TVET CERTIFICATE IV in Animal Health

COMMON PARASITIC DISEASES TREATMENT

ANHPD401

Treat Common Parasitic Diseases

Competence

Credits: 9

Learning hours: 90

Sector: Agriculture and Food Processing

Sub-sector: Animal Health

Module Note Issue date: July, 2020

Purpose statement

This core module provides the skills, knowledge and attitude for a learner to be competent in a range of routine tasks and activities that require the application of practical skills in a defined context of the treatment of the common parasitic diseases. Any veterinarian has to treat common parasitic diseases during his professional work. So, this competence is very important for the Veterinary assistants training.

Upon completion of this module, the trainee will be able to: Identify common parasites, Identify common parasitic diseases, Perform diagnosis of parasitic diseases and Perform treatment for parasitic diseases.

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Learning Unit 1 – Identify common parasites

Introduction

In addition to insufficient food quantity and quality, the low animal productivity is due to the persistence of several animal diseases, some of which are caused by parasites (tick borne diseases, worms, and protozoa.).

These diseases do not only cause the mortality but also the production loss (milk, meat, leather, eggs) loss. They also entail significant expenses related to their prevention, especially in their treatment.

Cross-breeds from local and exotic breeds and pure exotic breeds easily catch various parasitic diseases. However, the clinical diagnostic techniques are not always mastered.

Causation of parasitic disease

Parasitic disease must be understood in terms of the interrelationship among parasites, the host, and the environment. Some common **symbiotic** relationships between the hosts and the parasites are followings:

Commensalism: Commensalism is a relationship where one organism benefits without producing harmful effects for the other.

Parasitism: In parasitism, one organism benefits at the expense of the other. The parasite may be facultative or obligatory. If the parasite is able to live independently of the host, it is considered a facultative parasite. When the parasite has an absolute requirement for a host and cannot live outside the host, it is an obligatory parasite.

Parasite life cycle

It is the development of a parasite through its various life stages, for example ticks, from egg to adult. Each parasite has its own individual life cycle and may also have at least one definitive host and may be one or more intermediate hosts.

- **The definitive host** is the host that harbors the adult, sexual or mature stage of the parasite.
- **The intermediate host** is the host that harbors the larval, juvenile, immature or asexual stages of the parasite.
- **The transport or paratenic host** is a special intermediate host, in which a parasite does not undergo any development but instead remains arrested or encysted within the host's tissues.
- **A reservoir host** is a vertebrate host in which a parasite or disease occurs in nature and is a source of infection for human and domesticated animals.

Pathogenic role of parasites

The pathogenic role of parasites area:

- **Mechanical actions**: by causing injury to the tissues and blocking the organic ducts (biliary ducts, gastro-intestinal tract, and trachea).
- **Voracious actions**: by blood sucking and robbing the host of its food.
- **Toxins production**: most intestinal parasites produce toxins that injure the hosts.

L O 2.1 – Describe helminths

The word 'helminth' is a general term meaning 'worm', but there are many different types of worms. Prefixes are therefore used to designate types: platy-helminthes for flat-worms and nemathelminthes for round-worms.

Characteristics of helmenths

All helminthes are multicellular eukaryotic invertebrates with tube-like or flattened bodies exhibiting bilateral symmetry.

They are triploblastic (with endo-, meso- and ecto-dermal tissues) but the flatworms are acoelomate (do not have body cavities) while the roundworms are pseudocoelomate (with body cavities not enclosed by mesoderm). In contrast, segmented annelids (such as earthworms) are coelomate (with body cavities enclosed by mesoderm). The higher taxa (phylum) containing helminthes of veterinary importance are:

- Nemathelminthes (roundworms)
- Platyhelminthes (flatworms)
- Acanthocephalan (thorny headed worms)

- Topic 1: Nemathelminthes

A. Morphology of Nemathelminthes

Platyhelminthes also called Nematodes (roundworms) have long thin unsegmented tube-like bodies with anterior mouths and longitudinal digestive tracts. They have a fluid-filled internal body cavity (pseudocoelum) which acts as a hydrostatic skeleton providing rigidity (so-called 'tubes under pressure'). Worms use longitudinal muscles to produce a sideways thrashing motion. Adult worms form separate sexes with well-developed reproductive systems.

Picture 1: Common round worm (*Ascaris lumbricoides*)

B. Basic life cycle of nematodes

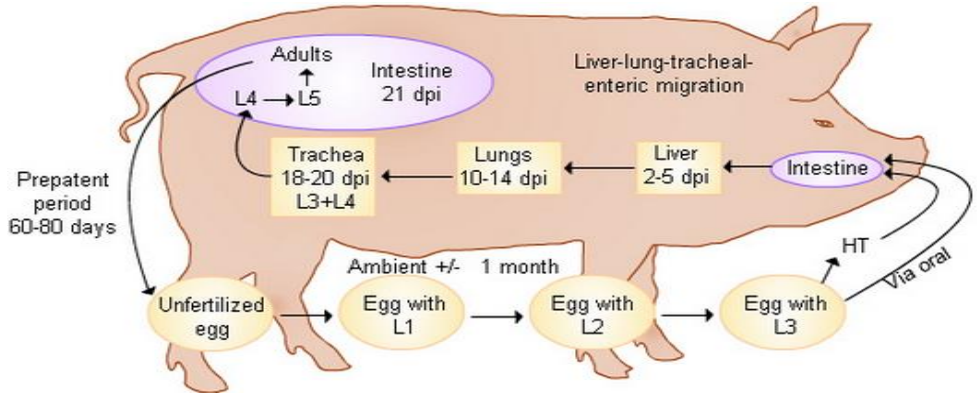
During development, a nematode moults at intervals shedding its cuticle. In the complete life cycle there are four moults, the successive larval stages being designed as L_1, L_2, L_3, L_4 and L_5 which is immature adult. Some development stages usually take place in the faecal part or in different species of animal, the intermediate host (I. H.), before the infection can take place.

In the common form of **direct life cycles**, the free-living larvae undergo two moults after hatching and infection is by ingestion of the free L_3. There are some important exceptions however, infection sometimes being by larval penetration of the skin or by ingestion of the egg containing a larva

In **indirect life cycles**, the first two moults usually take place in an intermediate host and infection of the final host is either by ingestion of the intermediate host or by inoculation of L_3 when the intermediate host feeds.

However, in many species the larvae travel considerable distances through the body before settling in their **final site** (predilection site) and this is the migratory form of life cycle.

Hepatic-tracheal-enteric route of Nemathelminthes



One of the most common routes is the **hepatic-tracheal-enteric route**. This takes developing stages from the **gut** via the portal system to the **liver** then via the hepatic vein and posterior vena cava to the heart and from there via the pulmonary artery to the **lungs**. Larvae then travel via the bronchi, trachea and esophagus to the **gut**.

C. Classification of Nemathelminthes

The phylum of nematode has two classes:

- Class of Secernentea
- Class of Adenophorea

The class of Secernentea includes Filarial worms, Roundworms, Hookworms and Pinworms

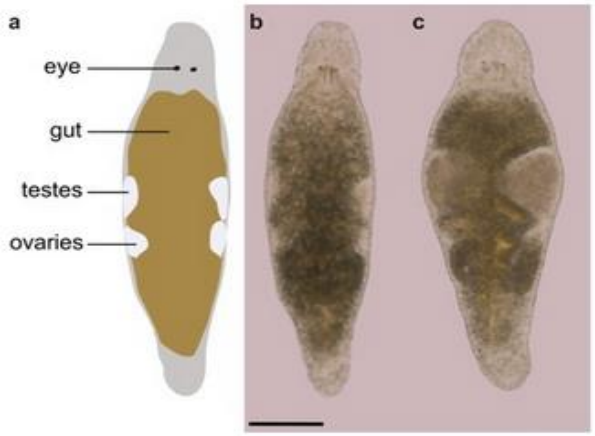
The class of Adenophorea includes Trichinella worms

• Topic 2: Platyhelminthes

A. Morphology of Platyhelminthes

The phylum name Platyhelminthes literally means "flatworms." Members of this phylum are soft, thin-bodied, leaf or ribbonlike worms, including the familiar planaria of ponds and streams, as well as the flukes and tapeworms parasitic in human and other animal bodies.

Some defining characteristics of the phylum are that flatworms are acoelomate (they have no body cavity), triploblastic (the body has three tissue layers), and bilaterally symmetric (they have symmetric right and left sides and usually a definite head), and they have organ systems, including an excretory, digestive, reproductive, and nervous system, but no respiratory system.

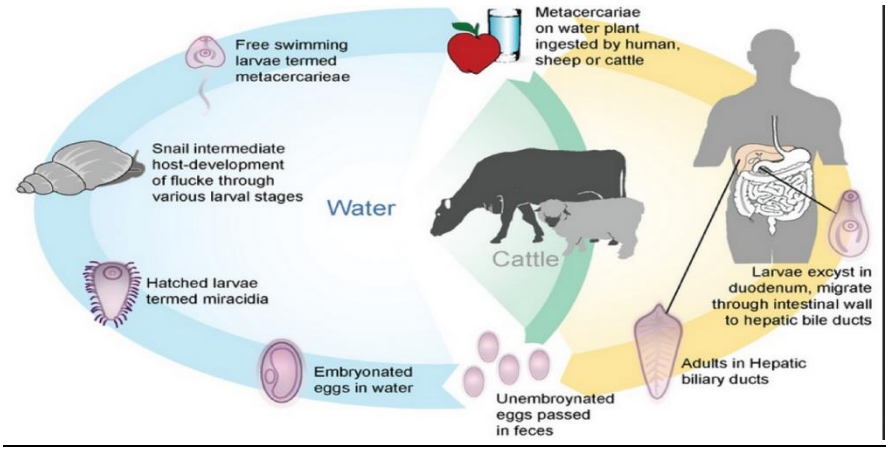


Picture 2: Morphology of plathelminthes

B. General life cycle of Platyhelminthes

The final host of plathelminthes are domestic animals or man. When unembryonated eggs hatched, they reach water bodies and develop into miracidium to infest the intermediate host; water snail and become metacercaria which is the infective form of plathelminthes.

The general life cycle of plathelminthes



C. Classification of Platyhelminthes

The phylum of plathelminthes includes flatworms (Planaria, Flukes and tapeworms).

It has four classes:

- Turbellaria: mostly free living flatworms (commensals)
- Monogenea: parasitic on the skin and gills of fish and amphibians
- Trematoda: all Digenea parasitic called flukes
- Cestoidea Formerly Cestoda: all parasitic tapeworms found in the small intestines of definitive host.

  The only two classes Trematoda and Cestoda have veterinary importance.

C.1. The class Trematoda

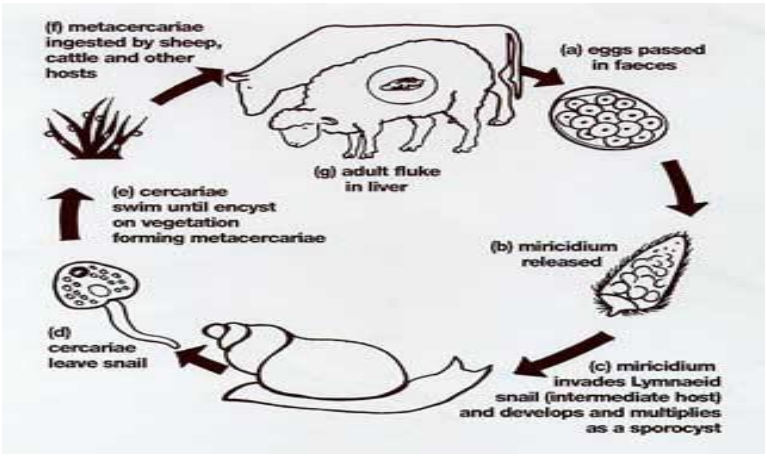
The class Trematoda falls into two main subclasses, the Monogenea, which have a direct life cycle, and the Digenea, which require an intermediate host. The adult digenetic Trematodes, commonly called “flukes” occur primarily in the bile ducts, alimentary ducts and vascular system.

Picture 3: Trematode (*Fasciola hepatica*)

Trematodes (flukes) have small flat leaf-like bodies with oral and ventral suckers and a blind sac-like gut. They do not have a body cavity (acoelomate) and are dorso-ventrally flattened with bilateral symmetry.

Most flukes have a blind alimentary tract, suckers for attachment and are hermaphrodite. There are many families in the class Trematoda, and those include parasite of major veterinary importance are the Fasciolidae, Dicrocoelidae, Paramphistomatidae, and Schistosomatidae.

General life cycle of digenetic Trematodes



The adult flukes are always oviparous and lay eggs with an operculum or lid at one pole. In the egg the embryo develops into a pyriform, ciliated larva called a **Miracidium**.

The cilia allow miracidium to swim in the water seeking out on an aquatic snail, the first intermediate host, then penetrate the skin of the snail and develop to the next stage, the **Sporocyst**, containing a number of germinal cells.

These cells develop into **Rediae** which migrate to the hepato-pancreas of the snail. From the germinal cells of the rediae arise the final stages, the **Cercariae**. The cercariae stage often has a tail and will emerge from the snail and swim in the water.

Depending on the species of the flukes, at this point, the cerariae will take one of the following three paths:

- The cercariae may directly penetrate the skin of the definitive host,
- The cercariae may attach to vegetation, lose its tail, secrete a thick cyst wall around itself, and thus develop into **Metacercariae**
- The cercariae may lose its tail, penetrate the second intermediate host, secrete a thick cyst wall around itself and develop into a **Metacercaria** within the second intermediate host.

The infestation occurs when the definitive host ingests one of the above infective forms:

- If the fluke takes the first option, the cercariae will migrate its predilection site and develop into the adult fluke,
- If the fluke takes the second option, the thick cyst wall will be digested by the host and the juvenile fluke released will migrate to the predilection site and develop into the adult fluke,
- If the fluke takes the third option, the second I. H. and the thick cyst wall are digested and the juvenile fluke released will migrate to the predilection site and then develop into the adult fluke.

The most of the predilection sites of the flukes are associated with the digestive system. The exceptions are *Paragonimus bellicotti*, the lung fluke of dogs and cats, and the *Schistosomes* in the blood vessels

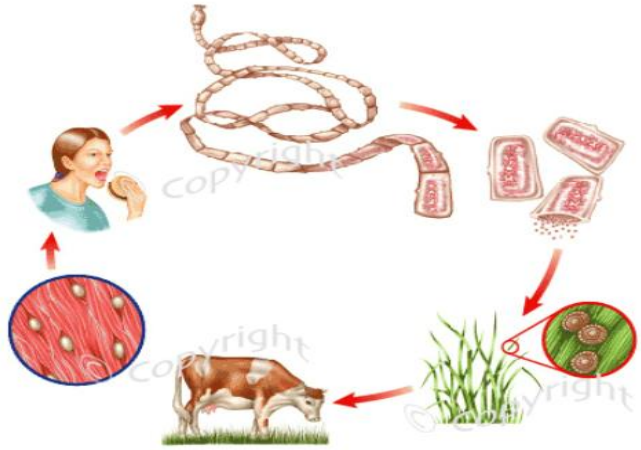
C.2. The class Cestoda

Cestoidea is the class of Platyhelminthes that is known as *tapeworms.* The Class is divided into 18 orders, but only two are of significance to most veterinary practitioners because most of the diverse forms are found in sharks and fish. The two groups of interest to veterinarians are the Cyclophyllidea, which are found mainly in terrestrial vertebrates, and the Diphyllobothriidea, which have aquatic stages as part of their life cycles.

Life cycle of cestodes

The typical life cycle of cestodes is indirect with one intermediate host. With few exceptions, the adult tapeworm is found in the small intestine of the final host, the segments and the eggs reaching the exterior in the feces.

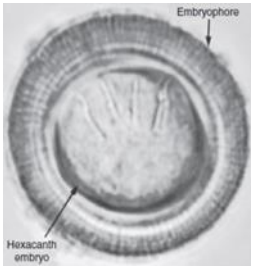
Life cycle of beef tapeworm



The egg of cestodes consists of the following:

- The hexacanth (6 hooked) embryo also known as Onchosphere
- A thick, dark, radially striated "shell" called the embryophore

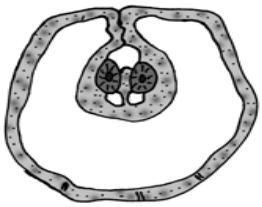
Egg of Cestoda

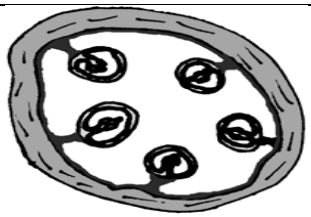
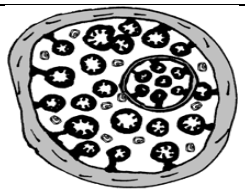
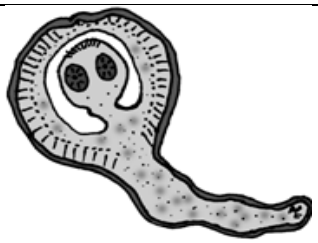
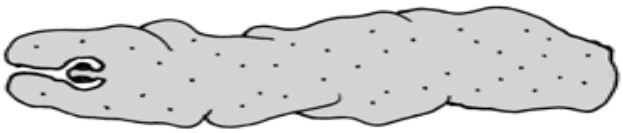


When the egg is ingested by the I H, the gastric and intestinal secretions digest the embryophore and activate the onchosphere. Using its hooks, it tears through the mucosa to reach the blood or lymph stream or, in the case of invertebrates, the body cavity.

Once in its predilection site the onchosphere loses its hooks and develops, depending on the species, into one of the following larval stages described in table below, often known as METACESTODES:

Table 1: Description of Metacestodes

Larval stage of Cestoda	Image of larvae
Cysticercus: is a fluid-filled cyst containing an attached single invaginated scolex, sometimes colled a Protoscolex	

Coenurus: similar to a cysticercus, but with numerous invaginated scolices	
Strobilocercus: the scolex is evaginated and is connected to the cyst by a chain of asexual proglottids	
Hydatid: this is a large fluid-filled cyst lined with germinal epithelium from which are produced invaginated scolices which lie free or in bunches, surrounded by germinal epithelium.	
Cysticercoids: is a single evaginated scolex embedded in a small solid cyst, found in very small I H such as Arthropods	
Tetrathyridium: is a worm-like larva with an invaginated scolex, found only in Mesocestoididae	

- Topic 3: Acanthocephalan

A. Morphology of Acanthocephala

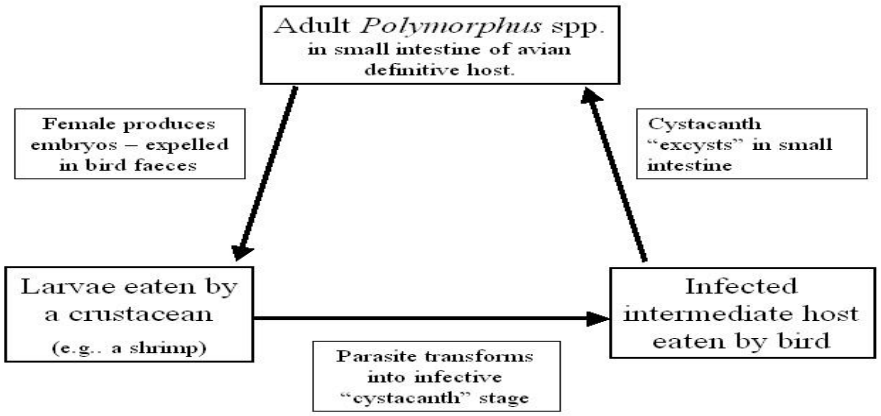
This is a separate phylum, closely related to the Nematoda, which contains a few genera of veterinary importance. They are generally referred to as “tornyheaded” worms due to the presence anteriorly of a hook covered proboscis and most are parasites of alimentary tract of vertebrates. The hallow proboscis armed with recurved hooks, which aid in attachment, is retractable and lies in a sac.

Picture 4: **Macracanthorhynchus**

B. The life cycle of Acanthocephala

The life cycle is indirect involving either an aquatic or terrestrial arthropod intermediate host. On ingestion by the intermediate host, the egg hatches and the acanthor migrates to the haermocoel of the arthropod where it develops to become a cystacanth after 1-3months. The definitive host is infected by ingestion of arthropod intermediate host, and the cystacanth, which is really a young adult, attaches and grows to maturity in the alimentary canal.

The life cycle of *Polymorphus spp.* Acathocephala parasite of birds



C. Classification of Acanthocephala

The phylum Acanthocephala is divided into 3 classes:

- Archiacanthocephala,
- Eocanthocephala and
- Palaeacanthocephala.

Class of Archiacanthocephala:

- Parasites of birds and mammals; insects, centipedes and millipedes act as intermediate hosts.
- Females about 70 cm in length and are 3 to 4 times larger than the males.
- The spines of the proboscis are concentrically arranged.
- Main lacunar canals are median.
- Protonephridia are present.

Examples: Gigantorhynchus, Macracanthorhynchus.

Class of Eoacanthocephala

- Most species parasitize fish, some amphibians and reptiles. Crustaceans serve as intermediate hosts.
- The spines on proboscis are radially arranged.
- Lacurinar canals are median.
- Protonephridia are absent.

Examples: Neoechinorhynchus, Octospimfer.

Class of Palaeacanthocephala

- Definite hosts include fish, amphibians, reptiles, birds and mammals. Crustaceans usually serve as intermediate hosts.
- The spines are alternately arranged on proboscis.
- Lacunnar canals are lateral.
- Protonephridia are lacking.

Examples: Acanthocephalus, Polymorphus.

The major genus of veterinary significance is *Macracanthorhynchus*

L O 1.2 – Describe protozoa

Protozoa are microscopic unicellular eukaryotes that have a relatively complex internal structure and carry out complex metabolic activities. Some protozoa have structures for propulsion or other types of movement. Protozoa, like other eukaryotic cells, have a nucleus, an endoplasmic reticulum, mitochondria and a Golgi body and lysosomes.

In addition, because they lead an independent existence they possess a variety of other subcellular structures or organelles with distinct organizational features and functions.

- Thus locomotion, in the genus *Trypanosoma*, is facilitated by a **single flagellum** and in some other protozoa by several flagella.
- Other protozoa, such as *Balantidium* move by means of **cilia** which are fine, short hairs, each arising from basal body; these cover much of the body surface and beat in unison to effect movement.
- A third means of locomotion, used by protozoa such as *Entamoeba* are **pseudopodia** which are prolongations of cytoplasm. Movement occurs as the rest of the cytoplasm flows into this prolongation.

- Finally some protozoa, such as the extracellular stages of the *Eimeria*, have no obvious means of locomotion, but are nevertheless capable of **grinding** movement.

Classification of protozoa

Classification of the phylum Protozoa is extremely complex. There are four subphyla of protozoa of veterinary importance:

Sarcomastigophora: locomotion by pseudopodia and/or flagella. This subphylum consists of two classes:

- Sarcodina (eg.: *Entamoeba*) for human being
- Mastigophora (eg.: *Trypanosoma*, *Trichomonas*, *Histomonas*)

Sporozoa: locomotion by gliding, lifecycle largely intracellular, and both sexual and asexual phases occur. This subphylum consists of three important classes:

- Coccidia (eg.: *Eimeria*, *Toxoplasma*, *Isospora*)
- Piroplasmidia (eg.: *Babesia*, *Theileria, Anaplasma*)
- Haemosporidia (eg.: *Plasmodium*)

Ciliophora: locomotion by cilia (eg: *Balantidium*)

Microspora: has a little veterinary importance. Intracellular parasites multiplying asexually.(eg: *Encephalitozoon*)

- Topic 1: Theileria

A. Morphology of Theileria

The genus *Theileria* differs from *Babesia* in that schizonts occur in lymphocytes and induce the infected lymphocytes to undergo division and proliferation. Also, typically no transovarial transmission occurs in the case of ticks infected with *Theileria* species.

Theileria parva, the causative agent of East Coast fever of African cattle, occurs in the erythrocytes, lymphocytes, and endothelial cells and is transmitted interstadially by *Rhipicephalus* and *Hyalomma* species. East Coast fever is characterized by dyspnea, emaciation, weakness, tarry feces, and exceptionally heavy mortality. There are vaccines available for preventing disease due to *T. parva* and to *Theileria annulata* in Africa

- Theileria parva: the cause of bovine *Theileriosis* and *East Coast fever*.
- Theileria annulata: the cause of bovine *Theileriosis*.
- Theileria equi: causing equine piroplasmosis.

B. Life cycle of Theileria

Theileria are transmitted via the *Haemaphysalis* and *Rhipicephalus* species of tick vectors. Sporozoites enter mononuclear cells of the host and develop into trophozoites and multinucleate schizonts by asexual reproduction. This process stimulates proliferation of the host cells, allowing further multiplication of the parasite. The local lymph nodes are first infected.

Schizonts then disseminate through the lymphoid tissues before differentiating into merozoites. The merozoites enter the erythrocytes and form piroplasms which are infective to ticks and capable of sexual reproduction.

C. Classification of Theileria

Theileriae are obligate intracellular protozoan parasites phylum Apicomplexa, order Piroplasmida, family Theileriidae, genus Theileria. They are most closely related to Babesia, from which they differ by having a developmental stage in leukocytes prior to infectionof erythrocytes.

This agent infects both wild and domestic Bovidae throughout much of the world and some species also infect small ruminants.

There are several identified *Theileria spp.* that infect cattle; the most pathogenic and economically important are *T. parva,* which causes East Coast fever(ECF), *T. annulata,* which causes Tropical theileriosis (TT) or Mediterranean theileriosis and *T. orientalis* (*T. orientalis*/buffeli group), which causes Oriental theileriosis (OT) or Theileria-associated bovine anaemia (TABA).

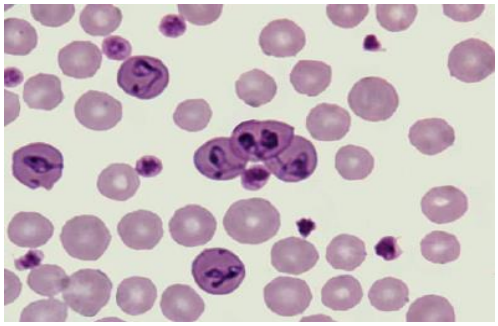
Theileria lestoquardi (*T. hirci*), which causes Malignant ovine theileriosis (MOT), *Theileria uilenbergi* and *Theileria luwenshuni* are the most pathogenic species of economic significance infecting small ruminants.

- Topic 2: Babesia

A. Morphology of Babesia

Babesia species are apicomplexan parasites of the erythrocytes of their vertebrate hosts; the erythrocyte is the only vertebrate host cell infected.

Typically, *Babesia* stages that are seen in red blood cells are large compared with the *Theileria* species, which occur in the same host, but unfortunately, there are exceptions. For members of the genus *Babesia*, sexual conjugation occurs within the intestinal lumen of the tick, and sporogony occurs within the epithelium of the tick's intestinal wall.



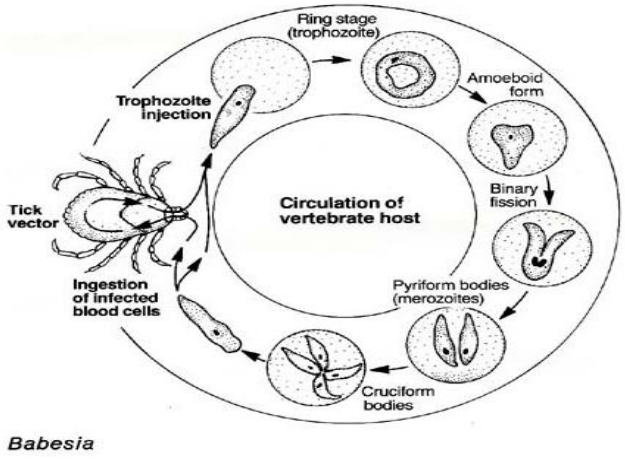
Picture 5: *Babesia bigemina* in Giemsa-stained blood film from a cow

B. Life cycle of Babesia

When an infected tick feeds on a vertebrate host, the Babesia parasite enters the host in trophozoite ("ring") form. The trophozoites invade the host's red blood cells, where they multiply through binary fission. It is here that the merozoite form of Babesia wreaks the most havoc on the host, as it destroys red blood cells and causes anemia.

Uninfected ticks ingest the vertebrate's blood when feeding, and the merozoitic parasites settle in the midgut. After multiplying in the vector's gut, Babesia migrates to the salivary glands, ready to be spread to a new vertebrate host. Like all apicomplexans, Babesia's cells invade the host's red blood cells by way of the apicoplast, an organelle unique to members of this phylum

The life cycle of Babesia



C. Classification of Babesia

Babesia are also obligate intracellular protozoan parasites phylum Apicomplexa, order Piroplasmida, family Babesiidae, genus Babesia. They are most closely related to Theileria.

Spicies

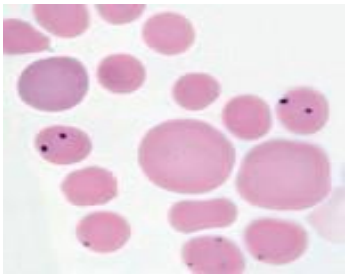
Babesia bicornis, B. bigemina, B. bovis, B. caballi, B. canis, B. divergens, B. duncani, B. equi, B. felis, B. microti, B. cf. microti, B. motasi, B. muratovi, B. odocoilei, B. orientalis, B. ovata, B. ovis.

- Topic 3: Anaplasma

A. Morphology of Anaplasma

Anaplasma is a genus of bacteria of the alphaproteobacterial order Rickettsiales, family Anaplasmataceae. Anaplasma species reside in host blood cells and lead to the disease anaplasmosis. The disease most commonly occurs in areas where competent tick vectors are indigenous, including tropical and semitropical areas of the world for intraerythrocytic Anaplasma spp.

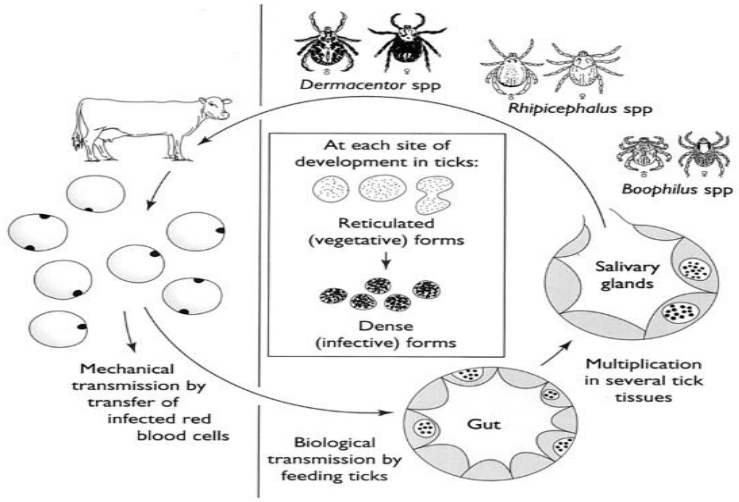
The dense forms were eventually released from the cultured cells by a process in which the inclusion membrane fused with the host cell membrane. Release of A. marginale was effected without the loss of host cell cytoplasm. In subsequent cell cycles, A. marginale reinfected cultured cells resulting in the development of multiple colonies per cell and eventual host cell destruction. Small vesicles were abundant within the colonies and appeared to form from individual rickettsiae.



Picture 6: ***Anaplasma Marginal* in infected erythrocytes**

B. Life cycle of Anaplasma

Anaplasma species are biologically transmitted by Ixodes deer-tick vectors, and the prototypical species, *A. marginale*, can be mechanically transmitted by biting flies and iatrogenically with blood-contaminated instruments. One of the major consequences of infection by bovine red blood cells by *A. marginale* is the development of nonhaemolytic anaemia, thus the absence of hemoglobinurea, which allows clinical differentiation from another major tick-borne disease, bovine babesiosis, caused by *Babesia bigemina*.



