

WOLLO UNIVERSITY SCHOOL OF VETERINARY MEDICINE

**Diagnostic veterinary helminthology (3101)
for second year VLT students**

By.

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Symbiotic associations

- **Phoresy**
 - organism merely provides shelter, support, or transport for another organism of d/t species
 - E.g. Bacteria is transported by the legs of flies.
- **Commensalism**
 - association when one is benefited and the other is neither harmed nor benefited
 - not usually obligatory for their existence
 - Eg. Intestinal bacteria

- **Mutualism**
 - an obligatory association when both animals benefited from the association
 - They live together with common vital importance to each other
 - One partner cannot live without the other
 - Eg. Ciliates in ruminants
- **Parasitism** a harmful association
 - an organism (the parasite) is metabolically dependent on another species of an animal (the host)
 - Those parasites which are harmful to their hosts in various ways

- Important features of parasitism
(host-parasite relationship)
 - always involves two species, the parasite and the host
 - produce **pathological changes** in hosts that may result in **disease**
 - The parasite is always the beneficiary and the host is always the provider
- **Major objective** ; Successful treatment and control of parasitic diseases
 - ☞ Requires not only information about the parasite itself
 - ☞ But good understanding of the nature of parasites' interactions with their hosts

Important terminologies

- **Hypobiosis-**
 - retarded larval development, larval inhibition, (inhibited larval development and arrested larval development).
- **Periparturient Rise (PPR)** - also called “**Post parturient rise**” or “**Spring Rise**”.
 - It refers to an increase in the number of nematode eggs in the faeces of female animals around parturition

Pathogenesis

– progressive development of a disease

Infective larval stage (L_3)

- in most nematodes the 3rd stage larva” – is stage infective for the host when grazed with pasture while grazing

1Predilection site

- preferred site within the host, which is characteristic for a parasite species.

Establishment of Host Parasite Relationship

- The entry of parasite into the body of the host is not necessarily followed by disease or infection.
- A parasite can be held as a **successful organism** as it fairly set up the infection/disease/ and survives in the host.
- Criterias

Entry of the parasite

Evolutionary change

Host-parasite compatibility

ROOTS OF ENTRY

– Natural opening

- Mouth, Excretory organs and External openings of reproductive organs

– Skin Penetration

– Transplacental entry

– Transovarian transmission

Is an infection of the ovary and it is very common in tick borne disease.

- Involvement of Vectors

Vectors are usually IH maintaining the larval stage of the parasite.

Evolutionary change

- Parasite establishment in the host is only possible if the host's defense mechanism is **suppressed**.
- Practically all parasites undergo a successive adaptation(evolutionary process).
 - A) Increasing resistance to enzymatic activities
 - B) Profound morphological adaptation
 - C) Development of specialized attachment organs
 - D) Highly developed organs of reproduction

Survival of parasite in the host

(Host parasite Compatibility)

- antigenic shift/ variation
- alteration of the function of host cells
- Hypobiosis----arrest in development

Effect of parasites on their hosts

- **Pathogenic** and **economic** importance of parasites
- Parasites harm their hosts in the following ways
 - Absorbing readily digested food material intended for the host.
E.g. Ascaris, Taenia
 - Sucking body fluids (blood, exudates, lymph)
E.g. Haemonchus
 - Feeding on the tissue of the host.

- Causing mechanical obstruction and cause pressure on tissue or organs
- Causing growth of nodules and perforating vessels
- Causing wounds through which infection may enter into the body.
- Causes irritation
- Reducing the immune status of the host
- Transmitting the causative agent of infection or disease

- **The pathogenecity (harmful effect) of the parasite depend on**
 - type of parasite
 - number of parasite burden
 - their habitat
 - presence or absence of migration in the host
 - degree of adaptation developed b/n the host and parasite

- **Successful parasitism**
 - ability of the parasites to adapt and integrate itself within the host's internal environment.
 - the parasite does not appear foreign to the host.
- **Parasitosis**
 - condition of parasitism in a host which may or may not be showing clinical signs of a parasitic disease

Classification Vet. Parasitology

- **Helminthology**

the study of helminthes (worms)

- **Protozoology**

protozoa which are unicellular organisms

- **Entomology**

deals with arthropods (arachnids and insects)

CHAPTER -1

DIAGNOSTIC VET. HELMINTHOLOGY

Objectives

- Define helminthology and knows its area of concern
- Understand taxonomic classification of helminthes, their morphology, life cycle, transmission and pathogenesis and diagnostic techniques
- Understand disease conditions that are caused by parasites, that are very common in Ethiopia and identifying the parasites for the diagnosis of the diseases that they cause

1.1. Introduction to diagnostic Helminthology

- Study relating to helminthes and their relationship with their hosts.
- “*helminthos*” = parasitic worms
- Diseases caused by worms are generally *Helminthosis*

Taxonomy of Helminth Parasite

- Living organisms are classified according to Kingdom, phylum, Class, Order, Family, Genus and Species.
- Each of these categories is known as **Taxon**.
- The study of this aspect in biology is called taxonomy

- The intervals between these groups are large so some organisms cannot be allocated precisely.
- So that intermediate taxa have been formed.
Eg. Suborder, supper family, subfamily.
- The generic name have capital letters followed by spp name. They should be italized or should be underlined separately.

Eg. Haemonchus contortus , Haemonchus contortus

1.2. Taxonomy of Helminthe Parasites

- The parasitic helminthes comprise three phyla of veterinary importance:
- Phylum : **Nemathelminthes**
Class : Nematoda (round worms)
- Phylum : **Platyhelminthes**
Class : Trematoda (Flukes)
Class ; Cestoda(Tapeworms)
- Phylum : **Annelida**
Class : Hirudina (Leeches)

Nomenclature of nematodes

- All living organisms organized into a hierarchy of groups called **taxa**.
- This structure is based primarily
 - Degrees of similarity among members of the same group
- The highest level of classification
 - Phylum and the lowest is the species.
- Nematodes belong to the animal kingdom
- **Kingdom, Phylum, Class, Order, Superfamily, Family, Subfamily, Genus, species**

A species

- collection of similar organisms that will only interbreed among themselves.
 - *Strongylus vulgaris* in horses
 - *Toxocara canis* in dogs
 - *Haemonchus contortus* in sheep.
- many different species are morphologically similar
- may share the same habitat in the same host
 - they are still distinct species because they do not interbreed.
- Genus *Strongylus* inhabit the large intestines of horses:
 - *Strongylus vulgaris*, *Strongylus equinus*, and *Strongylus edentatus*.
- Although they share the same host and the same habitat within that host, they do not breed with each other.

- Taxonomic groups have suffixes (endings) that are specific for the particular group.
 - Kingdom: Animalia
 - Phylum: Nematelminths
 - Class: Nematoda -----→ a
 - Order: Strongylida-----→ida
 - Suborder: Strongylina-----→ina
 - Super family: Trichostrongyloidea-----→oidea
 - Family: Trichostrongylidae-----→idae
 - Sub-family: Haemonchinae-----→inae
 - Genus: *Haemonchus*
 - Species: *contortus*

Chapter 2: Phylum Nematelminthes

- Although phylum Nematelminthes has six classes, only one of these (**Nematoda**) contains worms of parasitic significance.

2.1. Class – Nematoda

- Called round worms because are round in cross section.
- Are elongate cylindrical multicellular worms
- Body isn't metamerically segmented
- Cuticle looks smooth to the unaided eye. But show various cuticular structures under microscope.
- Are very complex & variable & come in all sizes & shapes ranging from 1mm as in *Strongyliodes* to 1 meter as in *Dictyophyoma* in length.

