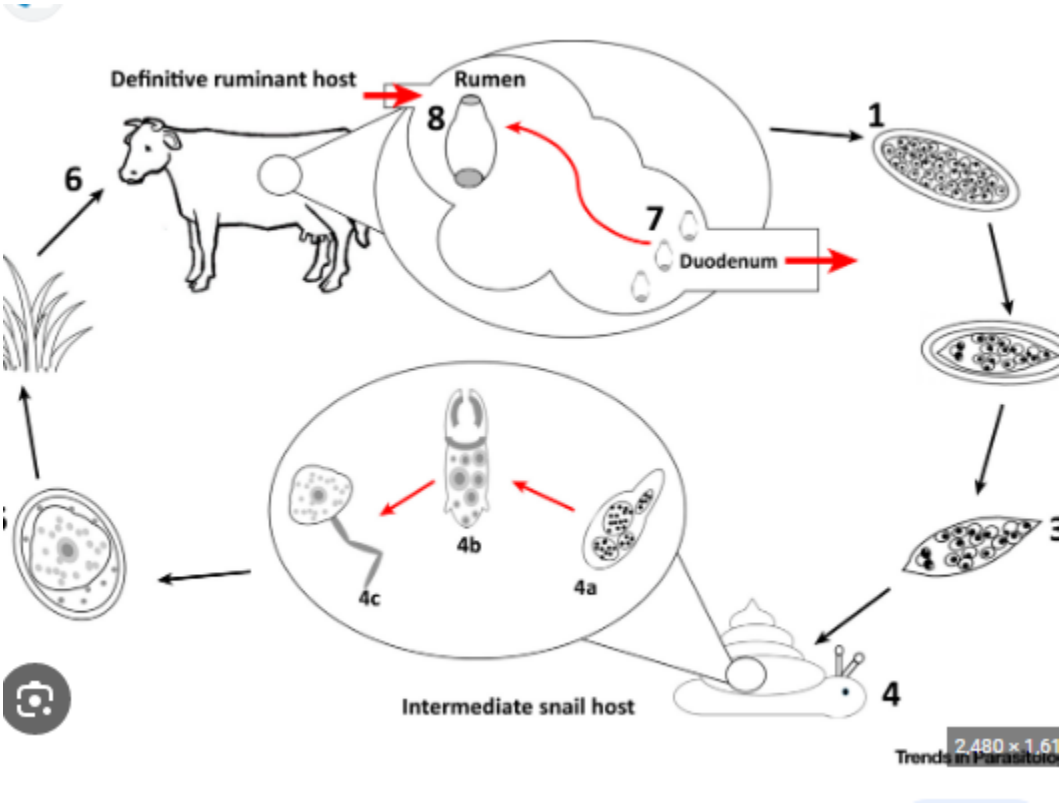


Figure 1.25. Life cycle of Paraphistomum

Clinical Sign

- profuse diarrhoea, anaemia, lethargy, and often result in death if untreated.
- decreased appetite, listlessness and weight loss.
- Fluid, foul-smelling diarrhoea, dehydration and death may result from these infections.
- reduced weight gains or milk production, or ill-thrift.

Diagnosis

- Diagnosis is commonly made at necropsy.
- Examination of the fluid feces may reveal immature flukes

Treatment

- Oxyclozanide (20 mg/kg, repeated after 72 hours) appears effective against mature flukes in shee
- Praziquantel remains the drug of choice for all trematode infections except fascioliasis.

Prevention and Control

Control measures are as for the control of fascioliasis

- Anthelmintics
- Pasture management

2.3.3. Schistoma

Schistosomiasis is an acute and chronic parasitic disease caused by blood flukes (trematode worms) of the genus Schistosoma. Estimates show that at least 251.4 million people required preventive treatment in 2021. Preventive treatment, which should be repeated over a number of years, will reduce and prevent morbidity. Schistosomiasis japonica, caused by *Schistosoma japonicum*, is an endemic, zoonotic parasitic disease. Domestic animals, particularly bovines, are thought to play an important role in transmission of the disease. Historically, China was the country mostly severely impacted by schistosomiasis japonica, but now prevalence and morbidity have been greatly reduced. Since the mid-1950s when China launched the National Schistosomiasis Control Program, the control of schistosomiasis in domestic animals has been carried out almost synchronously with that of human schistosomiasis, and this concept has been proven to be effective. Generally, the campaign of schistosomiasis japonica control in domestic animals in China went through four phases over the past six decades, namely, the large-scale epidemiological investigation phase, the case screening and small-scale chemotherapy phase, the mass chemotherapy phase, and the infection source control phase.

These distinct phases were responsive to changing disease epidemiology, socioeconomic development, and technological advances, resulting in successful attainment of disease control goals. **Diseases**- Schistosomiasis/Bilharziasis

Etiology- S. *Japonicum and S. mansoni* are considered to be true zoonotic disease.

Definitieve hosts: man, cattle, sheep, goats, and wild animals and birds.

Intermediate Hosts: different species of snails (amphibious snail)

Reservoir hosts: Cattle, buffaloe, pig, dog

Endemic to central and southwestern China, the Philippines and Indonesia.

In China and Phillipines 1.5–2.0 million people estimated to be infected

Host immune reaction to egg in liver may be fatal

Transmission: Cutaneous, ingestion, congenital

Risk groups

- Farmers working in irrigation (rice and sugar cane fields).
- Fishermen working in fish culture ponds.
- Children swimming in body of water contaminated with snails.
- women washing clothes and utensils at river banks

Transmission

How can I get schistosomiasis? Infection occurs when your skin comes in contact with contaminated freshwater in which certain types of snails that carry schistosomes are living. Freshwater becomes contaminated by Schistosoma eggs when infected people urinate or defecate in the water.

Symptoms

Without treatment, schistosomiasis can persist for years. Signs and symptoms of chronic schistosomiasis include: abdominal pain, enlarged liver, blood in the feces or stool or blood in the urine, and problems passing urine. Chronic infection can also lead to increased risk of liver fibrosis or bladder cancer.

Diagnosis

- Clinical signs, egg identification, and serology

- Shape of Eggs of *S bovis*, *S curassoni*, and *S mattheei* are spindle-shaped and oval eggs of *S japonicum*
- quantitative miracidial hatching techniques
- Molecular analysis based on the detection of *Schistosoma* DNA by PCR.

Treatment

- Praziquantel (25 mg/kg, PO, in water buffalo; 30 mg/kg, PO, in cattle) is highly effective However, for practical and economic reasons, schistosomiasis in domestic stock is rarely treated in certain areas.
- Triclabendazole showed adulticidal effect at a dosage of 20 mg/kg against female schistosome worms.
- (lithium antimony thiomalate, 15 mL, IM). Two or three injections usually produce a complete recovery.

Prevention and Control

The **most effective way to control cattle schistosomiasis** in endemic areas is to prevent contact between the animals and the parasite by fencing of dangerous waters and supplying clean water. Unfortunately, this is not always possible in parts of the world where nomadic conditions of management prevail. Other methods of control include destruction of the snail intermediate host population at transmission sites, either by chemical or biologic methods, or their removal by mechanical barriers or snail traps. Ecologic measures against the snails that aim to render their habitat unsuitable for survival, such as drainage, removal of water weeds, and increased water flow, have also proved valuable. These measures not only help reduce the transmission of schistosomiasis but also help control other parasitic trematodes such as *Fasciola gigantica* and paramphistomes, which also have water snails as intermediate hosts and frequently are found in the same localities as schistosomes.

2.4. Host, transmission, lifecycle, clinical sign, diagnosis, treatment and management of cestode parasites

2.4.1. Taenia

As members of the platyhelminths, the cestodes, or tapeworms, possess many basic structural characteristics of flukes, but also show striking differences. adult tapeworms are flattened, elongated, and consist of segments called proglottids. Tapeworms vary in length from 2 to 3 mm to 10 m, and may have three to several thousand segments. Anatomically, cestodes are divided into a scolex, or head, which bears the organs of attachment, a neck that is the region of segment proliferation, and a chain of proglottids called the strobila. The strobila elongates as new proglottids form in the neck region. The segments nearest the neck are immature (sex organs not fully developed) and those more posterior are mature.

2.4.2. Anoplocephala

Intestinal cestodes belonging to the family ***Anoplocephalida***e. These cestodes have an unarmed head or scolex with four suckers. There are three species of Anoplocefalidae: ***Anoplocephala perfoliata***, ***A. magna*** and ***A. mamillana***. The most frequently associated with clinical disease is ***Anoplocephala perfoliata***, usually located in the large intestine near the ileocecal valve. Between 8 and 25 centimeters long, *Anoplocephala perfoliata* is part of the order Cyclophillidea and is one of the three tapeworm species that can infect horses.[2] This parasite is the most common intestinal tapeworm of horses in the world and is one of the top causes of equine colic.[1] Any horses that have access to pastures and grazing are likely to have an *A. perfoliata* tapeworm in their body and experience no symptoms.

The other two species which can infect horses are *A. magna* and *Paranoplocephala mamillana*. *A. perfoliata* can be found in the intestinal tract of a horse at the ileocecal junction as well within the cecum and the ileum themselves.



Figure 1.34. Anoplocephala

Life cycle

This parasite follows the general Cyclophillidean life cycle diagrammed. These parasites prefer rich, grassy areas and are not often found in arid, desert climate areas such as Arizona. This life cycle is indirect, and requires an intermediate host, typically an oribatid mite which lives on vegetation. The eggs from the adult worms are passed in the feces into the pasture which the mite then consumes. While in the mite, the eggs develop into larvae until this mite is consumed by a horse eating grass.

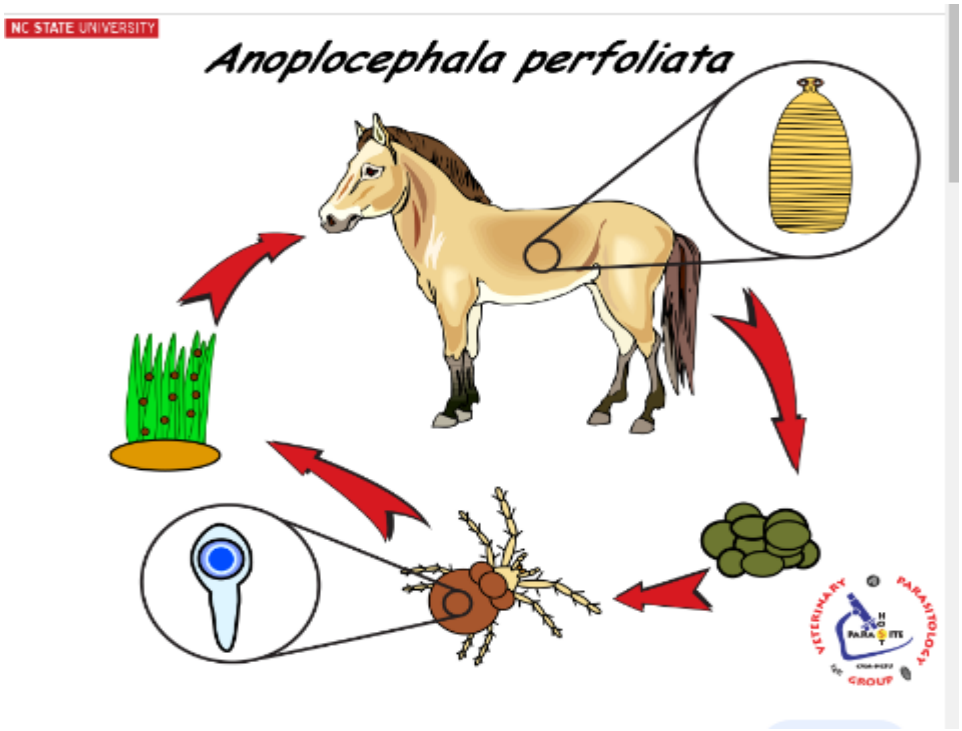


Figure 1.35. Lifec cycle of Anoplocephala

Clinical Sign

Severity is directly proportional to parasite infection intesity. Clinical signs associated with ***A. perfoliata*** are recurring diarrhea, progressive weight loss and anaemia of chronic disease. ***A. perfoliata*** is associated to spasmodic colic and ileal impactation. More serious clinical signs would be: ileocecal colic, intussusception of ileum and / or cecum and perforation and / or caecal torsion.

Diagnosis

A fecal float can diagnose the presence of an infection however they may not always be present in a sample, especially if the host has a light infection. Proglottids are released on an irregular basis

as well therefore multiple tests may need to be in order to confirm an infection of *Anoplocephala perfoliata*. Immunodiagnostic methods can be used, however they do not reliably distinguish between the two species of genus *Anoplocephala* which infect horses.

Treatment

The typical Pyrantel pamoate (pyrantel salts) dose of 6.6 mg per kilogram. At this dose, the treatment is anywhere between 80-87% effective. Doubling this dose only increases the overall efficacy to 93-95%.. Praziquantel at a dose of 1 mg per kg is 89-100% effective at eliminating an infection of *Anoplocephala perfoliata.* Ivermectin or Moxidectin in combination with Praziquantel are extremely effective against this parasite. No documentation of resistance to these treatments has been recorded yet.

Prevention

Horses cannot develop immunity to these parasites, so prevention is a key step in maintaining their health. Interval deworming is a common practice among horse owners to prevent heavy parasite infections from occurring and is a safe, effective way to prevent an infection in a horse. The typical drug for this is Ivermectin. Pyrantel salts can be administered every day that horses are grazing. The daily dose to eliminate *A. perfoliata* and *A. magna* is about 2.65 mg per kilogram.

Lastly, rather than treating every day or sporadically, horses can be dewormed right before grazing season begins and after at the end of the season in areas where horses are not out year round due to climate. This seems to be the most beneficial plan of prevention for horses as it can remove any parasites left over from the winter months and then after grazing season, eliminates the parasites they may have become infected with over the summer. For young horses that are just getting weaned off mother's milk, tapeworm treatment is highly recommended to prevent infection, as when infection occurs this early in life, it can put them at a high risk for ileocecal colic.

2.5. Host, transmission, lifecycle, clinical sign, diagnosis, treatment and management of protozoan parasites

2.5.1. Trypanosom

African Animal Trypanosomiasis Nagana, Tsetse Disease, Tsetse Fly Disease, African Animal Trypanosomosi.African animal trypanosomiasis is a parasitic disease that causes serious economic losses in livestock from anemia, loss of condition and effects on reproduction.

Losses in cattle are especially prominent. Animals other than livestock, including dogs, can also be affected. Untreated cases can be fatal, and the mortality rate is high in some outbreaks. The causative agents mainly circulate in parts of Africa that contain the biological vector, the tsetse fly; however, they can also be found in nearby regions due to mechanical transmission and the movement of infected animals. One organism, Trypanosoma vivax, has become established in South America, where it is mainly transmitted by biting flies acting as mechanical vectors. Tsetse fly control programs in Africa can help reduce the incidence of African animal trypanosomiasis, as well as other forms of trypanosomiasis that affect both humans and animals.

