

An abstract graphic featuring three blue circles of varying sizes. The largest circle is in the top right, a medium-sized one is in the center, and a smaller one is in the bottom right. Two thin, light blue diagonal lines intersect the circles. The text 'General Veterinary Parasitology and Helminthology VPA-211' is positioned in the lower-left area.

**General Veterinary
Parasitology and
Helminthology VPA-211**

COURSE OVERVIEW

- Outline on types of helminths, its life cycle, pathogenesis and clinical signs.
- Zoonotic significance of helminths and its control.
- Diagnosis, treatment and control of helminths.
- Economic loss due to helminths.

MODULE-1: PARASITES AND PARASITISM

Learning objectives

- The module, parasites and parasitism will enlighten the students on the need to study veterinary parasitology, their importance in the field of medicine and veterinary sciences, the relationship between various organisms and the types of parasitism.

INTRODUCTION

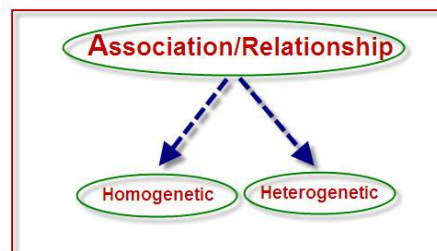
WHAT IS VETERINARY PARASITOLOGY ?

- In simple terms, Veterinary Parasitology is the study of parasites of domestic animals. On a broader platform, Veterinary Parasitology is the science that explores the relationship between various animal hosts and their parasites. Precisely, Veterinary Parasitology is the science that dwells on the anatomy and physiology of parasites, their host(s), transmission, development, impact and their detection and control.

IMPORTANCE OF PARASITES

- Parasitic diseases are known since time immemorial and yet many of the parasitic diseases have not been controlled and continue to evade and plague mankind.. Helminthic infections have been found in ancient Egyptian [mummies](#) (1210 – 1000 BC). According to the famous [Ebers papyrus](#) of 1550 BC at least four worm infections were recognized in ancient Egypt including *Ascaris lumbricoides*, *Taenia saginata*, *Dracunculus medinensis* (guinea worm) and *Schistosoma haematobium* . The papyrus also provides information on arthropods such as fleas, flies, and lice. More recently, evidence was obtained from examining tissue sections of ancient Egyptian mummies that *Trichinella spiralis* existed as an infection during that period. [Helminths](#) were also mentioned by Assyrian, Babylonian, and Greek physicians. Therefore, all of you should appreciate the fact that parasites have existed in this world for a long time and have become very successful in forging a good relationship with the host and discreetly utilize the relationship to their advantage and finally depriving the host of their nutrients.

- Parasitic diseases have their place in the history of the world and many wars were influenced by parasitic diseases. Alexander the Great could not continue his conquest of the world owing to malaria that killed many of his soldiers. Kingdoms in Northern Africa could not invade the Southern Africa owing to trypanosomosis. In many parts of the world, livestock could not be raised owing to parasitic diseases. Parasitic diseases continue to be the single most cause of mortality and morbidity in livestock resulting in immense economic loss to farmers. In India gastrointestinal nematodiosis in sheep and goats is so common and continue cause havoc to the small ruminant industry. Calves and lambs if infected with great numbers of parasites such as liver flukes or *Haemonchus* or coccidia may succumb to the infection. Diseases such as theileriosis continue to cause mortality in cross bred cattle. Cattle, pigs, sheep and goats infected with parasites fail to gain weight and may not reproduce there by affecting the economy of the livestock enterprise. In our country, coccidiosis is so common in broiler poultry farms, if the farm management is poor, the farm can be easily destroyed by coccidiosis.
- In the case of pet animals, toxocariosis and ancylostomosis are so common that is rare to come across a dog that is free of these worms. Puppies infected with hookworms and Ascarids die of the infection and heart worm infection in dogs continues to be a great challenge to veterinarians in certain parts of the world. Of course, heart worm infection is a problem only in North East India but it poses a great challenge to veterinarians in the United States. Cats infected with *Toxoplasma gondii* are a threat to other livestock and humans. If you take arthropods, mankind is yet to conquer so many of them. Mosquitoes continue to pose a big challenge to scientists. Only in the near past, the genome of mosquitoes and Plasmodium was sequenced.
 - During the course of evolution, living forms came together and associated as groups to ensure their survival in the environment. This association is referred to as symbiosis or symbiotic relationships.



- When the association is between members of the same genotype, it is known as [homogenetic association](#), for example, a flock of sheep or a pack of dogs.



A flock of sheep - Homogenetic association



Homogenetic association

- When the association is between members of different genotype as between a cattle and an insect, it is referred to as [heterogenetic association](#).

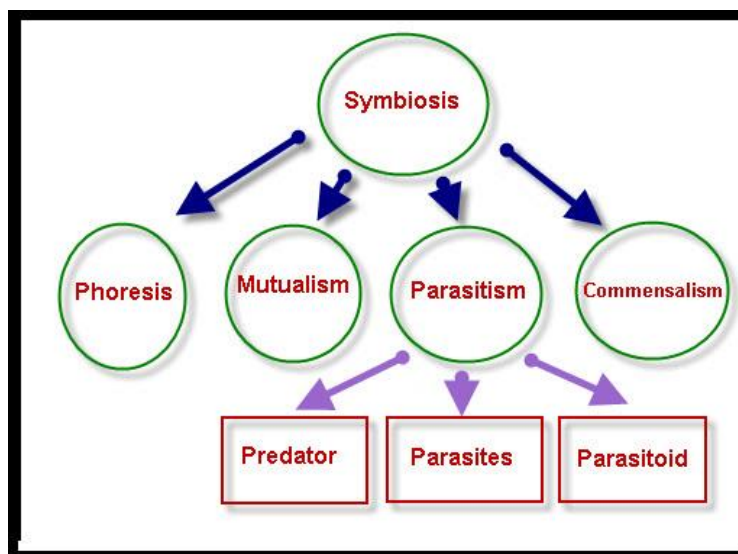


Flies infesting a cow - An example for heterogenetic association



SYMBIOSIS, COMMENSALISM AND PREDATORISM

- Symbiosis may be of different kinds viz., mutualism, commensalism, phoresis, parasitism or predatorism.



- Symbiosis** is association between two different organisms living in close physical association usually to the advantage of both as opposed to free living organisms or simply, living together.
- Any organism that is intimately associated with another organism of a different species is considered to be a [Symbiont](#).



Heterogenetic association

- **Phoresis** is a relationship between two symbionts which are merely traveling together and without physiological or biochemical dependence on each other.
- One will be larger than the other and the smaller phoront will be mechanically carried by the larger phoront, for example, mites carried by beetles or bacteria on the legs of a fly.



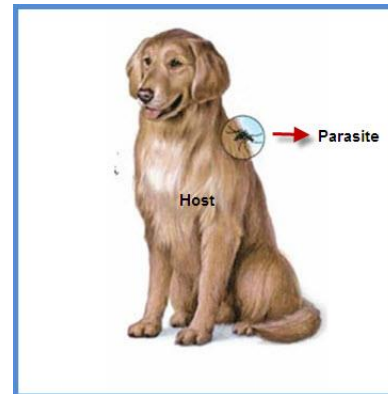
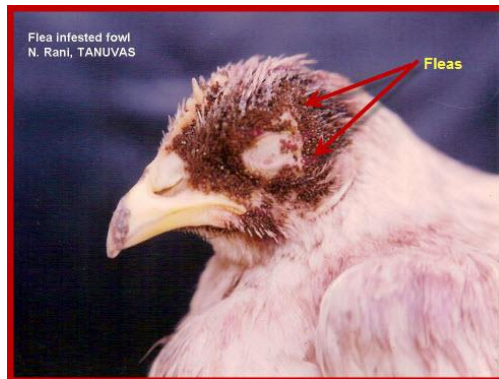
- **Mutualism** is relationship involving symbionts that are mutually dependant on each for food and shelter. This relationship is usually obligatory and one symbiont cannot survive without the other, e.g., intestinal protozoa in termites.
- Termites feed on food that they do not digest but protozoa living in their intestine digest the food.
- Termites do not have the enzyme cellulase, but still feed on food that is rich in cellulose.
- Protozoa living in the intestine of termites synthesize the enzyme cellulase and help in digesting the food ingested by the termite.
- **Commensalism** is a type of relationship, in which one symbiont derives benefit from the other symbiont (host) but the host is neither benefited nor harmed. The term commensalism refers to eating at the same table. Commensalism may be either facultative or obligatory.
- It is obligatory when the host is required for their survival and facultative when the host is not required for their survival.
- The amoeba, *Entamoeba gingivalis*, is an obligatory commensal that is present in the mouth of humans where it feeds on bacteria, food and dead epithelial cells but never harms the healthy tissue in the mouth.
- On the other hand, the ciliate, *Vorticella*, a facultative commensal, may associate with small crustaceans in a pond but are not dependant on them for their survival.

- **Predatorism** is the relationship between predators and prey where the predators kill the prey for food.
- Predators are animals or invertebrates that prey on other animals or invertebrates for food. Both [Parasites and Predators](#) live at the expense of the host or prey .
- Parasites seldom kill their host and are small in relation to the size of the host whereas predators kill the prey. Parasites have a higher reproductive potential than their hosts and are more numerous while predators have a lower reproductive potential than their prey, and are less numerous .
- **Parasitoids** are those, whose immature stages develop on other parasites and emerge by killing the parasite. E.g. Hymenopteran flies on dipteran flies



PARASITES AND PARASITISM

- Parasitism may be defined as association between two-species, in which one species, the parasite, lives in or on a second species, the host, for a significant period of its life and is metabolically dependant on the host for its survival.
- The organism that derives benefit from the association is called as the parasite while the other that is harmed is called as the Host.
- The word 'parasite' is derived from the Greek word and means 'besides food" (Para-beside, sitos - food).
- The parasite lives at the expense of the host without endangering the host.
- It does not kill the goose that lays the golden eggs.
- A predator kills the prey to feed upon the flesh but a parasite takes advantage of the host without killing it.

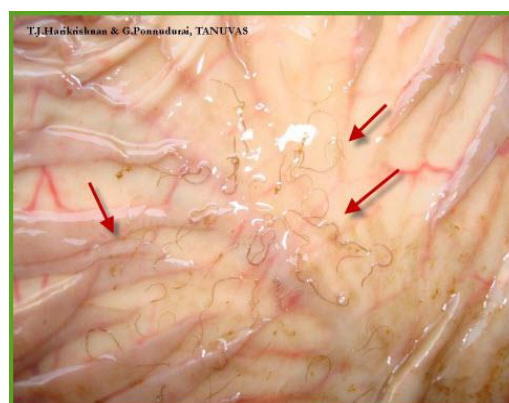


TYPES OF PARASITISM

- Ectoparasite is a parasite that lives on the surface of a host e.g. Fleas, ticks, lice, flies etc.

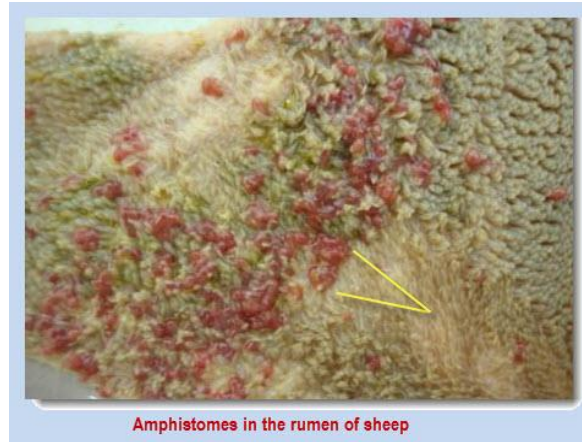


- Endoparasites are parasites that live inside the body of the host e.g. Flukes, tapeworms, roundworms etc.



The endoparasite, *Haemonchus contortus* in abomasum of a sheep

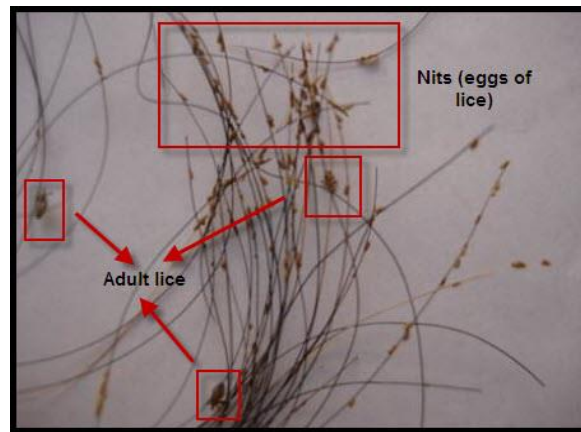
- Obligate parasites are parasites that cannot complete their life cycle without spending a part of their life cycle on the host e.g. trematodes.



- Facultative parasites are not normally parasitic but become parasitic when they accidentally find a host, E.g. *Naegleria fowleri* in humans causing primary amoebic meningitis and *Chrysomya* sp. larva in wound



- Accidental parasites are free living organisms that enter or attaches to the host by accident and leads a short parasitic existence. E.g. Larva of *Musca domestica* in hosts
- Incidental parasite is a parasite found in a host that is not its original host e.g. *Toxocara vitulorum* in goats
- Permanent parasites are those that spend their entire life cycle in or on the body of the host e.g. lice

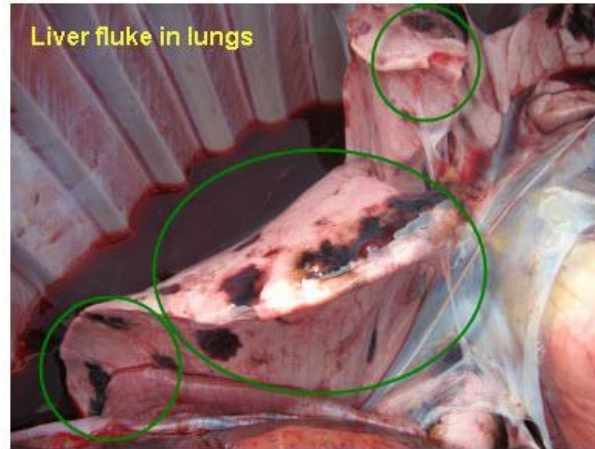


Eggs (Nits) and adult lice on tail switch of cattle

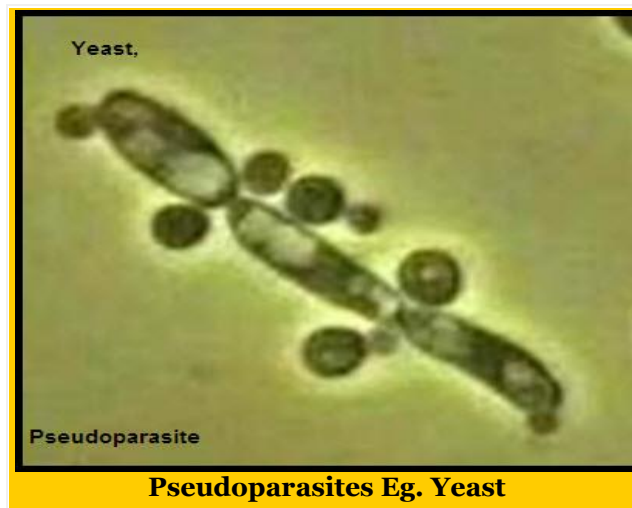
- Temporary parasites are those whose life cycles are not spent entirely on the host and are seen only for certain periods of time (short or long duration) E.g. *mosquitoes and bugs*.



- Erratic or aberrant or ectopic parasites are present in locations that are not their usual site e.g. *liver fluke in lungs*.

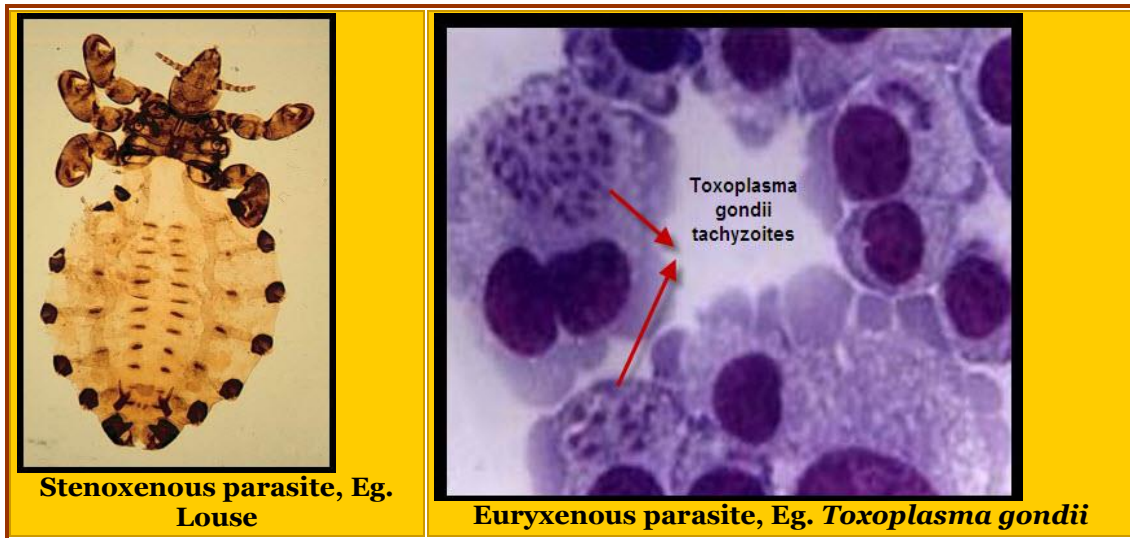


- Hyperparasites are those that parasitize other parasites e.g. *Plasmodium* on mosquitoes.
- Spurious parasites are parasites found transiently in the excretions of the host in view of the host having consumed a parasite of another host E.g. *Eggs of Moniezia* in dog faeces after ingestion of *Moniezia* infected sheep, goat and cattle intestines by the dog.
- Pseudoparasites or artifacts are parasites that resemble a parasite and which are present in the clinical specimens e.g. *plant fibres, yeast, pollen grains etc* .



- A monogenetic parasite is one where there is no alternation of generation (e.g. *Ascarids, amoeba, trichomonads*, etc.) while a digenetic parasite is one in which there is alternation of generation (Coccidia, Plasmodium, piroplasmids, trematodes of higher vertebrates where asexual generation alternates with the sexual generation, for e.g. in piroplasmids, the asexual stages are present in vertebrate host and the sexual stages in invertebrate host).
- Parasites that require more than one host to complete its life cycle, are termed Heteroxenous e.g. *Theileria annulata* – Cattle and ticks while parasites that require only one host to complete its life cycle are said to be Homoxenous/Monoxenous e.g. *amoeba, coccidia* etc.

- Parasites with a narrow host range are referred to as Stenoxenous e.g. *Haematopinus suis* in pigs and *coccidia* whereas parasites with a wide host range are known as Euryxenous parasites e.g. *Toxoplasma gondii*.



- Synanthropic parasites are parasites that are present or frequent human dwellings e.g. houseflies, cockroaches etc.



MODULE-2: TYPES OF PARASITES

On the basis of activity in a day, parasites are further divided into

- Diurnal parasites
- Nocturnal parasites
- Crepuscular parasites
- Diurnal parasites are those that are active in the day, e.g. *Musca domestica*, *Tabanus* sp. of flies

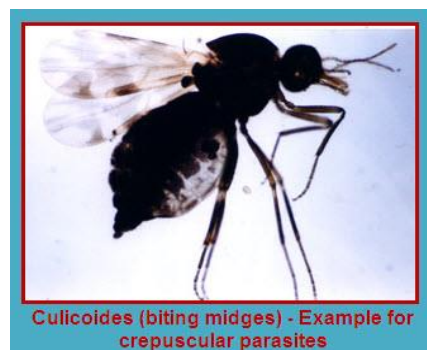


- Nocturnal parasites are those that are active at night, e.g. Anopheline mosquitoes



A mosquito feeding on a host

- Crepuscular refers to parasites that are active at twilight (dawn or dusk), e.g. the biting midge, *Culicoides* sp.

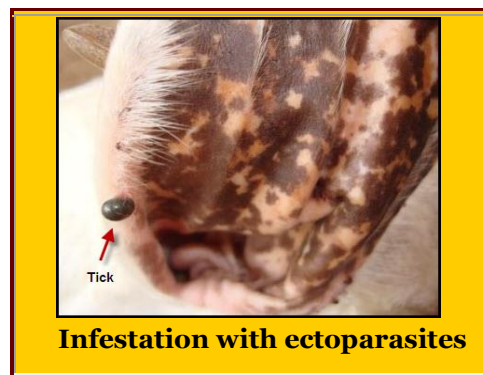


TYPES OF PARASITISM

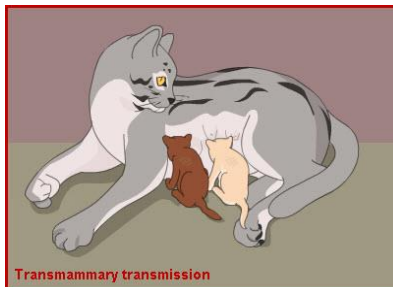
- [Infections](#) refer to parasitism of host by internal parasites (e.g. amphistomes in livestock)



- Infestations refer to parasitism of host by external parasites (fleas, lice, ticks, flies etc.).



- [Autoinfection](#) refers to a condition where the juvenile infective form of a parasite, without escaping from the host infects the same host e.g. *Enterobius vermicularis*
- [Hyperinfection](#) refers to a condition where the juvenile form of a parasite without exiting the host penetrates in an area adjacent to the site of predilection and establishes in the same host e.g. *Strongyloides* sp.
- [Congenital \(Transuterine or Transplacental\)](#) infection is transmission of parasites from the dam to the foetus across placental membranes e.g. *Toxocara canis* in dogs
- [Transmammary \(Transcolostral\)](#) is transmission of parasites from the dam to the young one through the milk of the infected dam e.g. (*Toxocara cati* in cats)



- **Transovarian transmission** is transmission of parasite from the female parent to the progeny through the ova. E.g. *Babesia bigemina* in ticks



- **Transtadial (stage to stage)** is transmission of parasite from one stage to another stage of the vector as in *Theileria annulata* in *Hyalomma* spp. of ticks
- **Zoonoses** are diseases common to humans and animals (Singular – Zoonosis). According to WHO (1959), zoonoses are diseases transmissible between vertebrate animals and man.



- **Anthropozoonoses** is a disease of animals transmitted to humans e.g., Cystic echinococcosis from animals to humans

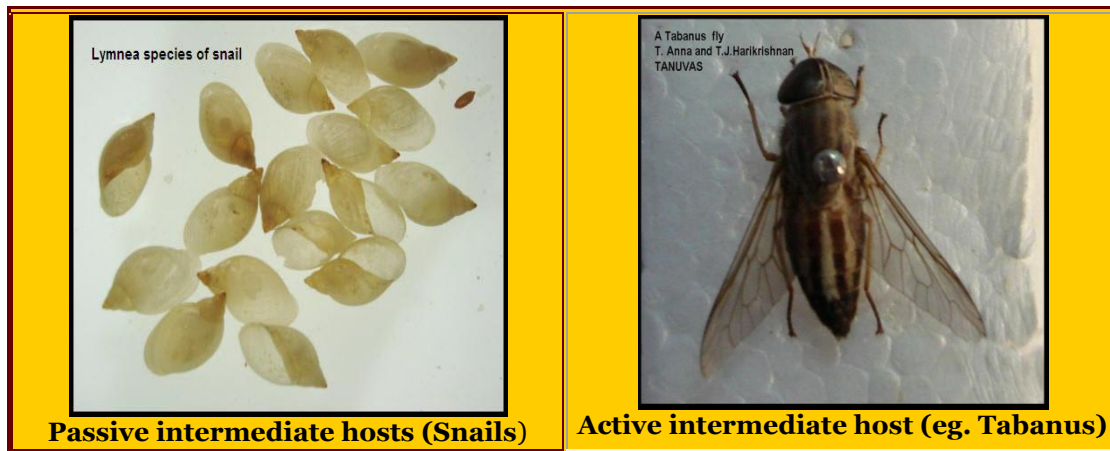


- [Zoonanthroponoses](#) is a disease of humans transmitted to animals e.g. *Taenia solium* from humans to pigs



TYPES OF HOST

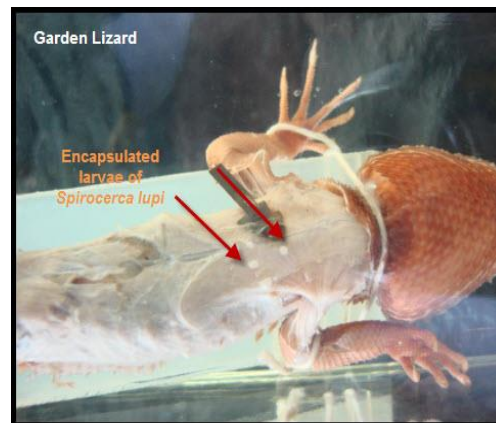
- **Definitive hosts** are hosts that harbour the adult (sexual) form of the parasite. e.g. Sheep for *Fasciola gigantica*, Dog for *Echinococcus granulosus*, and Horse for *Habronema muscae*.
- **Intermediate hosts** are those that harbour the asexual form of the parasite (only when there is an obligatory passage through the host).
 - Intermediate hosts may be divided into **passive intermediate hosts** (snails for trematodes) and **active intermediate hosts** (*Tabanus sp.* for *Trypanosoma evansi*)



- **Reservoir hosts** are hosts that harbour a parasite of another host without itself getting affected, but act a source of infection for the original host e.g. Game animals for the protozoan parasite *Trypanosoma gambiense*.
- **Carrier hosts** are hosts that have a residual population of the parasite and acts as a source of infection for the same type of host.
- **Transport hosts** are those that harbour the immature/infective stage of a parasite of another host and help in disseminating the parasite without any development in itself. E.g. Earthworms for Egg/larvae of *Ascaridia galli*



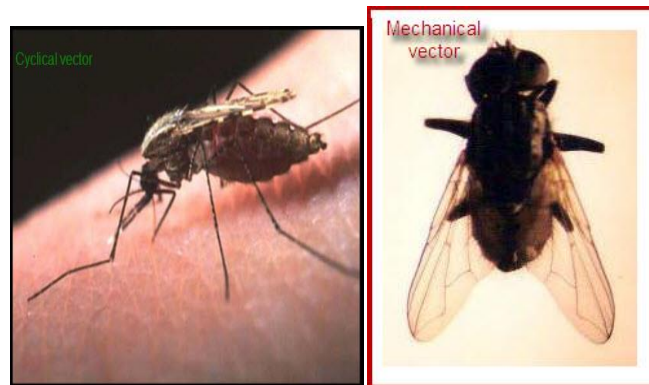
- **Paratenic hosts** are those that harbour the immature/infective stage of a parasite in an encapsulated form and helps in dissemination of the parasite to the definitive host e.g. *Calotes* for *Spirocerca lupi*.



- **Intercalary host** is a host that liberates the infective stages of a parasite of another trapped in the body of the original host E.g. Cats by eating mice liberate the eggs of *Capillaria hepatica* which are trapped in the liver parenchyma.

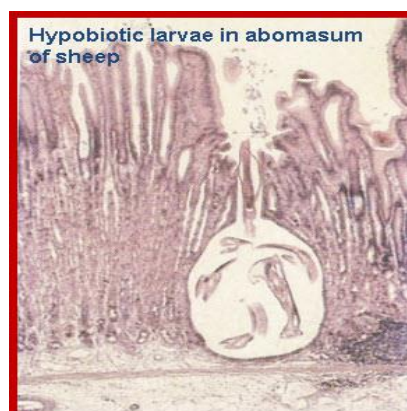
VECTOR

- A Vector is a latin word which means *bearer*. In the broad sense any agent that carries infectious organisms between animals. A vector is an arthropod that carries the parasite between two vertebrate hosts. E.g. Ticks and mosquitoes for various blood protozoans.
- Vector may be divided into [mechanical vector and cyclical vector](#).
- A mechanical vector is an arthropod that carries the infectious agent from, one vertebrate host to another without any development in its body. E.g. *Tabanus* transmitting *T.evansi* by interrupted feeding or house flies transmitting cysts of *Entamoeba histolytica* while a cyclical vector is a vector in which the parasite undergoes development/multiplication before being transmitted to the next host (Ticks transmitting haemoprotezoan parasites)



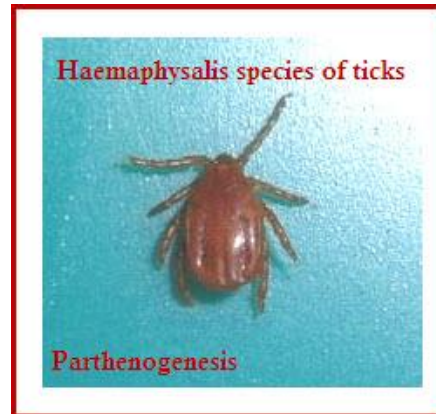
TERMINOLOGIES USED IN PARASITIC DISEASES

- **Epidemiology** is the study of a disease in relation to the population, aspects of disease such as incidence, prevalence and transmission in a population
- **Endemic** disease refers to a disease that occurs with a predictable frequency with minor fluctuations in a population
- Epidemic disease refers to diseases that occur at a higher level in a place at a time than expected for the disease in the place at that particular time
- Sporadic diseases are those diseases that occurs irregularly with widely dispersed incidence or a disease that occurs infrequently
- Pandemic is an epidemic occurring in a wide/larger area.
- Incubation period is the time lapse between the entry of the parasite and the first appearance or onset of clinical signs.
- Prepatent Period is the period in which the form of the parasite is demonstrated in the clinical material of the host.
- Hypobiosis refers to temporary cessation in the development of immature stages of some nematode parasites in the host due to adverse environmental conditions, for example, *Ostertagia ostertagi* in cattle

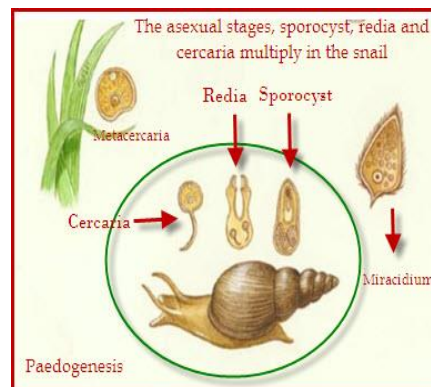


- **Hibernation**(Winter Sleep) refers to low activity of parasites in winter.
- **Aestivation** (summer sleep) is the period of low activity in summer
- **Diapause** is a period of low activity in arthropods.

- [Parthenogenesis](#) is a (Virgin birth) refers to formation of progeny without fertilization e.g. *Haemaphysalis* ticks and *Strongyloides* sp. of nematodes.



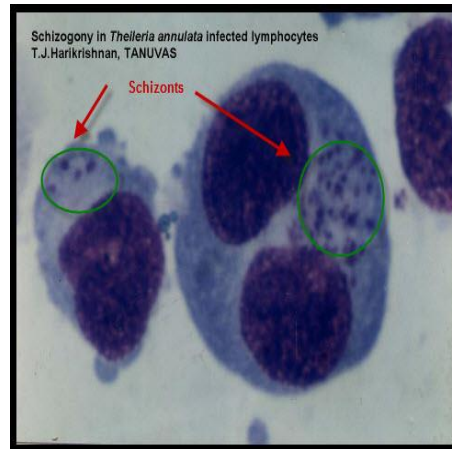
- [Paedogenesis](#) (Juvenile multiplication) refers to multiplication of juvenile/immature stages of parasite in intermediate hosts. E.g. Multiplication of immature stages of trematodes in snail



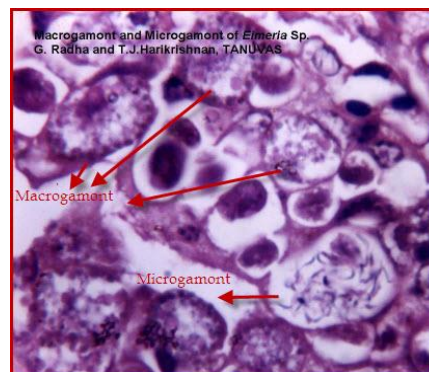
- [Metagenesis](#) (Alternation of generation) refers to sexual multiplication alternating with asexual multiplication, E.g. Trematodes, Sexual multiplication in definitive host alternating with paedogenesis in intermediate hosts



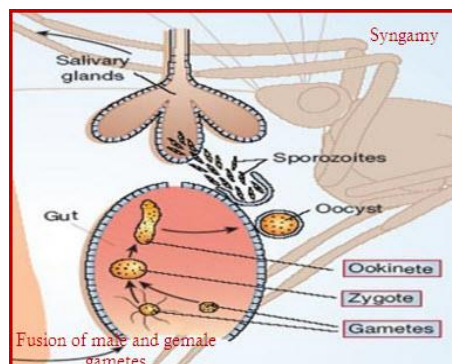
- **Schizogony** is a type of asexual division/multiplication in protozoa where a parasite multiplies into numerous individuals by multiple fission. The nucleus of the parasites undergoes repeated division without cytokinesis. e.g. apicomplexan parasites



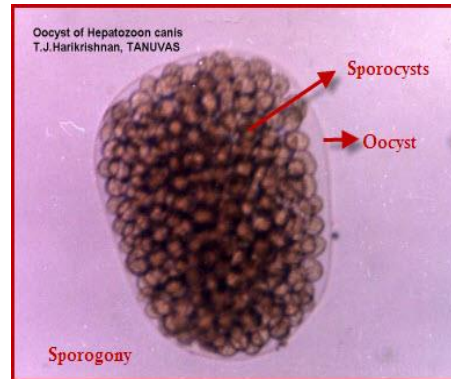
- Gametogony is the differentiation of gamonts into male and female gametes



- Syngamy is the union of the male and female gametes to form the zygote e.g. apicomplexan parasites



- [Sporogony](#) is the differentiation of zygote to oocyst, sporocyst and sporozoites e.g. all apicomplexan parasites.



HOST PARASITE RELATIONSHIP

- Host parasite relationship as the term implies is the relationship between two organisms namely the host and the parasite where the parasites invariably succeed in forging a relationship to its advantage.
- Parasites upon entering their hosts through various routes may survive or die in the host. If parasites survive they develop and multiply causing infection or disease in the host.
- In evolution, there is always a tendency towards mutual adjustment between two organisms and therefore both parasite and the host forge a relationship that may either limit the infection so that parasite control is achieved without any pathological changes. Therefore, successful parasites tend to become harmless to the host.
- Majority of the parasites that survive in their hosts do not endanger their hosts, since they do not want to lose their shelter and food. However, the host tries to expel or kill the parasite by immune responses. When the host attempts to kill the parasite by immune mediated mechanisms, immunopathological lesions develop leading to disease in the animal.
- Host-Parasite relationship is a dynamic process where the host employs innate and acquired immune mechanisms to destroy the parasite.
- The nutrition status of the host and the energy resources of the host, at the start of an infection play a role in determining the ultimate outcome of the host parasite relationship. Malnutrition will impede the animal's ability to respond immunologically to the parasite.
- As parasites are large and complex, the host finds it difficult to counter the parasites by immune mediated mechanisms, resulting in responses that are not always protective.
- In addition, parasites also evade the immune responses of the host by various ways to ensure their survival and propagation. Therefore, host parasite relationship is an intricate relationship where the host attempts to destroy the parasite but the parasite does not endanger their host lest that they so lose their shelter and food.
- Host parasite relationship occurs in a complex ecological setting where in vivo and external environments change continuously.

MODULE-3: MODES OF TRANSMISSION OF PARASITES

Learning objectives

- This module will focus on the various routes of transmission of parasites between hosts. In other words, how parasites are transmitted between hosts will be dealt in this chapter. Parasites are commonly transmitted through ingestion of parasite contaminated food and water besides other routes such as skin penetration, intra nasal transmission, coitus and congenital transmission. Each mode of transmission will be dealt in detail with suitable examples.

INTRODUCTION

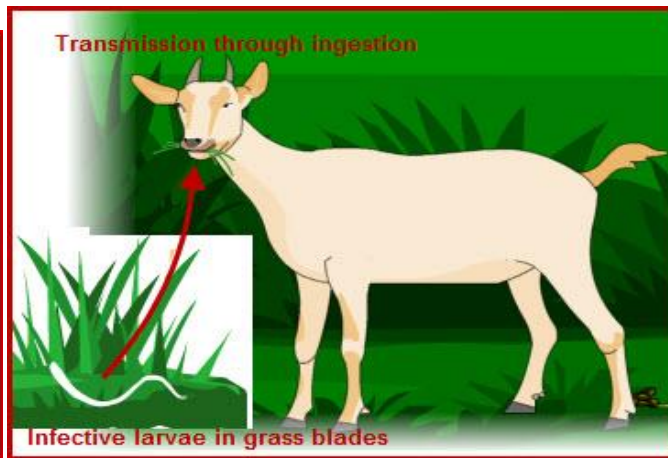
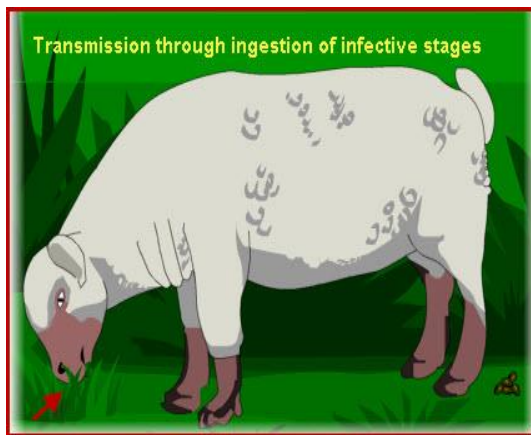
- Parasites have distinct routes of entering a host.
- Endoparasites enter the hosts in general through natural openings on the exterior of the body or through the skin.
- Parasites are transmitted from one animal to another by
 - Ingestion
 - Skin penetration
 - Contact
 - Predation
 - Coitus
 - Transplacental/Transmammary transmission

THROUGH INGESTION

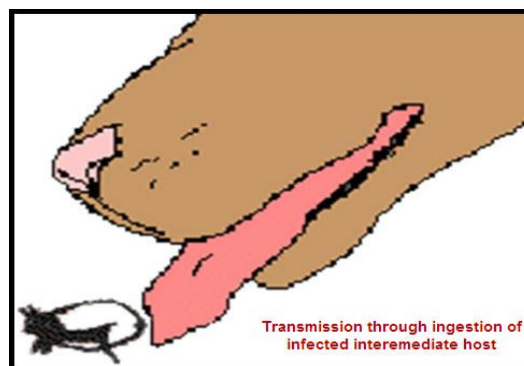
- The most common mode of transmission of parasites is through ingestion. The host may become infected by direct ingestion of an infective stage through contamination of food and water or by ingestion of an intermediate host or transport host containing infective stages.



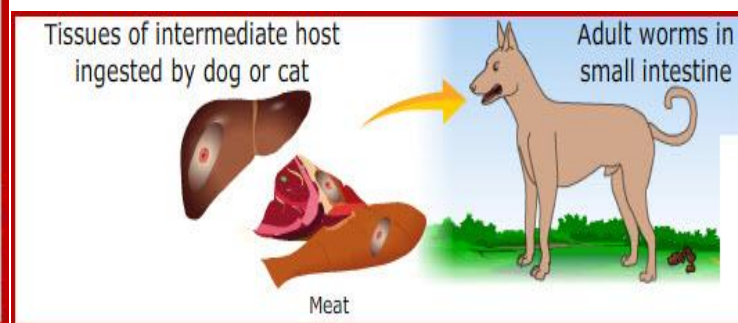
- For parasites with direct life cycles, the infective stages viz., egg containing the second larval stage (ascarids), cysts (*Entamoeba* and *Giardia*) and oocysts (coccidia) find their way into the feed and water of the host thereby gaining access to the host or the infective stages (third larval stage) may climb up the vegetations and wait for the host to ingest.



- For parasites with indirect life cycle, the host becomes infected by ingestion of an intermediate host or transport host containing infective stages.

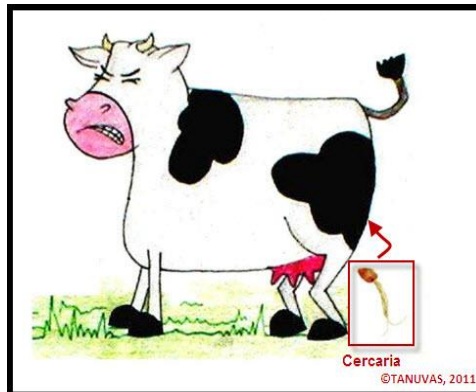


- In certain parasites, the definitive hosts are infected through ingestion of infective stages (larval stages) in intermediate hosts (fish for *Diphyllbothrium latum* and crabs for *Paragonimus* sp) and through ingestion of raw or undercooked meat/organs/milk of intermediate hosts (*Taenia solium*, *Echinococcus granulosus*).



THROUGH SKIN PENETRATION

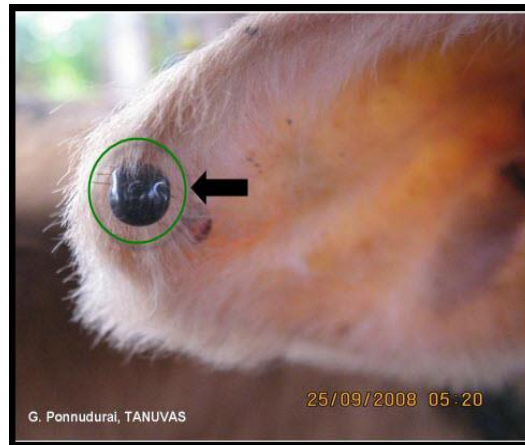
- After mouth, skin is the most common route of entry for many parasites.
- The infective stages of the parasite may actively penetrate the skin and in this process, the secretions of the parasite help to digest the host tissue (cercariae in schistosomes).



- Penetration of the host's skin is the predominant route by which the infective larvae of hookworms enter their hosts, although they also enter through the mouth.

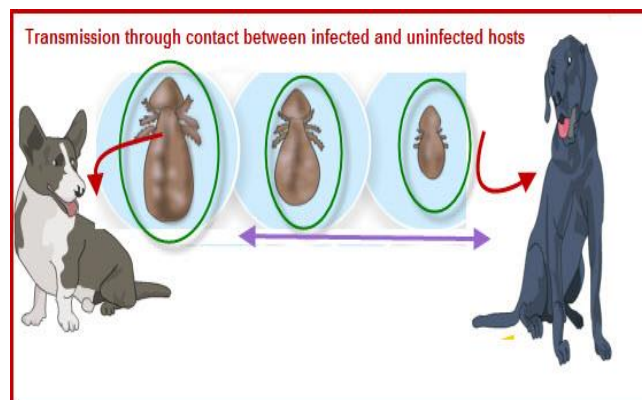


- Certain parasites enter the hosts via the bite of an intermediate host serving as a vector. These parasites develop in blood sucking insects and ticks and when these vectors feed to obtain the blood and tissue fluids from hosts, the infective stages of the parasites are introduced into the host.



CONTACT AND PREDATION

- Parasites are transmitted between animals by contact especially when they are confined in sheds or houses. Eg. Lice infestation and mange is chiefly transmitted between animals by contact.

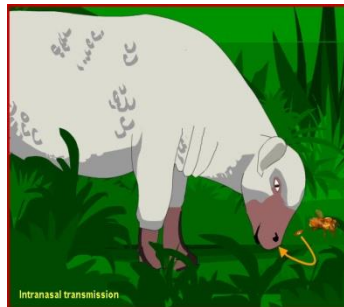


- Parasites are also transmitted during predation by a host. For example, when cats predate on rats, cats may acquire an infection with parasites.



ENTRY THROUGH NOSTRILS

- Oestrus ovis***, the nasal bot fly, deposit its young larvae around the nostrils of the host, where upon they crawl upward and enter the nasal sinuses.

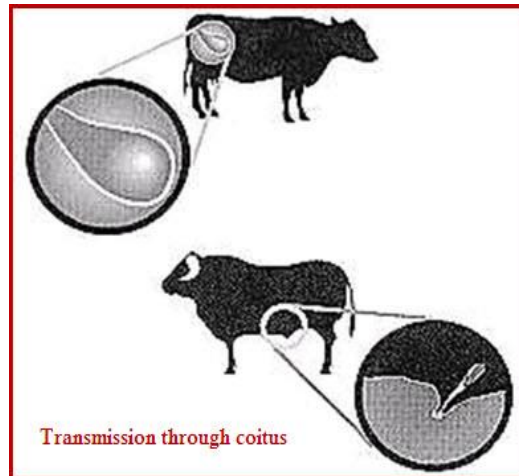


- Naegleria fowleri***, a fresh water amoebae present in surface water of polluted pools gains access to the hosts through the intranasal route.



ENTRY THROUGH EXTERNAL GENITALIA

- *Tritrichomonas foetus*, the protozoan parasite that causes abortion in cattle and *Trypanosoma equiperdum*, the protozoa that causes dourine in horses are transmitted during coitus.



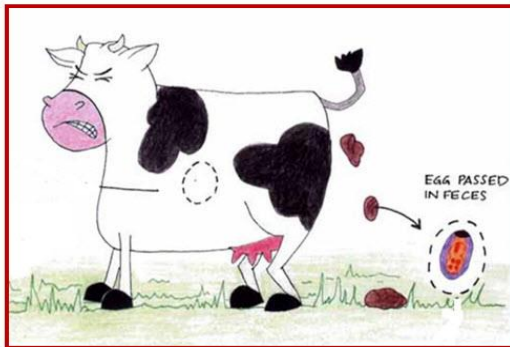
TRANSPLACENTAL/TRANSMAMMARY TRANSMISSION

- Transplacental or prenatal infection refers to transmission from mother to foetus across the placenta as in *Toxocara canis* and transcolostral or transmammary transmission refers to transmission from infected dams to nursing offspring via colostrum or milk as in *Toxocara vitulorum* or *Toxocara cati*.

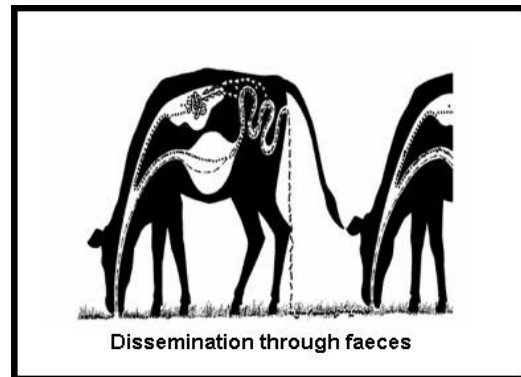
MODULE-4: METHODS OF DISSEMINATION OF THE INFECTIVE STAGES OF PARASITES

Introduction

- Most of the parasites leave the host passively through excretions of the host.
- Many exit from the hosts through faeces as eggs (ascarids, strongyles, flukes etc.), cysts (*Entamoeba*, *Giardia*) and oocysts (coccidia).

