VETERINARY PARASITOLOGY

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Vet Study

Lecture 1

What is Veterinary Parasitology ?

Veterinary Parasitology is the science that deals with the parasites of domestic animals. More specifically, it deals with the interactions between a host and the population of parasites that are found on or in that host. Parasitology is a multidisciplinary para-clinical subject which comprises the field of taxonomy, morphology, biochemistry, physiology, immunology and pharmacology. It is the science that dwells on the anatomy and physiology of parasites, their host(s), transmission, development, impact and their detection and control.

Brief history of Parasitology

- Father of Parasitology: **Platter**
- Father of Modern Parasitology: **Francesco Redi**

World

1500 B.C.	Ebers Papyrus recorded at least four worm infections in ancient Egypt including *Ascaris lumbricoides, Taenia saginata, Dracunculus medinesis* (guinea worm) and *Schistosoma haematobium* .
The Bible	Description of the disease caused by *Dracunculus medinensis*, known as guinea worm, was given by the Greeks. Pulling out guinea worms by winding them on a stick was a treatment used.
129-200	Galen recognised *Ascaris*, *Enterobius* and *Taenia*.
1532	Fitzherbert described the liver fluke, *Fasciola hepatica*
1592	Dunas discovered *Diphyllobothrium*
1674	Leeuwenhoek described the first coccidian oocysts of *Eimeria* from rabbit which was later named *Eimeria stiedae* by Donbell (1922).
1681	Leeuwenhoek first observed *Giardia* from his own stool
1717	Lancisco suggested that malaria is carried by mosquitoes
1843	Dubini described the hookworm *Ancylostoma duodenale*
1851	Bilharz described *Schistosoma haematobium, Hymenolepis nana* and *Heterophyes heterophyes*
1857	Leuckart and Virchow described independently the life cycle of *Trichinella spiralis*
1880-81	Thomas and Leuckart described independently the life history of *Fasciola hepatica*

1889 Theobald Smith described *Babesia* parasites

1893 Theobald Smith and Kilbourne demonstrated the transmission of *Babesia* by ticks

1898 Ross described the life cycle of avian malaria parasites in mosquitoes

1898 Koch described *Theileria parva*

1907 Tyzzer described *Cryptosporidium*

1909 Nicolle and Manceaux discovered *Toxoplasma gondii*

1910 Chagas first observed *Trypanosoma cruzi* and its life cycle through bugs

1959-63 Introduction of first commercially produced antiparasite vaccine against *Dictyocaulus* in sheep and cattle through work of Jarett and co-workers

1972 Rommel; Heydorn and co-workers discovered life cycle of *Sarcocystis*

1988 Dubey and co-workers discovered *Neospora caninum*

India

1878-79 Lewis reported first mammalian trypanosome, *Trypanosoma lewisi* in rats

1880 Griffith Evans discovered first pathogenic trypanosome, *Trypanosoma evansi* from blood of horses and camels suffering from 'Surra'

1885 Cunningham discovered *Leishmania tropica*

1894-99 Alfred Lingard made chemotherapeutic studies against Surra with arsenicals

1897 Ronald Ross found *Anopheles* mosquito as the vector of human malarial parasite

1898 Ross found Culex fatigans as the vector of bird malaria parasite, Plasmodium relictum

1903 Leishman and Donovan independently discovered *Leishmania donovani* at Dum Dum in Bengal

1906 Montgomery described blood flukes namely *Schistosoma spindale, S. indicum* and *Orientobilharzia bomfordi* of animals

1926 Chandler described *Schistosoma incognitum* from pig and dog

1932-33 M.A.N. Rao, discovered *Schistosoma nasale* in the veins of nasal mucosa of cattle

1935-36 P.G. Pande described *Stephanofilaria assamensis*, causing hump sore in cattle

1950 H.N. Ray described Stilbamidine test for diagnosis of latent trypanosomosis in bovines

1958 S.N. Singh described *Stephanofilaria zaheeri* causing ear sore in buffaloes

Parasites

The word parasite has been derived from the Greek word Parasitos (Para-beside, sitos-food). Parasite refers to an animal or organism that lives inside or on the body of a different animal called host (another species) and is metabolically dependent on it. In order to survive, the parasites have to overcome several problems some of which include:

1. Host finding and host cell invasion: They must develop successful strategies for finding a suitable finding. They must find methods for attachment and/or for partial or total penetration into their prospective hosts.
2. Metabolism: They must become able to feed on their host's tissues or fluids and must be able to metabolize the nutrients obtained.
3. Immune Response: They must develop mechanisms to protect themselves from the attacks of the host's defence systems, e.g., mechanisms for immune evasion.
4. Biotic potential: Parasites have to establish a high reproduction rate in places from which the offspring can be transmitted to other hosts.

Types of parasites and their classification:

A. According to the duration of parasite on/in the host

1. Temporary parasites: Also known as intermittent or periodic parasites, whose life cycles are not spent entirely on the host and are seen only for certain periods of time. E.g. mosquitoes and bugs.
2. Permanent parasites: Those parasites that spend their entire life cycle within or the host e.g. *Melophagus ovinus* (sheep ked).

B. According to the habitat of the parasite in or on the host

1. Ectoparasite: A parasite that lives on the surface of a host, e.g. Fleas, ticks, lice, flies etc.
2. Endoparasite: A parasite that lives inside the body (body cavity/ organ/ tissue/ cell) of the host, e.g. helminths, protozoan parasites etc. Endoparasites are further of different types**:**

   i) Intracellular parasites: Those parasites that live and reproduce inside the host cell, e.g. piroplasms of *Babesia bigemina* inside RBCs.

   ii) Intercellular parasites: Those parasites that inhabit spaces in the body of the host. They are also referred as extracellular parasites, e.g. *Trypanosoma evansi*.

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iii) Erratic or aberrant or ectopic parasites: Those parasites which are present in locations that are not their usual site, e.g. *Fasciola* in lungs or brain of sheep.

iv) Accidental or incidental parasites: Those parasites found in a host that is not their original host, e.g. *Toxocara vitulorum* in goats, *Dipylidium caninum* in children.

C. According to the degree of parasitism

1. Obligate parasites: Parasites that cannot complete their life cycle without spending a part of their life cycle on the host, e.g. trematodes.
2. Facultative parasites: Parasites that are not normally parasitic but become parasitic when they accidentally find a host, e.g. *Chrysomyia* sp. larva in wound.

D. According to the type of host required in the life cycle of parasite

1. Monoxenous parasites: Parasites that require only one host to complete its life cycle, e.g. eimerian coccidia etc.
2. Heteroxenous parasites: Parasites that require two or more hosts to complete its life cycle, e.g. *Babesia bigemina* requiring cattle and ticks.
3. Auto-heteroxenous parasites: Those parasites whose definitive host (vertebrate) at one stage of infection may act as an intermediate host for another susceptible vertebrate host, e.g. *Trichinella spiralis.*
4. Stenoxenous parasites: Parasites with a narrow host range e.g. *Haematopinus suis* in pigs, coccidian parasites etc.
5. Euryxenous parasites: Parasites with a wide host range, e.g. *Toxoplasma gondii*.

E. According to size of the parasites

1. Microparasites: Those parasites which can be seen with the aid of a microscope, e.g. *Theileria annulata*, *Hepatozoon canis*, *Trypanosoma evansi* etc.
2. Macroparasites: Those parasites which can be seen with naked eye, e.g. *Ascaris suum*, *Haemonchus contortus* etc.

F. According to the activity in the day

1. Diurnal parasites: Parasites that are active in the day, e.g. *Musca domestica*, *Tabanus* sp. of flies.
2. Nocturnal parasites: Parasites that are active at night, e.g. Anopheline mosquitoes.
3. Crepuscular parasites: Parasites that are active at twilight (dawn or dusk), e.g. the biting midge, *Culicoides* sp.

G. According to transmission to man or animals

1. Zoonotic parasites: Parasites that are transmissible from animals to man and vice versa e.g. Man getting *Taenia solium* infection after eating under cooked pork.
2. Non zoonotic parasites: Parasites which donot get transmitted from animals to man and vice-versa, e.g. *Strongylus vulgaris*.

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H. According to invasion of the tissues of the host

1. Histozoic parasites: Those which lives within the cells and tissues of the host, e.g. coccidia inside the intestinal cells of host.

2. Coelozoic parasites: Those which live in the lumen of the intestine or other hollow organs of the host, e.g. *Haemonchus contortus* in the abomasum of sheep.

I. According to the laying stages by the parasite

1. Oviparous parasites: Those parasites which lay eggs, e.g. *Strongylus vulgaris, Ancylostoma* sp.

2. Ovo-viviparous parasites: Those parasites which lay eggs containing fully developed larvae to hatch, e.g. *Habronema* sp.

3. Viviparous parasites: Parasites in which the embryonated eggs hatch in the uterus of the female and larvae are passed out, e.g. Filarid worms.

4. Pupiparous parasites: Those parasites in which the eggs hatch and larvae developed in the uterus and when passed outside they are ready to pupate, e.g. *Hippobosca*, *Melophagus* etc.

J. According to the specificity of the parasites

1. Host specific parasites: The parasites whose host range is confined to either one species of the host or closely related species of the host, e.g *Plasmodium vivax* is specific to human beings.

2. Non-host specific parasites: The parasites whose host range is not limited to one species of the host and do not show any marked preference for one species or group of related species of host, e.g. *Fasciola*, *Toxoplasma* sp.

K. According to development in the host

1. Proliferous parasite: The parasite enters the body of the host as one individual and grows, multiples and eventually produces number of daughter individuals. The daughter individuals further grow and multiply, so do their further progeny, thus the host, who begins with one parasites and finishes with harbouring many, e.g. *Theileria*, *Babesia*, etc.

2. Non-proliferous parasites: The parasites enters the body of the host as one individual and grows likewise but the daughter individuals do not multiply in the host in whom they are born. They must get into another host before they can multiply. Thus the host infected with one parasites will never have more than one in his body, e.g. helminths.

L. Other parasites

1. Hyperparasites: Those parasites that parasitize other parasites, e.g. *Histomonas meleagridis* parasitizing the eggs of *Heterakis gallinarum*.

2. Spurious parasites: These parasites are 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.

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3. Synanthropic parasites: are parasites that are present on human dwellings e.g. houseflies, cockroaches etc.
4. Pseudoparasites: Things that resemble a parasite and which are present in the clinical specimens e.g. *plant fibres, yeast, pollen grains etc* .

General Parasitology

Lecture 2

Animal association

All animals are in constant interaction with other organisms. These interactions can be divided into two basic types: intra-specific interactions and inter-specific interactions. Intra-specific interactions are those that occur between organisms of the same species, e.g. association between members of a flock of sheep. Inter-specific interactions are those that take place between different species of organism, e.g. association between a sheep and a dog.

Generally, an association/ interaction between two organisms living in close physical association is termed as **Symbiosis**. Any organism that is intimately associated with another organism of a different species is considered to be a symbiont. Symbiosis may be of different kinds viz., mutualism, commensalism, phoresis, parasitism, predation.

1. **Mutualism:** A kind of association where both partners (symbionts) are mutually dependant on each other**.** This relationship is usually obligatory and one symbiont cannot survive without the other, e.g., intestinal protozoa in termites, where termites lacking the enzyme cellulase still feeds on cellulose enriched food which is digested by the intestinal protozoa of the termites.
2. **Commensalism:** An association where one symbiont derives benefit from the other symbiont (host) but the host is neither benefited nor harmed, e.g. *Escherichia coli* in the intestine of man (host) where it feeds on the unwanted food of the host.
3. **Phoresis or phoresy:** An association between two symbionts which are merely traveling together and without physiological or biochemical dependence on each other, e.g. bacteria on the legs of a fly.
4. **Predation:** is the relationship between predators and prey where the predators kill the prey for food.
5. **Parasitism**: an association in which an organism (parasite) is metabolically dependent on another species of an animal (host).
6. **Parasitoids:** are those, whose immature stages develop on other parasites and emerge by killing the parasite. E.g. Hymenopteran flies on dipteran flies

Types of Parasitism

1) **Infestation:** Parasitism of host by external parasites e.g. fleas, lice, ticks in livestock.
2) **Infection:** Parasitism of host by internal parasites, e.g. amphistomes in livestock. Infection can be further of two types:

a) **Autoinfection:** Condition where the juvenile infective form of a parasite, without escaping from the host infects the same host e.g. *Cryptosporidium parvum*

b) **Hyperinfection:** 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.

3) **Congenital (Transuterine or Transplacental):** Transmission of parasites from the dam to the foetus across placental membranes, e.g. *Toxocara canis* in dogs.
4) **Transmammary (Transcolostral):** Transmission of parasites from the dam to the young one through the milk of the infected dam, e.g . *Toxocara cati* in cats.
5) **Transovarian:** Transmission of parasite from the female parent to the progeny through the ova, e.g. *Babesia bigemina* in ticks.
6) **Transtadial:** Transmission of parasite from one stage to another stage of the vector, e.g. *Theleria annulata* in *Hyalomma* spp. of ticks.

Types of host

1. **Definitive host or final host:** The host in which the parasite reaches maturity and undergoes sexual reproduction, e.g. Sheep for *Fasciola hepatica,* Dog for *Echinoccocus granulosus etc.*
2. **Intermediate host:** The host in which the parasite undergoes its developmental stages (asexually) but cannot develop into an adult or reproduce sexually. Intermediate hosts can be further classified as:
    a) First intermediate host: The first larval stage of the parasite develops in this host, e.g. snail for *Dicrocoelium dendriticum*.
    b) Second intermediate host: This host picks up the early larval stages developed in the first intermediate host. Here these larval stages develop further and reach the infective stages of the parasite, e.g. ants for *Dicrocoelium dendriticum*.
3. **Reservoir hosts:** They are vertebrate hosts that harbour a parasite of another host without showing any clinical symptoms, but act a source of infection for the original host, e.g. Game animals for the protozoan parasite *Trypanosoma gambiense.*
4. **Carrier hosts:** The vertebrate hosts that have suffered from an infection and in later stage carries the infective organism in its body without showing any clinical signs.
5. **Transport hosts:** 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.*
6. **Paratenic hosts:** 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. C*alotes for Spirocerca lupi.*
7. **Intercalary host**: The 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 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.

1. **Mechanical vector:** An arthropod that carries the infectious agent from, one vertebrate host to another without any developmental or biological change in its body, e.g. *Tabanus* transmitting *Trypanosoma evansi* by interrupted feeding.
2. **Cyclical vector:** A vector in which the parasite undergoes development or multiplication before being transmitted to the next host, e.g. *Hyalomma anatolicum* transmitting *Theileria annulata* infection.

Parasitic Zoonoses

These are diseases transmissible between vertebrate animals and man. They can be classified accordingly:

1. **On the basis of reservoir host:**

a) **Anthropozoonoses:** Infection transmitted to humans from lower vertebrates, e.g., Cystic echinococcosis from animals to humans, *Onchocerca volvulus* etc.
b) **Zooanthroponoses:** Infection transmitted to lower vertebrate animals from man e.g. *Taenia solium* from humans to pigs.
c) **Amphixenoses:** Infections shared by both man and lower vertebrates and are transmitted in either direction, e.g. Salmonellosis.

2. **On the basis of type of life cycle:**

a) **Direct zoonoses:** Infection is transmitted between the two vertebrate hosts through direct contact or a mechanical vector, e.g. Toxoplasmosis, Amoebiosis etc.
b) **Cyclo-zoonoses:** The zoonoses which involves two or more vertebrate hosts (but no invertebrates) for the completion of life cycle, e.g. Echinococcosis, Taeniosis etc.
c) **Meta-zoonoses:** This type of transmission requires an invertebrate host and a vertebrate host, e.g. Schistosomosis, Leishmaniosis etc.
d) **Saprozoonoses:** They include a vertebrate host and an environmental reservoir (food, soil, plant etc.), e.g. Myiasis, Larva migrans etc.

Other Terminologies

1. **Incubation period:** The time lapse between the entry of the parasite and the first appearance or onset of clinical signs.
2. **Prepatent Period:** The period in which the form of the parasite is demonstrated in the clinical material of the host.
3. **Hypobiosis:** Temporary cessation in the development of immature stages of some nematode parasites in the host due to adverse environmental conditions, e.g. *Ostertagia ostertagi* in cattle. The epidemiological importance of hypobiosis is that the resumption of development of hypobiotic larvae usually occurs when conditions are optimal for free-living development and so results in increased contamination of the environment.

4. **Diapause:** This phenomenon is observed in arthropods where they survive adverse conditions by cessation of growth and metabolism at a particular stage. It is most common in temporary arthropod parasites in temperate climates. In these, feeding activity is restricted to the warmer months of the year and winter survival is often accomplished by a period of diapause, e.g. *Hydrotoea irritans*, *Stomoxys calcitrans* etc.
5. **Parthenogenesis (Virgin birth):** refers to formation of progeny without fertilization, e.g. *Haemaphysalis* ticks and *Strongyloides* sp. of nematodes.
6. **Paedogenesis (Juvenile multiplication):** Multiplication of juvenile/immature stages of parasite in intermediate host, e.g. Multiplication of immature stages of trematodes in snail.
7. **Metagenesis (Alternation of generation):** Sexual multiplication alternating with asexual multiplication, e.g. In trematodes where sexual multiplication in definitive host alternates with paedogenesis in intermediate hosts.

General Parasitol

Lecture-3

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 except 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.

General Parasitol

Lecture 4

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.

Mode of transmission of the parasites

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 following routes:

1. **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. Examples, the infective stages of the parasites with direct life cycle like *Ascaris suum*, *Entamoeba histolytica* 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, e.g. *Diphyllobothrium latum*, *Taenia solium* etc.

2. **Skin penetration:** The infective stages of the parasite may actively penetrate the skin, e.g. cercariae in schistosomes, infective larvae of hookworms. Certain parasites enter the hosts via the bite of an intermediate host serving as a vector, e.g. Sporozoites of *Babesia bigemina* entering inside blood circulation of cattle by the bite of tick vector *Rhipicephalus* (*Boophilus*) *microplus*.
3. **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.
4. **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.
5. **Coitus:** *Tritrichomonas foetus*, the protozoan parasite that causes abortion in cattle and *Trypanosoma equiperdum*, the protozoa that causes dourine in horses are transmitted during coitus.
6. **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*.

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

1. **Water:** Important agent for dissemination of parasites. Example includes larvae of *Dracunculus* which 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 use of sewage and sludge to fertilize pasture, construction of irrigation canals directly facilitate breeding of snails' thereby aiding dissemination of trematode parasites besides aiding breeding of insects.
2. **Fomites:** 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.

Parasite induced host behavioural changes

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

Strategies adopted by parasites to infect hosts

1. 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.
2. 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.
3. Some parasites are endowed with sensory organs to locate hosts. Warmth, CO_2, fatty acids, amines etc., serve as stimuli for parasites especially arthropods to locate hosts.

General Parasitology

Lecture-5

Parasite specificity in relation to species, breed, sex and location

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 or 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 like protozoan parasite *Toxoplasma gondii*.

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

1. 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. Th normal resistance of various species of animals to various pathogens is due to the presence of antibodies on their erythrocytes called isohaemagglutinins.
2. Many parasites do not develop in hosts other than their natural hosts, e.g *Eimeria* sp.
3. The red worms (*Strongylus* sp) of horses are specific to equine hosts and cannot infect cattle, buffalo, sheep and goat.
4. 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.

5. 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

1. 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.
2. 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.
3. In case of female dogs (bitch) above six months old, development of *Toxocara canis* is influenced by hormonal changes. When they 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

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

Parasite specificity in relation to location in hosts

1. 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.
2. Other examples include the nematode *Dictyocaulus* species which affect only the lungs in ruminants and the trematode, *Paragonimus* species in lungs of dogs.

Parasite specificity in relation to locality

1. 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.

Tissue reactions caused by parasites to the host

1. **Injuries during entry of parasites:** Certain parasites cause direct injuries to the host, during the process of entering the host. The hookworms *(Anclystoma 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.
2. **Blood loss and anemia:** Some parasites suck blood (hookworms, barbers pole worm, mosquitoes, tick etc) from the hosts resulting in anaemia. *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.
3. **Changes in protein metabolism and mineral levels:** It 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. Mineral deficiences 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.
4. **Destruction of tissues:** Many parasites during their course of development undergo migration in the tissues of hosts, e.g., 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.
5. **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.Ticks also cause inflammation and leakage of serum from damaged skin in sensitized animals.
6. **Mechanical interference:** Interference can occur either due to occlusion or pressure atrophy by the parasite.

    a) 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. Gapeworms block the air passages causing dyspnoea and asphyxia in fowls.

    b) Pressure atropy: Pressure upon organs by larval stages of parasites, especially those of tapeworms result in atropy 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.

7. **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. 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.

General Parasitology

Lecture 6

Immunity against parasitic infections or infestations

1. **Innate or natural immunity:** Immunity possessed by the host animal from birth. In other words, innate immunity is physiological incompatibility between certain parasites and the host that prevents the entry and establishment of the parasite in the host. It is an inherited non-specific immunity and the first line of defense. It constitutes of physical and chemical barrier to infectious agents, identification and removal of foreign substances present in the host body by leucocytes like natural killer cells, mast cells, basophils, eosinophils, macrophages, neutrophils; activation of the complement cascade, production of cytokines etc. 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. Innate immunity can be complete / absolute or incomplete / relative.

- A host is said to possess complete innate 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 immunity.

2. **Acquired immunity:** Immunity acquired by the host during its life time as a result of exposure to a particular parasite or through inoculation of parasite antigen. Acquired immunity may be either active or passive.

- **Active immunity:** is acquired in response to the introduction of live or killed parasites. This may be either natural or artificial. Natural active 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, long lasting, and diverse and takes time to fully develop.
- **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.

Types of Natural Resistance

Different types of natural resistance or immunity viz., premunity, concomitant immunity and sterile/ solid immunity are seen in parasitic infections.

1. **Premunity:** Immunity that is established after the primary infection has become chronic and is 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 (Co-infectious immunity). Premunity is commonly associated with haemoprotozoan diseases such as theileriosis and babesiosis.
2. **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 schistosomiosis and cystic echinococcosis (hydatidosis).
3. **Sterile immunity:** Also known as complete immunity, it is maintained by the host even after the parasite is eliminated. The immune system remains sensitized for many years e.g. Cutaneous leishmaniosis (Oriental sore) in man, *Theileria annulata* infection in cattle, *Plasmodium cynomolgi* infection in monkeys, etc.

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 Immune Response

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.

Cell Mediated Immune Response

Cellular immunity is mediated by T cells subsets and their cytokines 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).

<u>Hypersensitivity reactions</u>

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:-

1. **Type –I (Anaphylactic shock):** acute or 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.
2. **Type-II (Cytotoxic):** it is a subacute reaction where antigen binds on IgG or IgM antibody. It is associated with development of anaemia in malaria and haemoglobinuria in babesiosis.
3. **Type-III (Immune Complex disease):** Circulating antigen antibody complexes (IgG or IgM) coupled with complement are deposited in tissue space causing damage to the tissues, e.g., glomerulonephritis in babesiosis, malaria, and granuloma formation around eggs of schistosomes.
4. **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 eosiniphil infiltration.

Kinds of parasitic antigens

1. **ES antigens**: Antigens from the excretions (E) and secretions (S) of the parasite are called ES antigens.
2. **Somatic antigens**: Antigens elaborated by the entire body of the parasite is called somatic antigen.
3. **Internal antigens**: Antigens obtained after the disruption of the parasites.
4. **External antigens**: Antigens manifested by intact parasites.
5. **Released antigens**: Metabolic products of parasites are released antigens and these are comparable to external antigens.
6. **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.

Evasion of immune response

Helminths and protozoa have the capability to survive within immunologically competent host and can thus evade the host's immune response by various mechanisms:

1. **Parasite location in sanctuary sites:** There is failure of phagosome fusion with lysosome (*Toxoplasma gondii*), escape into cytosol (*Trypanosoma cruzi*) or resistance to lysosomal enzymes (*Leishmania* sp.). In Toxoplasmosis, host antibodies are protective but the cystic stage of *Toxoplasma* is inaccessible to the humoral factors, hence the antibody cannot escape the cystic stage; as a result the overt clinical disease is rare but subclinical infection is relatively frequent.
2. **Antigenic variation:** In trypanosomosis and malaria, the parasites escape from the cytocidal action of humoral antibody on their cycling blood forms by altering their antigenicity.
3. **Antigenic mimicry:** In Schistosomosis, the host tissue materialgets attached to the surface of parasite and the host immune system fails to recognise the antigen to elicit immune response. Sometimes, antigenic determinants of parasite resemble host determinants and will not be recognised as 'non self' by the host and thus no immune response will be produced against them.

Effect of immune response/ immunity on parasites

1. Prevention or reduction in the rate of establishment
2. Structural or metabolic alterations in the parasites
3. 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.
4. 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.
5. Destruction of parasites *in situ*.

6. Elimination of existing infection.

Harmful effects of parasites on their hosts

Parasites have different types of effects on their hosts. Variability in the degree of harmful effects amongst various parasites is related to their number, invasive power, virulence, reproductive potentiality, propensity of release of toxic products and their localistaion in the body of their hosts, besides many other intrinsic and extrinsic factors in their surrounding within and outside of their hosts like general health condition, age, breed, sex, nutritional level, concurrent infections, state of hygienic condition,etc. The following types of pathological conditions are caused by different parasites:-

1. Compete with the host for food including vitamins (e.g. ascarid roundworms, tapeworms like *Diphyllobothrium latum*).
2. Decreased utilization and absorption of nutrients.
3. Reduction in the feed intake by the animals.
4. Increase in the passage of food, without proper digestion, through the digestive tract of animals.
5. Changes or reduction in the absorptive surface of the intestine (intestinal nodular worm of ruminants e.g. *Oesophagostomum* spp., *Paracooperia nodulosa* in buffaloes etc.)
6. Decreased synthesis of protein in skeletal muscles.
7. Alterations in the efflux and influx of water and sodium and chloride ions into the bowel (gastrointestinal nematodes).
8. Morphological and biochemical changes in the epithelial cells and their microvilli (e.g. coccidia).
9. Removal of host's body fluid including blood (e.g. hookworms, ticks etc.).
10. Parasites feed on host's solid tissues (kidney worm, liver fluke, immature amphistomes).
11. Destruction of host's tissue mechanically or by pressure of the growing size of the parasite (*Cysticercus*, hydatid cyst etc.).
12. Cause mechanical obstruction of gut lumen, air passages, ducts, blood vessels etc. (e.g. ascarid worms, lungworms, liver fluke etc.)
13. Destroy host cells like epithelial, endothelial, connective tissue, RBC, WBC (e.g. coccidian, *Babesia*, *Theileria* etc.).
14. Produce toxic substances causing haemolysis, neurotoxin, anticoagulants, toxic metabolites etc. (e.g. hookworms, tapeworms, ascarids).

General Parasitology

15. Various host's tissue reactions against parasites like inflammation, haemorrhage, necrosis, fibrosis, excessive mucus production, nodulation, tumor formation, atrophy, paralysis, swelling, blindness etc.
16. Immune responses to parasites to include immunopathological lesions (e.g. eosinophilic granuloma around eggs of blood flukes; auto-immunity in piroplasmosis).
17. Some worms carry pathogenic organisms (e.g. Heterakis gallinae transmits *Histomonas meleagridis*, *Metastrongylus apri* carries swine influenza virus).
18. Parasitic infections depress immune system of animals making them susceptible to other pathogens (e.g. trypanosomosis, theileriosis etc.).

General control measures against parasites

The following measures should be kept in mind to prevent healthy animals from getting infection:

1. Animals should be kept in hygienic and in dry surroundings.
2. Avoiding access of susceptible animals to various sources of infection including contact with diseased animals.
3. Providing balanced nutrition and clean drinking water.
4. Removing spots/ places of breeding of intermediate hosts/ vectors including their killing by using molluscicides/ insecticides.
5. Proper maintenance of slaughter houses and effective disposal of offals/ blood accessible to stray animals.
6. Proper investigation of new animals before introducing in the herd/ flock.
7. Use of sterilised equipments for mass vaccination, artificial insemination, feeding, watering, storing etc.
8. Frequent screening of animals to detect any latent/ carrier case and its immediate treatment.
9. Formulating mass drenching schedule for prophylaxis according to the requirement in the area against possible parasitic infection and to remove infection from sub-clinical or carrier cases, particularly at organised livestock farms. Frequent removal of ticks from the body of animals and their immediate burning.
10. Newly born and young growing animals should be reared separately from the adult animals and they should not be allowed to graze on infected pastures.
11. Segregation of clinical cases and their proper treatment (chemotherapy).

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12. All apparently healthy animals should be given preventive medication (chemoprophylaxis).
13. Vaccination where available and possible will also be helpful in curbing the disease.

General Parasitology

Lecture 1 (Eucestoda)

All parasites are classified into three major groups – Helminths, Arthropods and Protozoa.

Helminths : Name helminths is derived from a Greek word *helmins* or *helminthos* which means a worm and this is applied to only parasitic and non parasitic species belonging to phylum Platyhelminthes and phylum Nemathelminthes. The annelids (leeches, earthworms etc.) are fundamentally different and not regarded as helminths.

Characteristics of Phylum platyhelminthes :

1. The members of this phylum are dorsoventrally flattened and usually hermaphrodite with solid bodies.
2. They do not have body cavity. The organs are embedded in the parenchyma.
3. Respiratory and blood vascular systems are absent.
4. Flame cells are the excretory organs.
5. Like Nemathelminthes they are not metamerically segmented.
6. Life cycles are usually indirect.

Phylum platyhelminthes further divided in to four classes.
Class: Trematoda ; Class : Eucestoda ; Class : Turbellaria ; Class :Cotyloda.

1.Class: Trematoda :The parasites under this class have alimentary canal. Species are parasitic in man and animals.Commonly they are called as flukes. Further they are devided into two sub classes :1. Sub class- Digenea.
2. Sub class- Monogenea.

2. Class: Eucestoda: They are true cestodes .They do not have alimentary canal .Food is absorbed through the surface tegument. Commonly they are called as tape worms.
3. Class:Turbellaria : This class mainly comprised of the non parasitic species living in fresh water,sea water or on the land.These species have ciliated covering on their body.The species which are parasitic ,they are non parasitic in domesticated animals.
4. Class:Cotyloda : Shape of these parasites looks like tape worms. Forms are mainly parasitic in fishes except few (*Diphyllobothrium* & *Spirometra* sps.) whicg are parasitic in animals and man.

Wardle et al.(1974) divided the cestode into two classes. ; Cotyloda and Eucestoda. The class Eucestoda contains 15 orders, of which 7 contain important parasites of domestic animals, man and fishes.

Class: Eucestoda

They are true cestodes .They do not have alimentary canal .Food is absorbed through the surface tegument. Commonly they are called as tape worms.

- 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: Scolex is usually globular in shape. Has four suckers(acetabula) , which may be armed with hooks. A protrusible part ; the rostellum, often armed , may be present.
    - Neck
    - The remainder body is called as strobila which consists of number of segments called as proglottids. Proglottids are vary in shape and size and are separated by transverse constrictions. Each proglottid contains one or two sets of reproductive organs. Majority of tapeworms have metameric repetition of their reproductive organs called as proglottidization. Proglottids are formed from neck/growth region and mature segment are pushed away from the scolex. The posterior proglottids when fully mature are packed with eggs and are called as gravid segments

The body of tapeworms covered with tegument composed of syncytial outer layer formed by tegumental cells. The outer cytoplasm is extended into microtriches &microvilli (which are spine like processes covered with plasma membrane) containing micro tubular structure. The whole act as absorptive surface.

Excretory system:

Consists of Nephridial system with flame cells There is a presence of efferent canals ,usually on the either side two longitudinal canals joined by a loop in the scolex. Each pair may be connected in the posterior portion of the proglottids.

Nervous system:

- Central part of the nervous system is situated in the scolex generraly consists of a rosteller nerve ring and two lateral nervous ganglia, from which six cords run posteriorly.
- In addition to that there is a dorsal and ventral nerve cords.

Reproductive system:

There are one or two sets of reproductive organs per proglottids. Mature proglottids are those in which reproductive organs are fully mature and functional. Following fertilization of eggs , the reproductive organs degenerates leaving gravid proglottids full of eggs. Gravid proglottids are detached from the worm and passed out of the host singly or in chains. Eggs set free by disintegration of gravid proglottids. The process is called apolysis. In some species (eg. Taenia) the eggs are released by pressure of eggs in uterus and the muscular activity of proglottids through an opening called as *thysanus* created in the uterus when gravid proglottid break away from strobila. When egg production ceased the exhausted proglottids detached from strobila and

disintegrated , the process is called as pseudoapolysis. In cotyloda the proglottids are not detached and eggs are normally discharged from proglottids through an uterine pore opening on ventral side of proglottid.

Male reproductive organs developed first and it is termed as protandry or androgyny

Male reproductive system

- There is one/more but usually more number of testes.
- These testes discharge in vasa efferentia which join to form vas deference and ends in cirrus which is surrounded by cirrus sac.
- Male and female genital pore lies close together in genital sinus on lateral or ventral side.
- Self fertilization or cross fertilization between proglottids may occur.

Female reproductive System

- Female genital pore leads to vagina
- Receptaculum seminis.
- Ovary is usually bilobed.
- Vitellaria is compact in Eucestoda
- May be follicular in cotyloda
- Uterus arises from ootype.
- The uterus in cotyloda may have pore which communicate outside
- In eucestoda it is filled with eggs. Uterus (in eucestoda) may be found as a tube or diverticula or may develop branching (*Taenia*). In gravid segments uterus may degenerate and eggs may pass singly or may pass, in groups, in a protective structure with in the segment. Protective structure such as hyaline egg capsules which enclose and protect the group of eggs. This structure is formed from uterus. Paruterine organs made up of fibro-muscular tissue of parenchyma and dilation of uterus. Eggs when passed out of the host may be embryonated or unembryonated. Embryonated eggs contain onchosphere armed with three pairs of hooks. Hatching and activation of onchosphere occur in the intestine of the intermediate host. Hatching may be by a combination mechanical action of the host and digestive enzymes (*Taenia* sp.). The activated onchosphere penetrate the intestinal mucosa and migrate to the site of predilection via circulation.

General Life cycle of cestodes (tape worms):

In majority of cases tapeworms are found in intestine of animals and man. Therefore the eggs or gravid segments are found in the faeces of infected animals. In majority of cases these eggs contain hexacanth embryo or onchosphere. Onchosphere do not hatch from the eggs unless

the eggs have been swallowed by the intermediate host. Different species of invetebrate and vertebrate host act as intermediate hostfor different species of the tapeworms.

1) Life cycle of tape worm having invertebrate host as intermediate host:

In this life cycle the eggs are swallowed by different species of invertebrate animals (Oribated mites ,flies ,fleas, ants beetles, earthworm etc.) which act as intermediate host. In the gut of these intermediate hosts eggs hatch and onchosphere released which subsequently penetrate the gut wall and reach to the body cavity In the body cavity the onchosphere converted into infective stage called cysticercoids. Final host acquire infection by swallowing infected invertebrate hosts containing cysticercoids ,with contaminated food and water. In the intestine of final host cysticercoids are releasedand scolex attach to the intestinal mucosa. Lateron by the process of proglottidization adult tapeworms are formed.

2) Life cycle of the tapeworms having vertebrates as intermediate hosts:

In this life cycle eggs are swallowed by the herbivorous (cattle, sheep, goat, buffalo) omnivorous (pigs) and man which act as intermediate hosts.In the intestine of intermediate host eggs hatch and onchosphere released which invade the intestinal mucosa to reach in the blood circulation from where they are distributed throughout the body of Intermediate host. Only those onchosphere survived which reach to skeletal muscle, cardiac muscle and visceral organs depending upon the species of the tape worm. These later on develop into infective stage called as cysticercus, strobilocercus, coenurus, hydatid cyst). These are called as bladder worms because of their typical appearance like cyst or bladder /balloon. The final host acquire infection by swallowing uncooked/partially cooked meat and visceral organs containing these metacestodes. In the body of final host the scolices are released in the intestine and attach themselves to the intestinal mucosa and adult tapeworms are formed by the process of proglottidization.

Types of metacestodes

1. Cysticercus

Single invaginated scolex with cavity filled with fluid. Eg. *Cysticercus cellulosae* - larval stage of *Taenia solium.* And *Cysticercus bovis* - larval stage of *T. saginata*

2. Cysticercoid

- Single non-invaginated scolex with drawn into a small vesicle. Practically no cavity.
- Eg. Larval stage of ruminant and poultry tapeworm, *Moniezia expansa* has oribated mites as intermediate host.

3. Strobilocercus

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

4. Coenurus

- A large fluid containing bladder in which number of invaginated scolex attached to the wall.
- Eg. Larval stage of *Taenia. multiceps.*

5. 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.*
6. Procercoid
    - Is the larval stage of Cotylodan tapeworms which is occurs in the first Intermediate Host.
    - Procercoid is solid body and possess hooks on the posterior region/cercomere e.g.*Diphyllobothrium latum*
7. Plerocercoid
    - Is also larval stage of Cotylodan tapeworms which occurs in the second Intermediate Host.
    - It is elongated, solid and has a scolex like in adults e.g. *Diphyllobothrium latum.*
8. Tetrathyridium

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

Class Cotyloda

Contains the parasites which are important in fishes in the *plerocercoid* or adult stage of the parasite except *Diphyllobothrium* and *Spirometra* species which are parasitic in man ,dog and cat.

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 grooves.
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. *Taenid* egg	Eggs are unembryonated, operculated and hatch out in the water.eg. *Diphyllobothrium latum* egg
It requires only one I/H	It requires two I/H.
Apolysis is present.	Apolysis is absent.

Class Eucestoda

Order: Anoplocephalidea

Characters:

- They do not have rostellum or hooks
- Segments are usually broader than the length
- Each set has one or two sets of genital organs
- Genital pore located at the margin of the segments
- The transverse uterus may persist or replaced by eggs capsule or one or more paruterine organs.

Eggs have three covering, outer most vitelline membrane, a middle albuminous coat and inner most chitinous membrane which is frequently pear shaped bearing one side a pair of hooked projection called pyriform apparatus.

Family -Anoplocephalidae

- Uterus persists as transverse tube or network of tubes.
- Intermediate hosts are oribatid mites.

TAPE WORMS OF EQUINES

Genus *Anoplocephala*

Species *A. perfoliata*

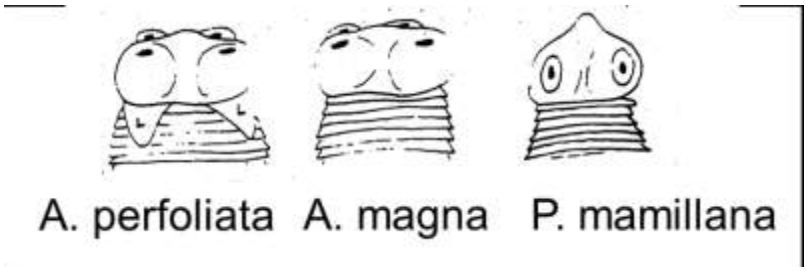
- Found in large intestine and small intestine of horse and donkeys.
- Cosmopolitan in distribution and common species in horses.
- Adult are up to 5 cm. Or up to 8 cm. And 1.2 cm. In width.
- The scolex is 2-3 mm. In dia. And provided with lappet behind each sucker
- Segments are wider than long & contain one set of reproductive organs.
- Gravid uterus is transverse sack like and lobed.
- Eggs measure 50-60 µm.

Species: ***Anoplocephala magna***

- Found in small intestine (jejunum)and stomach of horse and donkeys.
- They may reach to 80 cm X 2.5cm.
- Scolex is 4-6 mm (large) & without lappets.
- Eggs measure 50-60 µm.

Genus **Paranoplocephala**

Species ***P. mamillana***

- Found in small intestine, occasionally in stomach of equines.
- They are 6-50 X 4-6 mm. In measurement.
- The scolex is narrow.
- The opening of the suckers is slit like situated dorsally and ventrally.
- Lappets are absent.
- Eggs measure 51 X 37 µm.
- 

Life cycle

- Gravid segments are passed out with the faeces. They are eaten up by oribatid mites (I.H.) in which cysticercoid develop in 2-4 months after infection.
- Adult tapeworms are produced in the D.H. in 4-6 weeks after accidental ingestion of the infected mites during grazing**.**

Pathogenesis:

- Light infection goes unnoticed but heavy infection may cause ill health, unthriftiness and even death.

- *A. perfoliata* usually found near the ileo-caecal orifice so here it may cause dark depressed ulcerative lesions. There may be oedema and granulation tissue and rarely occlusion of ileocaecal orifice.
- *A. magna* if present in large number, may cause haemorrhagic / catarrhal enteritis.
- Perforation of intestine has been recorded by *A. perfoliata* and *A. magna*.
- *P. mamillana* is usually not causing ill health.

Diagnosis

By finding the eggs in the faeces of the host

Treatment:

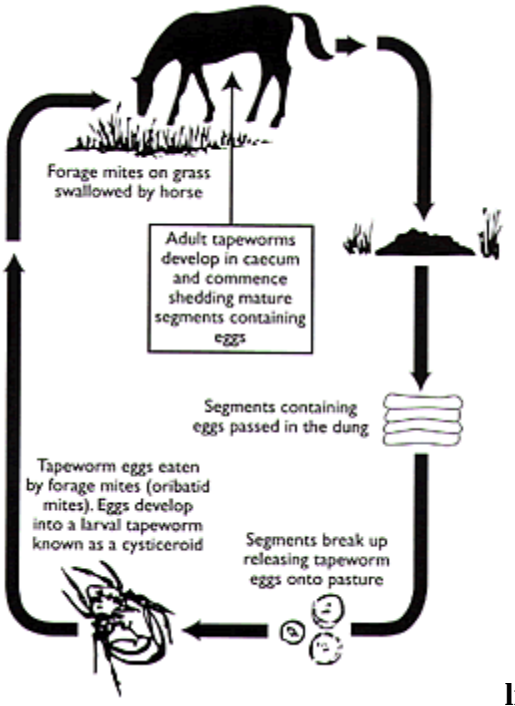
Niclosamide @ 88 mg./KG. B.Wt.

Bithional @ 7mg. / Kg. B. Wt.

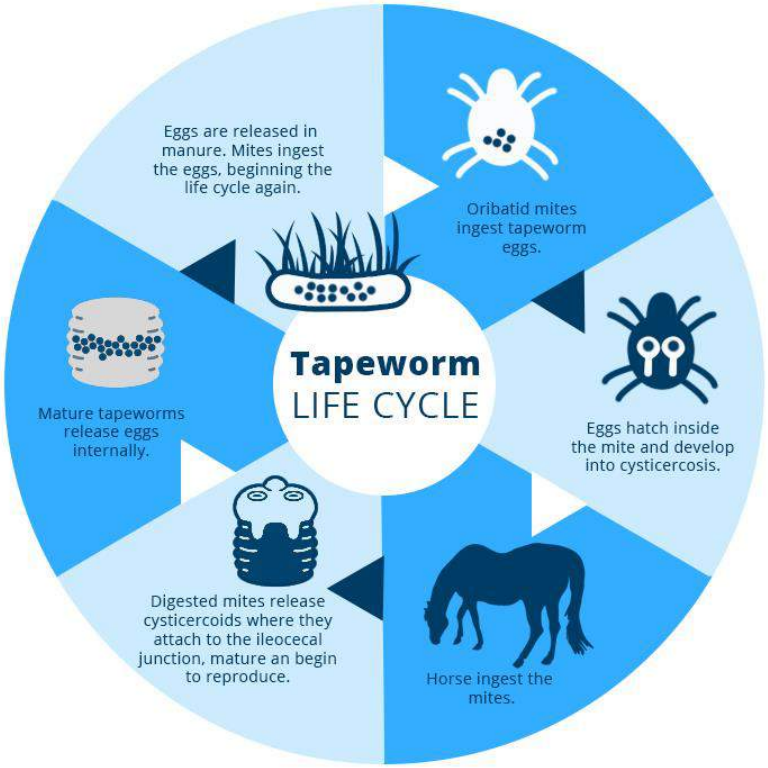
Micronised mebendazole @ 15-20 mg/kg. b.wt.

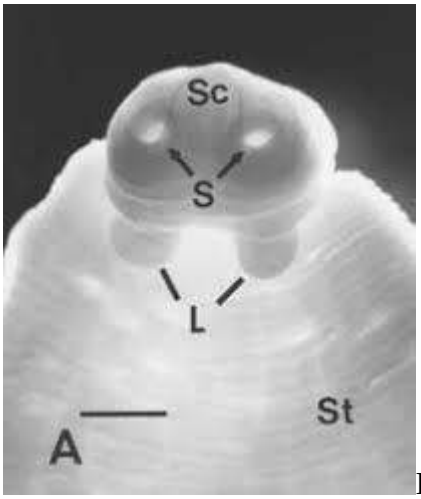


Egg


Forage mites on grass swallowed by horse
Adult tapeworms develop in caecum and commence shedding mature segments containing eggs
Segments containing eggs passed in the dung
Tapeworm eggs eaten by forage mites (oribatid mites). Eggs develop into a larval tapeworm known as a cysticeroid
Segments break up releasing tapeworm eggs onto pasture

life cycle


Eggs are released in manure. Mites ingest the eggs, beginning the life cycle again.
Oribatid mites ingest tapeworm eggs.
Tapeworm
LIFE CYCLE
Mature tapeworms release eggs internally.
Eggs hatch inside the mite and develop into cysticercosis.
Digested mites release cysticercoids where they attach to the ileocecal junction, mature an begin to reproduce.
Horse ingest the mites.



L=lappets

S=suckers

Anoplocephala perfoliata

TAPE WORMS OF RUMINANTS

Genus: Moniezia

Species : Moniezia expansa

- Found in small intestine of sheep, goat, cattle and other ruminants.
- Cosmopolitan.
- Measurement; 600cm.X 1.6cm. , scolex - 0.38-0.8 mm. Wide ,have prominent suckers.
- Rostellum and hooks are absent. Segments are broader than long and each segment contains two sets of reproductive organs with genital pore at the margins.
- Ovaries and vitelline glands form a ring on both sides median to longitudinal excretory canal.
- Testes are distributed throughout the central field.
- Interproglottidal are rosette like found at the posterior border of the segment and extended across the width of the proglottid.
- Uterus becomes sac like when filled with the eggs.
- Eggs roughly triangular in shape contain well developed pyriform apparatus .
- Eggs measure 56-67 um.

Genus: Moniezia

Species : Moniezia benedeni

- Small intestine of cattle , other ruminants .
- It is broader (2.6cm.) than *M. expansa.*
- Interproglottidal glands in a short continuous row close to midline of the segment.

Life cycle:

- Gravid segment in faeces → eggs eaten by oribatid mites (I.H.) →cysticercoids develop in mites in 4 months → animal is infected by eating of mites when grazing . P.P. 37-40 days.
- Infection is common in calves/ lambs/kids in first summer on pasture. Lambs may start passing ripe segments in faeces at the age of 6 week.

Pathogenesis:

Infection is common in lambs, kids and calves under 6 months age. May cause diarrhoea, unthriftiness, obstruction of intestine, depressed wool and meat production. Light infection has no clinical significance**.**

Diagnosis:

Faecal Examination

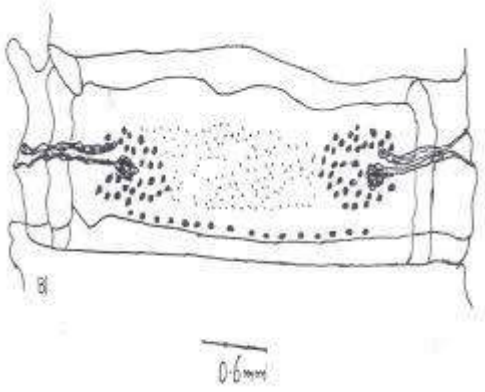
Gross examination may reveal Cooked rice like segments. Eggs are square in shape with pyriform apparatus

Treatment:

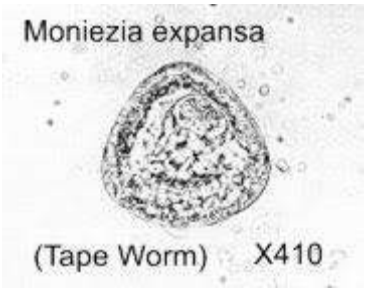
Niclosamide 75mg./kg. , Praziquantel 15mg/kg. ,

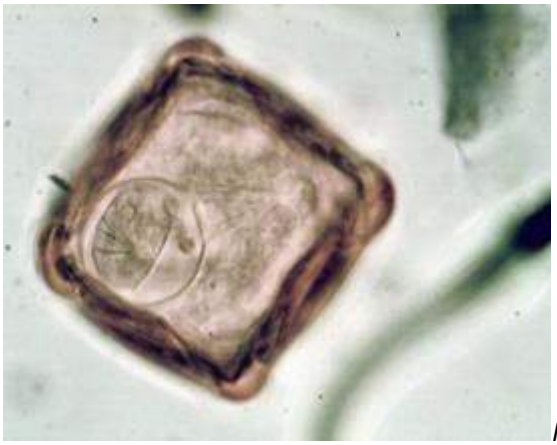
Fenbendazole – 5mg/kg ., Cambendazole 20 mg./kg.

Albendazole 10 mg./kg.

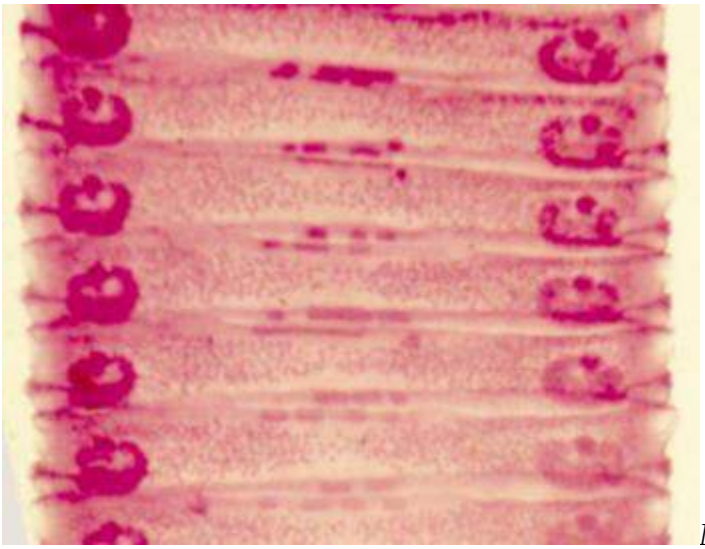


Moniezia expansa mature segment





Moniezia benedeni egg


Moniezia benedeni (mature segment)

Family : Thysanosomidae

In the members gravid uterus disappear and replaced by paruterine organ or capsule

Genus : Avitellina

Species: A. centripunctata

- Small intestine of sheep and other ruminants.
- Prevalent in Europe, Africa and Asia.
- Size: 300cm.X3mm.
- They are cylindrical in posterior end.
- Segments are short and not distinctly segmented.
- Genital organ is single set and G.P. Alternate irregularly.
- Uterus lies transversely in middle portion.
- Uterus and par uterine organs show a opaque line in the middle portion of segment.
- Eggs without pyriform apparatus measuring 21-45 um.

Life cycle:

Psocids(bark lice, dust lice, book lice) may serve as Intermediate host in which the cysticercoid develops.

Pathogenesis: same as Moniezia, considered to be less pathogenic.

Treatment: Bithional, praziquantal and niclosamide are given with same dose rates.

Genus : Stilesia

Species: Stilesia hepatica

- Found in the bile ducts of sheep, goat, cattle and wild ruminants in Africa and Asia.
- In Africa 90-100% sheep may be infected.
- Adults are 20-50 cm long and 3 mm wide.
- It has broad neck and prominent sucker.
- Proglottids are short.
- Reproductive organs are single set and genital pore alternate irregularly.
- Uterus long transverse and dumbel bell-shaped
- Eggs pass into two paruterine organs each containing about 30 eggs.
- Eggs have pyriform apparatus measuring 26x 16-19 um

Life cycle: Oribatid mites act as intermediate hosts

Pathogenesis:

Occurs in animals of all ages groups. In light infection non pathogenic, in heavy infection may cause slight cirrhosis. In most of the cases where whole bile ducts may be full of worms even these worms form dilatations but no icterus or other clinical signs are seen. Infected liver is condemned during meat inspection.

Treatment: praziquantel - @ 15 mg./kg.

Genus : Stilesia

Species: Stilesia globipunctata

Small intestine of sheep , goat, cattle & other ruminants. Common in Europe, Africa and Asia.

Morphology:

- Adult are 45-60 cm long and 8.5 mm wide.
- Characters same as *S. hepatica* except testes are arranged in two rows on lateral side of lateral canals.

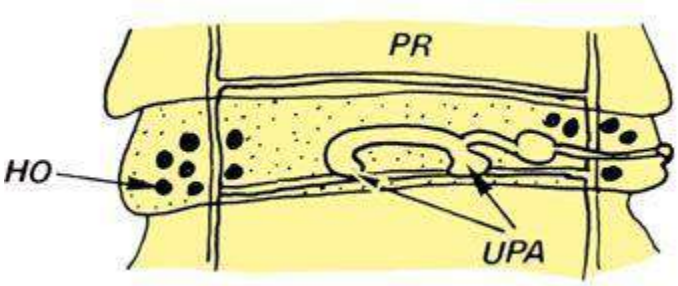
Life cycle: Psocids and oribatid mites act as intermediate host in which cysticercoid develop.

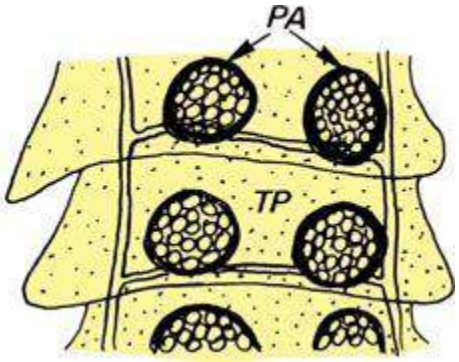
Pathogenesis:

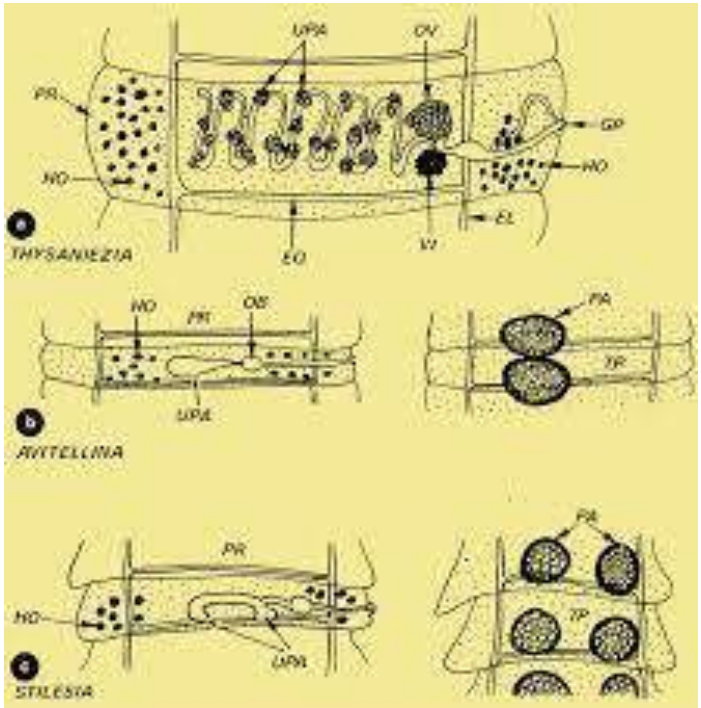
They are attached at the junction of duodenum and jejunum. Scolex of immature worms penetrate in the mucosa of intestine. At the site of the attachment there is nodule formation, cell infiltration, proliferative inflammation and desquamation of epithelial cells. Scolex and anterior part of segments are embedded in mucosa and rest of tapeworm is found free in the lumen.

Treatment:

Praziquantel @ 15 mg/ kg body weight.







Thysanosoma actinoides

- Called as fringed tapeworms and found in bile ducts, pancreatic ducts and small intestine of sheep, cattle and deer in western part of USA and South America
- Size:15-30 cm x 8mm, scolex 1.5 mm
- segments are short and fringed posteriorly
- Each segment contains two sets of genital organs and testes lie in the median field.
- Uterus is single undulating tube.
- Eggs pass in several paruterine organs each containing 6-12 eggs.

Eggs do not have pyriform apparatus

Life cycle: not well understoodnot well understood possibly cysticercoids develops in psocids.

Pathogenesis:

- It may obstruct the flow of bile and pancreatic juice and cause digestive disorders and unthriftiness. Infected liver is liable to be condemned.
- Treatment : Bithional @ 200mg./kg, niclosamide @ 400mg./kg, praziquantel @ 15 mg./kg

Genus : Thysaniezia

Species: Thysaniezia giardi

It occurs in the small intestine of sheep, goat and cattle of Europe, Russia, Africa And America.

Morphology

- Size 200 cm x 12mm wide
- Short segments
- Single pore
- Gential pore alternate irregularly
- The side of the segments, cirrus sac bulges out, thus the margins seem to be
- having irregular appearance

- No pyriform appartus
- Eggs pass from uterus to a large number of par uterine organs

Life cycle

Psosids act as intemediate hosts

Pathogenesis

Infect both large and small animals but rarely produce clinical symptoms

Treatment: Same as *Moniezia* sp.

TAPEWORMS OF POULTRY

ORDER: Davaineidea.

- Small to medium sized tapeworms.
- Rostellum is retractable and armed with numerous hammer shaped hooks.
- Suckers are usually armed.
- Genital organs are usually single.
- Uterus may persist or eggs may pass into egg capsule or in paruterine organ.

Family: Davaineidae

In this family uterus is replaced by egg capsule.

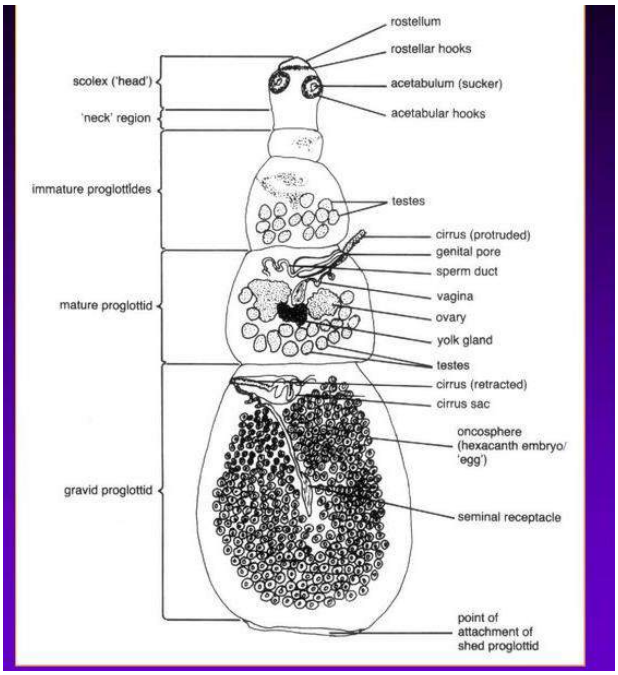
Genus: Davainea

Davainea proglottina

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

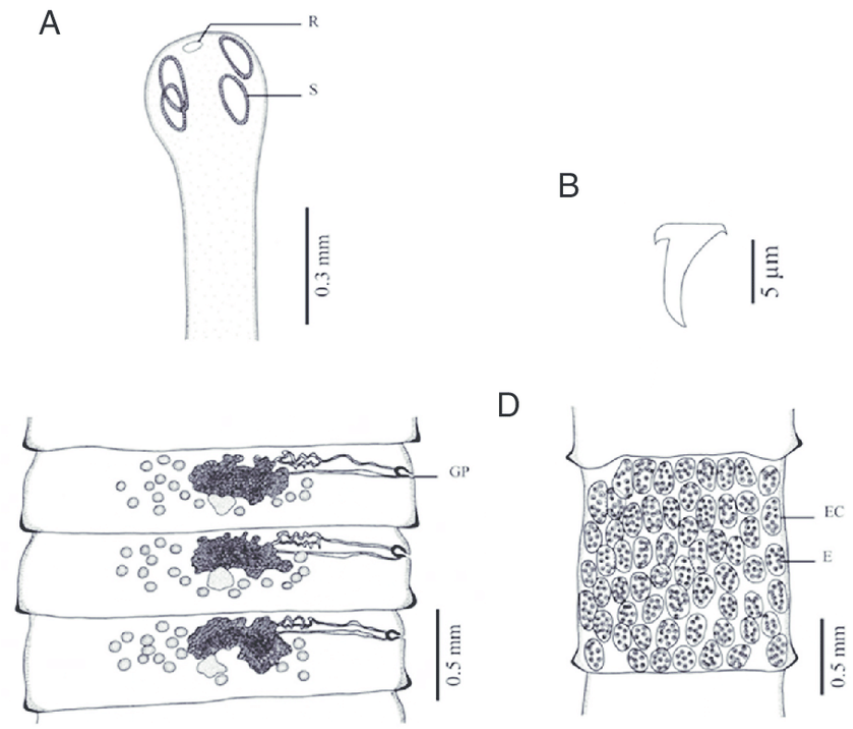
- Each egg capsule contains single egg.
- Eggs are 28-40 um. In diameter
- 

Raillietina tetragona

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

Morphology

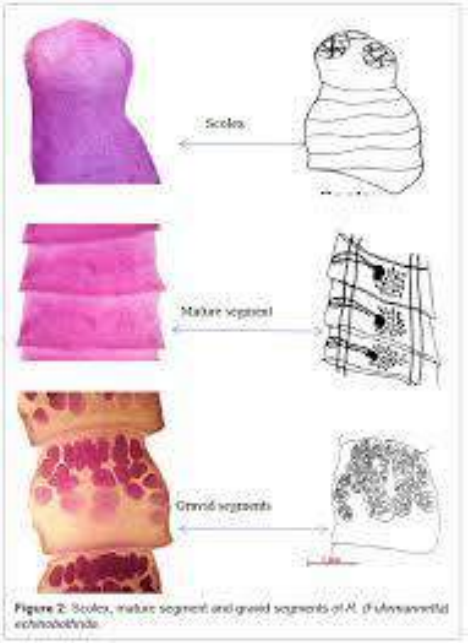
- Adults are up to 25 cm in length. Scolex is smaller than the *R.echinobothridia.* Rostellum is armed with 1 to 2 rows of hooks. Suckers are oval in shape and armed with hooks.
- Each segment has single set of reproductive organs genital pore opens unilaterally.
- Each egg capsule contains 6 to 12 eggs.
- found in posterior half of small intestine of poultry guinea fowl, pigeon and other birds..
- Cosmopolitan in distribution.
- It is one of the largest fowl tapeworm and may reach

- up to 25 cm in length.
- 

R. echinobothridia

Host	Chicken and turkey
Location	Small intestine
I/H	Ants. (*Tetramorium* 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.

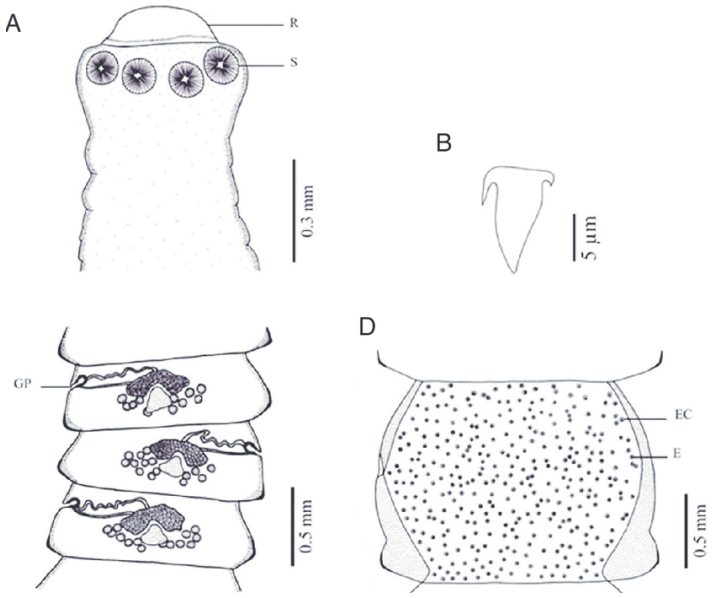


R. cesticillus

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.

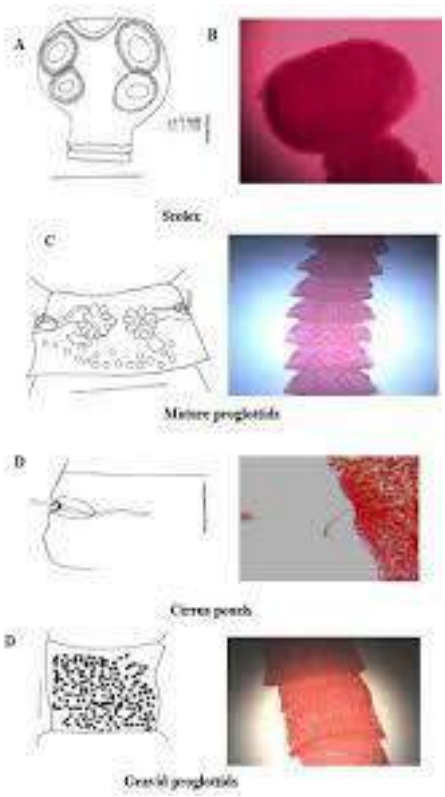
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Cotugnia digonopora

Common name	Double pored poultry tapeworm
Host	Chicken
Location	Small intestine
I/H	Ants. (*Pheidole* spp., *Monomoriumfloricola)*

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.

- 

Order – Dilepididea

Amoebotaenia sphenoides

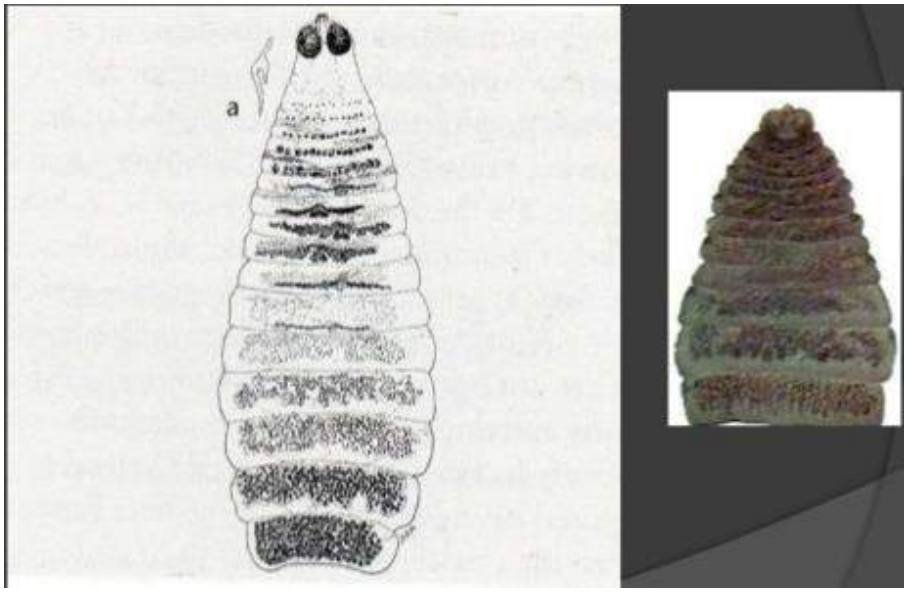
1. Rostellum is armed with two or more rows of rose thorn
2. shaped hooks.
3. Sucker are armed. Genital organs are single or double sets.
4. Uterus may be sac like or eggs pass in capsule or par uterine organ.
5. The species are found in birds.

Family_ Dilepididae: Uterus persists as transverse sac.

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.
- 

Family : Dipylidiidae

- In the members gravid uterus is replaced by egg capsule containing one or more eggs.

Genus : *Choanotaenia*

Species: *C. infundibulum*

- Found in the upper half of small intestine of fowl and turkey.
- Size: up to 23 cm. Segments are wide posteriorly
- Rostellum is armed with 16-20 slender hooks.
- G.Pore alternate regularly .
- Uterus is sac like.
- Proglottids leave host before maturity

Life cycle: *Musca domestica* and beetles of genera Calathus & Tribolium act as I.H



Hymenolepis carioca

Host	Chicken
Location	Small intestine
I/H	Dung bettles, flour beetles and *Stomoxys calcitrans*

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.

Host	Ducks
Location	Small intestine

I/H	Aquatic crustaeceans

H. lanceolata

Morphology

- Similar to *H.carioca*.

Life cycle

- 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.

Prepatent period

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

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.

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.
- Aricolinehydrobromide (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.

Order : Hymenolepididea

- Small to medium sized tapeworms.
- Rostellum is retractable with single circle of hooks.
- Scolex has four unarmed suckers.
- Single set of genital organs & genital pore unilateral.
- Eggs have three delicate membranes.
- Intermediate hosts are arthropods in which cysticercoid develops.

Family : **Hymenolepididae**
Genus: ***Hymenolepis***
Species ***H. nana*** *(Dwarf tapeworm of man)*

1. Found in small intestine of man and rodents.
2. They are 2.5-4cm. Long.
3. Eggs are oval; onchosphere has three pairs of hooks. It is most common tape worm of man in tropics and sub tropics.

Life Cycle:
Direct. In man cysticercoids develop in villi of small intestine and emerge out and develop in lumen as adult . P.P. 16 days. Auto infection has been recorded. In rodents life cycle can be direct or indirect where flour beetles and fleas act as intermediate host.

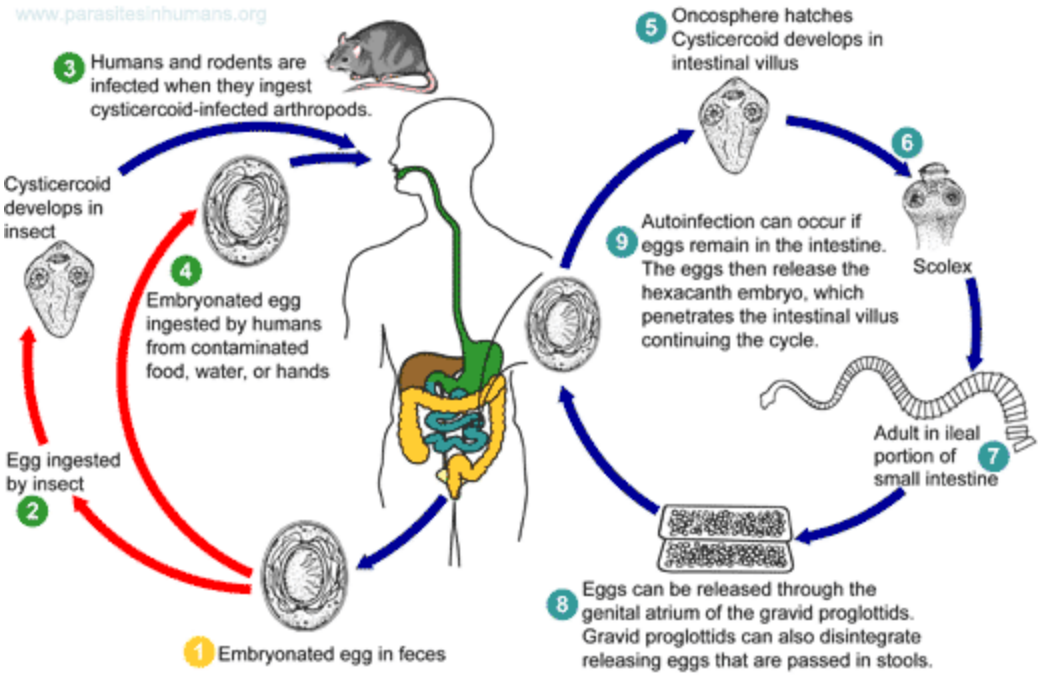
Pathogenesis

- Heavy infection in man may cause anorexia, vomition, diarrhea and pain

Diagnosis: eggs in faeces.

Treatment:
Rodents:

- Niclosamide 100-200mg/kg body weight in rats and hamster
- Bunamidine hydrochloride 200mg/kg bodyweight
- Praziquantel: 25 mg/kg body weight.

- 

-  *Hymenolepis nana* egg

Order Dilepididea

Family: Dipylidiidae
Genus : Dipylidium
Species*:* *D. caninum*

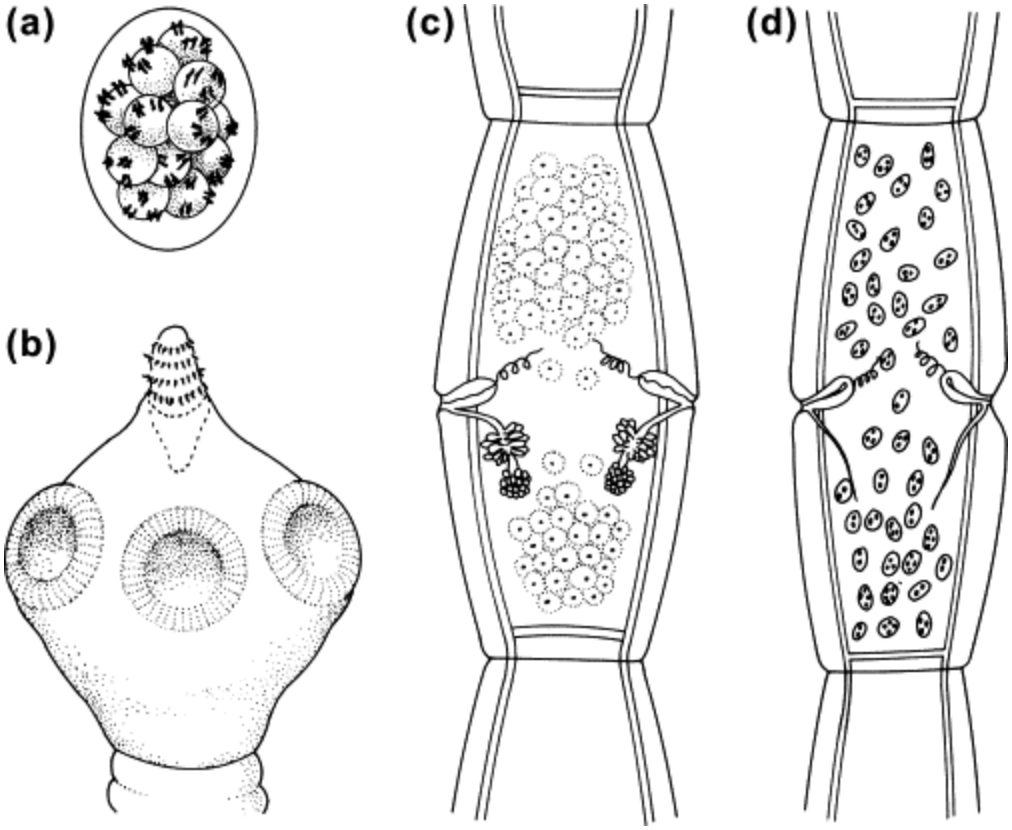
- Found in S.I. of dogs, cat, fox and occasionally in man.
- Worldwide in distribution,

- commonest species in dogs.

Dipylidium caninum

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

Morphology

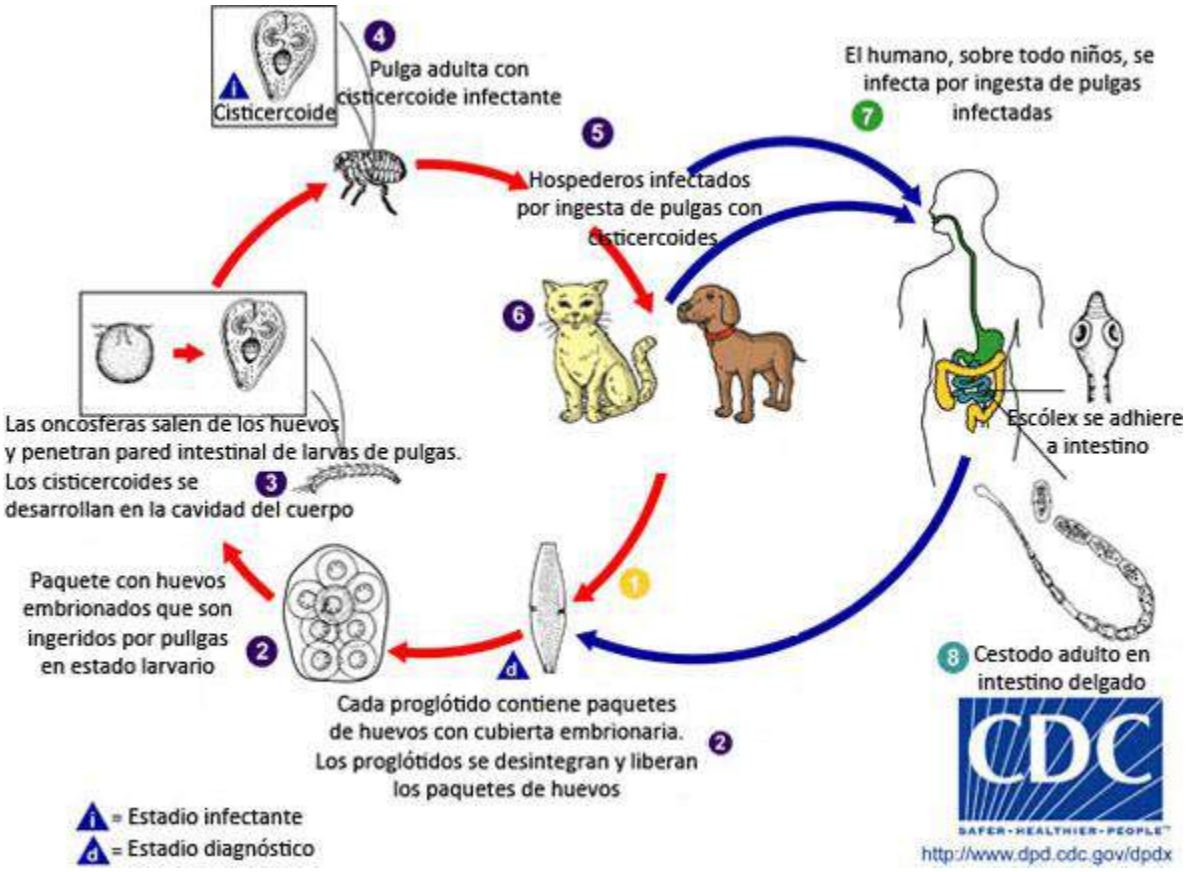


- Retractable rostellum armed with three or four rows of rose thorn shaped hooks.
- They are upto 50cm
- Each segment contains two sets of genital organ.
- Vitelline glands and ovary form a mass on either side resembling a bunch of grapes.
- In the gravid segment uterus are replaced by egg capsule or egg packets.
- Egg packets contain 30 eggs per packet. Gravid segments are elongate and oval in shape resembling cucumber seed shape.

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 cysticercoid 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



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".

Diagnosis and treatment

Diagnosis

- Demonstration of egg packets in faeces.

- Macroscopic examination of feaces for gravid segment.

Treatment

- Arecolinehydrobromide - 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 fo 5 days.
- Bithionol – 200 mg/Kg b wt.



***Dipylidium caninum* eggs**

ORDER : Taeniidea

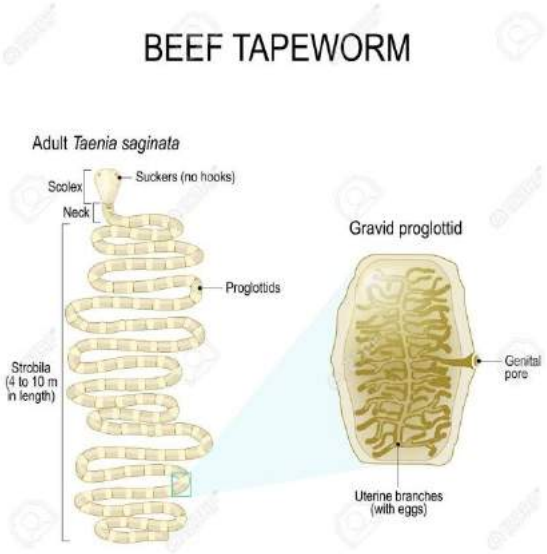
- It contains usually large tapeworms.
- Gravid segments are longer than wide.
- Rostellum may be absent or present (armed with double row of small and large hooks).
- Genital pore are single and alternate irregularly.
- Testes are in large number.
- Ovary in the posterior part of the segment.
- Uterus has median longitudinal stem and lateral branches.
- Eggs has outer envelop, inside it is inner envelope which develops into embryophore which is made of blocks and give characteristic appearance to eggs within this is present onchospheral membrane which surrounds the hexacanth embryo.
- Metacestodes are cysticercus, Strobilocercus, coenurus or a hydatid cyst.

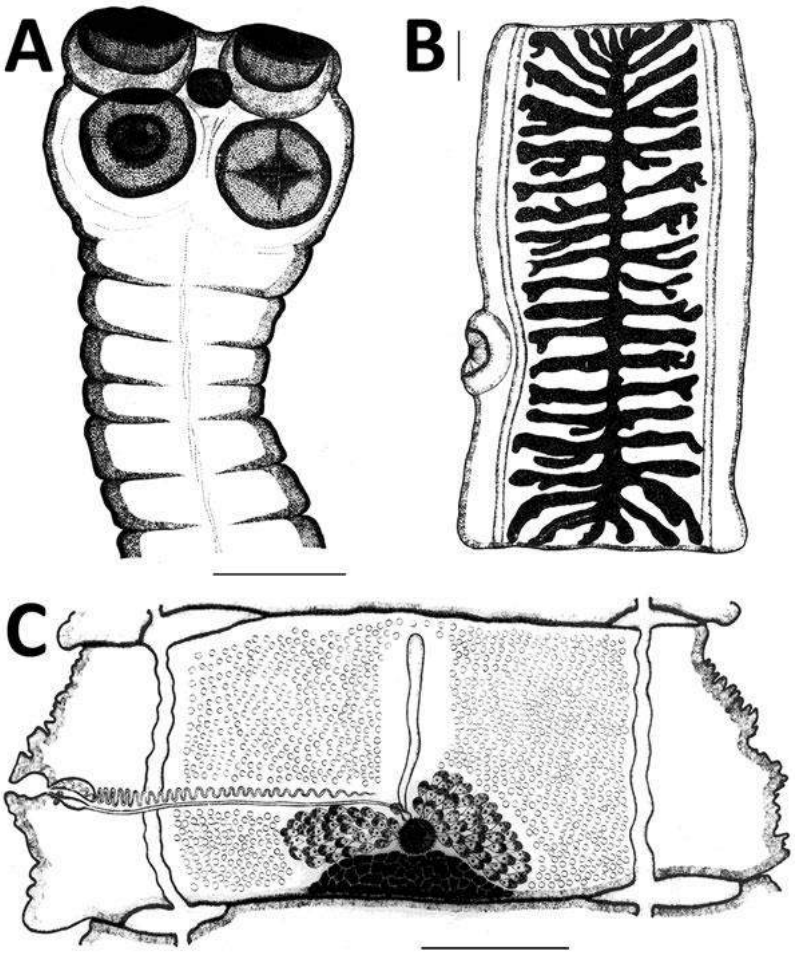
Genus: Taenia

Species: *Taenia saginata*

- Tapeworm is found in small intestine of man and *cysticercus* (metacestode) is found in cattle. Other animals such as llama, and reindeer may also serve as intermediate host. *T. saginata* survive many years in definitive host & cosmopolitan in distribution.
- It is most important in Africa, South America and Mediterranean countries.