Etiology -Trypanosomes are protozoan parasites in the family Trypanosomatidae. African animal trypanosomiasis is caused by those organisms that are transmitted by tsetse flies but do not ordinarily affect humans. The three most important species are Trypanosoma congolense (subgenus Nannomonas), T. vivax (subgenus Duttonella) and T. brucei subsp. brucei (subgenus Trypanozoon). There are three variants of T. congolense, called the savannah, forest and kilifi (or Kenya Coast) types. African animal trypanosomiasis can also be caused by T. (Nannomonas) simiae, T. (Pycnomonas) suis, T. (Nannomonas) godfreyi and T. (Duttonella) uniforme, and possibly by additional unnamed trypanosomes.

Two related parasites, T. brucei subsp. gambiense and T. brucei rhodesiense, cause human African trypanosomiasis, which is also known as sleeping sickness. The primary distinction between this disease and African animal trypanosomiasis is that these two organisms can evade the innate resistance humans possess against other tsetse-transmitted African trypanosomes.

Clinical Signs- Cattle, sheep and goats can be infected with or without clinical signs. Most clinical cases in ruminants are chronic, but acute disease, which may be fatal within weeks, can also be seen. The first sign may be a localized swelling (chancre) at the site of the fly bite, but this usually remains unnoticed. The primary clinical signs are an intermittent fever, signs of anemia

(e.g., pale mucous membranes, lethargy), lymphadenopathy and weight loss. Hypoglycemia may be seen in the acute stage. Over time, the animals can lose condition and become progressively emaciated, often with concurrent signs such as decreases in milk yield. They can have a decreased appetite, and may develop neurological signs, dependent edema (including submandibular edema), cardiac lesions, diarrhea and keratitis/ corneal opacity.

There may also be abortions, premature births, perinatal losses and damage to the male reproductive organs (e.g., orchitis, epididymitis), with reduced semen quality. Trypanosomes can cause immunosuppression, and concurrent infections may complicate this disease. Sudden deaths have been reported in small ruminants, and deaths are common in untreated animals with chronic clinical signs. Animals that recover clinically may relapse when stressed. An acute hemorrhagic syndrome has been seen sporadically in cattle infected with some isolates of Tvivax, mostly in East Africa. This condition might be mediated by antibodies directed against red blood cells. Affected animals have enlarged lymph nodes and signs of severe anemia, and they develop widespread visceral and mucosal hemorrhages, particularly in the gastrointestinal tract.

In one outbreak, the main hemorrhagic sign was bleeding from the ears. Weight loss can be severe. This syndrome can be rapidly fatal. There is limited information about trypanosomiasis in species other than cattle, sheep and goats, but the clinical signs are reported to be similar. Anemia, weight loss and other typical signs, including neurological signs, were seen in some pigs, while other pigs have had mild illnesses or asymptomatic infections. T. simiae is reported to cause a hyperacute illness in this species, with death often occurring within 48 hours of the initial signs. Severe illnesses and deaths have been seen infrequently in horses, while most infections in donkeys seem to be mild or asymptomatic.

Donkeys did not develop clinical signs after inoculation with a South American T. vivax isolate pathogenic for goats. There are very few reports of clinical trypanosomiasis in wildlife, but illnesses have apparently been seen in captive Artiodactyla, and T. b. brucei caused systemic signs, orchitis and several deaths in experimentally infected red-fronted gazelles (Gazella rufifrons). Both acute and chronic clinical cases, as well as subclinical infections, have been described in

dogs. The signs in naturally and/or experimentally infected dogs have been similar to those in ruminants, and included neurological signs or keratitis/ corneal opacity in some cases. A few unusual cases have also been seen.

In addition to other signs, one dog presented with seizures and severe hypoglycemia that was refractory to treatment with glucose and oral sugar supplementation but responded to elimination of the trypanosomes. Two dogs had clinical signs that included acute hemorrhagic vomiting and diarrhea. Bloody diarrhea was also seen in some experimentally infected dogs; however, these animals were inoculated intraperitoneally, which could have resulted in atypical signs. Several sudden deaths were reported in naturally infected dogs. Other animals died during the course of the illness, especially during episodes of cardiovascular collapse or other events. A few dogs with clinical signs recovered spontaneously, and some dogs were treated successfully with antiparasitic drugs. Relapses have also been reported in treated dogs, and corneal opacity may be permanent.

Life cycle

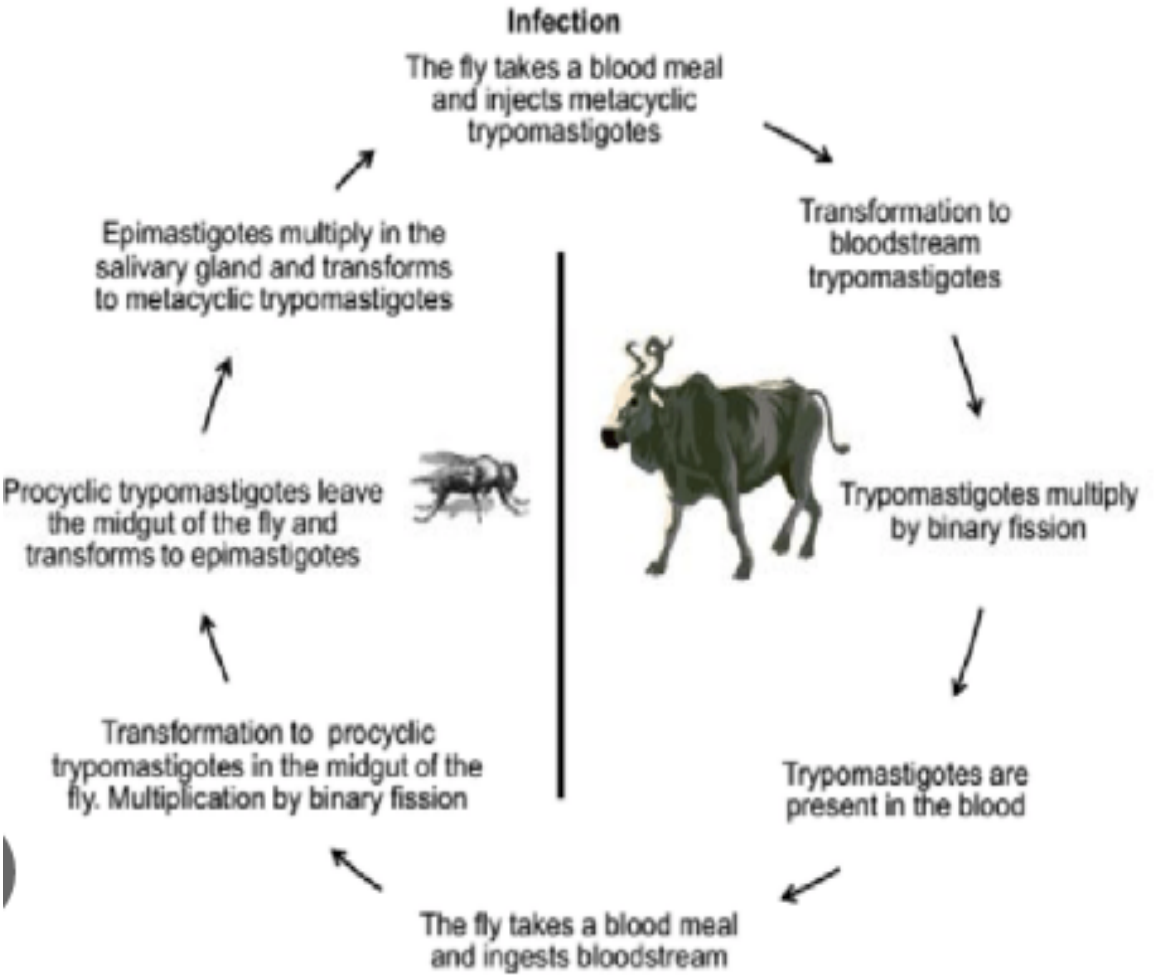

Infection
The fly takes a blood meal and injects metacyclic trypomastigotes
Transformation to bloodstream trypomastigotes
Trypomastigotes multiply by binary fission
Trypomastigotes are present in the blood
The fly takes a blood meal and ingests bloodstream
Transformation to procyclic trypomastigotes in the midgut of the fly. Multiplication by binary fission
Procyclic trypomastigotes leave the midgut of the fly and transforms to epimastigotes
Epimastigotes multiply in the salivary gland and transforms to metacyclic trypomastigotes

Figure 1.36. Trypanosoma species life cycle

Diagnostic Tests -A presumptive diagnosis can be made if trypanosomes are observed by direct microscopic examination of blood, lymph nodes (e.g., smears of needle biopsies), edema fluid or tissues collected at necropsy. Organisms are most likely to be found in the blood during the initial stages of the infection. They are less likely to be detected in chronically ill animals, and are almost never seen in healthy carriers. Samples for microscopy can include unstained wet blood films and thick or thin smears stained with Giemsa or other agents. Wet films are examined by light microscopy (condenser aperture, phase-contrast or interference contrast), using 400x power to detect the motile trypanosomes. Thick or thin blood films should also be examined at high magnification. Thick films are more likely to detect trypanosomes when their numbers are low, but parasite morphology is easier to observe in thin films.

Parasite concentration techniques, such as mini anion– exchange chromatography, hematocrit centrifugation, the quantitative buffy coat method and the dark-ground/phasecontrast buffy coat technique, can increase the probability of detecting the organisms. The trypanosome's morphology can provide a presumptive species identification, but some species cannot be distinguished by this method, and unusual morphologies have occasionally been reported. Animals can be infected concurrently with more than one species of trypanosome.

Treatment A small number of drugs have been licensed as veterinary treatments for trypanosomiasis. Diminazene aceturate and isometamidium chloride are used most often, but resistance to these agents is common in some regions. Various systems to maximize effectiveness and reduce the development of resistance, such as alternating drugs that are unlikely to induce cross-resistance, have been proposed for livestock. Drug resistance or inadequate treatment (or poor quality drugs) can result in clinical cure but persistence of the infection. Some of the drugs used in livestock, especially isometamidium chloride, have been employed in clinical cases in dogs. Agents used to treat trypanosomiasis in humans (e.g., pentamidine) were used in a few cases when other drugs were unavailable. Two dogs recovered after treatment with pentamidine,

although one later relapsed. Treatment is most effective when begun early in the course of the disease.

Control

Disease reporting -Veterinarians who encounter or suspect African animal trypanosomiasis should follow their national and/or local guidelines for disease reporting. In the U.S., this disease should be reported immediately to state or federal authorities.

Prevention Although most of the organisms that cause African animal trypanosomiasis can only become established where tsetse flies are present, T. vivax has become endemic in some areas where it is transmitted mechanically by other insects. Stamping out (e.g., quarantines, movement controls and the euthanasia of infected animals), combined with vector controls, may be able to eliminate this organism from a disease-free area if its introduction is recognized promptly

In Africa, trypanosomiasis can be controlled by reducing or eliminating tsetse flies with traps, insecticides and other means, and by treating infected animals. The specific control methods may differ depending on the epidemiological importance of various livestock species and wildlife in a region, and the predominant species of tsetse flies. A tsetse fly eradication campaign, the Pan African Tsetse and Trypanosomosis Eradication Campaign (PATTEC), is being conducted in Africa.

2.5.2. Trichomonas

Trichomoniasis is a venereal disease of cattle characterized primarily by early fetal death and infertility, resulting in extended calving intervals. Distribution is likely worldwide. Diagnosis is confirmed by isolation of the organism. Imidazoles have been used to treat infected bulls, but none is both safe and effective. Control is by culling infected bulls.

Etiology and Epidemiology of Trichomoniasis in Cattle

The causative protozoan of trichomoniasis, *Tritrichomonas foetus*, is pyriform and ordinarily 10–15 × 5–10 mcm, but there is considerable pleomorphism. It may become spherical when cultured in artificial media. At its anterior end, there are three flagella approximately the same

length as the body of the parasite. An undulating membrane extends the length of the body and is bordered by a marginal filament that continues beyond the membrane as a posterior flagellum. Although *T foetus* can survive the process used for freezing semen, it is killed by drying or high temperatures. *T foetus* is **found in the genital tracts** of cattle. When cows are bred naturally by an infected bull, 30%–90% become infected, suggesting that strain differences exist. Variation in breed susceptibility to trichomoniasis may also exist.

Bulls of all ages can remain infected indefinitely, but this is less likely in younger males. By contrast, most cows are free of infection within 3 months after breeding. However, immunity is not longlasting and reinfection does occur. Transmission can also occur when the semen from infected bulls is used for artificial insemination.

Clinical Findings of Trichomoniasis in Cattle

The most common sign of trichomoniasis is **infertility caused by embryonic death**. This results in repeat breeding, and attending stock persons often note cows in heat when they should be pregnant. This, along with poor pregnancy test results (eg, too many "nonpregnant normal" and late-bred cows) is usually the presenting complaint. In addition to a reduced number of cows estimated to calve during the regular calving season, an increased number of cows with a "nonpregnant abnormal" reproductive tract diagnosis is seen. These include cows with pyometra, endometritis, or a mummified fetus. Fetal death and abortions can also occur but are not as common as losses earlier in gestation. *T foetus* has been found in vaginal cultures taken as late as 8 months of gestation and, apparently, live calves can be born to infected dams. Pyometra occasionally develops after breeding.

Diagnosis of Trichomoniasis in Cattle

- Diagnosis is confirmed by culture of *T foetus*

History and clinical signs are useful but are essentially the same as those of bovine genital campylobacteriosis. Confirmation depends on isolation of *T foetus*, which may be difficult to differentiate from other trichomonads resident in the digestive tract. Diagnostic efforts are directed at bulls, because they are the most likely carriers. Suction is applied to a pipette while it is used to vigorously scrape the epithelium in the preputial fornix. Alternatively, douching with

saline or lactated Ringer's solution (without preservatives) can be used. Aspirates or douches, concentrated by centrifugation, are examined using darkfield contrast microscopy.

This material is also transferred immediately to the surface of a liquid culture medium such as Diamond medium. Better success culturing the organism has been reported when using commercially available media-filled pouches. In addition, incubating the media beyond the standard 48 hours may also enhance the accuracy of the diagnosis. Sampling every 48 hours for 10 days from the bottom of the tube and examining at 100–400× may reveal the rolling jerky movements of *T foetus*. Studies have examined the possibility of using PCR assays to identify *T foetus* directly from the preputial samples without an intervening culture. These tests are being used by some laboratories to reduce the time required for a definitive diagnosis of trichomoniasis. Some diagnostic laboratories are suggesting that the scraping be placed in phosphate buffered saline before transport to the diagnostic laboratory. A sample prepared in this way can be effectively examined for both trichomoniasis and campylobacteriosis by using a specific PCR application. Studies suggest that 90%–95% of infected bulls will be positive on culture, and that three successive cultures at weekly intervals will detect ~99.5% of infected bulls.