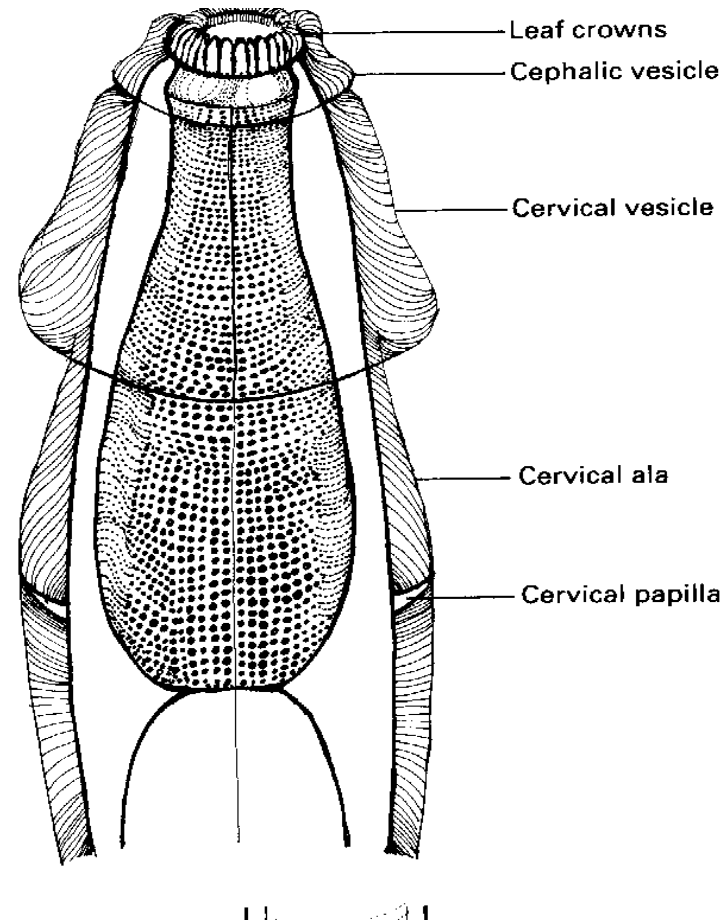
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- Infect a variety of organs & organ systems & cause significant economic losses.
- Are most numerous only 2nd to Arthropods.
- Are characterized by having sexual dimorphism
- Nematodes don't multiply inside their host (except strongyloides, Probstmayria).
- Feed on intestinal debris, mucus, bacteria, intestinal mucosal cells, some haematophagus, etc.
- Significant pathology is caused by adult & larval stages.
- Consists of 10 superfamilies of vet-importance.
- Are divided in to bursate & non non-bursate groups

A. External Morphology

- Body is covered by colour less translucent layers called **cuticle**
 - which is the external non-cellular hyaline layer covering the nematode) secreted by underlying hypodermis.
- The cuticle extends into all body openings like mouth, esophagus, rectum & genital organs.

- **Cuticle is modified to form different structures like:**
 - ✓ **Leaf crowns** - used to pin a patch of mucosa in position while feeding.
 - ✓ **Cervical & Caudal papillae** - have sensory or supportive function.
 - ✓ **Cervical & caudal alae**
 - ✓ **Cervical & cephalic vesicle**
 - ✓ **Copulatory bursa**
 - ✓ **Plaques & cordons.** Ex. occurs in superfamily Spiruroidea.
- Mouth is located at anteriorly & anus is usually sub-terminal



B. Internal Structures

- Digestive & reproductive systems are the most important internal systems

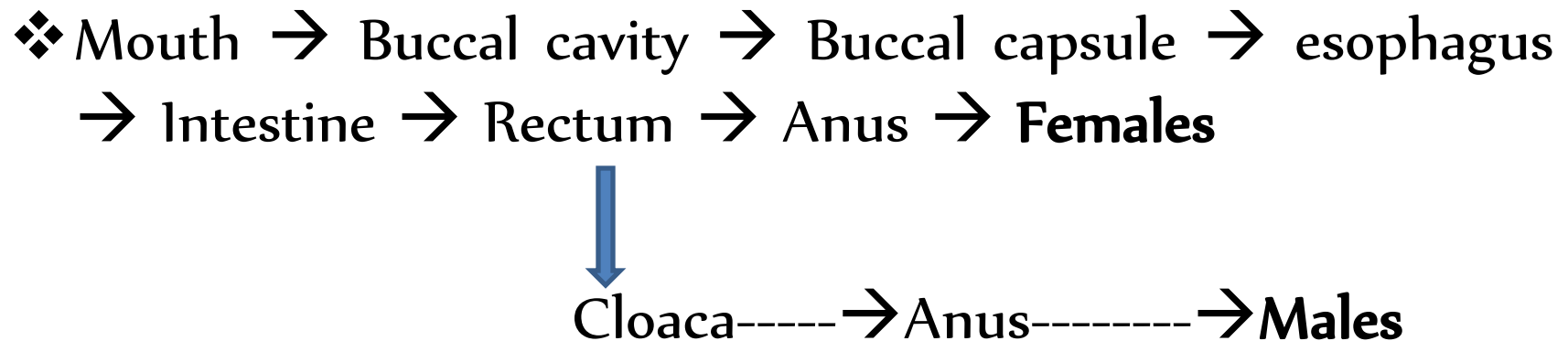
1. Digestive system

- Is tubular, long and straight & it extends from mouth to anus.
- Looks a tube within a tube in cross section
- In some species **mouth** is surrounded by leaf crowns
- **Mouth** is a simple opening in many nematodes.
- Where as in others it is large & opens into a buccal capsule & may contain teeth.

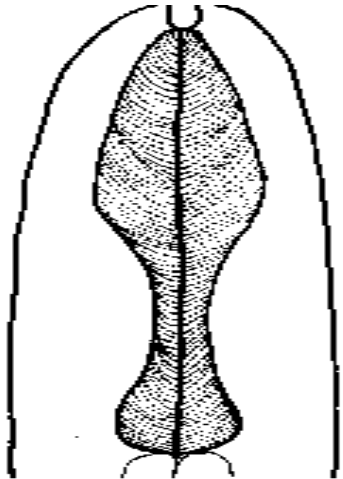
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- **Esophagus** is usually muscular & pumps food into the intestine.
- The form of Esophagus is variable & is important in preliminary identification of groups of parasites. It could be one of the following:
 - **Filariform esophagus** - simple & slightly thickened posteriorly. Ex. Bursate nematodes.
 - **Bulb shaped** - has a large posterior swelling. Ex. Asacridoids.
 - **Double bulb-shape** - Ex. Oxyuroids.
 - **Muscular-glandular** - muscular anteriorly but glandular posteriorly. Ex. Filarioids, Spiruroids.

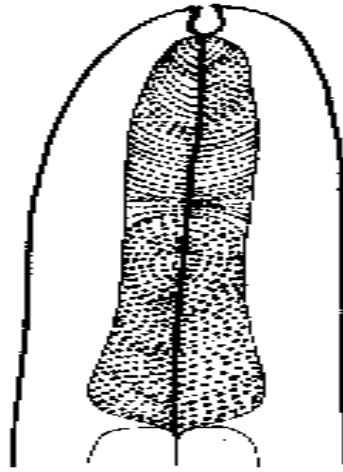
- **Trichuroid** - a capillary form esophagus passes via a single column of cells the whole being called stichosome.
- **Rhabditiform** - has slight anterior & posterior swellings. Ex. Preparasitic & adult free living stages of many nematodes.