Morphology:

- Adult worm is 4-8 meter long and may reach 25 meter.
- Scolex has 4 suckers without hooks.
- Rostellum is absent. (present in *T. solium* with 2 rows of hooks)
- Gravid segment contains 80000 eggs. (40000 in *T. solium*)
- Uterus has 14-32 lateral branches. (7-16 in *T. solium)*
- Vaginal sphincter muscle is present (absent in *T.solium*)
- Cirrus pouch does not extend to excretory vessel (reach in *T. solium*)
- Ovary is bilobed (trilobed in *T. solium*)
- Gravid segments leave host spontaneously (In *T.solium* they do not)
- 



A=scolex, B=gravid segment, C= mature segment

Life cycle:

- About 10 segments are shed daily which migrate out of anus. They are motile and migrate for few centimeters over body, clothed, grounds or beds and discharge the eggs. The eggs remain viable for several weeks- months in sewage in river or pasture. By variety of ways neonatal calves become infected such as if they are handled by infected person who also disperse eggs in pastures.
- The places which are closely infected are camping ground, spots visited by tourists, irrigation ditches, hay fields etc.
- Eggs can survive for 71 days in liquid manure, 16 days in city sewage 33 days in river water and 159 days on pastures. When eggs are ingested by cattle→onchosphere hatch→after activation by gastric and intestinal juice penetrate intestinal mucosa to reach in general circulation→disseminated throughout body and skeletal and cardiac muscles, in fat and other organs. Cysticerci are found in all muscles but muscles of heart, messeters, diaphragm and tongue have highest concentration *of Cysticercus bovis.* Cysticerci become infective in 10 weeks and remain viable for 9 months or longer. Man is infected by ingestion of raw or under cooked infected (measly beef) gravid segmens start passing after 100 days. Cysticerci degenerate 4-6 months after infection and by 9 months large number may be dead.

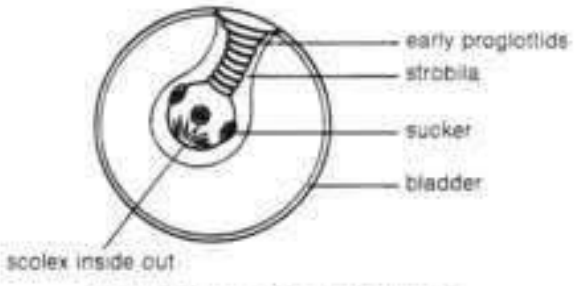
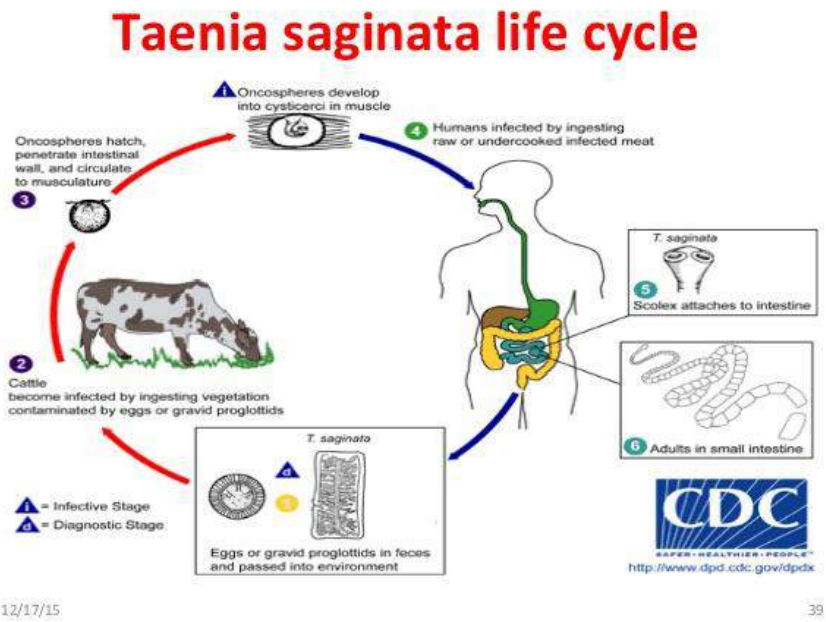


Fig. 136 **Cysticercus**. Generalized structure.

- In neonatal calf, cysticerci remain viable for longer period perhaps for whole life.

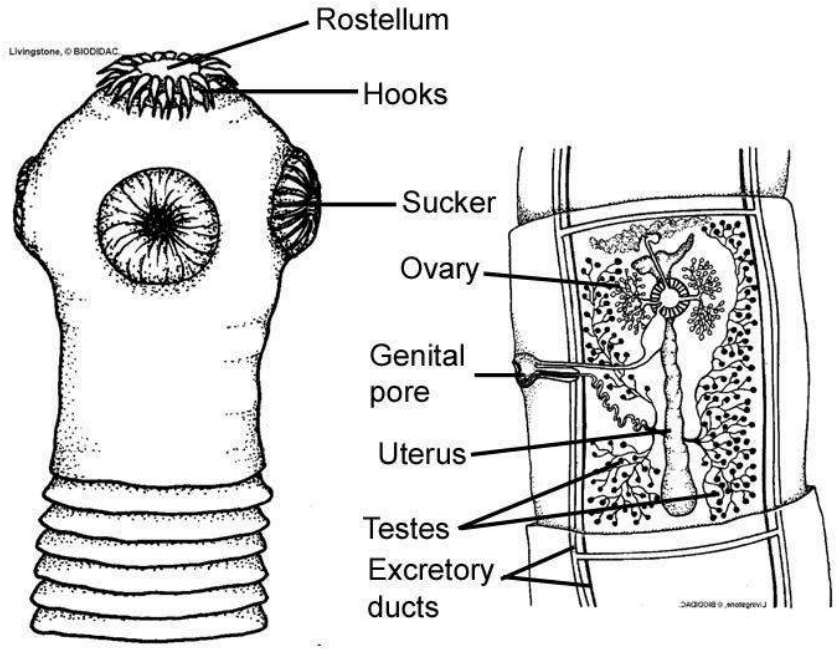


Taenia solium

- Small intestine of man. The pigs and wild boars act as intermediate host. Man act as definitive host.

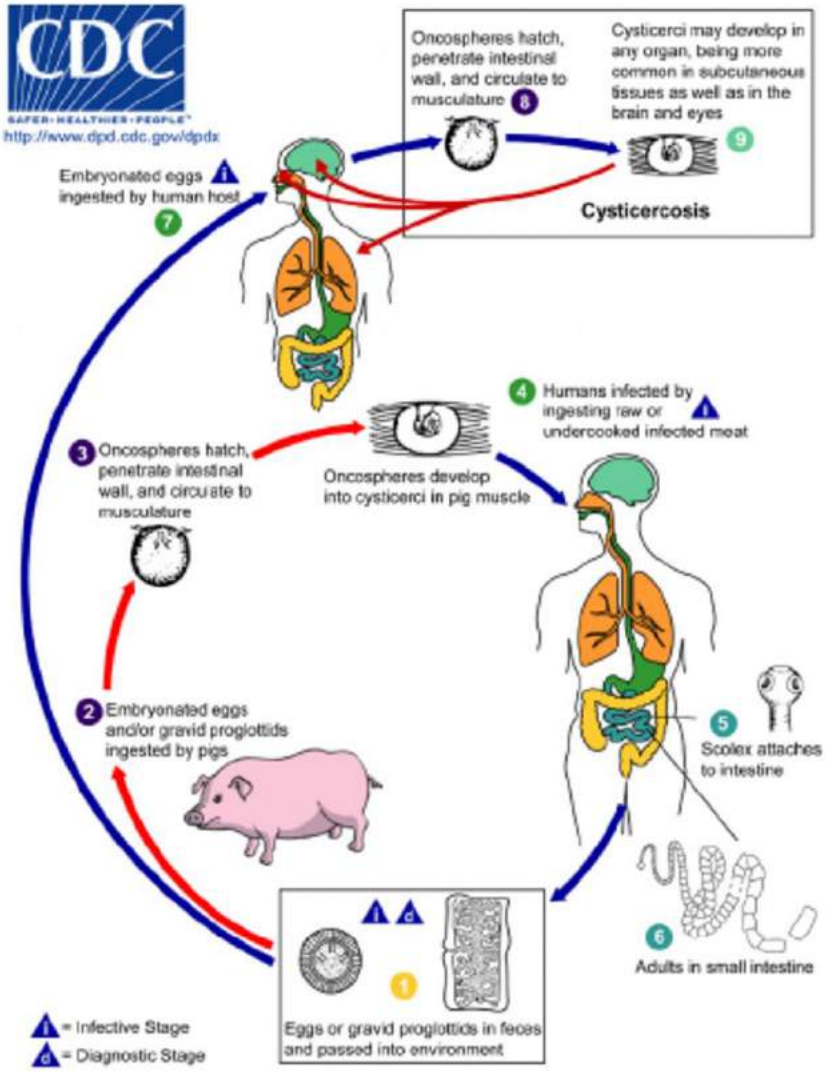
Morphology:

- Adults are 3-5 meters long and may reach 8 mater and may survive for 25 yars.
- Scolex bears a rostellum which has 2 rows of hooks.
- Gravid segment 10-12 meter long and 5-6 mm wide.
- Uterus has 7-16 lateral branches.
- Vaginal sphincter muscle is absent . Cirrus pouch reach to excretory vessel.
- Ovary is trilobed in *T solium*.
- Gravid segment has about 40,000 eggs.
- Gravid segments do not leave host spontaneously and voioded in facese in chain.
- Eggs are 26-34 µm in diameter in T. saginata they are 46-50 by 39-41 µm



Life cycle

- Life cycle is similar to *T saginata,* but here pigs act as intermediate host. *Cysticercus cellulosae* primarily develop in skeletal and cardiac muscles.
- Cysticerci are 20 mm long 10mm wide when fully grown and become infective after 9-10 weeks.
- Definitive host become infected when eat raw or under cooked pork.
- Man can act as intermediate host when ingest eggs with contaminated food and water or from dirty hands.
- In man cysticerci develop in s/c tissues, brain and ocular tissue. In brain, it develop in ventricle and proliferate.
- Infection causes pain, paralysis, epileptic attacks and death. Disease is called as neurocysticercosis. In Latin America 3 lakh people suffer from neurocysticercois every year.
- In pigs cysticerci are found in messeters, heart, tongue, shoulders but may be found throughout body.
- Longevity of cysticerci in pigs not known. Pigs are heavily infected as segments of *T solium* do not migrate away from the site of deposition.



-

Epidemiology:

- Infection of *Taenia solium* is common in pork eating countries and restricted in regions of low socioeconomic development. Infection is endemic in Latin America, South Africa, South- East Asia and Indian subcontinent. Common in those areas where pigs has easy access to human faeces.
- There are approximately 50 million cases of *Taenia solium* infection worldwide. This number is most likely an underestimate as many cases of *T. solium* are asymptomatic.
- Highest prevalence is found in areas where pork is undercooked or general environmental conditions are unsanitary. These areas of highest prevalence are Latin America, Southeast Asia, Africa, and Eastern Europe.
- Areas inhabited primarily by Jews or Muslims who do not eat pork have the lowest prevalence around the world, illustrating the necessity of the pig as an intermediate host to perpetuate the T. solium life cycle.
- In America- New York, southern California, and parts of Texas are all considered limited endemic areas. These areas have unusually high prevalence of *T. solium* due to their large immigrant population (especially immigrants from Mexico where *T. solium* prevalence is extremely high).

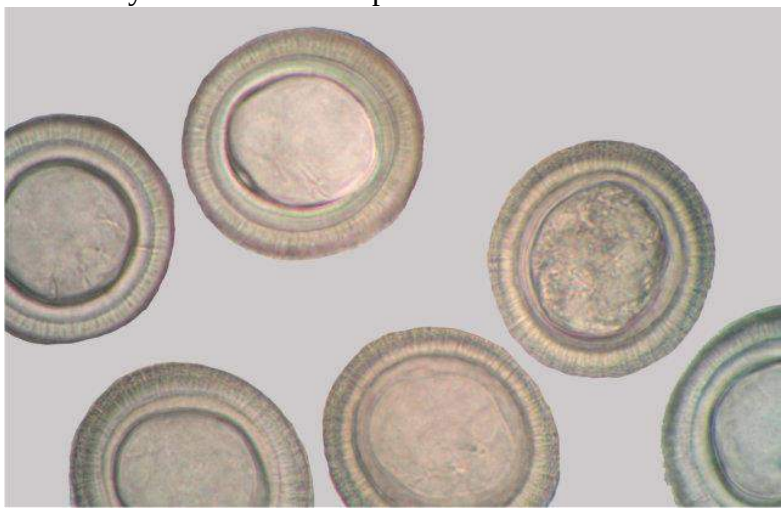
- It is also important to note that certain types of cysticercosis only have prevalence in certain areas. For example, Latin America has an overall high prevalence of cysticercosis, but subcutaneous cysticercosis is very rare.
- However in India subcutaneous cysticercosis has a relatively high prevalence (sometimes as high as 10%) even though overall cysticercosis prevalence in India is lower than that of Latin America.

Clinical signs.

Adult tapeworms of both species in man cause non-specific symptoms such as diarrhea, constipation and epigastric pain. Neurological symptoms may be found in man in neurocysticercosis. In pigs and cattle the infection is asymptomatic extremely heavy infection in cattle may cause myoisitis, myocarditis and stiffness in legs. Bovine and swine cysticercosis have major economic significance due to condemnation or dowm grading of infected carcasses.

Diagnosis

T. saginata

- Perianal swab is useful.
- Examination of faeces for segments.
- Ijn animals diagnosis of cysticercosis is usually during meat inspection.
- Antemortem examination / diagnosis with serological tests not possible due to non availability of sensitive and specific test.
- 

Treatment

- Adult parasites

1. praziquantel 10 mg/kg body weight
2. Niclosamide 2g/case is effective but scolex is partially digested.

Prophylaxis

1. Treatment of infected persons.
2. Proper meat inspection
3. Infected carcasses should be condemned for human consumption ot treated at -10 ºC for 10 days – 2 weeks or cooking at 50-60 ºC

Treatment of Cysticerci

At -5 ºC for 15 days.
-10 ºC for 10days.
-15 to -30 ºC for 6 days.
Old cysticerci are less effective for freezing.

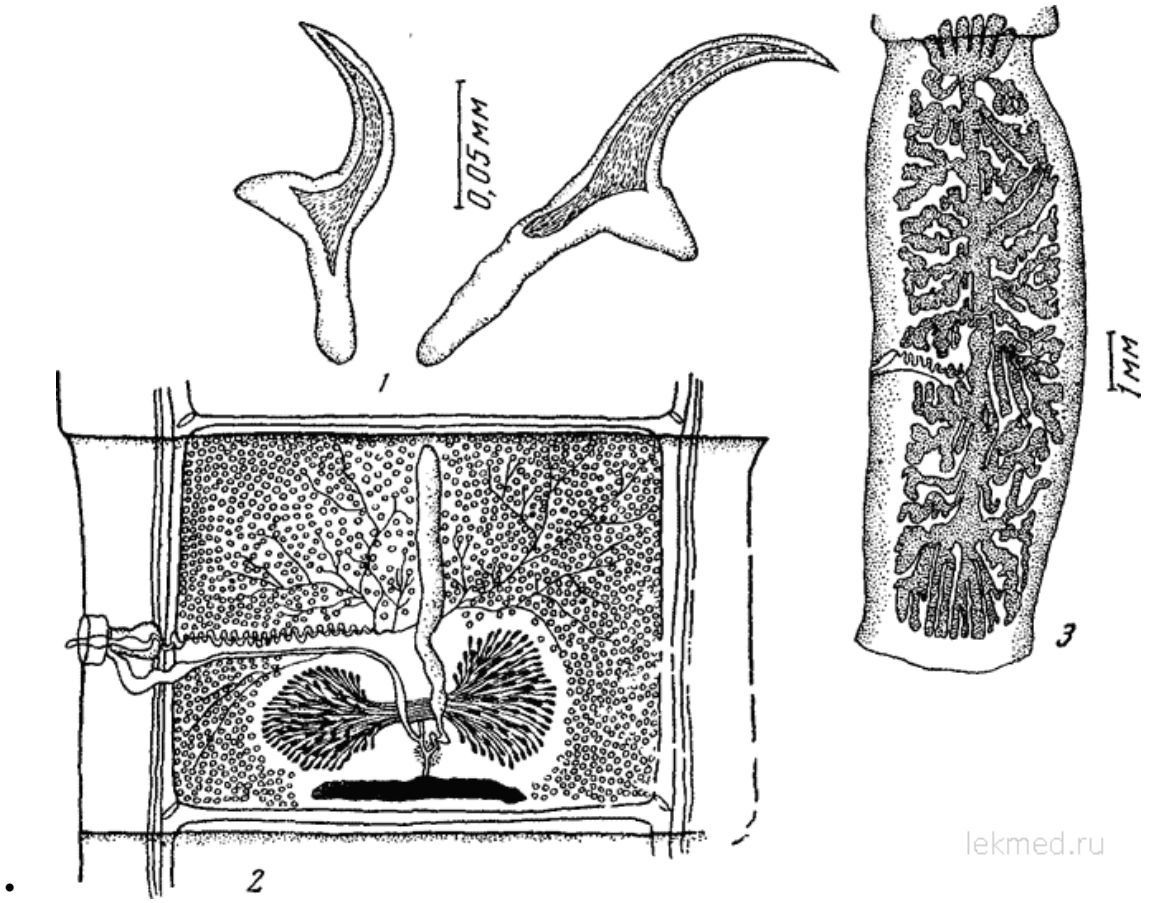
Family:Taeniidae

Taenia hydatigena

- Host: Dog and wild carnivores
- Location: Small Intestine
- Intermediate host: Domestic and wild ruminants (Sheep, cattle, sometimes pig)
- Metacestode stage: *Cysticercus tenuicollis*

Morphology:

- Found in small intestine of dogs, wolves and other wild carnivores.
- Adults are 75-500 cm long.
- Rostellum has two rows of hooks.
- Gravid proglottids are 12x6 mm
- Uterus has 6-10 lateral branches.
- Genital organs is single set and genital pore alternate irregularly.
- Testes are 600-700 in number.
- Ovary look like *T. solium*
- Vitellaria same as *T. solium.*
- Eggs are oval and measure 36-39x31-35 μm

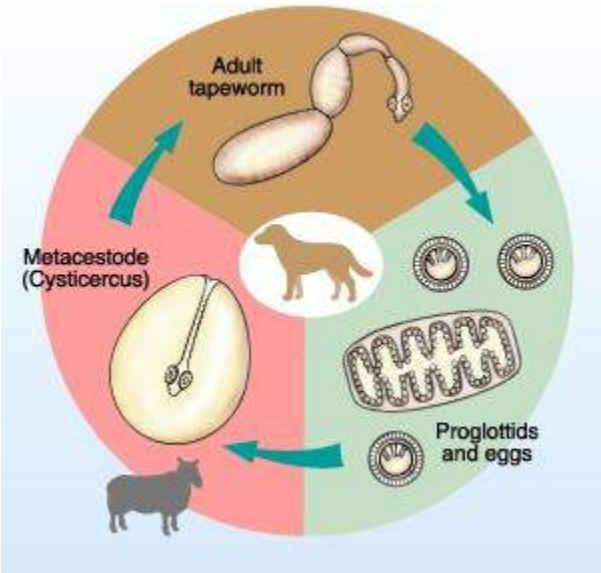


Life cycle:

Intermediate host are domestic and wild ruminants, particularly sheep. Pigs may also be infected. Ingested eggs hatch in small intestine and reach liver via blood and embryo migrate in liver parenchyma for 30 days causing hemorrhages and fibrosis. Developing cysticerci migrate to peritoneal cavity from 18 days - 4 weeks and reach to maturity between 34-53 days and found attached to greater omentum, intestinal mesentry and serosal surface of organs.

Cysticercus tenuicollis are upto 6 cm long and contain a single scolex which is invaginated in a long neck.

Carnivores become infected on ingestion of *C. tenuicollis*. In dog, prepatent period is 51 days. Dogs remain infected for a year or more.



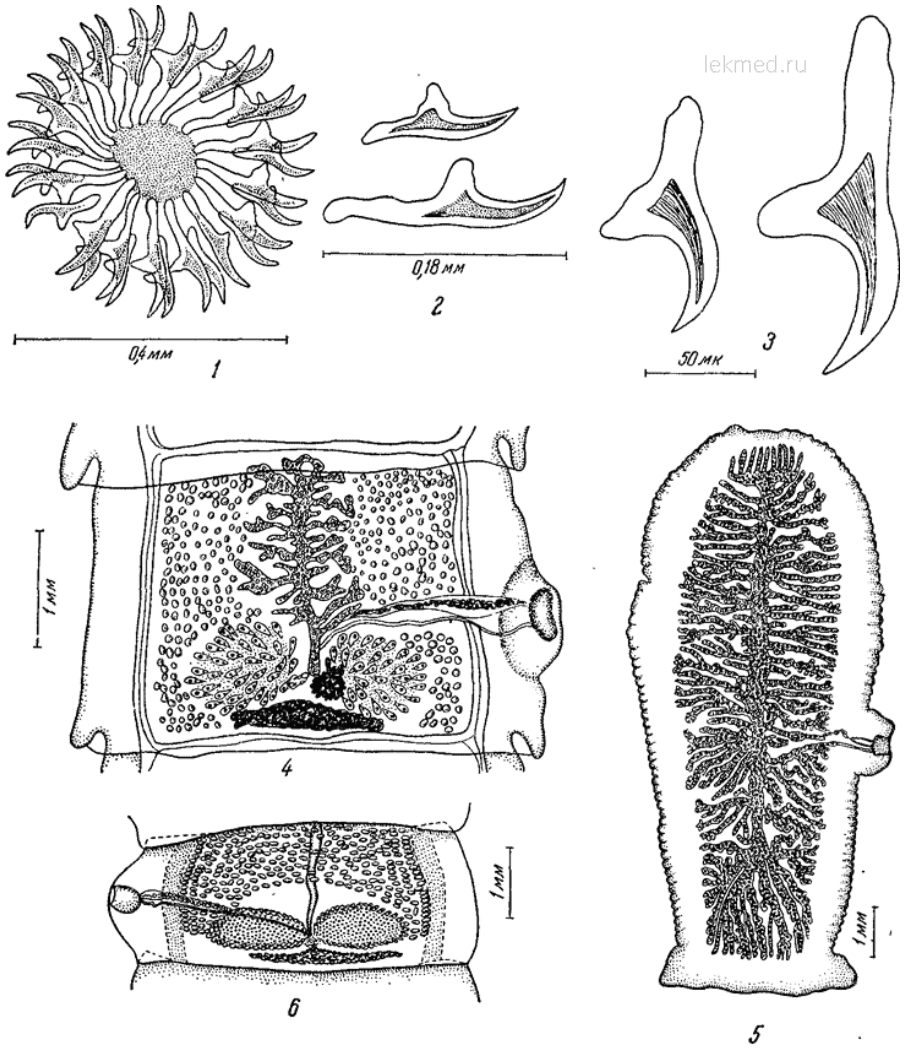
Pathogenesis and diagnosis

- 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.

Taenia ovis

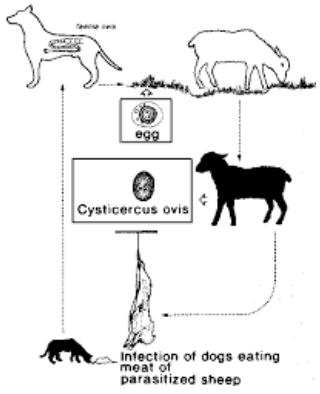
- It is a common tapeworm of dog and wild carnivores in most parts of the world. Intermediate hosts are sheep and goats in which metacestode (Cysticercus ovis) develop.
- Adults are 1-2 cm long,
- Uterus has 11-20 branched lateral branches.
- Eggs are oval, measure 19-31x24-26 μm.

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Life cycle:

- Similar to*T.saginata*. Onchosphere via blood disseminated and develop in cardiac and skeletal muscles. Heart, diaphragm and messeter muscles are important sites. Mature cysticerci about 6 mm long and infective at 46 days. After infection to dog Prepatent period is 60 days.



Pathogenesis

- Clinical signs are not usually observed in intermediate host. But presence of cystierci cause financial losses due to non-aesthetic appearance of meat.

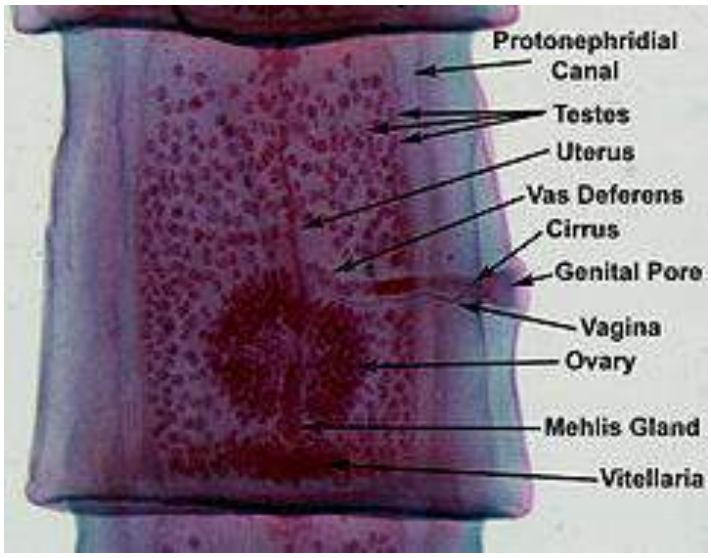
Taenia krabbei

- Tapeworm of wild carnivores and dog and metacestode (*Cysticercus tarandi*) found in reindeer and other wild ruminants. It is found in northern countries.

Taenia pisiformes

- Found in small intestine of dog, fox, wild carnivores and in cat. Intermediate hosts are lagomorphs mainly rabbit, hares and rodents (in case of cats).

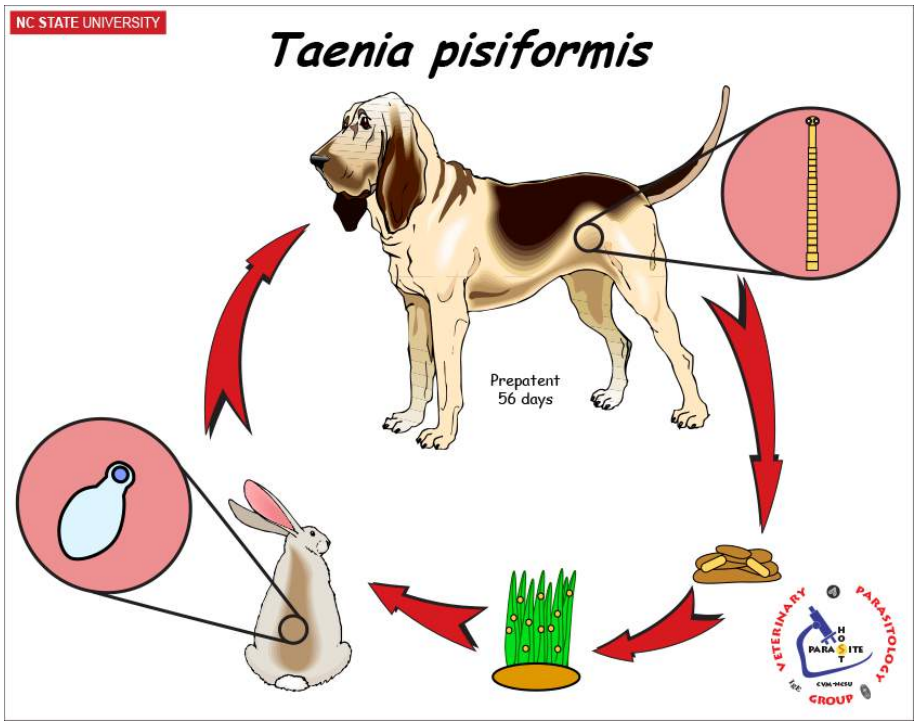
Morphology

- Measurement: upto 2 meter.
- Rostellum has 24-48 hooks in two rows.
- Gravid segments measure 8-10x4-5 mm
- Uterus has 8-14 lateral branches.
- Eggs slightly oval, measure 43-53x43-49 μm
- 

Life cycle:

- Similar to *T. hydatigena*

- Embryo after migrating and developing in liver for 15-30 days reach to peritoneal cavity and *Cysticercus pisiformis* (metacestode) found attached to the visceral organs. Cysts are of pea size.



Pathogenesis

- Light infection may cause digestive disturbances but heavy infection may cause severe damage to liver resulting in death.

Taenia taeniaeformis

- Found in small intestine of cat and related species. Larval stage is called *strobilocercus* found in rodents and lagomorphs.

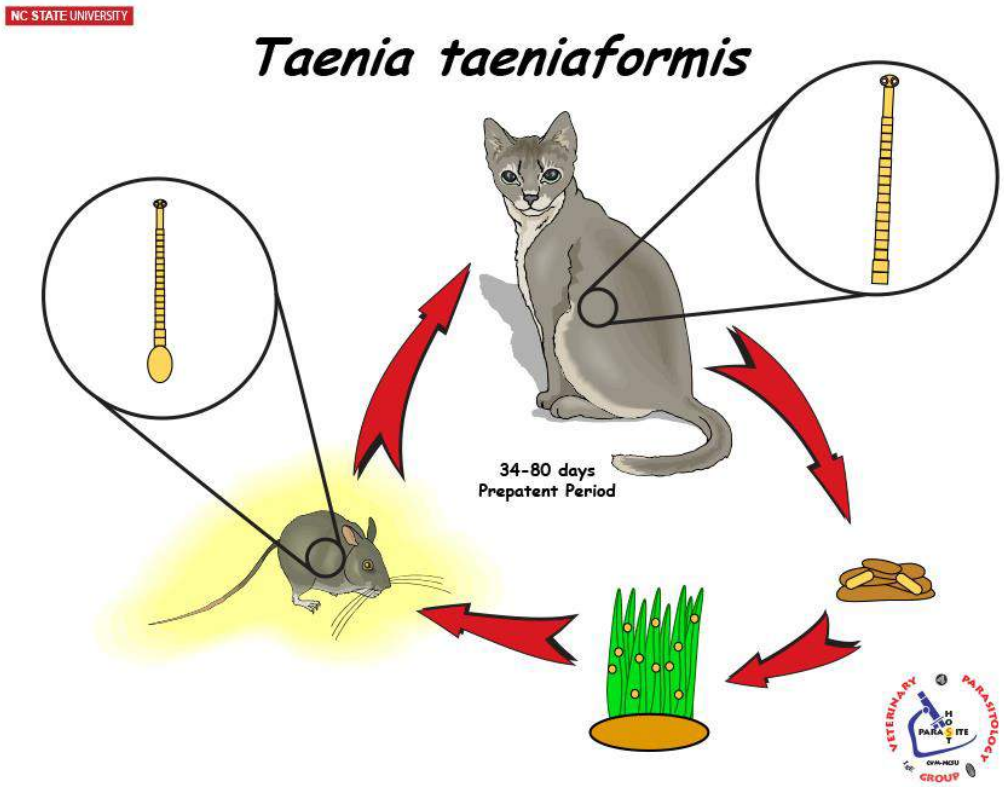
Morphology:

- Adults may reach upt 60 cm in length.
- They lack neck and have bell shaped posterior proglottids.
- Scolex is large and rostellum has two rows of hooks.
- Uterus has 5-9 lateral branches.
- Eggs are 31-36 µm diameter

Life cycle:

- After ingestion of eggs by intermediate host ,by day 30 ,*Cysticercus fasciolaris* develops and on day 42 scolex evaginates and is connected to bladder by segmented strobila looking like tapeworm. It is called *strobilocercus.*

- It is infected for cat after 60 days of infection. On ingestion by cat posterior bladder is dissolved, scolex attach to small intestine. Prepatent period is 36-42 days and cats remains infected for 2 years.



Taenia multiceps (Multiceps multiceps)

- Adults found in small intestine of dog, fox, jackal throughout the world. Intermediate stage *Coenurus* develops in the brain of sheep goat and other ungulates.

- Adults are 40-100 cm in length,

- Uterus has 14-20 lateral branches

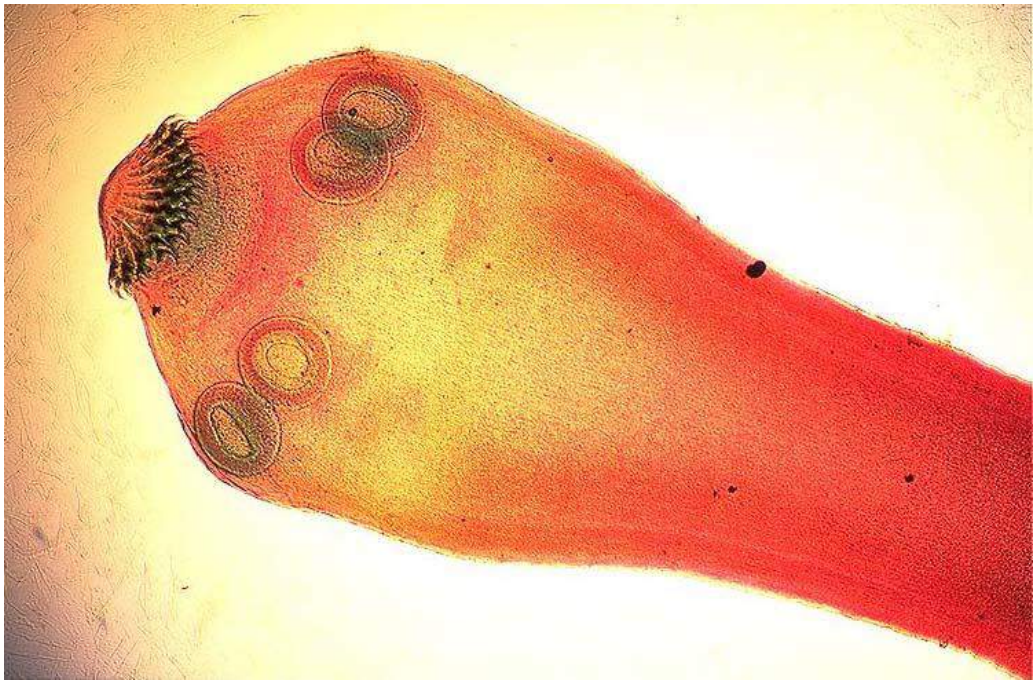
- Scolex pyriform and 1mm in diameter and four suckers.

- Rostellum armed with double rows of 22-32 hooks.

- Single genital organs set, genital pore alternate irregularly.

- Testes are 200/segment

- Vitellaria transversely elongated situated behind the ovary.



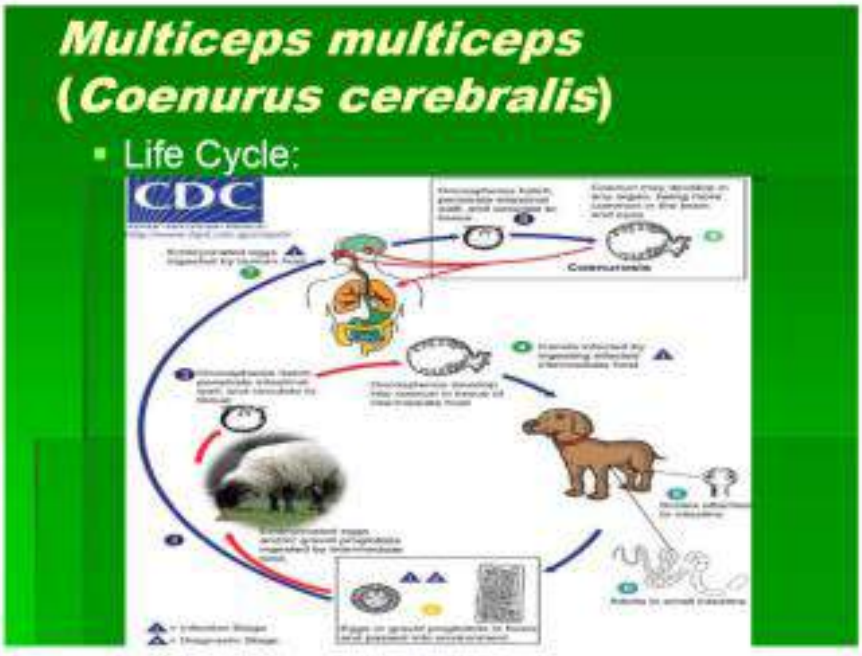


Mature segment

Life cycle:

- *Coenurus cerebralis* develop in brain and spinal cord of sheep and goats, cysts may be found in other organs, intermuscularly and subcutaneously.

- Developing stages first migrate in brain and spinal cord and produce tortuous yellowish grey to reddish straek. Mature cyst may reach 5 cm or more in diameter. *Coenurus* in infective after 6-8 months.



-

<u>Pathogenesis and clinical symptoms</u>

- Immature stage cyst may migrate in brain and cause meningeoencephalitis. In common cases or say commonly this infection is founs in chronic and associated with the presence of 1-2 coenuri in brain 4-6 months after infection.

- As cyst develop there is increasing degree of damage in brain. After neurological symptoms are called as **GID or Staggers.**

Symptoms:

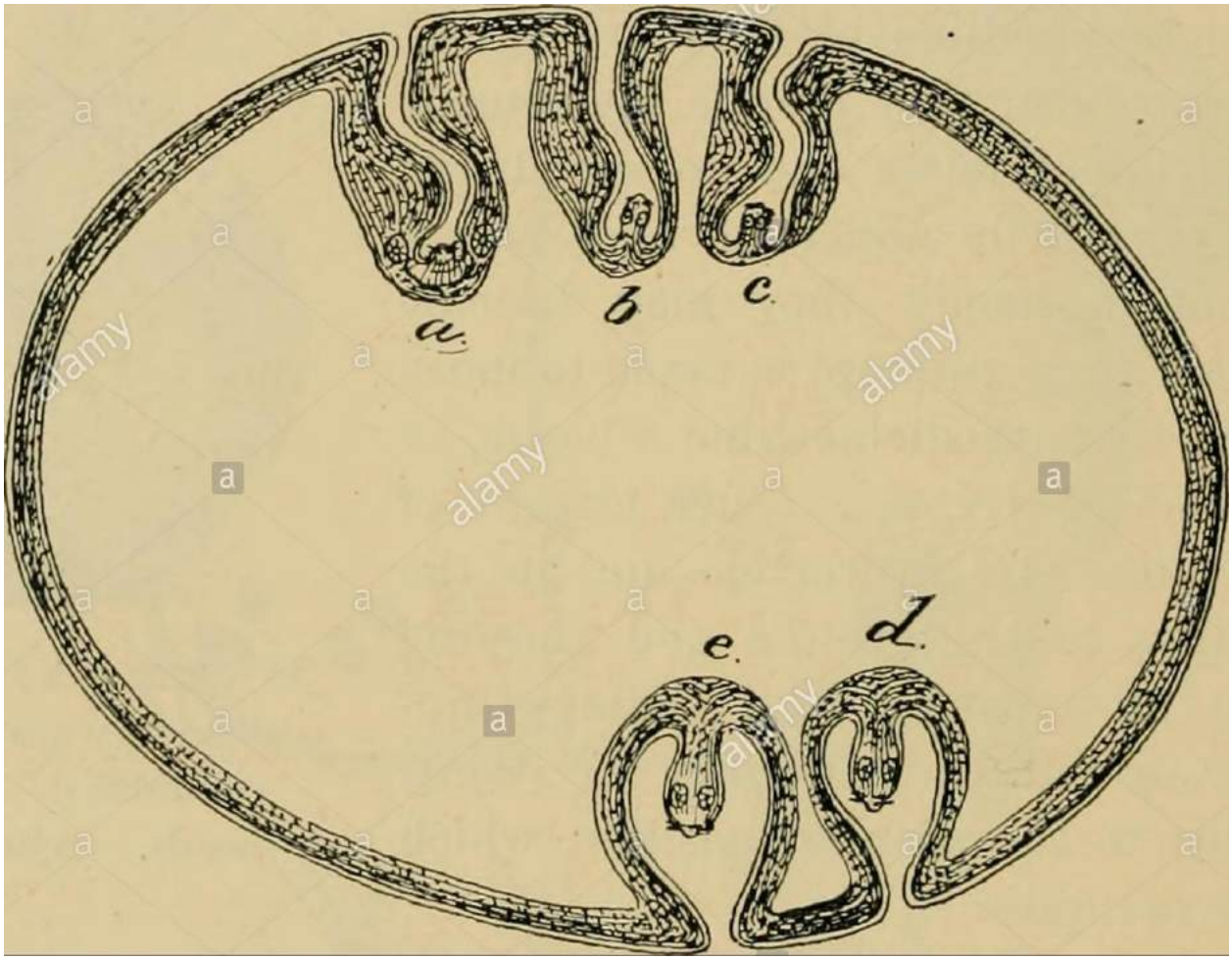
Depend upon the location of the cyst.

Most often cyst is found in parietal region on the surface of one of the cerebral hemispheres. In this situation animal holds its head on one side and turn in circle towards the affected side, may be blind in the eye of opposite side.

If cyst is present on anterior side of brain the head is held against the chest and animal steps high and walk in straight line until it meets an object, tehn remain motionless for some time.

- Reverse movements may be found if cyst is located in ventricle

- If cyst is found in cerebellum, then animal become hyperaesthetic and has staggering gait or prostrate.
- If in spinal cord, may cause paresis of one or both the limbs.
- Clinical signs are intermittent, cease to feed and become emaciated.
- If parasite present on the surface of brain, skull. Undergoes pressure atrophy, even perforated
- Such cyst may be palpated and by putting pressure symptoms may be incited.



Coenurus cerebralis

<u>Diagnosis</u>

- If on surface by palpation.

- If deep located, difficult to differentiate.

Treatment

- Surgical removal if parasite is present on surface.
- However albendazole may be tried in early stage of infection.

Taenia serialis

- Tapeworm found in dog and fox, cosmopolitan in distribution. Intermediate host are lagomorphs (rabbit and hare)in which *Coenurus serialis develops* in subcutaneous and intermuscular connective tissue.
- Tapeworm may grow up to 72 cm in length.
- Rostellum has two rows of 26-32 hooks.
- Gravid uterus has 20-25 lateral branches.

Coenurus may reach 4 cm in diameter or longer



Family: Taeniidae

Genus: *Echinoccocus granulosus*

- In this genus four species are accepted they are *E. granulosus, E. multilocularis, E.oligarthus and E. vogeli.*
- These species are differentiated on the basis of form of strobila, position of genital pore in mature and gravid segments. Number of testes, rostellar hooks and shape of uterus.

E. granulosus

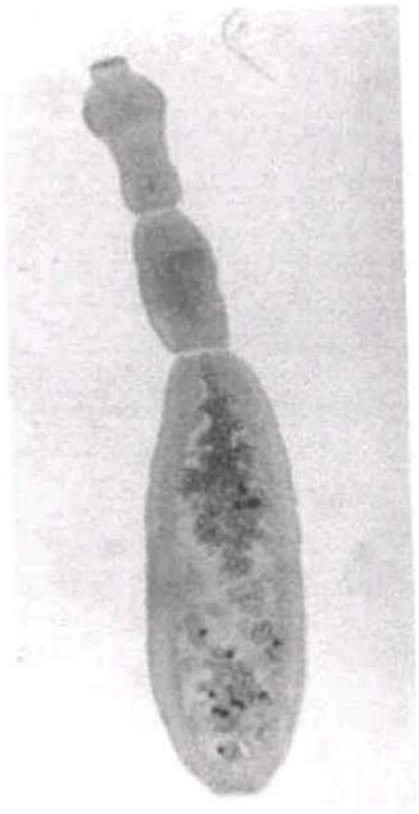
- Found in small intestine of dog and other carnivores.

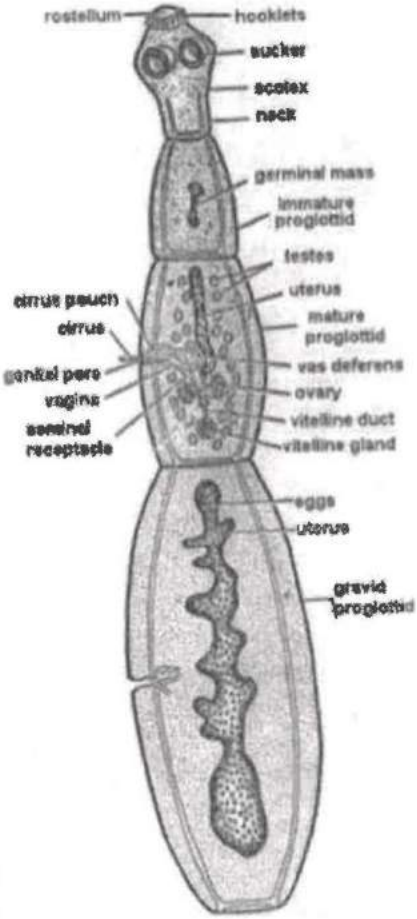
  larval stages(Hydatid cyst) is found in primates including Human being, marsupials, ruminants, lagomorphs, equines and occasionally in avian host

Morphology:

- Adults are 2-7 mm long and usually possess 3-4 (rarely upto six) segments
- Penultimate is mature segment.
- Gravid segment (last) about half of the total length of parasite.
- Rostellum has two rows of hooks.
- Ovary is kidney shaped.
- Vitelline gland is large and spherical.
- Genital pore alternate irregularly and open in posterior half of the mature and gravid segments.
- Uterus (gravid) has well developed diverticula
- Uterus usually disintegrate in intestine and only eggs are found in faeces.
- Eggs like Taenia (32-36x25-30 μm)

E. granulosus



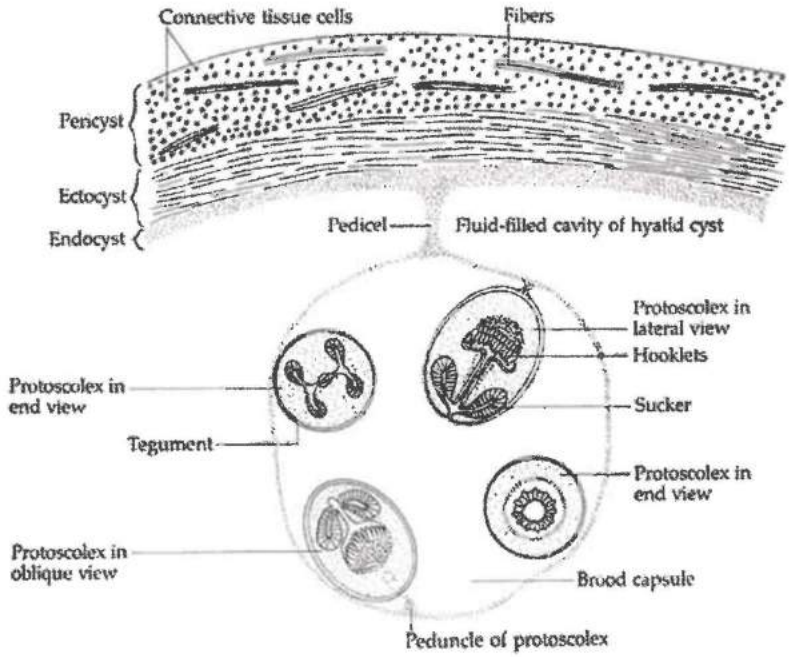


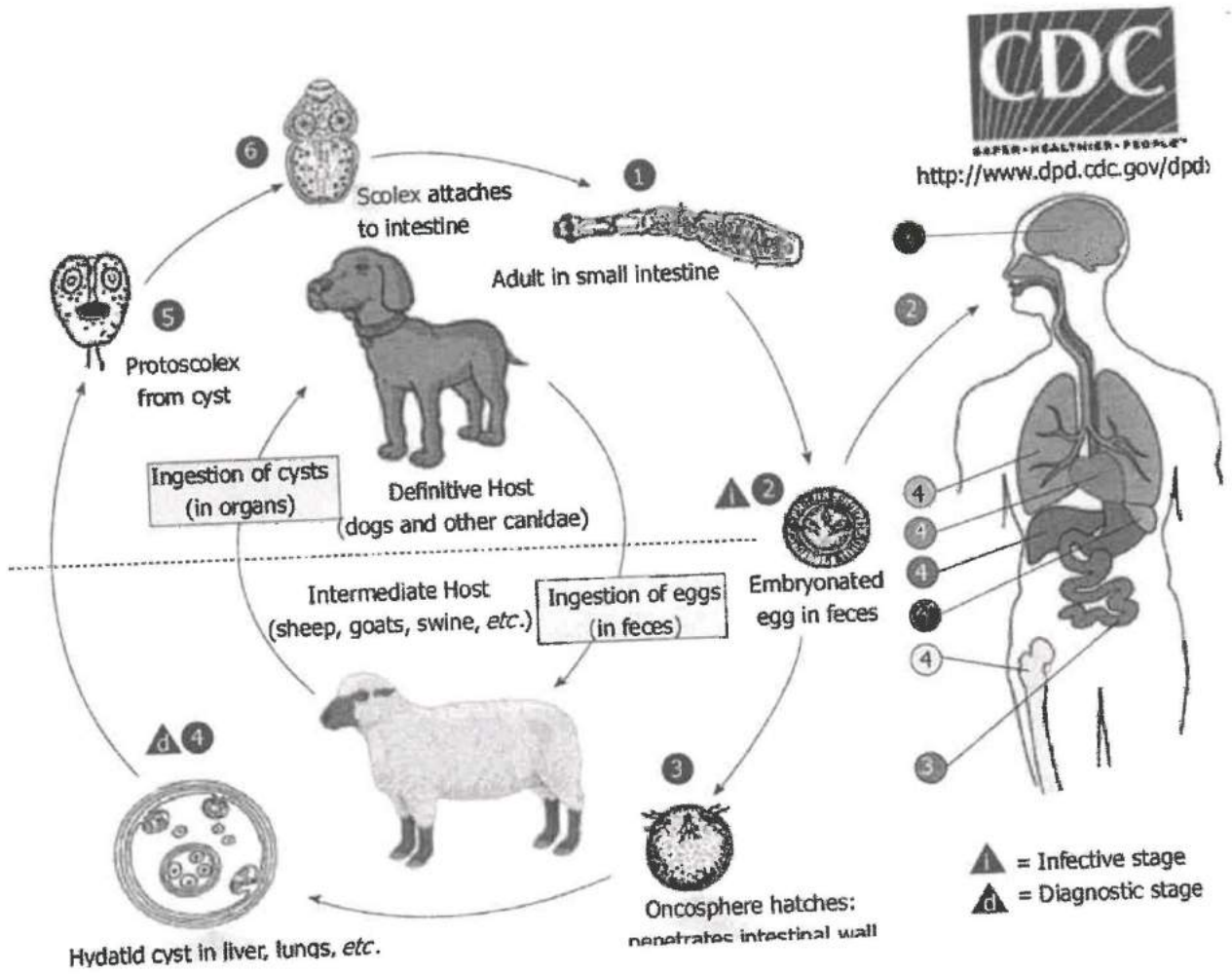
Life cycle:

- Eggs are present in faeces of carnivores and immediately they are infective and on ingestion by ungulate onchosphere penetrate intestinal vein and lymph vessels and reach to lung, liver and other organs. Hydatid cyst develops slowly over several months. These cysts are 5-10 cm in diameter (in man cyst of 50 cm diameter and 16 liter fluid)
- Hydatid cyst are unilocular and composed of thick outer laminated membrane and within this a germinal membrane from this brood capsule, each containing protoscolices develop after 5 months of infection. At this time cyst is infective for definitive host, brood capsule detach and float in cyst fluid called as hydatid sand.
- Occassionally daughter cyst develop into other cyst within. Sometime when cyst rupture protoscolices and brood capsule can develop into other external daughter cysts.
- External daughter cyst may also form, if a piece of germinal membrane become enclosed in laminated layer.

- When dog ingests the cyst/protoscolices, tapeworm is produced in dog in about 47 days. Dogs remain infected for about 2 years. In heavy infection intestine may be carpeted with worms.
- Hydatid cysts which produce brood capsule are called as fertile and those do not called sterile cysts.
- It was found 27% of horse hydatid cyst and 51% of sheep hydatid cysts are to be sterile.
- Cyst in cattle are frequently sterile, pigs usually have sterile cysts.
- Hydatid cyst in sheep generally found in lungs where they are frequently multilocular.
- In Pigs, cyst found in liver and lungs,
- Cattle/ horse primarily in liver.
- In horses cysts are unilocular.
- In man may be found in variety of organs.
- Cyst fluid is pale yellow in colour and has 17-200 mg protein/100ml. it has similarity with serum and has immunoglobulins.

Hydatid cyst





Distribution : cosmopolitan

- Endemic in previous USSR, Mediterranean countries and areas of Africa, Latin America and Australia.

E.multilocularis

- Found in northern hemisphere, central and eastern Europe particularly Canada and earlier USSR. Adults found in foxes.

Morphology:

- Adults are 1.2-4.5 mm (smaller than E. granulosus)
- They have 2-6 progl;ottids.
- Rostellar hooks ate 14-34 μm
- Uterus does not have lateral branches in gravid segments.

- Genital pore near anterior margin.
- Testes are 14-35 in number.

Life cycle:

- Adults are found in foxes and cyst which alveolar hydatid cyst found in rodents. Cysts is multivesicular cyst. Field mouse, vole, ground squirrel, man) are main intermediate host.
- These intermediate host are called as micotine rodents.
- For man, main sourse of infection is fallen fruits and vegetables contaminated by faeces of foxes.

Echinococus oligarthus

- Found in wild felids Puma ,jaguar and Geoffroys cat in central and south America. Rodents serve as intermediate host .

Morphology

- measure 1.9-2.9 mm and adult possess three segments and penultimate is mature.
- Genital pore anterior to middle in mature segment and at middle in gravid segment.
- Testes 15-46 in number.
- Gravid uterus sac like.
- Rostellar hooks are 26-40 in number
- The hydatid cyst is polycystic. Cysts have tendency to septate and form multichambered growth.

E. vogeli

- Tapeworm found in central and northern south America. Definitive host: domestic dogs and bush dogs and rodents act as intermediate host.

Morphology

- They are 3.9to 5.6 mm long and has three segments and penultimate segment is mature.
- Genital pore in mature and gravid segment is siruated posterior to the middle.
- Testes are 50-67 in number.
- Gravid uterus has no lateral branches.

- Rostellar hookjs are 28-36 in number.
- Cystic stages are polycystic.

Pathogenesis of Tape worms:

- Adult tapeworms are comparatively harmless, however in large number *Echinococcus* may cause enteritis in dog.
- Pathogenesis of cyst (hydatid) depend upon severity of infection and organ infected. In animals clinical signs are not usually observed inspite of heavy infection.
- In man, symptoms are pronounced and function of organ is frequently impaired. Rupture of cyst lead to fatal anaphylactic shock, and released protoscolices and brood capsule develop into numerous daughter cysts.

Diagnosis: difficult to differentiate the eggs from *Taenia* spp. For this dog must be purged with arecoline hydrobromide (1-2mg/kg) and the mucous portion of the purged material is examined for minute adult parasites.

- Diagnosis of hydatid in animals is usually done during autopsy.
- Radiographic examination is also helpful to detect thoracic hydatid cyst in sheep.
- In man Immunodiagnostic tests such as CFT, indirect haemagglutination (IHA) CIEF (counter immune electrophoresis, RIA (radioimmune assay) and ELISA has been used with success. Besides these ,radiographic and ultrasound examination are most useful.

TAPEWORM INFECTION IN DOGS AND CATS

- Normally, infection with adult cestodes are not very harmful in dogs and cats. However, heavy infection in young animals can cause non specific abdominal symptoms. There may be diarrhea or constipation and animal may have pot bellied appearance.
- Rarely there may be obstruction of intestine. Gravid segments,particularly of *D.caninum* migrate out of anal sphincter and in perianal area and may cause irritation which may lead to dragging of anus over the ground.

Diagnosis

- .Many infections are usually diagnosed on presence of proglottids in faeces or in surrounding. Differentiation is based on examination of these segments.
- Egg capsule of *D. caninum* contain upto 30 eggs on rupture releasing eggs.

- Proglottids of *Taenia* species are differentiated on presence of single eggs (not in capsule) with thick radially striated embryophore.

Treatment

Arecoline Hybrobromide 1-2 mg/kg bodyweight

Bithional: 200 mg/kg bodyweight

Dichlorophen 0.3 mg/kg bodyweight

Bunamidine hydrochloride 25-50 mg/kg bodyweight

Niclosamide 100-150 mg/kg bodyweight

Praziquantel 5 mg/kg bodyweight

Metacestodes:

Albendazole 50 mg/kg bodyweight

Praziquantel 50 mg/kg bodyweightat asingle dose

Mebendazole 50 mg/kg bodyweight for 14 days Bid

Metacestode Infection in Intermediate host.

- The spread of metacestode infection in human being can be checked by adopting proper washing of fruits and raw vegetables before consumption. Regular checkup ant treatment of pets, restricting stray dogs and cats in premises

Titender

Class: Cotyloda

Order: Diphyllidea

- Order contains small to medium sized tape worms.
- Suckers are replaced by deep narrow muscular groove one on dorsal side and one on ventral side calledas bothria.
- Rostellum and suckers are absent.
- One set of reproductive organs in one segment.
- Testes are numerous and vitellaria are scattered.
- Ovary is bilobed and uterus form spiral tube.
- Uterine pore opens on the ventral side of proglottid.
- Eggs are operculated & unembryonated when laid.
- Life cycle include free living Coracidium (a ciliated embryo), a Procercoid found in crustaceans (1st I.H.) , and Plerocercoid found in fish (2nd I.H.).

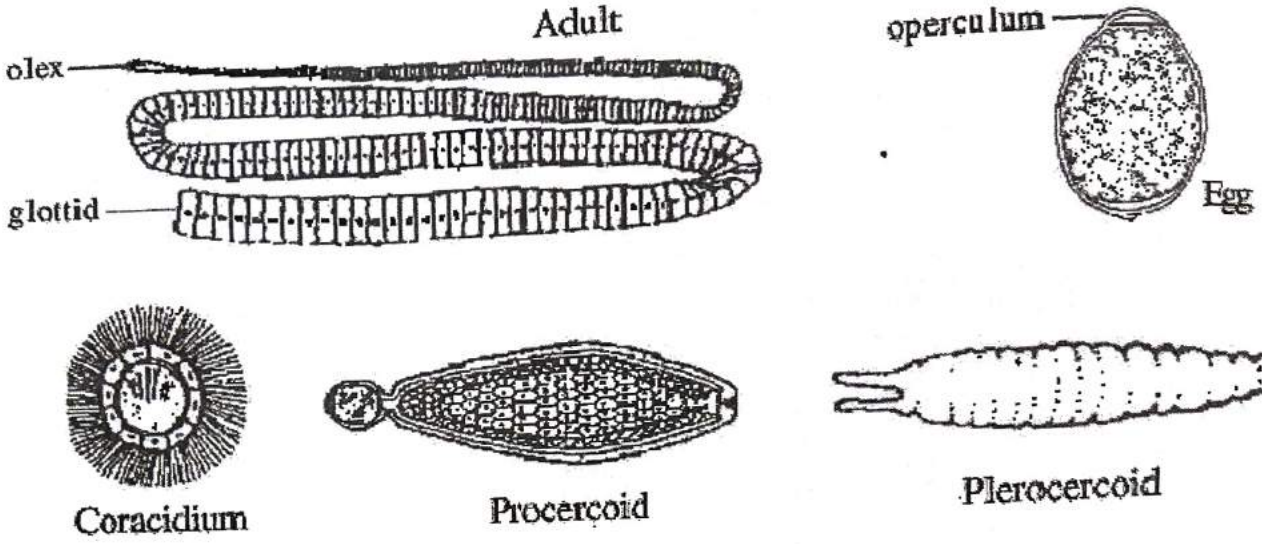
Genus: *Diphyllobothrium*

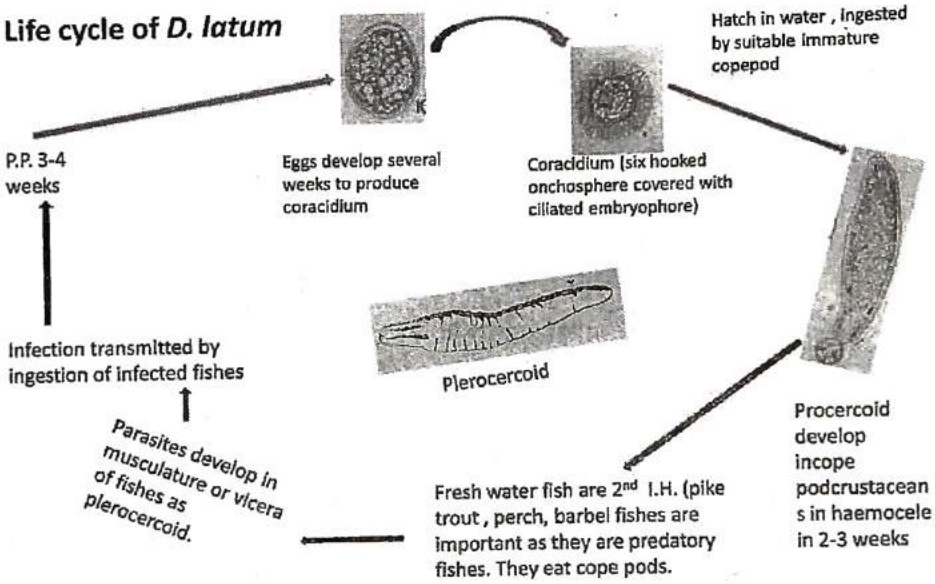
Species: *D.latum (broad fish tapeworm)*

- Found in small intestine of man , dog , cat, polar bear and other fish eating animals in many parts of the worlds including India.

Morphology:

- Adult are 2-12 mts. Long & 10-20 mm wide, yellowish in colour with dark central marking caused by uterus and eggs.
- There are numerous testes & vitellaria lies in central field.
- Uterus is coiled and in centre and open ventrally in uterine pore which lies immediately behind the genital pore.
- Eggs are passed continuously in faeces which are Light brown in colour, operculated measuring 67-71 X40-51 um.





Pathogenesis :in man cause non specific abdominal symptoms and as parasite compete for Vit. B12 so causing macrocytic hypochromic anaemia

Diagnosis : symptoms and by finding operculted eggs in faeces.

Treatment: In dogs same as for other tape worms. Praziquantel and niclosamide are more effective compounds.

Control:

1. Proper cooking of fishes.

2. Freezing of fishes

3. Avoid feeding raw fishes or fish offals to dogs and cats.

Genus: Spirometra

The parasites of this genus are small to medium size tapeworms. Morphologically similar to *D.latum.*

- Uterus is spiral instead of rosette shaped.
- Uterus and vagina open seperately on ventral surface of proglottids.
- Eggs have pointed ends rather than rounded ends as in ***D.latum.***
- Adult parasites are found in wild carnivores and in domestic dogs

Species:

- ***Spirometra mansonoides*** (found in cats and dogs). It is pink in colour, found in jejunum of cats and dogs & live for several years.
- ***S. mansoni*** (cats and dogs).
- ***S erinacea***

Life cycle:

- Eggs are passed in faeces and develop to produce coracidium which hatch and swallowed by fresh water crustaceans (cyclops). Procercoid is produced in 10-14 days. 2nd I.H. Are water snakes , tadpoles or other amphibians, birds, mammals and man in which plerocercoid develop. They also called spargana. Plerocercoid white ribbon like structure which may reach several cm. in length. Cats become infected by ingestion of infected 2nd I.H.. P.P. 10-30 days. Dogs and cats may also be infected with plerocercoid.
- **Pathogenesis:** adult is non pathogenic in most of cases but plerocercoid are of public health significance causing sparganosis in man. Man may become infected by following ways.

1. By accidental ingestion of crustaceans infected with *procercoid. Procercoid* migrate in S/C tissue and musculature.

2. By ingestion of *plerocercoid* in 2nd I.H. Pigs which are raised for human consumption may become infected. When infected pork is eaten as raw these *plerocercoid* are usually mistaken for nerves. After ingestion they migrate to tissues and re-establish .

3. When infected frog or snake flesh is used as a dressing for wounds or eyes , the plerocercoids migrate in flesh or in eyes.

In man spargana migrate subcutaneously causing inflammation, urticaria, oedema and eosinophilia. Spargana may also found in muscles or in periorbital area.

Treatment: In man surgical removal of plerocercoid .

In dogs, treatment similar to adult tapeworms

Class Trematoda

Characters of Class Trematoda:

1.The parasites are dorsoventrally flattened , unsegmented and leaf like.
2.they do not have body cavity and all organs are embedded in the parenchyma.
3.they have suckers , hooks and clamps as organs of attachment.
4.They have mouth , oesophagous , pharynx and intestinat caeca which may be branched but there is no anus.
5. They have branched excretory system , flame cells and excretory bladder which has a posterior opening.
6. They are hermaphrodite except the members of family Schistosomatidae.
7. Life cycle may be direct or indirect.

The class trematoda is further devided into two sub classes.
Sub Class : Monogenea and
Sub Class : Digenea

Characteristics of Sub Class Monogenea :

Monogenean trematodes are parasitic in cold blooded aquatic or amphibious vertebrates or sometimes in aquatic invertebrates. Mainly they are ectoparasites of gills , skin , fins and/or buccal cavity. In these parasites there is a posterior adhesive organ called as haptor which has suckers , hooks and clamps . haptor has great significance in identification of these species.

The life cycle is direct. The eggs are usually operculated and its both or one pole has filamentous structure which help in attachment of these eggs to the host or other objects. The larvae is called as oncomiridium and after hatching it bears cilia .

Characteristics of Sub Class Digenea: Flukes under this sub class are parasitic in domestic animals and birds.

1. Generally digenetic trematodes are dorsoventrally flattened. Morphologicall they are leaf shaped , some are long and narrow(schistosomes) while others may have fleshy bodies like amphistomes .
2. The tegument of flukes is a metabolic active surface which may be smooth or spiny.
3. The organs of attachment are anterior sucker(oral sucker) located at the anterior end and ventral sucker or acetabulum found usually at the anterior third of ventral side but its position may be variable and in some may be absent.

4. Digestive system opens at mouth which is surrounded by the anterior sucker. Posteriorly there is muscular pharynx then a oesophagus, this leading into a intestine which usually divides into two blind caeca which may be branched in few species. Epithelial cells lining the gut caeca , have both absorptive and excretory functions. Only a few species of trematodes may have an anus.

5. The parasites utilize blood, mucous, host tissue and gut contents in theit diet. The food is drawn into parasite by combined action of oral sucker and pumping action of muscular pharynx and /or oesophagus.

6. They have developed excretory system consists of a bladder which may be from sac like , in simple form to various shapes and opens to the posterior extremity of the body.This bladder is connected with numerous blind ending tubules are embedded in parenchyma which end in flame cells which are terminal organs. These cells collect the waste products from its surroundings which is propelled in connecting tubes with the help of cilia present in these cells and finally these substances from different tubes are accumulated in bladder. The number of flame cells and arrangement of tubes is characterstic and used in diagnosis of species.

7.They are hermaphrodite except the members of family Schistosomatidae where the sexes are separate.

8. Nervous system comprises of cicumoesophageal ring of fibres and paired ganglia from where 3 pairs of nerves run forward and 3 pairs backward to the all parts of the body. Sence organs as such are not present in adults , although free living larval forms(miracidium and cercaria) may be provided with patches of pigments called as eye spots.

Life cycles are indirect.

Reproductive system in digenic trematodes : Digenetic trematodes are hermaphroditeexcept schistosomes which are unisexual

The male reproductive organs consists of two testes , which may be spherical , lobed , branched or devided into number of bodies. From these testes vasa efferentia rise which unite to form vas deference which widens distally to form vesiculum seminalis. This duct latter on becomes narrow and surrounded by prostate glands and finally ends in cirrus. These terminal structures from vesiculum semanalis to cirrus are enclosed in a sac like structure called as cirrus sac. The

genital pore which usually opens interiorly towards ventral side but its location may be variable. Genital pore is surrounded by genital sinus which is also called atrium in which female pore is also situated.

Female organs consists of an ovary which discharge the ova into oviduct. Oviduct bears a pouch like structure called as receptaculum seminis.

There is a pair of viteline glands which consists of number of follicles which situated in the lateral field of body and discharge into yolk duct which joins the oviduct at wider portion that is ootype in which the eggs are formed. The ootype is surrounded by unicellular glands called as Mehlis's glands. The shell of the egg is formed by the material contributed by mehlis's glands and yolk glands.

In trematodes self fertilization usually takes place, Cirrus evaginates and enters into the uterus.

On leaving the ootype eggs enter in the uterus which opens at the genital pore. In some species, the distal part of uterus form a muscular portion called as metraterm. Eggs usually have an operculum and many species develop in uterus.

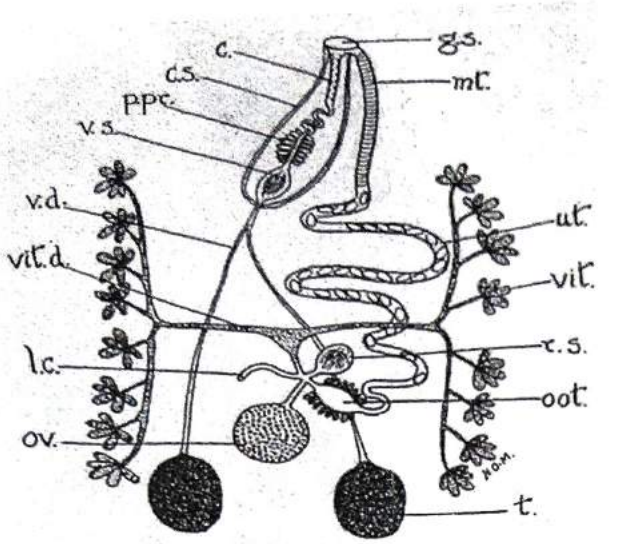


Fig 1.4 Reproductive organs of a trematode.

c	cirrus
cs	cirrus-sac
gs	genital sinus
lc	Laurer's canal
mt	metraterm
oot	ootype surrounded by Mehlis's glands
ov	ovary
ppr	pars prostatica
rs	receptaculum seminis
t	testis
ut	uterus
vd	vas deferens
vs	vesicula seminalis
vit	vitellaria
vitd	vitelline duct

Digestive system in digenetic trematodes: Digestive system is incomplete, starts from mouth which is surrounded by anterior sucker followed by a muscular pharynx then oesophagus which leads to intestinal caeca which are usually in shape of two blind tubes with no anus.In some species (fasciola) secondary branching of caeca may occur.

Excretory system in digenetic trematodes:

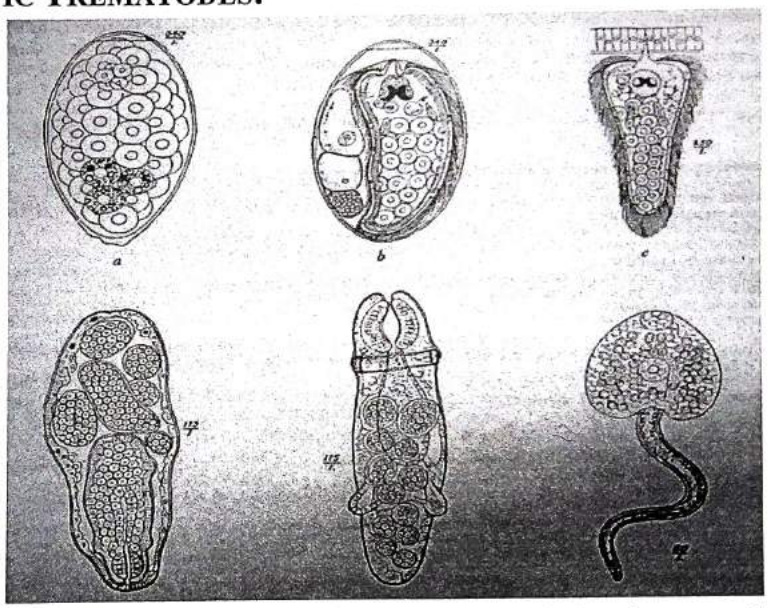
The excretory system consists of a bladder which may be from sac like, in simple form , to various shapes and usually open to the posterior extremity of the body. From central collecting organ fine branches are embedded in parenchyma which end in the flame cells which are terminal organs. These cells collect the waste products from its surroundings and propelled in connecting tubes with the

help of cilia present in these cells and finally these substances from different tubes are accumulated in bladder . The numberof flame cells and arrangement of tubes is characteristics and used in diagnosis.

Nervous system in digenetic trematodes:

The nervous system is composed of circumoesophageal ring of fibres and paired ganglia from which three pairs of nerves run forward and three backwards to all parts of the body. Sense organs,as such , are not present in adult but miracidium and cercaria may be provided with patches of pigments called as eye spots.

LIFE CYCLE OF DIGENETIC TREMATODES:



Eggs of parasites are usually passed in the faeces of the host and under suitable conditions miracidium hatches. Eggs can not withstand dessication. Hatching is controlled by light,temperature and salinity. Miracidium triangular in shape and anterior end being broader. Miracidium is usually covered with ciliated covering and have an anterior spine for boring into first intermediate host. The first intermediate host is snail , if second I.H. is present then it may be other snail or a wide range of other invertebrates or even vertebrates. The miracidium is usually provided withexcretary and nervous system , a sac-like gut and an eye spot. This larval stage does not feed It enters in snail . Penetration is assisted by the boring action of the miracidia and probably enzymes secreted from the apical glands.

Following the penetration the ciliated covering is lost and form becomes as sporocyst which has a undifferentiated mass of germinal cells which multiply to produce either daughter sporocysts or rediae. Rediae are not found in the life cycle of those digenetic trematodes in which daughter sporocysts are formed. One or more generations of rediae may occur. The final stage cercaria is produced either by sporocyst or redia. The redia has an oral sucker, a pharynx , a sac like intestine , a excretory system and a birth pore through which cercariae which are produced inside escaped. Cercaria has suckers and an intestine like that of adult , excretory and nervous system ,special glands and sometimes an anterior spine. It frequently provided with a tail which help in propelling in the water.Cercariae leaves the snails either through an opening , or through integument or expelled passively in masses.Cercariae encyst on or in 2nd intermediate host or on the vegetation.the encyted form mature to form metacercaria which is the infective stage.

Metacercaria is final stage of life cycle and has to reach to definitive host.It enters the host through herbage , through contaminated water of through 2nd I.H. However In family schistosomatidae cercariae actively penetrate the skin of the definitive host..

Reproductive potential in trematodes is very high , millions of cercariae may be produced from a single miracidium.

When metacercaria reaches in the intestine of host excystation take place in intestine and immature stages migrate to their site of predilection.

Dr. Devika Sharma

Dicrocoeliidae

Species of this family small to medium sized flukes, parasitic in biliary and pancreatic ducts ofamphibians, birds, reptiles and mammals. Body is flattened, elongated with loose parenchyma through which internal organs are visible. Cuticle usually lacks spines. Suckers are close together.intestinal caeca simple not reaching to the posterior part of body. Excretory bladder is simple and tubular. **Testes** are close to ventral sucker and ovary is post testicular in position. Most of the space behind gonads is filled with uterine coils. Vitelline glands lie in the lateral field.

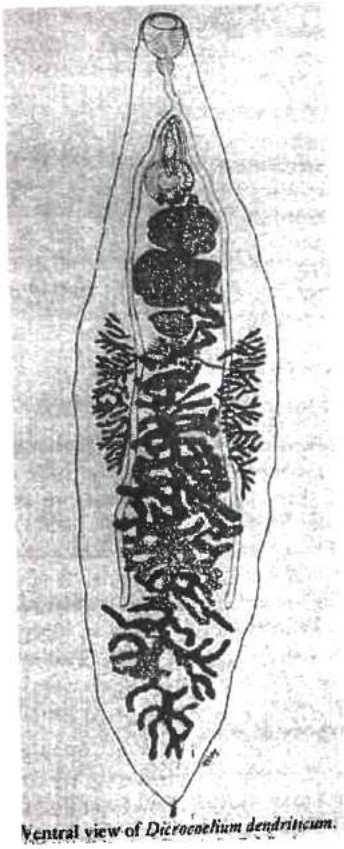
Genus: ***Dicrocoelium***

Species: ***D. dendriticum*** (commonly called as lancet liver fluke of ruminants and is commonly found in the Himalayan regions of India)

Found in the bile ducts of sheep, goat, cattle, pig and other mammals

Morphology:

1. They are 6-10 mm. Long and 1.5 to 2.5 mm wide.
2. Body is elongate, narrow anteriorly widest behind middle.
3. Cuticle is smooth
4. Oral sucker is less developed thsn ventral sucker.
5. Testes are slightly lobed and tandem and located behind the ventral sucker.
6. Ovary is situated immediately behind testes.
7. Vitellind glands in middle third of the body in the lateral field.
8. Uterus fills the central field behind gonads.
9. Intestinal caeca is simple, unbranched & not reaching to the posterior part of the body.



Ventral view of *Dicrocoelium dendriticum*.

Life cycle:

The parasite may live more than 8 years in the final host. Eggs contain miracidia when they are laid and pass out in the faeces of the host. They are ingested by several species of the land snails. In India *Macrochlamys cassida* and *Luastenia monticola* act as the first intermediate host. Miracidia hatches in

side theintestine of snail from where it migrate to the hepatopancreas to develop into sporocyst. The spocysts produces daughter sporocysts which lateron producelong-tailed, stylet cercariae. The cercariae accumulate in the mantle cavity of snail. Subsequently, 200-400 cercariae are held together by a sticky, gelatinous substance. Such clumps of cercariae are called as slime balls.These slime balls are expelled out from the snails when there is drop in the temperature in the environment and adhere to the vegetation. Many more slime balls are deposited if rainfall follows after some days of dryness or draught.

The ant, formica fusca and other species act as 2nd intermediate host.the ants ingest cercariae and metacercariae are formed in the abdominal cavity in 26-62 days.The infected ants climb on the blades of grass and form clusters and therefore, more easily available to the grazing animals.

In final host, young parasite reach to bile ducts andliver via the portal blood system in 5-8 days. The young flukes migrate in the larger bile ducts at the time of maturity and during this phase of life cycle they cause pathological changes.

P .P. 47-54 days.

Pathogenesis: The pathology includes subacute to chronic non supportive cholangitis and cholangiectasis (dilation of the bile ducts, usually as a sequel to obstruction) and finally leading to cirrhosis of liver. Continuous infection builds up a massive infection because there is no immunity to the infection. Liver become indurated (callus, unfeeling) and scarred and whole organ becomes hard.. Bile ducts become thickened and cystic duct may be occluded resulting in jaundice leading to interference in digestion of fat. There is proliferation of fibroblasts around the bile ducts, veins and arteries. In later stage extensive cirrhosis is foundIn liver of sheep there may be encrustation of calcium salts of necrosed areas of the connective tissue, granular dystrophy hepatic cellswith simultaneous proliferation of liver cells and occasional thrombosis of blood vessels.This infection is less severe than fascioliosis.

Clinical symptoms: In severe cases the animals may show the symptoms of anaemia , oedema and emaciation.

Diagnosis : by finding the characteristic eggs in the faeces of infected animals.

Treatments:

Albendazole	7.5mg/kg b.w. (two dose 2-3 weeks apart.
Cambendazole	5mg/kg b.w.
Fenbendazole	150mg/kg.b.w.
Thiabendazole	200- 300 mg/kg.b.w.
Praziquantel	50mg/kg.b.w.

Control:

1. Include the treatment of infected animals and control of snails and ants.
2. Using poultry to control the snail population
3. Ploughing of field help in reducing the ant population. Ants may also be killed by using the insecticides such as benzenehexachloride.

Family: Opisthorchiidae

Family contains small to medium sized flukes. They are found in gallbladder bile ducts of reptiles, birds and mammals. They are much flattened and have translucent body with narrow anterior end. Oral sucker and pharynx are well developed. Ventral sucker is comparatively smallin anterior third of the body. The testes are diagonal one bebind other, near posterior end of the body. Ovary is pretesticular. Receptaculum seminis is large. Vitelline follicles are in lateral field in the mid body.genital pore lies just anterior to the ventral sucker.

Genus: ***Opisthorchis***

Species:*O.tenuicollis* the species occur in the bile ducts and more rarely in intestine and pancreatic ducts of dog,cat,fox,pig and man. This species occurs in India.

Opisthorchis viverrini **:** Species occur in the bile ducts of civet,domestic and wild cats,dogs and man in south East asia

Morphology:

1. They are 7-12 and 1.5-2.5 mm. Long, reddish in colour when fresh.
2. Oral sucker is terminal, pharynx and oesophagus is short, intestinal caeca terminate posteriorly.
3. Testes are lobed ,slightly diagonal and situated in the posterior half of the body.
4. Typically Y shaped excretory bladder passes between the two testes. The cirrus and prostate gland are absent.
5. The pre-testicular ovary lies in the midline at the beginning of theposterior third of the body.
6. Uterus located in between ventral sucker and ovary and genital pore opens in middle just anterior to the ventral sucker.
7. Vitelline glands consists series of transversally arranged follicles and occupy the middle third of body in lateral field.
8. Eggs are slightly brown in colour, 26-30X 11-15micron in size, electric bulb shaped having an operculum which fits into thickened rim of shell and contain miracidium when laid.

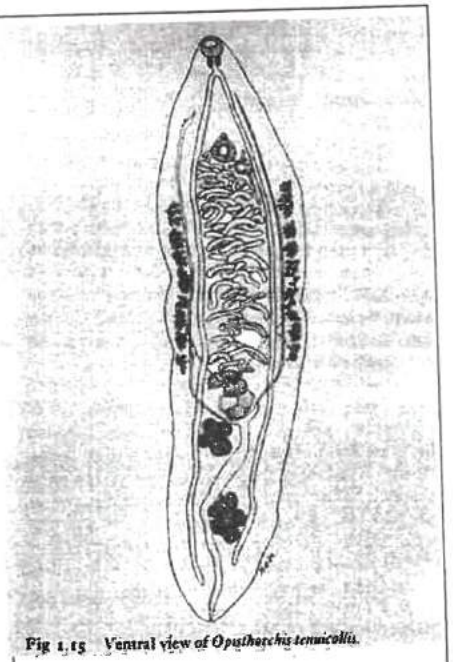


Fig 1.15 Ventral view of *Opisthorchis tenuicollis*.

Life Cycle: First I.H. are fresh water snails of sp. *Bithynea leachi, Bulinus infata* and 2nd I.H are Cyprinid fishes belonging genera *Idus, cyprinus, Tinca , Babus, Abramis, blicca* etc. cercariae are shed by the snails about 2 months after infection. The cercariae penetrate through skin and encyst in the s/c tissues of the fishes which act as 2nd I.H.The metacercariae require about six weeks in muscles of fishes to matureInfection reaches to the definitive host when raw or partially cooked fishes are eaten by them.parasites reach to liver by way of common duct and mature in 3-4 weeks. The whole life cycle is completed in 16-18 weeks.