A vaginal discharge (after treatment of pyometra) or vaginal mucus (obtained toward the end of a luteal phase) may also be of diagnostic value. The number of times the bull battery should be sampled and cultured to ensure the bulls are negative depends on the prevalence of fetal wastage in the cow herd. With more open and late cows, the frequency of testing should increase to improve the probability that bulls are negative for the protozoan.

Treatment and Control of Trichomoniasis in Cattle

- Imidazoles have been used to treat bulls, but none are both safe and effective
- Control is by culling infected bulls

Various imidazoles have been used to treat bulls with trichomoniasis, but none is both safe and effective. Ipronidazole is probably most effective but, due to its low pH, frequently causes sterile abscesses at injection sites. In addition, bulls are probably susceptible to reinfection after

successful treatment. Resistance to ipronidazole may also be a concern. The biggest problem, however, is that the success of treatment is measured by repeated sampling, which may mean the individual bull can never be definitively called negative. Therefore, an unqualified recommendation for the bull's use cannot be given.

Control consists of eliminating the infection from the bull battery by culling all bulls and replacing them with virgin bulls or by testing and culling positive bulls. Repeated testing in older bulls may be unsatisfactory, and it may be prudent to cull them all. Reinfection is prevented by exposing only the uninfected (clean) bulls to uninfected (clean) cows.

Clean cows are assumed to be those with calves at foot (even though some infected cows may produce a live calf) and virgin heifers. In situations in which several herds are commingled on the same range, caution must be exercised to ensure that cows and heifers are not exposed to potentially infected bulls at the home ranch before they are turned out on the common grazing pasture. *T foetus* can be safely eliminated from semen with dimetridazole. Vaccines developed some time ago for use in cows and evaluated in the field were not highly effective, especially in the absence of other control measures. However, the efficacy of whole-cell *T foetus* vaccines has recently been critically reviewed. Although there is some evidence to suggest that timely vaccination will improve reproductive performance in heifers, there is a distinct lack of evidence that vaccination will reduce "bull-associated outcomes."

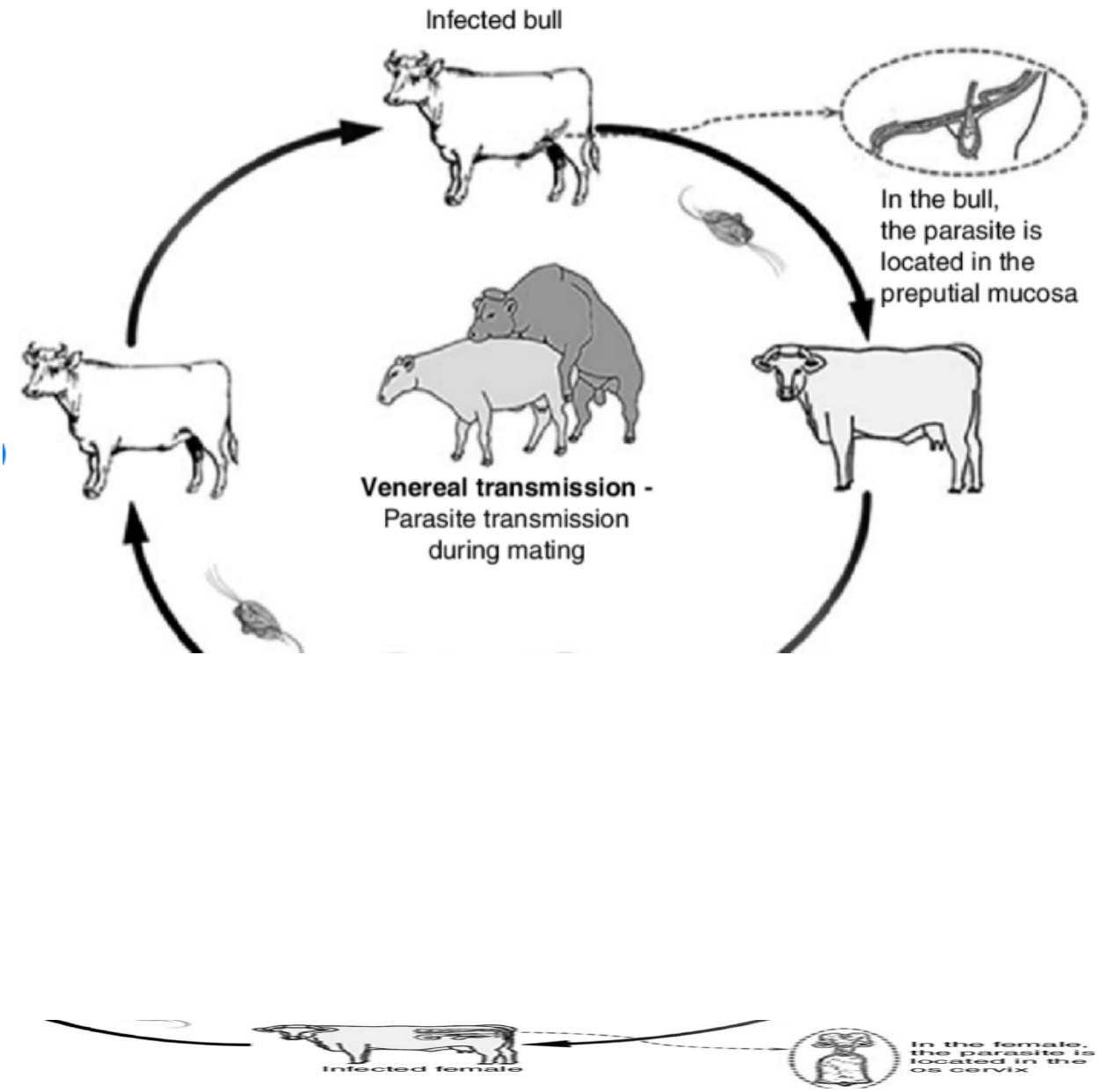


Figure 1.37. Life cycle of bovine Tritrichomonas foetus

2.5.3. Coccidia- Emeriidae

Coccidia are single-celled obligate intracellular protozoan parasites in the class Conoidasida within the phylum Apicomplexa. The main clinical sign of coccidiosis is diarrhea. Oocysts can be identified in feces by use of salt or sugar flotation methods, direct intestinal smears, or a McMaster counting chamber. Antiprotozoal treatment can shorten the length of illness, decrease

discharge of oocysts, alleviate clinical signs, and reduce likelihood of secondary infections and death. Coccidia are obligate intracellular protozoan parasites in the class Conoidasida within the phylum Apicomplexa. The genera *Eimeria* (sporulated oocyst contains four sporocysts) and *Isospora* (sporulated oocyst contains two sporocysts) contain many species that infect a wide variety of birds, mammals and reptiles, but almost all are species host-specific.

Infections occur throughout the world. Other genera cause besnoitiosis, cryptosporidiosis, sarcoc ystosis, and toxoplasmosis.

Infection is oral from ingestion of infective sporulated oocysts from a contaminated environment. Most animals are exposed to infection early in life. Clinical signs of infection usually only occur in the young. The parasites mainly invade the intestinal epithelial lining and underlying connective tissue of the mucosa, sometimes leading to catarrhal inflammation and hemorrhage. In rabbits, the liver is also affected. During their lives most animals become infected but show no illness, and the disease is self-limiting. However, when clinical signs occur, they range from diarrhea and decreased growth in mild cases to dysentery, dehydration, and tenesmus in severe cases, usually occurring with widespread hind gut involvement. Mortality rate is generally low.

Chronic infections can occur, leading to pasty feces, staring coats, poor growth, and variable sizes within an age group. In production animals, infection can have serious economic consequences. In cats, dogs, and horses, infection is mainly from *Isospora* spp, and clinical signs are less common and usually less severe than with *Eimeria* spp. Various treatments are available but those approved vary in different countries. Control depends on decreasing environmental contamination and the stresses to which susceptible animals are subjected. Coccidiostats can also be used to decrease oocyst production and disease.

Etiology and Pathogenesis of Coccidiosis in Animals

All species of *Eimeria* complete their life cycle within a single host (monoxeny), as do most *Isospora* spp. However, some *Isospora* spp can use optional transfer or paratenic hosts. Development does not occur in these hosts, but allows the parasite to be dispersed to new areas.

A new genus name, *Cystoisospora*, has been proposed for those species of *Isospora* with paratenic properties. Otherwise, coccidia are host-species specific and there is no cross-immunity between different coccidial species in the same host. Intercurrent infections are common and may be exacerbated because coccidiosis can be immunosuppressive.

The life cycle is complex but is all completed within the one host producing oocysts. There are three stages in the life cycle: sporogony, merogony and gametogony.

Sporogony/sporulation: Oocyst passed in the host's feces are not immediately infective. They have a thick, resistant wall but require moisture, oxygen, and warmth to sporulate. In sporulation, the oocyst nucleus divides a number of times, dependent on the species, to produce sporocysts. The divided nucleus is surrounded by protoplasm forming a conical body, a sporoblast, which in turn secretes a wall around itself and divides into two sporozoites to become a sporocyst. The development of this infective sporulated oocyst can occur in hours but it may take days or weeks. These infective oocysts are resistant in the environment and can survive long periods, including over winter.

Merogony/schizogony (asexual stage): Once the infective sporulated oocyst is ingested, it releases the sporozoites in the small intestine and, after activation by the bile or trypsin, they invade the intestinal mucosa. Here, they become rounded trophozoites, which then divide into nucleated elongated merozoites that together form a meront. Once developed, the meront ruptures, as does the containing intestinal cell wall, thereby releasing the merozoites. Depending on the coccidial species, the merozoites may repeat the process of meront production in one or more cycles before they ultimately progress to the gametogony stage.

Gametogony (sexual stage): The last wave of merozoites enters other intestinal cells where they develop into large cells (macrogametocytes), which are female, or a large number of small flagellated male cells (microgametocytes). The male gametocytes are released by the rupture of the containing intestinal cell. One microgametocyte penetrates a macrogametocyte, resulting in their nuclei fusing. The resultant cell develops a thick wall and becomes an oocyst, which is ultimately released into the feces. With this life cycle, depending on the coccidial species, one

ingested oocyst after two asexual generations could produce 24×10^6 merozoites and many million oocysts.

Clinical Findings of Coccidiosis in Animals

Most infected animals are young when exposed to oocysts in the environment and show no observable clinical signs. Animals with subclinical infection may have decreased growth rates and decreased feed conversion, which can be important in production animals. For example, the initial decrease in weight gain in cattle may remain for at least one grazing season. The clinical signs of coccidiosis are due to destruction of the intestinal epithelium and, frequently, the underlying connective tissue of the mucosa. This may be accompanied by hemorrhage into the lumen of the intestine, catarrhal inflammation, and diarrhea. The main clinical sign is diarrhea. Dehydration may result. Mainly when there is hind gut involvement, hemorrhage and tenesmus may occur. Typically, mortality rate is low. Serum protein and electrolyte concentrations (typically hyponatremia) may be altered. Anemia with decreased blood hemoglobin or PCV occurs uncommonly and then only in severely affected animals, which may die.

Chronic infection occurs in some animals, causing economic loss due to decreased feed intake, feed conversion efficiency, and weight gain. Some of these animals have poor-quality coats and are unthrifty. Infection can lead to nutritional deficiencies and digestive problems. In some cases, it can result in exacerbation of any intercurrent infections, causing animals to have more severe clinical signs and lengthening their duration or recovery period, either due to immunosuppression or for other reasons.

Diagnosis of Coccidiosis in Animals

- Presence of fecal oocysts
- History (age of affected animals, environment, and management)
- Clinical signs (diarrhea in several young animals is indicative)

Isospora amphiboluri oocysts

Fecal samples should be taken from several animals in a group with and without (recorded) clinical signs of coccidiosis. They usually indicate oocyst presence. Oocysts can be identified in feces by use of salt or sugar flotation methods, direct intestinal smears, or a McMaster counting chamber. However, the severity of diarrhea may not correlate with the peak period of oocyst shedding.

Finding notable numbers of oocysts of pathogenic species in the feces is diagnostic (>100,000 oocysts/g of feces in severe outbreaks). Usually a count >5,000 oocysts/g with clinical signs is suggestive of infection with coccidia.

However, in some host species, oocyst counts >2,500 oocysts/g may be important depending on number of animals affected with and without clinical signs and may indicate potential problems. The number of oocysts in feces is influenced by the genetically determined reproductive potential of the species, the number of infective oocysts ingested, stage of the infection, age and immune status of the animal, prior exposure, consistency of the fecal sample (free water content), and method of examination. Therefore, the results of fecal examinations must be related to clinical signs and, when deaths occur, the intestinal lesions (gross and microscopic).

Speciation should be undertaken even if treatment is required before results. Although there is some overlap, many of the species in a particular host can be identified by oocyst size, shape, and wall thickness. Artificial sporulation of the oocyst facilitated with potassium dichromate may be required to confirm identification of some species. When a diagnosis of coccidiosis is established, the possibility of intercurrent disease should be considered. Coccidial infection may need to be differentiated from other causes of diarrhea that occur at the same age, which include those due to bacteria, viruses, other protozoal parasites, helminths, nutritional deficiencies, and feeding and management faults.

Treatment of Coccidiosis in Animals

- Stress reduction
- Anticoccidial treatment

During outbreaks, ill animals should be separated and provided with good-quality feeds and a nonstressful environment. This also reduces reinfection pressures on others within the group that are less affected. If possible, in-contact animals should be moved to a less contaminated and stress-free environment with a lowered stocking density. Vacated areas should be thoroughly cleaned and suitably disinfected. The new accommodation should be regularly cleaned, generously bedded, and disinfected as necessary.

Those animals outside should also be moved to new areas and the stocking density decreased. All feed and water supplies should be sited in well-drained areas and fecal contamination avoided. Any medications used should be administered individually to the affected animals to ensure that they receive the correct full dose. It is not sufficient to place the anticoccidial in the feed or water as consumption of both is decreased in the ill animals. Provision of anticoccidial treatment to the rest of the group can usually be justified to prevent further illness and decrease oocyst environmental contamination.

Prevention

Preventive management is based on limiting intake of sporulated oocysts by young animals so that immunity is induced but not clinical signs. Good feeding practices and good management, including sanitation, contribute to this goal. With some *Eimeria* species, it may at times be necessary to treat pregnant dams to decrease oocyst shedding. Neonates should receive adequate amounts of good-quality colostrum. Young, susceptible animals should be kept in clean, dry quarters. Feeding and watering devices should be clean and must be protected from fecal contamination; this usually means using troughs raised above the ground and positioning them to minimize fecal contamination. Stresses (eg, weaning, grouping, sudden changes in feed, and shipping) should be minimized. Any management alterations should not take place in extreme weather conditions or when weather is very variable.