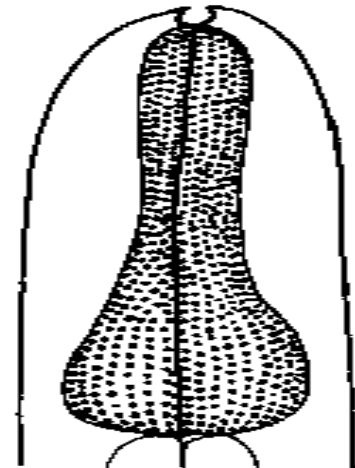
Different forms of oesophagus



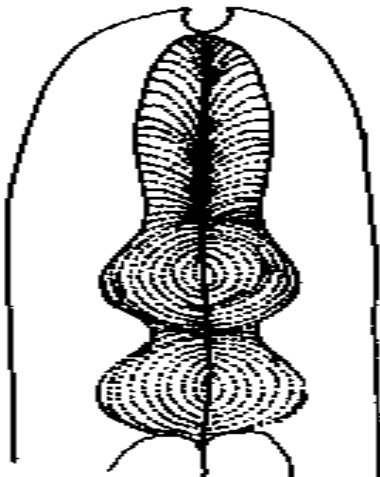
Rhabditiform



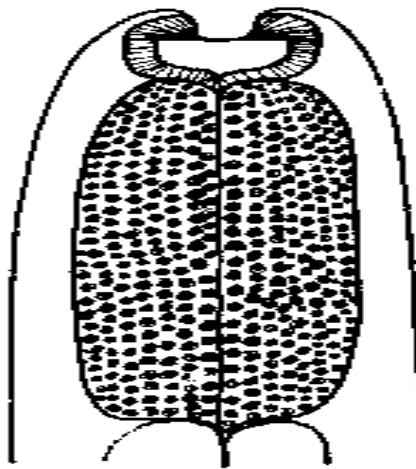
Filariform



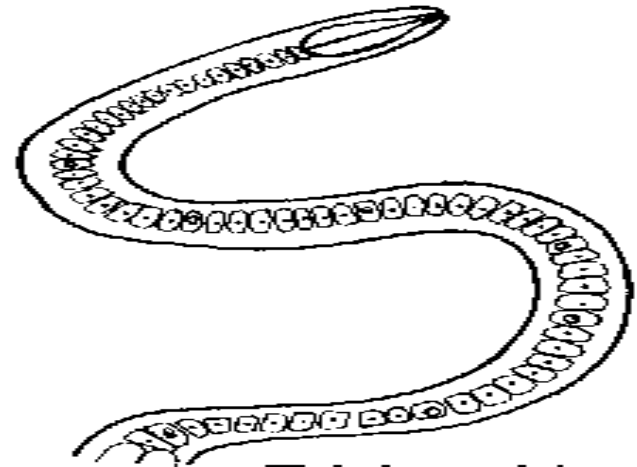
Bulb



Double-bulb



Muscular-glandular



Trichuroid

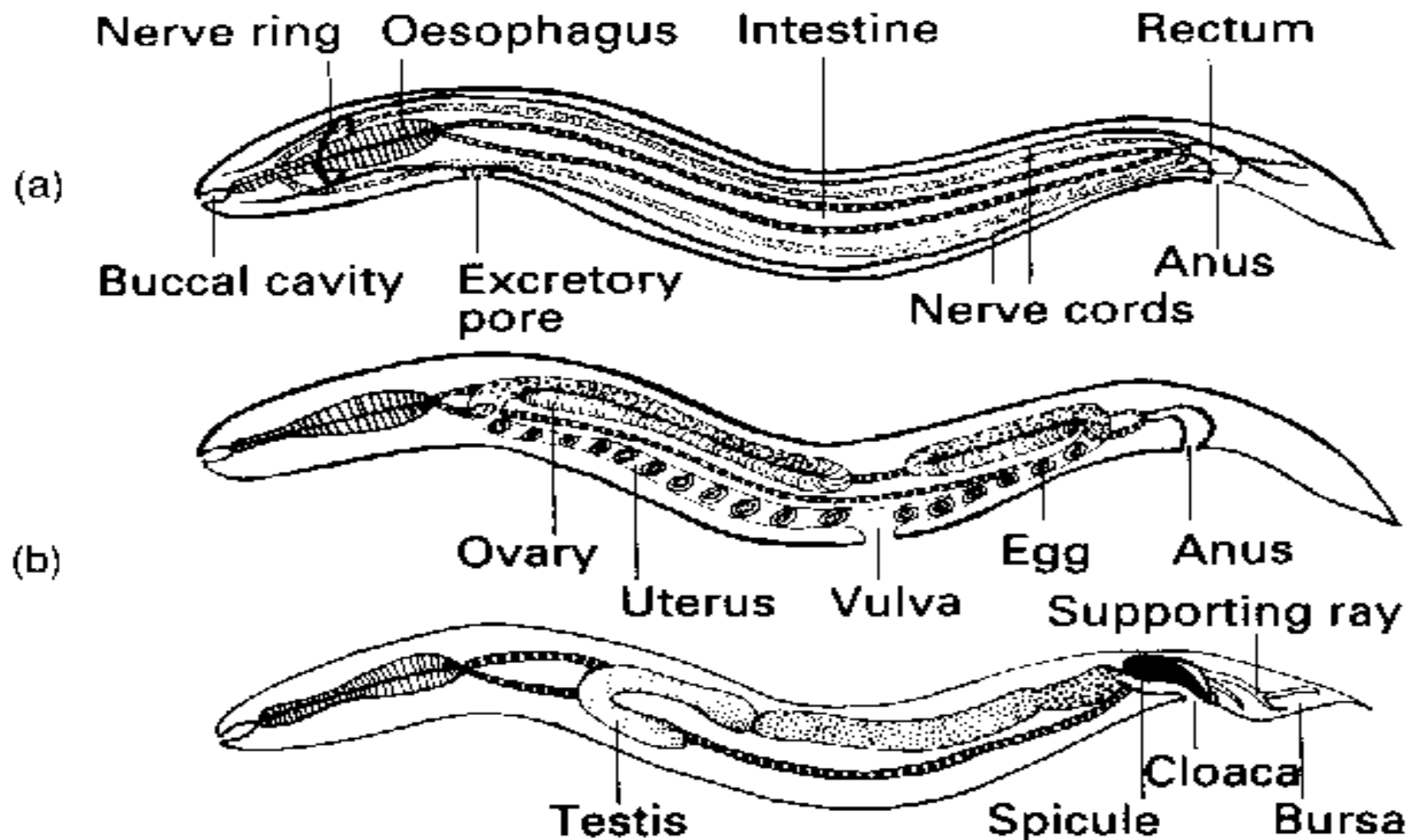


Fig. 2 Longitudinal sections of a nematode illustrating: (a) Digestive, excretory and nervous system; (b) Reproductive system of female and male nematodes.

2. Reproductive system

- Sexes are separate-----**Dioecious.**
- Males smaller than females in size

A. Male Reproductive Organs

- Single testis----->Vas deference----->Ejaculatory duct----->Cloaca.
- Accessory genital organs are copulatory bursa, chitinous spicules, gubernaculum, & bursal rays (ribs).
- **Copulatory bursa:**
 - Is well developed and derived much from expanded caudal alae
 - Has 2 lateral & 1 dorsal lobes
 - Supported by bursal rays (ribs) which are fingerlike projections

- Has function of embracing or holding on or grasping the female during copulation & is very important for species identification

Bursal rays

- Are modified or elongated caudal papillae supporting copulatory bursa derived from much expanded caudal alae.
- These rays contain muscle fiber & are arranged in a definite order.

Spicules:

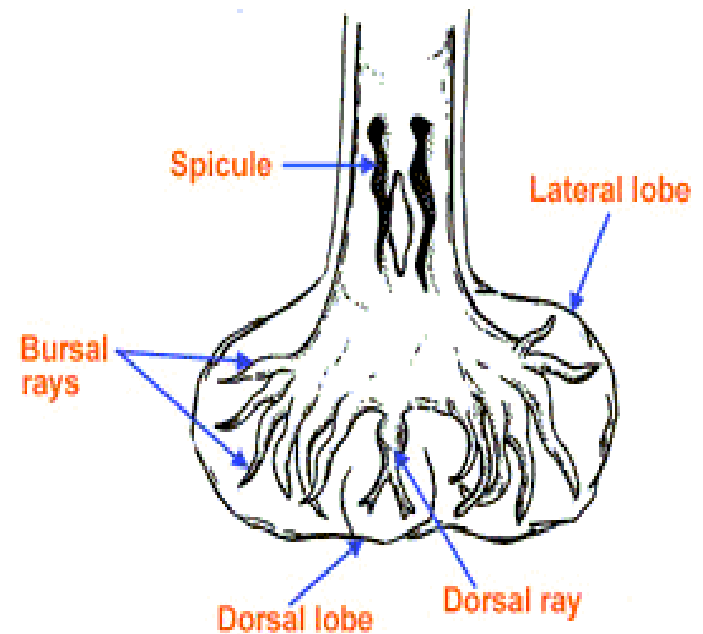
- ✓ Are accessory male genital organs situated dorsally cranial to cloaca
- ✓ Are interomittent organs or penis
- ✓ Are usually paired & equal in size

- ✓ Are highly chitinous & pigmented\ serve as organs of attachment
- ✓ Inserted into vulva to dilate & open during copulation direct flow of sperm

Gubernaculum:

- Is also chitinous accessory male organ on the dorsal wall
- Function in guiding the spicules

Nematodes - cuticle modifications copulatory bursa



Posterior modification of cuticle

B. Female Reproductive Organs

- Comprise paired ovaries, oviduct, paired uteri, ovejectors that end in common short vagina.
- Uterus can be teared to see characteristic eggs or larvae.
- Some females are highly prolific Ex. Haemonchus, etc.