C. Classification of Anaplasma

Species of veterinary interest include:

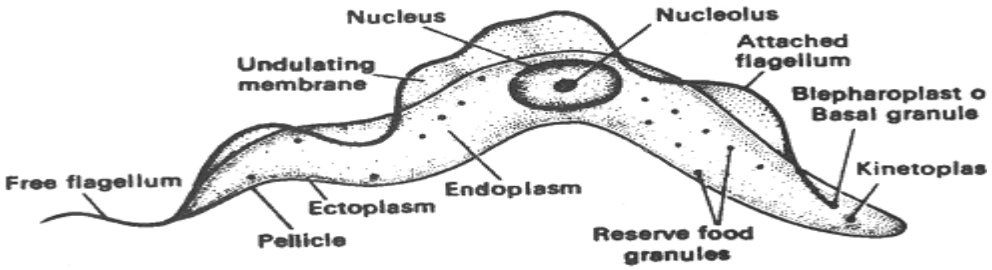
- *Anaplasma marginale* and *Anaplasma centrale* in cattle
- *Anaplasma ovis* and *Anaplasma mesaeterum* in sheep and goats[2]
- *Anaplasma phagocytophilum* in dogs, cats, and horses (see human granulocytic anaplasmosis)
- *Anaplasma platys* in dogs

Topic 4: Trypanosoma

A. Morphology of Trypanosoma

A trypanosome is an elongated, spindle-shaped cell with a single nucleus lying near the middle of its length and a single flagellum that arises near a large mitochondrion with copious DNA called a kinetoplast and passes out of the anterior end of the cell.

A Trypanosome

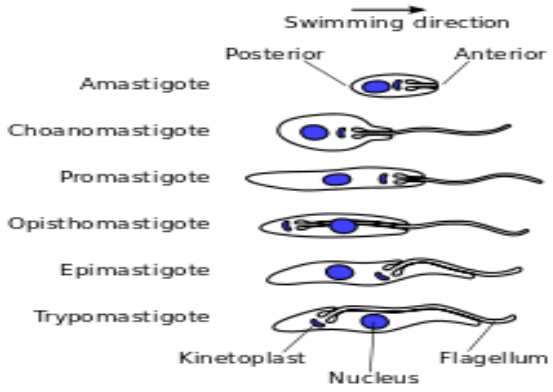


B. Life cycle of Trypanosoma

During development in both mammalian and arthropod hosts, trypanosomes can undergo considerable morphologic change.

Sexual reproduction occurs within the nymph and larval stages of the tick and the final infective stage is present within the salivary glands and is transmitted to mammalian hosts when blood feeding. Transmission in the tick is then trans-stadial.

Development stages of Trypanosoma



Two different types of trypanosomes exist, and their life cycles are different, the salivarian species and the stercorarian species. Salivarian trypanosomes develop in the anterior gut of insects, most importantly the Tsetse fly, and infective organisms are inoculated into the host by the insect bite before it feeds.

As trypanosomes progress through their life cycle they undergo a series of morphological changes as is typical of trypanosomatids. The life cycle often consists of the trypomastigote form in the vertebrate host and the trypomastigote or promastigote form in the gut of the invertebrate host. Intracellular lifecycle stages are normally found in the amastigote form. The trypomastigote morphology is unique to species in the genus Trypanosoma.

C. Classification of Trypanosoma

Trypanosoma is a genus of kinetoplastids (class Trypanosomatidae), a monophyletic group of unicellular parasitic flagellate protozoa. Trypanosoma is part of the phylum Sarcomastigophora.

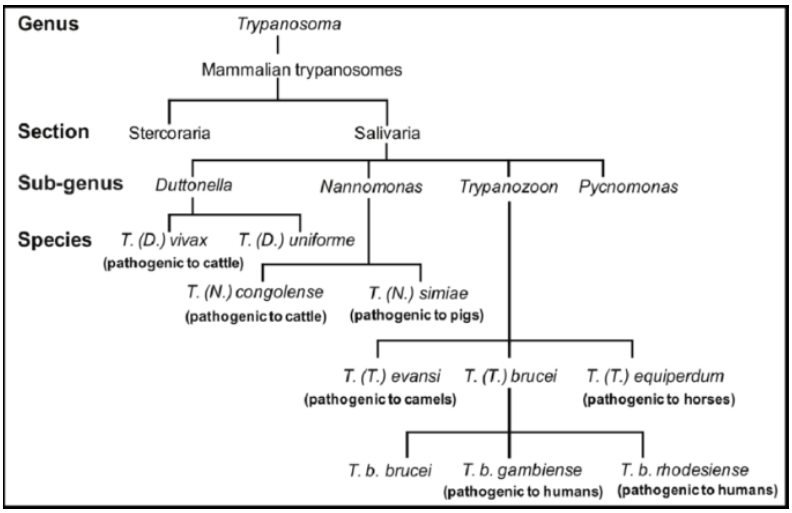
The trypanosomes are passed to the recipient in the saliva of the tsetse fly (Glossina spp.). Antigenic variation is a characteristic shared by the Salivaria, which has been particularly well-studied in T. brucei.

Other species of veterinary importance are:

- *T. avium,* which infects birds and blackflies
- *T. bennetti,* which infects birds and biting midges
- *T. brucei,* which causes sleeping sickness in humans and nagana in cattle
- *T. cruzi,* which causes Chagas disease in humans

- *Trypanosoma culicavium*, which infects birds and mosquitoes
- *T. congolense,* which causes nagana in ruminant livestock, horses and a wide range of wildlife
- *T. equinum*, in South American horses, transmitted via Tabanidae,
- *T. equiperdum,* which causes dourine or covering sickness in horses and other Equidae, it can be spread through coitus.
- *T. melophagium*, in sheep, transmitted via Melophagus ovinus
- *T. simiae,* which causes nagana in pigs. Its main reservoirs are warthogs and bush pigs
- *T. sinipercae*, in fishes
- *T. theileri,* a large trypanosome infecting ruminants
- *T. vivax,* which causes the disease nagana, mainly in West Africa, although it has spread to South America.

Classification of Trypanosoma

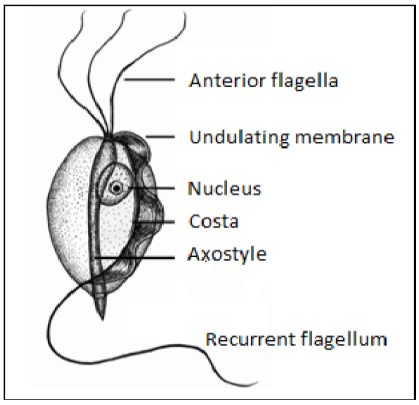


- Topic 5: Trichomonas

A. Morphology of Trichomonas

Trichomonas foetus is found in the vagina, uterus, macerated fetus, prepuce, penis, epididymis, and vas deferens. The organism varies from 10 to 25 µm in length, and has three anterior flagella and a long, trailing flagellum that extends beyond the undulating membrane.

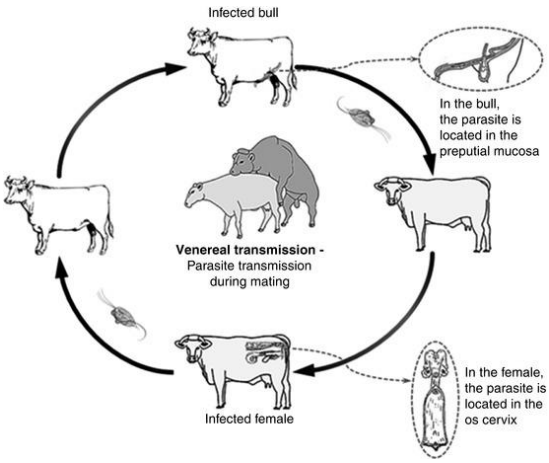
Trichomonas foetus



B. Life cycle of Trichomonas

Trichomonas has a trophozoite form, its pear-shaped form, which is most commonly observed, and an amoeboid form, which appears during host colonization. In its trophozoite form, Trichomonas undergoes cell division through an interesting process called cryptopleuromitosis. There are three common forms of mitosis: open, closed, and semi-open. In open mitosis, the nuclear envelope disappear so that mitotic spindles can interact with the chromosomes. In closed mitosis, the nuclear envelope does not disappear but mitotic spindles appear within the nucleus to separate the chromosomes. In semi-open mitosis, the nuclear envelope remains intact but the mitotic spindles pierce through the nuclear envelope to divide the chromosomes.

The life cycle of Trichomonas



C. Classification of Trichomonas

Trichomonas is a genus of anaerobic excavate parasites of vertebrates. It was first discovered by Alfred François Donné in 1836 when he found these parasites in the pus of a patient suffering from vaginitis, an inflammation of the vagina. Donné named the genus from its morphological characteristics.

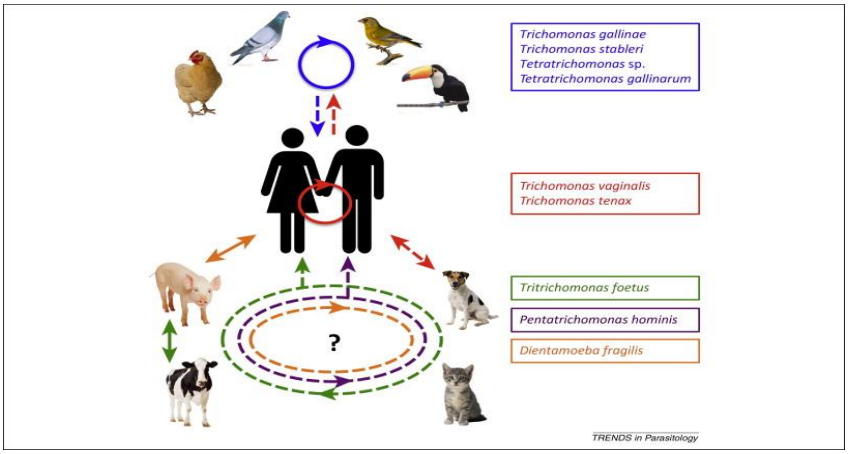
The prefix tricho- originates from the Ancient Greek word θρίξ (thrix) meaning hair, describing Trichomonas's flagella. The suffix-monas (μονάς – single unit), describes its similarity to unicellular organisms from the genus Monas. This is the classification of Trichomonas:

- Phylum: Metamonada
- Subphylum: Trichozoa
- (unranked): Parabasalia
- Order: Trichomonadida
- Family: Trichomonadidae
- Genus: Trichomonas

The following are the species of Trichomonas:

- *Trichomonas brixi*: inhabits the oral cavity of dogs and cats.
- *Trichomonas gallinae*: inhabits the upper digestive tract of primarily pigeons and doves, but also other birds. *Trichomonas gypactinii*: inhabits the upper digestive tract of scavenging birds of prey, such as vultures. *Trichomonas stableri*: inhabits the upper digestive tract of pigeons.
- *Trichomonas tenax*: inhabits the oral cavity of humans.
- *Trichomonas vaginalis*: inhabits the urogenital tract of humans
- *Trichomonas Foetus*: Inhabits in the vagina and prepuce of cattle

Different species of Trichomonas and their habitats.

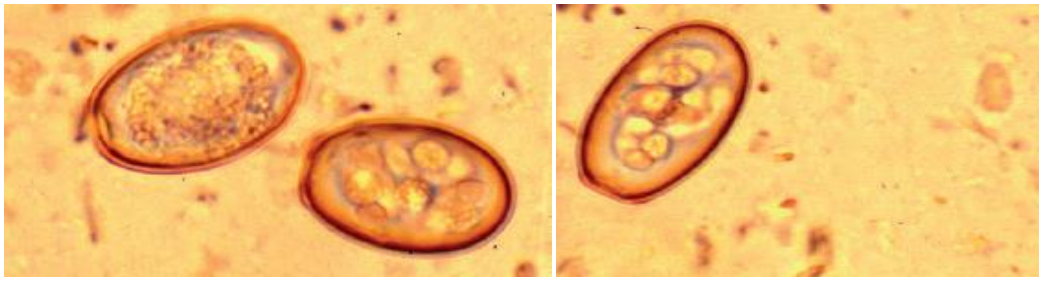


- Topic 6: Coccidia

A. Morphology of Coccidia

Eimeria, genus of Coccidia is characterized by the presence of oocysts that when sporulated have four sporocysts, each with two sporozoites.

The wall of the oocyst is often brownish in color, and many species have a polar cap. Members of this genus all have direct life cycles. Thus, the general form of the direct coccidian life cycle is represented by the genus *Eimeria*, species of which include gastrointestinal parasites of a wide range of vertebrate hosts.

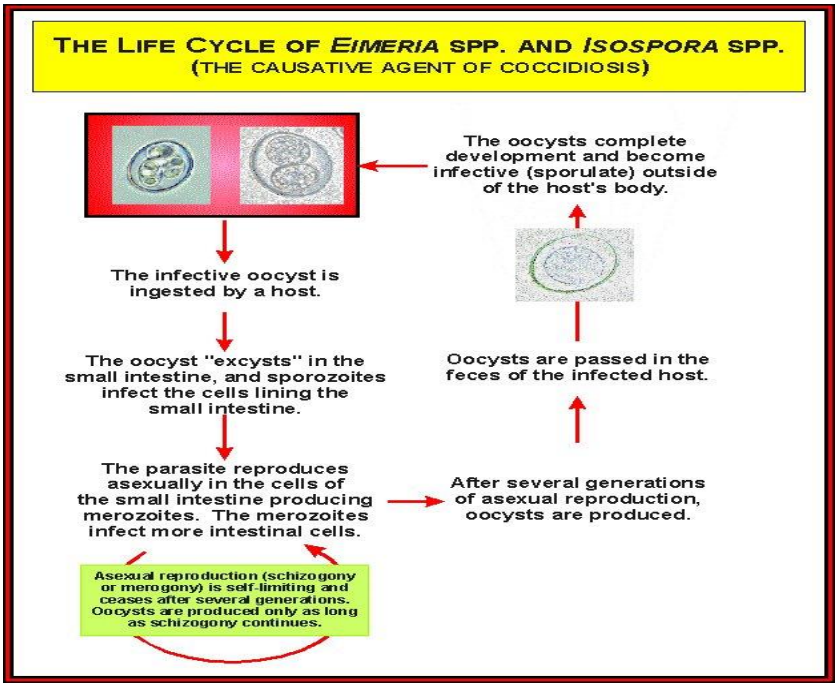


Picture 7: **Eimeria magna** ***oocysts, sporulated, from the feces of a domestic rabbit***

B. Life cycle of Coccidia

Coccidia are transmitted mainly by fecal contamination and reproduce through rigid sequences of asexual and sexual phases of multiplication and development that, in an important minority of cases, require an alternation of hosts.

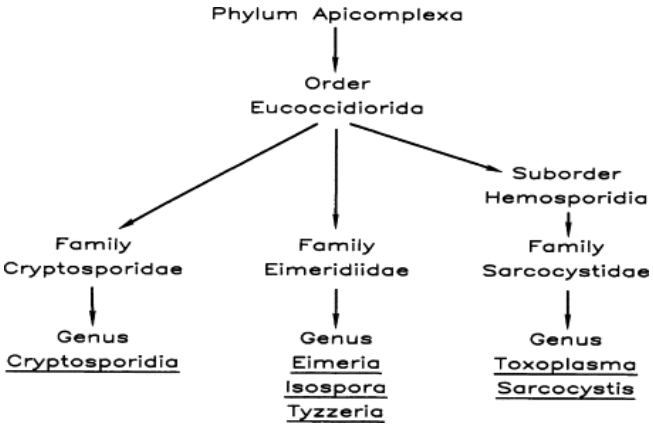
Life cycle of Coccidia a typical *Eimeria* and *Isopora* species.



C. Classification of Coccidia

Coccidia (Coccidiasina) are a subclass of microscopic, spore-forming, single-celled obligate intracellular parasites belonging to the apicomplexan class Conoidasida.

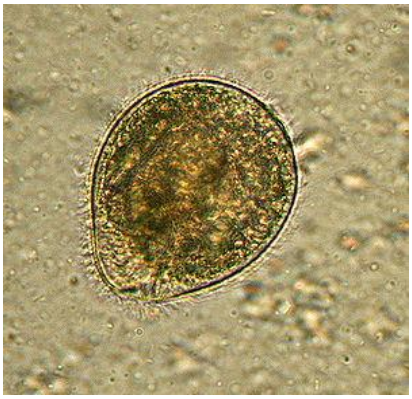
As obligate intracellular parasites, they must live and reproduce within an animal cell. Coccidian parasites infect the intestinal tracts of animals, and are the largest group of apicomplexan protozoa. Most species of coccidia are species-specific in their host. An exception is Toxoplasma gondii, which can infect all mammals.



- Topic 7: Balantidia

A. Morphology of Balantidia

Balantidia are cysts in wet mounts. Trophozoites are characterized by: their large size (40 µm to 200 µm), the presence of cilia on the cell surface, a cytostome, and a bean shaped macronucleus which is often visible and a smaller, less conspicuous micronucleus.



Picture 8: Cyst of *Balantidium Coli* with cilia

B. Life cycle of Balantidia

Balantidium coli has 2 developmental stages: a **trophozoite** stage and a **cyst** stage. Trophozoites live in the large intestines of the host animals. They are covered in cilia and have boring or rotary motility. The cyst is the infective stage of the Balantidium coli life cycle.

Encystation is the process of forming the cyst; this event takes place in the rectum of the host as feces are dehydrated or soon after the feces have been excreted. Excystation produces a trophozoite from the cyst stage, and it takes place in the large intestine of the host after the cyst has been ingested.

The life cycle of *Balantidium Coli*

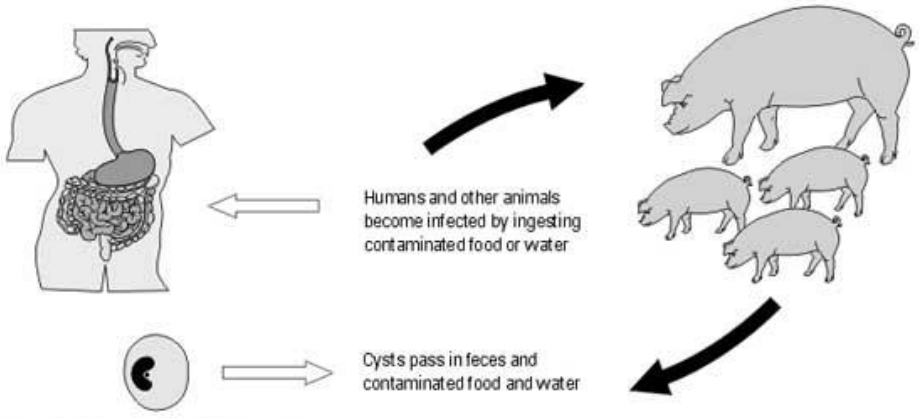


Figure 1. Life cycle of Balantidium coli

C. Classification of Balantidia

The Family of Balantidiidae. These ciliates are monoxenous (one-host) endocommensals in vertebrates, some species of which can become histophagous parasites. The trophozoites have a uniform covering of somatic ciliary rows and a cytostome at the base of an anterior vestibulum.

It includes the species *Balantidium coli*: this species causes balantidiasis in vertebrates, especially in pigs and humans.

L O 1.3 – Describe mites

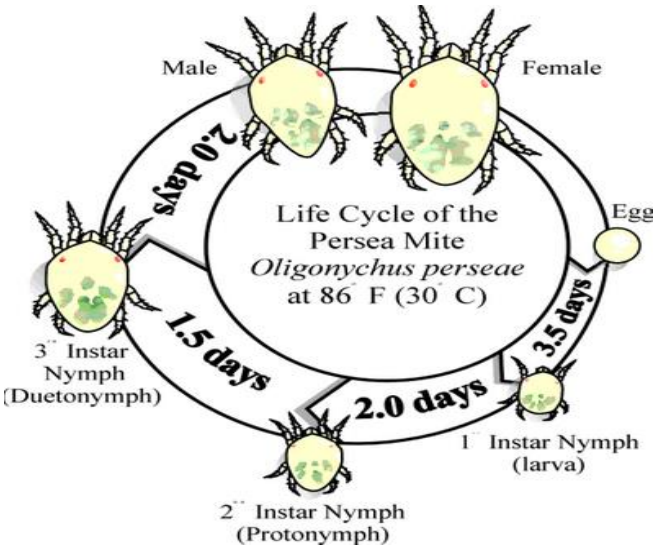
The phylum Arthropoda contains over 80% of all known animal species and consists of invertebrates whose major characteristics are a hard chitinous exoskeleton, a segmented body and jointed limbs. There are two major classes of arthropods of veterinary importance, namely the *Insecta* and *Arachnida*. These two major classes can be differentiated by the following general characteristics:

- **Insecta**: these have three pairs of legs, the head, thorax and abdomen are distinct, and they have a single pair of antennae. This class contains the following important orders: *Diptera* (flies), *Phthiraptera* (lice) and *Siphonaptera* (fleas).
- **Arachnida**: the adults have four pairs of legs, the body is divided into a cephalo-thorax and abdomen, and there are no antennae. These are grouped in the *Acarina* order (mites and ticks).

The parasitic mites are small, most being less than 0.5mm long, though a few blood-sucking species may attain several mm when fully engorged. With few exceptions they are in prolonged contact with the skin of the host, causing various forms of the condition generally known as **mange**.

Although, like the ticks, mites are obligate parasites, they differ from them in the important respect that most species spend their entire life cycles, from egg to adult, on the host so that transmission is mainly by contact.

General life cycle of mites



General classification of Mites

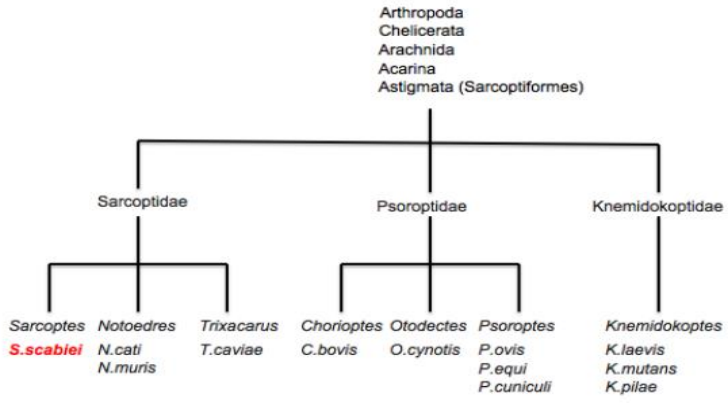
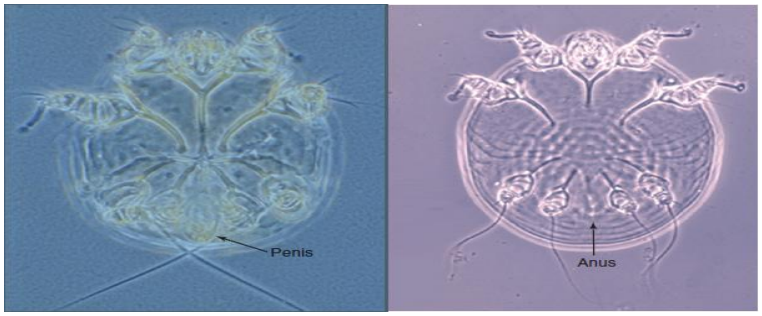


Figure 1. Simplified classification of mites of veterinary importance

- Topic 1: Sarcoptes

A. Morphology of Sarcoptes

The pretarsi have long, unsegmented pedicels, and the anus is at the posterior edge of the body. *Sarcoptes scabiei* causes sarcoptic mange or scabies of humans, dogs, foxes, horses, cattle, and others. Sarcoptic mange of cattle is reportable.



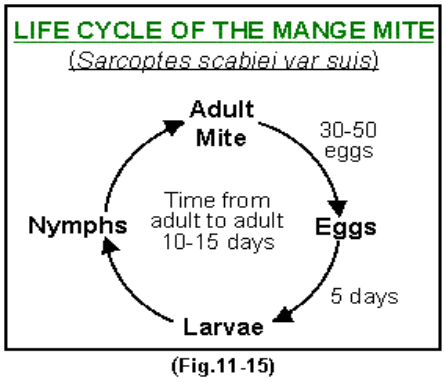
Picture 9: *Sarcoptes* male *(left)* and female *(right)*.

B. Life cycle of Sarcoptes

The scabies mite *Sarcoptes scabiei* goes through four stages in its lifecycle: egg, larva, nymph, and adult.

Upon infesting a human host, the adult female burrows into the stratum corneum (outermost layer of skin), where she deposits two or three eggs per day. These oval eggs are 0.1–0.15 mm (0.0039–0.0059 in) long and hatch as larvae in three to four days. A female can lay up to 30 eggs, then dies at the end of a burrow. Upon hatching, the six-legged larvae migrate to the skin surface and then burrow into molting pouches, usually into hair follicles, where vesicles form (these are shorter and smaller than the adult burrows). After three to four days, the larvae molt, turning into eight-legged nymphs.

This form molts a second time into slightly larger nymphs, before a final molt into adult mites. Adult mites then mate when the male penetrates the molting pouch of the female. Mating occurs only once, as that one event leaves the female fertile for the rest of her life (one to two months). The impregnated female then leaves the molting pouch in search of a suitable location for a permanent burrow. Once a site is found, the female creates her characteristic S-shaped burrow, laying eggs in the process. The female will continue lengthening her burrow and laying eggs for the duration of her life



(Fig.11-15)

C. Classification of Sarcoptes

Sarcoptes scabiei is a burrowing mite and the causative agent of sarcoptic mange.

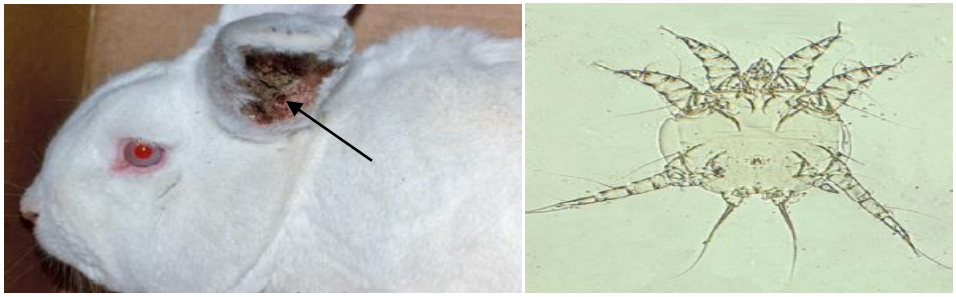
Mites of the genus Sarcoptes are generally considered to be one species, S. scabiei, but are often further identified by a variety name corresponding to the host species (e.g., S. scabiei var. cuniculi). The organisms are commonly referred to as itch or scab mites.

- Topic 2: Psoroptes

A. Morphology of Psoroptes

The legs are long, and the pretarsi have long, three-segmented pedicels. *Psoroptes ovis* causes a very serious and reportable form of mange (scabies or "scab") in cattle, sheep, and horses. *Psoroptes cuniculi* is very common and causes ear canker in rabbits and a less severe form of otic acariasis in goats and horses.

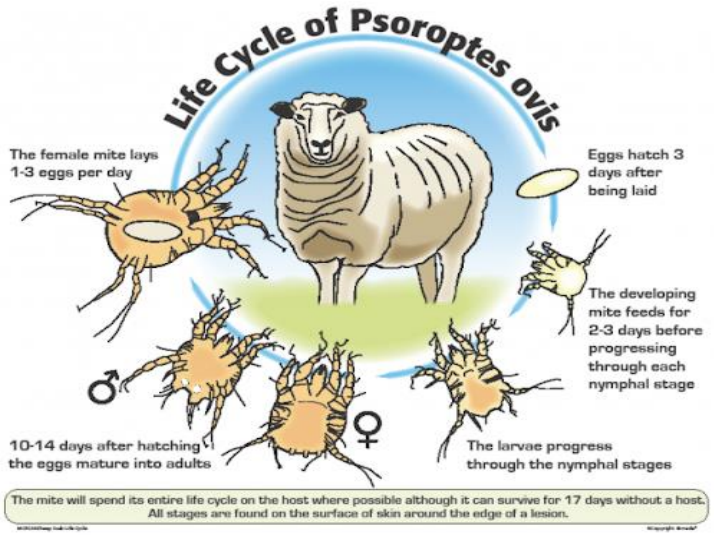
Psoroptes ovis does not burrow in the epidermis but remains at the base of the hairs and pierces the skin with its stylet-like chelicerae. This manner of feeding results in exudation of serum, which hardens to form a scab.



Picture 10: Psoroptic mange on a rabbit *(left)* and an adult *Psoroptes (right).*

B. Life cycle of Psoroptes

Psoroptes ovis. In sheep, live at the base of the fleece and feed on skin exudates. The life cycle is approximately 11 to 19days from egg to egg under optimal conditions, and adult mites can live for 40 days. P. ovis is highly contagious, and a single gravid female mite can establish an infestation. The Mite will spends its entire life cycle on the host where possible although it can survive for 17 days without host. All stages are found on the surface of the skin around the edge of a lesion.



C. Classification of Psoroptes

- Kingdom: Animalia
- Phylum: Arthropoda
- Class: Arachnida
- Subclass: Acari
- Order: Sarcoptiformes
- Family: Psoroptidae
- Genus: Psoroptes

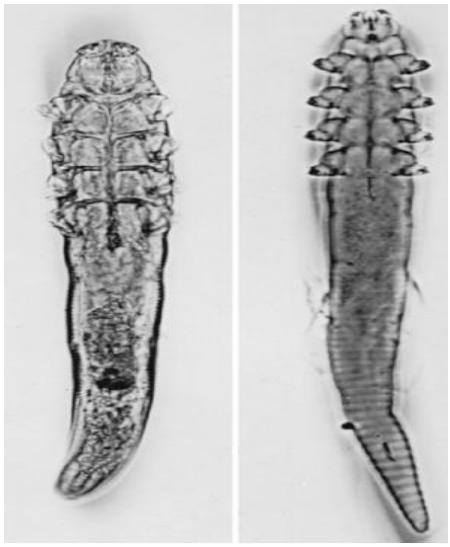
The mites used in the present study were obtained from the ears of rabbits, and had previously been described as *Psoroptes cuniculi*. Therefore *P. ovis, P. cervinus, P. equi, P. cervinus* and *P. natalensis*), are identified primarily on the basis of their localization and their hosts.

Psoroptes has been traditionally considered to include five species living on different host species, but genetic analysis has reduced the genus to a single species, *Psoroptes ovis*.

• Topic 3: Demodex

A. Morphology of Demodex

These tiny, wormlike mites with short, stubby legs live in the hair follicles and sebaceous glands of mammals. Several distinct species of *Demodex* often parasitize the same host animal, but each species tends to be restricted to a particular habitat.

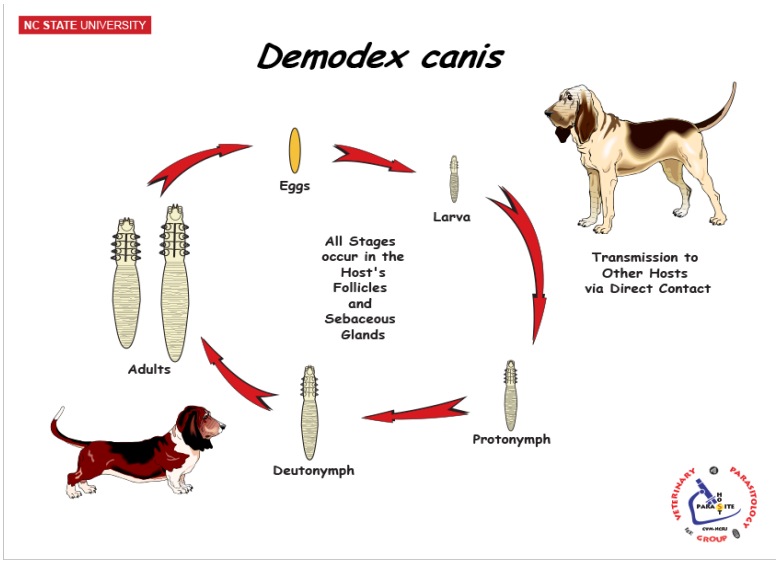


Picture 11: Demodex canis (left) and Demodex cati (right).

B. Life cycle of Demodex

The typical Demodex life cycle is usually 2 to 3 weeks. A female Demodex mite lays 15 to 20 eggs inside the hair follicle near the sebaceous glands. The eggs develop into larvae, which eventually become an adult eight-legged mite. The adult male Demodex mite will leave the follicle in search of a mate, while the adult female mite remains in the follicle. The mites are capable of walking approximately 10 mm/h and tend to be more active in the dark.