Oocysts are resistant to most disinfectants. Some ammonia-based disinfectants kill oocysts but can only be used in areas vacated by animals. Infected areas should be cleaned, disinfected, and then steam cleaned. Animals should only occupy the areas once dry and after a fallow period. Ideally, coccidiostats should not be administered prophylactically. However, when animals under various

management regimens can be predictably expected to develop coccidiosis, then medication may be necessary to prevent clinical disease. This does not mean that management procedures to improve matters can be ignored. In virtually all cases of clinical coccidiosis, *Eimeria* spp are implicated. *Isospora* spp tend to mainly involve the small intestine, and so are less likely to produce illness except in piglets and very occasionally in kittens and puppies, mainly in catteries and kennels. Sulfonamides in the drinking water or in the feed can be helpful in preventing clinical disease in cattle, goats, sheep, and pigs but they are not licensed for such a purpose in many countries.

Key points

Coccidiosis is a gastrointestinal illness caused by a protozoan.

Clinical signs include diarrhea (with or without mucus), hematochezia, lethargy, weight loss, vomiting, signs of abdominal pain, pallor, and anorexia.

Oocysts can be identified in feces by use of salt or sugar flotation methods, direct intestinal smears, or a McMaster counting chamber.

Antiprotozoal treatment can shorten the length of illness, decrease discharge of oocysts, alleviate clinical signs, and reduce likelihood of secondary infections and death.

Lifecycle

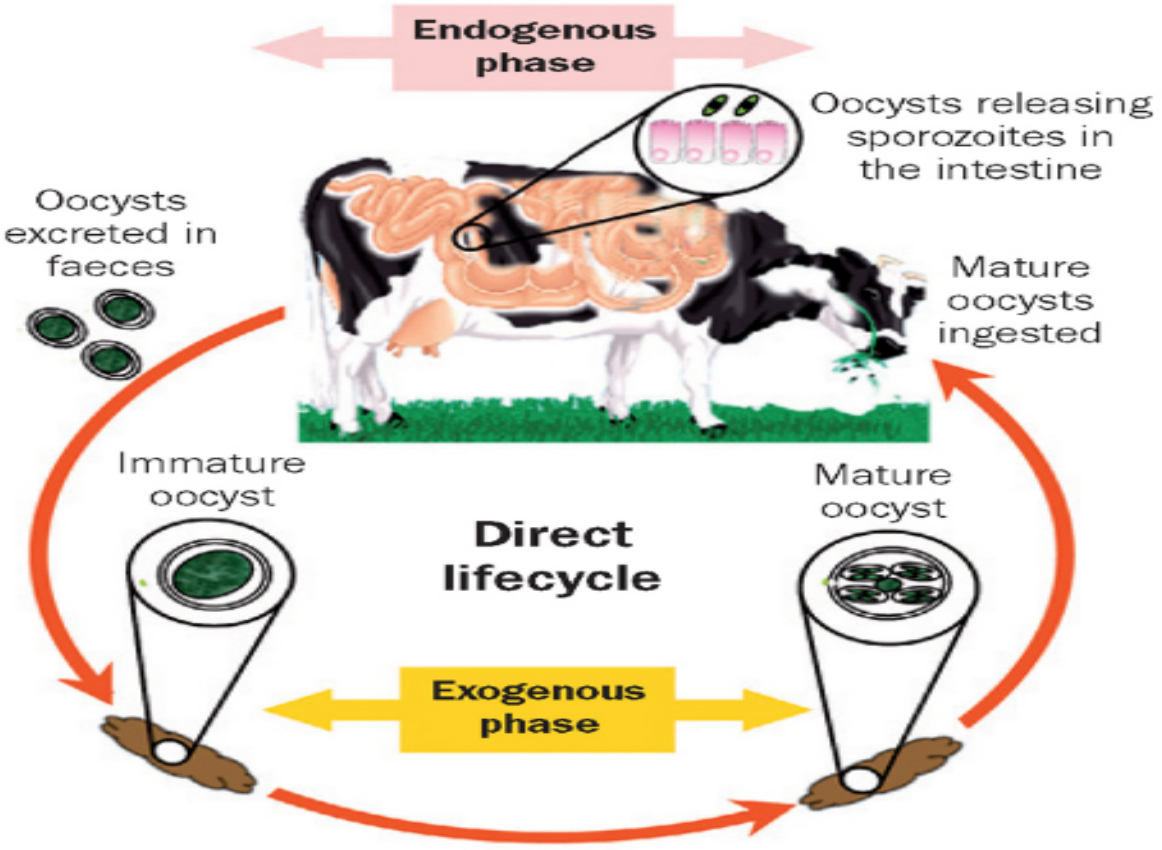


Fig ure1.38-direct lifecycle

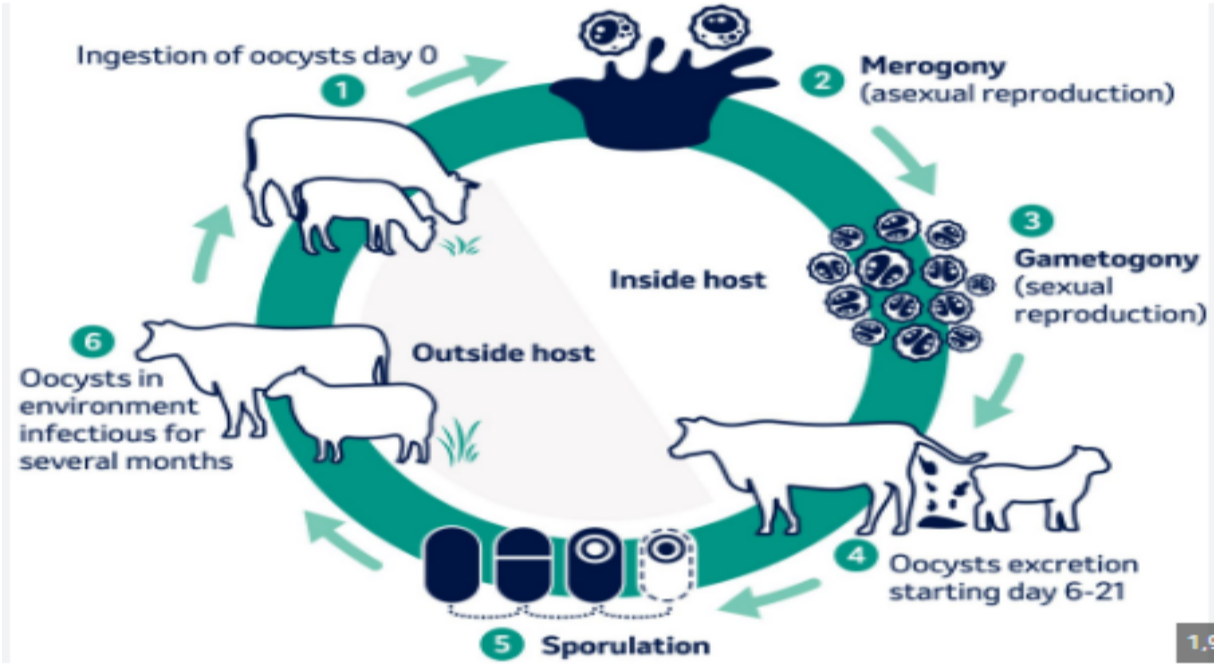


Figure 1.39-Coccidiosis in ruminants

2.5.4. Babesia

Babesia spp blood parasites are transmitted by ixodid ticks. Babesiosis is typically characterized by fever and intravascular hemolysis leading to progressive anemia, hemoglobinuria, and jaundice, and may result in death. Diagnosis is principally by light microscopic evaluation of blood smears. The most common treatments are imidocarb dipropionate and diminazene aceturate. Control is via chemotherapy, vector control, and vaccination in some countries. Babesiosis is the clinical disease associated with infection with protozoa of the genus *Babesia*, which are blood parasites transmitted by ixodid ticks. Among other wild and domestic mammals, cattle, horses, sheep and

goats, swine, and cats and dogs are susceptible. In addition, babesiosis is a zoonotic disease affecting humans. The disease, which may be fatal, is typically characterized by fever and intravascular hemolysis leading to progressive anemia, hemoglobinuria, and jaundice. Diagnosis is principally by light microscopic evaluation of blood smears. The most common treatments are imidocarb dipropionate and diminazene aceturate. Control is via chemotherapy, vector control, and vaccination in some countries.

Etiology and Pathogenesis of Babesiosis in Animals

Babesia are intraerythrocytic protozoan parasites from the phylum Apicomplexa, order Piroplasmida. More than 100 species of *Babesia* exist, affecting domestic animals (cattle, horses, sheep, goats, pigs, dogs, and cats), wildlife, and, occasionally, humans. Traditionally, *Babesia* spp were classified based on morphology and vector and host specificity; however, recent molecular characterizations suggest greater complexity. *Babesia bovis* is a much more virulent organism than *B bigemina*. With most strains of *B bigemina*, the pathogenic effects relate more directly to erythrocyte destruction. With virulent strains of *B bovis*, a hypotensive shock syndrome, combined with generalized nonspecific inflammation, coagulation disturbances, and erythrocytic stasis in capillaries, contribute to the pathogenesis. The following are indicative of *Babesia* spp affecting domestic animals; however, the list is far from complete.

Cattle

Babesia divergens and *B major* are two temperate-zone species with features comparable to those of *B bovis* and *B bigemina*, respectively. *Babesia divergens* is a small, pathogenic *Babesia* of considerable importance in the British Isles and northwest Europe, whereas *B major* is a large *Babesia* of lower pathogenicity. *Babesia divergens* is transmitted by *Ixodes ricinus*, and *B major* by *Haemaphysalis punctata*.

Horses

Equine piroplasmosis is caused by *Theileria* (formerly *Babesia*) *equi* or *B caballi*. *Theileria equi* is a small parasite and is more pathogenic than *B caballi*. *Theileria equi* was reclassified as a *Theileria* in 1998. Equine babesiosis is found in Africa, Europe, Asia, South America, Central

America, and the southern US. It is transmitted by ticks of the genera *Rhipicephalus*, *Dermacentor*, and *Hyalomma*. Intrauterine infection, particularly with *T equi*, is also relatively common.

Sheep and Goats

Although small ruminants can be infected by several species of *Babesia*, the two most important species are *B ovis* and *B motasi*, transmitted by *Rhipicephalus bursa* and *Haemaphysalis* spp, respectively. Infection is of importance in the Middle East, southern Europe, and some African and Asian countries.

Transmission and Epidemiology of Babesiosis in Animals

The major economic impact of babesiosis is to the cattle industry in tropical and subtropical geographic areas, and is attributed to *Babesia bovis* and *Babesia bigemina*; however, the diseases caused by different *Babesia* have many common features. Babesiosis is a tickborne disease and its geographic distribution is therefore determined by the distribution of tick vectors. The main vectors of *B bigemina* and *B bovis* are 1-host *Rhipicephalus (Boophilus)* spp ticks which are widespread in tropical and subtropical areas. Transmission occurs transovarially. Although these *Babesia* spp can be readily transmitted experimentally by blood inoculation, mechanical transmission by insects or during surgical procedures has no practical importance. Intrauterine infection has also been reported but is rare.

In *Rhipicephalus* spp ticks, the blood stages of the parasite are ingested during engorgement and undergo sexual and asexual multiplication in the replete female, infecting eggs and subsequent parasitic stages. Transmission to the host occurs when larvae (in the case of *B bovis*) or nymphs and adults (in the case of *B bigemina*) feed. The percentage of larvae infected can vary from 0%–50% or higher, depending mainly on the level of parasitemia of the host at the time the female ticks engorge. Under field conditions, the rate of tick transmission is generally higher for *B bigemina* than for *B bovis*. NAHRS) Reportable Diseases, Infections, and Infestations List.

Clinical Findings of Babesiosis in Animals

Acute babesiosis generally runs a course of ~1 week or less. The first clinical signs are lethargy, weakness, depression, and fever (frequently ≥106°F [41°C]), which persist throughout, and these are accompanied later by inappetence, anemia, jaundice, and weight loss; hemoglobinemia and hemoglobinuria occur in the final stages. Involvement of the CNS due to adhesion of parasitized erythrocytes in brain capillaries can occur with *B bovis* infections. Late-term pregnant cows may abort, and temporary infertility due to transient fever may occur in bulls. Many animals recover; however, some may die if not treated. Animals that recover from the acute disease remain infected for a number of years with *B bovis* or for a few months with *B bigemina*. No clinical signs are apparent during this carrier state.

Diagnosis of Babesiosis in Animals

- Physical examination and history
- Light microscopic evaluation of blood smears
- PCR assay
- Serologic testing

Clinical findings and history may provide a presumptive diagnosis of babesiosis; however, other conditions also cause similar clinical signs. Examination of Giemsa-stained blood or organ smears by light microscopy is essential to confirm the diagnosis. Light microscopy is rapid and inexpensive but does require some expertise. From the live animal, blood smears should be prepared from capillaries, for example from the ear or tail tip, to improve the sensitivity of detection of *B bovis*, because *B bovis*-infected erythrocytes adhere to capillary endothelium. Smears of brain, muscle, kidney, spleen, and from a blood vessel in an extremity should be taken at postmortem examination. Note that detection of *Babesia* spp using light microscopy in the carrier state has poor sensitivity.

Microscopically, the species of *Babesia* involved can be determined morphologically; however, expertise is required, especially in *B bovis* infections in which few organisms may be present. *Babesia bovis* is generally small, with the parasites in paired form at an obtuse angle to each other and measuring ~1–1.5 × 0.5–1 mcm. *B bigemina* is larger (3–3.5 × 1–1.5 mcm), with

paired parasites lying almost parallel or at an acute angle to each other. Single forms of both parasites are also commonly seen.

Treatment, Control, and Prevention of Babesiosis in Animals

- Babesiacides
- Supportive treatment
- Tick control
- Use of resistant breeds
- Vaccination

Babesiacides

A variety of babesiacidal drugs have been used to treat bovine babesiosis; however, only diminazene aceturate and imidocarb dipropionate are still in common use. These drugs are not available in all endemic countries, or their use may be restricted. Manufacturers' recommendations for use should be followed. For treating cattle, diminazene is administered at 3.5 mg/kg, IM, once. For treatment, imidocarb is administered at 1.2 mg/kg, SC, once. At a dosage of 3 mg/kg, imidocarb provides protection from babesiosis for approximately 4 weeks and may also eliminate *B bovis* and *B bigemina* from carrier animals.

Supportive Treatment

Supportive treatment is advisable, particularly in valuable animals, and may include the use of anti-inflammatory drugs, corticosteroids, and fluid therapy. Blood transfusions may be lifesaving in very anemic animals.