2.2. Features of nematode eggs

- Nematode eggs differ greatly in size & shape.
- Shell has variable thickness & has 3 layers.
- Inner membrane is thin, lipid & impermeable.
- Middle layer is tough, chitinous, if thick the egg appears yellow colour & when interrupted at one or both ends the egg has operculum as in Oxyurus, etc.

Cont'd

- Outer layer is protein, very thick & sticky in some species. Ex. Ascaridioids (important for their epidemiology).
- Females nematodes lay or give birth to the following type of eggs or larvae:
 - Trichostrongyle (Typical strongyle), Ascaroid (Ascarid), Spiruroid (spirorid), Trichuoroid type eggs
 - some produce egg containing Larvae
 - or Some give birth to L₁ ex. Filaria
- Survival potential of the egg is affected with the thickness of the shell. Ex. infective larvated eggs usually have thick-shelled egg & can survive for years.

- The degree of development of the egg when laid varies considerably with species & it has diagnostic & ecological importance & is as follows:
 - Unsegmented eggs - contain only single cell when deposited with faeces. Ex. Ascaroid type eggs, Trichuroid type eggs.
 - Segmented eggs - contain 8-16 cells when deposited with faeces. Ex. Typical strongyle eggs, Nematodirus eggs, etc
- Eggs containing fully developed larva Ex. Metastrongylus, Dictyocaulus, etc.

Cont'd

- Based on the stage of the egg they lay or the larvae born female nematode worms can be divided into 3 as:
 1. **Oviparous females** - lay egg that hatch after they have laid
Ex. Strongylidae, Ascaris.
 2. **Ovoviviparous females** - lay eggs containing fully developed 1st stage larvae (embryo), which may hatch in the hosts' intestine & deposited with faeces. Ex. Spiruroida & most lungworms like Metastrongylus
 3. **Viviparous females** - hatching occurs within the uterus itself hence discharge fully developed free L₁ from the uterus. Ex. Filariidae, Trichenella, etc.

2.3. Basic Life Cycle of Nematodes

- Females lay eggs, L_1 containing egg or give birth to L_1 , which pass in the faeces of the host.
- Important feature in nematodes is that immediate transfer of infection from one final host rarely occurs before some development either in the faecal pat or in the IH.
- Life cycle can be direct or indirect.
- A nematode molts at intervals shedding its old cuticle (sheath) during development.
- Usually there are 4 molts in complete life cycle.

- The nematode life cycle consists of an egg, four larval or juvenile stages (usually designated by L_1 , L_2 , L_3 , L_4 and L_5 that mature to an adult stage, with a molt between each stage.
- With a few exceptions the L_3 stage of parasitic nematodes is usually the infective stage.

Free-living stages are:

- **First larval stage (L_1)**- hatches from egg feeds on lipid reserve & bacteria, grows & then undergoes second molt (M_2).

con...

- **Second larval stage (L_2)** – also feeds on lipid reserve & bacteria & grows to molt to L_3 . **Third larval stage (L_3)**– in most species the sheath of 2nd larval is retained in which it is enclosed hence it doesn't feed & is the infective stage.
- **Molting** is controlled by hormones & is used to control development & to increase probability of completing life cycle.
- **Temperature & humidity** are the two most important component of external environment.
- **Maximum number of larvae** in the shortest feasible time generally develops in the optimum temperature of 18-26^{oc} & 100% humidity.

Parasitic stages are:

Ingested L_3 :

- L_3 casts off (exsheaths) the sheaths of L_2 & called third parasitic stage, start feeding & grows to under go the third molt (M_3).
- After infection further molts takes place to produce the L_5 (immature adult) i.e. ingested $L_3 \rightarrow L_4 \rightarrow L_5$
- **Fourth larval stage** - feeds & grows; 1st evidence of sexual differentiation is noted.
- **Fifth larval stage** - is immature initially
Feeds, grows & copulate to become an adult.
- Nematodes don't multiply inside the final host except Strongyloides & Probstmayria.
- The worm can grow only to a certain size & then must molt in order to grow larger.

Cont'd

- The parasitic phase of development in the body of the host can be of two types:
- **Migratory:** where larvae travel considerable distance through the body before arriving in their final (predilection site)
 - *Hepatotracheal route*
 - *Lymphatic route*
- **Non-migratory:** it completes its development in the GIT

Two phenomena which affect the normal parasitic life cycle of nematodes

1. Arrested larval development (Inhibited larval development, Hypobiosis)

Epidemiological importance of hypobiosis

- ✓ it ensures survival of nematodes during periods of adversity
- ✓ subsequent maturation of arrested larvae increases contamination of env't and sometimes result in clinical disease

2. Periparturient rise in faecal egg counts (Postparturient rise, spring rise)

Sources of PPR may be

- ✓ Maturation of larvae arrested due to host immunity
- ✓ Increased establishment of infections acquired from the pastures and a reduced turnover of existing adult infections
- ✓ An increased fecundity of existing adult worm populations
- Phylum Nematelminths has six classes,
 - **Nematoda** contains worms of parasitic significance
- Nematodes of veterinary importance
 - the broad group of **bursate** and **non-bursate** parasites
- divided into 10 super families

A. Bursate nematodes

Order	Super family	Family	Genus
Strongylida	Trichostrongyloidea	Trichostrongylidae	<i>Ostertagia, Cooperia , Nematodirus, Haemonchus, Trichostrongylus</i>
		Dictyocaulidae	<i>Dictyocaulus</i>
	Strongyloidea	Strongylidae	<i>Strongylus</i>
		Trichonematidae	<i>Oesophagostomum</i>
		Syngamidae	<i>Syngamus</i>
		Stephanuridae	<i>Stephanurus</i>
	Ancylostomatoidea	Ancylostomatidae	<i>Ancylostoma</i>

B. None bursate nematodes

Order	Super family	Family	Genus
Ascaridida	Ascaridoidea	Ascarididae	<i>Ascaris</i> , <i>Toxocara</i> , <i>Parascaris</i> , <i>Toxascaris</i>
Oxyurida	Oxyuroidea	Oxyuridae	<i>Oxyuris</i>
		Heterakidae	<i>Ascaridia</i> , <i>Heterakis</i>
Rhabditida	Rhabditoidea	Strongyloididae	<i>Strongyloides</i>
Spirurida	Spiruroidea	Thelaziidae	<i>Thelazia</i> , <i>Spirocerca</i>
	Trichuroidea	Trichuridae	<i>Trichuris</i>
		Trichinellidae	<i>Trichinella</i>
	Filarioidea		

2.1.1. Super family: Trichostrongyloidea

- Family: Dictyocaulidae
 - Genus: Dictyocaulus
- Family : Trichostrongylidae
 - Genera: Haemonchus, Trichostrongylus, Cooperia, Nematodirus, Ostertagia

Characterstics of the super family: Trichostrongyloidea

- Small, often hair-like worms in the bursate group.
- Parasitize the alimentary tract of animals and birds,
 - the exception of *Dictocaulus*.