The life cycle of *Demodex canis*



C. Classification of Demodex

Class: Arachnida

Order: Prostigmata

Family: Demodecidae

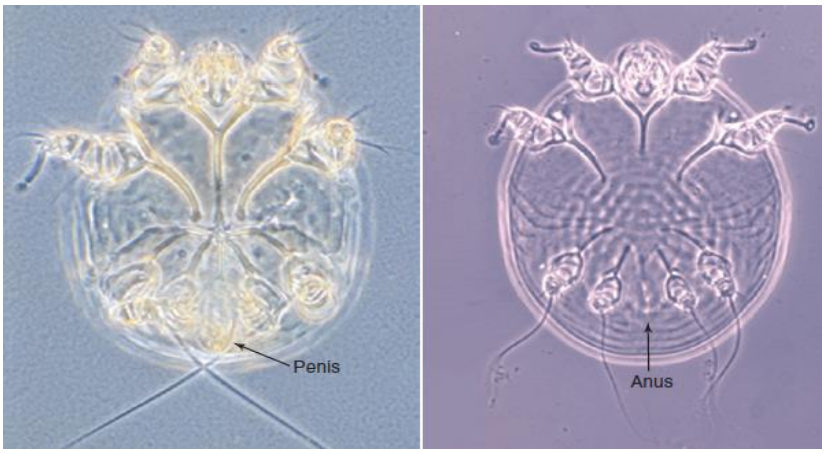
Genus: Demodex

- Species Demodex bovis: Cattle
- Species Demodex canis: Dog
- Species Demodex caprae: Goat
- Species Demodex cati: cat
- Species Demodex equi: Equidea
- Species Demodex folliculorum: Human
- Species Demodex ovis: Sheep

• Topic 4: Notoedres

A. Morphology of Notoedres

A parasite of cats, rats, rabbits, and occasionally and temporarily of humans, *Notoedres* much resembles *Sarcoptes* in that the pretarsi have long, unsegmented pedicels, but it is smaller and its anus is on the dorsal surface instead of on the posterior margin of the body.



Picture 12: Notoedres male *(left)* and female *(right)*.

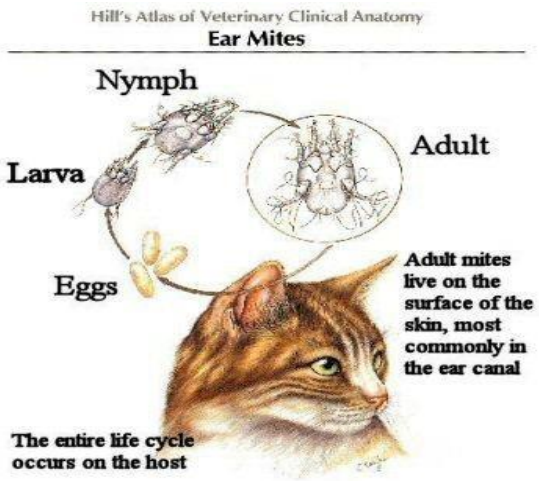
B. Life cycle of Notoedres

The life cycle of Notoedres is not clearly understood. Ova are deposited within epidermal tunnels. These ova hatch into six-legged larvae within 3–4 days. These larvae undergo several more molts, transforming into eight-legged nymphs in approximately a 3-day period.

These nymphs continue to molt as they burrow within the skin, eventually becoming adults. The entire cycle is estimated to take 10–20 days. All three mobile life stages can be found on the skin.

Epidermal burrowing by mites causes pruritus and increases potential for secondary bacterial infections following mechanical damage to the skin by mites and the host's attempts to alleviate discomfort through scratching.

The life cycle of *Notoedres cati*



C. Classification of Notoedres

- Class: Arachnida
- Order: Sarcoptiformes
- Family: Sarcoptidae
- Genus: Notoedres

The genus of Notoedres has only two spices of veterinary importance:

- *Notoedres cati*
- *Notoedres muris*

• Topic 5: Chorioptes

A. Morphology of chorioptes

Pretarsi of *Chorioptes bovis* have short, unsegmented pedicels on the first, second, and fourth pairs of legs of the female and on all legs of the male; the male has two turret-like lobes on the posterior margin of the body.

Picture 13: *Chorioptes* male *(left)* and female *(right).*

The female has pretarsi on the first, second, and a fourth pair of legs and the males have pretarsi on all four pairs.

B. Life cycle of Chorioptes

Female mites lay eggs on skin surface: Females can lay 15-20 eggs. Eggs hatch after 5-6 days into hexapod larvae. First nymph turns into second octopod protonymph stage; followed by tritonymph and adult. The whole life cycle takes about 3 weeks.

Transmission by direct contact with an infected animal. Can also be transmitted in contaminated bedding and housing.

C. Classification of Chorioptes

- Class: arachnida.
- Sub-class: acari.
- Order: astigmata (Sarcoptiformes).
- Family: psoroptidae.
- Genus: Chorioptes

There are different species of veterinary importance:

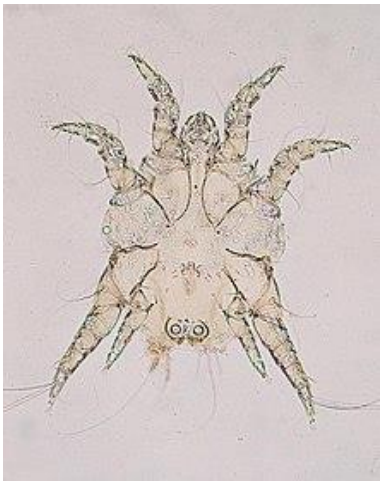
- Chorioptes equi and Chorioptes cuniculi are mites of equine and rabbits respectively.
- *Chorioptes spp.* are skin mites that cause mange in domestic and wild animals. These mites are commonly found in herbivorous hosts, including cattle, sheep, goats, horses, camelids and moose.
- *Chorioptes ovis* for Sheep, *Chorioptes caprae* for goat. Chorioptic mange appears to be the only common mange mite in small ruminants.
- *Chorioptes texanus* is considered to be relatively non-host specific and capable of infesting a wide range of wild cervids and domestic livestock.

- *bovis* of cattle is of the greatest importance because can infest other domestic livestock.

- Topic 6: Psorergates

A. Morphology of Psorergates

Psorergates is a genus of itch mites (family Cheyletidae) parasitic in cattle, sheep, and goats. These are a small, shallow cup on a long stalk. Psorergatic mites as adults are just large enough to see with the naked eye and can crawl readily using long legs.



Picture 14: Psorergates

B. Life cycle of Psorergates

The life cycle of Psororgatos ovis is completed in 5-6 weeks, and there are six stages: the egg, larva, protonymph, doutonymph, tritonymph, and the male or female adult. All stages are found under the stratum corneum, but adults and occasional tritonymphs are also found on the surface of the skin. Only the adult is motile and is responsible for the spread of infestation over the body of sheep. Adult mites transferred most readily to contact, materials and bedings which were placed next to the skin, which indicated that they primarily are responsible for transference of infestation from sheep to sheep.

Classification of Psorergates

This family of Psorergatidae includes the species *Psorergates bovis* which infests cattle and *Psorergates ovis* which infests sheep. They are similar in appearance to species in the Psoroptidae and infest the skin in a similar way, feeding superficially.

Learning Unit 2 – Identify common parasitic diseases

L O 2.1 – Characterize helmenthiasis

- Topic 1: Nemathelminthiasis

A. Ascaridiosis

Definition

The ascaridoids responsible of ascaridiosis are among the largest nematodes and occur in most domestic animals, both larval and adult stages being of veterinary importance. While the adults in the intestine may cause unthriftiness in young animals, and occasional obstruction, an important feature of this group is the pathological consequences of the migratory behavior of the larval stages.

Etiology

Table 2: Genera of Ascardoids and their respective definitive hosts

Genus	Species	Definitive hosts
Ascaris	*Ascaris suum*	Pig
Toxocara	*Toxocara canis* *Toxocara cati* *Toxocara vitulorum*	Dogs Cat Cattle
Toxascaris	*Toxascaris leonine*	Dog and Cat
Parascaris	*Parascaris equirum*	Horses and Donkey
Ascaridia	*Ascaridia galli* *Ascaridia dissimils* *Ascaridia columbae*	All birds Turkeys Pigeons
Heterakis	*Heterakis gallinarum*	Domestic and wild birds

A.1. Ascariasis

Host: Pigs

Site: Small intestine

Species: *Ascaris suum*

Distribution

Worldwide

Mode of transmission

The life cycle of *Ascaris suum* is direct. The single preparasitic moult occurs at about 3 weeks after the egg is passed, a period of maturation is necessary and it is not infective until a minimum of four weeks after being passed. The egg resists to extremes temperature and is viable for more than four years.

After infection, the egg hatches in the small intestine and L2 travels to the liver where the first parasitic moult takes place. The L3 then passes in the bloodstream to the lungs and hence to the small intestine via trachea and the final two further parasitic moults occur.

The eggs are ingested by an earthworm or dung beetle they will hutch, and the L2 to the tissues of these paratenic hosts, where they can remain fully infective for pig for a long period.

Pathogenesis

The migrating larval stages in large numbers may cause transient pneumonia, in the liver the migrating L2 and L3 can cause "**milk spot**".

The adult worms in the intestine cause little apparent damage to the mucosa, but occasionally if large numbers, there may be obstruction, and rarely a worm may migrate into the bile duct causing obstructive icterus and carcass condemnation.

Clinical signs

- The main effect of the adult worms is to cause production loss in term of diminished weight gain
- General clinical signs are absent except in the occasional case of intestinal or biliary obstruction
- Clinically evident transient pneumonia in piglets of fewer than 4 months old.

Epidemiology

Partial age immunity operates in pigs (over 4 months of age). The main source of infection is the highly resistant egg on the ground. *Ascaris suum* may occasionally infect cattle causing an acute, atypical, interstitial pneumonia, which may prove fatal.

Young adults of *Ascaris suum* are occasionally found in the small intestine of sheep. There are a few recorded cases of patent *Ascaris suum* infection in man.

Diagnosis

Diagnosis of clinical ascarosis frequently depends on clinical and necropsy findings because the main pathologic events occur during the prepatent stage.

Clinical signs of severe respiratory distress in a group of growing pigs and the discovery of extensive petechial and ecchymotic pulmonary hemorrhages and edema contribute to a diagnosis of acute ascarosis.

Pieces of lung tissue should be minced and placed in a Baermann apparatus for demonstration of the migrating larvae.

Treatment

- Benzimidazoles for intestinal ascariosis
- Levamizole and Avermectin in case of ascaris pneumonia

Control

- In housed pigs, strict hygiene in feeding and bedding with frequent hosing of walls and floors
- In pig of free range, it is necessary to discontinue the use of paddocks for several years
- It is good to treat in pig sows at the entry to the farrowing pen and young pigs should receive anthelmintic treatment when purchased or on the entry to the finishing house.
- Boars should be treated every 3-6 months

A.2. Toxocara

Through the member of this genus are in many respects typical Ascardoids, their biology is sufficiently varied for it to be necessary to consider each species separately.

Etiology

- Toxocara cati: cat
- Toxocara canis: dogs
- Toxocara viturolum: cattle and buffalo

Toxocara canis

Apart from its veterinary importance, this specie is responsible for the most widely recognized from of visceral larva migrans in man.

- **Host**: dogs
- **Site**: small intestine

Distribution:

Worldwide

Identification

Toxocara canis is a large white worm up to 10cm in length, and in dog can be confused with *Toxascaris leonina*. The egg is dark brown and sub-globular, with a thick, pitted shell.

Life cycle

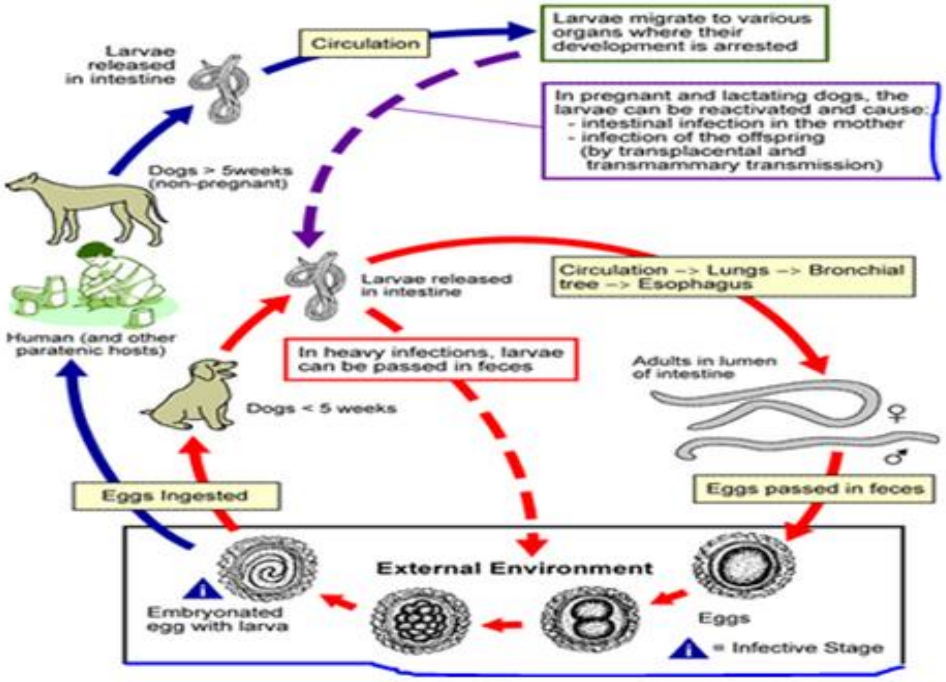
This specie has four possible modes of infection:

- The basic form is typically ascaridoid, the **egg containing the L2** being infective at optimal temperatures, four weeks after being passed. After **ingestion** and hatching in the small intestine, the L2 travel by the blood stream via the liver to the lungs, where the second moult occurs, the L3 returning to intestine via trachea and the final two moults take place.

  In dog over 3months of age, the hepatic-tracheal migration occurs less frequently. The larva L2 travels to a wide range of tissues including the liver, lungs, brain, heart and skeletal muscles and the walls of the alimentary tract.

- In pregnant bitch, **prenatal infection** occurs, larvae becoming mobilized at about three weeks prior to parturition and migrating to the lungs of fetus where they moult to L3 just before birth. In the newborn, the larvae travel to the intestine via trachea to complete final moults.
- The suckling pup may also be infected by **ingestion of L3 in the milk** during the first three weeks of lactation. There is no migration in the pup following infection by this route.
- The **paratenic hosts** such as rodents or birds may ingest the infective eggs and the L2 travel to their tissues where they remain until eaten by a dog.

Life cycle of *Toxocara canis*



The known minimum prepatent periods are different according to the mode of infection:

- Direct infection following ingestion of eggs or larvae, in paratenic host is 4-5 weeks
- Prenatal infection: 3 weeks.

Pathogenesis

- In moderate infections, the larval migratory phase is without any apparent damage to the tissues, and the adults provoke little reaction in the intestine.
- In heavy infections, the pulmonary phase is associated with pneumonia, sometimes accompanied with oedema. The adult worms cause mucoid enteritis; there may be partial or complete occlusion of the gut and in rare cases perforation with peritonitis or blockage of bile ducts.

Clinical signs

- In mild to moderate infections, there are clinical signs during pulmonary phase.
- The adults worms in intestines may cause pot-belly, with failure to thrive, and occasional diarrhea,
- Entire worms are sometimes vomited or passed in the faeces,
- In heavy infections, clinical signs include coughing, increased respiratory rate and a frothy nasal discharge.
- Most facilities from *Toxocara canis* infection occur during pulmonary phase,
- Heavily transplacentally infected pups may die within a few days of birth.
- Nervous convulsions

Epidemiology

Surveys of Toxocara canis prevalence of infections in dogs have shown the highest prevalence in dogs of less than six months of age. Three factors depends the distribution and intensity of infection:

- The females are extremely fecund (700 eggs for each gram of faeces per day)
- The eggs are highly resistant to climatic extremes and can survive for years on ground
- There is a constant reservoir of infection in the somatic tissues of the bitch and larvae are insusceptible to most anthelmintics.

Diagnosis

Clinical: only a tentative diagnosis possible (during pulmonary phase) is based on simultaneous appearance of pneumonic signs in a litter, often within two weeks of birth.

Faecal examination: using simple faecal smears

Treatment and control

- Anthelmintics to remove adult worms are (piperazine, benzimidazoles, febendazlo, mebendazole).
- The control includes the followings:

  - All pups should be dosed at 2 weeks of age and again 2-3 weeks later
  - A further dose should be given to the pups at two months old.
  - Adult dogs should be treated every 3-6 months throughout their lives.
  - Daily administration of febendazole to bitch from 3 weeks pre-partum to 2 days post partum to limit transmammary and prenatal infection of the pups.

B. Syngamiasis

Etiology

With the exception of the three genera, *Syngamus* and *Mammonogamus*; parasites of respiratory tract and *Stephanurus*; parasite of kidney, all other genera of *Trichostrongyloidea* are found in the gastro intestinal tract.

- **Hosts**: Domestic fowel and game birds (non aquatic birds)
- **I H**:
- **Site**: Trachea
- **Specie**: Syngamus trachea

Identification

The reddish, large female (up to 2cm) and small male (up to 0.5cm) worms are permanently in copulation forming a "**Y**" shape. They are only the parasites found in the trachea of domestic birds. The ellipsoidal egg of *Syngamus trachea* has an operculum at both ends.

Transmission mode

Up the trachea, contained in the excess mucus produced in response to infection, eggs are swallowed and pass in the feces. The L3 develops within the egg. Infection occurs by one of the following three ways:

- By ingestion of L3 in the egg,
- By ingestion of hatched larva L3
- By ingestion of a transport host (earthworms, slugs, snails and beetles) containing L3

After penetrating the intestine, the L3 travels to the lungs via blood and are found in alveoli 4-6 after ingestion. Two other moults take place in the lungs within 5 days to reach 1-2mm long. Copulation occurs around day 7 in the trachea or bronchi after which the female grows rapidly. Prepatent period is 18-20 days.

Pathogenesis

The effects of *Syngamus trachea* are most severe in young birds in which migration through the lungs in heavy infections may result in pneumonia and death. In less severe infections, the adult worms cause a hemorrhagic tracheitis with excess mucus production which leads to partial occlusion of airways and difficulty in breathing.

Clinical signs

- Pneumonia during prepatent phase
- Dyspnea and depression
- Asphyxia (adult worms + excess mucus in trachea)
- Head shaking and coughing
- Gape picture (mouth open), from gasping (breathe painfully), dyspnea and death to weakness, anemia and emaciation in less severely affected animals

Epidemiology

- Gapeworm infection primarily affects young birds
- However, turkeys of all ages are susceptible
- Adult birds often act as carriers
- Eggs may survive for up to 9 months in soil and L3 for years in transport hosts.

Diagnosis

- It is based on clinical signs and the finding of egg in the feces
- Postmortem examination to find worms attached to tracheal mucosa.

Treatment and Control

- In feed: Benzimidazole and Fenbendazole are administered usually over period of 3-14 days
- Nitroxynil and Levamisole, in water
- Do not rear young with adult birds
- Yards should be kept dry and prevent contact with wild birds
- Drug prophylaxis may be practiced over the period when outbreaks are normally expected.

C. Oxyuriasis

Etiology

The most known species of Oxyuriasis is *Oxyuriasis equi*

- Hosts: horses and donkeys
- Sites: coecum, colon and rectum
- Specie: Oxyuris equi

Distribution

Worldwide

Transmission mode

After fertilization in colon, the gravid female migrates to the anus and lays eggs in clumps. In 4-5 days after, the eggs contains infective L3. After ingestion of eggs, larvae are released in small intestine where they migrate into the mucosal of coecum and colon and moult to L4 takes place within 10days mucosa before maturing to adult stages which feed to intestinal contents. Prepatent period is 5 months.

Pathogenesis

Most of the pathogenic effects are due to the feeding habits of L4 which result in small erosions of the mucosa and in heavy infections; these may be wide spread and accompanied by an inflammatory response. The more important effect is the perineal irritation caused by the adult females during the egg laying.

Clinical signs

The condition rarely causes any clinical signs. However, intense pruritis around the anus causes the animal to rub, resulting in broken hairs and inflammation of the skin over the rump and tail head.

Diagnosis

It is based on signs of **anal pruritis** and the finding of grayish-yellow egg masses on the perineal skin. White long-tailed female worms are often seen in feces. Rarely eggs are seen on fecal examination (samples taken in rectum).

Treatment and control

Routine chemotherapy by anthelmintics used for horse parasites

D. Trichostrongylosis

Etiology

- Hosts: Ruminants, horses, pigs, rabbits, and fowel.
- Site: Small intestine, except *T.axcei* and *T. tenuis*
- Species:
  - *Trichostrongylus axcei* (abomasums of ruminants and stomach of horses and pigs)
  - *T. colubriformis* (ruminants)
  - *T. vitrinus* (sheep and goat)
  - *T. capricola* (sheep and goat)
  - *T. retortaeformis* (rabbit)
  - *T. tenuis* (small intestine and caeca of birds)

Distribution

Worldwide

Life cycle and transmission mode

The life cycle is direct and the pre-parasitic phase is typically Trichostrongyloid. Under optimal conditions, development from egg to infective stage occurs in 1-2 weeks. The parasitic phase is non-migratory and the prepatent period in ruminants is 2-3weeks.

In the horse, *T. axcei* has a prepatent period of 25 days while 10 days for *T. tenuis* in birds.

Pathogenesis

Following ingestion, L3 of the intestinal species penetrates between the epithelial glands of mucosa with formation of tunnels beneath the epithelium. When about 10-12 days after infection young worms are liberated, there is a considerable hemorrhage and edema and proteins are lost into the lumen of the gut.

Grossly there is enteritis, particularly in the duodenum. There is reduction of the area available for absorption of nutrients and fluids. In heavy infections, diarrhea occurs and this, together with the loss of plasma protein into the lumen of the intestine leads to weight loss. A reduced deposition of proteins, calcium and phosphorus has also been recorded.

Clinical signs

The principal clinical signs in heavy infections are rapid weight loss and diarrhea. At lower levels of infection, inappetence and poor growth rates, sometimes accompanied by soft feces are the common signs. It is often difficult to distinguish the effects of low infection from malnutrition.

Epidemiology

The embryonated eggs and infective L3 of *Trichostrongylus* have a high capacity for survival under adverse conditions whether there are extreme cold or dessications.

More commonly, larval numbers increase on pasture in summer and autumn giving rise to clinical problems during these seasons.

In the southern hemisphere larvae accumulate in the late winter outbreaks are usually seen in spring until recently hypobiosis was not considered to be a feature of this genus.

Immunity to *Trichostrongylus* is slowly acquired and in sheep and probably goats it wanes (weakens) during the periparturient period.

Diagnosis

This is based on clinical signs, seasonal occurrence of disease and if possible, lesions at post mortem examination. Fecal egg counts are useful aid to diagnosis; although fecal cultures are necessary for generic identification of larvae.

Treatment and control

- Benzimidazole (Albendazole)
- Levamisole
- Avermectins (Ivermectin

E. Thelaziosis

Etiology

Members of the genus *Therazia* are principally found in or around the eyes of animals and can be responsible for a Keratosis.

- **Definitive hosts**: Cattle and other domestic animals and occasionally man.
- **Intermediate hosts**: Muscid flies (*Musca spp*)

- **Sites**: Ocular region especially the conjunctival sac and lachrymal duct.
- **Species**:
  - Therazia lacrymalis (equine)
  - Therazia californiensis (dog, cat, sheep)
  - Therazia rhodesi
  - *Therazia gulos* } Cattle
  - *Therazia skrjabini*

Identification

Small thin white worms of 1-2cm long

Life cycle and transmission mode

The worms are viviparous. The L_1 passed by the female worm into the lachrymal secretion is ingested by the fly intermediate host as it feeds. Development from L_1 to L_3 occurs in the ovarian follicle of the fly in 15-30 days during the summer months.

L3 migrate to the mouth parts of the fly and are transferred to the final host when the fly feeds. Development in the eye takes place without further migration. Prepatent period is between 3-11 weeks depending on the species.

Pathogenesis

Lesions are caused by the serrated cuticle of the worm and most damage results from movement by the active young adults causing lachrymation, followed by conjunctivitis. In heavy infections the cornea may become cloudy and ulcerated.

There is usually complete recovery in about two months although in some cases areas of corneal opacity can persist.

Clinical sings

- Lachrymation
- Conjunctivitis
- Photophobia
- Flies are usually clustered around the eye because of the excessive secretions
- In severe cases, the whole cornea can be opaque.

Epidemiology

Therazia infections occur seasonally and are linked to the period of maximum fly activity. The parasite can survive in the eye for several years.

Diagnosis

This is based on observation of the parasites in the conjunctival sac. It may be necessary to instill a few drops of local anesthetic to facilitate manipulation of the third eyelid.

Treatment and control

Treatment is based on manual removal of the worms and administering an effective anthelmintic such as Levamisole or avermectin 1% or a mixture of 1.5ml oxytetracycline +0.5ml Ivermectin. Prevetion is difficult because of the ubiquitous nature of the fly vectors.

F. Metastrongylosis of pig

Etiology

The most worms in the superfamily of Metastrongyloidea inhabit the lungs or blood vessels adjacent to the lungs. The typical life cycle is indirect and the intermediate host is usually a mollusk. Only one genus occurs in pigs, Metastrongylus, and it is exceptional in having earthworms, rather than mollusks, as intermediate hosts.

- **Site**: small bronchi and bronchioles especially those of the posterior lobes of the lungs.
- **Species**:
  - *Metastrongylus apri (*synonym*: elongatus)*
  - *Metastrongylus salmi*
  - *Metastrongylus pudendotectus*

Distribution

Worldwide

Identification

Slender white worms up to 6cm in length; the host, site and long slender form are sufficient for generic identification.

Life cycle and transmission mode

In cold temperatures the eggs are very resistant and can survive for over a year in soil. Normally, they hutch almost immediately and the I H ingest the L_1. In the earthworms, development to L_3 takes about 10 days at optimal temperature of 22-26^{o}C.

The longevity of the L3 in the earthworms is similar to that of the I H itself, and may be up to seven years. The pig is infected by ingestion of earthworms and the L3 released by digestion, travel to the mesenteric lymph nodes, moult and the L4 then reach the lungs by the lymphatic vascular route, the final moult occurring after arrival in the air passage. The prepatent period is about 4 weeks.

Pathogenesis

During the prepatent period area of pulmonary consolidation, bronchial muscular hypertrophy, and peribronchial lymphoid hyperplasia develop, often accompanied by area of over inflation.

- When the worms are mature and the eggs are aspirated into the smaller air passage and parenchyma, consolidation increases and emphysema is more marked.
- Hypersecretion of bronchiolar mucus also occurs during this stage.
- About six weeks after infection, chronic bronchitis and emphysema are established and smaller grayish nodules may be found in the posterior part of the diaphragmatic lobes; these may aggregate to form larger areas.

Clinical signs

- Most infections are light and asymptomatic.
- In heavy infections, coughing is marked and is accompanied by dyspnea and nasal discharge.
- Secondary bacterial infection may complicate the signs

Epidemiology

Metastrongylosis shows a characteristic age distribution, being most prevalent in pigs of 4-6 months old.

Diagnosis

For fecal examination saturated magnesium sulfate should be used as the flotation solution because of the heavy density of the eggs.

Treatment and Control

- Many anthelmintics including the modern benzimidazoles, levamisole and the avermectin are highly effective.
- When pig husbandry is based on pasture, control is extremely difficult because of the ubiquity and longevity of the earthworms, intermediate host.
- On farms where outbreaks have occurred pigs should be housed, dosed and the infected pasture cultivated or grazed by other stocks.

G. Schistosomiasis

Etiology

- **Definitive hosts**: All domestic animals, mainly important in sheep and cattle.
- **Intermediate hosts**: Water snails
- **Site**: Usually mesenteric veins
- **Species**:
  - *Schistosoma bovis* (Cattle)
  - *Schistosoma mattheei* (Ruminants + man)
  - *Schistosoma japonicum* (man)

Distribution

Tropics and subtropics regions

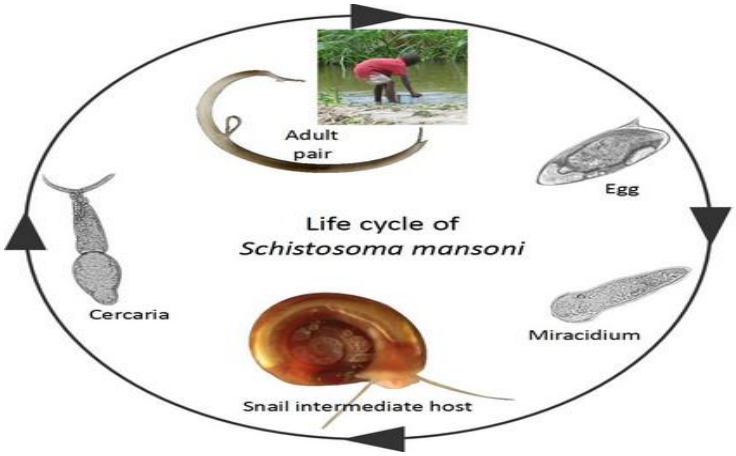
Identification

Grossly, the sex are separate, the male, which is broad and flat and about 2cm long, carrying the female in hallow of its inwardly curved body.

Life cycle

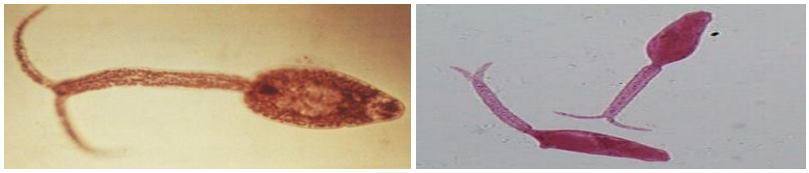
The female in the mesenteric vein inserts her tail into a small venule and since the genial pore is terminal, the eggs are deposited, or even pushed, into the venule. There, aided by their spines and by proteolytic enzymes secreted by the unhatched miracidia, they penetrate the endothelium to enter the intestinal submucosa and ultimately the gut lumen; they are then passed out in the feces.

Life cycle of *Schistosoma mansoni*



The eggs hatch and miracidia penetrate appropriate snails. Development to cercarial stage occurs without a redial form and there is no metacercarial phase. The final host is infested by penetration of cercaria through the skin or by ingestion in drinking water.

After penetration or ingestion the cercariae lose their forked tail, transform to Schistosomula, or young flukes, and travel via the blood stream through the heart and lungs to the systemic circulation. In the liver they locate in the portal veins and become sexually mature before migrating to their final site, the mesenteric veins. The prepatent period is 6-7 weeks.



Picture 15: Cercariae of *Schistosoma spp*

<u>Pathogenesis</u>

Schistosomiasios is generally considered to be a much more serious and important infection in sheep than in larger ruminants. Following massive infection death can occur rapidly, but more usually the clinical signs abate slowly as the infection progresses.

As this occurs, there appears to be a partial shift of worms away from the intestinal mucosa and reactions to these migrating parasites and their eggs can occur in the liver. In sheep, anemia and hypoalbuminemia have been shown to be prominent during the clinical phase apparently as a result of mucosal hemorrhage, dyshaemopoeisis and an expansion in plasma volume.

Clinical signs

- There are diarrhea, sometimes blood stained and containing mucus,
- Anorexia,
- Thirst,
- Anemia and emaciation

Epidemiology

The epidemiology is very similar to that of *Fasciola gigantic* and *Paramphistomum spp*, *Schistosoma spp* being totally dependent upon water as a medium for infection of both the intermediate host and final host. The fact that percutaneous infection may occur encourages infection where livestock are obligate to wade in water.

Diagnosis

- This is based mainly on the clinic-pathological picture of diarrhea, wasting and anemia, coupled with a history of access to natural water sources;
- The demonstration of the characteristic egg in the feces;
- Post mortem examination of the mesentery (if the mesentery is stretched, the presence of numerous schistosomes in the veins).
- Serological tests may be of value.

Treatment and control

Care has to be exercised in treating clinical cases of Schistosomiasis since the dislodgement of damaged flukes may result in emboli being formed and subsequent occlusion of major mesenteric and portal blood vessels with fatal consequences.