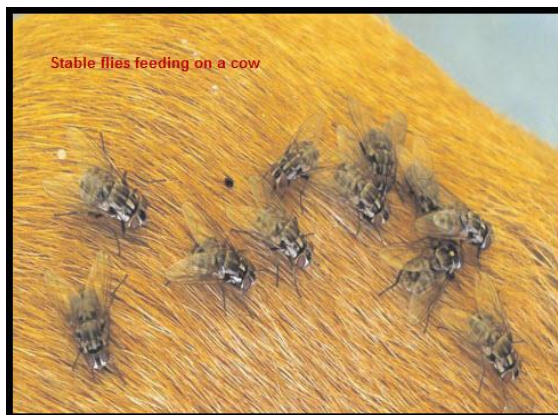


Parasites disseminated through faeces

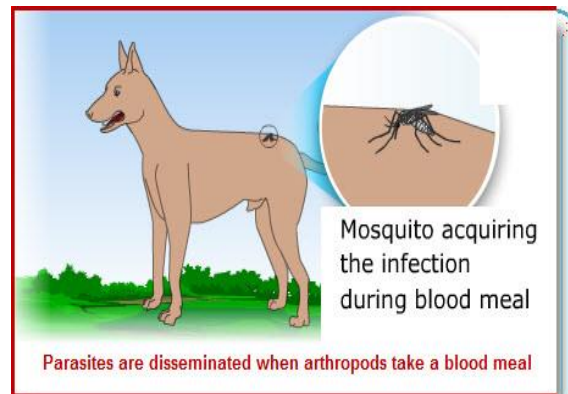


Dissemination through faeces

- Few exit through urine (Eg. *Schistosoma haematobium*, *Stephanurus dentatus*) and some leave through genital discharges (Eg. *Tritrichomonas foetus*).
- Some parasites are imbibed by arthropods inadvertently when they feed on a host (eg. Malarial parasites transmitted by mosquitoes) while the extraintestinal stages of parasites may be removed from the host during predation and the parasite subsequently infects the predator and leaves it passively through their faeces (eg. Cyst forming coccidia).



Stable flies feeding on a cow



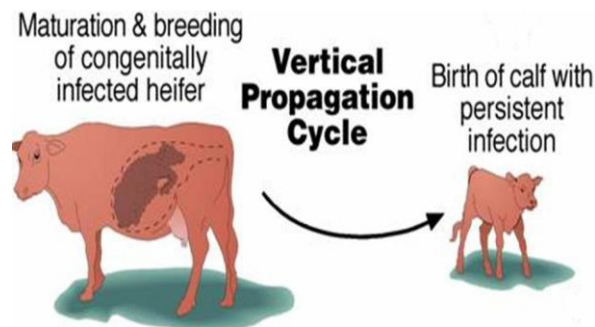
Parasites are disseminated when arthropods take a blood meal

DISSEMINATION OF PARASITES

- Permanent ectoparasites such as louse and flea exit the hosts actively by either crawling or jumping on to another host during moments of contact between hosts.



- Few parasites exit actively from the hosts (mother) to their foetuses through placenta (transuterine route) eg. *Toxocara canis*, *Toxoplasma gondii*



- Some parasites leave the host through the medium of milk to infect the young ones (transmammary route), eg. *Toxocara vitulorum*, *Toxocara cati*.



METHODS OF DISSEMINATION OF INFECTIVE STAGES OF PARASITES

- Once parasites are outside the hosts, they have to survive in the environment until they find a suitable host. As the environmental conditions are generally adverse to their survival, the infective stages of parasites are endowed with resilience. These resistant stages have the capability to survive the adverse conditions and remain infective to find a host. Temperature and moisture are two important factors that facilitate or deter parasite survival and development.
- Parasites generally do not develop below 10°C or above 40°C. When climatic conditions are hostile (freezing temperatures), parasites cease development in hosts (arrested development) and wait for the conditions to improve before they resume development and discharge their eggs in faeces.

Dispersal of parasites

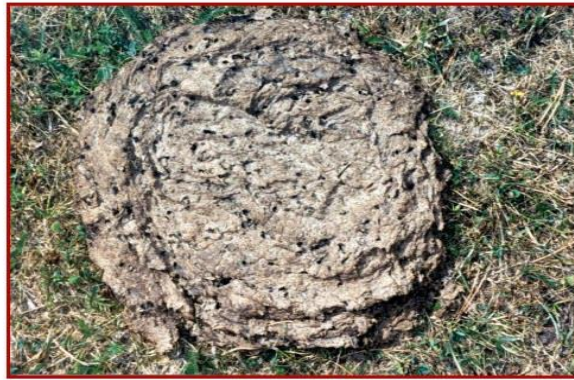
- Parasites are disseminated in the environment mechanically through the agency of water and fomites beside human interventions.
- Water is an important agent for dissemination of parasites especially for those that require an aquatic habitat for development (eggs of many trematodes, larvae of *Dracunculus* etc).
- Trematodes whose intermediate hosts are aquatic snails are carried by the surface water into water bodies where they develop in aquatic snails. Similarly, larvae of *Dracunculus* require water bodies for gaining access to cyclops.
- Parasites that require aquatic habitats for developments such as mosquitoes and black flies are carried to long distances by streams and rivers.



- Human interventions such as construction of irrigation canals directly facilitate breeding of snails' thereby aiding dissemination of trematode parasites besides aiding breeding of insects.



- The use of sewage and sludge to fertilize pasture is another potential method of dissemination of parasites.
- Fomites such as insects mechanically disseminate the parasites from one place to another.
- The fungus *Pilobolus* aids in the dispersal of the lungworm infective larvae from faecal pats to the pasture thereby facilitating infection of cattle hosts.
- Wild animal hosts may serve as a reservoir of infection for domesticated livestock.



The fungi, *Pilobolus* that grows in faecal pats helps in dissemination of lungworm larvae

- Wild reservoir hosts are important factor in the epidemiology of Fasciolosis. Overcrowding of hosts also facilitates dispersal of parasites.

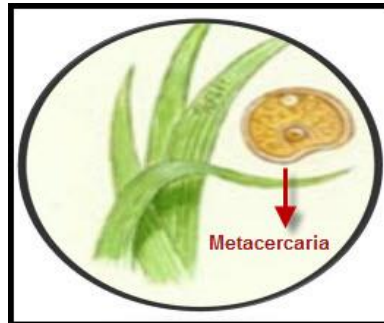
Parasite induced host behavioural changes

- Metacercariae of *Dicrocoelium* enters the brain of ants and paralyse them thereby aiding dissemination of parasite infected ants for herbivores during grazing.
- Grasshoppers infected with tetrameres and beetles infected with cysticercoids of tapeworms become sluggish thereby facilitating ingestion of definitive hosts.
- Ruminants heavily infected with hydatid cysts become debilitated, making them easier prey for carnivores.



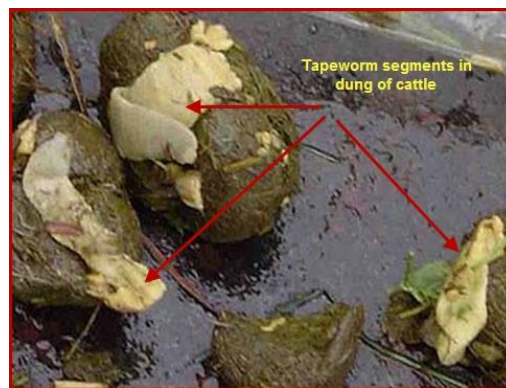
Strategies adopted by parasites to infect hosts

- Some parasites employ certain strategies to augment their chances of finding a host. Liver fluke encysts on green parts of plants and on higher parts of the plants to facilitate ingestion by herbivore hosts.



Metacercaria about to encyst on grass blades

- As cattle are reluctant to feed on herbages near faeces, the motile gravid segments of *Taenia saginata* leave the faeces to contaminate the herbage and increase its chances of being eaten by an herbivore as against the non motile gravid segments of *Taenia solium* which does not have to leave the faeces because pigs being coprophagic consume the faeces and pick up the infection.



- Some parasites are endowed with sensory organs to locate hosts. Warmth, CO₂, fatty acids, amines etc., serve as stimuli for parasites especially arthropods to locate hosts.

MODULE-5: PARASITE SPECIFICITY IN RELATION TO SPECIES, BREED, SEX AND LOCATION

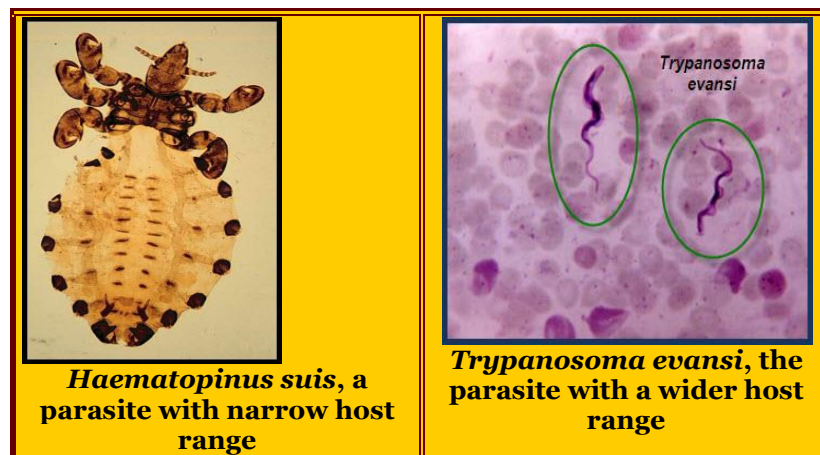
Learning objectives

This module will focus on

- parasite specificity in relation to host species, breed, sex, location within the host and the geographical location of parasites.
- Parasite specificity determines the host range of parasites.

PARASITE SPECIFICITY IN RELATION TO SPECIES, BREED, SEX AND LOCATION INTRODUCTION

- Parasite specificity or Host Specificity is the natural adaptability of a species or groups of parasite to certain species or groups of host and is dependent upon the compatibility of a host to a parasite.
- In natural host parasite relationships, the parasite must be precisely adapted to the structural and physiological conditions that characterize the host species. This adaptation, which develops over long periods of evolutionary change, is the basis for the phenomenon of host specificity or parasite specificity.
- Parasite specificity is usually defined in terms of establishment or failure to establish in a host. However, a range of parameters, such as establishment, number, size, developmental stage of worms, duration, level of egg production and duration of infection can give indication of the degree of adaptation to a particular host. For example, the eggs of the human ascarid, *Ascaris lumbricoides* can hatch in a variety of mammalian hosts but can develop into adults in humans alone. However, in some cases, the restriction is absolute or total as occurring in *Eimeria* species or the restriction may be very loose and parasite can undergo development in and be transmitted between, a wide variety of hosts as occurring in the nematode *Trichinella spiralis*.
- Parasite specificity may be supra specific where, groups of parasites are associated with groups of hosts or infra specific where a specific parasite is associated with a specific species of host.
- Parasite specificity determines the host range of a parasite and accordingly, the parasite may have a narrow or wider host range. Certain parasites such as *Haematopinus suis* infest only pigs (narrow host range), while some such as *Trypanosoma evansi*, have a wider host range and infect many hosts.



- Specificity may also vary between larval and adult stages of the same parasite to hosts.
- In some, the larval stages have strong host specificity to intermediate hosts (snails) and less for definitive hosts as in trematodes while in some such as *Toxoplasma gondii*, the asexual stages have a wider host range and infect a wide variety of intermediate hosts while the sexual stages infect only one host.
- In some parasites, as in *Eimeria*, the host specificity is less at the generic level where they infect a wide variety of hosts but very strong at the individual species level with very high niche specificity in hosts.

FACTORS INFLUENCING PARASITE SPECIFICITY

- Parasite specificity is also governed by anatomical, physiological and nutritional, beside ecological factors.
- The anatomy of the host animal may prevent the establishment of a parasite.
- The intestinal villi or crypts may be too short or intestinal movements too rapid to prevent the establishment of a parasite.
- Physiological factors such as composition of the bile, dissolved carbon dioxide in hosts, pO_2 , redox potential and the normal temperature of a host may also regulate host specificity.
- In unnatural hosts, the unsuitable body temperature may fail to provide the right stimulus for the parasite to establish in hosts.

PARASITE SPECIFICITY IN RELATION TO HOST SPECIES

- Host animals are susceptible to some parasites and resistant to others. For example, majority of the helminths of cattle are incapable of infecting sheep and goats. The normal resistance of various species of animals to various pathogens is due to the presence of antibodies on their erythrocytes called isohaemagglutinins.
- Many parasites do not develop in hosts other than their natural hosts. A good example for host specificity is *Eimeria* sp.
- The red worms (***Strongylus* sp**) of horses are specific to equine hosts and cannot infect cattle, buffalo, sheep and goat.

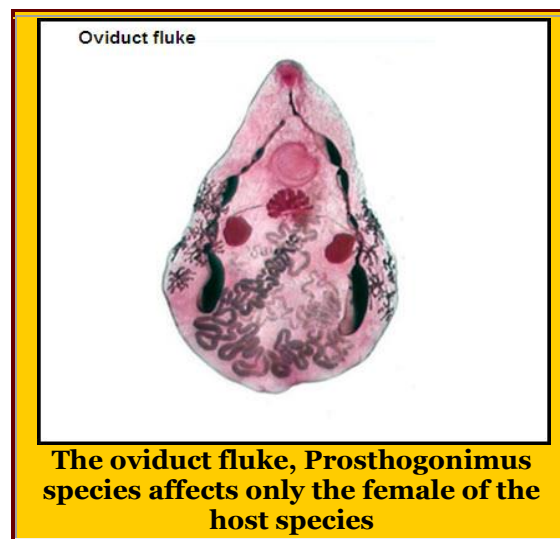


- The nematode ***Ancylostoma caninum*** is a parasite of both dogs and cats. However, the strain adapted to dog survives better and produces more eggs in dog, as compared to the strain found in cat. Similarly, the cat adapted strain develops better and produces more eggs in the cat as compared to dogs.

- Limited degree of development of parasite occurs in unnatural host in some cases as in the case of larvae of *Ostertagia ostertagi* (cattle parasite) in sheep where only few reach to adult stage or the dog parasite, *Toxocara canis* which undergoes limited development in children causing the condition visceral larval migrans.

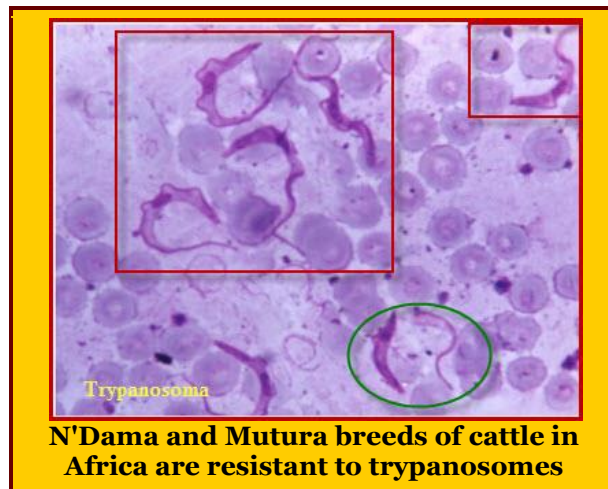
PARASITE SPECIFICITY IN RELATION TO SEX

- Some parasites affect only the females and not the males as in the case of the trematode, *Prosthogonimus* species which is mostly found in the oviduct of female gallinaceous birds. The influence of sex on helminth burden appears to be largely hormonal. In animals whose oestrus cycle is seasonal, parasites tend to synchronize their development with that of the host. For example, ewes show a spring rise in faecal egg counts after lambing and onset of lactation. Similarly, in the case of bitches above six months old, development of *Toxocara canis* is influenced by hormonal changes. When bitches become pregnant, the dormant larvae are activated and transported to the foetus and so puppies are born with *Toxocara canis* infection. However in the case of male dogs older than 6 months, dormant larvae are not activated but become calcified. Therefore, older male dogs serve as dead end hosts for *Toxocara canis*.



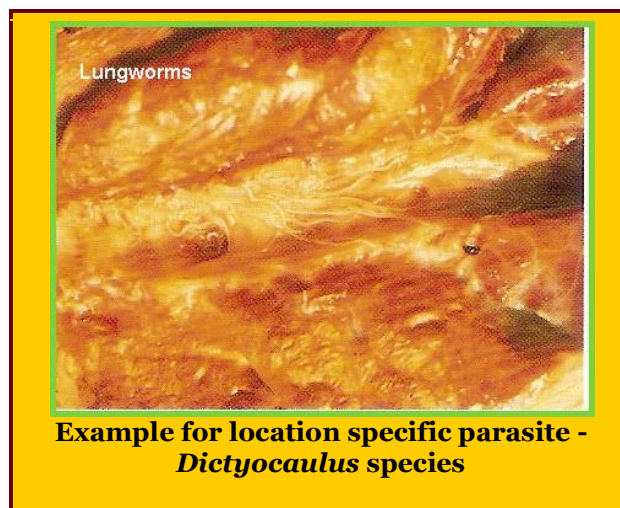
PARASITE SPECIFICITY IN RELATION TO BREEDS

- Different parasites have varied susceptibility to different breeds.
- Indigenous breeds of cattle suffer less from tick infestation and tick borne diseases in comparison to cross breeds and exotic breeds.
- N'Dama & Mutura breeds of cattle in Africa and their crosses are tolerant to trypanosomes.



PARASITE SPECIFICITY IN RELATION TO LOCATION IN HOSTS

- Each parasite has a specific predilection site or a location in hosts. The best example for site specificity is *Eimeria* species which parasitize only certain areas of the intestine.
- Other examples include the nematode *Dictyocaulus* species which affect only the lungs in ruminants and the trematode, *Paragonimus* species in lungs of dogs.



PARASITES IN RELATION TO LOCALITY

- When parasites are limited to certain ecological or geographical areas as in the case of African animal trypanosomes and human trypanosomes that are restricted to Western, Central and Eastern Africa, it is referred to as ecological or geographical restriction.

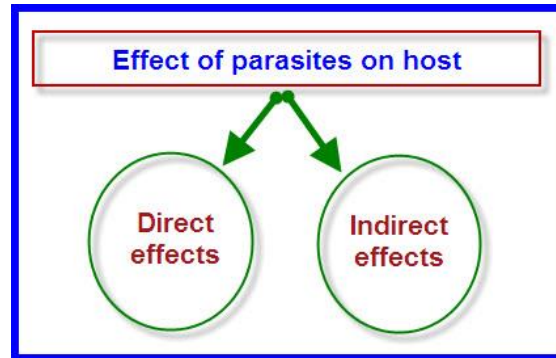
MODULE-6: TISSUE REACTIONS CAUSED BY PARASITES TO THE HOST

Learning objectives

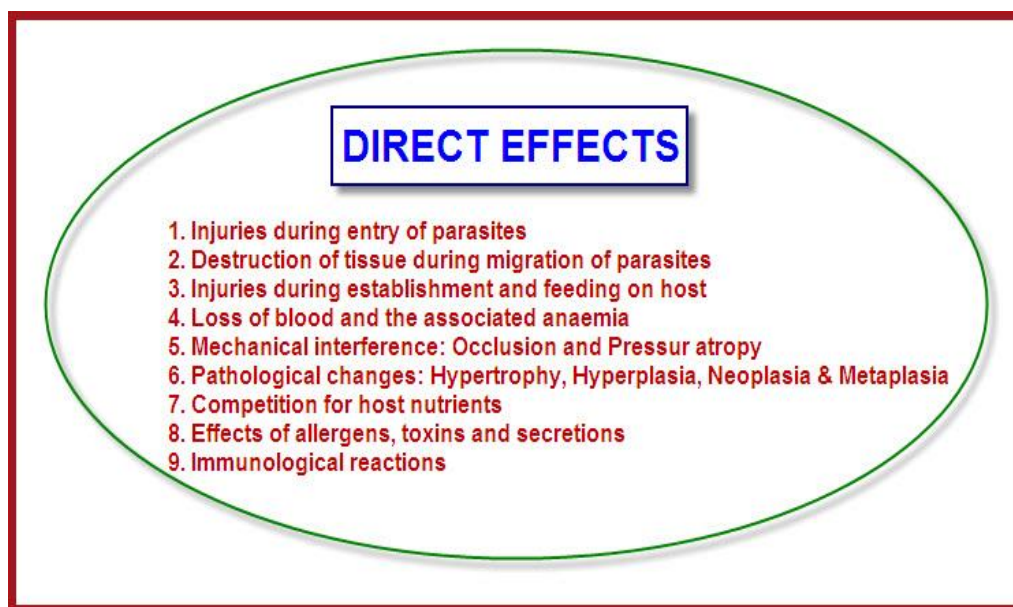
- This module will inform the students about the effects of parasites on hosts or in other words the injury and lesions inflicted by parasite during its invasion, establishment and development in the host.

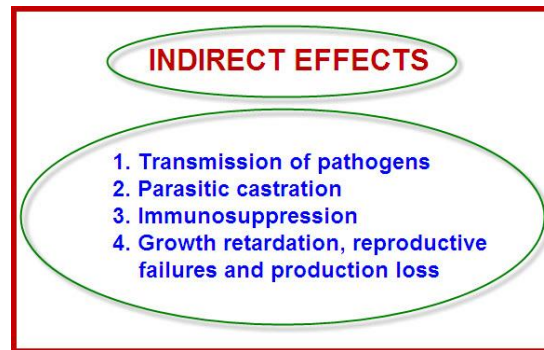
TISSUE REACTIONS CAUSED BY PARASITES TO THE HOST INTRODUCTION

- Parasites affect the host by two major ways. They may cause direct injuries during the process of entry, establishment, feeding and multiplication in the host and indirectly by transmitting pathogens that may kill or affect the well being of the host.



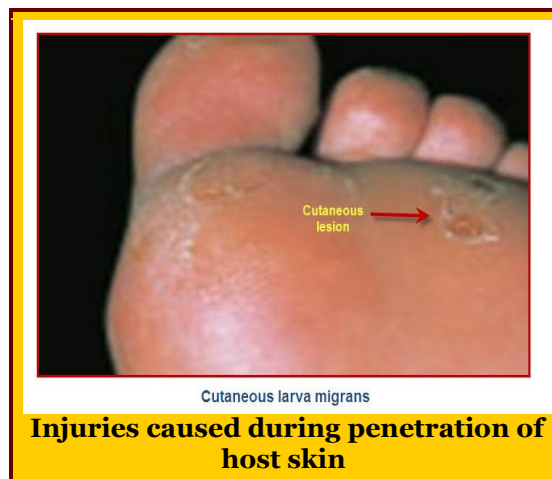
EFFECTS OF PARASITES ON HOST



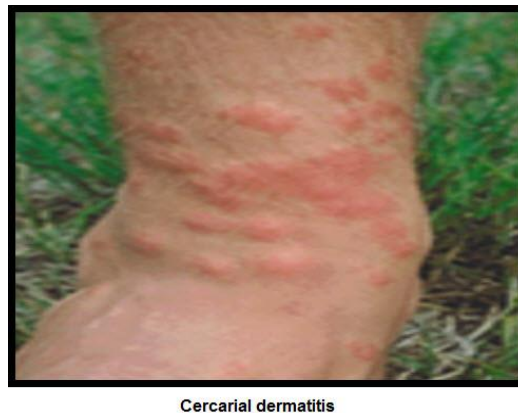


INJURIES DURING ENTRY OF PARASITES

- Certain parasites cause direct injuries to the host, during the process of entering the host.
- The hookworms (*Anchylstoma sp.*, *Bunostomum sp.*) cause injuries to the cells and underlying connective tissue when they penetrate the skin of the host.



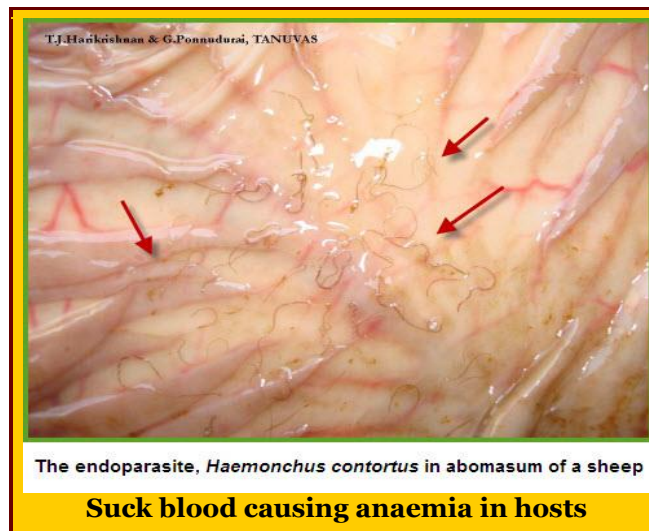
- Cercaria of certain schistosomes causes dermatitis while penetrating the host's skin.



Cercarial dermatitis

LOSS OF BLOOD AND THE ASSOCIATED ANAEMIA

- Some parasites suck blood (hookworms, barbers pole worm, mosquitoes, tick etc) from the hosts resulting in anaemia.
- The quantum of blood imbibed by insects and ticks and the resultant blood loss may not impair the health of the host, but the quantum of blood imbibed by some species of helminths, such as barber's pole worm, hookworms and red worms of equines is immense and may prove fatal to the hosts.
- *Haemonchus contortus*, in large numbers may bleed the animals causing severe anaemia and mortality in lambs.

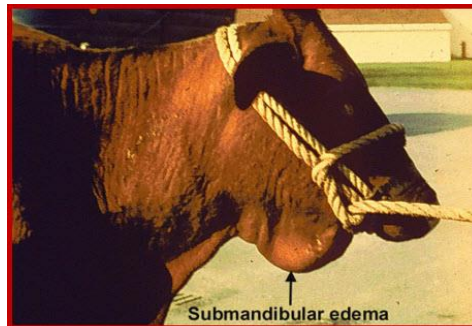


- Heavy infestations with ticks can lead to severe anaemia in animals. A single adult female tick is reported to suck around 0.5 to 2.0 ml of blood and therefore if an animal is heavily infested with ticks, substantial blood loss may occur.



Development of oedema in hosts

- Loss of plasma proteins especially albumin (hypoalbuminaemia) results in oedema where fluid accumulates in the submandibular space, between the mandibles which is commonly known as **bottle jaw** or in the peritoneal cavity (**ascites**) and thoracic cavity (**hydrothorax**).
- Exchange of fluid between blood and interstitial spaces is controlled by Hydrostatic Pressure (HP) and Plasma Colloid Osmotic Pressure (COP). If the hydrostatic pressure is high, fluid will leave the blood vessels and seep into the interstitial spaces. On the contrary, if Colloid Osmotic Pressure is high, then fluid will be drawn into the blood vessels.
- The plasma protein, albumin balances these two forces. Loss of blood results in hypoalbuminaemia and as a sequel Colloid Osmotic Pressure is reduced and Hydrostatic Pressure is increased. This results in seepage of fluid from the blood vessels into interstitial spaces resulting in oedema. Oedema is commonly seen in parasitic diseases where loss of blood occurs as in Haemonchosis, Fascioliosis, Amphistomosis etc.



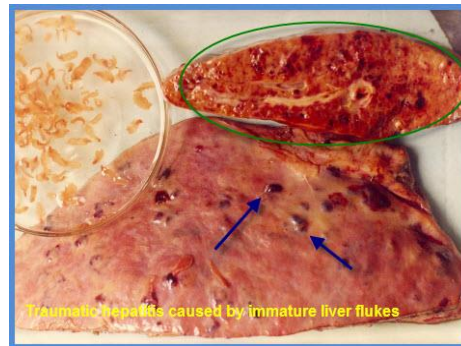
CHANGES IN PROTEIN METABOLISM AND MINERAL LEVELS

- Changes in protein metabolism and mineral levels commonly occur in parasitic infections especially in helminthic infections.
- Reduced level of aminoacid incorporation in muscle protein results in reduced weight gain and weight loss.
- Reduced incorporation of aminoacids into protein by wool follicles affects the wool quality.
- The wool of helminths affected sheep become brittle and wither away.
- Mineral deficiencies affects the growth rates since skeletal size (bone size) ultimately determines the capacity of growing animal to accumulate muscle.
- All these results in reduced weight gain, reduced wool growth and reduced milk production in infected animals.



DESTRUCTION OF TISSUES

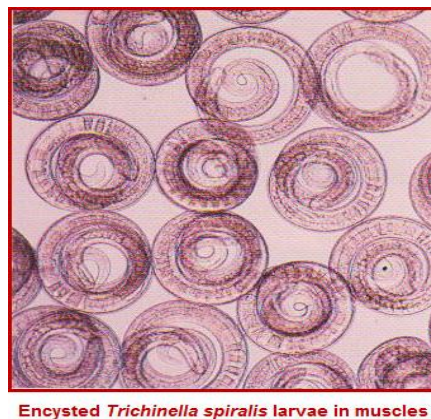
- Many parasites during their course of development undergo migration in the tissues of hosts.
- Examples include, immature stages of liver flukes that migrate in the liver of cattle, sheep and goats causing traumatic hepatitis.



- Larvae of *Toxocara canis*, *T. cati*, *T. vitulorum* and *Ascaris suum* migrate through liver and lungs inflicting physical damage, especially to lungs.



- *Trichinella spiralis* larvae cause severe myositis as they migrate through the muscles of their hosts.



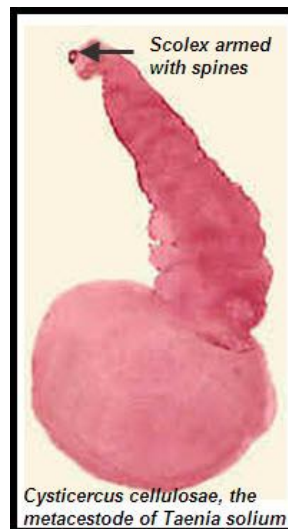
MODULE-7: TISSUE REACTIONS CAUSED BY PARASITES TO THE HOST

Learning objectives

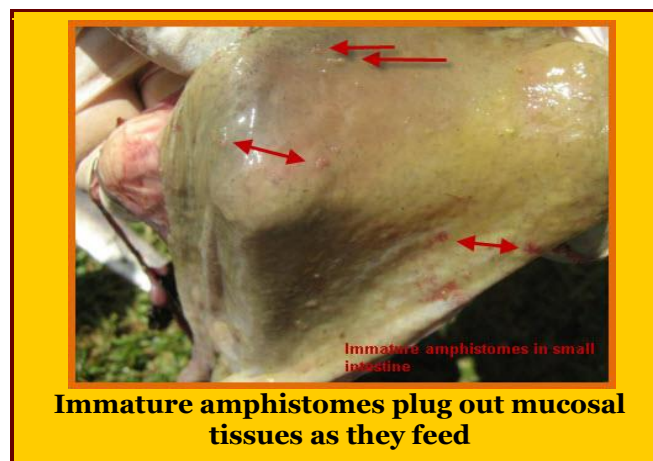
- This module will continue to inform about the effects of parasite on hosts.
- The tissue reactions caused by different parasites during establishment and feeding on hosts, mechanical interference, pathological changes, effect of secretions or excretions and the immunological reactions of the host to parasites resulting in immunopathological lesions will be taken up in this chapter.

INJURIES DURING ESTABLISHMENT AND FEEDING

- Flukes, tapeworms and acanthocephalans armed with suckers, hooks and spines cause irritation to the mucosal surfaces of intestines.



- Immature stages of amphistomes ingest plugs of mucosal tissues as they feed.



- Biting lice such as *Bovicola bovis* browse on the epidermis and cause intense irritation, inflammation and leakage of serum from damaged skin.
- Ticks also cause inflammation and leakage of serum from damaged skin in sensitized animals.



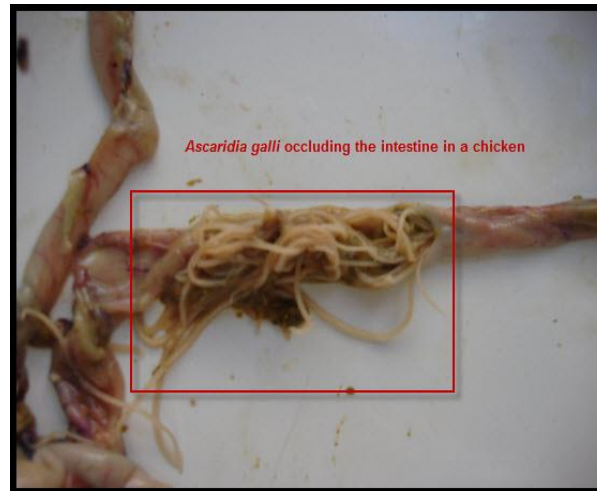
- Sarcoptes mites pierce and burrow into the skin of animals leading to irritation, itching, and inflammation of the skin characterized by keratinization, thickening and wrinkling of skin.



MECHANICAL INTERFERENCE

Occlusion

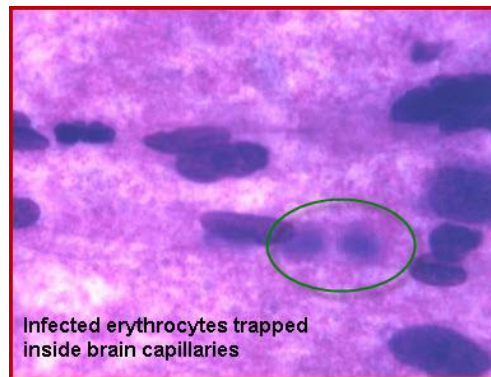
- Death due to intestinal obstruction by parasites is common, eg. ascarids and tapeworms.
- Occlusions of bile ducts by liver flukes obstruct the flow of bile resulting in icteri.



- Adult filarids lodged in lymphatic ducts cause aggregation of connective tissue that results in blockage of lymph flow and oedema in hosts.
- Gapeworms block the air passages causing dyspnoea and asphyxia in fowls.
- Lungworms such as *Dicytocaulus viviparous* cause blockage of bronchi leading to atelectasis and emphysema in lungs of cattle.



- Occlusion of blood vessels with thrombus as in the case of *Strongylus vulgaris* in horse may prove fatal if the coronary artery or brachiocephalic trunk is involved.
- In haemoprotozoan diseases, such as babesiosis and malaria, protozoa infected erythrocytes get trapped in the microcirculation of organs, impeding blood flow resulting in hypoxia and necrosis of tissues.



Pressure atrophy

- Pressure upon organs by larval stages of parasites, especially those of tapeworms result in atrophy or distortion of the organ involved.
- *Coenurus cerebralis*, the larval stage of the tapeworm *Taenia multiceps* exert pressure on the brain of sheep causing the condition known as Gid or Staggers.

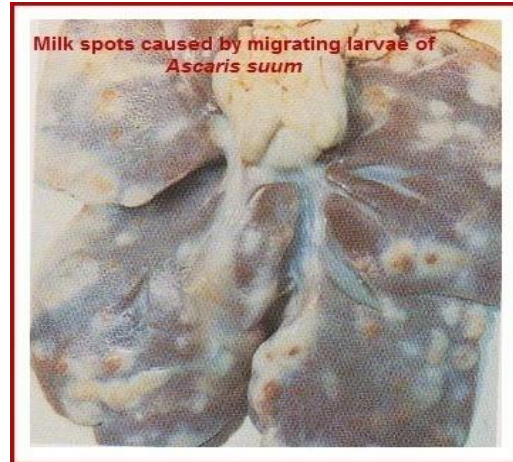


- Similarly, the larval stages of *Echinococcus granulosus*, form hydatid cysts in various organs, especially in liver and lungs of animals and man that causes severe problems in intermediate hosts, especially humans.



PATHOLOGICAL CHANGES

- One of the effects of parasites on hosts includes changes in tissues that are affected by the parasite.
- Some parasites cause chronic inflammatory reactions resulting in fibrosis and formation of nodules in the inflamed tissues.
- In *Ascaris suum* infections, spots of white fibrous tissue (Milkspots) are formed in the liver of affected piglets.



- In sensitized sheep, larvae of *Oesophagostomum columbianum* are encapsulated within nodules owing to inflammation and infiltration of eosinophils and foreign body giant cells to cause the condition known as pimply gut.



Pimply gut caused by *Oesophatostomum columbianum* in sheep

- Larvae of warble flies form cysts in the back of cattle and develop within it until ready to leave the hosts for pupation on the ground.



Hypertrophy

- Hypertrophy commonly occurs in all intracellular parasites, for example, in coccidiosis, the intestinal epithelial cells are markedly enlarged.



Intestinal epithelial cells are markedly enlarged in Coccidiosis infected goat.

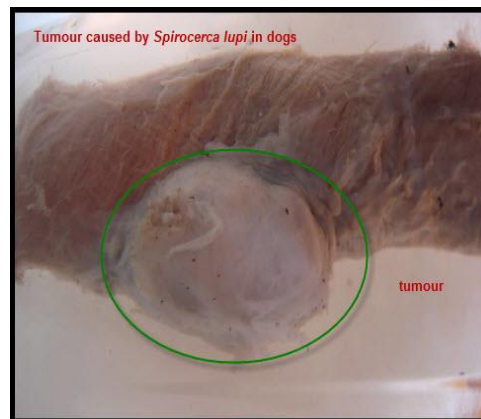
Intestinal cells are hypertrophied (eg. coccidiosis)

Hyperplasia

- Hyperplasia is associated with some parasites as in liver flukes that cause increased division of cells lining the bile ducts resulting in thickening of the bile duct.

Neoplasia

- Another tissue change is neoplasia which is associated with *Spirocerca lupi*, the gullet worm of dogs. Oesophageal nodules, transformed into a granuloma and later into sarcoma are common in *S. lupi* infections.

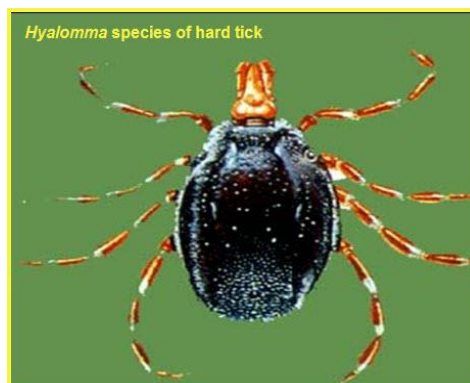


Metaplasia

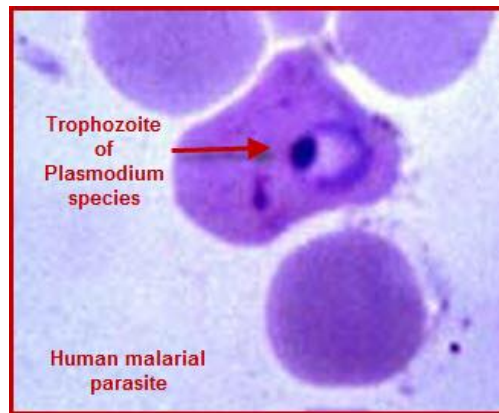
- In infections caused by the lung fluke, *Paragonimus westermani*, the parasite is surrounded by epithelial cells and fibroblast cells in lungs. This is a classical example of metaplasia where one type of tissue transforms into another without the intervention of embryonic tissue.

EFFECTS OF SECRETIONS AND EXCRETIONS

- Ticks secrete a toxin that causes the condition known as tick paralysis in mammals. Eleven hard ticks and one soft tick is associated with tick paralysis. Tick paralysis is characterized by acute ascending flaccid motor paralysis.

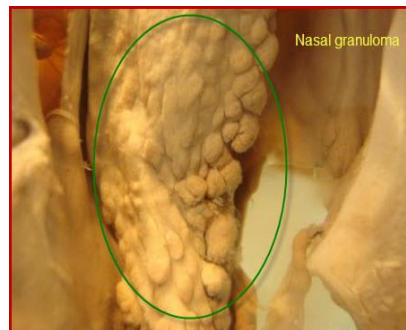


- Release of parasite internal antigens results in anaphylactic shock in some cases.
- Metabolic products released during erythrocyte destruction in malaria causes febrile responses in hosts and toxins/metabolic products secreted by trypanosomes cause injuries to the host.



IMMUNOLOGICAL REACTIONS

- Host immune response to eggs of *Schistosoma nasale* cause nasal granuloma in cattle.



- The nodular worm of sheep and goats, *Oesophagostomum columbianum*, may cause calcified nodules in sensitized hosts impairing digestion and absorption of nutrients in their hosts.



MODULE-8: RESISTANCE OF HOSTS TO PARASITIC INFECTIONS/INFESTATION

Learning objectives

- This module will introduce the students to parasitic immunity, factors that influence the host immunity, natural and acquired resistance to parasitic infection, types of parasitic immunity etc.

INTRODUCTION

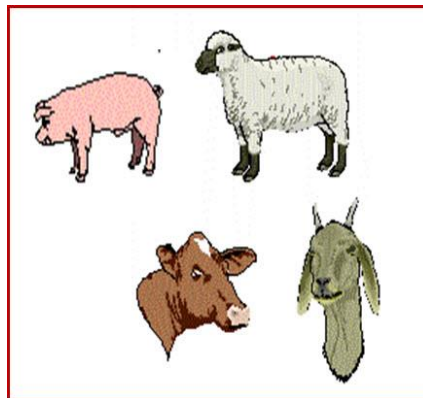
What is Resistance?

- Resistance is the defense mechanism exhibited by the host against a foreign body or a pathogen.
- The host's defense mechanism, which is directed against the parasites either kills or retards their establishment and limits their multiplication.
- A host is said to be susceptible if it is capable of being infected by a specific parasite. This implies that the physiological state of the host is such that a parasite is capable of establishing itself in the host.
- A host is said to be resistant if its physiological state prevents the establishment and survival of a parasite.

FACTORS THAT INFLUENCE HOST RESISTANCE

Species resistance

- Host animals are susceptible to some parasites and resistant to others.
- For example, majority of the helminths of cattle are incapable of infecting sheep and goats.
- The normal resistance of various species to various pathogens is due to presence of antibodies on their erythrocytes called isohemagglutinins.



Breed Resistance

- Certain breeds of animals show natural / innate resistance to some parasitic infections, e.g., West African hump less cattle (NDama and Mutura) are resistant to trypanosomiasis and they are called as trypanotolerant breeds.

- Similarly, Red masai sheep of East Africa is more resistant to *Haemonchus contortus*. Zebu (*Bos indicus*) shows high resistance to *Boophilus* ticks than exotic cattle (*Bos taurus*).
- This is probably a result of selection of the most resistant animals over many years.



Age of the host

- Young animals are generally more susceptible than adults to parasitic infections, eg. pigs less than 4 months old are more susceptible to *Ascaris suum*.
- Similarly, lambs are more susceptible to *Moniezia spp* (tapeworm) and *Trichostrongyles* (round worms).
- Conversely, young animals are more resistant to babesiosis than older cattle and this phenomenon is known as inverse/reverse age resistance.



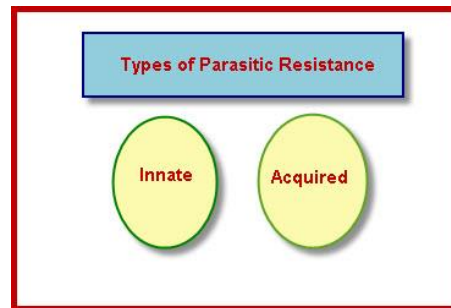
Nutritional status of the host

- Poorly fed animals are more susceptible to parasitic infections, e.g., Vitamin (A, D & B Complex) and iron deficiency may interfere with establishment and maintenance of host resistance to blood sucking parasites.

Genetic constitution of host

- The best analyzed example of genetically determined resistance is sickle cell anaemia and its role in resistance to malaria in humans. Humans who inherit the sickle cell anaemia trait possess haemoglobin S (HbS) which confers resistance on humans to malaria.
- Sheep with Haemoglobin A show superior resistance to infections with *Haemonchus contortus* and *Ostertagia circumcincta* as compared to sheep with Haemoglobin B. Sheep with Haemoglobin A mount an effective self cure phenomenon than sheep without Haemoglobin A.

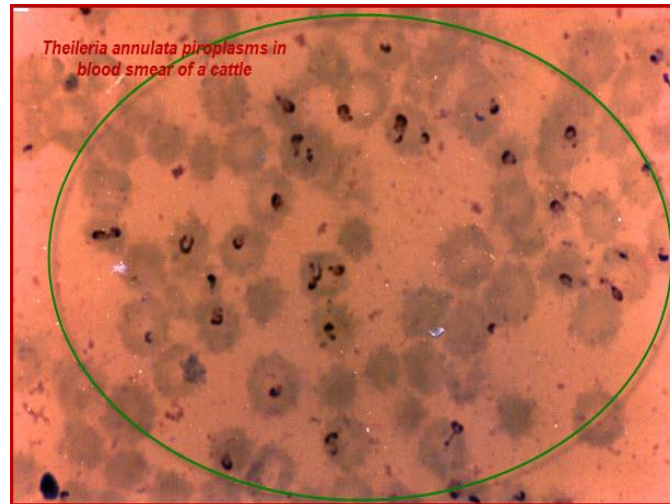
TYPES OF PARASITIC RESISTANCE



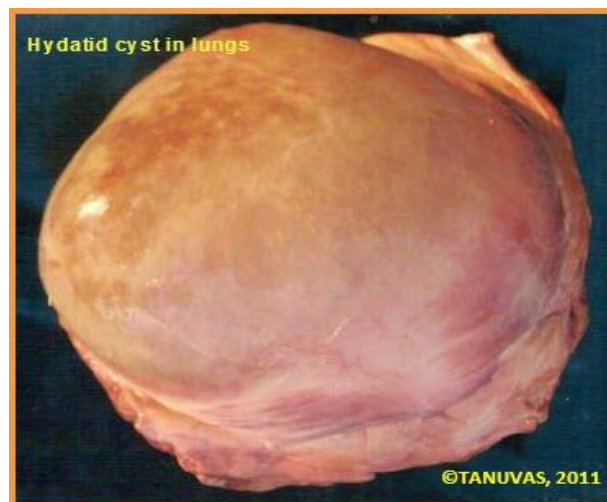
- **Innate resistance or natural resistance to parasites** is the resistance or immunity possessed by the host animal from birth. In other words, innate resistance is physiological incompatibility between certain parasites and the host that prevents the entry and establishment of the parasite in the host. Eg. Helminths such as hookworms and schistosomes may not be able to penetrate the skin and gain entry into the host, Trypanosomes upon reaching the blood stream may be destroyed by substances present in serum. Intracellular parasites such as *Theileria annulata* of cattle may be unable to enter other hosts because of deficiency of essential surface molecules in the host cells of unnatural hosts.
- **Innate resistance** or immunity can be complete / absolute or incomplete / Relative.
- A host is said to possess *complete innate resistance or immunity* when a parasite is not able to infect a host under any circumstance, eg. sheep cannot be infected with ascarids.
- When a host innately resistant to a species of parasite becomes infected owing to lowered health, it is *incomplete innate resistance*.
- **Acquired resistance or immunity** is resistance acquired by the host during its life time as a result of exposure to a particular parasite or through inoculation of parasite antigen.
- **Acquired resistance** may be either active or passive. *Active acquired resistance* is resistance acquired in response to the introduction of live or killed parasites. This may be either natural or artificial.
- **Natural active resistance or immunity** is derived following repeated exposure to an infection while artificial active immunity is derived by inoculation of parasites or their stages. It is stronger, longer lasting, and diverse and takes time to fully develop.
- An **immunity** that is developed in host following natural exposure to parasites is actively *acquired natural resistance or immunity*.