- Males have a well developed bursa and **two spicules**.
- The life cycle is **direct** and usually **non-migratory**
- L_3 is the infective stages.
- Buccal capsule is vestigial
- Configuration of spicules is used for differentiation
- Responsible for considerable economic loss
 - mortality & morbidity

Family : Trichostrongylidae

A. Genus *Haemonchus*

- blood sucking abomasal parasite
- Responsible for extensive losses in sheep & cattle in tropics & subtropics

Site - Abomasum

Species

- *H. contortus* → sheep & goats
- *H. placei* → cattle
- *H. similis* → cattle & deer
- *H. longistipes* → camel

Morphology

Gross

- 2-3 cm in length being the largest parasite in the abomasum.
- In fresh specimens egg filled white ovaries are observed winding spirally around the blood filled red intestine to produce a “barbed pole” effect appearance.
- Males are smaller in size & has even red colour



Microscopic

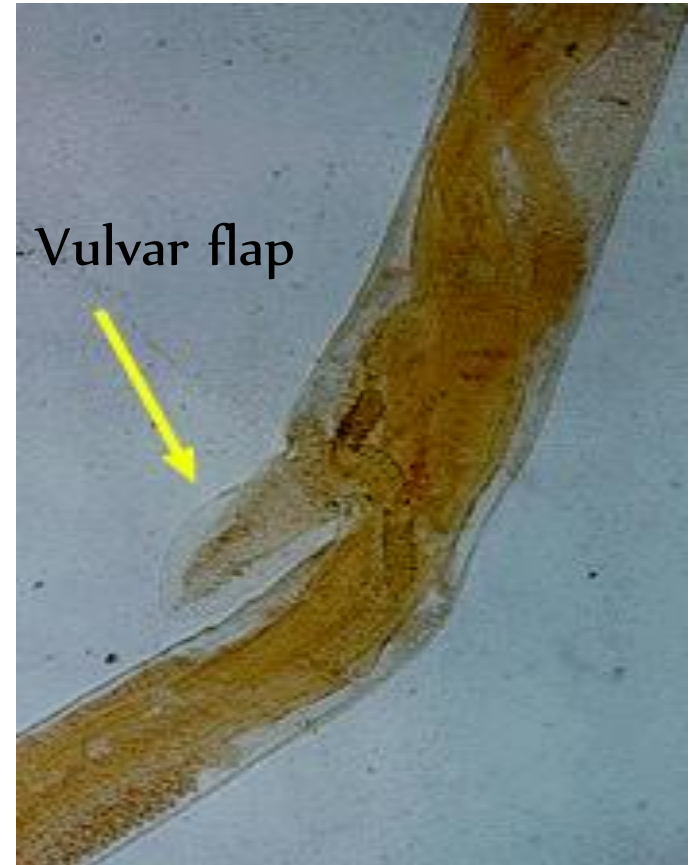
Male

- Has large bursa especially the lateral lobes supported by long slender rays.
- Has asymmetrical dorsal lobe situated against the left lateral lobe & is supported by “Y” shaped dorsal ray
- Each spicule is provided with small barb near its proximity hence called **barbed spicules**.



Female

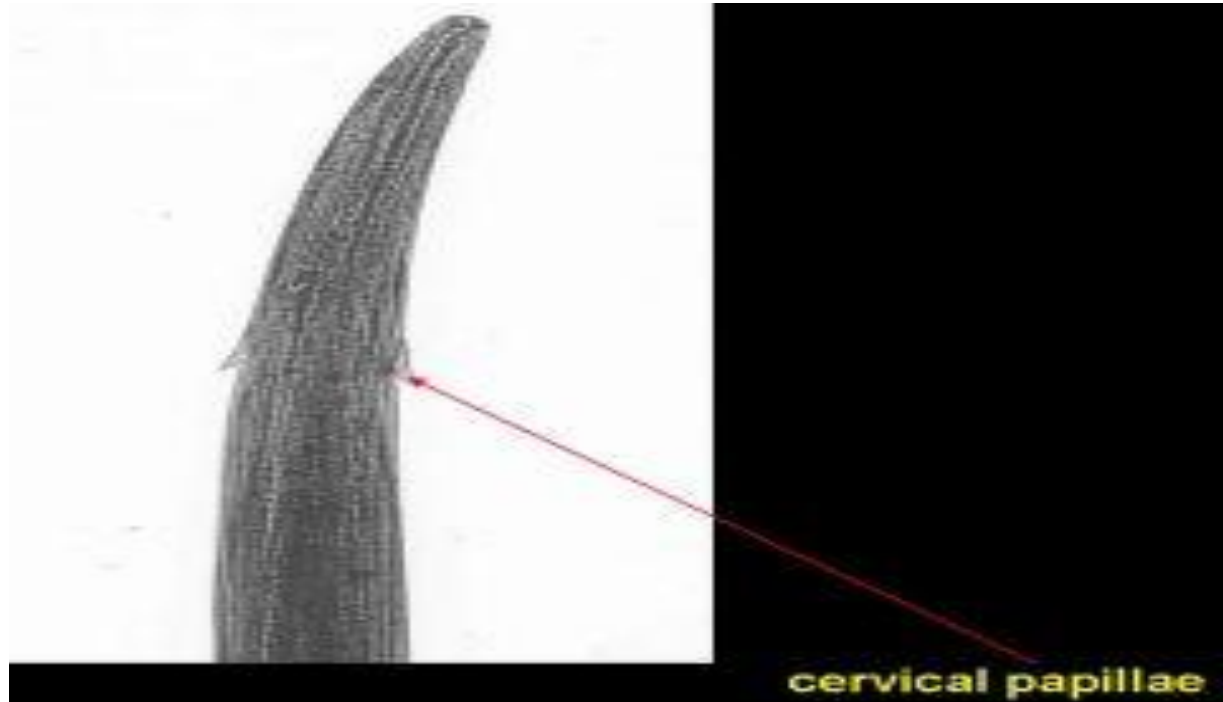
- Vulva is posterior & usually covered by vulvar flap.
- The shape of vulva can be linguiform, knobbed or Smooth.
- Females are highly prolific hence numerous eggs can be seen inside the uteri



Both sexes

- Have a small buccal cavity with slender lancet
- Possess prominent cervical papillae