Tick Control

Tick control, via acaricides or management practices, can be useful in reducing tick burdens, which can lower transmission rates. However, this can lead to naive cattle populations, with consequent risk of outbreaks of disease should tick populations increase. Chemical tick control cannot be relied upon to prevent transmission of *Babesia*, and outbreaks often occur after introduction of susceptible cattle to endemic areas despite acaricide use. Acaricide resistance is also an increasing problem. However, acaricidal tick control before moving animals from tick-infested areas is useful to prevent the introduction of ticks and babesiosis to tick-free areas.

Eradication of the tick vector is rarely feasible on individual premises but may work on a regional level in well-coordinated programs.

Vaccination

Vaccination using live attenuated strains of the *Babesia* parasites has been used successfully in countries such as Argentina, Australia, Brazil, Israel, South Africa, and Uruguay. The vaccine is available in either a chilled or frozen form. One vaccination produces adequate immunity for the typical productive lifespan of animals raised in commercial settings. Commercial vaccines based on recombinant antigens are not yet available.

Life cycle

Life cycle of bovine Babesia parasites . The parasite undergoes merogony in the bovine, and gamogony and sporogony in the tick. Sporozoites (Sz) injected into the bloodstream of a bovine during the blood meal of an infected tick, invade erythrocytes and differentiate into trophozoites (T), which divide asexually into usually two merozoites (M). Merozoites eventually exit erythrocytes and invade new ones, continuing their replicative cycle. Some merozoites stop division and transform into gamonts or pre-gametocytes (G), which are taken up by a tick, and differentiate in the tick gut into gametes, also known as ray bodies or Strahlenkörper (Sk), that fuse forming a diploid zygote (Z). Zygotes undergo meiosis giving rise to motile haploid kinetes which multiply by sporogony and access the hemolymph, invading and replicating in several tick organs, including the tick ovaries and eggs. Kinetes transform into sporotozoites in the salivary glands (Sg) of the next generation larvae (transovarial transmission, To). Additionally, it has been shown that some babesias can persist in the tick after it has molted, and infective sporozoites are produced in the Sg of the next stage (transstadial transmission, Ts).

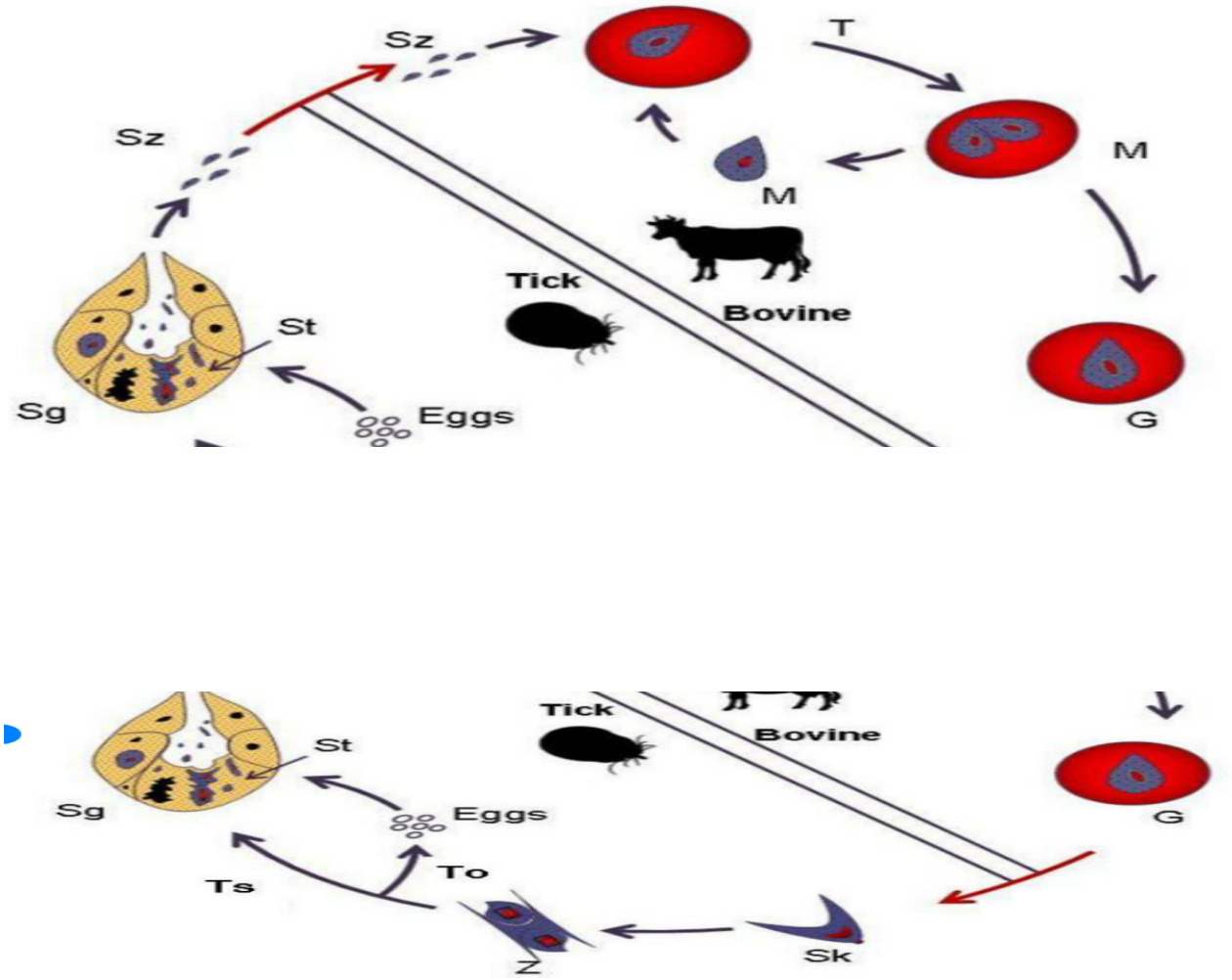


Figure1. 40. Life cycle of Babesia

2.5.5. Toxoplasma

Toxoplasmosis is an important zoonotic protozoal infection worldwide. All homoeothermic animal species may be infected. Infection is generally asymptomatic and chronic in immunocompetent individuals. Common clinical signs are abortion and reproductive failure, especially in small ruminants, cervids, and pigs. Some susceptible animal species (e.g., Australian marsupials, New World monkeys, and meerkats), as well as immunocompromised individuals,

suffer multiorgan failure, referred to asfatal toxoplasmosis. Humans may experience retinochoroiditis (ocular toxoplasmosis) or fetal neurologic disorders (congenital toxoplasmosis). Monitoring by means of serologic testing and anticoccidial treatments is recommended.

Etiology and Pathogenesis of Toxoplasmosis in Animals

Toxoplasma gondii is an apicomplexan protozoan that infects humans and other warm-blooded animals worldwide, including birds and marine mammals. This intracellular parasite has a facultative, indirect life cycle, reproducing sexually in enterocytes from definitive hosts and asexually in nucleated cells from intermediate hosts. Felids are the only definitive hosts of T gondii, which contaminates the environment with oocysts transmitted in feces. T gondii has three infectious stages: tachyzoites (rapidly multiplying form), bradyzoites (slow division form contained in tissue cysts), and sporozoites (in mature or sporulated oocysts). There are different T gondii genotypes, which exhibit different behaviors and virulence. Genotypes detected in South America, Africa, and Asia are considered more virulent than those predominating in Europe and North America.

T gondii is transmitted by consumption of infectious oocysts from a contaminated environment, by consumption of tissue cysts in infected meat, and by transplacental transfer of tachyzoites from mother to fetus. T gondii initiates enteroepithelial replication in unexposed felids after ingestion of uncooked tissues containing cysts. Bradyzoites are released from tissue cysts by digestion in the stomach and small intestine, invading intestinal epithelium, and undergoing asexual and sexual replication, culminating in the release of oocysts (~10 mcm in diameter) in the feces. Oocysts are first evident in the feces 3 days after infection and may be released for up to 20 days.

Oocysts sporulate (become infectious) outside the cat within 1–5 days, depending on aeration and temperature, and remain viable in the environment for several months. Cats generally develop immunity to T gondii after the initial infection, and they shed oocysts only once in their lifetime; however, immunocompromised animals can re-shed oocysts. Felids are also intermediate hosts for extraintestinal asexual multiplication of the parasite, especially in nervous system cells. Neurologic and ocular lesions can arise in immunocompromised and elderly cats.

After being consumed in uncooked meat containing tissue cysts (carnivores) or in feed or drink contaminated with cat feces containing oocysts (all warm-blooded animals), T gondii initiates extraintestinal replication. Bradyzoites and sporozoites, respectively, are released and infect intestinal epithelium and other tissues. After asexual replication, tachyzoites emerge and disseminate via the bloodstream and lymph. Tachyzoites infect tissues throughout the body and replicate intracellularly until the cells burst, causing tissue necrosis. Tachyzoites are banana shaped and measure 4–6 × 2–3 mcm. Susceptible species, and young, immunocompromised animals (e.g., puppies, kittens, piglets) may die from generalized toxoplasmosis at this stage of illness.

Adult animals mount a powerful, cell-mediated immune response (mediated by cytokines) and control infection, driving the tachyzoites into the tissue cyst or bradyzoite stage. Tissue cysts usually occur in neurons; however, they also occur in other tissues, such as muscles. Individual cysts are microscopic, up to 100 mcm in diameter, and may enclose hundreds of bradyzoites in a thin, resilient cyst wall.

Clinical Findings of Toxoplasmosis in Animals

The tachyzoite is the stage responsible for tissue damage in toxoplasmosis; therefore, clinical signs depend on the affected tissue, the number of tachyzoites released, and the ability of the host immune system to limit replication and transmission. Because adult immunocompetent animals control tachyzoite transmission efficiently, toxoplasmosis is usually a subclinical illness. However, in susceptible species, as well as in young and immunocompromised animals (particularly puppies, kittens, and piglets), tachyzoites are transmitted systemically and cause interstitial pneumonia, myocarditis, hepatic necrosis, meningoencephalomyelitis, chorioretinitis, lymphadenopathy, and myositis. The corresponding clinical signs include fever, diarrhea, cough, dyspnea, icterus, seizures, and death.

T gondii is also an important cause of abortion and stillbirth in sheep, goats, cervids, and sometimes pigs. After infection of a pregnant ewe, tachyzoites are transmitted via the bloodstream

to placental cotyledons, causing necrosis. Tachyzoites may also be transmitted to the fetus, causing necrosis in multiple organs. Finally, immunocompromised adult animals (eg, cats infected with feline immunodeficiency virus) are extremely susceptible to developing acute generalized toxoplasmosis, mainly expressed as neurologic and respiratory disorders.

Women who acquire T gondii during pregnancy can develop a fever, and transplacental passage of tachyzoites to the fetus may occur. Veterinarians have a key role in preventing this zoonotic disease by recognizing contamination of the environment and water by oocysts, by observing the presence of cysts in animal tissues, and by helping to develop, disseminate, and apply control measures.

Diagnosis of Toxoplasmosis in Animals

Indirect diagnosis by serologic testing

Postmortem diagnosis via histologic, bioassay, and molecular methods Toxoplasmosis is diagnosed by biologic, serologic, or histologic methods; or some combination of these. Clinical signs of toxoplasmosis are nonspecific and are not sufficiently characteristic for a definitive diagnosis. Antemortem diagnosis may be accomplished by indirect hemagglutination assay, indirect fluorescent antibody assay, latex agglutination test, or ELISA testing. IgM antibodies appear sooner after infection than IgG antibodies; generally, however, IgM antibodies do not persist past 3 months after infection. Increased IgM titers (>1:256) are consistent with recent infection. In contrast, IgG antibodies appear by the fourth week after infection and may remain increased for years during subclinical infection. To be useful, IgG titers must be measured in paired serum samples obtained during the acute and convalescent stages (3–4 weeks apart) and must show at least a fourfold increase/decrease in titer (seroconversion). In addition, CSF and aqueous humor samples may be analyzed for the presence of tachyzoites or anti–T gondii antibodies.

Postmortem, tachyzoites may be evident in tissue impression smears. Also histologic examination of tissue sections may reveal the presence of tachyzoites or bradyzoites, which could be

specifically marked by immunohistochemical staining. T gondii is morphologically similar to other protozoal parasites and must be differentiated from Sarcocystis species and Neospora caninum, which may also be involved in abortions and infection of neurologic tissues. Application of specific PCR assays allows diagnosis from tissue DNA samples. Once the presence of parasitic DNA is confirmed, the sample can be further processed for parasite genotyping.

Treatment of Toxoplasmosis in Animals

For acute toxoplasmosis: anti-coccidial drugs, administered as soon as possible after infection is suspected For primary infection: specific treatment to avoid or minimize vertical transmission For animals other than humans, treatment of toxoplasmosis is seldom warranted. Sulfadiazine (15–25 mg/kg, PO, every 12 hours for 4 weeks) and pyrimethamine (0.44 mg/kg, PO, every 12 hours for 4 weeks) act synergistically and are widely administered for treatment of toxoplasmosis. Trimethoprim-sulfamethoxazole (15 mg/kg, PO, every 12 hours for 4 weeks) is also widely recommended in dogs and cats. Although these drugs are beneficial if given in the acute stage of the disease when there is active multiplication of the parasite, they will not usually eradicate infection.

Other drugs, including diaminodiphenylsulfone, atovaquone, and spiramycin, may also be administered to treat toxoplasmosis in difficult cases. For dogs and cats, clindamycin is the treatment of choice, administered PO every 12 hours for 3–4 weeks at 10–12.5 mg/kg in dogs and 25–50 mg/kg in cats. Toltrazuril, ponazuril, and diclazuril may be administered to treat acute toxoplasmosis, as well as to reduce the shedding of oocysts by cats. No drugs are particularly effective at the bradyzoite stage.

Prevention and Zoonotic Risk of Toxoplasmosis in Animals

Toxoplasmosis is an important zoonotic disease. In some areas of the world, as much as 60% of the human population has serum IgG titers for T gondii and is likely to be persistently infected. Toxoplasmosis is a major concern for people with compromised immune systems. In these individuals, toxoplasmosis usually presents as meningoencephalitis and results from the emergence of T gondii from tissue cysts located in the brain as immunity wanes (reactivation) rather than from primary infection. Toxoplasmosis is also a concern for pregnant women because tachyzoites can migrate transplacentally and cause neurologic disorders in the fetus. Infection with T gondii may occur after ingestion of undercooked meat or accidental ingestion of oocysts from cat feces thorough contaminated water or vegetables.

To prevent infection, hands should be washed thoroughly with soap and water after contact with meat or potentially contaminated environments. The stages of T gondii are killed by cooking and contact with boiling water, and boiling water should be used to clean all cutting boards, sink tops, knives, and other materials. T gondii can also be killed by freezing. Tissue cysts are killed by heating throughout to 67°C (152.6°F) or by cooling to −13°C (8.6°F). Toxoplasma individuals in tissue cysts are also killed by exposure to 0.5 kilorad of gamma irradiation. Meat of any animal should be cooked to 67°C (152.6°F) before consumption, and tasting meat while cooking or while seasoning should be avoided.