TYPES OF NATURAL RESISTANCE

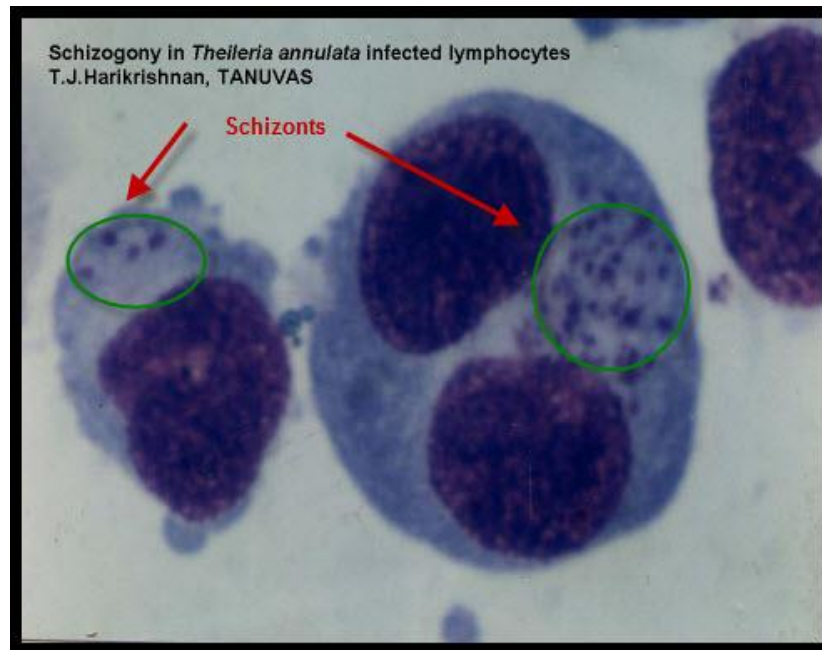
- Different types of natural resistance or immunity viz., premunity, concomitant immunity and sterile/ solid immunity are seen in parasitic infections.
- **Premunity** is a term used to describe resistance that is established after the primary infection has become chronic and is only effective only if the parasite persists in the host or in other words Premunity is immunity developed due to presence of residual parasites in the host.
- It is also known as incomplete immunity or infection immunity (Coinfectious immunity).
- Premunity is commonly associated with haemoprotozoan diseases such as theileriosis and babesiosis.



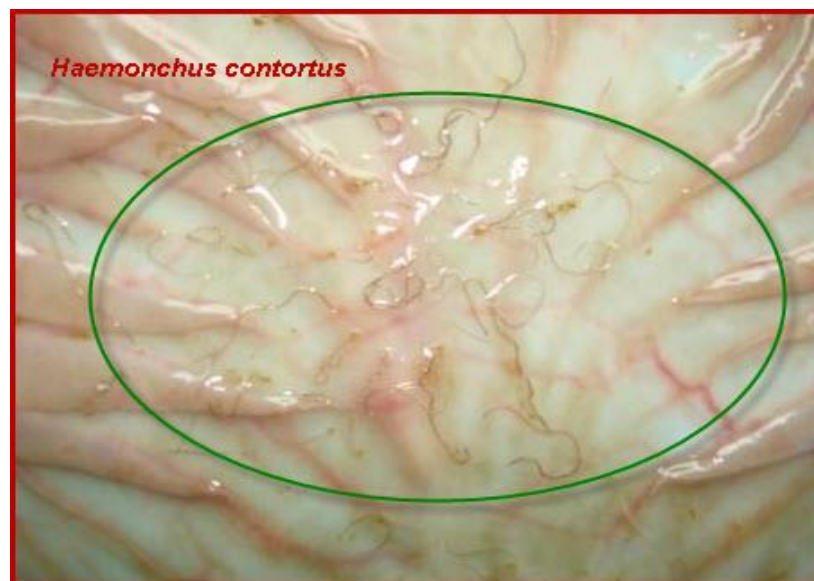
- **Concomitant immunity:** It is one type of premunity developed as a result of stimulation of the host's immune system by existing adult parasites. This immunity does not interfere with the survival of existing adult parasites but prevents the entry and establishment of newly invading larvae. This type of immunity is seen in schistosomiasis and cystic echinococcosis (hydatidosis).



- **Sterile immunity:** Immunity maintained by the host even after the parasite is eliminated. The immune system remains sensitized for many years e.g. *Theileria annulata* infection in cattle, *Plasmodium cynomolgi* infection in monkeys, etc.



- **Self cure** is a phenomenon where adult worm populations are expelled following larval challenge.
- It is a natural remedial mechanism by which the host gains a temporary respite from persistent blood loss while aging parasite population is eventually replaced by robust young generation.
- The expulsion of adult worm population is considered to be a sequel to immediate type hypersensitivity reaction (mediated by Ig E) to antigens derived from developing larvae. Self cure will not protect the animal since larval challenge often develops to maturity.
- Self cure phenomenon is commonly associated with *Haemonchus contortus* infection in small ruminants and cattle.



PASSIVE IMMUNITY

- Passive immunity refers to transfer of antibodies or lymphocytes from immune to non immune animals.
- It is only temporary and does not last long. It could be either natural or artificial.
- Natural passive immunity refers to transfer of immunity naturally from mother to offspring by placenta or through colostrum and these antibodies protect the young for a certain period.



- Artificial passive immunity is immunity transferred to a non immune animal through injections of sera produced in another animal by repeated injections of particular antigens.
- The process of producing excess antibodies against pathogens is termed as hyperimmunisation.

MODULE-9: IMMUNITY AGAINST PARASITIC INFECTIONS

Learning objectives

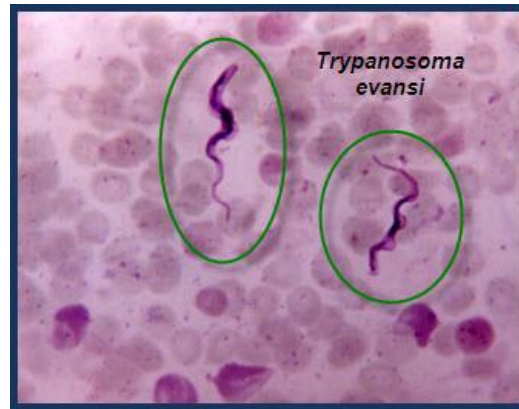
- The objective of this module is to elucidate the types of immune response against parasitic diseases and how the immune response prevents the establishment and development of the parasite in the host besides immunopathological lesions or effects that occur following an immune response to the parasite. The kinds of parasite antigens is also dealt in this chapter.

TYPES OF IMMUNE RESPONSE IN PARASITIC DISEASES

- If the parasite successively overcomes the animal's innate defence mechanisms, it will elicit an immune response specific to the parasite and these immune responses may be humoral mediated or cell mediated. Humoral immunity is mediated through antibodies which are large glycoproteins released by B cells. These antibodies are present in blood, lymph and cerebrospinal fluid. There are five classes of antibodies, each of which is distinguished from the other by their heavy chains.
 - *Immunoglobulin G (IgG)*: Major antibody in serum. Long half life
 - *Immunoglobulin M (IgM)*: Largest and the first antibody to be produced in an immune response. Presence denotes acute infection. These antibodies are able to neutralize, fix complement, agglutinate and immobilize parasitic antigens.
 - *Immunoglobulin A (IgA)* : Predominantly found in secretions (mucous, saliva, tears) and colostrums and hence called secretory Ab. It is also known as mucosal antibody.
 - *Immunoglobulin E (IgE)*: Also called as reaginic antibody. This Ab binds to the surface of mast cells and causes degranulation of the cell and release of histamine into circulation. This Ab is associated with helminthic infections or allergies.
 - *Immunoglobulin D (IgD)*: The function of this Ab with respect to parasitic infections is not known.
- Parasites are not composed of a single antigen but a mosaic of antigens. Animal hosts therefore will recognize a number of these different antigens resulting in a polyclonal antibody response. In addition, the host will produce different immunoglobulin classes having the same antigen specificity.
- In general, humoral immune response (antibodies) controls extracellular parasites in the intestine, blood stream and tissue fluids. Humoral immune response is predominant in helminthic infections involving IgE, IgA and IgG antibodies. Many helminth infections are associated with characteristic type I hypersensitivity, including eosinophilia, oedema, asthma and urticarial dermatitis where IgE levels and eosinophil levels are increased. One of the major tasks of eosinophils is the destruction of helminths.
- **Complement** is the name given to a complex of proteins that are present in the serum and which are capable of being activated to become enzymatically active. When complement is attached to an antigen antibody complex, a series of reactions occur with each component acting enzymatically to activate subsequent components in a cascade fashion. Activation of complement can also occur in the absence of immune complexes by molecules present on the surface of parasites.

HOW ANTIBODIES PREVENT THE ESTABLISHMENT OF PARASITES ?

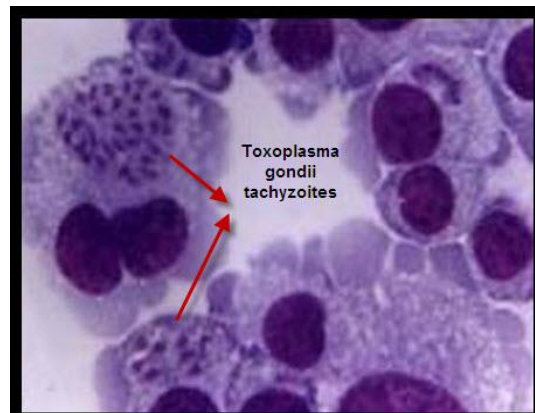
- **Opsonins** are antibodies that render the antigens more sensitive for phagocytosis by leukocytes. The presence of complement is essential for opsonisation.
- **Ablastins** are antibodies that inhibit the reproduction of parasites. These antibodies have been demonstrated in trypanosome infections. They can be transferred from mother to offspring through the milk.



- **Neutralisation:** Antibodies that bind to parasites and neutralize their toxins and inactivate the enzymes. Antibodies neutralize the proteases used by larvae to penetrate tissues, antibodies also bind with the excretory and secretory products of helminths and prevent moulting, antibodies also act against exsheathing antigens and inhibit larval development. Antibodies also block the enzyme pathways of helminths resulting in cessation of egg production and development of adults.
- **Immobilization:** Antibodies bind to flagella and prevent these parasites from phagocytosis
- Lysis of bound parasites with the help of complement.
- Presence of IgA in secretions will prevent the attachment of parasites in the mucosal surfaces.
- Masking of certain target cells by antibodies so that parasites are not able to attach to them
- The openings and orifices of parasites are blocked by the antigen antibodies complexes causing lysis/death.

CELL MEDIATED IMMUNITY

- Cellular immunity is mediated by T cells and like the antibody response, there is involvement of macrophages and the presentation of antigens in combination with specific macrophage surface molecules to the T cells. Specific cytotoxic cells can directly kill parasite infected cells. Some parasites preferentially stimulate T helper cells (subsets Th-1 or Th-2) and each helper T cell subset secretes a characteristic set of cytokines, referred to as a T cell cytokine profile. For example, in Leishmania infections, there will be Th-1 response with a characteristic cytokine profile of interleukins (IL2, IL3, IL-12 and Interferon). Cellular immunity is often directed against intracellular parasites (e.g. Theileria, Toxoplasma etc).



IMMUNOPATHOLOGICAL EFFECTS

Immunopathological effects due to immunological reactions against parasitic diseases

- Immune response evoked by the host against parasitic antigens, causing variety of harmful pathological effects to the host.
- Pimply gut in oesophagostomiosis



- Oesophageal nodules in *Spirocerca lupi* infections



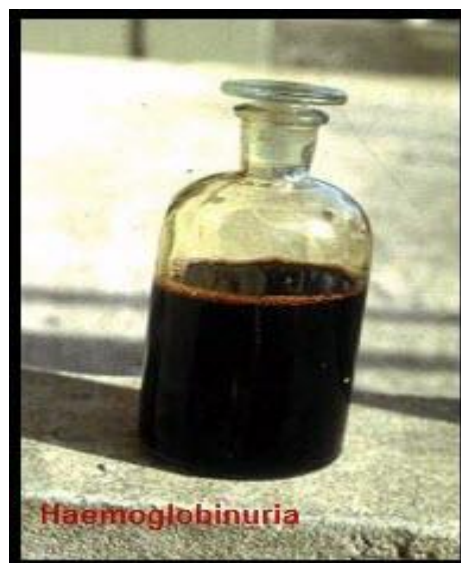
- Avascular granuloma in hepatic schistosomiosis
- Hypersensitivity reactions.

HYPERSENSITIVITY REACTIONS

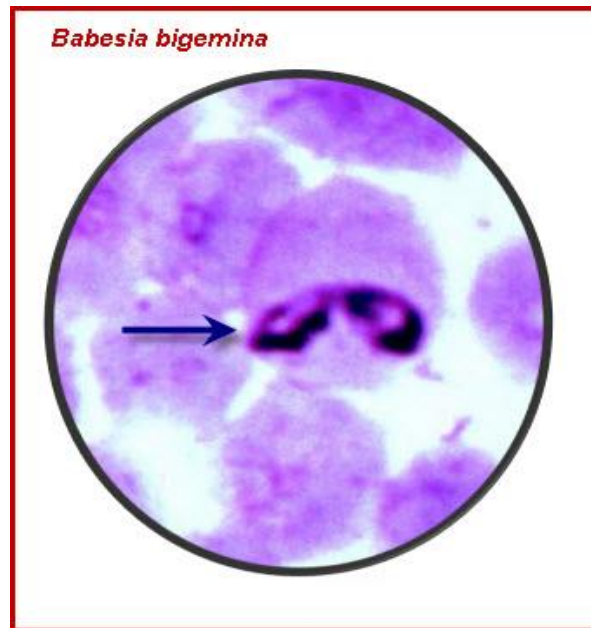
- During the process of destruction and removal of parasites by the host immune system, surrounding body tissues may be damaged. These harmful effects are called as hypersensitivity reaction.
- The different types of hypersensitivity include.
 - *Type -I (Anaphylactic shock)* immediate reaction due to binding of antigen with IgE fixed to mast cells and basophils leading to release of vasoactive, amines (histamine & serotonin) e.g., anaphylactic shock following rupture of hydatid cyst and release of hydatid fluid into the system.



- *Type I hypersensitivity* is a feature of trichomoniosis and causes local irritation and inflammation in the genital tract.
- *Type-II (Cytotoxic)* is associated with development of anaemia in malaria and haemoglobinuria in babesiosis.



- **Type-III (Immune Complex disease):** Circulating antigen antibody complexes coupled with complement are deposited in tissue space causing damage to the tissues, e.g., glomerulonephritis in babesiosis, malaria, and schistosomiasis.



- **Type-IV (Delayed):** Biting fleas secrete saliva into the skin wound and some low molecular weight components present in the flea saliva act as haptens by binding to dermal collagen resulting in local type IV hypersensitivity reaction characterized by mononuclear cell infiltration. This is commonly known as flea bite dermatitis. However, in some sensitized animals, after some months, type I reaction takes over resulting in eosinophil infiltration.



KINDS OF PARASITIC ANTIGENS

- **ES antigens:** Antigens from the excretions (E) and secretions (s) of the parasite are called ES antigens.
- **Somatic antigens:** Antigens elaborated by the entire body of the parasite is called somatic antigen.
- **Internal antigens:** Antigens obtained after the disruption of the parasites.
- **External antigens :** Antigens manifested by intact parasites.
- **Released antigens:** Metabolic products of parasites are released antigens and these are comparable to external antigens.
- **Concealed or Hidden antigens:** Antigens present on the intestinal surfaces of helminths and arthropods which are not recognized by the host. However, these concealed antigens are excellent targets for artificially induced responses.

EFFECT OF RESISTANCE ON PARASITES

- Prevention or reduction in the rate of establishment
- Structural or metabolic alterations in the parasites
- Retardation or inhibition of development, for example adult female *Ostertagia ostertagi* recovered from immunized calves show absence of vulvar flaps as compared to those recovered from non immunized calves, Similarly, *Cooperia* males derived from immunized animals showed altered morphology of spicules.
- Inhibition of reproduction, for example in ticks that fed on immunized animals there will be extended oviposition, reduced egg laying and diminished hatchability of eggs.
- Destruction of parasites in situ
- Elimination of existing infection

MODULE-10: STANDARDIZED NOMENCLATURE OF ANIMAL PARASITIC DISEASES (SNOAPAD)

Learning objectives

- This module will deal about classification of parasites on the basis of relationships and naming of parasites in accordance with the Standardized Nomenclature of Animal and Parasitic Diseases (SNOAPAD)

STANDARDIZED NOMENCLATURE OF ANIMAL PARASITIC DISEASES (SNOAPAD)

- The standard and uniform nomenclature formulated by the World Association for Advancement of Veterinary Parasitology (WAAVP) is Standardized Nomenclature of Animal Parasitic Diseases (SNOAPAD).
- Nomenclature is the process of naming the parasites.
- Members belonging to the animal kingdom are classified into phyla, classes, orders, families, genus and species. Later, additional categories such as super or sub were created to accommodate the increasing number of species discovered over the years. Super/Sub was prefixed to the existing categories (Superfamily/ Suborder). The family group includes taxa ranked as at the family and tribe levels (including super and subfamilies). The genus group includes taxa below subtribe and above species. The species group includes taxa ranked as species or subspecies.
- The names of parasites must be from Latin or Greek and not in the local language. The genus name should be a noun and the species names should be either noun or adjective.
- The name of the order ends in 'ida', e.g. Strongylida, superfamily ends in 'oidea', e.g. Ancylostomatoidea, family ends in 'idae', e.g. Ancylostomatidae and subfamily ends in 'inae', e.g. Ancylostominae.
- The first letter of the genus should always be in capital letter while the first letter of the species should be in small letter excepting for those whose names have been derived from persons, where, it shall be either in small letter or in capital letter, e.g., *Trypanosoma evansi* or *Trypanosoma Evansi*, *Cotugnia bhaleraoi* or *Cotugnia Bhaleraoi*. In printed formats, the names of the genus and species should be italicized while in written text, the names of the genus and species should be underlined. The genus and species are also named after morphological characteristics, host, anatomical location, geographical location, scientists etc.
- The law of giving importance to the scientist who first named the parasite is known as Law of Priority. If the genus and species names are after scientists, the name of the scientists and the year should follow the scientific name. While writing, a comma should be used after the scientists and not anywhere, e.g. *Schistosoma nasale*, Rao, 1932. If the name of the original author is changed subsequently for valid reasons, his name and the year have to be written within brackets after the new classified name for e.g. *Ancylostoma caninum* (Ercolani, 1859) Hall, 1913. If a parasite name is changed subsequently for valid reasons, the earlier name is considered as the synonym e.g. *Neoascaris vitulorum* is the synonym for *Toxocara vitulorum*.

Recommendations of SNOAPAD include

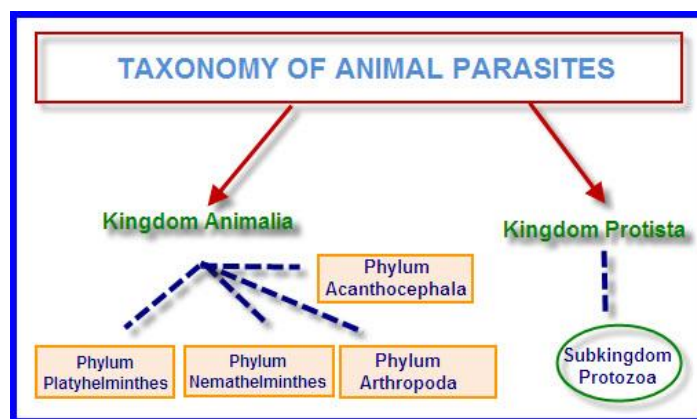
- The practice of using the suffix 'osis' to denote parasitic disease with apparent clinical signs and the suffix 'iasis' for subclinical infections are to be discontinued.
- The suffix 'osis' should be added to the full generic name or the stem of an appropriate higher taxon name or from the genitive name of the parasite by deleting the last one or two letters.
- In some, the suffix 'osis' is added to the full generic name of the parasite, eg., *Hepatozoon* - Hepatozoonosis, *Leucocytozoon* - Leucocytozoonosis
- For certain parasites, the 'osis' is added to the appropriate higher taxon name as in the case of *Schistosoma* eg. Schistosom+osis = Schistosomosis, *Ancylostoma* -Ancylostom+osis = Ancylostomosis, *Babesia* - Babesi+osis = Babesiosis
- When the taxa, ends in 'x' as in Demodex, the stem is formed from the genitive as in the given example Demodex, Demodicis - Demodic+osis=Demodicosis
- Use of nomenclature such as Malaria, Surra, Myiasis, Mange etc., to be continued in addition to the new names as these are well established names and have been in use for a long period.

MODULE-11: GENERAL DESCRIPTION OF HELMINTH PARASITES AFFECTING DOMESTIC ANIMALS AND BIRDS

Learning objectives

- After reading this module the students will understand the following
 - Phylum Platyhelminthes
 - Phylum Nematelminthes
 - Phylum Acanthocephala
 - Phylum Arthropoda
 - Kingdom Protista

TAXONOMY



PHYLUM PLATYHELMINTHES

- Dorsoventrally flattened worms, Unsegmented or pseudosegmented body, parasites of gastrointestinal tract or viscera, hermaphrodites, body cavity absent, organs are embedded in tissue parenchyma, covered by thin membrane called cuticle which may be smooth, spiny or tuberculated, organs of attachments such as suckers, hooks or bothria are present, excretory organ is the flame cell or solenocytes.
- There are two classes, viz., Trematoda (flukes) and Cestoda (tapeworms).





CLASS TREMATODA (FLUKES)

- Endoparasites with complex life cycles, involving snail as intermediate hosts and vertebrate hosts as definitive hosts.
- Adults live in gut or tissues of vertebrate host and reproduce sexually.
- Larval stages live in tissues of snails (mollusk) and undergo asexual reproduction.



Adult liver flukes (Obligate parasites)



Lymnaea species of snail

CLASS CESTODA (TAPEWORMS)

- Adults are endoparasites in gut of vertebrates.
- Pseudosegmented and hermaphroditic.
- Uptake of nutrients occurs through the tegument.
- The body divided into a scolex, neck and strobila.
- Strobila consists of a linear series of proglottids.
- Each proglottid carries a set of female and male reproductive organs.
- Life cycle is indirect and involves one or two intermediate hosts.
- Immature forms (Bladderworms or larval tapeworms) in tissues of intermediate hosts.



PHYLUM NEMATHELMINTHES

Phylum Nematelminthes

- Unsegmented roundworms with complete digestive tract.
- Mouth and anus are on opposite ends to the body.
- No circulatory or respiratory systems.
- Class Nematoda is only of parasitic importance.

Class Nematoda

- Includes many free living and parasitic roundworms.
- Cylindrical and metamerically segmented.
- Body cavity (pseudocoelum) is present.
- Cuticle is present with modifications that help to identify the different species.
- Digestive tract consists of mouth, pharynx, oesophagus, intestine, caecum and anus.
- Anus is not terminal as in annelids so that a tail is present.
- Sexes are separate, flame cells or cilia are absent.
- Life cycle is either direct/indirect.



PHYLUM ACANTHOCEPHALA
(ACANTHUS - THORN, KEPHALE - HEAD)

- Thorny headed worms, cylindrical with thick cuticle and retractable proboscis provided with spines / hooks, alimentary canal absent, sexes separate, life cycle indirect with an invertebrate intermediate host. e.g., *Macracanthorhynchus hirudinaceus* in the stomach of pigs, intermediate hosts are crustaceans.

MODULE-12: VETERINARY HELMINTHOLOGY

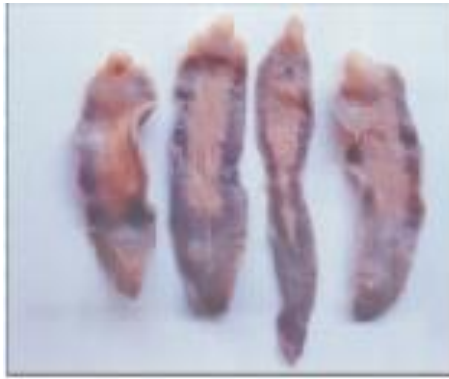
Learning objectives

After reading this module the learner will understand the following

- Helminthic infection affecting livestock, its morphology, life cycle, transmission, pathogenesis, clinical signs, diagnosis, treatment and control.
- Various trematodes, cestodes and nematodes affecting man and animals.
- Recent advances in the development of vaccine and other international regulations for use of novel control measures.

VETERINARY HELMINTHOLOGY - AN INTRODUCTION

- The name 'Helminth' is derived from the greek word 'helmins' or 'helminthos' means 'a worm'. Helminths is applied only to the parasitic and non-parasitic species belonging to the Phylum-Platyhelminthes [fluke, flat worm and tape worm] and Phylum Nematelminthes (round worm).



CHARACTERS OF PHYLUM PLATYHELMINTHES

- They are dorso-ventrally flattened.
- They are bilaterally symmetrical.
- Usually hermaphrodite.
- Body cavity is absent.
- Organs are embedded in specialized connective tissue known as 'Parenchyma'.
- Excretory organs are called 'flame cells'.
- No definite anus.
- Respiratory and circulatory systems are absent.
- Life cycle is usually indirect.
- Phylum consists of 4 classes.

CLASS

Turbellaria (Eddy worm)

- They are non-parasitic species living in fresh water, sea and land.
- Body has ciliated covering.
- They are not occurring in domestic animals.

Trematoda (Flukes)

- They are ecto/endo parasitic.
- The teguments are syncytial and absorptive.
- Alimentary canal and adhesive organs are well developed.

Eucestoda (True cestodes)

- They are elongated, tape like endo-parasites.
- They have no alimentary canal, food is absorbed through surface.
- Teguments are syncytial and highly absorptive, usually divided into segments. Adhesive organ at anterior end.

Cotyloda

- They are previously classified as "Cestodaria".
- Mainly parasites of fish except few genus occurs in man and domestic animals.
- Scolex lacks true hold fast organ.
- The segmentation may be absent or indistinct.

CHARACTERS OF CLASS TREMATODA

- **Common name** - Trematodes/flukes or flat worms
- "Trematoda" derived from the Greek word 'Treme' means holes. They are equipped with hooks/suckers for anchor

Characters

- Body is dorso-ventrally flattened, unsegmented and leaf-like.
 - All organs are embedded in parenchyma and there is no body cavity.
 - Suckers/hooks or clamps are present to attach to the host.
 - The mouth(oral aperture) and alimentary canal is present but no anus.
 - Mouth leads to muscular pharynx, oesophagus followed by two (branched or unbranched) branched caeca.
 - The branched excretory system has flame cells and it discharges the waste product into excretory bladder, which usually has a posterior opening.
 - All trematodes are hermaphrodites except the members of the family "Schistosmatidae", which are unisexual.
 - The life cycle is usually indirect in Digenea and direct in Monogenea.
- Three subclasses under the class Trematoda are:

Subclass:Aspidogastrea

- Most of them have only one host, a mollusc.
- Few are found in turtle and fishes with molluscs and lobsters as intermediate hosts, but none are found in domestic animals.
- Adhesive organs on ventral side known as "Baer's disc" or "opisthaptor".

Monogenea

- Parasites of cold aquatic vertebrates, fishes, amphibians and reptiles.
- Most of them are ectoparasite.
- None on domestic animals.
- Life cycle is direct.
- They have posterior adhesive organ called as 'HAPTOR' which serves as suckers, clamps or hooks.
- They are oviparous or viviparous.

Digenea

- All the species are parasitic in animals, birds and humans.
- They have 2 suckers.
 - Oral suckers: Found on dorsal side of the anterior end.
 - Ventral sucker: Found on ventral surface.
- Life cycle is indirect. It requires one or two intermediate hosts. Invariably Snail act as first I/H.

Common forms of Adult digenetic Trematodes

- Distome
- Monostome
- Gasterostome
- Amphistome
- Echinostome
- Strigeoid
- Schistosome

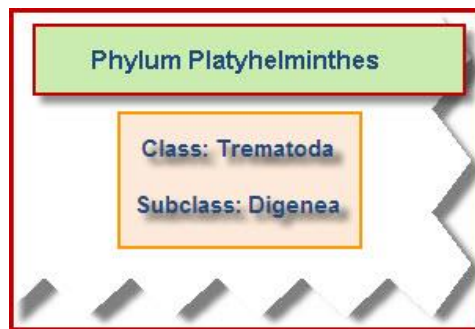
MODULE-13: PHYLUM - PLATYHELMINTHES

Learning objectives

After reading this module the learner will understand the following

- General characters of digenetic trematodes
- Various systems of digenetic trematodes
- Eggs and larval stages of digenetic trematodes

PHYLUM: PLATYHELMINTHES INTRODUCTION



Characters of subclass: Digenea

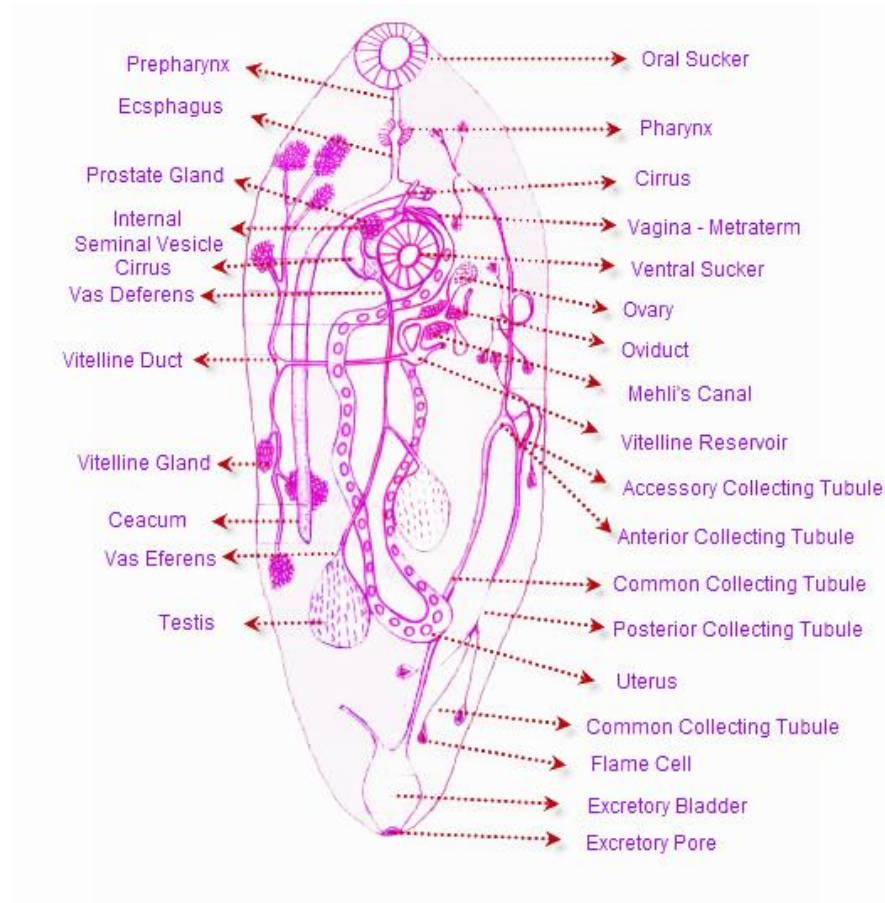
- Trematode is derived from the Greek work which means worms with holes
- Trematodes are dorsoventrally flattened flukes that are slender and leaf like or possess fleshy bodies.
- Absence of body activity (Acoelomate)
- Cuticle or tegument may be smooth or spiny.
- Parasites of alimentary canal or associated organs or in blood vessels.
- Most are hermaphrodite except schistosomes.
- Life cycle is complex and indirect.
- Organs of attachment include an anterior sucker known as oral sucker placed at the anterior end of the body and the ventral sucker also known as acetabulum at the anterior third of ventral surface, but position varies from species to species or may be absent, for example in liver flukes and lancet flukes, the acetabulum is at the anterior third of the ventral surface while in amphistomes it is located at the posterior end.

CHARACTERS OF DIGENETIC TREMATODES

- [Digestive system](#)
- [Excretory system](#)
- [Nervous system](#)

DIGESTIVE SYSTEM

- Digestive system opens at mouth with oral sucker, followed by muscular pharynx then oesophagus.
- Oesophagus leads to two branched caeca ends in a blind manner. There is no anus in trematodes. The absorption and digestion may occur through suckorial mouth, muscular pharynx, extra-corporeal digestion or by pinocytosis in trematodes.



EXCRETORY SYSTEM

- Consists of bladder a collecting organ from which number of branched tubes, run out to the parenchyma and they end in flame cells.
- Each flame cell collects the waste products from its surrounding area and discharges into the bladder.
- Bladder opens externally through excretory pore, situated at the posterior extremity of body.
- Flame cells are arranged bilaterally in a definite pattern. These arrangements may be expressed by simple formula known as “flame cell formula”.
- Flame cell formula is important for identification of species.
- **Eg.** $2[(3+3+3) + (3+3+3)] = 36$.

NERVOUS SYSTEM

- Nervous system consists of circum oesophageal ring of fibre and paired ganglia.
- From which 3 pairs of nerves forwardly and 3 pairs of nerves run posteriorly which supplies to the entire body.
- Sense organ is absent in adults but seen in young larval stages.
 - **Eg.** Miracidium and Cercaria posses eye spots (patches of pigments).

REPRODUCTIVE SYSTEM

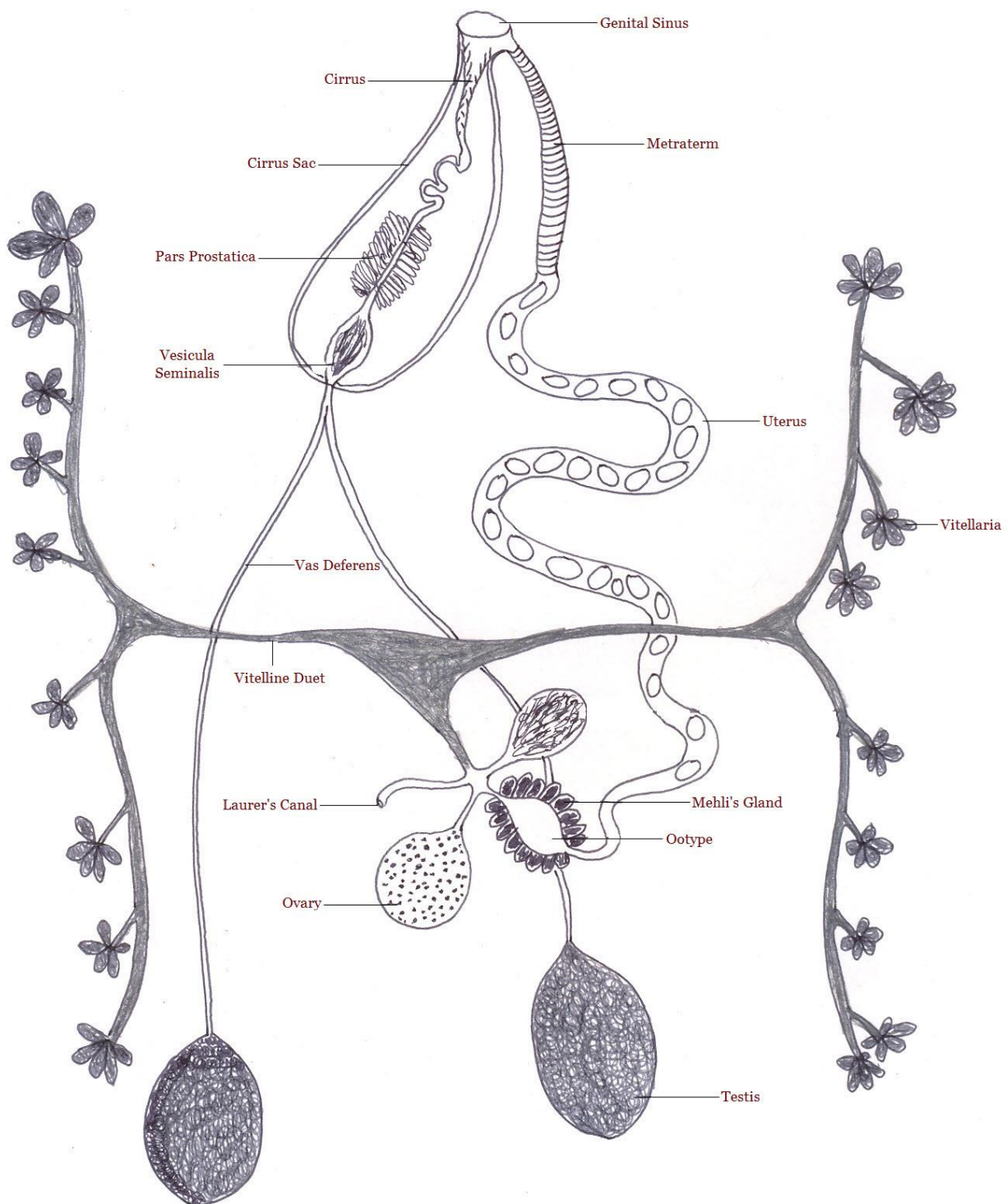
Digenetic trematodes are hermaphrodite except the members of family Schistosomatidae.

Male reproductive system

- In male, two testes are present, which may be spherical, lobed or divided into number of small bodies.
- The vasa efferentia unite to form a vas deferens. The vas deferens widens distally to form vesiculum seminalis and ends in a cirrus or penis. Cirrus may be enclosed by cirrus sac.

Female reproductive system

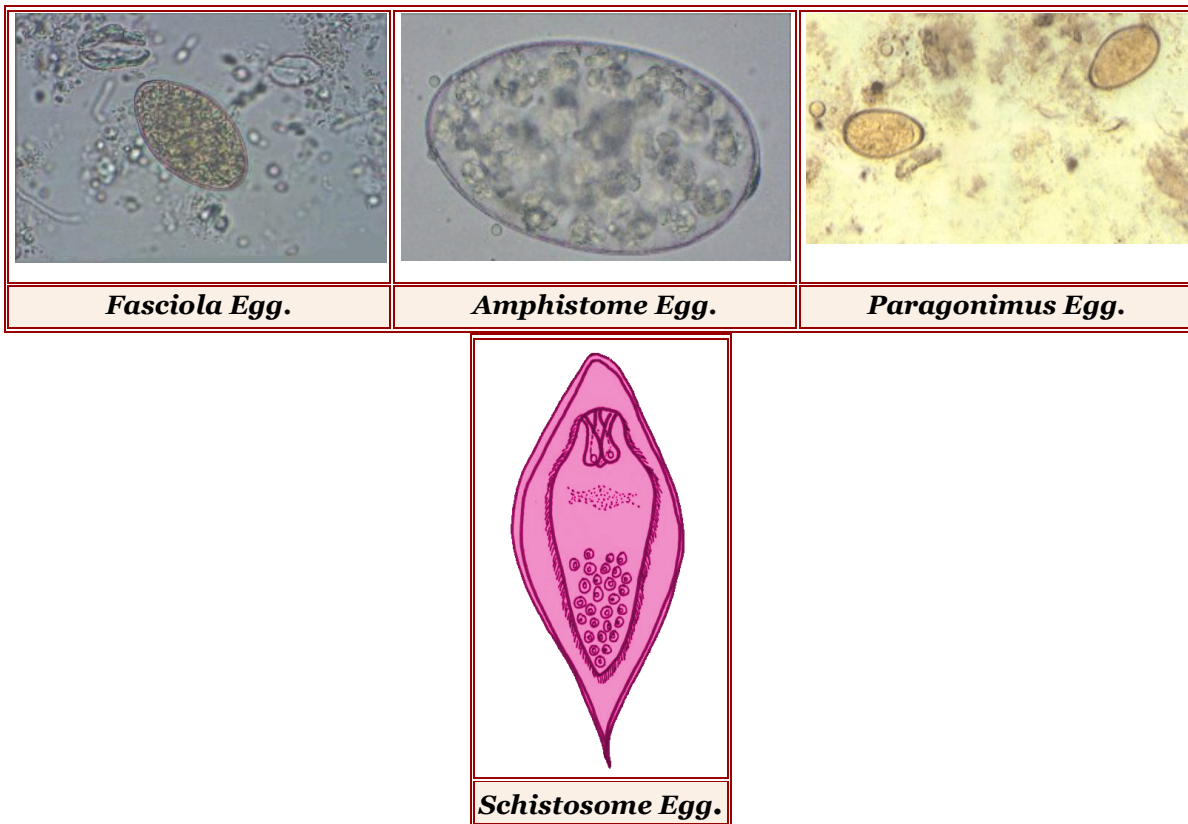
- Consists of ovary, which discharges the ova into the oviduct. Oviduct bears “receptaculum seminalis” and a narrow a canal (Laurers canal).
- A pair of vitelline gland or vitellaria with number of follicles are placed laterally, which discharges into the yolk duct. Yolk duct joins with oviduct in a special widen portion – Ootype.
- Ootype is surrounded by Mehlis (unicellular) gland. Mehlis and yolk gland collectively known as shell gland.
- Eggs after leaving the ootype enter into the uterus. The uterus may be short or much convoluted and open through genital pore is situated antero-ventral surface or postero-lateral.
- Genital pore is surrounded by sinus or atrium. In some species, the sinus or atrium develop into sucker known as genital sucker.
 - **Eg.** Cotylophoran cotylophorum.



EGG OF TREMATODES

- Usually the trematode eggs have operculum - a lid or cap like structure at the anterior end. But schistosome egg has terminal spine.
- In most of the species, the eggs are developed in the uterus, so they are ready to hatch immediately after laying.
- Generally the trematode eggs hatch in the water but in *Dicrocoelium dendriticum*, the egg hatch only after ingested by the snail intermediate host.
- Hatching of eggs is controlled by number of factors like light, temperature and salinity of water.
- Eggs cannot with stand desiccation. Under favourable condition larvae hatches out from the eggs.

Example



MIRACIDIUM

- Roughly triangular in shape.
- Anterior end is broad and has a spine.
- Body is covered with ciliated ectoderm.
- Miracidium usually provided with excretory and nervous system, sac like intestine and eyespots.
- Number of germ cells attached to the wall of the body.
- It does not feed, further development occurs only after entering into the snail I/H.
- Penetration into the snail I/H is assisted by boring action of miracidium and enzyme secreted by apical glands and penetration glands.
- After entering into the snail I/H its ciliated covering is lost and becomes sporocyst.

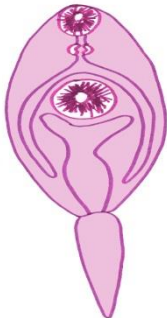
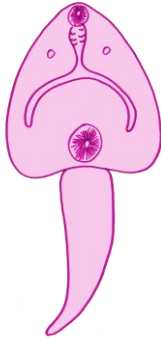
MODULE-14: LIFE CYCLE OF TREMATODE

Learning objectives

After reading this module the learner will understand the following

- Various life cycle patterns of parasitic trematodes.
- Various developmental stages encountered in the life cycle of parasitic trematodes.
- Importance of intermediate hosts in the transmission of parasitic trematodes.

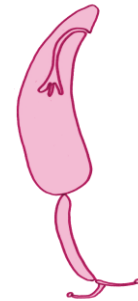
TYPES OF CERCARIA

<i>Gymnocephalus cercaria</i> • Posses two suckers situated close to each other. • Tail is simple. • Stylet or spine is absent. Eg. <i>Fasciola gigantica</i>.	
<i>Xiphidocercus cercaria</i> • Have two suckers situated close to each other. • Oral sucker armed with stylet. • Tail is simple. Eg. <i>Dicrocoelium dendriticum</i>.	
<i>Amphistome cercaria</i> • Have two suckers situated at each end i.e., not close to each other. • Tail is simple. • Eye spots are present Eg. <i>Amphistome</i>	

Furcocercus cercaria

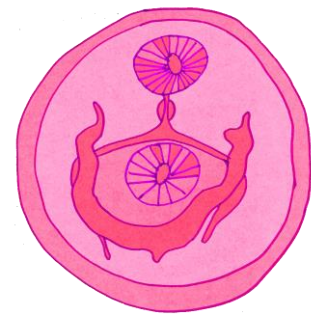
- Tail is usually forked.
- Suckers are close to each other.

Eg. *Schistosoma*



Metacercaria

- After escape from the snail I/H the cercaria encyst on or in the second I/H or on the vegetation or herbage. The encysted form undergoes physiological maturation and become infective stage.
- Encysted cercaria is known as Metacercaria. Cystogenous glands in the cercaria form a cyst. It is the last and infective stage of trematode so it has to reach the D/H to complete the life cycle.
- Metacercaria may enter into the D/H passively along with contaminated water or vegetation.
- After entering, "excystation" occurs in the intestine of D/H and the immature fluke moves to site of predilection.



[metacercaria in grass blade that survives for upto 6 months in tropical climate](#)

BASIC LIFE CYCLE PATTERN OF TREMATODES

- Invariably all trematode parasites require snail as a first I/H in which various developmental changes occur. Sometimes daughter sporocyst and daughter redia may occur.
- Some species of trematode requires only one I/H i.e., snail for completion of their lifecycle. Eg. *Fasciola gigantica*. While others, *Dicrocoelium dendriticum* requires two I/H. Snail as a 1st I/H, Brown ant -- 2 I/H. Based on the developmental changes in the life cycle of trematode, life cycle can be classified into 4 types.
- In this life cycle pattern, redial stage is absent but metacercarial stage is present. Cercaria is produced by daughter sporocyst. Infective stage is metacercaria found in the second I/H. Eg. *Dicrocoelium dendriticum* - D/H acquire infection by ingestion of infected 2nd I/H.
 - Both redial and metacercarial stages are absent. Cercaria is produced by daughter sporocyst. The infective stage is cercaria. Final host - D/H acquire infection by penetration of cercaria. Eg. Schistosome species.
 - All the developmental stages occur except daughter sporocyst or daughter redia. The cercaria is produced by redia. Infective stage is metacercaria, occur on the vegetation or bottom of the water. D/H host acquire infection by ingestion of metacercaria along with water or herbage. Eg. Fasciolidae, and Paramphistomatidae.