Pathogenesis: Severity depends on the intensity of of the infection.most of the cases are usually asymptomatic with few or no changes in liver. These worms live in the proximal part of bile ducts and cause dilatation with adenomatous thickening of the epithelium,catarrhal cholecystitis, desquamation of bile duct epithelium and increased production of mucousand wide spread periductal fibrosis. Carcinoma of liver or pancreas of cats may be found.

Diagnosis: by finding the eggs in the faeces of the infected host.

Treatment: No complete satisfactory treatment yet available in animals.

Family : Paragonimidae

These are plum diastome. Cuticle is spiny. In the members of this familyOral sucker is ventroterminal. Ventral sucker near the midway. Genital pore behind V.S. Testes are in posterior half of the body. Ovary is pretesticular in position.

Family Prosthogonimidae

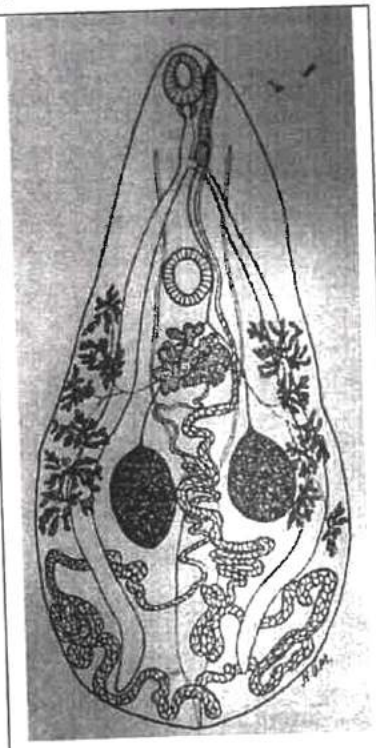
The members of this family small flukes of birds and rarely mammals. Flukea are anteriorly pointed and posteriorly rounded.Spines are present on the cuticle. Ventral sucker is found in the anterior half.Testes are symmetrical and behind the ventral sucker ovary is present between these or dorsal to the ventral sucker. Vitelline glands are in the lateral sides.

Genus: *Prosthogonimus*

Prosthogonimus ovatus: the fluke is found in the bursa of fabricius and oviduct of domestic fowl,duck and geese and in a wide variety of wild birds n Europe, Africa and Asia.

Commonly called as oviducal fluke of the poultry andfound commonly in birds in India.

1. It measures 3-6mmx1-2 mm.
2. The body is oval to elliptical or pyriform with greater diameter in its middle.
3. Oral sucker is elongated and oval.
4. Ventral sucker is twice as large as oral sucker and lies in anterior onethird of the body.
5. There is small pharynx, long caeca ending above the margin of the body.
6. Genital opening is located on the left side of the oral sucker.
7. Testes are elongated lie side by side behind the middle part of the body.
8. Ovary is deeply lobed , pretesticular,behind the ventral sucker.
9. Vitellaria on the lateral side, extending between the level of the ventral sucker and posterior margin of the testes.
10. Uterine coils spread in the posterior region.



Life Cycle:

Two intermediate snails are required in the life cycle of this fluke.

First intermediate host is water snail(Bithynia leachi. Gyraulus albus,G.gredleri) and 2nd I.H. isnymphal stages of various species of dragon flies(In India *sympatrum decoloratum* is commonly found harbouring the metacercariae in India). Sporocysts directly produce cercariae without radial stage. Cercariae after liberating from the snail swim in the water for a while and these are drawn in the anal opening of the naiads of the dragon flies with the help of breathing movement of these insects. These cercariae penetrate the muscles and encyst in the haemolymph of the nymph to

become metacercariae. Final host become infected by ingesting the naiads or adult of dragon flies.In final host liberated fluke migrate to cloaca and bursa of fabricius wher they become adult. In adult birds where bursa is atrophied, the flukes enter the oviducts. They may reach to ovary or in peritoneal cavity.

Pathogenesis: flukes cause marked irritation, resulting in acute inflammation of the oviduct, the production of abnormal eggs , and discharge of albumin from the cloaca. The oviduct performs retroperistaltic movements, causing broken yolks,albumin, bacteria and parasitic material to enter in the peritoneal cavity causing peritonitis usually leading the death of the birds.

Clinical symptoms. Infected hen lay soft shelled eggs or eggs without any shell.the birds usually sit in nest more than the required. There is milky discharge from the cloaca containing calcareous material which soil the feathers of perianal region. Other clinical signs include listlessness, a pendulous abdomen, legs are held wide apart in walking. Subsequently egg laying is stopped and peritonitis develops, comb and wattles become cyanotic.The birds become prostrated and die.

PM lesions: Reveals inflammation of oviduct and its obstruction with cheesy mass of puss with or without yolk or concretions. Haemorrhages or congestion of peritoneum. Flukes may be seen adhered to the mucosa of oviduct and in even ovary.

Diagnosis:

1. symptoms.
2. Detection of eggs in the cloacal discharge.
3. Finding of flukes in the affected oviduct.

Treatment and control: Albendazole and praziquentel may be useful.Infection is only possible in those birds which are reared in pens and have access to ponds and water collections where breeding of snails and dragon flies occur. The birds reared in cages or fenced rooms are less likely to be infected. Other species are *P. pellucidus* and *P. macrochis* in north America in. *P.ovatus* is considered the only valid species by some workers.

Dr. Devina Sharma

Family Fasciolidae

Family Characters:

1. Flukes of this family are parasitic in bile duct, liver, gall bladder and intestine of mammals.
2. They are large sized, leaf shaped, covered with spiny tegument.
3. Anterior and ventral suckers are found close together, oral sucker is followed by a pharynx, a short oesophagus and intestinal caeca which are much branched.
4. Genital pore situated to the anterior of the ventral sucker.
5. Testes may be lobed or branched and tandem in position
6. Vitelline glands are strongly developed filling the lateral fields.
7. Receptaculum seminis is absent and eggs are operculated.

This family has three important genera:

1. Fasciola 2. Fascioloides 3. Fasciolopsis.

Genus *Fasciola* has two important species. ie. *Fasciola gigantica and F. hepatica.*

Fasciola gigantica is commonly found in low lying areas.

Commonly these parasites are called as liver fluke and disease caused by them is known as fascioliosis, liver rot or liver fluke disease.

These parasites are found in bile ducts, liver ,gall bladder of sheep, goat, cattle, buffalo and wild ruminants. They may also occur in pig, horses, dog, cat and elephant.

- Adult flukes are leaf like, grayish brown in color, large and measure 25-75 mm by 12mm
- The tegument is spiny.
- Cephalic cone, though present, is not marked off from rest of the body because of inconspicuous shoulders
- Oral sucker is smaller than ventral sucker; both lie close to each other in the anterior third of the body.
- Intestinal caeca are profusely branched both inward and outward, and extend up to the posterior end.
- Testes are highly branched, Tandem and fill up the median field.
- Ovary is also branched and lies to the right of the middle in front of the testes.
- Uterus runs anterior to the testes.
- Genital pore is situated anterior to the ventral sucker.
- Vitelline glands are voluminous and occupy the lateral fields on the either sides and extend from anterior to posterior end.
- Eggs are large, ellipsoidal, yellowish-brown, operculate and measure 156-197 by 90-104µ.

Differentiating features in between *Fasciola gigantica and Fasciola hepatica.*

S.No.	Fasciola hepatica.	Fasciola gigantica
	30mmx 13mm	25-75mm x 12mm.
	They are leaf like in shape	They are more elongated.
	Cephalic cone is more prominent	Less prominent.
	Shoulders are more prominent.	Comparably less prominent.
	Intestinal caeca mainly diverticulated externally	Intestinal caeca diverticulated externally and internally.

Life cycle:

The adult fluke ,they are known to live for upto 11years in vertebrate hosts. Though normal life is about a year or less. During this period an individual fluke may produce 3000 to 3500 eggs per day. The eggs of F. gigantica hatch in about 17 days at optimum temperature (26°C) at high temperature hatching may take place in about nine days and at lower temperature (above 10°C) in approximate 90 days with less survival percentage. Eggs can survive at a temperature of 2-4°c for 16 months but at 37oC they are killed. Miracidium actively swim in water and may survive up to 24 hours. During this time it seek a suitable intermediate host.

for ***Fasciola hepatica*** 1.*lymnaea truncatula*

2.*L. luteola*

For *Fasciola gigantica* 1. *Lymnaea auricularia var rufescens*

2.*Lymnaea Luteola*

Miracidium can penetrate any part of the snail and develop to next larval stage ie. sporocyst. The sporocyst produces a number (usually 5-8) ofgerm balls; each developing into next larval stage, redia with blunt processes at the beginning posterior quarter. Usually a second generation of redia stage is produced which in turn produce the cercaria, the next larval stage. Cercariae are shed 68-89 days after infection which last for 10-77 days. At 25°C cercariae are shed on 38the day after infection of the snails.shedding of cercariae is stimulated by several factors like change in temperature, sudden flooding of the environment. Agter coming out from the snails the cercariae they live a short free life and then attach themselves to the vegetation or even inanimate object and shed their tail. Later on cytogenous gland cell secrete a covering and form a cyst.This is called as metacercaria. Metacercaria remains alive for considerable period of time and under favourable condition they may live for 12 months.Vertebrate host become infected when metacercaria is ingested along with the vegetation.

After infection the cyst of metacercaria is digested in duodenum with the help of acid pepsin followed by trypsin and bile. Liberated metacercaria makes its way to bile ducts and liver usually by penetrating the intestinal wall and then liver from outside, or by travelling up to the bile duct, or is carried with the blood stream but majority of young parasites migrate via portal vein. Young flukes . Usually parasites take 6 days to reach the liver or only 48 hours in exceptional cases. The young baby flukes move around in the liver parenchyma for about 4-5 weeks causing extensive damage finally settling down in the bile duct. Parasites grow quickly and start laying eggs in 11-12 weeks after infection. The young flukes appear in the body cavity 2 hours after infection and in liver after 48 hours but majority take in 4-6 days.

Pathogenesis: During early stage, the flukes migrate extensively through the tissue of liver and cause mechanical destructionand migratory tract are produced on the liver capsule.Damage is also caused by the feeding of the parasites on hepatic cells. In older infection there are excessive leucocyte infiltration and the terminal part contains large number of leucocytes , broken hepatic cell and blood. The spines of the parasites irritate the tissue of liver resulting in inflammation and fibrosis. A progressive eosinophilia occurs during migratory phase, accompanied , later on,fall in serum protein and rise in serum globulin. When the parasites reach to the bile duct, there is fall in the number of the erythrocytes,level of serum calcium leading to hypoalbuminaemia.

The pathological menifestations mainly depends on the number of metacercariae ingested in first attempt. This results in:

1. Acute or Per -acute fascioliosis
2. Sub -acute fascioliosis
3. Chronic fascioliosis

Acute fascioliosis: It is basically a traumatic hepatitis produced by continuous migration of large number of immature flukes of 6 to 8 weeks of age. It causes extensive destruction of liver parenchyma and severe haemorrhages .If flukes are large in number ,they cause rupture of liver capsule leading to haemorrhages in peritoneal cavity and animal may die with in a fews days of the onset of clinical symptoms.Infected liver becomes enlarged, pale and friable and numerous haemorrgagic tracts may be found on on liver surface.Fibrinous clots on liver surface and throughout the peritoneal cavity are seen.small flukes of 0.7-2mm may be obtained from peritoneal flude or from liver after giving incision.

A complication of acute disease is black disease caused by *Clostridium Clostridium novyi.* This anaerobic bacilli may be normally present in liver without causing harm but they multiply rapidly in damaged liver causing black disease.which is not recognisable before death as the animals die so suddenly.

Sub acute Fascioliosis: In this affected liver is seen covered with migratory tracts. Infiltration of WBC in more evident and early fibrosis is seen. If sub acute infection is superimposed then a more marked cellular reaction may be observed, indicating a form of immune response to the second infection.

Chronic fascioliosis: It is most common form of disease found in sheep/goat/cattle and other animals including man. This type of disease occur in in light and repeated low dose of infection. The main consequence of it is liver fibrosis. The disease entity may be devided into:

- **Hepatic fibrosis**
- **Hyperplastic cholangitis**

Hepatic Fibrosis: It is caused by migration of immature flukes in liver parenchyma producing migratory tracts, which leads to traumatic destruction of liver parenchyma,haemorrhage and necrosis.Migratory parasites may also lead to thrombus formation in hepatic veins and liver sinusoids, subsequently causing obstruction of blood leading to ischemia and coagulative necrosis of liver parenchyma.Subsequently healing process starts 4-6 weeks after infection in which collegen fibres are laid down and fibrosis take place. Contraction of these scars leads to distortion of hepatic architecture, which restored 12-20 weeks after infection.

Hyperplastic cholangitis: It is caused by adult flukes in bile ducts.bile duct become fibrosed and thickened with proliferation of epithelial cells. In heavy infection fibrosis and inflammation spread almost to the whole of the liver, the bile ducts show hypertrophy and calcification. The whole organ become hardened. In cattle the wall of bile ducts are commonly calcified and protrude markedly from the surface and are difficult to cut from knife. They resemble the stem of a clay pipe, giving the common name of pipe-stem liver to the infection. The anaemia in fascioliosis is associated primarily with intrabiliary haemorrhages due to blood sucking activity of the adult parasite. The calculated blood loss is 0.5 ml /day/fluke. The rapid drop in PCV at early infection is related with loss of RBC into intestine resulting in anaemia.

Symptoms: In acute cases sheep dies suddenly without showing symptoms. Blood stained froth appears at the nostrils and blood is discharged from anus. In acute and sub –acute cases animal disinclined to move, there is anorexia, distended abdomen which is painful to touch.

In chronic cases first sign appears when parasites burrow the liver parenchyma. Sheep becomes off colour,anaemic,loss of appetite,mucous membrane become pale and intermandibular oedema (bottle jaw) may develop. If condition continues the skin becomes dry and doughy . Wool starts falling in patches.animals become debilitated .

there may be occasionally diarrhoea or constipation.At this stage animals may die. If recovers, the symptoms are gradually subsided.

In cattle, constipation is marked and diarrhoea is only seen at extreme stages. Animal becomes emaciated,dull and weak lead to prostration, especially calves.

Diagnosis:

1. Symptoms.
2. Confirmed by finding the eggs of parasites in faeces. These eggs must be differentiated with eggs of paramphistomes.

   Fasciola eggs are smaller, operculum is indistinct, embryonic mass is indistinct and egg shell is of yellow in colour.

   Amphistomes eggs are larger, operculum is distinct, embryonic cells are clear, egg shell is transparent and egg may have a posterior knob at posterior extremity.

Treatment:

1. Carbon tetrachloride and hexachloroethane are old drugs. CTC is cheaper but more toxic, especially in cattle. One to 2.5 ml of CTC in a blend oil, such as liquid paraffin, is given as a drench to sheep anf goats, but care should be taken that drug should not reach to lungs. For small and weaker animals, s dose of 2 to 3 ml is indicated. Intramuscular administration of CTC reduce the risk of toxicity with a disadvantage that at the site of injection there is necrosis or abscess formation. For S/c injection 1-3 ml dose is recommended in sheep.
2. **Hexachloroetane:** 90% effective against mature flukes.
   Well tolerated by sheep /goat and cattle.
   Cattle- 220-400 mg/Kg.B.W in 3-4 doses is quite effective in cattle.
   Sheep- the dose varying from 20-30 gram/animal is safe drug.
3. **Hexachlorophene:** 90% effective against mature flukes.
   Sheep- Routine dose 15-20mg/Kg.B.W.
   Acute case- 40mg./kg.b.w.
   Given either s/c as oily solution or orally.
   Cattle - 10-20 mg/kg.b.w.
4. **Oxyclozanide:** 100% effective against mature flukes.Three times doses effective against immature flukes in sheep.
   Sheep/goat - 15-20mg./kg.b.w.
   Cattle - 10-15 mg./kg.b.w.
5. **Rafoxanide:** over 99% effective against mature flukes, up to 98% effective against six-week old flukes 50-90% effective against 4-5 weeks old flukes.
   Sheep/cattle/goat - 7.5mg./kg.b.w.
6. **Nitroxynil:** effective against Mature – 100%, Immature 90% , given s/c.
   Cattle – S/acute and chronic 10mg./kg.b.w.
   Acute 15mg./kg./b.w.

7. **Triclabendazole:** Effective against Adult, Immature & to a extend early immature flukes in sheep.

   Sheep/goat- 10mg./kg.b.w.

   Cattle- 12mg./kg.b.w. Effective against adult & to a extend against immature. (Fasinex a product of Hindustan Ciba-Geigy Ltd. In cattle and buffalo given at a rate of one bolus (99mg.)/75kg.b.w. In sheep/goat one bolus(250mg.) 25/kg. b.w.).

   Some benzimidazole compounds , such as albendazole and oxfendazole are effective in addition to their efficacy against gastrointestinal nematodes

8. **Albendaziole:** Sheep/goat- 7.5mg./kg.b.w.

   Cattle- 15mg./kg.b.w.

Control: Control of Fasciola depends on three factors 1. Destruction of snails which act intermediate host 2. Treatment of infected animals. 3. Control of wild animals as they serve as reservoir host.

The snails are found in shallow water and capable of migrating a long distance over mud and wet pasture.It should be borne in mind that snails are prolific breeders and one snail in course of two generations may produce from 25000-160,000 individuals Since one miracidium may produce as many as 629 cercariae or more and infected snail discharge 7179 cercariae in 75 days.

Snails may be reduced by proper drainage and by removal of vegetation, which may not be feasible on large areas.

Chemical use:

1. Copper sulphate: In a dilution of 1 part in 150 million parts of water kills the snails in 12 hours. The chemical should be applied several time during the year as single application is sufficient to kill all the snails. It is highly toxic to fishes and other species of animals
2. Di-nitro-0-cyclohexyl-phenol (DCPH) at a concentration 3-5 ppp in water is more effective than copper sulphate. Pentachlorphenol and 2-4,dinitro-6-phenylphenol, Copper sulfate pentahydrate crystals and copper chelates are alsoactive molluscicides. These molluscicides are used in spring and mid summer. Animals should not be allowed to graze until a rain is fallen.

   For *Fasciola gigantica* Molluscan vector is an aquatic species of snail, their elimination by molluscicides is rather difficult.

All the animals infected with Fascila should treated twice in a Year rin a systematic way. Control of snails and treatment of animals should be carried out simultaneously to get better results.

Immunity and immunization: some animals like cattle and pigs have moderate to high degree of resistance to primary infection Sheep is highly susceptible to reinfection and also do not develop protective immune response to reinfection.

The ability of cattle to develop protection against reinfection suggested that immunization might be feasible.

Workers immunized the calves with 1000 irradiated (20 K rad) metacercariae in a single dose found that calves exposed to infected pasture showed 69% reduced worm burden in comparion to control animals.

Fasciola jaksoni

Found in the liber ,bile ducts of elephant and known as liver fluke of elephant.

Fasciolopsis buski

Called as giant intestinal fluke.
It is found in small intestine of pig and man.
Morphology:

1. Its body is thick, flat and large measuring 30-75 mm. In length and8-20mm in width.
2. It is elongated,oval in shape,slightly broader posteriorly.
3. Shoulders and cephalic cone are absent.
4. Ventral sucker is larger and situated near the anterior extremity.
5. Pharynx and oesophagus are short , followed by unbranched intestinal caeca, which run to the posterior extremity.
6. Testes are branched, tandem and situated in posterior part of the fluke.
7. Ovary is branched and lies on the right of the midline.

Life Cycle: The life cycle is similar to that of *Fasciola*. In India *Planorbis* and *Gyraulus* spp. of snails act as intermediate host, the miracidia requires 2-3 weeks to hatch and within a few weeks of infection of snails, the sporocyst, mother and daughter redia and cercariae are produced. The cercariae resemble those of Fasciola and encyst on the tubers and water chest nuts, which are cultivated for food and are usually fertilized with human night soil, and other water plants.

Pathogenesis: Pathology is known in human being where parasite produce local inflammation, lesions, deep ulcerative enteritis. Flukes are found firmly attached to intestinal mucosa leading to inflammation and erosion.

Symptoms: In human being ,symptoms include abdominal pain,diarrhoea,oedema and ascitis.

Symptoms in pigs are not clearly reported. Pigs usually act as reservoir.

Treatment:

Hexyl resorcinol- 1gm./Patient.

Tetra Chloroethylene – 0.08-0.14ml/kg.

Niclosamide- 160mg./kg.

Prophylaxis:

1. Proper disposal of night soil of huma being and faeces of pigs.
2. Tubers and nuts of the plants should not be used as food without cleaning in boiling water.
3. Health education for cooking water nuts and to avoid drinking of unfiltered water.

Family: Paramphistomatidae

Trematodes classified in this family are commonly known as amphistomes or rumen flukes. These are thick and circular in transverse section. Body is conical or oval in shape. Oral sucker is situated in the anterior extremity. Ventral sucker is situated at the posterior extremity is called acetabulum. Pharynx is absent. Oesophagus is with or without muscular bulb. Intestinal caeca are simple. Testes are lie in tandem, diagonal or side by side. Ovary is post testicular in position. Uterus runs forward in the dorsal part of the body and coiled. Genital pore is ventral vitellaria is well developed laterally.

There are eight genera of veterinary importance;

1. *Paramphistomum*
2. *Cotylophoron*
3. *Gastrothylax*
4. *Fischoederius*
5. *Carmyerius*
6. *Gastrodiscus*
7. *Gastrodiscoides*
8. *Pseudodiscus*

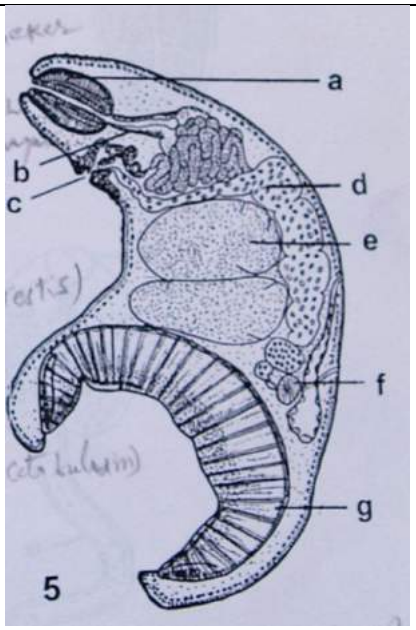
These species are found in the digestive tract of ruminants, equines and pigs.

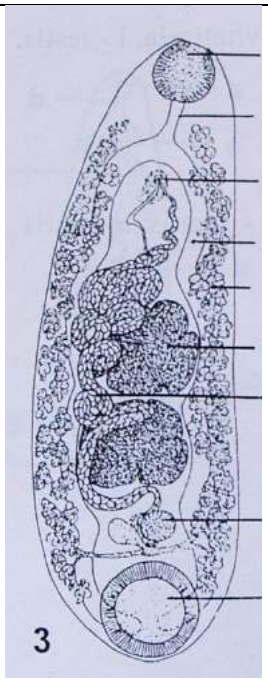
Life cycle is indirect , require snail as intermediate host. The eggs are operculated.

1. Genus - *Paramphistomum :* Body is elongated or conical and convex dorsally, ventral pouch is absent.Oral sucker is without pouch. Intestinal caeca simple, long with deep bends.tstes are tandem or diagonal in position. Genital pore is surrounded by genital bulb. Ovary is posttesticular in position.vitellaria in lateral fields. Genus have many species.these flukes are commonly called as conical flukes.

Paramphistomum Cervi: adult fluke is found in rumen and reticulum of sheep, goat, cattle and buffalo. The body is pear shaped with convex dorsal surface and concave ventral surface and with a posterior sub terminal sucker. They measure 5-13mm in size. Genital pore is situated in the	

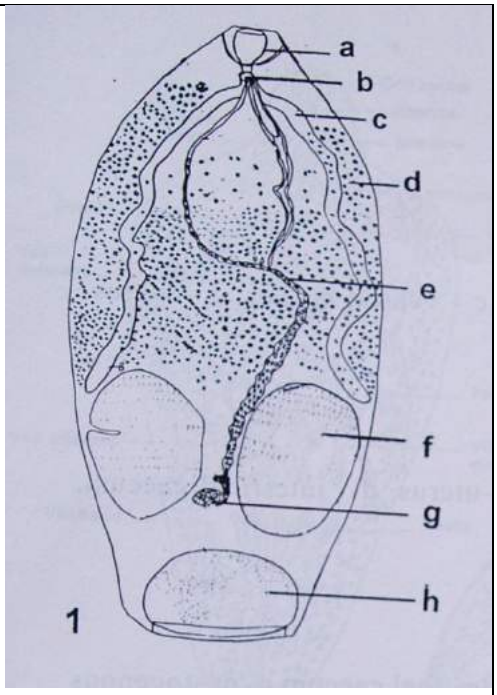
anterior third of the body. Two lobed testes lie anterior to the ovary and tandem in position. Vitellaria are in groups of follicles.the intermediate host is *Indoplanorbis exustus.*	

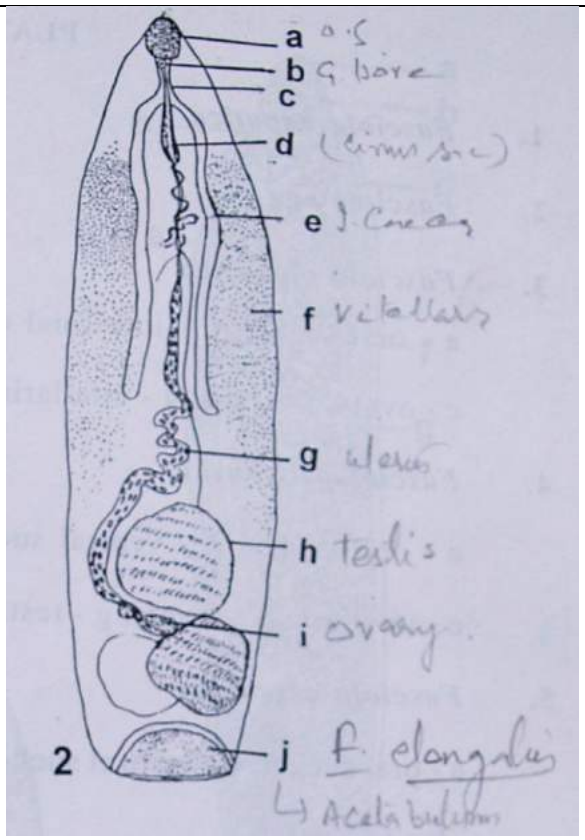
Paramphistomum explanatum (Gigantocotyle explanatum) : the fluke is found in the bile ducts inside the liver, gall bladder and duodenum ,mostly in buffaloes and rarely in cattle,sheepand goats. The posterior sucker is very large and deep. The testes are placed diagonally. The ovary is posterior to the testes, just above the posterior sucker .Life cycle involves *Gyraulus convexiusculus* as intermediate host.	

Cotylophoron cotylophorum: Adult fluke found in rumen and reticulum of sheep,goat,cattle and buffalo. A genital sucker encloses the genital openingjust behind the intestinal bifurcation. Testes are tandem. Life cycle involves *Indoplanorbis exustus* as intermediate host.	

Gastrothylax: Body is elongate and flashy. A large pouch is placed on the ventral aspect is called ventral pouch which opens anteriorly. Intestinal caeca are straight and terminate in front of the ovary. Uterus crosses the middle of the body from one side to the other.

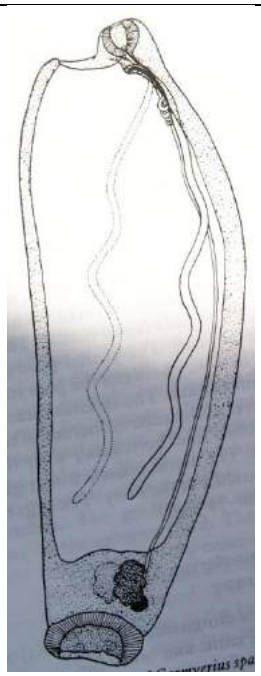
G. crumenifer*:* adults are found in rumen and reticulum of cattle , buffalo,sheep and goat. They measure 9-18mmx 5mm. Thee is large ventral pouch which opens anteriorly and extends over the whole ventral surface up to the posterior sucker. Genital poe opens into the ventral pouch behind the bifurcation of the intestine. Intestinal caeca run almostnear the testes. Testes are side by side, immediately anterior to the posterior sucker. Ovary just behind the testes. Uterus crosses the body in the middle from one side to other side. The life cycle involves *Gyraulus convexiusculus* as Intermediate host.



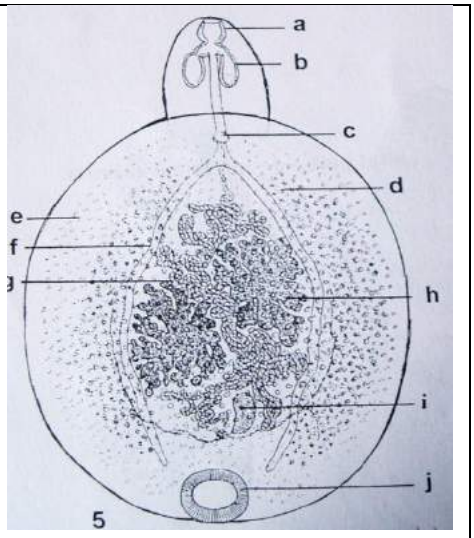
Fischoederius: *the flukes resembles Gastrothylax in having a ventral pouch. The intestinal caeca are short or extend upto the posterior end. Uterus lies in the middle of the body and runs straight throughout the course. Testes are placed one behind the other.* ***Fischoederius elongates****:* Adults are found rumen and reticulum of cattle, buffalo, sheep and goats. Measure 10-20mm in size. Small ventral pouch is present. Testes are dorsoventral in position. Uterus runs straight in the midline. The intestinal caeca run upto the mid region. The life cycle involves *Lymnaea luteola* as Intermediate host.	

Fischoederius cobboldi: Adult fluke occur in rumen and reticulum of cattle,buffalo,sheep and goat. These measure 8-10mm in size. Intestinal caeca are long,extending upto the posterior end. The life cycle involves *Lymnaea luteola* as Intermediate host.	

***Carmyerius spatiosus*:** the species is found in rumen and reticulum of cattle and buffalo. The flukes are 11-15mmx3-4.25 mm. The body is cylindrical with anterior end bluntly pointed. The ventral pouch is present. The testes lie side by side.the ovary lies in between the posterior border of the testes.the genital pore opens a little anterior to the intestinal bifurcation surrounded by a large sucker like genital bulb. The intermediate host is not known.



<u>Gastrodiscus aegyptiacus;</u> the flukes are found in caecum and other part of the intestineof equine and pigs. The fluke measure 9-17mmx 9-11m. They are pink in colourwhen fresh.anteriorly there is more or less cylindrical part which is upto 4 mm long while rest of the body is saucer shaped. The ventral surface is covered with large number of regularly arranged papillae. The acetabulum issmall and terminal. The intestinal caeca reach at the anterior border of the wide portionand the caeca continue tonear the hind end of the body. Testes are lobed and slightly diagonal and lie behind the middle of the body. The ovary is post testicular in position. Vitellaria occupy the lateral fields. The genital pore opens at the level of intestinal bifurvcation. The intermediate host are *Cleopatra* sp. of snail.



Gastrodiscus secundus:

This species is found in colon of horse and elephant of Inia. These flukwe measure 7.8mmx4.5-5mm. anterior part is globoid and posterior part is discoid. A pair of oral pouches are present. Testes ar placed diagonally. I.H are *Indoplanorbis exustus*.

Genus: ***Gastrodiscoides***

Species- ***G hominis*** the species occur in the caecum and colon of pig and man. The flukes usually pyriform in shape and bright red in colour when fresh. The body is divided in two portion – anterior is smaller with posterior without any papillae. The oral pouches are present. The acetabulum has prominent deep cleft. Intermediate host are snails of species ***Helicorbis coenosus.***

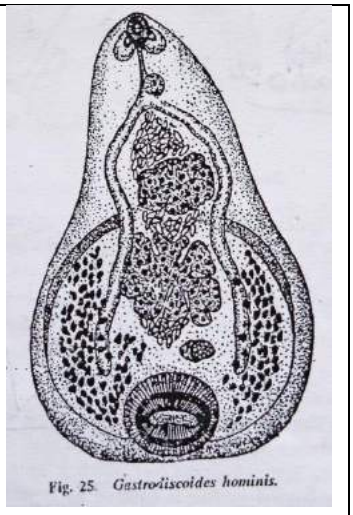
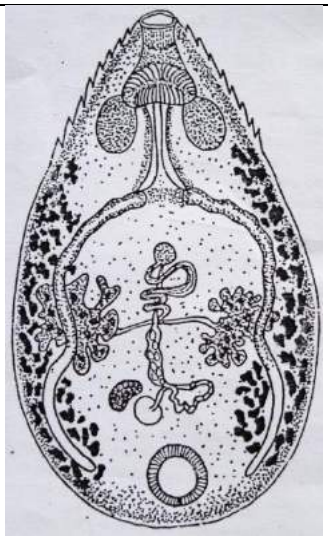


Fig. 25. *Gastrodiscoides hominis.*

Genus ;***Pseudodiscus***

Species- ***P. collinsi***

Flukes are found in large intestine of equines and elephant of India.fresh flukes are brich red in colour and somewhat oval or conical in shape.the oral sucker has two pairs or oral pouches. The acetabulum is subterminal. Intestinal caeca reach upto middle of the acetabulum. Testes are deeply lobed and lie side by side on both side of median line. The ovary is post testicular .genital pore present in about one-third of the body length from anterior end. Intermediate host are *Planorbis, bulinus, pseudosuccinea, Fossaria, Indoplanorbis and Lymnaea.*



Pseudodiscus hawkinsi

The species is found in the small intestine of elephant and man.The flukes measure 3.5-6mm x 2-3 mm. The body is oval or pyriform. The ventral pouch is absent. The oral sucker has two diverticula. The acetabulum is sub terminal.The intestinal caeca are slightly wavy. The tstes are slightly lobed and tandem. The ovary lies just above the acetabulum. The vitellaria are extracaecal. Genital pore lies posterior to the intestinal bifurcation and pretesticular in position. The intermediate host is not known.

Life cycle: the life cycle of amphistomes is generally similar except to for the different species of snails acting as intermediate host..The eggs are passed in the faeces of the definitive host. These are oval in shape and fairly large in size. They are whitish to transparent in colour except those of Gigantocotyle explanatum which are yellowish like Fasciola eggs.shell has distinct operculum and frequently a knob like thickening present on the posterior border. The eggs embryonate in open and depending upon the temperature and huminity, the incubation period varied in different species of amphistomes ranging from from 1-3 weeks when miracidium hatches out. Miracidia swim in water for specific species of snails. In few hours miracidia penetrate the snail and turn into sporocyst.The young sporocyst are irregular , pyriform or oval in shape. As the sporocyst grows, the germinal balls derived from miracidial stage develop further and differentiate into the next larval stage, the redia.The sporocyst generally attain maturity in about 6-9 days in summer and 16-38 days in winter. The mature sporocyst contains developing rediae.. the rediae lodged in the tissue of snail. Under optimum condition, they mature in about 10-15 days.Mature rediae are sausage shaped . The germinal elements of the redia consist of germinal cells and balls and developing cercariae. Sometime rediae may also produce daughter rediae in addition to cercariae.The amphistomes redia do not posses lappets like the redia of Fasciola..the cercariae emerge out from redia stage have pigmented body(called as *Cercaria pigmentata*) with numerous cytogenous cells and unpigmented simple tail. The acetabulum is located on the posterior end of the body. The two cjharacterstics features are the presence of a pair of pigmented eye spots and globular masses of high refractile material in the main excretory canal. The cercariae develop for a further period of 10-25 days in the digestive gland and other tissue of the snail.after emerging of the cercariae the life span is short, varying from several minutes to a few hours. They encyst on vegetation and become metacercariae which is infective for the definitive host. The metacercariae may remain viable for atleast 29 days or 5-6 months at laboratory condition.

On ingestion the metacercariae excyst in the small intestine which is assisted by ruminal fluid, pepsin and hydrochloric acid in abomasums, and trypsin and bile salts in small intestine, and thenyounf flukes gradually migrate anteriorly against the flow of ingesta.The Prepatent pediod in cattle is 56 days, 71 days in sheep, and 69 days in goats.

Pathogenesis:

In ruminants , clinical immature amphistomiasis results from massive invasion of mucosa of duodenum and adjacent parts of the alimentary tract by immature flukes following heavy intake of metacercariae. Superimposition is important factor in the pathogenesis of the disease.

The diseae is commonly called as immature amphistomiasis (locally called as pittu gillar or bissi rog). In massive infection, it may be fatal in young animals.the commonly involved species are

Cotylophoron cotylophorum, Paramphistomum Cervi, G. crumenifer and fischoderius spp. the disease generally occur after rainy season.The incubation period of the disease ranges from 4-6 weeks and the disease runs from 5 days to three months. In heavy infection mortality may reach upto 90% in sheep and goats and 40-60 percent in cattle and buffaloes.The immature fluke penetrate the mucosa of intestine and become attachedby drawing a plug of mucosa into their acetabula. The pieces of the affected parts of mucosa become necrosed and eventually slough off. There is extensive erosion and haemorrhages on the intestinal mucosa causing discomfort and loss of appetite. Anorexia coupled with impaired food assimilation results in loss of weight. The animal looses plasma albumin by seepage through the erosion in the digestive tract.It also leads to dyspepsia which,along with anorexia and the decomposition of plasma protein in the intestine, account for the loose and foetid nature of the faeces.

REDUCED PLASMA CONCENTRATION LEADS TO HYDROPERICARDIUM, HYDROTHORAX, PILMONARY, ASCITIS, OEDEMA OF abomasum and in the intermandibular space(bottle jaw).Pilmonary oedema.starvation and exhaustion from diarrhoea apper to be the immediate cause of the death.

Symptoms:

Most common symptoms are oedema, anorexia and diarrhoea. The oedema starts as small flabby swelling in subcutis of the intermandibular space which gradually increases in size extends upto cheeks in sheep and sternum of cattle. The oedema is soft to touch. There is decrease in appetite and may be complete cessation of feed intake. Animals generally show

increased thirst ,it drinks small quantities of water at frequent intervals and may keep its muzzle dipped in water for considerable time.

With in weeks of oedema , animals develop diarrhoea. With the passage of time faeces becomes watery and foetid. The faeces may contain mucous and immature flukes. In chronic cases rectal haemorrhages may occur due to rectal straining with the presence of blood clots in the faeces.

With the advanced diarrhoea, animal may show symptoms of anaemia. The disease follow the sub acute course in cattle and buffaloes (10-20 days) but in sheep and goats acute to sub acute lasting for 5-10 days emaciation and prostration precede death. Some time young animals may die within few days without showing many symptoms.

Pathogenesis of *Gigantocotyle explanatum:* it is found bile ducts,gall bladder and liver of buffaloesand rarely in other ruminants including goats. This fluke causes **biliary amphistomiosis.** Young flukes migrate through intestine to bile ducts ,gall bladder and connecting ducts in side liver and mature there. They cause superficial erosion of mucosa and haemorrhages due to firm attachment with tissue by powerful acetabula and subsequently fibrosis of bile ducts ducts. In heavy liver may become pale and extensive fibrosis.

Gastrodiscoides hominis: Flukes draw the mucosa of caecum in pigs in their acetabula.After detachment of flukes, the mucosal plug on the surface of mucosa look likeraised papillae in the wall of caecum.There is desquamation of epithelial lining. The lamina propria shows infiltration with eosinophils,lymphocytes,plasma cells anf macrophages. The presence of these flukes disturb the function of absorption of fluid of the caecum.

The amphistomes fluke (*G.aegyptiacus* and *G. secundus*) occurring in caecum and colon of equines are mildly pathogenic in heavy infection.

Control of Immature amphistomes: routine preventive medication to all the susceptible animals should be undertaken to prevent outbreaks.The time of dosing should be determined on the basis of epizootiological data concerning the seasonal and regional occurrence. The animals should be removed from the source of infectionbefore treatment.Preventive treatment before appearance of the symptoms leads to more effective control.

Control of Mature amphistomes: Animals carrying adult/mature flukes are the source of pasture contamination leading to infection of the snails. Preventing dosing should in these animals about a month before the onset of rainy season.

Treatment: two doses of oxyclozanide at a rate of 18.7 mg/kg. B.wt./day or a single dose hexachlorophene at a dose rate of 20mg/kg give consistent result against immature amphistomes in cattle but hexachlorophene show toxicity at this rate. Niclosamide at 160mg/kg as a single dose or as two devided doses 3 days apart is effective in cattle, while in sheep it has good results at a dose rate 50-100mg/kg. Against immature flukes. Resorantel has also high efficacy against immature and adult form in sheep and at a dose rate65mg/kg, remove 84% adult flukes in cattle.
Bithional 15mg/kg, ctc and hexachloroethane are effective against adult flukes only. However, these drugs are toxic.
Brotianide at 15mg/kg is also effective against only adult flukes.

Treatment with intestinal sedatives to control the enteritis and parental fluids to repair the dehydration are recommended. During outbreak, the animals should not be allowed to go on infected pastures.

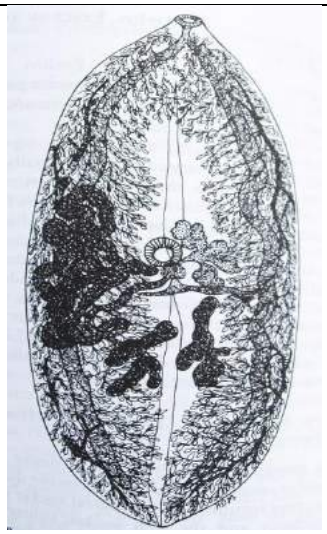
Snail control: as in fascioliosis.

Family: Paragonimidae

Genus: *Paragonimus*

Species: *P. westermanii*

It is known as lung wormoccurs in the lungs and rarely in brain, spnal cord and other organs ofdog,cat.goat,cattle, wild carnivores and in man.It has also been reported in Tigers from India. Paragonimus kellicoti:It is found in wild animals and and cat,dog and pig.

1. Parasites are reddish brown in colour. 2. They measure 7.5-16mm by 4-8 mm. 3. Tegument is covered with spines,the spines of P.westermanii are larger with bifid tips while of P.kellicoti spines have number of points.. 4. Ventral sucker is situated slightly anterior to the middle. 5. Eggs are yellowish brown in colour measuring 75-118x 42-67 um.	

Life cycle: Parasites generally live in pairs inside the cysts in the lungs and eggs are deposited in the cystic pockets from where they are voided through connecting channels into the bronchi or else when cysts rupture. They pass up to mouth and may be found rusty colour in sputum, subsequently pass out with the faeces. The eggs require 2-7 weeks to hatch and miracidium penetrate the snails of genera *Melania, Hua, Thiara, Brotia* etc. It produces one generation of sporocyst and two generation of rediae. The daughter rdiae produce cercariae with very short tail .After a short free life, they encyst on crayfishes and crabs . The final host become infected by eating infected crabs raw or partially cooked. After reaching inside host, metacercariae penetrate the wall of the small intestine within 30-60 minutes. The immature parasites enter the peritoneal cavity, penetrate through diaphragm and pleural cavity to reach in lungs.PP 30-36 days.

Pathogenesis and clinical symptoms: There is leucocytic infiltration around the parasites and fibrous tissue formation around the hazel shaped cyst.these cysts measure 6-10 mm in

diameter. There may be diffuse cirrhosis, cystic dilatation of the bronchi,and tubercle like lesions may be present.Parasites while migrating through the body of the host may reach other organs and these abnormal sites generally produce abscess and pseudotubercles.The migrating parasites cause eosinophilic peritonitis, bronchiolitis and granulomatous pneumonia.

Symptoms: infected animals are lethargic with intermittent cough,dyspnoea and pneumothorax. Sometimes parasites migrate to brain forming cyst there.

Diagnosis: finding eggs in the sputum.

Lesions may be detected by radiography.Lesions are found more frequently in the right caudal lobe of the lung.

Treatment: Praziquantel : 20mg/Kg.B.wt. given on two consecutive days is the drug of the choice.

Niclofolan 2mg/kg.B.Wt. 2 doses reduces egggg count by 89%.

Niclosamide: two doses of 100mg/kg.b.wt. reduces egggg count by 67%.

Albendazole: 50-100 mg/KG.B.Wt. for 14-21 days reduces the number of eggs in faeces and reduces pulmonary lesions in cats.

Prophylaxis:

Removal of snails.

Fresh water crustaceans should not be eaten as raw.

Family : Schistosomatidae

- All the members of this family found in the blood vessels of their definitive host and are commonly called as blood flukes.
- These are elongated and unisexual
- The male is stout and short and offen carries a female, which is longer, in ventral gutter-like groove called gynaecophoric canal, especially during copulation.
- The canal is formed by incurved lateral edges of the body. Pharynx is absent and two intestinal caeca usually unite posteriorly which may again bifurcate and unite, extending to the hind end.
- The genital pore is located behind the ventral sucker.
- Male may possess three to many testes depending upon the genus and the tstes may be situated anteriorly or posteriorly.
- The ovary is elongated and situated infront of the union of two intestinal caeca and also anterior to the vitellaria.
- The eggs are thin shelled, non operculated, often possessing a lateral or terminal spine.
- These are laid in the small blood vessels of the intestine and mesentery attached to it or of bladder or nasal passages. From here eggs pass through tissue and come out of the host alongwith the faeces or urine or nasal discharge.
- Redia and metacercaria stages are absent in the life cycle. Cercariae have bifurcated tail and , therefore called as furcocercus cercariae. They enter the final host through skin penetration.

The family include many genera, but important are schistosoma which has number of species parasitizing domestic livestock. The other genus of importance is Orientobilharzia having more than 30 testesThe species of other gernera are mostly found in birds and their wild counterparts.

Schistosoma:

male is stout and short with tuberculated tegument.

Female is longer, slender with smooth tegument.

The oral and ventral suckers are lie anteriorly.

The digestive system starts with the oral sucker followed by oesophagus and two intestinal caeca which reunite prior to its termination as a blind tube at the hind end.

The reproductive system is well developed. In male number of testes are 3-8 , placed dorsal posterior to the ventral sucker.

The female reproductive system consists of an ovary, an oviduct,

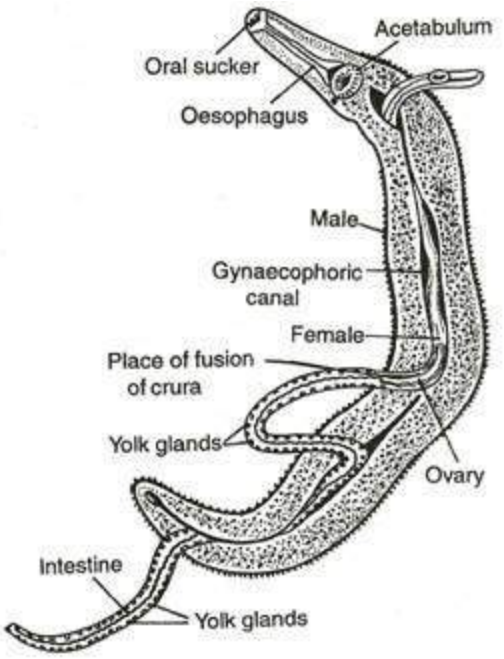
Vitelline glands, shell gland and ootype which passes forward to the uterus. T

he uterus contains one or many eggs at a time open through genital pore posterior to the genital sucker.

The Vitelline glands occupy most of the field behind the ovary.

The eggs are this shelled. Non operculated and provided with a spine either terminally or subterminally.

The shape and size of egg is specific to the species. The egg contains fully formed miracidia.



Schistosoma indicum: it is tuberculated blood fluke found in the mesenteric veins of equines, camel, sheep, goat, cattle and buffaloes in Indian subcontinent. Male are 4-22 mm and female are upto 26mm in length. The eggs have terminal spine and measure 57-140X 18-72um. Indoplanorbis exustus acts as intermediate host.

Schistosoma spindale

The flukes reside in the mesenteric veins of cattle, buffalo, sheep, goat, equines and dog in India, and far eat countries. The males are 5-13mm and females 7-16 mm long , the eggs are spindle shaped with a terminal spine and measure 200-400µm x 70-90µm. *Indoplanorbis exustus* acts as its intermediate host.

Schistosoma nasalis

The flukes resemble S. spindale and develop commonly in the veins of nasal mucosa of buffalo and cattle, sometimes in goat and sheep rarely in horses in the Indian subcontinent. The disease caused by this fluke is called nasal granuloma or nasal schistosomoisis. The eggs are typically boomerang shaped and measure 350-380µmx50-80µm, with a terminal spine at one end. *Indoplanorbis exustus* acts as its intermediate host.

Schistosoma incognitum

The flukes occur in the mesenteric veins of pig and dog in the Indian subcontinent. The parasite has also been reported from sheep, goats and rodents. The male is stout and measures 5.8mmx0.3mm.

There is a constriction behind ventral sucker. The cuticle bears prominent tubercles and spines except at the anterior end. There are 4-7 testes. The female is slender and measures 5.7x0.11mm in size with smooth cuticle. The uterus contains not more than one egg at a time. The sub oval and yellowish brown egg is flat at one side with a terminally placed short stout spine. These measure 86-148µm x36-81µm. *Lymnaea luteola* acts as intermediate host.

Schistosoma haematobium
The flukes occur in the posterior mesenteric arteries and the eggs are laid in the walls of urinary bladder, urethra and ureter of man. This infection has also been found in some animsl viz. baboons, monkeys, rodents and pigs in African countries. The blood flukes, resembling this species have been reported from Ratnagiri district (Maharashtra) in India. Adult males are 10-15mm and females are up to 20m long. The eggs have a terminal spine and measure 112-170µmx40-70µm. Snails of the genus *Bulinus* act as its intermediate host.

Genus: Orientobilharzia (Syn Ornithobilharzia)
The species of the genus have the intestinal caeca which anastomose in front of the median posterior intestinal caecum. The ovary forms a spiral at the anterior region of the body. The testes are numerous and the uterus is short containing only one egg at a time,
O.bomfordi: the flukes ocuur in the mesenteric and hepatoportal veins of sheep, goat, camel, horse, donkey, mule, buffalo, cattle and the cat in Russia, Iraq, Iran, China, France, Pakistan and in hilly tracts of India. The male tegument is tuberculated. The males are 4-8mm and the females are 3.8-8mm long. There are 70-80 testes and the ovary is spirally coiled. The eggs measure 72-77µmx18-26µm. they have a terminal spine and a short appendage at the other end. Species of *Lymnaea* act as its intermediate host.

Bivitellobilharzia nairi is another blood fluke which has been reported from elephants in India. There are other genera and species under fanily Schistosomatidae which parasitize canidae, felidae and other mammals, and variety of birds.

Life cycle:

The female flukes penetrate deep into the blood vessels of mucosa or submucosa of the intestine to deposit their eggs. The spinose eggs penetrate first the capillary and then the intestinal wall to reach intestinal lumen from where these are expelled along with the faeces. Peristaltic movements of the intestine and the proteolytic enzymes secreted by the miracidia within eggs help in expulsion of the eggs which takes place in 10-12 days. As the infection progresses, the adults withdraw a little upwards and lay eggs also in the mesenteric veins. From here, many eggs may be carried by blood to different organs like liver, lungs and brain. This causes involvement of other organs in chronic schistosomosis. The eggs laid by the females continue to develop during tissue phase as fully mature eggs with miracidium within them before voided in the faeces.

The eggs discharged through faeces when come in contact with water, hatch immediately depending upon temperature, availability of light and salinity of water. Thus, a ciliated pyriform miracidium is released from egg swims with the help of cilia in water in search of a suitable aquatic snail-an intermediate host. The miracidium has a short life span of 12-20 hours and does not feed outside. The miracidium penetrates the appropriate snail species, casts its cilia and changes into a sac like structure inside the snail called mother sporocysts possessing a number of germ cells. Each germ cell develops into a daughter sporocyst and settles in the digestive glands of the snail. Each daughter sporocyst produces germ balls which in turn develop to the forked tailed cercaria called *furcocercus cercaria*. The cercariae are the final larval stages of the blood flukes. Time taken by the miracidium to develop upto the cercaria stage ranges between 24-60 days. In India, mostly, the cercaria are liberated from the snail in the morning hours.

The furcocercus cercariae neither feed outside nor convert to metacercariae. They swim actively in the water in search of a suitable definitive host. As soon as they come in contact with the

body of the host, the cercariae penetrate the hosts' skin with the help of the hyaluronidase enzyme secreted by them. Percutaneous infection is the usual route of infection but ingestion of cercariae may also set up the infection. During penetration of the hosts' skin the cercaria lose its tail with other physiological changes resulting into a globular body called schistosomulum. In nature, large number of schistosomulae pass to the lungs and then to liver where further development and pairing takes place. Finally, these flukes pass to the mesenteric veins where they become adults and start laying eggs.

In case of *Schistosoma nasalis* infection, the developing flukes reach the veins of the nasal mucosa of the host where they become adults. In case *of S.haematobium*, the developing flukes reach to the posterior mesenteric arteries and lay eggs in the walls of the urinary bladder.

Pathogenesis

Amongst all helminth parasites, the group of blood flukes is an exception where the eggs produced by the females are mainly responsible for the pathogenesis inflicting harm to the host both mechanically and immunologically. Most of the species of Schistosomes viz. *S. indicum*, *S. spindale*, *S.incognitum* and others which reside in the portal and mesenteric veins of animals and man cause hepatointestinal schisosomosis. *S.nasalis* of domestic ruminants is responsible for causing nasal schistosomosis while *S. hamatobium* of man causes urinary or visceral schsitosomosis. Since the pathogenesis of blood flukes starts as soon as the furcocercus cercariae penetrates the hosts'skin there is another condition named as cercarial dermitis which occurs mainly in human beings whose skin is penetrated by the furcocercus cercariae of birds and of some mammals. Therefore, the pathogenesis is of four different kinds of schistosomosis in animals and man is given separately.

a) Hepato-intestinal schistosomosis (Visceral schistosomosis) it has mainly two types of clinical syndromes-an acute intestinal syndrome and chronic hepatic syndrome. The intestinal syndrome is seen in acute schistosomosis with the beginning of laying eggs by the females. The passage of eggs through the intestinal walls cause severe inflammatory changes and haemorrhagic lesions particularly in the posterior small intestine and caecum. In very heavy infection, the involvement from fore stomach to rectum may be seen. The intestinal mucosa becomes oedematous and covered with haemorrhagic foci. Numerous eggs are found in the mucosa and blood stained mucus covering the intestinal surface. The lamina propria is seen oedematous and infiltered with large number of eosinophils, lymphocytes, marophages and plasma cells. The eggs may be found free, in small granulomata or in small abscesses. Marked granulomatous response to eggs laid in submucosa is seen. Microscopically, the eggs are seen distributed in various depths of mucosa and submucosa of the intestine.in some cases, the eggs are attacked by phagocytes with some fibroblastic activity around the disintegrated shells of the eggs. Long standing infections are characterized by granulomatous lesions in all layers of the intestinal walls including its thickenings. Adult parasites cause phlebitis in the mesenteric veins in ruminants, in *S.indicum* infection, small pseudoabscesses develop around eggs in the intestinal walls with heavy accumulation of eosinophils and lymphocytes.eggs of *S. indicum* in the submucosa may be entrapped with the increase of fibroblastic cells. Similar pathological changes occur in *S. spindale* infection in the mucosa and submucosa of the intestine with marked inflammatory changes in domestic ruminants. In *S.indicum* infection in equines, thickened areas can be easily be identified with black pigmented raised character of the mucosa of intestine. In older cases, nodule likestructure in the intestine may seen surrounding number of eggs. Adults of *S. indicum* and other species in heavy infections may be found in pulmonary arteries. The lungs may be enlarged, brownish black in colour and contain reddish or grayish foci surrounding the adult flukes.
b) Hepatic Schistosomosis: it is observed in long standing cases and it is an immunologically disease resulting from hosts's cell mediated immune response to schistosome eggs in the liver. The soluble antigens of the eggs evoke inflammatory reaction and formation of

granuloma. Eventually, the eggs are destroyed and healing is accompanied by deposition of collagen and fibrous tissue formation which in heavy infection leads to clay pipe stem fibrosis. Chronic hepatic pathology occurs in schistosome infected animals which are repeatedly exposed to heavy infections. Granulomatous lesions and periportal inflammation occurs, followed by a progressive portal fibrosis. In *S. indicum* infection in domestic ruminants, the liver either assumes a dark grayish colour without development of cirrhotic nodules on its surface or the liver undergoes varying degree of nodulation, the nodules being white or pin head sized without any calcification in advanced cases. In S. *indicum* infection in equines, the liver undergoes varying degree of cirrhosis leading to calcification as a result of accumulation of eggs. The visible lesions on the surface of liver are pearl white or chalky white in character as minute pin head sized which become calcareous in later stage. In hepato- intestinal schistosomosis, clinical manifestations include profuse diarrhea or dysentry dehydration and anorexia in acute heavy infection. Anaemia, hypoalbuminaemia and sometimes oedema are present. There is marked decrease in production and loss of body weight. In chronic cases, animals are emaciated. There is eosinophilia, hypoalbuminaemia and someties neurological signs are seen.

c) Nasal Schistosomosis: *S. nasalis* infection produces severe clinical disease in cattle whereas buffaloes usually carry the infection without significant symptoms and lesions. The eggs of the fluke enter the mucous glands of the nasal cavity evoking a cellular reaction with formation of miliary abscesses. Rupture of abscess liberated the ova and repeated formation of abscess leads to fibrosis and cauliflower like growths in nasal passages of cattle. Such lesions show infilteration of eosinophils, lymphocytes, plasma cells, macrophages, a few giant cells and fibroblast cells. In buffaloes, the lesions develop only to the size of a pea or millet without spectacular clinical signs including snoring sound during respiration. The affected animals show rhinitis, mucopurulent dischsrge, manifested clinically by coryza, sneezing, dyspnoea and snoring (snoring disease). In other animals like sheep and goat, slight snoring symptoms are seen. The snoring disease in cattle adversely affect production, gain in body weight and drought capacity in bullock. The disease may sometimes become fatal.

d) Urinary or visceral schistosomosis: *S haematobium* adult worms are widely distributed throughout the pelvic and mesenteric plexus. Oviposition takes place mostly in the pelvic organs particularly in the wall of the lower urinary tract especially the urinary bladder. Excretion of eggs in urine is accompanied by mild inflammatory reaction. The presence of eggs and their secretion evoke development of delayed type of hypersensitivity reaction in the host leading to the formation of extensive granulomas. In chronic cases, size of the granuloma around the eggs decreases progressively. The obstructive lesion is well marked in the urinary bladder and less frequently in the gastrointestinal tract. There is ureteritis, appendicitis and unusual ectopic lesions in other body parts and are frequently associated with severe *S. haematobium* infection in man.

e) Cercarial dermatitis: it is schistosome dermititis also commonly known as Swimmers itch or dhobi itch in India. And also as clam digger's, hunter's itch, rice paddy itch, lakeside disease (Japan) Bader dermitits (Germany) or gae des nageurs (France). This disease can be a serious occupations problem for man e.g washerman (dhobhi) or others who frequently come in contact with water in ponds, river sides, ditches, rice paddy fields and other natural water collections for long hours repeatedly. It is also important in recreational areas throughout the world. Cercarial dermatitis is caused by the active penetration of furcocercus cercariae of non human schistosomes including birds into the skin of the man. In the beginning there is mild erythema, and oedema at the site of cercarial penetration. Repeated exposure causes a severe pruritus and a popular or pustular eruption followed by a severe dermatitis. The condition may persist for several days to several weeks if the lesions become infected secondarily. Besides non human mammalian schistosomes, avian schistosomes infecting various water fowl are very important for causing cercarial dermatitis. Cercariae of

S. spindale (water buffalo) may be the cause of rice paddy itch in rice growing countries. The disease in humans can be prevented by the use of protective water proof clothings during work in water. Repellants like dibutylpthalate andbenzyl benzoate may be applied on the skin of the body parts frequently coming in contact with water.

Diagnosis

- Examination of fresh faecal samples after fixation in 10% formaline since eggs containing miracidium hatch soon after coming in contact with water.
- Besides, fresh faeces may be diluted several times in water and exposed to light for searching miracidia under stereoscopic microscope.
- Diagnosis in living animals can be attempted by certain specific serological tests viz. Miracidia immobilization test (MIT), cercarien-hullen-reaktion test (CHR), circumoval precipitation (COP), HA test and CFT
- In case of nasal schistosomosis, clinical signs help in diagnosis. Demonstration of typical boomerang shaped eggs of *S. nasalis* in nasal washings and nasal scrapings. 1 ml of nasal mucus is mixed with 9ml of 10% KOH and kept under room temperature for 10-20 minutes. The ova pesent in the lower 1ml solution are counted. The process can be hastened by just boiling the mixture of 1gm mucus with 10% KOH and centriguging the solution, this removes mucus.
- *S. Haematobium* infection in humans may be diagnosed on the basis of history of fever and haematuria. Eggs of the bllod flukes are detected in urine.

Treatment

For treatment of haepatointestinal schistosomosis in animals, the drug of choice are tartar emetic, antimosan, lucanthone, hycanthone, niridazole and trichlorophon. For treatment of nasal schistosomosis, the drugs pf choive are antimosan, anthiomaline, tartar emetic, trichlorophon, niridazole and praziquantal.

Control

Chemotherapy with effective drugs is the only practical approach to reduce the infection rate in a population particularly where host range of the blood fluke is narrow. Treatment of all the clinical and sub clinical cases is recommended. So far, there is no prophylactic medication adopted in animal population. Proper disposal of faeces of animals, suspected for blood fluke infection, without any contact with water is suggested. The control measures also include elimination of snails, acting as intermediate hosts in ponds and water collections visited frequently by the animals, by both chemical and biological methods. Introduction of fish in water collections helps in the reduction of snail population.

Lecture-1

The name helminth is derived from the Greek words helmins or helminthos, a worm, and is usually applied only to the parasitic and non-parasitic species belonging to phylum Platyhelminthes and Nemathelminthes.

General Characteristics:

1. Nematodes are commonly called roundworms because they are round when viewed in cross section.
2. They are cylindrical, unsegmented, bilaterally symmetrical in structure and taper towards their anterior and posterior ends.
3. Sexes are separate in most species, a few are hermaphrodite.
4. Life cycle maybe direct or may include an intermediate host.
5. Nematodes that parasitize domestic animals are found in all parts of the body but are most commonly found in the digestive and respiratory tracts and in circulatory system.

External Morphology

Cuticle

1. Nematode bodies are covered by cuticle which is colourless and partly translucent.
2. It also lines the buccal cavity, oesophagus, excretory pore, vagina, cloaca and rectum.
3. The cuticle is also resistant to host digestive enzymes and in most nematodes is relatively impervious, allowing only the passage of water molecules and certain small water- soluble ions.
4. It maintains the form and structure of parasite and also provides an anchor for muscles.
5. It is provided with circular annulations or it may be smooth or it may have longitudinal striation

Cuticular modifications

The cuticle can be modified to form various structures:

1. **Papillae:** They are paired spine or finger like projections which maybe soft, fleshy and short. They may occur inside the rim of the mouth opening (buccal capsule) as **internal leaf crown** or on the rim itself as **external leaf crown**. When they occur anteriorly on both sides of oesophageal region they are known as **cervical papillae** and when they are present posteriorly on the tail region, then known as **caudal papillae.**
2. **Vesicle:** They are inflations of the cuticle around the mouth as **cephalic vesicles** and the anterior oesophagus as **cervical vesicle.**
3. **Alae: Cervical** and **caudal alae** are flattened wing or fin-like expansions of the cuticle in the oesophageal and tail regions, respectively. The **bursa** of the strongylids is the most exuberant development of alae, where caudal alae have become elongated lobes and caudal papillae have become elongated to form bursal rays.
4. **Plaques and cordons:** They are plate-like and cord-like ornamentations present on the cuticle of many nematodes of the superfamily Spiruroidea.

Nematodes

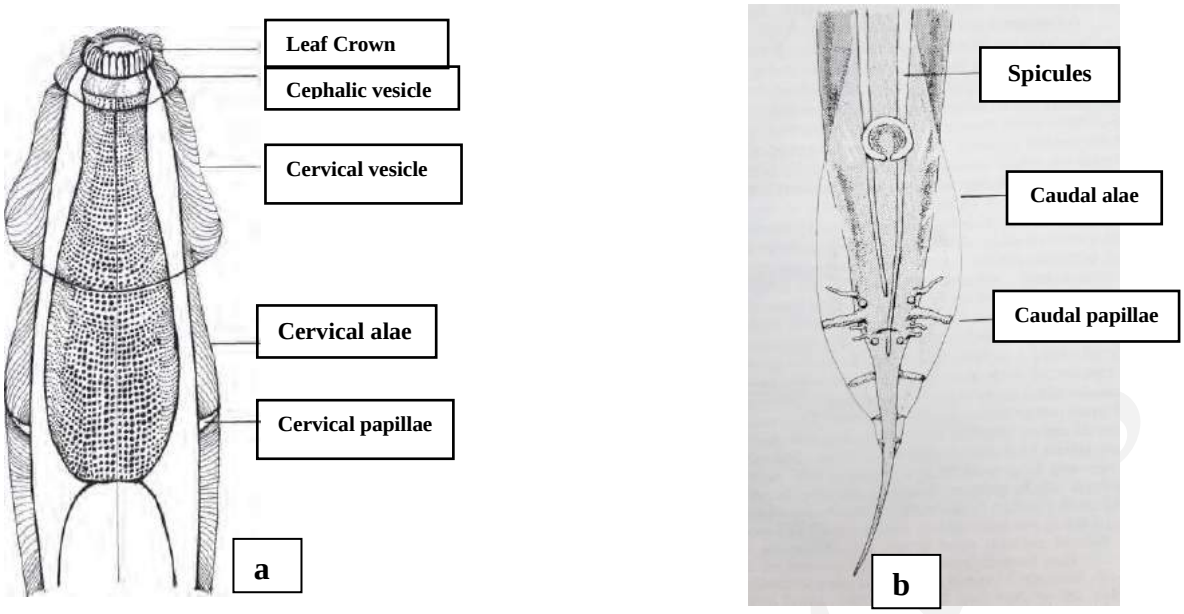
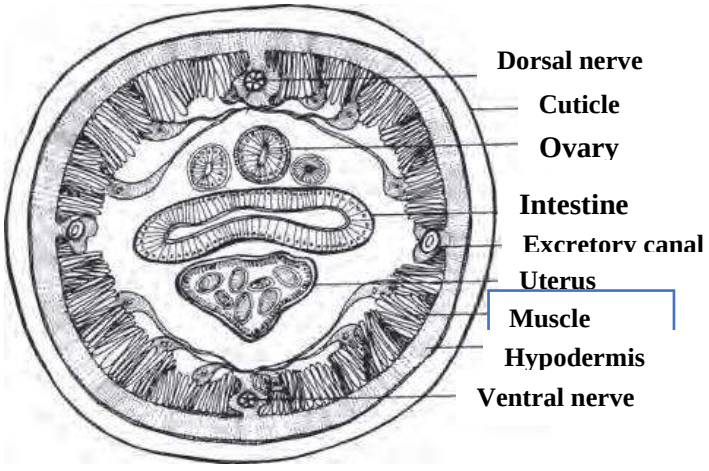


Fig. Cuticular modification of a generalised nematode (a) anterior region (b) posterior region of a male

Internal Morphology

1. Nematode body consists of an outer tube (the body wall) enclosing an inner tube (the digestive tract).
2. The fluid-filled body cavity (pseudocoelom) lies between these two tubes and contains the reproductive tract.
3. Unlike a true coelom, pseudocoelom does not possess a cellular lining or peritoneum.
4. There is no vascular system in the nematodes, instead the circulation of nutrients in the pseudocoelom is assisted by body movements and locomotion.
5. Body wall has three layers: cuticle, hypodermis and an inner layer of muscle cells. Hypodermis lies beneath the cuticle and it basically secretes the cuticle.
6. Hypodermis projects into the body cavity forming two lateral cords, which carry the excretory canals, and a dorsal and ventral cord carrying the nerves.
7. Locomotion is affected by undulating waves of muscle contraction and relaxation that alternate on the dorsal and ventral aspects of the worm.

Transverse section



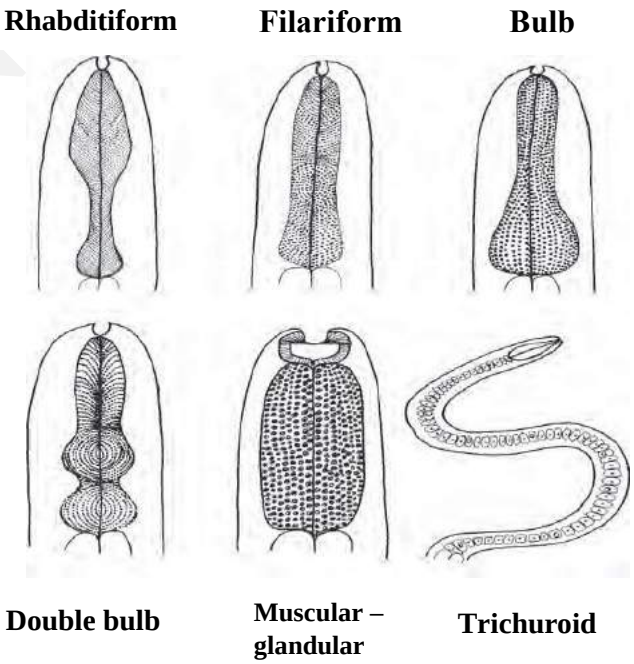
Digestive System

1. Consists of mouth/ stoma (in simple forms), buccal capsule/ cavity (in specialised forms), oesophagus, intestine, rectum (females), cloaca (males) and anus. The cloaca is a common termination for the intestinal tract and the vas deferens in adult males. In either case, the cloaca (males) and rectum (females) lead to an anus which usually opens to the outside at the posterior end of the body.
2. The mouth maybe surrounded by two or three lips, and leads directly into the oesophagus.
3. In some nematodes, instead of lips leaf crown is present, e.g. *Oesophagostomum* spp.
4. In some, the buccal capsule has a thick cuticular wall or teeth like structure, eg. *Ancylostomum* spp.

Oesophagus and its types:

It is muscular and pumps food into the intestine. It is a useful preliminary identification character for groups of worms. Various types of oesophagus include:

1. **Rhabditiform**: slight anterior and posterior swellings, is present in the pre-parasitic larval stages of strongylids, and in many free-living and plant-parasitic adult nematodes.
2. **Filariform**: simple and slightly thickened posteriorly, present in the infective, invasive and adult stages of strongylids.
3. **Bulb**: with a large posterior swelling, as in the ascaridoids (*Heterakis gallinarum*).
4. **Double bulb-shaped**: bulb constricted behind its middle, as in the oxyuroids (*Enterobius vermicularis*).
5. **Muscular–glandular**: oesophagus which is muscular anteriorly, the posterior part being glandular, seen in the spirurids.
6. **Trichuroid**: oesophagus has a capillary form, passing through a single column of cells (stichocytes), the whole being known as a stichosome.



Nematodes

Excretory and Nervous System

1. Excretory system is primitive, excretion occurs via osmotic regulation. This system consists of a pair of unbranched lateral vessels embedded in hypodermis which opens through excretory pore situated anteriorly.
2. Nervous system consists of number of ganglia connected by nerve fibres, forming the nerve ring around the oesophagus, from this central nerve ring, 6 nerve trunks runs anteriorly and 6 trunks to posterior part.

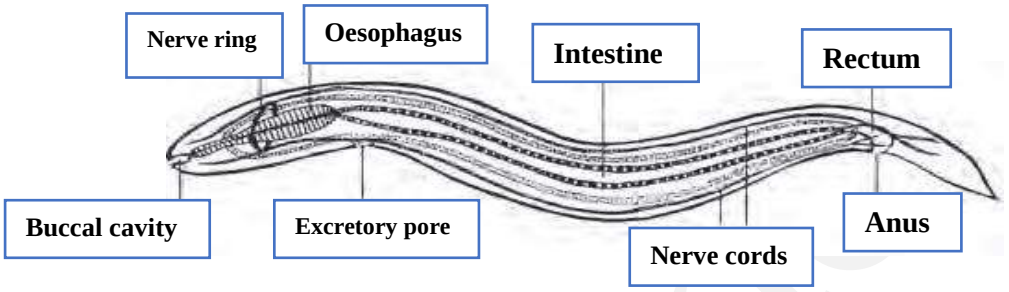


Fig. Longitudinal section of a generalised nematode showing digestive, excretory and nervous system

Reproductive System

- Sexes are usually separate, sexual dimorphism occur.
- Usually, male worms are smaller than the female because females need to accommodate the production of large quantities of eggs.

Male Reproductive System

1. Consists of single testis, vas deferens, seminal vesicle and ejaculatory duct. These ejaculatory duct opens into the cloaca.
2. Spicules are made of cuticle, are often paired and used in copulation to dilate the female vulva. Most of the species have two spicules. In some species they are absent (e.g. *Trichinella)* or only one spicule is present (e.g. *Trichuris)*
3. In many species, the wall of the cloaca is provided with cuticular thickening. This thickening assists the spicule movement.
4. Cuticular thickening on the dorsal wall of cloaca is called as *'gubernaculum'*, cuticular thickening on the ventral wall of cloaca is called as *telamon.*

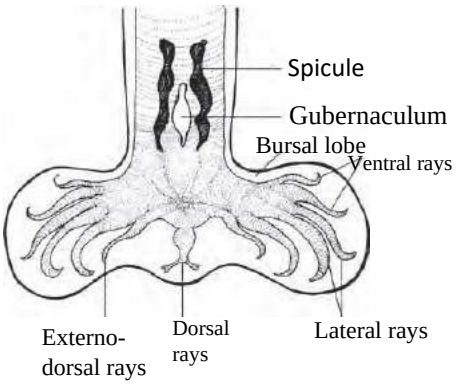


Fig. Male trichostrongyloid nematode bursa showing spicules and bursa

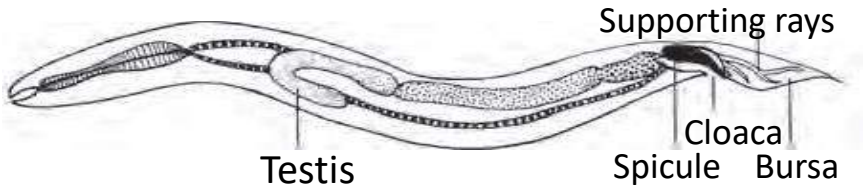


Fig. Longitudinal section of a generalised nematode showing reproductive system of a male nematode.

Female Reproductive System

The female organs comprise ovary, oviduct and uterus, which may be paired (didelphic) or sometimes single (monodelphic), ending in a common short vagina, which opens at the vulva. The location of the vulva can be a useful aid in diagnosis:

1. **Opisthodelphic:** vulva situated at the anterior end, uterus and ovaries running backwards.
2. **Amphidelphic:** vulva situated in the middle, the uterus may run in opposite directions.
3. **Prodelphic:** vulva situated at the posterior end, uterus and ovaries running forwards.

- Ovejector: A short muscular organ situated at the junction of uterus and vagina which regulates and assists in egg laying.
- A vulval flap may also be present in some nematodes.

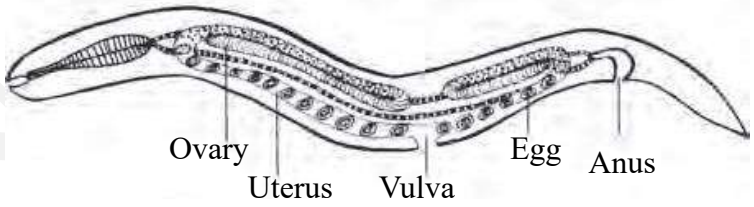


Fig. Longitudinal section of a generalised nematode showing reproductive system of a female nematode.

<u>Egg: Structure, Development and Hatching</u>

Nematode eggs differ greatly in size and shape, and the shell is of variable thickness, usually consisting of three layers:

1. **Inner membrane:** thin, has lipid characteristics and is impermeable.

Nematodes

2. **Middle layer:** tough and chitinous, gives rigidity and egg its shape and, when thick, imparts a yellowish colour to the egg. In many species this layer is interrupted at one or both ends with an operculum (lid) or plug (e.g. *Trichuris* eggs).

3. **Outer layer (embryo membrane):** consists of protein, this is an exogenous product of the nematode uterus which is very thick and sticky in the ascaridoids in which it is often referred as the 'mamillated' albuminous layer.

- Egg cell divides into 2, 4, 8, 16, 32, and so on. The developmental stage consists of morula and tadpole stage. Hatching may occur either:

1. Outside host (in the environment)
2. After ingestion by I.H or D.H.

- In environment hatching is controlled by temperature, moisture, larval movement and enzymes secreted by larva.
- Inside the host it is controlled by CO_2 concentration.

Basic Life Cycle of Nematodes

- Stages in nematode life cycle:

Egg → Larva (L1, L2, L3, L4, L5/immature adult) → Adult

- Two phases:

1. **Pre-parasitic phase:** occur either as free-living phase in external environment or inside an intermediate host. These include L1, L2 and L3.
2. **Parasitic phase:** Definitive host. These include L3, L4, L5 and adult.

- L3: connecting link between pre-parasitic and parasitic stages

Larval bionomics

- Nematodes moult four times during each life cycle with a moult occurring at the end of each larval stage.

(L1 to L2), (L2 to L3), (L3 to L4) and (L4 to L5)

- During moulting, the old cuticle is replaced by new one. The larva produces exsheathing fluid (leucine aminopeptidase), stimulated by carbon dioxide, which allows old cuticle to loosen and rupture. The L1 and L2 feed on bacteria.
- L3 can't feed because the cuticle of L2 is retained as a protective sheath around L3 larvae (infective stage), hence they are completely dependent on their stored reserves.
- In most of the nematode species, L3 is the infective stage where as in Ascarid L2 inside egg is considered as an infective stage.

Nematodes

Lecture-2

Order Ascaridida

The members of this order possess three large lips, which surround the mouth and may have caudal alae which are laterally placed.

The order consists of two super families:

- Ascaridoidea
- Oxyuroidea
- Subuluroidea

Super family Ascaridoidea

- Contains large nematodes
- Oesophagus lacks the posterior bulb.
- Male has two spicules but do not have a bursa.

Family Ascarididae

- In the members of the family, buccal capsule and pharynx is absent.
- Worms have three well developed lips, one dorsal and two sub ventral.
- The esophagus is club shaped.
- Tail of the male is usually without well developed alae but has several caudal papillae.
  Genera: *Ascaris, Parascaris, Toxocara, Toxascaris*.

Genus: *Ascaris*

Species *Ascaris suum* (pig) small intestine (Common name: Large Intestinal roundworm of swine)
Ascaris lumbricoides (man) small intestine.

- Size: male-15-25cmx3mm
- Female-up to 41 cm x 5mm
- Male has two spicule and each about 2mm long.
- There are a large number of precloacal papillae.
- Vulva situated at the posterior $1/3^{rd}$ of the body.
- Eggs: Measure: 50–75 by 40–55 μm, ovoid in shape with thick eggshell whose outermost layer is irregularly mamillated

Nematodes

Life cycle

Each female may lay up to 2 million eggs daily. In environment within 10 days at optimum condition, these eggs become infective containing second stage larvae. Infection occurs by ingestion of infected eggs with food and water or from the soiled skin of mother in suckling piglets. In this parasite, second larval stage inside the egg is the infective stage. After reaching inside the definite host eggs hatch and larvae penetrate the intestinal wall and reach liver through hepatoportal blood stream (24 hrs PI). From liver they are carried to lungs with blood where they moult to L3 stage. Larva in lungs is found after 4-5 days of infection. Larvae break open the alveolar capillaries and reach to bronchioles-------trachea--------pharynx-------mouth--------swallowed and reach in the intestine where they moult to L4 stage. In intestine they may be seen in between 14-21 days. Last moulting (L5) occurs 21-29 DPI and maturity reaches after 50-55 days. Prepatent period is 60-62 days.

Pathogenesis

Larvae may cause severe damage in liver and lung during migration. They may cause haemorrhages in alveoli and bronchioles, desquamation of alveolar epithelium, oedema, and eosinophilic infilteration in lungs. In repeated heavy infection marked pathological changes are observed in lungs. In such cases death may occur 6-15 days of infection.

Adult nematodes in the intestine are comparatively less harmful; they may obstruct the lumen of intestine if present in large numbers. Sometimes they may reach bile duct or may perforate the intestine and cause peritonitis.

Clinical symptoms

Depends on the severity of infection. Young pigs are chiefly affected. Symptoms of pneumonia are very common in suffering piglets. Adult parasites may cause diarrhea and poor growth rate.

Post mortem lesions

In the liver, the migrating larvae can cause milk spot or white spots, which appears as cloudy whitish spots of up to 1.0 cm in diameter on the surface of the liver, and represents the fibrous repair of inflammatory reactions to the passage of larvae in the livers of previously sensitised pigs.

Diagnosis

- On the basis of symptoms
- By faecal examination and larvae in sputum.
- Prevalence of infection is usually highest in pigs of around 3–6 months of age

Nematodes

Treatment

- Levamisole 7.5 mg/kg body weight
    - 8 mg/kg as drench.
    - 7mg/kg of feed (also effective against the majority of nematodes)

- Tetramisole 15 mg/kg in feed
  250g/25kg feed for herd management (mature and immature stages)
- Parbendazole 30 mg/kg orally (broad spectrum)
- Fenbendazole 5 mg/kg (ovicidal and broad spectrum)
- Cambendazole 20 mg/kg (feed supplement as top dressing)
- Piperazine compounds (piperazine citrate, adipate, dihydrochloride)-100-400 mg/kg. Administered along with food.

Prophylaxis

- Pig pens should be cleaned with caustic soda or by using boiling water.
- The sow should be treated before farrowing for ascarids.
- Skin should be scrubbed and washed to remove the eggs before farrowing.
- Young pigs should receive anthelmintic treatment when purchased
- Boars should be treated every 3–6 months.

Genus Parascaris
Species *Parascaris equorum*

- Found in the small intestine of equines.
- Males are 15-28 cm and females are up to 50cmx8mm.
- Rigid worm with large head.
- The large lips present at the anterior extremity by three small lips
- The vulva is situated at the end of the first quarter of the body.
- The mail tail has small lateral alae.
- There are five pairs of post cloacal papillae.
- A single median papillae found on the anterior border of the cloaca
- Spicules are 2-2.5mm long.
- Eggs are subglobular with thick, pitted, albuminous layer and measures 90-100µ in diameter

Life cycle

Similar to that of *A. suum.* Worms reach maturity in 80-83 days of infection.

Nematodes

Pathogenesis and Clinical symptoms.