Praziquantel which is used in the treatment of human schistosomiasis is also effective in animals. The control is similar to that outlined for *Fasciola gigantica* and *Paramphistomum* infections.

H. Haemonchosis

Etiology

- **Hosts:** Cattle, sheep and goats
- **Site:** Abomasum
- **Species:**
  - *Haemonchus contortus (*sheep*)*
  - *Haemonchus placei (*cattle*)*

- *Haemonchus smilis*

Distribution

Worldwide. Most important in tropical and subtropical areas

Identification

- Grossly: the adults are easily identified because of their specific location in the abomasum and their large size (2-3cm).
- Microscopically: the male has an asymmetrical dorsal lobe and barbed spicules; the female has a vulval flap. In both sexes there are cervical papillae and a tiny lancet inside the buccal capsule.

Life cycle

This is direct and the females are prolific egg layers. The eggs hutch to L1, on the pasture and may develop to L3 in as short a period as 5 days, but development may be delayed for weeks or months under cool conditions.

After ingestion by the host, the larvae moult twice and develop the piercing lancet which enables them to obtain blood from the mucosal vessels. These adult parasites may move freely on the surface of mucosa. The prepatent period is 2-3 weeks in sheep and 4 weeks in cattle.

Pathogenesis

Essentially the pathogenesis of haemonchosis is that of an acute hemorrhagic anemia due to the blood sucking habits of the worms. In acute haemonchosis anemia becomes apparent about two weeks after infection and is characterized by a progressive and dramatic fall in the packed red-cell volume.

When ewes are affected, the consequent agalactia may result in death of the suckling lambs. At necropsy, between 2000 and 20000 worms may be present on the abomasal mocosa which shows numerous small hemorrhagic lesions.

Less commonly, in heavier infections of up to 30000 worms, apparently healthy sheep may die suddenly from severe hemorrhagic gastritis. This is termed hyperacute haemonchosis. Perhaps as important as acute heamonchosis in tropical areas is the lesser known syndrome of chronic haemonchosis.

Clinical signs

- In hyperacute cases, sheep die suddenly from hemorrhagic gastritis.
- Acute haemonchosis is characterized by anemia, variable degrees of oedema of which the submendibular form and ascites are most easily recognized, lethargy, dark colored feces and falling wool. Diarrhea is not generally a feature.

- Chronic haemonchosis is associated with progressive weight loss and weakness, neither severe anemia nor gross oedema being present.

Epidemiology

The epidemiology of *Haemonchus contortus* is best considered separately depending on whether it occurs in tropical and subtropical or in temperate areas. In tropical and subtropical areas, because larval development of *Haemonchus contortus* occurs optimally at relatively high temperatures, haemonchosis is primarily a disease of sheep in warm climates.

However, since high humidity, at least in the microclimate of the feces and the herbage, is also essential for larval development and survival, the frequency and severity of outbreaks of disease is largely dependent on the rainfall in any particular area.

The sudden occurrence of acute clinical haemonchosis appears to depend on two factors:

- The high fecal worm egg output of between 2000 and 20000 eggs per gram (EPG), means that massive pasture population of L3 may appear quickly.
- In contrast to many other helminth infections, there is little evidence that sheep in endemic areas develop an effective acquired immunity to haemonchosis, so that there is continuous contamination of the pasture.

The survival of the parasite is also associated with the ability of Haemonchus contortus larvae to undergo hypobiosis. The survival of Haemonchus contortus infection on tropical pastures is variable depending on the climate and degree of shade, but the infective larvae are relatively resistant to dessication and some survive for 1-3 months on pasture or in feces.

Diagnosis

- The histories of grazing and clinical signs often are sufficient for the diagnosis of the acute syndrome especially if supported by feces worm egg counts.
- Necropsy, paying attention to both the abomasums and the marrow changes in the long bones, is also useful.
- The diagnosis of chronic haemonchosis is more difficult because of the concurrent presence of poor nutrition.

Treatment

When an acute outbreak has occurred the sheep may be treated with one of the following anthelminthics: Benzimidazole, Levamizole and Ivermectin.

Control or prevention

In the tropics and subtropics, this varies depending on the duration and number of periods in the year when rainfall and temperature permit high pasture levels of Haemonchus contortus larvae to develop.

- Use anthelmintics at interval of 2-4 weeks depending on degree of challenge.
- Treat sheep at the start of dry season and preferably before the start of prolonged rain to remove persisting hypobiotic larvae.

I. Ostertagiosis

Etiology

Ostertagiosis is a helminthosis of digestive tract (stomach).

- **Hosts**: Ruminants
- **Site**: Abomasum
- **Species**:
  - *Ostertagia ostertagi* (cattle)
  - *Ostertagia circumcincta* (sheep and goats)
  - *Ostertagia trifurcata* (sheep and goats)

Distribution

Worldwide

Identification

The adults are reddish-brown worms of the size 6-10mm long, occurring on the surface of the abomasal mucosa.

Ostertagia ostertagi is perhaps the most common cause of ostertagiosis in cattle. Bovine ostertagiosis is characterized by weight loss and diarrhea and typically effects young cattle during their first grazing season, although, herd outbreaks and sporadic individual cases have also been reported in adult cattle.

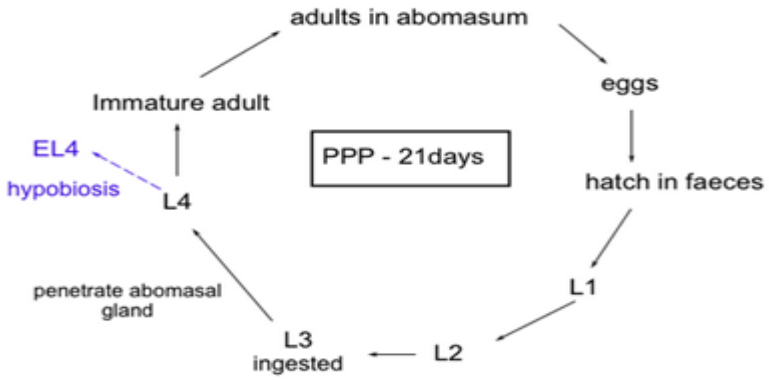
Life cycle

Ostertagia ostertagi has a direct life cycle. The eggs are passed out in the feces, under optimal conditions develop the infective L3 stage within the fecal pat and migrate from the feces on to the herbage.

After ingestion, the L3 exsheaths in the rumen and further development takes place in the lumen of an abomasal gland. Two parasitic moults occur before the L5 emigres from the gland around 18 days after infection to become sexually mature on the mucosal surface.

The entire parasitic life cycle usually takes 3 weeks, but under certain circumstances many of the ingested L3 become arrested in development at early fourth stage (L4) for periods of up to six months.

Life cycle of *Ostertagia sotertagi*



Pathogenesis

The presence of Ostertagia ostertagi in the abomasum in sufficient numbers gives rise to extensive pathological and biochemical changes and severe clinical signs. These changes are maximal when the parasites are emerging from the gastric glands. This is usually about 18 days after infection, but it may be delayed for several months when arrested larval development occurs.

The results of these changes are a leakage of pepsinogen into the circulation leading to elevated plasma pepsinogen levels and the loss of plasma proteins leading to hypoalbuminaemia. Clinically, the consequences are reflected as inappetence (anorexia), weight loss, and diarrhea.

These disturbances of course influenced by the level of nutrition, being exacerbated by a low protein intake.

Clinical signs

The main clinical signs include:

- A profuse watery diarrhea,
- Submendibular oedema is often present due to hypoalbumineamia,
- The loss of body weight is considerable during the clinical phase and may reach 20% in 7-10 days

- Morbidity can reach 75% or more; however mortality is low.

Epidemiology

The epidemiology of ostertagiosis can be conveniently considered under the headings of dairy herds and beef herds. The important differences in subtropical climates are described below.

Dairy herds

From epidemiological studies, the following important factors have emerged:

- A considerable number of L3 can survive the winter on pasture and in soil.
- A high mortality of overwintered L3 on the pasture occurs in spring and only negligible number can usually be detected.
- The eggs deposited in the spring develop slowly to L3; this rate of development becomes more rapid towards mid-summer as temperature increases, and as a result, the majority of eggs deposited during April, May and June all reach the infective stage from mid-Jully.
- As autumn progresses and temperatures fall, an increasing proportion (up to 80%) of the L3 ingested do not mature but become inhibited at the early fourth larval stage

Beef herds

Although the basic epidemiology in beef herds is similar to dairy herds, the influence of immune adult animals grazing alongside susceptible calves has to be considered. Thus, in beef herds where calving takes place in the spring, ostertagiosis is uncommon since egg production by immune adult is low and the spring mortality of the overwintered L3 occurs prior to the suckling calves ingesting significant quantities of grass. Consequently, only low numbers of L3 become available on the pasture later in the year.

Diagnosis

In young animals this is based on the following:

- The clinical signs of inappetence, weight loss and diarrhea,
- The season
- The grazing history
- Fecal egg count (1000 eggs per gram)
- Post-mortem examination: the appearance of the abomasal mucosa is characteristic. There is putrid smell from the abomasal contents due to accumulation of bacterial and a high pH. The adult worms are reddish in color and 1cm in length can be seen on close inspection of mucosal surface.

In older animals, the clinical signs and history are similar but laboratory diagnosis is more difficult since fecal egg count and plasma pepsinogen levels are less reliable. A useful technique is to carry out a pasture larval count on the field on which the animals had been grazing.

Treatment and Prevention

- Benzimidazole (Albendazole)
- Levamisole
- Avermectins (Ivermectin)
- Anthelmintics: young cattle over the period when the pasture larval levels are increasing.
- Limit the exposure of animals to infection (more efficient method of control)
- A better policy is to permit young cattle sufficient exposure to larval infection to stimulate immunity but not sufficient to cause a loss in production.

J. Oesophagostomosis

- **Hosts**: Ruminants
- **I H**:
- **Sites**: Coecum and colon
- **Species**:

  - *Oesophagostomum radiatum* (cattle)
  - *Oesophagostomum columbianum* (sheep and goat)
  - *Oesophagostomum venulosum* (sheep and goats)
  - *Oesophagostomum dentatum* (pig)

Distribution

Worldwide, more important in tropical and subtropical areas.

Life cycle

Infection is by ingestion of L3 although there is limited evidence that skin penetration is possible, at least in pigs. The L3 enter the mucosa of any part of the small or large intestine and in some species, become enclosed in obvious nodules in which the moult to L4 takes place.

The L4 then emerge on to mucosal surface, migrate to the colon and develop to the adult stage. The prepatent period is about 45 days.

Pathogenesis

All species are capable of causing severe enteritis including *Oesophagostomum venulosum* which does not provoke nodule formation.

In the intestine *Oesophagostomum columbianum* L3 migrate deep into the mucosa, provoking an inflammatory response with the formation of nodules which are visible to the naked eye. When the L4 emerge there may be ulceration of the mucosa.

In heavy infections, there may be ulcerative colitis and the disease runs a chronic debilitating course with effects on the production. The nodules in the gut also render the intestines useless for processing as sausage, skins and surgical suture material.

Necropsy reveals a severity inflamed mucosa with yellowish-green purulent nodule. In the later stages of the disease, anemia and hypoalbuminaemia develop due to the combined effects of protein loss and leakage of blood through the damaged mucosa. The disease in pig is less clinical but responsible for poor productivity.

Clinical signs

- In acute infections of ruminants, severe dark-green diarrhea is the main clinical sign and there is usuallya rapid loss of weight and sometimes submendibular oedema,
- In chronic infections, which occur primarily in sheep, inappetence and emaciation with intermittent diarrhea and anaemia are the main signs of oesophagostomosis.
- Pregnant sows show inappetence, become thin and following farrowing milk production is reduced with effects on litter performances.

Epidemiology

In tropical and subtropical areas, Oesophagostomum columbianum and *Oesophagostomum radiatum* in sheep and cattle respectively are especially important. In *Oesophagostomum columbianum* infections, the prolonged survival of L4 within the nodules in the gut wall and the lack of an effective immunity make control difficult until the advent of effective anthelmintics.

In contrast, cattle develop a good immunity to *Oesophagostomum radiatum*, partly due to age and partly to previous exposure so that it is primarily a problem in weaned calves.

Diagnosis

- It is based on clinical signs and post mortem examination.
- In acute disease (prepatent period) eggs are not usually present in the feces.
- In chronic disease, the eggs are present and L3 can be identified following fecal culture.

Treatment and control

They are similar to that of *Trichostrongyles* in ruminants. Infection in pigs can be controlled by anthelmintic treatment regimes recommended for the control of *Ascaris suum*

K. Dictyocaulosis

Etiology

Dictyocaulus, the genus of Trichostrongyloidea, living in the bronchi of cattle, sheep, horse and donkeys is the major cause of parasitic bronchitis in these hosts.

- **Hosts**: Ruminants, horses and donkeys
- **Sites**: Trachea and bronchi, particularly of the diaphragmatic lobes.
- **Species**:
  - *Dictyocaulus viviparous*: cattle and deer
  - *Dictyocaulus filarial*: sheep and goats
  - *Dictyocaulus arnfieldi*: donkeys and horses.

Identification

The adults are slender thread-like worms up to 8cm in length. Their location in the trachea and bronchi and their size are diagnosed.

Life cycle

The female worms are ovoviviparous, producing eggs containing fully developed larvae L1 which hatch almost immediately. L1 migrate up the trachea, are swallowed and pass out in the feces. Under optimal conditions the L3 stage is reached within 5 days, but usually takes longer in the field.

After ingestion, the L3 penetrate the intestinal mucosa and pass to the mesenteric lymph nodes where they moult. The L4 travels via the lymph and blood to the lungs, and breaks out of the capillaries into the alveoli about one week after ingestion.

The final moult occurs in the bronchioles a few days later and the young adults then move up the bronchi and mature. Prepatent period is around 3-4 weeks.

Pathogenesis

It may be divided into four phases:

- **Penetration phase (days 1-7)**: During this period the larvae are making their way to the lungs and pulmonary lesions are not yet apparent.
- **Prepatent phase (days 8-25)**: this phase starts with the appearance of larvae within the alveoli causing alveolitis followed by bronchiolitis and finally bronchitis. Toward the end of this phase bronchitis develops, characterized by mucus containing immature lungworms in the airways.

  Heavily infected animals, whose lungs contain several thousand developing worms, may die from day 15 onwards due to respiratory failure following the development of severe interstitial emphysema and pulmonary oedema.

- **Patent phase (days 26-60)**: this is associated with two main lesions:

  A parasitic bronchitis characterized by the presence of hundreds or even thousands of adult worms in frothy white mucus in the bronchi. A parasitic pneumonia caused by the aspiration of eggs and L1 into the alveoli.

- **Post-patent phase (days 61-90)**: in untreated calves, this is normally the recovery phase after the adult lungworms have been expelled. The clinical syndrome is often termed “Post-patent parasitic bronchitis”.

Clinical signs

Within any affected group, differing degrees of clinical severity are usually apparent:

- Mildly affected animals cough intermittently, particularly when exercised.
- Moderately affected animals have frequent bouts of coughing at rest, tachypnea and hyperpnea.
- Severely affected animals show severe tachypnea (>80 respiration/min) and dyspnea and frequently adopt the class “**Air hunger**” position of mouth.
- There is usually a deep harsh cough, squeaks and crackles over the posterior lung lobes, salivation, anorexia and sometimes mild pyrexia.
- Most animals gradually recover although complete return to normality may take weeks or months.

Epidemiology

Generally only calves in their first grazing season are clinically affected, since on farms where the disease is endemic older animals have a strong acquired immunity. In endemic areas in the northern hemisphere infection may persist from year to year in 2 ways:

- Overwinted larvae: L3 may survive on pasture from autumn until late spring in sufficient numbers to initiate infection or occasionally to cause disease.
- Carrier animals: small numbers of adult worms can survive in the bronchi of infected animals, particularly yearlings, until the next grazing season.

Diagnosis

- Usually clinical signs, the time of the year and a history of grazing on permanent or semi permanent pasture are sufficient.
- Fecal examination (larvoscopy)

Treatment and control

The treatment is based to use anthelmintics: modern Benzimidazole, Levamisole or Avermectin
The best control is to immunize all young calves with lungworm vaccine and the use of prophylactic anthelmintic regimes.

L. Parafilariosis

The adult of *Parafilaria*, the genus of primitive filarioids live under the skin where they produce inflammatory lesions or nodule and, during egg laying, hemorrhagic exudates or "bleeding points" on the skin surface.

- **Hosts:** Cattle and buffalo
- **Intermediate host:** *Muscid* flies
- **Sites:** Subcutaneous and intermuscular connective tissue
- **Species:**
  - *Parafilaria bovicola*
  - *Parafilaria multipapillosa*

Identification

Slender (slim) white worms 3-6 in length. In the female the vulva is situated anteriorly near the simple mouth opening. Small embryonated eggs are laid on the skin surface where they hatch to release the microfilariae or L1 which are about 200µm in length.

Life cycle

Eggs of larvae present in exudates from bleeding point in the skin surface are ingested by muscid flies, for example *Musca autumnalis* in Europe, *Musca lusoria* and *Musca xanthomelas* in Africa, in which they develop to L3 within several weeks to months, depending on air temperature.

Transmission occurs when infected flies feed on lachrymal secretions or skin wounds in other cattle and the L3 deposited then migrate and develop to the adult stage under the skin in 5-7 months. Bleeding points develop 7-9 months after infection.

Pathogenesis

When the gravid female punctures the skin to lay her eggs there is a hemorrhagic exudates or bleeding point which streaks and mats the surrounding hairs and attracts flies.

Individual lesions only bleed for a short time and healing is rapid. At the sites of infections which are predominantly on the shoulders, withers and thoracic areas, there is inflammation and oedema which, at meat inspection, resemble subcutaneous bruising in early lesions and have a gelatinous greenish-yellow appearance with metallic odor in longer standing cases.

The affected areas have to be trimmed at marketing and further economic loss is incurred by rejection or down-grading of the hides.

Clinical signs

The signs of parafilariosis are pathognomonic.

- Active bleeding lesions are seen most commonly in warm weather
- An apparent adaptation to coincide with the presence of flies intermediate hosts

Diagnosis

This is normally based on clinical signs, but if laboratory confirmation is required, the small embryonated eggs or microfilariae may be found on examination of exudates from bleeding points.

Treatment and control

Patent infections in beef and non lactating dairy cattle may be treated with ivermectin or nitroxynil. The control is difficult because of the long prepatent period during which drugs are through not to be effective.

- Topic 2: Plathelmintiasis

A. Trematodoses (flukes)

A.1. Paramphistomosis

Etiology

This is a trematodosis condition of ruminants. The adult paramphistomes are mainly parasitic in the forestomachs of ruminants, although a few species occur in the intestine of ruminants, pigs and horses. Their shape is not typical of the trematodes, being conical rather than flat. All require a water snail as an intermediate host.

- **Dedinitive host**s: Ruminants
- **Intermediate hosts**: Water snails, principally *Planorbis* and *Bulinus*
- **Sites**: Adults are found in the rumen and reticulum and immature stages in the duodenum
- **Species**: there are at least 14 species of which *Paramphistomum cervi* and *Paramphistomum microbothrium* are the most common

Distribution

Worldwide, but are of little veterinary significance in Europe and America

Identification

Glossily, the adults are small, conical maggot-like flukes, about 1cm long. One sucker is visible at the tip of the cone and the other at the base. The larval stages are less than 5mm fresh specimens having a pink color.

Picture 16: Paramphistomum spp on the forestomachs

Microscopically, the eggs resemble that of *Fasciola hepatica* being large and operculate, but are clear than yellow.

Life cycle

Development in the snail intermediate host is similar to that of Fasciola and under favorable conditions (26-30^{o}C) can be completed in 4 weeks. Infestation occurs when the definitive host ingests the encysted metacercariae with herbage. The prepatent period is between 7-10 weeks.

Pathogenesis

Any pathogenic effect is associated with the young flukes (intestinal parasites) which cause severe erosions of the duodenal mucosa. In heavy infections, these cause enteritis characterized by oedema, hemorrhage and ulceration.

The adult parasites in the forestomachs are well tolerated even when many thousand are present and feeding on the wall of the rumen or reticulum.

Clinical signs

In heavy duodenal infections, the most obvious sign is diarrhea accompanied by anorexia and intense thirst.

- Sometimes in cattle, there is rectal hemorrhage following a period of prolonged straining.
- Mortality in acute outbreaks can be as high as 90%.

Epidemiology

Paramphistomosis often depends for its continuous endemicity on permanent water masses, such as lakes and ponds from which snails are dispersed into previously dry areas by flooding during heavy rains. Paramphistomes eggs deposited by animals grazing these areas hatch and infect snails. Subsequent production of cercariae often coincides with receding water levels making them accessible to grazing ruminants.

A good immunity develops in cattle and outbreaks are usually confined to young stock. However, adults continue to herbour low burdens of adult parasites and are important reservoir of infection for snails. In contrast, sheep and goats are relatively susceptible throughout their lives.

Diagnosis

- It is based on clinical signs usually involving young animals in the herd and a history of grazing around snail habitats during period of dry weather.

- Fecal examination is of little value since the disease occurs during the prepatent period. Confirmation can be obtained by a post mortem examination and recovery of small flukes from the duodenum.

Treatment and Control

Resorantel and oxyclozanide are considered to be anthelmintics of choice against both immature and adult rumen flukes in cattle and sheep.

- Prevent piped water supply to animals and access of animals to natural water
- Regular application of molluscicides at source (eg: Copper sulphate)
- Manual removal of snails

A.2. Fascilosis

The members of the genus Fasciola are commonly known as liver flukes. They are responsible for widespread morbidity and mortality in sheep and cattle characterized by weight loss, aneamia and hypoproteinaemia.

The two most important species are *Fasciola hepatica* found in temperate areas and in cooler areas of high altitude in the tropics and subtropics, and *Fasciola gigantica* which predominantes in tropical areas.

- **Definitive host**: Most mammals; sheep and cattle are most important.
- **Intermediate host**: Snail of genus Lymnea, *Lymnea truncatula*
- **Site:** the adults are found in the bile ducts and immature flukes in the liver parenchyma, occasionally, flukes are found in lungs (erratic location).

Distribution

Worldwide

Identification

The young fluke when entering the liver is 1-2mm long and lancet like. Mature fluke in bile ducts is leaf shaped, grey-brown in color. The size of *Fasciola hepatica* is around 3.5cm in length and 1cm wide while *Fasciola gigantic* larger than *Fasciola hepatica* can reach 7.5cm in length. The anterior end is conical. The egg is oval, operculated, yellow and large.

Life cycle and transmission

The adult flukes are always oviparous and lay eggs with an operculum or lid at one pole. In the egg the embryo develops into a pyriform, ciliated larva called a MIRACIDIUM.

The cilia allow miracidium to swim in the water seeking out on an aquatic snail, the first intermediate host, then penetrate the skin of the snail and develop to the next stage, the SPOROCYST, containing a number of germinal cells.

These cells develop into REDIAE which migrate to the hepato-pancreas of the snail. From the germinal cells of the rediae arise the final stages, the CERCARIAE. The cercariae stage often has a tail and will emerge from the snail and swim in the water.

Depending on the species of the flukes, at this point, the cerariae will take one of the following three paths:

- The cercariae may directly penetrate the skin of the definitive host,
- The cercariae may attach to vegetation, lose its tail, secrete a thick cyst wall around itself, and thus develop into METACERCARIAE
- The cercariae may lose its tail, penetrate the second intermediate host, secrete a thick cyst wall around itself and develop into a METACERCARIA within the second intermediate host.

The infestation occurs when the definitive host ingests one of the above infective forms:

- If the fluke takes the first option, the cercariae will migrate its predilection site and develop into the adult fluke,
- If the fluke takes the second option, the thick cyst wall will be digested by the host and the juvenile fluke released will migrate to the predilection site and develop into the adult fluke,
- If the fluke takes the third option, the second I. H. and the thick cyst wall are digested and the juvenile fluke released will migrate to the predilection site and then develop into the adult fluke.

Epidemiology

There are three main factors that influence the production of large numbers of metacercariae necessary for outbreaks of fasciolosis:

- **Availability of suitable snail habitats**: *Lymnea truncatula* prefers wet mud to free water, and permanent habitats include the banks of ditches or streams and the edges of small ponds.
- **Temperature**: a mean day/night temperature, of 10^{o}C or above is necessary both for snail to breed and for the development of *Fasciola hepatica* and *Fasciola gigantic*
- **Moisture**: the ideal moisture condition for snail breeding and the development of *Fasciola spp* within the snails are provided when rainfall exceeds transpiration and field saturation is attained.

Pathogenesis and symptoms

These vary according to the phase of parasitic development in the liver and host involved. Essentially the pathogenesis is two folds:

- The first phase occurring during the migration in the liver parenchyma and is associated with liver damage and hemorrhage.
- The second occurs when the parasite is in the bile ducts and results from the hemorrhagic activity of adult flukes and from damage to the biliary mucosa.

A.2.1. Ovine fasciolosis

The disease may be acute, subacute or chronic.

The acute form

This form occurs 2-6 weeks after ingestion of large numbers of metacercariae, usually over 2000 and is due to the severe hemorrhage associated with the migration of young flukes in liver parenchyma, rupturing blood vessels.

At necropsy, the liver is enlarged, hemorrhagic and honey combed with tracts of migrating flukes. The surface over the ventral lobe is frequently covered with fibrous exudates.

Outbreaks of this form are generally presented as sudden death during autumn and early winter. Survival sheep are weak, with pale mucous membranes and sometimes have palpable enlarged livers associated with abdominal pain and ascites.

The subacute form

It occurs 6-10 weeks after ingestion of approximately 500-1500 metacercariae. It appears in late autumn and winter. It is presented as a rapid and severe hemorrhagic anemia with hypoalbuminaemia and if untreated can result in high mortality rate.

Affected sheep may show clinical signs for 1-2 weeks prior to death: rapid loss of condition, a marked pallor of the mucous membranes, and an enlarged and palpable liver, submendibular or facial oedema and ascites may be present.

Chronic form

Seen mainly in late winter/early spring is the most common form. It occurs 2-4 months after infection with moderate number of 200-500 metacercariae. Pathologic effects are mainly anemia and hypoalbuminaemia (more than 0.5ml of blood can be lost daily).

At necropsy, liver has irregular outlines and is pale and firm. It is characterized hepatic fibrosis and hyperplastic cholangitis. Clinically, this form is characterized by a progressive loss of condition, anemia and hypoalbuminaemia that can result in emaciation, pallor of mucous membranes, submendibular oedema and ascites.

A.2.2. Bovine fasciolosis

Although acute and subacute disease may occasionally occur under condition of heavy challenge, especially in young calves, the chronic form of the disease is by far the most important, as in sheep, is seen in the late winter/early spring.

The pathogenesis is similar to that in sheep but has the added features of calcification of bile ducts and enlargement of gall bladder. Aberrant migration is common in cattle and parasites are seen in lungs.

In heavy infections, where anemia and hypoalbuminaemia are severe, submendibular oedema frequently occurs. Diarrhea is not a feature of bovine fasciolosis unless it is complicated by *Ostertagia spp*. Combined infection with these two parasites has been referred to as the Fasciolosis/Ostertagiosis complex.

Diagnosis

It is based on clinical signs, seasonal occurrence, weather patterns and a previous history of fasciolosis on the farm or identification of snail habitats.

Haematological tests and faecal examination are useful and may be supplemented by the following two laboratory tests:

- The estimation of plasma levels of enzymes released by damaged liver cells,
- The detection of antibodies against components of flukes, ELISA and passive haemoagglutination tests being the most reliable.

Treatment and Control

Recent and more effective drug compounds are followings: Nitroxynil, Albendazole, Closantel, Rafoxanide and Oxyclozanide which may be used in lactating cows.

The Control is done in two ways, either by reducing population of intermediate hosts or by using anthelmintics.

Reducing of intermediate hosts snail population

After determining whether snails are localized or widespread, the best long term method for reducing mud-snail populations is drainage since it ensures permanent destruction of snail habitats.

When the snail habitat is limited a simple method of control is to fence (protect) off this area or treat annually with a molluscicide such as copper sulfate.

Use of anthelmintics

The prophylactic use of fluke's anthelmintics is aimed at:

- Reducing pasture contamination by fluke eggs at a time most suitable for their development (eg: April to August)
- Removing fluke population at a time of heavy burdens or at a period of nutritional and pregnant stress to the animals.

A.3. Dicrocoeliosis

- **Final hosts**: sheep, cattle, deer and rabbits
- **Intermediate hosts**: Land snails and Brown ants
- **Sites**: bile ducts and gall bladder
- **Specie**:
    - *Dicrocoelium dendriticum*

Identification

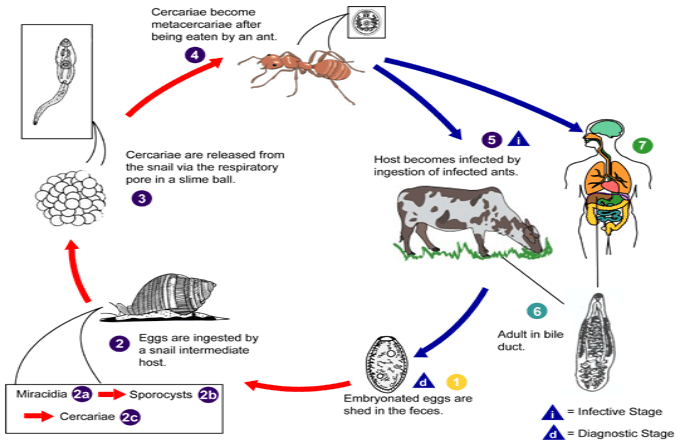
Dicrocoelium is less than 1cm long. The egg is small, dark brown operculated, usually with a flattened side. It contains a miracidium when passed in feces.

Life cycle

After being ingested by land snail, intermediate host, the egg hatches and two generations of sporocysts develop to produce cercariae. This takes at least 3 months.

The cercariae are ingested by ants in which they develop to metacercariae. Final hosts are infected by ingesting the ants. Ingested metacercariae hatch in small intestine and young flukes migrate to main bile ducts and the smaller ducts in the liver. There no parenchymal migration. The prepatent period is 10-12 weeks.

Life cycle of *Dicrocoelium dendriticum*



Pathogenesis

Although several thousand *Dicrocoelium dendriticum* are found in bile ducts commonly, the livers are relatively normal because of the absence of parenchymal migration. However in heavy infections, there is fibrosis of smaller bile ducts and extensive cirrhosis can occur.

Clinical signs

In many instances these are absent. Anaemia, oedema and emaciation have been reported in severe cases.

Epidemiology

There are two important features which differentiate the epidemiology of Dicrocoerium from that of Fasciola:

- The intermediate hosts are independent of water and are evenly distributed on the terrain.
- The eggs can survive for months on dry pasture, presenting a reservoir additional to that in the intermediate and final hosts.

Diagnosis

This is entirely based on fecal examination for eggs and necropsy finding. The eggs contain a miracidium and are very small (40 × 25 µm), lopsided, and yellowish brown. Fecal flotation with a solution of high gravity is recommended to detect *D dendriticum*.

Treatment and control

Effective anthelmintic treatments in both cattle and sheep are albendazole at 15–20 mg/kg in a single dose or two doses of 7.5 mg/kg on successive days, or netobimin at 20 mg/kg, Praziquantel (50 mg/kg), thiabendazoleand fenbendazole.

The control is difficult because of the longevity of *Dicrocoelium dendriticum* eggs, the widespread of intermediate host and the number of reservoir host. The regular anthelmintic treatment is required.

B. Cestodoses

The adults *Taeniidae* are found in domestic carnivores and man. The scolex has an armed rostellum with a concentric double row of hooks. The gravid segments are longer than they are wide. The intermediate stage (larva) is a *Cysticercus*, *Strobulocercus*, *Coenurus* or *Hydatid cyst*. And occur only in mammals. The main important genera of this family "*Taeniidae*" are ***Taenia*** and ***Echninococcus*.**

B.1. Taeniosis

Etiology

Taenia is the most important genus of *Taeniidea*, both the adult and larval stage being of importance in human health and veterinary medicine. The final host, larval stage, intermediate host and larval predilection site of the major species are given in table below.