No sporocyst stage. Redia is produced directly by miracidium. The first intermediate host will be invariably snails and may also involve other intermediate hosts such as Ants, Dragonfly nymphal stages, crabs, crustaceans or fishes as second intermediate hosts.

MODULE-15: FAMILY - FASCIOLIDAE

Learning objectives

After reading this module the learner will understand the following

- Importance of liver fluke infection in cattle, sheep, goats and buffaloes, wild ruminants and human beings.
- Life cycle, pathogenesis, clinical signs, diagnosis, treatment and control of liver fluke infection in animals and man.
- Zoonotic significance

FASCIOLA SPP. INTRODUCTION

Family: Fasciolidae

- Genus: Fasciola
- Species: *Fasciola gigantica*, *Fasciola hepatica*
- Common name: Liver fluke.
- Distribution: World wide. Commonest liver fluke in India.
- Disease: It causes fluke cirrhosis or liver rot, fascioliasis or fasciolosis or clay pipe cirrhosis, pipe stem liver or gall stone formation.
- Host: Cattle buffalo, sheep, goat, and also in man.
- Location: Bile duct and Liver parenchyma.

Fasciola gigantica



FASCIOLA SPP.
INTERMEDIATE HOST

Aquatic snail

- [Lymnaea](#) acuminata
- L.rufescens
- L.auricularia
- L.natalensis



MORPHOLOGY OF FASCIOLA HEPATICA AND F.GIGANTICA

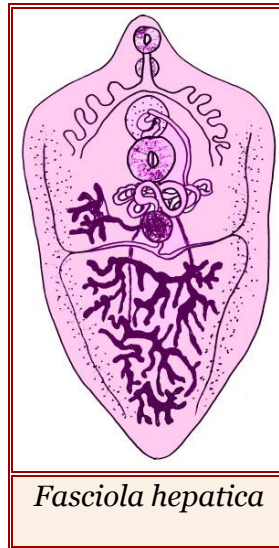
F.gigantica

- Leaf shaped large size. 25 - 75 mm in length, 12 mm in breadth and broader anteriorly than posteriorly.
- Anterior cone is smaller. They have indistinct shoulder (not so prominent).
- Cuticle have spine (spinose tegment). The body is more transparent.
- The oral and ventral suckers are present. The ventral sucker situated at the level of shoulder and is larger than oral sucker. The intestinal caeca is branched and diverticulated both externally and internally.
- Two follicular branched testes placed in the middle region. Single branched ovary with a coiled uterus lies anterior to the testes.
- Genital opening anterior to the ventral sucker. Numerous vitellaria placed laterally and 2 transverse vitelline ducts.

F.hepatica

- OS and VS are same size.
- Prominent shoulder.
- Testes placed at the posterior region.

- Comparatively smaller than *F. gigantica* in size
- Presence of anterior cone and distinct shoulder.
- Intestinal caeca branched and diverticulated only externally.



FASCIOLA SPP.
LIFE CYCLE

Fasciola-Egg



- *Egg is composite egg. Large in size. 156 - 197 X 90 - 104µm.*
- *They are oval in shape and yellowish tint with indistinct operculum.*
- *They have yolk cells and germ cells (hexagonal shape).*
- *The eggs are voided in the faeces of the host.*
- *It survives 2 - 3 months in humid faeces. But quickly destroyed by desiccation within few hrs / days.*
- *Miracidium develop only after the eggs have been laid*
 - *Development in invertebrate host*
 - *Development in vertebrate host*

FASCIOLA SPP.
DEVELOPMENT IN THE INVERTEBRATE HOST

- Eggs hatch in about 10 - 12 days at 26°C. (depends on temperature) release miracidium.
- Miracidium is broad anteriorly, with small papiliform protrusion, tegument is ciliated and has a pair of eyespot. (Within 3 hrs it should reach the snail.)
- Miracidium actively penetrates into aquatic snail (*L. acuminata*), casting off its ciliated covering and develops into sporocyst. Each sporocyst gives rise to 1 - 6 rediae by polyembryony.
- Redia has a circular thickening behind the level of pharynx and a pair of blunt processes at the beginning of the posterior quarter. They are 1 - 3 mm long.
- Usually rediae produce cercariae (daughter rediae may develop under unfavorable conditions).
- Cercaria is *Gymnocephalus*, there is no spine and eyespot but dark granular cystogenous glands in the lateral part of the body and long tail are present.
- Time taken for development of cercariae in the snail is 5-7 weeks under favorable conditions. Within few minutes to 2 hrs of the release of cercariae from snail, they settle on grass blades / other plants just below the water level (submerged vegetation) casting off their tails and secrete a covering by the cystogenous glands. From the spherical cyst is called as metacercaria.
- Small numbers may encyst on the surface of the water and sink into the bottom of the water. Metacercariae are infective to D/H. They are pearly / milky white in color with pinhead size.
- D/H acquire infection by ingestion of metacercariae along with herbage / drinking water.

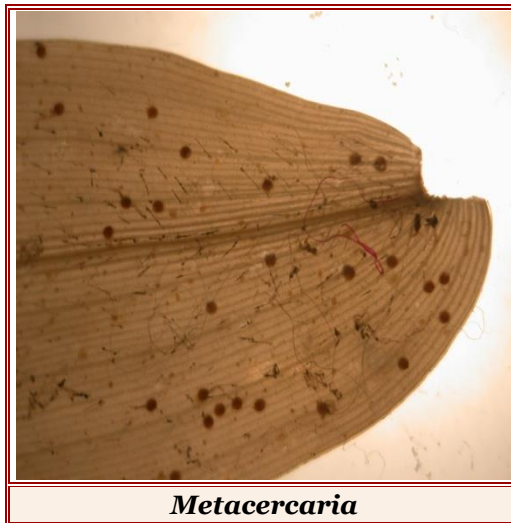
NOTE: Infection of snail with one miracidium can produce over 600 metacercaria. They survive on the herbage for 4 – 6 weeks at R.H 70%. It survive for 270 – 340 days on moist hay for 8 months and on silage for 35 – 57 days. Under normal condition 3 – 6 months but die quickly in hot and sunny days. This process of one embryo giving rise to numerous offsprings is unique to trematode and is known as paedogenesis.

FASCIOLA SPP.

DEVELOPMENT IN THE VERTEBRATE HOST

- Following ingestion of metacercaria excystation occurs in the duodenum. Within 24 hrs of infection, the majority of immature trematodes occur in abdominal and peritoneal cavity.
- By 4 - 6 days after infection, majority will penetrate the liver capsule and found migrating in the liver parenchyma. Young fluke may reach the liver through blood stream but it is unusual. Usual route is via peritoneal cavity and migration in the liver may occurs upto 5 - 6 weeks.
- From 7th week onwards they enter the bile duct and reach the sexual maturity, some flukes may be seen in gall bladder also. During 8th week of infection eggs are passed in the faeces. Prepatent period is normally 8 - 12 week.
- Occasionally in cattle, the immature flukes may be found in the other organs, such as in lungs and in the pregnant animals, the parasite may occur in the foetus.
- Minimum period of completion of one life cycle (egg to egg) is 17 - 18 weeks in *F.gigantica*, under favourable condition.

The longevity of *Fasciola* in untreated sheep may be years; in cattle, it is less than one year.



Metacercaria

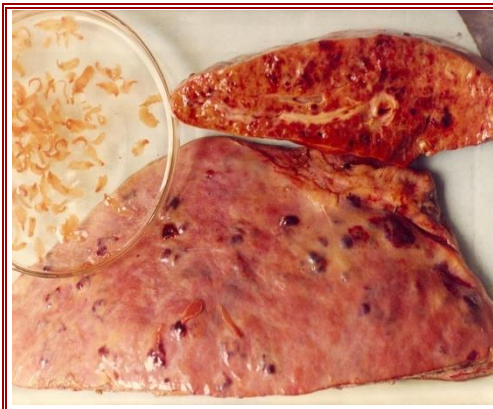
FASCIOLA SPP.

PATHOGENESIS

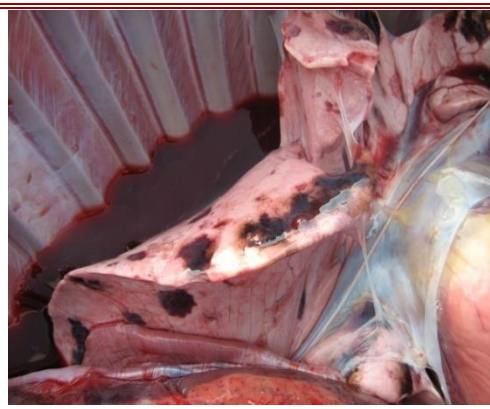
- The pathogenesis depends on the number of metacercaria ingested
- They are two forms of this disease.
 - [Acute fasciolosis](#)
 - [Chronic fasciolosis](#)

FASCIOLA SPP. ACUTE FASCIOLOSIS

- This is due to the Immature fluke
- It is a less common form compared to chronic form in sheep
- The condition usually results in Traumatic hepatitis which is mainly due to the migration of immature flukes.
- By about 6-8th week after infection, extensive destruction of liver parenchyma with hemorrhage
- If the number of parasites are more, it results in rupture of the liver capsule with hemorrhages in peritoneal cavity. In such animals death occurs in a few days time from the onset of clinical signs.
- Liver is enlarged, pale in color with hemorrhagic tracts on the liver surface.
- In less acute sub acute forms the liver is covered with migratory tracts with early fibrosis
- Sub acute form is common in animals of all ages
- A complication of acute fasciolosis is the occurrence of black's disease – A condition caused by the anaerobic bacteria *Clostridium novyi* which are mainly seen in the anaerobic lesions caused by the immature trematodes.



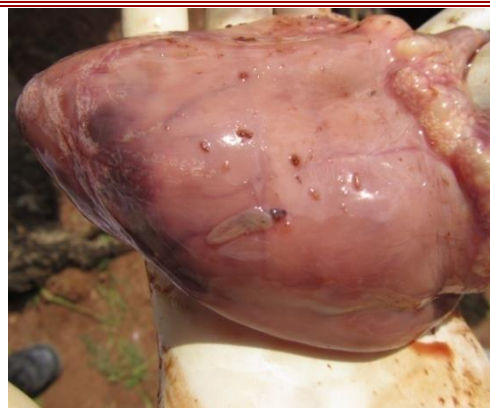
Acute Fasciolosis



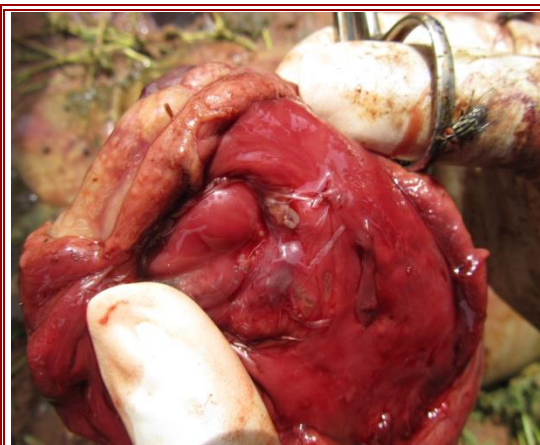
Fasciolosis in lung



Acute Fasciolosis in lung



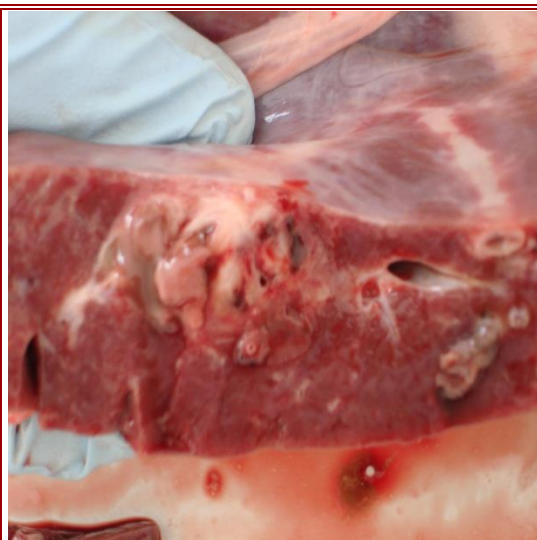
Fasciolosis in Heart



Fasciolosis in Heart

FASCIOLA SPP.
CHRONIC FASCIOLOSIS

- This is due to the activity of the Adult flukes.
- Most common form seen in sheep, cattle and other ruminants.
- The normal sequelae is hepatic fibrosis
- The chronic form is always associated with the presence of adult flukes in liver or bile duct.
- The condition may result in *hyperplastic cholangitis* in the liver and bile duct due to the presence of adult flukes.
- In such cases, the bile duct wall epithelium gets thickened giving the appearance of pipe stem liver and resulting in a condition called *clay pipe cirrhosis* or *pipe stem liver*



Chronic Fasciolosis



Sheep died due to fasciolosis

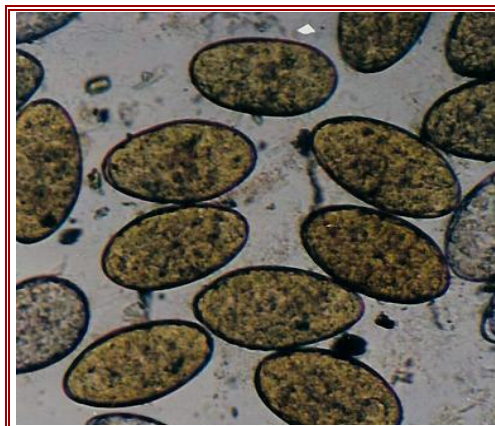
FASCIOLA SPP.
CLINICAL SIGNS

- The disease is common in animals of all the age groups.
- Affected animals shows anorexia, looks weak, emaciated.
- The most important sign is the appearance of odema especially in the sub mandibular region is referred commonly as bottle jaw (jowl odema) caused due to loss of proteins/ hypoalbuminaemia or loss of absorption of proteins by the host.
- In some cases especially in acute fasciolosis there will be diarrhea followed by constipation.
- Fasciolosis affected calf with sub mandibular swelling



FASCIOLA SPP.
DIAGNOSIS

- By clinical symptoms
- Weather based forecasting
- By examination of the dung sample for the presence of fasciola egg.
- Immunological test.



Fasciola Egg

FASCIOLA SPP. TREATMENT AND CONTROL

Treatment

- Oxyclozanide – 15 – 20 mg/kg b.w orally
- Rafoxanide – 10 mg / kg bw orally
- Nitroxylin – 10 mg/ kg B.W s/c
- Diamphenithide – ideal for fasciolosis
- Triclabendazole -10 mg/kg bwt

Control

- Eradication of Intermediate host– molluscicide – $\text{CuSO}_4 \cdot 7 \text{H}_2\text{O}$, Baylucide
- Treatment of infected animal -segregate
- Flukicides- twice a year trichlobendazole, oxyclozanide to healthy animal . Of all the drugs above, Triclabendazole is considered as the drug of choice for fasciolosis since it was found to be effective against both immature and adult stages of Fasciola species. In addition to treatment with flukicidal drugs, the flock should be moved to snail free pasture. The control include snail control in the endemic areas with efficient water management system. More over new water bodies / check dams, reservoir created should be made snail free. This may be achieved by application of herbal molluscidal drugs in the new areas.

FASCIOLOPSIS BUSKI INTRODUCTION

<i>Definite Host</i>	Man and pigs
<i>Location</i>	Small intestine of man & pig
<i>Common names</i>	Intestinal fluke of man.

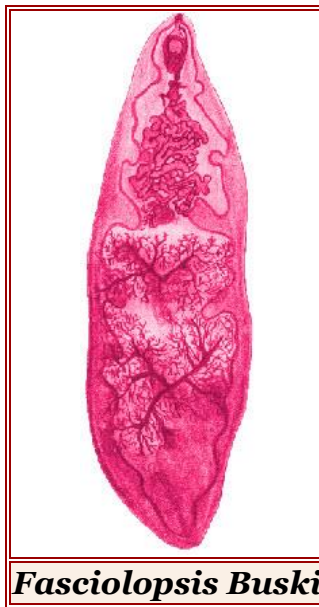
FASCIOLOPSIS BUSKI INTERMEDIATE HOST

- Planorbis sp, Segmentina sp,
- Water plants -- water calotrop, water chesnut.

MORPHOLOGY OF FASCIOLOPSIS BUSKI

Fasciolopsis buski

- Largest fluke 3 – 7.5 by 0.8 – 2 cm.
- Body is oval shaped and shoulders are absent.
- Cuticle is spiny.
- Ventral sucker is larger than the oral sucker.
- Intestinal caeca simple.
- Testes in the posterior third of the body are branched and lie in tandem position.
- Ovary is branched anterior to testes in the middle region, vitellaria occupy lateral fields.



Fasciolopsis Buski

**FASCIOLOPSIS BUSKI
LIFECYCLE**

- Eggs are similar to *Fasciola gigantica* like thin shelled, oval, operculate, brown colored.
- This cercariae encysts on the tubers or the nuts of the plants which are eaten raw by the humans.
- These possibly also other plants may carry the infection to pigs.

**FASCIOLOPSIS BUSKI
PATHOGENESIS**

- The parasite is chiefly of importance as a cause of disease in man.
- It attaches itself to the intestinal mucosa causing a local inflammation or deep ulcerative lesions in heavy infections.
- It produces abdominal pain, diarrhoea, oedema and ascites.

FASCIOLOPSIS BUSKI

CLINICAL SIGNS, DIAGNOSIS, TREATMENT AND CONTROL

Clinical signs

- common in children.
- Human – Abdominal pain, diarrhea, edema, ascites.

Diagnosis

Based on clinical signs, eggs in faeces.

Treatment

- Niclosamide – 60 mg /kg BW orally.
- Praziquantel – 20 mg /Kg BW orally.

Control

- Intermediate host control.
- Proper disposal of faeces of human and pig .
- Proper cooking.

DICROCOELIUM DENDRITICUM

INTRODUCTION

Definite host	Cattle, sheep and goat .
Location	Bile duct .
Common name	Lancet flukes/blade/needle fluke.

DICROCOELIUM DENDRITICUM

INTERMEDIATE HOST

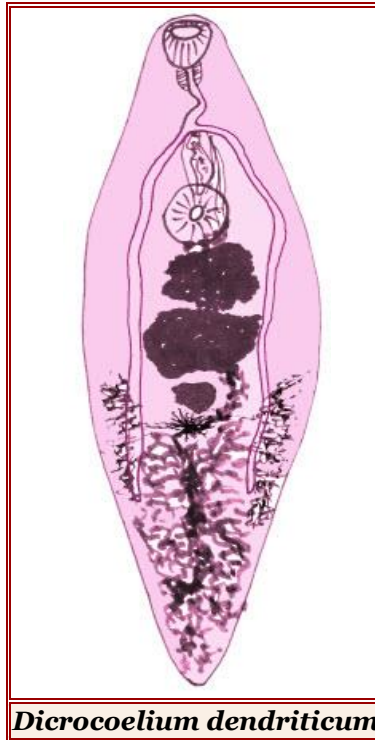
- *Land snail* - Macrochlamys cassida
- *Ant* – Formica fusca

MORPHOLOGY OF DICROCOELIUM DENDRITICUM

Dicrocoelium dendriticum

- Small fluke, 1.6 – 1 by 0.15 – 0.25 cm.
- Body is elongate, lanceolate shape with smooth cuticle.
- Oral sucker is smaller than the ventral sucker.
- Intestinal caeca are simple, not reaching the posterior end of the body.

- Testes are slightly lobed and are tandem in position just below the ventral sucker
- Ovary is posterior to testes.
- Uterus is quite distinct (filled with brown eggs) and coiled occupying the posterior part of the body.
- Vitelline glands are restricted to mid lateral region.



DICROCOELIUM DENDRITICUM

LIFECYCLE



- Eggs are small dark brown, operculate.
- Usually with one side flattened.
- It contains miracidium (embryonated).
- Redia stage absent.

DICROCOELIUM DENDRITICUM
PATHOGENESIS

- Extensive cirrhosis and scarring of liver surface.
- Bile ducts are distended in fibrosis in portal triads, marked proliferation of bile duct epithelium.
- In severe cases, edema, anemia and emaciation common in hill region – dicrocoeliosis.

DICROCOELIUM DENDRITICUM
TREATMENT

- Hetolin- 19-22 mg/kg BW orally.
- Albendazole - 7.5 mg/kg BW orally .
- Fenbendazole - Large doses 150 mg/kg BW orally.

DICROCOELIUM DENDRITICUM
CONTROL

- Snail control.
- Ant control .
- Treatment .

MODULE-16: FAMILY-PROSTHOGONIMIDAE

Learning objectives

After reading this module the learner will understand the following

- About the morphology, life cycle, pathogenesis, diagnosis, treatment and control of oviduct fluke.
- Importance of this fluke in poultry production.

PROSTHOGONIMUS OVATUS **INTRODUCTION**

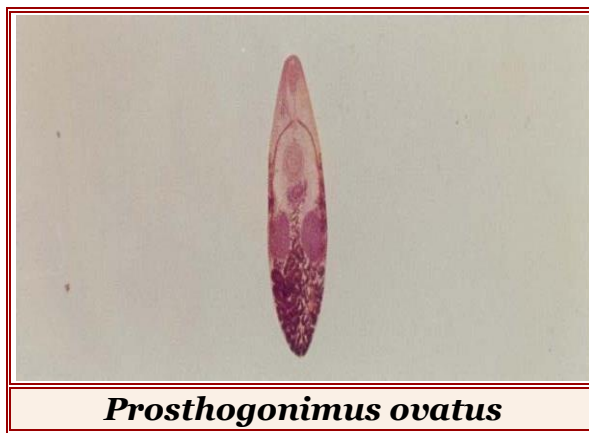
<i>Definitive host</i>	Domestic fowl, ducks, geese and wild birds
<i>Location</i>	Oviduct / Bursa of fabricius
<i>Common name</i>	Oviduct fluke

PROSTHOGONIMUS OVATUS **INTERMEDIATE HOST**

- *Gyraulus* (Water Snails)
- Naiads of dragon fly

PROSTHOGONIMUS OVATUS **MORPHOLOGY**

- Small flukes, 0.3 – 0.6 by 0.1 – 0.2 cm.
- Body is broad posteriorly.
- Ventral sucker present in the middle.
- Ovary is deeply lobed and is nearer the ventral sucker.
- Testes are elongated, horizontally placed in the middle of the body behind the ovary.
- Genital pore opens near the oral sucker.



Prosthogonimus ovatus

PROSTHOGONIMUS OVATUS
LIFE CYCLE

- Similar to *D. dendriticum*.
- First I.H: *Gyraulus* snails
- Second I.H: Naiads of Dragon fly.

PROSTHOGONIMUS OVATUS
PATHOGENESIS

- Irritation in oviduct of acute inflammation, malfunctions of eggs.
- Shell less eggs formation - naked yolk material and albumin may enter peritoneal cavity leads to peritonitis, sudden death.

PROSTHOGONIMUS OVATUS
CLINICAL SIGNS, DIAGNOSIS TREATMENT AND CONTROL

Clinical signs

- Cheesy material discharge from cloaca.
- The bird will be listless, weak, abdomen pendulous.

Diagnosis

Examination of eggs in droppings.

Treatment

- Albendazole.
- praziquantel .

Control

- Snail control .
- Isolation.

MODULE-17: FAMILY-OPISTHORCHIIDAE

Learning objectives

After reading this module, the learner will understand the following

- Importance of morphology, life cycle, pathogenesis, clinical signs, diagnosis, treatment and control of Ophisthorchiid flukes.

OPISTHORCHIS TENUICOILLIS **INTRODUCTION**

<i>Host</i>	Dog, cat, fox, pig and man
<i>Location</i>	Bile duct, rarely in intestine and pancreatic ducts
<i>Common name</i>	Liver fluke of dogs and cats

OPISTHORCHIS TENUICOILLIS **INTERMEDIATE HOST**

- Normally, two intermediate hosts present in *Opisthorchis tenuicollis* . These are
 - *Bithynia* sp.
 - Cyprinid fish

OPISTHORCHIS TENUICOILLIS **MORPHOLOGY**

- Small fluke (translucent body), reddish when fresh.
- Elongate body and narrow anteriorly.
- Suckers weakly developed.
- Pharynx, Oesophagus present and intestinal caeca extend almost to the posterior end.
- Testes spherical / slightly lobed, diagonal, in the posterior half of the body.
- Ovary small, slightly lobed and is anterior to the testes.
- Uterus situated between ventral sucker and the ovary.
- Vitellaria present as series of transversely arranged follicle and occupy the middle third of the lateral fields.
- The excretory bladder is typically ‘ Y ‘ – shaped.

OPISTHORCHIS TENUICOILLIS **PATHOGENESIS**

- Marked proliferation of bile duct epithelium. Severe inflammation of bile duct.
- Tumor formation in liver - carcinoma of liver

OPISTHORCHIS TENUICOILLIS
CLINICAL SIGNS, DIAGNOSIS, TREATMENT AND CONTROL

- **Clinical signs**
 - Abdominal pain, diarrhoea.
- **Diagnosis**
 - Eggs in faeces.
- **Treatment**
 - Praziquantel.
 - Albendazole.
- **Control**
 - Feeding only cooked fish to dog and cats

MODULE-18: FAMILY: PARAGONIMIDAE

Learning objectives

After reading this module, the learner will understand the following

- Importance of morphology, life cycle, pathogenesis, clinical signs, diagnosis, treatment and control of lung fluke.
- Significance of verminous pneumonia in grazing livestock.

PARAGONIMUS WESTERMANII INTRODUCTION

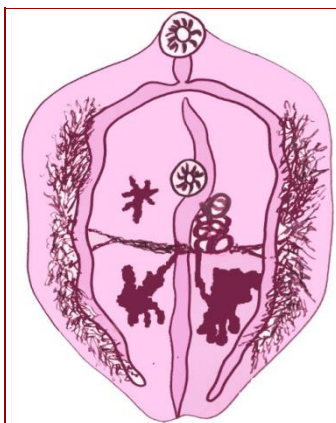
<i>Common name</i>	Lung fluke
<i>Location</i>	Lungs rarely in brain spinal cord
<i>Host</i>	Cats, dogs, pigs, wild carnivores and human

PARAGONIMUS WESTERMANII INTERMEDIATE HOSTS

- Two intermediate hosts are
 - Amphibious snails – *Melania* spp.
 - Crustaceans – crabs or cray fish .

PARAGONIMUS WESTERMANII MORPHOLOGY

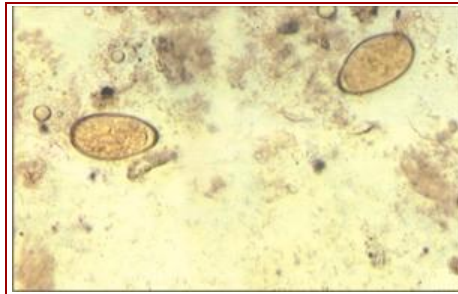
Paragonimus westermanii



- Adults live in pairs in cysts in lungs.
- Thick set, ovoid flukes, reddish brown in colour.
- Cuticle is spiny.
- Ventral sucker present in the middle of the body.
- Genital pore immediately behind ventral sucker.
- Testes in posterior half of the body.
- Ovary anterior to testes.

PARAGONIMUS WESTERMANII
LIFE CYCLE

- Intermediate host– snails aquatic – *Melania* sp *Semisulcospira* sp.
- Eggs are laid with in the cyst in which the worms live, escape into the bronchi, the eggs may pass up from the lungs with mucosa and may be found in sputum which has a characteristic rusty colour.
- Animals swallow the mucous and hence eggs are found in the faeces.
- By 2-7 weeks the miracidium hatches, penetrates the snail where it develops into the sporocyst redia and cercaria in 3 months.
- Cercaria comes out the snail which is eaten by crabs/ cray fish (II Intermediate host) to become metacercaria.
- The metacercaria are found mostly in the heart, liver and muscle of II Intermediate host.



***Paragonimus westermanii* Egg.**

PARAGONIMUS WESTERMANII
DEVELOPMENT IN DEFINITIVE HOST

- After ingestion young flukes penetrate the intestinal wall, enters the peritoneal cavity to diaphragm and then the lungs after 1 –3 weeks of infection.
- The parasite penetrate the pulmonary forming a cystic cavity where the parasite mature into adult.
- The cyst usually contains two parasites surrounded by a purulent fluid mixed with blood and eggs of the parasite.
- In cats, 1000-200 eggs are laid/ day flukes.

PARAGONIMUS WESTERMANII
PATHOGENESIS

- Migrating immature flukes causes eosinophilic peritonitis, pleuritis, myositis and multifocal pleural hemorrhages.
- In infected animals chronic bronchitis with hyperplasia of bronchiole epithelium and pneumonia associated with degenerating eggs in the tissue.

PARAGONIMUS WESTERMANII
CLINICAL SIGNS, DIAGNOSIS AND TREATMENT

Clinical signs

- Affected animals are lethargic with intermittent cough.
- Dyspnoea associated with Pneumothorax.
- Immature flukes may be carried away to the other organs and dislodging in the brain causing CNS disturbances.

Diagnosis

- Eggs demonstration in sputum / faeces.
- x-ray for identification of cyst in lungs.
- Very commonly observed in the right caudal lobe of lung.
- Serological test such as CIE (counter immuno electrophoresis)
- ELISA, IFAT (indirect fluorescent antibody technique)

Treatment

- Albendazole – 50 – 100 mg/kg day for 2-3 weeks
- Bithionol- 100 mg /kg / day for 7 days.
- Niclofolon- 2 mg/kg day for 3-5 days

MODULE-19: FAMILY-PARAMPHISTOMATIDAE

Learning objectives

After reading this module the learner will understand the following

- Importance of morphology, life cycle, pathogenesis, clinical signs, diagnosis, treatment and control of amphistomosis.
- Pathogenesis of immature amphistomosis.
- Bottle jaw in sheep and goats.
- Importance of snails in transmission and its control.

PARAMPHISTOMUM CERVI **INTRODUCTION**

<i>Host</i>	Cattle, buffalo, sheep and goat
<i>Location</i>	Adults in Rumen and reticulum, Immature flukes in Duodenum
<i>Intermediate host</i>	<i>Indoplanorbis, Gyraulus, Lymnaea and Bulinus</i>

PARAMPHISTOMUM CERVI **MORPHOLOGY**

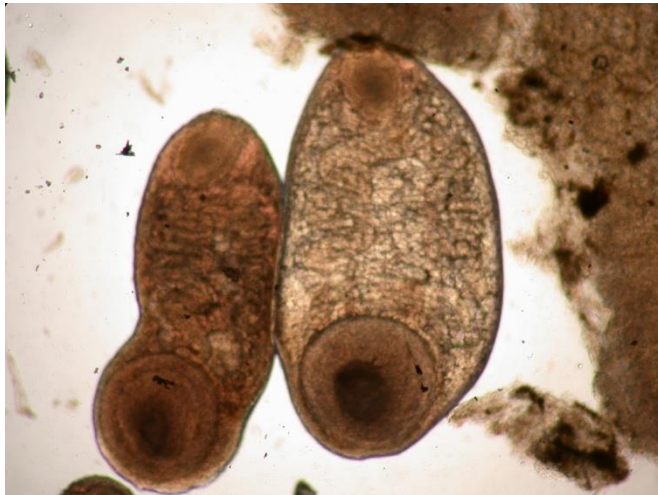
- Body is thick, conical, elongate with blunt ends.
- Ventral sucker is situated at the posterior end, large and strongly developed.
- Intestinal caeca coiled (3-4 coils) and terminate at about the level of middle of ventral sucker.
- Testes lobed and tandem.
- Genital sinus enclosing genital opening. The cuticle is spineless. The vitelline glands are lateral and are strongly developed.

PARAMPHISTOMUM CERVI **LIFE CYCLE**

- Eggs are composite, distinct operculum
- Metacercaria is dark in colour and remains viable for 3 months
- Infection of definitive host is by the ingestion of metacercaria along with the grass herbage.
- The ingested metacercaria gets excysted in the small intestine and gets attached to the mucosa of duodenum as immature flukes.
- After 6-8 weeks the immature flukes start migrating through various parts of intestine.
- I part of development Intestine – migrate -- Rumen/Reticulum

PARAMPHISTOMUM CERVI **PATHOGENESIS**

- The immature flukes are highly responsible for causing the pathogenesis by their presence in the small intestine.
- The immature flukes attach very strongly and get embedded in the mucosa of the intestine and they are commonly called as plug feeder.
- These immature flukes eat away pieces of mucosa through the sucker and pinch them off resulting in necrosis and severe hemorrhage in heavy infections the deeply placed immature flukes may reach the muscular coat of the intestine causing hemorrhagic duodenitis
- The adult flukes are normally non-pathogenic and are seen attached to the rumen feeding only on the seminal papillae resulting in loss of ruminal papillae.
- Due to the presence of the immature flukes in the intestine, extensive catarrhal hemorrhage occurs in the duodenum and jejunum with degeneration of intestinal glands. There will be anaemia, hypoproteinaemia, oedema and emaciation.



Pathogenesis caused by Immature amphistomes



Post mortem findings of immature amphistomosis



Immature Amphistome in Abomasum



Immature Amphistome nodules in Duodenum

PARAMPHISTOMUM CERVI
CLINICAL SIGNS

- Infected animals are weak emaciated showing proper foetid foul smelling diarrhea along with immature flukes present in faeces.
- Death may occur within 15-20 days after the onset of clinical symptom.
- In case of immature amphistomosis the rate may go up to 80-90 %
- Affected animals feel thirsty and drink water frequently, the animal also shows in bottle jaw (intermandibular swelling) which is characteristic of flukes infection.
- The immature amphistomes is called in Hindi as “gillor”, “pitto”
- PM lesions mucous membrane looks pale, subcutaneous fat is replaced by a gelatinous material, hydrothorax, hydro pericardium, and hemorrhagic deodentitis.
- The immature flukes are seen attached to duodenal mucosa in clusters which appear pink in colour.

PARAMPHISTOMUM CERVI
DIAGNOSIS

- By clinical signs.
- By faecal examination
- Presence of snail Intermediate host
- Immature flukes in diarrhoeal faeces
- Postmortem examination

PARAMPHISTOMUM CERVI
TREATMENT AND CONTROL

Treatment

- Oxyclozanide – 15 – 20 mg/kg BW orally 3-5 days.
- Niclofolon 6 – mg/kg BW.
- Bithionol 40 mg/kg.
- Niclosamide 90 mg/kg BW. As amphistomosis usually occurs along with GI nematode infection, a combination of Oxyclozanide with tetramisole is given to sheep and goats.

Control

- Control of snail Intermediate host.
- Proper drainage of water from lakes and ponds.

COTYLOPHORON COTYLOPHORUM
INTRODUCTION

<i>Host</i>	Cattle, buffalo, sheep and goat
<i>Location</i>	Rumen
<i>Intermediate host</i>	<i>Indoplanorbis exustus</i>

COTYLOPHORON COTYLOPHORUM
MORPHOLOGY

- Small to medium sized.
- Body is pear shaped.
- In fresh specimens, the dorsal surface is convex with slightly concave ventral surface.
- Ventral sucker sub-terminal.
- Intestinal caeca are simple.
- Testes are tandem in position.
- Genital sucker is distinct and surrounds the genital pore.
- Numerous vitelline glands fill the lateral aspect of the fluke.

GASTROTHYLAX CRUMENIFER

INTRODUCTION

<i>Host</i>	Cattle, buffalo, sheep and goats
<i>Location</i>	Rumen
<i>Intermediate host</i>	<i>Gyraulus convexiusculus</i>

GASTROTHYLAX CRUMENIFER

MORPHOLOGY

- Body is elongate.
- In fresh specimen, the flukes appear fleshy and red in colour.
- Ventral pouch appears dark red in colour.
- Ventral sucker is terminal.
- Intestinal caeca are straight and terminate in front of the ovary.
- Ventral pouch is large extending up to the ventral sucker and opens behind the oral sucker.
- Testes are placed side by side; the ovary is placed behind testes.
- At about middle of the body, the uterus crosses from one side to the other (right to left).

FISCHOEDERIUS ELONGATUS

INTRODUCTION

<i>Host</i>	Cattle, buffalo, sheep and goat
<i>Location</i>	Rumen
<i>Intermediate host</i>	<i>Lymnaea luteola</i>

FISCHOEDERIUS ELONGATUS

MORPHOLOGY

- Ventral pouch is comparatively smaller.
- Ventral sucker is terminal.
- Uterus lies in the middle through out its course and does not cross from one side to the other.
- Testes lie dorso-ventrally.
- Intestinal caeca extend up to the posterior end of the body.

FISCHOEDERIUS COBBOLDI

INTRODUCTION

<i>Host</i>	Cattle, buffalo, sheep and goat
<i>Location</i>	Rumen
<i>Intermediate host</i>	<i>Lymnaea luteola</i>

FISCHOEDERIUS COBBOLDI

MORPHOLOGY

- Ventral pouch is comparatively smaller.
- Ventral sucker is terminal.
- Uterus lies in the middle through out its course and does not cross from one side to the other.
- Testes lie dorso-ventrally.
- Intestinal caeca extend up to the posterior end of the body.

EXPLANATUM EXPLANATUM (*GIGANTOCOTYLE EXPLANATUM*)

INTRODUCTION

<i>Host</i>	Cattle, buffalo, sheep and goat
<i>Location</i>	Bile duct and liver
<i>Intermediate host</i>	<i>Indoplanorbis exustus</i>

EXPLANATUM EXPLANATUM (*GIGANTOCOTYLE EXPLANATUM*)

MORPHOLOGY

- Fleishy body with very large sized ventral sucker at posterior end.
- Testes are lobed and placed diagonally.
- Ovary is behind the testes just above the ventral sucker.
- Intestinal caeca are unbranched.

GASTRODISCUS SECUNDUS
INTRODUCTION

<i>Host</i>	Equines and elephants
<i>Location</i>	Caecum and colon
<i>Intermediate host</i>	<i>Indoplanorbis exustus</i>

GASTRODISCUS SECUNDUS
MORPHOLOGY

- Body is divided into two portions - an anterior globoid or cone shaped and a posterior papillated discoid or saucer shaped.
- Oral / oesophageal pouches present.
- Ventral sucker is small and placed sub terminally.
- Testes are branched and diagonally placed.

GASTRODISCOIDES HOMINIS
INTRODUCTION

<i>Host</i>	Man and pig (natural host)
<i>Location</i>	Caecum, colon
<i>Intermediate host</i>	Planorbid snail

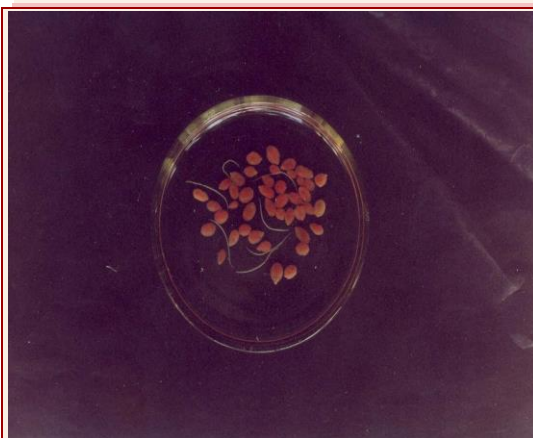
GASTRODISCOIDES HOMINIS
MORPHOLOGY

- Pyriform in outline and bright reddish in colour.
- Body is divided into two parts, anterior being smaller than posterior and without papillae.
- Oral pouches present.
- Ventral sucker has prominent deep cleft.

PSEUDODISCUS COLLINSI
INTRODUCTION

<i>Host</i>	Equines
<i>Location</i>	Caecum, colon
<i>Intermediate host</i>	<i>Indoplanorbis exustus</i>

PSEUDODISCUS COLLINSI
MORPHOLOGY



Pseudodiscus Sp.

- *Body tapering anteriorly and rounded posteriorly.*
- *Oral pouches present.*
- *Intestinal caeca wavy.*
- *Testes deeply lobed placed side by side.*
- *Ovary single, posterior to testes but lateral in position*

MODULE-20: FAMILY-SCHISTOSOMATIDAE

Learning objectives

After reading this module the learner will understand the following

- About snoring disease of cattle.
- Intestinal schistosomiasis of cattle and pigs.
- Zoonotic significance of cercarial dermatitis.
- Importance of snails in transmission of blood flukes.

SCHISTOSOMES GENERAL CHARACTERS

Amongst trematodes, the blood flukes are different in the following manner :

- Male and female are separate with marked sexual dimorphism. Male is short and thick; female is long and slender.
- Appear like a nematode.
- Pharynx absent.
- Intestinal caeca united posteriorly to form a common caeca.
- Male lodges the female in an incurved groove, gynaecophoral canal.
- Life cycle involves three larval stages viz. miracidium, sporocyst and furcocercous cercaria. Redial stage is absent.

SCHISTOSOMES MORPHOLOGY OF MALE FLUKES

- Short and stumpy
- Cuticle has tuberculation in the case of *Schistosoma nasale* whereas *S. spindale*, the cuticle is smooth.
- On the lateral sides of the body (ventral aspect), a curved structure forming a groove known as Gynaecophoric canal is seen. During copulation, the female flukes are lodged in this groove.

SCHISTOSOMES MORPHOLOGY OF FEMALE FLUKES

- Long and slender
- Single ovary placed in front of the common caecum
- After copulation, the female flukes leave the male and move away to lay the eggs
- Eggs are laid in the blood vessels of the intestinal wall/ nasal cavity or urinary bladder depending upon the species
- With the help of the terminal spines, the eggs are able to pass through tissue and reach the site of living

SCHISTOSOMA NASALE
INTRODUCTION

<i>Common name</i>	Nasal blood fluke
<i>Host</i>	Cattle, buffalo, rare in sheep, goats and horses
<i>Location</i>	Nasal veins
<i>Intermediate host</i>	<u>Indoplanorbis exustus</u>
<i>Disease</i>	Nasal schistosomosis or snoring disease or nasal granuloma in cattle

SCHISTOSOMA NASALE
MORPHOLOGY

- Suckers are weak and close together.
- Two or four testes in the anterior region behind the ventral suckers.
- The integument is coarsely tuberculated in male.
- Ovary in front of common caeca.
- Uterus with 1 or 2 eggs.

SCHISTOSOMA NASALE
PATHOGENESIS

- In the nasal granuloma a pseudotubercle formation occurs due to the constant irritation caused by the spines and metabolites released from the miracidium within the egg. Cut section of egg with miracidium surrounded by numerous phagocytic cells in a radial manner resulting in actinobody or pseudo tubercle formation.



*Nasal Granuloma lesion in a Cow infected with
*S.nasale**

SCHISTOSOMA NASALE
CLINICAL SIGNS

- Hemorrhagic enteritis, anemia, and emaciation
- Copious mucopurulent discharge, snoring, and dyspnea; milder cases frequently are asymptomatic.

SCHISTOSOMA NASALE
DIAGNOSIS

- Faecal examination and rectal scraping to find the eggs
- Nasal mucus
- In chronic cases, necropsy finding adult flukes in blood vessel

SCHISTOSOMA NASALE
TREATMENT AND CONTROL

- Praziquantel -25 mg/kg body weight
- Triclabendazole- 20 mg/kg body weight
- To provide clean drinking water
- Control the intermediate host (Snail)

SCHISTOSOMA SPINDALE
INTRODUCTION

<i>Host</i>	Cattle, buffalo, sheep and goats
<i>Location</i>	Portal and mesenteric veins
<i>Intermediate host</i>	<i>Indoplanorbis exustus</i>
<i>Disease</i>	Visceral schistosomosis

SCHISTOSOMA SPINDALE
MORPHOLOGY

- Similar to *S. nasale*.
- The integument is smooth in male.
- Three to seven testes in the anterior region behind the ventral suckers.

SCHISTOSOMA INDICUM
INTRODUCTION

<i>Host</i>	Cattle, buffalo, sheep, goats, equines and camels
<i>Location</i>	Portal and mesenteric veins
<i>Intermediate host</i>	<i>Indoplanorbis exustus</i>
<i>Disease</i>	Visceral schistosomosis

SCHISTOSOMA INDICUM
MORPHOLOGY

- Cuticle is finely tuberculated posterior to the ventral sucker.
- Five to twelve testes in the anterior region behind the ventral sucker.

SCHISTOSOMA INCOGNITUM
INTRODUCTION

<i>Host</i>	Pigs and dogs
<i>Location</i>	Portal vein
<i>Intermediate host</i>	<i>Lymnaea luteola</i>
<i>Disease</i>	Visceral schistosomosis

SCHISTOSOMA INCOGNITUM
MORPHOLOGY

- Very small fluke.
- Moderately tuberculated cuticle.
- Two to seven testes in the anterior region behind the ventral suckers.

VISCERAL SCHISTOSOMIASIS
PATHOGENESIS

- Two clinical syndromes are seen in animals infected with schistosomes, an acute intestinal syndrome and a chronic hepatic syndrome
- After heavy infection there is inflammatory and granulomatous responses to the deposition of eggs in the mesenteric veins and their subsequent infiltration in the intestinal mucosa
- During the acute phase of the disease there are marked haemorrhagic lesions in the mucosa of the intestine, but as the disease progresses the wall of the intestine appears greyish, thickened and edematous due to confluence of the egg granulomata and the associated inflammatory changes.
- The hepatic syndrome is an immunological disease resulting from the host's cell mediated immune response to schistome eggs in the liver
- Eggs swept back in the portal circulation lodge in perisinusoidal interlobular portal venules. Immunologically specific host reaction to the eggs leads to extensive damage to the portal vascular system.

VISCERAL SCHISTOSOMIASIS
CLINICAL SIGNS

- Acute heavy infections are manifested by profuse diarrhoea or dysentery, dehydration and anorexia.
- Anaemia and hypoalbuminaemia are present, sometimes with edema.
- There is marked decrease in production or loss of weight.
- Chronically infected animals are emaciated. There is eosinophilia, anemia, hypoalbuminaemia.

VISCERAL SCHISTOSOMIASIS

DIAGNOSIS

- Based mainly on the clinico-pathological picture of diarrhoea, wasting and anemia, coupled with history of access to natural water sources.
- The relatively persistent diarrhoea often blood stained and containing mucous, may helped to differentiate this syndrome from fasciolosis.
- The demonstration of characteristic egg in the faeces or in squash preparation of blood and mucous from the faeces.