Foals of 3-9 months are usually affected, immunity develops after 6 months of age. Sometimes more than 1000 worms may be present. Occasionally adult animals may be positive for the infection but there is no clinical significance. Migrating larvae may cause coughing and circulatory eosinophilia. Adult parasites may cause enteritis resulting into pale foetid coloured diarrhea. They may cause general weakness, harsh coat and animal become pot bellied. Sometimes parasites may migrate to bile duct or perforate intestine to reach peritoneal cavity. Sometimes the parasite may ball up and obstruct the intestine. In infected animals weight gain is depressed and infected foals have lower albumin.

Treatment.

- Thiabendazole 44mg/kg
- Mebendazole 10 mg/kg
- Fenbendazle 7.5 mg/kg
- Cambendazole 20 mg/kg

Prophylaxis

- Attention should be paid at the time of the birth.
- Foals should be allowed to run in clean paddocks.
- Boxes which are used for foaling should be cleaned at frequent intervals.
- Fresh food and water should be supplied to the animals.
- Manure should be disposed off at frequent intervals to a safe place.

Genus Toxocara
Species *Toxocara canis.*

- Adult parasites found in the small intestine of dog and fox.
- Males are up to 10cm and females are up to 18 cm in length.
- Adult head is elliptical due to the presence of a pair of large lanceolate cervical alae.
- Body is anteriorly bend ventral.
- There is no buccal capsule and the oesophagus lacks a posterior bulb.
- Male has caudal alae and spicules are .75 to .95mm.
- Female vulva at anterior 4th of the body.
- Eggs are sub globular, thick, finely pitted shells and measure 90-75µm.

Life cycle – Complex in nature

The life cycle may be completed by both ways –somatic as well as tracheal route.

Nematodes

If puppies are less then 3 months of age, life cycle occurs via tracheal route**.** Ingestion of eggs containing L2--------L2 free in duodenum----------penetrate intestinal wall and via lymph stream-------mesenteric lymph nodes-------portal blood----------liver (2 DPI)--------hepatic vein----------Heart----------pulmonary artery---------- Lungs(5DPI)----------trachea---------swallowed to reach stomach ----------Intestine. The 3rd stage larva is produced in lungs, trachea or oesophagus. 4th stage larva occur in intestine (2 weeks PI). Final moult to adult occurs in 3-4 weeks and patency occurs 4-5 weeks.

In pups above 3 months of age, somatic migration occurs. Following ingestion of embryonated egg, which hatch in the intestine, the liberated L_2 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 pregrancy) and enter into the foetus. When the larvae reaches the liver of the foetus, L_2 moult to become L_3. At the time of birth L_3 is present in the lungs of puppy. During 1st week of birth L_3 moults to L_4 in the lungs, by the 2nd week of birth L_4 moults to L_5 and pass to the intestine. Eggs appear in the faeces 23 to 40 days after birth of the pup.

In male dogs, larvae die out, usually after about a year. In female dogs, the encysted larvae remain infective for several years. *Toxocara canis* larvae can reactivate in bitches when they are in heat or before giving birth. This parasitic activity depends on the hormone cycle of the bitch and involves dormant larvae which are encysted in the mammary glands, uterus or muscle tissue.

Route of infection:

- Prenatal or transplacental
- Colostral (lactogenic). In this type of infection, larva develops into mature parasite in intestine without migration.
- By contaminated food and water. Bitch may also become infected by eating faeces of patent pup; eggs hatch in stomach and small intestine; molting, maturation without migration
- By predatory habits, when rodents (paratenic host) are eaten by dogs (no migration).

Genus Toxocara
Species *Toxocara cati*

- Location Small intestine

Nematodes

- Host Cat and wild felidae
- Worldwide including India but not very common
- Large stout worms but smaller than *T. canis*
- Male 3-7cm and females 4-12 cm long
- Cervical alea are large, narrow anteriorly and braod posteriorly giving arrow head appearance
- Spicules are 1.63-2.08mm long
- Eggs are similar to *T canis* (65-75 µm in diameter).

Life cycle
Direct. L2 (infective stage) develop within egg. There is no prenatal infection.

Main routes of infection:-

- Transmammary/lactogenic infection
- Contaminated food and water
- Consuming paratenic host

If cats acquire infection through *contaminated food and water*, then parasite follows the tracheal route of migration for development. First two days, larvae are found in the stomach wall. By 3rd day in liver and lungs----------lungs and trachea (5th day)----------stomach wall; many in lungs and trachea (10th day)----------stomach wall and contents (21 day). Later their number increase in intestinal contents. Migration occurs in L2 phase. L3 formed in stomach wall. L4 are larvae found in contents/ bowel wall/ bowel contents.

If infection is acquired through contaminated milk or paratenic host then larvae do not migrate through tracheal route and develop in the stomach wall (after entering as second stage) after changing into L3 stage and emerge as L4 again in the stomach contents on 13th day and get maturity in small intestine 21 day onwards.

Genus Toxascaris
Species *Toxascaris leonina*

Found in small intestine of dog, cat, fox and wild felidae and cainidae in most parts of the world.They are large stout worms and superficially resemble *T. canis* but different in following features:

- Males-2-7cm and female 2-10 cm
- Cervical alae, long narrow like *T canis* but anterior end is bend dorsally.
- Oesophagus lacks bulb at posterior extremity.
- Male bears two subequal spicules which are 0.7-0.15mm long.

Nematodes

- Female vulva is in anterior third of the body.
- Eggs are slightly oval with thick but smooth wall and measure 75-85 by 60-75 µm.

Life cycle

3-6 days after coming into the environment the eggs become infective (eggs containing L2). Infection may occur either through contaminated food and water or through paratenic hosts (mice, have L3). Prenatal and transmammary route of infection are absent. There is no tracheal migration. 2nd stage larva after hatching enter into intestinal wall and remain there for two weeks, here itself they moult to 3rd and 4th stage. At fourth stage they may be found in the lumen and mucosa. And fifth stage larvae are found in the intestinal lumen after six weeks. Pre patent period is 74 days.

Pathogenesis of ascarids in dogs and cats.

Heavy infection is most commonly seen in kennels and catteries maintained under poor hygiene. Heavy infection of young animals is very common. Heavy prenatal infection may lead to high mortalities in young pups. Eosinophilic granulomas in liver, lungs; causes a dry cough. Migration of large number of larvae may cause pneumonia. But more usual effect is uncomforting, vomiting and diarrheoa as worms mature in stomach and intestine. Each meal is rejected and animal is covered with stale vomit. Sometimes inhalation pneumonia may occur. Death frequently occurs 2-3 weeks after birth. In less severe infection of *T canis*, and infection of *T cati* and *Toxascaris leonina*, there is general weakness, intermittent diarrhea, pot bellied condition and anemia.

Adult worms sometimes migrate to aberrant sites (bile duct, intestinal wall) and symptoms come accordingly. Nervous symptoms sometimes also observed. They are probably due to irritation by adult worms in the intestine or by aberrant migration of larval stages to the brain.

Clinical symptoms

The animals are unthrifty; pot bellied with tucked up abdomen. The coat is dull, harsh, emaciation, anemia, restlessness, diarrhea and constipation. In pups, diarrhea and vomiting are very often observed.

Diagnosis

Disease may be suspected on the basis of symptoms and confirmed on the basis of faecal examination.

Nematodes

Treatment

The most popular drug used has been piperazine (overdose may induce vomiting and gastrointestinal disturbances in dogs), although this is being superseded by the benzimidazoles (fenbendazole and mebendazole) and by nitroscanate.

1. Treatment for reproductive females: Deworming females during reproductive periods and at the start of gestation. Females should be dewormed when they are in oestrus; this will destroy the adult worms and partially destroy dormant larvae which reactivate when the animal is in oestrus and at the beginning of gestation. Postpartum deworming of females:- Bitches and queens should be treated 15 days after the birth, then every 2 weeks until the litter is weaned (8th to 12th week).

2. Treatment for puppies: Puppies and kittens should be dewormed at 15 days old and then every 15 days until they are weaned. They should then be dewormed once a month until they are 6 months old. The deworming rate for puppies should be linked to the assumed presence of various stages of migrating larvae. Deworming needs to start before weaning because of the possible contamination from the mother's milk.

3. Treatment for adults: A quarterly deworming is advisable for adult pets, although faecal examinations can be performed at suitable intervals, from monthly to three-monthly, as an alternative to repeated treatments.

Prophylaxis

1. Good hygiene to prevent *T leonina*, *T cati* where oral infection leads to maturity. The routine treatment and cleaning of premises control these infections.
2. For *T. canis* where transplacental infection plays an important role in transmission, the long-term plan for regular treatment of adult animals for lowering down environmental contamination play an important role.

VISCERAL LARVA MIGRANS

This condition is caused in children, mainly by ingesting eggs of *T. canis*. Although larva of *T. cati* and *Toxascaris leonina* has also been incriminated to cause the condition. The larvae donot complete the normal migration but undergo developmental arrest. It is characterized by chronic granulomatous lesions in visceral organs, liver, lung, brain and eyes (ocular larva migrans) when larvae migrate in somatic manner. Pathological entity consists of enlarged liver, eosinophilic granulomatous lesions in liver, intermittent fever, pulmonary infiltration, eosinophil may reach upto 50%, loss of weight, loss of appetite, persistent cough. In ocular larva migrans, lesions are very severe and may resemble retinoblastoma. Condition is more usually seen in children of 1-5 years of age due to dirt eating habit or when they are allowed to play with puppies heavily infected with *T canis.*

Nematodes

Genus Toxocara

Species *Toxocara vitulorum.* (syn. *Neoascaris vitulorum*)

Found in the small intestine of cattle and buffaloes (in calves less than 6 months of age)

- Round worms, body does not taper much towards the extremities.
- The cuticle is thin and the worm appears translucent.
- Males upto 25cmx5mm and female 30cmx6mm.
- Three lips are present each with narrow extremity and broad base.
- Oesophagus, unlike ascarids, has a small ventriculus.
- Male has two equal spicules (measuring 0.99 to 1.25mm long) numerous precloacal and post cloacal papillae.
- Vulva in females is located $1/8^{th}$ of the body from the anterior end.
- Eggs are subspherical, thick walled and have pitted albuminous layer. Measures 75-95x60-75 µm.

Life cycle

A) **Calves below 6 months of age:** Harbour adult worms in S.I.---Gravid females produce eggs---outside, eggs undergo moulting and reach the infective stage (L2 inside egg shell).

B) **Newly born and suckling calves:** Get infected upon feeding on the milk/colostrum of the dam**,** in which larvae are present for up to 3–4 weeks after parturition. Ingested larva---grow in SI---moult twice---adult male and female worms. Worms are expelled gradually from 38 days after birth and by 6 months of age all worms are expelled.

C) **Calves above 6 months of age:** Infective eggs seldom results in patency. The larvae migrate to various tissues where they remain dormant. In female animals, resumption of development occurs in late pregnancy allowing further transmammary transmission.

Pathogenesis and clinical symptoms

Light infection may pass unnoticed, however, heavy infection may produce symptoms of diarrhea and steatorrhoea (passing of heavy, pale, greasy faeces). Animals pass mud colored and bad smelling faeces. Sometimes symptoms of severe colic resembling intestinal obstruction may be present. In India, this parasite contributes greatly to buffalo calf mortalities.

Diagnosis

Faecal examination for detection of eggs.

Nematodes

Treatment

- Preferably piperazine compounds are used-100-150 mg/kg
- Morantal tartarate 10 mg/kg body weight
- Fenbendazole 5 mg/kg
- Levamizole 7.5 mg/kg

Control

Calves must be dewormed within 10 to 15 days of birth.

SUPER FAMILY Oxyuroidea

- These nematodes do not have ventrolateral papillae or they are rudimentary.
- Two, one or no spicule.
- Oesophagus possessing a posterior bulb.

Family Oxyuridae

- Contains medium sized worms.
- Oesophagus has well developed posterior bulb.
- Male has a large number of papillae around cloacal opening.
- Females are much larger than males and has a long tapering tail.
- Vulva is situated near the anterior end. Eggs are flattened on one side

Genus Oxyuris

Species *Oxyuris equi* (pin worm of Horse)

- Found in caecum and colon of horse, mule and donkey.
- Cosmopolitan,very common in India.
- Male 9-12mm and female 40-150 mm long.
- Mouth is hexagonal surrounded by 3 inconspicuous lips.
- Male has obliquely truncated posterior end and bears a single pin shaped spicule. (120-150 µm) and a number of papillae.
- Body of female is thick anteriorly and narrow posteriorly into a tapering tail whose length increase with the age of the worm. The tail may be more than 3 times as long as the rest of the body.
- Vulva is situated anteriorly.
- Egg: ovoid, yellowish, thick-shelled, smooth and slightly flattened on one side with a transparent mucoid operculate plug at one end

Life cycle

After fertilization, gravid females contain large numbers of eggs in the gastrula stage. They migrate to the anus and extrude their anterior end, at the same time releasing

Nematodes

a viscous grey fluid containing enormous numbers of eggs (approx. 50,000 eggs/ female) which adhere to the perineal/perianal region. As she oviposits, the female shrinks and eventually passes out of the anus entirely and dies. Eggs do not hatch; the infective larva develops inside the egg. These eggs become infective in 3-5 days. Infection occurs by ingestion of infective eggs with feed and fodder, beddings or water. Larvae are liberated in the small intestine and L3 is found in mucosal crypts of colon and caecum. L4 produced after 8-10 days DPI. Sexually mature adult stage is reached in 4-5months after infection.

Pathogenesis

Fourth larval stage feed on intestinal mucosa while adults feed on intestinal contents. The chief feature of oxyuriasis in equines is anal pruritus caused by egg laying females.

Symptoms

Anal pruritus (irritation at anal region) may lead to restlessness and improper feeding which may lead to loss of condition and dull health. Intense pruritis around the anus causes the animal to rub on available solid objects, resulting in broken hairs, bare patches and inflammation, scarification and scaling of the skin over the rump and tail head. Tail acquires an ungroomed 'rat tailed appearance'.

Diagnosis

- Clinical symptoms
- Examination of perineal region for the presence of creamy colored eggs (Scotch tape test).
- The condition should be differentiated from mange mites and pruritus due to other reasons.
- The large white long tailed female worms are often seen in the faeces, having been dislodged while laying their eggs. *Oxyuris equi* eggs are rarely found on faecal examination of samples taken from the rectum, but may be observed in material from the perineum or in faecal material taken from the ground

Treatment

Regular treatment of equines for strongyles keep the animals infection free.

- Mebendazole 5-10mg/kg
- Cambendazole 20 mg/kg
- Other compounds such as Thiabendazole (40mg/kg), Fenbendazole (7.5 mg/kg), Oxibendazole (5-10 mg/kg) and Pyrantel (15-20 mg/kg) may be given.

Nematodes

Control

- Control depends on the good hygiene in the stables.
- Bedding should be removed frequently.
- Feeding appliances should not be contaminated with the bedding.

Genus Enterobius
Species *Enterobius vermicularis* (human pin worm or seat worm)

- Adults found in caecum, colon and appendix.
- Parasite resembles *O. equi.*

Female deposits eggs in the perianal and perineal region. Eggs become infective within 9 days and further infection occurs by the ingestion of infective eggs. Female produce an intense pruritus which cause restlessness and insomnia (sleeplessness). This parasite cause behavioral changes like inattention, lack of cooperation and probably feeling of shame.

Super family Subuluroidea

Family: Heterakidae

- Small to medium sized worms.with three lips around mouth.
- A small buccal cavity and pharynx.
- Lateral alae present.
- Oesophagus has three parts, small pharynx, a cylindrical middle part and bulbous posterior part.

Genus Heterakis
Species *Heterakis gallinarum* (caecal worm of poultry)
Found in caeca (large intestine) of fowl, guinea fowl, pea fowl, turkey,duck,goose and other birds.

Feature

- It is a small worm (male- 7-13 mm and female 10-15 mm long)
- Large lateral alae extends down the side of body.
- Oesophagus has well developed posterior bulb.
- Male at posterior end bears large alae, a circular precloacal sucker and 12 pairs of papillae.
- Spicules are unequal (right-2mm long and left-0.65 to 0.70mm long).
- Vulva in female opens slightly behind middle.
- Eggs measure 65-80x35-46 µm, have thick and smooth shell and are unsegmented when laid.

Nematodes

Life cycle

Eggs are excreted in faeces, become infected after 14 days at 27^0C. New birds become infected by ingestion of contaminated food and water. Earthworm may act as paratenic host and prepatent period is 24-30 days.

Pathogenesis and Clinical symptoms

Only heavy infection causes thickening of caecal mucosa with petechial haemorrhages. Whole pathogenic entity results in diarrhea and emaciation.

The main importance of this parasite is that the eggs of *Heterakis gallinarum* act as carrier of *Histomonas maleagridis* (protozoan in birds) causing Black head disease in birds. The organism can be transmitted from fowl to fowl in the egg of *Heterakis* and in earthworms containing hatched larvae of the worm.

Diagnosis

By finding the eggs or by postmortem examination.

Treatment

- Phenothiazine 1 gram/bird. It is mixed with 60 g feed given for six hours after overnight fast.
- Hygromycin B 0.25% as feed mix.
- Mebendazole 2g with 28 kg of feed.
- Tetramisole 10% solution in drinking water.

For prevention sanitary precautions are useful.

Genus **Ascaridia**
Species ***Ascaridia galli***

- Found in the small intestine of fowl, turkey and other gallinaceous birds.
- Large, stout worms, male- 30-80mm and female 60-120mm long.
- Three large lips are present.
- Posterior bulb in the oesophagus is absent.
- Male bears at the posterior end, small alae a circular precloacal sucker with thick cuticular rim and 10 pairs of papillae.
- Spicules are subequal and 4mm long.
- Vulva in females, near middle of the body.
- Eggs are ellipsoidal, have thick smooth wall, usually unsegmented when laid.

Nematodes

Lifecycle

Direct.

Eggs pass in faeces, develop in environment and become infective in 10 days. L_2 in eggs is the infective stage. Infection occurs through contaminated food and water. Eggs are very resistant and remain infective in a shaded place for three months. Earthworms act as paratenic host. Eggs hatch→for first 8 days, larvae live in the lumen of the intestine. Afterward (8-17days) enter intestine mucosa, subsequently they reenter the lumen and reach maturity in 6-8 weeks.

Pathogenesis and clinical symptoms

Young birds are more susceptible than adult exposed birds. Dietary deficiency of Vitamin A, B, B_{12}, many minerals, protein predispose. Chicken over three months more resistant due to increase goblet cells in mucosa. Most serious infection found in chicken of 1-3 months of age. Developing larval stages penetrate the duodenal mucosa causing haemorrhages and enteritis. At this stage birds become anaemic. Birds become weak, emaciated, egg production decreases. It may also cause intestinal obstruction.

Diagnosis

By finding eggs in the faeces.

Post mortem examination.

Treatment

Piperazine compounds are highly effective for *Ascaridia galli*.

Piperazine adipate	330-440 mg/kg of feed
Piperazine citrate	440 mg/litre of water for 24 hours.
Mebendazole	2g/28 kg of feed
Tetramizole	10% solution in drinking water.

Prevention

Young birds should be kept separate.

Poultry houses should be well drained.

Deep litter (moist one) around feeding and water pans should be changed with dry one.

Nematodes

Lecture – 3

Order Rhabditida
Super family Rhabditoidea

- Oesophagus in members of this order have long anterior or cylindrical portion and posteriorly a bulb, sometimes median bulbar swelling.
- Members have rhabditoid type of oesophagus characterized with anterior pseudobulb and posterior valved bulb.
- Excretory system is symmetrical and H- shaped.

Family Strongyloididae

- It has worms which are saprophytic in free living and parasitic generations are found in gut of vertebrates.
- They are heterogenetic.
- Parasitic generations have cylindrical oesophagus and free living generations have valvulated oesophageal bulb.

Genus *Strongyloides* (Common name: Thread worms of ruminants)

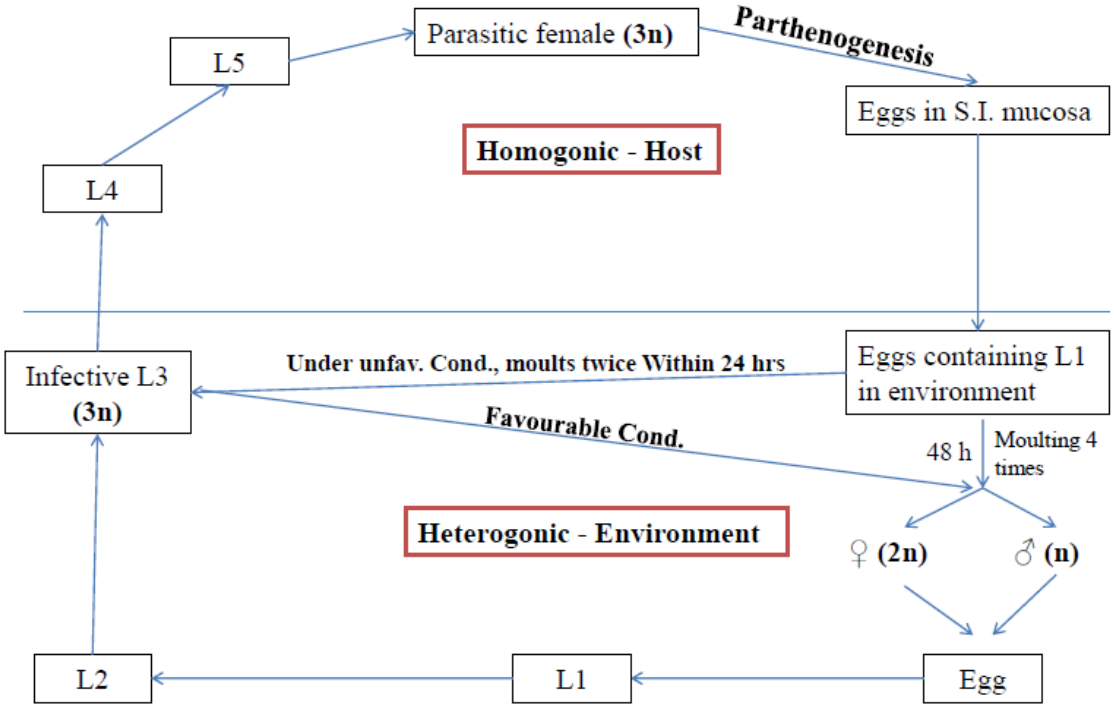
- Oesophagus is rhabditoid in free forms.
- Vulva near the middle of body. Uterus is intertwined with intestine giving the appearance of *twisted thread*.
- Non parasitic generations produce parasitic generations.
- In parasitic forms oesophagus is cylindrical without posterior bulb (filariform). The infective larvae of parasitic generations are capable to penetrate host skin.
- Adult females are parasitic, oesophagus occupy up to 1/3 of length

Species	Host	Location	Size	Eggs
Strongyloides papillosus	Sheep, goat, cattle.rabbit, wild ruminant	Small intestine	3.5-6mm	Eggs contains larva when passed in faeces
S. stercoralis	Man,dog,fox, cat	Small intestine	M-0.7mm and F-2.2mm	Larva is passed in faeces
S. cati	cat	Small intestine	2.73-3.33mm	Eggs contain larva which is poorly developed
S. westeri	Horse,pig, zebra	Small intestine	Up to 9mm long	The hatched L1 is passed out in the faeces.
S. ransomi	pig	Small intestine	3.33-4.49mm	Eggs Contain larva

Nematodes

Life cycle

- Unique, different from all other nematodes.
- Lifecycle may be direct heterogonic or homogonic.
- Life cycle can be complete parasitic or free-living or a combination of both.
- Parasitic Phase: composed entirely of female worms in small intestine and these produce larvated eggs by parthenogenesis. L.C. is direct homogonic.
- Free living stages: consists both of adult male and female. L.C. is direct heterogonic.



Route of Infection:- Skin Penetration, Oral Route

- Skin: larvae--- skin capillaries---venules---via blood to lungs---tracheal migration
- Oral: larvae---penetrate oral mucosa---systemic migration
- Prenatal and transmammary route of infection have been reported in pig, horse, sheep and cattle – no systemic migration occurs

Pathogenesis and clinical symptoms

Pathological changes by this parasite causes erosion of intestinal mucosa and fluidy gut contents. Pathological lesions are usually seen in young piglets. Infection is acquired orally from teat, udder, from penetration of larvae from soil and more commonly from milk. A protein losing enteropathy is commonly observed and mortality in young piglets may reach upto 50%. Main symptoms are anorexia, diarrhea which becomes haemorrhagic. *S. westeri* in foals produce diarrhea. Parasite in heavy infection may be responsible for high incidence of diarrhea in nursing foals. Foals usually develop a immunity to infection at 15-23 weeks after birth. From *S. stercolaris* (usually during summer season, when the weather is hot and humid) severe inflammation and sometimes sloughing, necrosis of intestinal mucosa of dogs may occur. In such cases, there is blood

stained diarrhea, dehydration followed by death. The larvae of *S. papillosus* is associated with the introduction of organism of foot rot *Bacteroides nodus* into skin around the feet of sheep.

Immunity
Only the young animals are the sufferers.

Diagnosis
Demonstration of eggs or larvae(dog) in the faeces.

Treatment

- Sheep

Thiabendazole 75mg/kg body weight is highly effective.

- Pig

Thiabendazole 50mg/kg body weight
Levamisole 5-10 mg/kg body weight

- Dogs

Diethylcarbamazine 100 mg/kg body weight
Dithiazinine (10 daily doses) 5 mg/kg body weight
Thiabendazole 50-75 mg/kg body weight

- Horse

Cambendazole 20 mg/kg body weight
Fenbendazole 50 mg/kg body weight

Prevention

- To provide clean,dry quarters as larvae are susceptible to dessication.
- Good hygienic management.

Order Strongylida

- The members possess 6, 3 or no teeth and if present then small.
- Corona radiata (leaf crown) may be present.
- Female reproductive system is well developed.
- Mouth with bursa and rays which are usually well developed.
- Oesophagus is club/filariform shaped.

Bursa in male
Copulatory bursa is well developed in posterior part of males. This structure consist of cuticular alae which usually form two lateral lobes and a dorsal lobe, enclosing the posterior extremity and supported by modified caudal papillae known as Bursal rays. These rays are arranged in a definite order and contain muscle fibers.
There are two ventral rays (ventroventral and lateroventral), three lateral rays (an anterolateral or externolateral, mediolateral and posterolateral) and a set of dorsal rays (comprising a externodorsal ray on either side and a dorsal ray which may be divided). The hind end of male is enclosed in the bursa is called genital cone. Therefore usually

Nematodes

two equal spicules, a gubernaculums (dorsal wall of cloaca) and a telamon (ventral wall of cloaca)

Superfamilies:

1. Strongyloidea
2. Ancylostomatoidea
3. Trichostrongyloidea
4. Metastrongyloidea

Superfamily: Strongyloidea

Include worms which have well developed mouth surrounded by corona radiate (leaf crown).
Teeth or cutting plates may be found in buccal cavity.
In males, copulatory bursa is well developed.

Family Strongylidae

Members have well developed buccal capsule.

Genus *Strongylus*

- Include large worms.
- Mouth opening is surrounded by corona radiata or leaf crown
- They have a well developed buccal capsule, may or may not have teeth.
- They have a well developed copulatory bursa with typical bursal rays (present in males).
- Spicules are simple, slender and equal.
- Vulva in female in 1/3rd of the body from posterior end.
- Eggs are oval, thin walled and segmented when laid.
- Members found in the large intestine (caecum, colon) of equines (horse, mule, donkey) throughout the world.

Species	**Size**	**Characters**	**Buccal capsule**
S. equinus	Male-26-35mm Female 38-47mm	Large, dark grey or red in color when fresh	Large oval base has one dorsal tooth which is bifid at the tip and two subventral teeth at the base.
S.edentatus	Male- 23-28mm Female 33-44mm	Large, head is separated from constriction	Large cup shaped, no teeth present at the base.
S. vulgaris	Male-14-16mm Female-20-24mm	Relatively small	Roughly oval, two ear shaped dorsal teeth at the base.

Nematodes

Lifecycle

Eggs are excreted in the faeces of the definitive host which become infected.

In ***S. equinus,*** larvae penetrate the wall of intestine (caecum and colon) and produce nodules. After 11 days, 4th larval stage develops in nodules which migrate to liver via peritoneal cavity. In liver, they wander for 6-8 weeks or more. From liver, they reach peritoneal cavity, appear in the large intestine, moult to L5 and reproduce in the lumen of large intestine. The prepatent period is 260 days.

In ***S. edentatus,*** infective larvae enters the wall of intestine and via portal system pass to the liver, where 4th larval stages are produced after 11-18 days. These stages wander in liver for about 9 weeks. From liver, they migrate to peritoneal cavity of right abdominal flank and cause haemorrhagic nodules. From peritoneum they reach intestine. Prepatent period-300-320 days.

In ***S. vulgaris***, third larval stages penetrate the intestinal wall when they produce 4th larval stage after 8 days. Now these larval stages penetrate blood vessels and migrate towards cranial mesenteric artery, causing thrombus formation and aneurysm (verminous aneurysm). After 45 days, these larvae again reach to the mucosa of caecum and colon via arterial system where they moult to 5th larval stage and reach maturity here. There is formation of nodules around the larvae, which rupture thus releasing adult parasites into intestinal lumen. Prepatent period is 6-7 months.

Pathogenesis and Clinical Signs

Adult parasites which attach to mucosa of large intestine suck blood. In heavy infection it may result to anaemia. Small superficial ulcers are also present. Migrating stages are responsible for severe losses. Primary lesions are produced in large intestine by migrating larvae of all three species.

The larvae of *S. vulgaris* produce severe lesions in arterial system (primarily in cranial mesenteric artery). The inflammatory lesions in arteries cause thrombus formation which may be of several centimeters in size. Detachment of such thrombi may lead to rapid death, if they present at anterior end of artery. After migration of larvae from arterial system; the arterial lesions are resolved. The consequences of such lesions in arterial system are varied. Detachment of large thrombi may lead to occlusion of iliac artery which may lead to temporary lameness. The larvae of *S. equines* produce haemorrhagic tracts in liver. The clinical symptoms include colic and anorexia. The larvae of *S. edentatus* may produce haemorrhagic nodules in peritoneum of right abdominal flank. In heavy infection the abdominal cavity is dotted with such nodules. Peritonitis, toxaemia, jaundice and fever are main consequences.

Diagnosis

Difficult to identify species by eggs for which culture is needed. But for treatment, presence of Strongyle eggs is sufficient indication and with 1000 EPG of faeces, treatment is recommended.

Treatment

- Thiabendazole 44mg/kg body weight (adult), 440 mg/kg body weight for migrating stages.

Nematodes

- Fenbendazole 7.5 mg/kg body weight, 50 mg/kg body weight for migrating stages.
- Cambendazole 20 mg/kg body weight (adult)
- Oxibendazole 5-10 mg/kg body weight(adult)
- Ivermectin - Effective for migrating stages

Control

- Routine treatment at the interval of 2 months by rotating the anthelmintics.
- Pasture management should not be over stocked or overgrazed.
- Provide clean pastures.
- Manual removal of faeces form pastures
- Alternate grazing with sheep and cattle.
- Proper disposal of manure by allowing fermenting which will generate heat.

Genus : Triodontophorus

- These parasites vary from 9-25mm in length.
- Found in the large intestine of equines.
- Buccal capsule is subglobular and thick walled.
- It has three pairs of teeth at its base.
- Spicules end in small hooks.

Developmental cycle of Triodontophorus is unknown but they are found restricted to bowel mucosa.

Species	**Location**	**Host**	**Distribution**
T. serratus	Large intestine	Horse, mule, ass and zebra	World wide
T.brevicauda	Large intestine	Horse and ass	World wide
T. minor	World wide	donkeys	World wide
T.tenuicollis	World wide	Horse	World wide

They may cause haemorrhages at the site of attachment. Other pathogenesis is same as previous species of adult parasites. Treatment: same

Genus : Creterostomum

Sp. Of this genus resembles Triodontophorus, but they have no teeth in buccal capsule. Genus has two species which are found in large intestine of equines.

C. acuticaudatum Found in horse, mules, ass and other equines. Worldwide in distribution.

C.tenuicauda Pony in India.

Life cycle is unknown.

Nematodes

Family Trichonematidae

These strongyles have short cylindrical or annular buccal capsule. Leaf crown present.

Genus: **Cyathostomum**

- Buccal capsule is short, thin walled and without teeth.
- Spicules of male have barbed tips.
- Vulva of female near anus.

C. tetracanthum	Caecum sometimes colon of of horse and other equines.
C. coronatum	Large intestine of equines throughout the world.
C. labiatum	Large intestine of equines throughout the world.
C. labratum	Caecum of ass

Genus: Cylicocyclus

- Genus is similar to Cyathostomum.
- The internal leaf crown is small and rod like.
- The buccal capsule is short with thin wall.

C. nassatus	caecum and colon of equines.
C. leptostomum	horse and ass

Genus: Cylicodontophorus

- These nematodes are 7-14mm long.
- Internal leaf crown elements are long and broad.
- Buccal capsule is short and thick walled.

C.bicoronatus	caecum and colon of horse(cosmopolitan).
C.mettami	caecum and colon of horse(cosmopolitan).

Life cycle

Larvae develop in caecum and colon. Larval stages invade mucosa and become encapsulated and subsequently return to lumen of large intestine of equines and reach to maturity. The period of encapsulation varies greatly from one species to another. Some species have prepatent period of 3 months while others remain encapsulated for several months.

Pathogenesis

These parasites are usually found in large number. Heavy infection may cause desquamation of epithelium and haemorrhagic enteritis. Larval stages produce large number of nodules which may lead to profuse diarrhea and colic.

Strongyle infection in Equines

Some of strongyles may be blood suckers. Faeces become soft and bad smelling. There is a diarhhoea, loss of appetite, animal becomes weak, emaciated and anaemic.Oedema may develop at abdomen and legs.

Nematodes

Genus: Oesophagostomum

- Small, stout, worms, commonly known as Nodular worms. Mouth is rounded with a mouth collar. Corona radiata is present.
- Buccal capsule is shallow, cylindrical or ring shaped.
- A ventral cervical groove is present near anterior end.
- Male has well developed bursa at posterior end and two equal spicules.
- Female, vulva is slightly anterior to anus.
- Eggs are oval, thin walled and segmented when laid. They measure 73-89x34-45µ.

Species	D.H.	Location	size	Leaf crown	Cephalic vesicle	Cervical alae	Cervical papillae
O.columbianum	Sheep, goat	LI (colon)	M-12-16mm F-15-21.5mm	External leaf crown has 20-24 and internal leaf crown has 40-48 elements	Present but not much inflated	Large hence anterior end is curved dorsally	Present just below cervical groove I oesophageal region
O. venulosum	Sheep, goat, camel	-do-	M-11-16mm F 13-24mm	Ext. leaf crown has 18 and internal leaf crown has 36 elements	-do-	Absent so ant. End is straight	Present; situated behind oesophageal region.
O. radiatum	Cattle, buffalo	-do-	M14-17mm F 16-22 mm	Ext. leaf crown absent and internal leaf crown has 36-40 elements	Large and inflated	present	Present, just below the cervical groove in oesophageal region
O. dentatum	pig	-do-	M 8-10mm F-11-14mm	Ext. leaf crown has 9and int. leaf crown has 18 elements	prominent	absent	Present but towards posterior end of oesophagus

Nematodes

Life cycle

Eggs are passed in faeces. Infective stage is reached in six to seven days at optimum condition. After ingestion, infective larvae exsheathed in small intestine and penetrate in wall anywhere from pylorus to rectum., get coiled up in muscularis mucosae and form cysts. Here third moult occur. Normally they return to lumen of gut after 5-9 days and pass to colon where they grow to adult after 4th moult. Prepatent period is 41 days. A few larvae is retained in mucosa especially in previously exposed animals.

Pathogenesis

The disease is called nodular worm disease /knotty gut/ pimply gut. In lambs or in older animals those who have no previous exposure, the larvae migrate but there is no tissue reaction, so large number of adult worms are found in colon but there are no nodules in the wall. If there is a previous sensitization, then during migration, severe reaction takes place around the fourth stage larvae (leucocyes especially eosinophils and giant cells). These cells accumulate around larvae. They are encapsulated by fibroblast. Usually after 3 months, the larvae either leave the nodule after casseation or parasite die. If free, then migrate in muscle fibre.

Nodules have opening through which pus is discharged. In such intestine, number of nodules will be high, but number of parasites in colon usually less. Extensive nodule formation may lead to interference with absorption and digestion. Nodules may rupture to peritoneal surface causing peritonitis and adhesions. Adults cause marked thickening of bowel wall. It has effect on appetite, growth and wool growth.

Clinical symptoms

There is persistent diarrhea, exhaustion and death until animal is removed from pasture. Faeces dark green in colour, contains mucous and blood. There is progressive emaciation and weakness. Skin becomes dry. Chronic symptoms of oesophagostomiasis in sheep is extreme emaciation, cachexia with atrophy of muscles ending complete prostration for 1-4 days and then death.

Diagnosis

In chronic cases, autopsy may be required of few representative animals.

Genus : *Chabertia*

Species: ***C. ovina***

- Found in colon of sheep, goat, cattle, buffaloes.
- Small, stout worm.
- Male-13-14mm and Female-17-20 mm long.
- Anterior end is slightly bent towards ventral side.
- A large bell/ cup shaped buccal capsule is present.
- Its opening is surrounded by a dorsal row of small cuticular elements, representing leaf crown.
- There is shallow ventral cervical groove and anterior to it an inflated cephalic vesicle.

Nematodes

- Male bursa is well developed and spicules are of equal size.
- Vulva, open posteriorly, slightly in front of anus.

Life cycle
Direct
Infection occurs per os. Third larval stages develop in intestinal wall and fourth stage comes out in lumen. Fourth ecdysis occur usually after 24 days. Immature adults pass to colon. Prepatent period-49 days.

Pathogenesis
These worms attach themselves to the mucosa of colon by means of buccal capsule and draw in plugs of mucosa. They suck blood only in accidental cases. The adjoining parts of mucosa show an increased activity of lymphocytes and eosinophils. More significant loss of plasma protein from mucosa causing hypoalbuminemia and weight loss.

Clinical symptoms
Include marked diarrhea with much mucous and blood. In severe condition animal becomes anaemic and die.
Chabertia infection may be responsible for reduction of wool growth in sheep.

Diagnosis
Culture of larvae for specific diagnosis.

Family Syngamidae

- Buccal capsule cup shaped.
- Vulva in anterior part of the body.
- Parasite of respiratory tract.

Genus: *Syngamus*
Species***: Syngamus trachea*** (gapeworm or forked worm)
- Occurs in trachea of turkey, fowl and other birds.
- Male-2-6mm and female-5-20mm long.
- Mouth opening wide without leaf crown.
- Buccal capsule is cup shaped bearing 6-10 small teeth at its base.
- The large reddish female and the small whitish male are permanently *in copulation forming a 'Y' shape*.
- Eggs: morulated, with a thickened operculum at each pole, 70-100 x 43-46 microns

Life cycle
Eggs escape under the bursa of the male and are carried up the trachea in the excess mucus produced in response to infection. Egg of parasite are coughed up and swallowed by the host which pass out through faeces. Unlike other strongyloids the L3 develops within the egg. Infection may occur by one of three ways: firstly by ingestion of the L3 in

Nematodes

the egg, secondly by ingestion of the hatched L3, or thirdly by ingestion of a paratenic host containing the L3. The most common paratenic host is the common earthworm, but a variety of other invertebrates including slugs, snails, beetles and some flies may act as transport hosts where larvae may live for several months or years. Through blood circulation, infective larvae reach lungs (60 hours); In lungs they are found in bronchi where they migrate towards large bronchi where copulation takes place and finally they reach trachea. In trachea they reach as early as 7th day. Prepatent period-17-20days.

Pathogenesis
Migration of larvae through lungs cause haemorrhages, oedema and pneumonia. In trachea adult parasites cause catarrhal tracheitis and suck blood. They also produce large amount of mucous.This mucous occludes air passage causing difficult breathing.

Clinical symptoms
Young birds (poultry) are mainly affected but turkey can suffer at any age. Symptoms are dyspnoea and asphyxia because of mucous accumulation in trachea. Birds shake its head, may cough or may extend the neck,opens beak and perform gaping movements. In severe cases death may occur due to asphyxia.

Diagnosis
Disease suspected on the basis of symptoms and finding the eggs in the faeces. Disease may be confirmed by sacrificing the representative birds.

Treatment
Thiabendazole .3-1.5g/kg orally
Mebendazole and fenbendazole are effective at level of 0.01% in feed for 7-14 days.

Family : Stephanuridae

- Cup shaped buccal capsule containing teeth.
- Vulva near anus.
- Contains parasites of kidney and perirenal tissue.

Genus: *Stephanurus*

Species *S. dentatus* (kidney worm of swine)
They occur in perirenal fat, pelvis of kidney and wall of ureters. In rare instances they have been reported from liver of cattle.in pigs as erratic parasite,they have been reported in liver, thoracic and abdominal organs and spinal cord.

- Female 30-45mm and male 20-20mm long.
- Cuticle is translucent.
- Buccal capsule is cup shaped with thick walls.
- Six triangular teeth are formed at the base. Rim of buccal capsule bears small cuticular leaf crown and six external cuticular thickening (epaulettes).
- Male has poorly developed bursa, spicules are of equal size(0.66-1.0mm long).
- Vulva situated close to anus.

Nematodes

- Eggs are thin shelled and appear only in the urine.

Life cycle
Direct or sometimes involve paratenic host
Modes of Infection:
- By ingestion of the free L3
- Ingestion of earthworms (paratenic host) carrying the L3
- Percutaneously

Adult parasites are found in cysts which may be present in kidney or in nearby areas through an opening. These cysts communicate with ureters. Eggs are passed in urine. At this time they are 32-64 cell stage. These eggs hatch in 24-36 hours and larvae reach the infective stage after 4 days. Eggs and larvae are sensitive to freezing and drying. In moist environment they can live for 5 months, but most of them die after 2-3 months. Earthworms may act as transport host in which larvae may survive for several weeks to months.

Infection may occur *per os* or skin penetration. After 70 hours, they moult to L_4 either in the wall of the stomach or in skin, depending upon source of infection. From both the sides of entry, larvae reach liver via portal blood vessels (oral route) or lungs (subcut route in 8-40 days). They migrate in liver for 3 months and then migrate to peritoneal cavity. After reaching the perirenal tissue they perforate the wall of ureter. Some of the larvae after percutaneous infection migrate to soft tissues where they be encapsulated.

Pathogenesis
Percutaneous infection causes formation of nodules in the skin with edema and enlargement of superficial lymph nodes. In liver, L4 produce inflammatory lesions. Sometimes there may be abscess formation, extensive liver cirrhosis and adhesions may occur. Adults are not markedly pathogenic. Adult parasites are usually found in pairs in cysts containing green pus. Cyst may occur in kidney. Ureter may be thickened and may be blocked in chronic cases.

Clinical symptoms
At early stage of infection, subcutaneous nodules may be seen. Sometimes may cause stiffness of legs if present at leg level. The general impact of disease is depressed growth rate, loss of appetite, emaciation

Diagnosis
Eggs may be found in urine if adult parasites are available and cyst communicating to ureter. Eggs are strongyle type (43-70x90-120µ). Eggs in urine - highest concentration of eggs in the last part of the first micturition in the morning.

Treatment
No satisfactory treatment available. However, Thiabendazole-0.1-0.4%in feed is effective in inhibiting larval stages.

Nematodes

Prophylaxis

1. Hygienic measures. Measures taken against *A. suum* are effective against it.
2. Adequate drainage of pig pens.
3. Segregation of clean animals from infected ones.
4. Periodic unstocking of animals form pens. These pens are allowed for drying.

Nematodes

Lecture-4

Superfamily Ancylostomatoidea

- Buccal capsule subglobular.
- Lips and leaf crown absent.
- Oral opening may be unarmed or having cutting plates or teeth.

Family: Ancylostomatidae

- Have well developed buccal capsule.
- Ventral margin armed with teeth or cutting plates.
- Anterior extremity usually turned dorsally.
- Bursa is normally developed and two spicules of equal size.
- Most species are voracious blood suckers and parasite in small intestine.

Genus: ***Ancylostoma***
Species: ***A. caninum***

Location and Host: Small intestine of dog, fox, wolf, and wild carnivores.

- Male-10-12 mm and female-14-16mm long.
- Worms are rigid, and grey or reddish in color.
- Buccal capsule is deep.
- There are three pairs of teeth on ventral margin of buccal capsule.
- Male bursa is well developed.
- Vulva is located at the end of $2/3^{rd}$ of body from anterior end.
- Spicules are 0.8-0.9mm long.
- Eggs: oval with a thin, smooth shell and they enclose one morula containing only 8 to 16 cells when shed

A. tubaeforme (hookworm of cat)
Location and host: Smaller than *A. caninum*
Male 9.5-11mm and female 12-15 mm long, mouth capsule similar to *Ancylostoma* but teeth on ventral margin are slightly larger. Spicules are longer than1 mm.

A. braziliense
Location and host: Small intestine of dog, cat, fox and wild canids.
It is slightly smaller (M 6-7.5 mm and female 7-10 mm long). On the ventral side of buccal capsule, two teeth are present; one larger and one smaller. Spicules are 0.7-1 mm long.

A. ceylanicum
Location and host: Small intestine of dog, cat and man.
Smaller than *A. braziliense* but inner pair of teeth on ventral margin is larger.

A.duodenale

Location and host: Small intestine of man (Hookworm of human beings).
Male 8-11 mm long. Female 10-18 mm long. Two pairs of teeth are present on the ventral margin.

Uncinaria stenocephala
Found in temperate climate. Host: dogs, cat, fox (small intestine)

Lifecycle

Direct.
Adult females may produce 16000 eggs per day. Pre-infective stage behaves similarly to that strongyle species. These stages cannot survive desiccation. Slightly sandy and moist soil is most suitable for infective stages. Optimum temperature for *A. caninum* is 23-30 °C in which one week is needed to reach the infective stage.

Routes of Infection:

1. Per cutaneous
2. Per os: Ingesting L3 along with food and water.
3. Transmammary/ lactogenic/ colostral
4. Prenatal: rare
5. Paratenic hosts: sometimes

1. Per cutaneous: Skin (L3) ---circulation (blood vessels/lymphatics)---heart---lungs---alveoli (L4)---coughed up---swallowed---small intestine (L5). Pre-patent period: 17 days

2. Per Os: Gut wall (L3)---circulation---heart---lungs (L4)---alveoli---coughed up---swallowed--- small intestine (L5). After oral infection the larvae which fail to penetrate in the tissues at different level enter in gastric glands or crypts of Liberkuhn for few days then return to lumen where they moult to become adult. Prepatent period 15-18 days in young pups and worms may persist for 6 months.

3. Transmammary/ lactogenic/ colostral: In susceptible bitches, a proportion of the L3 that reach the lungs migrate to the skeletal muscles where they remain dormant until she is pregnant. They are then reactivated and, still as L3, are passed in the milk of the bitch for a period of about 3 weeks after whelping. Infection of the bitch on a single occasion has been shown to produce transmammary infections in at least three consecutive litters.

4. Prenatal: Prenatal infection earlier reported to be the main source of infection but now it has been understood that it plays role in less than 2% infection.

Pathogenesis

A. caninum is more pathogenic, causing anaemia due to blood loss in intestine associated with feeding habits of the parasite. The severity of disease depends upon: a)

age b) intensity of infection c) nutritional status d) iron reserve e) acquired immunity. Initially anaemia is normocytic and normochromic but once an animal becomes iron deficient a microcytic and hypochromic enemy comes into the picture.

Small pups are more severely affected which acquire substantial worm burden through lactogenic route. Pups of small breed suffer more than larger breeds. Approximately 0.01-0.09 ml is lost per day per worm. Blood loss starts at about 8th day when fourth moult takes place along with the development of buccal capsule. The peak loss is found 10-15 days after infection. Death in small puppies found 10-24 days of infection while in older puppies due to sufficient iron reserve there is erythropoitic response which compensates the blood loss.

There is diarrhea and sometimes fresh blood mixed with mucous may be found. Skin lesions due to per cutaneous infection varied from moist eczema to ulcerations. These lesions are usually found after rain/otherwise when dogs are allowed on wet grass in endemic areas. Haemorrhagic pneumonia is also found which sometimes may cause death of animals in such cases lungs are consolidated and covered with haemorrhagic foci.

Clinical symptoms

Ancylostomosis usually occur in summers. Particularly in those animals which are confined to a small area of moist ground. Prenatal and colostral infection cause severe anaemia, coma and death within three weeks of birth. Other clinical signs include:

1. **Cutaneous signs:** penetration by L3 larvae can result in papular lesions covered with scales on the legs and ventral areas causing severe itching and thereby leading to pyoderma.
2. **Respiratory signs:** coughing, pneumonia, loss of sense of smell (often occurring in hunting dogs), change to the bark (to a higher pitch) and epistaxis.
3. **Intestinal disorders:** adult parasites will cause congestive haemorrhagic enteritis. Faeces are often diarrheic and contain bloody mucous or tarry nature.
4. **General disorders:** anaemia, hydraemia, oedema, chronic weight loss, muscular atrophy and development of a wasting syndrome.

Diagnosis

1. Symptoms
2. Finding strongyles type eggs in the faeces
3. P.M. liver light brown in color and show fatty changes. Intestinal contents are haemorrhagic.

Treatment

- Affected dogs should be treated with an anthelmintic, such as mebendazole, fenbendazole, pyrantel or nitroscanate.
- Supplementation with parenteral iron and possibly vitamin B12 supplements and to ensure that the dog has a protein-rich diet.
- Blood transfusion wherever necessary.

Prophylaxis

1. Periodic deworming of pups.
2. Hygienic maintenance of kennels. Preinfective stages are susceptible to dessication. The places where animals are kept should be dry.
3. Floor of kennels should be treated with common salt or sodium borate solution to kill the larval stages.
4. Proper disposal of faeces.

Cutaneous larva migrans (CLM) - Creeping Eruption

This condition is comparable to VLM, occurs in man and abnormal host caused by larvae of *A.braziliense, Uncinaria stenocephala, A.caninum, A.duodenale, Necator americanus, Bunostomum phlebotomum, Strongyloides* sp. These larvae enter in skin and migrate into it, causing tortuous erythematous inflammatory tracts within the dermis and severe pruritus. The severity of lesions is related to the degree of exposure. Later on it may lead to pustule formation. Sometimes these larvae may migrate to lungs and eyes where may produce lesions.

Genus *Bunostomum*

Species: ***Bunostomum trigonocephalum*** (Hookworm of sheep and goat)
Location: Found in small intestine (ileum and jejunum) and very common in India.

Morphology:

- They are small, stout worms.
- Male 12-17mm and female are 19-26 mm long.
- Anterior end is bent dorsally so buccal capsule opens anterodorsally.
- A large well developed buccal capsule is present. The ventral margin of its opening is armed with a pair of semilunar cutting plates and near its base pair of small subventral lancet.
- A prominent cone projecting in buccal capsule.
- Bursa well developed with asymmetrical dorsal lob.
- The right externodorsal ray is longer than the left one and arises much higher up on the dorsal stem. Spicules are 0.6-0.64 mm.

Lifecycle

Direct. Eggs hatch in open. L3 is the infective stage. Infection occurs via mouth or through skin penetration. Following penetration, larvae reach to the lungs where 4th stage (buccal capsule) moult and reach the small intestine after 17 days. Prepatent period 30-56 days.

Pathogenesis

Infection usually found during the summer. When a large number of hookworms are present, they cause severe anaemia. Diarrhea is common, may be dark in colour due to altered pigments of the blood. When infection is heavy it may lead to prostration and death.

Diagnosis

By finding eggs in faeces. Confirmed by culturing of faeces.

Bunostomum phlebotomum (Hookworm of cattle and buffalo)

- Found in small intestine (duodenum) of cattle and buffaloes.
- Small, stout worm, males are 10-18mm and female 24-28 mm long.
- It differs from *B. trigonocephalum*, only on following points:

➢ Dorsal cone is shorter.
➢ Spicules are filliform and longer.
➢ Eggs are slightly larger
➢ 2 pairs of subventral lancets.

Life cycle: Similar to *B.trigonocephalum*.

Pathogenesis

Skin penetration at feet, cause severe itching, which makes animals to stamp their feet. As they are blood suckers and cause anaemia, diarrhea, weakness especially in calves. Submandibular oedema may also be found.

Diagnosis

Strongyle type eggs. Confirmed by culturing.

Genus: *Gaigeria*

Species: ***G. pachyscelis*** (in India, very common)

Hookworm of sheep and goat. Found in small intestine (duodenum)
Size: male 20mm and female upto 36 mm (larger than *B. trigonocephalum*)

It resembles *B. trigonocephalum* but differs in following points:

1. Buccal capsule has large dorsal cone, but no dorsal tooth. At the base they have a pair of lancets.
2. In bursa of male, lateral lobes are small which are joined ventrally and dorsal lobe is large. Antero lateral ray is short, blunt and separated from other lateral rays.

Life cycle

Similar to that of other hookworms. Infection occurs mainly through skin penetration and here worms reach maturity in 10 weeks.

Pathogenesis

They are voracious blood suckers. Sometimes 20-30 worms may cause death of animal.
Other pathogenicity is same as other hookworms.

Treatment: as other gastrointestinal nematodes.

Prophylaxis: same as *B. phlebotomum*

Agriostomum vryburgi
Location: Small intestine of Zebu and Ox in India and Sumatra.

Morphology:

- Males are 9.2-11 mm long and females 13.5-15.5mm.
- The buccal capsule open antero-dorsally 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.
- Eggs: 125-195 by 60-92 micro meter.

Life cycle: Direct

Pathogenicity: anaemia and diarrhoea in heavy infection

Super family: Trichstrongyloidea

Family: Trichostrogylidae.

- Mostly small parasites.
- Buccal capsule absent or very small and do not have leaf crown and usually bear no teeth.
- Male bursa is well developed with large lateral lobe and small dorsal lobe.
- Parasites in the alimentary canal of sheep, cattle, equines and other vertebrates.

Genus: *Trichostrongylus* (hair worms)

- They are small, slender worms, usually reddish brown in colour when fresh.
- The head end is straight.
- Buccal capsule and cervical papillae absent.
- Excretory pore situated in the anterior end of the body and placed in the notch.

- Bursa has large lateral lobes and rudimentary dorsal lobe. Ventroventral rays are markedly thinner than lateroventral. Posterolateral rays lies near the externodorsal rays.
- Dorsal ray is slender and bifurcates at the tip and ends in two digits.
- Spicules are pigmented, brown, stout and rigid.
- Vulva in females situated a short distance behind the middle of the body and usually has prominent lips.
- Eggs are oval, thin walled, segmented when laid.

Species	Host	Location	Size
T. colubriformes (Black scour or bankrupt worm)	Sheep, goat, cattle, buffalo and camel	Anterior end of small intestine	Male-4-4.5mm and female 5-7mm. Spicules are equal.
T. axei (Stomach hair worm)	Sheep, goat, cattle,pig,equines and man	Abomasum/stomach	Male-2.5-6mm and female 3.5-8.0mm
T vitrinus	Sheep, goat, deer, occasionally in pig	Small intestine	
T capricola	Sheep, goat	Small intestine	
T. retortaeformis	Rabbit, hare	Small intestine and occasionally in stomach.	

Lifecycle

Eggs are strongyle type. Infective 3rd stage larvae produced in 4-6 days. Larvae migrate to grass blades and maximum number of larvae are found on grass blades in early morning and early evening. When temperature, moisture, and light intensity is optimum. Infection occurs by ingestion of L3 stages along with grass. Parasite L3 found in abomasum or small intestine 2-5 days after infection. L5 is found after 15 days after infection. The prepatent period is 20 days.

Pathogenesis

Trichostrongylus axei parasitizes the abomasum/stomach of sheep, goat, cattle, pig, equines and man. The histotrophic migration of larvae occurs and all stages of development are found in between abomasal epithelium and basement membrane in calf and gastric glands in horses. Infection causes hyperemia of mucosa which leads to catarrhal inflammation with necrosis and ulceration of epithelium. In calves, many

parasites may be found embedded in mucosa looking like raised, whitish-grey, circular lesions with demarcate borders in the pyloric and fundic region termed as plaques or ringworm lesions. In horses, hypertrophic (pedunculated or polyploid) gastritis develops.

T.colubriformes, T vitrinus , T. retortaeformis cause similar lesions in the small intestine. During development they are found in the tunnels beneath the epithelial cells, causing its displacement and disruption. Infection of *Trichostrongylus* lead to protein losing gastroenteropathy. Moderate to severe villous atrophy may develop in infected areas. The infected lamina propria becomes thickened and oedematous along with cellular infiltration causing increased permeability of plasma protein in the intestine resulting in hypoalbuminaemia.

Clinical symptoms

Horses and cattle may produce signs of gastrointestinal disturbance. In sheep and goats, heavy infection in young animals causes sudden death, just before dying the animals are unable to stand due to severe weakness in legs. In chronic cases, there is emaciation, skin becomes dry. Alternating diarrhoea and constipation may occur. Diarrhoeic faeces are dark in colour, so they are popularly known as *"Black scour worms."*

Immunity

Infection induces immunity to reinfection. Reinfection with infective larvae induces self cure. Immunity is not species specific. Animals sensitized to *T. colubriformes* may show protection, 98-100% against *T. colubriformes* and *T. vitrinus* and *N. spathiger.*

Genus: *Ostertagia* (brown stomach worm)

- Small, slender worms, brown in color when fresh, hence the common name.
- Cuticle of anterior extremity is slightly inflated to form head and transversely striated. Rest of the body has longitudinal ridges.
- Spicules are short, equal and end in 2-3 processes.
- Vulvas may or may not be covered with vulvar flap. Members are parasitic in the abomasum and rarely in the small intestine of ruminants.
- About 35 valid species belong to this genus of which the following two occur more frequently and considered pathogenic.

O. ostertagi

Found in the abomasum of cattle, buffalo, goat and rarely sheep. Male is 6. 5-7. 5mm and female 8.3-9.2 mm. There are two spicules and each end in three bluntly hooked processes. Vulva covered with flap.

O. circumcinata

Abomasum of sheep and goat. Male 7. 5-8. 5 mm and female 9.8 - 12.2 mm long. Spicules are long and each ends in a large knobbed and small acute process.

Lifecycle

Similar to the members of Trichstrongylidae. L3 is the infective stage which is picked up by host during grazing. Infective larvae exsheath in rumen and then penetrate gastric glands in abomasal mucosa. Adult parasites emerges 18-21 days after infection. Prepatent period- approximately 3 weeks.

Pathogenesis

The main pathogenic effect of *Ostertagia* is to reduce functional gastric glands. Enlarged parasitized gland stretches the surrounding glands. Larvae cause damage to parietal cells which produce hydrochloric acid, are replaced by rapidly dividing, undifferentiated, non-acid-secreting cells. A rapid division of epithelial cells leads to hyperplasia and thickening of mucosal layers. Normally, the abomasal pH is between 2 to 2.5 which may increase to 7 in the diseased condition. Changes in the abomasal pH, leads to the following consequences:

1. Pepsinogen is not activated to its active form, pepsin
2. Proteins are not denatured and digested
3. Increased bacterial load in abomasum
4. Pepsinogen leaking into circulation leading to its elevated concentration

Macroscopically, 2-3 mm diameter circular nodules are produced. In heavy infection whole mucosa may be hyperplastic with raised nodules (morocco leather appearance).

Clinical symptoms

The disease can be categorized into two forms in cattle:

1. Type I Ostertagiosis:
- Occurs in calves during their first grazing season (exposed to pasture for the first time) as a result of maturation of larvae ingested 3-4 weeks back.
- Reduced appetite and profuse watery green (bright) diarrhea due to failure of abomasum to denature chlorophyll

2. Type II Ostertagiosis:
- Occurs in yearlings
- Resumed development of larvae which have undergone hypobiosis (EL4)
- Occurs in late winter or spring
- Results from the maturation of larvae ingested during the previous autumn
- Severe chronic diarrhoea, emaciation, hypoalbuminemia, sub-mandibular oedema

Diagnosis

- Clinical signs of inappetance, weight loss and diarrhoea
- Increased plasma pepsinogen levels
- Post-mortem examination
- History of previous grazing.
- History regarding exposure for first time.
- Finding strongyle-type eggs in the faeces and doing their culture

Genus: Cooperia

- Small worms, reddish when fresh.
- Cuticle at anterior end extremely inflated to form cephalic swelling and the rest of the body bears longitudinal ridges which are transversely striated.
- Bursa in male has large lateral lobes and a small dorsal lobe; ventroventral rays are thinner and smaller than lateroventral rays. Externodorsal rays arise from the base of dorsal ray.
- Spicules are short, stout, and brownish and have ridged, wing like expansion in the middle. Vulva may or may not be covered with vulvar flap.
- Members are parasite in small intestine, rarely in abomasums of ruminants.

Cooperia punctata
Found in cattle, but rarely in sheep. Male-4.7-5.9mm and female 5.7-7.5mmm long. Spicules are equal

*C. **pectinata*** found in cattle but rarely in sheep. Male 7mm long, female 7.5 -9.0mm long.

*C **curticei*** sheep, goat, rarely cattle male 4.5-5.4 mm, female 5.8-6.2mm long.

Life cycle is similar to *Trichostrongylus* (direct). Infection of host occurs by ingestion of infective larvae from moist pasture. Infective larvae can survive in pastures for 9-26 weeks. Parasite may go for arrested development as in *Ostertagia* to survive in unsuitable environment. Worms penetrate mucosa of small intestine. Young animals may be severely affected where disease syndrome is similar to trichstrongylosis.

Diagnosis: by culturing larvae.

Treatment: with GIT nematodes.

Genus: ***Nematodirus***

- They are relatively long worms with filiform anterior portion.
- They have inflated cuticle around the anterior end and about 14-18 longitudinal ridges on the body cuticle.
- Anterior part of the body is thinner than posterior part.
- Male bursa has elongated lateral lobes.

- The dorsal lobe along with its rays split into two parts and each part is joining the lateral lobe. Ventral rays are parallel and close together. The mediolateral ray and posterolateral ray are close together except at their tips.
- Vulva in female open at the posterior third of the body.
- Triangular tail of female is short and truncate with slender terminal appendage.
- Eggs are very large in size and contain about 8 cells. They are so large that they are distinguished from other strongylids.

N. spathiger
Small intestine of sheep, cattle and other ruminants. Male 10-15 mm and female 15-23 mm long. Spicules terminating in spoon shaped structure.

N. filicollis
Small intestine of sheep, cattle. Similar to ***N.spathiger*** but spicules terminating in pointed end.

Lifecycle

Direct. L1 and L2 larvae moult inside the egg. L3 is infective stage. The larvae penetrate the mucosa of intestine and moult to 4th stage (4 dpi) and then moult to 5th stage (10 days DPI), return to lumen. prepatent period is 15 days. Infection is short lived in lambs. These parasites after few weeks are expelled out and such lambs become resistant to reinfection. Usually one cycle of parasites is completed in one year. Infection is again transmitted to lambs of new generation. Older animals may carry light infection.

Pathogenesis and clinical symptoms of *Nematodirus* inection.

These parasites penetrate the mucosa of intestine and cause tunneling. The posterior part remains outside hanging in lumen. The surface area of villi is reduced. After 20 days disease leads to villous atrophy. At this time parasite become surrounded (smeared by a mucous like material which may be a sign of rejection. Clinical symptoms in lambs are seen during prepatent period and are associated with in appetence (10-11 DPI) sudden acute onset of acute enteritis, severe blackish green diarhhoea which become pale in color followed by dehydration and prostration. There may be up to 30% mortality in lambs which are 6-10 weeks old. After 3 weeks there is improvement in clinical symptoms. Older animals may carry light infection and may increase pasture worm load. It is cycle which run from lambs to lambs of next generation.

Lecture-5

Genus: *Haemonchus*
Species: *Haemonchus contortus*
(Stomach worm/ wireworm/barber pole worm)

- Definitive hosts are sheep, goat, cattle and other ruminants.
- Location: abomasum (hence name Stomach worm)
- Found world-wide, very common in India where 3 sub species of this nematode has been reported from sheep and goats.
- ***H. longistipes***- camels in North Africa and India

Morphology:

- Relatively large but thin shaped worm, Male-10-20 mm and female 18-30 mm long.
- When fresh, males are reddish, but in females white ovaries spirally wound round the red intestine giving the worm barber pole appearance.
- Buccal capsule is very small (not visible) a dorsal lancet is present at its base.
- Cervical papillae, one on either side are present in the anterior part of the body, they are spine like.
- Bursa of male has elongate lateral lobes with long slender rays and a small dorsal lobe and supported by Y-shaped dorsal ray. Dorsal lobe is asymmetrical and situated against the left lateral lobe.
- Spicules are equal with barbed tips and .46-.506 mm long.
- Vulva, in female, is situated in the posterior part of the body and covered by a large and prominent vulvar flap. It may be reduced to knob like structure in some specimen.
- Eggs are elongate, thin walled and segmented when laid.

Life cycle

Similar to strongyle. Under satisfactory environment conditions infective larvae reached to infective stage in 4-6 days. Below 9^0C there is no development. Eggs at pre hatch stage are more resistant to adverse conditions (freezing and dessication) than other stages. Larvae and eggs are intolerant to these conditions. Maximum concentration of larvae during late spring, reach maximum at mid summer and decline through the winter. *H .contortus* survives the adverse condition through arrested development within the host. Resumptions of arrested larvae occur at a time when environmental conditions are suitable for survival of free living stages. Following ingestion of larvae exsheathment occur in rumen and parasitic larval stages migrate to abomasums and penetrate between gastric epithelial cells. Prepatent period for *H. contortus* is 15 days; *H. placei* is 26-28 days.

Pathogenesis

The principal feature of disease is anaemia; both adult and 4th stage larvae suck blood and cause wounds in abomasum. Average blood loss/worm/day is 0.05ml. Blood first appears in faeces 6-12 days after infection. Infected animals lose large quantities of serum protein into gut.

Clinical symptoms

Divided into-
-Hyperacute
-Acute
-Chronic

Hyperacute Haemonchosis
It is uncommon but if animals are suddenly exposed to heavy infection, then they cause rapid anemia. Dark colored faeces and sudden death from acute blood loss. There is severe hemorrhagic gastritis. Death usually occurs during prepatent phase. The necropsy examination reveals a tremendous number of larvae belonging to third, fourth and fifth stages along with haemorrhages on abomasal mucosa.

Acute Haemonchosis
When young susceptible animals become heavily infected. Anaemia development occurs in three stages:

1. First Stage: By immature worms in prepatent stage, there is rapid fall in PCV but serum iron remains normal
2. Second Stage: Further decrease in PCV, slight increase in serum iron levels due to absorption of the haemoglobin iron.
3. Third Stage: Marked decrease in PCV causing dyshaemopoiesis due to Fe deficiency. There is ascites and submandibular oedema (bottle jaw) indicating development of hypoproteinaemia.

Chronic haemonchosis
Very common form. It is due to low number of parasites. Mortality is low. Affected animals are weak, emaciated. Anemia may or may not be severe. EPG (eggs per gram of faeces) may be less than 2000. On postmortem there is hyperplastic gastritis. The clinical appearance is of malnutrition with progressive loss of weight, wool in adults and stunted growth in lambs.

Immunology of *Haemonchus contortus*

Probably the best known immunological response to helminths is the Self Cure Phenomenon. There is rapid expulsion of existing adult parasites following ingestion of a challenge dose of infective larvae. Reaction is initiated when larvae (l3) of challenge dose moult to fourth larval stage (probably molting fluid). The self cure reaction is accompanied by transient rise in blood histamine and increase in complement fixing antibodies titre. Histamine stimulates the gastric glands causing increased production of gastric volume and acidity. This acidity changes the mucosal environment and causes

mucosal oedema. This causes the worms to dislodge and expel from the abomasum. A latent period of 6-7 weeks is required between initial infection and infection which induces self cure reaction. Self cure reaction is more pronounced in those sheep which have experienced several suitable doses at a specific interval than those which carry initial infection. Antigen is associated with living stage of parasite. Self cure is not entirely specific it gives protection against *Trichostrongylus* sp.

Genus: *Mecistocirrus*

Mecistocirrus digitatus
Found in the abomasum of sheep, goat, cattle and buffalo (central America, Asia including India)

Morphological features
This worm superficially resembles *H. contortus* and often mistaken for it because of their common location.. However, they can be differentiated on the following features:

1. It is slightly larger (male upto 31 mm and female upto 43mm long)
2. Dorsal lobe of bursa is small but lateroventral ray and anterolateral rays considerably longer.
3. Spicules are very long, slender and united together almost for their entire length.
4. Vulvar flap in females is longer.

Life cycle: Direct like *H. contortus*. Prepatent period about 60 days.

Pathogenesis

It is an important pathogen of buffalo, cattle and goats. Effects are similar to *H. contortus.*

Gastrointestinal nematode infection in Ruminants.

Pathogenesis
The pathogenic effect caused by GIT nematodes are variable. The main pathogenic effect caused by *H. contortus* and *Mecistocirrus digitatus* are marked haemorrhages through wounds in abomasal mucosa. The infection with *Ostertagia* causes destruction of gastric glands of abomasum. Infection with *Nematodirus* and *Trichostrongylus* cause villous atrophy. *Oesophagostomum* species and *Chabertia ovina* cause ulceration and haemorrhages in the large intestine. In general in all GIT nematodes, there is anorexia leading to poor weight gain and lower production. Apart form whole blood loss by parasites there is protein loss by nematodes. Anaemia probably results from deficiency in the amino acid required for haemoglobin synthesis; in case of non blood sucking nematodes there is altered protein metabolism and reduced feed intake accompanied by changes in cholecystokinin level. There is local dysfunction of gastrointestinal tract. In villous atrophy there is deficiency in brush border enzymes (alkaline phosphatase, maltase and dipeptidase). GIT parasitism also causes disorder in mineral metabolism.

Reduced calcium, phosphorus and magnesium absorption results in reduced bone growth in young animals.

Treatment

-Thiabendazole	44 mg/kg body weight (sheep) 66 mg/kg body weight(cattle) (larval and adult stages)
-Mebendazole	15 mg/kg body weight (adult nematode) Also effective against *Moniezia*, *Dictyocaulus*.
-Fenbendazole	7.5 mg/kg body weight (cattle) 5 mg/kg body weight (sheep,goat) Highly effective against adult and immature. Also ovicidal (effective against *Moniezia*, lungworms)
-Oxfendazole	4.5-5 mg/kg body weight (mature, immature, ovicidal) also effective against *Moniezia*, lungworms
-albendazole	5-7.5 mg/kg body weight (also effective against *Moniezia*, lungworms, ovicidal)
-Febantel	5-7.5 mg/kg body weight (mature, immature, GIT nematodes, lungworms)
-Thiophanate	50-100 mg/kg body (GIT nematodes)
-Levamisole	7.5 mg/kg body (mature, immature, GIT nematodes, lungworms)
-Morantel tartarate	10 mg/kg body weight (mature, immature, GIT nematodes)
-Phenothiazine	220-440 mg/kg body weight (cattle). Maximum dose 40-80g) adult GIT nematodes.

Control

Control of parasitic diseases based on management, larval ecology and epidemiology of GIT parasites. It is affected by grazing management, provision of clean pastures, alternate grazing of different species of host and use of anthelmintics, safe pastures may be achieved by-

1. Pasture resting
   Stock may be withdrawn from pasture for few months in cool moist condition and two months under hot, arid conditions.
2. Variation in timing of reproductive event. May be timed in such a time that during parturition environmental conditions are unfavorable.
3. Stocking rate pasture should not be overburdened with population density.
4. Alternate grazing with hosts of different species.
5. Alternate grazing within same species.
   Usually adult animals are resistant to parasites in comparison to young one, so excrete low number of parasitic eggs. If young animals are grazing before adult animals there will be reduced pasture contamination.
6. Ploughing, seeding and burning of pastures.
7. Use of anthelmintics during spring and early summer will reduce the peak larvae concentration during the summer.

Lecture-6

Family: Dictyocaulidae

- Members of this family occur in respiratory passage of lungs.
- Bursa is well developed along with bursal rays.
- Spicules are short and reticulated.
- Life cycle is direct.

Genus: *Dictyocaulus*

General Morphology

1. Long, thin milky white worm. Intestine shows a dark line.
2. Mouth is surrounded with 4 lips.
3. Buccal capsule is present (shallow and small)
4. Medio and posterolateral rays are either completely or partially fused.
5. The externodorsal rays arise separately from dorsal stem which is split right from its base.
6. Spicules are equal, short, stout and dark brown and they have boot shaped appearance.
7. Eggs are thin walled, contain fully formed larvae when laid and measures 69-90 by 112-138µ. But in faeces L_1 are passed.

***D. filaria* (Thread lungworm of sheep)**

Occurs in the bronchi of sheep, goat and wild ruminants. Male 3-8 cm and female 5-10 cm long. Medio and posterolateral rays are fused together except at their extremities. Spicules are 0.4 to 0.64 mm long.

***D. viviparous* (Bovine lungworm)**

Occur in bronchi of cattle, buffalo and camel. Male is 4-5.5 cm and female 6-8 cm long. Medio and posteriolateral rays are completely fused together. Spicules are 0.195-0.215µm long.

D. arnfieldi

Found in bronchi of horse, donkey, mule and zebra. Male up to 36 mm long and female up to 60 mm long. Medio and posterolateral rays of male bursa are fused together for half their length. Spicules are 0.2-0.24 mm long.

Life cycle

The female worms are ovoviviparous, producing eggs containing fully developed larvae, which hatch almost immediately. The L1 migrate up the trachea, are swallowed and pass out in the faeces. L1 which is passed in the faeces is recognized by the presence of cuticular knob at the anterior extremity (present in *D. filaria* only) and brownish food granules in the intestinal cells. The free living larval stages of *Dictyocaulus* species do not feed and exist on these reserved food granules. Under

suitable conditions 6-7 days required to reach the infective stage (L3). The L3 leave the faecal pat to reach the herbage either by its own motility or through the agency of the ubiquitous fungus *Pilobolus*. Infection occurs per os. Larvae penetrate intestinal wall and via lymph vessels reach to mesenteric lymph glands where they moult to L4. Now via lymph vessels/blood vessels they pass to the lungs where they are arrested in the capillaries which they break open to reach in air passage and reach to maturity in 4 weeks.

Pathogenesis & Clinical Signs

D. viviparous

Young animals are more affected than adults. The disease occurs in four stages:

1. First Stage: Infective larva penetrates the intestinal mucosa in the first week of infection. There is no clinical sign except for mild diarrhea.

2. Second Stage (Pre-patent phase): around 1st to 3rd week of infection. There is a rupture of lung alveoli and blockage of respiratory bronchioles with eosinophilic exudates. This leads to pulmonary atelectasis and emphysema. The animals suffer from coughing producing a characteristic spasmodic coughing sound called husk or hoose (verminous bronchitis). There is marked epithelialization of alveolar epithelium and hyaline membrane formation.

3. Third Stage (Patent phase): around 3rd to 8th week of infection. Adult worms settle in the bronchi and bronchioles. Females lay eggs, L1 is passed in faeces. There is copious exudation, pulmonary hypoventilation and generalized hypoxemia. Animals are anaemic, emaciated and may die of heart failure.

4. Fourth stage (Post-patent phase): around 8th to 10th week of infection. Calves show recovery from infection. Respiratory rate decreases and coughing is less frequent. Epithelialization may persist in certain cases.

D. filaria

The worms produce catarrhal parasitic bronchitis. There is inflammation in the peribronchial tissues and the exudate passes in the bronchioles and alveoli causing atelectasis and catarrh, leading to pneumonia. On post mortem examination, lungs show atelectatic areas of different sizes, frothy mucous containing bunches of milky white worms with blood, lymphocytes and desquamated epithelial cells. Lobar type of pneumonia develops.

Young animals are mainly affected, but disease may be found in animals of any age group. Animals may cough; mucous may exude from nostrils (cough may not always present). There is dyspnoea, rapid respiration, abnormal lung sounds. Temperature may increase if pneumonia develops.

D. arnfieldi is not very pathogenic. Donkeys are assumed to be the natural host for this parasite suffering very little.

Diagnosis

1. Clinical Signs
2. Detection of L1 in the faecal samples

Treatment

Tetramisole	15 mg/kg body weight orally.
Levamisole	7.5 mg/kg body weight parenterally.
Benzimidazole	a number of compounds which have been used successfully in parasitic bronchitis
-Fenbendazole	5 mg/kg body weight

Prophylaxis

1. Animals should be removed from infected pastures and should be provided clean water for drinking.
2. Vaccination: Artificial immunization has been successful using X-irradiated infective larvae and a commercial vaccine available in Europe against *D. viviparous*. Vaccination consists of two doses of 1000 irradiated larvae given at interval of a month to young calves of 3-8 weeks of age. Dictol is a vaccine against cattle lungworms containing irradiated larvae. Similarly, in India the lung worm vaccine against sheep lungworm was named Difil.

Family: Protostrongylidae

- Hair like worms
- Bursa more or less reduced or absent.
- Parasitic in bronchioles and parenchyma of lungs.
- In lifecycle intermediate hosts are required.

Genus *Protostrongylus*

***P. rufescens* (Red lung worm)**

- Found in small bronchioles of sheep and goat.
- Male 16-28mm and females 25-35mm long.
- Worms are slender and reddish in colour.
- Bursa in male small but normally developed.
- Spicules are straight, tubular and with broad membranous expansion.
- Vulva, in females, situated near anus.
- Eggs hatch in lungs, L1 comes out in faeces.

Life cycle

The unembryonated eggs laid by the females embryonate and hatch within the lung parenchyma. First stage larva passes in faeces (tip of tail has a wavy outline). L1

larva penetrates the snail of genus *Helicella* for further development. 12-14 days require for larva to become infective. Final host become infected by ingesting infected snails while grazing. The larvae pass to lungs via mesenteric lymph glands. Pre-patent period - 30-37 days.

Pathogenesis

Found in small bronchioles where they produce local area of inflammation. Exudates fill the alveoli. Inflammatory process spread to peribronchiolar area. Ultimately small focus of lobular pneumonia are formed which are conical in shape. The pleura at the base may be involved causing pleuritis. Number of foci represents the number of parasites.

Diagnosis
By finding larva in faeces.

Treatment

Levamisole	20 mg/kg body weight; s/c
Ditrazine	100 mg/kg body weight; s/c
Fenbendazole	20-40 mg/kg body weight; orally.
Albendazole	5 mg/kg body weight

Prophylaxis

- Extermination of snail, if possible.
- Lambs should not be allowed on those pastures which have been earlier assessed by adult animals.
- Animals may be avoided to graze on pastures during early morning and evening when snails creep on plants.

Genus: *Muellerius*

Species: ***M. capillaris*** (Hair lung worm of sheep/goat)
They are found in lungs (alveoli and pulmonary parenchyma) of sheep and goats. Very common in Europe, has been reported from India.

Morphology

- Worms are small and thin. Males are 12-14 mm and females 19-23 mm long.
- Bursa in males is absent. The posterior end of male is spirally coiled. A number of papillae surround the cloacal opening.
- Spicules are curved.
- Vulva opens near anus.
- L1 comes out in faeces, larva has undulating tip and a dorsal spine.

Life cycle

L1 is ingested by the intermediate host which is a snail or slug. L_3 stages develop in these intermediate hosts. Infection occurs via mouth by accidental ingestion of these Intermediate hosts while grazing. Rest lifecycle is similar to *Protostrongylus*.

Pathogenesis

Lungworms of adult animals, and are not found in animals below 6 months of age. By three years of age, maximum goats may be affected in endemic areas. Adult worms live in alveoli and lung parenchyma. Worms produce greyish nodules which consist of necrotic mass. These nodules may calcify. Eggs after coming out also form small foci and adjoining tissue become hyperemic. After hatching of eggs lesions subside. Animals usually show no clinical symptoms, but heavy infections weakens the lungs and reduce the resistance of host. Sometimes secondary bacterial infection produces septic lesions. In some animals they may cause dyspnoea and persistant cough and even pneumonia.

Diagnosis
Finding larvae in faeces.

Treatment & Prophylaxis
Similar to *Protostrongylus rufescens*

Family: Metastrongylidae

Genus: *Metastrongylus*

M. elongatus

- Bronchi and bronchioles of pigs (rarely in sheep, ox, deer)
- Male up to 25 mm female up to 58 mm.
- Worms are white and have six small lips, or papillae around oral aperture.
- Bursa in males is small.
- Anterolateral ray is large with swollen tip. Mediolateral and posterolateral rays are fused and dorsal rays are much reduced.

Lifecycle

Indirect. Eggs pass in faeces, which may hatch soon after they have been swallowed by the suitable species of earthworms. Development in pigs similar to *D. filaria* Prepatent period is 24 days.

Pathogenesis and clinical symptoms

In general pathogenesis similar to *D. filaria*, but these lung worms are comparatively less pathogenic. In young pigs marked verminous bronchitis and pneumonia may be seen. Generally, they are responsible for loss of condition and retarded growth. *M. elongatus* spreads swine influenza virus.

Treatment

Same as *D. viviparous* (levamisole, tetramisole and fenbendazole may be given)

Prophylaxis

- Clean, concrete floor may be provided for pigs.
- Faecal samples should be disposed in such a way that it should not spread the infection.

Lecture-7

Order Spirurida

- Mouth is surrounded by 2 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.

Superfamily Spiruroidea

Family Spiruridae: Two genus of importance: *Habronema* and *Draschia*

Genus: *Habronema* (Stomach worm of equines)

Habronema muscae

Location: Stomach of equines
Morphology:

- Male: 8 – 13 mm in length; Female: 13 - 22 mm
- Mouth is surrounded by two lateral lips and each trilobed. Pharynx is cylindrical.
- It has small caudal alae.
- Spicules are unequal, left is five times longer (slender) than right (short and stout).
- There is presence of 4 pairs of precloacal papillae and 2 post cloacal papillae.
- Females are ovoviviparous. Eggs, thick shelled have larvae.

***Habronema majus* (Syn *H. microstoma*)**

Location: Stomach of equines
Morphology:

- Male: 16-22 mm in length; Female: 15-25 mm
- Pharynx is cylindrical and has a dorsal and a ventral tooth in its anterior part.
- Spicules are unequal, left is two times longer than the right one.
- Eggs are 45-49 µm x 16 µm and hatch in the uterus.

Genus *Draschia*
***Draschia megastoma* (Syn *Habronema megastoma*)**

Location: Inside nodules in stomach of equines
Morphology:

- Male: 7-10 mm in length; Female: 10-13 mm
- Head is constricted off from the body.
- Pharynx is funnel shaped.

- Spicules are unequal, left is two times longer than right one.
- The female lays embryonated eggs.

Life Cycle

The eggs/larvae when passed in the faeces are eaten by the maggots of flies breeding in the manure. For *Habronema muscae* and *Draschia megastoma*, the I.H. is *Musca domestica* (house fly) and for *H. majus,* the I.H. is *Stomoxys calcitrans* (stable fly). In the larva L1 moults to L2. The L2 moults to L3 in the pupa of the fly. In the adult fly, L3 migrates to the haemocoel (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. Sometimes, the flies fall into the water or feed, in which horse acquires infection by ingestion of larvae. Following deposition/ingestion, the L3 reaches the stomach of equine and become adults in about 2 months.