Pregnant women should avoid contact with cat litter, soil, and raw meat. Pet cats should be fed only dry, canned, or cooked food. The cat litter box should be emptied daily, preferably not by a pregnant woman. Gloves should be worn while gardening. Vegetables should be washed thoroughly before eating, because they may have been contaminated with cat feces. There is currently no vaccine to prevent toxoplasmosis in humans. Some countries have approved a vaccine to prevent abortions in sheep.

Life cycle

Toxoplasma gondii is a protozoan parasite that infects most species of warm-blooded animals, including humans, and causes the disease toxoplasmosis.

Life Cycle:

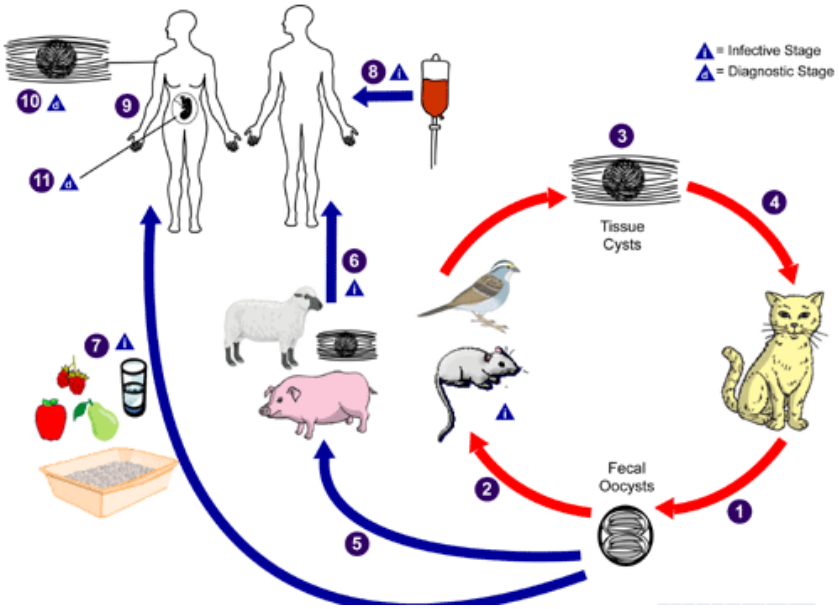


Figure 33. Toxoplasma life cycle

The only known definitive hosts for *Toxoplasma gondii* are members of family Felidae(domestic cats and their relatives). Unsporulated oocysts are shed in the cat's feces . Although oocysts are usually only shed for 1–3 weeks, large numbers may be shed. Oocysts take 1–5 days to sporulate in the environment and become infective. Intermediate hosts in nature (including birds and rodents) become infected after ingesting soil, water or plant material contaminated with oocysts . Oocysts transform into tachyzoites shortly after ingestion. Thesetachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites . Cats become infected after consuming intermediate hosts harboring tissue cysts. Cats may also become infected directly by ingestion of sporulated oocysts. Animals bred for human consumption and wild game may also become infected with tissue cysts after ingestion of sporulated oocysts in the environment .

Humans can become infected by any of several routes:

- Eating undercooked meat of animals harboring tissue cysts .
- Consuming food or water contaminated with cat feces or by contaminated environmental samples (such as fecal-contaminated soil or changing the litter box of a pet cat) .
- Blood transfusion or organ transplantation .
- Transplacentally from mother to fetus

In the human host, the parasites form tissue cysts, most commonly in skeletal muscle, myocardium, brain, and eyes; these cysts may remain throughout the life of the host. Diagnosis is usually achieved by serology, although tissue cysts may be observed in stained biopsy specimens . Diagnosis of congenital infections can be achieved by detecting *T. gondii* DNA in amniotic fluid using molecular methods such as PCR.

Key Points

Toxoplasmosis is a zoonosis that affects all warm-blooded animals worldwide.

A high proportion of animals and humans are infected without clinical signs. Different T gondii genotypes could generate different clinical outcomes.

Immunocompromised individuals may develop generalized toxoplasmosis with multiorgan failure.

T gondii may be transmitted vertically and produce fetal lesions and abortions.

2.5.6. Amoeba

The ameba (also spelled amoeba) is a protozoan that belongs to the Kingdom Protista. The name ameba comes from the Greek word “amoibe”, which means "change". Protists are microscopic unicellular organisms that don't fit into the other kingdoms. Some protozoans are considered plant-like, such as algae, and others are considered animal-like. The ameba is considered an animal-like protist because it moves and consumes its food, but it is not classified as an animal because it consists of a single cell; it is unicellular.

Protists are also classified by how they move, some have cilia or flagella, but the ameba has an unusual way of creeping along by stretching its cytoplasm into fingerlike extensions called

pseudopodia. The word "pseudopodia" means "false foot". On the coloring sheet, there are several pseudopodia, use a yellow highlighter or pencil to There are two types of cytoplasm in the ameba, the darker cytoplasm toward the interior of the protozoan is called endoplasm, and the clearer cytoplasm that is found near the cell membrane is called ectoplasm. On the coloring, the endoplasm is indicated by the dotted area, and the ectoplasm by the white area. Color the endoplasm blue , and leave the ectoplasm uncolored. By pushing the endoplasm toward the cell membrane, the ameba causes its body to extend and creep along. It is also by this method that the ameba consumes its food. The pseudopodia extend out and wrap around a food particle in a process call phagocytosis. The engulfed food then becomes a food vacuole. There are several food vacuoles on the drawing – color each brown . The food will eventually be digested by the cell's lysosomes.

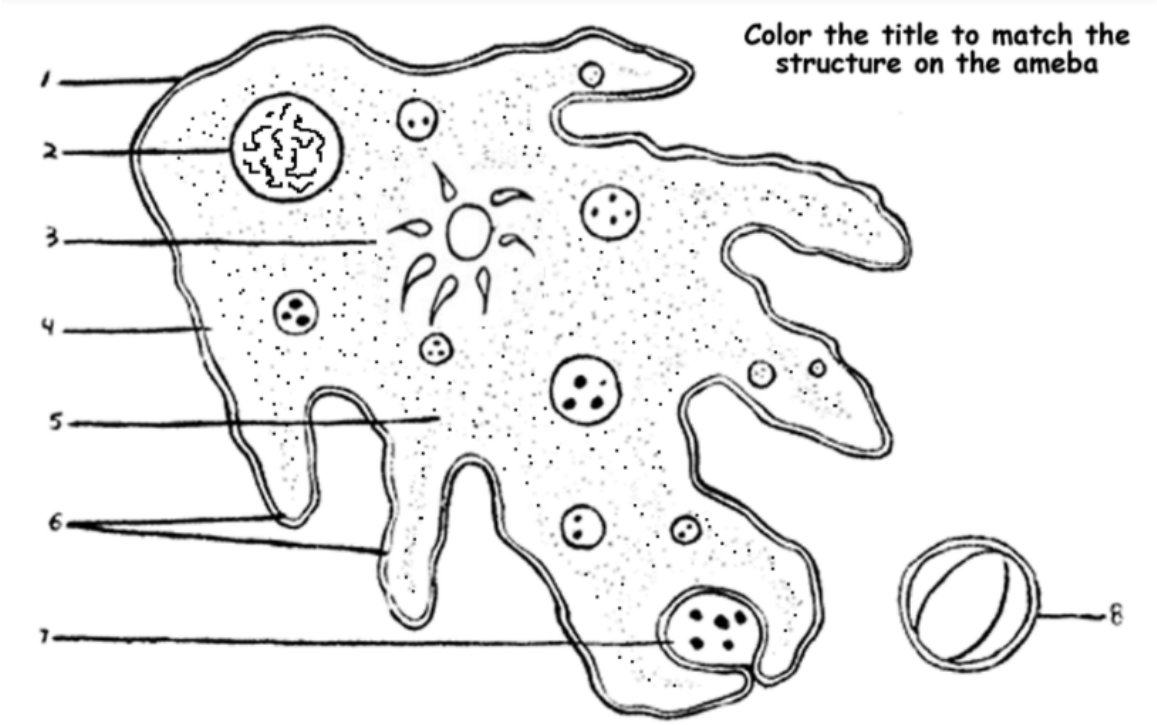


Figure 1.41. Ameba

2.5.7. Balantidium

Sixteen species of Balantidium have been described from india in different animals and valantidium coli var.bovis is reported from cattle.However, there is a lot of information on

balatidiosis of pigs there is little information on cattle balantidiosis. *Balantidium* ***coli*** is the only species in the genus ***Balantidium***. The trophozoite stage in the large intestine can cause ulcerative colitis and diarrhea. The cyst is the infective stage. ***B. coli*** is a commensal widely distributed in pigs. Water obtained from drainage areas contaminated by human or pig feces is the major source of human infection. The ***B. coli*** cyst can be identified from water after concentration by filtration or centrifugation. The cyst is large (45–65 μm) and surrounded by a distinct wall and it contains a large nucleus that is often bean-shaped. Both cysts and trophozoites can be identified by microscopic examination of suspect stool specimen mixed with physiological saline. In formalin-fixed samples trophozoites often resemble debris, artifacts, or eggs. *Balantidium coli* is a ciliated protozoan that primarily infects swine and nonhuman primates.

Balantidium coli (B. coli) is an emerging ciliated protozoan parasite of zoonotic importance which causes a disease balantidiasis in a variety of host species including pigs, camels, ruminants, equines and even human. This disease has a cosmopolitan distribution with high prevalence rates in tropical and sub-tropical areas of the world due to favorable geo-climatic conditions for the development and survival of the parasite in these regions. Main reservoir hosts for this pathogen are pigs and animals; acquire infection mainly by the ingestion of the food or water contaminated with the porcine feces. The infected animal manifests clinical signs of anorexia, dehydration, profuse watery diarrhea and retarded growth. Wet mount slide prepared from intestinal scrapings and fecal material is used for the identification of trophozoites and cysts stages of this parasite.

PCR can also be used to confirm the parasite. Secnidazole, oxytetracycline and metronidazole have varying efficacy against B. coli infection in various domestic animal species. There is no comprehensive literature available on the occurrence and distribution of the infection at international level. Therefore, the published data between 1989 and 2019 regarding this disease is critically analyzed to provide a detailed overview on this pathogen with special emphasis on geographical distribution of B. coli in domestic animals and different therapeutic agents used to treat this infection. This review will pinpoint the endemic regions which may be a source of

potential disease outbreaks and will also help in application of more effectual control strategies against balantidiasis.

Balantidium coli is an intestinal protozoan parasite that causes the infection called balantidiasis. While this type of infection is uncommon in the United States, humans and other mammals can become infected with ***Balantidium coli*** by ingesting infective cysts from food and water that is contaminated by feces. Mostly asymptomatic, ***Balantidium*** infection can cause such symptoms as diarrhea and abdominal pain.

prevent infection- Wash all fruits and vegetables with clean water when preparing or eating them, even if they have a removable skin.

Self-Check – 2	**Written Test**

Name…… ID…… Date…….

Directions: Answer the following questions accordingly.

Test I: Multiple choices (2 pt each)

1. From the following which is/are grouped under nematode parasites?

   A, Fasciola B, Heamonchus C, Trematode D, Flukes

2. ----------is known as lung worm

   A, Fasciola B, Heamonchus C, Trematode D, dictyocaulus

3. ----------Is common parasite of equine species

   A, Styrongyles B, Fasciola C, Cestode D, all

4. ----------is intermediate host for fasciola

   A, Animal B, Snail C human D, all

5. ----------is drug of choice for trematode treatment except fasciolosis

   A, pyrizequantel B, Triclabendazole C, Ivermectin D, all

Test II: Short Answer Questions (2 pt each)

1. Differentiate the following terms

   A,Schistosoma

   B, Schistosomosis

   C, Schistosomiasis

2. Explain the types of parasites

3. Write about eye worms
4. List protozoal parasites of veterinary importance
5. Write about diseases coused by teania and types of ticks

Note: Satisfactory rating – 20points Unsatisfactory - below 20points
You can ask you teacher for the copy of the correct answers.

Operation Sheet -2

5.2. Tools and procedures required for ssedimentation test

A. Tools and equipments

I. 250 ml beaker

II. Tea strainer or cheesecloth

III. Pipette

IV. Petridish

V. Methylene blue

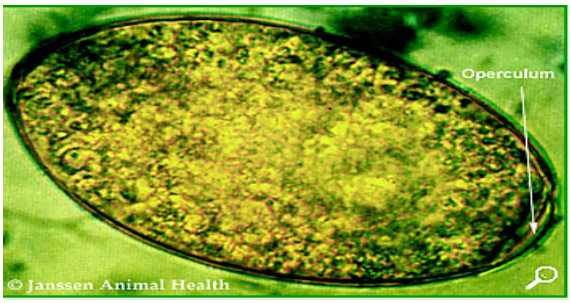
VI. Microscopy

VII. Test tube

B. Procedures/Steps/Techniques needed for fecal sedimantation

I. Measure of 3g/5 feces and put in to beaker
II. Pour 40-50ml of tap water in to a container/beaker and mix them thoroughly
III. Filter a fecal suspension through a tea sieve or double layer of a cheesecloth in to a second beaker
IV. Pour the filtered materials in to a test tube
V. Alow to sediment for 5minutes
VI. Remove the supernants very carefully with a pippets
VII. Repeat step 3 to 5 atleast 3times until the supernant is clear
VIII. After removing the supernant for the last time trabsfer the sediment to a Petridish, add adrop of methylene blue/malachite green and examine for eegs using microscopy(the methylene blue stains the background)

The result: +v granular yellowish-brown contents fillingwhole egg oneside operculated, larger egg, and regularly ellipsoid -fasciola



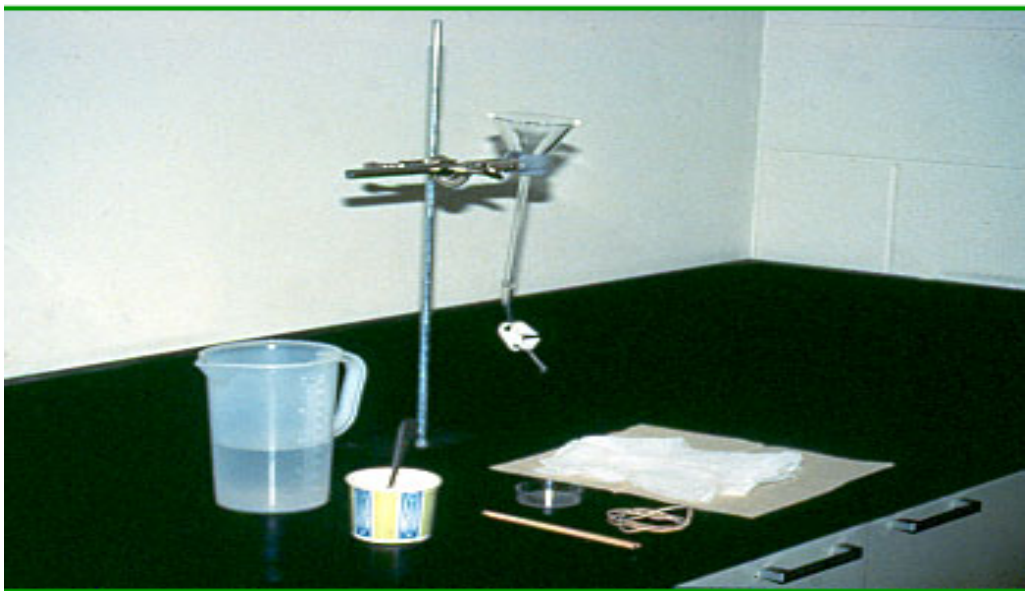
VIII.3. Tools and procedures required for Lung warm identification

A, Tools and Equipments

I. Funnel – size according to need
II. Funnel stand
III. Rubber or plastic tubing
IV. Clamp or spring clip
I. Pasteur pipette
II. Small petri dishes
III. Scissors
IV. Disposable paper towels

V. Cheesecloth or dental napkin
VI. Thin stick or metal rod
VII. Strainer
VIII. Microscope
IX. Test tube

V. Spoon or spatula
VI. Rubber band or length of string
VII. Jug or flask
VIII. Microscope slides and coverslips
IX. Iodine



B, procedures required for Lung warm identification

I. Take a funnel and fit a short piece of tubing to the stem.Close the tubing with a clamp or spring clip.Support the funnel on a single stand.
II. Place a double layer of cheesecloth or dental napkin on a disposable paper towel or equivalent on the bench.Using a spoon or spatula weigh or measure approximately 5-10 grams of faecal material.Place the faecal material in the centre of the cheesecloth.