VISCERAL SCHISTOSOMIASIS

TREATMENT AND CONTROL

Treatment

- Praziquantel- 60mg/kg

Control

- Measures to reduce population of the snail intermediate host
- Water troughs cleaned out regularly

ORNITHOBILHARZIA TURKESTANICUM

INTRODUCTION

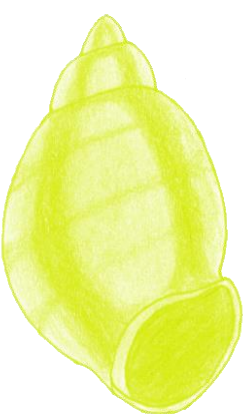
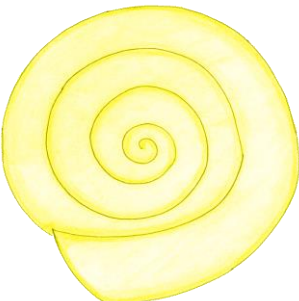
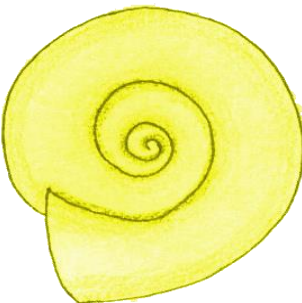
<i>Host</i>	Cattle, buffalo, sheep and goats
<i>Location</i>	Portal and mesenteric veins
<i>Intermediate host</i>	<i>Lymnaea acuminata</i>

ORNITHOBILHARZIA TURKESTANICUM

MORPHOLOGY

- Resembles *Schistosoma* in all characters.
- Possess large number of testes (above 60).

ORNITHOBILHARZIA TURKESTANICUM
INTERMEDIATE HOST (SNAILS)

			
<i>Bithynia sp</i>	<i>Brotia libertina</i>	<i>Bullinus contortus</i>	<i>Oncomelania hypensis</i>
			
<i>Lymnea stagnalis</i>	<i>Parafossarulus striatulus</i>	<i>Physopsis africana</i>	
			
<i>Gyraulus prashadi</i>	<i>Planorbis boissyi</i>	<i>Segmentina schmackeri</i>	

MODULE-21: CLASS - CESTODA

Learning objectives

After reading this module the learner will understand the following

- Various developmental stages of tape worms.
- Mode of transmission of developmental stages of tape worms in animals and man.

CESTODE **INTRODUCTION**

- 'Cestus' – Tape like structure.
- Tapeworms are hermaphrodite.
- They are endoparasites, elongate and flat in nature. Few millimeter to several centimeter in length.
- Body cavity, digestive, respiratory and circulatory systems are absent.
- Saprozoic nutrition.
- Tape worms have 3 important parts,
 - Head or scolex
 - Neck
 - Body or strobila

CESTODE **MORPHOLOGY**

Scolex

- Globular in shape, it has hold fast or adhesive organs is called as suckers in Eucestoda, where as in Cotyloda is known as 'Bothria'. Suckers are cup like structure, while bothria is longitudinal groove like structure.
- Scolex have a platform like structure is called as rostellum. It bears hooks or not if it has hooks known as 'armed rostellum'. Sometimes suckers also have hooks, they are known as armed suckers.

Neck

- Immediately following the head an unsegmented portion is called as neck.

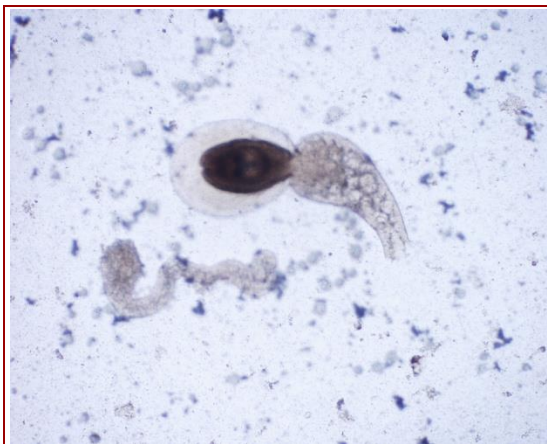
Body

- A portion behind the neck region is called as body or strobila.
- Strobila consists of numbers of segments or proglottids, they are separated by transverse constriction. The genital organs are ill developed in first few segments they are known as immature segments. Mature segments have fully and functional genital organs. Each segment contains one or two sets of genital organ.

- *Eg. Raillietina* spp - Single set of genital organ. *Cotugnia digonopora* - Double set of genital organ.
- Generally the genital pore opens at the lateral aspect in Eucestoda but in cotyloda situated on ventral aspect. Posteriorly the segment packed with eggs is known as gravid segment. Only the cotylodan tapeworms have separate uterine pore so they lay the eggs singly. Where as in Eucestoda the gravid segments are detached and passed in the faeces of the host and the eggs are released due to disintegration of the gravid segment this process is known as 'apolysis'.
- In some species after voiding the gravid segments move on the surface of faeces or bedding material or on the floor is called as "Anapolysis".

Nervous System

- Central part of nervous system situated in the scolex from which nerve cords run posteriorly.



Cysticercoid with tail



Subcutaneous cysticercus cysts in sheep

CESTODE LIFE CYCLE

- Eggs are embryonated or unembryonated when laid. If the eggs are embryonated it contains oncosphere or hexacanth embryo. The oncosphere is bilaterally symmetrical and has 3 pairs of hooks.
- Eggs having 4 layers.
 - Outer capsule.
 - Outer envelope.
 - Inner envelope.
 - Oncosphere membrane.
- In Eucestoda the eggs hatch out within the I/H (only after ingestion by I/H) and develops into larval stage. Where as in cotyloda eggs hatch out in water and release the larval stage is known as "Coracidium".
- The larval stage of tapeworm is called as metacestode or bladder worm stage.

TYPES OF LARVAL TAPEWORMS or BLADDER WORMS

Cysticercus

- Single invaginated scolex with cavity filled with fluid.
- Eg. *Cysticercus cellulosae* - larval stage of *Taenia solium*.
- [Cysticercus cellulosae cyst infected meat](#)



- *Cysticercus bovis* - larval stage of *T. saginata*

Cysticercoid

- Single non-invaginated scolex with drawn into a small vesicle. Practically no cavity.
- Eg. Larval stage of ruminant and poultry tapeworm.

Strobilocercus

- A single scolex which is not invaginated when fully developed and attached with the bladder by a long neck.
- Eg. Larval stage of *T. taeniaeformis*.

Hydatid cyst

- Large fluid containing bladder it can produce daughter cyst is called as “Brood capsule” in which the scolices are develop. The mature brood capsule detached and float free in the fluid is called as “hydatid sand”.
- Eg. Larval stage of *Echinococcus granulosus*.

Proceroid

- Is the larval stage of Cotylodan tapeworms which is occurs in the first I/H.
- Proceroid is solid body and possess hooks on the posterior region.

Coenurus

- A large fluid containing bladder in which number of invaginated scolex attached to the wall.
- Eg. Larval stage of *T. multiceps*.
- [Coenurus cyst from brain of a Sheep](#)



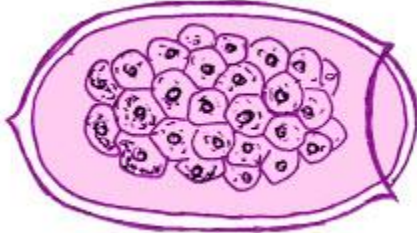
Plerocercoid

- Is also larval stage of Cylindroid tapeworms which occurs in the 2nd I/H.
- It is elongated, solid and has a scolex like in adults.

Tetrathyridium

- It is a larval stage of *Mesocostoides lineatus*. The body is elongate, solid and has a deeply invaginated Scolex.

EUCESTODA Vs COTYLODA

Eucestoda	Cotyloda
Hold fast organ is known as suckers . Suckers may be armed or unarmed and 4 in numbers.	Hold fast organ is known as Bothria , two longitudinal weak muscular groove.
Segmentation is present.	Segmentation is absent.
Genital pore is present on the lateral aspect.	Genital pore is on ventral aspect.
Eggs are embryonated when laid and hatch out within I/H. eg. <i>Taenid</i> egg 	Eggs are unembryonated, operculated and hatch out in the water. eg. <i>Diphyllbothrium latum</i> egg 
It requires only one I/H	It requires two I/H.
Apolysis is present.	Apolysis is absent.

MODULE-22: TAPEWORMS OF RUMINANTS

Learning objectives

After reading this module the learner will understand the following

- Importance of grass/soil mites in the transmission of ruminant cestodes.
- Moniezia infection in sheep, goats, cattle and buffaloes.
- Control of grass mites.

TAPEWORMS OF RUMINANTS INTRODUCTION

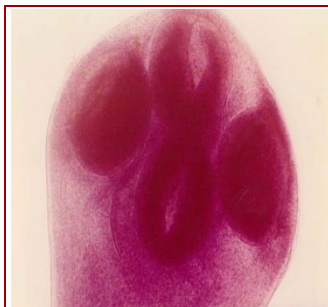
The tapeworms of ruminants are as below:

- [*Moniezia expansa*](#)
- [*Moniezia benedini*](#)
- [*Avitellina lahorea*](#)
- [*Stilesia globipunctata*](#)

MONIEZIA Spp. ([*Moniezia expansa*](#) and [*Moniezia benedini*](#))

Moniezia expansa

- Adults are up to 6 m in length. Scolex is wide and have prominent suckers.
- Segments are wider than long.
- It has double sets of reproductive organs, so it is known as “double pored ruminant tapeworm”.
- Vitelline glands and ovary form a ring on either side.
- Genital pore is marginal. Testes are numerous and distributed in the central field of the segment.
- At the posterior border of each segment a row of rosette like interproglottidal glands are present.
- The function of these glands are not known.
- The glands are extended along the full width of segments.



Moniezia Scolex

Eggs

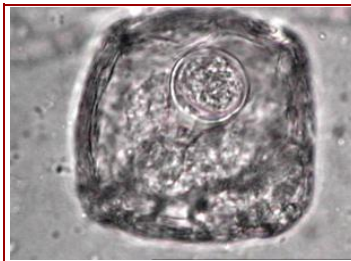
- Triangular in shape contains oncosphere or hexacanth embryo and has pyriform apparatus.

Morphology of *M. benedeni*

- Morphology of *M. benedeni* is similar to *M. expansa*, except that, interproglottidal glands are concentrate at the centre of the segment only.
- It commonly occurs in cattle than other animals.

Eggs

- Square shaped.



Moniezia Egg

Life cycle

- Oribatid mite act as a I/H common species – *Scheloribates leavigatus* and *Scheloribates madrasensis* (Anantaraman).
- Eggs are passed in the faeces of the host and are ingested by grass mite in which they develop into cysticeroid in four months.
- Final host acquire infection by ingestion of infected I/H.
- Prepatent period. 37 – 40 days.

Epidemiology

- Monieziosis commonly occur during November and December and infection is common in young lambs and calves.

Pathogenesis

- In adult animals, the infection is mild pathogenic but highly pathogenic in young animals causes malnutrition leading to reduced wool and meat production.
- The monieziosis predisposes the lambs to bacterial infection like Enterotoxaemia caused by *Clostridium Perfringens* - type D.

Diagnosis

- Faecal examination for presence of eggs.
- Gross examination of faeces for the presence of gravid segments which looks like “cooked rice grain”.

Treatment

- Albendazole - 10mg/Kg b wt. (oral)
- Fenbendazole - 5mg/Kg b wt. (oral).
- Praziquantel - 15mg/Kg b wt. (oral).
- Niclosamide - 75 – 150 mg/Kg b wt. (oral).

Control

- Elimination of grass mite on pasture by ploughing and cultivation.
- Deworming the animal in early summer.

AVITELLINA LAHOREA

Host

- Commonly occur in sheep and goats.

Location

- Small intestine.

Morphology

- 3 m in length. Segments are short and cylindrical and at the posterior end segmentation is not clear. Each proglottid contains single set of reproductive organ. The genital pore opens irregularly alternate.
- In the gravid segment eggs are passed into the par uterine organ (thick walled sac like organ).
- The par uterine organ resembles snail shell.
- Pyriform apparatus is absent.

Life cycle

- Psocid (Blook lice) act as a I/H.

Pathogenesis and treatment

- Similar to Moniezia.

STILESIA GLOBIPUNCTATA

Host

- Commonly occurs in sheep, cattle and goat.



Stilesia-Scolex

Location: Small intestine.

- Adults are 60 cm in length. Each segment contain single set of genital organ. Genital pore opens irregularly alternate. Uterusdump bell shaped.
- Eggs are passed into two par-uterine organ and eggs have no pyriform apparatus.



Stilesia Sp.

I/H: Oribatid mite.

Pathogenesis

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

Treatment

- Praziquantel – 150 mg / Kg b. wt.

MODULE-23: TAPEWORMS OF EQUINE

Learning objectives

After reading this module the learner will understand the following

- Importance of tapeworms in horses.
- Also distinguish fluke like appearance of the tape worms in equine.

ANOPLOCEPHALA PERFOLIATA

Family	Anoplocephalidae
Species	Anoplocephala perfoliata
Common name	Dwarf tapeworm of horse, 'lappet tapeworm'.
Location	Small and large intestine

Morphology

- Adults are upto 5 – 8 cm in length.
- Scolex has lappet behind the each sucker.
- Segments are wider than long.
- Segments have single set of reproductive organ.
- Genital pore is marginal.



Life cycle

- Oribatid mite (grass/soil mite) act as a I/H - common species *Scheloribates laevigatus*.
- [Eggs](#) are passed in the faeces of the host and are ingested by oribatid mite.
- Eggs are hatch out in the I/H and develops into cysticeroid in about 4 months.
- Final host get the infection by ingestion of infected mite along with herbage.
- Prepatent period is 4 to 6 weeks.

Pathogenesis

- Common tapeworm of horse. Light infection cause no clinical signs.
- But large numbers causes ill health, unthriftiness and even death may occur. Usually the worms are localized in the ileo-caecal orifice, it causes partial occlusion of ileo-caecal orifice.
- At the site of scolex attachment a small dark depressed ulcerative lesion may be seen.
- Perforation of intestine also recorded.

Treatment

- Mebendazole: 15 to 20 mg / Kg b wt. (Oral)
- Niclosamide: 88 mg / Kg b wt. (Oral)
- Bithionol: 7 mg / Kg b wt. (Oral)

OTHER SPECIES (*Anoplocephala magna* and *paranoplocephala mamillana*)

- **Species:** *Anoplocephala magna*
 - **Host:** Equines
 - **Location:** Small intestine
 - **Intermediate stage:** Cysticeroid
 - **Intermediate host:** Oribatid mites
 - **Scolex**
 - Lappets are absent.
- **Species:** *Paranoplocephala mamillana*
 - **Host:** Equines
 - **Location:** Small intestine
 - **Intermediate stage:** Cysticeroid
 - **Intermediate host:** Oribatid mites
 - **Scolex**
 - Scolex is narrow.
 - Opening of suckers are slit like.
 - Lappets are invariably absent.

MODULE-24: TAPEWORMS OF POULTRY

Learning objectives

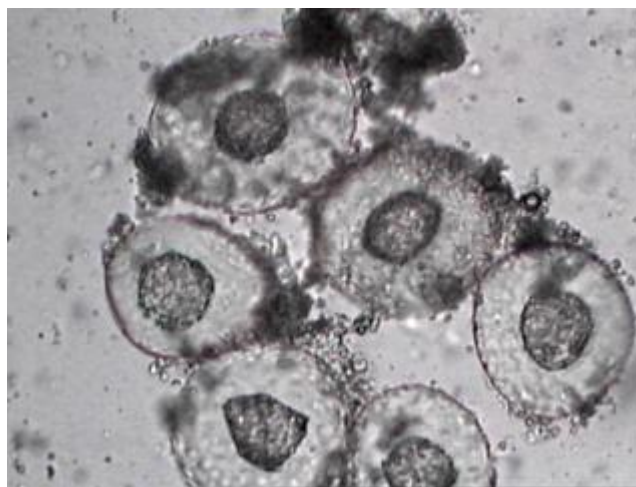
After reading this module the learner will understand the following

- Role of various arthropods present in the poultry farms in the transmission of tape worm infection.
- Significance of tape worms in birds reared under in cages and in backyard system.
- Need for deworming in commercial poultry.

**TAPEWORMS OF POULTRY
INTRODUCTION**

There are 10 species of tapeworms affecting poultry.

- [*Davainea proglottina*](#)
- [*Raillietina tetragona*](#)
- [*R. echinobothridia*](#)
- [*R. cesticillus*](#)
- [*Cotugnia digonopora*](#)
- *Choanotaenia infundibulum*
- [*Hymenolepis carioca*](#)
- *H. contaniana*
- [*H. lanceolata*](#)
- *Fimbriaria fasciolaris*



Poultry Tapeworm Eggs

DAVAINEA PROGLOTTINA

Common name	Dwarf tapeworm of poultry
Host	Chicken and pigeon
Location	Duodenum
I/H	Slug (snail without shell). <i>Limax</i> and <i>Arion</i> species

Morphology

- The worms are microscopic in nature, about 0.5 to 3mm in length. They have only 4 to 9 segments.
- Rostellum is retractable and armed with hammer shaped hooks.
- Suckers also armed with hooks.
- Each segment has single set of genital organ.
- Genital pore opens regularly alternate.
- In the gravid segment, the uterus is replaced by egg capsule.
- Each egg capsule contains single egg.

RAILLIETINA TETRAGONA

Common name	Largest poultry tapeworm
Host	Chicken, pigeon and guinea fowl
Location	Posterior half of the small intestine
I/H	Ants. (<i>Pheidole</i> spp. and <i>Tetramorium</i> spp.)

Morphology

- Adults are up to 25 cm in length. Scolex is smaller than the *R.echinobothridia*. Rostellum is armed with 1 to 2 rows of hooks. Suckers are oval in shape and armed with hooks.
- Each segment has single set of reproductive organs genital pore opens unilaterally.
- Each egg capsule contains 6 to 12 eggs.

R. ECHINOBOTHRIDIA

Host	Chicken and turkey
Location	Small intestine
I/H	Ants. (<i>Tetramorium</i> spp.)
Causes	Nodular taeniosis

- Scolex is large in size when compared to *R. tetragona*.
- Rostellum heavily armed with two rows of hooks. Suckers are circular in shape.
- Each segment has single set of genital organ. Genital pore irregularly alternate
- Gravid segments are separated by windows in progottids.
- Each egg capsule contains 6 to 12 eggs.

R. CESTICILLUS

Host	Chicken
Location	Small intestine
I/H	Dung beetles

Morphology

- Usually 4 cm in length. Rarely it attains 15 cm. Scolex is very wide.
- Large rostellum armed with 400 to 500 small hooks.
- Suckers are indistinct and are not armed.
- Each segment contains single set of genital organs. Genital pore unilateral.
- Each egg capsule has single egg.

COTUGNIA DIGONOPORA

Common name	Double pored poultry tapeworm
Host	Chicken
Location	Small intestine
I/H	Ants. (<i>Pheidole</i> spp., <i>Monomorium floricola</i>)

Morphology

- Rostellum is armed with two rows of hooks.
- It has cup like muscular suckers.
- Each segment contains two sets of genital organs.
- Eggs capsule contain single egg.

A.SPHENOIDES

Host	Chicken
Location	Small intestine
I/H	Earthworm

Morphology

- Small worm. Elongate triangular shape. 4 mm long and 1 mm wide.
- Rostellum bears 12-14 hooks.
- There are about 20 proglottids.
- Testes are 12 or more in number and lie near the posterior border of the segment.
- Uterus is sac-like and slightly lobed.

HYMENOLEPIS CARIOCA

Host	Chicken
Location	Small intestine
I/H	Dung beetles, flour beetles and <i>Stomoxys calcitrans</i>

Morphology

- Rostellum armed with spanner shaped hooks.
- Segments are very small. Each contains single set of reproductive organ. Genital pore is unilateral.
- Each segments contains three testes. One testes on poral side while the other two on aporal side.
- Eggs are covered with 3 layers and is rugby ball shaped.

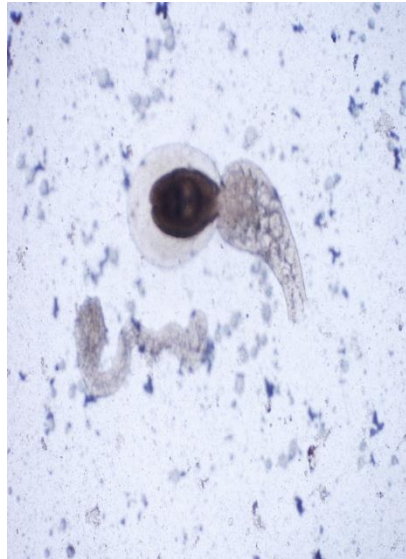
H. LANCEOLATA

Host	Ducks
Location	Small intestine
I/H	Aquatic crustaceans

Morphology

- Similar to *H.carioca*.

LIFE CYCLE OF POULTRY TAPEWORMS



- The gravid segments are passed in the droppings of birds and are crawling on the surface of droppings, during this process, eggs are released. Egg contains hexacanth embryo.
- The eggs are ingested by intermediate hosts where they hatch and develops into cysticercoid in about 3 weeks time. Infection of poultry by ingestion of infected I/H.

TAPEWORMS OF POULTRY
PREPATENT PERIOD

<i>D. proglottina</i>	14 days
<i>R. tetragona</i>	21 days
<i>R. echinobothridia</i>	20 days
<i>R. cesticillus</i>	13 days
<i>C. infundibulum</i>	15 days
<i>C. digonopora</i>	20 days
<i>Hymenolepis</i> spp.	20 days

TAPEWORMS OF POULTRY
EPIDEMIOLOGY AND PATHOGENESIS

Epidemiology

- Tapeworm infections are common in free range birds than the intensive system of rearing. Since free range birds have more access to eat I/H than birds reared under confined environment.
- Sometimes heavy tapeworm infection occurs in intensive system of management due to this system provide conducive environment for breeding of I/H like flies, beetles and ants.

Pathogenesis

- *D. proglottina* is most pathogenic tapeworm. The worms are penetrate deeply between the villi causes necrosis and haemorrhagic enteritis. Sometimes death may occur due to intestinal obstruction.
- Chronic infection characterized by reduced growth rate, emaciation and weakness.
- *R. echinobothridia* is most pathogenic causes nodules formation in the intestine is called as “**Nodular taeniasis**” in poultry. Hyperplastic enteritis may also occur.
- All other tapeworms are less pathogenic but in heavy infection results in reduced egg production and general weakness.

TAPEWORMS OF POULTRY
DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- Macroscopic or gross examination of dropping for the presence of gravid segment.
- PM examination of representative bird from affected flock.

Treatment

- Niclosamide - 75 mg/Kg b wt.
- Fenbendazole - 5 mg/Kg b wt.
- Aricoline hydrobromide (Arica nut).
- Praziquantel - 15 mg/Kg b wt.
- Closantel - 7.5 mg/Kg b wt.

Control

- Elimination of I/H is very important by
 - Hygienic maintenance of poultry shed.
 - Applying chemical compounds like BHC and HCH.
 - Insect growth regulators like larvadex may be used against *Musca* spp.
 - Laris (Cyromazine) - Chitin inhibitor may be used against I/H develop.
- Periodical deworming of birds.

MODULE-25: TAPEWORMS OF DOG

Learning objectives

After reading this module the learner will understand the following

- Zoonotic Importance of bladder worms/larval tapeworms affecting human beings.
- Importance of personal hygiene in humans
- Preventing contamination of grazing areas with dog faeces.
- Control of neurocysticercosis and other zoonotic diseases in humans.
- Importance of deworming pets and stray dogs.

TAPEWORMS OF DOG INTRODUCTION

The tapeworms of dog are as follows:

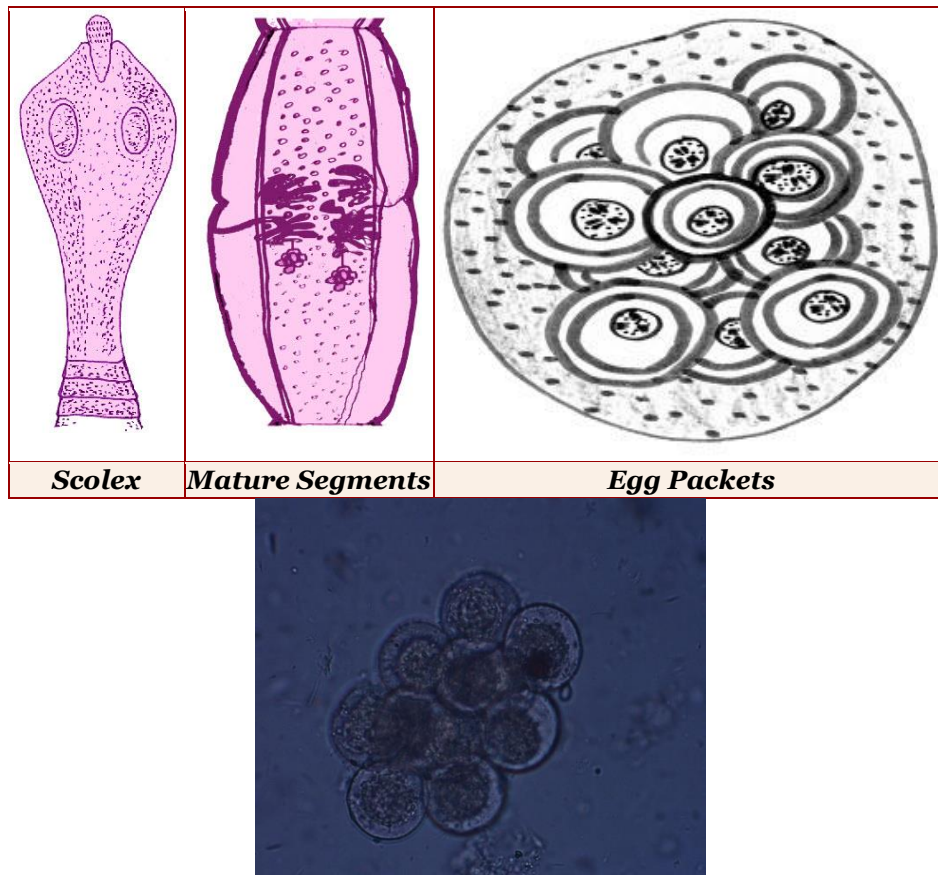
- *Dipylidium caninum*
- *Taenia hydatigena*
- *Taenia multiceps*
- *Echinococcus granulosus*
- *Mesocestoides lineatus*
- *Diphyllbothrium latum*

DIPYLIDIUM CANINUM

Common name	Double pored dog tapeworm. It also occurs in man and cat.
Location	Small intestine
I/H	Dog flea: (<i>Ctenocephalides canis</i>), Dog lice: <i>Trichodectes canis</i> and <i>Heterodoxus spiniger</i>
Metacestode stage	Cysticercoid

Morphology

- Retractable rostellum armed with three or four rows of rose thorn shaped hooks.
- Each segment contains two sets of genital organ.
- Vitelline glands and ovary form a mass on either side resembling a bunch of grapes.
- In the gravid segment uterus are replaced by egg capsule or egg packets.
- Egg packets contain 30 eggs per packet. Gravid segments are elongate and oval in shape resembling cucumber seed shape.



DIPYLIDIUM CANINUM
LIFE CYCLE

- The gravid segments are passed in the faeces or spontaneously leaving the host and crawling on the body surface of the host or on the floor, during this process eggs are released.
- [Eggs](#) are ingested by larval stage of fleas, but cysticeroid development occurs in the adult flea.
- D/H acquires infection by ingestion of infected adult flea.
- Man acquires infection by accidental ingestion of flea while playing with dog and cat.

Egg ----> Larval flea ----> Adult flea ----> D/H

DIPYLIDIUM CANINUM
PATHOGENESIS

- It depends upon the age of host. Adult worms are not pathogenic to dog but heavy infection causes abdominal pain, unthriftiness, diarrhoea or constipation and rarely intestinal obstruction may occur.
- When gravid segment leave the intestines they cause severe irritation around the perianal area and due to constant irritation the dog will drag its anus over the ground. This condition is known as “anal pruritus”.

DIPYLIDIUM CANINUM
DIAGNOSIS AND TREATMENT

Diagnosis

- Demonstration of egg packets in faeces.
- Macroscopic examination of faeces for gravid segment.

Treatment

- Arecoline hydrobromide - 1 to 2 mg/Kg b wt.
- Praziquantel – 5 mg/Kg b wt.
- Niclosamide – 100 to 150 mg/Kg b wt.
- Mebendazole – 100 to 200 mg/Kg b wt. Twice daily for 5 days.
- Bithionol – 200 mg/Kg b wt.

TAENIA HYDATIGENA

Host	Dog and wild carnivores
Location	Small intestine
I/H	Domestic and wild ruminants (sheep, cattle, sometimes pigs also act as I/H)
Metacestode stage	<i>Cysticercus tenuicollis</i>

Morphology

- Rostellum armed with 2 rows of pen knife shaped hooks. [1st row – 26 and 2nd row – 46].
- Adults are upto 75 – 500 cm in length
- Each segment contains a single set of genital organ. Genital pore is irregularly alternate
- Ovary is situated at the posterior border of segment
- Uterus has median stem
- Testes are numerous
- Gravid segments are longer than wide. In gravid segment, uterus has 6 to 10 lateral branches. (Important character for identification of Taenid tapeworms).

Egg

- Contain hexacanth embryo.
- They have 4 layers, made up of blocks giving radiate appearance or ‘cart wheel’ appearance.

TAENIA HYDATIGENA

LIFE CYCLE

- Eggs hatch in the small intestine and liberate the hexacanth embryo.
- Hexacanth embryo penetrate the intestine wall and reach liver via circulation.
- In the liver, hexacanth embryo break out of portal vessel and migrate in the parenchyma for about one month. The developing cysticercus migrate to the peritoneal cavity and attain maturity in about 53 days.
- The matured cysticercus attach to the omentum and mesenteric serosal surface of the intestinal wall. The metacestode/larval tapeworm/bladder worm stage is known as "*Cysticercus tenuicollis*".

Cysticercus tenuicollis

- 6 cm in length, consists of single invaginated scolex, attached to a fluid containing bladder by a long neck. Final host acquires infection by ingestion of *Cysticercus tenuicollis* infected meat or offals.
- Prepatent period is 51 days.

TAENIA HYDATIGENA

PATHOGENESIS AND DIAGNOSIS

Pathogenesis

- The prevalence of infection is high in sheep but level of infection is low.
- The migration of *Cysticercus* in the liver cause haemorrhagic and fibrotic tract.
- Heavy infection in lambs with *C.tenuicollis* causes the condition traumatic hepatitis which is called as "hepatitis cysticercosa". This must be differentiated from acute fasciolosis. *Cysticercus* in the peritoneal cavity do not cause any harmful effect.

Diagnosis

- PM examination of I/H.

TAENIA MULTICEPS

Host	Dog and wild carnivores
Location	Small intestine
I/H	Sheep and Goats
Metacestode stage	<i>Coenurus cerebralis</i>

Morphology

- Adults are upto 40 to 100 cm in length. In the gravid segment, uterus has 14 to 20 lateral branches.
- Metacestode stage occurs in the brain and spinal cord, sometimes in goats, it may occur in s/c tissue.
- The species that occur in goats is considered as a separate spp. *Taenia gaigeri*.

TAENIA MULTICEPS

LIFE CYCLE

- Eggs are ingested by I/H which hatch in the small intestine of the intermediate host. The oncosphere penetrates the intestine wall and reach the brain and spinal cord via circulation.
- Developing larval stages migrate in the brain and spinal cord leaving a tortuous yellowish grey to reddish streaks.
- Larval stage matures in 8 months. *Coenurus* is large, fluid containing bladder in which number of invaginated protoscolices are attached to the wall.
- Final host acquires infection by ingestion of infected meat.

TAENIA MULTICEPS

PATHOGENESIS

- Adult worms are not pathogenic in D/H (Dog) but the larval stages are highly pathogenic in I/H (Sheep). In lambs the migrating larval stage causes, “acute meningo encephalitis” which is chronic in nature and associated with one *Coenurus* or two *Coenuri*.
- The developing *Coenurus* produces high degree of brain tissue damage causing neurological condition which is referred to as Gid or Staggers or Sturdy. The neurological symptom depends upon the location of cyst in the CNS.
- If the cyst is located in one of the cerebral hemisphere the animal will hold its head to one side and turn in a circle towards the affected side. The eye on the opposite side may be blind.
- If the cyst is situated in the anterior part of the brain, the animal will hold its head against the chest and may walk with high steps or may walk in a straight line until it meets an obstacle and remain motionless.
- If the cyst is present in the ventricle, the movement will be backward.
- If the cyst is present in the cerebellum, the animal will be hyperaesthetic and may have incoordination, jerky or staggering gait or may remain motionless.
- If the cyst is present in the surface of the brain, the skull may be subjected to “pressure atrophy” and so perforation of the skull may occur.

TAENIA MULTICEPS
DIAGNOSIS AND TREATMENT

Diagnosis

- By PM examination.
- X – ray.

Treatment

- Uneconomical.

ECHINOCOCUS GRANULOSUS

Common name	Smallest dog tapeworm (Important Zoonotic tapeworm).
Host	Dog
Location	Small intestine
I/H	All mammals including man
Larval stage	Hydatid cyst

Morphology

- Worms are 3 to 7 mm in length and have 3 to 4 segments.
- The rostellum has two rows of hooks.
- The penultimate segment is the mature segment and the last one is gravid.
- Each segment has a single set of reproductive organ. Genital pore irregularly alternate.
- Ovary is kidney shaped. In the gravid segment number of lateral branches of uterus may occur.
- Eggs are taenid type.

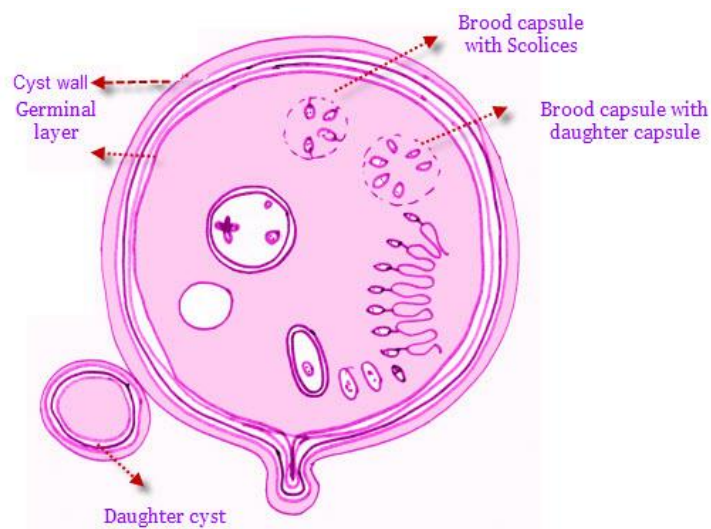


ECHINOCOCUS GRANULOSUS
LIFE CYCLE

- Eggs are ingested by I/H (Sheep, cattle, goat, pig, horse and man) and these hatch in the small intestine and upon hatching the oncosphere penetrates the intestine wall and reach the liver via blood and lymphatic circulation.
- In liver and lungs, oncosphere develops into a cyst - [hydatid cyst](#).
- Cyst may also occur in other organs.
- Cyst develops slowly and takes several months to attain maturity.

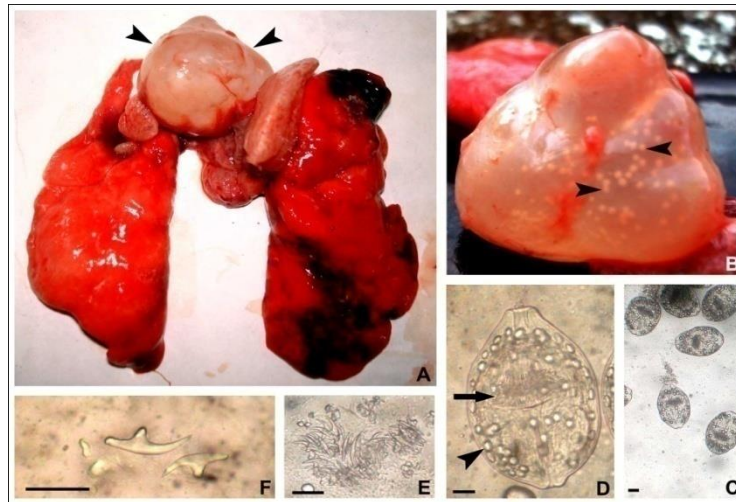
HYDATID CYST

- 5 to 10 cm in diameter, unilobular and consists of two layers
 - Outer laminated membrane and
 - Inner germinal membrane.
- From the germinal membrane, brood capsules develop in about 5 months after infection.
- Each capsule contains a number of protoscolices.
- Sometimes, the brood capsule detaches and floats free in the hydatid fluid which is called as “hydatid sand”.
- If the cyst is ruptured, the brood capsule and protoscolices produce “external daughter cysts”.
- All cysts do not produce brood capsules and protoscolices. Cysts which do not have brood capsules and protoscolices are known as “sterile cysts”.
- In sheep, 51% of cysts are sterile.
- D/H acquires infection by ingestion of protoscolices along with infected meat.
- In dog, the protoscolices penetrate between the villi and reach maturity in about 4 to 7 days.
- Man can acquire infection by ingestion of eggs along with contaminated food or entry of protoscolices through cut wounds during slaughter.



ECHINOCOCCUS GRANULOSUS PATHOGENESIS

- In dogs, adult tapeworms are not pathogenic whereas in humans and other domestic animals, the pathogenesis varies and may be severe, owing to larval tapeworms i.e., hydatid cyst.
- Clinical signs depend upon the location of the cyst. The function of affected organ is impaired. If the cyst is ruptured, it results in anaphylactic shock.



ECHINOCOCUS GRANULOSUS
DIAGNOSIS

Diagnosis in dog

- Examination of faeces for presence of eggs but *E. granulosus* eggs cannot be differentiated from other taenid eggs such as *T. multiceps* and *T. hydatigena*.
- So confirmation is only based upon the demonstration of adult worm.
- For collection of adult worms from the infected animals, the dogs must be treated with Arecoline hydrobromide, 1 to 2 mg/Kg b wt. The treated dogs will purge out the intestinal contents and expel all the adult worms.
- Examine the mucous portion of the faecal sample to obtain the adult worms.

Diagnosis in man (Hydatidosis)

- CASONI's skin test.
- Counter immuno electrophoresis.
- ELISA.
- AGPT.

ECHINOCOCUS GRANULOSUS
TREATMENT AND CONTROL

Treatment for tapeworm infection in Dogs

- Similar to *Dipylidium caninum* .

Treatment for Hydatid Cyst in Humans

- Surgical removal.
- Aspiration of cyst fluid, Marsupilization and Sterilization of cyst.
- Inject 2.5 to 10% formalin which will destroy the germinal membrane and protoscolices. However, this procedure is dangerous, because spillage of hydatid fluid may cause anaphylatic shock and dissemination of protoscolices to various parts.
- Albendazole – 10 mg/kg bwt. Two divided dose.
- Mebendazole – 400 to 600 mg/kg b. wt. thrice for 21 to 30 days.

Control of tapeworm infection in dogs

- Control of lice and fleas using deltamethrin 1% [**Butox**] or by using flea collar.
- Hygienic maintenance of kennel.
- Avoid providing raw meat or offals to dogs.
- Periodical deworming.

Control of hydatid cyst in man

- Personal hygiene.
- Public education.

MODULE-26: TAPEWORMS OF MAN

Learning objectives

After reading this module the learner will understand the following

- Importance of tapeworms in human health .
- Mode of transmission of human tapeworms through vegetables, fruits etc.
- Decontamination of water and food sources.

TAPEWORMS OF MAN INTRODUCTION

- The following are the tapeworms of man
 - *Taenia saginata*.
 - *Taenia solium*.
 - *Diphyllobothrium latum*.

TAENIA SAGINATA

Host	Man
Location	Small intestine
I/H	Cattle
Metacestode	<i>Cysticercus bovis</i>
Morphology	• Adults are 4 to 8 m and may rarely attain 25 m in length. Scolex has 4 suckers. • Rostellum and hooks are absent. • In the gravid segment, uterus has 14 to 32 lateral branches.

TAENIA SAGINATA LIFE CYCLE

- Gravid segments are passed in the stools and these gravid segments migrate and contaminate the soil or grazing area of livestock.
- During migration eggs are released which are ingested by the I/H (cattle) and in the small intestine of cattle they hatch where upon the oncosphere penetrates the intestinal wall and reach different parts of body via general circulation and develops into *Cysticercus* in heart, masseter muscles, tongue, diaphragm etc. However, high density occurs in the heart and masseter muscle.
- The cysticercus attains maturity in 10 weeks time and remain viable for 9 months.
- The larval stage is *Cysticercus bovis* and the beef infected with *Cysticercus bovis* is called as “**measley beef**”. Humans acquire infection by ingestion of uncooked or improperly cooked infected beef.

TAENIA SAGINATA
PATHOGENESIS AND DIAGNOSIS

Pathogenesis

- In man: Abdominal pain, diarrhoea and constipation may occur.
- In cattle: Usually asymptomatic but heavy infection causes myositis, myocarditis and stiffness of muscle may be seen.

Diagnosis

- Perianal swab for the presence of egg in man.
- Cattle: PM examination.

TAENIA SAGINATA
TREATMENT AND CONTROL

Treatment

- Niclosamide – 2 g.
- Paromomycin – 5 mg/Kg b wt.
- Quinacrine 7 – 10 mg/Kg b wt. (It should not be given for *T. solium* infection because it induces vomiting).
- Praziquantel – 10 mg/Kg b wt.

Control

- Personal hygiene.
- Proper disposal of night soil.
- Public education.
- Avoid eating of raw or under cooked beef.

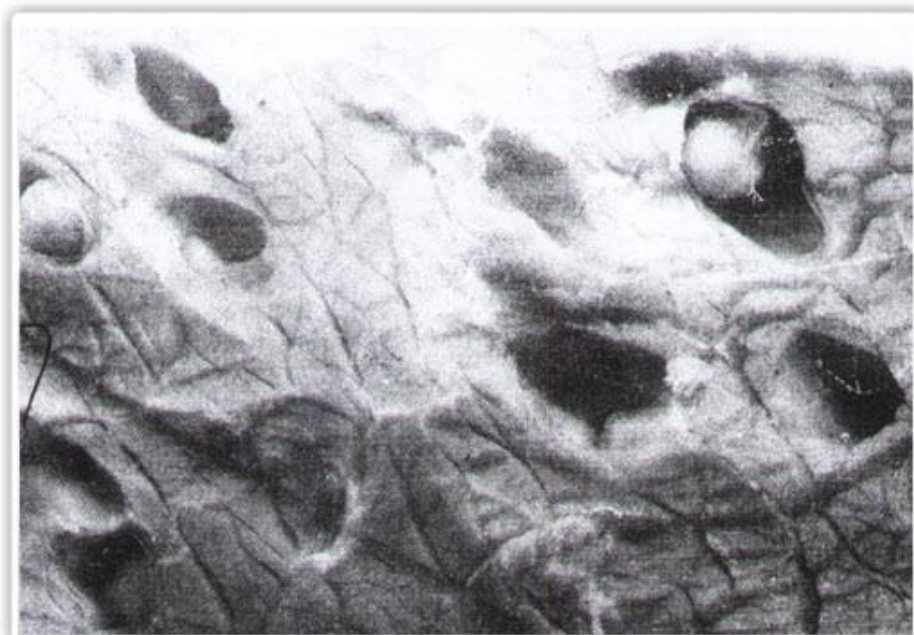
TAENIA SOLIUM

Host	Man
Location	Small intestine
I/H	Pig (Man also acts as I/H)
Metacestode/Bladder worm/Larval Tapeworms	<i>Cysticercus cellulosae</i>. The condition caused by this larval stage is Porcine cysticercosis or Measely pork. 
Morphology	• Adults are 3 to 5 m in length. • Scolex has four suckers. • Rostellum armed with penknife shaped hooks (2 rows). • In the gravid segment, uterus has 7 to 16 lateral branches. • Gravid segments do not leave the host spontaneously. • Segments are voided in the faeces in a chain. • All the other characters are similar to <i>T.hydatigena</i>. • In pigs, cysticerci occur in tongue, neck, oesophagus intercostal and cardiac muscle. • The larval stage is known as <i>Cysticercus cellulosae</i>. It reaches maturity in about 9 to 10 weeks time. <i>Cysticercus cellulosae</i> infected pork is called as “measley pork”. Sometimes man also acts as I/H in which man acquire infection either by ingestion of eggs along with contaminated food or by autoinfection. • Autoinfection occurs only in persons already infected with adult worms. Due to reverse peristalsis, the eggs are released from the gravid segment while they are passing through and hatch in the intestine itself. Then the oncospheres penetrate the intestinal wall and reach various parts of the body where they develop into cysticerci. • Cysticercus in man mainly occurs in brain, s/c tissue and ocular tissue (eye). •

TAENIA SOLIUM
PATHOGENESIS AND DIAGNOSIS

Pathogenesis

- In pigs: Myositis, myocarditis, and muscular stiffness may occur.
- In man: The adult worms may cause abdominal pain, diarrhoea and constipation.
- The pathogenesis is severe in man due to the larval stage of the *Taenia solium*. The cysticercus mainly occurs in the ventricle of brain and cause pain and neurological signs such as epileptic seizure. This condition is called as “**neurocysticercosis**”.



Cysticercus in pork

Diagnosis

- Stool examination for gravid segments in humans to diagnose taeniosis and MRI, CT-SCAN to detect neurocysticercosis. In pigs, post mortem and serodiagnosis may help to diagnose the presence of larval tapeworms.

TAENIA SOLIUM
TREATMENT AND CONTROL

- Same as *T. saginata* except quinacrine should not be used.
- Proper meat inspection.

MESOCESTOIDES LINEATUS

Order	Mesocestoididea
Family	Mesocestoididae
Genus	Mesocestoides
Species	<i>M.lineatus</i>
Host	Dog, cats, wild carnivores and also humans.
Location	Small intestine
I/H	• Oribatid mite or coprophagus beetles - 1st I/H. • Amphibia, reptiles, birds, dog and cat – 2nd I/H.
Metacestode stage	Cysticercoid and Tetrathyridium.
Morphology	• Small to medium sized worm. • Scolex has four elongate oval suckers which are unarmed • Rostellum is absent. Genital pore is situated on the mid ventral line of the ventral aspect. • Ovary is bilobed. • Testes are 50 in number. • In the gravid segment uterus is replaced by par-uterine organ. • No separate uterine pore.

**MESOCESTOIDES LINEATUS
LIFE CYCLE**

- Not fully known. Only based on experimental infection the scientists suggested that it requires two I/H.
- Oribatid mite or coprophagus beetles act as 1st I/H in which cysticercoid like developmental stage occur.
- When infected mite or beetles are ingested by 2nd I/H in which tetrathyridium is formed. It mainly occurs in the peritoneal cavity of 2nd I/H and they multiply asexually by longitudinal splitting of parent scolex.
- The final host acquire infection by ingestion of infected 2nd I/H. Prepatent period is 16 to 20 days

MESOCESTOIDES LINEATUS PATHOGENESIS AND TREATMENT

Pathogenesis

- Adult worms are not pathogenic. But heavy infection causes severe diarrhoea in man.
- If dog act as 2nd I/H, the tetrathyridium causes peritonitis and ascites.