Pathogenesis

H. muscae* and *H. majus

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

D. megastoma (a.k.a. H. megastoma)

Adult worms penetrate the stomach mucosa causing "nodule formation" due to constant irritation. In severe case more number of nodules fuses to form large 'tumor' like abscess. 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.

Cutaneous Habronemiasis a.k.a Summer Sore/ Bursati/ Granular dermatitis

- 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, medial canthus of eye and prepuce of male.
- 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.
- There is intense itching of the affected skin that affected animals may rub and bite themselves, causing secondary bacterial and mycotic infections.

Diagnosis

- Detecting embryonated eggs or larvae in faeces.
- Finding adults during necropsy.
- Cutaneous habronemiasis: identifying larvae in skin scrapings or biopsy of the lesion.

Treatment and Control

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

Family Thelazidae

Genus *Thelazia*

Members of this genus are parasites of the conjunctival sac and lacrimal duct and because of their location are also known as 'eyeworms'. Worms have a large vestibule and lips are absent in this genus.

Thelazia rhodesii

Location and host: Eye, conjunctival sac, lacrimal duct of cattle, buffalo, sheep, goats and wild ruminants.

- Milky white worms.
- Prominent cuticular transverse striations on the body.
- The male worms have about 14 pairs of pre-cloacal and three pairs of post-cloacal papillae.
- Females are viviparous.

T. gulosa

Location and host: Conjunctival sac of cattle and buffaloes.

- Thin transverse cuticular striations.
- Cup shaped buccal capsule.
- Females are viviparous.

T. callipaeda

Location and host: Nictitating membrane of the eye in dog and conjunctival sac of man

- The cuticle bears fine transverse striations.

- The male has one single and five pairs of pre-cloacal papillae and two pairs of post-cloacal papillae.
- Eggs are embryonated when laid.

T. lacrymalis (Equine eyeworm)
Location and host: Conjunctival sac of horse and cattle

Life Cycle

- Indirect and I.H. are the house-flies of genus ***Musca***.
- 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 into the ovarian follicle,where it moult to become L2 and L2 to L3.
- L3 reaches the mouth parts of the fly and are transferred to susceptible animals when the infected fly feed.

Pathogenesis

The lesions are caused by the serrated cuticle of the worm and most damage results from movement by the active young adults, causing lacrimation, conjunctivitis, ulcerated cornea, photophobia, sometimes causing corneal opacity and keratitis leading to complete blindness. Infection may predispose to infectious keratoconjunctivitis ('pink eye') caused by *Moraxella*.

Diagnosis

- Detection of mature and immature worms in the conjunctival sac.
- Examination of lacrimal secretions.

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.

Genus *Oxyspirura*
Sp. *O. mansoni* (Poultry eye worm)
Location and host: Nictitating membrane of the eye of chicken, turkey and other gallinaceous birds.

- Cuticle is smooth
- Pharynx is hour glass shape.
- Eggs are embryonated when laid.

Life Cycle
Indirect. Intermediate host is the cockroach.

Genus *Spirocerca*
Sp. *Spirocerca lupi*
Location and Host: Walls of oesophagus, stomach, aorta and rarely found freely in the lumen of stomach of dogs.

- Usually spirally coiled and pink in color when fresh.
- Male: 30 – 54 mm; Female: 54 – 80 mm.
- Male tail end has caudal alae with one single and 4 pairs of papillae, one paired median papillae and two pairs of post cloacal papillae.
- Group of small papillae occurs at the tip of tail. Spicules are unequal.
- Eggs are thick shelled and contains fully developed larvae.

Life Cycle

Indirect. I.H. are the coprophagus beetles. 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 a paratenic host (garden lizard, mice and domestic chicken) the larvae again encyst on oesophagus, mesentery and other visceral organs. Dog acquires infection by ingestion of either infected I/H or infected paratenic host. Following ingestion, larvae are liberated in the stomach, penetrate stomach wall to reach the gastric 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. Eggs, however, may not be found in the faeces of a proportion of animals with adult infections where the granulomas have no openings into the oesophageal lumen.

Pathogenesis

- The migratory larvae causes haemorrhage, scarring and formation of fibrotic nodules in the internal wall of aorta, which at times can rupture during heavy infection.
- Aortic lesions include haemorrhage and necrosis with eosinophilic inflammation. Widely dispersed thickening of the *tunica intima* and *tunica media*, which may weaken the arterial wall and result in aneurysm and potential rupture.
- The adult worms are associated with the formation of granulomas in the oesophageal wall. They are responsible for a variety of clinical signs, including dysphagia and vomiting arising from oesophageal obstruction and inflammation.
- Secondary complication due to *S. lupi* are development of malignant tumors in oesophagus (fibrosarcoma or osteosarcoma). Fibrosarcoma may metastasize to lungs and other organs.
- Sometimes there is occurrence of spondylitis of thoracic vertebrae or hypertrophic pulmonary osteoarthropathy of the long bones. Development of hypertrophic pulmonary osteoarthropathy with characteristic signs of marked hypertrophy of the long bones, oedema in the legs and considerable locomotion difficulty. This

syndrome, called the Cadiot syndrome, is characteristic of canine spirocercosis in regions where the parasitosis is enzootic.

Clinical Signs

A large oesophageal tumor, interfere with deglutition, respiration and circulation. Persistent vomition and rapid loss of condition will be seen.Vomiting and nausea often occurs at the sight of food and dogs are often polydipsic due to fluid loss from vomiting. Polyphagia is sometimes seen because the animal regurgitates any food that is ingested and can therefore be very hungry. Haemorrhage from oesophageal region causes anaemia and haemoptysis. Rupture of the surrounding weakened oesophageal wall and escape of ingesta in to the pleural cavity causes "Pleuritis". General weakness, emaciation and anaemia commonly occur.

Diagnosis

- Examination of faeces and vomition for eggs.
- Endoscopic examination for the presence of characteristic granulomatous nodules.

Treatment and Control

- Their location makes it difficult for anthelmintic products to reach the nodules containing *Spirocera* because these products do not circulate well in this kind of tissue.
- Nitoxynil @ 10 mg/kg b.wt s/c is effective.
- Ivermectin @ 0.2 mg /Kg b. wt. s/c.
- The affected dog should be isolated, their vomiting and faeces should be disposed of properly.
- Dogs should be prevented from eating of paratenic host.

Genus *Gongylonema* (Commonly known as gullet worms)

Gongylonema pulchrum

Location and host: Mucosa and Submucosa of oesophagus in sheep, goat, cattle, buffalo, pig and horse.

- The males are up to 62 mm long and females 145 mm long.
- The worm lies in a zig-zag fashion resembling 'Tailors stiches' embedded in the oesophageal mucosa.

G. verrucosum

Location and host: wall of rumen in sheep, goat, cattle, buffalo, pig and horse

G. ingluvicola

Location and host: Crop of fowl

Life Cycle

Indirect. I.H. are coprophagous beetles

Family Physalopteridae

Genus *Physaloptera*

Sp. *Physaloptera praeputialis*
Location and host: stomach of cat and wild felids
Life Cycle

Indirect. I.H. are beetles. Cockroach and field crickets may act as paratenic host.

Pathogenesis

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

Treatment
Using broad spectrum anthelmintics

Family Gnathostomatidae

Genus *Gnathostoma*
Sp. *Gnathostoma spinigerum* (Stomach worm of cat and dog)
Location: Stomach of cat, dog and erratic parasite of man

- The males are 1–2.5 cm and the females up to 3–4 cm long.
- The head is swollen and is covered with transverse rows of 6–11 small hooks, and contains four sub-median cavities or ballonets each of which communicate with a cervical sac.
- The ballonets contain fluid and serve to fix the head of the worm in the tissues of the host by contraction of cervical sac.
- The left spicule is about three times longer than the right spicule.

Life cycle

Indirect. The adult worms live in tunnels in the gastric nodules, and the eggs pass in the faeces of the host, they hatch in the water and liberate L1. L1 are ingested by Cyclops (crustaceans), the first intermediate hosts where they develop to L2. The crustaceans are themselves ingested by second intermediate hosts such as fish, frogs and reptiles, and development of L3 occurs and the larvae become encysted. The L3 can also encyst in many mammals such as mice, rats and dogs. The final host is

infected by ingestion of the vertebrate vector and further development occurs in the stomach wall, where the worms provoke the growth of fibrous lesions.

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 a cavity filled with sanguinopurulent fluid. Later on the cavity develops into thick cyst or tumor connected to lumen of stomach by small canal which may cause peritonitis.

Diagnosis

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

Gnathostomosis in man

Infection is most commonly acquired by consuming inadequately cooked domestic poultry and fish. The worms never fully mature and the immature forms commonly migrate under the skin and mucous membrane resulting in pruritic swellings and eosinophilia with occasional abscess formation. Ocular gnathostomosis is characterised by haemorrhage, uveitis and perforation of the iris. A severe form of infection is central nervous system (CNS) gnathostomosis, leading to haemorrhage and eosinophilic meningitis.

Lecture-8

Superfamily Filaroidea

- Long and relatively thin worms.
- Genital opening is near the anterior extremity (opisthodelphic).
- Females are viviparous.
- Larva laid by the females are known as microfilaria. In certain species microfilariae are enclosed in thin membrane or sheath known as sheathed microfilariae whereas the ones without sheath are known as unsheathed microfilariae.

Family Filaridae

Genus *Dirofilaria*
Sp. *Dirofilaria immitis* (Canine heartworm)
Location and host: Right ventricle and pulmonary artery of dog, cat, man and wild carnivores

- 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 right is smaller and ends bluntly.
- In female vulva is situated at the anterior extremity. They are larviparous or viviparous.
- Unsheathed microfilariae have long cylindrical tail, have tapered anterior end and blunt posterior end. They measure 200-332 μm in length.

Life Cycle

Indirect. The intermediate hosts are mosquitoes (*Culex*, *Aedes*, *Anopheles*. The adults live in the heart and adjacent blood vessels and the females release microfilariae directly into the bloodstream. Microfilariae in the peripheral blood of the infected host are ingested by female mosquitoes during feeding. Development to infective L3 takes place in the malpighian tubules of the mosquito. The infective L3 then migrate to the mouthparts and the final host is infected when the mosquito takes a further blood meal. In the dog the L3 migrate to the subcutaneous or subserosal tissues in the thorax or abdomen and undergo two moults over the next few months; only after the final moult do the young *D. immitis* pass to the heart via the venous circulation. The minimum prepatent period is about 6 months. Circulating microfilariae may survive upto 2 years.

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 cause endocarditis in the cardiac valves and a proliferative pulmonary

endarteritis. Long term infection of heartworm primarily causes pulmonary hypertension due to narrowing of pulmonary peripheral artery. This condition is compensated by hypertropy of right ventricle ultimately resulting in congestive heart failure, liver failure syndrome and peripheral oedema.

The mass of worms lodged in the posterior vena cava can cause obstruction leading to an acute, sometimes fatal, syndrome known as the vena caval syndrome. This is characterised by haemolysis, haemoglobinuria, bilirubinaemia, icterus, dyspnoea, anorexia and collapse. Death may occur within 2–3 days.

Clinical Signs

Heavily infected dogs are often listless and there is a gradual loss of condition, decreased stamina and exercise intolerance. They have a chronic soft cough with haemoptysis, and in the later stages of the disease become dyspnoeic and may develop oedema and ascites. Lighter infections in working dogs may be responsible for poor performance during periods of sustained exercise.

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.
- In case, microfilariae are not detected in infected dogs, thoracic radiography and electro-cardiography is recommended. ECG will be normal at rest but after exercise T-wave maybe inverted.
- Serological test: IFAT, ELISA.
- Differential diagnosis is to be made from the microfilariae of *Dipetalonema reconditum* (found in the subcutis of dog). They are less than 300 μm in length and have a blunt head and a hooked posterior end.

Treatment

- Melarsomine @ 2.5 mg/kg b.wt repeated after 24 hrs.
- Mebendazole: 80mg/Kg b wt. for 30 days.
- Levamisole: 10mg/Kg b wt.- 15 to 20 days.
- Ivermectin: 0.2 mg/Kg b wt s/c

Genus *Parafilaria*

- The anterior end of worm bears numerous papillae and circular ridges in the cuticle.
- Remaining portion of body is transversely striated.
- Oesophagus is very short and not divided into two parts.
- In female, vulva is situated at anterior end.
- Eggs are embryonated when laid.

Parafilaria bovicola

Location and host: Subcutaneous and intermuscular connective tissues of cattle and buffalo. Disease caused by this species in cattle is known as summer bleeding.

Life Cycle

Indirect. Intermediate host is the housefly ***Musca autumnalis***. Eggs or free L1 present in exudates from bleeding points in the skin surface are ingested by the fly in which they develop to L3.Transmission occurs when infected flies feed on lacrimal secretions or skin wounds in other cattle and the L3 deposited then migrate in the subcutaneous tissue and develop to the adult stage under the skin in 5–7 months. Bleeding points develop 7–9 months after infection.

Pathogenesis

Disease caused by this parasite in cattle is known as haemorrhagic dermatitis or summer bleeding. It occurs suddenly during hot summer seasons. Adult worms in the subcutaneous connective tissue induce small inflammatory lesions and haemorrhagic nodules, predominantly on the shoulders, neck, head, withers and thoracic area. When the gravid female punctures the skin to lay her eggs, there is a haemorrhagic exudate or 'bleeding point' which streaks and mats the surrounding hairs and attracts flies. Individual lesions only bleed for a short time and healing is rapid. Nodules may become infected with pyogenic bacteria leading to abscess formation. The affected areas have to be trimmed at marketing and further economic loss is incurred by rejection or downgrading of the hides.

Diagnosis

- Examination of fresh exudates from the bleeding points for the presence of embryonated eggs or microfilariae.
- Extracting female worms from the hole of the freshly burst out nodule.

Treatment

- Nitroxynil 20 mg/Kg b wt.s/c.
- Fenbendazole and albendazole can be used.

Parafilaria multipapillosa

Location and host: Subcutaneous and intermuscular connective tissues of horse, donkey and mule.

Life Cycle

Indirect. I.H. is the horn fly ***Haematobia.***

Family Setariidae

Genus *Setaria*
Setaria labiato-papillosa
Location and host: Peritoneal cavity of cattle and buffalo.

- Worms are long and milky white in colour.
- Mouth is surrounded by chitinous ring, called as *"peribuccal ring"* having 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.
- The tail of the female ends in a marked button, which is divided into a number of papillae.
- Microfilariae are sheathed.

Setaria equina
Location and host: Predominantly peritoneal cavity but sometimes in the pleural cavity, lungs, intestine and eye of the horse, donkey, mule, zebra and occasionally in cattle and man.

Setaria digitata
Location and host: Peritoneal cavity of cattle, buffalo and zebu. Adult worms maybe found in the lesions in urinary bladder.

Life Cycle

Indirect. Intermediate hosts are mosquitoes (***Aedes*, *Culex*, *Anopheles***)

Pathogenesis

- Adult parasites in the peritoneal cavity are non-pathogenic. They become pathogenic when they migrate erratically to different organs.
- *Setaria equina* migrates erratically to the eyes of horse where it can cause occular opacity, photophobia, hypopyon.
- The immature forms of *Setaria digitata* and *S. labiato-papillosa* can be encountered in the CNS of unnatural hosts like sheep, goats and horses.
- **Enzootic cerebrospinal nematodosis or lumbar paralysis** is a condition caused by erratic migration of immature stage of *S. digitata* in sheep and goats whereas in horses this condition is known as **Kumri**. 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. This disease is often fatal. There is locomotor

disturbance usually of the hind legs. If the parasites are high in the spinal canal, there may be paraplegia.

Clinical Signs

- It depends upon the site of lesion.
- Muscular weakness, ataxia, paralysis of hind limbs and sometimes blindness may also occur.

Diagnosis

- Finding microfilariae in blood smear examination
- Adult worms in the peritoneal cavity during PM examination.
- Examination of spinal cord and CSF for the larval forms in case of cerebrospinal nematodosis

Treatment

There is no effective treatment for setarial paralysis.

Genus *Stephanofilaria*

Sp. *Stephanofilaria assamensis*

Location and host: Dermis in cattle, goat and buffalo

It causes Hump sore in cattle. The worm occurs in skin in the region of hump and adjoining areas causing chronic dermatitis/verminous dermatitis/Cascado. In Assam and W. Bengal about 90% of cattle are affected and 59% in Andaman.

Sp. *Stephanofilaria zaheeri*

Location and host: Skin, ears, legs, teats of buffalo and cattle.

It causes Ear sore in the skin of ears of buffalo in eastern and central parts of India.

Life Cycle

Indirect. I.H. is the fly *Musca conducens*.

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 readly squeezed. The lesion extends outward with the central thick dry crust.
- The lesion resolves during the dry cool season but reappear during rainy season.

Diagnosis

Examination of deep skin scrapings: The scraping should be made only after the crust is formed for the presence of microfilariae.

Treatment

- Trichlorphon ointment - 6 to 8% in petroleum jelly or castor oil.
- Ivermectin @ 0.2mg/kg b.wt

Family Onchocercidae

Genus *Onchocerca*

Microfilariae occur in the tissue spaces rather than in the peripheral blood stream.

Various species of Genus ***Onchocera*** are as follows:

S.No.	Species	Location and host	Intermediate host
1.	*O. gibsoni*	Brisket and the hindlimbs of cattle and buffalo	*Culicoides pungens*
2.	*O. gutturosa*	Ligamentum nuchae, scapular cartilage, in the hip, stifle and shoulder regions of cattle and buffalo	*Simulium*
3.	*O. volvulus*	Human being, rarely in animals	*Simulium*
4.	*O. armillata*	Aorta of cattle, buffalo, sheep and goats	Unknown
5.	*O. cervicalis*	Ligamentum nuchae of horse and mule	*Culicoides nubeculosus*
6.	*O. reticulata*	Connective tissue of the flexor tendons and the suspensory ligament of the fetlock of horse, mule and donkey	*Culicoides nubeculosus*

Pathogenesis

O. gibsoni produces fibrous and calcareous nodules in the subcutaneous tissues. These nodules are called worm nests containing coiled up worms surrounded by fibrous tissue capsule. These nodules increases in size as the animal grows in age and becomes calcified or caseation occurs. The infected carcasses are unsuitable for sale.

Adult parasites of *O. cervicalis* and *O. gutturosa* are non-pathogenic. The parasites occur in tunnels and nodules in the connective tissue with usually little reaction to the live parasites. Heavy infections of *O. reticulata* in the flexor tendons and suspensory ligaments of horses have been associated with lameness.

O. armillata adults form nodules in the aorta. In older infections, aortic wall becomes thickened, the intima shows numerous nodules containing yellow caseous fluid and coiled worms. Clinical signs are usually not attributable to the adult parasites.

The adult worms of *O. volvulus* live in subcutaneous nodules but almost the entire pathogenic effect is caused by the microfilariae; dermatitis and elephantiasis are common, but the most important effect is ocular onchocercosis ('river blindness'). Dying microfilariae cause a sclerosing keratitis in the cornea that leads to corneal opacity and retinochoroiditis.

Diagnosis

Depends on the finding of microfilariae in skin biopsy samples

Treatment and Control

- Diethylcarbamazine
- Ivermectin
- Using microfilaricides to control fly population

Family Dracunculidae

Genus *Dracunculus*
***Dracunculus medinensis* (Common name: Guinea worm, Medina worm, Serpent worm and Dragon worm)**

Location and host: Subcutaneous tissues of man and rarely in dogs.

- Male worms are 10 – 30 mm and female worms are 60 cm – 1 m in 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.

Life Cycle

Indirect. Intermediate hosts are *Cyclops*. Infection of the definitive host takes place through drinking water which contains the infected cyclops and the worms develop to maturity in about one year.

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. Healing of the worm will only be completed if the worms are totally removed or expelled.

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.
- This disease has now been eradicated from India.

Lecture-9

Order: Enoplida

Superfamily:Trichuroidea
Muscular tissue of oesophagus is much reduced. Males have one or no spicule.

Family: Trichinellidae
Male has neither copulatory bursa nor spicules. Females are larviparous.

Genus: *Trichinella*
Sp. ***T. spiralis***

Location and host: Adults in small intestine and larvae in muscles. They are found in man, pig, cat, rat and many other mammals (mostly rodents).

- These worms are very small, slender and having uniform width.
- Male 1.4-1.6 mm and female are 3-4 mm long.
- Mouth is simple, oesophagus is surrounded by a single row of cells.
- Anus in male and female is terminal.
- The posterior extremity of the male bears two lateral flaps one on either side of the anal opening, spicules are absent.
- Vulva in females, is situated in the middle esophageal region. The female is viviparous.

Life cycle

The adult parasites and infective larvae (muscle trichinae) are unusual in being present within a single host (i.e. development from larva to adult to larva in a single host). *Trichinella* does not have a free-living stage. The very small developing adults lie between the villi of the small intestine. After fertilisation, the males die while the females burrow deeper into the intestinal mucosa. About a week later, they produce L1 which enter the lymphatic vessels and travel via the bloodstream to the skeletal muscles (diaphragm, tongue, pharynx, eyes, masticatory muscles). There, still as L1, they penetrate striated muscle cells where they are encapsulated by the host, grow and assume a characteristic coiled position; the parasitised muscle cell is transformed by microvascularisation into a 'nurse cell'. These cells provide nutrition and handle the waste products. This process is complete within about 3–4 weeks, by which time the larvae are infective and may remain so for many years. Development is resumed when muscle, containing the encysted trichinae, is ingested by another host, usually as a result of predation or carrion feeding. The L1 is liberated in the stomach and in the intestine undergoes four moults to become sexually mature within about a week. Patent infections persist for only a few weeks at the most.

Pathogenesis

Disease associated with this nematode is called Trichinellosis and important from human point of view. Pigs (and other animals) serve as reservoir host of human infection. Thus, it is a zoonotic disease. It is the larval stages in the muscle of man, responsible for pathological effects which vary greatly; they include myositis, eosinophilia, myocarditis, diarrhea, fever, pain and stiffness in affected muscles, dyspnoea, oedema of face. Heavy infection may lead to death due to paralysis of respiratory muscles.

Diagnosis

In human beings:

- clinical symptoms (muscular pain, edema of face and eyelids)
- Demonstration of larvae in biopsied muscles either by pressing muscle in between two slides or by acid pepsin digestion.

In animals:
Depends usually during meat inspection

- by immunodiagnostic tests: (CFT, ELISA, Haemagglutination)
- By trichinoscope; compressed muscle is examined in screen.
- Digestion of pooled muscles.

In humans, bentonite flocculation test (comes positive after 4 weeks of infection).

Treatment

In human beings: Thiabendazole, mebendazole, fenbendazole and anti-inflammatory may be tried against larval stages.
Treatment is rarely employed in animals.

Prophylaxis

- Man usually picks up infection from pig meat when eaten raw or partially cooked e.g sausages. So care should be taken.
- Pigs should not be allowed to consume raw garbage containing scraps of trichinous meat.

Family : Trichuridae

The name of the genus under this family, *Trichuris* is misleading in that it means hair tail, but infact it is hair head. Members of this family medium to large worm, the posterior part of body is much thicker than the anterior.

Genus: *Trichuris*

The worms of this genus are called as *Whip worms* because anterior part of body is long and slender. Posterior part is much thicker.

Characters:

- Worms are relatively long, body is divided into two parts. Anterior part is long and slender, posterior part is much thicker and shorter. The feature gives the worm a whip like appearance hence the common name whip worm.
- The posterior end of male is spirally coiled.
- Single spicule is present and provided with spicular sheath which is protrusible and usually covered with fine spines.
- Vulva, in female, is situated at the junction of the two part of the body.
- Eggs have a typical barrel shaped, they are thick walles, brownish with plugs at either poles.

The members are parasites in large intestine (caecum and colon) of mammals; the following species are more common in India:-

Species	Location and host	Dimensions	Morphological characters	Egg
Trichuris ovis	Caecum of sheep, goat and cattle	Male 50-80 mm and female 35-70 mm long	Spicules 5-6 mm long. Sheath bears an oblong swelling, a short distance from its dorsal extremity.	Brown, barrel shaped with transparent plug at either pole. Measure 70-80 by 30-42 μ.
T. discolor	Caecum and colon of cattle, buffalo, sheep and goat	Male 45-59mm and female 43-55 mm	Females are orange yellow when fresh. Spicule is 0.85-3.0 mm long, sheath is covered with spines.	Eggs are 60-73 by 25-35 μ.
T. globulosa	Caecum of sheep, goat, cattle and camel	Male 40-70 mm and female 42-60 mm.	In male spicule is 4.2-4.8 mm long. Sheath has a terminal spherical expansion and covered with spines.	Eggs are 68x 36 μ in size.
T. vulpis	Caecum and other parts of intestine of dog and fox.	Worms are 45-75 mm long.	Spicule is 9-11 mm long. Sheath is covered with spines only in proximal part.	Eggs are 70-89 μ in size.
T. suis	Occurs in pig.	Male is 30-50 mm, female is 35-50mm long.	Spicule is 2-3.5 mm with blunt end. Sheath has variable shape and bears spine.	Eggs are 50-60 μ by 21-15 μ.

Life cycle

Direct. At optimum conditions, eggs reach to the infective stage in about a month. Host acquires infection by ingestion of infective eggs. The larval stage is released in the intestine and then they move to caecal mucosa. After 4 months, adult emerges on the mucosal surface. Prepatent period of *T. ovis* is 7-9 weeks, for *T. vulpis* 11-12 weeks and *T. suis* 6-7 weeks.

Pathogenesis

T. vulpis and probably other species are blood suckers. Adult parasite makes tunnel into intestinal mucosa with their anterior end (mouth stylet). Stylet is used to enter in vessels creating a pool of blood which is taken by parasite. Natural acquired infection in cattle, sheep and pig are generally not severe enough to produce clinical disease. Sometimes in pig mortality may be seen where there is anaemia, dehydration, anorexia, dysentery. As *T. suis* make tunnels in mucosa of large intestine, it may cause typhlitis and colitis. In dogs, generally there are no symptoms, but in heavy infection may cause profuse diarrhea, loss of weight and sometimes faeces may have frank blood.

Diagnosis
By demonstration of barrel shaped eggs in faeces.

Treatment

Dog
Dichlorovos 30-40 mg/kg body weight.
Mebendazole 100 mg twice a day for 3-5 days.
Fenbendazole may also be used.

Swine
Levamisole 7.5 mg/kg body weight
Tetramisole 15 mg/kg body weight
Thiophanate 0.0225-0.045% in feed.
Fenbendazole 3-25 mg/kg body weight

Sheep/goat/cattle
Oxifendazole 2.5 mg/kg body weight
Fenbendazole 5-20 mg/kg body weight

Prophylaxis
1. Proper hygiene measures, houses should be cleaned regularly.
2. Heavily infected soil should be avoided to assess by the animals and allowed to be sun-dried for killing of eggs.

Family Capillaridae
Genus *Capillaria*
- The species resemble the trichurid worms but they are small and slender, posterior part is not much thicker than anterior part.

- Life cycle can be direct or in direct.
- Eggs: Colourless shell, barrel-shaped body with nearly parallel sides and the bipolar plugs donot project.

Species found in birds

Capillaria caudinflata: Duodenum of fowl. I.H. is earthworm.
C. obsignata: Small intestine of fowl and turkey. Lifecycle: Direct
C. anatis: Caeca of domestic anatid and gallinaceous birds. Lifecycle: Direct
C. annulata: Crop and oesophagus of fowl and turkey. Lifecycle: Indirect

Pathogenesis
Mild inflammation, thickening of digestive tract and sometimes diarrhea mixed with blood can be observed.

Diagnosis and Treatment
Finding eggs in the faeces. Treating birds by providing levamisole in drinking water.

Species found in mammals

C. bovis: Small Intestine of cattle, sheep and goat.
C. plica: Urinary bladder, pelvis of kidney of dog, cat and fox. I.H. is earthworm. The worms can cause cystitis and difficulty in urination.
C. hepatica: Liver of rodents. Eggs must be released from the liver by an intercalary host (predator) or by cannibalism and the eggs are passed out in the faeces of the predator or cannibal.
C. aerophila: Trachea, bronchi, rarely nasal cavities and frontal sinuses of dogs, cats and other carnivores. In severe infection, rhinitis is observed; there may be profuse nasal discharge along with chronic inflammation.
C. philippinensis: Intestine of man in Philippines and Thailand.

Diagnsosis
Eggs in the faeces, urine, sputum or nasal discharge.

Superfamily Dioctophymatoidea.

- Oesophageal glands are multinucleated.
- Vagina is tubular.
- The reproductive system is highly developed.
- Male with one spicule, a muscular caudal sucker is present.

Family Dioctophymidae
Tail of male bears a terminal cup shaped bursa; without rays. Male has one spicule. Eggs have thick pitted wall.

Genus *Dioctophyma*
Species: ***D. renale*** **(Synonym *Eustrongylus gigas*) a.k.a. Giant kidney worm**

Location and host: Kidney of dogs and wild carnivores and occasionally in pig, horse, cattle and man.

- It is the largest nematode known in animals.
- Males are 35-45 cm by 3-4mm and females up to 103 cm by 5-12 mm.
- Worms have blood red color.
- Spicule is 5-6 mm long.
- Eggs are barrel shaped and brownish yellow (71-84x 46-52μ) and pitted except at poles. They are unsegmented when laid.

Life cycle

Eggs are passed in urine and develop slowly in water requiring 1-7 months. They do not hatch until swallowed by intermediate host which is an annelid *Lumbriculus variegatus*. In annelid they take about 100 days to reach to infective stage. Defintive host become infected by ingestion of infected annelids. Paratenic host (frogs) may also play role in transmission of disease. In final host, initially larvae penetrate the wall of GIT and finally penetrate the kidneys. Right kidney is more commonly affected than left.

Pathogenesis

Worms enter in pelvis of right kidney and destroy the parenchyma. There is severe damage to kidney parenchyma and worms are oftenly found projecting in the ureter which may be blocked. It may lead to uraemia, sometimes worms may come down to bladder and may pass out through urethra. Sometimes worms may be found in the peritoneal cavity and pleural cavity causing peritonitis and pleuritis; respectively. Generally, no symptoms are observed. Infected animals look weak, show signs of kidney trouble and nervous symptoms. If retention of urine is there then it may lead to uremia and death.

Diagnosis
By finding eggs in urine, if parasite in kidneys.

Treatment
Surgical removal.

Phylum Acanthocephala

It is a group of parasites usually considered as being closely related to nematodes. They are commonly called as thorny headed worms. The body tegument has five layers and its absorptive surface is considerably expanded. There is no alimentary canal and they feed like cestodes. There is a proboscis armed with transverse or longitudinal rows of hooks.

Order: Archiacanthocephala
Family: Oligacanthorynchidae
Genus: *Macracanthorhynchus*

Species: *M. hirudinaceus*

Location and host: Small intestine of pigs. They are found in most of the countries (except Western Europe) including India.

Features:

- They are large, stout worms with transversely wrinkled cuticle; they are pale reddish in colour. Male up to 10cm and female up to 35 cm x 4-10 mm.
- They have a non retractable proboscis at anterior extremity. It is small and armed with six transverse rows of hooks (each row has six hooks each).
- Alimentary canal is absent.
- Male has two testes and a female has a single ovary.
- Eggs have 4 layered walls of which second is dark brown and pitted; they measure 67-110 by 40-65μ.

Lifecycle

Adult worms attach to the intestine of pigs by their hooked proboscis. Fully embryonated eggs are passed in the faeces of the pigs containing a hooked larva called acanthor. Intermediate host is a dung beetle. Beetles become infected when they feed on manure. In beetles these eggs hatch and they encyst in its haemocoele. Larvae change to acanthella larvae which mature to become infective larvae called cystacanth. Pigs become infected after ingesting the adult beetles.

Pathogenesis

Worms cause inflammation and occasionally a granuloma at the site of their attachment in small intestine. Rarely may it lead to perforation and peritonitis resulting in death.

Treatment

Carbontetrachloride , tetrachloroethylene and nicotine sulphate are used for treatment.

Diagnosis

Finding eggs in faeces.

Lecture 1
Phylum Arthropoda

Introduction

The name of the phylum derived from Greek words- arthros, a joint, and podos, a foot, refers to the fact that the members of this phylum have jointed limbs.

Characteristics:

- Have jointed appendages
- Segmented bodies.
- The outer covering of Chitin forms exoskeleton which covers the external surface of the body. This chitin is secreted by chitogenous cells. The chitin not only covers the external surface of the body but also extends inside. Anteriorly, forming stomodaeum and posteriorly, forming proctodaeum.
- Appendages found on the body are typically paired.

Following are the **classes** of importance in this phylum—

1.Crustacea

- Includes the cray fishes, lobsters, shrimps, crabs, wood lice and their relatives
- Aquatic
- Breathe by means of gills.
- Two pairs of antennae.
- Numerous pairs of limbs on thorax and abdomen.
- Sub classes- Entomostraca and malacostraca

2. **Myriapoda**

- Includes centipedes and millipedes
- Their body consist of a number of segments which are, with the exception of head, not grouped into definite body areas. 2 orders are- Diplopoda(millipedes) and Chilopoda (centipedes).

3. **Insecta**

- Bodies are divided into three parts, namely, a head, which bears one pair of antennae, a thorax, consisting of three segments, which bears three pairs of legs, and typically, two pairs of wings, and an abdomen, consisting of variable number of segments (either with no appendages or modified appendages)
- Insects breathe by means of trachea.
- Three pairs of legs
- Presence of antennae.

4. **Arachnida**

- Includes king crabs, scorpions, spiders, ticks, mites and their relatives
- Four pairs of legs.
- Head and thorax are fused to form a cephalothorax.
- Absence of antennae
- No wings.

5. **Pentastomida**

- These are the tongue worms and are of uncertain systematic position

- Some consider them as arthropods and others regard them as annelids but still a distinct class is assigned to them.

Why do we study arthropods?

Arthropods are of veterinary and medical importance,

1). Arthropods cause pathological condition by **direct harm** to the host e.g. scabies due to mites. They may act as ectoparasites when sucking blood on the surface of their hosts (e.g.mosquito) or may become endoparasites when entering the skin, and the intestinal and / or respiratory tracts on a variety of hosts (e.g flies leading to myiasis). The arthropods can produce pathological condition(s) (direct harm) to the host by causing-

- Dermatitis: skin irritation resulting from bites of arthropods. Damage may be local (e.g.mosquito bites) or extensive (e.g. mite infestations).
- Envenomation: Some arthropods inject toxic substances (venom) when they bite e.g. bites (spiders), stings (wasps), urticating hairs (caterpillars). Types of poisons: Hemolytic, Hemorrhagic, Neuro-toxic, Vesiccating.
- Allergy: The allergens may be present as apart of the organism itself or a part of a secretion produced by the parasite e.g flea bite allergy.
- Myiasis: The invasion of tissue by larvae of dipterans (e.g bot fly, warbles).
- Entomophobia. The fear of arthropods regardless if harmful or not.

2) In addition to this method of parasitism, the insects may be directly or indirectly **involved in the life cycles** of a large number of parasites

- Arthropods as intermediate hosts for various parasitic helminths e.g orbatid mites for cestodes, *Musca domestica* act as intermediate host for *Theilezia rhodesii*, *Habronema muscae* etc.
- As final hosts of important parasites of humans and animals (e.g. Plasmodium)
- As true/biological vectors of pathogens such as bacteria, rikettsiae, and viruses (e.g.*Xenopsylla cheopsis* for *Pasturella tularensis*, *Boophilus annulatus* for *Babesia bigemina*)
- As mechanical vectors of some parasites (e.g. Entamoeba, Giardia cysts)

Types of Transmission

a) Mechanical-

When pathogenic organisms are carried passively by an arthropod. This can occur by attachment to body parts of the arthropod or by contaminated mouth parts of blood feeding arthropods e.g. *Musca domestica* in case of Entamoeba and Giardia cysts

b) Biological- Specific changes in the organism take place inside the vector including various morphological, physiological, or developmental changes before it is infective for the new host. e.g. case of Babesia, Theleria, Anaplasma organisms inside ticks etc.

These changes are classified as:

-cyclico-developmental. Developmental changes in the organism but no changes in numbers. Examples include canine heartworm in mosquitoes.

-cyclico-propagative. Developmental changes in the organism as well as increases in numbers of organisms. Examples include malaria in mosquitoes.

-propagative. When no development or changes occurs in the organism but increase in numbers occur. Examples include plaque in fleas.

Methods by which arthropods transmit organisms:

- Bites –as a result of blood feeding activities e.g. mosquito transmits *Dirofilaria immitis*
- Mechanical transfer- When the organism is transmitted passively as a result of either contact or feeding activities e.g *Musca domestica* responsible for the transfer of Giardia, Entamoeba cysts.
- Transtadial and transovarian e.g ticks are responsible for biological transmission of Theleria and babesia
- Faecal transmission of an organism via faeces rather than through bites or contact e.g kissing bugs spread Chagas fever

3). Arthropods act as **reservoir** for disease agents

Some arthropods play an important role in the natural distribution of arthropod borne diseases and maintaining their existence in nature when the disease is not exhibited in the host population.

4) Annoyance : cause annoyance to humans and animals.

Aim of Veterinary Entomology and Acarology-

- To control and prevent arthropods related animal diseases,

To attain the aim we must have knowledge of life cycle, habits/biology, control and pertinent animal factors,

Evolution

- Insects and arthropods preceded man on earth by 400 million years.
- Parasitism of vertebrates by arthropods seems to have begun chiefly and most successfully in lair, nests and other host habitations.
- Parasitism may have developed through arthropods scavenging on hair detritus (such as lice and mites),
- There might have been general tendency of arthropods to attack so as to feed on variety of substances. From this certain arthropods might have grown or flourished on vertebrate host for nutritious food.
- General structural adaptation would have taken place to improve their existence, like piercing or sucking mouthparts to take blood meal and tissue fluids.
- Probable nutritional dependency is also one of the reasons for parasitism because insects require high protein diet, which is present in the blood meal to eggs to develop.

Historical aspects

With the vast numbers, diversity, and economic importance of insects, it is not surprising that entomology is rooted in nearly all human cultures from prehistoric times. However, scientific study only began as recently as the sixteenth century.

- Early interest of humans in insects is shown in the rock painting of bees from approximately 13,000 B.C.E
- The first documented forensic entomology case is reported by Song Ci around 1250 C.E. in the medico-legal text book *Xiyuan Jilu*, where he describes the use of insects in the case of a stabbing near a rice field.
- **Aristotle** knew about mites. He could observe that one type of them can be removed from dry boils. After the discovery of microscope, it was known that he had observed mites, which caused scabies.

- **Conrad Gesner** in 1551, zoologist published the first volume of *Historia animalium* ("History of Animals"), a work that includes some mention of insects.
- **Mercurialis (1577)**: Flies carry organism of plague from ill man to food.
- **Gabriel Soares De'souza (1587)**: Fly suck poison from soars and leaves on the skin aberration of the healthy man.
- **Cosmo Bueno (1764)** Cutaneous Leishmaniasis is transmitted in country Peru by a fly Uta (sandfly)
- **Josiah Knott (1848)** published that mosquitoes give rise to malaria and yellow fever.
- **Melnikoff (1869)** Bitting louse *Trichodectes canis* act as intermediate host for *Dipylidium caninum.*
- One of the first discoveries that incriminated arthropods in the transmission of diseases was that of Theobold Smith and F.L. Kilbourne who proved that a tick was responsible for the transmission of the protozoan parasite that caused Texas cattle fever. This discovery was quickly followed by others that implicated arthropods in the transmission of such diseases as malaria, yellow fever, trypanosomiasis and typhus.
- In 1900, **Walter Reed**, a United States Army major, was appointed president of a board "to study infectious diseases in Cuba paying particular attention to yellow fever." He concurred with **Carlos Finlay** in identifying mosquitoes as the agent of transmission.
- In 1902, **Ronald Ross** was awarded the Nobel Prize for Medicine for his discovery that malaria is carried by mosquitoes. The awarding committee made special mention of the work of Giovanni Battista Grassi on the life history of the "Plasmodium" parasite (a protozoan that uses the mosquito as a host).
- In 1934, **Vincent B. Wigglesworth**, the "Father of Insect Physiology," wrote the first book on the subject, *The Principles of Insect Physiology*.
- **David Bruce** (1920-30), reported that tsetse fly (*Glossina palpalis*) transmits African Trypanosomosis.
- **Carlos Chagas** (1900's) reported that Chagas fever is transmitted by cone nose/kissing bug**s.**

Lecture 2
Phylum Arthropoda

The name of the phylum derived form Greek words- *arthros*, a joint, and *podos*, a foot, refers to the fact that the members of this phylum have **jointed limbs**.

Characteristics:

- Have jointed appendages.
- There is an outer covering of **chitin** which forms exoskeleton, secreted by chitogenous cells. Chitin also passes through the mouth into the anterior part of alimentary system called the **stomodaeum** and through the anus into the posterior part of the alimentary canal forming **proctodaeum**.
- The exoskeleton is present in the form of chitinous plates, called **sclerites**. A typical part of the body having dorsal sclerite is called **tergum**, a ventral sclerite is called **sternum**, and a lateral sclerite is called a **pleuron**.
- As the arthropod grows it becomes too big for its chitinous covering and periodically this chitinous covering is cast off and a new exoskeleton is formed. Each casting of exoskeleton is called **ecdysis**
- Bodies are metamerically **segmented** having head, thorax and abdomen.
- There is a presence of paired **appendages.** On the head there is a presence of one or two pairs of sensory antennae and, behind these, paired appendages modified for feeding. There is one pair of mandible and two pairs of maxillae. Walking legs are found on thorax.
- The general body cavity is a space full of blood called the **haemocele**. The blood in it bathes all the organs of the body.
- **Respiratory organs** of arthropods

Gills	of various kinds found in larvae, nymphs and adults of species that are **aquatic**
Tracheae	are fine elastic tubes which are held open by chitinous spiral thickenings. These tracheae **branch** and **ramify** among the **internal organs** as air sacs. They take air that enters them through their **external openings** or **stigmata.** Characteristic of **insects** Stigmata are not present on head and prothorax.
Lung books	spiders
Gill books	crabs
Cuticle	parasitic mites

Alimentary canal

Consist of foregut/ stomodaeum followed by midgut/mesentron and then hind gut/ proctodaeum (fig.5)

Buccal cavity (salivary glands, epi- and hypo-pharynx→ pharynx →proventriculus/gizzard → crop/ oesophageal diverticulum→mid gut / mesenteron → proctodaeum/hindgut.

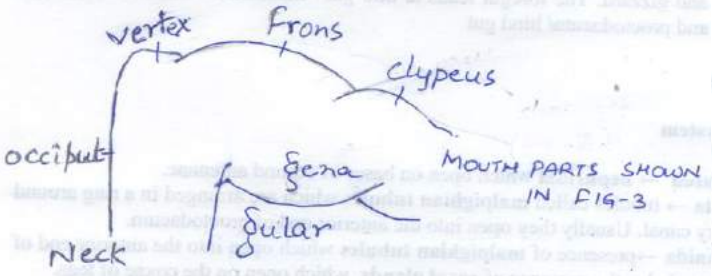
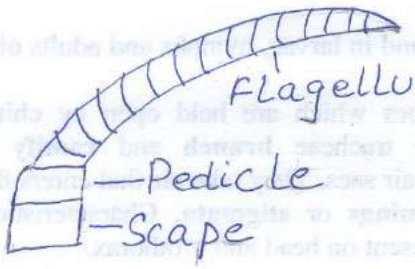


Fig2 HEAD OF INSECT



PARTS OF FLAGELLA

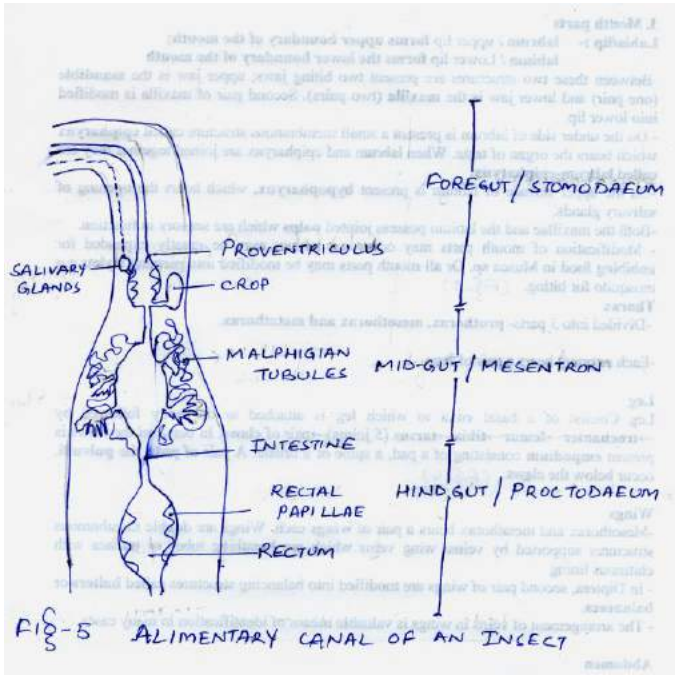
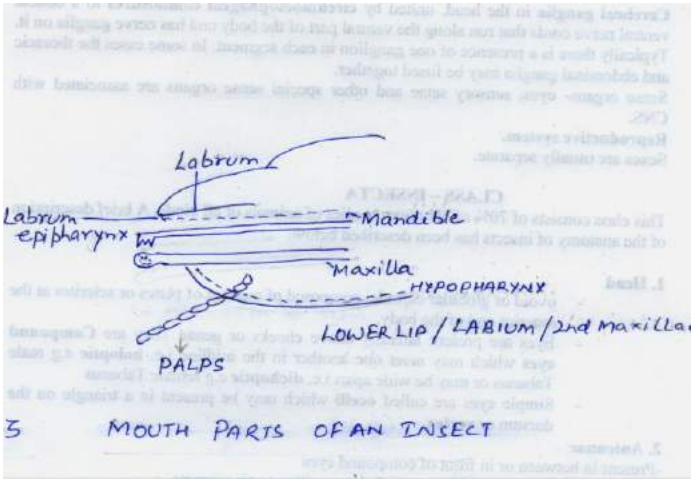
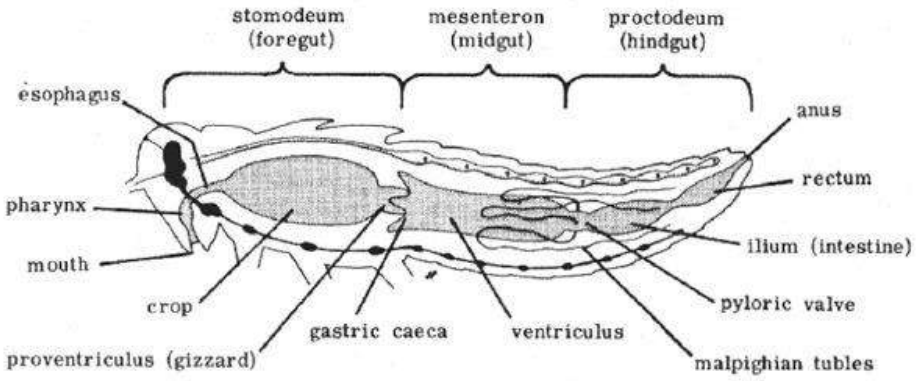


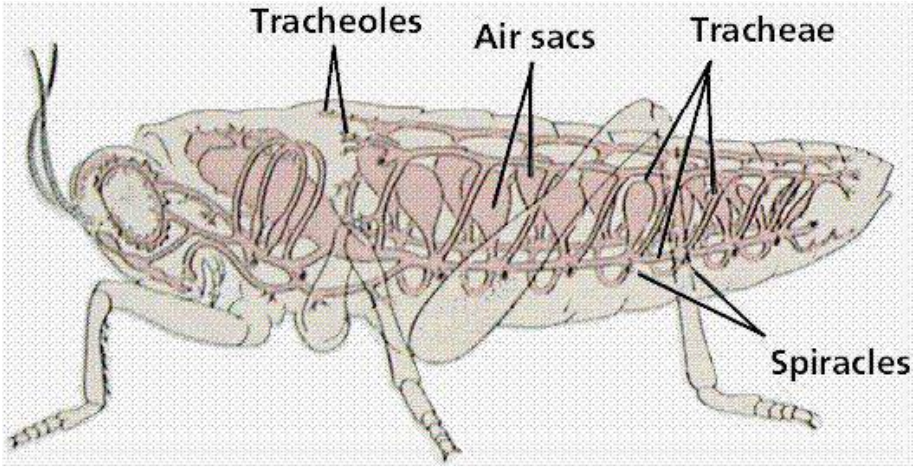
Fig-5 ALIMENTARY CANAL OF AN INSECT



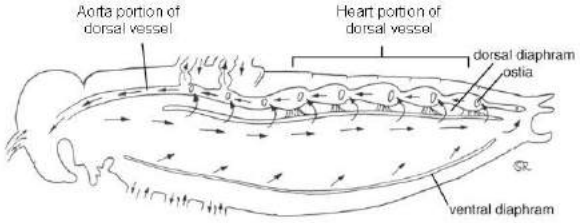
3 MOUTH PARTS OF AN INSECT

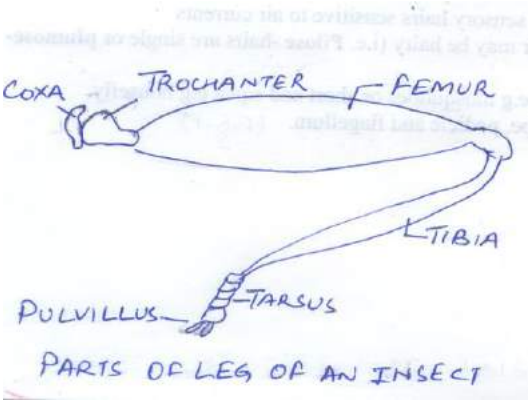


The "generalized" digestive system of insects.

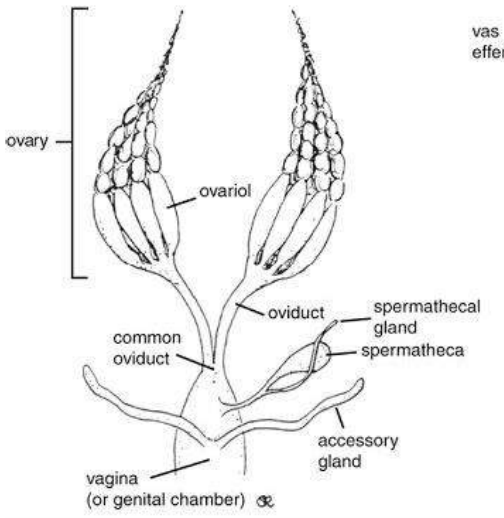


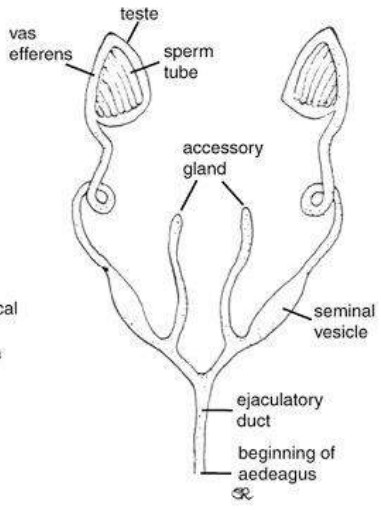
Respiratory system of an insect





Circulatory system of an insect





Female reproductive system

Male reproductive system

Excretory system

Class Crustacea → nephridia which open on the bases of second antennae.

Class Insecta → tubules called **malpighian tubules** which are arranged in a ring around the alimentary canal. Usually they open into the anterior part of proctodaeum.

Class Arachnida →presence of **malpighian tubules** which open into the anterior end of proctodaeum and also the presence of **coxal glands,** which open on the coxae of legs.

Nervous system

Cerebral ganglia in the head united by **circumoesophageal commisures** to a double ventral nerve cords that run along the ventral part of the body and has nerve ganglia on it. Typically there is a presence of one ganglion in each segment. In some cases the thoracic and abdominal ganglia may be fused together.

Sense organs- eyes, sensory setae etc are associated with CNS

TympanalOrgans

Legs, wings, abdomen, and antennae

Chemo receptors

Mouthparts, antennae, tarsi, etc.

Olfactory Sensilla

Antennae

CLASS – INSECTA

1. Head

- ovoid or globular capsule, composed of number of plates or sclerites at the anterior end of the body.
- Eyes are present laterally above cheeks or genae They are **Compound eyes** which may meet one another in the midline i.e. **holoptic** e.g male Tabanus or may be wide apart i.e. **dichoptic** e.g female Tabanus
- Simple eyes are called **ocelli** which may be present in a triangle on the dorsum or **vertex.** (Fig.2)

2. Antennae

-Present in between or in front of compound eyes

-These are tactile organs with sensory hairs sensitive to air currents

-May carry bristles (aristae) or may be hairy (i.e. **Pilose** -hairs are single or **plumose-** hair are numerous)

-May be segmented and long e.g mosquitoes or short and squat e.g housefly.

-Divided into three parts- scape, pedicle and flagellum.(figure1)

-Presence of olfactory pits (for smell probably)

3. Mouth parts

Labia/lip :- **labrum /** upper lip **forms upper boundary of the mouth;**
labium / Lower lip **forms the lower boundary of the mouth**

-Between these two structures are present two biting jaws; upper jaw is the **mandible** (one pair) and lower jaw is the **maxilla** (two pairs). Second pair of maxilla is modified into lower lip.

- On the under side of labrum is present a small membranous structure called **epipharynx** which bears the organ of taste. When labrum and epipharynx are joined together they are called **labrum-epipharynx.**

-On the upper surface of labium is present **hypopharynx,** which bears the opening of salivary glands.

-Both the maxillae and the labium possess jointed **palps** which are sensory in function. (fig.3)

- Modification of mouth parts may occur e.g labium may be greatly expanded for imbibing food in Musca sp. Or all mouth parts may be modified into piercing stylets e.g mosquito for biting.

Thorax

-Divided into 3 parts- **prothorax, mesothorax and metathorax**.

-Each segment bears a pair of **legs**.

Leg

Leg Consist of a basal **coxa** to which leg is attached to the body followed by **→trochanter→femur→tibia→tarsus** (5 joints)→pair of **claws**. In between the claws is present **empodium** consisting of a pad, a spine or a bristle. A pair of pads, the **pulvulli**, occur below the claws.

Wings

-Mesothorax and metathorax bears a pair of **wings** each. Wings are double membranous structures supported by veins/ wing veins which are breathing tubes or trachea with chitinous lining

- in Diptera, second pair of wings are modified into balancing structures called **halters** or **balancers**.

- The arrangement of veins in wings is valuable means of identification in many cases.

Abdomen

-May consists of 10-11 segments.

-Various structures may be present on the abdomen such as the external genitilia, copulatory claspers/ hooks (male) or an ovipositor (female)

Respiratory system

Consists of **Tracheae** which are fine elastic tubes which are held open by chitinous spiral thickenings. These tracheae **branch** and **ramify** among the **internal organs** as air sacs. They take air that enters them through their **external openings** or **stigmata.** Stigmata are not present on head and prothorax.

Alimentary canal

Consist of foregut/ stomodaeum followed by midgut/mesentron and then hind gut/ proctodaeum

Buccal cavity(salivary glands, epi- and hypo-pharynx→ pharynx →proventriculus/gizzard → crop/ oesophageal diverticulum→mid gut / mesenteron → proctodaeum/hindgut.

Of the stomodaeal origin are the buccal cavity with the salivary glands, the hypo- and epi- pharynx, the pharynx, the proventriculus or gizzard and the crop, or oesophageal

diverticulum or food reservoir. The mesentron forms the midgut. At its posterior end is the ring of malphigian tubes which have an excretory function. The proctodaeum consists of the intestine and the rectum, together with the malphigian tubes and the papillae of rectum.

Vascular system

Consists of a dorsally situated heart, an aorta and general body cavity or haemocoele. The heart is a tube surrounded by pericardial cells and its lumen is divided into a number of compartments by valves which allow the blood to pass forward only. Each compartment opens into haemocoele through a pair of ostia. Anteriorly there is an aorta which carries the blood to the head, whence it enters enters the haemocoele and bathes all the organs.

Blood enters the heart through the ostia as the alary muscles dilate the heart chamber, but the ostia close when circular muscles of the chamber contract, and their valvelike arrangement prevents the blood from returning to the hemocoeles or posterior chambers. Because the contractions start at the posterior chamber of the heart and pass forward, the blood is pushed anteriorly, passing through the aorta, a constricted portion of the anterior part of the heart. The blood is pumped from the aorta into a series of cavities or sinuses and veins throughout the insect. The blood bathes the "brain," and from there it passes posteriorly through the hemocoeles, bathing the internal organs and passing into the appendages and wings. The circulation of the blood is aided by the contraction of a ventral diaphragm, and often by accessory hearts at the bases of legs and wings. One function of the blood is to carry digested food materials from the alimentary canal to all tissues of the body. In contact with the alimentary canal, the blood picks up the products of digestion by osmosis. While the blood is bathing the network of Malpighian tubules, waste products are removed. Insect blood has no red blood corpuscles and plays no part in oxygen transport, although the plasma of chironomid larvae does contain hemoglobin which may aid in respiration.

Nervous system

Cerebral ganglia in the head united by **circumoesophageal commisures** to a double ventral nerve cords that run along the ventral part of the body and has nerve ganglia on it. Typically there is a presence of one ganglion in each segment.. In some cases the thoracic and abdominal ganglia may be fused together.

Reproductive system

Male The male has two testes, each with a vas deferens forming a vesicular seminalis and then fusing to form a common duct. A penis with sheath and other accessory structures are frequently present.

2 testes→vas deferens→seminal vesicle→duct→penis/hypopygidium with sheath

Female has two ovaries which consist of groups of ovarian tubes, all arising from an apical filament. The ducts join to form a common duct which bears the receptaculum seminis and ends in an ovipositor.

2 ovaries→ovarian ducts / tubes→receptaculum seminis→ →ovipositor.

Insects may be:-

- Oviparous
- Viviparous or larviparous
- Pupiparous e.g Glossina and Pupipara i.e. they give birth to larvae that are ready to pupate. The larvae are nursed in the common portion of the oviduct, the uterus

which is provided with milk glands that secrete a milky fluid through a teat to which the mouth of the larva is applied. The larva usually lies with its stigmatic plate at the vulva of the fly and is thus able to breathe.

Life cycle

Ecdysis

Casting of outer exoskeleton

Stadia

Different stages separated by ecdysis

Instar

Form of insect during each stadium

During their development insects undergo a variable degree of change. The changes that mark the growth of an insect from the egg to an adult are expressed by the term **Metamorphosis. It may be of two types:-**

1. **When the metamorphosis is complete i.e. Holometabolus**

   Egg→larvae (feeds actively)→pupa/chrysalis (quiescent stage)→Imago/adult e.g in housefly or butterfly

Larva

The larvae of species with complete metamorphosis take various forms.

- **Polypod larva** – has a well marked head, a thorax of three segments, each of which bears a pair legs and an abdomen of ten segments which bears five pairs of legs, called prolegs.e.g. caterpillar of butterfly

- **Oligopod larva-** has well marked head and three pairs of thoracic legs but no abdominal legs.e.g larvae of beetles

- **Apodous larva-** no legs either on thorax or abdomen and the head is also reduced. e.g. the larvae of houseflies, blow flies and of all diptera are apodous and are called maggots.

Pupa

The pupa of insects can take three forms:-

Free or exarate pupa: The wings and legs of the adult insect can be seen externally and free from the rest of the body e.g pupa of beetle

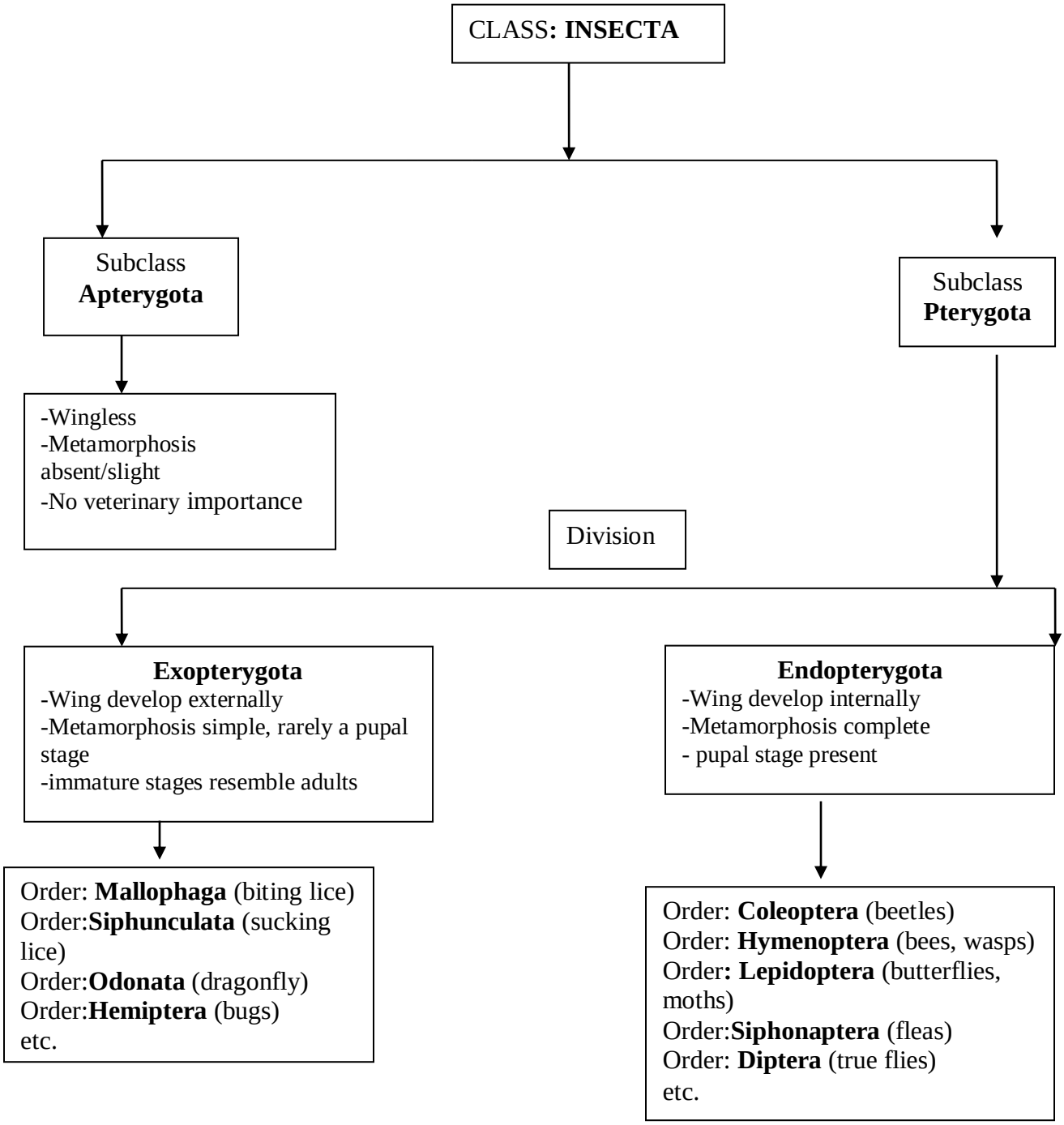
Obtectate pupa:- In this pupae, the legs and the wings are bound down by moulting fluid, but they can be seen externally. e.g pupa of butterfly and moth, horse flies (Brachycera), mosquito and their relatives (Nematocera).

Coarcotate:- The pupae is enclosed in the cast of skin of the last larval phase, which is called *puparium*. Their skin hardens and the insect cannot be seen externally.e.g the pupae of house fly.

2. **When the metamorphosis is slight or simple/ Hemimetabolus** i.e. the form that leaves the egg is more like an adult and it simply casts off its skin several times to form an imago e.g members of the subclass Apterygota.

Egg→nymph→adult

CLASSIFICATION OF INSECTA



Lecture 3
Sub class: Pterygota
Division: Exopterygota

1. **Order Mallophaga (biting lice)**

Sub orders:

1. **Amblycera**
2. **Ischnocera**
3. **Rhynchophthirina**
4. **Anoplura**

1. Order Mallophaga (biting lice)

Characters:

- Small and wingless
- Dorsoventrally flattened bodies
- Antennae are short and with 3-5 segments
- Vestigeal eyes and indistinct segmentatation of thorax.
- Presence of prothorax, and meso and meta thorax are fused.
- Spiracles on ventral side of mesothorax. (1 pair)
- On abdomen, presence of 6 pairs of abdominal spiracles.
- Tarsus has two segments with 1 or 2 claws.

Lifecycle

- Egg→nymph1→nymph-2→nymph 3→adult.
- The operculated eggs are cemented to hairs/ feathers of the host.
- Whole life cycle is passed on the host.
- Uninfected hosts are infected by close contact with the infected host or by farm equipment and personnel.

Sub order: Amblycera

Characters:

- Antennae lie in the grooves in the side of the head and may not be readily seen.
- Antennae have 4-5 segments and 3rd segment is stalked like egg cup.
- Maxillary palps present segmented (2-5 segments) and 3rd segment is not stalked.
- Mandibles bite horizontally.
- Head is broader and more rounded than members of order Siphunculata..
- Parasitic in birds and mammals.
- Birds amblycera→tarsi have 2 claws
- Mammal amblycera→tarsi has 1 claw.

Amblycera of birds

1. *Menopon gallinae* (shaft louse)

- Males-1.7mm and females-2.04mm
- Thoracic and abdominal appendages have one dorsal row of bristles.
- Ducks, fowl, pigeons are the hosts.
- Eggs are laid in clusters on the feathers.

2.*Menacanthus stramineus* (yellow body-louse of poultry)

- Found on less densely feathered parts like breasts, thighs and around anus.
- Occurs on fowl, turkey, peacock, peacock, Japanese pheasant
- males→2.8mm and female→3.3mm
- Abdominal segments have 2 dorsal rows of bristles.
- Eggs have filaments on anterior half of shell and on operculum and laid in clusters.

Amblycera of mammals

- *Gyropus ovalis (guinea pig)*
- *Gliricola porcelli(guinea pig)*
- *Heterodoxus spiniger (lice of dog)*
- *H. longitarsus (on kangaroos and dogs)*

Suborder : Ischnocera

Characters:

- Antennae filiform and is visible at the sides of head with 3-5 segments.
- Head is broader than sucking lice.
- No maxillary palps.
- Mandibles bite vertically.
- In abdomen, 1&2 and 9&10 segments are fused.
- Parasitic on both mammals and birds.

Ischnocera of birds

1. *Lipeurus heterographus* (head louse of poultry)
    - Males-2.4mm and females-2-6mm.
    - Occurs on skin and feathers of head and neck of fowl and patridges.
    - In males→ first segment of antenna is long and bears a posterior process. And abdomen is elongated.
    - In females, abdomen is barrel shaped with brown tergal plates.
    - Eggs are laid singly on the feathers.
2. *Lipeurus caponis* (wing louse of poultry)
   Found on the underside of wings of fowl and moves very little.
2. *Gonoides gigas* / *Goniocetes gigas* (large louse occurring on body and feathers of fowl)
3. *Goniocotes gallinae* (fluff louse). Occurs in fluff at the base of the feathers of fowls, pheasants and pigeons) Male-1mm and female-1.6 mm body is broad and the head is short and wide.

Ischnocera of mammals

1. ***Damalinia bovis (D. bovicola)*** → found on neck and shoulders of cattle Also known as ***Trichodectes scalaris*** (little red cattle louse)
2. ***Damalinia equi***
    - Found on head and thorax of equines.
    - Lifecycle of 29 days
    - Legs are reddish brown and these form scab like lesions abdomen has 8 dusky transverse bands.
3. ***Damalinia ovis*** →on sheep.
4. ***Damalinia caprae*** → goats.
    - Retards growth and condition of flesh and also causes reduction in mohair clip.

5. ***Bovicola painei*** (goats)
6. ***Damalinia limbata*** (angora goats).
7. ***Trichodectes canis*** (dogs)
8. ***Felicola subrostratus*** (cats). Has triangular head.

2. Order: Siphunculata (sucking lice)

Characters:

- Wingless.
- Suck blood and tissue fluid of host.
- 2 antennae on the side of the head with 5 segments.
- No sexual dimorphism.
- Thoracic segments are fused.
- Abdomen has 7 segments.
- Abdominal Segments bear dark brown areas of thickened chitin called paratergal plates.
- Eyes are absent or reduced.

* Exception (eyes are present)

-***Pediculus humanus*** (human head louse)

-***Phthirus pubis*** (human pubic louse)

- First pair of legs is smaller and the third is the largest.
- 2 segments of tarsus are indistinguishable and each tarsus has one claw.
- Thumb like process on ventral apical angle of tibia (hair of the host is held by claw and this process).
- In Haematopinidae, hair is held by tibial pad.
- One metathoracic and 6 pairs of abdominal spiracles.

Life cycle

Egg —(12 days)→ nymph 1 → Nymph 2 →Nymph 3 → adult (sexual maturity in 3 days). (12 days)

Important families:

1. Haematopinidae
2. Linognathidae
3. Pediculidae

Family: Haematopinidae

- Eyes are absent
- Head has forward prolongations (temporal angles) behind antennae.
- Thorax is broad with paratergal plates.
- One row of spines on each abdominal segment.

1. *Haematopinus asini* (sucking louse of equines)
2. *Haematopinus bufali (*sucking louse of *buffaloes in S.Africa)*
3. *Haematopinus suis (very large louse of pigs)*
4. *Haematopinus eurysternus (short nosed cattle louse)*
5. *Haematopinus tuberculatus (large buffalo louse. Size- 5.5mm)*

Family : Linognathidae

- Eyes are absent.
- Abdomen is membranous with numerous hairs.
- The first pair of legs is the smallest.
- Mostly found on ungulates.

1. *Linognathus ovillus* (body louse/blue louse of sheep/face louse). Occurs chiefly on the face. Head is longer and also longer than thorax.
2. *Linognathus vituli* (long nosed cattle louse)
3. *Linognathus africanus* (African blue louse of sheep)
4. *Linognathus pedalis* (foot louse of sheep) occurs on foot and legs where there is no wool.
5. *Linognathus stenopsis* (goats)
6. *Linognathus setosus* (dogs and foxes)
7. *Solenopotes capillatus* (cattle)

Family : Pediculidae

- pigmented eyes
- abdomen has paratergal plates.
- Wide thorax and small abdomen.
- First pair of legs being slender with slender claws.

1. *Pediculus humanus*
2. *Phthirus pubis*

Effects of lice on host.

1. They cause irritation to the host.
2. lice is found more in winters because of
   – longer hair on host's coat
   - closer contact of animals.
   - lack of general vigour.
3. Animals are restless, do not sleep well, they bite and scratch themselves and damage their feathers, wool and hair.
4. Decreased egg production and milk production.
5. Wounds and bruises due to scratching.
6. In sheep, wool is damaged and also soiled by faeces of the lice. The coat of the the animal becomes rough and shaggy
7. In calves, excessive licking may result in the formation of hair balls in the stomach.
8. The foot louse of sheep may cause lameness.

Diagnosis:

By finding the lice or eggs/nits on hairs and feathers**.**

Control and treatment.

Control includes the use of insecticides like organophosphorus compounds, organochlorides carbamates and pyrethroids.

1.Poultry

- Painting of perches with a strong extract of nicotine or tobacco
- Dusting with sodium fluoride **.**

- Dust or sprays of -Carbaryl (5%),
  -Coumaphos/asuntol (0.06%)
  - Lindane/HCH
  -Malathion (0.1%)

2. **Cattle**

- Crotoxyphos (3% dust)
- Ciovap spray (1% crotoxyphos+ 0.25% dichlorovos)
- Coumaphos (0.06%) spray
- Crufomate (dip-35%; spray 25%, pour-on 13.5%)
- Ronnel (.25% spray, 1% solution in oil, 25% as pour on)
- Methoxychlor (0.5% as spray or dip)
- Malathion (0.5%)
- Cypermethrin (150 ppm as dip or spray)
- ivermectin s/c 0.2mg/kg is 100% effective for *L vitulli* and *H. eurystrenus.*

3. **Sheep**

Control of lice depends on the ability of insecticide to penetrate the fleece of the sheep. Jets, showers, sprays are generally satisfactory.

-Ronnell (24%) or Diazinon (0.06%)

- organophosphorus compounds and synthetic pyrethroids are useful.

4.Pigs

-Coumaphos (0.06%)
-Crotoxyphos (1%)+ DDVP (0.25%).
-Malathion (0.5%)
-Ronnell (0.25%)

5.Horses

- Malathion (0.5%) spray

6. Dogs

- Coumaphos (0.5%) dust.
-Carbaryl shampoo.
- Ronnell (.25-1%) solution or 110mg/kg as systemically.

7. Cats.

- Chlorinated hydrocarbons should not be used in cats. Dichlorovos may be incorporated into a flea collar.
- Pyrethrum or carbaryl may be used as a shampoo or powder.

8. Man

- hexachlorocyclohexane (1%)
- Pyrethrins (2%)+ 2% piperonyl butoxide
- copper oleate (0.03%)

Lecture 4

Order: Hemiptera (bugs)

- Mouth parts are adapted for piercing and sucking.
- 2 pairs of wings. Anterior one is often harder than posterior one.
- Blood suckers.

Family Cimicidae

Genus *Cimex lectularius* (bed bug)

- Attack man and animals to suck blood.
- 4-5mm long, flat bodied, yellowish brown to dark brown in colour.
- Antennae has 4 joints.
- Compound eyes project at the side of the head.
- Prothorax is large and deeply notched where head is inserted in it.
- Wings are vestigial.
- Abdomen has 8 visible segments.
- Whole body is covered with spinose bristles and some hairs.
- Tibia is long and tarsus has 3 joints.
- Adult has a pair of ventral thoracic stink glands which emits a characteristic odour.
- Mouth parts are adapted for piercing and sucking.
- Labrum is immovable. Labium forms a tube with 4 joints and contains piercing mandible and maxillae.

Life cycle.

Eggs (150-200 in no.) are laid in dark crevices. Eggs are about 1mm long with operculum. Larva hatches in 3-14 days at 23ºC→ 5 nymphal stages (N1→N2→N3→N4→N5) → adult.

Under favorable conditions, the adult stage is reached in 8-13 weeks after hatching. Bugs live in crack and crevices of wood near sleeping places of their host. Mainly they are nocturnal. After blood meal they defaecate in or near the wound and transmit diseases. Apart from being annoying insects, they may cause severe irritation and anaemia in poultry.

Control

- HCH, Chlordane, Dieldrin (sprays, powders, smokes)
- Synergised pyrethrins and synthetic pyrethroids.
- Organophosphorus compounds as sprays (1% dimethoate or ronnell 1%)
- Dry baits (e.g DDVP-2,3 dichlorovinyl dimethyl phosphate) or resin impregnated strips (DDVP).
- permethrin

Family Reduviidae

Intermediate Hosts of *Trypanosoma evansi*

Well developed wings. Cone shaped head. Known as cone nosed bugs, kissing bugs , assassin bugs. Transmits Chagas fever/ *Trypanosoma cruzi* infection.

Genus – Triatoma is the largest and contains most vector species.

Life cycle
5 nymphal stages.

Division : Endopterygota

- Pupal stage is present.
- Metamorphosis is complete.
- Wings develop internally.

Order: Coleoptera

- 2 pairs of wings.
- Anterior mesothoracic(hard and leathery) and posterior (metathoracic).
- Mouth parts are adopted for chewing.
- Metamorphosis is complete.
- Free pupal stage.

Disease transmission.

1. Act as intermediate host for various tapeworm and nematodes e.g *Spirocerca lupi*, *Ascarops strongylina*, *Physocephalus sexalatus*, *Gongylonema pulchrum*, *Macrocanthorhynchus hirudinaceous*, *Raillietina cesticillus* .
2. Scavenger beetles feeding on carcass shown to carry Anthrax bacilli.
3. Dung beetles spread bacteria of faeces.

Order: Siphonaptera (Fleas)

- *Wingless* insects.
- *Laterally compressed* bodies, about 1.5-4mm long.
- Thick brown chitinous covering.
- Compound eyes are absent.
- Abdomen has 10 segments.
- 9th abdominal segment bears a dorsal plate called *sensilium or pygydium* which is covered by sensory *setae*.
- Tergum of 9th abdominal segment is modified to form *claspers*.
- Penis is chitinous, coiled and its structure is complex.
- A number of *large spines* are present on head and the thorax known as *combs or ctenidia.*
- On the cheeks (gena), there are *genal combs* and on the posterior border of the first thoracic segment *pronotal combs*.
- The short, clubbed *antennae* are sunk in the *antennal grooves* on the side of head.
- The legs are strong, long and adapted to leaping.

Different species are:

1. ***Ctenocephalides felis***

Ctenocephalides felis felis

On cat, dog, man, mouse, rat and primates

2. ***Ctenocephalides canis***

On dogs and related species

3. ***Spilopsyllus cuniculli*** (rabbit and hare)

4. ***Ceratophyllus fasciatus***
Rats and house mouse.
5. ***Xenopsylla cheopsis*.** Also known as Oriental or black rat flea and is associated with the transmission of Plague. Present on rodents in ports and occurs on rat, mice and other rodents.
6. ***Pulex irritans***. / Human flea.
Also in pigs, dog, cat rat
7. ***Tunga penetrans/*** Jigger or chigoe flea (on man).
8. **Bird fleas**
-Ceratophyllus gallinae.(on chicken)
-Ceratophyllus columbae. (pigeon)
-Echidnophaga gallinacea/ stick tight flea (chicken)

Life cycle

Female lays 20 eggs at a time **(**in a lifetime around 400-500 eggs**)** and deposited in dust and dirt, or may be laid on the host. **Eggs** are around 0.5mm long and drop off soon, rounded and pearly white in appearance

↓

2 -16 days

↓

Larvae breaks the egg by chitinous spine on the first larval instar
- 6mm long, creamy yellow
-elongate slender maggot like having 3 thoracic and 10 abdominal segments with few long hairs
- Last abdominal segment bears 2 hooked processes called *anal struts* for holding or locomotion.
- Larvae feed on dry blood, faeces and other organic matter and are c*oprophagus*.
-Moderate temperature and high degree of humidity is favourable for development.

↓

Pupa

↓

10-17 days

↓

Imago

↓

Adult

Habits and significance.

-Longevity depends upon the degree of moisture and also whether they are fed or not.
-Not specific for their host and leave it and feed on other hosts.

Pathogenesis.

1. Direct effect

- Generally chronically debilitating and poorly kept animals are affected more.
- Animals become restless, loose condition and spoil their coat by biting and scratching.
- The reaction to the flea bite is determined by the stage of sensitization of an animal to the saliva of the flea. The hapten in the saliva of the flea becomes immunogenic by fixation to skin collagen.

- Immediate type hypersensitivity reaction is the most usual reaction seen and is characterized by elevated levels of immunoglobulin E and eosinophilia at the site of the reaction.however, Delayed type of hypersensitivity response can also be seen.
- Animals vary greatly in their sensitivity to flea bites
- Dogs and cats that do not become hypersensitive to flea saliva show mild pruritus which is often unnoticeable.
- Those which become hypersensitive show moderate to severe pruritic reaction esp. on lumbosacral, abdominal, hindleg and neck region resulting into moist dermatitis.
- Secondary infection may occur resulting in the formation of papules with scales.
- Acanthosis, hyperpigmentation and hyperkeratinization may occur.
- Cats show military dermatitis (small papules associated with pruritus) due to hypersensitivity.
- In poultry, Stick tight flea/ *Echidnophaga gallinacea* found mainly on comb, wattles and around eyes and flea does not jump away when disturbed. The female burrows into the skin cause swellings which ulcerate. The females lay eggs here and after hatching the larvae fall out and develop to adult in about 4 weeks.

2. Disease transmission

1. Bubonic plague (*Yersinia pestis*)

Transmitted by *Xenopsylla cheopsis*. There is blocking of the chitinous teeth of the proventriculus with blood and bacilli. A blocked flea is unable to feed, so it wanders from host to host in attempt to feed. In doing so it contaminates the new hosts with bacilli.

Enzootic plague/sylvatic plague

Rats →squirrel →humans.

2. Endemic or Murine Exanthematous Typhus

Caused by ***R****ickettsia mooseri*. Spread by fleas of rats and mice in human dwellings.

Rickettsia multiply in flea gut and are excreted in the faeces of flea and transmitted to man

3. Tularaemia

Disease is Caused by Francisella tularensis . Transmission occurs mechanically by flea.

4**. Myxomatous virus** of hares and rabbits

Transmitted by *Spilopsyllus cuniculli*. Virus spreads through the contaminated mouth parts of the flea.

5. **Salmonellosis *(Salmonella enteritidis).*** The bacteria multiply in the stomach of flea resulting in the contamination of both the oral and anal routes. There is blockage of proventriculus.

6. *Ctenocephalides canis*, *C. felis, Pulex irritans* act as intermediate host for ***Dipylidium caninum.***

7. *C.canis, C. felis* act as intermediate host for filarial worm of dog ***Dipetalonema reconditum***

Control.

Includes

Treatment of animals to kill fleas, elimination of developmental stages and prevention of reinfestation of environment**.**

-Flea collars of dichlorvos (DDVP), 9.3% for dogs and 4.65% for cats, gives protection for 3 months)

-Combination of Dichlorovos and fenitrothion

-Carbaryl shampoos for dogs and cats.

-Coumaphos/asuntol (0.5%) dust

-Ronnell 1% solution for topical application.

- Premises should be thoroughly cleaned; insecticide should be blown into likely breeding places. Beds and beddings of dogs and cats should be cleaned or destroyed.

Poultry

-4%Malathion (dust) and BHC (0.1-0.2%) dust.

Lecture 5

Order Diptera

1. Single pair of functional membranous wings (mesothoracic pair)
2. Metathoracic pair is modified to halters/balancers.
3. Mouth parts are adapted for piercing and sucking or can be of lapping type.
4. Mesothorax is usually large and fused to pro and meta thorax.
5. Tarsi usually have 5 joints.
6. Metamorphosis is usually complete, Larvae being apodous with reduced heads.
7. Pupa may be coarcotate or obtectate.
8. No of veins in the wings are reduced.