III. Using a rubber band or length of string close the cheesecloth pouch.Push the stick or short metal rod under the rubber band or string so that the pouch can be suspended.

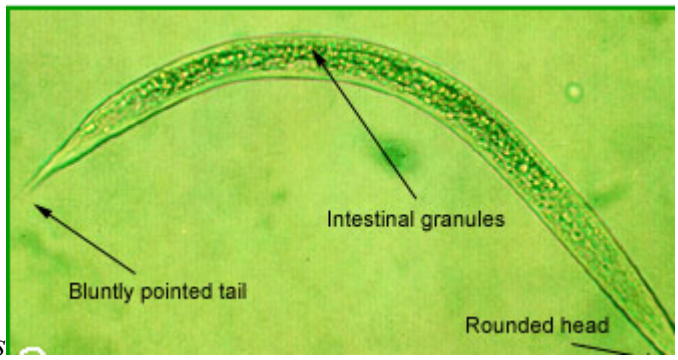
IV. Place the pouch containing the faecal material in the funnel. Trim off the excess cheesecloth.

V. Draw off a few millilitres of fluid from the stem of the funnel into a test tube. Then either: leave to sediment for at least 30 minutes Or if a centrifuge is available, the fluid can be drawn into a centrifuge tube and spun at 1000 rpm for 2 minutes.

VI. Check sedimented sample in a petri dish for the presence of larvae.

VII. Use a Pasteur pipette to transfer a small droplet of the sedimented fluid from the petri dish to a microscope slide. Add drop of iodine to fix the larvae and gently place a coverslip over the drop.

Result: -ve



Dictyocaulus viviparus

Length 390-450 µm

Larva often curved
Sluggish movemen

LAP TEST-2	**Performance Test**

Name……. ID……..

Date…….

Time started: _____2:30_____ Time finished: __3:30_____

Instructions: To perform the following tasks carefully.

Task-1 Perform Sedimentation technique

Task 2 Perform baerman technique

LG #7	LO#3-Recoginize common ectoparasitess of animals and manage associated problems

Instruction sheet 3.

This learning guide is developed to provide you the necessary information regarding the following content coverage and topics:

- Characteristic feature, stages of development, geographic distribution, clinical effect on animals, economic effect on animal product/by-product, treatment and prevention/control measures of important and prevalent ***tick*** of farm animals' are identified and implemented.
- Characteristic feature, stages of development, geographic distribution, clinical effect on animals, economic effect on animal product/by-product, treatment and prevention/control measures of important and prevalent ***mites*** of farm animals' are identified and implemented.
- Characteristic feature, stages of development, geographic distribution, clinical effect on animals, economic effect on animal product/by-product, treatment and prevention/control measures of important and prevalent ***cutaneous myasis,*** fleas***, flies, lice, true bugs*** and leech of farm animals' are identified and implemented.

This guide will also assist you to attain the learning outcomes stated in the cover page. Specifically, upon completion of this learning guide, you will be able to:

- Identify and implement Etiology, transmission, ecology, clinical sign, diagnosis, treatment and prevention/control measures of economically important and prevalent ***tick*** parasitic diseases of farm animals'
- Identify and implement Etiology, transmission, ecology, clinical sign, diagnosis, treatment and prevention/control measures of economically important and prevalent ***mites*** parasitic

diseases of farm animals

- Identify and implement Etiology, transmission, ecology, clinical sign, diagnosis, treatment and prevention/control measures of economically important and prevalent prevalent ***cutaneous myasis,*** fleas***, flies, lice, true bugs*** and leech parasitic diseases of farm animals

Learning Instructions:

1. Read the specific objectives of this Learning Guide.
2. Follow the instructions described below.
3. Read the information written in the information Sheets
4. Accomplish the Self-checks
5. Perform Operation Sheets
6. Do the "LAP test"

Information Sheet 3:

3.1. Tick

Ticks (order **Ixodida**) are parasitic arachnids that are part of the mite superorder Parasitiformes. Adult ticks are approximately 3 to 5 mm in length depending on age, sex, species, and "fullness". Ticks are external parasites, living by feeding on the blood of mammals, birds, and sometimes reptiles and amphibians. The timing of the origin of ticks is uncertain, though the oldest known tick fossils are from the Cretaceous period, around 100 million years old. Ticks are widely distributed around the world, especially in warm, humid climates.

Ticks belong to two major families, the Ixodidae or hard ticks, and the Argasidae, or soft ticks. *Nuttalliella,* a genus of tick from southern Africa, is the only member of the family Nuttalliellidae, and represents the most primitive living lineage of ticks. Adults have ovoid/pear-shaped bodies (idiosomas) which become engorged with blood when they feed, and eight legs. Their cephalothorax and abdomen are completely fused. In addition to having a hard shield on their dorsal surfaces, known as the scutum, hard ticks have a beak-like structure at the front containing the mouthparts, whereas soft ticks have their mouthparts on the underside of their bodies. Ticks locate potential hosts by sensing odor, body heat, moisture, and/or vibrations in the environment.

Ticks have four stages to their lifecycle, namely egg, larva, nymph, and adult. Ticks belonging to the **Ixodidae** family undergo either a one-host, two-host, or three-host lifestyle. Argasid ticks have up to seven nymphal stages (instars), each one requiring blood ingestion, Argasid ticks undergo a multihost lifestyle. Because of their hematophagous (blood-ingesting) diets, ticks act as vectors of many serious diseases that affect humans and other animals.

Ticks are the most important ecto-parasites of livestock in tropical and subtropical areas and are responsible for severe economic losses in livestock and are effective disease vectors, second only

to mosquitoes in transmitting infectious disease. Ticks belong to the phylum Arthropod, class Arachnid and order Acari. The families of ticks parasitizing livestock are categorized into two, the ***Ixodidae*** (hard ticks) and ***Argasidae*** (soft ticks).

Though, sharing certain basic properties, they differed in many structures, behavioural, physiological and feeding and reproduction pattern. They are obligate, blood feeding ectoparasites of vertebrates, particularly mammals and birds. It has been estimated that about 80% of the world population of cattle are infested with ticks. The life cycle of ticks (both ixodids and argasids) undergo four stages in their development; eggs, 6-legged larva, 8-legged nymph and adult. According to the number of hosts, Ixodids ticks are classified as one host ticks, two host ticks, three host ticks and Argasids classified as multi host ticks.

In one host ticks, all the parasitic stages (larva, nymph and adult) feed on the same hosts; in two host ticks, larva attach to one host, feed and moult to nymphal stage and engorged, after which they detach and moult on the ground to adult; and in three host ticks, the larva, nymph and adult attach to different hosts and all detach from the host after engorging and detaching from the hosts. Ticks are relatively large and long lived, compared to mites, surviving for up to several years. Although, only relatively few of more than 889 species of tick in the world are important to man and his domestic animals, these few species must be controlled if livestock production is to meet world needs for animal protein.

Over 79 different species of ticks are found in Eastern Africa and many of these appear to be of little or no economic importance. In Ethiopia, there are 47 species of ticks found on livestock and most of them have importance as vector and disease causing agents and also have damaging effect on skin and hide production. The Genus Amblyomma and Rhipicephalus ticks are predominating in many parts of the country, Boophilus and Hyalomma ticks also have a significant role. ***Amblyomma cohaerence*** is prevalent and abundant in western humid highland areas of Ethiopia. ***Boophilus decoloratus*** and ***Rhipicephalus evertsi-evertsi*** are widely distributed in most altitudinal ranges.

Ticks, besides being important vectors for diseases like theilerosis, anaplasmosis, babesiosis, streptothricosis (Mekonnen) and rickettsiosis (heart water) in domestic animals; they also cause nonspecific symptoms like anaemia, dermatosis, toxicosis and paralysis. Besides to disease transmission ticks inflict a huge economic loss. Production losses due to Ticks and Tick-Borne Diseases (TTBDs) around the globe have been estimated at US$ 13.9 to US$ 18.7 billion annually leaving world's 80% cattle at risk and estimated an annual loss of US$ 500,000 from hide and skin downgrading from ticks, and approximately 65.5% of major defects of hides in Eastern Ethiopia are from ticks. Due to economic and veterinary importance of ticks, their control and the transmission of tick borne diseases remain a challenge for the cattle industry in tropical and subtropical areas of the world and it is a priority for many countries in tropical and subtropical regions. Investigations directed toward determining the magnitude of infestation and the type of species involved will play a magnificent role in designing strategic control toward these parasites. Moreover a species level identification will assist the diagnosis of different tick borne diseases and their respective control programs.

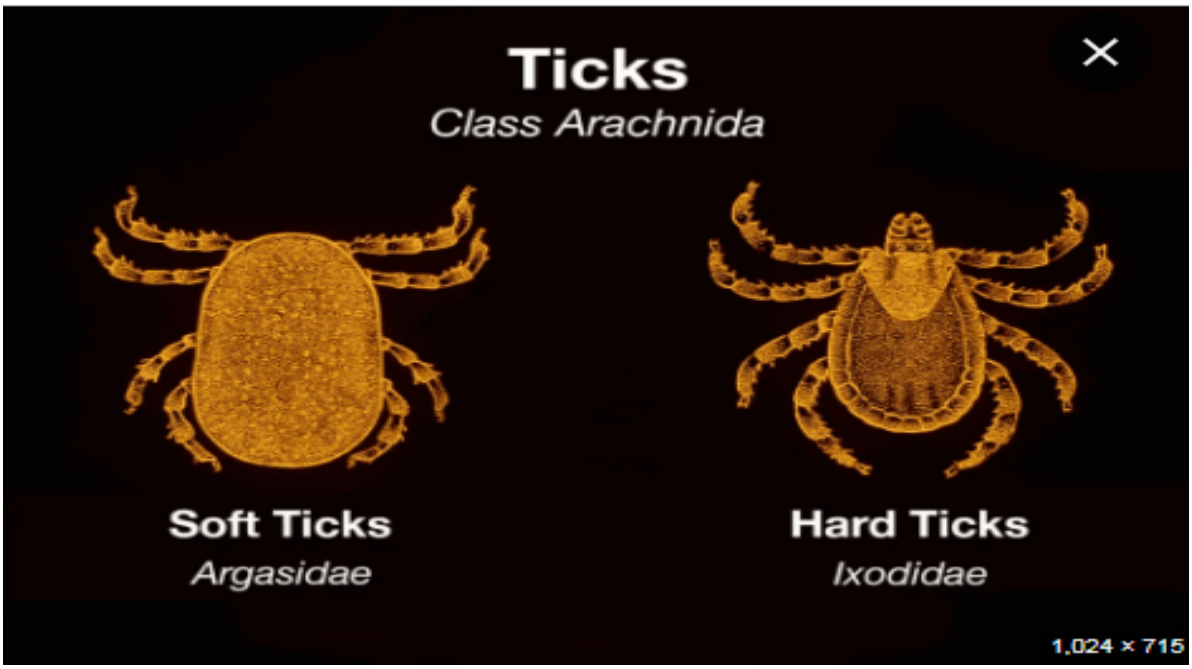


Figure 3.1. different species of ticks

Life cycle

Most hard ticks are either a one, two or three-host ticks. After the tick's egg stage hatches, one larval, one nymphal and an adult stage follows. Mature, blood-fed female adult ticks drop from the host, lay a large mass of a few to several thousand eggs and then die. Hard ticks usually mate while on host animals. Depending on the tick species and environmental conditions, the typical longevity of hard ticks ranges from about 2 months to 3 years. Adult female soft ticks feed and lay eggs several times during their lifetime. Most species of soft ticks do not bury themselves for a long stay on a host. Instead, they feed quickly, detach and then return later for another blood meal.

Also, soft tick nymphs may feed several times and go through several nymphal stages. Female adults that take multiple blood meals will lay small batches of 20-50 eggs after taking each blood meal during their lifetime. Soft ticks can live up to 16 years. All three tick families ticks have four lifecycle stages: egg, larva, nymph, and adult.

Ixodidae

Ixodidae ticks have three different life cycles. Depending on the species, Ixodids can either possess a one-host lifecycle, two-host lifecycle, or three-host lifecycle.

One-host ticks

In one-host ticks the tick remains on the host through the larval, nymphal, and adult stages, only to leave the host to lay eggs. Eggs laid in the environment hatch into larvae, which immediately seek out a host in which to attach and feed. Fed larvae molt into unfed nymphs that remain on the host. After engorging on the host's blood, the nymphs molt into sexually mature adults that remain on the host in order to feed and mate. Once a female is both fed and ready to lay eggs, only then does she leave the host in search of a suitable area to deposit her eggs. Ticks that follow this life cycle are called one-host ticks. The winter tick *Dermacentor albipictus* and the cattle tick *Boophilus microplus* are examples of one-host ticks.