Table 3: Final and intermediate hosts of Taenia spp

Adult tapeworms	Final host	Larva	Intermediate host	Larval site
Taenia saginata	Man	*Cysticercus bovis*	Cattle	Muscle
Taenia solium	Man	*Cysticercus cellulosae*	Pig, man	Muscle
Taenia multiceps	Dog	*Coenurus cerebralis*	Sheep, cattle	Central nervous system
Taenia hydatigena	Dog	*Cysticercus tenuicolis*	Sheep, cattle, pig	Peritoneum
Taenia ovis	Dog	*Cysticercus ovis*	Sheep	Muscle
Taenia pisiformis	Dog	*Cysticercus pisiformis*	Rabbit	Peritoneum
Taenia serialis	Dog	*Coenurus serialis*	Rabbit	Connective tissue
Taenia taeniaeformis	Cat	*Cysticercus fasciolaris*	Mouse, rat	Liver
Taenia krabbei	Dog	*cysticercus tarandi*	Reindeer	Muscle

B.1.1. *Taenia saginata* (Taenia of Man)

It is Taeniasis of man is mainly caused by *Taenia saginata*.

The intermediate stages of this tapeworm found in the muscles of cattle, frequently present economic problems to the beef industry and are public hazard.

Identification

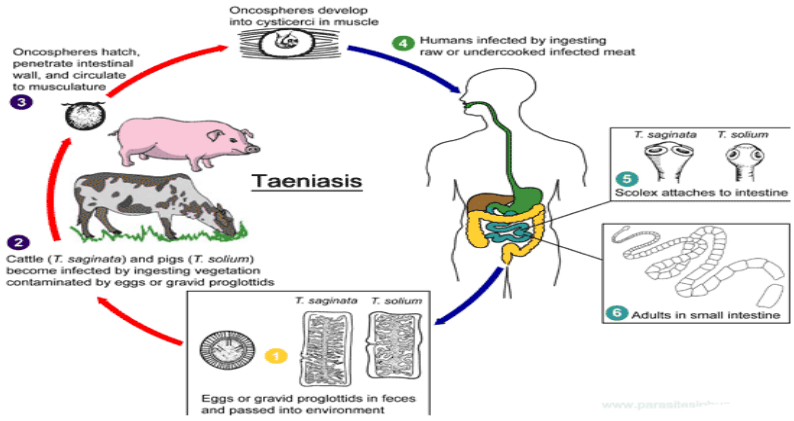
The adult tapeworm, found only in man, range from 5-15m length. The immature stage in bovine animals is *Cysticercus bovis* is white about 1cm in diameter. It may occur anywhere in the striated muscles the predilection sites, at least from the viewpoint of routine meat inspection, are the heart, the tongue and the masseter and intercostals muscles.

Life cycle

An infected human may pass millions of egg daily in feces or in intact segments, and these can survive on pasture for several months. After ingestion by a susceptible bovine the onchosphere travels via the blood to striated muscles.

It is grossly visible about 2 weeks later as pale, semi-transparent spot about 1mm in diameter. Man becomes infected by ingesting row or inadequately cooked cattle meat.

Life cycle of Taeniae of human



Pathogenesis and clinical signs

Under natural conditions the presence of cysticerci in muscles of cattle is not associated with clinical signs, even though calves given massive infection of *Taenia saginata* eggs have developed severe myocarditis and heart failure. In man, the adult tapeworm may produce diarrhea and hunger pains, but the infection is usually asymptomatic.

Diagnosis

Diagnosis is based to meat inspection. Individual countries have different regulations regarding the inspection of carcasses, but invariably the masseter, tongue, and heart are incised and examined and the intercostal muscles and diaphragm inspected; triceps muscle is also incised in many countries. The inspection is inevitably a compromise between detection of cysticerci and the preservation of the economic value of the carcass.

Treatment and Control

- As yet there no licensed drug available effectively to destroy all of the cysticerci in the muscles but praziquantel may be used.
- The control of bovine cysticercosis depends on a high standard of human sanitation, on the general practice of cooking meat thoroughly and on compulsory meat inspection.
- Regulations usually require that infected carcasses are frozen at – 10^{o}C for at least 10 days which is sufficient to kill the cysticerci although the process reduces the economic value of the meat.

B.1.2. *Taenia solium* (Taenia of Man)

This is the other Taenia specie of man caused by *Taenia solium*, the larval stage, *Cysticercus cellulosae*, occurring in the muscles of the pig. On occasions the cysticerci may also develop in man and the disease, human cysticercosis is the most serious aspect of this zoonosis.

Identification

The adult is similar to *Taenia saginata* except that the scolex is typically Taeniid, having a rostellum armed with concentric rows of hooks.

Life cycle

This may occur either from the accidental ingestion of *Taenia solium* eggs or, apparently, in a person with an adult tapeworm, from the liberation of onchosphere after the digestion of a gravid segment which has entered the stomach from the duodenum by reverse peristalsis (autoinfection).

Pathogenesis and clinical signs

Clinical signs are unapparent in pigs naturally infected with cysticerci and so in human with adult tapeworms. However, when man is infected with cysticerci, various clinical signs may occur depending on the location of the cysts in the organs, muscles, or subcutaneous tissues.

Most seriously, cysticerci may develop in the CNS producing mental disturbances or clinical signs of epilepsy or increased intracranial pressure; they may also develop in the eye with consequent loss of vision.

Diagnosis

For all practical purposes, diagnosis depends on meat inspection procedures similar to those outlined for *Taenia saginata*. In man the diagnosis of cerebral cysticercosis depends primarily on the detection of cysticerci by scanning techniques.

Treatment and Control

No effective drugs are available to kill cysticerci in the pig although in man praziquantel and albendazole are considered to be of some value as possible alternatives to surgery.

The control depends ultimately on the enforcement of meat inspection regulations and deep freezing procedures as described for *Taenia saginata*.

- The exclusion of pigs from contact with human feces
- Cooking of pork and proper standards of personal hygiene, reduce the prevalence of infection

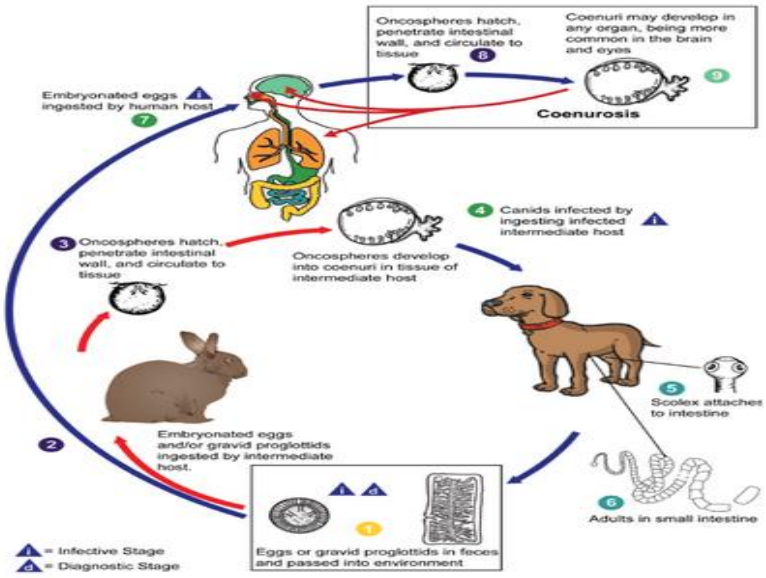
B.1.3. *Taenia multiceps* (Taeniosis in dog and cat)

The adult is up to 100 cm lon. Hosts (final) are dogs and wild canids, intermediate host is sheep or other ruminants. The onchospheres, when ingested by sheep or other ruminants, are carried in the blood to the brain or spinal cord where each develops into larval stage "***Coenurus cerebralis".***

When mature, this larva is readily recognized as a large fluid-filled cyst up to 0.5cm or more in diameter which bears clusters of scolices on its internal wall.

The coenurus takes about 8 months to mature in the CNS and, as it develops, clinical signs commonly occur. Thes depend on the location of the cyst(s) and include circling, visual defects, periculiarities in gait, hyperaesthesia (abnormal increased sensitivity to stimuli) or paraplegia. The clinical syndrome is often known as "**gid syndrome**".

Life cycle of *Taenia multiceps*



Generally, there is no treatment but however, surgical removal is possible if the cyst is situated on the brain surface.

B.1.4. *Taenia hydatigena* (Taeniosis in dog and cat)

This is a large tapeworm, up to 500cm long, found in dog and wild canids. The onchosphere, infective to the sheep, cattle and pigs, are carried in the blood to the liver in which they migrate for about 4 weeks before they emerge on the surface of this organ and attach to the peritoneum.

Within a further 4 weeks each develops into the characteristically large *Cysticercus tenuicolis* which is up to 8 cm in diameter. The infection is prevalent, particularly in sheep, but is generally only detected at meat inspection.

Infrequently, however, large numbers of developing cysticerci migrate in the liver of the sheep or pig producing “hepatitis cysticercosa”, a condition whose gross pathology resembles acute fasciolosis which is often fatal.

B.1.5. *Taenia ovis* (Taeniosis in dog and cat)

This common tapeworm of dogs and wild carnivores is up to 200cm long. The life cycle is similar to that of *Taenia saginata*, the intermediate stage, *Cystscercus ovis* being found in the muscle of sheep. It is economical important, because of the appearance of the cysts in sheep meat can cause economic loss through condemnation at meat inspection.

To eradicate this tapeworm it is better to use a regular chemotherapy and by prohibiting the feeding uncooked sheep meat to dogs.

B.1.6. *Taenia pisiformis* (Taeniosis in dog and cat)

The life cycle of this tapeworm, up to 200cm long, of dogs and related carnivores is similar to that of *Taenia hydatigena* except that the intermediate stage, the pea-like *Cysticercus pisiformis*, is found on the peritoneum of the rabbit and hare.

B.1.7. *Taenia serialis* (Taeniosis in dog and cat)

This is another tapeworm of dog, around 70cm long, whose intermediate stage is *Coenurus serialis* is found in the rabbit, usually subcutaneously or in the intermuscular connective tissue. The numerous scolises in the coenurus are arranged in the lines as the name “serialis” implies.

B.1.8. *Taenia taeniaeformis* (Taeniosis in dog and cat)

This is a cat tapeworm, about 60cm long, whose intermediatr stage, *cysticercus fasciolaris*, is found in the liver of mice and other rodents. Each strobilocercus is found within a pea-sized nodule partially embedded in the liver parenchyma.

Diagnosis

Diagnosis depends on the demonstration of segments or individual taeniid eggs in the feces. Specific identification of the adult tapeworm is a specialized task.

Treatment and Control

For adult tapeworms a number of effective drugs are available including Praziquantel, Mebendazole, Fenbendazole, Nitroscanate and Dichlorophen.

Control depends on dietary methods which exclude access to the larval stage in the intermediate hosts.

B.2. Echinococcosis

Etiology

Echinococcus is the smallest genus cestode of Taeniidae of domestic animals. In veterinary medicine, two species are important, *Echinococcus granulosus* and *Echinococcus multilocularis*, intermediate stage develops in a very wide range of intermediate hosts including man. The definitive hosts are dogs and foxes.

Identification

The cestode is about 6cm long and is difficult to find in freshly opened intestine. It consists of a scolex and three or four segments, the terminal gravid one, occupying half the length of the total worm.

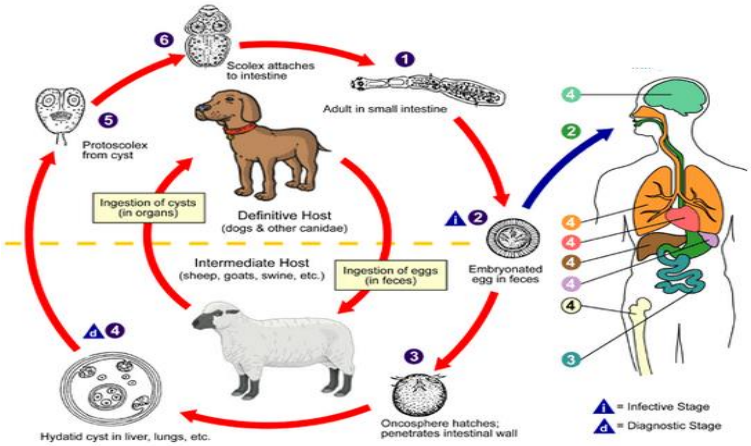
Picture 17: *Echinococcus granulosus* (large) and *Echinocuccus multilocularis*

Life cycle

After a prepatent period of 40-50 days in final host, a gravid segment is weekly released. On ground, the onchospheres are variable for about 2 years. After ingestion by intermediate host, onchosphere travels to the liver via blood or lymph. Location in other organs and tissues is occasionally possible through blood circulation. Maturity is reached in 6-12 months.

Hydatid cyst may develop complete daughter cysts inside or externally; if externally formed, they may be carried to the other parts of the body to form new hydatids. In sheep, about 70% of hydatids occur in lungs, 25% in liver, the rest in other organs. In horses and cattle, more than 90% are found in liver.

Life cycle of *Echinococcus spp*



Pathogenesis

The adult is not pathogenic and is associated with no clinical signs, even presence of thousands. In domestic animals (I H), hydatids are usually tolerated without no clinical signs, the majority being revealed at the abattoir.

In case of locations other than liver and lungs, associated clinical signs may be seen. In man as intermediate host, the hydatid in its pulmonary or hepatic sites is often of pathogenic significance.

Epidemiology

The epidemiology of Echinococcus granulosus is based on two cycles, pastoral and sylvatic.

- In the pastoral cycle, the dog always is involved, being infected by the feeding of ruminant offals containing hydatid cysts. The pastoral cycle is the primary source of hydatidosis in man, infection being by accidental ingestion of onchospheres from the coats of dogs or from vegetables and other foodstuffs contaminated by dog feces.
- The sylvatic cycle occurs in wild canids and ruminants and is based on predation. It is less source of infection of man except in hunting communities.

Diagnosis

- No clinical diagnosis
- Laboratory diagnosis is difficult in dogs with adult tapeworm, because segments are small and shed only sparsely (in small amount). When found, the size is about 2-3mm, ovoid shapeand single genital spore.
- In man, are commonly used serological tests, scanning techniques may also be used to locate the cysts.

Treatment and Control

- Praziquantel is highly effective
- After treatment, dogs are confined for 48hours to facilitate collection and dispersal of infected feces
- In man, surgical treatment, mebendazole, albendazole and praziquantel, therapies have been reported to be effective.
- Regular treatment of dogs to eliminate adult tapeworms,
- Prevent dogs infection: do not give to dog ruminant offals, limit the access of dogs to abattoirs,
- In some countries, these measures have been supported by legislation, with penalties when they are disregarded.

- Topic 3: Acanthocephales

Etiology

As described above, the major genus of veterinary significance is *Macracanthorhynchus*.

- **Definitive host**: Pig
- **Intermediate hosts**: Various dugs beetles
- **Specie**: *Macracanthorynchus hirudinaceus*

Distribution

Worldwide, but absent from certain areas, for example parts of Western Europe

Identification

Grossly, adult resemble to *Ascaris suum*, but taper posteriorly. The males are up to 10cm long and the females up to 65cm in length. When placed in water, the spiny proboscis is protruded, thus aiding differentiating from *Ascaris*. Microscopically, the egg is oval with a thick brown shell and contains the acathor larva when laid.

Pathogenic significance

Macracanthorynchus hirudinaceus produces inflammation and may provoke granuloma formation at the site of attachment in the small intestinal wall. Heavy infections may cause weight loss and rarely, penetration of the intestinal wall results in fatal peritonitis.

Diagnosis and treatment

- Diagnosis is based on finding the typical eggs in the faeces.
- For treatment, pigs should be prevented from access to the intermediate hosts. However, levamizole and ivermectine are reported to be effective.

L O 2. 2 – Characterize protozoal diseases

- Topic 1: Tick-Borne Diseases

Importance of ticks and tick borne diseases:

- They are parasites that lead to anemia, toxicosis and secondary bacterial infections
- They are vectors of diseases in domestic animals and human
- They are associated with direct and indirect losses

In pathogenic reactions of the tick, we will include:

- Trauma: mental condition caused by shock. If the tick challenge on animal is high, it will cause the trauma on the host.
- Toxicosis: the toxin can be produced, the can cause problems being deposed in body tissues.
- Anemia: result from blood sucking done by ticks.

The severity of TBDs depends on below factors:

- Number of ticks on individual cow
- Rate of infection, meaning the % of infected ticks
- Cattle breed and age

Tick borne diseases, especially in tropical countries are diseases caused by rikettsial (heart water) or rikettsial-like (Amaplasmosis) and protozoa (Theileriosis=ECF and Babesiosis). These are the most important TBDs in East-Africa including Rwanda.

A. Theileriosis (East Coast Fever)

Etiology

Theileriases are a group of tick borne diseases caused by *Theileria spp*. A large number of *Theileria spp* are found in domestic and wild animals in tick-infested areas of the World. The most important species affecting cattle are *T parva* and *T annulata*, which cause widespread death in tropical and subtropical areas of the World.

The infective sporozoite stage of the parasite is transmitted in the saliva of infected ticks as they feed.

East Coast fever, an acute disease of cattle, is characterized usually by high fever, swelling of the lymph nodes, dyspnea, and high mortality. Caused by *Theileria parva*, it is a serious problem in east and central Africa.

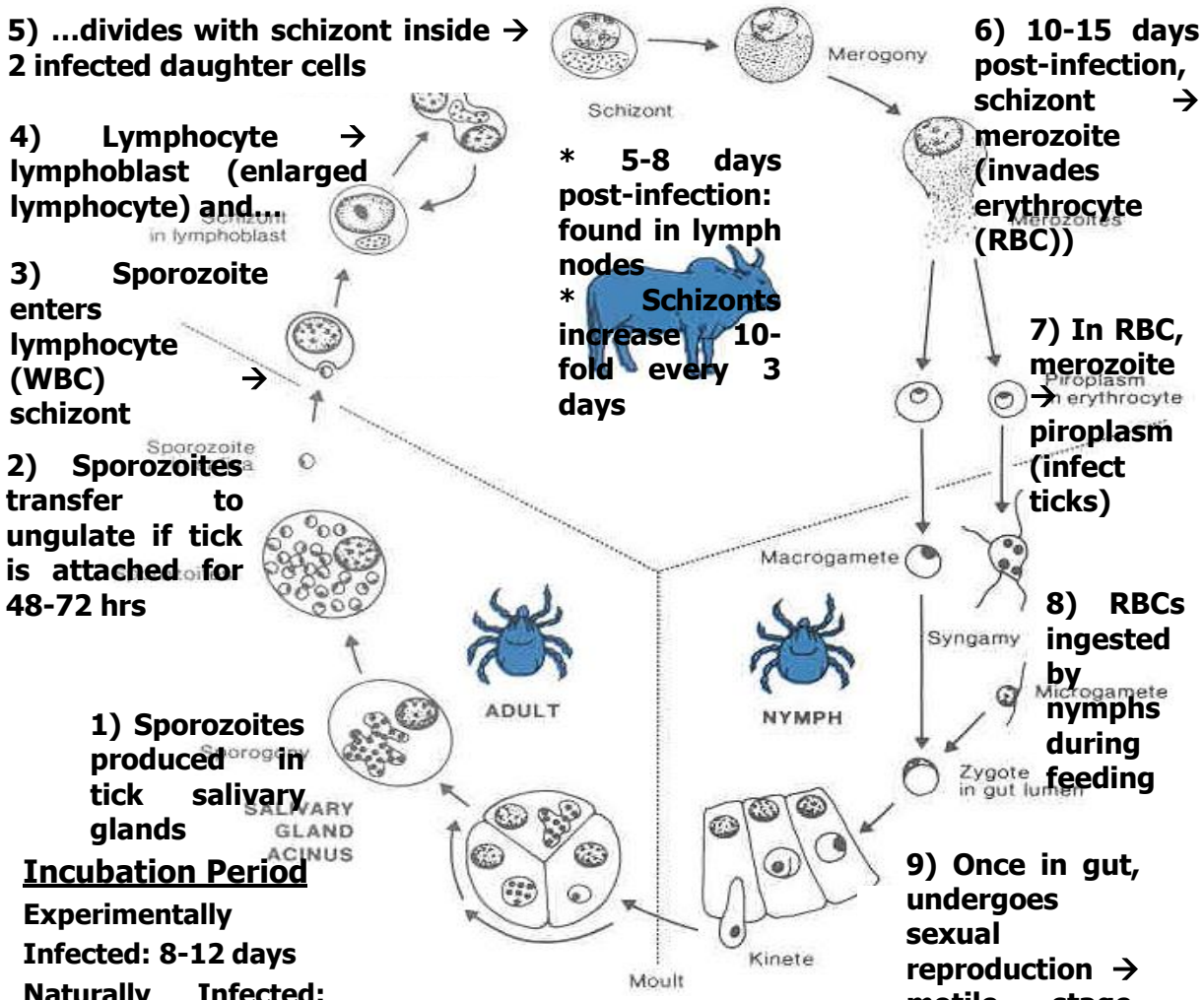
Transmission

T parva sporozoites are injected into cattle by infected vector ticks, *Rhipicephalus appendiculatus*, during feeding. Based on clinical and epidemiologic parameters, 3 subtypes of T parva are recognized, but these are probably not true subspecies. T parva parva and T parva bovis are both maintained by transmission between cattle.

Pathogenesis

The number of parasitized cells increases rapidly throughout the lymphoid system, and from about day 14 onward, cells undergoing merogony are observed. This is associated with widespread lymphocytolysis, marked lymphoid depletion, and leukopenia

Life cycle of Theileria parva



5) ...divides with schizont inside → 2 infected daughter cells

4) Lymphocyte → lymphoblast (enlarged lymphocyte) and...

3) Sporozoite enters lymphocyte (WBC) → schizont

2) Sporozoites transfer to ungulate if tick is attached for 48-72 hrs

1) Sporozoites produced in tick salivary glands

*** 5-8 days post-infection: found in lymph nodes**

*** Schizonts increase 10-fold every 3 days**

6) 10-15 days post-infection, schizont → merozoite (invades erythrocyte (RBC))

7) In RBC, merozoite → piroplasm (infect ticks)

8) RBCs ingested by nymphs during feeding

9) Once in gut, undergoes sexual reproduction → motile stage, moves to tick's salivary gland

<u>Incubation Period</u>

Experimentally Infected: 8-12 days

Naturally Infected: up to 3 weeks

Clinical signs

- Clinical signs vary according to the level of challenge and range from inapparent or mild to severe and fatal.
- Typically, fever occurs 7–10 days after parasites are introduced by feeding ticks, continues throughout the course of infection, and may be >107°F (42°C).
- Lymph node swelling becomes pronounced and generalized. Lymphoblasts in Giemsa-stained lymph node biopsy smears contain multinuclear schizonts.
- Anorexia develops and the animal rapidly loses condition; lacrimation and nasal discharge may occur. Terminally, dyspnea is common.
- Just before death, a sharp fall in body temperature is usual, and pulmonary exudate pours from the nostrils.
- Death usually occurs 18–24 days after infection.

Diagnosis

The most striking postmortem lesions are lymph node enlargement and massive pulmonary edema and hyperemia.

Treatment and Control

Treatment with parvaquone and its derivative buparvaquone is highly effective when applied in the early stages of clinical disease but is less effective in the advanced stages in which there is extensive destruction of lymphoid and hematopoietic tissues.

Immunization of cattle against T parva using an infection-and-treatment procedure is practical and is gaining acceptance in some regions.

Incidence of East Coast fever can be reduced by rigid tick control, but this is not feasible in many areas because of cost and the high frequency of acaricidal treatment required.

B. Anaplasmosis

Anaplasmosis, formerly known as gall sickness, traditionally refers to a disease of ruminants caused by obligate intraerythrocytic bacteria of the order Rickettsiales, family Anaplasmataceae, and genus *Anaplasma*. Cattle, sheep, goats, buffalo, and some wild ruminants can be infected with the erythrocytic Anaplasma.

Transmission and Epidemiology

Up to 19 different tick vector species (including *Boophilus, Dermacentor, Rhipicephalus, Ixodes, Hyalomma*, and *Ornithodoros*) have been reported to transmit *Anaplasma spp*. Not all of these are likely significant vectors in the field. *Boophilus spp* are major vectors in Australia and Africa, and *Dermacentor spp* have been incriminated as the main vectors in the USA.

After feeding on an infected animal, intrastadial or trans-stadial transmission may occur. Transovarial transmission may also occur, although this is rare, even in the single-host Boophilus spp. There is a strong correlation between age of cattle and severity of disease.

Calves are much more resistant to disease (although not infection) than older cattle. This resistance is not due to colostral antibody from immune dams. In endemic areas where cattle first become infected with *Anaplasma marginale* early in life, losses due to anaplasmosis are minimal.

Clinical Findings

In animals <1 year old anaplasmosis is usually subclinical, in yearlings and 2 years olds it is moderately severe, and in older cattle it is severe and often fatal. Anaplasmosis is characterized by progressive anemia due to extravascular destruction of infected and uninfected erythrocytes.

Lesions

- Lesions are typical of those found in animals with anemia due to erythrophagocytosis.
- The carcasses of cattle that die from anaplasmosis are generally markedly anemic and jaundiced.
- Blood is thin and watery.
- The spleen is characteristically enlarged and soft, with prominent follicles.
- The liver may be mottled and yellow-orange.
- The gallbladder is often distended and contains thick brown or green bile.
- Hepatic and mediastinal lymph nodes appear brown.

Diagnosis

At necropsy, thin blood films of liver, kidney, spleen, lungs, and peripheral blood should be prepared for microscopic examination.

Microscopic examination of Giemsa-stained thin and thick blood films is critical to distinguish anaplasmosis from babesiosis and other conditions that result in anemia and jaundice, such as leptospirosis and theileriosis.

Treatment

Tetracycline antibiotics and imidocarb are currently used for treatment. Cattle may be sterilized by treatment with these drugs and remain immune to severe anaplasmosis subsequently for at least 8 months.

A commonly used treatment consists of a single IM injection of long-acting oxytetracycline at a dosage of 20 mg/kg. Imidocarb is also highly efficacious against *Anaplasma marginale* as a single injection.

C. Babesiosis

Babesiosis is caused by intraerythrocytic protozoan parasites of the genus *Babesia*. The disease, which is transmitted by ticks, affects a wide range of domestic and wild animals and occasionally people. While the major economic impact of babesiosis is on the cattle industry, infections in other domestic animals, including horses, sheep, goats, pigs, and dogs, assume varying degrees of importance throughout the world.

Two important species in cattle are *Babesia bigemina* and *Babesia bovis* which are widespread in tropical and subtropical areas.

Transmission and Epidemiology

The main vectors of *Babesia bigemina* and *Babesia bovis* are 1-host *Rhipicephalus (Boophilus) spp* ticks, in which transmission occurs transovarially. While the parasites can be readily transmitted experimentally by blood inoculation, mechanical transmission by insects or during surgical procedures has no practical significance.

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.

Clinical signs

- The acute disease generally runs a course of about 1 week.
- The first sign is fever (frequently 106°F [41°C] or higher), which persists throughout, and is accompanied later by inappetence,
- Increased respiratory rate, muscle tremors, anemia, jaundice,
- Weight loss; hemoglobinaemia and hemoglobinuria occur in the final stages.
- CNS involvement due to adhesion of parasitized erythrocytes in brain capillaries can occur with *B bovis* infections.
- Either constipation or diarrhea may be present.
- Late-term pregnant cows may abort.

Lesions

Lesions include an enlarged and friable spleen; a swollen liver with an enlarged gallbladder containing thick granular bile; congested, dark-colored kidneys; and generalized anemia and jaundice. The urine is often, but not invariably, red. Other organs, including the brain and heart, may show congestion or petechiae

Diagnosis

Clinically, babesiosis can be confused with other conditions that cause fever, anemia, hemolysis, jaundice, or red urine.

Therefore, confirmation of a diagnosis by microscopic examination of Giemsa-stained blood or organ smears is essential. From the live animal, thick and thin blood smears should be prepared, preferably from capillaries in the ear or tail tip.

Smears of heart muscle, kidney, liver, lung, brain, and from a blood vessel in an extremity (eg, lower leg) should be taken at necropsy.

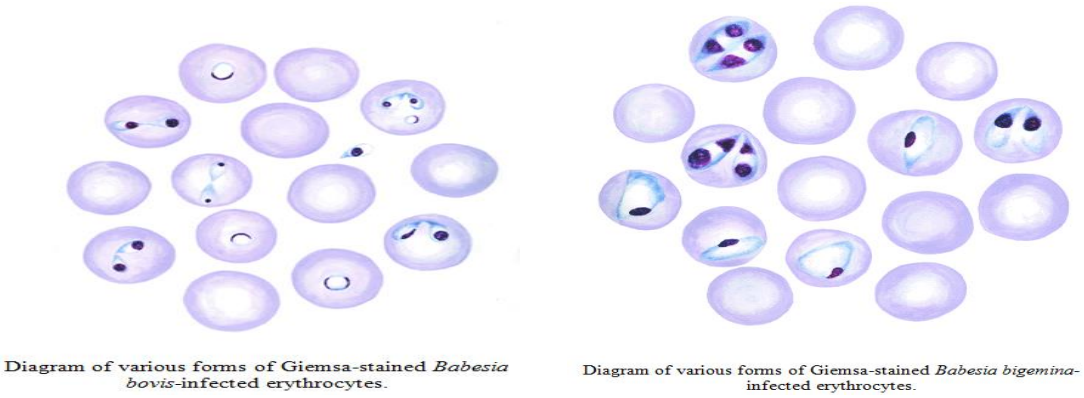


Diagram of various forms of Giemsa-stained *Babesia bovis*-infected erythrocytes.

Diagram of various forms of Giemsa-stained *Babesia bigemina*-infected erythrocytes.

Picture 18: Diagram of Babesia bovis (left) and Babesia bigemina (right)

Serologic tests have been described for the detection of antibodies to *Babesia* in carrier animals. The most commonly used are the indirect fluorescent antibody test and ELISA; commercially produced ELISA for *Babesia bovis* and *B bigemina* are available.

Treatment

A variety of drugs have been used to treat babesiosis in the past, but only Diminazene aceturate and Imidocarb dipropionate are still in common use. Manufacturers' recommendations for use should be followed.

Long-acting tetracycline (20 mg/kg) may reduce the severity of babesiosis if treatment begins before or soon after infection.

Supportive treatment is advisable, particularly in valuable animals, and may include the use of anti-inflammatory drugs, antioxidants, and corticosteroids.

Control

- Imidocarb provides protection from babesiosis
- Vaccination using live, attenuated strains of the parasite
- Controlling the tick vector can break the transmission cycle

D. Heart water (Cowdriosis)

Heartwater is an infectious, noncontagious, tick-borne rickettsial disease of ruminants. The disease is seen only in areas infested by ticks of the genus *Amblyomma variegatum.* Cattle, sheep, goats, and some antelope species are susceptible to heartwater. In endemic areas, some animals and tortoises may become subclinically infected and act as reservoirs.

Etiology and Transmission

The causative organism is an obligate intracellular parasite, previously known as *Cowdria ruminantium*. Molecular evidence led to reclassification of several organisms in the order Rickettsiales, and it is now classified as *Ehrlichia ruminantium*. Under natural conditions, *E ruminantium* is transmitted by *Amblyomma* ticks. These three-host ticks become infected during either the larval or nymphal stages and transmit the infection during one of the subsequent stages (trans-stadial transmission).

Pathogenesis

The pathogenesis of heart water has not been elucidated; however, the tick probably infects the host via organisms in the saliva or regurgitated gut content while feeding.

During the febrile stages and for a short while thereafter, the blood of infected animals is infective to susceptible animals if subinoculated. Signs and lesions are associated with functional injury to the vascular endothelium, resulting in increased vascular permeability without recognizable histopathologic or even ultrastructural pathology.

The concomitant fluid effusion into tissues and body cavities precipitates a fall in arterial pressure and general circulatory failure. The lesions in peracute and acute cases are hydrothorax, hydropericardium, edema and congestion of the lungs and brain, splenomegaly, petechiae and ecchymoses on mucosal and serosal surfaces, and occasionally hemorrhage into the GI tract, particularly the abomasum.

Clinical Findings

The clinical signs are dramatic in the peracute and acute forms.