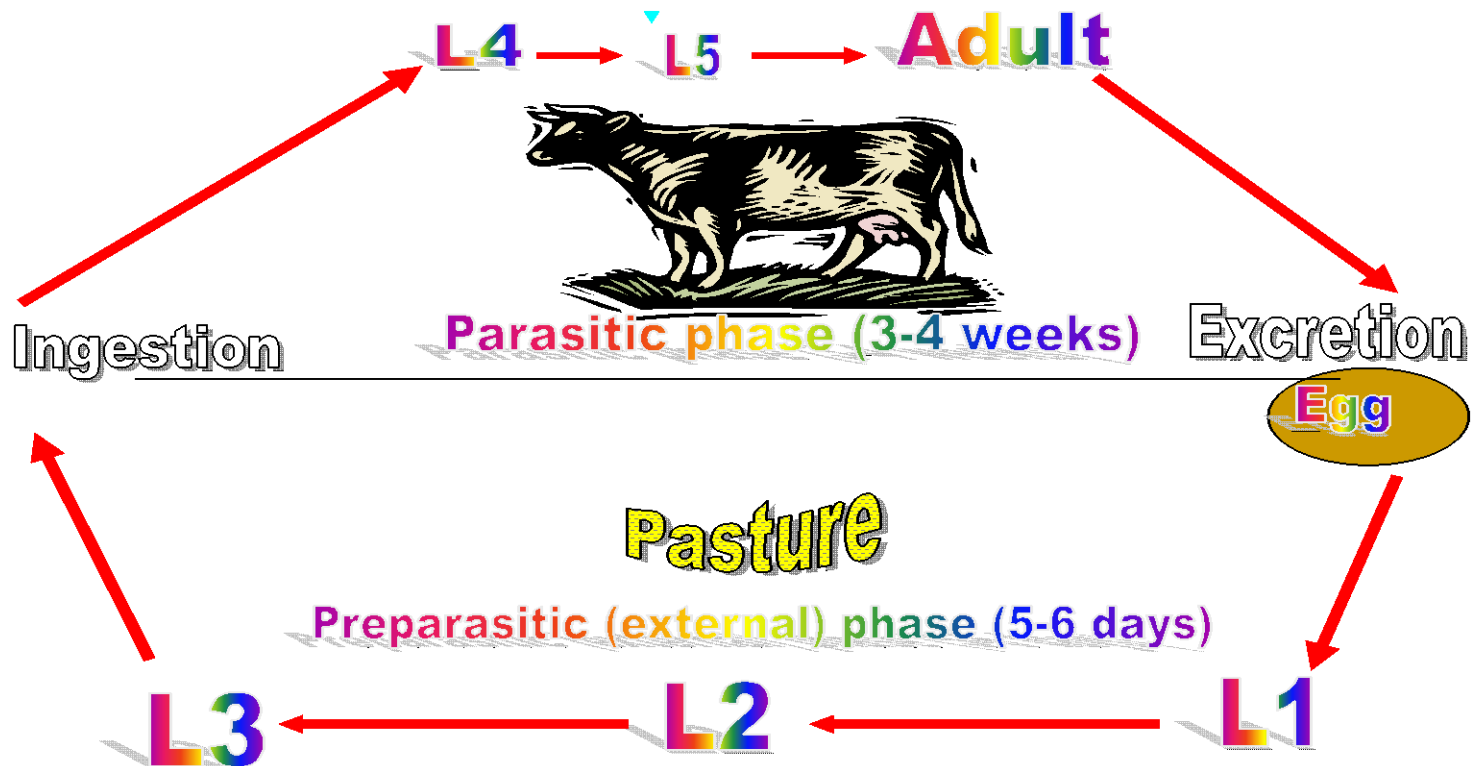
lancet



Life Cycle

- Direct and the preparasitic phase is typically trichostrongyloid
- Females are prolific egg layers. The eggs hatch to L₁ on the pasture and may develop to L₃ in as short a period

Haemonchus spp (Life cycle: direct)



Pathogenesis:

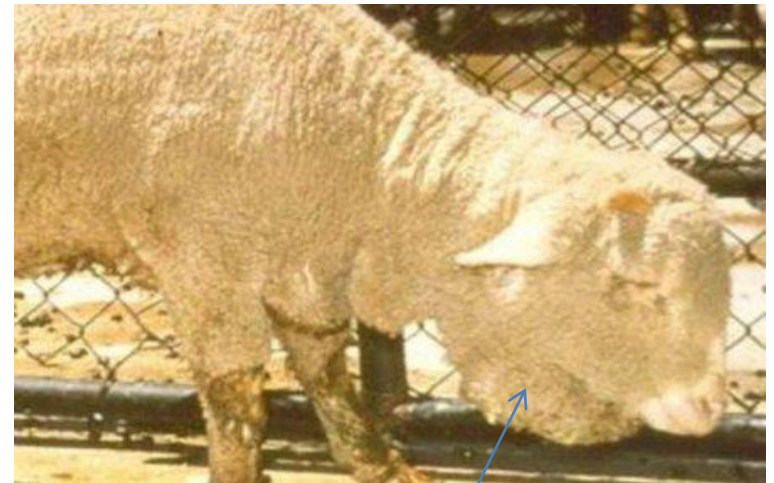
- Related with its voracious blood sucking habit
- Each parasite removes 0.05 ml of blood per day by ingestion & seepage.
- Continual loss of Fe & protein into the GIT → reduced pcv & Hb.
- Numerous hemorrhagic lesions
- Pale & edematous carcass

Diagnosis

- History & clinical signs pale mucus membrane and edema (bottle jaw) supported by high epg.
- Coproculture to identify L_3 .
- Definitive diagnosis is by conducting autopsy & examination of abomasums.
- Necropsy such as pale carcass, organs



Pale mucus membrane(anemia)



Edema (bottle jaw)

SELF-CURE

- Is the expulsion of the major parts of adult worm burden after a period of heavy rain causing drop in the faecal egg counts sharply to near zero level.
- Is caused by immediate hypersensitivity reaction to antigens of L_3 .
- Has multibenefit to both the host & the parasite

Treatment and Control

- Regular use of modern benzimidazoles at 2-4 weeks as prophylactic treatment
- Use of genetically resistant breeds of animals like Red Massi of Kenya, etc
- Recombinant vaccine based on a membrane glycoprotein of intestinal microvilli of parasitic stages of *H. contortus* is understudy.

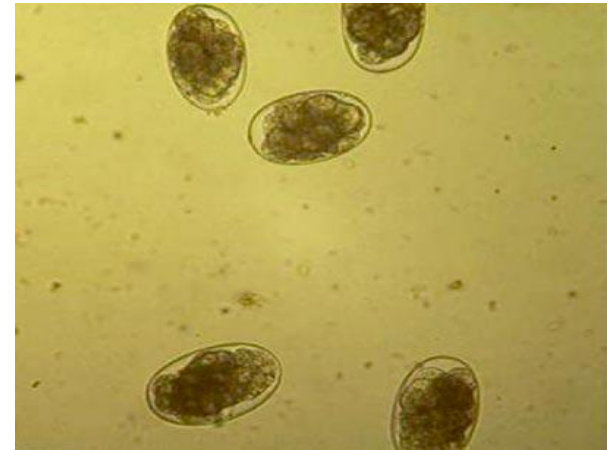
B. Genus: Trichostrongylus

- In subtropics it is one of the most important causes of parasitic gastroenteritis.
- **Hosts** – ruminants, horses, pigs, rabbits & fowls
- **Site** – small intestine except *T.axei* & *T.tenuis*

Parasite species	Host	Site
<i>T. vitrinus</i> , <i>T. capricola</i>	Sheep & goats	Small intestine
<i>T. colubriformis</i>	Ruminants	Small intestine
<i>T. axei</i>	Horses, ruminants & pigs	Stomach, abomasum
<i>T. tenuis</i>	birds	Small intestine , caeca

Morphology

- Adults are hair-like and small, 1cm long and difficult to see with the naked eye.
- Have no obvious buccal capsule.
- The main generic character is the distinct excretory notch in the esophageal region.
- The eggs are typical **strongyloid**.
- Females have bluntly tapered tail and lack vulval flap



Life Cycle

- This is direct and the preparasitic phase is typically Trichostrongyloid.
- Under optimal conditions development from the egg to infective stage occurs in 1-2 weeks.
- The parasitic phase is **non-migratory**.
- The prepatent period is 2-3 weeks in ruminants, 25 days in horses and 10 days in birds.

Pathogenesis

- L_3 of intestinal species penetrate between the epithelial glands of the mucosa with formation of tunnels containing the developing worms.

- Considerable hemorrhage, edema & loss of plasma proteins into gut lumen.
- Distorted & flattened villi reduces nutrient & fluid absorption
- *T. axei* penetrate between the glands causing alteration in p^H & increased in permeability of the mucosa.
- Plaques & ring-like lesions are formed when nodules coalesced.

Diagnosis

- Based on **clinical signs**, **seasonal occurrence** of disease and lesion at **postmortem examination**.
- Fecal egg counts
- Faecal culture for **generic** identification of L_3

Epidemiology

- Embryonated eggs & infective L_3 have a high capacity for survival under extreme cold or desiccation.
- Ability to under go hypobiosis at L_3 stage.
- Immunity is slowly acquired & **it wanes** during perparturient period.

C. Genus: **Ostertagia**

- Major cause of parasitic gastritis in **ruminants** in temperate areas of the world.
- Also called **brown stomach** worm.

Species :

- *O. ostertagi* ----- *cattle*
- *O. circumcincta* ----- *sheep and goat*
- *O. trifurcata* ----- *sheep and goat*

Morphology

- **Slender reddish-brown** worms up to 1cm long, visible on the abomasal mucosa on close inspection.
- **Larval stages** can be microscopically identified in the **gastric glands** following processing of gastric mucosa.
- Species differentiation is based on the structure of the **spicules** which usually have three distal branches

Life cycle

- Is direct
- L₃ exsheaths in the rumen

Pathogenesis

- The emerging parasites from the gastric glands cause extensive pathological and biochemical changes and severe clinical signs
- Reduction in functional gastric glands mass responsible for the production of HCl.
- Parietal cells are replaced by rapidly dividing undifferentiated cells
- Macroscopically raised nodule with central orifice in heavy infection that coalesce to form a condition called **morocco leather**.