Treatment

- Same as that of *Dipylidium caninum*.

DIPHYLLOBOTHRIUM LATUM

Subclass	Cotyloda
Order	Diphyllidea
Family	Dipyllobothridae
Genus	Dipyllobothrium
Species	D. latum
Common name	Broad fish tape worm
Host	Man, dog, cat, pigs, and other fish eating mammals
Location	Small intestine
I/H	1. 1st I/H - Cyclops (Diaptomus gracilis – copepod crustacean). 2. 2nd I/H – Fresh water fish. (Pike, trout and perch)
Morphology	• Worms are medium to large in size, scolex has a narrow week, deep muscular groove known as “Bothria” (hold fast organ) situated on both dorsal and ventral side. • Scolex is unarmed and almond in shape.

- Each segment contain single set of reproductive organs.
- Genital pore and uterine pore open separately on the ventral aspect, whereas in mesocestoides, there is no separate uterine pore.
- Ovary is bilobed. Vitelline gland and testes distributed in the lateral margin of segment.
- In the gravid segment uterus is spiral tube in shape.

DIPHYLLOBOTHRIUM LATUM

LIFE CYCLE



Diphyllobothrium egg

- Eggs are passed in the faeces of host, they are light brown in colour, operculated and unembryonated when laid.
- The development of eggs occur in the environment, takes several weeks for the development of 'coracidium'. It is a six hooked oncosphere covered with ciliated embryosphere.
- The fully developed coracidium hatch out and swim in the water for short period then the coracidim is ingested by 1st I/H – Cyclops in which develops into "Proceroid" in about 3 weeks time. These infected 1st I/H are ingested by fish (2nd I/H).
- Within the 2nd I/H it develops into plerocercoid in the viscera and musculature.
- D/H acquire infection by eating of infected raw fish. Prepatent period is 4 weeks

DIPHYLLOBOTHRIUM LATUM

PATHOGENESIS AND DIAGNOSIS

Pathogenesis

- In man: It causes non-specific abdominal symptom and macrocytic hypochromic anaemia - pernicious anaemia due to competition between the host and parasite for vitamin B12.

Diagnosis

- Based on clinical signs.
- Faecal examination for the presence of eggs.

DIPHYLLOBOTHRIUM LATUM
TREATMENT AND CONTROL

Treatment

- Praziquantel – 25mg/Kg b wt.
- Niclosamide – 75 – 150mg/Kg b wt.
- Quinacrine – 7 – 10mg/Kg b wt.

Control

- Avoid eating of raw fish.

MODULE-27: PHYLUM-NEMATHELMINTHES

Learning objectives

After reading this module the learner will understand the following

- Importance of nematodes in animals and man.
- Ideal environment for development of larval stage nematodes.
- Various patterns of nematode life cycle and its stages.
- Control of larval stages.

INTRODUCTION - NEMATODES

- Nematodes or roundworms are very important group of helminths which infect animals and humans.
- The nematodes have both direct and indirect life cycle affecting wide range of animals.
- Nematodes are free living/ parasitic and unsegmented, cylindrical in shape.
- Nematodes cannot be stained for identification
- Some of the nematodes have on their cuticle specialized adhesive structure such as hooks in *Tetrameres* sp. Cephalic vesicle in *Oesophagostomum* sp.
- Some of the nematodes have lateral flat cuticular expansion called as ‘Alae’
- If the alae is situated in the cervical region it is called as cervical alae and in posterior region as caudal alae.
- Alimentary canal is present.
- They have a body cavity or coelom. But it is not true coelom. (Pseudo coelom).
- The extremities are tapering except the females of *Tetrameres* spp. which become almost spherical after copulation.
- The sexes are separate the body is covered with cuticle. The cuticle is provided with circular annulation or it may be smooth or it may have longitudinal striation.
- The cuticle is relatively thick in nature and may extend into the buccal capsule, oesophagus, rectum and distal portion of genital duct.
- Sometimes the cuticle forms a special adhesive structure for Eg. Hooks in *Tetrameres* spp. – male
- Simple or complicated cuticular thickening in *Gongylonema* spp.
- Cervical collar or cephalic collar in *Physaloptera* spp.
- Cephalic vesicle in *Oesophagostomum* spp.
- Cuticle is formed by under lying subcuticular layer and muscular layer, the subcuticular layer is called as ‘hypodermis’. In free living form the hypodermis is composed of number of cells. Whereas in parasitic form hypodermis consist of syncytium with a number of nuclei. Subcuticular layer form a four longitudinal thickening on the inner aspect which are situated on dorsal, ventral and lateral aspect.
- Muscular layer: Situated next to subcuticular layer and is divided into 4 quarter by longitudinal thickening.

DIGESTIVE SYSTEM OF NEMATODES

- Mouth is situated at anterior extremity sometimes it may be subdorsal or subventral.
- Mouth is surrounded by lips. In *Ascarids* the mouth is surrounded by three lips one dorsal and two lateral and each lip possess two papillae.
- In *spirurids* the mouth is surround by two lips each lip possess three papillae and subdivided into three parts. In the members of *Strongylidae* the lips are absent.
- Instead of lips the secondary structures may be developed known as 'leaf crown'. It consists of number of pointed process. The leaf crown arises from the rim of the mouth called as *external leaf crown*.
- Those leaf crown arising from brim of the buccal capsule is known as *internal leaf crown*. Eg. *Oesophagostomum* spp.
- In some spp. the buccal capsule has a thick cuticular wall or teeth like structure. Eg. *Ancylostomum* spp.

OESOPHAGUS

- The structure of oesophagus is very important character for identification of spp.
- It is a strongly muscular organ with triradiate lumen. The lumen is covered with thick cuticular lining and has 3 glands, secreting digestive enzymes.
- At the posterior end of oesophagus a bulbar swelling may occur in some specices and it is known as '*Oesophageal bulb*' which contains *valvular apparatus*.
- **Eg.** *Heterakis gallinarum*.

TYPES OF OESOPHAGUS

- Rhabditiform.
- Filariform.

Rhabditiform oesophagus

- Club shaped anterior portion is connected by a narrow neck to pear shaped posterior bulb.
- This type occurs in the 1st larval stage of nematode and adult form of non-parasitic generation [of adult worm]. Eg. Rhabditida.

Filariform oesophagus

- Oesophagus is club shaped without posterior bulb.
- It occurs in the 2nd and 3rd larval stage of nematode and also in parasitic generation of Rhabditida.

EXCRETORY SYSTEM OF NEMATODES

- Excretory system is not carried out by flame cells as in the case of trematodes.
- Excretory regulation by osmotic regulation. This system consists of a pair of unbranched lateral vessels embedded in hypodermis which opens through excretory pore.
- Excretory pore is situated short distance behind the anterior extremity.

NERVOUS SYSTEM OF NEMATODES

- Nervous system consists of number of ganglia connected by nerve fibers, forming the nerve ring around the oesophagus, from this central nerve ring, 6 nerve trunks runs anteriorly and 6 trunks to posterior part.

CLASS: NEMATODA SENSE ORGANS

Amphids

- Amphids consist of a pair of depression on each side of head end well supplied by nerve fibres.
- The amphids are situated either lateral or posterior lateral aspect of head end.
- The function is 'chemoreceptor'.

Phasmids

- A pair of depression occurs on each side of tail end.
- Earlier the phasmids were considered as important character for identification.
- Nematodes with phasmids comes under Phasmidia, and without phasmids comes under Aphasmidia.

Cervical papillae

- Eg. *Oesophagostomum* species. A pair of papillae occurs in the cervical region.
- Papillae in the middle of body.

Genital papillae

- In females, a pair of papillae occurs in vulval region.

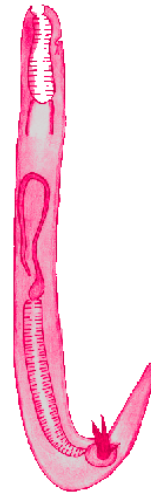
REPRODUCTIVE SYSTEM OF NEMATODES

- Sexes are usually separate, sexual dimorphism occur.
- Usually male worms are smaller than the female.

MALE REPRODUCTIVE SYSTEM OF NEMATODES

- It consists of single testis, vas deferens, seminal vesicle and ejaculatory duct. These ejaculatory duct opens into the cloaca. In most of the species have one or two spicules.
- *Spicules* are lying in the sheath, which opens into the cloaca, spicules are chitinized, pigmented, they vary in size and shape. The number, size and shape is very important character for identification of species.

- *Function of spicules:* For attachment during copulation and expand the vagina for direct the flow of sperms.
- In many species, the wall of the cloaca is provided with cuticular thickening. This thickening assists the spicule movement.
- Cuticular thickening on the dorsal wall of cloaca is called as '*gubernaculum*', cuticular thickening on the ventral wall of cloaca is called as *telamon*.



FEMALE REPRODUCTIVE SYSTEM OF NEMATODES

- It consists of a pair of ovary, oviduct and uterus which ends in a short vagina and opens at vulva. In some species like *Haemonchus contortus* the vulva is covered with vulval flap or knob.
- At the junction of uterus and vagina, a small muscular organ may occur and it is known as *ovijector*, its function is assisting in laying eggs.
- One female may lay several thousand eggs per day.



EGGS OF NEMATODE

- Nematodes are oviparous or ovoviviparous or viviparous.
- Oviparous: Nematodes that lay eggs which contains only yolk material when laid. Eg. Ascarid.
- Ovoviviparous: Nematode that lay eggs it contains fully developed larvae when laid. Eg. *Subulura brumpti*.
- Viviparous: Giving birth to larvae. Eg. Filarid nematode.
- The larvae of filarid nematode is called as Microfilaria.
- Nematode eggs vary greatly in shape and size. Usually they are round, oval and ellipsoidal in shape and covered with 3 layers. In some species, the middle layer is interrupted with operculum or plug. The plug may be occur at one end or at both the ends of egg.
- Eg. Plug at one end – *Oxyuris equi*. Plug at both end – *Trichuris ovis*.

DEVELOPMENT AND HATCHING OF NEMATODE EGGS

- Original egg cell divide into 2, 4, 8, 16, 32, and so on. The developmental stage consists of morula and tadpole stage. After completion of larval development the eggs are ready to hatch. Hatching of eggs depends on the species of worms.
- Eggs may hatch outside the host i.e., in the environment or may hatch after ingested by D/H or I.H.Eg. In Ascarid – the egg hatch only after ingested by the D/H.
- In *Spirocerca lupi* – the egg hatch only after ingested by the I/H.
- In *Strongyle* spp. – the egg hatch in the environment, that is out side the host.
- Hatching of the eggs in the environment is controlled by temperature, moisture, larval movement and enzymes secreted by larva.
- Hatching of eggs inside the host is controlled by host stimuli like CO₂ concentration.

BINOMICS OF LARVAE

- Usually 4 moults or ecdyses (L1, L2, L3, and L4) occurs in nematode.
- During moulting, the old cuticle is replaced by new one. The L1 and L2 feed on bacteria.
- L3 can't feed because the cuticle of L2 retained as a protective sheath around L3 larvae (infective stage).
- In most of the nematode species, L3 is the infective stage where as in Ascarid L2 is considered as a infective stage.

LIFE CYCLE PATTERN OF NEMATODE

Direct life cycle

- Eggs develop and hatch outside the host. The larvae live as a free living for some time and later become infective. The infective larvae enter the host by *two ways*.
 - In some species the infective stage enter into the host through mouth along with feed and water. *Eg. Strongylidae and Trichostrongylidae*.
 - But the hookworm larvae (infective stage) enter into the host by skin penetration. *Eg. Ancylostomum and Bunostomum spp.*
- The development of eggs occur in the environment but eggs do not hatch outside the host (i.e., in the environment). Host getting infection by ingestion of embryonated eggs. *Eg. Ascarid*.

Indirect life cycle

- Eggs hatch outside the host or ovoviviparous. The larvae are free living for some time. Then enter into the suitable I/H and become infective stage. Final host acquire infection by ingestion of infected I/H or infective stage injected by the I/H while feeding. *Eg. Metastrongylidae and Habronema*.
- Eggs do not hatch outside the host. They hatch only after ingestion by I/H. D/H acquire infection by ingestion of I/H. *Eg. Spirocerca lupi*
- Viviparous in which the adult female worm directly give birth to larvae, they enter into the blood circulation of the host. From the blood it is taken up by blood sucking I/H, within the I/H the larvae become infective. The infection of final host by biting of infected I/H (by inoculation). *Eg. Setaria digitata*.

MODULE-28: ASCARIS OF LARGE ANIMAL

Learning objectives

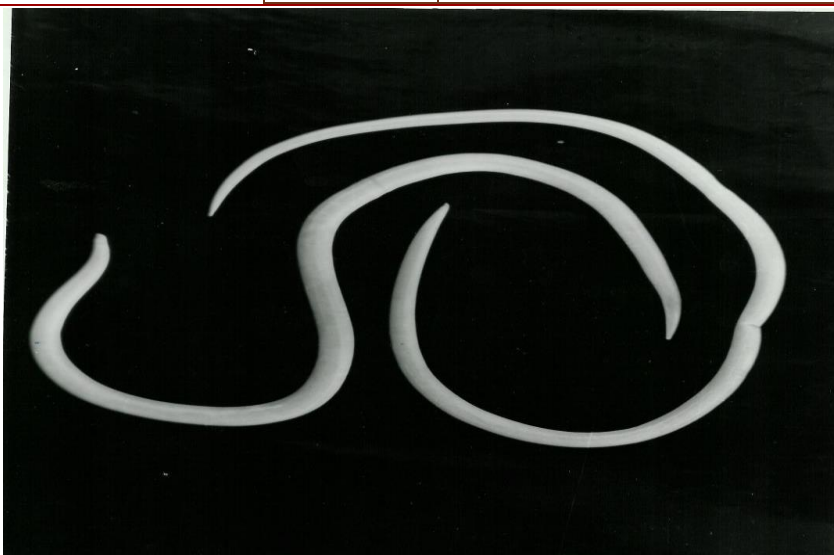
After reading this module the learner will understand the following

- Importance of soil borne nematode infection in animals and man.
- Transmission of milk borne ascarid infection in buffalo calves.
- Importance of deworming of calves, piglets and foals.

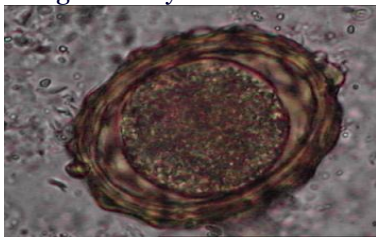
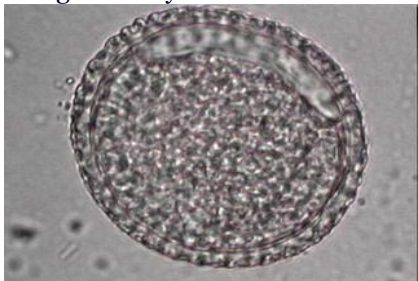
ASCARIS OF LARGE ANIMALS MORPHOLOGY

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Order	Ascaridida
Super Family	Ascaridoidea
Family	Ascarididae
Genus	<i>Ascaris</i>
Family character	Large nematode, buccal capsule is absent. Mouth is surrounded by 3 lips. Oesophagus is devoid of posterior bulb.



Ascaris

<i>Ascaris suum</i>	<i>Parascaris equorum</i>	<i>Toxocara vitulorum</i>
Host: Pigs	Horse	Cattle and buffaloes, common in buffalo calf.
Location: Small intestine	Small intestine	Small intestine
Morphology Mouth: Surrounded by 3 lips. Dorsal lip has 2 papillae	Surrounded by 3 lips. They are separated by intermediate lips	Surrounded by 3 lips. Base of the lip is broad.
Tail end of male Pointed, precloacal papillae are numerous and 5 pairs of postcloacal papillae is present 	Numerous precloacal papillae and 5 pairs of postcloacal papillae. Median papillae also present.	They have <i>spike like appendage</i> . 5 pairs of precloacal and 5 pairs of postcloacal papillae. The anterior postcloacal papillae is large and double in number.
Spicule: Sub equal	Sub equal	Sub equal
Egg Sub globular in shape. Eggshell is thick. Provided with <i>knob or cuticular bosses</i> . It contains unsegmented yolk. 	Sub globular in shape. Eggshell is thick. Provided with <i>finely pitted</i> structure. It contains unsegmented yolk. 	Sub globular in shape. <i>Coarsely pitted</i> structure. It contains unsegmented yolk. 
Male: 15 to 20cm Female: 40cm	15 to 18cm 50cm	25cm 30cm
Prenatal infection No prenatal infection	No prenatal infection	Prenatal infection occurs.
Cuticle: Normal	Thick and rigid in nature. It has distinct head and neck	Smooth and translucent

LIFE CYCLE OF *Ascaris suum* and *Parascaris equorum*

- A single female lays about two lakhs eggs per day. Eggs are passed in the faeces of host and develop to infective stage in about 10 days or above depending upon the environmental temperature.
- The eggs are resistant to adverse conditions like drying, freezing and chemicals and remain viable for 5 years.
- The infection of host by ingestion of embryonated eggs containing *L₂ larva*. Ingested egg hatch in the intestine and liberate *L₂*.
- The liberated *L₂* penetrate the intestinal wall reach the peritoneal cavity and then to liver. Majority of *L₂* reach the liver via hepatportal system.
- From the liver, the larvae carried to the right atrium via superior venacava by blood and then to lungs.
- In the lungs larvae are arrested in the capillaries where the *L₂* moult to *L₃*. These *L₃* break out of the alveolar capillaries and pass through alveolar duct and small bronchioles. Then the larvae gradually ascend the bronchial tree and reach the trachea. From the trachea the larvae migrate to pharynx and mouth and finally swallowed to reach the intestine. This migration is called as, "*Tracheal migration*".
- Large number of *L₃* reach intestine in about 7 to 8 days after infection.
- In the intestine *L₃* moult to *L₄* in about 14 to 21 days and *L₄* to *L₅* in about 21 to 29 days. The worms reach maturity in about 50 to 55 days. Eggs appear in faeces in about 62 days.
- In *P. equorum*, the worm reach maturity in 80 to 83 days.

LIFE CYCLE OF *Toxocara vitulorum*

- Adult worms are exclusively found in calves. Transmammary route is the major source of infection to calves.
- Eggs develop into infective stage in about 15 days.

Transmammary route

- Ingestion of embryonated eggs by neonates, juvenile or adult does not directly lead to patent infection.
- After hatching of eggs in the intestine, *L₂* are distributed to various organs and tissues where they remain dormant until later part of the pregnancy of animals (8th month).
- During 8th month of pregnancy the larvae migrate to mammary gland and occurs in colostrum after parturition.
- The calf acquire infection by ingestion of larvae along with colostrum, larvae reach stomach, intestine and attain maturity in 4 weeks.
- The natural expulsion of adult worm occurs in about 38 days after birth and by 4 – 6 months no adult parasite remain.

ASCARIS OF LARGE ANIMALS PATHOGENESIS AND CLINICAL SIGNS

Pathogenesis and clinical signs of *A. suum*

- Young pigs are commonly affected. Heavy infection in neonatal pigs causing pneumonia, cough and exudate in the lungs. These symptoms similar to that of symptoms occurs in “thumps” or piglet anaemia.

PM lesions

- Varying degrees of fibrosis occurs in liver, the fibrosis is localized in the form of “Milk spot”. Petechial haemorrhage occurs in lungs. Desquamation of alveolar epithelium, edema of lungs and eosinophilic infiltration is seen.

Pathogenesis and clinical signs of *P. equorum*

- Foals 3 to 9 months of age are commonly affected. Heavy infection causes coughing and circulating eosinophilia. Adult worm causes catarrhal enteritis and foetid diarrhoea.
- General malaise, debility and pot belly may occur. Hair Coat becomes very rough, sometimes the adult worms enter into the bile duct causing jaundice and also the adult worm enter into the intestinal wall causing generalized or localized peritonitis.

Pathogenesis and clinical signs of *T. vitulorum*

- Light infections pass unnoticed. Heavy infection causes diarrhoea, steatorrhea (fat in faeces), colic and passing mud coloured evil smelling faeces. In India and Sri Lanka calf hood mortality due to *T. vitulorum* is common.

ASCARIS OF LARGE ANIMALS DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- Demonstration of eggs in faeces.
- Demonstration of larvae in sputum especially for *A. suum*.

Treatment

A. suum

- Piperazine compounds: 100 to 400 mg/Kg b wt.
- Fenbendazole – 5 mg/Kg b wt.
- Levamisole – 7.5 mg/Kg b wt.
- Morantel tartrate – 12.5 mg/Kg b wt. for sow.

P. equorum

- Thiabendazole – 44 mg/Kg b wt.
- Mebendazole – 10 mg/Kg b wt.

T. vitulorum

- Piperazine compounds – 250 mg/Kg b wt..
- Fenbendazole – 7.5 mg/Kg b wt.
- Levamisole – 7.5mg/Kg b wt.

Control

A. suum

- Protection of young pigs are very important. In USA *Mac lean county system* is followed for the control of *A. suum* in this system, pregnant sow is treated for ascaris little time before farrowing.
- Sows should be thoroughly washed and scrubbed within 3 days of farrowing in order to remove the eggs adhering to the body. After that, the sow should be placed in farrowing pen made of concrete floor and washed with hot caustic soda.

P. equorum

- Foals must be kept in clean paddock and should not be allowed to graze along with the mother. The pregnant mare should be treated for ascaris before foaling.

T.vitulorum

- Calf must be dewormed within 10 to 15 days of birth.

MODULE-29: ASCARIS OF DOG AND CAT

Learning objectives

After reading this module the learner will understand the following

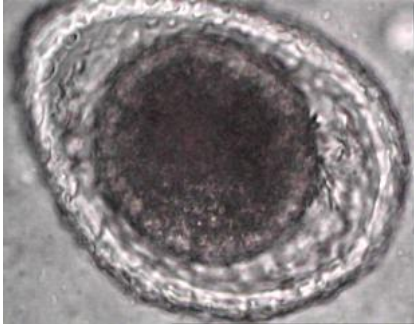
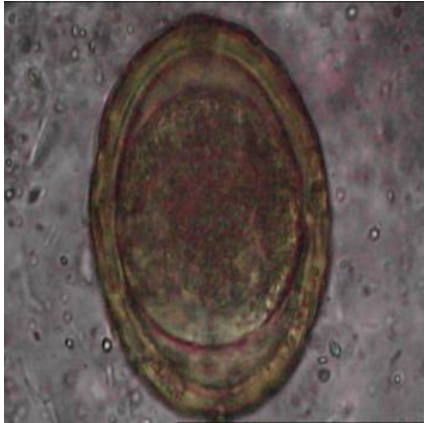
- Visceral larval migrans in children.
- Congenital mode of transmission in bitches.
- Need for regular deworming of puppies and kittens.
- Neonatal ascarid infection in puppies and kittens.

MORPHOLOGY OF *TOXOCARA* SP.

The ascaris of dog and cat are

- *Toxocara canis*
- *T.cati*
- *Toxascaris leonina*

<i>T. canis</i>	<i>T. leonina</i>	<i>T. cati</i>
Host: Dog and fox. Common in below 3 months of age	Dog and cat	Cat and wild felidae
Morphology: Male - 10cm Female -18cm in length.	7cm 10cm in length.	6 cm 9cm in length.
Anterior end :bent ventrad 	Anterior end bent dorsad	Same as that of <i>T.canis</i>
Cervical alae is lengthy.	Lengthy	Cervical alae is broad and striated.
Cervical alae looks like a “arrow”. So these worms are commonly called as <i>Arrow headed worms</i> .		

Tail end of male: Tail ends in a finger like appendage called as “digitiform appendage” 	Pointed tail end	Pointed tail end
Spicules: Sub equal	Sub equal	Sub equal
Eggs: Sub globular in shape. Egg shell is thick, and provided with finely pitted structure with unsegment yolk 	Oval in shape. Shell is thick and smooth 	Similar to <i>T.canis</i> 

LIFE CYCLE OF T. canis

- Very complex. It depends on the age of the host.

Types of life cycle

- [Direct](#)
- [Prenatal](#)
- [Transmammary](#)
- [Paratenic host involvement](#)

Direct

- Eggs developed to infective stage within 10 to 15 days.
- *Pups upto 3 months of age:*
 - Following ingestion of embryonated eggs hatch in the duodenum, the liberated L₂ penetrate intestinal wall and follow the tracheal route of migration.
 - The L₂ moult to become L₃ in lungs about 10 days after infection.
 - After reaching the intestine L₃ moult to L₄ in 2 weeks of infection. The L₄ moult to L₅ in about 5 weeks of infection.
 - Tracheal route of migration occur only upto 3 months pup. If the *pup is above 3 months*, tracheal route is less frequent but somatic migration is common.

Prenatal infection

- In pups above 3 months of age, following ingestion of embryonated egg hatch in the intestine, the liberated L₂ are distributed to various tissues and organs like liver, kidney, brain and lungs, where they remain dormant.
- The dormant larvae begins to migrate in the pregnant bitches about 3 weeks prior to whelping (after 42nd day of pregnancy) and enter into the foetus. When the larvae reaches the liver of the foetus, L₂ moult to become L₃.
- At birth L₃ present in the lungs of puppy. During 1st week of birth L₃ moult to L₄ in the lungs, by the 2nd week of birth L₄ moult to L₅ and pass to the intestine.
- Eggs appear in the faeces 23 to 40 days after birth of the pup.

Transmammary route

- Larvae passed to suckling pups via colostrum. These larvae reach the intestine and directly develop to adult without migration.

Paratenic host

- Rodents may act as a paratenic host. Rodent may ingest the embryonated eggs. In which the liberated L₂ distributed to various organs and remain dormant. Infection of dogs is by ingestion of paratenic host. In the intestine of dog larvae develop to adult directly without migration.

LIFE CYCLE OF *Toxascaris leonina*

- Direct
- Paratenic host

Prenatal and transmammary route of infection is absent.

- **Direct**
 - Ingestion of embryonated eggs hatch in the intestine. The liberated L₂ enter into the intestinal wall where L₂ moult to become L₃ and L₃ moult to become L₄ in about 10 days and 5 weeks of infection respectively.
 - The L₄ comes to the lumen and moults to become L₅ in 6 weeks after infection. Prepatent period is 74 days.
- **Paratenic host**
 - Mice acts as a paratenic host. In mice L₂ are distributed to various organs and remain dormant until eaten by dogs and cats.
 - Following ingestion of infected mice by dog, larvae are released in the intestine and develop to adult without migration.



LIFE CYCLE OF *T. cati*

- Prenatal infection is absent. But transmammary route of infection is seen.
- Direct and paratenic host cycle commonly occurs in cat.

Direct

- Similar to *T. canis* as it occurs in 3 months and below age groups.

Paratenic host

- Earthworm, cockroach, chicken and sheep may act as a paratenic host.
- Infection of final host by ingestion of infected paratenic host. There is no migration of larvae.

ASCARIS OF DOG AND CAT

PATHOGENESIS AND CLINICAL SIGNS

Pathogenesis of *T.canis*

- Heavy infection occurs in young pups due to poor hygienic condition of kennels. Heavy prenatal infection leads to death of whole litters of puppies.
- The migrating larvae causes pneumonia in pups and heavy infection causes vomition and diarrhoea. So the pups are frequently covered with stale vomit and may suffer from inhalation pneumonia. Death occurs about 2 to 3 weeks of birth. Adult worm causes mucoid enteritis. Sometimes the adult worms enter into aberrant site like bile duct causes serious pathogenic effect.

Clinical signs

- Unthriftiness, dullness, harsh coat and pot belly appearance or tucked up abdomen may occur. Sometimes the entire worms are vomited or passed in faeces. Emaciation and anaemia are common.

ASCARIS OF DOG AND CAT

DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- By clinical signs
- Confirmation by finding the eggs in the faeces.

Treatment

- Piperazine compounds – 200 mg/Kg b wt.
- Diethyl carbamazine – 50 mg/Kg b wt. [DEC]
- Pyrantel pamoate – 5 mg/Kg b wt. [Combandrin]
- Mebendazole – 10 mg/Kg b wt. Twice daily for 2 days.
- Fenbendazole – 100mg/Kg b wt. Single dose or divided over five days.

Control

- Hygienic maintenance of kennel.
- Regular treatment of pups and pregnant bitches.

VISCERAL LARVAL MIGRANS [VLM]

- It is a condition which occurs in *unnatural host* (in human being especially children) caused by larvae of *T.canis*, *T.cati*, *Toxascaris leonina*, *Capillaria hepatica* and *Lagochilascaris major*.
- Commonly occurs in one to 5 years age group children. Because of the habit of dirt eating.
- The soil may be contaminated with eggs of *T.c anis*. Following accidental ingestion of embryonated eggs hatch in the intestine, the liberated L₂ penetrate the intestinal wall and migrate to various visceral organs like liver, lungs, kidney, brain and eye.

Pathogenesis

- Hepatomegaly, *eosinophilic granulomatous* lesions in the liver, intermittent fever, persistent coughing, eosinophil may reach upto 50%, pulmonary infiltration and loss of appetite. The eye lesion may resemble retino blastoma.

Diagnosis

- Based on clinical signs.
- Hepatomegaly.
- Leucocytosis.
- Immuno diagnosis.
- Demonstration of larvae in biopsy.

MODULE-30: ASCARIDS OF POULTRY

Learning objectives

After reading this module the learner will understand the following

- Role of arthropods such as beetles in transmission of poultry ascarids.
- Control of arthropods in poultry farms.
- Need for regular deworming of poultry.

ASCARIDS OF POULTRY INTRODUCTION

The following are the poultry ascarids

- *Ascaridia galli*
- *Heterakis gallinarum*
- *Subulura brumpti*

ASCARIDIA GALLI

Common name	Large round worm of poultry
Host	Fowls
Location	Small intestine

Morphology

- Worms are stout and densely white, female – 12cm.
- Three large lips and club shaped oesophagus without posterior bulb.
- Tail end of male has small caudal alae and bears 10 pairs of sessile papillae.
- Circular preloacal sucker with a thick cuticular rim. Spicules are sub equal.
- Eggs are oval with smooth shell.



ASCARIDIA GALLI **LIFE CYCLE**

- Is direct, eggs are passed in the droppings developed into infective stage in 10 days or longer.
- Infection of chicken by ingestion of egg having L₂ along with food and water.
- Eggs hatch in the lumen of intestine and larvae live for 8 days in the lumen.
- Majority of them enter into intestinal mucosa from 8 to 17th day. Finally the larvae again reenter into the lumen and reach maturity in 6 to 8 weeks.
- Earthworm may act as transport host.

ASCARIDIA GALLI **PATHOGENESIS AND CLINICAL SIGNS**

- Young birds are more susceptible (1 to 3 months of age) than the adults.
- Dietary deficiencies such as vitamin A, B and B₁₂, various minerals and proteins leads to heavy infection.
- Chicken over 3 months are more resistant to infection and this may be accompanied with increase in goblet cells number in gut mucosa during this age.
- Marked lesions are produced when large number of young parasite penetrate into duodenal mucosa may cause severe haemorrhagic enteritis.
- Birds become anaemic and suffer from diarrhoea. Affected birds become unthrifty, markedly emaciated and egg production is decreased. In heavy infection, intestinal obstruction may occur.



Ascaridia galli-chicken

ASCARIDIA GALLI **POST - MORTEM LESIONS AND DIAGNOSIS**

Post – mortem lesions

- Haemorrhagic enteritis with larval stage about 7 mm are found in mucosa, carcass emaciated and anaemic, adult worms are found in lumen of the intestine.
- Viable or calcified parasites may be found in the albumin part of hen's egg.



Diagnosis

- Eggs in droppings or worms in the intestine at autopsy.

ASCARIDIA GALLI TREATMENT AND CONTROL

Treatment

- Piperazine compounds are highly effective. Piperazine adipate should be given 300 to 400mg/Kg b. wt.
- Phenothiazine + Piperazine mixture can also be used.
- Hygromycin – B @ 8 g per tonne of feed. Administered for 8 weeks.
- Mebendazole and Tetramisole also useful.

Control

- Young birds should be separated from older birds.
- Poultry runs should be well drained.
- Provide clean feeding troughs and drinking water appliances.
- Litter materials should always be kept in dry condition.

Heterakis gallinarum

Common name	Caecal worm of poultry
Host	Fowl, Turkey, duck and Pea fowl
Location	Large intestine and Caecum

Morphology

- Whitish small sized worm, 1.5 cm long.
- Large lateral alae extending down the side of the body.
- Oesophagus has strong posterior bulb having valvular apparatus.
- Tail end of male has large alae, circular precloacal sucker and 12 pairs of pedunculated papillae. Spicules are unequal, right one is slender and long. Left one is short and broad.
- Eggs are oval, smooth shelled.

LIFE CYCLE *Heterakis gallinarum*

- Is direct, eggs developed into infective stage in 14 days. Infection of chicken by ingestion of egg containing L₂ stage.
- L₂ hatch in the intestine move to caecal mucosa and remain there for 2 to 5 days and moult to L₃ in the lumen of caecum on 6th day of infection.
- L₄ in 10 days and L₅ in 15 days. Prepatent period is 24 to 30 days, earthworm may act as transport host.

PATHOGENESIS AND DIAGNOSIS *Heterakis gallinarum*

Pathogenesis

- Usually non pathogenic, but heavy infection cause thickening of caecal mucosa with petechial haemorrhage on the surface.
- The significance of the worm is that the protozoan viz. *Histomonas meleagridis* causing black head or entero hepatitis in turkeys is incorporated in the eggs of *Heterakis gallinarum*.

Diagnosis

- Finding eggs in the feces.

TREATMENT AND PREVENTION *Heterakis gallinarum*

Treatment

- Phenothiazine is effective at the dose rate of one gram per bird.
- Phenothiazine + Piperazine mixture is used to eliminate the mixed infections of *H. gallinarum* and *A. galli*.
- Hygromycin B - 0.25% mix in feed is highly effective.
- Mebendazole is also be used.

Prevention

- Strict sanitation of poultry houses is essential.

MODULE-31: FAMILY-OXYURIDAE

Learning objectives

After reading this module the learner will understand the following

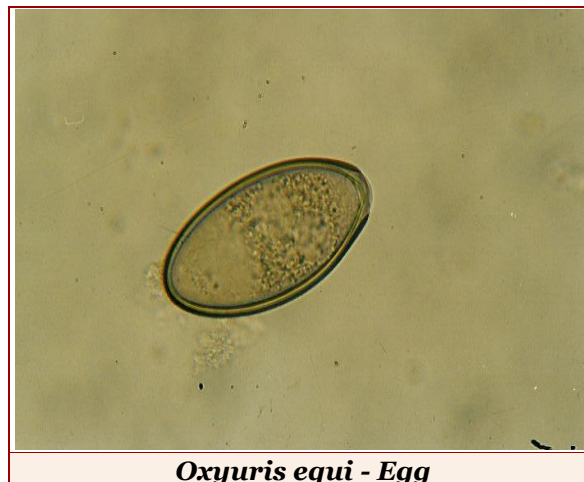
- Anal pruritus in horses.
- Direct mode of transmission.

OXYURIS EQUI **MORPHOLOGY**

Genus	<i>Oxyuris</i>
Species	<i>O.equi</i>
Common name	Pin worms of horses or false whipworm

Morphology

- Small or medium sized worm with inconspicuous lips. Mature female are 10cm in length. Male less than one cm.
- Oesophagus – Hour glass shaped.
- Male tail end is truncated having single pin shaped spicule. Females are usually much larger than males and have long tapering tail end (whip like).
- Eggs are elongate, flattened on one side provided with mucoid plug at one pole.



***Oxyuris equi* - Egg**

OXYURIS EQUI **LIFE CYCLE**

- Adult worms found in caecum and colon. After fertilization the gravid female wander down to rectum and crawl out through anal opening, eggs are laid in the clusters in the perineal region. Eggs appear as yellowish white streaks – gelatinous.
- Development of egg is rapid usually eggs fall off to the ground and reach infective stage in 3 to 5 days. Sometimes infective stage is reached in perineal region itself. Eggs survive for several weeks in moist surroundings.
- Eggs are commonly found in stable and bedding materials.
- Infection of horse occur by ingestion of embryonated eggs contain L₂, hatch in the intestine. The liberated L₂ moult to L₃ and L₃ migrate to caecal mucosa where it becomes L₄ in 8 – 10 days of infection. L₄ come down to caecal lumen, in which they become adult. Prepatent period is 4 to 5 months.

OXYURIS EQUI **PATHOGENESIS AND CLINICAL SIGNS**

Pathogenesis

- L₄ feed on caecal mucosa while adult worms feed on intestinal contents.
- The chief clinical feature of oxyurosis is ‘anal pruritus’ produced by egg laying females.

Clinical signs

- Restlessness, improper feeding and dull coat, animal rubs its base of the tail against any suitable hard object causing hair to break off and the tail to acquire an ungroomed ‘rat-tailed appearance’.

OXYURIS EQUI **DIAGNOSIS, TREATMENT AND CONTROL**

Diagnosis

- Examining the perineal region for eggs. This condition should be differentiated from mange.

Treatment

- Mebendazole – 5 to 10 mg/Kg b.wt oral
- Piperazine compounds – 400 mg/Kg b.wt oral
- Thiabendazole – 100 mg/Kg orally

Control

- A high standard of stable hygiene should be followed.
- Frequent removal of bedding materials.
- Perineal region and underside of tail should be cleaned properly.

MODULE-32: ORDER-RHABDITIDA

Learning objectives

After reading this module the learner will understand the following

- Pre-parasitic and parasitic larval stages.
- Types of larval stages under various families.
- Pathogenesis due to L4 stages in animals.

STRONGYLOIDES PAPPILOSUS

MORPHOLOGY

Super family	Rhabditoidea
Family	Strongyloididae
Genus	Strongyloides
Species	<i>S. papillosus</i>
Host	Sheep, goat and cattle
Location	Small intestine
Common name	Thread worms of ruminants

Morphology

- Slender hair like worms less than one cm long.
- Free living generation are saprophytic, parasitic generation are living in gut of vertebrate.
- Oesophagus is *rhabditiform* in free living and *filariform* in parasitic phase.
- Adult females are parasitic, long filariform oesophagus occupy upto 1/3 of length and uterus is intertwined with intestine giving the appearance of *twisted thread*.
- Parasitic forms are *parthenogenetic* they lay the eggs which may either give rise to another parasitic or free living generation of males and females.
- Free living generation produces larvae of parasitic generation which are able to penetrate through skin of host and pass to lungs via circulation, then move to trachea and finally reach the intestine.
- Eggs are small oval, thin shelled with both end blunt and contain fully developed embryo when laid.

STRONGYLOIDES PAPPILOSUS
LIFE CYCLE

Parthenogenetic female egg ---> L1 ---> L2 ---> L3 (infective stage)

Free living ♀ ♂

D/H

Egg ---> L1 ---> L2 ---> L3

- Lifecycle may be direct heterogonic or homogonic.
- Completely parasitic and completely free living cycles or combination the of both can occur.
- The parthenogenetic female is found buried in the mucosa of S.I and prodces thin shelled transparent eggs which are passed in faeces.
- L1 may develop either directly to become L3 (infective stage) or develop to free living female and male which may subsequently produce infective larvae (Heterogonic cycle). When environmental conditions are satisfactory (temp & humidity) heterogenic cycle predominates. But when environmental conditions are unfavourable homogonic lifecycle predominates.
- In heterogonic lifecycle, L1 rapidly transformed to sexually matured free living males and females within 48 hrs. Following copulation the free living females produces egg which hatch in a few hrs and these larvae metamorphose to become infective larvae.
- In homogonic lifecycle, L1 rapidly develop to become infective larvae [L3] within 24 hrs.
- Infection of the vertebrate host is by skin penetration and though oral infection may also occurs.
- During oral infection, larvae penetrate the mucosa of mouth or oesophagus may lead to systemic migration.
- After skin penetration the larvae reach the skin capillaries and venules then they are carried by blood to lungs.
- In the lungs they breakout into alveoli then migrate up in smaller bronchioles to bronchi, trachea and mouth finally swallowed to reach the intestine, where they nature. Prepatent period is 5 to 7 days.
- Sometimes prenatal and transmammary route of infection is also possible in sheep and cattle.

STRONGYLOIDES PAPPILOSUS
PATHOGENESIS

- Skin penetration by infective larvae may cause an erythematous reaction.
- Erosion of intestinal mucosa.
- Young animals are severely affected.

STRONGYLOIDES PAPPILOSUS
CLINICAL SIGNS

- Anorexia, loss of condition, diarrhoea and moderate anemia. Field out break of disease are associated with catarrhal enteritis.
- The larvae of *S.papillosus* are associated with introduction of foot-rot organism caused by *Bacteroides nodosus* into the skin around the feet of sheep.

STRONGYLOIDES PAPPILOSUS
DIAGNOSIS

- Demonstration of eggs or L1 in faeces.

STRONGYLOIDES PAPPILOSUS
TREATMENT

- Benzimidazoles are useful.

STRONGYLOIDES PAPPILOSUS
CONTROL

- Good hygienic management.

STRONGYLOIDES STERCORALIS

- Occurs in the small intestine of man, various other primates, dogs, fox and cattle
- Size of female parasite 2.2mm long and 0.034 mm thick, the male parasite is 0.7mm long
- The larvae are found in fresh faeces

STRONGYLOIDES AVIUM

- Its occurs in the small intestine and caeca of the fowl, turkey and wild birds
- It is 2.2 mm long, the esophagus is 0.7 mm long

MODULE-33: ORDER-STRONGYLIDA

Learning objectives

After reading this module the learner will understand the following

- Importance of strongyle infection with respect to colic in horses.
- Pimplygut condition in sheep and goats.
- Pathogenesis due to L4 larval stage in intestine.

STRONGYLIDA - AN INTRODUCTION

The members belonging to this super family are known as “bursate nematodes”

Family

- Strongylidae
- Syngamidae

Strongylidae

Characters

- It has a well developed buccal capsule. A median thickening in buccal capsule is known as dorsal gutter.
- It possess both external and internal leaf crown or “corona radiata”.
- Teeth or cutting plates may be present.
- Leaf crowns resemble “palisade” so it is commonly called as palisade worm.
- In male, the bursa is well developed. Bursal lobes are supported by modified caudal papillae, known as bursal rays.
- Spicules are equal.

Genus: Strongylus

Species

- *S.vulgaris*
- *S.edentatus*
- *S.equinus*

Host: Equine

Location: Large intestine.

MORPHOLOGY OF *Strongylus vulgaris*

Worms are smaller than other two species. Buccal capsule roughly oval in shape.

- Buccal capsule contain two ear shaped dorsal teeth.
- Both internal and external leaf crowns are present.
- External leaf crowns fringed at distal extremity.
- Spicules are equal.

LIFE CYCLE OF STRONGYLOIDES

- Eggs are passed in faeces of host, oval in shape, thin shell contains early segmentation of yolk.
- The development and hatching of eggs is controlled by various factors like moisture, temperature and O₂.
- At 26°C the first stage larvae is produced within 24 hrs.



Strongyle Larva

BIONOMICS OF STRONGYLE LARVAE

The L1 hatch out from the eggs, L1 have rhabditiform oesophagus.

- L1 mainly feeds on bacteria and grows rapidly, then enter into the lethargic state and moult to L2.
- L2 also feeds on bacteria, soon after lethargic state the L2 moult to L3. L2 have less rhabditiform oesophagus.
- L3 have filariform oesophagus, it is a infective stage. Cuticle of L2 is retained as a protective sheath around L3. Which is very important for survival of infective stage. Since L3 wrapped with cuticle of L2 it does not feed. L3 thrive on the stored food materials in the intestinal cells. Infective stage is negatively geotropic and positively phototropic to mild sunlight, but it will be repelled by strong sun light. L3 larva crawls on grass blades in early morning, evening and during dull weather. L3 survive upto 3 months.
- Infection of horse by ingestion of L3 along with herbage or vegetations.

- Exsheathment occurs in the small intestine of horse.
- After exsheathment, L3 penetrate the intestine wall where L3 moult to become L4 in about 3 days of infection. These L4 penetrate the intima of submucosal arterioles and migrate towards the cranial mesenteric artery. In the cranial mesenteric artery it produces thrombus and later aneurysm in about 14 days of infection. From 45 days of infection onwards the L4 pass back to submucosa of caecum and colon via arterial system. In the submucosa of caecum and colon L4 moult to L5. Then L5 enter into the lumen of intestine and reach maturity in about 3 months.

STRONGYLIDA **PATHOGENESIS**



- L4 in the cranial mesenteric artery causes inflammatory reaction lesions like endarteritis and thrombus formation.
- Due to thrombus formation, initial thickening and later dilatation of arterial wall may occur.
- Sometimes the thrombus detached leads to fatal results like occlusion of coronary artery or brachio cephalic trunk.
- Infarction of iliac artery leads to temporary lameness.
- Thrombus in the testicular artery results in passive congestion of one or both testicle.
- Diarrhoeic syndrom may occur and is associated with ulceration of caecum and colon due to thromboembolism caused by migrating larvae.



STRONGYLIDA

CLINICAL SIGNS

- Colic is due to pressure of cranial mesenteric artery on nerve plexuses.
- Rough coat, diminished appetite, diarrhoea, edematous swelling on the abdomen and leg, emaciation and anaemia.
- Adult worm causes heavy blood loss due to blood sucking activity resulting in anaemia. (Normocytic, normochromic anaemia).
- Large number of haemorrhagic ulcers are seen in the intestine which indicates the site of attachment of worm.

STRONGYLIDA

DIAGNOSIS

- Faecal examination for eggs
- Detection of anterior mesenteric aneurysm by rectal palpation.
- PM examination reveals ascitis, emaciation and anaemia.

STRONGYLIDA

TREATMENT

- Fenbendazole – 7.5mg / Kg b wt. (oral).
- Thiabendazole – 440mg / Kg b wt. (oral).
- Ivermectin 0.2mg / Kg b wt s/c.

STRONGYLIDA

CONTROL

- Periodic deworming of horse.
- Proper disposal.
- Use clean pasture.

OESOPHAGOSTOMUM SP.

MORPHOLOGY

Species

- *O. columbianum*
- *O. radiatum*

Character

- Cylindrical buccal capsule. Cervical groove, cephalic vesicle and cervical papillae are present.
- Generally called as *nodular worm*.