Two-host ticks

The lifecycle of a two-host tick often spans two years. During fall the pregnant female tick will drop off her second host and lay her eggs. The eggs hatch during winter, the following spring the larvae emerge and attach to their first host. Newly hatched larvae attach to a host in order to obtain a blood meal. They remain on the host then develop into nymphs. Once engorged, they drop off the host and find a safe area in the natural environment in which to molt into adults, this typically occurs during the winter. Both male and female adults seek out a host on which to attach, which may be the same body that served as host during their early development but is often a larger mammal. Once attached, they feed and mate. Gravid females drop from the host to oviposit in the environment. Ticks that complete their life cycle in this manner are called two host ticks, like *Hyalomma anatolicum excavatum.*

Three-host ticks

Most ixodid ticks require three hosts, and their lifecycles typically span three years. The female tick drops off its host, often in the fall, and lays thousands of eggs. The larvae hatch in the winter and emerge in the spring. When the larvae emerge, they attach and feed primarily on small mammals and birds. During the summer the larvae become engorged and drop off the first host to molt and become nymphs, this often occurs during the fall. The following spring the nymphs emerge and seek out another host, often a small rodent. The nymphs become engorged and drop off the host in the fall to molt and become adults. The following spring the adult ticks emerge and seek out a larger host, often a large mammal such as cattle or even humans. Females will mate on their third host. Female adults then engorge on blood and prepare to drop off to lay her eggs on the ground, while males feed very little and remain on the host in order to continue mating with other females.

Argasidae

Argasid ticks, unlike ixodid ticks, may go through up to seven nymphal stages (instars), requiring a meal of blood each time. Often, egg laying and mating occurs detached from the host in a safe environment. The eggs hatch and the larvae feed on a nearby host for anywhere from a few hours to several days, this depends on the species of tick. After they feed the larvae drop and molt into their first nymphal instars, then the nymph seeks out and feeds on its second host, often this is the same as the first host, within an hour. This process occurs repeatedly and until the last nymphal instar occurs, thus allowing the tick to molt into an adult. Once an adult these ticks feed rapidly and periodically their entire lifecycle. In some species an adult female may lay eggs after each feeding. Their lifecycles range from months to years. The adult female argasid tick can lay a few hundred to over a thousand eggs over the course of her lifetime. Both male and female adults feed on blood, and they mate off the host. During feeding, any excess fluid is excreted by the coxal glands, a process that is unique to argasid ticks.

3.2. Mites

Mites- Mites are a common cause of itching and scratching in dogs and cats. They are microscopic and therefore require a microscope to be seen. There are a variety of mites, but the 3 most common species that cause disease in dogs and cats include Demodex, Sarcoptes and Otodectes.

Demodex mange or demodicoses can be localized (only a certain area is affected) or generalized (whole body is affected). Both forms can affect young dogs and adult dogs. Normal dogs have a small number of demodex mites on their bodies and do not cause them any harm. It is when there is an overgrowth of mites that disease develops. An overgrowth occurs then an animal has an underlying disease, an immune defect or an underdeveloped immune system, such as with puppies.Localized demodicosis is characterized by patchy hair loss on the head or limbs and will often occur in puppies less than six months of age. Generalized demodicosis can cause significant disease characterized by hair loss, skin redness and skin infections.

Sarcoptic mange or scabies is a fairly common skin mite that causes dogs and cats to be extremely itchy. This is due to the motion of the mite under the skin and in addition, the eggs and mites can cause an allergic reaction. Sarcoptic mites prefer hairless skin or less haired areas. The ear pinnae, elbows and abdomen are most likely to be affected. The skin on these areas will become red, scaly and very itchy. It is important to note that sarcoptic mange can be transmitted to other animals by direct contact. People can become infested with the mites present on their dogs. The lesions that are produced will be highly itchy but will almost always clear without the need of specific treatment for the mite infestation. Sarcoptic mange is diagnosed by microscopic examination of deep skin scrapes from affected areas of hair loss. There may be cases where severe skin disease can occur even though repeated skin scrapings recover no mites.

Otodectes mites Ear Mites- Ear mites are small mites that live and reproduce in the ears of cats and dogs. They cause intense itching, inflammation and result in ear infections. When an animal has an ear infection due to ear mites, the ears will contain thick, black discharge that resembles coffee grounds. Not all animals that have ear infections will have ear mites. Ear mites are much

more likely to occur in cats with infections, whereas dogs will have ear infections due to other causes. Otodectes mites are spread by direct contact between animals.

Diagnosis is made on clinical signs and examination of an ear swab under the microscope. Please see your veterinarian if your pet is exhibiting clinical signs of an ear infection so ear mites can be ruled out.

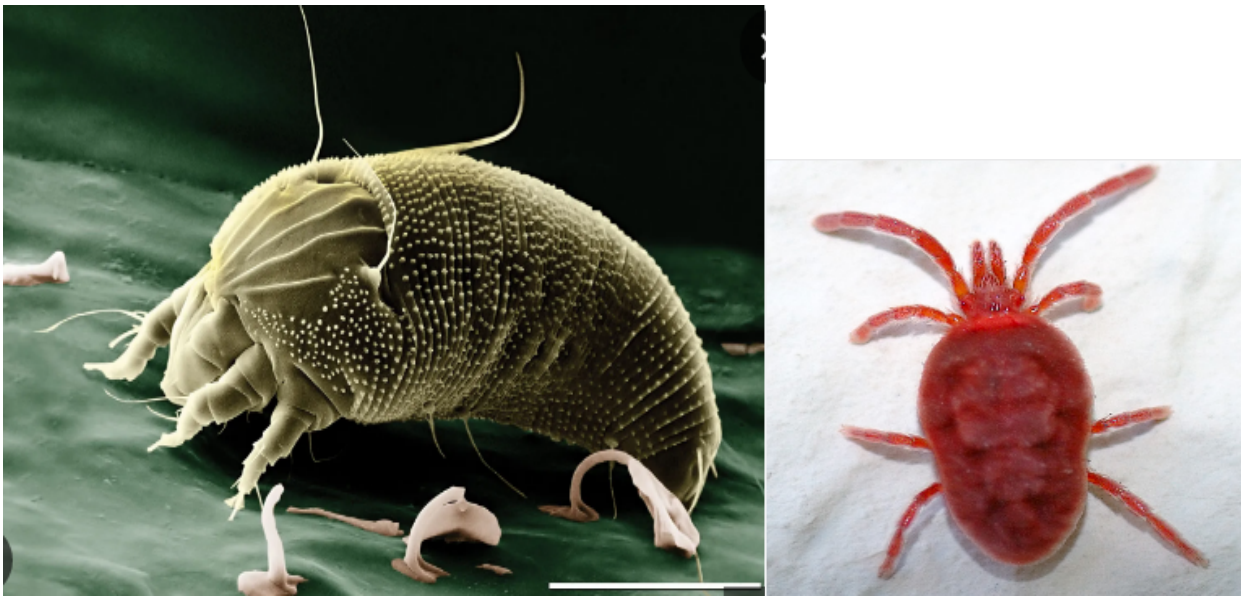


Figure 3.2. Diagram of different mite species

Life cycle

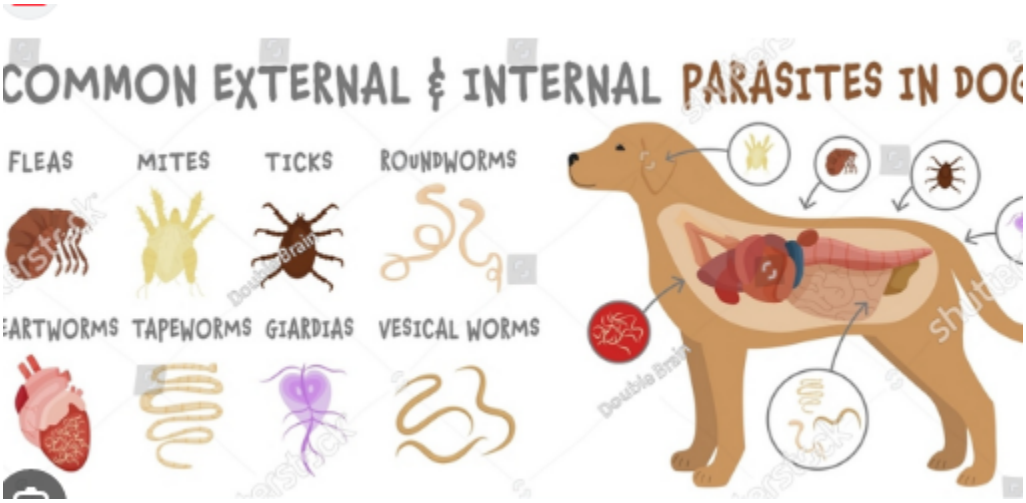


Figure 3.3. mite life cycle

3.3. Cutaneous myasis /Fleas, Flies, Lice, bed bugs and leech /

3.3.1.Lice(Louse)

Lice are small, wingless insects that infest the hairs, skin, and feathers of animals. Clinical signs include pruritus, alopecia, and occasionally anemia. Diagnosis is based on the inspection of predilection sites and visualization of lice or nits (eggs on hair). Many effective oral and topical treatments are available (eg, pyrethroids, macrocyclic lactones). A key component of prevention and control is addressing management practices that lead to stress, overcrowding, and poor nutrition. Most lice are host-specific, obligate ectoparasites that depend on the host to complete their life cycle. In general, they are divided into two categories: bloodsucking (or sucking) lice (order Anoplura) and chewing (or biting) lice (formerly the order Mallophaga, now composed of

three suborders). Bloodsucking lice are parasites of mammals; chewing lice infest both mammals and birds.

Lice live within the microenvironment provided by a host's skin and its hair or feathers, and they are transmitted primarily by contact between hosts. The predilection site depends on the lice species. All life stages occur on the host, but lice may survive off the host for a period of time. In temperate regions, lice are most abundant during the colder months and often are difficult to find in the summer. Infestations most often occur on stressed animals, and husbandry and individual health are important in the treatment and management of these parasites. (Also see Ectoparasites.) Lice infestations can lead to secondary dermal infections, and lice can transmit diseases and be intermediate hosts for other parasites.

Etiology and Pathogenesis of Lice in Animals

Lice eggs (nits)

Lice are wingless, flattened insects, ranging from 1 to 8 mm long and visible to the naked eye; however, magnification is often required to identify the species. On mammalian hosts, louse eggs, sometimes called nits,are glued to hairs near the skin surface and are pale, translucent, and suboval. The three nymphal stages, of increasing size, are smaller than adults; in habits and appearance, however, they resemble adults. Nymphs and adults use their claws and mouthparts to attach to the host.

Completion of one generation takes ~3–4 weeks. Usually, lice are transmitted by direct host contact. Lice dropped or pulled from the host die in a few days, and few, if any, nits develop after being removed from the host. However, fomites can be involved in transmission; therefore, the bedding of small animals and the premises of large animals that have been recently vacated by infested animals should be disinfected before being used again for uninfested animals.

Approaches to treating lice differ depending on whether the lice are chewing or sucking lice. Head shape, size and shape of claws on the legs, predilection site, and host are useful characteristics for identification.

Chewing lice:

- infest mammals and birds
- have a head with a blunted appearance that is wider than the thorax
- have ventral chewing mandibles
- feed on epidermal debris, primarily skin scales, sebaceous secretions, and feathers, if applicable
- Are active and can sometimes be observed moving through the hair

Sucking lice:

- infest only mammals
- have a head with a pointed appearance and narrower than the thorax
- have piercing mouthparts
- feed on blood

Clinical Findings and Diagnosis of Lice in Animals

- Pruritus, self-trauma
- Presence of lice and/or nits
- Parting feathers to see lice on animal

Pediculosis is manifested by pruritus and dermal irritation, resulting in scratching, rubbing, and biting of infested areas. A generally unthrifty appearance, rough coat, and lowered production in farm animals is common. In severe infestations, there may be loss of hair and local scarification. Extreme infestation with bloodsucking lice can lead to anemia; in rare cases, extreme infestation with chewing lice on dogs also can result in anemia. The constant crawling and piercing or biting of the skin may lead to restless behavior in hosts, and the wounds resulting from feeding by lice and rubbing by hosts can become infected.infestations can be more common in the winter in production animals and more common in stressed animals and in animals in a poor system of management. Crowding (eg, in the winter housing of production animals, or among caged and indoor free-roaming birds) also can increase lice infestations.

Treatment of Lice in Animals

- Ivermectin
- Permethrin lotion 1%

Prevention and Control

- Extermination of adult and nymphal life stages
- Prevention of transmission within a herd, flock, or household
- Washing of animal equipment and bedding to prevent transmission via fomites
- Modification of animal management to avoid overcrowding, poor nutrition, and stress Successful louse
- treatment of the affected animal(s),
- treatment of contact animals,
- environmental control, and elimination of stressors
- debulking the infested coat, if weather and coat type permit.



Figure 3.5. lice

Key Points

- Lice are host-specific ectoparasites.
- A poor coat, pruritus, dermal irritation, and, in heavy infestations, anemia, are common clinical signs.
- Many effective treatments are available; however, they can differ for chewing and sucking lice.
- Control and prevention must also address underlying conditions and management methods that lead to overcrowding and poor nutrition.

Self-Check – 3	**Written test**

Name…… ID…… Date…….

Directions: Answer the following questions accordingly.

Test I: Multiple choices (2 pt each)

1. From the following which is/are grouped under Ecto parasites?

   A, Fasciola B, Heamonchus C, Tick D, Flukes

2. Identify known as tick born diseases

   A, Fasciola B, Babesiosis C, Trematode D, dictyocaulus

3. From the following which is/are mites?

A, Demodex B, flies C, Tick D, all

4. ----------is/are hard tick/s

A, Amblyoma B, Hyaloma C Boofilus D, all

5. ----------is drug of choice for the treatment of lice

A, pyrizequantel B, Triclabendazole C, Ivermectin D, all

Test II: Short Answer Questions (2 pt each)

1. Differentiate the following terms

A, Ticks

B, Lice

C, mite

D, Flies

2. Discuss about tick born diseases

Note: Satisfactory rating - 14points Unsatisfactory - below 14points
You can ask you teacher for the copy of the correct answers.

Reference Materials

Books and journals

Food and Agriculture Organization of the United Nations. Manual on Meat Inspection for Developing Countries http://www.fao.org/docrep/003/t0756e/T0756E00.HTM The Merck Veterinary Manual https://www.merckvetmanual.com/Identification of Ixodide Tick Species on Bovine in and Around Mesela (Shanan Dhugo) District, Eastern Ethiopia, Hassen Yusuf Bekere, Department of Veterinary Science, Haramaya University, Haramaya, Ethiopia,)

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http://www.oie.int/international-standard-setting/terrestrialmanual/access-online/ OIE Terrestrial Animal Health Code

http://www.oie.int/international-standar DVM, DACVM, DEVPC, Department of Microbiology, Immunology, and Pathology, College of Veterinary Medicine and Biomedical Sciences, Colorado State University

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