In peracute cases, animals may drop dead within a few hours of developing a fever, sometimes without any apparent clinical signs; others display an exaggerated respiratory distress and/or paroxysmal convulsions.

In the acute form, animal often shows anorexia and depression along with congested and friable mucous membranes. Respiratory distress slowly develops along with nervous signs such as a hyperaesthesia, a high-stepping stiff gait, exaggerated blinking, and chewing movements.

Terminally, prostration with bouts of opisthotonus; “pedaling,” “thrashing,” or stiffening of the limbs; and convulsions are seen. Diarrhea is seen occasionally. In subacute cases, the signs are less marked and CNS involvement is inconsistent.

Diagnosis

In clinical cases, heart water must be differentiated from a wide range of infectious and noninfectious diseases, especially plant poisonings that manifest with CNS signs. In acute clinical cases in endemic areas, clinical signs alone may suggest the etiology, but demonstration of colonies of organisms in the cytoplasm of capillary endothelial cells is necessary for a definitive diagnosis.

Treatment and Control

Oxytetracycline at 10 mg/kg/day, IM, or doxycycline at 2 mg/kg/day will usually affect a cure if administered early in the course of heartwater infection. A higher dosage of oxytetracycline (20 mg/kg) is usually required if treatment begins late during the febrile reaction or when clinical signs are evident.

Corticosteroids have been used as supportive therapy (prednisolone 1 mg/kg, IM), although there is debate as to the effectiveness and rationale for their use. Diazepam may be required to control convulsions.

Control of tick infestation is a useful preventive measure in some instances but may be difficult and expensive to maintain in others. Excessive reduction of tick numbers, however, interferes with the maintenance of adequate immunity through regular field challenge in endemic areas and may periodically result in heavy losses.

Chemoprophylaxis involves a series of oxytetracycline injections to protect susceptible animals from contracting heartwater when introduced into endemic areas while also allowing them to develop a natural immunity.

- Topic 2: Trypanosomiasis

Etiology

This group of diseases caused by protozoa of the genus *Trypanosoma* affects all domestic animals. The major species are *T congolense, T vivax, T brucei*, and *T simiae*. For the animals mainly affected by these tsetse-transmitted trypanosomes and the geographic areas where tsetse-transmitted Trypanosomiasis.

Table 4: Trypanosoma spp and their definitive hosts

Trypanosoma spp	Animals Mainly Affected	Major Geographic Distribution
T congolense	Cattle, sheep, goats, dogs, pigs, camels, horses, most wild animals	Tsetse region of Africa
T vivax	Cattle, sheep, goats, camels, horses, various wild animals	Africa, Central and South America, West Indies
T brucei	All domestic and various wild animals; most severe in dogs, horses, cats	Tsetse region of Africa
T simiae	Domestic and wild pigs, camels	Tsetse region of Africa

Cattle, sheep, and goats are infected, in order of importance, by *T congolense, T vivax*, and *T brucei*. In pigs, T simiae is the most important. In dogs and cats, *T brucei* is probably the most important.

Transmission and Epidemiology

Most tsetse transmission is cyclic and begins when blood from a trypanosome-infected animal is ingested by the fly. The trypanosome loses its surface coat, multiplies in the fly, then reacquires a surface coat and becomes infective.

Tsetse flies (genus *Glossina*) are restricted to Africa. Mechanical transmission can occur through tsetse or other biting flies. In the case of *T vivax, Tabanus spp* and other biting flies seem to be the primary mechanical vectors outside the tsetse areas.

Pathogenesis

Infected tsetse inoculates trypanosomes into the skin of animals, where the trypanosomes grow for a few days and cause localized swellings (chancres). They enter the lymph nodes, then the bloodstream, where they divide rapidly by binary fission. In *T congolense* infection, the organisms attach to endothelial cells and localize in capillaries and small blood vessels. *T brucei* species and *T vivax* invade tissues and cause tissue damage in several organs.

Clinical Findings

Severity of disease varies with species and age of the animal infected and the species of trypanosome involved. The clinical signs include the followings:

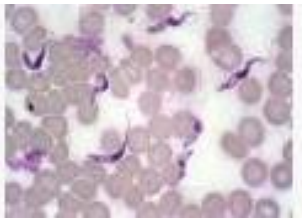
- The incubation period is usually 1–4 weeks.
- The primary clinical signs are intermittent fever, anemia, and weight loss.
- Cattle usually have a chronic course with high mortality, especially if there is poor nutrition or other stress factors.
- Ruminants may gradually recover if the number of infected tsetse flies is low; however, stress results in relapse.

Lesions

- Necropsy findings vary and are nonspecific.
- In acute, fatal cases, extensive petechiation of the serosal membranes, especially in the peritoneal cavity, may occur.
- Also, the lymph nodes and spleen are usually swollen. In chronic cases, swollen lymph nodes, serous atrophy of fat, and anemia are seen.

Diagnosis

A presumptive diagnosis is based on finding an anemic animal in poor condition in an endemic area. Confirmation depends on demonstrating trypanosomes in stained blood smears or wet mounts. Other infections that cause anemia and weight loss, such as babesiosis, anaplasmosis, and haemonchosis, should be ruled out by examining a stained blood smear.



Picture 19: *Trypanosoma vivax*, bovine blood smear

Treatment and Control

Several drugs can be used for treatment. Drug resistance occurs and should be considered in refractory cases.

Table 5: Drugs commonly used for Trypanosomiasis in domestic animals

Drugs	Animal	Trypanosome	Main action
Diminazene aceturate	Cattle	vivax, congolense, brucei	Curative
Homidium bromide	Cattle, equids	vivax, congolense, brucei	Curative, some prophylactic activity
Isometamidium chloride	Cattle	vivax, congolense	Curative and prophylactic
Quinapyramine sulfate	Horses, camels, pigs, dogs	vivax, congolense, brucei, evansi, equiperdum, simiae	Curative

Control can be exercised at several levels, including eradication of tsetse flies and use of prophylactic drugs.

Tsetse flies can be partially controlled by frequent spraying and dipping of animals, aerial and ground spraying of insecticides on fly-breeding areas, use of insecticide-impregnated screens and targets, bush clearing, and other methods. The Sterile Insect Technique (SIT) has been used with success.

- Topic 3: Coccidiosis

Etiology

Coccidiosis is a usually acute invasion and destruction of intestinal mucosa by protozoa of the genera Eimeria or Isospora.

Coccidiosis is an economically important disease of cattle, sheep, goats, pigs, poultry and also rabbits, in which the liver as well as the intestine can be affected. In dogs, cats, and horses, coccidiosis is less often diagnosed but can result in clinical illness.

Epidemiology

Eimeria and *Isospora* typically require only one host in which to complete their life cycles.

Some species of *Isospora* have facultative intermediate (paratenic or transfer) hosts and a new genus name, Cystoisospora, has been proposed for these species of *Isospora*. Coccidia are host-specific, and there is no cross-immunity between species of coccidia.

Coccidiosis is seen universally, most commonly in young animals housed or confined in small areas contaminated with oocysts. Coccidia are opportunistic pathogens; if pathogenic, their virulence may be influenced by various stressors.

Therefore, clinical coccidiosis is most prevalent under conditions of poor nutrition, poor sanitation, or overcrowding, or after the stresses of weaning, shipping, sudden changes of feed, or severe weather.

Most animals acquire *Eimeria* or *Isospora* infections of varying severity when between 1 mo and 1 yr old. Older animals usually are resistant to clinical disease but may have sporadic inapparent infections. Clinically healthy, mature animals can be sources of infection to young, susceptible animals.

Pathogenesis

Infection results from ingestion of infective oocysts. Oocysts enter the environment in the feces of an infected host, but oocysts of *Eimeria* and *Isospora* are unsporulated and therefore not infective when passed in the feces. Under favorable conditions of oxygen, humidity, and temperature, oocysts sporulate and become infective in several days. During sporulation, the amorphous protoplasm develops into small bodies (sporozoites) within secondary cysts (sporocysts) in the oocyst.

In Eimeria spp, the sporulated oocyst has 4 sporocysts, each containing 2 sporozoites; in Isospora spp, the sporulated oocyst has 2 sporocysts, each containing 4 sporozoites.

When the sporulated oocyst is ingested by a susceptible animal, the sporozoites escape from the oocyst, invade the intestinal mucosa or epithelial cells in other locations, and develop intracellularly into multinucleate schizonts (also called meronts).

Each nucleus develops into an infective body called a merozoite; merozoites enter new cells and repeat the process. After a variable number of asexual generations, merozoites develop into either macrogametocytes (females) or microgametocytes (males).

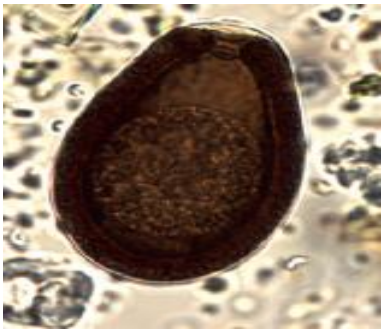
These produce a single macrogamete or a number of microgametes in a host cell. After being fertilized by a microgamete, the macrogamete develops into an oocyst. The oocysts have resistant walls and are discharged unsporulated in the feces.

Clinical Findings

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. Signs may include discharge of blood or tissue, tenesmus, and dehydration.

Diagnosis

Oocysts can be identified in feces by salt or sugar flotation methods. Finding appreciable numbers of oocysts of pathogenic species in the feces is diagnostic (>100,000 oocysts/g of feces in severe outbreaks), but because diarrhea may precede the heavy output of oocysts by 1–2 days and may continue after the oocyst discharge has returned to low levels, it is not always possible to find oocysts in a single fecal sample; multiple fecal examinations of one animal or single fecal examinations of animals housed in the same environment may be required.



Picture 20: Eimeria oocysts

Treatment

The life cycles of Eimeria and Isospora are self-limiting and end spontaneously within a few weeks unless reinfection occurs. Sick animals should be isolated and treated individually whenever possible to ensure delivery of therapeutic drug levels and to prevent exposure of other animals.

However, the efficacy of treatment for clinical coccidiosis has not been demonstrated for any drug, although it is widely accepted that treatment is effective against reinfection and should therefore facilitate recovery.

Most coccidiostats have a depressant effect on the early, first-stage schizonts and are therefore more appropriately used for control instead of treatment. Soluble sulfonamides are commonly administered orally to calves with clinical coccidiosis and are perceived to be more effective than intestinal sulfonamide formulations (boluses).

Amprolium is also administered orally to calves, sheep, and goats with clinical coccidiosis. Preventive treatment of healthy exposed animals as a safeguard against additional morbidity is an important consideration when treating individual animals with clinical signs.

Prevention

Prevention is based on limiting the intake of sporulated oocysts by young animals so that an infection is established to induce immunity but not clinical signs. Good feeding practices and good management, including sanitation, contribute to this goal. Neonates should receive colostrum. Young susceptible animals should be kept in clean and dry quarters.

Preventive administration of coccidiostats (sulfonamides and amprolium) is recommended when animals under various management regimens can be predictably expected to develop coccidiosis.

- Topic 4: Trichomoniasis

Etiology

Trichomoniasis is a venereal disease of cattle characterized primarily by early fetal death and infertility, resulting in extended calving intervals. The most important pathogen is *Trichomonas foetus* (a venereally transmitted), multi-flagellated organism of the reproductive tract of cattle

Epidemiology

The causative protozoan, *Trichomonas foetus*, is pyriform and ordinarily 10–15 × 5–10 µm, 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. Transmission can also occur when the semen from infected bulls is used for artificial insemination.

Clinical Findings

In the bull, there are no clinical signs once the infection is established. In the cow, early abortion is a characteristic feature although this is often undetected because of the small size of the foetus and the case may present as one of an irregular oestrus cycle.

The most common sign 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 mo of gestation and, apparently, live calves can be born to infected dams. Pyometra occasionally develops after breeding.

Diagnosis

- History and clinical signs are useful but are essentially the same as those of bovine genital campylobacteriosis
- Apart from a problem of infertility, which usually follows the purchase of a mature bull, diagnosis depends on the demonstration of the organism (*T foetus*).
- Diagnostic efforts are directed at bulls, because they are the most likely carriers.
- Vaginal mucus collected from the anterior end of the vagina by suction into a sterile tube, or prepucial washing from the bull, may be examined using a warm-stage microscope for the presence of the organisms.
- However, since the organism is often only present intermittently, the examination may require to be repeated several times

- Alternatively, on a herd basis, samples of vaginal mucus may be examined in the laboratory for the presence of specific agglutinins against laboratory culture of *T. foetus*.

Treatment and Control

Since the disease is self-limiting in the female only symptomatic treatment and sexual rest for three months is normally necessary. In the bull, slaughter is the best policy, although dimetridazole orally or intraveinously has been reported to be effective.

Artificial insemination from non-infected donors is the only entirely satisfactory method of control. If a return to natural service is contemplated, recovered cows should be disposed of since some may be carriers. *T foetus* can be safely eliminated from semen with dimetridazole.

L O 2.3 – Identify different types of manges

- Topic 1: Sarcoptic Mange (Scabies)

A. Definition

Sarcoptic mites as adults are microscopic, nearly circular in outline, and their legs are short, adapted for burrowing. The females, after mating with males on the surface of their host's skin, burrow into the living layers of the epidermis (mainly the stratum spinosum). They make tunnels horizontal to the surface of the skin. Here, eggs are laid and development of larvae and nymphs occurs.

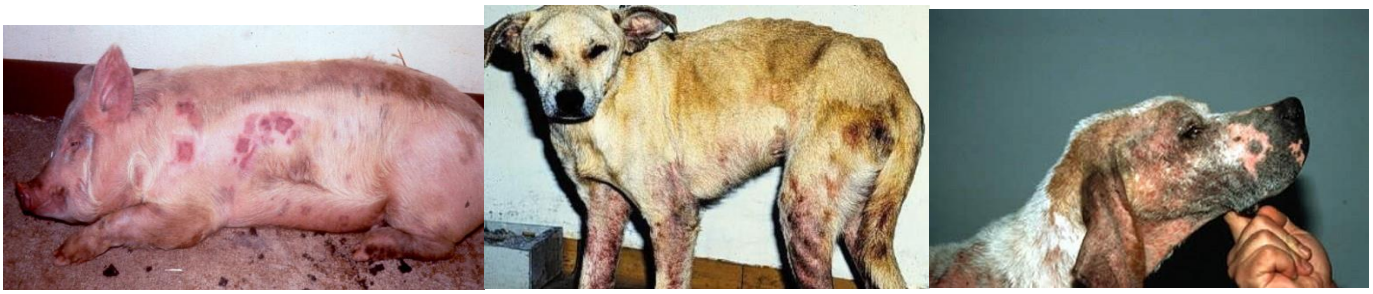
B. Etiology

- *Sarcopte scabiei* is the only species of Sarcoptes and to specify the causative agent of mange in different animal species it has varieties:
- *Sarcoptes scabiei* var *canis* infestation is a highly contagious disease of dogs found worldwide.
- *Sarcoptes scabiei var bovis* is a highly contagious disease spread by direct contact between infested and naive animals or by contaminated fomites. It can be transmitted to humans and is a reportable disease.
- *Sarcoptes scabiei var ovis* infests sheep, and *S scabiei var caprae* infests goats, throughout the world. However, *S scabiei var ovis* is rare in the USA. This mite infests nonwooly skin, usually on the head and face.
- Sarcoptes *scabiei var equi* is rare but is the most severe type of mange in horses.
- Sarcoptes *scabiei var suis*, is of primary importance in pigs worldwide.

C. Transmission and Distribution

The sarcoptic mange is worldwide and the mites have low mobility and transmission between hosts occurs only during close contact.

D. Clinical signs



Picture 21: Sarcoptic mange in pig and dogs

The first sign of infestation is intense pruritus due to hypersensitivity to mite products.

Affected animals have decreased reproduction, meat gain, and milk yield. In goats, *S scabiei* var *caprae* is responsible for a generalized skin condition characterized by marked hyperkeratosis.

In pigs, itching can be intense and associated with a hypersensitivity reaction to the mites. As the hypersensitivity subsides, typically after several months, the thickened, rough, dry skin is covered with grayish crusts.

In Cattle, Pruritus is intense, and papules develop into crusts; the skin thickens and forms large folds.

E. Lesions

In Dogs, primary lesions consist of papulocrustous eruptions with thick, yellow crusts, excoriation, erythema, and alopecia. Secondary bacterial and yeast infections may develop. Typically, lesions start on the ventral abdomen, chest, ears, elbows, and hocks and, if untreated, become generalized. Dogs with chronic, generalized disease develop seborrhea, severe thickening of the skin with fold formation and crust buildup, peripheral lymphadenopathy, and emaciation; dogs so affected may even die.

In Cattle, lesions caused by this burrowing mites start on the head, neck, and shoulders and can spread to other parts of the body. The whole body may be involved in 6 wk.

In small ruminants, lesions start usually on the head and neck and can extend to the inner thighs, hocks, brisket, ventral abdomen, and axillary region. Both *S scabiei var ovis* and *S scabiei var caprae* are zoonotic.

In Horses, Lesions start as small papules and vesicles that later develop into crusts. Alopecia and crusting spread, and the skin becomes lichenified, forming folds. If infestations are not treated, lesions may extend over the whole body, leading to emaciation, general weakness, and anorexia.

In Pigs, Lesions due to infestation with *S scabiei var suis* usually start on the head, especially the ears, then spread over the body, tail, and legs.

F. Diagnosis

Diagnosis is best performed by combining different approaches: dermatitis score recorded at slaughter, scratching index, observation of clinical signs of mange, ear or skin scrapings for microscopic examination, and ELISA for detection of specific antibodies.

G. Treatment

Injectable doramectin and ivermectin are labeled for use against *S scabiei var suis* and are considered highly effective treatments. In some instances, a second dose of macrocyclic lactone 14 days later may be necessary for complete resolution in swine, lime sulfur dips are repeated at intervals of 3–7 days to treat mange, unlike in other species in which they are repeated every 12 days, lime sulfur spray can be applied to suckling pigs

Certain spray formulations of permethrin are labeled for use against mites on swine, but it is generally not considered the compound of choice. If permethrin is used, animals should be wet thoroughly with the product and re-treated in 14 days.

A single treatment of oral moxidectin at 400 mcg/kg, have effectively treated psoroptic, chorioptic, and sarcoptic mange in horses.

The following compounds are approved for use against sarcoptic mange mites in cattle at the labeled injectable and pour-on dosages: doramectin, eprinomectin, and ivermectin. Hot lime sulfur dips or sprays may be used, following the label instructions for species-specific dilution, with treatment repeated at 12-day intervals as needed, usually for a maximum of three treatments.

In dogs, Systemic treatments of scabies are based on administration of macrocyclic lactones, selamectin is given as a spot-on formulation at 6 mg/kg. Even in ivermectin-sensitive breeds. Another is the imidacloprid-moxidectin formulation. The recommended dosage for milbemycin oxime is 2 mg/kg, PO, weekly for 3–4 week;

In some countries, moxidectin is also registered for treatment of scabies. Other endectocides, such as milbemycin oxime and ivermectin, which are not registered for treatment of sarcoptic mange in dogs,

For topical treatment, hair can be clipped, the crusts and dirt removed by soaking with an antiseborrheic shampoo, and an acaricidal dip applied. Lime sulfur is highly effective and safe for use in young animals; several dips 7 days apart are recommended. Amitraz is an effective scabicide, although it is not approved for this use. It should be applied as a 0.025% solution at 1- or 2-wk intervals for 2–6 wk. In addition, the owner must observe certain precautions to avoid self-contamination. Fipronil spray was reported to be effective but should be considered an aid in control rather than a primary therapy. Benzyl-benzoate is an insecticide with an unknown mode of action. It is effective against most ectoparasites, but is used only on dogs infested with sarcoptic mange.

- Topic 2: Psoroptic Mange

A. Definition

This reportable disease, caused by *Psoroptes ovis*, does not spread to humans.

B. Etiology

Psoroptic mange in cattle is caused by infestation with *Psoroptes ovis*. Current taxonomic and systematic classification of Psoroptes spp indicates that *P ovis* and *P cuniculi* (ear canker in rabbits, ear mange in sheep and goats) are strains or variants of the same species, with *P ovis* being found primarily on the backs and flanks of infested animals and *P cuniculi* in the ears.

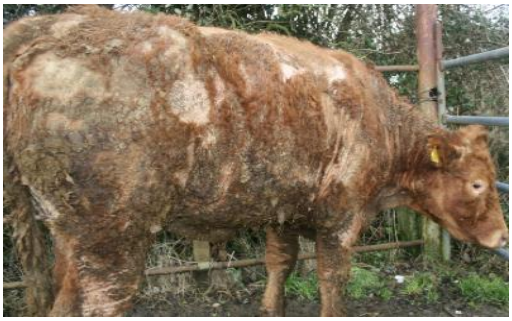
Psoroptes ovis is a highly contagious and severe infestation of sheep. Psoroptes ovis (formerly P equi) and P cuniculi (likely a variant of P ovis) both infest horses. P ovis is rare in horses.

C. Transmission and distribution

Transmission between hosts is readily accomplished by contagion during flocking contact and also on fomites such as scraps of sheep's wool because these relatively large and robust mites can survive for one to two weeks off their host.

P ovis is a common parasite of cattle with a distribution limited to continental Europe and parts of the USA. This mite has been eradicated from sheep in Canada, New Zealand, and the USA. However, sheep scab persists in many countries, including Africa and some in Europe.

D. Clinical signs



Picture 22: Psoroptic Mange in cattle

P cuniculi typically infests the ears of goats but can spread to the head, neck, and body. Intense pruritus leads to large, scaly, crusted lesions that develop in more densely haired or woolly parts of the body.

Disease can range from subclinical to scaling, crusting, inflammation, alopecia, ear scratching, head shaking, and rubbing of ears and head to alleviate irritation. Although the course is chronic, the prognosis is good with appropriate treatment.

Clinical signs of P ovis infestation are rare in dairy cattle. These mites pierce host tissue and feed on serum and other fluid secretions from the bite wound. Exudates coagulate to form thick, scabby crusts. Alopecia is common with exudative dermatitis. Infestations are intensely pruritic, with papules, crusts, excoriation, and lichenification on the shoulders and rump initially, spreading to cover almost the entire body. Secondary bacterial infections are common in severe cases. Death in untreated calves, weight loss, decreased milk production, and increased susceptibility to other diseases can occur.

E. Lesions

In cattle, lesions may cover almost the entire body; secondary bacterial infections are common in severe cases. Death in untreated calves, weight loss, decreased milk production, and increased susceptibility to other diseases can occur.

In Horses, lesions start as papules and alopecia and develop into thick, hemorrhagic crusts. Psoroptic mites are more easily recovered from skin scrapings than are sarcoptic mites.

However, infestations can produce lesions on thickly haired regions of the body, such as under the forelock and mane, at the base of the tail, under the chin, between the hindlegs, on the udder, and in the axillae. *P cuniculi* can sometimes cause otitis externa in horses and may cause head shaking.

In small ruminants, Lesions begin on the back and side but may become generalized and cover a large portion of the body. Animals bite, lick, and scratch in response to the pruritus, which results in wool loss and secondary bacterial infection. If affected sheep are not treated, infested animals may become emaciated and anemic and possibly die.

F. Diagnosis

Based on clinical signs and Microscopical examination from deep scratching of the affected area.

G. Treatment

Treatment can be done by spray dipping or vat dipping; topical application of nonsystemic acaricides; and oral, topical, or injectable formulations of systemic drugs.

The following treatments are labeled for use against *P ovis* in cattle at the labeled dosages: injectable doramectin, injectable ivermectin, and moxidectin (pour-on and injectable formulations). Hot lime sulfur dips or sprays may be used, following the species-specific dilution labeled on the package, with treatment repeated at 12-day intervals as needed, usually for a maximum of three treatments. Of these, only pour-on moxidectin and hot lime sulfur are approved for use on dairy animals.

If permethrin is used, the animals should be thoroughly wet with the product and re-treated in 10−14 days. Other treatments are available and labeled for the control of mange, including flumethrin pour-on (2 mg/kg, repeated 10 days later), 0.3% coumaphos, 0.1% phoxim, 0.075% diazinon, and 0.025–0.05% amitraz.

H. Control

Control is done by cleaning and disinfection of these sites, or by using traps for the mites. Vaccination against some species of mites has been tested experimentally.

The sheep-scab mite Psoroptes ovis is the target for such control because of its wide distribution, serious economic importance, and because it feeds on inflammatory exudate which contains antibodies reactive against antigens in the mite's gut.

- Topic 3: Chorioptic Mange (Leg Mange)

A. Definition

This reportable disease, caused by *Chorioptes bovis*, does not affect humans. The pastern areas of the legs are preferred sites for the mites.

B. Etiology

Chorioptic mange in cattle is caused by infestation with *Chorioptes bovis or C texanus.* Species of Chorioptes are not host specific, and *C bovis* can be found on domestic ruminants and horses throughout the world.

Chorioptes bovis infests sheep and goats worldwide. Prevalence of *C bovis* is more common in rams than ewes or lambs.

Chorioptic mange is caused by infestation with *Chorioptes bovis* (formerly *C equi*) and is the most common form of mange in horses.

C. Transmission and distribution

Transmission is by direct contact of infested and naive hosts. *C bovis* can live off their host for up to 3 weeks and can be transmitted to cattle through contact with contaminated fomites and housing. The mange is worldwide.

D. Clinical signs

Picture 23: Chorioptic mange caused by Chorioptes bovis

A high proportion of cattle can be infested without showing clinical signs. Most sheep are subclinically infested with *C bovis.* However, *C bovis* can cause exudative dermatitis on the lower legs and scrota of rams (scrotal mange). While feeding, *C bovis* irritates the host skin, causing abrasions that become contaminated with secretions and feces from the mites. Most cattle are subclinically infested with *C bovis*.

E. Lesions

Lesions start as papules, crusts, and ulcerations on the legs and can spread to the udder, scrotum, tail, and perineal area.

Lesions caused by C bovis start as a pruritic dermatitis affecting the distal limbs around the foot and fetlock. Papules are seen first, followed by alopecia, crusting, and thickening of the skin. A moist dermatitis of the fetlock develops in chronic cases. Infested horses may stamp their feet or rub one foot against the opposite leg or object. Chorioptic mange is a differential diagnosis for “greasy heel” in draft horses.

In Cattle, Lesions include nodules, papules, crusts, and ulcers that typically begin at the pastern and spread up the legs to the udder, scrotum, tail, and perineum. Self-trauma and alopecia may be evident.Lesions and clinical signs appear in late winter and spontaneously regress during summer months.

F. Diagnosis

Chorioptic mange is less pathogenic than sarcoptic or psoroptic mange in cattle. Diagnosis is by observation of mites in skin scrapings.

G. Treatment

The following treatments are approved for use against C bovis at the labeled dosages: pour-on doramectin, eprinomectin (both pour-on and injectable formulations), and pour-on moxidectin. Hot lime sulfur dips or sprays are labeled for use against chorioptic mites and may be used following the label directions for species-specific dilution. Lime sulfur treatment should be repeated at 12-day intervals if needed.

Hot lime sulfur is labeled for use against Chorioptes in horses (see above). Treatment is aided by clipping long hair from infested areas. Although not labeled for treatment of mange in horses, oral ivermectin at 200 mcg/kg given for two doses 14 days apart (field studies), or a single treatment with oral moxidectin at 400 mcg/kg, has effectively treated chorioptic mange in horses.

- Topic 4: Demodectic Mange

A. Definition

Demodicosis can occur as dense, localized infestations with inflammation of the epidermis in hair follicles. This leads to pustular folliculitis and indurated (thickened) plaques within the skin. Demodex mites infest hair follicles and sebaceous glands.

B. Etiology

Demodectic mange in **horses** is caused by infestation with *Demodex equi* or *D caballi*. **In pigs**, caused by infestation with *Demodex phylloides*. *Demodex ovis* infests sheep, and *D caprae* infests goats. Demodectic mange in **sheep** is not common, whereas *D caprae* are relatively common in **goats**. Three species of Demodex are known to infest **cattle**: *D bovis, D ghanensis,* and *D tauri. D bovis* is the most common and infests hair follicles of cattle worldwide.

In **cats**, Demodex cati is thought to be a normal inhabitant of feline skin. One other species of Demodex (named D gatoi) is shorter, with a broad abdomen, and is found only in the stratum corneum. **Canine** demodicosis occurs when large numbers of *Demodex canis* mites.

C. Transmission and distribution

In dogs, the mites are transmitted from dam to puppies during nursing within the first 72 hours after birth. The mites spend their entire life cycle on the host, and the disease is not considered to be contagious. In cattle, transmission of *D bovis* occurs through close contact of infested and naive hosts, with the transfer of mites from infested dams to neonates being the primary route. *Demodex bovis* is transferred from cow to calf while nursing and may cause considerable damage to hides.

D. Clinical signs

Picture 24: Demodectic Mange in cattle and Juvenile-onset generalized demodicosis in dog

In Pigs, clinical signs of *D phylloides* infestation include reddening of the skin, pustules, and alopecia. Although rare in domestic pigs, *D phylloides* infestation can be common in wild boars without overt signs of clinical disease. In wild boars, the highest prevalence and greatest numbers of D phylloides were found in sebaceous glands in eyelids and cheeks. D phylloides can also be found around the eyes, mouth, snout, ventral neck, ventrum, and thighs.

In horses, Demodex mites infest hair follicles and sebaceous glands. *D equi* lives on the body, and *D caballi* on the eyelids and muzzle. Demodectic mange is rare in horses but can manifest as patchy alopecia and scaling or as nodules.

In goats, nonpruritic papules and nodules develop, especially over the face, neck, shoulders, and sides or udder. Infestation with *D bovis* in cattle, is usually subclinical, and infestation may extend for many months.

In feline, it causes a contagious, transmissible, superficial demodicosis that is frequently pruritic and can be generalized. In follicular localized demodicosis, there are one or several areas of focal alopecia most commonly on the head and neck. In generalized disease, alopecia, crusting, and potential secondary pyoderma of the whole body are seen.

E. Lesions

Demodectic mange in goats occurs most commonly in kids, pregnant does, and dairy goats. The nodules contain a thick, waxy, grayish material that can be easily expressed; mites can be found in this exudate.

In cattle, Lesions consist of follicular papules and nodules, especially over the withers, neck, back, and flanks. Invasion by *D bovis* results in chronic inflammation, with formation of ulcers, abscesses, and fistulae due to follicular rupture or secondary staphylococcal infection. Pruritus is absent.

In dogs, Lesions consist of areas of focal alopecia, erythema and/or hyperpigmentation, and comedones. Pruritus is usually absent or weak.

F. Diagnosis

Diagnosis is not difficult; deep skin scrapings or hair plucking typically reveal mites, eggs, and larval forms in high numbers.

G. Treatment

A number of other protocols are commonly used for refractory generalized demodicosis. Among macrocyclic lactones, milbemycin oxime (0.5–1 mg/kg/day, PO), moxidectin, and ivermectin have all demonstrated varying degrees of effectiveness. Moxidectin is available as a spot-on formulation in combination with a flea product (imidacloprid) and should be given at 2.5 mg/kg at 1–4 weeks intervals.

Weekly lime sulfur dips (2%) are safe and usually effective; amitraz (0.0125%–0.025%) has been used but is not approved for use in cats and can cause anorexia, depression, and diarrhea.

In horses, therapy is rarely done, although there is limited evidence that the macrocyclic lactones may be effective. Lesions have also been reported to resolve without treatment and there is no reliable treatment in pigs.

- Topic 5: Psorergatic Mange (Itch Mite)

A. Definition

It is caused by *Psorergates spp*. Affected animals show mild, patchy alopecia and pruritus. The disease does not cause significant economic losses; thus, animals are usually not treated.

B. Etiology

Psorobia ovis (formerly *Psorergates ovis*) is a common skin mite of sheep in Africa, Australia, New Zealand, and South America.
Psorobia bos (formerly *Psorergates bos*) is a small mite that lives in the superficial layers of cattle skin.

C. Transmission and distribution

This species is prevalent in Australia, New Zealand, southern Africa, and the Americas. All breeds of domestic sheep are susceptible. Transmission is by direct contact or by fomites.