Suborder :Nematocera	**Suborder:Brachycera**	**Suborder: Cyclorrhapha**
1. Antenna of adult longer than head and thorax. More than 8 segments. First two segments are different and rest all is same.	1. Antenna is shorter than thorax. Less than 6 segments (3 being usually present). Last segment is annulated.	1. Antenna has 3 segments.
2. Arista is absent	2. Arista, if present, then terminal. Maxillary palps are held stiffly forward. Abdomen with seven visible segments	2. Arista present on the dorsal side of antenna. Maxillary palps with one joint only. Abdomen fewer than seven visible segments.
3. Larva is aquatic, has well developed head. Mandibles bite horizontally	3. Larva has retractable head. Mandibles bite vertically	3. Larva has vestigial head.
4. Pupa is Obtectate type	4. Obtectate type	4. Coarcotate
5.Mandibles and first pair of maxillae are present	5. Present	5. Absent.
Families: 1.Ceratopognidae (biting midges) 2.Simuliidae (black flies) 3.Psychodidae (sand fly) 4.Culicidae (mosquitoes)	Family: Tabanidae (horse fly)	**Series:** 1.Aschiza and 2.Schizopora 3. Pupipara **1.Aschiza:** Not of veterinary importance **2. Schizopora** **Sections**: 1.**Acalypterae** Family: Gastrophilidae 2**.Calypterae** Families: i. Muscidae (house flies and stable flies) ii Calliphoridae (blow flies) iii. Oestridae (bot flies) iv. Glossinidae (tse-tse fly) **3.Pupipara** Family : Hippoboscidae

Suborder: Nematocera

Family: Culicidae

1. Family consists of mosquitoes with small spherical head and long legs.
2. Antenna has 14-15 segments.
3. Plumose male and pilose female.
4. Proboscis is long and slender.
5. Abdomen is elongate.
6. Thorax is wedge shaped.
7. Wings are long, narrow and folded flat over abdomen during rest and bears leaf like scales along the margins and on the veins.

Family: **Culicidae**

Division into Culicine and Anopheline species.

Life cycle

1. **Eggs** are laid in water/floating vegetable matter. They are laid in form of masses/ raft in case of Culex (anterior side towards water). Aedes and anopheles lay eggs singly. Anopheline egg is boat shaped and provided with the structure called float.
2. **Larvae**
    - Well developed head, distinct thorax and abdomen. Head bears eyes, antenna and several hairs.
    - Mouth parts are masticatory and surrounded by brushes which produce currents in water ultimately bringing food material near to mouth.
    - Unsegmented thorax bears feathered hairs and abdomen is segmented and haired.
    - The stigmata from which trachea passes through cuticle are situated on the fused 8th and 9th segment of abdomen. 10th segment bears hairs, gills and paddles.

Special feature.

- Anopheles has palmate hairs. It helps in tilting larva parallel to water surface.
- Culicine larvae have siphon tube at dorsal aspect of 8th and 9th abdominal segment and surround the stigmata. At the apex of the tube it has chitinous wall. (It hangs down in the water).
- In Aedes, anal gills are well developed. They feed on the bottom of water.

Larva molts four times to give rise to Pupa.

3. **Pupa**
    - Has rounded body, head, thorax and abdomen
    - Through its delicate cuticle, wings and appendages of adult can be seen.
    - From dorsal aspect of thorax, attach to lateral stigmata there arise a pair of tubes or respiratory trumpets through which it breathes.
    - Pupa is not quiet as active like larva.
    - Abdomen has 8 segments with terminal pair of pedals or fins.

Anopheline pupa- respiratory trumpet is short and broad.

Culex Pupa - Long and tubular respiratory trumpets.

4. **Adults.**

	Anopheles	Culex
Female	Palps = proboscis Antenna is pilose	Palps are very short Antenna is pilose
Male	Palps = proboscis but palps are clubbed Antenna is plumose	Proboscis = palps Antenna is plumose
	It rests with the abdomen directed away from resting surface i.e proboscis and abdomen are in the straight line	Rests with the abdomen pointing towards the resting surface i.e. proboscis and abdomen are at an angle and gives a humped back appearance.

Biology

- Development time differs according to species. Cold period may extend larval stage. Usually 7-16 days are required for the development.
- Great variation in seasonal prevalence of different species mainly due to temperature. Most species are seen in warm climate.
- Some species are seen in winter only. If the female is too late to lay its eggs, it may go hibernate to next season.
- Humidity also plays an important role.
- Adult fly may travel fair distance especially Anopheles. It can travel several kilometers from breeding grounds
- They can be transported in vehicles and readily enter buildings.
- Females are blood suckers. They require blood to lay eggs. Females are attracted towards warmth radiating from the host. They are active in night and attracted by light.

Disease transmission

1. Mosquitoes can be of a great nuisance and their bites cause painful reactions.
2. The definitive hosts of species of the protozoan Genus Plasmodium, which causes *Human malaria* are various species of *Anopheles* and those of the species of Plasmodium which cause B*ird malaria* are *Culex, Theoboldia and Aedes*.
3. Various Culicine and Anopheline species are intermediate hosts for filarid nematodes.e.g.
   - *Wuchereria bancrofti* → Culex, Aedes, Mansonia
   - *Brugia malayi* → Mansonia, anopheles.
   - *Dirofilaria immitis* → Anopheles, Culex, Mansonia,
4. Spirochaete: *Borrelia anserina* is transmitted to fowl by Aedes.
5. Viruses
   Arbovirus
   -Rift valley fever of man
   - Yellow fever transmitted by *Aedes aegypti*
   - Eastern and western encephalitis
   - St. Louis and Japanese ß encephalitis of man.

Both encephalitis is transmitted by species of Aedes and Culex
- Dengue fever in man is transmitted by *Aedes aegypti*
- La crosse (Californian encephalitis) *Aedes triseriatus*

Distinguishing characters of Anopheline and Culicine mosquitoes

	Anopheline	Culicine
Eggs	Laid singly, boat shaped with paired lateral floats	Laid in rafts or singly
Larvae	-No siphon tube -Rests parallel to water surface and feeds there. -Palmate hairs on dorsal surface of abdomen	-Well developed siphon tube -Hangs head down from water surface and feeds below it -Palmate hairs are absent.
Pupa	Breathing trumpet short and broad in lateral view.	Breathing trumpet long and narrow in lateral view.
Adults	-Rests with abdomen directed away from resting surface i.e. proboscis and abdomen in a straight line -Female palps as long as proboscis, male palps are clubbed. -Scutellum evenly curved	-Rest with abdomen pointing towards resting surface. Proboscis and abdomen at an angle, imparting a humped back appearance -female palps are very short. male palps as long as proboscis, not clubbed. -Scutellum trilobed.
Example	*Anopheles maculipennis* *Anopheles gambiae*	*Culex pipiens* *Aedes aegypti*

CONTROL

Measures to taken against larval stages and adults.

1. **Measures against larvae**
   - Biological control by larvivorous fish (mainly Fundulius, Gambusia sp.)
   - Larvicidal compounds during breeding season e.g Paris green+kerosene, pyrethrums, organophosphorus compounds (fenthion, difenphos), organochlorides (dieldrin, chlordane etc)
   - Abolishment of breeding places of mosquitoes.
   - Reclamation of swamp areas for agricultural and other uses.
2. **Measures against the adult stages.**
   - residual spraying of the indoor surfaces of houses for malaria control
   - various insecticides used for the mosquito are:

   i. **Chlorinated hydrocarbons e.g.**

   -DDT(dichlorodiphenyltrichloroethane) – excellent residual insecticide but it persists in the environment and tends to concentrate in the fat of the animals in

food chains. Its use is banned in certain countries. Apart from food chain toxicity, mosquitoes develop a high degree of resistance to DDT.

- HCH (hexachlorocyclohexane:lindane) @ 0.25-0.30g/m^2– its use is banned in certain countries.
- Dieldrin

ii **Organophosphorus compounds e.g.**

- Malathion (5% dust , 25% wettable powder, residual spray @ 2g/m^2)
- Crotoxyphos

Ciovap(1%) and ciodrin(3%dust)

Others like dichlorovos, fenitrothion can also be used.

iii **Carbamates** e.g

- carbaryl@ 2g/m^2

iv Pyrethrins and Pyrethroids

They are used as sprays or mists e.g permethrin, cypermethrin, fanvalerate and decamethrin serve as contact insecticides with advantages over others in that they are more toxic for insects, more resistant to light and more stable.

Prevention against mosquitoes

1. mosquito proof buildings
- tightly screened doors and windows
- White walls.
2. Use of protective clothing.
3. use of mosquito repellents e.g indalone, benzyl benzoate, oil of citronella)

Lecture 6

Family: Ceratopognidae (biting midges/punkies/no-see-ums/Gnats)

Characters:

1. Mandibles act like scissors.
2. Proboscis is short and adapted for sucking blood.
3. Thorax is humped over head.
4. Antenna is pilose in females and plumose in males.
5. Wings are folded flat over the abdomen, spotted, have hairs and no scales.

Life cycle:

Eggs are laid in water

Larva is aquatic, whitish in colour.

- Consists of head, thorax (3 segments) and abdomen (9 segments).
- Terminal segment bears a few locomotory spines. At the posterior end, there are 3 retractile anal gills.

Pupa

- Brown, inactive and obtectate.
- 2 long respiratory trumpets on the sides of mesothorax
- 9 abdominal segments which end in 2 spines with which pupa anchors itself
- Whole pupa is covered with spines and tubercles.

Members of Ceratopognidae are predatory in adult stage.

Genus: Culicoides has veterinary importance.

- 1-3 mm long.
- Females attack man and animals and suck blood.
- Bites case inflammatory responses.

Disease transmission.

1. Act as intermediate host for filariid nematodes

Nematode	Intermediate host
Dipetalonema perstans	*Culicoides grahami, C. austeni.*
Dipetalonema streptocara	*Culicoides grahami,*
Onchocerca cervicalis	*Culicoides nubeculosus*
Onchocerca gibsoni	*C. pungens*
Mansonella ozzardi	*C. furens*

2. Viruses

- Blue tongue
- African horse sickness

3.Allergic dermatitis of horses

Queensland itch/sweet itch/sweat itch/summer dermatitis *C. robertsii*

Loss of hair on dorsal part of the body accompanied by itching, pruritus

Treatment : antihistaminic and application of insecticides like dichlorovos and benzyl benzoate

Control

- Use of insecticides, screens with repellants.
- Control of breeding grounds(ditching, dyking, pumping water)
- 5% dieldrin by aircraft was found to be effective.
- Parathion, malathion and bayer are highly effective.

Family: Simuliidae (black flies/ buffalo Gnats)
Characters:

- Thorax is humped over the head.
- Piercing proboscis is short
- Antennae are long, have 11 segments and are not pilose or plumose.
- Wings are broad and have no spots, scales or hairs.
- Presence of bristles on the thick anterior veins.
- Body is covered with short golden or silvery hairs.
- Palps have 5 segments.

Life cycle
Eggs are laid on stones or plants just below the surface of the water in running streams. The female inserts her ovipositor into the water to lay and deposit several hundred eggs at a time**.**

↓
After 4-12 days
↓
Larva

- Cylindrical
- carnivorous
- mouth parts have a pair of brush like organs
- posteriorly there is a presence of sucker like organ with hooks for attachment and movement.
- Presence of arm like appendage called proleg with circlet of hooks near anterior surface ventrally (for movement)

↓
Moulting for six times
↓

Pupa (triangular cocoon)
Obtectate
One dorsal and one ventral respiratoty tube, branches of which float out of the cocoon
Pathogenesis and clinical symptoms
1. Cause great annoyance and irritation.

- The fly is troublesome in mainly in the warm countries but can be found in all parts of the world.
- Associated with petechial hemorrhages particularly in the fine areas of skin and there is oedema of throat and abdomen.

Simulium erythrocephalum
Swarms of flies keep cattle from grazing, bite on legs and abdomen, head and ears. Bites give rise to vesicles and papules especially on teats.
In poultry, there is loss of blood resulting in anaemia.
Flies are active in morning and evening and not in the day.

Simulium indicum/potu fly	Found in Himalayan regions
S. columbaczense	Near River Danube. Killed 20000 horses.
S. pecuarum/Southern buffalo Gnat	Mississippi valley (cattle and mules are affected)

S. arcticum	In Canada (loss in milk and beef production)

2. Act as vectors for the transmission of Viruses

Eastern equine encephalitis
Vesicular stomatitis.

3. Transmission of Protozoan diseases

Protozoa	fly
Leucocytozoon anatis	*Simulium venustum*
L. smithi	
L. caulleryi	
Haemoproteus nettionis	*Simulium rugglesi*

4. Act as intermediate host for various filariid nematode infections

Nematode	Fly
Onchocerca gutturosa (cattle)	*S. ornatum*
Onchocerca volvulus (man)	*S. damnosum (Africa)*
Cause of river blindness	*S metallicum*
	S ochraceum
	S callidum(Mexico)

Control

- As the adults can fly 3-5 km, the control is difficult.
- Use of repellents and medicated screens.
- Protective clothing.
- Dust baths (e.g.0.5% lindane)

Family: Psychodidae (sand fly/owl midges)

Characters:

- Rarely over 5mm long.
- Body and wings are hairy.
- Long legs.
- Wings are lancet shaped, have parallel wing venation and are held roof like over the abdomen.
- Antennae have 16 segments, beaded and thickly covered with hairs.
- Palps are recurved and hairy. Flies are active during the night only and some species are blood suckers.

Life cycle

Eggs are laid in dark and moist places (40-80 eggs at a time) e.g roof crevices and between stones**.** At around 15°C, 6 weeks are required for the completion of the life cycle.

Larvae

Resembles small caterpillar and feed on the faeces of lizards, bats, and other animals and on dried leaves. Flies are active at night only and hide during the day in dark corners.

Disease transmission

Disease	fly
1.Cutaneous Leishmaniasis	*Phlebotomus papatasii*
caused by *Leishmania tropica*	*Phlebotomus sergenti, P. major*
2.South American Cutaneous	*Phlebotomus* species.

Leishmaniasis (Espundia) by
Leishmania braziliense

3. Kala-azar / Visceral leishmaniasis *Leishmania donovani*	*P. argentipes* (India) *P. chinensis* (China) *P.papatasii* (Mediterranean) *P. mongolensis* (china)
4. Carrion's disease/Oroya fever by *Bartonella bacilliformis*	*P. verrucarum* *P.columbianum*

Control

- Removal of dense vegetation.
- Use of insecticides in the interior of humans and animal dwellings.
- Rest same as mosquito control.

Lecture 7.

Suborder: Brachycera

Family: Tabanidae (horse flies/breeze flies)

- Large, Robust flies with powerful wings and large eyes.
- Eyes are holoptic in males and separated by a narrow groove in females (dichoptic) and project posteriorly beyond the lateral margins of the thorax.
- Labella with pseudotracheal membrane.
- The antennae have two short basal segments and third which is large and is usually ringed.

Genus: Tabanus, Chrysops, Haematopota

	Tabanus	**Haematopota**	**Chrysops**
Proboscis	Proboscis short, soft and hangs down	Short and hangs	Long, soft and hangs down.
Antennae	First two segments are small, third segment has tooth like projection on its basal part and 4 annulations.	First segment is large and second is narrower, terminal segment has 3 annulations	1&2 segments long and 3rd segment has 4 annulations
Wings	Clear wings and hold their wings horizontal at rest.	Mottling in wings	Dark band passing from anterior to posterior border of wings. The wings are held divergent at rest
Abdomen	Brown longitudinal strips on the abdomen	Slender abdomen	Ovoid abdomen, yellow and dark strips
Distribution	worldwide	Africa	World wide

Life cycle

Eggs are laid near water, usually on leaves of plants.

(500-600 eggs are laid, about 2mm long, light in color but turn dark in color after a while.)

↓

After 4-7 days

↓

Larvae drop in water, or on mud

- inconsipicuous head
- maggot like with 11 segments
- each segment with 8 fleshy tubercles
- larvae is carnivorous and mouth parts are masticatory
- 3 jointed antennae
- Large lateral trachea opens on penultimate segment which bears a retractable siphon tube (on 10th segment).
- Carnivorous in nature.

↓

2-3 months

Pupa

- Brown and subcylindrical,
- movable abdominal segments and appendages are distinguishable

↓

Imago

↓

Adult

Whole life cycle = 4-5 months.

Biology

Seen in summer and sultry days, are very fond of sunlight.

Females are blood suckers of large animals such as horse and cattle and feed on underside of abdomen, around navel, neck, withers, bite the animal and cause wounds.

They bite at number of places several times before they replete.

Flies feed every 3 days and rest on underside of leaves, stones or trees.

Pathogenesis and disease transmission

1. These flies cause wounds and weal formation in animals and cause restlessness in animals.
2. These flies as well as Muscidae which come after them Act as mechanical carriers of Anthrax, Equine infectious anemia and Anaplasmosis.
3.

Disease	Vector
• *Pasteurella tularensis*	*Chrysops discalis*
• Loa loa	*C dimidiata* and *C silacea*

4. Various species act as mechanical vectors of
   - *Trypanosoma evansi* (Cause of Surra in dogs and equines) ,
   - *Trypanosoma equinum* (Mal de Caderas of horses),
   - *T. simiae* (pigs),
   - *T gambiense, T. rhodesiense* (Human African Trypanosomiasis) and
   - *T.vivax* and *T. brucei* (Nagana in cattle, sheep and equines).
   - *T. theleri* (cattle) transmitted by Tabanus and Haematopota.

Control

- Breeding places be destroyed by drainage.
- Pouring of kerosene in the water as they dip their bodies into water.
- Residual sprays for inside walls of animal houses Chlorinated hydrocarbons may be used. Organophosphorus compounds like Malathin and crotoxyphos can be used.
- Measures similar to those for the mosquito control are applicable.

Sub order: Cyclorrhapha
Series: Schizopora
Section: Acalyptera

Features

-Squamae is small and does not conceal the halteres.
-Thorax without distinct transverse sutures.

Family Gastrophilidae
-Hairy flies. Mouth parts are reduced and functionless.
Genus Gastrophilus in equines and **Cobboldi** in elephants.
Genus : **Gastrophilus**
-Larvae (L3) of this genus are parasites of equines and are called **bots.** Are also rarely found in dogs, pigs, birds and man.
-Adult flies are brown in color, hairy and look like bees.
-18 mm long
-Dark irregular transverse bands run across wings.
Life cycle
Adult flies occur during the later half of summer and live only for 3 weeks.
Female fly, with its ovipositor darts at the host to glue an egg to hair. Large number of eggs may be laid in succession.
Gastrophilus intestinalis lays its eggs around fetlock, high in the legs, scapular region.
G nasalis → (intermandibular region)
G haemorrhoidalis and *G inermis*→ (on hairs around mouth and on cheeks).

Eggs
G pecorum & *G haemorrhoidalis* (dark in colour, pointed at the attached end and operculated at the other)
Other species → pale yellow, elongate, operculate at one end and pointed at the other
↓
Hatch in 5-10 days (eggs near mouth hatch earlier, others hatch in response to an increase in temperature on licking)
↓
Larvae penetrate the mucosa of mouth
G intestinalis & *G haemorrhoidalis* penetrate the mucosa of tongue.
G pecorum and *G inermis* penetrate the mucosa of cheek.
G haemorrhoidalis may even pierce the skin of face and wander in it to the mouth leaving consipicous tracts. In man, these cause cutaneous swellings at the point of penetration of larva. L1 can occasionally be found in forearm, human stomach and may even migrate to thoracic and abdominal organs of humans.
↓
After 21-28 days, Larvae (L3) is found attached to the cardiac portion of stomach, has red colour (e.g. *Gastrophilus intestinalis*)
Pylorus and duodenum, pale yellow in colour (*G. nasalis*)
Pharynx and upper part of oesophagus and fundus region of stomach, blood red in color (*G pecorum*)
Pharynx and settle in stomach, red in colour (*G haemorrhoidalis*)
The larva remains in the host for 10-12 months and reach the third stage when they measure up to 20 mm in length, are brown in colour and have dense spines on anterior border of segments. There is a presence of distinct mouth hooks on the first segment. The larvae then pass out through the intestine and usually leave the host usually in late

autumn or in spring. The larvae of *G haemorrhoidalis* again attach themselves for a few days in the rectum

↓

After 3-5 weeks

↓

Pupa (equine faeces)

↓

Adult fly

Pathogenesis

- Fly cause annoyance in animals.
- Migrating larvae in superficial layers of buccal epithelium cause inflammation.
- *G pecorum* cause stomatitis.
- In pharynx, oesophagus, duodenum and stomach there is inflammatory reaction and ring like thickening around larvae.
- There is ulceration of oesophageal region of stomach by larvae of *G. intestinalis*.
- Mostly larvae are found in the non glandular region of stomach and causes extensive ulceration. There may be abscess formation, rupture of stomach can occur with peritonitis or there may be stricture of the pylorus with tissue reaction.
- Larvae which attach to rectum cause irritation and inflammation (*G. haemorrhoidalis)*

Diagnosis

- Eggs at the sites where they are deposited
- Larvae can be diagnosed by endoscopy.

Treatment

- Carbon disulphide via mouth tube 2-5 ml/kg body weight after 18 hour fast.
- Piperazine + phenothiazine @ 110mg/kg body weight.(effective against Gastrophilus larvae, ascarids and strongyles)
- Trichlorophon- 39.5mg/kg via stomach tube or drench. (effective against Gastrophilus larvae, ascarids and Oxyuris)
- 2g thiabendazole+18g trichlorophon/50kg body weight in feed, or by stomach tube or drench. effective against Gastrophilus larvae,Strongyles)
- Dichlorovos (organophosphorus) @ 26-52mg/kg in feed (effective against Gastrophilus larvae, ascarids and Strongyles).
- Butonate @45 mg/kg by stomach tube (for Gastrophilus and Parascaris)

Treatment should be given twice a year.

Prophyllaxis.

- Frequent grooming to remove the eggs.
- Eggs can be hatched by sponging chest, forelegs and chin with water at (40-43°C) containing insecticide to kill hatched larvae.
- Use of fly evaders like masks and sheets.
- Use of fly traps.

Genus: **Cobboldia**

Cobboldia elephants: found in stomach of elephants.

LECTURE 8

Genus : Stomoxys

Species : ***Stomoxys calcitrans*** (stable fly)

- Prominent proboscis and small labella (piercing and sucking)
- M1+2 curves forward and R5 is open.
- Thorax is grey, 4 longitudinal dark strips, lateral pairs are narrow and not reach upto the scutum.
- Abdomen is shorter, broader than Musca and there are 3 dark spots on each of 2nd and 3rd segment.

Life cycle

Eggs

- Eggs are laid on horse manure and decaying vegetable matter like straw and hay (moist/contaminated with urine)
- 25 to 50 eggs are laid in one batch and total 800 eggs are laid.
- 1mm
- Dirty white to yellow in color.
- Longitudinal groove on one side.

↓

1-4 days

↓

Larvae

Stigmal plates are wide apart and 3 S- shaped winding slits are present.

↓

14-24 days

↓

Pupa is formed on drier parts of manure, bedding material

↓

After 6-9 days

↓

Fly (oviposition after 9 days)

Life cycle – 30 days.

Habits and significance

- Live for about a month and seen in autumn and summer.
- Preferable seen in strong light, swift fliers and do not travel long distances.
- Both male and females are Blood suckers.

Disease transmission.

1. Cause fly worry
2. Various species act as mechanical vectors of
   - *Trypanosoma evansi* (Cause of Surra in dogs and equines) ,
   - *Trypanosoma equinum* (Mal de Caderas of horses),
   - *T gambiense, T. rhodesiense* (Human African Trypanosomiasis) and

- *T. vivax* and *T. brucei* (Nagana in cattle, sheep and equines).

3. Intermediate host for *Habronema majus*.
4. Equine infectious anemia.
5. Anthrax

Control

Destruction of breeding places e.g. moist bedding, faeces from stables and yards, food wastes from feeding troughs and vegeatable refuse etc..
Use of insecticides.

Genus : Haematobia/ Lyperosia

- Pests of cattle
- Flies are the smallest of the blood sucking muscids, 4mm in length.
- Face is silvery gray.
- Thorax is silvery grey medially and dark laterally with 2 well defined stripes.
- Palps are yellowish and as lons as proboscis.
- Aristae are haired on the dorsal surface only.
- Piercing and sucking mouth parts.

Haematobia exigua (buffalo fly)

Transmission.

T evansi (Surra)
Intermediate host for *Habronema majus*.

Haematobia irritans (horn fly)

- On the base of horns and also on the back, shoulders, belly of cattle.
- Fly rarely leaves the host and leaves it only to pass to another host or to lay eggs when animal defaecates.
- Blood sucker and heavy burdens cause injury and irritation in animals.
- Animal may develop sores and wounds and attract other flies.
- Cause great losses in production and performance.

Transmission

Stephanofilaria stilesi (skin).

Control

Regular spraying with insecticides.
Self application with dust bags/ back rubbers.
Regular removal of dung and faeces.

Family : Glossinidae

Genus: Glossina/ tsetse fly

- Blood sucking flies
- Yellowish to dark brown 6-13.5mm
- Wings are held over the back, overlapping almost scissor like.
- Thorax is greenish ground in color and has inconspicuous strips or spots.
- Abdomen is light to dark brown with 6 segments visible from dorsal aspect.
- Proboscis is long, held horizontal and unsheathed in long palps which are of uniform thickness throughout.

- Antenna has large, elongate 3rd antennal segment which ends in blunt, forwardly directed point.
- Arista bears 17-29 dorsal branching hairs.

Life cycle

Female produces one larva at a time. The larva grows in the uterus of the female, its mouth being attached to a teat from which milk is obtained for nourishment; posterior extremity which bears the stigmal plates near the vulva.

Gestation period is 10 days.

The female can produce 8-10, sometimes 12 larva in a life. Larva is oval 7mm long→wrigglesinto soil 2cm deep→60-90minutes→pupa. Larva has two respiratory lobes at its posterior end, each perforated by about 500 spiracular openings.

Pupa

6-7mm long. Lasts for 35 days. In summers-31days and in winters-92 days.

Habits

Found mainly in central parts of Africa. Their presence depends on altitude, moisture, vegetation and presence of hosts. Humidity and temperature and presence of shade have an important bearing on the life of the fly.

Blood sucking flies.

Glossina palpalis → feed on blood of warm blooded animals.

G morsitans→ an any large mammal or bird or big game.

G palpidipes → big game, cattle, wart hogs.

Fly flies low near the ground and hunt by sight. They are susceptible to the effects of light and shade and are attracted especially by moving objects.

Fly may reach new sites after leaving its host or giving up the chase.

The flies feed about every 3 days, depending on the temperature and humidity.

As the fly engorges, its abdomen becomes distended. Finally the fly leaves its host and settles on the underside of a leaf or on a log or on the bark of a tree to rest.

Control

1 catching and trapping of fly (by use of and nets)

2. bush clearing.

3. destruction of big game.

4. fly screens, repellents.

5. Insecticides Organochlorines: DDT, Dieldrin
   By use of helicopters, ground spray, endosulphan, isobenzan.

6.use of sterile insects, physiological control.

Disease transmission

Trypanosomiasis

Family : Calliphoridae

- Larvae are saprophagous, flesh feeders or parasites of other arthropods.
- These are bristly flies with the presence of a row of bristles on the hypopleuron
- Two sub families: Calliphorinae (blow flies, blue/green) and sacrophaginae (flesh flies-grey longitudinal striped thorax with a check board marking in abdomen)

Sub family: **Calliphorinae**

Genus: Lucilia/ Green bottle flies

L cuprina (Australia, S.Africa),
L sericata(Britain)

- Eyes are brownish red.
- Bright metallic color (bright green/bronze colour)
- Green bottle or copper bottle flies.

Genus Calliphora/Blue bottle flies
Have metallic blue sheen.
C erythrocephala
12mm
Buzzes very loudly.
Eyes are red
C australis, C. augur, C fallax, C vomitoria.

Genus: Phormia/black blow flies.
P regina
Thorax is black with metallic blue green sheen.
P terraenovae
(in Britain and Canada)

Genus : Chrysomia/steel blue blow fly
C chloropyga.
C albiceps
C rufifacies
C bezziana/old screw worm
C micropogon

Calliphorine myiasis of sheep.

Known as **Strike** and caused by the larvae of various species belonging to the genera Lucilia, Calliphora, Phormia and Chrysomia

Life cycle
Eggs are laid in carcasses, wounds or soiled wool, being attracted by the odour of decomposing matter. 1000-3000 eggs are laid and 50-150 in one batch. →after 8 hours to 3 days→larvae→after 2 ecdysis (2-19 days) →maggots grayish white/ pale yellow. Anterior end has a pair of oral hooks and on posterior end the posterior stigmal plates are situated. The stigmatic openings consist of 3 long slender slits, more or less parallel to each other on the spiracle. → pupa in ground or dry parts of carcass→after 3-7 days or longer in winter→fly emerges (ptilinal suture)
Total life cycle is completed in 7 days.
Epidemiology
Governed by:

1. Prevalence of flies.
2. Susceptibility of the sheep.

Prevalence of flies.
More in late spring, early summer and early autumn

Abundance and suitability of food.

Flies

1. Primary flies are those which initiate strike by laying eggs on living sheep e.g *Lucilia sericata, L. cuprina, Calliphora* sp.
2. Secondary flies are those which usually lay their eggs on sheep already struck. Larvae extend the injury done by the larvae of primary flies e.g *Chrysomia rufifacies, C. micropogon, Microcalliphora varipes.*
3. tertiary flies are those which come last of all, the larvae of which do little further damage. E.g. *Musca domestica, Fannia australis, Peronia rostrata*. They occur both on living animals and carrion.

Primary flies (occur during autolytic and bacterial decomposition satge) →secondary flies (liquefaction) →tertiary flies come when carcass begins to dry out.

Types of Strike.

1. Breech strike/crutch strike/ tail strike

-area is frequently soiled with diarrhea and by urine.
-narrowing of breech
-wrinkling of skin.

2. Pizzle strike(in rams)

-Sheath has narrow opening, so it soils the wool of this region.

3.Poll strike.

-deep head folds
-horns lying close to head→cause seaty condition→attract blow flies.

4. Wound strike

-in wounded areas
-castration
-tail docking

5. Body strike

Myiasis of the dorsal region of the body during wet weather cause fleece rot and dermatitis

Pathogenesis

In the strike, wound and bacterial activity is important. In wool that is kept moist by prolonged wet conditions, the yolk, wool scales and skin scales become pasty and form a suitable media for bacteria. The odour produced by decomposition attracts flies and exudative reaction provides food for the larvae. When the skin becomes soiled by urine and faeces, it causes inflammation and bacteria aggravate the condition and attract flies. The larvae of primary blow flies initiate the attack. Proteolytic enzymes liquefy and digest the tissues and secondary flies are then able to produce the lesions. Large wounds are produced and larvae of secondary flies form deep tunnels in the skin. The central portion of the lesion may heal with thick scab, while the action of the larvae extends outwards. The smell again attract other flies. The lesion and parasites are irriating and animal does not feed properly and becomes poor and weak. The cause of death is toxaemia and septicaemia. Decreased value of fleece, reduced meat production, death of animals and labour costs incurred in the surveillance and handling of blow fly strike cause economic losses.

Clinical signs

- Sheep stands with its head down and does not feed.
- Attempts to bite the affected area.
- When the lesions are located on tail or buttocks, then animal will stamp or jerk the hind legs and wag the tail.
- Discoloured, grayish brown, moist wool with an evil odor.
- The maggots may be found.
- Inflamed ragged wound with exudates with foul smell.
- There is malnutrition, loss of milk, elevation of temperature and even death.
- Lightning strike- sheep dies within few days. Bacteria cause septicaemia and toxaemia.

Diagnosis.

- clinical symptoms
- finding larvae in the wound.

Treatment

- kill the larvae in the lesion, promote healing and prevent reinfestation.
- Clipping of hair/wool and removal of larvae which should be killed especially larvae of primary flies (L cuprina, L sericata)
- Dressing of wound.
- Dipping solution (2% diazinon, or 3% coumaphos)

Prophyllaxis

1. treatment of the sheep to render them less attractive to the flies
2. treatment of sheep with insecticides.
3. genetic manipulation approach.
4. carcass disposal.

1.**Treatment of the sheep to render them less attractive to the flies**

a) Selective breeding

narrow breech and folds of skin predispose the animals to strike. So, breed sheep with plain breeches.

b) Surgical removal of the breech folds.

By Mulesing operation. The crescentric area on either side of the urogenital area of skin of merino sheep is removed.

Application of 40% phenol on preputial area after shearing cause dry encrustation this heals leaving scar which eliminates the skin folds.

c) Docking.

Docking of tail at/ behind 4th coccygeal vertebra. Long tail tends to flatten the skin folds on either side of the vulva. Longer tail is also held away from the body when the ewe urinates.

d) Crutching.

Clipping of wool from around the tail and breech. Machine crutching is more effective.

2.Treatment of sheep with insecticides.

- Chlorinated hydrocarbons-DDT, HCH, Dieldrin (0.05%)

- Organophosphates (bromophos, chlorfenvinphos, coumaphos, 0.05%, diazinon, 0.04%, Butacarb. 0.05%)
- Synthetic pyrethroids
-Avermectins
-Insecticides with acrylic emulsions.

c) Genetic manipulation approach
Females are blind and males are partially sterile and these transmit the genes for blindness to female offsprings.

d) Carcass disposal.

Screw worms
Cause Myiasis in man e.g *Callitroga hominivorax, C. macellaria and Chrysomyia bezziana* (old screw worm)

Life cycle
150-500eggs on the edge of the wound →after 10-12 hours→larva→3-6days→pupa→3days to several months

Pathogenesis
Eggs are laid near the wound→cause liquification→formation of wound, foul smelling exudates oozes out→secondary bacterial invasion→death

Sub family: Sarcophaginae

- Flesh flies
- Medium to large size.
- Light and dark grey in colour.
- Thorax has 3 longitudinal stripes.
- Dorsum of the abdomen has dark spots or is chequered dark and grey.
- Larviparous in nature.

Genus : Sarcophaga
These lay their larvae in decomposing flesh, wounds, ulcers etc.

Lecture 9
Family : Oestridae

Genera :
Oestrus
Hypoderma

- Adults are hairy flies with rudimentary mouth parts and do not feed.
- Lay their eggs on animals. Larvae are parasitic maggots and consist of 12 segments of which first two are fused together. Oral hooks are present but there is no head. The posterior stigmata open through semicircular plates which may be retractible. Larva moults two times and pupate on ground. They feed on body fluids of host or on exudates surrounding them.

Species: ***Oestrus ovis/Sheep nasal fly*.**

- Dark grey colour with small black spots prominent on thorax and is covered with light brown hair.
- Mostly seen from spring to autumn.
- Larvae are found in the nasal sinuses or cavities in sheep. Rarely in goats and camel.
- *O. ovis* can deposit its eggs on eyes, nostrils and even on lips of man and cause serious trouble.

Life cycle

Fly deposit their young larvae around nostrils

↓

They crawl upwards. Sometimes they enter cavities with small openings, like those of turbinate bones or branch of frontal sinuses and are not able to get out and so die there. First instar may remain in the nasal passage for 2 weeks to 9 months during the cold months. The second instar passes into the frontal sinuses develop rapidly leaving sheep 25 days after infection. Fully grown larvae crawl out and pupate on ground for 3-6 weeks and fly emerges.

Larvae of *Oestrus ovis*

- Dark transverse bands on dorsal aspect of segments.
- Taper anteriorly and flat posteriorly. Large black oral hooks are connected to cephalopharyngeal skeleton. Black stigmal plates are consipicuous at posterior end.
- Ventral surface has row of small spines.

Pathogenesis

- Adult fly causes Annoyance, restlessness, interference in feeding of animal.
- The animal shakes its head and press the nose against the ground and other sheep.
- The irritation of nasal mucosa by larvae causes mucous exudates.
- Nasal discharge and sneezing.

- Erosion of bones of skull may occur, there may be injury to brain and there may be signs like high stepping gait and incordination. These symptoms resemble those of *Gid* caused by *Coenurus cerebralis*, hence this condition is also called the *False Gid.*

Diagnosis

clinical symptoms

Treatment

-instillation of HCH in oil (1-4%) into the nostrils.
-55-88 mg/kg of a mixture of 2g Neguvon and 0.2g of asuntol.
-crufomate 35%(dip) or pour on 13.5%)
-Rafoxanide drench (7.5mg/kg)

Prophyllaxis

Use of narrow troughs with edge smeared with tar. Tar acts as repellent to flies.

Genus: Rhinoestrus

Obligate parasite of equines

Rhinoestrus purpurensis

In nasal sinus and larynx of horses and mules of Europe, Asia, Africa. Causes ophthalomyiasis of man.

Genus: Hypoderma

Larvae of ***Hypoderma bovis*** and ***Hypoderma lineatum*** are known as **Ox warbles**. Parasites of cattle, rarely of man and horse in northern hemisphere

H bovis	***H. lineatum***
15mm long	13 mm long
Thorax is greenish yellow	Thorax is yellowish white
L3 is 27-28mm	L3 is 25 mm
Northern cattle grub in North America	Common cattle grub/ heal fly
Lay eggs singly	Deposits a row of six or more on hairs
L1 is found in spinal cord and epidural fat	L1 in sub mucosal connective tissue of oesophagus.

The flies are hairy and no functional mouth parts.

Life cycle

Flies occur in summer (may- July)

↓

Attack cattle to lay eggs. Limited flight not exceeding 5 kms. Eggs are fixed on hairs of legs (0.1mm) by means of terminal clasps. Fly lays around 100 eggs or more

↓

4 days

↓

Larvae crawl down the hair to the skin and penetrates the skin

↓

Wanders in the subcutaneous tissue, up the leg and towards diaphragm and increase in size. L1 may be found in different parts of the body, especially intermuscular connective tissue, surface of internal organs The winter resting sites of L1 is the submucosal connective tissue of the oesophageal wall in *H lineatum* and spinal cord and epidural fat for *H bovis*. 12-16 mm in length (found in autumn)

↓

January-February

L2 is formed and travel towards the dorsal aspect of the body and reach subcutaneous tissue of the back (spring resting phase).Swelling of 3cm diameter and skin gets perforated and larva lies with its posterior stigmal plate directed towards the pore. This is known as warble stage larvae.

↓

Formation of L3

H bovis (27-28mm)

H lineatum (25mm)

L3 bears a number of flat tubercles. Spines present on all segments except last in *H lineatum* and in all except last 2 in *H bovis*.

↓

Pupa falls on the ground. Pupal case is black.

↓

35-36 days

↓

Adult fly emerges.

Pathogenesis

- Calves and young cattle are affected more. Adults possibly develop immunity.
- There is gadding and aimless walking to avoid the flies. Animal becomes apprehensive and disturbed and can injure itself and even die.
- There is reduced weight gain and milk yield (10-15% and even 50%)
- Carcass and hide depreciation are important aspects of Hypoderma infection..
- There is discolouration of meat along the tracks of larvae and meat becomes gelatinous. Also known as *Licked beef.*
- Local abscess formation.
- In horses, aberrant migration of L1 into the cranial cavity lead to neurological disorder. May occur on back as well.
- Death of larvae in the wall of oesophagus result in oesophagitis causing boat and in spinal cord may result in temporary or permanent paralysis
- In man, abdominal pain, sub-dermal migratory tracts.

Clinical symptoms.

- Poor growth and reduced milk yield
- Swellings can be felt and seen.
- Larvae lies in the cyst and contains yellow purulent fluid.

Diagnosis

Presence of larva on skin of the back.

Eggs on hair.

Treatment
1) Mechanical removal of larvae. Rupture of larvae may lead to localized inflammation, abscess formation and generalized anaphylaxis
2) Insecticides
-derris/rotenone
Systemic insecticides
Oral, parental or dermal or pour ons
Organophosphorus compounds (crufomate, fenthion, phospmet)
Ivermectin @ 0.2 mg/kg is 99% effective.

Section : Pupipara

- Live on blood of mammals or birds.
- Body is broad and dorsoventrally flattened.
- Abdomen is indistinctly segmented and wall is soft and leather like.
- Antenna has one joint and lies in pit on forehead.
- Feet are provided with claws
- Female gives birth to larva which is ready to pupate.

Family : Hippoboscidae
Known as Forest flies and attack horse and cattle.
Genus : Hippobosca

Species: ***H equina***
H rufipes
H maculates

- 1 cm long, reddish brown with pale yellow spots.
- One pair of wings, veins of which are crowded together towards the anterior border.
- Short thick palpi ensheath the tip of the slender proboscis, the main portion of which is withdrawn into head during rest.

Life cycle
Fly is larviparous which lays one larva at a time in sheltered spots where there is soil or humus.

↓
Larva (5×4mm)
↓
Immediately converted to subglobular Pupa
↓
Fly

Flies are most frequent in summers. Remain for long periods on the host. Cluster in perineal region, between hindlegs to the pubic region.

Transmission
Trypanosoma theileri cattle

Haemoproteus to anatid and other birds.

Control

Pyrethrins, Ciodrin, DDVP

Genus: Mallophaga

Melophagus ovinus/sheep ked.

- Wingless, hairy, leathery insect, 4-6mm long.
- Head is short, broad and not freely movable
- Thorax is brown and abdomen is grayish brown. Legs are strong, armed with stout claws.

Life cycle

Permanent ectoparasites. Female attaches its larva to the wool

↓

Larva is immobile and soon turns to chestnut brown pupa which is ovoid in shape with broad ends (3-4mm)

↓

19-23 days or more in winters (36 days)

↓

Adult

After 3-4 days copulation occurs. Female ked lives for 4-5 months on a sheep. Female produces 10-15 larvae. Spread from sheep to sheep occurs by contact. Most numerous in autumn and winter.

Pathogenesis

- Cause damage to the wool.
- Suck blood and cause anemia.
- Cause irritation due to which sheep scratch, bite and damage wool.
- Staining of wool by the faeces of ked.

Transmission

Trypanosoma melophagium.

Control

- Shearing of wool.
- OP insecticides (ronnel, diazinon 0.06%, ciodrin 1%)

Genus : ***Pseudolynchia canariensis***

- Dark brown fly, 6mm long.
- A pair of transparent tapering wings with venation reduced and concentrated at anterior end.
- Claws are strong and spurred. Lives on pigeons, wild birds.

Life cycle

Attach to feathers→suck blood→cause wounds especially in 2-3 weeks old. Female produces 4-5 youngs in life cycle of 43 days. Copulation occurs on host. Larvae are laid on dark crevices → pupate→23-31 days→adult

Transmission

Haemoproteus columbae

Control (organophosphorus compounds-coumaphos, carbaryl)

CLASS: ARACHNIDA

- Scorpions, spiders, ticks and mites.
- Differ in structure and function from insects.
- Antennae, wings, compound eyes and division of body into head, thorax and abdomen is absent.
- Mouth of arachnids is small and feed on tissue fluid of other animals.
- Carnivorous
- 1st and 2nd pair of appendages are modified to help in feeding
- 1st pair is called chelicerae and 2nd pair is called pedipalps and between these, a median toothed structure called the hypostome. They are borne on the gnathosoma, which consists of a plate called the capitulum and the mouth parts.
- Prosoma consists of chelicerae, pedipalps and 4 pairs of legs. The legs are borne on podosoma.
- These segmentation is not evident on ticks and mites as they have lost the external signs.
- Most of them show-

-anterior gnathosoma (capitulum) consisting of chelicerae, pedipalps, median hypostome (adapted for sucking blood or tissue fluids or holding to host)
-posterior fused called idiosoma consisting of opisthosoma and podosoma

- Mites absorb oxygen through cuticle.

Order : Acarina

- Species are hard and soft ticks, minute mites.
- Cause mange and inflict injury on farm animals.
- Mouth parts : chelicerae, pedipalps and median toothed hypostome. They are borne on gnathosoma which consist of plate called capitulum.
- Segmentation of the rest of the body is absent.

Life cycle

Egg→larva (3 pair of legs) →nymph (resemble adult but there are no sexual organs)
N1 (protonymph); N2 (deutonymph) and N3 (tritonymph) → adult.

There are 6 suborders (2 are not of economic importance)

1. **Ixodoidea** (these include hard and soft ticks)
   Family:
   - Argasidae (Argas, otobius, ornithodorus)
   - Ixodidae (Ixodes, boophilus, Hyalomma, Rhiphicephalus, amblyomma, Dermacentor)
2. **Trombidiformes**
   Family:
   Demodicidae (Demodex)
3. **Sarcoptiformes**
   Family:
   Sarcoptidae (Sarcoptes, Notoeders, Cnemidocoptes)
   Psoroptidae (Psoroptes, Chorioptes, Otodectes)
   Analgesidae (Megnina)

4. **Mesostigmata** (gamasid mites
   Family:
   Dermanyssidae (Dermanyssus, Ornithonyssus, Pneumonyssus)

Class Arachnida

↓

Order Acarina

Suborders

I Ixodoidea

Family

1. **Argasidae-** Genus- (Argas, Otobius**, Ornithodorus)**
2. **Ixodidae**

Genus
Ixodes, Boophilus, Hyalomma, Rhiphicephalus, Amblyomma, Dermacentor

II. Trombidiformes

Family: Democidae

Genus: Demodex

III. Sarcoptiformes
Family:

1. **Sarcoptidae**

Genus- Sarcoptes, Notoeders, Cnemidocoptes

2. **Psoroptidae**

Genus- Psoroptes, Chorioptes, Otodectes

3. **Analgesidae**

Genus- Megnina

IV. Mesostigmata
Family:
1.Dermanyssidae
Genus-
Dermanyssus
Ornithonyssus
Pneumonyssus

Suborder Mesostigmata

- Species of this suborder usually armed with brown or dark brown plates.
- Body has minute gnathosoma (bears mouth parts) and posterior idiosoma.
- Mesostigmata – single pair of stigmata are lateral and outside the coxae of leg.

Family : Dermanyssidae

Genus : Dermanyssus, Ornithonyssus and Pneumonyssus

***Dermanyssus gallinae* (Red mite of Poultry)**

- Red colour when recently fed otherwise whitish, grayish or black.
- 1mm, dorsal shield do not reach the posterior end of the body. Anus is on posterior half of the anal plate

- Chelicerae are long, whip like.
- Attack fowl, pigeon, canary, cage birds, wild birds and also feed on man.

Life cycle

After blood meal, eggs are laid (7 in cracks and crevices of poultry house, nests)
↓
After 48-72 hours, moult to
↓
Six legged larvae, do not feed
↓
After 24-48 hours, moult to
Protonymph (feed on host blood)
↓
24-48 hours
↓
Deutonymph (blood meal)
↓
Adult (live for 4-5 months)

Pathogenesis

- Cause irritation and anaemia
- Birds become listless, decreased egg production, loss of blood cause death.
- Vector of Borrelia anserine (spirochaetosis), St. Louis encephalitis, Eastern and Western encephalitis
- Humans-skin lesions

Diagnosis: can be seen with unaided eye.

Control

- Check cracks and crevices, remove nests, 0.25% carbaryl (sevin) applied to the houses and perches. Repeat after 2-3 weeks
- Amitraz (synthetic pyrethrin.
- Malathion (dusts and sprays)
- HCH sprays on premises (not to be used on birds)
- 0.5% lindane, 2% chlordane, 2% malathion dust-good for premises treatment@225g/m^2

***Ornithonyssus sylviarum (*Northern mite of poultry)**

- Elongate to oval adult mite, 1mm
- Dorsal plate wide for 2/3rd of its length and then suddenly tapers to form tongue like and reach posterior end.
- Anus is on anterior half of the anal plate.
- Found on fowl and other birds in temperate climate generally (Britain and Newzealand)

Life cycle (5-7 days)
1-5 whitish sticky eggs are laid on host
↓

24 hours
↓
Six legged larvae (do not feed)
↓
After 8-9 days
↓
Protonymphs (feed on host, need two meals)
↓
1-3 days
↓
Deutonymph (do not feed)
↓
3-4 days
↓
Adult

Pathogenesis

- Mites found on birds, or in nests, or in houses, feed intermittently on birds, seldom attack young chicks
- Irritation, loss of weight, decrease in egg production, loss of blood leads to death.
- Vector for fowl pox, St. Louis encephalitis

Control

Sevin dust or emulsion, dust of fenchlorovos, trichlorophon, coumaphos, 4% malathion, chlorodane, lindane and toxaphene- found to be toxic in birds.

***Ornithonyssus bursa* (tropical fowl mite)**

- Dorsal plate gradually tapers to a blunt posterior end.
- Anus on anterior half of the plate.
- Found on fowl, pigeon, sparrow and other birds in warmer parts of the world (southern Africa, India, China, Columbia, Panama, USA)
- Cannot survive without host longer than 10 days.
- Attack man and cause pruritus.

Life cycle

On sparrow life cycle take place in nests. On poultry most mites are found on fluff of feathers, especially on those around vent

Eggs are usually laid on litter
↓
3 days
↓
Larva (do not feed)
↓
17 hours
↓
Protonymph (host blood)
↓
1-2 days
↓

Deutonymph (feed on host blood)

↓

Adult

***Ornithonyssus bacoti* (tropical rat mite)**

- Adult female 0.65-1mm long.
- Dorsal plate is narrow than other species and tapers gradually to a blunt point.
- Chelicerae have no teeth.
- Spur on the distal segment of pedipalp.
- Anus is on anterior half of anal plate.

Life cycle

Eggs laid in nets and burrows (1-2)

↓

1-2 days

↓

Six legged larvae(do not feed)

↓

24-36 hours

↓

Protonymph (take blood meal)

↓

Deutonymph(do not feed)

↓

24 hours

↓

Adult

Pathogenesis

- Can transmit Yerseenia pestis, Murine typhus, Q fever
- Act as intermediate host for Litomosoides carinii

Control

DDT-10%, dust is applied on burrows.

Pneumonyssus caninum

- Oval, pale yellow, 1-1.5x0.6-0.9mm.
- Single dorsal palte is irregular in shape.
- Genital opening is a transverse slit between fourth coxae.
- Mature females often contain eggs which give birth to larvae.
- Mature females often contain eggs which give birth to larvae.
- Lives in nasal passage and sinuses of dog 9any breed, sex or age)
- Found in USA, Hawaii, Australia, Republic of South Africa.
- It causes reddning of mucosae, sneezing, shaking of head and rubbing of nose.

Pneumonyssus simicola

- Bronchi of Rhesus monkey.

- 100% in some batches.
- Sneezing and coughing.

Histologically, pulmonary lesions, pale spots or yellowish foci to several hundred of tubercle like lesions with associated pneumonitis and cellular infilteration.

Sub order:
Trombidiformes

Family: Democidae

Genus : Demodex

- Elongate, 0.25mm long, have head, thorax which bears 4 stumpy legs, elongate abdomen which is transversely striated all around
- Mouth parts(pair of pedipalps, chelicerae, and single hypostome)
- Penis on dorsal side of thorax, vulva on ventral side. Eggs are spindle shaped.
- It lives in hair follicles and sebaceous glands of various mammals causing demodectic/follicular mange.
- Species are called after their host e.g *D. canis, D. caprae, D. bovis, D folliculorum(man), D. phylloides (pigs)*

Life cycle (18-24 days)
Entire life cycle completed on host
Eggs (20-24 in number, are laid in hair follicles or sebaceous glands)
↓
Larvae
↓
Protonymph
↓
Deutonymph
↓
Adult stage (in hair follicles or sebaceous glands)
Larvae and nymph flow with sebaceous secretions to the mouth of follicle where they mature and repeat life cycle.
Males occur near skin surface.
Transmission is by direct contact and neonatal contact.
Predisposing factors- age, poor condition, intercurrent infection, undue use of alkaline shampoos. In dogs it is associated with poor cell mediated immune response.

Pathogenesis
Lesions vary from simple squamous epithelial shedding or depilation to small or large papules or nodule.
Secondary bacterial infection may occur, leading to suppurative pustule formation.
It becomes evident in 3-9 months dog, number of mite inskin is proportional to severity of clinical disease.
Lesions initially occur on head, forelimbs, then extend to body, hairless parts of the body are seldom involved.
In dog, the disease occur in two forms:
1.**Squamous form-**

- hair follicles are distended with mites and cellular debris.
- Follicular epithelium is atrophic, hyperkeratosis is progressively evident.
- Pieces of cornified material exfoliate from surface.
- Hair may be separated and shed or splintered or disintegrated.
- Changes in the sebaceous gland vary, some atrophy and other hypertrophy.

- Hyperpigmentation occurs giving copper red colour.
- Alopecia, thickened and wrinkled skin with mousy odour

2. **Pustular form**

Invasion of Staphylococcus sp. bacteria in skin

Extensive PMN leucocytes, lymphocytes, plasma cells in pustule/abscess formation with marked inflammation.

Death due to toxaemia or emaciation.

In pigs,

Large pustules (walnut size)

Horse

Pruritus, loss of hair, pustule formation

Goat

Pustular form

Sheep

Rare, usually scaly form occurs.

Suborder: Sarcoptiformes

- Legs end in suckers, claws or hairs.
- Some species in unfavorable environment produce nymphs adapted to resist adverse conditions. They are called hypopial nymphs or hypopi.
- These nymphs often provided with group of suckers and hooks on their legs which they can use to adhere to transport host and carry them to favourable place.

Family: Sarcoptidae

Genera

1.Sarcoptes – cause scabies of man. Sarcoptic mange in sheep, goat, cattle, pig ,equines, fox,dog, rabbit.

2. **Notoeders** Notoedric mange of cats, rabbits, rats.

3. **Cnemidocoptes**

C. mutans (scaly leg)

C. gallinae (depluming itch)

Sarcoptes

- Globose body.
- Dorsal dentate spines with sharp points.
- Two vertical setae occur on the dorsum of propodosoma
- Legs are short
- In males, tarsi of 1st ,2nd , 4th pair of legs and in females, tarsi of 1st ,2nd pair of legs end in bell shaped suckers.
- In male, tarsi of 3rd pair and in females, tarsi of 3rd ,4^{th}pair of legs end in bristle
- Pedicles which bear suckers or bristles is not segmented.

Sarcoptes scabiei

- roughly circular
- Female- 330-600x250-400µm and male 200-240x150-200µm.
- 3rd and 4th pair of leg do not project beyond the margins of the body.

- Ventral surface has distinct epimeres (chitinous extension of coxae).
- Dorsal surface covered with fine folds and grooves, bears small triangular scales.

Life cycle

Female burrow in skin and lay 40-50 eggs in tunnel
↓
3-5 days
↓
larva
Six leg .
some larva escape the breeding tunnel and some remain in the parent tunnel
↓
Nymph
Stages on surface perish.
Few burrow in stratum corneum
↓
Construct invisible moulting pocket into a tunnel
↓
Female remain in this pocket until fertilized by male
↓
She then extends this pocket into a tunnel

Pathogenesis
Parasite pierce skin, suck lymph and feed on young epidermal cells, cause irritation resulting into itching and scratching. This seults in the inflammation of skin accompanied by ab exudates, which coagulates and forms the crust on surface. This is further characterized by excessive keratinization and proliferation of connective tissue. Skin becomes thickened and wrinkled. There is loss of hair.
Man
Pruritus with urticarial response.
Dog
Axillary and inguinal region .also on head, around eyes and ears. There is erythema, papular dermatitis followed by the rupture of papules and encrustation of lymph. The skin becomes dry and thickened with the loss of hair.
Pigs
Ears and back are mostly affected. Skin becomes thickened abd cracks open leaving deep wounds. Secondary bacterial infection may occur.
Horse
Starts at head, neck and shoulder and extend to the whole body.
Sheep.
Non wooly portion affected. Starts at mouth (lip and nostrils) and extend to other parts of the face, tarsal and carpal joint.
Rabbit
Head and ear
Primates

Back, neck and shoulders.

Treatment

Lindane 0.016-.03% x 3 days (10-14 days interval)
Benzyl benzoate+soft soap/methylated spirit.
Tetaethylthiuram monosulphide 5% (tetmosol soap)
Ciovap (10% ciodrin and 2.5% DDVP)
Coumaphos (30%), HCH (30%)
Carbaryl 4.25%
Ivermectin 200 ug/kg body weight

Notoedres

Anus is dorsal in Notoedres as compared to terminal location in Sarcoptes.

Notoedres cati

Minute mites, attack cat occasionally rabbit.
Occur in ear and back of neck, may extend to face, foot, hind paws, whole body in young cats
Mites buurow into the skin
Characteristic lesion, yellowish crust in region of ear, face or neck, skin is thickened, wrinkled.
Benzyl benzoate and tetmosol soap

N. muris ear mange of rat (ear, nose, tail). Lesions are wart like and horny.

Cnemidocoptes

No spine on dorsal surface as Sarcoptes have.
2 chitinous bars run from bases of pedipalps to the level of legs where they are united by transverse bar.
Legs and pedicles are short and stumpy.
male- 1,2,3,4 legs bears suckers and in female no leg bears suckers.

C . gallinae (depluming itch in fowls)

Mitws burrow into the skin alongside shaft of the feathers.
Itching, inflammatory condition, feathers break off readily, pulled out by birds.
Lesions seen on back and wings, rarely on head and neck.
10% benzyl benzoate daily x3 and Lindane (0.2%)

C. mutans (scaly leg of fowl)

Mite get on to feetfrom ground, pierce skin underneath scales and cause inflammation with exudates. This hardens on surface and displaces the scale. There is marked keratinization leading to lameness, malformation (rare cases comb and neck is involved)
Treatment

0.1% HCH, 10% sulphur,).5% sodium fluoride. 1% coumaphos or carbyryl)

Family Psoroptidae

Genera:
Psoroptes Psoroptic mange in sheep (sheep scab), goat, cattle and equine.
Otodectes Otodectic mange in dog, fox, cat
Chorioptes Chorioptic mange in equines and cattle.

Psoroptes
Oval body
No dorsal spine
No vertical setae on the dorsum of propodosoma.
Legs long and project beyond the margin of body.
Bell shape sucker on tarsi of some or all legs.
Pedicle long and composed of three segment in Psoroptes.
Anus is terminal
Posterior margin of abdomen is prolonged into 2 lobes and is prominent in Psoroptes and Chorioptes
P ovis cause sheep scab.
Terminal tuberclesbears several hairs
Male- 1,2,3 segmented pedicles and bear sucker.
Female-1,2,4 jointed peidcle and bears sucker.

Life cycle
Eggs are laid on skin of edge of lesion
↓
1-3 days
↓
Larva (feed for 2-3 days)
↓
Nymphal stages (last for 3 to 4 days)
↓
Adult (female appear before male)
↓
Copulation

Pathogenesis
Mite rupture / puncture epidermis and suck lymph
↓
Local inflammatory reaction, rich with serum
↓
Serum exudates on surface, coagulate and forms crust
↓
Wool becomes loose, pulled out by sheep bite and scratch
↓
Crusty patch unsuitable for mite so they migrate to margin

Signs

All parts of body (covered with hair or wool). Frequently around shoulder, side of body, back, sternum, dorsal aspect of tail

P. cervinus occur in ear canal of big horn sheep in North America.
P. cuniculi in ears of rabbit (ear canker)
P equi body mite of horse

Treatment
Dip .03% dieldrin
Ivermectin, coumaphos, pyrethroids

Chorioptes
Tarsal suckers have unjointed pedicles.
Sucker in male, on 1,2,3,4 and 1,2,4 in female.
Male has marked abdominal lobe which bears several hairs.
C eqiu fetlock of horses
C ovis pasterns of sheep.
C cuniculi ears of rabbit
C bovis foot mange or itchy leg in horses
Scab like lesions on fetlock
Horse rub, stamp, scratch, bite legs, kick
Occurs especially on pasterns, hind legs, papules
In cattle, camels and wild ruminants- root of tail. Spread to sacral region and other parts of the body.
Scrotum of rams, sheep legs.
HCH, Fenchlorovos, diazinon.

Otodectes cynotis
Resemble chorioptes
Sucker on Male- 1,2,3,4 and Female-1,2 (unjointed pedicles)
Abdominal lobes are not marked.
Occur in ear of dog,cat, red fox, Arctic fox, carnivores and cause ear mange.
Life cycle same as Psoroptes. Eggs are singly laid.
Mites spend all their lives in the ears of host.
Feed on epidermal cells/ debris
Pierce skin obtain body fluids.
Signs seen in dogs at early stage wherase cat and foxes show signs during advanced stage.
Ear droops, purulrnt discharge, haematoma in dog, torticollis, epileptic fits, perforation if tympanic membrane, ulceration, serious infection of middle and inner ear and brain.
0.1%HCH, benzyl benzoate (20%), antibiotics.

Genus	**Male**	**Female**

	1	2	3	4	1	2	3	4
Sarcoptes	s	s	-	s	s	s	-	-
Notoedres	s	s	-	s	s	s	-	-
Cnemidocoptes	s	s	s	s	-	-	-	-
Psoroptes	s	s	s	-	s	s	-	s
Chorioptes	s	s	s	s	s	s	-	s
Otodectes	s	s	s	s	s	s	-	-

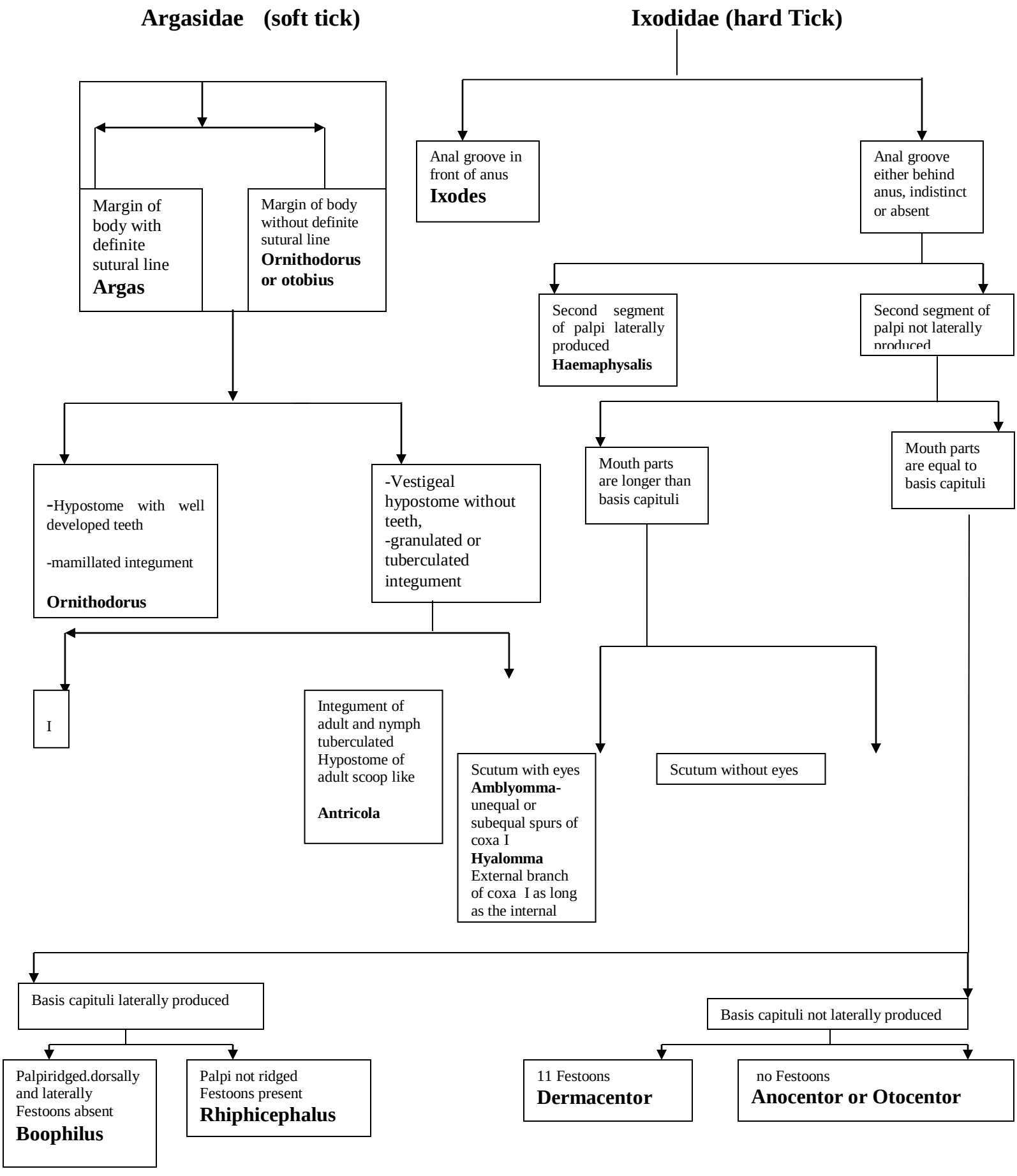
S= sucker on tarsus

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Lecture:12

Sub order: Ixodoidea

Family	**Argasidae**	**Ixodidae**
1.	Tegument is leathery, frequently mammilated, no dorsal shield	Hard, chitinous shield/ scutum covers whole dorsal surface in males and covers only small surface behind head in larvae, nymph and female.
2.	Mouth parts are located anteriorly on ventral surface, not visible form dorsal side (in nymph and imago)	Mouth parts are anterior and well visible from dorsal aspect.
3.	Sexual dimorphism is not marked	Sexual dimorphism is marked.
4.	One pair od spiracles, posterolateral on third coxae	One pair of spiracles posterolateral to foruth coxae.
5.	eyes are absent, if present (2pairs) situated laterally in supracoxal fold.	Eyes are absent, if present (one pair) situated on lateral margins of scutum.
Life cycle		
1.	Adults feed on host several times	Adult feed on host for one time
2.	Adults lay eggs in batches	Female lay her eggs in one batch and then dies.


Argasidae (soft tick)
Ixodidae (hard Tick)
Anal groove in front of anus
Ixodes
Anal groove either behind anus, indistinct or absent
Margin of body with definite sutural line
Argas
Margin of body without definite sutural line
Ornithodorus or otobius
Second segment of palpi laterally produced
Haemaphysalis
Second segment of palpi not laterally produced
-Hypostome with well developed teeth
-mamillated integument
Ornithodorus
-Vestigeal hypostome without teeth,
-granulated or tuberculated integument
Mouth parts are longer than basis capituli
Mouth parts are equal to basis capituli
I
Integument of adult and nymph tuberculated Hypostome of adult scoop like
Antricola
Scutum with eyes
Amblyomma-
unequal or subequal spurs of coxa I
Hyalomma
External branch of coxa I as long as the internal
Scutum without eyes
Basis capituli laterally produced
Basis capituli not laterally produced
Palpiridged.dorsally and laterally Festoons absent
Boophilus
Palpi not ridged Festoons present
Rhiphicephalus
11 Festoons
Dermacentor
no Festoons
Anocentor or Otocentor

Family : Ixodidae

Hard, chitinous shield/ scutum covers whole dorsal surface in males and covers only small surface behind head in larvae, nymph and female.

The basis capituli which is inserted into the body anteriorly bears mouth parts and shows two dorsal porose areas in female.Mouth parts are anterior and well visible from dorsal aspect

Sexual dimorphism is marked

One pair of spiracles posterolateral to fourth coxae

Eyes are absent, if present (one pair) situated on lateral margins of scutum.

The genital opening is a natural transverse slit in front of the middle, anus being posterior.

Ornate ticks have coloured enamel like areas on the body, inornate ticks do not.

Bionomics

Ticks lay eggs in the sheltered spots: under stones, soil, crevices and cracks of wood near ground. Eggs are yellowish brown to dark brown in colour. All the eggs are laid in one batchby female which dies after laying egg. The newly hatched larvae or seed ticks climb on to grass and shrubs and wait there to find a suitable host, to which they attach by means of their claws. After having engorged, larva mpults and becomes a nymph. The integument of later requires some time to harden and then nymph engorges and moults to become an imago. After hardening of integument and often after copulation (on host or on ground) female engorges, drops off and seeks a sheltered spot to lay her eggs. Males remain attached to the host for a longer period of time. According to the number of hosts they require during their life time, ticks can be classified into 3 groups-

1 One host ticks.

All the instars engorge on the same animal, the 2 ecdysis take place on the host e.g *Boophilus decoloratus, B. annulatus*

2. Two host tick

The larva engorges and moults on the host and the nymph drops off after also having engorged, it moults on the ground and the imago seeks a new host e.g *Rhiphicephalus evertsi, R. bursa*

3. Three host tick

A different host is required for every instar, they drop off each time after having engorged and moult on the ground. E.g *Ixodes ricinus, R. appendiculatus*

Various species of Ixodidae not only cause direct harm to the host but also are vectors and reservoirs of important viral, rickettsial and protozoan parasites of man and animals

Genus : Ixodes

Anal grooves surrounds anus anteriorly.

Long palpi

Inornate

Eyes and festoons are absent

Species:

Ixodes ricinus (castor bean tick or sheep tick)

Hosts- sheep, dog, domestic and wild animals

Transmission-

Babesia divergens and *B. bovis* (Babesiosis of cattle), *Anaplasma marginale*,

Louping ill virus, Tick borne rickettsial fever, tick pyaemia (*Staphylococcus aureus*),encephalitis, Coxiella burnetti

Other species-

I persulcatus,

I hexagonus,

I canisuga,(dogs)

I scapularis/shoulder tick or black legged tick (cattle,horse,dog,cat)

I pilosus, I rubicundus (tick paralysis), sheep,goat,cattle

I holocyclus (man,dog, cats).

Genus : Boophilus

Anal groove absent in females and surround anus posteriorly in males.

Inornate, eyes present

Festoons absent

Palps and hypostome short

Palps with prominent transverse ridges.

Spiracle are oval/circular

Species:

Boophilus annulatus (north American tick)-one host tick on domestic and wild ungulates

Transmission

Babesia bigemina (Texas fever or piroplasmosis), *Borrelia theileri*, Anaplasmosis

Boophilus decoloratus (Blue tick)

Host: Cattle, equines, sheep, goat, dogs)

Transmission: *Babesia bigemina*, *B. ovis*, *Borrelia theileri*

Boophilus microplus- cattle, horse, goat, sheep.

Transmission- *Babesia bigemina*, *B. argentina*, *Borrelia theileri*, *Anaplasma marginale*, *Coxiella burnetti*

Rhiphicephalus

Bears capituli hexagonal dorsally

Eyes and festoons present.

Usually inornate.

Hypostome and palpi short.

Spiracle are comma shaped

Species-

R haemaphysaloides cattle and buffaloes, goat, sheep, camel wild animals.

Other species

R. appendiculatus (brown ear tick-three host tick on cattle , equine, sheep, goat, even dog)

Transmission-

East coast fever (Theileria parva), *Hepatozoon canis, Theilerai mutans, B bigemina, Rickettsia conori*, Nairobi sheep virus, Louping ill virus

R capensis (3 host tick on cattle)

R sanguineus (brown dog tick/ 3 host tick)

Transmission- *Babesia canis, Ehrlichia canis, B vogeli*

B equi, B caballi (horses) *Anaplasma marginale, Hepatozoon canis, Coxiella burnetti, Rickettsia conori, R. canis, R rickettsi, Pasteurella tularensis*, Nairobi sheep virus

R evertsi (red legged tick/ 2host tick)on domestic and wild mammals,

R bursa

Transmission-

East coast fever, *Babesia. Bigemina (red water disease), Theileria mutans, Borrelia theileri, Rickettssia conori.*

Hyalomma

Inornate, sometimes ornate

Eyes present.

Festoon present or absent.

Male with adanal shields sometimes accessory adanal shields.

Spiracles are comma shaped in male and triangular in females.

Hyalomma sp are usually two host tick.

Hypostome and palps are long (long mouth parts)

Species of Hyalomma are-

a. *Hyalomma anatolicum anatolicum*
b. *H aegyptiacum*
c. *H savigyni*
d. *H. marginatum*.

Larvae and nymph of these ticks feed on small wild mammals and birds and adult tick feed on domestic ruminants and horses.

Transmission

Babesia caballi in horses

Babesia equi

Theileria annulata

T parva

Coxiella burnetti

Rickettsia bovis

R conori.

Haemaphysalis

Inornate

Eyes absent

Palps are short and conical, IInd articlea are laterally produced.

Spiracles in female ovoid or comma shaped, ovoid in males.

Species are of usually small size.

H bispinosa (india)

Mainly found in cattle abd buffaloes but sometimes from sheep, goat, horse and dogs. It is a three host tick.

Other Indian species.

H spinigera	cattle
H. montgomeryi	Cattle, sheep
H flava	cattle, dog
H howletti	Hill pony
H sewelli	goat.
H sundrai	sheep.

H leachi leachi (yellow dog tick) Africa, Asia and Australia

Mainly parasite in domestic and wild carnivores.

May transmit *Babesia canis, Rickettsia conori, Coxiella burnetti*

Amblyomma

Usually inornate

Eyes and festoons present

Hypostome and palpi long.

Male without ventral plate.

Species are usually large and broad.

Indian species

Amblyomma intergrum	Cattle, buffalo
A testudinarium	cattle

Other species of genus are:

A habraeum (bont tick)	wild mammals

A variegatum/ tropical bont tick.

A americanum / lone star tick.

Dermacentor

In this genus *D auratus,* the only known Indian species

Usually ornate

Eyes and festoons present.

Hypostome and palpi short.

Coxa I bifid and coxa IV of male much larger than coxa I to III

No plates on ventral surface of male.

Other species are-

D marginatus

D andersoni (Rocky mountain wood tick).

Transmits Rocky Mountain spotted fever (*Rickettsia rickettsi*)

D variabilis (American dog tick) Transmits Rocky mountain spotted fever and St. Louis encephalitis.

D albipictus/ Winter tick or moose tick.

*D occidentalis/*Pacific coast tick.

D marginatus Asia

Ticks and Disease

1. **Bite injuries**

Bite and blood loss are important. A single female may suck 0.5-2ml blood and if animal carries thousand of ticks, and then there is a substantial blood loss. It is more usual to carry several hundred of ticks by an animal. The effect caused by them is known as Tick worry. Tick worry a combination of irritation by tick bites, local skin infection, blood loss and attack by flies on tick created bite/ wounds.The secondary bacterial infections may produce suppurative lesions(ear, legs etc.) and in lambs they become pyaemic (called as tick pyaemia). It also cause damage to hide.

2. **Tick Paralysis** This is characterized by an acute paralysis (ascending motor paralysis). This condition may be fatal if ticks are not removed. The genus Ixodes, Dermacentor (*D. andersoni*) are more concerned to cause this condition. The degree of tick paralysis is proportional to the length of time the tick has been feeding and no of ticks attached. The condition is completely reversible after removal of tick, if heart and lungs are not involved. Nature of toxin is not known. In ticks, toxins accumulates in ovary and in the late stage of engorgement they reach to salivary glands. Toxins affect neuromuscular transmission. Animals survive paralysis after removal of tick, develop immunity and remain refractory to paralysis for 8 weeks to 8 months. Eleven ixodid and one argasid species can cause paralysis in mammals and in poultry.

The main species responsible for causing tick paralysis are-

Ixodes holocyclus,I rubicundus,I crenulatus, Dermacentor andersoni, Dermacentor variabilis , Amblyomma maculatum, A americanum, Haemaphysalis inermis, H punctata

3.**Tick toxicosis / sweating sickness**

By Hyalomma species, there is eczema and hyperemia of mucous membranes.

4. Disease transmission.

Apart from protozoal infections, ticks may transmit-

- Rocky mountain spotted fever.

- Siberian tick typhus
- Colorado tick fever
- Kyasanur forest disease
- Congo haemorrhagic fever

Treatment and control

As ticks are found on whole body so treatment should be carried out to whole body.

1. Dipping of animal in a tank containing acaricide - aqueous solution/ suspension /emulsion

2. Alternatively spray, showers etc. may be utilized, it saves labour cost. Dipping of animals should be planned keeping in mind the stay of tick on host, time of year when infestation is observed, knowledge of biology of tick, duration of its stages of its host, feeding time and whole life history. The one host tick can be easily controlled than two/three host ticks as all the stages feed on same individual host. The different stages may show differently to different acaricides (chemicals killing ticks).

In last two decades, control of ticks is achieved by chlorinated hydrocarbons (DDT, HCH, Toxophene etc.) but due to their residual effect in meat and development of resistance by Boophilus and other species the emphasis was given to organophosphorus and carbamates.

Newer applications include use of ear tags, ear bands and neck bands

Acaricides

Arsenic- 0.16-.2%, butacarb (20%) with HCH (38%) and phenol (38%), chlorfenvinphos (10%), Coumaphos 0.05%, diazinon 0.05%, dioxathion 0.01% etc can be used.

Synthetic pyrethroids that are being used are- Flumethrin (30-75 ppm), decamethrin (25 ppm), cypermethrin (150 ppm for dips and sprays), cypothrin (150 ppm).

Other methods of control of ticks (especially two and three host ticks) include-

1. Burning of pastures: This may kill the numbers of larvae and other stages when they are expected to be off the host.

2. Cultivation of land. This tends to reduce the tick life by controlling the movement of animals and also create situations unsuitable for ticks e.g exposure of eggs to sunlight, burying the eggs by ploughing. Good drainage reduces the humidity. Pasture spelling can be practiced where the cattle are removed from the pastures for long time to ensure that

the larval ticks die of starvation or climatic effects. Certain species of grass e.g Cynodon which act as tick repellents while others act as larvicidal can be cultivated.

3. Repellants e.g indalone, dimethyl phthalate can be used.

4. Natural enemies e.g hymenopterous parasites of ticks, certain ants and birds destroy a large number of ticks.

5. Use of Sterile hybrids where male offsprings are sterile and females produce sterile males.

6. Access of smaller animals like rats, rabbits, pigs, birds, bats etc to the animal houses should be checked especially in control of 2 and 3 host ticks.doors and windows should be proper so that smaller animals especially rodents do not enter.

7. Wood, furniture and other material should not be stalked in the animal shelters as they provide site for the females to lay eggs.

8.Cracks and crevices in the walls and floors of the animal houses should be filled up and acaricides should be sprayed in these cracks and crevices as females lay their eggs here also.

9. longer shrubs and bushes around the animal houses should be cut because the larval stages climb on these shrubs andd bushes and wait for the hosts to attach to them.

Vaccines:

GAVAC: recombinant vaccine containing rBm-86 mid gut antigen produced in the yeast *Pichia pastoris*. Given 2ml intramuscular. At 0, 4 and 7weeks.

Tick guard:

Family Argasidae

Argas persicus

- common parasite of warm and temperate countries, attacking fowl, turkey, pigeon and man
- 4-10 x 2.6-6mm, ovalin shape, narrow anteriorly than posteriorly
- Edges of the body are sharp.
- Engorged tick is slaty blue in colour, starved tick is yellowish colour with dark intestines.
- Genital opening situated anteriorly on ventral surface and is larger in female than male.

Life cycle and habits.

Egg (small, brown spherical, in batches of 20-100→crack and crevices of fowl houses→after 3 weeks→ larva (six long legs, circular body which become spherical after feeding, attach under the wings, engorge in 5-10 days, can survive without feed for even 3 months) →after engorging→drop off and hide away→N1 (2 weeks) →N2 (2 weeks) →hide away→ adult (hide in sheltered spots, attack their host in night→feed for 2 hours and adults feed only once in a month) →female lay eggs in batches after each meal.

Nymphs and adults can survive for 5 years without food.

Pathogenesis

- worries the birds in night.
- Birds sleep restlessly
- in heavy infection, birds become anaemic.
- decreased egg production or egg production stops completely.
- tick paralysis of ducks.

Transmission of diseases

-*Anaplasma marginale*

-*Aegyptianella pullorum.*

- *Borrelia anserine* (spirochaetosis)

Diagnosis

Look for ticks in cracks and crevices of fowl house

Control

Change of house→put the birds in wooden crates→ larvae will fall down within 10 days→then return fowl in house (clean it by that time)

Spray HCH (0.05% in houses, OP, pyrethroids.

Otobius megnini

- found in North and south America, Southern Africa and India.
- Larval and nymphal stages are most often the parasites in the ears of dogs, sheep, horse and cattle. Also found in goats, pigs, cat, ostriches and man, rabbits, deer.
- Engorged larvae are almost spherical.
- Nymphs are widest at middle, their skin is mamillated, bears numerous spine like processes.
- Nymph body is bluish grey, white legs, mouth parts and spines are pale yellow.
- Adults which are not parasitic have constriction at the middle.

Life cycle and habits

Infection is mainly associated with sheds, yards, hardly seen in animals that remain in open pastures. Eggs are laid in sheltered spots, cracks, under food boxes, stones and crevices

↓

After 3-8 weeks

↓

Larva (can live without food even for 2-4 month

- Attach themselves in ear of suitable host
- Attach below the hair line
- Engorge in 5-10 days
- Suck lymph
- Engorged larva 2-3mm length, yellowish white or pink in colour
- Spherical shape, legs are small

↓

Moults in ear to spined 8 legged Nymph (remain in ear for 1-7 months, if dislodged then accidentally.

↓

Fully grown, engorged nymph measures 7-10mm long

↓

Drop off the host. Seek dry, protected place, buildings, fence, trees

↓

After few days, moult to adult (not spiny)

Females and males do not feed but female lays 500-600 eggs (oviposition six months)

↓

Female dies and unmated males can live longer

↓

After 10days

↓

Eggs may hatch

Pathogenesis

- Suck blood and cause irritation and inflammation
- Secondary bacterial infection may extend inward with serious results
- Infected animal is dull, do not feed well and loose condition.
- Dog shakes head and scratch ears.
- A waxy or oily exudates from ear
- Heavy infection can lead to blood loss.

Diagnosis

Heavy infections are easily diagnosed (dislodge the parasite with wire loop and examine)

Waxy exudates

Treatment

1 part HCH (15%) + 2 parts xylol+17 parts pine oil (weight basis). This penetrates the wax and protects for 3-4 weeks.

Or

1 part 40% chlorodane+15 parts pine oil. Spray in houses.

Ornithodorus moubata

- Eyeless tympan of Africa
- Lives in native huts, in sand under trees where man and animal takes shelter.
- Margin of the body lack definite sutural lines, thick and rounded.

Life cycle

Female lay batches of 100 eggs in sand

↓

Broods over eggs. Eggs hatch in 8 days.

↓

Larva comes out and remain quiescent till it has moulted to Nymph

↓

Nymph (moults)

↓

Adult

Pathogenesis

- Nymph like adult attack their host for short period only to feed
- Suck blood of man and animals
- Transmits *Borrelia duttoni,* Cause of African relapsing fever of man
- This tick is reservoir host of African swine fever virus.
- Vector of Q-fever.
- May transmit *Borrelia anserina* and *Aegyptianella pullorum*

Control

Residual sprays HCH, Op, Pyrethroids, replace material of huts.

O savignyi Tympan which possess eyes (Africa, India, attack camel, fowl).May bite man
O.tholozani without eyes, integument mamillated/ granular
O. megnini Integument pitted, no eyes.
O lahorensis integument finely wrinkled, no eyes

Class Pentastomatida

- Body flattened, fluke like, convex dorsal side
- Digestive tract in the axis of body surrounded by convulations of uterovagina.

***Linguatula serrata* (tongue worm)**

- Cosmopolitan
- Occurs in nasal and respiratory passage of dog, fox, wolf rarely man, horse dog and sheep.
- Parasite tongue shaped, convex dorsally and flattened ventrally.
- Cuticle transversely striated.
- Male-1.8-2cm and female 8-13 cm long.

Life cycle

Eggs expelled in respiratory passage
↓
Swallowed by suitable herbivores (sheep, goat, bovine, rodents, rabbit)
↓
Hatch in alimentary canal. Larvae reach mesenteric lymph glands
↓
Develops to infective nymphal stage 4-6mm
↓
Lies in small cysts
↓
Dogs are infected by eating viscera of infected animal especially sheep.

Pathogenesis

Parasite attach in the nasal passage and cause irritation leading to sneeze/cough, difficult breathing, uneasiness, restlessness, mucous discharge, often blood stained. Parasite live for 15 months.