<i>O.columbianum</i>	<i>O.radiatum</i>
Common name : Nodular worm of sheep and goat	Nodular worm of cattle
Location : Colon	Colon
Morphology: Worms are stout, white in color. 1 to 2 cm in length	Worms are stout, white in color. 1 to 2 cm in length
Cuticle forms a <i>cone shaped</i> mouth collar, which is fairly high and separated from the rest of the body.	Rounded mouth collar
Both external and internal leaf crown are present.	External leaf crown is absent
Possess large cervical alae, produces dorsal curvature of worm (anterior part)	Possess large cervical alae, produces dorsal curvature of worm (anterior part)
Cuticle anterior to cervical groove forms a vesicle known as “ <i>cephalic vesicle</i> ” which is slightly inflated.	Constriction at the middle of cephalic vesicle
A short distance behind the cervical groove a pair of “ <i>cervical papillae</i> ” is present	A short distance behind the cervical groove a pair of “ <i>cervical papillae</i> ” is present



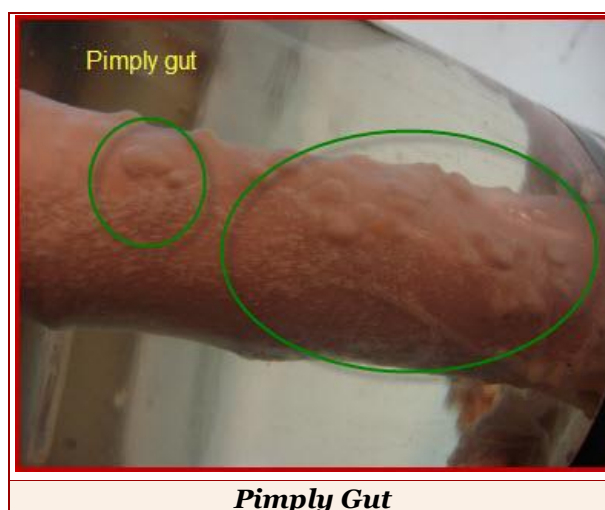
Oseophagostomum radiatum

OESOPHAGOSTOMUM SP. **LIFE CYCLE**

- Eggs are passed in the faeces of host. The development and bionomics of the larvae are similar to *Strongylus* spp.
- L1 emerges from the eggs in about 20 hrs and L1 to L2 in about 3 days. Infective stage is reached in about 6 to 7 days of time.
- Infection of final host by ingestion of infective larvae along with herbage. Exsheathment occurs in small intestine.
- L3 penetrate the intestinal mucosa and form a nodule in which the L3 moult to become L4 in about 4 days.
- Then the L4 enters into the lumen of the intestine and pass to the colon where L4 moult to become L5 and reach maturity in 41 days.
- Eggs are strongyle type.

OESOPHAGOSTOMUM SP. **PATHOGENESIS**

- In the intestine they produce a condition known as pimply gut or knotty gut.
- Lambs and older sheep that are exposed for very first time to this parasite will not have any resistant to this parasite, hence the larvae incite practically no reaction during migration into intestinal mucosa.
- Eventually large number of adult worms emerge occur but nodule formation will not take place.
- Where as lambs and sheep which have previous exposure to this parasite (i.e., immunologically sensitized sheep), the larvae produces localized reaction during migration into the mucosa.
- During localized reaction leucocytes especially eosinophils, foreign body giant cells collect around the each larva and encapsulated by fibroblast. The larvae stay in the nodules for about three months.
- When the contents of the nodule caseate and calcify the larvae either dies or leaves the nodules.
- After leaving the nodule, larvae wander in the muscle fibre. But majority of the larvae don't return to the lumen of the intestine. In such case, large number of nodules and very few adult worms are seen.
- Nodules are suppurative in nature and may rupture the peritoneum causing peritonitis.



OESOPHAGOSTOMUM SP.
CLINICAL SIGNS

- Marked and persistent diarrhoea, faeces are dark green in color mixed with mucous and sometime with blood.
- Diarrhoeic syndrome occurs in about 6 days of infection and coincides with the time when the larvae leave the nodule.
- In chronic cases initial diarrhoea and later constipation may be seen and extreme emaciation and atrophy of muscle may occur.

OESOPHAGOSTOMUM SP.
POST MORTEM LESIONS

- Marked emaciation, complete absence of fat and intestinal walls studded with nodules. Worms are embedded in the mucosa and covered with mucus.

OESOPHAGOSTOMUM SP.
DIAGNOSIS

- Faecal examination for eggs.
- In acute cases, L4 stage may occur in the faeces

OESOPHAGOSTOMUM SP.
TREATMENT

- Fenbendazole – 7.5mg/Kg b wt.
- Thiabendazole – 44mg/Kg b wt
- Albendazole – 5 to 10mg/Kg b wt
- Piperazine compounds - 5 to 15mg/Kg b wt.
- Levamisole – 7.5mg/Kg b wt.

Supportive medication may be necessary for severe case of diarrhoea.

OESOPHAGOSTOMUM SP.
CONTROL

- Clean pasture and periodical deworming.

CHABERTIA OVINA
MORPHOLOGY

These worms occur in the colon of sheep, goats, cattle and other ruminants.

- The anterior end is curved slightly ventral and the large buccal capsule opens antero-ventrally.

- Oral aperture is surrounded by a double row of small cuticular elements representing leaf crowns.
- There is a shallow ventral cervical groove and anterior to it is a slightly inflated cephalic vesicle.
- Male bursa is well developed with a gubernaculum.

CHABERTIA OVINA

LIFE CYCLE

- Life cycle is direct and infection of the host occurs by ingestion of infective larva.
- Infective larvae go and attach to the mucosa of the upper colon and enter its wall.
- Then the larvae develop into adults.
- Eggs appear in the faeces 48-54 days after infection.

CHABERTIA OVINA

PATHOGENESIS

- The worms attach themselves firmly to the mucosa of the colon by means of their buccal capsule and draw in a plug of mucosa of the glandular layer and digest the layer by their secretions.
- The adjoining parts of the mucosa show an increased activity of goblet cells and infiltration with lymphocytes and eosinophils.
- The worms appear to move about frequently causing extensive destruction of the mucosa and thereby leading to bacterial infiltration.
- They also have a toxic action.
- In severe infections sheep lose condition and become anemic and die.

CHABERTIA OVINA

POST MORTEM LESIONS

- Worms are found attached to the mucosa of the colon, which is congested and swollen with
- transparent mucus in severe cases. Punctiform hemorrhages may be present.

CHABERTIA OVINA

DIAGNOSIS

- Faecal examination for eggs.
- Identification of larvae in faecal cultures.

TREATMENT

- Fenbendazole – 7.5mg/Kg b wt.
- Pyrantel pamoate-15 mg/kg b.wt.
- Supportive medication may be necessary for severe case of diarrhoea.

CONTROL

- Clean pasture and periodical deworming.

MODULE-34: FAMILY-SYNGAMIDAE

Learning objectives

After reading this module the learner will understand the following

- Y shaped worms in birds like peafowls.

SYNGAMUS SP. **MORPHOLOGY**

Genus	Syngamus
Species	trachea
Common name	Gape worm of poultry
Host	Fowl and turkey
Location	Trachea

Morphology

- It has cup shaped buccal capsule.
- Leaf crowns are absent. But 6 to 10 teeth occurs at the base of buccal capsule.
- The female and male are always in copulation giving “Y” shaped appearance.
- Eggs are ellipsoidal in shape, thick shelled, operculated at both end. It contains 16 celled (Yolk) stage.



Syngamus Sp.

SYNGAMUS SP. **LIFE CYCLE**

- Eggs of the worms are usually coughed up and swallowed by the birds and voided in the droppings.
- Under optimal condition, the infective stage (L3) is reached in about 3 days. L3 occur inside the egg.
- The infective stage hatch out from the egg, soon it becomes inactive. Infective stage may be ingested by transport host like earthworm, snail and slug.
- Inside the transport host the larvae are encysted and remain viable for several months. Sometimes the egg may not hatch in the environment or outside the host.
- Infection of final host occurs in 3 ways,
 - Ingestion of embryonated eggs having L3 stage.
 - Ingestion of L3 larva along with feed.
 - Ingestion of infected transport host.
- After ingestion, L3 stage penetrate the intestinal mucosa and reaches blood stream and then carried by blood to lungs in about 6 hrs.
- In the lungs, L3 stage is found in alveoli, where further two moults (L4 and L5) occurs in 3 days of infection.
- Young worms migrate to larger bronchi, where copulation takes place and reach the trachea in about 7th day of infection.
- Prepatent period is 17 to 20 days.

SYNGAMUS SP. **PATHOGENESIS**

- Heavy infection causes ecchymoses, edema and lobar pneumonia due to migration of large number of larvae.
- Adult worms are attached to tracheal wall and suck blood causing catarrhal trachitis and excess secretion of mucus results in blockage of air passage and difficulty in breathing.
- Male worms are deeply embedded into the tracheal wall causing nodular formation.

SYNGAMUS SP. **CLINICAL SIGNS**

- Young chicken are mainly affected and dyspnoea, asphyxia, shaking and tossing the head is seen.
- Coughing and extension of neck may occur. Birds open the mouth and performs gaping movements [gapes].

SYNGAMUS SP. **PM LESION**

- Carcass emaciated, anaemic and worms are found in posterior part of trachea.

SYNGAMUS SP. **DIAGNOSIS**

- Clinical signs.
- Faecal examination for egg.

SYNGAMUS SP. **TREATMENT**

- Mebendazole or Fenbendazole – 0.01% in feed for 1 to 2 weeks.

SYNGAMUS SP. **CONTROL**

- Birds should not be reared in moist localities.
- Chicken and turkey should not be kept in the same pen or ground.
- In turkey all age groups may be affected and may act as a carrier of the disease.

STEPHANURUS DENTATUS **MORPHOLOGY**

Genus	<i>Stephanurus</i>
Species	<i>dentatus</i>
Common name	Kidney worm of swine
Host	Pig
Location	Kidney, Ureter

Morphology

- Stout worms.
- Buccal capsule is cup-shaped and thick-walled with six variable cusped teeth at its base.
- Its rim bears a leaf crown of small elements and six external cuticular thickenings or epaulettes.
- Male bursa is small and its rays are short.

STEPHANURUS DENTATUS **LIFE CYCLE**

- Eggs are passed out in the urine of the host.
- At 26°C, eggs hatch after 24-36 h and larvae reach infective stage in four days.
- Infection of the host occurs per os or through skin. Earthworms may act as transport host.
- Larvae undergo ecdysis either in the wall of the stomach after oral infection or in the skin and abdominal muscles after percutaneous infection.
- Larvae then enter the liver and then reach the peritoneal cavity.
- Then they reach the perirenal tissues and perforate the walls of ureters and produce cysts.
- Migratory larvae can also go to lungs, spleen and psoas muscles.

STEPHANURUS DENTATUS **PATHOGENESIS**

- Percutaneous infection causes the formation of nodules in the skin, with oedema and enlargement of the superficial lymph glands.
- Abscess formation, extensive liver cirrhosis and multiple adhesions may occur.
- Aberrant migration of larvae may lead to lesions in the spinal cord.
- Cysts may occur in the kidney tissue.
- Ureters thickened and in chronic cases ureter may be occluded.

STEPHANURUS DENTATUS **CLINICAL SIGNS**

- Stiffness of leg due to precrural nodules.
- Posterior paralysis
- Depressed growth rate, loss of appetite and later emaciation.
- Ascites

STEPHANURUS DENTATUS **PM LESION**

- Liver is enlarged and cirrhosis may be marked.
- Perirenal tissues show hypertrophy and surface of kidneys may bear small scars of healed abscesses.
- Portal and mesenteric lymph vessels are enlarged or indurated.

STEPHANURUS DENTATUS **DIAGNOSIS**

- Finding eggs in the urine.
- Postmortem.
- Gel precipitin test.

STEPHANURUS DENTATUS **TREATMENT**

- Broad spectrum anthelmintics are preferred

STEPHANURUS DENTATUS **CONTROL**

- Good sanitation and adequate drainage of pig pens and yards is essential.
- Treatment of soil with polyborate for destroying the eggs and larvae.
- Segregation of young animals from infected ones.

MODULE-35: FAMILY-ANCYLOSTOMATIDAE

Learning objectives

After reading this module, the learner will understand the following

- Importance of cutaneous larval migrans in children and man.
- Congenital, milk borne and cutaneous mode of transmission.
- Zoonotic significance of hook worm infection in man.

ANCYLOSTOMUM SP. MORPHOLOGY

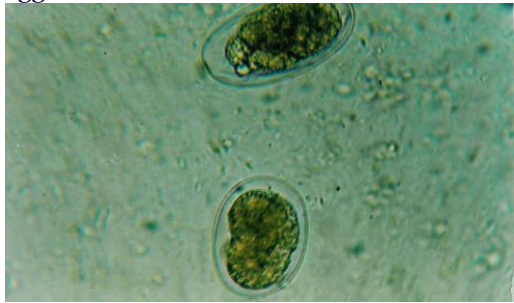
Family	Ancylostomatidae
Subfamily	Ancylostominae

Character

- The anterior end of the worm is bent in a dorsal direction. So they are called as "hook worm"
- It has well developed sub globular buccal capsule.
- Leaf crowns are absent.
- They have either teeth or cutting plates.
- In male the bursa is normally developed.
- They are voracious bloodsuckers.



Genus	Ancylostoma
Species	A. caninum , A. braziliense
Common name	Hook worms of dog.

A. caninum	A.braziliense
<i>Host:</i> dog, cat; fox	Dog, cat, and fox
<i>Location:</i> S.I	S.I
Morphology	
They are fairly rigid Grey or reddish in colour depending upon the amount of blood in the intestine	Size is smaller than the A.caninum
Buccal capsule is well developed It contains 3 pair of ventral teeth	Only 2 pairs of ventral teeth. The lateral pair is very large and inner pair is small
Eggs are oval thin shelled contains 8 cell stages 	
The ant end bend dorsally	

ANCYLOSTOMUM SP.

LIFE CYCLE

- Female lays about 16,000 eggs per day. Bionomics and development of larvae is similar to Strongyle spp.
- Desiccation is very lethal to hook worm larvae so the more suitable place for survival of hook worm larvae is slightly sandy and moist soil.
- L1 emerges from egg in about 3 days and moult to become L2 in 6 days. Under optimal (23 to 30°C) condition infective stage is reached in one-week time. Infection of final host either by oral ingestion of L3 or skin penetration by L3.
- In dogs and cats oral ingestion is very common because they have a habit of licking the grass. Skin penetration is common in human being and cattle.

In *A. caninum*, infection occurs in four ways,

- Oral ingestion
- Skin penetration.
- Prenatal.
- Transmammary infection.

Oral ingestion

- Following oral ingestion of infective larvae either directly develops to mature or the infective larvae penetrate the oral mucosa and follow the tracheal route of migration.
- Finally the infective stage reaches the intestine and develops to adult.

Skin penetration

- Following skin penetration, the infective larvae reaches the lungs via circulation and then follow the tracheal route of migration.
- Finally enter the intestine where it develops to adult.

Prenatal infection

- In older bitches following oral ingestion or skin penetration, a few larvae directly develops to adult but majority of the larvae follows somatic migration and remain dormant in the tissue until pregnancy.
- In the pregnant bitches dormant larvae is activated by the hormonal influence.
- These activated larvae enter the foetus via placental circulation. The worms do not mature until birth of the pups.
- They mature within 30 days of birth of pups and eggs can be seen in faeces. Prenatal infection is common in pups.

Transmammary infection

- The larvae passed to pups via colostrum they directly develop to adult without any migration.
- Paratenic host may also involved (rodents) infection is by ingestion of infected rodents.

ANCYLOSTOMUM SP. **PATHOGENESIS**

- Heavy infection occurs in young puppies below one year old. Smaller breeds are severely affected than larger breed.
- Worms are attached to intestinal wall (mucosa) with the help of well-developed buccal capsule and suck the blood.
- The worms frequently change the site of attachment. Hence numerous necrotic foci are seen. Since anticoagulants are found in the secretion of worms the blood continuously oozes from the site of attachment.
- In heavy infection puppies become anaemic because each worm sucks about 0.001 ml per day.

- Development of anaemia coincides with emerging L5 stage because it has developed buccal capsule (the anaemia is microcytic hypochromic).
- In dogs due to skin penetration by L3 causes dermatitis and swelling of s/c tissue.
- There will be decrease in RBC - Hb content and also haemorrhage in the lungs and pneumonia due to larval migration.

ANCYLOSTOMUM SP.

CLINICAL SIGNS

- Anaemia, it depends upon the age of the host, nutritional status and iron reserve.
- Mucous membrane will be pale, diarrhoea with bloody mucous and passing tarry red coloured faeces.
- Oedema of legs and dependant part, coat become dry and harsh in nature and stunted growth will be seen.

ANCYLOSTOMUM SP.

DIAGNOSIS

- Clinical signs.
- Faecal examination.

ANCYLOSTOMUM SP.

TREATMENT

- Bephenium bromide or iodide - 20 mg / kg b wt.
- Disophenol - 7.5 mg / kg b wt. - s/c.
- Mebendazole - 40 mg / kg b wt.
- Tetramisole - 7.5 mg / kg b wt. – s/c .
- Fenbendazole - 20 mg / kg b wt or 100 mg / kg single dose 100% effective.

ANCYLOSTOMUM SP.

CONTROL

- Periodic deworming of pups.
- Hygienic maintenance of kennels.
- Floor of kennels should be treated with common salt or sodium borate solution.
- Proper disposal of faeces.

CRISIS

Immunological phenomenon

- The infective stage (L3) can be used as vaccine after subjected to radiation treatment. Graded doses of larvae given orally or s/c will protect the animals.
- During initial infection the egg count is rising and attaining maximum in about 2 months.
- After that egg production is gradually decreases and finally the adult worms are expelled out.
- This phenomenon is similar to *self-cure* occurs in *Haemonchus contortus*.
-

Cutaneous Larval Migrans - Creeping Eruption: (CLM)

- Is a condition that occurs in unnatural hosts like human being especially children due to the skin penetration of larvae of *A.braziliense*, *A.caninum*, *A.duodenale*, *Necator americana*, *Bunostomum trigonocephalum* and *Gnathostoma spinigerum*.
- During initial exposure there will be inflammatory tract, oedema, pruritus.
- But repeated exposure leads to erythematous lesion, vesicle formation and burning sensation at the site of penetration.

MORPHOLOGY OF *BUNOSTOMUM TRIGONOCEPHALUM* AND *B.PHLEBOTOMUM*

<i>B.trigonocephalum</i>	<i>B.phlebotomum</i>
Common name: Hook worm of sheep and goat.	Hook worm of cattle
Location: S.I, especially jejunum, ileum.	S.I, especially in duodenum
Morphology: Anterior end of the worm bent in dorsal direction, well developed buccal capsule contains cutting plates.	Anterior end of the worm bent in dorsal direction, well developed buccal capsule contains cutting plates.
Dorsal cone is larger.	
Dorsal teeth is absent.	Dorsal teeth is absent.
A pair of sub ventral lancet at the base of buccal capsule is present.	A pair of sub ventral lancet at the base of buccal capsule is present.
Eggs are larger than Strongyle egg, but both the ends are blunt and contains darkly granulated yolk material.	Eggs are larger than Strongyle egg, but both the ends are blunt and contains darkly granulated yolk material.
Spicules are long and slender.	Spicules are long and slender.

LIFE CYCLE

- Eggs are passed in faeces of host. Development and bionomics of larvae are similar to strongyle spp under optimal condition infective stage is reached in 5 days.
- Infection of definitive host by skin penetration (by L3) and also by oral ingestion. Following skin penetration, larvae reaches the lungs via blood stream.
- In the lungs L3 moult to become L4, then the L4 migrate up from trachea to pharynx and finally swallowed, reach the intestine. In the intestine L4 moult to become L5.
- Prepatent period is 30 to 56 days.

BUNOSTOMUM TRIGONOCEPHALUM **PATHOGENESIS AND CLINICAL SIGNS**

- Adult worms are blood suckers causing progressive anaemia, hydraemia (watery blood), Oedema in intermandibular region (bottle jaw) and occasional diarrhoea.
- The faeces is dark in colour due to altered blood pigments.

BUNOSTOMUM TRIGONOCEPHALUM **PM LESION**

- Hydrothorax and pericarditis .

BUNOSTOMUM TRIGONOCEPHALUM **DIAGNOSIS**

- Faecal examination.

BUNOSTOMUM TRIGONOCEPHALUM **TREATMENT**

- Fenbendazole - 5 mg/kg b wt.
- Albendazole - 5 mg / kg b wt.

BUNOSTOMUM TRIGONOCEPHALUM **CONTROL**

Larvae commonly occurs on moist pasture hence avoid such a place for grazing

- Proper disposal of manure.
- Keeping the floor and bedding are dry.
- Ground around the water troughs should keep hard and dry.

AGRIOSTOMUM VRYBURGI

- Agriostomum vryburgi occurs in the small intestine of the zebu and the ox in India and sumatra.
- Males are 9.2-11 mm long and females 13.5-15.5mm.
- The buccal capsule open anterodorsally and is relatively shallow. It is followed by a very large oesophageal funnel which contains two small subventral lancets.
- Spicules are equal 0.83-0.87 mm long and accompanied by a gubernaculum.
- The vulva is posterior and eggs measure 125-195 by 60-92micro meter.
- Life cycle is direct.
- Pathogenicity: anaemia and diarrhoea in heavy infection.

MODULE-36: FAMILY-TRICHOSTRONGYLIDAE

Learning objectives

After reading this module the learner will understand the following

- Blood feeding G.I nematodes of ruminants.
- Bottle jaw condition in sheep and goats.
- Protein losing Parasitic gastroenteritis condition.
- Wire worm

TRICHOSTONGYLUS SP. **MORPHOLOGY**

- The members of the genus *Trichostrongylus* are small and slender hair like worm, pale reddish brown in colour.
- Buccal capsule, leaf crown and teeth are absent.
- A distinct excretory pore is situated at anterior end.
- Male bursa has well developed lateral lobes but dorsal lobe is indistinct.
- Spicules are pigmented either equal or unequal or dissimilar.

T. axei	T. colubriformis
Common name: Black scour worm	
Host: Sheep, goat, cattle, horse, pig and man	Sheep, goat and cattle
Location: Abomasum of sheep, goat and cattle, Stomach of horse, pig and man	Abomasum of sheep, goat and cattle
Eggs are oval, thin shelled contains segmented yolk materials	Eggs are oval, thin shelled contains segmented yolk materials.
Spicule: Dissimilar	Equal

TRICHOSTONGYLUS SP.

LIFE CYCLE



Trichostrongylus Sp.

- Eggs are passed in the faeces of host (Bionomics of larvae similar to Strongylus spp).
- Infective stage found on grass blades during early morning and late evening hours.
- Infection of final host occurs by ingestion of infective larvae along with vegetation.
- Following ingestion exsheathment occurs in the intestine.
- Infective stage penetrates into the abomasal and intestinal mucosa where L3 moults to become L4 in about 5 days and L4 moults to become L5 in about 5 days time.
- Finally, L5 returns to the lumen of the intestine and reaches maturity.

TRICHOSTONGYLUS SP.

PATHOGENESIS

- In the stomach of horse and abomasum of cattle, the worms burrow into the mucosa, causing haemorrhage and necrosis.
- The histotrophic phase (migration) of larvae causes catarrhal inflammation with necrosis and ulceration of intestinal epithelium.
- In calves, the parasite embedded in gastric mucosa causes "raised plaque" like lesions. This lesion comprises of greyish flat areas with sharply demarcated borders, looking like "ring worm lesions".

[The Trichostrongylid infection causes Moroccoleather appearance of Abomasum in ruminants](#)



Horse

- In horse there is hypertrophic gastritis with pedunculated polypoid lesion and is associated with protein losing gastropathy and hypo albuminemia in heavy infection.

Sheep

- In sheep acute inflammation of abomasal mucosa and death may occur.



TRICHOSTONGYLUS SP. **CLINICAL SIGNS**

- In sheep and goats young animals are commonly affected. Heavy infection of *T. axei* causes gastro intestinal disturbance.
- If sheep and goats acquire heavy infection in short time results in sudden death, leg weakness and unable to stand shortly before they die.
- Alternating diarrhoea and constipation may occur. Diarrhoeic faeces dark in colour, so they are popularly know as "*Black scour worms*"



TRICHOSTONGYLUS SP.

DIAGNOSIS

- Faecal Examination.

TRICHOSTONGYLUS SP.

TREATMENT

- Fenbendazole - 7.5 mg /kg. b.wt.
- Oxfendazole - 4.5 - 5 mg /kg. b.wt.
- Albendazole - 5 mg /kg. b.wt.
- Thiabendazole - 220 - 440 mg /kg. b.wt.

TRICHOSTONGYLUS SP.

CONTROL

- Periodical deworming.
- Provision of clean pasture.

HAEMONCHUS CONTORTUS

MORPHOLOGY

Species	H.contortus
Common name	Stomach worm, Barber's pole worm, wire worm of ruminants
Host	Sheep, goat and cattle
Location	Abomasum

- It is a most pathogenic parasite of sheep
- Morphology**



Haemonchus Contortus

- Worms are 10-30 mm in length.
- It has prominent cervical papillae.
- Cuticle is transversely striated.
- Longitudinal indistinct striations are also present.
- They have small buccal cavity with small dorsal lancet or teeth.
- Male bursa has well developed lateral lobe and asymmetrical dorsal lobe supported by inverted 'Y' shaped dorsal ray.
- Spicules are equal barbed at the tip.
- In female, vulva is situated at the posterior extremity and is covered by vulval knob or vulval flap.
- Ovaries of female, wound around the intestine (twisted) giving the appearance of barber's pole.

HAEMONCHUS
LIFE CYCLE

- Direct life cycle.
- L3 is reached in about 4 - 6 days.
- Infection of D/H is by ingestion of infective stage along with herbage.
- Following ingestion exsheathment occurs in rumen, then L3 migrate to abomasum and penetrate between the gastric epithelial cells.
- Where L3 moult to become L4 and L4 moult to become L5.
- Finally, L5 comes to the surface of abomasum and reach maturity.
- Prepatent period - 15 days.

HAEMONCHUS
PATHOGENESIS



- Adult male, female and L4 larvae are blood suckers.
- Each worm remove 0.05ml per day.
- Important clinical feature is anaemia in haemonchosis.
- Worms are frequently changing the site of attachment, so numerous biting wound may be seen.
- From the wound, haemorrhage occurs into the abomasum.
- Anaemia occurs in 3 stages.

HAEMONCHUS
CLINICAL SIGNS

- Hyperacute haemonchosis
- Acute haemonchosis
- Chronic haemonchosis.

Hyper acute haemonchosis

- It is uncommon, and occurs when animals are exposed to sudden massive infection.
- It causes rapidly developing severe anaemia and death due to acute blood loss.
- Faeces is dark coloured.
- Haemorrhagic gastritis occurs.

Acute haemonchosis

- It occurs when young animals are exposed to heavy infection.
- Causes anaemia accompanied by hypoproteinaemia and edema (*bottle jaw condition*).
- Death may occur within prepatent period.

Chronic haemonchosis

- It is very common and causes heavy economic loss.
- Morbidity 100% but mortality is low.
- Affected animals are weak unthrifty and emaciated.
- Hyperplastic gastritis and chronic expansion of the bone marrow will be seen.

HAEMONCHUS PM LESIONS



- Mucous membrane, skin and internal organs pale in colour.
- Blood is watery in nature.
- Liver is light brown in colour and shows fatty changes.
- The fat is replaced by gelatinous tissue.
- Abomasum contains brownish fluid ingesta in which the worms swim actively.
- Abomasal mucous membrane is swollen and covered with biting red marks.

HAEMONCHUS DIAGNOSIS

- Clinical signs
- Faecal examination



Strongyle Eggs

HAEMONCHUS TREATMENT

- Fenbendazole - 7.5mg/kg b.wt.
- Thiabendazole - 44 mg/kg b.wt.
- Ivermectin - 200 mg/kg b.wt.- s/c
- Albendazole - 5mg/kg b.wt.

SELF CURE PHENOMENON

- Is an immunological phenomenon in haemonchosis in endemic areas in which as a result of this phenomenon adult worms are expelled out.
- This phenomenon is induced by newly entering infective larvae (L3) in sensitised sheep.
- This reaction is initiated when newly entered L3 moult to become L4.
- The self cure phenomenon commonly occurs in sheep which have several previous exposure to this parasite.
- It doesn't occur in sheep which carry an initial infection (first exposure).
- It is an immediate type of hypersensitivity reaction and minimum period of 6-7 weeks is required between initial infection and challenge infection for initiating self cure.
- In self cure phenomenon, there is a transient rise in blood histamine level, complement fixing antibody and intense mucosal edema.
- This phenomenon is not species specific. A challenge infection of *H. contortus* may expel the adult worm of *trichostrongyles*.

MORPHOLOGY OF MECISTOCIRUS DIGITATUS

Species	M. digitatus
Host	Sheep, goats and buffaloes
Location	Abomasum

Morphology

- Males: 31 mm
- Female: 43 mm
- Cuticle has 30 longitudinal striations.
- Morphology: Similar to *H. contortus* except in female vulval knob or flap is absent.
- In males the bursa has pincer like lateral lobe and small, symmetrical dorsal lobe.
- Spicules are long, slender and united together for whole length.

MORPHOLOGY, LIFECYCLE AND PATHOGENESIS

Genus: **Ostertagia**, Genus: **Cooperia**, Genus: **Nematodirus**

Characters	Ostertagia	Cooperia	Nematodirus
Host	Sheep, goat and cattle	Sheep, goat and cattle	Sheep, goat and cattle
Location	Abomasum	Small intestine	Small intestine
Pathogenesis	More common in temperate and sub tropical regions. Pathogenesis similar to <i>Haemonchus contortus</i> . Morocco leather appearance of mucosa in heavy infection.	Worms penetrated into small intestine seen mostly after monsoon rain. Pathogenesis similar to <i>Trichostrongylus</i> infection.	Extensive destruction and tunnelling of intestinal mucosa with villus atrophy. Blackish green and yellowish diarrhoea.
Species	<i>O. ostertagi</i> , <i>O. circumcincta</i> , <i>O. pinnata</i>	<i>C. curticei</i> , <i>C. punctata</i> <i>C. saptulata</i>	<i>N. spathiger</i> , <i>N. battus</i> <i>N. abnormalis</i>

MODULE-37: PARASITIC GASTROENTERITIS IN RUMINANTS

Learning objectives

After reading this module the learner will understand the following

- Protein losing gastroenteropathy.
- Increased egg count during parturition.
- Bottle jaw condition in sheep and goats.

PARASITIC GASTRO ENTERITIS IN RUMINANTS

Nematodes responsible for enteritis are

- *Oesphagostomum columbianum*
- *O.radiatum*
- *Bunostomum trigonocephalum*
- *B.phelebotomum*
- *Trichostrongylus axei*
- *T.colubriformis*
- *Haemonchus contortus*
- *Mecistocirrus digitatus*
- *Trichuris ovis*
- *Strongyloides papillosus*
- *Toxocara vitulorum*
- *Cooperia sp.*
- *Ostertagia sp.*
- *Chabertia sp.*

PARASITIC GASTRO ENTERITIS IN RUMINANTS PATHOGENESIS

- Heavy blood loss due to blood sucking activity of haematophagus nematodes like *H.contortus*.
- Protein losing gastropathy in *T. axei*.
- Malabsorption of protein, fat and sugar due to villus atrophy and nodular formation in the intestine in Oesophagostomiasis.
- Malabsorption of Ca, P and Mg leads to poor bone growth in young animals.

PARASITIC GASTRO ENTERITIS IN RUMINANTS CLINICAL SIGNS

- Diarrhoea, reduced milk and meat production, anemia and weakness.

**PARASITIC GASTRO ENTERITIS IN RUMINANTS
TREATMENT**

- Fenbendazole - 7.5 mg/kg b.wt
- Thiabendazole - 44 mg/kg b.wt
- Mebendazole - 10 mg/kg b.wt
- Oxfendazole - 4.5 - 5 mg/kg b.wt
- Albendazole - 5 mg/kg b.wt
- Levamisole - 7.5 mg/kg b.wt – s/c - (immuno modulator)
- Morantel tartrate - 10 mg/kg. b.wt
- Phenothiazine - 220 - 440 /kg b.wt[very effective for Ascarids]
- Ivermectin - 50 - 200 mg/kg b.wt - s/c

**PARASITIC GASTRO ENTERITIS IN RUMINANTS
CONTROL**

- Periodical deworming.
- Pasture resting (6 months).
- Stocking rate should be optimal .
- Alternate grazing of different species-Sheep (4months)- Goats (4 months)- Cattle (4 months).
- Alternate grazing of same species – young –old animals.
- Ploughing and cultivation of land .
- Burning of contaminated pasture.

**PERIPARTURIENT RISE
(Rise in egg count)**

- Periparturient rise occurs during late pregnancy and during early lactation.
- During this period relaxation of immunity occurs due to hormonal influence like prolactin. Since the hypobiotic larvae resume its activity and all become sexually mature to produce eggs it is called as "*periparturient rise*".

MODULE-38: DICTYOCAULIDAE

Learning objectives

After reading this module the learner will understand the following

- Importance of fungi in transmission of lung worm larva.
- Verminous pneumonia.

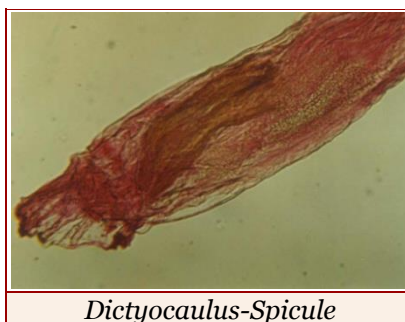
MORPHOLOGY OF *DICTYOCAULUS* SP.

- Those parasites which require I/H for completion of their life cycle are called as *biohelminths* those parasite which do not require I/H for completion of their life cycle are called as *geohelminths*.
- The members of the Dictyocaulidae live in the lungs, and are commonly called as "*lung worm*".

Genus	<i>Dictyocaulus</i>
Species	<i>D. filaria</i>
Common name	Lung worm of sheep & goat. Common in Ooty

Morphology

- The worms are milky white in colour.
- Female -3 - 8 cm, male 5 - 10 cm in length
- The buccal capsule is small and surrounded by 4 lips.
- The location of parasite is bronchi.
- In male the bursa is ill developed. The medio lateral & posterio lateral rays are fused together except at the tips.
- The spicules are short, stout, reticulate, dark brown in colour, equal and are *boot or sock shaped*.
- In female the vulva is situated just behind the middle
- The egg contains fully developed larvae
- In the lungs, the worms are entangled together giving the appearance, "*Lumps of broken threads*".



DICTYOCAULUS SP.
LIFE CYCLE

- The eggs are coughed up & swallowed.
- The L1 hatch out from the egg while they are passing through the intestine of the host and voided in the faeces.
- L1 stage can be recognised by its cuticular knob at the anterior end and brownish food granules stored in intestinal cells. Since food granules are present the L1 does not feed.
- In about 24 hours L1 moult to become L2, the cuticle of L1 encloses the L2 until the larva moult to L3.
- Sometimes the L3 may be enclosed with two cuticles. (L1 and L2).
- L2 moult to become L3 in 6 days.
- The infective larvae climb on the grass blades during early hours or cool climates.
- Infection of D/H is by ingestion of infective larvae along with herbage.
- Following ingestion of the infective larvae, it penetrates the intestinal wall and reach the lymphatic vessel.
- They are carried by the lymph to the mesenteric lymph gland where L3 moult to become L4 in 4 days. This L4 reaches the lungs via lymph and blood vessel.
- In the lungs they are arrested in alveolar capillaries then break through into air passage and become adult in the bronchi in four weeks.

DICTYOCAULUS SP.
PATHOGENESIS

- The worms in the small bronchioles cause "*parasitic catarrhal bronchitis*". The condition is called as "*verminous bronchitis*" (hoose or husk).
- The inflammatory process extends to the surrounding peribronchial tissue and the exudate passes back into bronchiole and alveoli causes *atelectasis (trapping of air in the lungs)* and *pneumonia*.
- The secondary bacterial infection leads to severe pneumonia.

DICTYOCAULUS SP.
CLINICAL SIGNS

Young animals are commonly affected, animals may cough but it is not always present.

- The mucous exudate from the nostril and dyspnoea is very common.
- Increased respiratory rate and abnormal lung sounds may be heard.

DICTYOCAULUS SP.
PM LESIONS

- Various sizes of atelectatic area is seen.
- The affected parts of bronchi contains adult worms and large amount of mucous mixed with blood.
- It is opaque in nature due to the presence of desquamated epithelial cell, leucocyte and parasite egg.

- The bronchial mucosa and peribronchial tissue is inflamed and cone shaped pneumonia may be seen.
- Compensatory emphysema and proliferation of epithelium of bronchi may occur.

DICTYOCAULUS SP.

DIAGNOSIS

- Faecal examination.
- Sometimes the larvae can be seen in sputum or nasal discharge.

DICTYOCAULUS SP.

TREATMENT

- Tetramisole - 15 mg / Kg b wt.
- Levamisole - 7.5 mg / Kg b wt. – s/c.
- Benzimidazoles and Ivermectin.

DICTYOCAULUS SP.

IMMUNOPROPHYLAXIS

- The vaccination consists of two doses of 1000 x-ray irradiated larvae each.
- The vaccination done at two months of age.
- The interval between first and second dose is one month.
- The animal must be prevented from exposure to infection until two weeks after 2nd dose.
- Reinfection is necessary after two weeks for maintaining of immunity.
- Dictol is a vaccine against cattle lungworms containing irradiated larvae.
- Difil is a vaccine against sheep lung worms containing irradiated larvae.

GENUS: PROTOSTRONGYLUS RUFESCENS

Genus: Protostrongylus rufescens

Location: Small bronchioles

Host: Sheep, goats and deer

Morphology: The male is 16-28 mm long and female is 25-35 mm long.

- The spicules are 0.26 mm long, tubular, with broad membranous expansions.
- The vulva opens near the anus.
- The eggs are unsegmented when they are laid and measure 75-120 by 45-82 µm.

Life cycle:

- The eggs develop in the lungs of the host and the first stage larvae which is passed in the faeces.
- The life cycle requires intermediate host snail species viz. *Helicella*, *Theba*, *Abida*, *Zebrina*, *Arianta*. The first stage larvae penetrate the foot of the snail and development takes place in two weeks time with two moulting takes place.
- The final host infected on ingestion of snail along with food and the larvae pass to the lungs of the host via mesenteric lymphatic glands, in which the third ecdysis takes place.

Pathogenesis:

- The worms live in the small bronchioles.
- They produce a local area inflammation, the inflammatory process spreads to the peribronchial tissues.
- The affected alveolar and bronchial epithelium is desquamated, blood vessels are occluded and an infiltration with round cells and proliferation of connective tissue takes place in the area.

Diagnosis: Finding the eggs in faeces.

Treatment: Levamisole-20mg/kg, dithiazine-100mg/kg both given subcutaneously, fenbendazole-20-80 mg/kg, albendazole-5mg/kg.

MUELLERIUS CAPILLARIS

Location: Lungs

Host: Sheep, goats and chamois

Morphology

- Male 12-14mm long and female 19-23 mm long.
- The posterior end of the male is spirally coiled and there is no bursa, but a number of papillae surround the cloacal opening.
- The spicules are 0.15mm long.
- The eggs measure about 100x20 µm.

Life cycle

- The eggs develop in the lungs of the host and the first stage larvae are passed in the faeces.
- The tail of the larvae has an undulating and dorsal spine.
- They require snail intermediate host eg: Species of the nude slugs *Limax*, *Agriolimax* and *Arion*, and snails *Helix* and *succina*.
- Infective larvae can live in snails one week even after death of snails.

Pathogenesis

- This parasite is usually not found in lambs or kids under six months of age. Thereafter the prevalence increases with age and in goats may reach 100% by three years of age.
- The worms produce greyish nodules up to 2cm in diameter.
- Smaller separate foci are formed by the eggs which are surrounded by leucocytes and epithelioid cells.
- An adenoma-like proliferation of the bronchial epithelium is seen in some cases.
- In goats it causes widespread interstitial pneumonia.

Diagnosis

- Finding the larvae in fresh faeces. Treatment: Albendazole 5 mg/kg.

DICTYOCAULUS VIVIPARUS

Host: Cattle, deer, buffalo and camel

Location: Bronchi

Morphology

- Male is 4-5.5 cm long and female is 6-8 cm long.
- The worm closely resembles the preceding species, but the medio and posterolateral rays are completely fused and the spicules are only 0.195-0.215 mm long.
- The egg measure 82-88 by 33-38 μ m.

Life cycle

- Similar to that of *D.filaria*.
- The infective larvae exsheath in the small intestine, penetrate the bowel wall and are carried to the local mesenteric lymph nodes. Here they moult to fourth stage larve and then continue their migration to the lungs via the thoracic duct and right heart.
- In massive infections third-stage larvae may be found in the lungs as early as 24 hours after infection.

Pathogenesis and clinical signs

- The major pathogenic phases are the prepatent phase in the lungs, the patent phase and the post patent phase.
- The prepatent phase is associated with blockage of many respiratory bronchioles with an eosinophilic exudate and collapse of alveoli.
- The patent phase lasting from 25 to 55 is associated with adult parasites in the bronchi and trachea.
- Onset of tachypnoea and coughing. Emphysema may develop.

- There is severe damage to the epithelium of these organs, marked exudation into the bronchi and blockage of air passages.
- In some animals a sudden exacerbation of dyspnoea is seen. This condition is often fatal and is characterized by proliferation of the alveolar epithelialization to involve entire lung lobes.

Diagnosis

- Clinical signs of bronchitis, rapid breathing and coughing.
- Demonstration of larvae in the faeces.

Treatment

- Dietycarbamazine- 22 mg/kg/day for three days.
- Tetramisole- 15 mg/kg, Levamisole 7.5 mg/kg.

Control

- Grazing management should be improved.

MODULE-39: METASTRONGYLIDAE

Learning objectives

After reading this module the learner will understand the following

- Lung worm infection in pigs.
- Piglet pneumonia.
- Role of earthworm in transmission.

METASTRONGYLUS ELONGATUS **MORPHOLOGY**

Family	Metastrongylidae
Genus	<i>Metastrongylus</i>
Species	<i>M. elongatus</i>
Common name	Lung worm of pig
Location	Bronchi
I/H	Earth worm

Morphology

- Male 28 mm, female 58 mm in length.
- The mouth is surrounded by 6 lips.
- In male the bursa is ill developed. The anterior lateral rays are swollen at the tip while mediolateral and posterior lateral rays are fused together.
- Spicules are very long slender and ending in a hook like structure.
- The tail end of female bent in a ventral direction.

METASTRONGYLUS ELONGATUS
LIFE CYCLE

- The eggs are coughed up and swallowed and voided in the faeces. They hatch immediately in the environment or swallowed by a suitable I/H.
- Even though the eggs hatch outside the host further development occurs only after ingestion by I/H. The larvae can survive upto three months in moist place.
- Further development of larvae occurs only after ingested by suitable I/H like earthworm.
- In the earthworm, the development of larvae occurs in the blood vessels, wall of oesophagus and proventriculus.
- They reach infective stage in about 10 days. The infection of D/H by ingestion of infected earthworm.
- Lifecycle and pathogenesis are similar to the Dictyocaulus species.
- Highest incidence of infection occurs in 4 to 6 months old pigs.
- Metastrongylus elongatus spreads swine influenza virus.

METASTRONGYLUS ELONGATUS
PM LESIONS

- Petechial haemorrhages in the lungs.
- Compensatory emphysema.
- The lesions are similar to T.B nodule.

METASTRONGYLUS ELONGATUS
DIAGNOSIS AND TREATMENT

Diagnosis

- Faecal examination.
- Sometimes the larvae can be seen in sputum or nasal discharge.

Treatment

- Diethylcarbamazine (DEC) - 22 mg /day - for days.
- Methyridine - 200 mg / Kg b wt. - s/c.
- Tetramisole - 15 mg / Kg b wt.
- Levamisole - 7.5 mg / Kg b wt. – s/c.

METASTRONGYLUS ELONGATUS
CONTROL

- The animal should be placed on the concrete floor.
- Provision of clean water.

MODULE-40: SPIRURIDAE

Learning objectives

After reading this module the learner will understand the following

- Importance of flies as vector for helminthic infection.
- Seasonal dermatitis in cattle and buffaloes.
- Economic loss due to damaged skin and hide.

SPIRURIDA INTRODUCTION

Character

- Nematodes have two lateral lips.
- Oesophagus is divided into two portions, the anterior muscular and posterior glandular region.
- Nematodes live in vertebrate host.
- Indirect lifecycle.
- Arthropods act as I/H.
- Eggs are oval in shape, thick shelled and contain fully developed larvae.

Super family

- Spiruroidea.
- Filaroidea.
- Physalopteroidea.
- Dracunculoidea.

MORPHOLOGY OF *HABRONEMA* SP.

Family	Spiruridae
Genus	Habronema.
Common name	"stomach worm of equine"
Species	H.muscae, H.majus (or) H.microstoma, H.megastoma (or) Draschia megastoma

H.muscae	H.microstoma	H.megastoma
Location: Stomach of equine	Stomach of equine	It doesn't occur freely, present inside the tumours
Morphology: Male : 8 – 13mm in length Female: 22mm Medium in size	Male : 22mm in length Female: 25mm Lengthy	Male : 7 – 10mm in length Female: 10 – 13mm Smaller than the other two spp.
Mouth is surrounded by two lateral lips and each trilobed. Pharynx is cylindrical.	Pharynx have dorsal and ventral tooth Lips are undivided.	Pharynx is <i>funnel shaped</i> . Head is distinct 
Male tail end - It has small caudal alae. - Spicules are unequal, left is long and slender while right is short and stout. 4 pairs of precloacal papillae and 3 number of post cloacal papillae is present. - Female ovoviviparous. Eggs, thick shelled have larvae. Hatch in I/H or D/H	- It has small caudal alae. - Spicules are unequal, left is long and slender while right is short and stout. 3 number of post cloacal papillae is present. Viviparous	- It has small caudal alae. - Spicules are unequal, left is long and slender while right is short and stout. 2 numbers of post cloacal papillae is present
I/H: House fly, Musca spp	Stable fly	Stomoxys calcitrans, Musca species

LIFE CYCLE OF *HABRONEMA MUSCAE* AND *H. MICROSTOMA*

Life cycle of *H. muscae*

- Embryonated eggs or larvae are voided in the faeces of the host (depending upon the species).
- Eggs are ingested by the larva of *Musca* species.
- In the *Musca* larva the L1 hatches out and enter the malpighian tubules and moult to become L2, by the time the larva of *Musca* become pupa.
- In the pupa L2 moult to become L3.
- In the adult fly, L3 migrate to the haemocoel cavity (body cavity) and then to the proboscis.
- Infected adult fly deposits the larvae in the lips, nostrils or existing wounds on D/H while feeding.
- Following deposition the L3 reaches stomach of equine and become adults in about 2 months.