- In heavy infection of 40,000 or more adult worms the p^H increases from 2.0 to 7.0
- This results in failure to activate pepsinogen to pepsin & to denature protein
- Loss of bacteriostatic effect in the abomasum.
- Macromolecules like pepsinogen & plasma proteins pass into & out of the epithelial sheet.
- This leads to elevated plasma pepsinogen & hypoalbuminaemia.
- In heavy infection there is a reduction in the acidity of the abomasal fluid.
- Results in failure to activate pepsinogen to pepsin and loss of bacteriostatic effect in the abomasum.

Diagnosis

❖ In young animals this is based on:

- The clinical signs (inappetance, weight loss and diarrhea).
- The season
- The greathing history
- Fecal egg counts
- Plasma pepsinogen level
- Post-mortem examination

Control

- Routinely treating young cattle with anthelmintics over the period when **pasture larval levels** are increasing.
- Limiting exposure to infection

D. Genus *Cooperia*

- Some species are very important in tropics & sub-tropics
- **Host** -ruminants
- **Site** -Small intestine
- **Species**
 - ✓ *C. onchophora*, *C. punctata* & *C. pectinata* → cattle
 - ✓ *C. surnabada*(*C. macmasteri*) → cattle & sheep
 - ✓ *C. curticei* → sheep & goats

Morphology

- In size similar to *Ostertagia*
- Reddish color when fresh

Life cycle

- Direct & typical of the superfamily

Pathogenesis

- *C. punctata*, *C. pectinata* & *C. surnabada* penetrate epithelial surface leading to villous atrophy the reduction in absorption.
- But *C. oncophora* & *C. cruticei* are mild pathogens & associated with inappetance & poor growth.



Diagnosis, Treatment & Control

- The principle is as for Haemonchosis & Ostertagiosis

E. Genus *Nematodirus*

- Very important & common in lambs in temperate regions
- **Host** _ ruminants
- **Site** _ small intestine

Species

- *N. battus* - sheep rarely calves
- *N. filicollis* - sheep & goats
- *N. spathiger* - sheep & goats rarely cattle
- *N. helvetianus* – cattle

Morphology

- Are relatively long with filiform anterior portion.
- Adults are slender about 2.0 cm in length
- Intertwining thin & twisted worms produce **cotton wool** appearance.

Life cycle

- Unique in that development to L₃ takes place inside eggs shell in 2 months.
- There is a large period before hatching
- Non-migratory like other too

Pathogenesis

- Is attributable to larval stages due to disruption of iliac mucosa
- L₄ → L₅ coincides with villous atrophy & erosion of the mucosa
- Fluid & nutrient exchange ability of intestine is grossly reduced.

Diagnosis

- Clinical signs and post-mortem examination
- Ovaculture

Family: Dictyocaulidae

A. Genus : *Dictyocaulus* (Lung Worms)

- Causes a disease called **vermnious (parasitic) pneumonia**, -- **bronchitis** or simply Dictyocaulosis.
- Host** - Ruminants, horses & donkey
- Site** - tracheae & bronchi especially the diaphragmatic lobe

Dictyocaulus SPECIES	HOST SPECIES	PREDILECTION SITE
<i>Dictyocaulus viviparus</i>	Cattle and deer	Trachea and bronchi
<i>Dictyocaulus filaria</i>	Sheep and goats	Trachea and bronchi
<i>Dictyocaulus arnfeldi</i>	Donkeys and horses	Trachea and bronchi

Morphology

Gross

- Slender thread-like worms up to 8cm length
- Have a milk-white colour.

Microscopic:

- Intestine shows a dark line
- Very small, shallow buccal capsule
- Have 4 very small lips

Male

- Are 3 – 8 cm in length & have well developed bursa with typical rays
- Gubernaculum is oval & semi transparent

Female

- 5-10 cm in length
- Vulva is situated not far behind the middle of the body
- Ovoviviparous hence eggs contain fully formed larvae when laid.

Larvae

- Intestinal cells of L_1 are filled with dark brown food granules hence preparasitic stages don't need to feed on bacteria.
- Sheath of L_1 & L_2 are retained externally on sheath of L_3 for protection from adverse environmental conditions.

Life cycle

- L_1 migrate up the trachea & swallowed to pass out with faeces.
- L_3 leave faecal pat to reach the herbage either by their own motility
- Most eggs of *D. arnfieldi* don't hatch before being passed in the faeces.
- $L_3 \rightarrow$ penetrate intestinal mucosa $\rightarrow$ mesenteric lymph nodes to moult to $L_4 \rightarrow$ travel via lymph & blood to lungs $\rightarrow$ break out of capillaries into $\rightarrow$ alveoli air passages $\rightarrow$ $L_4 - L_5$ in bronchioles $\rightarrow$ adult matures in bronchi

Pathogenesis

- **Penetration phase** → larvae move to lungs & lesions aren't apparent
- **Prepatent phase** → alveolitis → bronchiolitis → bronchitis
- Cellular infiltrated of neutrophils, eosinophils & macrophages plug bronchial lumina causing collapse of other alveoli first signs.
- Death is due to interstitial emphysema & pulmonary edema.
- **Patent phase** → two main signs
 - **Parasitic bronchitis** → frothy white mucus , hyperplastic & infiltration of cells
 - **Parasitic pneumonia** → caused by aspiration of eggs & L₁ into alveoli as foreign bodies provoke cellular infiltration & consolidation of lobules.
- **Postpatent period** → recovery phase → post patent parasitic bronchitis

Diagnosis

- Clinical signs, time of the year & history of grazing
- Demonstration of L_1 in fresh samples taken from rectum

Treatment:

- Benzimidazoles, Imidazothialoles, avermectines & moxidectin

Control

- Vaccination by attenuated larval vaccines in endemic areas
 - There is only one successful commercial parasite vaccine but all the other yet not been commercialized.
- Treat potential worm carriers
- Avoid overstocking

2. Metastrongylus of Sheep & Goats

- **Host** - shoats
- **IH** – Mulluscs (snail & slugs)

Site

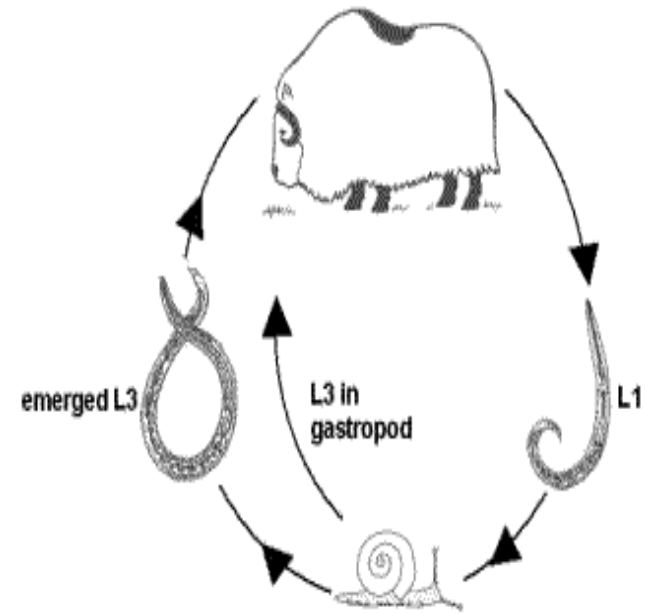
- *Muellerius capillaries* is the commonest one → alveoli.
- Protostrongylus → Small bronchioles

Morphology

- Are brown hair like worms up to 3 cm in length
- Are found embedded in lung tissue nodules

Life cycle

- Females are ovoviviparous → L₁ passes in the faeces
- L₁ penetrates foots of **mollusc** & develop to L₃
- Sheep is infected by ingesting L₃ containing mollusc freed by digestion
- L₃ travel to lungs by **lymphatic-vascular** route
- Parasitic moults occur in the mesenteric lymph nodes & lungs.