D. Clinical signs and lesions

Psorergates bos causes pruritus, but little clinical harm to cattle. In contrast, *Psorergates ovis* feeding on sheep induces inflammatory and hypersensitive reactions in the epidermis, resulting in intense pruritus and formation of scabs. Further damage to the skin and fleece of sheep occurs when the sheep groom compulsively. Economic loss is incurred by damage and depilation of the wool fleece.
In most instances, *P bos* is nonpathogenic, and few cattle exhibit clinical signs of infestation. On rare occasions, mild pruritus, alopecia, and increased licking and rubbing have been attributed to infestation with *P bos*.

E. Diagnosis

Diagnosis is based on clinical signs. Because of their small size, the psororgates are difficult to find in skin scrapings.

F. Treatment

Treatments effective against sarcoptic, chorioptic, and psoroptic mange in sheep are expected to be efficacious for psorergatic mange, like the macrocyclic lactone products. Several dips and injectable ivermectins and milbemycins are effective in controlling this infestation.

• Topic 6: Notoedric Mange (Feline Scabies)

A. Definition

This rare, highly contagious disease of cats and kittens is caused by *Notoedres cati*, which can opportunistically infest other animals, including humans.

B. Etiology

Nontoedric mange (Feline scabies) is a contagious disease of cats and kittens is caused by *Notoedres cati*.

C. Transmission and distribution

This mange is worldwide and the mite and its life cycle are similar to the sarcoptic mite. Pruritus is severe.

D. Clinical signs and lesions

Crusts and alopecia are seen, particularly on the ears, head, and neck, and can become generalized. Mites can be found quite easily in skin scrapings.

E. Treatment

Mites can be found quite easily in skin scrapings. Treatment consists of both topical and systemic therapies. Non-approved but effective and safe treatments include selamectin (6 mg/kg, spot-on) and moxidectin (1 mg/kg, spot-on, in the imidacloprid-moxidectin formulation). Ivermectin (200 mcg/kg, SC) has also been used. Another effective topical therapy is lime sulfur dips at 7-day intervals.

• Topic 7: Otodectic Mange

A. Definition

Otodectes cynotis mites are a common cause of otitis externa especially in cats but also in dogs. Mites that belong to the family Psoroptidae are usually found in both the vertical and horizontal ear canals but are occasionally seen on the body.

B. Etiology

Otodectes cynotis is the only species likely to cause otodectic mange also called Otodectes surface feeding mite causing ear canker.

C. Transmission and distribution.

The disease is extremely contagious, and spreads by direct contact between cats. The disease is often seen in dogs as well, and it has also been described in humans. Infestations in dogs or cats can be transferred between hosts by close contact. Survival of mites in the environment is not thought to be a significant factor in transmission.

D. Clinical signs and Lesions

Clinical signs include head shaking, continual ear scratching, and ear droop. Pruritus is variable. Pruritus is variable but may be severe. Dark brown cerumen accumulation in the ear and suppurative otitis externa with possible perforation of the tympanic membrane may be seen in severe cases.

E. Diagnosis

Mites in the ear may be observed with an otoscope or on swabs of the ear canal. It is important to remember that ear mites can be found at other locations and may cause signs or lesions specific to those locations.

F. Treatment and Control

Affected and in-contact animals should receive appropriate parasiticide treatment in the ears. Systemic therapies have been approved and include topically applied selamectin and moxidectin. Direct applications to the external ear canal of cats using approved ivermectin and milbemycin formulations are also effective. As a general rule, ear cleansing with an appropriate ceruminolytic agent is indicated with any therapy.

Several products have demonstrated efficacy against *O. cynotis,* but are not label approved. These include other formulations of ivermectin (dog and cat; variable dosages), doramectin (cat), fipronil (cat), afoxolander (dog and cat; label dose), fluralaner (dog and cat; label dose), and sarolaner (dog; label dose).

Cleansing of the ear canal prior to treatment is always recommended.

When secondary infections with bacteria or fungi occur, these conditions should also be treated.

Learning Unit 3 – Perform diagnosis for parasitic diseases

L O 3.1 – Perform anamnesis

- Topic 1: Techniques of history taking

Taking a history from a patient is a skill necessary for examinations and afterwards as a practicing technician. It tests both your communication skills as well as your knowledge about what to ask. Specific questions vary depending on what type of history you are taking but if you follow the general framework below you should gain good marks in these stations. This is also a good way to present your history.

Procedure (Steps)

Step 01: Introduce yourself, identify the owner and your patient and gain consent to speak with them. You should wish to take notes as you proceed.

Step 02 - Presenting Complaint (PC): This is what the patient tells you is wrong, for example: chest pain.

Step 03 - History of Presenting Complaint (HPC): Gain as much information you can about the specific complaint.

Step 04 - Past Medical History (PMH): Gather information about a patients other medical problems (if any).

Step 05 - Drug History (DH): Find out what medications the patient is taking, including dosage and how often they are taking them, for example: once-a-day, twice-a-day, etc. At this point it is a good idea to find out if the patient has any allergies.

Step 06 - Review of Systems (ROS)

Gather a short amount of information regarding the other systems in the body that are not covered in your HPC.

These are the main systems you should cover: Cardio Vasculo System, Respiratory, GI, Neurology, Genitourinary/renal, Musculoskeletal and integumentary and ophthalmology.

Step 07 - Summary of History

Complete your history by reviewing what the owner has told you about the patient. Repeat back the important points so that the owner can correct you if there are any misunderstandings or errors.

You should also address what the patient thinks is wrong with them and what they are expecting/hoping for from the consultation. A useful acronym for this is **ICE** [I]deas, [C]oncerns and [E]xpectations.

Step 8 - Patient owner Questions / Feedback

During or after taking their history, the owner may have questions that they want to ask you. It is very important that you don't give them any false information. As such, unless you are absolutely sure of the answer it is best to say that you will ask your seniors about this or that you will go away and get them more information (e.g. leaflets) about what they are asking. These questions aren't necessarily there to test your knowledge, just that you won't try and 'blag it'.

Step 9: When you are happy that you have all of the information you require, and the owner has asked any questions that they may have, you must thank him for his time.

- Topic 2: Owner identification

A. Owner identification

The farmer's full identification should be provided for the following reasons:

- To make a follow up of the animal after the first treatment
- If the animal has been stolen, it will help to get it back to the owner
- In case of any outbreak after the treatment, this information will help the District Veterinary Officer or other person who has in charge of animal health to trace the situation of that disease.

The farmer identification should contain the main information as follows:

- Farmer's name
- Address: Village/ Cell/ Sector and District
    - Phone number if he/she has any.
    - Age/ Sex

B. Animal signalment

Signalment: Assists with proper identification of the patient, diagnosis, and predilections to traits and conditions as some conditions may be species, breed, gender, age, and color specific.

Note: Most signalment information does not change over time. Exceptions to this include acquired markings, age, reproductive status, and means of identification.

Objectives

- To collect, classify, and record required patient information.
- To identify the relevance and interrelationship of the information as it relates to: Diagnosis, Treatment, Nursing care and Patient progress

What to Include

- **Date and time of admittance:** Establishes a starting point for intervention and monitoring
- **Patient identification:** Name, tag number or electronic ID
- **Species**
- **Breed**
- **Gender and reproductive status**
- **Age:** In years, months, weeks, or days depending on age of patient
- **Color:** In order of predominance

- Topic 2: Animal history

History taking or anamnesis is the process of obtaining information on the animal patient about its illness, onset of illness and feeding practice through careful questioning of the owner. In Veterinary practice, the disease is presented indirectly in the form of a complaint by the owner or the attendant. Thus, it is very necessary to have all the information from the owner.

A. Immediate history (present history)

This relates to the sequence of events associated with the period of time that the animal has been ill. It is important to determine the chronological order in which the more important changes in behaviour and in physiological functions were observed.

Specific questions should therefore be centred on such aspects as appetite for food or drink, defaecation, urination, respiration, sweating, physical activity, milk production, growth, gait, posture, voice, odour, etc. The questions should be designed to ascertain the degree and nature of any departure from normal in any of these functions.

Locations of the problems

Following up and attention at the complaint that a farmer has to say and from there you can tentatively say the likely system involved in that condition, for instance:

- Digestive system involvement will be shown as absence of rumination, appetite, bloat or diarrhea.
- Respiratory system involvement will be indicated presence of nasal discharge, coughing, dyspnea.
- Urinary system involvement will be manifested as frequent urination, passing red coloured or cloudy urine.
- Musculo-skeletal and nervous system involvement will be manifested as lameness, inco-ordination, and paralysis.

Nature of illness

The clinician should be able to assess and find out the time of onset of disease, any change management practices and signs noticed by the farmers.

- To assess to know the duration of disease whether it is peracute, acute, subacute or chronic
- To know number of animal diseased and morbidity rate and mortality rate of animals
- Determine whether any drug has been given for animals, before patience come to clinic for assurance

And the following question should be pointed:

- When did the farmer notice the disorder? (time)
- Did it occur suddenly/slowly? (acute /sub-acute / chronic nature)
- What were the signs noticed? (anorexia/drop in milk yield/ others)
- Are the animal fed / grazed in pasture / forest grazed? (getting information on management practices e.g. ketosis seen in stall fed animals, while babesiosis seen in forest grazed animals)
- Is there any other animal affected with similar condition in the same herd / in other farmer's herd in the village (to find out if the disease is rapidly spreading)?
- Ask if there has been any introduction of new animal to the herd / village (sick animal may have been bought from affected area and disease has started)
- Is the affected animal vaccinated against food-and-mouth disease (FMD), anthrax, hemorrhagic septicemia (HS), Black quarter (BQ) (to find out if the animal is protected against common diseases)

B. Past Histories

History of past disease may be co-related to the present illness. Past history will also give idea if such condition prevailed previously in the area.

Ask if such condition was reported previously too (reveal endemic nature of disease, or occurrence of a new disease)

- Does this occur at certain period of time? (find out the seasonal occurrence of the disease)
- Was the disease reported form other places in the locality? (area of spread / occurrence can be found out)
- Has any animal recovered from such a sickness? (to aid in prognosis)
- Is the disease restricted to certain age group / sex? (BQ is seen in animals between 1 – 3 years of age in both sexes.

C. Nutritional history

Nutritional diseases are, in most instances, group problems, so that a number of animals are more or less simultaneously affected. Domestic pets, however, are still occasionally found to be suffering from serious nutritional deficiency syndromes.

During the grazing season, a study of the pasture composition, along with identification of specific poisonous species, including ergotized grass or rye, or those which possess the ability selectively to absorb potentially toxic elements (selenium, copper), is advisable in certain circumstances. Stall-fed animals, in comparison with those at pasture, are in most instances fed on a nutritionally balanced diet; the quality of pasture is not easily assessed, so that, with the exception of certain recognized diseases, a nutritional deficiency may exist for quite a time before it is identified. A sudden change from stall to lush pasture feeding, during the spring season, may predispose to hypomagnesaemic tetany or rickets, even though the herbage com-position is normal. Grazing animals, more particularly when adolescent, are exposed to the risk of acquiring various parasitic infestations, e.g. various forms of parasitic gastroenteritis in cattle and sheep, lungworm infestation in cattle, strongylosis in horses.

Housed animals are exposed to the risk of being over- or underfed or of receiving diets which are incomplete or inadequate in respect of some essential constituents. Farm compound foods are more likely than those commercially produced to be nutritionally inadequate. The quality of the ingredients may have an undesirable effect.

Feeding methods should also be investigated because they may contribute to low productivity or disease. Inadequate trough space for pigs and calves and for older cattle in yards leads to over¬eating on the part of the vigorous animals and partial starvation in the smaller, weaker ones.

- Topic 3: Environmental History

The environment in which the animals were kept at the time of the onset or just before the onset of the illness should be carefully examined. The animals may be housed or outside or inside.

A. Farm history

Environmental history can be divided into outdoor environment and indoor environment:

Outdoor environment history: It regard to the topography of land where animals are reared, vegetation, type of agriculture practiced in the locality, use of chemicals in agriculture (pesticide, weedicide) and system garbage disposal in the area. Animals that are grazed are likely to be infested by parasites and prone to vector borne diseases like babesiosis, trypanosomiasis, or animals that are grazed in the marshy area including paddy filed are likely to be infested by liver fluke etc.

Indoor environment history: It regards with the types of animal house. The following should be look assessed how is the house of animals is designed:

- If there is proper ventilation,
- In the rural area, traditionally animals are housed in the ground floor of the house where there hardly any ventilation and this will predispose the animals to respiratory diseases,
- If the animals are stall fed check
- If the animals are provided with enough drinking water,
- If the floor is dry and clean, damp and dirty floor may lead to mastitis in milking animals,
- If the bedding materials are used
- If the materials are changed daily of topped daily to keep the animals dry and clean.
- If the height of the roof is at a required level,
- If it is too low, in hot places animals may be subjected to heat stress in summer months,
- If it is too high animals will be exposed to rain.

B. Vicinity and Climate

The consideration of the surroundings and circumstances should include an enquiry into such aspects of animal husbandry as nutrition, breeding policy, housing, etc., which might reveal information of diagnostic significance. In relation to diet, any recent change in character or constitution should be ascertained.

This brings into focus the need to determine whether the ailing animal is being house-fed or grazing. Within this context, it is necessary to consider the geographical and seasonal incidence of disease for a particular region. Knowledge of the local topography is of value in relation to vector-borne diseases including louping-ill, babesiasis, anaplasmosis, trypanosomiasis, African horse sickness, Rift Valley fever and blue tongue, etc., and a number of other diseases such as fascioliasis, hypocuprosis, cobalt deficiency, etc.

L O 3.2 – Carry out general examination

General examination

A medical practitioner examines a patient for any possible medical signs or symptoms of a medical condition. It generally consists of a series of questions about the patient's medical history followed by an examination based on the reported symptoms. Together, the medical history and the physical examination help to determine a diagnosis and devise the treatment plan. This data then becomes part of the medical record.

- Topic 1: Animal observation

A. At distance and Close

Most farmers know their animals very well and can notice even subtle changes in an animal's behavior such as isolation, loss of appetite, lethargy, etc., which can be the first indications of an illness or injury. Animals exhibiting these minor changes in behavior should be monitored closely and a veterinarian should be contacted if the signs do not disappear in a short period of time.

More visible signs such as coughing, excessive salivation, diarrhea, abortions, and neurological disorders (e.g., circling, head pressing, stumbling, blindness) are obvious concerns. At the first sign of problems, a veterinarian should be called to make a farm visit for evaluation. If possible, the animals' caretakers should be trained to determine heart rate, respiratory rate, temperature, and dehydration status.

How to observe

Observe each animal daily to assess its health and well-being. Carefully observe each animal from head to tail, including each part of its body and behavior, for abnormalities that may indicate a potential problem. Also, look at the feces and discharge from the animal if there is any, for signs of abnormalities. Establish a consistent method for conducting your daily observations. If you aren't sure how, consult with a veterinarian on how to perform daily observations of animals. Checklists and similar tools are helpful for ensuring daily observations are consistent and effective in assessing animal health and well-being.

When to observe

Things to consider when conducting your observations (not limited to the following):

Physical Attributes: How does the animal look? Are there any abnormalities? Consider handling the animal if safe and appropriate to do so. Not only will you be able to use touch as an observational tool, you will also promote social interactions and neurological well-being.

Body: The hair coat may be Glossy or dull, hair loss. Under or over weight: You can see the ribs. Observe Itching, Scabbing, Lumps, Bumps, Parasites.

Head: Eyes – clear, discharge, winking or blinking excessively. Ears/Nose – clean, discharge or buildup. Mouth/Teeth/Gums – clean, buildup (tartar), bleeding or injuries.

Limbs/Extremities: Feet – Nails/Claws/Hooves proper length, wounds or abnormalities. Joints – calluses, mobility, lameness.

Illness in Poultry: Signs could include: Sudden increase in bird deaths in your flock; Sneezing, gasping for air, coughing, and nasal discharge; Watery and green diarrhea; Lack of energy and poor appetite; Drop in egg production or soft- or thin-shelled, misshapen eggs; Swelling around the eyes, neck, and head; Purple discoloration of the wattles, combs, and legs (avian influenza; Tremors, drooping wings, circling, twisting of the head and neck, or lack of movement (exotic Newcastle disease).

Behavior: Is the animal acting normal? Is the movement or gait normal? Is the animal lethargic or displaying behaviors consistent with sickness, stress, or boredom? Does the behavior change when you move closer or farther away from the animal? For example, does the animal act stoic and appear to be normal upon closer examination, but when walking away or from a distance the animal appears to have a limp or injury or displays different behavior.

Environment: Is the animal's environment safe? Are environmental controls (temperature, humidity, shelter from elements) adequate for the species and the season? Is the animal: Huddling or shivering due to cold? Panting, or laying stretched out due to heat? Food and water receptacles used or left alone? Eating and drinking enough? Elimination habit normal? Loose or abnormal stools? Vomit? Regurgitated food/water?

Record Keeping

Keep records of daily observations and contacts with the attending veterinarian

- Animal identification.
- Date.
- What the problem was.
- Method of contact to the attending veterinarian (phone, text, email, visit).
- The attending veterinarian's advice.

Capture: Capture information on daily observations, including photographs and videos, using a tablet or smartphone that can immediately connect to a main office computer for review.

Create: Create a system to log daily observation recordings, such as notebook/binder or spreadsheet/database.

Look: Look at every animal and enclosure at the beginning of each day. Make a list of what needs to be fixed, repaired, clipped, moved, medicated, etc. This creates a "to-do list" for the day for planning and maintenance in addition to accomplishing daily observations.

Provide: Provide training to all staff with responsibility for conducting daily observation on recognizing physical and behavioral concerns, and, the established protocol within the facility for documenting and communicating with the attending veterinarian.

Monitor: Monitor appetite and diet consumption as potential early indicators of concern. Include weekly weight checks in the observation program.

- Topic 2: Supplementary information (related to the suspicion)

All observations regarding the animal including behavior, physical appearance, feces, vomit, and leftover food should be recorded on the Animal's Observation Log in the left-hand side. All observations regarding the enclosure including integrity, fallen branches, low areas needing dirt etc should be reported on the Maintenance Observation Log.

L O 3.3 – Carry out specific examination

- Topic 1: Examination of body systems

The body systems are vital while performing specific examination:

- Cardiovascular system (chest pain, dyspnea, ankle swelling, palpitations) are the most important symptoms and you can ask for a brief description for each of the positive symptoms.
- Respiratory system (cough, haemoptysis, wheezing, pain localized to the chest that might increase with inspiration or expiration).
- Gastrointestinal system (change in weight, flatulence and heart burn, dysphagia, abdominal pain, vomiting, bowel habit).
- Genitourinary system (frequency in urination, pain with micturition, urine color, any urethral discharge, altered bladder control like urgency in urination or incontinence, menstruation and sexual activity).
- Endocrine system (weight loss, polydipsia, polyuria, increased appetite and irritability).
- Musculoskeletal system (any bone or joint pain accompanied by joint swelling or tenderness, aggravating and relieving factors for the pain and any positive family history for joint disease).

- Skin (any skin rash, recent change in cosmetics and the use of sunscreen creams when exposed to sun).

- Topic 2: Systematical physical examination

Temperature: The body temperature is taken using a mercury or digital electronic thermometer placed carefully into the rectum. Thermometer reading will reveal if the temperature of animal being examined is normal, above normal (fever) of below normal (subnormal). Based on this finding action taken will vary. Fever: denotes the elevation of body temperature of animal above normal.

Palpation and hydration status: Palpation: Palpation is used to detect the presence of pain in a tissue by noting increased sensitivity and use fingers, palm, back of the hand, and fist, in order to get the information about the variation in size, shape, consistency and temperature of body parts and lesions, e.g., the superficial lymph nodes.

Percussion: Method of examination in which part of body to be examined is struck with sharp blow using fingertips to produce audible sound. Sound thus emitted will indicate the nature of the tissue / organ involved for example rumen when bloated will emit drum like sound. Some of the organs that can be examined by percussion are: gastro-intestinal tract, abdomen and thorax, frontal and nasal sinuses.

Auscultation (heart and lungs): This is a technique of listening to the sounds produced from organs in the abdominal and thoracic cavities. This method used to examine the lung, trachea, heart and certain parts of the alimentary tract.

Abdominal auscultation and assessment of rumen function: This is to listen the sounds produced by the rumen motion in the abdomen. The examination of rumen is performed by inspection, palpation, percussion and auscultation; stomach tube can be used as well. In bloat case, the left side would be bulged, and the motility would be decreased.

Succussion: It is the method used to determine the presence of fluid in the body cavities like thoracic and abdominal cavity. Here the animal is shaken from side to side to set fluid into motion so that audible fluid sound is produced. This is difficult in large animals and can be applied only in small animals like dogs and cats

Olfactory and visual inspection: Formal testing of vision divides this function into two basic aspects: (1) central or cone vision, and (2) peripheral or rod vision. Peripheral vision is the greatest part of the visual field, whereas central vision represents a relatively small segment of the projected visual world. Nevertheless, it is mainly central vision that is responsible for visual acuity and color vision.

Olfaction is tested by having a patient with eyes closed, sniff a relatively familiar odor from a small vial, occluding the nares alternately to test each side separately.

Examination of mucous membranes: Visible mucous membrane examined by visual inspection to note the presence of lesions, discharge, glaucoma, nystagmus. The perspective of this examination is to identify pale and discolored mucus membranes; assess problems of oral cavity and deranged appetite

L O 3.4 – Make diagnosis and prognosis

- Topic 1: Concepts and definitions

A. Suspicion

Information about a group or individual coupled with suspicions or prejudices of medical staff could influence how diagnoses are made, by affecting what examinations are performed and how quickly people are investigated, which can affect rates of diagnosis. This can be termed diagnostic suspicion bias.

B. Presumption

Similarly, in medicine, a presumptive diagnosis identifies the likely condition of a patient, and a confirmatory diagnosis is needed to confirm the condition.

C. Confirmation

Confirmatory tests provide a positive identification of the disease in question. This is called qualitative analysis, and determines what diseases are present.

Importance: The purpose of a presumptive test for a sample is to determine whether or not it is likely that this agent is present; in other words, a positive result indicates the possibility of agent. A positive result of a confirmatory test for sample allows one to conclude that agent is present

- Topic 2: Types of diagnosis

Clinical diagnosis

The estimated identification of the disease underlying a patient's complaints based merely on signs, symptoms and medical history of the patient rather than on laboratory examination or medical imaging.

Arriving at a diagnosis is often complex, involving multiple steps:

- taking an appropriate history of symptoms and collecting relevant data
- physical examination
- generating a provisional and differential diagnosis
- testing (ordering, reviewing, and acting on test results)
- reaching a final diagnosis
- consultation (referral to seek clarification if indicated)
- providing discharge instructions, monitoring, and follow-up
- documenting these steps and the rationale for decisions made

Differential diagnosis

In medicine, a differential diagnosis is the distinguishing of a particular disease or condition from others that present similar clinical features. Differential diagnostic procedures are used by physicians to diagnose the specific disease in a patient, or, at least, to eliminate any imminently life-threatening conditions. Often, each individual option of a possible disease is called a differential diagnosis (e.g. acute bronchitis could be a differential diagnosis in the evaluation of a cough, even if the final diagnosis is common cold).

A differential diagnostic procedure is a systematic diagnostic method used to identify the presence of a disease entity where multiple alternatives are possible.

A standard of care differential diagnosis has four steps. Patient safety requires that the physician:

- Gathers all information about the patient and create a symptoms list. The list can be in writing or in the physician's head.
- Lists all possible causes (candidate conditions) for the symptoms.
- Prioritizes the list by placing the most urgently dangerous possible causes at the top of the list.
- Rules out or treats possible causes, beginning with the most urgently dangerous condition and working down the list.

Etiological (Laboratory) diagnosis

The act or process of identifying or determining the nature and cause of a disease or injury through evaluation of patient history, examination, and review of laboratory data.

For example, direct identification of parasitic infections are usually diagnosed from samples of faeces, urine, blood and tissue by microscopic observation of simple saline, stained, blood and tissues smears.

A. Diagnosis of helminthes infections

To diagnose gastro-intestinal parasites, the parasites or their eggs/larvae must be recovered from the digestive tract of the animal or from faecal material. These are subsequently identified and quantified. This chapter presents diagnostic techniques within the reach of most laboratories to identify and quantify parasite infections from the examination of faecal material.

The following are the main tasks involved in this process:

- Collection of faecal samples
- Separation of larvae from faecal material, and their concentration of eggs
- Microscopical examination of prepared specimens
- Preparation of faecal cultures or incubation
- Isolation and identification of larvae from cultures

It is important to understand the following basic limitations of faecal examination in the diagnosis of gastro-intestinal parasitism.

- The demonstration of parasite eggs or larvae in the faeces provides positive evidence that an animal is infected but does not indicate the degree of an infection.
- The failure to demonstrate eggs or larvae does not necessarily mean that no parasites are present; they may be present in an immature stage or the test used may not be sufficiently sensitive.
- There is generally no correlation between the numbers of eggs/larvae per gram of faeces and the number of adult nematodes present in cattle. An exception to this may occur in a primary infection in young grazing animals during their first exposure. There are some indications that the correlation is stronger in sheep and goats with mixed infections.

Various factors can limit the accuracy and significance of a faecal egg count.

- There is a fairly regular fluctuation in faecal egg output.
- Eggs are not evenly distributed throughout the faeces.
- The quantity of faeces passed will affect the number of eggs per unit weight.
- The egg output is influenced by the season of the year (large infections may be acquired during rainy seasons).
- The resistance of the host can depress or entirely inhibit the egg production of parasites.
- Immature worms do not indicate their presence by producing eggs.
- Immunity may result in a marked extension of the prepatent period and a lower egg output by female parasites.

- An egg count often refers to the total number of eggs of a mixture of species, which differ widely both in their biotic potential and their pathogenicity.
- Eggs may not be detected due to low numbers of them or due to low test sensitivity.

A.1. Collection of faeces

Faecal sample should preferably be collected from the rectum and examined fresh. If it is difficult to take rectal samples, then fresh faeces can be collected from the field or floor. A plastic glove is suitable for collection, the glove being turned inside out to act as the receptacle. For small pets a thermometer or glass rod may be used.

Ideally, about 5g of faeces should be collected, since this amount is required for some of the concentrations methods of examination.

Since eggs embryonate rapidly the faeces should be stored in the refrigerator unless examination is carried out within a day. For samples sent through the post the addition of twice the volume of 10% formalin to the faeces will minimize development and hatching.

A.2. Methods of examination of faeces

Several methods are available for microscopic examination to detect the presence of eggs or larvae. However, whatever method of preparation is used, the slides should first be examined under lower power since most eggs can be detected at this magnification.

If necessary, higher magnification can be employed for measurement of the eggs or more detailed morphological differentiation.

A.2.1. Qualitative techniques for separating and concentrating eggs, larvae and oocysts in faecal samples

Three methods are described as follow:

- Simple test tube flotation
- Simple flotation
- Sedimentation technique (for trematode eggs)

Simple test tube flotation

The simple test tube flotation method is a qualitative test for the detection of nematode and cestode eggs and coccidia oocysts in the faeces.

It is based on the separating of eggs from faecal material and concentrating them by means of a flotation fluid with an appropriate specific gravity. This is a good technique to use in initial surveys to establish which groups of parasites are present.

Equipment

- Beakers or plastic containers
- A tea strainer (preferably nylon) or double layer cheesecloth
- Measuring cylinder or other container graded by volume
- Fork, tongue blades or other type of stirring rod
- Test tube
- Test tube rack or a stand
- Microscope
- Micro slides, cover slips
- Balance or teaspoon
- Flotation fluid (see the Appendix to this handbook for formulation)

Procedure

- Put approximately 3 g of faeces (weigh or measure with a pre-calibrated teaspoon) into Cont 1.
- Pour 50 ml flotation fluid into Container 1.
- Mix (stir) faeces and flotation fluid thoroughly with a stirring device (tongue blade, fork).
- Pour the resulting faecal suspension through a tea strainer or a double-layer of cheesecloth into Container 2.
- Pour the faecal suspension into a test tube from Container 2.
- Place the test tube in a test tube rack or stand.
- Gently top up the test tube with the suspension, leaving a convex meniscus at the top of the tube and carefully place a cover slip on top of the test tube.
- Let the test tube stand for 20 minutes.
- Carefully lift off the cover slip from the tube, together with the drop of fluid adhering to it, and immediately place the cover slip on a microscope slide.

Simple flotation method

This is another good technique for use in initial surveys. In addition, it can be used in conjunction with the McMaster technique to detect low numbers of eggs (when present below the McMaster sensitivity of 50 eggs per gram of faeces).

Equipment

- Two beakers or plastic containers
- A tea strainer or cheesecloth
- Measuring cylinder or other container graded by volume
- Fork, tongue blades or other type of stirring rod
- Test tube (dry)
- Microscope
- Micro slides, cover slips
- Balance or teaspoon
- Flotation fluid

Procedure

- Put approximately 3 g of faeces (weigh or measure the faeces with a pre-calibrated teaspoon) into Container 1.
- Pour 50 ml of flotation fluid into Container 1.
- Mix (stir) the contents thoroughly with a stirring device (tongue blade, fork).
- Pour the resultant faecal suspension through a tea strainer or a double-layer of cheesecloth into Container 2.
- Leave the container to stand for 10 minutes.
- Press a test tube to the bottom of the filtrate, lift it quickly and transfer a few drops adhering to the surface to a micro slide.
- The test tube ought to touch the microslide for at least 2-4 seconds for the drops to run off.
- Mount the cover slip on the micro slide for microscopical examination.

Sedimentation technique (for trematode eggs)

Principle

The sedimentation technique is a qualitative method for detecting trematode eggs *(Paramphistomum)* in the faeces. Most trematode eggs are relatively large and heavy compared to nematode eggs. This technique concentrates them in sediment.

Application

This is a procedure to assess the presence of trematode infections. It is generally run only when such infections are suspected (from previous post-mortem findings on other animals in the herd/flock area), and is not run routinely. The procedure can be used to detect liver fluke *(Fasciola)* and *Paramphistomum* eggs.

Equipment

- Beakers or plastic containers
- A tea strainer or cheesecloth
- Measuring cylinder
- Stirring device (fork, tongue blade)
- Test tubes
- Test tube rack
- Methylene blue
- Micro slide, cover slips
- Balance or teaspoon
- Microscope

Procedure

- Weigh or measure approximately 3 g of faeces into Container 1.
- Pour 40-50 ml of tap water into Container 1.
- Mix (stir) thoroughly with a stirring device (fork, tongue blade).
- Filter the faecal suspension through a tea strainer or double-layer of cheesecloth into Container 2.
- Pour the filtered material into a test tube.
- Allow to sediment for 5 minutes.
- Remove (pipette, decant) the supernatant very carefully.
- Resuspend the sediment in 5 ml of water.
- Allow to sediment for 5 minutes.
- Discard (pipette, decant) the supernatant very carefully.
- Stain the sediment by adding one drop of methylene blue.
- Transfer the sediment to a microslide. Cover with a coverslip.

Microscopical examination of prepared samples

The prepared samples on micro slides from the simple test tube flotation method, the simple flotation method and the sedimentation method are examined under a microscope at the magnifications listed in Table below.

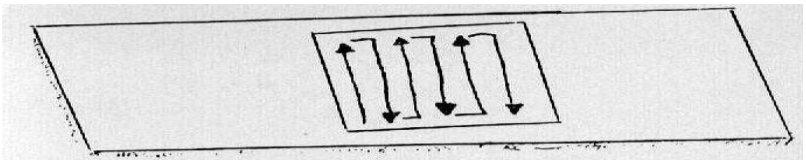


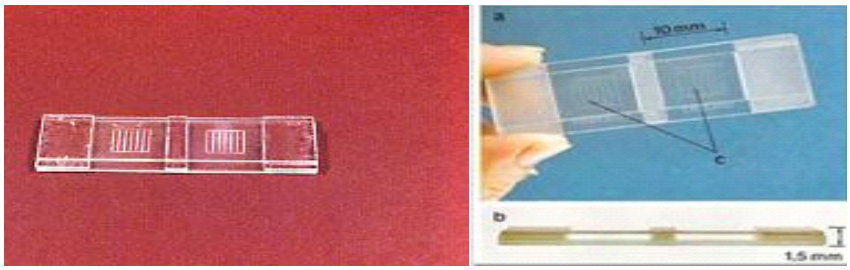
Table 6: Microscopical examination magnification

Magnification	Parasites
10 x 10	Nematode and cestode eggs
10 x 40	Coccidia oocysts
10 x 4	Trematode eggs

WARNING: In case of a time delay between processing the sample and reading the count, egg numbers may decline dramatically. Also, eggs may change their appearance, becoming created and "ghost-like". It is therefore advisable to prepare only a few samples at a time. These changes can be prevented by keeping prepared samples in the refrigerator after mixing. Using the salt-sugar solution as flotation fluid also reduces the morphological changes.