Diagnosis

Clinical signs and eggs in the faeces

Treatment

- Surgical removal of parasite
- Organophosphorus compounds

Lecture-1
General Protozoology

General characteristics:

- Subkingdom Protozoa falls under the kingdom Protista.
- Unicellular, eukaryotic organisms with nucleus enclosed in membrane.
- Contains cell wall and organelles (differentiated portions of cell) like nucleus, an endoplasmic reticulum, mitochondria, a Golgi body and lysosomes.
- However, they differ from rickettisia which are prokaryotic like bacteria with nucleus dispersed in cytoplasm and devoid of well developed cell wall.

STRUCTURE OF PROTOZOA

Nucleus

It is of two types: Vesicular and Compact

1. **Vesicular:** Vesicular nucleus has large amount of nucleoplasm and small amount of chromatin material. The nuclear membrane binds nucleoplasm enclosing an intranuclear body called endosome (karyosome) or nucleolus.
    - If the intranuclear body is endosome, it is devoid of DNA. Example: Mastigophora
    - If the intranuclear body is nucleolus, it possesses DNA and tests positive with fuelgen stain. Example: Apicomplexa

2. **Compact:** Compact nucleus contains large amount of chromatin and small amount of nucleoplasm. In cliates: Two types of nuclei found
    - *Micronucleus:* Relatively small, diploid, divides mitotically during fission. Controls reproductive function of the organism.
    - *Macronucleus:* Relatively large and divides amitotically during fission – polyploidy – controls vegetative function (metabolic activities) of the organism.

Cytoplasm

- This is the extra nuclear part of the cell.
- Consists of outer dense hyaline homogenous layer ectoplasm and inner more voluminous fluids or granular endoplasm**.**
- They have carbohydrates, protein, lipids, salts, metals and non-metals.

1. **Ectoplasm:** The outer most thick layer is the ectoplast which gives shape to the cell. If it is a membrane it is known as pellicle, if it is a layer it is called periplast. Protection, locomotion and adhesion are some of the functions of ectoplasm.

2. **Endoplasm:** It is granular because of the inclusion. It contains vacuoles – food vacuoles masses of glycogen, fat globules, proteins, Golgi bodies, centrosome. It is involved in providing nutrition and reproduction.

Locomotion

1. **Gilding:** The body glides smoothly along with changes in shape (or) size appreciable under light microscope. However, exact mode of locomotion is still not clear. **E.g.** *Toxoplasma, Sarcocystis* and coccidian merozoites.
2. **Pseudopodia:** Temporary locomotor organelle that can be formed and retracted as needed. Eg. Amoebae.
3. **Flagella:** Flagella are whip - like organelle composed of central axoneme and outer sheath which arise from kinetosome (or) blepharoplast in the cytoplasm. It is composed of a central axial filament, axoneme, which is surrounded by a contracticle cytoplasmic sheath. Electron microscopic studies indicate that axoneme is composed of 2 central filaments surrounded by 9 peripheral filaments (9+2 structure). Eg: Mastigophora
4. **Cilia:** Eyelash - like organelle, fine short flagellum like structure arising from kinetosome embedded in the pellicle or ectoplasm. They also help in ingestion of food and serve as tactile structures. Cilia are found in ciliphora.

Nutrition of Protozoa

1. **Holophytic Nutrition:** Carbohydrates as syntherised by means of chlorophyll contained in chromatophores which vary in size shape, and number – characterstic of phytoflagellates.
2. **Holozoic Nutrition:** Particular food materials (preformed food material including host tissue) are ingested through a temporary or permanent mouth. Temporary mouth is formed by amoeba when they engulf food (pseudopodia). Permanent mouth is a cytosome seen in ciliates. Undigested materials are extruded out though a temporary opening (or) a permanent opening "cytopyge" (cell anus).
3. **Pinocytosis:** Many protozoa ingest nutritious material in solution by a process known as pinocytosis (pinching - off vacuoles of fluid through a temporary opening in the wall). E.g. *Eimeria* sp, *Plasmodium.*
4. **Saprozoic Nutrition:** Nutrition being absorbed through body wall by simple osmosis.

Excretion

1. Through body surface /wall.
2. Cytopyge (or) cell anus – In ciliates, these are seen in opposite poles where cytostome is present.
3. Contractile vacuoles simple (or) associated with feeder canals or vacuoles. They are more important as osmoregulating organelles that are found in fresh water protozoa.

Reproduction of Protozoa

Asexual reproduction: Types of asexual reproduction:-

1. **Binary fission**: Two individual daughter cells are formed with cytoplasm division following nuclear fission. Parent cell gives rise to two nearly halved daughter cells. It may be irregular as in *Entamoeba*, longitudinal as in flagellates (*Trypanosoma*) and transverse as in ciliates (*Balantidium*).
2. **Budding:** Two (or) many daughter forms are produced from "Parent Cell". In this, a small daughter individual separates from the side of the mother and grows to full size. Division may be internal (or) external and number of buds may vary with species. Types of budding:-

    1. **Endodyogeny:** It is a type of internal budding in which two daughter cells are formed with in mother and breaks out destroying it e.g. *Toxoplasma, Sarcocystis*
    2. **Endopolygeny:** If more than 2 daughter cells are produced by internal budding the process is known as endopolygeny.
    3. **Ectopolygeny:** If more than 2 daughter cells as produced by external budding, the process is known as ectopolygeny.

3. **Multiple fission or schizogony or merogony:** It is found in the phylum Apicomplexa. In this, the nucleus divides several times before cytoplasm divides. The dividing cell is known as **meront** (or) **schizont** (or) **segmentor** (or) **agamont**. The daughter cells are known as **merozoites** (or) **schizozoites**.

    Agamont is a schizont in which merozoites repeat schizogony.

    Gamont is a schizont in which merozoites become gametocytes initiating sexual phase.

Sexula reproduction: Two types of sexual reproduction:-

1. **Conjugation:** This is seen in ciliates. Two individuals come together temporarily and fuse along part of their length. The macronucleus undergoes disintegration and disappears. Part of their micro nuclei divide a number of times and one of the haploid pronuclear passes from each conjugant another. The conjugants then separate and nuclear reorganization takes place.
2. **Syngamy:** It is a process of fusion of two gametes to form a zygote. If the gametes are same in size they are called as isogametes and if they are dissimilar, they are called as anisogametes.

In the anisogametes, the smaller one is called microgamete which is a male gamete. They divide by mitosis. Nucleus divides into a number of bits each surrounded by cytoplasmic fragment forming microgametes. They are actively motile by means of flagella. The large gamete is called macrogamete, a female gamete. Contains large amount of cytoplasm with compact nucleus of more food reserve; they divide by meiosis into a single macrogamont which is non-motile and ovum like.

- The gametes are produced by a special cell called gamonts; the microgamont (or) microgametocyte and macrogamont (or) macrogametocyte. The process of gamete formation is known as gamogony or gametogony.
- **Isogamy:** It is a process of fusion of two identical gametes. Eg. *Babesia*
- **Anisogamy or heterogamy:** Gametes of different size fuse resulting into a zygote. *Eimeria*

Sporogony

Zygote may be divided to form a number of sporozoites. The process is known as Sporogony which normally follows syngamy. This is an asexual process of multiple fission.

Economic importance of Protozoa:

The nature of harm done and consequently the host reaction to protozoa are varied. Broadly, the most common damages caused by various protozoa are as follows:-

1) **Mechanical obstruction:** Protozoa, being microscopical rarely cause mechanical obstruction except for occasional clumping of the organisms in the infected cells and blocking the capillaries of the organs including brain and spleen. Eg. *Plasmodium falciparum*, *Babesia* species and Trypanosomes.
2) **Damage to the host cells:** All intranuclear protozoans destroy many cells of various organs and tissue of the host, e.g. Erythrocytes by haemoprotozoa, epithelial cells of gastrointestinal tract by coccidian species.
3) **Haematological changes:** All blood protozoans cause a variety of changes in the blood due to the destruction of red blood cells leading to anaemia and inhibitin of haemopoesis (*Trypanosoma congolense*), hypoglycemia due to impairment of vital organ functions (*T. evansi*).
4) **Host reaction:** Protozoa may cause various host reactions such as inflammation, hypertrophy, hyperplasia, increased peristalsis, enlarged lymph nodes etc.
5) **Toxin production:** Necrosis and lysis of tissues may occur due to the release of various toxic substances and proteolytic enzymes by the protozoa having cytolytic effect.
6) **Autoimmunity:** The immune response of the host against some intracellular protozoa cause severe destruction of infected as well as non infected blood cells. The self character of the non infected erythrocytes is changed making themselves susceptible to phagocytosis by the host body defence cells.

Phylum	Subphylum	Class	Order	Family	Genus
Sarcomastigophora	Mastigophora	Zoomastigophorea	Kinetoplastida	Trypanosomatidae	*Trypanosoma, Leishmania*
			Retortamonadida	Retortamonadidae	*Chilomastix*
			Diplomonadida	Hexamitidae	*Giardia, Hexamita*
			Trichomonadida	Monocercomonadidae	*Histomonas*
				Trichomonadidae	*Tritrichomonas, Tetratrichomonas, Trichomonas, Trichomitus, Pentatrichomonas*
	Sarcodina	Lobosea	Amoebida	Entamoebidae	*Entamoeba, Endolimax, Dientamoeba, Iodamoeba*
				Hartmannellidae	*Hartmannella, Acanthamoeba*
Apicomplexa		Sporozoea	Eucoccidiida	Eimeriidae	*Eimeria, Isospora, Tyzzeria, Wenyonella*
				Cryptosporidiidae	*Cryptosporidium*
				Sarcocystidae	*Sarcocystis, Toxoplasma, Neospora, Besnoitia, Hammondia*
				Haemogregarinidae	*Hepatozoon*
				Plasmodiidae	*Plasmodium, Haemoproteus, Leucocytozoon*
			Piroplasmida	Babesiidae	*Babesia*
				Theileriidae	*Theileria*
Microspora		Microsporea			*Encephalitozoon*
Ciliophora		Kinetofragminophorea	Trichostomatida	Balantidiidae	*Balantidium*

General Classification of Protozoa

Lecture-2

Phylum: Sarcomastigophora
Subphylum: Mastigophora
Class: Zoomastigophorea

The Zoomastigophorea are flagellate protozoa possessing one or more thread like flagella; some also possess pseudopodia. In some forms a flagellum may pass along the body, being attached to it by an undulating membrane (e.g. *Trypanosoma*, *Trichomonas*). The nucleus is usually vesicular and reproduction is by longitudinal binary fission. The Zoomastigophorea lack chromatophores and feed in holozoic manner. The following orders are of veterinary and medical importance:-

a) Kinetoplastida (e.g. *Trypanosoma*)
b) Retortamonadida (e.g. *Cochlosoma*)
c) Diplomonadida (e.g. *Hexamita*)
d) Trichomonadida (e.g. *Trichomonas*)

Order: Kinetoplastida
Family: Trypanosomatidae

They are parasitic and are commonly found in blood, plasma, lymph and tissue fluids of mammals and birds, hence called as haemoflagellates.

Morphology of Trypanosomes

- Characteristic leaf- like body with vesicular nucleus.
- Posterior to the nucleus, the **kinetosome** or **basal granule** or **blepharoplast** is located, from which arises a single flagellum. In some genera, this single flagellum is attached to the body by an undulating membrane.
- Posterior to the kinetosome is a rod-shaped or spherical **kinetoplast** containing DNA.
- Under light microscope, kinetoplast and kinetosome appear close together giving a fused appearance.

Developmental Stages in Trypanosomes:

1. **Trypomastigote stage:** The most advanced form. Here, the kinetoplast and kinetosome are near the **posterior end and the flagellum forms the border of an undulating membrane** that extends along the side of the body to the anterior end. This stage is found in the vertebrate host and the arthropods. The arthropods carry this stage as the infective stage for the vertebrate host.
2. **Epimastigote stage:** The kinetoplast and kinetosome are just **anterior to the nucleus and the undulating membrane is short**. It has a small free flagellum. This is principally a stage in arthropods, though in certain species it maybe found in the vertebrate developmental cycle.

3. **Promastigote stage:** The kinetoplast and kinetosome lie towards the **anterior tip of the body and there is no undulating membrane.** Flagellum is quite short and free. It occurs in arthropods.
4. **Amastigote stage:** The body is **round and the flagellum is absent or represented by short fibrils.** Kinetoplast is present. The stage is seen in vertebrates and arthropods.

Genera: The family includes the following genera of veterinary importance:

1. ***Trypanosoma*:** Occurs in vertebrates and arthropods and development may include the trypomastigote, epimastigote, promastigote and amastigote stages.
2. ***Leishmania*:** Found in vertebrates and arthropods. Developmental stages include amastigote and promastigote with the latter being encountered in invertebrates.

Most of the members of Trypanosomatidae undergo **cyclical development**. In the above discussed genera, the life cycle alternates between vertebrate and invertebrate hosts. The invertebrate host gets infected from the vertebrate hosts usually involving a developmental stage different from that in the vertebrate host. Thus, in the genus *Trypanosoma* the typomastigote form occurs in the vertebrate host and epimastigote form predominates in arthropods.

Genus: *Trypanosoma*

Location and host: Blood and tissue fluids of vertebrates.

Transmission of Trypanosomes in animals:

With the exception of *Trypanosoma equiperdum*, all trypanosomes are transmitted by blood sucking insects in which transmission is either cyclical or non-cyclical (mechanical).

1. **Cyclical transmission:** The arthropod is a necessary intermediate host in which the trypanosomes multiply, undergoing a series of morphological transformations before forms infective for the next mammalian host are produced. Cyclical transmission maybe of two types:

a) **Anterior station development:** Here the developmental stages (epimastigotes) multiply in the gut of the arthropods and the infective stages a.k.a. **metacyclic trypomastigotes** accumulate in the proboscis or salivary glands, so that the new infection is transmitted when taking a blood meal. The various species of trypanosomes which use this process are often considered as a group, the **Salivaria** and hence the transmission is also known as Salivarian transmission. Eg. *Trypanosoma congolense, T. vivax* and *T. brucei* are transmitted by tsetse flies (*Glossina* spp.).

b) **Posterior station development:** In other trypanosomes, multiplication and transformation occur in the hindgut and the metacyclic trypomastigotes (infective

forms) migrate to the rectum and are passed in the faeces of the arthropod. Infection of the vertebrate occurs by contamination of the skin or skin wound. The trypanosome species are grouped together as the **Stercoraria** and hence the transmission is also known as Stercorarian transmission. E.g. *Trypanosoma theileri* and *T. melophagium* transmitted by tabanid flies and sheep keds respectively; *T. cruzi* transmitted by reduviid bugs.

2. **Non-cyclical transmission:** This is essentially mechanical transmission in which the trypanosomes are transferred from one mammalian host to another by the interrupted feeding of biting insects. The trypanosomes in or on the contaminated proboscis do not multiply and die quickly so that cross-transmission is only possible for a few hours.

General characteristics of Salivarian Trypanosomes

a) **Morphology:** Kinetoplast smaller, terminal or subterminal. Posterior extremity blunt and there maybe no free flagellum, undulating membrane varying in development.
b) **Biology in definitive host:** Reproduction in the mammalian host is continuous, taking place in the trypomastigote stage in the blood or in the lymph glands. Salivarian trypanosomes are highly pathogenic for certain mammals.

General characteristics of Stercorarian Trypanosomes

a) **Morphology:** Kinetoplast large and not terminal, posterior extremity tapering, free flagellum present, undulating membrane not well developed.
b) **Biology in definitive host:** Multiplication in the mammalian host is discontinuous, typically taking place in the epimastigote or amastigote stages with the tryptomastigotes typically not pathogenic.

Immune response

Trypanosomes are known for the antigenic variation phenomenon as they constantly change their antigenic character because of periodical change of its surface coat also known as variable surface glycoprotein (VSG). Since antigenic character of strain keeps on changing the prospects of producing a vaccine against trypanosomes is bleak.

THE PATHOGENIC TRYPANOSOMES OF DOMESTIC ANIMALS

SECTION: SALIVARIA (Anterior Station forms)

***Trypanosoma vivax*:**

Morphology: It is a monomorphic species. The posterior part is distinctly broader and bulbous, the kinetoplast is large and terminal and the free flagellum is short. The organism is very motile in fresh blood.

Hosts: Cattle, buffalo, sheep, goat, camel and horses. The dogs and pigs are refractory to infection.

Transmission: Cyclical transmission by *Glossina morsitans* and *G. tachinoides* (tsetse flies). The development taking place in the anterior station and in the proboscis only.

Geographical distribution: The parasite occurs throughout Africa. *T. vivax* causes the most common form of trypanosomosis of cattle in West Africa.

Pathogenesis and Clinical Signs: The parasite causes an asymptomatic infection or a chronic, acute or peracute disease. In the peracute infection, a high and persistent parasitaemia occurs, although at the time of death parasitaemia maybe very low. Extensive haemorrhages are seen on the mucosal and serosal surfaces of the digestive tract, body cavity, muscles, heart and lymph nodes. Enlargement of spleen and lymph nodes and there is depression of haemoglobin and red blood cell levels. Chronic disease in cattle may last for several months.

***Trypanosoma congolense*:**

Morphology: It is a small form and smallest of the African trypanosomes. The posterior extremity is blunt and kinetoplast is typically marginal, being at some distance from the posterior end. Characteristically there is no free flagellum although the tapering anterior extremity may inadvertently give the appearance of one. The parasite exhibits marked but non progressive movements in fresh blood.

Hosts: All domestic animals and wild game animals, sucha s antelopes, zebra and even the elephants. The chief association of the organism is with the cattle and is the principal cause of the disease **'nagana'**, the word derived from Zulu, meaning 'to be in low or depressed spirits.'

Transmission: Cyclical transmission by *Glossina* species.

Geographical distribution: It is widely distributed in tropical Africa and it produces the most important form of animal trypanosomosis in East Africa.

Pathogenesis and Clinical Signs: *T. congolense* is regarded as a plasma (haematic) parasite. It may produce peracute, acute or chronic diseases. Haemorrhages occur on serosal and mucosal surfaces and in lymph nodes of the infeted animals, which may also be enlarged. Splenomegaly and multiple perivesicular accumulations of lymphocytes in the liver, kidney and other vital organs may be seen.

***Trypanosoma brucei brucei*:**

Morphology: It is a polymorphic trypanosome, slender, intermediate and stumpy forms occurring. The undulating membrane is conspicuous in all forms. The kinetoplast is subterminal and tail pointed.

Transmission: Cyclical by *Glossina morsitans, G. tachinoides* and *G. palpalis* and may also be mechanical by biting flies. The development occurs in the anterior station, in the midgut and the salivary glands of the fly.

Geographical Distribution: The parasite is widely distributed in tropical Africa.

Pathogenesis and clinical signs: *T. brucei* is classified as a **humoral** parasite, being recovered in the intercellular tissue fluids, such as connective tissue fluid and fluids of the body cavity as well as in the plasma. The parasite is more severe in equines. Acute disease lasts for two to four weeks, while chronic infection maybe of several months duration. Clinical signs include intermittent fever, ocular and nasal discharge, icterus and subcutaneous oedema. Reductions in packed cell volume, haemoglobin and red cells are generally encountered as a result of depression of haemopoetic tissues. At autopsy, emaciation, anemia, enlargement of lymph nodes and splenomegaly had also been reported.

Trypanosoma brucei gambiense: It is the cause of **Gambian sleeping sickness** or trypanosomosis of humans. The principal tsetse flies associated with transmission are the riverine types: *Glossina palpalis* and *G. tachinoides*. Development occurs in the anterior station and mechanical transmission due to biting flies may also occur.

Trypanosoma brucei rhodesiense: It causes **Rhodesian trypanosomosis** or **East African sleeping sickness** of humans. The organism is polymorphic. The principal transmitting flies are the savannah tsetses, chiefly *Glossina morsitans*. *T. rhodesiense* is only mildly pathogenic for cattle; experimental infection of goats and sheep has been found to cause keratitis, encephalitis and death within two months of infection.

MECHANICALLY TRANSMITTED FORMS

Trypanosoma evansi (Syn. *Trypanosoma brucei evansi*)

This is the first trypanosome noticed to be pathogenic for mammals and was identified by **Griffith Evans in horses of Dera Ismail Khan, Montgomery Province** (now in Pakistan)

Morphology: In majority of infections, parasite is monomorphic in character but polymorphism occurs sporadically. The kinetoplast is subterminal, the undulating membrane is well developed and there is a substantial free flagellum.

Host:
Natural hosts: Camel, horse, donkey, mule, ox, goat, pig, dog, water buffalo, elephant, mongoose, deer and other wild animals like fox, hyena and tiger.

Experimental hosts: Many laboratory animals including mouse, rat, rabbit, guinea pig and chicken.

Transmission: Mechanically by biting flies such as ***Tabanus, Stomoxys, Lyperosia*** and ***Haematopota***. An essential factor in mechanical transmission is interrupted feeding on the part of the flies which go from one host to another in order to become replete. Trypanosomes donot survive for more than 10-15 minutes in the proboscis of a fly. In Central and South America, the vampire bats may also act as a vector, although the bat may die of infection about a month after an infected blood meal. In the case of dogs, the infection can be transmitted by the ingestion of the tissues from infected carcasses.

Geographical distribution: North Africa, Central and South America, central and southern Russia, parts of Asia (India, Burma, Malaysia, southern China, Indonesia, Philippines)

T. evansi infection in Equines

The classical entity in the Indian subcontinent occurs in horses and is known as '**Surra**'(a hindi word meaning **rotten**). Surra is always nearly fatal to horses and if treatment is not applied, death occurs in a few days to a few months depending upon the virulence of the strain of the organism. Emaciation and oedema are the most common clinical signs, the oedema varying from urticarial plaques on the neck and the flanks to the oedema of the legs and the lower parts of the body. The plaques may necrose at the centre and the haemorrhages occur at the junction of the skin and the mucous membrane, especially at the nostrils, eyes and the anus. An intermittent fever maybe present but in some cases the condition is so acute that this is not obvious. On post mortem, there is marked oedema, emaciation, enlargement of lymph nodes and splenomegaly. Petechiae occur on the serous surfaces and in the parenchyma of liver and the kidneys.

T. evansi infection in Camels

Camels are highly susceptible to the infection, the disease running an acute or chronic course, terminating fatally in untreated cases. The animals become progressively weaker and the infection runs a course of about three years. Based on which the disease is also known as '**Tibersa**.' In Sudan, the infection is completely limited to camels being known as **Gufar**. Clinical symptoms include fever, progressive emaciation and anaemia with oedema of the dependent parts. Their hump disappears and they become anorectic. Oedema generally appears on pads, abdomen, scrotum and sheath of males.

T. evansi infection in Cattle and Buffalo

Cattle and buffalo are considered to be the main reservoirs of the infection for equines. In these the infection is subclinical in nature. But occasional outbreaks of the acute disease occur with quick deaths. This is often associated with introduction of the parasite of a new strain of it into a new area or to additional stress on animals, such as FMD vaccination. In acute conditions, the animal becomes dull and sleepy, show staggering gait, eyes staring and wide open, breathing hard and noisy, circling movements, nervous excitements, hitting of the head on hard objects, apparent blindness, stamping of the feet, bellowing, groaning, frequent micturition, profuse salivation, twitching of muscles, shivering of body followed by coma, collapse in 6-12 hours. Rise in body temperature is seen but it is not a consistent sign. Peracute cases show nervous

signs in 2-3 hours. The disease thus should be differentially diagnosed from snake bite, anthrax, rabies, poisoning by organic or inorganic chemicals, nervous form of ketosis and initial stage of milk fever.

T. evansi infection in Dogs

In case of dogs, the exotic breeds get severely infected than the native ones. The course of disease is more serious in pups than in adults. Incubation period is 5-10 days. The disease is marked by fever, anorexia, oedema of the head and throat, corneal opacity and even blindness. Oedema of the larynx changes the voice of the dogs which is similar to that seen in rabies.

Diagnosis

Diagnosis of acute infections is usually relatively easy since the organisms are readily demonstrable in freshly stained blood smears. However, the diagnosis of chronic infections may preent difficulty because parasitaemia is usually low. In such cases thin and thick blood smears taken on consecutive days, or lymph node puncture smears should be screened. Thus the diagnosis is based on (1) the history of the prevalence of *T. evansi* infection and of biting flies especially tabanids; (2) clinical symptoms; (3) laboratory examination of the blood and body fluids by direct examination, chemical tests, animal inoculation tests and immunological tests.

1) **Direct examination:** Demonstration of trypanosomes in fresh blood smears from the infected animals is the surest way of diagnosing the acute infection since the organisms are readily demonstrable in freshly stained blood smears. However, the diagnosis of chronic infections maybe difficult because parasitemia is usually low. Blood collected at the peak of the fever should be used for screening as it may clearly demonstrate the parasites.
2) **Chemical tests:** The following tests have been designed for detecting chemical changes in composition of the blood:
    a) **Mercuric chloride test:** A drop of test serum is added to 1 ml of 1:25,000 solution of mercuric chloride in a test tube, which is gently shaken to mix the two. Appearance of white precipitates after 15 minutes denotes a positive reaction. The utility of the test is limited to camels having one month plus infection.
    b) **Formal gel test:** The test is reliable in camels. Two drops of commercial formalin (40% formaldehyde) are added to 1 ml of test serum in a test tube. The tube is shaken and allowed to stand. The gel starts forming in positive cases in around an hour. Final reading maybe taken after 24 hours of incubation at 4˚C. Distinct opalescence with or without gelatin, indicates positive reaction for Surra.
    c) **Thymol turbidity test:** The test is similar to mercuric chloride test as it assesses the excess of serum globulin fraction in infected individuals. Alkaline thymol buffer (3 ml) is mixed with one drop of test serum inactivated at 56˚C

for 30 min. Immediate white turbidity, increasing maximum upto 1 hour, appears in positive cases.

d) **Stilbamidine test:** Utility of this test is limited to Surra in cattle. About 0.5 to 2.5 ml of freshly prepared 10% aqueous solution of stilbamidine isothionate is taken in a small test tube. One drop of inactivated test serum is added on the surface of the above solution. A positive case shows coagulated mass which begins to sink down in half a minute and dissolves in 5-10 minutes.

3) **Animal inoculation: Albino mice and rats** are most suitable hosts for detecting subpatent infection. Guinea pigs and rabbits are less suitable because of their greater inner resistance to *T. evansi.* In white mice, 0.5ml of suspected blood with an anticoagulant is inoculated intraperitoneally. Cryptic (or) sub- clinical trypanosomes in blood will multiply in mice and cause death within 48-72 hours. Animal inoculation test is considered to be more reliable than the direct microscopic examination as it can detect subpatent infections.
4) **Immunodiagnostic tests:** A number of immunodiagnostic tests have been developed and applied for the diagnosis especially in case of animals. Indirect haemagglutination (IHA), Complement fixation test (CFT), Immunofluorescent antibody test (IFAT), Enzyme Linked Immunosorbent Assay (ELISA) *etc*.have been recommended for the diagnosis of trypanosomes in animals. The antigen based ELISA and Suratex are useful in detection of current infections, whereas antibody based ELISA and card agglutination test for trypanosome (CATT) are useful for serodiagnosis.

   Nowadays, polymerase chain reaction (PCR) and DNA probes have been used for detection of trypanosomes in animals.

Treatment

Drugs for the treatment of trypanosomosis are classified under four major chemical groups:

1) Quinapyramine (Antrycide): (a) Antrycide methyl sulphate @ 3mg/kg body wt. as 10% aqueous solution subcutaneous injection is used for therapeutic purpose. (b) Antrycide chloride at similar dose rate is used for prophylaxis. (c) Antrycide prosalt (3 parts of Antrycide methyl sulphate + 2 parts of Antrycide chloride) @ 3 mg/kg body wt. as 10% aqueous solution subcutaneous injection. The prosalt is both therapeutic and prophylactic, which provides protection for 3 months. Most useful for control and treatment of surra in cattle, horse, camel, elephant and dogs.
2) Suramin (Sulphonated naphthylamine): In horses, a 10% sol. in sterile distilled water is used @ 4g/45 kg body wt. (single or in 3 divided doses over a period of three weeks) intravenously. The drug can prove toxic in equines so slow intravenous injection is recommended.
3) Diamidines (Diaminazine aceturate): 3.5 mg/kg b.wt. in dogs and 10 mg/kg b.wt. in cattle, subcutaneous or deep intramuscular injection.
4) Phenanthridine compounds: 1mg/kg body wt. intramuscular injection.

***Trypanosoma equinum*:**
Morphology: It is a large monomorphic form differing from *T. evansi* only in **lacking a kinetoplast**.

Host: *T. equinum* is chiefly an infection of equines, the horse being more seriously affected and the clinical condition is referred to as **mal de caderas**. Mules and donkeys are less susceptible while dogs, cattle, sheep, goats can be infected with a mild disease entity. The capybara is susceptible to infection and may serve as a reservoir host for the equine infections. The parasite is responsible for high mortality of the equines in the wet areas, particularly swamps, where the incidence of blood sucking flies is high.

Transmission: Mechanical by biting flies.

Geographical Distribution: It occurs in Central and South America.

Pathogenesis and clinical signs: The disease in horses is rarely acute but in such cases death occurs a few weeks after the onset of the clinical signs. More usually it runs a chronic course with the death occurring two to six months after infection. The incubation period is four to ten days, after which the pyrexia and parasitemia occurs. Emaciation commences early in the infection and a **marked weakness of the hindquarters (mal de caderas)** results in staggering gait. This is progressive and the animal final becomes recumbent. Associated lesions are conjunctivitis, keratitis and oedema of the eyelids. Transient cutaneous plaques occur over the neck and the flanks, which lose their hair and later scab over. On PM, there is splenomegaly, enlargement of lymph nodes and anemia.

Diagnosis: In the acute stage of the disease is based on the demonstration of the parasites in the peripheral blood. In more advanced infections, the organisms are difficult to find and inoculation of blood into laboratory rodents (eg. mice) is the most satisfactory means of diagnosis.

***Trypanosoma equiperdum*:**
It causes a venereal disease of horses, called as **Dourine** (Arabic: unclean).

Host: The disease is naturally found in **horse and donkeys**. The **jackass** maybe a symptomless carrier. Of other species, dogs maybe infected with some strains of the parasite; cattle are generally resistant to infections, but laboratory mice, rabbits and rats are susceptible.

Transmission: *T. equiperdum* is ordinarily mechanically transmitted by **coitus** and rarely by blood sucking biting flies and by infective discharges contaminating mucous membranes.

Geographical Distribution: The disease presently occurs in North and South Africa, Central and South America, the Middle East and Russia. The disease has been eradicated from India under the 'Dourine Act'and last case was reported in 1921.

Clinical Signs: The clinical entity of dourine usually progresses through three distinct phases following an incubation period of 2-12 weeks or several months.

The first phase, **the stage of oedema,** is initiated by a mucoid vaginal or urethral discharge, a degree of nymphomania or a mild fever with the oedema of the genitalia. In stallion, the prepuce and scrotum are swollen and the oedema may extend under the belly as far forward as the chest. In the mare the vaginal mucosa is hyperaemic and ulcers maybe present. There is deep pigmentation of circumscribed areas of vulva and the penis. This phase lasts 4-6 weeks and in mild cases may go unnoticed. But in severe forms, frequent micturition and even abortion in pregnant mares maybe evident.

The second phase, **urticarial phase**, is characterized by the appearance of oedematous plaques under the skin, especially of the flanks but any part of the body maybe affected. The plaques are circular, sharply circumscribed and are 2-10 cm in diameter. They are classically referred to as '**Dollar spots**' since they appear as if a silver dollar has been inserted under the skin. They may persist for a few hours to three to four days, then disappear but may reappear. The presence of dollar spots is almost pathognomonic for the disease and the affected areas maybe left depigmented when the swelling resolves, giving a good appearance of their previous existence.

The third phase of infection is the **phase of paralysis.** The muscles of the face and nostrils are usually affected first but later paralysis of the muscle is associated with complete paralysis and recumbency, which is followed by death.

At post mortem, the carcass is emaciated with marked muscular atrophy. The clinical disease is typical enough to allow diagnosis in an endemic area.

Diagnosis: Trypanosomes are not readily detected in the blood but they maybe found in the vaginal and preputial discharges or in the serous fluid squeezed from the urticarial plaques.

SECTION: STERCORARIA (Posterior Station Forms)

***Trypanosoma theileri*:**
Largest species amongst other trypanosomes.

Morphology: The posterior end is long and pointed and the kinetoplast lies some distance from the posterior end. The undulating membrane is well developed and the free flagellum is well defined.

Host: Bovines

Transmission: *T. theileri* is transmitted cyclically in the posterior station by tabanid flies such as ***Tabanus*** and ***Haematopota*** and infection of bovine takes place by contamination.

In the bovine both trypomastigote and epimastigote forms occur and multiplication takes place chiefly in the lymph nodes and inner organs.

Under ordinary circumstances, *T. theileri* is non pathogenic organism and probably occurs in cattle throughout the world.

***Trypanosoma melophagium*:**
It is a non pathogenic form infecting sheep. It is transmitted cyclically in the posterior station by the **sheep ked**, ***Melophagus ovinus***. Infection is by contamination of the skin and also if a ked is eaten, the metacyclic trypanosomes may enter the buccal mucosa.

***Trypanosoma cruzi*:**
It is the main pathogenic species of Stercoraria. It is the cause of **American Human Trypanosomosis** or **Chaga's Disease** in South America.

Morphology: In the blood, it is monomorphic and crescent shaped with a pointed posterior end. The kinetoplast is large and subterminal. There is moderately well developed undulating membrane and free flagellum. No division takes place in trypomastigote stage and it generally takes place in **amastigote stage.**

Host: A large number of animals get infected and serve as reservoir hosts including dogs, cats, pigs, foxes, ferrets, squirrels and monkeys. *T. cruzi* is of greatest importance for man, chiefly affecting young children and infants. The principal reservoir hosts in South America are armadillo and the opossum.

Transmission: Under natural conditions, the parasite is transmitted by blood sucking bugs of the family **Reduviidae,** development occurring in the posterior station. Metacyclic trypanosomes are passed in the faeces of the infected bugs. The human or mammalian hosts get infected when metacyclic trypanosomes are rubbed into the wound made by the insect or into other skin abrasions or through the mucous membranes. **Reduviids (kissing bugs)** usually defaecate after feeding and they usually feed on the soft skin of lips and eyes. The infected faecal material then easily gets rubbed into the mucous membranes and the wounds.

Pathogenesis: Following infection of the wounds by metacyclic trypomastigotes, the organisms enter histiocytes and proliferate in amastigote form. There is a local inflammation and later encapsulation by a fibrous tissue leading to blockage of local lymphatics and producing oedema of the local area. This is the primary lesion or **'chagoma'**. Amastigotes pass from the primary site to the local lymph nodes and then by the lymphatic system to the whole body. The lungs, liver, spleen, bone marrow, cardiac muscles and brain cortex are affected. The organisms now multiply as amastigotes. Large scale rupture of the host cells release trypomastigote form into the blood and this is associated with fever. In dogs, debility, anemia and splenomegaly occur. Young animals are more susceptible to infection. Myocardial involvement also takes place in dogs but not as extensive as in man.

Diagnosis: The acute stage is detected by demonstration of the trypomastigote stage in thick blood smears. However, as the infection progresses the organism becomes infrequent in the blood and are present in detectable numbers only during the bouts of fever.

Xenodiagnosis, that is the feeding of the triatomid bugs on the suspect or allowing bugs to feed on infected blood through a membrane, maybe a valuable technique. A number of serological tests such as Complement Fixation Test (CFT), an Indirect Fluorescent Antibody Test (IFAT) and an Indirect Haemagglutination Test (IHA) *etc.* are also employed for diagnosis.

Control of trypanosomes:

The control measures include both treatment and prevention of the disease. The main factors responsible for the disease prevalence are the latent cases among cattle and buffaloes, wild reservoirs, the development of drug resistant strains and the widespread presence of vectors. The control programme consists of:
(1) treatment of infected animals;
(2) chemoprophylaxis;
(3) regular spray with insecticides, proper disposal of manure and treatment of breeding places of the vectors to reduce the population of the flies;
(4) identification of the enzootic areas by regular examination of blood of animals for effective implementation of control measures.

Though control of tabanid flies is difficult, proper drainage, sprinkling of kerosene oil over the water in ponds, ditches, streams, spraying of insecticides to kill adults and their aquatic larvae; removal of moist hay, bedding and dung from the stables, proper disposal of excreta and keeping animals clean by regular grooming can prove helpful in controlling the vector's population. During outbreaks, the sick animals should be segregated from the healthy ones in fly proof shed and should be provided with proper prophylactic treatment.

Lecture-3

Genus: *Leishmania*

Developmental stages of this genus occur in the **amastigote form in the vertebrates** and in the **promastigote form in the insect vectors** and **in the culture**.

Morphology: The amastigote stages in the vertebrates are found in the endothelial and macrophage cells of the body. They are circular or oval in outline. When stained with Romanowsky stains, the cytoplasm is blue, the oval nucleus is red and lies to one side and at right angle to it is red to purple staining kinetoplast. The parasite multiplies in the cytoplasm of the cells forming cluster of organisms.

The cycle in the insects: A variety of ***Phlebotomus*** species (**sandflies**) act as vector for the parasite. During the blood meal the sand fly ingests the leucocytes and large mononuclear cells containing **amastigotes** also known as **Leishman-Donovan bodies (LD bodies).** These develop in the midgut of the sandflies and enormous number of promastigotes are produced. These pass to the oesophagus and the pharynx of the fly and their number be so great as to block the food canal. When a fly attempts to feed, a plug of organisms maybe dislodged and injected into the mammalian hosts. Infection may also occur when infected sandflies are crushed on the skin.

The two most common types of leishmaniosis encountered are: (1) Visceral leishmaniosis (2) Cutaneous leishmaniosis

Visceral leishmaniosis

***Leishmania donovani*:** It is the cause of Kala-azar, dumdum fever or visceral leishmaniosis in humans. Different types of visceral leishmaniosis have been recognised and classified as follows:-
Indian kala-azar, African kala-azar, Russian kala-azar and American kala-azar.

Three ecological forms of *L. donovani* have been recognised:

(1) *Leishmania donovani donovani*: Found in Indian Subcontinent and Burma. It causes Indian kala-azar or Dumdum fever in humans. It affects especially the young aduls (60%) and children of 5-15 years of age. Dogs are not natural hosts although they can be experimentally infected. It is transmitted by *Phlebotomus argentipes*. In India, epidemics have been reported from Assam, West Bengal, Bihar. Endemic foci are located in Tamil Nadu and sporadic cases were seen in Gujrat, Uttar Pradesh, Punjab, Jammu region and Puducherry.

(2) *Leishmania donovani sensu lato* of Kenya, southern Ethopia, Somalia and Uganda. This forms a part of 'African kala-azar'. It resembles Indian kala-azar, as 66% of the cases are reported in young adults.

(3) *Leishmania donovani sensu lato* of Sudan, western Ethopia, Chad, Niger Republic and Gabon. It is a zoonosis and carnivores and rodents serve as reservoirs. It is transmitted by *P. langeroni orientalis*.

Leishmania infantum: It causes Chinese kala-azar, Mediterranean (infantile) kala-azar and Russian kala-azar. Reservoir hosts are fox, wolf, jackals and porcupines, with the dog as domestic reservoir host. In China, transmission is mainly caused by *Phlebotomus chinensis*, whereas in other parts, *P. major* plays the role of vector.

Pathogenesis of Visceral Leishmaniosis

Visceral leishmaniosis, especially the Indian form, is frequently a fatal disease in man. Following inoculation promastigotes proliferate at the local site in macrophages. There is inflammation and chemotaxis, receptor mediated phagocytosis. After a period of weeks or months, they invade the inner organs especially spleen, liver, bone marrow and elsewhere to multiply, destroying the macrophages in process. The kala-azar starts with an irregular fever followed by malaise, headache, abdominal pain, dysentery or diarrhoea and bleeding from the mucous membranes of mouth and nostrils. The skin becomes dark with brittle hair. Macrophages, myelocytes and neutrophils of the bone marrow are filled with the parasites. In advanced cases there is ulceration of the digestive tract with enlargement of liver and spleen. The recovering cases after treatment show whitish spots as lentil sized nodules on the skin particularly face and the neck. The nodules are called **post kala-azar dermal leishmanoids (PKADL).** These contain numerous leishmanial organisms which results in premonition state giving a life-long immunity against *L. donovani* infection in humans.
In dogs, there is initial development of 'spectacles' due to depilation of hair around the eyes. Generalised loss of body hair and eczema. Intermittent fever, anemia, cachexia and generalised lymphadenopathy are typical signs.

Diagnosis: The only certain method of diagnosis is the demonstration of the organisms in spleen pulp, lymph nodes, bone marrow, liver or fixed smears of peripheral blood. In the dogs, scrapings should be made from the periphery of the skin ulcers or eczematous areas. Cultivation of biopsy or post mortem materials on **NNN (Novy, McNeal and Nicolle)** medium or similar media will demonstrate the promastigotes, however, the growth may take one to several weeks. Various immunodiagnostic tests exist for visceral leishmaniosis: complement fixation, indirect immunofluorescence and indirect haemagglutination test.

Treatment: Pentavalent antimony compounds are widely used for the treatment. Compounds such as Pentavalent sodium stibogluconate (Pentostam) and Trivalent sodium antimonyl gluconate are widely used for visceral leishmaniosis treatment. The diamidine compounds, Pentamidine isothionate is very effective for the treatment of *L. donovani* infection.

Control: A major control measure is routine control of sand flies. The various chlorinated hydrocarbons and organophosphate insecticides are useful, but measures against the breeding places of the flies are also necessary. The latter involve the removal of the decaying vegetation and clearing of the dense vegetation around the habitation. Where kala-azar is a zoonosis involving dogs, these should be treated or destroyed. Control of stray animals should be instituted.

Cutaneous Leishmaniosis

***Leishmania tropica*:** It causes cutaneous leishmaniosis. The disease is also known as **'Oriental sore', 'Delhi Boil', 'Mucocutaneous leishmaniosis' or 'Old world cutaneous leishmaniosis'**. The usual hosts are man, dog, gerbil and other wild rodents. Dogs may suffer from dermal lesions.

The classical oriental sore is found in Middle Eastern and Eastern countries with a hot dry climate. It mainly occurs in countries like Sudan, Egypt, West Africa, in Middle East from Lebanon to Turkey and then Eastwards to India, Pakistan and Ceylon. Dogs are commonly infected in India. Anthroponotic cutaneous leishmaniosis had been reported from Rajasthan and other adjacent northern parts of India.

L. major: Also causes cutaneous leishmaniosis in Turmenistan, Uzbekistan and Iran. Reservoir hosts include Giant Gerbils and hedgehogs.

Pathogenesis: Cutaneous leishmaniosis is differentiated as dry or moist or mildly ecthymatous. The dry form is more common in dogs while the moist form is prevalent in gerbils and other rodents which effects man.

In dry cutaneous form, the promastigote forms, introduced into the skin by sandflies (*P. papatasi* or *P. sergenti*) are taken up by the macrophages in which they multiply. After maturity, the resultant amastigote forms are released after the rupture of the cells and infect other cells. The first detectable lesions occur 3-6 weeks after the bite of the sandfly as a reddish papule which gradually develops into a crust, forming a shallow ulcer. The ulcer gradually enlarges and may reach several centimetres in diameter. Ulcers may coalesce and spread over a large area. In uncomplicated cases, ulcers may heal in 2-12 months, leaving a deeply pigmented depressed scar. The infection is seldom fatal. In dogs, skin lesions are similar to that in man.

Diagnosis: The organisms are detected in the epithelial and mononuclear cells by microscopic examination of the material taken from the edge of an ulcer or a local lymph node. Biopsy material from the proximal lymph gland maybe attempted in case organisms are not detected in ulcer scrapings. Culture of materials in NNN medium should also be made. Gel diffusion test and passive agglutination test are also used for diagnosis.

Treatment: Lithium antimony thiomalate is given as intramuscular injection, 1 ml raising by 0.5-2.5 ml in subsequent injections given on alternate days 4-6 times.

Control: The control measures as mentioned in visceral leishmaniosis should be employed in cutaneous leishmaniosis as well.

Lecture-4

Order: Trichomonadida
Family: Trichomonadidae

The body is usually pyriform with rounded anterior and pointed posterior ends. There is single nucleus in the anterior part of the body and anterior to which is blepharoplast from which arises 3-5 anterior flagella and recurrent posterior flagellum. The posterior flagellum runs along the undulating membrane and often extends beyond the body. An accessory filament maybe associated with it. A filamentous costa arises from the blepharoplast and runs along the base of the undulating membrane. A parabasal body (Golgi apparatus) lies posterior to the blepharoplast. A clear, rod like axostyle also arises from the blepharoplast and passes through the centre of the body to emerge from the posterior end. The anterior end of the axostyle is enlarged to form a capitulum. Anterior to the blepharoplast a pelta arises from the capitulum. There maybe various granules within or along the axostyle and costa.

The family has several genera and speciation is based on the number of anterior flagella. They are usually found in the digestive tract or may also be present in the reproductive and other organs.

Important genera are: *Tritrichomonas*, *Trichomonas*, *Trichomitus*, *Tetratrichomonas* and *Pentatrichomonas.*

Genus: *Tritrichomonas*

The members of this genus have three anterior flagella and are found in the digestive tract and genital organs of domestic animals.

Tritrichomonas foetus

The organism causes a genital disease called **bovine trichomonosis** or **bovine trichomonad abortion** in cattle. It may also occur in zebu, buffalo, pig, horse and deer but pathogenic only in cattle. The disease is widespread all over the world but its incident has now been reduced substantially with the introduction of artificial insemination and decrease of number of bulls kept for breeding. From India, bovine trichomonosis has been reported from West Bengal, Bihar, Rajasthan, Jammu region and Odhisha.

The organism is spindle to pear shaped and 10-25 × 3-15 µm in size. It has three anterior flagella and a posterior flagellum that trails as a free flagellum. It moves with a characteristic jerky movement. The undulating membrane runs along the full length of the body and there is an accessory flagellum. The costa is prominent. The axostyle is well developed and emerges from the end of the body through a chromatic ring. The anterior end of the axostyle is enlarged to form a capitulum. The basal body is 3-4 µm long and is sausage shaped. Multiplication of the parasites takes place through longitudinal binary fission. There is no sexual process or encystment.

Transmission: Bovine trichomonosis is transmitted by coitus under natural conditions. Artificial insemination, teaser bulls and gynaecological examination may become the source of the infection if no proper precautions are taken. Bulls, once infected remain a permanent source of infection. In cows, on the other hand, the infection is self limiting and the parasites may gradually disappear. In untreated cases, infected cows may also serve as a carrier of infection. Experimental infection is possible in laboratory animals.

Pathogenesis

Cows: The organism multiply primarily in vagina, causing vaginitis by third day post infection, which may become severe by ninth day. The initial lesions may vary in intensity which maybe slight as to pass unnoticed or it may produce a muco-purulent discharge. The organisms are most numerous in vagina by 14-18 days post infection. They invade the uterus through the cervix and may disappear from the vagina or may remain there producing low grade inflammation and catarrh. Slight reddening of the vulva and vagina may be seen.

Bovine trichomonosis in cows is characterised by early abortion. Rarely, despite the infection, the animal may conceive normally, retains the calf up to full term and gives birth to normal healthy calf. Abortion usually occurs 8-16 weeks after conception. If abortion occurs at a very early stage (one to two weeks of gestation) the foetus and membranes frequently pass unnoticed and this may lead to the assumption that there has been an irregular oestrus cycle. Abortions beyond six months of pregnancy are rare.

Placentitis with detachment of placental membranes and death of the foetus may lead to an abortion usually between 8-16 weeks after the infected service. It is followed by uterine discharge and the animal may show a series of irregular heat periods. In some cases, if the foetus and membranes are not completely eliminated, chronic catarrh or purulent endometritis occurs. When the cervix is closed and there is a retained corpus luteum, a closed pyometra occurs. The uterus may contain several litres of thin, greyish white fluid swarming with trichomonads. In the absence of bacterial contamination this fluid is almost colourless. There is associated anoestrus and the animal appears apparently pregnant during this period. In long standing cases, the trichomonads may disappear from the uterine fluid.

Bulls: The most common site of predilection of organisms is preputial cavity. Testes, epididymis and seminal vesicles may also be involved in some cases. Early clinical signs are pain during micturition and a disinclination to serve cows. A muco-purulent discharge maybe present. Small red nodules in the mucous surface of the prepuce maybe visible. Such signs diappear one to two weeks after the infection. In chronic cases, the swelling and pain subside. Spontaneous recovery is rare and infected bulls may remain permanently infected. In some bulls, clinical signs may not be apparent.

Diagnosis: The diagnosis of the bovine trichomonosis is based on the herd history, demonstration of the organisms and serological investigations. The herd history may include information on early abortions, more number of cows returning to the service and failure of animal to conceive except after repeated service, and an increases vaginal

discharge and incidence of pyometra in the herd. Infertility cases may increase with the introduction of the new bull into the herd.

The diagnosis can be confirmed by demonstration of the organisms in the vaginal or uterine discharge or in the foetus. Vaginal mucus or exudate or saline washings from the vagina and preputial cavity can be examined microscopically. Saline washings or irrigation materials from the uterus are settled for an hour or two, or centrifuged for examination of the sediments. Uterine discharges may also be collected from the cow and this is best done 2-3 days before the time of next estrus. In pyometra, the pus should be examined as it may contain plenty of *T. foetus*. Trichomonads are found readily in the mouth cavity and stomach contents of an aborted foetus, in the aminotic and allantoic fluids, and in the uterine discharges after abortion. Following abortion, the organisms often disappear within 48 hours. Care should be taken to avoid contamination of aborted materials with faeces. Samples should be kept warm throughout by adding saline. Low power objective of the microscope shows small moving objects. At higher magnification, characteristic features of the organisms can be observed.

In very light infection, the preparation should be stained or put in the cultures. Smears fixed in osmic acid vapours and stained with Giemsa give good results. Organisms may be cultured *in vitro* in Diamond's medium. Serological investigations include cervical mucus agglutination test and skin test.

At least three negative tests and two normal oestrus periods can indicate absence of infection in a cow. A bull is considered uninfected when six tests at weekly intervals are negative and at least two virgin heifers (free from infection) bred show no infection.

Immunity: The organisms persist in bulls for life and they remain a continuous source of infection for cows. In cows, there is spontaneous disappearance of the organisms from the genital tract and there is immunity to disease after 3-4 months of infection. In some cases, there maybe immunity to re-infection followed by one or more abortions. Despite of development of immunity to infection, permanent sterility may result if there has been extensive involvement of uterine mucosa.

Treatment:

Bulls: Infected bulls are difficult to treat. Pudendal anaesthesia is used first to relax the retractor penis muscle. The penis is thus washed with weak solution of detergent and dried. Flavin ointment is then applied in the preputial cavity and massaged for 15-20 minutes. Less than 1% solution of acriflavin is injected into the prepuce and retained for 15 minutes along with thorough massage of prepuce. One to two courses with five successive treatments per day eliminate the infection. Preputial washings with 3% hydrogen peroxide is also partially effective. Dimetridazole is also found effective when given parentrally @ 50 mg/kg body weight daily for five days. Similar dose is effective for cows.

Since the infection is self limiting and there is adequate immunity, animals should be given a breeding rest and subsequently artificial insemination should be done with clean uninfected semen. This will avoid infection to clean bulls.

Control: As the treatment is difficult and unsatistfactory, the following preventive methods may be adopted:

1. All infected bulls must be either castrated or slaughtered.
2. Use of untested bulls should be checked and only tested authorised bulls should be permitted for breeding.
3. Artificial insemination with proper care can reduce the incidence of the disease.
4. The aborted cows should be given breeding rest for three consecutive oestrus periods.
5. Proper maintenance of records of service dates, calving etc. This will help in early diagnosis and control.

Genus: *Trichomonas*

Trichomonas gallinae*:* occurs primarily in the upper digestive tract of pigeons, turkeys and rarely in chicken and other birds and causes avian trichomonosis. The organism is small, elongate, ellipsoidal or pyriform in shape and measure 6-19 µm × 2-9 µm. Axostyle is narrow and protrudes 2-8 µm from the body posteriorly. Costa is present as a fine rod. The undulating membrane does not reach the posterior end of the body. Typically four anterior flagella are present.

The disease occur in young birds through drinking water contaminated by infected birds. Nestling pigeons (squabs) get infected through infected crop milk of parent pigeon. The earliest lesions appear as small yellowish circumscribed areas in the oral cavity. The lesions later extend to the oesophagus, crop and proventriculus and form disc like thick, caseous, necrotic masses occluding the lumen and called **'yellow buttons.'** Usually lesions donot extend beyond proventriculus. Invasion of the liver by the organisms cause abscess and even death may occur.

Affected birds show depression, ruffled feathers, weakness and emaciation. Greenish fluid or cheesy material accumulates in the mouth and crop. Foul smell emits from the mouth. Organisms are plenty in mouth and crop. On post-mortem, necrotic lesions are seen in mouth, crop, oesophagus and liver. Recovered birds are immune to re-infection but survive as carriers.

Treatment: Furazolidone @ 25-30 mg/day orally for seven days in gelatine capsules is effective. 2-4 amino-5-nitrothiazole (Enheptin) @30-45 mg/day for seven days is effective to control the disease.

Tetratrichomonas gallinarum: occurs in lower digestive tract and sometimes in the liver of the turkey, domestic fowl and guinea fowl. It is cosmopolitan in distribution. The organism is pyriform. The axostyle is long and slender and projects from the posterior part of the body.

T. gallinarum produces liver lesions resembling those of histomonosis. Transmission occurs by ingestion of trichomonads in contaminated feed and water. The liver lesions are more irregular and raised above the liver surface instead of being depressed as in case of *Histomonas meleagridis* infection.

Lecture-5

Order Trichomonadida

Flagellate protozoa under this order are characterised by the presence of 4-6 flagella, one of which maybe a trailing flagellum attached to the undulating membrane. Multiplication occurs by binary fission. Cysts are produced by some forms. Most of the species are non-pathogenic and are found in the alimentary canal of animals.

Family: Monocercomonadidae

Species of this family have three to five flagella directed forward and one trailing flagella, which is usually free.

Genus *Histomonas*

The organisms are amoeboid with a single nucleus. The shape is spherical or elongated with one flagellum.

Histomonas meleagridis

It is found in the caeca and liver of birds and causes histomonosis, infectious entero-hepatitis or black head disease in turkey, chicken, peafowl, guinea-fowl, pheasant, partridge and quail. The disease is worldwide in distribution but quite uncommon in India.

The organisms are pleomorphic as the appearance depends upon the location, stage of the organism and stage of the disease. As such there are four forms of the parasite viz., invasive form, vegetative form, resistant form and flagellated form.

1. **Invasive form:** It is found in early caecal and liver lesions and at the periphery of the older lesions. It is amoeboid, extracellular and 8-17µm long. These forms feed by phagocytosis.
2. **Vegetative form:** It is found near the centre of lesions and in slightly older lesions. They are larger i.e. 12-15 µm×12-21 µm in size. They are less active than invasive forms and are packed tightly together and cause disruption of the tissue.
3. **Resistant forms:** These forms do not form cysts and are more resistant than others. Their sizes are 4-11 µm in diameter and are compact and seen to be enclosed in the dense membrane. These forms may occur singly or packed together and are extracellular. These maybe taken up by the phagocytes or the giant cells.
4. **Flagellated forms:** They occur in the lumen of the caeca. The same form is found in cultures. The body is amoeboid and 5-30 µm in diameter. A basal granule or blepharoplast is present near the nucleus from which the flagella arise. Commonly, there is a typical single short flagellum but there may be as many as four. There is no axostyle and undulating membrane.

Life Cycle and Transmission

The trophozoites are fragile and do not survive more than few hours outside the body. There is no cystic stage and the trophozoites multiply by binary fission. Transmission occurs through ingestion of the embryonated eggs of the caecal worm, *Heterakis gallinarum*. The protozoan has also been demonstrated in the larvae of *H. gallinarum.* The infective eggs hatch in the intestine and liberate *H. meleagridis*.

Infection is also possible by the ingestion of the protozoans freshly discharged from acutely infected turkeys. Earthworms can transmit both *Heterakis* worms and *Histomonas* organisms and are probably responsible for long duration survival of the *Histomonas* in the soil. Chicken is less susceptible than turkeys. Birds between 3-12 weeks of age show high mortality.

Pathogenesis and Clinical Signs

H. meleagridis organisms are released from embryonated *H. gallinarum* eggs or larvae. The organisms invade the caecal wall and multiply producing characteristic lesions. Later, they migrate through blood stream to liver. The principal lesions of histomonosis are observed in liver and caeca. The incubation period is 15-21 days. The first sign of the disease is droopiness. The birds appear weak, drowsy and stand with lowered head, ruffled feathers and drooping wings. There is sulphur yellow coloured diarrhoea. The origin of the name "blackhead" is obscure and misleading; birds may or may not display cyanosis of the head and wattles.

One or both caeca maybe involved and the earliest lesions consist of small-raised pin point ulcers in which large number of organisms are present. Gross lesions develop after 6-8 days, which become more enlarged and may involve whole of the caecal mucosa. The mucosa becomes thickened and necrotic and is covered with characteristic, foul smelling yellowish exudates forming a dry, hard cheesy plug that fills the caecum and adheres tightly to its wall. The caeca are markedly inflamed and often enlarged. Sometimes the ulcers perforate the caeca and cause peritonitis and adhesions. From caeca, the organisms enter the hepatic portal system and are carried to the liver. The pathognomonic lesions in liver are focal necrosis which produce circular, depressed, and yellow to yellowish green areas. Spleen, kidney and lungs may occasionally be affected.

Post-mortem lesions

There are typhlitis and hepatitis. Caeca are eroded, inflamed, distended and pouched out, lining with cheesy core like blood-stained mass. Perforation of the caeca leads to peritonitis and adhesions. The liver surface shows characteristic sunken, ulcer like areas with yellow tan colour borders with depressed reddish or white coloured centres.

Diagnosis

The characteristic sulphur yellow droppings in sick birds are suggestive of enterohepatitis. On post mortem, the lesions of the liver are pathognomonic. Lesions maybe confused with those caused by leukosis, tuberculosis or mycosis which can be differentially diagnosed by demonstrating *H. meleagridis* histologically. The caecal lesions maybe differentiated from those of *Eimeria tenella* and trichomonosis.

Treatment and Control

Thiazole derivatives like enheptin, acetylenheptin and nithiazide are quite effective against histomonosis.

Enheptin is given orally in feed or water. It should be given in feed @ 0.05% as preservative or 0.1% as curative.

Nithiazide is more effective and less toxic and is given @ 0.02% and 0.025% in feed and drinking water respectively.

Furazolidone is also quite effective in prevention and treatment @ 0.015% and 0.04% in feed, respectively.

Proper management, sanitation and preventive medication are more important than therapy alone. The following preventive measures should be followed for effective control of histomonosis:

1. Chicken should not be reared with turkeys.
2. Birds should be kept free from *H. gallinarum* infection by regular treatment.
3. Proper sanitation, regular removal of droppings, rotation of ranges at regular intervals should be undertaken.
4. Birds should not have access to earthworms.
5. Sick and infected birds should be segregated and treated.

Order Diplomonadida

Family: Hexamitidae

Organisms of this family are bilaterally symmetrical, with two nuclei and six-eight flagella.

Genus: *Hexamita* / *Spironucleus*

Hexamita meleagridis / Spironucleus meleagridis

It occurs in the duodenum and small intestine of young turkey, peafowl, partridge, chick, domestic duck and quail. The parasite may also occur in caecum and bursa of fabricius of the birds acting as carriers.

The organisms are pyriform, binucleate and measure 6-12 µm × 2-5 µm. The two nuclei contain rounded endosomes, occupying 2/3 the diameter of the nucleus. The six anteriorly directed flagella arise from blepharoplast, lying anterior to the two nuclei in two groups of three in each. The caudal flagella arise on each side from a distinct blepharoplast posterior to the large anterior blepharoplast and pass posteriorly emerging at the posterior end of the body.

Life Cycle:

The organisms divide by longitudinal binary fission and are transmitted through contaminated feed and water.

Pathogenesis:

Hexamitosis/Spironucleosis is a disease of young turkeys. It is spread by old birds acting as symptomless carriers. Wild birds like quails, pheasants and partridges may also act as source of infection. Hexamitosis occurs in birds kept in unhygienic condition. Hot weather and overcrowding predispose birds to this disease.

Turkeys up to 2 months of age are most susceptible. Hexamitosis in turkey poults is commonly called **infectious catarrhal enteritis**. The essential lesions are in the upper

digestive tract showing catarrhal enteritis. The intestinal contents are thin watery and foamy. Myriads of organisms are found in the bulbous part of the caeca.

Genus: *Giardia* (Grand old man of intestine)

Giardia species are pyriform to elliptical in shape and are bilaterally symmetrical. The anterior end is broadly rounded and posterior end is drawn out and somewhat pointed. The dorsal side is convex and there is a large adhesive disc on the ventral side. There are two anterior nuclei, two slender median axostyles, eight flagella arranged in four pairs and a pair of darkly stained body placed medially. Cysts are produced which are oval or elliptical and possess two or four nuclei and a number of fibrillar remnants of the trophozoite organelles.

The species of *Giardia* occurring in vertebrates have similar morphology. Though the parasite is euryxenous, but due to lack of cross transmission studies it is customary to name the species according to their hosts e.g. *Giardia intestinalis* (man), *G. canis* (dog), *G. muris* (rat), *G. cati* (cat), *G. chinchilla* (rabbit), *G. bovis* (ox), *G. caprae* (goat), *G. equi* (horse) and *G. caviae* (guinea pig). Parasites usually occur in the upper digestive tract of the host.

Giardia intestinalis causes giardiosis in man, monkey and pig and inhabits in duodenum, jejunum and upper ileum. It is worldwide in distribution. It is widely prevalent in man in India. The trophozoites measure 9-20 µm × 5-10 µm. The cysts are ovoid and retractile. Reproduction is by binary fission. Transmission is by ingestion of the cysts with contaminated food and water. The cysts remain viable in moist surroundings up to 2 weeks.

Giardia canis occurs in duodenum, jejunum and upper ileum of dogs. The trophozoite is 12-17 µm × 7-10 µm in size. In dogs, it causes diarrhoea and dysentery. The stool is soft, pale, mucoid and greasy. A poor hair coat is attributed to deficiency of fat soluble vitamins. Organisms may also occur in large numbers in apparently healthy dogs.

Diagnosis: Done by demonstrating cysts and trophozoites in the faeces.

Treatment: Metronidazole @ 250 mg twice daily for 10 days is also effective. Good hygiene and frequent check up help in the control.

Giardia cati: Occurs in small intestine of cat. Pathogenesis is unknown.

Giardia bovis: Occurs in small intestine of ox. The trophozoites and cysts are 11-19 µm × 7-10 µm in size and 7-16 µm × 4-10 µm in size, respectively.

Lecture-6

SUBPHYLUM: SARCODINA

The amoebae are classified under this subphylum. They possess primarily pseudopodia for movement and do not possess pellicle or cilia. Most amoebae are holozoic, ingesting their food (bacteria, protozoa and small metazoan) by means of pseudopodia. Cytoplasm consists of endoplasm having food vacuoles, nucleus and ectoplasm are relatively clear. Contractile vacuoles are absent in parasitic amoebae or those living free in salt water unlike fresh water forms. Usually, reproduction is asexual by binary fission. Most species form cysts. Only a few species are parasitic.

Family: Endamoebidae

This family includes parasitic amoebae which occur in the digestive tract of vertebrates and invertebrates and usually do not cause any serious disease in domestic animals except *Entamoeba histolytica* which causes amoebic dysentery in man. Cysts have 1-8 nuclei and may have rod like chromatoid bodies. Species of the genus *Entamoeba* have been divided into four groups on the basis of trophozoite and cyst morphology.

1. **Histolytica group (with quadrinucleate cysts)**: ***Entamoeba histolytica*** (man, other primates, dogs, cats and rarely pigs); ***Entamoeba hartmanni*** (man, other primates, dogs etc.); ***E. equi*** (horses)
2. **Coli group (with eight nucleate cysts)**: ***Entamoeba coli*** (man, other primates, dogs and pigs); ***E. wenyoni*** (goats); ***E. gallinarum*** (chicken, turkeys etc.); ***E. muris*** (rats, mice and rodents)
3. **Bovine group (with uninucleate cysts)**: ***E. bovis*** (cattle); ***E. ovis*** (sheep); ***E. bubalis*** (buffalo/caribou); ***E. suis*** (pig)
4. **Gingivalis group (with unknown number of cysts)**: ***E. gingivalis*** (man, other primates, dogs and cats); ***E. equibuccalis*** (horse); ***E. canibuccalis*** (dogs)

Entamoeba histolytica is the only pathogenic species infecting mammals particularly man.

Entamoeba histolytica:

This species cause amoebic dysentery in man, but young dog, cats and mice are also highly susceptible. Other hosts are monkeys, pigs and rats which may serve as sources of infection to man. Besides, gorilla, chimpanzee, gibbon, languor, many species of macaques, baboons, spider monkeys etc. act as reservoirs. Natural infection is quite common in monkeys. Rats, guinea pigs and hamsters can be infected experimentally. *E. histolytica* rarely infects cattle.

Morphology:

The trophozoite of *E. histolytica* measures from 10-60 µm, the size being larger in freshly voided dysenteric stools than in stools of an asymptomatic carrier. The trophozoites move by its pseudopodia and the movement is rapid in warm environment. The pseudopodia are finger like, appearing suddenly and the endoplasm flowing rapidly into them. The spherical nucleus is 4-7 µm in diameter and is indistinct in living amoebae. Trophozoites also possess food vacuoles containing ingested erythrocytes. The encystations of the trophozoites start with the dehydration of the faeces in the lumen of the colon. The trophozoite discharges undigested food and condenses into a spherical mass called the cyst. The cyst matures by mitotic division

of the nucleus and form four nuclei. Rarely eight nuclei maybe found. The cysts can survive for several days at 4-8˚C. These are sensitive to putrefaction, desiccation and temperature above 40˚C or below -5˚C. They can also pass unharmed through the digestive tract of the filth flies. Chlorination of water does not kill the cysts, but 5% acetic acid kills them in a few minutes.

A small form or race of the organism which is non pathogenic is named *E. hartmanni*, while the large on (*E. histolytica*) maybe divided into an avirulent and a virulent race.

Life cycle and transmission:

The trophozoites multiply by binary fission. Before encysting, the amoeba rounds up, eliminates its food vacuoles and become smaller. The cyst wall develops and the nucleus divides into two and then four nuclei. On ingestion, cysts excyst in the intestine and the released metacystic forms undergo nuclear and cytoplasmic divisions to produce eight uninucleate amoebae which pass to the large intestine and grow into larger forms and invade bowel mucosa.

The infection is transmitted through food and water contaminated with cysts, as trophozoites do not survive for more than half an hour after passing of stools. Eating of raw and unclean vegetables also cause infection. Lack of personal hygiene, use of water polluted by night soil, contamination of food by flies, handling of food and water by contaminated hands are other common modes of transmission of *E. histolytica* cysts.

Pathogenesis:

Amoebiasis in man is a common and serious disease caused by the large forms of *E. histolytica. Escherichia coli* or *Aerobacter aerogenes* play an important role in the growth and invasive character of the amoeba. Disease maybe acute or sub-acute, but chronic infections are most common. The parasite penetrates the intestinal wall by lyses of the epithelium. The amoebae multiply in epithelium forming small colonies and then penetrate into the submucosa. They invade deep into the mucous membrane and produce inverted flask shaped ulcers, having a narrow canal or neck opening into bowel lumen. Amoebae are mainly found at the periphery of the ulcer and the cavity of the ulcer is filled with necrotic tissues.

Amoebae may reach other parts of the body through lymph or blood vessels. In liver, it may produce inflammation and abscesses. Active amoebae maybe found in the wall of the abscess. Amoebae through blood maybe carried to lungs and brain.

Clinical Symptoms:

Secondary complicated cases show fever. In acute amoebic dysentery, the faeces consist almost entirely of blood and mucus with amoebae. There is severe abdominal pain with straining. In chronic cases, recurring symptoms may continue for a long time. There is occasional abdominal pain, nausea, flatulence, irregular bowel, headache and fatigue. The symptoms stimulate appendicitis and peptic ulceration. Rectal straining may cause prolapsed of the rectum. In monkeys, *E. histolytica* usually does not invade the intestinal wall and there are no symptoms but the infection maybe of public health significance. Other non human primates suffer from clinical condition. Pigs show similar symptoms. Dogs may have lesions in the caecum and mostly without symptoms, but may suffer, at times, from acute or chronic amoebiosis. Kittens may show acute dysentery with extensive intestinal ulceration.

Diagnosis:

Diagnosis can be done by gross and microscopic examination of freshly discharged faeces, clinical symptoms, radiography and various serologic and skin tests. Cultivation of amoebae on artificial media may also help.

Treatment:

Metronidazole is the drug of choice @ 750 mg orally thrice daily for 5-10 days. It is effective against intestinal and extra-intestinal infections. Tinidazole is considered superior to metronidazole.

Control:

Amoebiosis can be controlled by adoption of proper sanitation, personal hygiene, supply of clean drinking water, proper disposal of sewage and night soil, prevention of faecal contamination of food by flies and cockroaches etc.

PHYLUM: CILIOPHORA

CLASS: KINETOFRAGMINOPHOREA

***Balantidium coli*:**

The species occur worldwide in caecum and colon of pig, man, monkey, chimpanzee and other non human primates and dogs. These species cause balantidial dysentery in man and other hosts. The species is most common in pigs.

B. coli is the largest protozoan parasite parasitizing man. The species has a trophozoite and a cystic stage. The trophozoites are ovoid or pear shaped or occasionally sub cylindrical. The body surface is covered with slightly oblique longitudinal rows of cilia. The peristome is subterminal in the narrow end which leads to a tiny mouth called as cytostome. The posterior end has an excretory organelle called cytopyge. The trophozoite has two nuclei; the large macronucleus is kidney or sausage shaped. The micronucleus is small and lies in the depression of the macronucleus.

Life cycle:

Infection occurs either by ingestion of the infective cysts or trophozoites of *B. coli*. The pig is the usual source of infection for man. The parasites multiply by transverse binary fission. Conjugation also takes place.

Pathogenesis:

In pigs, the parasite lives almost as a commensal in the lumen of the large intestines and usually does not penetrate the mucosa of the intestine. However, if some lesions develop due to some other reason, the ciliates act as opportunists and invade and may be found deep in the ulcer. It produces enzyme hyaluronidase which may cause enlargement of lesions.

In humans, B. coli is pathogenic and produces diarrhoea and dysentery.

Diagnosis:

It is based on the identification of the cysts in the faeces or by histological examination of the intestinal lesions.

Treatment:

It is not necessary in pigs. In humans, metronidazole, paramycin and imidazole are used.

Lecture-7

PHYLUM: APICOMPLEXA

Class: Sporozoea

All the members under this class are parasitic and spore forming. This group of protozoa is distinct because of the apical complex generally consisting of polar ring present in some stage, and a conoid, micronemes, rhoptries, micropores and subpellicular tubules as revealed by electron microscopy. There is a single nucleus. Cilia and flagella are generally absent except that the microgametes of some groups are flagellated. Reproduction is both asexual (schizogony) and sexual (gametogony). After the formation of zygote, there is spore formation (sporogony).

There are two subclasses, **Coccidia** and **Piroplasmia**, with one order under each, **Eucoccidia** and **Piroplasmida**, respectively.

Order Eucoccidia has three sub-orders

1. Eimeriina with three families, Eimeriidae, Cryptosporiididae and Sarcocystidae
2. Haemosporina with one family, Plasmodiidae
3. Adeleina with two families Klossiellidae and Haemogregarinidae

Order Piroplasmida has two families, Babesiidae and Theileriidae

Family: **Eimeriidae**

Organisms of this family are commonly called **coccidia**, are typically intracellular parasites of the intestinal epithelial cells of the vertebrates in general with few exceptions. All forms have a single host in which they undergo asexual (schizogony or merogony) or sexual (gametogony) cycles of reproduction. A zygote results from the union of the male gamete (microgamete) and the female gamete (macrogamete) and develops independently inside the epithelial cells of the intestine of the host and leaves the host after developing a protective cystic wall in the form of an oocyst. This stage, outside the host, undergoes a process of sporogony. A variable number of spores (sporocysts) contain one or more sporozoites.

Tyzzeria **(asporocystic octasporozoic)**: after sporogony, the oocyst contains eight free sporozoites, without any sporocyst.

Isospora / Cystoisospora (**bisporocystic tetrasporozoic**): after sporogony, the oocyst contains two sporocysts with each containing four sporozoites.

Eimeria (**tetrasporocystic bisporozoic**): after sporogony, the oocyst contains four sporocysts with each containing two sporozoites.

Wenyonella (**tetrasporocystic tetrasporozoic**): after sporogony, the oocyst contains four sporocysts with each containing four sporozoites.

Cryptosporidium **(asporocystic tetrasporozoic**): The species of this genus possess four sporozoites, not enclosed in a sporocyst. They differ from species of other genera and develop just under the surface membrane of the host cell or within its brush border instead of within the cell.

The coccidian species in domestic livestock and birds are generally host specific, especially of ***Eimeria*** genus, which are in majority. All species under the family Eimeriidae have different developmental stages inside and outside the host.

The stage which comes out of the host in all the cases is an oocyst with a zygote. The **oocysts** are usually oval, egg shaped, ellipsoidal, sub-spherical or spherical. The cyst wall is generally bilayer, clear and transparent. The outer layer maybe lightly coloured and smooth, scabrous or spiny. The inner layer is usually thinner. There maybe small thinning at the narrower pole called **micropyle**. It maybe covered by a cap like structure of different shapes called as **micropylar cap or polar cap.**

Sporulation:

It begins after the oocysts have been discharged by the host. Inside the unsporulated oocyst, the cytoplasm of the uninucleate zygote is usually coarsely granular which usually fills the entire space in the beginning and later it contracts into a ball (**sporont**) leaving some surroundings. Some dark granules maybe seen near the micropyle as **polar granules**. Sporogony starts with the sporulation of the oocysts which begins with the division of the sporont either into two **sporocysts** (*Cystoisospora* / *Isospora*) or into four small sporocysts (*Eimeria*, *Wenyonella*). In some species, the part of the protoplasm, left after the spore formation, is called **oocystic residuum**. **Stieda body** is present at the more pointed end of each sporocyst in some species. Each sporocyst then divides to form two or four banana shaped elongated bodies called **sporozoites**. The sporozoites are usually sausage or comma shaped or banana shaped with a central nucleus.

The sporulation of the oocysts, under natural conditions is affected by certain factors like type of soil, exposure to direct sunlight, amount of humus in the soil, moisture, atmospheric temperature etc. a temperature of 29±3˚C with sufficient moisture and oxygen is considered optimum for sporulation. The sporulation time, in different species of coccidia varies from few hours to few days depending upon the atmospheric temperature. In general, sporulated oocysts are more resistant to desiccation and cold and may survive for up to two weeks at a temperature of 12-20˚C. Unsporulated oocysts are killed within 96 hours at these temperatures.

Infection and Asexual Reproduction:

Infection is transmitted to the host by ingestion of numerous oocysts through water or feed. Freshly released sporozoites show contraction, elongation and gliding movements. The process of penetration of host's cell takes only in a few seconds.

Schizogony/ Merogony (asexual cycle) commences after the entry of the sporozoites in the host cells which can be either epithelial cells of villi, crypts, bile ducts, kidney tubules, endothelial cells lining the lacteals inside the core of the villus or in the connective tissue cells. Inside the cells, the sporozoite becomes spherical, which is known as trophozoite. Within few days, the nucleus of each trophozoite divides by multiple fission. Later the cytoplasm surrounds each daughter nucleus, resulting in the production of a number of elongate uniform bodies, called as merozoites. The number and size of the merozoites in each first generation schizont / meront varies in different species. Each mature schizont is enclosed in a thin distinct wall containing 8 to many hundreds or thousands of merozoites of banana shape with centrally placed nucleus. When division is completeand the meront is mature, the

host cell and the meront rupture and the merozoites escape to invade neighbouring cells. The parasitised host cell is generally enlarged, distorted and protrudes in different directions. The nucleus of the host cell is seen hypertrophied and shifted to one side during the early phase of schizogony. The number of subsequent generations of schizonts maybe up to four (*Eimeria acervulina* and *Eimeria mivati*). Mature schizonts are mainly of two types:

1. Giant schizonts: are always the first generation schizonts of 100-700 µm in diameter (*E. ovina, E. bovis* etc.). These can be seen grossly on the mucosal surface as pin-point cyst like bodies, each containing millions of merozoites.
2. Epithelial schizonts: are microscopic and recognised as (a) Large Epithelial schizonts: containing few hundred merozoites (*E. tenella, E. necatrix*) and (b) Small Epithelial Schizonts: containing approximately 8-36 merozoites (*E. maxima, E acervulina*)

Gametogony (Sexual Phase) and oocyst formation:

It starts when merozoites, liberated from the second or subsequent generation schizonts, enter new cells of the host to develop into male and female gamonts. Most of these merozoites turn into female gametocytes called as macrogametocytes (macrogamonts) which mature into macrogametes. The young macrogamonts develop equal to the size of oocysts. A few of the merozoites become male gametocytes called microgametocytes (microgamonts). The young microgamonts develop in the same way as macrogamonts except that the nucleus undergoes multiple divisions with the production of a large number of comma-shaped microgametes which accumulate on the periphery of the cell, leaving the residuum of the cytoplasm. The microgametes are tiny slender, slightly bent with two flagella for locomotion. Hundreds of the liberated microgametes fertilize the macrogametes. The resultant zygote develops to oocyst with the formation of oocyst wall. The oocysts break out of their host cells, enter the intestinal lumen and pass out in the faeces. In the absence of reinfection, coccidial infections are self-limiting. Sporogony begins after the oocysts have been discharged by the host.

The prepatent period is the time between the ingestion of the first infective oocyst to the time when first oocyst appears in the faeces. It varies from few days to few weeks in different species. Oocysts continue to discharge for a number of days. The patent period is the time from the first discharge of the oocysts to the time of the last discharge in the faeces of the host after single infection. It also varies from few days to few weeks in different species. In the absence of re-infection, coccidial infections are self-limiting.

Coccidia of Cattle and Buffalo

Thirteen Eimerian species of coccidia are recognised to parasitise the intestines of cattle and water buffalo in India and many other countries.

1. ***Eimeria alabamensis*:** occurs primarily in the small intestine but also has been located in the caecum and upper colon of cattle. It is an intranuclear parasite of the intestinal epithelial cell. It is worldwide in distribution.
2. ***E. auburnensis*:** occurs in the lower third of the small intestine of cattle and buffalo. It is fairly common and worldwide in distribution. Oocysts are ovoid or ellipsoidal with a narrow micropylar end.

3. ***E. bareillyi:*** has been described from water buffalo in India. Oocysts are pyriform having narrow end with truncated or slightly flattened surface. Oocyst's wall is smooth. Micropyle is present.
4. ***E. bovis***: one of the commonest species which occur in cattle and buffaloes and has worldwide distribution. The species is most commonly involved with *E. zuernii* in clinical coccidiosis in cattle.
5. ***E. brasiliensis:*** is the only capped Eimerian species of cattle and buffalo reported from most of the countries of the world. Micropylar cap is present. Oocysts are ellipsoidal or ovoidal.
6. ***E. bukidnonensis*:** is the biggest among all the Eimerian species found in cattle and buffalo. Oocysts are pyriform or pear shaped to oval. Oocyst wall is dark yellowish to brown with interrupted radial striations and spotted or speckled with irregular refractile dark protuberances.
7. ***E. canadensis*:** occur in cattle and buffalo all over the world. Oocyst wall is light yellow in appearance. Sporocysts are elongate and ovoid.
8. ***E. cylindrica*:** occurs in cattle and buffaloes in most of the countries. Oocysts are cylindrical or sub-cylindrical or elongated ellipsoidal. The oocyst wall is smooth.
9. ***E. ellipsoidalis***: occurs in cattle and buffalo throughout world. Oocyst wall is smooth and is ellipsoidal to slightly ovoid.
10. ***E. pellita***: is known only from the oocysts from bovine in Australia and England and is not common.
11. ***E. subspherica***: is the smallest Eimerian species found in the small intestine of cattle and buffalo. The species is widely prevalent. Oocysts are typically spherical to occasionally spherical to bluntly ovoid.
12. ***E. wyomingensis***: is worldwide in distribution. Oocysts are ovoid or egg shaped. Oocystic wall is thick, yellowish brown in colour with uniformly punctuate or rough layer.
13. ***E. zuernii***: occurs commonly in small and large intestines of cattle and buffalo. Oocysts are spherical to sub-spherical, bluntly ellipsoidal or ovoidal. Sporocysts are elongate-ovoid.

Coccidiosis in cattle and buffalo

Coccidiosis in cattle and buffalo is primarily a disease of young animals, particularly calves of 3 weeks to 6 months of age. Older calves and adult animals are also affected if the infection is heavy. Adult animals mostly act as a source of infection for susceptible young animals. Under natural conditions, the disease with a single species of coccidium is rare and mixed infections are common.

The commonly occurring species of *Eimeria* in cattle and buffalo are: *E. auburnensis, E. bovis, E. ellipsoidalis* and *E. zuernii*. Of these the most pathogenic species are *E. zuernii* and *E. bovis*. Concurrent parasitic or other infections also play a role in the clinical course of bovine coccidiosis.

Transmission of the infection to calves occurs through ingestion of the sporulated oocysts with feed and water. The severity of the disease mainly depends upon the number of oocysts they ingest besides other factors influencing the susceptibility of the animals. Overcrowding and lack of sanitation increase chances of transmission and infection of large number of

calves occur when kept with adults. Mortality maybe considerably high after abrupt climatic changes.

Clinical Symptoms:

E. zuernii is considered to be the most pathogenic species. Initially intestinal catarrah is manifested by watery faeces for 1-2 days with an extremely unpleasant odour which soils the posterior parts of the sick animals. In severe cases animals may not survive. Repeated infections result in chronic disease characterised by alternate discharge of bloody and bloodless pasty mucoid faeces. Anorexia, rapid respiration, convulsions, emaciation, tremors and weakness are other symptoms of chronic disease and animals may die of pneumonia. **Winter coccidiosis** in animals is primarily attributed to *E. zuernii*.

In *E. bovis* infection, the enormous number of merozoites swarming out from the giant schizont and gametogonic stages developing from them give rise to intestinal bleeding of varying durations, bloody diarrhoea and copious oocyst discharge. Recurrent infections may result in aggravation of the large intestinal lesions.

In mixed infections, the symptoms are characteristic of the disease. In acute cases, there is abdominal pain, foul smelling diarrhoea with or without blood and masses of mucus and clots of blood. Other symptoms include rough coat, drooping ears, listlessness, soiled hind quarters, anorexia, tenesmus, inability to rise and partial paralysis of anal sphincters. The last stage of the disease is of variable duration in which the animals become too weak to rise and refuse food and water.