Life cycle of *H. microstoma*

- Larva of stable fly ingests L1.
- Development in the I/H is similar to *H. muscae*.
- Normal feeding habit of *S. calcitrans* is by biting and sucking.
- They feed on moist surface like nostrils, lips or existing wounds and help in transmission.
- Sometimes, the flies fall into the water or feed, in which horse acquire infection by ingestion of larvae.

PATHOGENESIS OF *HABRONEMA MUSCAE* AND *H. MICROSTOMA*

Pathogenesis of *H. muscae*

- Worms penetrate into the stomach mucosa causing catarrhal gastritis with large amount of mucus and ulceration of stomach mucosa.

Pathogenesis of *H. megastoma*

- Adult worms penetrate the stomach mucosa causing "nodule formation" due to constant irritation.
- In severe case more number of nodules fuse to form large 'tumor' like fibrous growth.
- This tumor protrudes into the lumen of stomach leads to mechanical interference with stomach function.
- Frequently the tumors are located in the fundus region and having cavity filled with pus and cheesy material in which the worms live and also have 1 or 2 openings.

**SUMMER SORE - BURSATI - GRANULAR DERMATITIS (OR) CUTANEOUS
HABRONEMIASIS**

- It is very common in India during summer, because during summer fly activity will be more than winter.
- It is caused by larvae of Habronema especially H.megastoma.
- Lesions are commonly seen on those parts of the body liable to injury like legs, withers, canthus and sheath.
- Condition occurs as a result of deposition of infective larvae on existing wound sore, the larvae causing irritation leads to delay in wound healing and also formation of reddish brown material covered with granulation tissue.
- The lesions do not respond to ordinary treatment and often heals spontaneously during winter.
- In chronic cases, granulomatous lesions may occur. In the eye, due to deposition of larvae it causes in "*granular conjunctivitis*"(wart like).

Summer Sore



HABRONEMA SP.
TREATMENT

- Surgical removal.
- Topical application of chromic acid 2%.
- Fenbendazole 15 - 60mg/ Kg. b.wt.
- Trichlorphon 25mg/Kg. b.wt.

HABRONEMA SP.
CONTROL

- Proper disposal of manure.

MODULE-41: SPIROCERCA AND GONGYLONEMA

Learning objectives

After reading this module the learner will understand the following

- Importance of chicken and garden lizard as paratenic hosts.
- Dry cough in stray dogs due to spirocercosis.

SPIROCERCA LUPI **MORPHOLOGY**

Species	<i>Spirocerca lupi</i> .
Host	Dog, and also recorded in goat.
I/H	Coprophagous beetles (<i>Scarabeus</i> spp).
Paratenic host	Garden lizard (<i>Calotes</i> spp) and chicken.
Location	Walls of oesophagus, stomach, aorta and rarely found freely in the lumen of stomach.

Morphology

- Usually spirally coiled and pink in color when fresh.
- Male: 30 - 50mm; Female: 54 - 80mm.
- Worms are stout.
- Lips are trilobed.
- Male tail end has small lateral alae and have 5 pairs and one median precloacal papillae and two pairs of post cloacal papillae.
- Group of small papillae occurs at the tip of tail.
- Eggs are gelatin capsule shaped, thick shelled and contains fully developed larvae.
- Spicules are unequal.

SPIROCERCA LUPI **LIFE CYCLE**

- Eggs are passed in the faeces of host. They hatch only after ingestion by coprophagous beetles.
- In the beetles, larvae encyst on the tracheal tube. If the infected beetle is ingested by unsuitable host (i.e. paratenic host - like garden lizard, mice and domestic chicken) the larvae again encyst on oesophagus, mesentery and other visceral organs.
- Domestic chicken is considered to be important paratenic host in the transmission of infection to dog.
- Dog acquires infection by ingestion of either infected I/H or infected paratenic host.
- Following ingestion, larvae liberated in the stomach, then they penetrate stomach wall to reach the arteries and migrate to thoracic artery via coeliac artery in about 3 weeks of infection.

- After 3 months of infection, larvae migrate from thoracic aorta to oesophagal wall where they stimulate the granuloma development, within the granuloma they reach maturity in about 3 months.
- Prepatent period - 6 months.

SPIROCERCA LUPI **PATHOGENESIS**

- The migratory larvae causes haemorrhage, inflammation, necrosis, abscess and purulent streaks in various organ.
- Adult worm causes nodule formation in walls of oesophagus, stomach and aorta.
- In the thoracic aorta lesions and scarring are pathognomonic in *S.lupi* infection.
- The intima of aorta become rough and granular and eosinophilic granulomatous reaction forming nodule around a parasite.
- Initially the elastic tissue of aorta degenerated and replaced by collagen, later on calcification and ossification of vessel results in stenosis and rupture of vessel.
- In severe infection, the oesophageal nodule may become large, pedunculated and protrude into the lumen of oesophagus, leads to digestive disturbance and occasionally persistent vomiting and emaciation may occur.
- Secondary complication due to *S.lupi* are development of malignant tumor in oesophagus (fibrosarcoma or osteosarcoma).
- Fibrosarcoma may metastasize to lungs and other organs.
- Hypertrophic pulmonary osteoarthropathy.
- Spondylitis - due to constant irritation of periosteal tissue by parasite and blockage of intervertebral arteries.
- Pyaemic nephritis.
- Aplastic anaemia.

SPIROCERCA LUPI **CLINICAL SIGNS**

- A large oesophageal tumor, interfere with deglutition, respiration and circulation.
- Persistent vomiting and rapid loss of condition will be seen. Sometimes adult worms may occur in the vomitus.
- Haemorrhage from oesophageal region causes anaemia and haemoptysis. Rupture of the surrounding weakened oesophageal wall and escape of ingesta into the pleural cavity causes "Pleuritis".
- General weakness, emaciation and anaemia commonly occur.

SPIROCERCA LUPI **DIAGNOSIS**

- Examination of faeces and vomition.
- Contrast radiography.
- Endoscopy.

SPIROCERCA LUPI
TREATMENT

- Disophenol - one ml/5 Kg b.wt. s/c.
- Diethyl carbamazine (DEC) - 20mg/Kg b. wt. oral .
- Ivermectin 200mg /Kg b. wt. s/c.
- Albendazole

SPIROCERCA LUPI
CONTROL

- Affected dog should be isolated , their vomition and faeces should be disposed off properly.
- Dogs should be prevented from eating of paratenic host.

GONGYLONEMA PULCHRUM

Host: Sheep, goat, cattle, buffalo, pig and horse.

Location: Oesophagus.

Morphology

- Male 62mm long and female 145 mm long.
- The worm lies in zig zag fashion embedded in the mucosa.

Life cycle

- Transmitted through coprophagous beetle of the genus Aphodius and Blaps.

Pathogenesis

- Adult not pathogenic, slight chronic inflammatory reaction with hypertrophy and cornification of epithelia.
- Gongylonema verrucosum is found in rumen of sheep goat and cattle in India.

MODULE-42: THELAZIIDAE

Learning objectives

After reading this module the learner will understand the following

- Role of flies as vector in transmission.
- External eye worm in cattle and dogs.

THELAZIA SP. MORPHOLOGY

Character

- Pseudolabia is absent.
- Male tail end has both pre and post cloacal papillae.
- Spicules are unequal.

Genus	<i>Thelazia</i>
Species	<i>T. rhodesii</i> , <i>T. callipaeda</i>
Common name	Extra ocular worm

T.rhodesii	T.callipaeda
Eye worm of cattle	Eye worm of dog
Location: Conjunctival sac.	Nictitating membrane.
Morphology Male: 8 – 12mm Female: 12 – 18mm	Male: 8 – 12mm. Female: 12 – 18mm .
Transverse striation is very prominent.	It has fine transverse striations.
Male tail end 14 pairs of precloacal and 3 pairs of post cloacal papillae are present	5 pairs and 1 single precloacal papilla and 2 pairs of post cloacal papillae are present.
Female vulva is situated in the oesophageal region	Female vulva is situated in the oesophageal region.

Eggs contain fully developed larvae when laid. Immediately after laying larvae extend themselves within the egg and the shell as well. So appear as 'sheathed larvae'	Eggs contain fully developed larvae when laid. Immediately after laying larvae extend themselves within the egg and the shell as well. So appear as 'sheathed larvae' .
<i>I/H: Musca species</i>	<i>I/H: Musca species</i>

LIFE CYCLE

- Adult fly ingests first stage larvae (L1) while feeding on the eye secretions of D/H. Following ingestion.
- L1 reaches the gut of the fly, then penetrate in to the ovarian follicle, where it moults to become L2 and L2 to L3.
- These L3 reaches the mouth parts of the fly and are transferred to susceptible animals when the infected fly feed.

PATHOGENESIS

- Adult worms are found behind and in the nictitating membrane, lachrymal, naso lachrymal duct and the surface of conjunctival sac.
- Generally adult parasites are not pathogenic in adult animals.
- In some cases, they cause keratitis and ophthalmia.

CLINICAL SIGNS

- Lesions are found in one or both the eyes.
- Initially there is mild conjunctivitis and later on there will be congestion of conjunctiva and cornea.
- In severe cases, cornea becomes cloudy and marked lacrimation, the affected eye is markedly swollen and covered with exudate and pus.
- If the condition is not treated properly progressive keratitis and ulceration in the cornea leads to protrusion of anterior chamber of eye.

DIAGNOSIS

- Demonstration of adult parasite under local anesthesia.
- Examination of lachrymal secretion for presence of L1 stage.

TREATMENT

Removal of worms with fine forceps under local anesthesia

- Methyridine - 20ml s/conjunctival route.

- Tetramisole - 15mg/kg. b.wt.
- Levamisole - 5mg/kg. b. wt. oral route.

THELAZIA LACRYMALIS

Host

- Cattle; other domestic animal and occasionally man

Intermediate Host

- Muscid flies, particularly Musca, Fannia and Morellia

Location

- Ocular region especially the conjunctival sac and lachrymal duct

Morphology

- Small thin white worms 1.0-2.0 cm long
- Mouth capsule is present and cuticle has prominent striations at the anterior end

Life cycle

- L1 passed by the female worm into the lachrymal secretion is ingested by the fly intermediate host as it feeds
- L1 to L3 occurs in the ovarian follicles of the fly 15-30 days during the summer months
- L3 migrate to the mouthparts of the fly and are transferred to the final host when the fly feeds

Pathogenesis

- Active young adults causing lachrymation followed by conjunctivitis
- In heavy infection the cornea may become cloudy and ulcerated

Clinical signs

- Lachrymation, conjunctivitis and photophobia

Diagnosis

- Observation of the parasites in the conjunctival sac

Treatment and Control

- Manual removal of worms under a local anaesthetic
- Levamisole or avermectin

MODULE-43: TETRAMERIDAE

Learning objectives

After reading this module the learner will understand the following

- Mustard like worms in lizard.
- Female worms leaf like different from male.
- Fly borne poultry nematodes.

TETRAMERES MOHTEDAI **MORPHOLOGY**

Genus	<i>Tetrameres</i>
Species	<i>T.mohtedai</i>
Host	Fowl
I/H	Cockroach and Grasshopper
Location	Proventriculus and Gizzard

- Commonest nematode found among desi fowl

Morphology

- Sexual dimorphism is marked. Females are spherical, blood red in colour and deeply embedded in the glands of proventriculus and gizzard, so they look like dark object in the depth of tissue.
- Eggs contains fully developed larvae
- Male worms are slender, fili form, white in colour and cuticle usually provided with 4 rows of spine.
- They are commonly found in the lumen of proventriculus, but temporarily enter into the glands proventriculus and gizzard for copulation.

TETRAMERES MOHTEDAI **PATHOGENSIS**

- Generally, female worms suck blood, but more damages is done by migrating young worms causing irritation and inflammation leads to death of young birds.

TETRAMERES MOHTEDAI **CLINICAL SIGNS**

- Anaemia, emaciation, thickened walls of proventriculus and exfoliation of walls of proventriculus may occur.

TETRAMERES MOHTEDAI
TREATMENT

- Piperazine adipate for 3 days.

TETRAMERES MOHTEDAI
CONTROL

- Birds should be prevented from eating of I/H.
- Birds must be reared on cage system.

MODULE-44: PHYSALOPTERIDAE

Learning objectives

After reading this module the learner will understand the following.

- Blood sucking intestinal nematodes of cats.
- Prepuce like thickening in the mid of the worm and hence the species name praeputialis.

PHYSALOPTERA PRAEPUTIALIS MORPHOLOGY

Family	Physalopteridae Gnathosomatidae, Physlopteridae
Genus	<i>Physaloptera</i>
Species	<i>P.praeputialis</i>
Host	Cat, dog, and wild felidae
Location	Stomach
I/H	Flour beetles, cockroach and field cricket

Morphology

- Pharynx is absent and cuticle forms collar like projection at the anterior extremity.
- In both sexes the cuticle posteriorly extend to form a sheath or prepuce like structure, which projects beyond the caudal end of worm.
- In fertilized female, the vulva is covered by ring of brown cement material.
- Eggs are oval, containing fully developed larvae. In male, the tail end has large lateral alae and spicules are unequal.
- Left is longer than right.

PHYSALOPTERA PRAEPUTIALIS LIFE CYCLE

- Eggs are passed in the faeces of host and they are ingested by suitable I/H.
- In the I/H L1 hatches out from egg and enter into the haemocoel cavity where L1 moult to become L2 in about 11 days.
- D/H acquire infection by the ingestion of infected I/H.
- The infective stage reach stomach and becomes adult.

PHYSALOPTERA PRAEPUTIALIS
PATHOGENESIS

- Adult worms are firmly attached to the mucosa of stomach and suck the blood.
- They also feed on the mucosa of stomach wall, so the erosion of mucosa occur at the site of attachment.
- Heavy infection causes acute gastritis ,anaemia due to blood loss, anorexia and vomition may also occur.
- Faeces is dark, tarry red in colour.

PHYSALOPTERA PRAEPUTIALIS
CLINICAL SIGNS

- Weight loss, anorexia, anaemia, quarrelsome nature and shaggy fur.

PHYSALOPTERA PRAEPUTIALIS
TREATMENT

- Broad spectrum anthelmintics may be tried.

PHYSALOPTERA PRAEPUTIALIS
CONTROL

- Prevent access to I/H.

MODULE-45: GNATHOSOMATIDAE

Learning objectives

After reading this module the learner will understand the following

- Zoonotic significance in humans as accidental eye worm.
- Spiny headed worms of the cats.

GANATHOSTOMA SPINIGERUM MORPHOLOGY

Genus	<i>Ganathostoma</i>
Species	<i>G. spinigerum</i>
Host	Cat, dog, and erratic parasite of man
I/H	Ist- Cyclops (copepod) and IInd Fresh water fish
Location	Stomach

Morphology

- The mouth is surrounded by two large lips and are trilobed. The cuticle forms tooth like ridges on the medial surface of lips (interlocked).
- They have large head bulb consists of four submedian cavity or "ballonet" contains fluid.
- The head bulb is armed with 6 to 11 rows of transverse spines.
- Anterior 2/3 of the body also armed with large flat cuticular spine which is denticulate at the posterior margin.
- In male, the caudal and ventral surface is covered with small spine and armed with 4 pairs of pedunculated and several papillae
- Eggs are oval, thick shelled, greenish ornamented and have small cap at the anterior pole.
- Egg contains one cell or morula stage.

GANATHOSTOMA SPINIGERUM LIFE CYCLE

- Eggs are passed in the faeces of host, they hatch in the water and liberate L1.
- These L1 swim in water for few days and then should be ingested by the cyclops.
- In the haemocoel cavity of cyclops L1 develops to L2.
- When infected cyclops is ingested by fresh water fish, the L2 moult to become L3 in the body cavity of fish.
- The L3 encyst on various muscles of fish.
- D/H acquire infection by ingestion of infected fresh water fish.
- Man acquires infection by ingestion of under cooked infected fish.

GANATHOSTOMA SPINIGERUM
PATHOGENESIS

- Young worms migrate through liver causing destruction of liver tissue leaving characteristic "yellow mosaic marking" on the surface and the tracts are filled with necrotic materials.
- Adult worms penetrate into the stomach mucosa producing cavity filled with sanguino purulent fluid and each contains nine worms.
- Later on the cavity develops into thick cyst or tumour connected to lumen of stomach by small canal which may causes peritonitis.

GANATHOSTOMA SPINIGERUM
DIAGNOSIS

- Examination of faeces for egg, but eggs are not always present in faeces.

GANATHOSTOMA SPINIGERUM
TREATMENT

- Benzimidazole compounds.
- Gnathostomiasis in human beings causes creeping eruption.

MODULE-46: FILARIDAE

Learning objectives

After reading this module the learner will understand the following

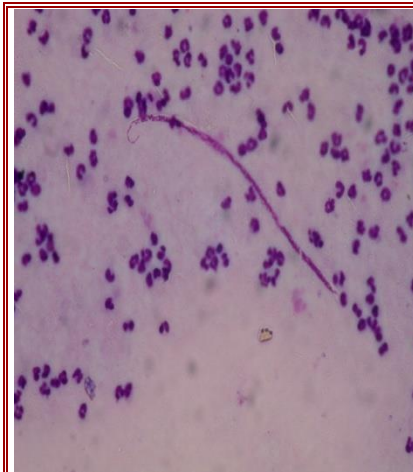
- Vector borne nematodes of ruminants and other animals
- Microfilaria-the infective stage present in the circulatory system.
- Filarid dermatitis cause economic loss.

DIROFILARIA IMMITIS **MORPHOLOGY**

Family	Filaridae
Genus	<i>Dirofilaria</i>
Species	<i>D.immitis</i>
Location	Right ventricle and pulmonary artery
I/H	Mosquitoes (Culex, Aedes & Anopheles)
Common name	Heart worm of dog.

Morphology

- Worms are long, thin and white in colour.
- Mouth is very small and lips are absent.
- Male is smaller than the female.
- Posterior end of male is spirally coiled.
- The spicules are unequal and the left spicule is blunt.
- In female vulva is situated at the anterior extremity. They are larviparous or viviparous.
- Larvae of filarid is known as "Microfilaria" which are found in the blood of the host.



Dirofilaria Immitis

DIROFILARIA IMMITIS
LIFE CYCLE

- Adult mosquitoes ingest the microfilaria along with blood meal while feeding on the D/H.
- Ingested microfilaria are first found in the stomach of mosquito then migrate to the malpighian tubule, where the further development occurs.
- In about 4 days it will develop into a sausage form (i.e., 2nd stage larvae). The sausage form become "elongate sausage form" in nine days.
- It feeds on the cells of malpighian tubule and enters into the haemocoel cavity.
- From there, they migrate through the thorax and reach the mouthparts, where it becomes the infective stage.
- The dogs get infected when the infected mosquitoes feed on them. (by inoculation).

DIROFILARIA IMMITIS
PATHOGENESIS

- Mild infection causes no clinical signs . where as heavy infection causes circulatory distress due to mechanical interference.
- Presence of large number of worms in the heart will interfere with the function of the cardiac valves.
- Heartworm primarily causes pulmonary circulatory disturbances leads to pulmonary hypertension due to narrowing of pulmonary peripheral artery.
- Compensatory hypertrophy of right ventricle results in "*congestive heart failure*", *liver failure syndrome and peripheral oedema*.

DIROFILARIA IMMITIS
CLINICAL SIGNS

- Deep soft cough, reduced stamina, haemoptysis, dark brown colour faeces containing "Hb" and bilirubin.
- Anorexia and anemia may also be seen.

DIROFILARIA IMMITIS
DIAGNOSIS

- Blood smear examination- stained, Wet film examination.
- *Knotts method*: One ml of blood + 9 ml of 2% formalin, centrifuge for 5 min. then the sediment is to be stained with methylene blue.
- X ray.
- Serological test.
- ELISA.
- IFAT.

TREATMENT
(Against microfilaria)

- DEC: 5.5mg/Kg b wt. for 2 months.
- Mebendazole: 80mg/Kg b wt. for 30 days.
- Levamisole: 10mg/Kg b wt.- 15 to 20 days.
- Ivermectin: 200mg/Kg b wt.- s/c.
- Against adults ?.....

CONTROL
(Against microfilaria)

- Control of mosquitoes.

MORPHOLOGY OF *STEPHANOFILARIA ASSAMENSIS*

Species	S.assamensis
I/H	Musca conducens

- It causes "*Hump sore*" in cattle. It is a "*chronic dermatitis*" *Verminous dermatitis/Cascado*.
- In Assam and W.Bengal about 90% of cattle are affected and 59% in Andaman.

Morphology

- Mouth is surrounded by cuticular rim which is denticulated.
- Spicule is unequal.



STEPHANOFILARIA PATHOGENESIS

- Poor condition and high rainfalls are the predisposing factors for this condition. Following infection, the lesions initially appear as large number of small papule. Later on, they join together and form a large lesion which is covered with thick crust.
- There will be inflammatory changes and pruritus. This leads to constant rubbing of the body against hard objects and the condition is aggravated by self mutilation.
- Hyperkeratosis, acanthosis and alopecia may also occur. The lesions are rich in blood and lymph and readily squeezed. The lesion extends outward with the central thick dry crust.
- The lesion may reach upto 25mm in dia and resolve during the dry cool season but reappear during rainy season.

STEPHANOFILARIA DIAGNOSIS

Examination of deep skin scrapings

- The scraping should be made only after the crust is formed. (Microfilaria will be found)

STEPHANOFILARIA TREATMENT

- Trichlorphon ointment - 6 to 8% in petroleum jelly or castor oil.

MORPHOLOGY OF PARAFILARIA BOVICOLA

Species	P.bovicola
Host	Cattle and buffalo
Location	Inter muscular and S/c
I/H	Musca species, (Musca lusoria)

- It causes haemorrhagic nodules, on the skin of cattle and buffaloes. The condition is known as "*Summer bleeding*".

Morphology

- The anterior end of worm bears 13 rows of cuticular elevation or thickening.
- Remaining portion of body is transversely striated. In female, vulva is situated at anterior end.
- Musca gets infected, when feeds on lacrimal secretion or existing wound.
- The larvae enter into the host via lacrimal duct or wound.

PARAFILARIA PATHOGENESIS

- S/C nodules are seen mainly on the shoulder region and on the dorsal aspect of (loins, withers and neck) the body.
- The nodules are 12 to 15 mm in dia and 5 to 7 mm in height.
- The gravid female moves to dermis for oviposition. (egg laying).
- During this period, the nodules become enlarged and painful haemorrhage may occur.
- There will be marked edema and infiltration of lymphocytes and neutrophils.

PARAFILARIA TREATMENT

- Nitroxylin 20mg/Kg b wt.s/c.
- Fenbendazole and Albendazole can be used.

DIROFILARIA REPENS

Host

- Dog and cat

Location

- Adults found in subcutaneous tissues and the microfilariae in the blood and lymph

Pathogenesis

- Being transmitted thru mosquitoes, these worms are frequently found in the anterior chamber of eye in human beings as accidental parasites causing difficulties in vision.

DIPETALONEMA RECONDITUM

- *Dipetalonema reconditum* is a parasite rarely causes significant disease.
- Its importance lies in the fact that its microfilariae can be easily confused with those of *Dirofilaria immitis*.
- *D. reconditum* lives in the body cavity and subcutaneous (just below the skin) tissues of dogs.

Life cycle

- The life cycle of *D. reconditum* includes an intermediate host such as flea, tick, or louse instead of the mosquito.

Morphology

- The adult worms which are ½ to 1-inch long may be found on necropsy or as an incidental finding during surgery for some other reason.
- The microfilaria can be found in the bloodstream. Based on their size and shape, your veterinarian should be able to distinguish them from heartworm microfilariae under the microscope.
- The presence of a *D. reconditum* infection should not affect the common serologic test used to diagnose heartworm.

Pathogenesis and control

- Usually no treatment of a *D. reconditum* infestation is required, however, a single treatment of ivermectin (0.11 mg/lb) will eliminate the microfilaria.
- It is unknown if any antihelmintics will kill the adult worms. Since it is not considered a pathogen, few efforts have been made toward its prevention or control. Controlling the flea, tick, and lice populations would be beneficial.

MODULE-47: SETARIDAE

Learning objectives

After reading this module the learner will understand the following

- Mosquito borne filarid nematodes commonly seen in cattle and buffaloes.
- Internal eye worm of cattle.
- Lumbar paralysis in accidental host like sheep.

MORPHOLOGY OF *SETARIA DIGITATA*

Genus	<i>Setaria</i>
Species	<i>S. digitata</i>
Host	Cattle and buffalo
Location	Peritoneal cavity and urinary bladder
I/H	Mosquitoes - <i>Culex</i> , <i>Aedes</i> and <i>Anopheles</i>

Morphology

- Worms are long and milky white in colour. Mouth is surrounded by chitinous ring, called as "*peribuccal ring*".
- It has lateral, ventral and dorsal prominence. Body of the worm tapers towards the tail end and the posterior end is spirally coiled.
- Tail end of both sex has small lateral appendages.
- Female tail end has a small knob like conical projection and may be armed with small spine.



Setaria Sp.

SETARIA DIGITATA
PATHOGENSIS

- Adult parasite are not pathogenic, but in some cases they cause peritonitis and lesions in the urinary bladder.
- Accidental setaria infection can occur in cattle in the Anterior chamber of the eyes of Horses with immature or adult *Setaria digitata* or *S.labiopatapillosa*

[Anterior chamber of eye in a Horse clinically affected with Setaria infection](#)



SETARIA DIGITATA **ENZOOTIC CEREBROSPINAL NEMATODIASIS OR KUMRI OR LUMBAR PARALYSIS**

It is a condition caused by erratic migration of immature stage of *S. digitata* in the unnatural host like horses, sheep and goat.

- The condition is more prevalent in summer since the mosquito activity is more.
- The affected animals suffer from acute focal encephalomyelomalacia.
- The migrating worms produce tracks and degeneration of tissues in CNS

SETARIA DIGITATA **CLINICAL SIGNS**

- It depends upon the site of lesion.
- Muscular weakness, ataxia, paralysis of hind limbs and sometimes blindness may also occur.
- Microfilaria of setaria in bovines also cause a syndrome consisting of systemic disease in affected animals with variety of symptoms

SETARIA DIGITATA **TREATMENT**

- DEC - 40mg/Kg b wt .

ONCHOCERCA GIBSONI

Host

- Cattle

Location

- Brisket and the external surface of hind-limbs

Morphology

- Male 30-53 mm long and the female 140-190 mm long
- Tail of the male is curved ventrad; it bears small lateral alae and six to nine papillae on either side
- The spicules 0.14-0.22 mm and 0.047-0.094 mm respectively

Intermediate host

- *Culicoides pungenis*

Life cycle

- The life cycle of the onchocerca spp. is typically filarioid, with exception that the microfilariae occur in the tissue spaces of the skin, rather than in the peripheral bloodstream

Diagnosis

- Depends on the finding of microfilariae in skin biopsy samples

Treatment

- Diethylcarbamazine

Control

- Use of microfilaricides
- Reduce the numbers of infected flies

ONCHOCERCA VOLVULUS

Host: Human being rarely in animals

Intermediate host: Black fly (*Simulium*).

Morphology

- Adults of *Onchocerca volvulus* are long and slender, have a smooth cuticle, and have blunt anterior and posterior ends. There are no lips or buccal capsule, and the mouth is surrounded by 2 circles made up of four papillae each.
- The esophagus doesn't appear to be divided. Males range in length from 19-42 cm and range in width from 130-210 micrometers.
- Lacking alae, their tails are curled ventrally and bear 6 or 8 pairs of postanal and 4 pairs of adanal papillae. Females are larger than males, ranging in length from 33.5-50 cm and in width from 270-400 micrometers.
- Their vulva is directly behind the posterior end of the esophagus. The microfilariae released by adult females are 250-300 micrometers long, are unsheathed, and have sharply pointed and curved tails.

Life cycle

- *O. volvulus* begins when a parasitised female black fly of the genus *Simulium* takes a blood meal. The microfilariae form of the parasite found in the dermis of the host is ingested by the black fly.
- The microfilaria then penetrates the gut and migrates to thoracic flight muscles of the black fly, entering its first larval phase (J₁). After maturing into J₂, the second larval phase, it migrates to

the proboscis where it can be found in the saliva . Saliva containing stage three (J_{3s}) *O. volvulus* larvae passes into the blood of the host.

Treatment and control

- Ivermectin
- Control of black fly formation
- Affected people treated by Ivermectin

ONCHOCERCA ARMILLATA

- Cattle are the most susceptible species. The diagnosis of the infection by skin snip examination and some also at post-mortem examination.
- Infection is more common between nine months and eight years. Prevalence rate and number of microfilariae per gram of skin were higher in male than in female cattle.
- Severe pathological changes were seen in the thoracic aorta, brachiocephalic, costocervical and brachial arteries and posteriorly in the abdominal aorta to its bifurcation into the iliac arteries.

MODULE-48: DRACUNCULIDAE

Learning objectives

After reading this module the learner will understand the following

- One of the Ancient nematode recorded in man and animals.
- Zoonotic nematodes transmitted through poorly drained water source.
- Cyclops is a intermediate host.

MORPHOLOGY OF *DRACUNCULUS MEDINENSIS*

- It is a oldest nematode parasite of man in India and Pakistan . It is also seen in dogs, cattle and horse

Family	Dracunculidae
Genus	<i>Dracunculus</i>
Species	<i>D.medinensis</i>
Location	S/C connective tissue also seen in dogs, cattle and horse.
Common name	Guinea worm, Medina worm, serpent worm and Dragon worm.

Morphology

- Male worms are 10 - 30mm in length.
- Female worms are 300 - 400cm length and vulva is absent.
- The gravid female migrates from body cavity to s/c connective tissue, where it produces papule or blister mainly on legs.
- Later on this papule develops into ulcer around the anterior extremity of worm.
- When the lesion comes in contact with water, the uterus of worm comes out through the mouth of worm and rupture, discharges large number of larvae that swim in water.
- The larvae ingested by I/H Cyclops in which they develop to infective stage. D/H acquire infection by ingestion of infected I/H along with water.

DRACUNCULUS MEDINENSIS LIFE CYCLE

- These larvae have to develop in a species of cyclops to become infective for the final host
- Infection of the latter takes place through drinking water which contains the infected cyclops and the worms develop to maturity in about one year

DRACUNCULUS MEDINENSIS
PATHOGENESIS

- The metabolic wastes produced by female worms migrating in the skin elicit a violent allergic reaction to the host's system.
- Allergic reactions produce rashes, nausea, diarrhea, dizziness, and localized edema.
- Rupture of the blister, allergic reactions subside but skin ulcers form, through which the worm can protrude.
- Healing of the worm will only be completed if the worms are totally removed or expelled.

DRACUNCULUS MEDINENSIS
CLINICAL SIGNS

- Urticaria, itching and rise of temperature in affected patients. In some cases, the adult female worm may be seen as a stumb in the frontal view of the legs.

DRACUNCULUS MEDINENSIS
TREATMENT

- Best method of treatment is manual removal of worm. During manual removal, worm should not be broken. In this method the worm is tied with small stick and gradually rolled up for few days or Weeks with great care.

DRACUNCULUS MEDINENSIS
CONTROL

- Prevention of drinking water from infected areas.
- Use boiled water for drinking purpose.

MODULE-49: ENOPLIDA

Learning objectives

After reading this module the learner will understand the following

- Directly transmitted nematodes of man and animals.
- Auto infection in trichinellosis infection.
- whip like worm with barrel shaped eggs seen in mammals and birds.

ENOPLIDA INTRODUCTION

Character

- Small - medium in size.
- The anterior portion is long filiform while the posterior portion is short and thick.
- Male has single spicule or absent.
- In female, vulva is situated at the junction of anterior and posterior portion.
- Female are ovo-viviparous or oviparous.

There are 3 families

- Trichinellidae.
- Trichuridae .
- Capillaridae .

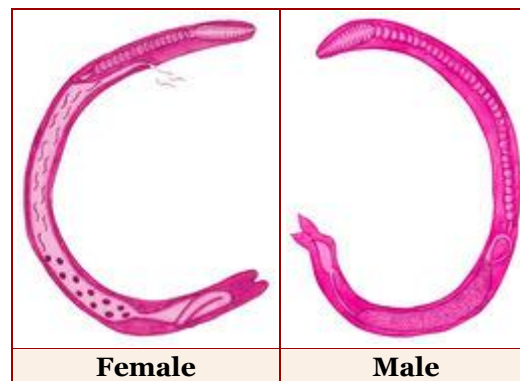
MORPHOLOGY OF *TRICHINELLA SPIRALIS*

Genus	<i>Trichinella</i>
Species	<i>T.spiralis</i>
Host	Pig, rat, man and also seen in dog, cat
Location	Adult worms are seen in the small intestine while larvae are seen in striated muscles like diaphragm, tongue, intercostal and mandibular muscle
Common name	Garbage worm and Trichina worm

- The worm causes - *Trichinellosis* or "*Raw pork disease in man*".

Morphology

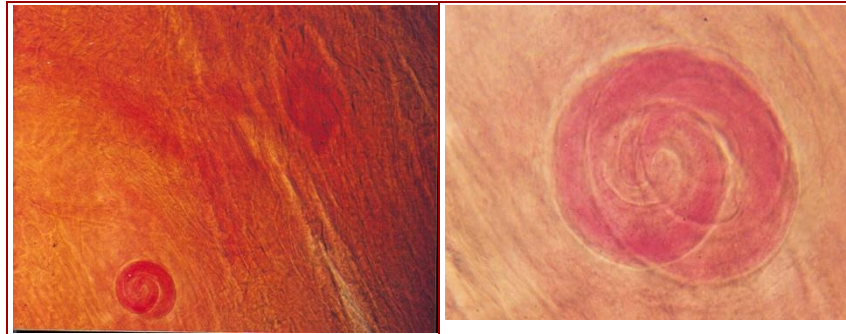
- Male worm is about 1.5mm. Female is about 3 - 4mm. In both the sexes anterior end is blunt and Oesophagus is very long (about 1/3 of total body length).
- In male, spicule is absent, in female vulva is situated near the middle of oesophageal region.
- Male tail end has a pair of lateral flap on either side of cloacal opening and 2 pairs of papillae occur just behind the lateral flap.
- Female is ovo-viviparous. But egg hatch in the uterus itself .



TRICHINELLIDAE **LIFE CYCLE**

- Man acquires infection by ingestion of infected meat containing viable cyst. Following ingestion, the cyst reach the stomach.
- In the stomach, larvae escape from the cyst in about 5 hrs of infection. Liberated larvae penetrate the stomach mucosa, where it moult to become L1 - L2 and L2 - L3.
- L3 reaches intestine and moult to become L4. Sexual differentiation and copulation occurs in the intestine.
- After copulation, the male will die, while the female enter into the intestinal mucosa, where it produces large number of larvae.
- The newborn larvae enter into the lymphatic vessel and then migrate to left superior vena cava via thoracic duct and finally enters into systemic circulation.
- Through blood, it is distributed to different parts of body.
- Further development occurs in various organs like tongue, diaphragm etc. It is also found in kidney and liver.
- The larvae enter the striated muscle, become encapsulated by muscle fibre.
- These parasitised muscle cell undergoes modulation or redifferentiation in the structure is called as "Nurse cell".
- Function of nurse cell is larval nutrition and handling of waste product. The larvae occurs in lemon shaped cyst in a coiled position.

***Trichinella* Larva in Musle cyst**



TRICHINELLIDAE
EPIDEMIOLOGY AND PATHOGENESIS

Epidemiology of *T. spiralis*

- There are two important cycles, which play a major role in the transmission of infection to the man.

Sylvatic cycle: (Wild animal)

- In which carnivores like jackal and wild boars maintain the infection by cannibalism. Man may be infected by ingestion of wild animals meat.

Synanthropic zoonotic cycle: (Domestic animal)

- Domestic animals are getting infection from wild animals due to close contact.

Pathogenesis

- In domestic animals the infection cause no clinical signs, but in man the infection causes irritation and marked enteritis. The larva in the muscle fibre causing acute myositis, myocarditis, ascitis, periorbital oedema, eosinophilia and fever. In heavy infection death may occur due to paralysis of respiratory muscle.

TRICHINELLIDAE

DIAGNOSIS

- Based on the clinical signs.
- During meat inspection, the encysted larvae seen as a white spot can be visible to naked eye.
- For routine examination in meat industry small piece of muscle pressed between the slide, then viewed under trichinoscope.
- Digest the muscle piece in acid pepsin then put in the Baermann's apparatus for larval separation.

TRICHINELLIDAE

TREATMENT

Treatment

- Benzimidazole drugs.

Control

- Cooked garbage should be provided to pigs.
- Avoid eating of under cooked pork.
- Freezing of pork at 25 ° C for 10 - 20 days, in which all larvae are killed.

MORPHOLOGY OF TRICHURIS OVIS

Genus	Trichuris
Species	T.ovis
Host	Goat, sheep, cattle and ruminants
Location	Caecum and colon
Common name	Whip worm

Morphology

- Anterior portion of the worm is long and slender, while posterior part is short and stout.
- Male tail end is spirally coiled and has single spicule enclosed in a spiny sheath.
- Female is oviparous.
- Eggs are brown in colour, barrel shaped, transparent and has plug at both ends.

TRICHURIS OYIS **LIFE CYCLE**

- Unembryonated egg passed in faeces of the host .Under favourable condition the infective stage(L1) is reached in about a weeks (infective stage is L1).
- The D/H acquires infection by ingestion of embryonated egg containing L1.
- The eggs are hatched in the small intestine, the liberated L1 penetrate the intestinal mucosa, where all the moulting occurs .
- The adult worms reenter into the lumen and moves to the caecum. In the caecum the filamentous anterior end embedded in the caecal mucosa.

TRICHURIS OYIS **PATHOGENESIS**

- Generally not pathogenic. But heavy infection is causative for *diphtheretic inflammation*.
- Mainly occurs due to continuous moving of worm for attachment.

TRICHURIS OYIS **DIAGNOSIS**

- Faecal examination for the presence of eggs.

TRICHURIS OYIS **TREATMENT**

- Levamisole - 7.5mg/Kg.b.wt
- Fenbendazole - 5mg/Kg. b.wt
- Methyridine - 200mg/Kg. b.wt
- Thiabendazole- 44mg/ Kg b.wt (very effective)

MORPHOLOGY OF CAPILLARIA ANNULATA

Genus	Capillaria
Species	C.annulata
Host	Fowl and turkey
Location	Crop and Proventriculus
I/H	Earth worm

Morphology

- They are very small, thin and hair like worm.
- At the anterior end of worm ,the cuticle forms collar swelling behind the head.
- Capillaria egg is similar to T.ovis egg, but it is colorless , more barrel and have lateral striations.



CAPILLARIA ANNULATA
LIFE CYCLE

- Earthworm act as I/H.
- D/H acquires infection by ingestion of infected earthworm.



CAPILLARIA ANNULATA
PATHOGENESIS

- Mild infection causes inflammation and thickening of crop but heavy inflammation results in marked thickening of crop and oesophagus. Catarrhal or croupous inflammation may occur.
- The birds become emaciated and weak.
- Sloughing of mucosa may occur.

CAPILLARIA ANNULATA
DIAGNOSIS

- Feecal and PM examination.



Capillaria Sp.

CAPILLARIA ANNULATA
TREATMENT

- Coumaphous 0.08 % in feed for 10 – 14 days.

MODULE -50: DIOCTOPHYMIDAE

MORPHOLOGY OF *DIOCTOPHYMA RENALE*

Genus	<i>Dioctophyma</i>
Species	<i>renale</i>
Common name	Giant Kidney worm
Host	Dog, Fox and other carnivores
Location	Kidney

Morphology

- This is the largest nematode known.
- Blood red coloured worms.
- Male measures upto 35 cm by 3 to 4 mm and female upto 103 cm by 5 to 12 mm.
- Spicule is 5 to 6 mm long.

LIFE CYCLE

- Eggs are passed out in the urine of the host.
- Eggs are swallowed by intermediate host *Lumbriculus variegatus*, a free living oligochaete annelid.
- The definitive host may become infected by ingesting the infective larvae in annelids or by ingestion of paratenic hosts (Pike, Frogs) which are infected by in the ingestion of infected annelids.
- In final host infective larvae penetrate the bowel wall, developing body cavity and then penetrate the kidney.

PATHOGENESIS

- The worms enter the pelvis of kidney and destroy the parenchyma.
- The worms block the ureter leading to uraemia.
- In abdominal cavity, encapsulated worms cause chronic peritonitis with adhesions.

CLINICAL SIGNS

- Infected animals grow thin and shows signs of kidney trouble together with nervous signs.

DIAGNOSIS

- Finding eggs in the urine.

TREATMENT

- Surgical removal.

MODULE-51: PHYLUM-ACANTHOCEPHALA

Learning objectives

After reading this module the learner will understand the following

- Atypical helminths seen in pigs, man and wide variety of birds.
- Life cycle is complex similar to cestode and nematode.
- Tri-layered resistant eggs.

ACANTHOCEPHALA INTRODUCTION

Common name: *Thorny headed worm.*

Character

- At the anterior end of the worm there is a proboscis which is armed with recurved hooks.
- The proboscis kept or lie in a sac is called as proboscis sac.
- On either side of proboscis sac a pair of elongated hollow organ occur –known as lemnisci. It contains proboscis fluid.
- Function of lemnisci is for in attachment of worms.
- 4 pairs of suspensory ligament which provide attachment to the genital organ.
- Digestive and excretory system is absent. Nutrition – saprophytic.

MORPHOLOGY OF MACRACANTHORYNCHUS HIRUDINACEUS

Genus	<i>Macracanthorynchus</i>
Species	<i>M.hirudinaceus</i>
Host	Pig
Location	S.I and also occurs in stomach
I/H	Dung beetle

Morphology

- The worms are more or less curved.
- The proboscis is small and armed with 6 rows of recurved hooks.
- The cuticle is transversely wrinkled.
- Female – 10 cm; Male – 35cm.
- Tail end of male is coiled and has 2 testes.

- In the female the ovary discharge the ova into the body cavity, where fertilization occurs. After fertilization the embryo develop and covered with 3 to 4 layer. The female has special organ called uterine bell. This uterine bell swallows the immature egg through its anterior opening.
- After maturation of egg they will be passed to vagina. The eggs contain larva called “**ACANTHOR**”. It has hooks at anterior end. The eggs are covered with 3 layers and are passed in the faeces of the host.

MACRACANTHORYNCHUS HIRUDINACEUS

LIFE CYCLE

- Eggs contain acanthor.
- Dung beetles ingest the eggs which develop into infective stage called *cystacanth* [like cestode - cysticercoid].
- The pigs acquire infection by ingesting beetles.

MACRACANTHORYNCHUS HIRUDINACEUS

PATHOGENESIS

- The worms are attached to the mucosa of the small intestine causes inflammation and also provokes granuloma at the site of attachment.
- Sometimes the worm penetrate the intestinal wall causing peritonitis.

MACRACANTHORYNCHUS HIRUDINACEUS

CLINICAL SIGNS

- Weight loss.

MACRACANTHORYNCHUS HIRUDINACEUS

DIAGNOSIS

- Faecal examination for the presence of egg.

MACRACANTHORYNCHUS HIRUDINACEUS

TREATMENT

- Broad spectrum of anthelmintics

MODULE-52: MODE OF ACTION OF ANTHELMINTICS

Learning objectives

After reading this module the learner will understand the following

- Groups of anthelmintics currently used.
- The use and abuse of anthelmintics.
- Dosage and presentation of anthelmintics.
- How the anthelmintics work within the host.
- International regulations on control of different helminthic diseases.

I. BENZIMIDAZOLE COMPOUNDS

- There are 5 groups of anthelmintics which have different modes of action.
- *Benzimidazoles compounds* act by inhibiting the fumarate-reductase enzyme system and also binding with tubulin thus preventing polymerisation of tubulin to form microtubules in the intestinal cells of nematodes or in the cuticle of cestodes causing reduced uptake of glucose thereby depriving the energy to the parasite.
- Both tubulin binding and inhibition of fumarate - reductase results in the parasite being deprived of energy.
- Tubulin binding anthelmintics
 - Mebendazole
 - Thiabendazole
 - Parbendazole
 - Fenbendazole
 - Albendazole
 - Oxfendazole
 - Febantel

II. IMIDOTHIAZOLE GROUP

- *II. Imidothiozole group* : This group acts as a nerve ganglion stimulant and also depolarising the neuromuscular system causing rapid muscle reversible paralysis.
- Ganglion binding anthelmintics include
 - Levamisole.
 - Morantel tartrate.
 - Tetramisole .

III. SYSTEMIC ORGANOPHOSPHATE

- **Systemic organophosphate:** The organophosphates acts by inhibiting acetylcholinesterase resulting in a continual stimulation of nerve ending or muscle because the transmitter Acetylcholine is not degraded.
- Acetylcholinesterase Antagonist
 - Dichlorovos
 - Trichlorphon
 - Naphthalphos

IV. SUBSTITUTED PHENOLS AND SALICYLANILIDES

- **Substituted phenols and Salicylanilides:** These compounds uncouple the mitochondrial reactions involved in electron transport and associated phosphorylation. These compounds are effective against cestodes and trematodes but not nematodes.
- Uncoupling oxidative phosphorylate anthelmintics
 - Oxyclozanide
 - Bromsalans
 - Nitroxylin
 - Niclosamide
 - Closantel
 - Rafoxanide

V. AVERMECTIN

- **Avermectin:** This group to potentiate the release and binding of gamma aminobutyric acid (GABA) in nerve synapses. Thus blocking GABA mediated transmitters of nerve signals results in paralysis of the parasite.
- GABA antagonist
 - Ivermectin
 - Doramectin

INTERNATIONAL REGULATIONS FOR CONTROL OF HELMINTHIC DISEASES

As the control of helminthic diseases of animals is found to be more beneficial and effective for controlling helminths of animals and man, the following international bodies are involved in putting up a co-ordinated effort for control of helminthic diseases. They are

- Office International des Epizooties (OIE)
- Food and Agricultural Organisation (FAO)
- World Health Organisation (WHO)
- International Laboratory for Research on Animal Diseases (ILRAD)
- Biotechnology and Biological Research council (BBSRC)
- Indian Council for Agricultural Research (ICAR)

Sustainable control of helminthic infection is achieved through

- Restriction of movement of infected live animals, biologicals or infectious agents
- Funding for development of vaccines against helminthic diseases through National and International funding agencies
- International collaboration on emerging helminthic infections
- Control of vectors and intermediate hosts through chemical and non chemical methods
- Development of novel control strategies
- Formulation of Regional Deworming calendar
- Awareness on helminthic infection to field veterinarians and farmers
- National level Anthelmintic drug regimen system
- Awareness on control of Anthelmintic resistance
- Market regulation on abuse of anthelmintic drugs
- International assistance for development of new drug molecules
- Development of forecasting model of helminthic diseases