A.2.2. McMaster counting technique

The McMaster counting technique is a quantitative technique to determine the number of eggs present per gram of faeces (epg.). A flotation fluid is used to separate eggs from faecal material in a counting chamber (McMaster) with two compartments. The technique described below will detect 50 or more e.p.g. of faeces.



Picture 25: Mac Master Cell

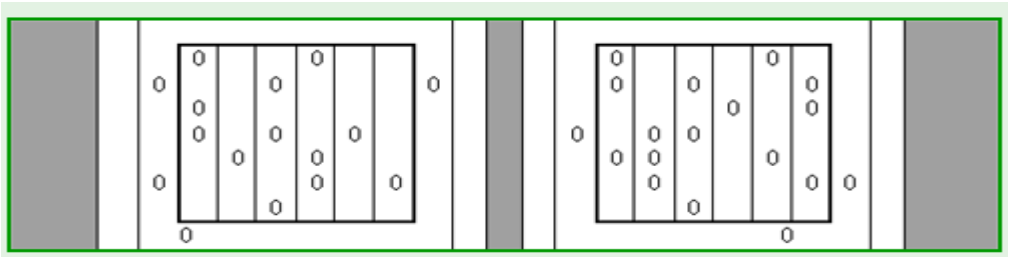
This technique can be used to provide a quantitative estimate of egg output for nematodes, cestodes and coccidia. Its use to quantify levels of infection is limited by the factors governing egg excretion.

Equipment

- Beakers or plastic containers
- Balance
- A tea strainer or cheesecloth
- Measuring cylinder
- Stirring device (fork, tongue depressor)
- Pasteur pipettes and (rubber) teats
- Flotation fluid (see the Appendix to this handbook for formulation)
- McMaster counting chamber
- Microscope

Procedure

- Weigh 4 g of faeces and place into Container 1.
- Add 56 ml of flotation fluid.
- Mix (stir) the contents thoroughly with a stirring device (fork, tongue blade).
- Filter the faecal suspension through a tea strainer or a double-layer of cheesecloth into Container 2.
- While stirring the filtrate in Container 2, take a sub-sample with a Pasteur pipette.
- Fill both sides of the McMaster counting chamber with the sub-sample.
- Allow the counting chamber to stand for 5 minutes (this is important)
- Examine the sub-sample of the filtrate under a microscope at 10 x 10 magnifications.
- Count all eggs and coccidia oocytes within the engraved area of both chambers.
- The number of eggs per gram of faeces can be calculated as follows: Add the egg counts of the two chambers together.
- Multiply the total by 50. This gives the e.p.g. of faeces. (Example: 12 eggs seen in chamber 1 and 15 eggs seen in chamber 2 = (12 + 15) x 50 = 1350 e.p.g.)
- In the event that the McMaster is negative (no eggs seen), the filtrate in Container 2 can be used for the simple flotation method (in the steps f, g and h).

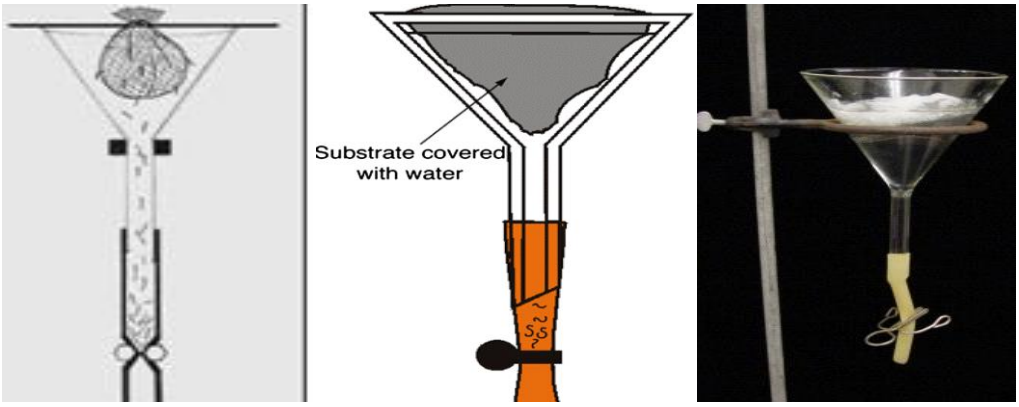


12 eggs seen in chamber 1 and 15 eggs seen in chamber 2
= (12 +15) x 50 = 1350 e.p.g.

A.2.3. Isolation and identification of lungworm larvae and infective larvae harvested from faecal cultures (the Baermann technique)

The Baermann technique is used to isolate lungworm larvae from faecal samples and infective larvae from faecal cultures. It is based on the active migration of larvae from faeces suspended in water and their subsequent collection and identification.

This is a procedure for harvesting infective larvae for identification purposes.



Picture 26: The Baermann technique

Equipment

- Funnel (size according to need)
- Funnel stand
- Rubber or plastic tubing
- Rubber bands
- Clamp or spring clip
- Cheesecloth or screen
- Simple thin stick (about 15 cm long)
- Strainer
- Microscope
- Test tube
- Pasteur pipette and Small petri dish(es)

Procedure

- Fit a short piece of tubing which is closed at one end with a clamp or spring clip, to the stem of a funnel of appropriate size.
- Support the funnel by a stand.

- Weigh or measure about 5-10 g of faecal culture/faeces and place it on a piece of double-layer cheesecloth.
- Form the cheesecloth around the faeces as a "pouch".
- Close the pouch with a rubber band.
- Fix a supporting stick under the rubber band - Step 1
- Fix a supporting stick under the rubber band - Step 2
- Place the pouch containing faecal culture material or faeces in the funnel. Trim the surplus cheesecloth off.
- Fill the funnel with warm water about 35^{o}C, covering the faecal material.
- Leave the apparatus in place for 24 hours, during which time larvae actively move out of faeces and ultimately collect by gravitation in the stem of the funnel.
- Examination for longhorns
- Draw a few ml of fluid from the stem of the funnel into a small petri dish.
- Examine under dissecting microscope for live lungworm larvae (L1).
- For positive samples a transfer of larvae to a micro slide for identification at 10 x 10 magnifications may be required. It is important to differentiate between *Muellerius capillaris* and other species as the treatment is different.
- Examination for infective larvae from faecal cultures
- Draw 10-15 ml of fluid from the stem of the funnel into a test tube or other container.
- Leave the tube to stand for 30 minutes. Remove the supernatant with a Pasteur pipette.
- Transfer a small aliquot of the remaining fluid using a Pasteur pipette to a micro slide, add a drop of iodine and cover with a cover slip.
- Examine fewer than 10 x 10 magnifications.
- Repeat steps m and n until 100 larvae have been identified.
- The counts for each species provide an estimate of the composition (%) of the parasite population of the host.

B. Protozoal infections diagnosis

B.1. Giemsa Stain

Giemsa stain is valuable for staining large number of slides simultaneously, for the laking and staining of thick films and for the diagnosis of parasitic diseases. It is not as good as Wright'stain for routine hematological procedures because the staining time is longer, preliminary fixation is required and the neutrophilic granular detail is not good.

Preparation

- *T*ake 0.3g of Giemsa stain in mortar. Add 25.0ml of glycerin in small amount while grinding. Add 25.0ml acetone free methyl alcohol. This makes stock solution.
- Sometimes difficult is encountered in the preparation of Giemsa stain, so it is recommended that this stain be purchased in liquid form.

Procedure

Thin blood films (only)

Wear gloves when performing this procedure.

- Fix the blood film for 3-5 minutes in methyl alcohol and air dry
- Prepare working solution by adding 1ml of Giemsa stain, to 9ml neutral distilled water or buffered distilled water (pH 6.8).
- Place the blood film in Coplin jar having diluted stain for 30-45 minutes.
- Wash the film with neutral water, then drain dry and examine. *Note: Excessive washing will decolorize the film.*
- Let air dry in a vertical position.

Thick blood films (only)

- Allow film to air dry thoroughly for several hours or overnight. Do not dry films in an incubator or by heat, because this will fix the blood and interfere with the lysing of the RBCs. *Note: If a rapid diagnosis of malaria is needed, thick films can be made slightly thinner than usual, allowed to dry for 1 h, and then stained.*
- DO NOT FIX.
- Stain with diluted Giemsa stain (1:50, vol/vol) for 50 min. For a 1:50 dilution, add 1 ml of stock Giemsa to 50 ml of buffered water in a Coplin jar.
- Wash by placing film in buffered water for 3 to 5 min.
- Let air dry in a vertical position.

Thin and thick blood films on the same slide

- Allow the thick film to air dry thoroughly
- Fix air-dried film in absolute methanol by dipping the film briefly (two dips) in a Coplin jar containing absolute methanol. Be sure not to get the alcohol or its fumes on the thick film by slightly tilting the slide.
- Remove and let air dry with the *thick film up*. Be sure slide is thoroughly dry before staining. Introducing even a minute amount of methyl alcohol into the stain dilution will interfere with the lysing of the RBCs in the thick films.

- Stain the entire slide with diluted Giemsa stain (1:50, vol/vol) for 50 min. For a 1:50 dilution, add 2 ml of stock Giemsa to 40 ml of buffered water in a Coplin jar. Place the slide in the stain, *thick film down* to prevent the debris caused by dehemoglobinization from falling onto the thin film.
- Rinse the thin film by briefly dipping the film in and out of a Coplin jar of buffered water (one or two dips). Wash the thick film for 3 to 5 min. Be sure that the thick film is immersed but *do not allow the water to cover any part of the thin film.*
- Let air dry in a vertical position with the **thick film down**

C. Diagnosis of Mites

Arthropods of veterinary interest are divided into two major groups, the Insecta and the Arachnida. Most are temporary or permanent ectoparasites, found either in or on the skin, with the exception of some flies whose larval stages may be found in the somatic tissues of the host.

The two groups of Arachnids of veterinary importance are the ticks and mites. In all cases diagnosis of infection of mites will depend on the collection and identification of the parasite(s) concerned.

Diagnosis is based on the history of severe pruritus of sudden onset, possible exposure, and involvement of other animals, including people. Making a definitive diagnosis is sometimes difficult because of negative skin scrapings. Concentration and flotation of several scrapings may increase chances of finding the mites, eggs, or feces.

Several extensive superficial scrapings should be done of the ears, elbows, and hocks; nonexcoriated areas should be chosen. A centrifugation fecal flotation using sugar solutions may reveal mites or eggs. A specific and sensitive commercially available ELISA to detect specific antibodies has been developed and may be useful. Because mites can be difficult to detect, if Sarcoptes is on the differential diagnosis list but no mites are found, a therapeutic trial is warranted.

• Topic 3: Prognosis

Prognosis (Greek: "fore-knowing, foreseeing") is a medical term for predicting the likely or expected development of a disease, including whether the signs and symptoms will improve or worsen (and how quickly) or remain stable over time; expectations of quality of life, such as the ability to carry out daily activities; the potential for complications and associated health issues; and the likelihood of survival (including life expectancy).

A prognosis is made on the basis of the normal course of the diagnosed disease, the individual's physical and mental condition, the available treatments, and additional factors.

Prognosis vs. Diagnosis

People often confuse the terms prognosis and diagnosis. The difference between the two is that while a prognosis is a guess as to the outcome of treatment, a diagnosis is actually identifying the problem and giving it a name, such as depression or obsessive-compulsive disorder.

Factors That Affect Prognosis

Different factors can affect the prognosis of each individual. These factors include:

- Age
- Gender
- Medical and/or family history
- How the disease or disorder is presenting
- Response to treatment
- Particular symptoms and how long they have been present
- Whether or not there are other illnesses or conditions present
- What treatment or treatments are being used

A. Favourable

A favorable prognosis means a good chance of treatment success. For example, the overall 5-year relative survival rate for testicular cancer is 95%. This means that most men diagnosed with the disease have a favorable prognosis. Prognosis depends on the stage of the cancer at diagnosis.

B. Unfavourable

An unfavorable prognosis means a bad chance of treatment success For example, the overall 5-year relative death rate for testicular cancer is 5%. This means that men diagnosed with the disease have an unfavorable prognosis.

Antiparasitic drug or *parasiticide* is a poison that is more toxic to parasites than to their hosts. This is the principle of selective toxicity.

As a matter of fact, it is sometimes easier to explain the deleterious effects that parasiticides frequently exert on the host than to explain how they kill parasites. Parasiticide drugs are divided into three main groups: insecticides drugs, anthelmintic drugs and antiprotozoal drugs, although not every drug fits nicely into just one of these sections.

L O 4.1 – Select appropriate treatment

- Topic 1: Identification of antiparasitic drugs

A. Anthelminthic drugs

Table 7: The major groups of anthelminthics

Parasites	Chemical group	Drugs
Nematodes	Piperazines	Piperazine salts, diethylcarbamazine
	Imidazothiazoles/tetrahydropyrimidines	Tetramisole, levamisole, morantel, pyrantel
	Benzimidazoles/pro-benzimidazoles	Thiabendazole, mebendazole, parbendazole, fenbendazole, oxfendazole, albendazole, oxibendazole, cambendazole, flubendazole, febantel, thiophanate, netobimin
	Avermectins/milbemycins	Ivermectin, doramectin, abamectin, moxidectin, milbemycin oxide
	Organophosphates	Dichlorvos, haloxon, trichlorfon (metriphonate)
	Salicylanilides/substituted phenols	Nitroscanate, closantel
Trematodes	Salicylanilides/substituted phenols	Nitroxynil, rafoxanide, oxyclozanide, brotianide, niclofolan, closantel
	Benzimidazoles/pro-benzimidazoles	Triclabendazole, albendazole, netobimin
Cestodes	Salicylanilides/substituted phenols	Niclosamide
	Others	Praziquantel, bunamidine, arecoline

B. Antiprotozoal drugs

Amprolium: is a thiamine analogue that is used to prevent and treat intestinal coccidiosis (Table 10-1). It is available as a feed additive for livestock and is sometimes administered in food or drinking water to puppies and kittens.

Benzimidazoles (Fenbendazole and Albendazole): Benzimidazoles bind to β-tubulin within a variety of helminths and protozoa. Fenbendazole is widely used to treat giardiasis in dogs and cats. It is safer than metronidazole, can be administered to young animals, and has higher efficacy, although treatment failure can still occur. A second course of treatment or administration of fenbendazole in combination with metronidazole can be effective in refractory cases.

Nitroimidazoles: Protozoa reduce nitroimidazoles to nitro anion free radicals, which cause damage to parasite DNA. Metronidazole, ronidazole, and tinidazole have primarily been used to treat enteric protozoal infections. Benznidazole is specifically used to treat infections with *Trypanosoma cruzi*.

Metronidazole

Metronidazole: is used to treat giardiasis in dogs and cats, although efficacy may be as low as 50%. It also has activity against amoebic infections. Metronidazole can be combined with fenbendazole for refractory giardiasis.

Tinidazole: is a 5-nitroimidazole that has amoebicidal, giardicidal, trichomonicidal, and anaerobic bactericidal activity.

Ronidazole: Ronidazole is the drug of choice for treatment of Tritrichomonas foetus infections, which are less responsive to metronidazole and tinidazole.

Nitozoxanide: Nitazoxanide is a nitrothiazolyl-salicylamide derivative that has activity against Giardia spp., Cryptosporidium spp., Sarcocystis neurona, some anaerobic bacteria. Reports of nitazoxanide use in dogs and cats have been rare, and its efficacy in dogs and cats is largely unknown.

Tick borne diseases

The treatment of Babesiosis is dependent upon babesia and availability of a particular drug in the market. Diminazene aceturate and imidocarb are used.

Theileriosis (*Theileria parva* and *T.annulata*) is best treated by Buparvaquone in most countries and The Tetracyclines (Chlortetracycline, Tetracycline) can be used.

The efficacy of Iimidocarb dipropionate against anaplasomosis and babesiosis in cattle and Oxytetracycline are used for the treatment. Diminazene (Diminazene acceturate) also can be used.

C. Drugs for mites

The efficacy of **ivermectin** long-acting injection (3.15 % ivermectin w/v) formulation was evaluated in cattle with induced Sarcoptes *scabiei var. bovis* or Psoroptes ovis infestations.

In dogs, ivermectin may be used in the treatment of mites (demodectic mange, scabies, and ear mites), intestinal parasites (hookworms, roundworms), and capillaria. In cats, ivermectin may be used to treat ear mites and cat scabies.

Selamectin is given as a spot-on formulation at 6 mg/kg. This drug appears to be safe, even in ivermectin-sensitive breeds.

Another is the **imidacloprid-moxidectin** formulation, which may be used on dogs as young as 7 wk of age. In some countries, moxidectin is also registered for treatment of scabies

Other endectocides, such as milbemycin oxime. The recommended dosage for milbemycin oxime is 2 mg/kg, PO, weekly for 3–4 wk; potential.

Acaricide treatment prevents adrenocortical hyperplasia as a long-term stress reaction to psoroptic mange in cattle.

- Topic 2: Selection criteria of the antiparasitic drugs

A. The selection criteria

The selection criteria of the parasiticid depends on the followings: animal species, breeds, physiological status, parasite species, diseases, age, treatment, value of animals, presentation of medicine, side effects, availability, financial resources of animal owner.

The choice of chemical product to be used in diseases prevention is very important in companion animals breeding. It must take into account:

- Type of animal (animal species, breeds, age and physiological status): one drug may be used for a given animal but no useful for another.
- Causal agent of diseases :vaccines are used particularly for microbial diseases, antiparasitics are used in parasitic diseases prevention ;
- Prevalence of the disease in the area :some diseases may be found in region while there not found in other

- Localisation of the disease :some medicines can be used in internal area of the body (products used for deworming),while others are used in external area to fight against for example external parasites;
- Efficiency and specificity of the drugs (some drugs are more indicated more than others);
- Price of drug (the choice is more oriented to cheaper drugs);
- Side effects: more the drug has no or less side effects more is likely to be more chosen;
- Period of action: more the drug has short period of action more is likely to be more chosen.

Note that, before use of any drug, the information given by manufacturers must read and strictly respected.

B. Properties of anthelmintic compounds

An ideal anthelmintic should possess the following properties:

- **It should be efficient against all parasitic stages of a particular species**: it is also generally desirable that the spectrum of activity should include members of different genera.
- **It should be non-toxic to the host**: or at least have a wide safety margin. This is important in the treatment of groups of animals such as a flock of sheep, where individual body weights cannot easily be determined, rather than in the dosing of individual companion animals.
- **It should be rapidly cleared and excreted by the host**; otherwise long withdrawal periods would be necessary in meat and milk producing animals. However, in certain circumstances and in certain classes of animals, drug persistence is used to prophylactic advantage.
- **Anthelmintics should be easily administered**, otherwise they will not be readily accepted by owners; different formulations are available for different domestic animal species.
- **The cost of an anthelmintic should be reasonable**. This is of special importance in pigs and poultry where profit margins may be narrow.

L O 4.2 – Administer appropriate medicine

- Topic 1: Drug administration route and techniques

A route of administration in pharmacology and toxicology is the path by which a drug, fluid, poison, or other substance is taken into the body. Routes of administration are generally classified by the location at which the substance is applied. Common examples include oral and intravenous administration.

Routes can also be classified based on where the target of action is. Action may be topical (local), enteral (system-wide effect, but delivered through the gastrointestinal tract), or parenteral (systemic action, but delivered by routes other than the Gastro-Intestinal tract).

A. Oral route (Per Os)

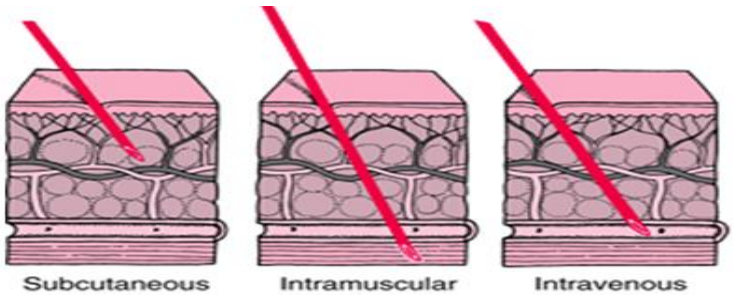
Many drugs can be administered orally as liquids, capsules, tablets, or chewable tablets. Because the oral route is the most convenient and usually the safest and least expensive, it is the one most often used. However, it has limitations because of the way a drug typically moves through the digestive tract. For drugs administered orally, absorption may begin in the mouth and stomach. However, most drugs are usually absorbed from the small intestine.

Picture 27: Oral route administration

B. Parenteral route

Administration by injection (parenteral administration) includes the subcutaneous, intramuscular, intravenous, and intrathecal routes.

Parenteral administration by injection



C. Local application of drug /Transdermal route (ointment)

Some drugs are delivered bodywide through a patch on the skin. These drugs are sometimes mixed with a chemical (such as alcohol) that enhances penetration through the skin into the bloodstream without any injection.

L O 4.3 – Follow up the case

- Topic 1: Regular monitoring for evolution and possible complications

Utilize daily monitoring forms for each animal in the shelter and provide training for care staff in utilizing these forms for recording all daily observations:

- Look for urine/fecal output before beginning cleaning and any signs of vomit.
- Record attitude prior to feeding in the morning
- Monitor appetite during feeding
- In general housing areas, if an animal appears sick, mark the cage so others are alerted
- Unless an animal requires immediate intervention, cleaning staff should not enter the cage of an ill animal until feeding and cleaning of the general population has been completed
- Follow appropriate procedures to notify veterinary staff or supervisors of problems or concerns
- Include weekly weight checks as part of a complete monitoring program (have weighing as part of the intake process as well)
- When animals remain in the shelter for longer than one month, perform a full physical exam including weight and body condition score by trained staff on a monthly basis

- Topic 2: Post treatment check up

After the treatment has finished, you will have regular checkups. These will allow the health care technician to monitor the animal patient health and wellbeing.

The care plans usually are:

- Provide a summary of the treatment set out a clear schedule for follow-up care,
- details for the health professionals involved in the treatment and any screening tests
- list any symptoms to watch out for, complications and possible long-term side effects
- identify any medical or psychosocial problems that may develop after treatment and ways to manage them
- Suggest ways to adopt a healthier lifestyle after treatment.

What do post treatment check-ups involve

During check-ups veterinarian may:

- assess animal patient recovery
- monitor and treat any ongoing side effects
- talk to the owner about any late treatment side effects to watch out for
- check any new symptoms
- do a physical examination
- ask if the owner have any concerns or questions
- discuss the general health and give healthy lifestyle advice

References

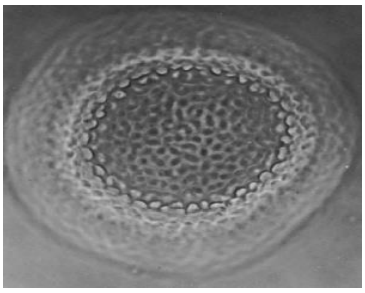
1. Bowman *Georgis'* (2014) *Parasitology for Veterinarians*, 10th Edition.
2. Dr. Leland S. Shapiro (2010) *Pathology and Parasitology for Veterinary Technicians*, Second Edition.
3. G. M. Urquhart, J. Armour, and Al (1996), *Veterinary parasitology*, Second Edition.
4. Dr. Anne M. Zajac, (2010) *Veterinary Clinical Parasitology,* eighth edition.
5. Kahn CM (2010) Merck *Veterinary Manual*. 10th edn. Whitehouse Station,Merck, NJ, USA
6. Radostits OM, Mayhew IG, Houston DM (2000) *Veterinary clinical examination and diagnosis*. London
7. Ajello SE (1998), *the Merck Veterinary Manual*, 8th edition, published by Merck and Company, INC, USA.
8. http://www.merckmanuals.com/home/infections/parasitic_infections/.html. (Retrieved on 20th July, 2020).
9. http://www.cdc.gov/ticks/life_cycle_and_hosts.html (Retrieved on 20th July, 2020).
10. http://www.med-chem.com/pages/lab_procedures/pdf/giemsa_blood_stain.pdf (Retrieved on 20th July, 2020).
11. https://www.msdvetmanual.com/integumentary-system/mange/mange-in-cattle (Retrieved on 24th July, 2020)
12. https://www.msdvetmanual.com/integumentary-system/mange/mange-in-sheep-and-goats (Retrieved on 24th July, 2020)
13. https://www.msdvetmanual.com/integumentary-system/mange/mange-in-dogs-and-cats (Retrieved on 24th July, 2020)
14. https://www.msdvetmanual.com/integumentary-system/mange/mange-in-pigs (Retrieved on 24th July, 2020)

15. https://www.msdvetmanual.com/integumentary-system/mange/mange-in-horses (Retrieved on 24th July, 2020)
16. https://veteriankey.com/antiprotozoal-drugs/ (Retrieved on 24th July, 2020)
17. https://www.researchgate.net/publication/49612208_Efficacy_of_various_antiprotozoal_drugs_on_bovine_babesiosis_anaplasmosis_and_thileriosis (Retrieved on 24th July, 2020)
18. https://pubmed.ncbi.nlm.nih.gov/25399813/ (Retrieved on 24th July, 2020)

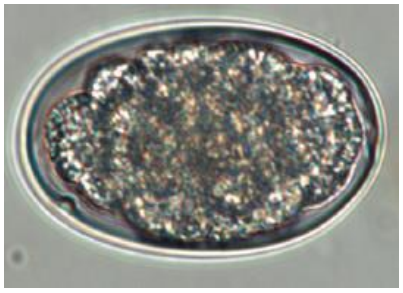
Appendices

Appendix 1: General identification of eggs, cysts, and larvae

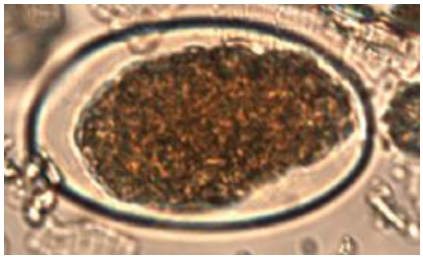
Oxyurid eggs



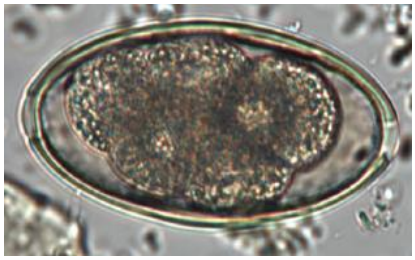
Toxocara canis egg



Strongylid egg

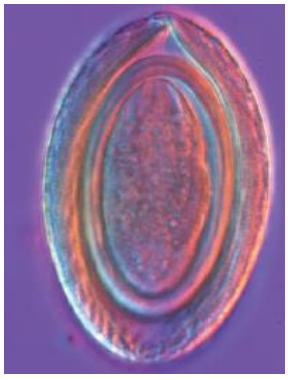


Oesophagostomum sp.



Syngamus sp. Egg

Trichinelloid egg



Acanthocephalan egg

Appendix 2: Brief description of TBDs diseases

TBD	Etiology	Transmission	Signs	Diagnosis	Treat, control
Theileriosis	T. parva T. annulata T. hirci (of shorts)	By *R. appendiculatus*, protozoa are inoculated in the host and reach in the lymph nodes	Increased t^{o} up to39.5^{o}C, lymphadenopathy, anorexia, emaciation, lacrima-tion, very low t^{0}, recumbency, dyspnea and death	Is by: .clinical signs .patholog y (smear) .serology (ELISA)	Buparvaquone (Butalex) and tetracycline. Control of ticks, animal vaccination
Anaplasmos is	A.marginale A. centrale A. ovis	It is transmitted by tick bite, mechanically by biting flies and contami-nated surgical instruments	.Fever (40^{0}C) .anorexia .constipation .anemia .Emaciation .Death in 24 to48 hrs	.Pathoge-nesis .clinical signs .pathologi cal feature Epidemi-ology	.Imidocarb (carbesia) .Tetracycline .water+oil Control is by acaricide, fencing, hand picking
Babesiosis	B. divergens B. major B. bigemina B. bovis B. motasi B. ovis B. canis B. caballi B. equi B. trautmani	Organism gets in cell of host by tick bite. It divides asexually (binary fission) within the RBC. The host cell rupture to liberate the organisms for next cell infection	.Fever .Haemoglobunuria (red water) .Jaundiced mucous .Increased respiration .Abortion .Death may occur .If not convulsions, emaciation and milk production loss. Diarhoea followed by constipation.	It is by: .History .Clinical signs .For confirmati on use blood film stained with GIEMSA	Different drugs: .Quinuroneum .Amicarbilide .Imidocarb .Pantamidine .Diminazene aceturate .The control is by tick control, cattle immunization by blood from carriers
Heart water	Cowdria ruminantium	Larvae of Abryomma tick become	It is characterized by: .High fever (to 42^{0}C	In general it is clini-cally	Tetracycline administered as early as

		infected when feeding on infected cattle. Transmission to the vertebrate host by gut contents or through the saliva of infected tick while feeding	in peracute form) .Nervous signs .Hydropercardium .Hydrothorax .Oedema of lungs .Oedema of brain .Death The disease is developed in: Peracute , acute or chronic forms	difficult, but it is best done at post mortem inspection (Looking on lesions).	possible may be effective. Control is by: .vector control .chemoprophylaxis (use of tetracycline) . Immunopro-phylaxis

Appendix 3: Brief description of other protozoal diseases

Disease	Etiology	Transmission	Sings	Diagnosis	Treatment and control
Coccidiosis	Coccidia in families of: Eimeridae and sarcocystidae In eimeridae, we will identify three genera: .Eimeria .Isospora and .Cryptosporidium	The infection to the host is by ingestion of sporulated oocyst. The next will be the liberation of sporocyst and sporozoi-tes to penetra-te the epithet-lial cells	The disease is characterized by: .anorexia .diarrhoea .Poor growth .vomiting may also occur .in poultry, the coecal coccidiosis ia character-rized by: soft faeces cont-ainning blood, chicks are dull with drooping	Diagnosis is based at post mortem examination of dead birds. Oocysts may be detected by fecal examination	.Sulfonamide .Sulphaquinoxaline .Amprolium .thopabate. The prevention of avian coccidiosis is based on the combination of good management and the use of anticoccidial medications in feeds and

			feathers		water.
Toxoplasmosis	Toxoplasma gondi	The direct transmission will be cat to cat ingesting mature bradyzoites. Indirect cycle is the the cat is infected by ingesting toxoplama infected animals	In dog: .fever .anorexia .diarrhoea .pneumonia .neurvous signs In cats: .rarely found .enteritis .encephalitis .pneumonia In ruminant: .fever, dyspnea, nervous signs,	It is based on serology and the demonstratio n of the organism in the tissue. More recently the ELISA test has been developed	No complete satisfactory treatments but the combinttion of antimalarial drugs (pyrimethamine and sulphadiazine) are effective against the tachyzoite. Control is by proper cleaning of litter of cat and disposal of feces. Raw meat should not be given to cat, vaccination of sheep
Trypanosomias is	T. vivax T. congolense T. brucei T. simiae T. evansi T. swis	1. cycliical transmission: is where the arthropod (tsetse) is necessarily an intermediate host. 2. non cyclical transmission: is essentially	In ruminant: .anemia .enlargement of superficial lymph nodes .emaciation .Fever and loss of appetite .the disease is chronic .decreased	Is by: .clinical signs .parasitology ..Serology to isolate trypanosome from blood.	.diminazene aceturate .homidium bromide .homidium chloride .isometadium chloride .quinapyramine chloride The control is by

		mechanical transmission most of time by biting insect 3. Carnivores can be infected by eating flesh carcass dead by trypanosomiasis	fertility and death In pig: .mild and chronic by T. congolense and hyperacute by T. simiae		keeping animal in the zone free from Tsetse flies, keep trypanotolerant animals,reduce the possibility of reducing the drug resistance, isometadium has a prophylactic activity.