Lesions:

On post-mortem, there is thickening and petechiation of the bowel but mucosal scrapings will reveal masses of gamonts and oocysts. Giant meronts may be seen in the mucosa of the small intestine as pin-point white spots, but unless they are present in vast numbers, they cause little harm. The most pathogenic stages are the gamonts.

Diagnosis:

Bovine coccidiosis can easily be diagnosed by its characteristic symptoms like bloody diarrhoea, loss of appetite, fever, debility etc. In *E. bovis* infection the oocysts are always demonstrable in the faeces. Oocysts of *E. zuernii* appear in faeces soon after clinical peak has been reached. Coccidiosis in young suckling calves should be differentiated from bovine dysentery and in older calves with anthrax. Confirmation of the diagnosis of acute coccidiosis is done by detection of characteristic intestinal lesions on post mortem examination of recently died calves.

Treatment:

Since, the signs of the disease do not become noticeable until it is advanced. Hence, treatment in most of the cases is ineffective. Of the range of coccidiostats, sulphonamides are the most frequently tested against bovine coccidiosis.

1. Amprolium: The most effective drug against 1st generation Giant schizonts. It is given @ 10 mg/kg body weight/day in feed and water for 5 days for treatment and @ 5 mg/kg body weight/ day in feed and water for 3 weeks for prevention.

2. Sulfamethazine: @140 mg/kg body weight/ day in feed for three days for treatment and @ 5 mg/kg body weight/ day in feed for 15 days for prevention.
3. Monensin: @ 1mg/kg body weight/ day or 0.006% in feed for 5 weeks for prevention.
4. Sulfaquinoxaline: @ 13mg/kg body weight/ day in feed for 2-3 weeks for prevention.
5. Nitrofurazone: is used as curative drug @ 15 mg/kg body weight/ day orally for 7 days.

Prevention:

General hygienic measures are essential for keeping the animals from infection. Frequent faecal examination of different age groups of animals and treatment positive cases may keep the herd free of disease. However, complete eradication of coccidiosis from the domestic animals is neither practicable nor advisable. Calves should be isolated within 24 hours after birth. Growing calves should not be kept with adult animals. The intake of sporulated oocysts can be reduced by avoiding contamination of feed and water with infected faeces and by washing floors and lower parts of the walls of the byres at regular intervals. Beddings and surroundings maybe sterilized with 1.25% sodium hypochlorite or 0.5% cresol or phenol by fumigation with formaldehyde. The floors of the byres should be kept dry as much as possible.

Coccidia of Sheep and Goat

Different Eimerian species have been recovered from different parts of the world from sheep and goats. These are as follows:

Sheep: *Eimeria ahsata, E. crandallis, E. faurei, E. granulosa, E. intricate, E. ovina, E. ovoinoidalis, E. parva and E. gilruthi*

Goats: *E. alijevi, E. asphreronica, E. arloingi, E. caprina, E. christenseni, E. hirci, E. jolchijevi, E. kochari, E. ninakohlyakimovae* and *E. gilruthi*

Coccidiosis in sheep and goat:

Coccidiosis is one of the commonest and economically important disease of lambs and kids up to 6 months of age. The species of clinical importance are: *Eimeria ahsata, E. ovina, E. ovoinoidalis* and *E. parva* in sheep and *E. alijevi, E. arloingi, E. christenseni* and *E. ninakohlyakimovae* in goats. Other species contribute in enhancing clinical severity of the disease with pathogenic forms in heavy infections.

Coccidia of Domestic Pig

Eimeria debliecki, E. neodeblieki, E. perminuta, E. polita, E. porci, E. scabra, E. spinosa and Cystoisospora suis are the most common coccidian species of domestic pigs recorded from India.

Eimeria debliecki: Occurs commonly in the small intestines of domestic pigs and wild boars worldwide. The oocysts are ellipsoidal to ovoid and measure 20-23µm × 14-29µm and have a smooth wall. Micropyle is absent.

E. neodebliecki: Worldwide in distribution. The oocysts are ellipsoidal or ovoid and measure 17-26 µm × 13-20 µm. Oocyst wall is smooth and sporocysts are elongate or ovoid.

E. perminuta: Worldwide in distribution. The oocysts are spherical to sub-spherical and measure 12-20 µm × 9-17 µm. The oocyst wall is rough. The oocyst wall is rough. The sporocysts are ellipsoidal to broadly ovoid.

E. polita: Occurs in small intestines of pigs worldwide. The oocysts are ellipsoidal or ovoid and measure 22-39 µm × 17-26 µm. The oocyst wall is generally smooth or slightly rough.

E. porci: The oocyst measures 18-27 µm × 13-18 µm in size.

E. scabra: It is found in the small intestine and possibly in caecum and colon of domestic pigs and wild boar throughout the world. The oocysts are ellipsoidal or ovoid and measure 25-45 µm × 18-28 µm. The oocyst wall is yellowish brown, rough and striated. The sporocysts are ovoid.

E. spinosa: Occurs in small intestine. The oocysts are ovoid or ellipsoidal and measure 14-26 µm × 12-21 µm in size. The cyst wall is brown, opaque and studded with spines. The sporocysts are elongate-ovoid.

Cystoisospora suis: Occurs commonly in small intestine of domestic pigs throughout the world. The oocysts are spherical to sub-spherical and measure 17-25 µm × 16-21 µm with a smooth thin wall. The two sporocysts are ellipsoidal.

Pathogenesis:

Pigs are commonly infected with coccidian species but clinical signs are not frequently seen. Mostly young animals suffer showing signs of enteritis. Older pigs remain as asymptomatic carriers. *Eimeria debliecki* and *E. scabra* are the most pathogenic species. The clinical signs include diarrhoea, emaciation and constipation. Crowding and lack of sanitation greatly increases the chances of severe diarrhoea. Repeated infections lead to immunity.

Diagnosis:

It is based on the clinical signs and the detection of oocysts in the faeces. On post-mortem endogenous stages could be looked for confirmation. The lesions are commonly confined to small intestines which show catarrhal haemorrhagic inflammation with distension of blood vessels. Lesions of *E. spinosa* may reach up to large intestine.

Treatment and Control:

1) Nitrofurazone @ 0.44% in the feed for seven days
2) Amprolium @ 25-65 mg/kg body weight once or twice daily.
3) Other drugs of choice are sulphadimethyl pyrimidine and sulphamethazine.

Control measures include improved hygiene, cleanliness of pens, better feeding and avoidance of overcrowding. Affected animals should be segregated.

Coccidia of Equine

Eimeria leuckarti: Biggest size coccidian oocyst occurring in small intestine of horse and jackass in Europe, North America and Indian subcontinent. The oocysts are ovoid or pyriform, somewhat flattened at the small end and measure 70-90 µm × 49-69 µm in size. The oocyst wasll has an outer layer 5-9 µm thick, dark brown, opaque and granular and inner

layer 1 µm thick and colourless. Micropyle is distinct and micropylar cap is absent. Oocyst residuum and polar granule are absent.

Pathogenesis: Diarrhoea, weight loss and even death has been reported in heavy infections.

E. solipedum: Occurs in domestic horses, mule and donkey in Russia. The oocysts are distinctively orange red or yellowish brown in colour with no micropyle.

E. uniungulati: Occurs in domestic horse and mule.

Klossiella equi: Occurs in horse, ass, donkey and zebra from North America, Europe, Turkey and Australia. Oocysts occur in the epithelial cells of the loop of Henle.

Coccidia of Rabbit

Thirteen Eimerian species have been recognised to parasitize the intestine and one species (***Eimeria stiedae***) parasitizes the liver of rabbits. Most of the species occur in India. Some of them are highly pathogenic. *Eimeria elongate, E. exigua, E. intestinalis, E. coecicola, E. flavescens, E. magna, E. media, E. nagpurensis, E. neoleporis, E. perforans, E. piriformis, E. matsubayashii, E. irresidua* are the species recorded from the intestines of the rabbits.

***E. stiedae*:** It is of historical importance in protozoology as oocysts of this species were seen by Leeuwenhoek in 1674 in the gall bladder of an old rabbit which made it the first parasitic protozoan observed. The oocysts are ovoid to ellipsoidal, flattened slightly at the micropyle end and measure 31-42 µm × 17-25 µm in size. The species parasitizes the wall of the bile ducts in the liver of domestic rabbits.

Pathogenesis: Young rabbits are most susceptible to coccidiosis. The disease at times appears as an outbreak where intensive breeding and rearing are done under poor sanitation.

Liver coccidiosis: also known as hepatic coccidiosis. The clinical symptoms usually appear 12 days after the infection but the rabbits may die without showing obvious signs in heavy infections. In mild infections, there is only temporary decrease in weight gain, resulting from functional disorder of the liver. Massive infection shows inappetence, diarrhoea or constipation. Advanced coccidiosis is accompanied by icterus and also fever particularly in secondary infections.

Intestinal coccidiosis: The disease is quite frequent. Only exceptionally a coccidium free rabbit maybe found. It is difficult to determine the exact species causing clinical infection due to frequent mixed infections. Although, *E. irresidua* and *E. magna* are considered as primary etiological agents but also *E. media, E. intestinalis* and *E. piriformis* may be pathogenic. The clinical signs in intestinal coccidiosis include indigestion, diarrhoea, bloat, listlessness, inappetence and reduced weight gain. Rabbits either die suddenly without showing symptoms or show convulsions and sometimes paralysis. In massive infection, eosinophilia is common.

The intestine shows acute or sub-acute catarrhal inflammation which seldom becomes chronic. The intestinal wall becomes thickened and the mucosa is seen covered by greyish white deposits. The intestinal contents are always pasty and may contain streaks of blood and mucus.

Diagnosis: It is based on the demonstration of oocysts in the faeces. Post-mortem examination of the dead animals confirms the diagnosis.

Treatment:

Preventive Treatment:

1. Sulphaquinoxaline @ 0.025% in feed for 30 days or 0.1% for two weeks
2. Sulfachloropyrazine @ 0.1% in water for 1-2 months old rabbits
3. Monensin @ 0.005-0.02% in feed.

Therapeutic Treatment:

1. Sulfamethoxine @ 200mg/kg b.w. orally on first day followed by 100 mg/kg b.w. orally for 4 subsequent days
2. Sulphaquinoxaline and sulfamerazine @ 0.03% in drinking water for 10 days. Both are most widely used drugs against hepatic coccidiosis in rabbits.
3. Toltrazuril @ 2.5% in water for 2 days is effective in hepatic and intestinal coccidiosis.

Control of coccidiosis in rabbits mainly depends upon the hygienic measures and regular prophylactic dosing/ treatment.

Coccidia of Camel

There are only three species of coccidia reported from Indian camels:

Eimeria cameli: It is probably common species reported worldwide. It occurs in small intestine of dromedary and Bactrian camels. The oocysts are truncate-ovoid, 81-100 µm × 63-94 µm in size. The oocystic wall consisting of three layers is very thick, brown and opaque. Micropyle is present. The sporocysts are elongate and pointed at both the ends.

E. dromedarii: Most commonly observed in camels of India, Pakistan and Iraq. The oocysts are ovoid with brown coloured oocystic wall.

E. rajasthani: It has been described from the faeces of dromedary camel calves in Rajasthan. The oocysts are ellipsoidal. The sporocysts are ovoid and sporozoites are comma shaped.

Coccidia of Domestic Fowl

The following species have been reported from the domestic fowl in India and other parts of the world: *Eimeria acervulina, E. necatrix, E. maxima, E. mivati, E. mitis, E. brunetti, E. tenella, E. praecox* and *E. hagani*.

E. acervulina: It is most commonly found in domestic fowl throughout the world. It occurs in the epithelial cells of the anterior portion of small intestine mainly the duodenum of the chicken. The oocysts are ovoid with smooth wall and measure 12-23 µm × 9-17 µm in size.

E. brunetti: Occurs in the mucosa of the lower portion of the small intestines, caecum, rectum and cloaca of the chicken throughout the world. The oocysts are ovoid and measure 14-34 µm × 12-36 µm in size. Oocyst wall is smooth.

E. hagani: It is not very common and occurs in the anterior part of the small intestines of chicken in India and North America. The oocysts are broadly ovoid and measure 16-21 µm × 14-19 µm in size.

***E. maxima*:** It is worldwide in distribution and occurs in mucosa of the middle of the small intestines of the chicken. The oocysts are ovoid, egg shaped or ellipsoidal and measure 27-24 µm × 16-28 µm in size. Oocysts discharged in the early stage of the infection are probably smaller than those passed later. The oocyst wall is slightly rough and yellowish.

***E. mitis*:** It is found throughout the world and occurs mostly in the anterior half of the small intestines of fowl. The oocysts are sub-spherical and measure 10-21 µm × 9-18 µm in size.

E. mivati: It occurs in the epithelial cells of the small intestines and may extend from duodenum to rectum. The developmental stages are mostly observed in the duodenal loop and other parts of the small intestine. The oocysts are ellipsoidal to broadly ovoidal and measure 11-20 µm × 10-15 µm in size. Oocyst wall is colourless and smooth.

E. necatrix: It occurs in jejunum, midgut, caecum and other parts of large intestine. The oocysts are broadly ovoid and measure 15-25 µm × 14-20 µm in size. The oocysts wall is smooth.

E. praecox: It occurs in the upper third of small intestine. The oocysts measures 20-25 µm × 16-20 µm in size.

E. tenella: It is the most common and highly pathogenic coccidium of domestic fowl throughout the world. It is responsible for heavy economic losses and is the primary etiological agent for producing caecal coccidiosis as its all developmental stages occurs in the caecum of the chicken. The oocysts are broadly ovoid and measure 14-31 µm × 9-25 µm in size.

Pathogenesis:

Coccidiosis affects all the ages of the birds and is ubiquitous in distribution. The clinical entity is required to be distinguished from sub-clinical infection. The mere presence of oocyts in the droppings of the older birds (carriers) has little significance because symptoms of the disease, particularly in younger birds may appear even before the discharge of the oocysts. The lesions in coccidiosis are often due to mixed infections. The post mortem lesions and the detection of typical endogenous stages in the intestinal lining are of diagnostic value. Clinical outbreaks are generally observed after monsoon. However, clinical cases including chronic form of the disease may be seen throughout the year.

Of all the coccidian species infesting poultry, *Eimeria tenella* is the most pathogenic, followed by *E. necatrix*. *E. brunetti* maybe markedly pathogenic but is not so common. *E. mivati, E. acervulina* and *E. maxima* are also common but hold moderate pathogenicity.

Susceptibiliy of domestic fowl to different species of coccidian is related to age groups. *E. tenella* is most commonly encountered in 4 weeks old chicks, while 2 weeks old chicks are comparatively resistant. Chicks less than 1 week old are more susceptible to *E. brunetti*. *E. necatrix* infection commonly occurs in birds of six week age and above. *E. maxima* can cause severe disease in chicken of 1 month age and above. *E. acervulina* is quite common in young and old birds.

Clinical Symptoms:

1. **Intestinal coccidiosis:** In *E. acervulina* infection, there is watery diarrhoea without blood. The malabsorption of feed leads to stunted growth and there is decline in egg

production. In *E. mivati* infection, the chicken shows listlessness, anorexia and watery diarrhoea. There is stunted growth, decrease in egg production and impaired feed conversion. In *E. maxima,* the droppings contain more mucus and occasionally contain flakes or streaks of blood. It occurs mostly in older chicken and laying hens. In *E. necatrix*, both acute and chronic forms of clinical disease occur. The acute form occurs less often and show droppings stained with blood. Death occurs usually 5-7 days post infection.

2. **Caecal coccidiosis:** *E. tenella* is the most pathogenic coccidium and causes most severe disease of poultry rated next only to that caused by *Salmonella pullorum*. Acute and peracute cases of caecal coccidiosis prove invariably fatal but the chronic cases may go unnoticed. Initially the chicken droop, stop feeding and may continue to take water, pass large quantities of blood in droppings (red diarrhoea), become anaemic and die. Some of the recovered birds may show paralysis. In cases, having mixed infections with *E. necatrix*, the symptoms maybe more aggravated.
3. **Rectal coccidiosis:** *E. brunetti* inhabits regions of the lower small intestine to cloaca. The affected birds pass white fluid droppings mixed with blood and mucus casts. There is loss of body weight due to severe dehydration and complete inappetance or reduced feed intake.

Lesions: The coccidian lesions in the fowl are characteristic to different species, but may vary depending upon the stage of the life cycle. Generally, the birds in a flock suffer from mixed infections. The lesions can be differentiated easily by examining different parts of the intestine. Microscopically, suspended lesions on the mucous membrane reveal characteristic endogenous stages of individual species.

In *E. acervulina* infection, upper part of the small intestine especially duodenum shows greyish white pin-point foci or streaks running transversely in the intestinal mucosa. The thickened intestinal mucosa shows catarrhal exudates without haemorrhages.

In *E. maxima* infection, middle portion of the small intestine becomes flaccid and dilated. The inflamed mucosa is seen covered with orange or pinkish coloured mucoid exudates. In *E. mivati* infection, the intestinal mucosa reveals oedematous petechiae. The lesions are similar to those of *E. acervulina*, while in *E. mivati* infection, the greyish white mucosal lesions, representing the colonies of gamonts and oocysts are circular and petechial haemorrhages may also be evident.

In *E. necatrix* infection, the main lesions occur in the middle small intestines. On gross examination of the serosal surface of the small intestine, pin point to pin head sized greyish white foci are observed. These white spots may coalesce to become large ones due to accumulation of second generation schizonts located deep into the mucosa.

In E. *tenella* infection, petechial haemorrhage is evident in caecum initially especially during first three days which become haemorrhagic by fourth day. The caecal pouch becomes greatly enlarged by sixth day to the size of a thumb and the contents become consolidated forming a dark brown to blackish caseous mass (caecal core) containing faeces, necrosed cellular debris and blood. Presence of caecal core is quite characteristic of caecal coccidiosis.

In *E. brunetti* infection, the lesions are characteristically confined to the posterior part of the intestines. The gut wall is thickened and covered with catarrhal exudates. The intestinal lumen maybe filled with blood tinged mucus and sloughed necrotic material.

Diagnosis: For correct and early diagnosis of coccidiosis in domestic fowl, knowledge of history, characters of various types of lesions and their locations and important features of the life cycles different coccidian species are essential. Diagnosis by faecal examination alone is of little significance in severe clinical disease and in outbreaks as the mortality starts well before the discharge of the oocysts. However, after the acute phase, a large number of oocysts could be detected in faeces. In outbreaks, the diagnosis is confirmed by sacrificing one or more moribund cases to locate the characteristic lesions and endogenous stages, if the clinical symptoms are suggestive of coccidiosis.

Treatment: Mortality of the birds due to coccidiosis could be checked by timely and proper use of prophylactic and therapeutic treatments with drugs, singly or in combination.

The emergence of drug resistant strains of coccidia is major problem in the treatment of this disease which limits the use of most of the drugs. A plan to avoid drug resistance which includes switching around thirteen classes of drugs in a grow-out of birds' flock is called as shuttle programme, which is generally adopted.

Sulphonamides: The common drugs of this group are sulphaquinoxaline, sulphadimethoxine, sulphaguanidine, sulphadimidine and sulphanitran. They have a coccidiostatic action and stop the onset of disease by acting against the schizontal stage especially the second generation schizonts of *E. tenella* and *E. necatrix*.

Sulphaquinoxaline: @0.5% in feed is used as therapeutic whereas Sulphadimidine is given in the feed @0.4%.

Amprolium: It is most effective against *E. tenella*, *E. necatrix* and *E. acervulina*. The spectrum of activity of amprolium is extended and strengthened in combination with ethopabate, sulphaquinoxaline and pyrimethamine. It is available as premix and is given prophylactically to birds @ 0.012%.

Nitrofurazone: It is used for therapeutic purpose @ 0.22%, but if this level is continued for more than 10 days, toxic effects like nervous signs maybe seen.

Furazolidone: @ 0.011% in feed.

Salinomycin: Has significant anticoccidial activity @ 0.01% in feed.

Halofuginone: If used at a concentration of 3 ppm, the drug is effective against pathogenic Eimerian species.

Other important drugs include Polyether ionophorous antibiotics (such as Monensin, Lasalocid and Salinomycin), Nicarbazin, Nitrophenide, Toltrazuril and nucleoside analogues etc.

Control: In general, mortality due to coccidiosis in poultry can be controlled by any of the drugs. Anticoccidial drugs are commonly used in starter rations for broilers raised under floor pen management. Other methods of prevention include:

A) Immunoprophylaxis: Various methods have been employed to induce sufficient immunity to provide flock protection against mortality and morbidity in poultry against this disease. Acquired immunity is built up in chicken by repeated successive exposure to small numbers of oocysts. A suspension of live oocysts containing a mixture of eight species, is given in the feed or drinking water to chicks at 4 and 14 days of age. Although the primary exposure starts the process of immunization, development of substantial immunity depends upon oocysts production arising from initial exposure as a source of infective oocysts that reinfect the birds. A number of vaccines have also been introduced from time to time containing varying number of oocysts strains for the development of immunity in the birds.

1. **Coccivac:** It consists of eight *Eimeria* spp. of chickens. Birds are administered the vaccine at 3 days of age and low-level medication is started at about 13 days of age and continued until the birds are 5-6 weeks of age. It is reported that these chickens develop a solid immunity by 4 weeks of age.
2. **CoxAbic:** It is a subunit commercial vaccine against coccidiosis. It consists of purified gametocyte antigen of *Eimeria maxima* and used to control coccidiosis in chicks up to 7 weeks of age by maternal immunization
3. Likewise, there are other vaccines as well such as **CoxATM, Paracox, Livacox, Immunocox** etc. which are used to provide immunity to birds.

B) Genetic resistance of the host: The control programme of coccidiosis through selective breeding is best applicable to the domestic fowl.

C) Nutritional supplementation: Nutritional status of the birds is also important in the control programme of coccidiosis. Supplementation with some vitamins may prove beneficial in this disease. Vitamin A though does not reduce morbidity or mortality but promotes host's recovery. Vitamin K reduces mortality due to *E. tenella* and *E. necatrix* infection where mortality is due to excessive haemorrhages.

D) Sanitation and management practices: The primary objective of sanitation is to prevent chicken from coming in contact with faeces containing oocysts. Although prophylactic medication has become the major method of coccidiosis control but this alone is entirely inadequate in preventing outbreaks and sanitation practices are still necessary in modern control operations. Avoidance of deep litter system, regular prophylactic treatments, litter management, dryness of the floors, induction of immunity through immunoprophylaxis, most frequent removal of droppings where large flocks are maintained are some of the most important steps to prevent outbreaks of coccidiosis in birds.

Coccidia of Duck and Geese

Eight species of coccidia have been reported from domestic geese and duck and some of these have been associated with severe disease and death.

The species of importance are: *Eimeria anatis, E. anseris, E. battakhi, E. kotlani, E. parvula, E. saitame, E. stigmosa, E. truncata, Tyzzeria anseris, T. perniciosa, Wenyonella anatis, W. gagari* etc.

Coccidiosis of duck and geese: very limited information regarding clinical and economic importance of coccidia in duck and geese is there. With the exception of *Eimeria truncata* (where development occurs in kidney tubules) which may cause 100% mortality in acute cases of renal coccidiosis, the other species appear to be

important only in sporadic cases, presumably where the environment is overcrowded and unhygienic.

Coccidia of Cats and Dogs

Dog

Five species of genus *Cystoisospora* (syn. *Isospora*) are found in dogs that are monoxenous. Coccidia of dogs also include one species of *Hammondia* and nearly two dozen species of genus *Sarcocystis*, that are heterogenous involving one or more vertebrates as intermediate hosts. Important species are *Cystoisospora burrowsi, Cystoisospora canis, Cystoisospora neorivolta* and *Cystoisospora ohioensis*.

Cat

Coccidia of cat include two species of *Cystoisospora*. The other coccidia are from family Sarcocystidae which include about ten species of genus *Sarcocystis*, two each of *Besnoitia* and *Hammondia*. The other one of the most important parasites is *Toxoplasma gondii*.

Pathogenesis: Dog and cat usually harbour coccidian parasites but rarely suffer from a severe disease. Though experimental evidence does not indicate any coccidian species to be pathogenic, clinical disease may occur especially in kennels where sanitation is poor and animals are crowded together. At times, blood maybe passed in the faeces or diarrhoeic syndrome maybe there with the passage of large number of oocysts. Species of heterogenous genera may be responsible for clinical disease.

Diagnosis: Based on clinical signs and on the presence of oocysts in the faeces. Young puppies and kittens are more prone to infection. During post-mortem examination a large number of developmental stages may be detected in mucosa of intestines.

Lecture-8

Family: Sarcocystidae

There are two subfamilies: Sarcocystiniae and Toxoplasminiae

Genus: ***Toxoplasma***

Oocysts with two sporocysts, each with four sporozoites and are facultative or obligate heteroxenous. Felines act as definitive hosts. Merogony occurs in both intermediate and definitive hosts. Schizonts and gametocytes occur in the enteric cells of felines. Sporogony occurs outside the host. A single species is recognised i.e. *Toxoplasma gondii.*

Toxoplasma gondii

Definitive hosts: Domestic cat, jaguarondi, ocelot, mountain lion, leopard cat and bobcat.

Intermediate hosts: There is little host specificity and almost all warm-blooded animals, including man as intermediate host.

Geographical distribution: Worldwide in distribution and prevalence varies according to climatic region, presence of cats etc. The name *Toxoplasma* (toxon-arc, plasma-form) is derived from crescent shape of the 'tachyzoite' stage. The type definitive host is the domestic cat beside other felids viz., jaguarondi, ocelot, mountain lion, and leopard cat.

Structure and life cycle: *Toxoplasma gondii* is an obligate intracellular parasite transmitted in three primary ways viz., congenital, carnivorism and faecal oral route. The resistant oocysts in the cat faeces ensure dissemination of the parasite.

There are three infectious stages of *Toxoplasma* etc., the tachyzoites (in pseudocysts), the bradyzoites (in tissue cysts) and sporozoites (in oocysts). The term bradyzoites (brady means slow in Greek) and tachyzoites (tachos = speed in Greek) have been coined to describe the slowly and rapidly dividing forms respectively, multiplying in any cell of the definitive host. Host cells containing numerous crescent shaped tachyzoites are called as clones or groups or pseudocysts. The term pseudocyst has been used because there is no well-defined parasitic membrane covering the groups of tachyzoites. A cyst is a collection of bradyzoites within a well-defined parasitic membrane. Gametes are the products of gametogony occurring in the intestinal cells of the definitive host which shed unsporulated oocysts.

Development in definitive hosts: The primary process of infection of a feline is by ingesting prey containing tissue cysts, or oocysts containing sporozoites excreted by another feline. After ingestion of the cysts, the cyst wall is dissolved by the proteolytic enzymes in the stomach and small intestines. The released bradyzoites penetrate the epithelial cells of the small intestines and then turn into schizonts. During schizogony, successive nuclear divisions and cytoplasmic divisions occur resulting in the formation of merozoites inside the host cell. Rupture of these enterocytes releases many merozoites, which are also able to infect new enterocytes and divide again by schizogony. Each schizogonic cycle gives rise several merozoites that will be released to readily invade new enterocytes, exponentially enhancing the number of parasites. Gametocytes occur throughout the small intestines but most commonly occur in the ileum, 3-15 days after infection. Mature male gametocytes are oval to ellipsoidal in shape and few in number. The microgametes penetrate mature macrogametes. An oocyst wall forms around the fertilized gamete. Oocysts are discharged into the intestinal

lumen by rupture of the intestinal epithelial cells when they are mature. Unsporulated oocysts are sub-spherical to spherical. Sporulation occurs outside the host within 1-5 days. Sporulated oocysts are sub-spherical to ellipsoidal.

Development in intermediate hosts: The intermediate hosts become infected by ingesting sporulated oocysts or infected meat of animals or congenitally through placenta. After ingestion, the oocysts rupture in the intestine, releasing 8 sporozoites. Sporozoites multiply intracellularly in the intestinal epithelium and in associated lymph nodes and tachyzoites are formed. These spread to the rest of the body through lymph and blood and eventually encyst in brain, skeletal and cardiac muscles and liver. A tissue cyst is the collection of the bradyzoites surrounded by a well-defined host cell membrane. These are microscopic and survive in tissues as long as the host lives.

On ingestion of the infected meat having tissue cysts of *T. gondii*, proteolytic enzymes dissolve the cyst wall, releasing the bradyzoites. After entering the host cells, the bradyzoites transform into the tachyzoites. In the host cells, tachyzoites may undergo repeated divisions ultimately encysting in tissues. The cycle of *T. gondii* is completed when tissue cysts are ingested by the cats.

Transplacental infection can occur when a previously non infected host becomes infected during pregnancy. *T. gondii* multiplies in the placenta and then spread to the foetal tissue. Although transplacental infection can occur at any stage of the gestation, the foetus is affected more severely if the dam becomes infected during the first half of the gestation. Although, *T. gondii* can be transmitted by transfusion of the platelets and blood cells and organ transplants, these modes are less common than oral routes. However, fatal toxoplasmosis may result from organ transplant if the patient is provided with immunosuppressive therapy.

Toxoplasmosis in domestic animals:

Pathogenesis: Severity of toxoplasmosis varies from an inapparent infection to an acute or fatal disease. All species of animals are not equally susceptible. Asymptomatic toxoplasmosis is most common and rarely does it cause any serious clinical manifestation. A host may die due to necrosis of the intestines and mesenteric lymph nodes before other organs are severely damaged. Necrosis is caused by the intracellular growth of tachyzoites.

Cattle: Fatal toxoplasmosis has not been reported in cattle.

Sheep: Abortion occurs in ewes of all age groups which acquire infection during pregnancy. Less than 1% of ewes abort in subsequent pregnancies. Lambs maybe mummified, macerated, aborted, still born or may born weak or die within a week of their birth. The ewes which abort return to heat and conceive normally.

Goat: The parasitaemia develops within one week and *Toxoplasma gondii* encysts in the subsequent week. There may be fever, anorexia, diarrhoea and respiratory distress and goat may die of enteritis. Abortion may occur at any point of the time within three weeks of the infection. *T. gondii* can cause early embryonic death, mummification, abortion, still-births or birth of weak kids in goats.

Pigs: *T. gondii* infection in pigs is highly prevalent in most countries. The main source of infection from the environment is through ingestion of the infective oocysts. Pigs can be

infected with tachyzoites through parenteral route. The porcine toxoplasmosis is more important because of pork containing tissue cysts being one of the major sources of human infection. *T. gondii* has been isolated from the diaphragm, abdominal muscles, heart, brain, stomach, large intestines, lungs, liver, spleen, kidney, lymph nodes and small intestines.

Equines: are generally resistant to clinical toxoplasmosis.

Dogs: Clinical toxoplasmosis in dogs with mortality has also been reported. Fatal toxoplasmosis has been reported in young dogs having concurrent infection with canine distemper virus due to immunosuppression. Clinical signs are divided into four major forms: generalised, neuromuscular, respiratory and gastrointestinal.

The generalised form is characterised by intermittent fever, tonsilitis, dyspnoea, diarrhoea and vomiting. The neuromuscular form is characterised by presence of lesions in brain and spinal cord with the symptoms of progressive paresis and may last for several weeks. The respiratory form may be fatal within a week, while there is diarrhoea in gastrointestinal form. There are few reports of congenital toxoplasmosis in canine.

Cats: Clinical toxoplasmosis in cats is a rare occurrence. Congenital toxoplasmosis has been confirmed in cats. There may be anorexia, lethargy, pneumonia, icterus, vomiting, fever, diarrhoea and occasional encephalitis. Retinitis may result in one or both eyes.

Diagnosis: It can be done only on the basis of clinical signs which are non-specific. It is usually made by biological, serological and histological techniques.

T. gondii can be isolated from the infected animals from secretions, excretions, body fluids and tissues. Diagnosis can be made either by finding *T. gondii* in host tissues or inoculation in laboratory animals and tissue cultures. A rapid diagnosis may be made by microscopic examination of impression smears stained with Giemsa technique. Immunohistochemical staining can confirm the diagnosis within few hours.

Sabin-Feldman dye test has been employed for the diagnosis of the toxoplasmosis in humans. Apart from this various serological test involving IHA, IFA, MAT, LAT, ELISA and CF have been used.

Treatment: Sulphadiazine and pyrimethamine are two widely used drugs for the treatment of toxoplasmosis. Recently, a live toxoplasmosis vaccine ‘Toxovax’ has been introduced in U.K. to protect ewes from toxoplasmosis, which are intended for breeding. The vaccine contains live tachyzoites of S48 strain. Dose is 2 ml intramuscularly in the neck region. Given to sheep from the 5th month onwards or 4 months prior to mating. Not used in sheep less than 6 weeks of age and in pregnant ones.

Lecture-9

Genus: *Neospora*

Species*: Neospora caninum*

Neospora caninum misidentified and misdiagnosed as *Toxoplasma gondii* till 1988, when it was first recognised in the dogs in Norway as a new genus and species. It causes a serious disease in cattle and dogs worldwide. Abortions and neonatal mortality are the major problems encountered in cattle.

N. caninum is a coccidian parasite with a wide host range. In general, it is very similar to structure and life cycle of *Toxoplasma gondii* with most important difference: Neosporosis is primarily a disease of cattle and dogs, whereas toxoplasmosis is primarily a disease of humans, sheep, goats and cats. Like *T. gondii*, the life cycle is typified by three known infectious stages: tachyzoites, tissue cysts and oocysts. The first two stages are found in the intermediate hosts (herbivores) and they occur intracellularly. The environmentally resistant stage of the parasite is oocyst that is excreted through the faeces of dogs. Oocysts sporulate outside the hosts in as few as 24 hours.

Carnivores probably get infected by consumption of tissues of herbivores containing tissue cysts (bradyzoites) and herbivores by the consumption of infected water and fodder with sporulated oocysts. Transplacental infection can occur when tachyzoites are transmitted from infected dam to her foetus during pregnancy. Neonatal calves may also become infected by the ingestion of colostrum and milk containing tachyzoites.

In dogs, neosporosis is the most severe disease of young congenitally infected pups. Puppies / young dogs develop an ascending paralysis with the hind limbs more severely infected than the forelimbs. There is difficulty in swallowing due to paralysis of jaw.

Detection of antibodies in serum or individual or bulk milk samples of cattle by techniques like Indirect Fluorescent Antibody Test (IFAT) and various ELISA tests are optimal for the identification of the infected herds. **Neoguard** was a commercial vaccine developed to reduce abortion in pregnant cattle resulting from infection with *Neospora caninum* and was available in the USA, New Zealand and some other countries.

Genus: *Sarcocystis*

Asexual stages occur only in the intermediate hosts which in nature is often a prey animal. Sexual stages develop only in the definitive host which is carnivorous (predator). There are different intermediate and definitive hosts. The parasites derive their name from the intramuscular cyst stage (sarcocyst) present in the intermediate (prey) host.

Sarcocysts (in Greek, Sarcos = flesh, Kystis = bladder) are the terminal stages found encysted, primarily in the striated muscles of mammals, birds and poikilothermic animals (intermediate hosts). These have also been found in the CNS and Purkinje fibres of the heart and muscle bundles, though in relatively low numbers. Sarcocysts vary in shape and size depending on the species of the parasite. These are either microscopic (e.g., *S. cruzi* of cattle-dog cycle) or macroscopic (e.g., *S. fusiformis* of buffalo-cat cycle). Macrosarcocysts are almost always found in the skeletal muscles or oesophageal muscles and appear filamentous,

globular, spindle or like rice grain. Micro-sarcocysts vary from very long and narrow to small and wide. Sarcocysts are always located in parasitophorous vacuole in the host cell cytoplasm. More than one cyst may be found in the host cell.

Histologically, the sarcocyst wall may be smooth, hirsute, striated or may possess complex branches protrusions. Internally, group of zoites may be divided into compartments by septa. Immature sarcocysts contain globular parasites called metrocytes (mother cells). Each metrocyte produces two progenies by an internal form of multiplication called endodyogeny. Some of the metrocytes, through the process of endodyogeny, produce banana shaped zoites called bradyzoites. Metrocytes are generally located in the cortex whereas bradyzoites are located in the medulla. Live sarcocysts are probably fluid filled with the metrocytes and bradyzoites moving within.

Host specificity: The species of *Sarcocystis* are more hosts specific for their intermediate hosts than for their definitive hosts. For example: *S. cruzi* ox and bison are the only intermediate hosts but dog, wolf, coyote, jackal and fox can act as definitive hosts. However, none of the species transmissible to dogs are transmissible to cat and vice-versa.

Life Cycle: The definitive host gets infection by ingesting the muscular or neural tissues containing mature sarcocysts. Bradyzoites are liberated in the stomach and intestines. Bradyzoites move actively and penetrate the mucosa of the small intestines, transforming into male (micro) and female (macro) gamonts. Micro and macrogamonts develop as early as 6 hours of ingestion of the infected tissues in approximately 95:5, respectively. The gametogonic stages either develop in goblet cells at or near the surface of the intestines or in enterocytes next to the basement membrane adjoining lamina propria. After fertilization, a wall develops around the zygote and oocyst is formed which, unlike those of most other enteric sporozoans, sporulate within the body. Two sporocysts are formed, each containing four sporozoites. Usually the fragile oocyst wall ruptures and free sporocysts are found in the faeces.

The intermediate hosts get infected by ingesting sporocysts during grazing on contaminated pastures or through feed or water contaminated with faeces of dogs, cats or man or related species of animals containing sporocyts. On reaching the intestines, sporozoites are released. They penetrate the intestinal wall and reach the mesenteric lymph nodes, arteries. The route of migration is quite unclear. First generation schizonts develop in the endothelial cells of small blood vessels of various organs. All species infect herbivores, from second generation schizonts in vascular endothelium of different organ/tissues including brain and spinal cord. Merozoites liberated from second generation schizonts initiate sarcocyst formation in striated muscles including cardiac muscles in different body parts. These intracellular merozoites in muscle cells first become round to ovoid. After repeated asexual divisions, the sarcocyst is filled with crescent shaped bradyzoites which become the infective stages on maturation for the definitive hosts. Sarcocyst may persist in the body of the intermediate host for the whole life or may start disappearing after 3 months. Generally, microsarcocysts belong to such species of *Sarcocystis* which have the definitive hosts as canids, man or other primates, while the species having macrosarcocysts involve felids as definitive hosts.

Sarcocystosis in different animals:

Cattle: There are three species of *Sarcocystis* found in cattle: - *S. cruzi* (cattle-dog cycle), *S. hirsuta* (cattle-cat life cycle) and *S. hominis* (cattle-man life cycle). *S. cruzi* is the most pathogenic species of cattle.

Water buffalo: There are two species of importance: *S. fusiformis* (buffalo-cat cycle) and *S. lavinei* (buffalo-dog cycle).

Sheep: There are four species of *Sarcocystis* in sheep: *S. tenella* (sheep-dog cycle), *S. arieticanis* (sheep-dog cycle), *S. gigantea* (sheep-cat cycle) and *S. medusiformis* (sheep-cat cycle). *S. tenella* is highly pathogenic for lambs.

Goat: Two species of *Sarcocystis* are infecting the goats' viz., *S. capracanis* (goat-dog cycle) and *S. hircicanis* (goat-cat cycle). *S. capracanis* is the most pathogenic species in goats and may even lead to abortions.

Pigs: There are three species of *Sarcocystis* reported from pigs, viz. *S. miescheriana* (pig-dog cycle), *S. suihominis* (pig-human cycle) and *S. porcifelis* (pig-cat cycle). *S. suihominis* hold significant zoonotic potential.

Equine: Five species of *Sarcocystis* have been named from equids: S. *neurona, S. bertrami, S. equicanis, S. fayeri* and *S. asinus*. *S. neurona* is responsible for causing Equine Protozoal Myeloencephalitis (EPM).

Control:

Interruption of the life-cycle is the key method to minimize the spread of sporocysts. This can be performed by:

1. Keeping away carnivores from animal houses and their feed, water and bedding;
2. Uncooked meat or offal should never be offered to carnivores;
3. Dead animals should be buried or incinerated to avoid access to carnivores;
4. Use of therapeutic and prophylactic anticoccidial drugs.

Family: Cryptosporidiidae

The family represents a single genus *Cryptosporidium*, the species of which parasitizes gastrointestinal and respiratory epithelium without formation of the tissue cysts in a wide range of animals including human beings. Several species of the genus have been named and described from the fish, reptiles, birds and mammals. At least 22 species of *Cryptosporidium* have been classified, based on host occurrence, parasite morphology, host predilection and site of infection. But, only 18 species are considered to be most commonly occurring validated species. *C. parvum* is the most commonly reported species in numerous species of mammals including humans. Various pets possess different species of *Cryptosporidium* and are responsible for communicating the disease conditions to human beings.

Species	**Host Range**
C. parvum	*Mus musculus* (mouse), *Bos taurus* (cattle), *Homo sapiens* (humans), *Sus scrofa* (pig), *Ovis aries* (sheep), *Capra hircus* (goat), *Equus caballus* (horse)
C. hominis	*Homo sapiens* (humans)
C. muris	*Mus musculus* (mouse), *Homo sapiens* (humans)
C. nasorum	*Naso lituratus* (fish)

C. molnari	*Sparus aurata* and *Dicentarchus labrax* (fish)
C. meleagridis	*Meleagridis gallopavo* (turkey), *Homo sapiens* (humans)
C. baileyi	*Gallus gallus* (chicken)
C. serpentis	*Elaphe guttata* (corn snake), *E. subocularis* (rat snake), different species of lizards
C. wairi	*Cavia porcellus* (guinea pig)
C. felis	*Felis catis* (cat), *Homo sapiens* (humans)
C. canis	*Canis familaris* (dog), *Homo sapiens* (humans)
C. andersoni	*Bos taurus* (cattle)
C. saurophilum	*Eumeces schneideri* (lizard), different species of snakes, different species of lizards

The life cycle of most *Cryptosporidium* species is completed within the gastrointestinal tract (primarily small intestine and colon) of the host, with developmental stages being associated with the luminal surface of the mucosal epithelial cells. *Cryptosporidium* oocysts are spherical, measuring only 3-6 µm in diameter. Thick-walled oocysts are excreted from the infected host in faecal material and represent the infective stage of the parasite. Infection of *Cryptosporidium* in a new host results from the ingestion of these oocysts, which release sporozoites that invade the epithelial cells and undergo asexual and sexual reproduction to produce thin-walled and thick-walled oocysts. Thin-walled oocysts can excyst endogenously, resulting in **autoinfection,** which helps to explain the mechanism of persistent infections (in AIDS patients) in the absence of successive (thick-walled) oocyst exposure.

Cryptosporidium infection is easily transmissible due to:

1. Large number of oocysts are being shed by the hosts;
2. Only a small infective dose is required i.e. only a few oocysts are capable of transmitting the infection;
3. Thick walled oocysts are hardy and hence can withstand chlorination easily;
4. Environmental factors have limited effect on hardy wall of thick walled oocysts.

Cryptosporidium has a typical coccidian life cycle in the brush border of the infected epithelial cells and differs from that of other coccidian protozoans (*Eimeria* and *Cystoisospora* species) in various aspects. These include, recycling from the type meronts, the production of thin walled auto infective oocysts, sporulation of the oocysts within the host (oocysts lack sporocysts) and the location of the parasitophorus vacuole containing the **intracellular extyracytoplasmic stage** of the *Cryptosporidium* in the brush border of the enterocytes. After ingestion of the sporulated oocysts (4 sporozoites), the sporozoites invade the microvillus brush border of the enterocytes and the trophozoites rapidly differentiate to form schizonts with 4-8 merozoites. Two types of schizonts develop. The first type of schizonts develops up to 8 merozoites, which recycle, producing either more first type schizonts or second generation of schizonts. The second type of schizonts produces four merozoites, which become gametocytes. Microgamont is wedge shaped and contains compact nucleus. It produces 14-16 microgametocytes. Macrogamont is spherical and contains large nucleus. Oocysts sporulate within the host and contain four sporozoites. The oocysts are produced in 72 hours. The oocysts are minute, spherical and colourless. Two types of oocysts are formed. The thick walled oocysts are passed in the faeces, whereas the thin walled ones excyst within the gut and release their sporozoites causing autoinfection. The extensive

recycling of merozoites from the type first schizont or autoinfection within the hosts is responsible for massive number of *Cryptosporidium* in clinically affected hosts.

Transmission: Transmission of *Cryptosporidium* infection is direct by ingestion of sporulated oocyst through contaminated food and water. Cross infection can occur between domestic, laboratory animals and man. Autoinfection also takes place due to excystment of thin wall cyst within the gut of the host. Transmission of cryptosporidiosis is frequently from person to person, animal to animal, animal to man or through indirect routes like contaminated water and food. *Cryptosporidium* oocysts are extremely resistant to a variety of commonly used disinfectants, but infectivity of oocysts can be destroyed after heating to 73˚C for one minute.

Pathogenesis: *Cryptosporidium* is not strictly host specific. Isolates of humans are infectious to a variety of mammals. *Cryptosporidium* infects primarily the intestinal epithelium but maybe disseminated to epithelial cells of other organs e.g. bile duct or the respiratory tract. In clinically overt disease, the main symptoms are profuse water diarrhoea and abdominal pain/discomfort. Young animals are most commonly susceptible for the infection. Pathogenesis mainly includes mild to moderate villous atrophy, degeneration and sloughing off of the enterocytes, crypt hypertrophy and inflammatory changes involving sub-epithelial lamina propria and Peyer's patches. In the respiratory tree there may be excessive mucous.

Diagnosis: Oocysts may be demonstrated using Ziehl‑Neelsen stained faecal smears in which the sporozoites appear as bright red granules. Speciation of Cryptosporidium is difficult, if not impossible, using conventional techniques. A range of molecular and immunological techniques has been developed that includes the use of immunofluorescence or ELISA. More recently, DNA ‑ based techniques have been used for the molecular characterisations of *Cryptosporidium* species**.**

Treatment and Control: There is no known treatment against Cryptosporidiosis. Quinolones, sulphaquinoxaline and halofuginone lactate have shown some therapeutic effect. Immunocompetent individuals recover by themselves.

Family: Haemogregarinidae

Genus: *Hepatozoon*

Hepatozoon canis: Occurs in canines involving dogs, jackals, hyena, also in palm civets and cats throughout the world. It was first reported in India in 1905.

Description: Gamonts, found in circulating neutrophils, are ellipsoidal in shape, about 11 by 4 μm, and are enveloped in a thick membrane. Meronts are usually round to oval, about 30 μm in diameter, and include elongated micromerozoites with defined nuclei, which in cross-section have a 'wheel-spoke' appearance.

Life Cycle: The life cycle involves two hosts. The tick is a final host in which syngamy occurs, and the dog is an intermediate host in which asexual reproduction occurs. Nymphal ticks engorge with gamont-infected leucocytes in an infected dog. Following ingestion the gamonts are freed from the leucocytes, associate in pairs and transform into male and female gametes leading to the formation of zygotes and oocysts. Each mature oocyst contains

numerous sporocysts, each containing 10–26 sporozoites. After the tick moults, oocysts are found in the haemocoel and each tick may carry thousands of infective sporozoites. Since the sporozoites remain in the body cavity, the dog is apparently infected by ingesting the tick. Once ingested the sporozoites are released from the oocysts, penetrate the intestinal wall and are transported to target tissues and organs in the blood and lymph. They primarily infect the spleen, lymph nodes and bone marrow where merogony/schizogony occurs in macrophages and endothelial cells. Two forms of meronts/schizonts are found in infected tissues, one type containing two to four macromerozoites, and a second type containing 20 elongated micromerozoites. When the meront matures and ruptures, merozoites are released and penetrate circulating neutrophils, in which they develop into gamonts that circulate in peripheral blood. The cycle is completed when the tick ingests infected blood. The period of development in the dog from infection to the appearance of gamonts is about 28 days.

Pathogenesis: Organism maybe found in apparently healthy dogs. There may be a mild infection associated with a limited degree of inflammatory reaction. However, infection may vary from asymptomatic in dogs with a low parasitaemia, to life-threatening in animals that present with a high parasitaemia. High parasitaemias can cause direct injury to the affected tissues and affect the immune system, leading to extreme loss of weight and cachexia, although infected dogs may maintain a good appetite.

Clinical signs: Irregular fever, anaemia, weight loss and progressive emaciation. There is enlargement of spleen. Lumbar paralysis also occurs. Death occurs 4-8 weeks after the onset of clinical signs.

Diagnosis: Diagnosis is usually made on the identification of gamonts in the cytoplasm of neutrophils in Giemsa or Wright's stained blood smears or the schizonts in the biopsy from the spleen or the bone marrow.

Treatment: Infection is treated with imidocarb dipropionate 5‑6 mg/kg every 14 days until gamonts are no longer present in blood smears. Oral doxycycline 10 mg/kg daily for 21 days in combination with imidocarb may also be used.

Lecture-10

Suborder: Haemosporina

The genera under this suborder resemble in their development with coccidia under the suborder Eimeriina except that the schizogony occurs in a vertebrate and gametogony and sporogony in a blood sucking arthropod.

Family: Plasmodiidae

The schizonts occur in red blood cells and also in the endothelial cells of the blood vessels of inner organs, macrophages, fibroblast and parenchymal cells of liver of vertebrate hosts. Thus, schizogony = vertebrate host and gametogony and sporogony = invertebrate vectors.

Three genera of importance: *Plasmodium, Haemoproteus* and *Leucocytozoon*

Genus: *Plasmodium*

- Schizogony occurs in RBC's and endothelial cells of internal organs.
- Sexual phase occurs in blood sucking insect vectors.
- Vector for avian malaria are culicine mosquitoes and mammalian malaria are anopheline mosquitoes.

Avian Malaria

Various species are responsible, but certain species which displace the nucleus of the host are: *Plasmodium cathemerium, P. gallinaceum* and *P. juxtanucleare.*

Species with elongate gamonts, which usually do not displace the host cell nucleus, are: *P. durae, P. circumflex, P. elongatum* and *P. fallax.*

Developmental Stages: The infective sporozoites do not enter the RBC's directly but develop as exoerythrocytic forms in the cells of reticuloendothelial system prior to invasion of the erythrocytes.

Following the introduction of the sporozoites from infected mosquitoes, numerous pre-erythrocytic schizonts (cryptozoites) are found in the macrophages and fibroblasts of the skin near the point of entry. Merozoites from this first generation of pre-erythrocytic schizonts form a second generation of pre-erythrocytic schizonts, the metacryptozoites, which reach maturity at about 72 hours. Merozoites from the metacryptozoites enter erythrocytes and cells of the lymphoid–macrophage system in the skin, spleen, lungs and capillary endothelial cells of the major organs. In this species, the exoerythrocytic developmental stages may be added to by forms which are derived from the erythrocytic cycle. These are known as phanerozoites, being derived from the merozoites of the meronts in the erythrocytic cycle.

The erythrocytic cycle is initiated 7-10 days after infection of merozoites from metacryptozoites. On entering the erythrocytes merozoites round up to form trophozoites, which are small rounded forms containing a large vacuole and displace the cytoplasm and nucleus to one side giving the young form a 'signet ring' appearance. Early trophozoites undergo schizogony and result in formation of merozoites. During each cycle of schizogony, the parasite takes in host cell cytoplasm by invagination, haemoglobin is digested and the

residual haematin pigment is deposited in granules with food vacuoles. After a number of asexual generations, merozoites undergo sexual development with the formation of macrogamonts and microgamonts. Female forms (macrogamonts) are more numerous than male forms and stain more intensely blue with Romanowsky stains. Further, development takes place in suitable mosquito and is rapid. Within 10-15 minutes the nucleus of microgamonts divide and exflagellation takes place. Six to eight thin, flagella like microgametes are extruded from parent cell and these remain attached to parent cell for a few minutes, lashing actively. They then detach, swim away and fertilize macrogamete. Zygote resulting from fertilization is motile and is called ookinete. Ookinete penetrates midgut mucosa, comes on surface and results in formation of oocysts. Nucleus of oocyst further divides repeatedly to form a very large number of sporozoites. After complete maturation of cyst, oocysts rupture and release the sporozoites in the body of the mosquito which eventually reach the salivary glands. These are infective to new host and these mosquitoes remain infective for the whole life; transmitting malarial parasites each time it takes a blood meal.

Pathogenesis: Chickens are more susceptible than adult birds. Mortality may reach up to 80%. Affected birds may show progressive emaciation and anaemia as the disease advances. Spleen and liver becomes enlarged. Cerebral stroke due to blockage of capillaries may cause death.

Diagnosis: Clinical signs, blood smear examination and post mortem lesions are confirmatory.

Treatment and Control: Mosquitoes should be controlled in poultry houses / farms with the help of insecticides. The breeding places in the form of water accumulations around the poultry farm should be prevented.

Chloroquine @5mg/kg body weight, act as schizonticide. Pyrimethamine @ 0.3mg/kg body weight is another effective drug for protecting chicken.

Genus: *Haemoproteus*

Organisms are transmitted by blood sucking insects. Gametocytes occur in erythrocytes and possess a halter shaped appearance encircling the nucleus known as Halteridium. Schizogony occurs in endothelial cells of blood vessels of inner organs, especially lungs.

Haemoproteus columbae

Commonly occurs in domestic pigeons, wild pigeons and doves.

The only form occurring in erythrocytes is gamont, which encircle the nucleus in form of halter. Macrogamonts stain dark blue with Romanowsky stain with compact dark purple to red nucleus. Microgamonts stain pale blue to pinkish. Infected hippoboscid fly *Pseudolynchia canariensis* injects the sporozoites in the blood stream of the birds.

Sporozoites thus enter the endothelial cells of the blood vessel and form early schizonts. Growth and nuclear division of early schizonts in small unpigmented masses (15 or more) take place, which are known as cytomeres. Each cytomere continues to grow and its nucleus undergoes repeated division. Each cytomere thus produces enormous number of minute merozoites. Further, break down of endothelial cells takes place and cytomeres are liberated

and accumulate in blood capillaries, which further may get blocked due to their abundance. Cytomeres rupture and merozoites are released in the blood stream. Merozoites enter the RBC's and transfer into macrogametes and microgamonts. Further development takes place in Hippoboscid flies. Development involves exflagellation of microgamonts in the midgut of fly. Microgametes fertilize macrogamete and result in formation of ookinete. Ookinete migrates to the surface of the midgut. Sporogony takes place with the production of sporozoites. Sporozoites are liberated in the body cavity of insects and are passed to salivary glands. These flies are now capable of further transmitting the infection.

Pathogenesis: Adult birds usually show no clinical signs. In heavy infections, the birds may appear restless and do not feed. Anaemia may result due to destruction of erythrocytes. Liver and spleen maybe enlarged.

Diagnosis: It is based on demonstration of intra-erythrocytic gametocytes in blood smears. On post-mortem examination, schizonts in the endothelial cells of lungs, liver, spleen and kidney may be found.

Genus: *Leucocytozoon*

In this genus, gametocytes occur in leucocytes or in some species, in erythrocytes of birds. Schizogony occurs in the epithelial and parenchymatous cells of liver, heart, kidney and other organs. The vectors are black flies of the genus *Simulium* or midges of the genus *Culicoides*. The most common species are: *Leucocytozoon simondi* in wild ducks and geese, *L. smithi* in domestic and wild turkey and *L. caulleryi* in domestic chicken.

Leucocytozoon simondi: Occurs in the domestic ducks and geese. The species is highly pathogenic to young ducks and geese.

Life cycle: Sporozoites, after injection by bite of fly vector, are carried to different cells of the body by blood. Two types of schizonts develop. The first generation schizonts develop in kupffer cells of the liver. These are small. The merozoites from these either enter erythrocytes to become gametocytes or enter parenchymal cells of the liver and form hepatic schizonts, each forming a number of cytomeres. After multiple fission, a large number of merozoites develop. Megaloschizonts more numerous than hepatic schizonts, develop in lymphoid cells or macrophages in brain, lungs, liver, kidney, intestinal tissues and lymphoid tissues 4-6 days after the infection. They contain a large number of cytomeres which in turn produce much large number of merozoites. Merozoites from hepatic schizonts and megaloschizonts are released into the blood and develop as gamonts in peripheral circulation in 6-7 days after the infection possibly inside lymphocytes or may be inside erythrocytes, mature erythrocytes are elongate, oval bodies. Development inside the vectors is essentially the same as *Plasmodium* in mosquitoes.

Pathogenesis: The young birds suffer more than the adults. The clinical signs include anaemia, listlessness, loss of appetite and diarrhoea. Post mortem changes include haemorrhages in lungs, liver and kidneys. There is enlargement of spleen and white spots on the heart muscles. After 14 days of infection of sporozoites, gamonts in chicken have been demonstrated.

Treatment: It is recommended with sulphamonomethoxine sodium @ 1g/litre of drinking water. For prophylaxis, continuous medication at the level of 1g/20 litres of drinking water is effective.

Lecture-11

Subclass: Piroplasmia

Order: Piroplasmida

Haemoprotozoan parasites of the cells of haemopoetic system of vertebrates are classified under this order. These are small than those classified under Plasmodiidae. They are pyriform, amoeboid, rod like, round or pleomorphic. Reproduction is by binary fission or schizogony. Pigment (haemozoin) is not formed from the host cell haemoglobin. The vectors are ixodid ticks. Two families are of importance.

Family: Babesiidae

Developmental stages occur in the erythrocytes of the vertebrate host. These are large round to pyriform or amoeboid organisms. The family has one genus *Babesia*. The species of the genus are parasites of the RBC's of the mammalian hosts.

Genus: *Babesia*

The parasite is worldwide in distribution and has 70 species recorded from different categories of domestic animals causing a tick borne haemoprotozoan disease 'babesiosis' in cattle, sheep, goat, dogs, cats, equine. The disease is recognised as a problem of major economic importance, particularly in domestic ruminants.

Organisms multiply in the erythrocytes by asexual division producing two, four, or more parasites without pigment. *Babesia* species in red blood cells are seen singly as round, ovoid, elongate trophozoites or as paired pyriform merozoites or in tetrads as cruciform merozoites.

In general, *Babesia* is divided into two major groups: large forms with an average length of more than 2.5µ or more and small forms with less than 2.5µ size. The paired large forms generally lie with their narrow ends in apposition having an acute angle of divergence while small forms lie at an obtuse angle. Generally, the infection with large forms can be successfully treated with trypan blue while infection with small forms does not respond to it.

Life cycle: In vertebrate hosts, *Babesia* organisms occur in the erythrocytes and multiply by binary fission or budding process (endodyogeny or endopolygeny) or schizogony to form two, four or more trophozoites. These are liberated from erythrocytes and further infect/invade other cells. This process is repeated until a large number of erythrocytes are parasitized. Young erythrocytes are preferably invaded by the merozoites, especially in acute *Babesia bigemina* infection. Sometimes, a red blood cell may show multiple infections with large number of trophozoites which may result due to series of binary fissions. These blood forms are readily transmissible by mechanical means to other animals.

Babesia species are transmitted by ticks. Essentially the transmission of Babesia species in ticks is either 'transovarian' (in one host tick) or transstadial or stage to stage (two/three host ticks). The former mode of transmission is only possible by one host tick e.g. species of *Boophilus* transmitting *B. bigemina* in cattle.

Transovarian transmission: After repeated multiplication of parasites inside erythrocytes of the vertebrate hosts, the stages are ultimately differentiated as microgamonts and

macrogamonts proving that further development inside the tick vector is sexual. These stages are taken up to the suitable tick and further developments take place in the gut of the tick. The gametes are set free by the lysis of the infected erythrocytes and unite to form a zygote. The zygote undergoes further development and multiplication. The zygote becomes motile and comes to serous surface and ruptures. The vermicules thus released invade ova and finally reach to gut epithelium of the larva emerging out of the infected eggs. The infective organisms finally reach the salivary glands of the larva and after maturation are able to infect the vertebrate host during feeding on its blood. Subsequent development stages viz., nymph and adult ticks continue to transmit the infection.

Stage to stage transmission: In case of feeding of larval and nymphal stages, the vermicules formed from the zygote migrate to the muscle sheaths and divide repeatedly to form a large number of small ovoid forms. Further development and migration to the salivary glands occur when after moulting the next stage feed on the blood of the host. On reaching the salivary glands, the parasites enter the cells of the acini and undergo repeated binary fission to form a large number of small, ovoid infective stages.

Babesiosis in cattle and buffalo:

Four species of *Babesia* are described in cattle: *Babesia bigemina, B. bovis, B. divergens* and *B. major*.

Babesia bigemina: is the cause of cattle tick fever/ red water fever/ Texas fever or piroplasmosis in cattle and buffalo. *B. bigemina* is a large piroplasm, 4-5µ × 2µ in size in erythrocytes. Round forms are 2-3µ in diameter. Oval, pyriform or irregular forms may also occur. The organisms are characteristically pear shaped in form and lie in pairs forming an acute angle in erythrocytes.

Life Cycle: *B. bigemina* is transmitted by various species of ixodid ticks: *Rhipicephalus (Boophilus) microplus, Rhipicephalus (Boophilus) australis, Rhipicephalus (Boophilus) decoloratus, Rhipicephalus (Boophilus) calcaratus, Rhipicephalus appendiculatus, Rhipicephalus evertsi, Rhipicephalus bursa.*

Pathogenesis: *Babesia bigemina* causes higher mortality in adults than in young ones (Inverse age immunity) and symptoms are more marked in exotic breeds than indigenous breeds. The natural resistance of young calves to infection usually disappears at 9-12 months of age.

The first symptom of the disease is high fever (40-41˚C) which may further rise up to 45.5˚C and last for a few days to a week. During the early phase of the fever, the blood smears show parasitemia. A parasitemia of more than 1% can give rise to acute form of the disease. In acute infection, a very large number of erythrocytes are destroyed.

The symptoms following fever include anorexia, depression, weakness and cessation of rumination, salivation, dryness of muzzle, lacrymation and fall of milk yield. Initially, there is profuse diarrhoea followed by marked constipation. The conjunctivae appear brick red in the beginning and then become anaemic in advance stages. In advance stages, there is severe anaemia, haemoglobinuria, jaundice which terminates in death. Death is due to unabated destruction of red blood cells because of autoimmune mechanism, failure to recoup blood loss, cerebral anoxia and accumulation of by-products. Animals become thin and

emaciated. Thus, the pathological changes involve haemolytic destruction of erythrocytes in *B. bigemina* infection, whereas in *B. bovis* infection, there is kinin production which further leads to stasis and intravascular coagulation, eventually leading to shock and death.

Haematological changes include reduction in TEC and haemoglobin values, which may lead to death of the animals. The biochemical changes involve decrease in calcium, inorganic phosphorus and total serum protein levels, marked increase in blood glucose level, total serum bilirubin and SGOT levels indicating liver damage and increased activity of aspartate amino transferase attributable to liver necrosis and damage of erythrocytes.

Post mortem lesions include emaciation, subcutaneous and intramuscular oedema and icterus, pale yellow to distinct yellow discolouration of the subcutis, swelling and congestion of the internal organs, serous surface of the intestines showing ecchymotic haemorrhages, marked enlargement of the spleen with soft pulp enlargement of liver.

Immunity: In *B. bigemina* infection, there is premunity and the animals recovered from the infection may continue to harbour the organisms and remain immune to reinfection for about more than a year. The infection with *B. bigemina* or *B. bovis* confers species specific immunity. In nature, the young animals are resistant to infection, whereas adults get infected. The phenomenon is called as inverse age immunity, which is rendered by colostral antibodies for the age of about 9 months.

Endemic stability: A dynamic epidemiological state in which clinical disease is rare in spite of a high incidence of infection within a population. Inverse age immunity and premunity play important role in maintenance of endemic stability.

Diagnosis: It is based on clinical symptoms, detection of piroplasm in the peripheral blood and serological tests. The important symptoms include high fever, haemoglobinuria and anaemia. Infected animals may show heavy infestation with ticks. Detection of piroplasms in stained blood smears may not always be possible. Both thin and thick smears maybe examined for the presence of piroplasms in erythrocytes. Occasionally, differential diagnosis from other diseases including anaplasmosis, leptospirosis, post parturient haemoglobinurea, plant poisoning etc. is required. A number of organisms are demonstrable in blood. The various serological tests have been employed to detect the infection, especially in sub-clinical state where organisms are demonstrable in blood. The various serological tests that are used for diagnosis involve complement fixation (CF), indirect fluorescent antibody test, indirect haemagglutination assay etc.

Treatment:

1. Diminazine aceturate @ 3-5 mg/kg body weight through subcut or I/M route for two consecutive days.
2. Imidocarb dipropionate @ 1.2 mg/kg body weight as single dose through subcut route.
3. Phenamidine isethionate @ 0.03 ml of 40% solution per kg body weight as a single dose through subcut route
4. Trypan blue @ 50-100 ml of 1% sol. to be given only through I/V route.

Control: Life cycle should be interrupted by applying the control measures against the ticks. Thus, animals should be dipped at regular intervals for the control of ticks. The inverse age resistance of cattle has been exploited for control by immunization.

***Babesia bovis*:** Occurs in cattle and some cervids in Europe, Russia, Africa, Central and South America and Iran. Its occurrence in India has been confirmed by some workers from South India and also from Haryana. It is small piroplasm, 2.4µm × 1.5µm and slightly larger than *B. divergens*. The trophozoites in the erythrocytes of the host are pyriform, round or irregular.

Life cycle: The tick vectors are *Rhipicephalus (Boophilus) miroplus, Rhipicephalus bursa, Ixodes ricinus* and *Ixodes persulcatus*.

Transmission: Trans-ovarian

Pathogenesis: Clinical signs are similar to those for *B. bigemina.* The incubation period is 4-10 days. The first sign is high fever (40-41˚C) for about a week after infection. A cerebral form of the disease has been reported. Other symptoms include anaemia, icterus, diarrhoea and rapid heartbeat. Animals may die after acute phase of infection. Post-mortem changes consist of congestion of white and grey matter of brain and generalised dilatation of capillaries by erythrocytes. There is perivascular perineuronal and interstitial oedema through the brain and spinal cord.

***Babesia divergens*:** the species is a European form found in cattle in Western and Central Europe.

***Babesia major*:** Occurs in cattle in South America, North and West Africa, Europe and Russia. The species is smaller than *B. bigemina* but larger than *B. bovis*.

Babesiois in Sheep and Goat:

There are four species of *Babesia* in sheep and goats. There is one large form *B. motasi* and three small forms: *B. foliata, B. ovis* and *B. taylori*.

Babesia motasi: causes babesiosis in sheep and goats in Southern Europe, Middle East, Southern East Asia, Russia and Africa. The pyriform stages are similar to *B. bigemina*.

Life cycle: similar to *Babesia bigemina*. They may occur singly or in pairs forming acute angels. The tick vectors are *Dermacentor sylviarum, Haemaphysalis puctata* and *Rhipicephalus bursa*.

***Babesia ovis*:** smaller form of Babesia. Most of the piroplasms are round and lie in the margins of the erythrocytes. The paired pyriform trophozoites usually form obtuse angle.

***Babesia foliata*:** has been reported from sheep in India. It resembles *B. ovis* but is leaf shaped. The trophozoites lie centrally in the red blood cells.

***Babesia taylori*:** has been reported from goats in India. This is small form of *Babesia* measuring 1.5-2µ in size.

Babesiosis in Horse

Babesia caballi: occurs in erythrocytes of horse, donkey, mule and zebra in Asia, South Europe, Africa and Russia. The species is a large form resembling *B. bigemina*. Parasites commonly occur in pairs forming an acute angle. These are pyriform in shape and measure 2.5-4 µm in length.

Life Cycle: Similar to that of *B. bigemina*. The species of tick vectors involved in different countries in the life cycle are: *Dermacentor marginatus, D. reticulates, R. bursa* and *R. sanguineus.*

Pathogenesis: The incubation period is 6-10 days for *B. caballi* infection. The disease may be acute, subacute and chronic. Mixed infection with *Theileria equi* are found in nature. Fever, gastroenteritis anaemia, icterus are common symptoms. Haemoglobinurea is rare. In acute cases death may occur within 1-4 weeks after the onset of clinical signs. Involvement of CNS is common resulting in paralysis of the hindquarters. Other nervous symptoms include restlessness, nervousness and walking in circles with incoordination.

Babesiosis in horses is more marked in old animals and the inverse age resistance is comparable to that seen in *B. bigemina* infection in cattle. Post mortem lesions include emaciation, anaemia, jaundice, oedema, gelatinous fat, bile-stained internal tissues, splenomegaly with soft pulp, hepatomegaly, and enlargement of lymph nodes.

Diagnosis: Depends upon identification of parasites in stained blood smears. The most satisfactory site to obtain blood sample is ear vein. Besides clinical signs, reduced Hb and TEC values and an increased ESR help in the diagnosis. Different serological tests have been used for diagnosis.

Treatment:

1. Diminazine aceturate @ 5 mg/kg body weight through subcut or I/M route for two consecutive days.
2. Phenamidine isethionate @ 0.03 ml of 40% solution per kg body weight.
3. Amicarbalide @ 5-10 mg/kg body weight as 50% solution.

Babesiosis in dogs

Babesia canis: It is recognised to represent three subspecies: -

S.No.	Subspecies	Distribution	Vector
1.	*Babesia canis canis*	Southern and Central Europe	*Dermacentor reticulatus*
2.	*Babesia canis rossi*	Southern Africa	*Haemaphysalis leachi*
3.	*Babesia canis vogeli*	Asia, Africa, North and South America, Australia, Europe	*Rhipicephalus sanguineus*

These are large piroplasms, pyriform in shape, 4–5 µm in length, pointed at one end and rounded at the other. Amoeboid forms have been described that are 2–4 µm in diameter and usually contain a vacuole.

Pathogenesis: The first sign is fever which is quickly followed by marked anaemia, icterus, inappetence, marked thirst, weakness, prostration and often death. Haemoglobinuria is seen

sometimes in peracute cases where the loss of erythrocytes is marked. Usually there is bilirubin in urine. Emaciation become extreme but animal usually dies before this stage is reached in acute cases. Active phagocytosis of both parasitized and non-parasitized erythrocytes by macrophages in the circulating blood and in spleen, bone marrow and liver are largely responsible for the loss of erythrocytes in cases where haemoglobinuria is not present. However, in acute cases the death in 4-5 days is associated with acute respiratory failure, often with extensor spasms. There may be involvement of CNS causing locomotor disturbances, paresis and epileptiform fits.

Diagnosis: It is based on symptoms and by finding the organisms in blood smears. Buffy coat smear diagnosis plays an important role in identification of the parasites mainly *Babesia* (targets the immature RBCs associated to buffy coat), *Ehrlichia canis*, *Hepatozoon canis* as well as *Trypanosoma evansi*.

Treatment: In every case, chemotherapy with imidocarb, phenamidine, diminazene aceturate or trypan blue is advisable immediately after clinical diagnosis, since death may occur rapidly. In dogs with severe anaemia, blood transfusion should be considered.

Babesia gibsoni: Found in dogs in China, India and Sri Lanka. Wild carnivores also harbour the infection. It is a small form and pleomorphic. The trophozoites are typically annular or oval and not usually pyriform.

Life cycle: Transmitted by *Rhipicephalus sanguineus*

Pathogenesis: The parasite causes chronic form of the disease which shows relapse of fever. There are periods of exacerbations of fever and progressive anaemia and haemoglobinuria. Jaundice is less common. The affected dog becomes extremely weak and has staggering gait.

Diagnosis is done as in other cases of babesiosis. Berenil is effective.

Zoonotic Babesiosis: the first case was reported from Yugoslavia. *B. microti* plays an important role in transmission of infection to humans in American subcontinent, whereas *B. divergens* is important in Europe and infects humans.

Lecture-12

Family: Theileridae

Genus: *Theileria*

Genus *Theileria* mainly involves three commonly occurring species which infect cattle and buffalo, one species of equine and two species which infect sheep and goats. The parasites are pleomorphic and occur as minute, round, ovoid. Rod like, comma shaped and irregular forms in lymphocytes, histiocytes and erythrocytes of vertebrate hosts. They do not produce pigments. Ixodid ticks are vectors of these parasites.

Theileriosis in cattle and buffalo

There are two main species infecting cattle and buffalo: *Theileria annulata* and *T. parva*

***Theileria annulata*:** Occurs commonly in cattle and buffalo of North Africa, Middle and Far East, Russia, Southern Europe, Asia Minor and India. The species cause a highly fatal disease in dairy animals transmitted by ixodid ticks of the genus *Hyalomma*. In India, disease caused by *T. annulata* in cattle and buffalo is called Bovine Tropical Theileriosis. Though the disease was recognised as early as 1905 but the economic importance was only realised after 1970 when several cases of exotic and cross bred cattle were reported.

The erythrocytic forms in the red blood cells are generally indistinguishable from those of *T. parva* but usually 80 percent are round or annular. And rest are oval or comma shaped. In peripheral blood smears, one or more parasites are found in each erythrocyte. The erythrocytic forms of the parasite undergo binary fission with the formation of two or rarely four daughter organisms.

Macroschizonts and microschizonts are found in the lymphocytes of the spleen and the lymph nodes. Biopsy smears from superficial lymph nodes reveal the presence of schizonts in the lymphocytes, monocytes as well as in free state.

Life Cycle:

Under natural conditions, the susceptible animals are infected by tick vectors possessing a number of uninucleate infective particles (sporozoites 1.5 µ in diameter) during its attachment for the engorgement. Transmission does not occur immediately on attachment because sporozoites develop in the salivary glands during first 2-4 days of engorgement of nymphal and adult stages of the ticks. These stages in the lymphocytes and lymphoblasts are small spherical bodies of 2 µm diameter, with round nucleus in the cytoplasm. The entry of the sporozoites in the lymphocytes stimulates lymphoproliferation and so the parasite and host cells divide synchronously. After several days, the parasites become apparent in various lymph nodes and elsewhere in the lymphoid and reticuloendothelial tissues as multinucleated macroschizonts as **Koch's blue bodies**, each containing about 8 nuclei of about 1 µm size. A portion of the macromerozoites develops into microschizonts after 14 days of infection. The micoschizonts are initially compact bodies in lymphocytes and reticuloendothelial cells or macrophages, which subsequently produce 50-120 nuclear particles (micromerozoites) by budding. The microschizonts appear simultaneously with the appearance of morphological

similar piroplasms in erythrocytes which indicate that erythrocytic stages are derived from microschizonts. Two types of erythrocytic stages viz. comma and ovoid forms have been demonstrated. They probably represent micro and macrogamonts from which gametes are formed when larval or nymphal stages feed on the blood of infected animals. In ticks, two types of gametes unite to form zygotes which increase in size by about 12 days. Subsequently, the zygote becomes elongated (ookinete) which leave the gut cells and enter into haemolymph after 14 to 17 days. The ookinete pass to the acinar cells (type II or III) of the salivary glands and develop into sporonts which subsequently commence sporogony. Further development occurs after moulting into subsequent stages of the ticks i.e. the nymphal stage or the adult which are capable of transmitting the infection during their feeding on the susceptible animals. This is called stage to stage or transstadial transmission. The ticks responsible for transmission include *Hyalomma anatolicum anatolicum, H. dromedarii, H. marginatum issaaci* and *H. dentritum*. The infective stages in the salivary glands develop only after one day of feeding in the adult tick and two days of feeding in the nymph. Experimental infection could be established in calves by inoculation of ground-up tick supernatant (GUTS) containing infective particles (sporozoites). *Theileria annulata* can also be transmitted mechanically to susceptible animals by inoculation of infective blood and tissue suspensions made from spleen, liver of infective animals.

Pathogenesis: The pathogenic strains of *T. annulata* occur in Russia, Israel, Iran and India. The disease maybe: acute, subacute or chronic. The acute disease occurs in all breeds and of all age groups of cattle, buffalo and zebu but the latter two recover more readily from the disease. The incubation period is 9-25 days. In acute form, the course of disease is 3-4 days, while in other forms it may last for about 20 days.

Initially the clinical symptoms observed include enlargement of regional superficial lymph nodes (due to lymphoproliferation), high fever, increase in heart and respiratory rate, laboured breathing, rough hair coat and slight anorexia. Petechial haemorrhages on the conjunctivae and depression are also noted.

Cerebral form of theileriosis has also been recorded in which nervous symptoms predominate and the impression smears formed from brain reveal the presence of Koch's blue bodies. Some cross bred and indigenous animals continue to suffer a few weeks and show low parasitemia, irregular temperature and marked anaemia. Finally such animals become recumbent and die. Recovered animals remain premune carriers. This status persists due to subsequent infection by the infected ticks. In nature, there may be breakdown of the premune status due to various stress factors and there may be recrudescence of parasitemia, which may lead to death.

Haematological changes involve fall in haemoglobin level, decrease in TEC and PCV. The post mortem changes include emaciated carcass, pale mucous membranes, and enlargement of superficial lymph nodes, spleen and liver, distension of gall bladder with thick bile and punched necrotic ulcers in abomasums which are pathognomonic. In young calves, abomasums is full of undigested clots of milk.

Immunity: Presence of cell mediated immunity in tropical theileriosis has been demonstrated by the leukocyte migration inhibition test. Cattle recovered from tick infection are immune against subsequent infection by the ticks, schizonts and erythrocytic forms.

Diagnosis: It is based on clinical signs, presence of ticks, and demonstration of the blood forms in erythrocytes, Koch's blue bodies in lymphocytes or biopsy smears from lymph nodes. For the diagnosis of latent cases, serological tests viz., complement fixation test, capillary tube agglutination test and fluorescent antibody techniques are opted.

Treatment and Control: Mainly involve prophylaxis and chemotherapy. Prophylaxis contains of chemoprophylaxis, immunoprophylaxis and control of ticks. Drug of choice are naphthaquinone compounds like parvaquone and buparvaquone.

Tissue culture vaccines have been developed in different countries. These are based on the principal of serial passages of schizontal stages of the parasites to attenuate virulence. In India, Rakshavac-T, a commercial live attenuated tissue culture macroschizontal vaccine has been used in cross bred and exotic cattle aged 2 months and above. One subcutaneous injection of 3 ml is given annually.

Theileria parva: occurs in cattle, African buffalo in most parts of Africa and cause a classical disease called East Coast Fever.

Life Cycle: *Rhipicephalus appendiculatus* is the tick vector involved in the life cycle. The disease condition is not reported in India due to the absence of the vector. Transstadial transmission is reported.

***Theileria mutans*:** Occurs in cattle in Africa, Australia, Russia and Asia including India. It causes benign tropical theileriosis which is less pathogenic. The forms in the RBCs are round, oval, pyriform or *Anaplasma* like. More than two parasites may also be recorded in one erythrocyte. The life cycle is comparable to other species. The ticks involved in transmission are mainly of *Boophilus, Hyalomma* and *Haemaphysalis* species.

Theileriosis in Equines

***Theileria equi* (**previously ***Babesia equi*)**: Occurs in horse, mule, donkey and zebra throughout the world. The species is smaller than *Babesia caballi*. The parasite has a tendency to divide into four daughter organisms which appear like a 'Maltese cross'. In addition to the small pear-shaped forms, round amoeboid or annular forms are also found. There may be as many as eight organisms in one cell.

Life cycle: exoerythrocytic schizogony has been reported *in vivo* and *in vitro* with the development of micro and macroschizonts in the lymphocytes and invasion of the erythrocytes by the merozoites.

Pathogenesis: It is more pathogenic than *B. caballi*. Incubation period ranges from 10-21 days. The first clinical sign is high rise of body temperature, which maybe remittent or intermittent. Following rise of temperature, organisms appear in large number in blood. The fever may last from a day to several weeks. Haemoglobinuria may be present. There is listlessness, depression and inappetence. Oedema of the dependent parts of the body and the head may occur. Pregnant mares may abort.

Diagnosis: It is based on clinical signs and detection of the parasites as typical 'maltese cross form' in blood.

Theileriosis in Sheep and Goat

There are two species of *Theileria* infecting sheep and goats.

Theileria hirci: Responsible for causing malignant theileriosis in sheep and goats. The erythrocytic forms are mostly round or oval (approximately 80%) and few forms are rod like.

Theileria ovis: Responsible for causing benign theileriosis in sheep and goats. The tick vectors are species of *Rhipicephalus* and *Haemaphysalis*.

Order: Rickettsiales

Genus: *Anaplasma*

The parasite of this genus parasitizes the red blood cells of cattle, buffalo, sheep, goat and related animals. Blood films prepared from infected animals and stained with Romanowsky stains, show deep red coloured organisms inside the erythrocytes with no cytoplasm and slight halo around them. One erythrocyte may occasionally have more than one organism. There are three species of the genus responsible for causing anaplasmosis in animals:

Anaplasma marginale and *A. centrale* in cattle and *A. ovis* in sheep and goat.

***Anaplasma marginale*:** It is mainly responsible for the disease in cattle and wild herbivores. The primary mode of transmission is by ixodid ticks of species *Boophilus, Dermacentor, Hyalomma, Ixodes, Rhipicephalus*. *Argas* and *Ornithodoros* also act as vectors. There may be mechanical transmission by blood sucking flies viz. tabanid fly, deer fly and *Stomoxys*. The disease may also be transmitted during surgical operations such as dehorning, castration, vaccination, blood sampling etc. In 80% cases the parasites are seen at the margin of the erythrocytes.

Pathogenesis: Clinical anaplasmosis is mostly seen in cattle of more than 18 months of age and is less frequent in younger animals. Extravascular haemolysis is common in anaplasmosis. The incubation period varies with the mode of infection and the number of infective stages introduced. In peracute cases, there is sudden onset of fever, anaemia, icterus, sudden dyspnoea and death often in 24 hours. In acute and chronic cases, there is anaemia, loss of milk yield and abortion in pregnant animals.

Diagnosis: It is based on clinical manifestation, detection of organisms in blood smears and by serological tests viz. Complement Fixation Test, Capillary tube agglutination test and fluorescent antibody test.

Treatment: Tetracyclines @ 6-10 mg/kg body weight given as single I/M injection is effective. Imidocarband berenil may also be used in treatment.

Genus: *Ehrlichia*

Species of this genus occur in white blood cells in variety of mammals either singly or in groups called **morula.** The important species are: *E. bovis* (cattle), *E. canis* (dog), *E. ovina* (sheep) and *E. phagocytophilia* in cattle, sheep and goat.

E. canis occurs in India and leads to canine ehrlichiosis or tropical canine pancytopaenia. The organisms are found inside monocytes. In acute form, there is fever, severe anaemia and

thrombocytopaenia. *Rhipicephalus sanguineus* transmits the infection. *E. canis* is mostly found as mixed infection with *B. canis* in blood of dogs in India.

Pathogenesis: During acute phase, the organisms enter the blood stream and lymphocytes and localise the macrophages in liver, kidney and spleen. Clinical signs include mild depression, anorexia, lethargy, conjunctivitis, and anterior uveitis, oedema of hind limbs, haematuria and neurological symptoms.

Diagnosis: It is difficult due to vague and non-specific clinical symptoms. The parasites in the form of morula are the source of diagnosis of disease. Various serological tests have also been developed.

Treatment: Oxytetracycline @ 66 mg/kg body weight divided into 2-3 daily doses for 2 weeks. Doxycycline @ 10mg/kg body weight for 2-3 weeks is given.

Ehrlichia bovis is responsible for causing bovine ehrlichiosis, also called as NOPI or NOFEL.

Treatment is performed with the help of tetracyclines.