Pathogenesis

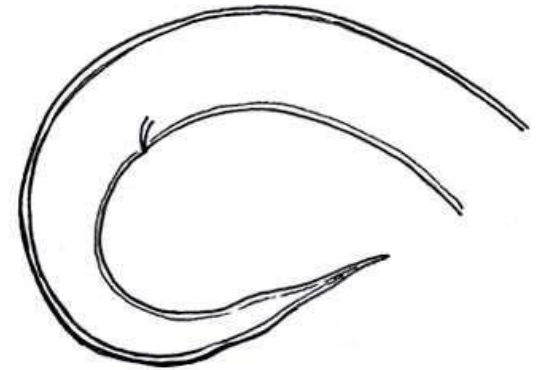
- Muellerius causes small, spherical & nodular lesions on lung surfaces
- Protostrongylus involves larger lung area causing **occlusion of a small bronchus**

Clinical signs

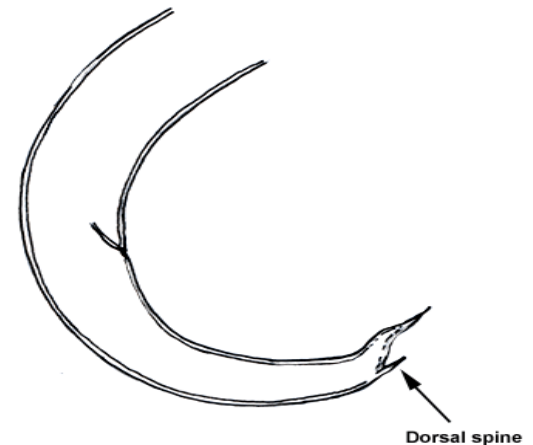
- Pneumonic signs are rarely observed
- Infections are almost always inapparent being encountered in **necropsy**

Diagnosis:

- Fecal examination for L₁
- L₁ of nodular lungworms don't have anterior protoplasmic knob
- Smaller in size than Dictyocaulus
- Transparent without dark intestinal granulation
- **Protostrongylus** has sharp end of tail unlike the blunt tail end in Dictyocaulus
- The tail end of **Muellerius** is "S" shaped with spine.



Protostrongylus



Muellerius

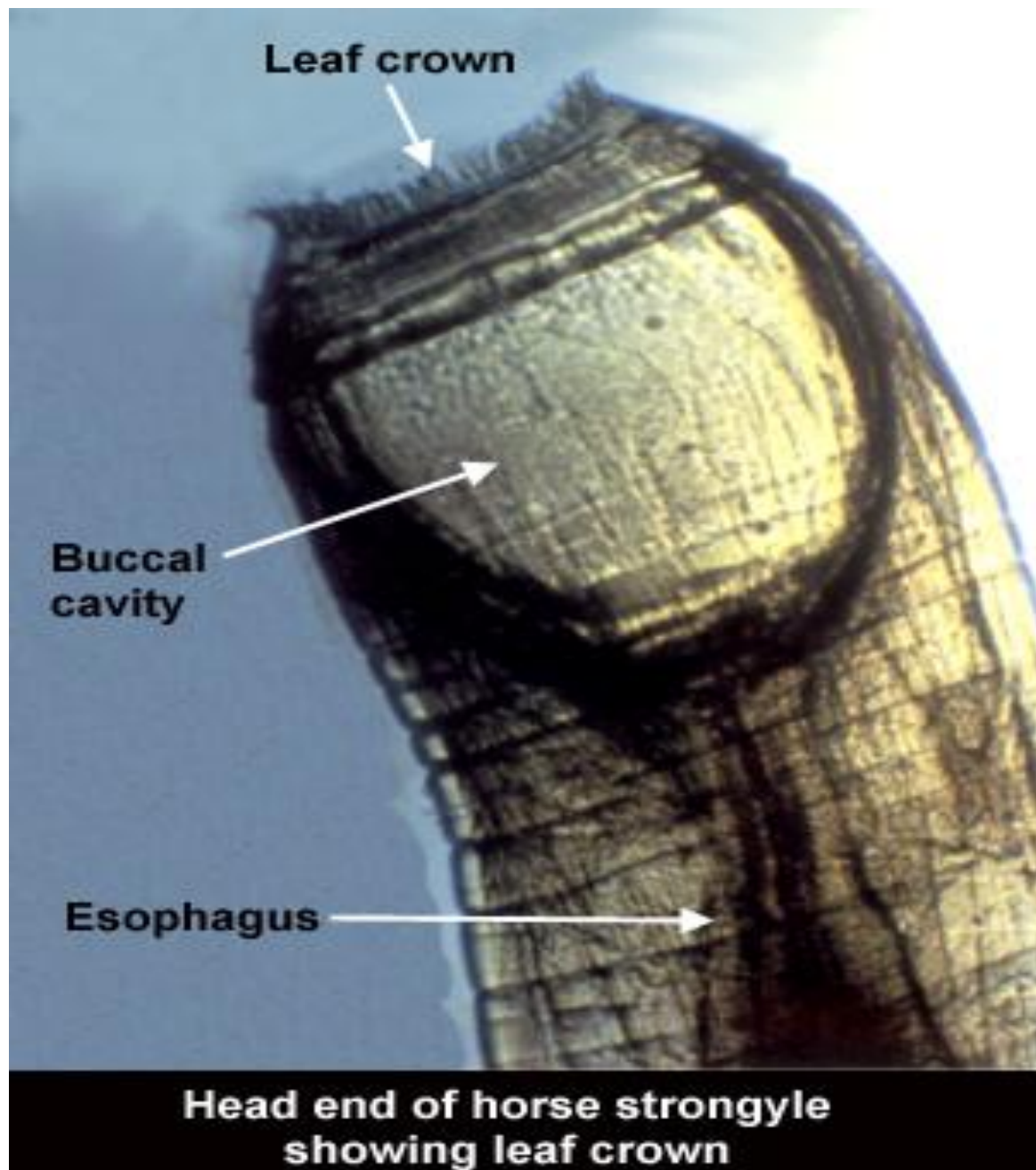
2.1.2. Superfamily: Strongyloidea

- Unlike the above two superfamilies they are characterized by the following features:
 - Large buccal capsule
 - Have teeth or cutting plates
 - Prominent leaf crowns (corona radiata) surrounds the mouth
 - Feeding is by ingestion of **plugs** of **mucosa**
- Are divided into two

A. STRONGYLES

- parasitic in the **large intestine**

B. HOOK WORMS



- The genera of veterinary importance are:
 - Genus Strongylus
 - Genus Triodontophorus}large Strongyles of equ.
 - Genus Cyathostomum(Trichonema)....small Strongayles of equ.
 - Genus Chabertia
 - Genus Oesophagostomum
- }Ruminant strongayles

1. Strongyles

1.1. Genus : *Strongylus* (Equine Strongyles)

- commonly called **large** or **migratory** strongyles
- **Host** – horse and donkey
- **Site** – caecum & colon
- **Species** – *S. vulgaris*, *S. edentatus* & *S. equinus*

Morphology

Gross

- Robust dark red
- Have well developed prominent buccal capsule

Microscopic

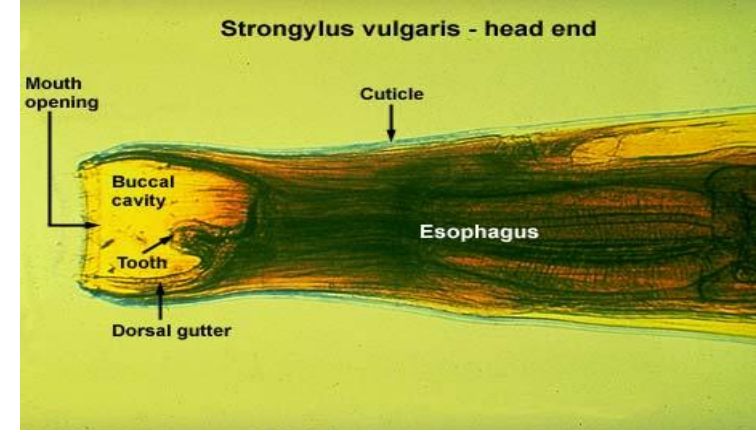
S. vulgaris

- Is from 1.5 – 2.5 cm in length & has 2 ear shaped rounded teeth
- L₄ migrate to cranial mesenteric artery & its main Branches
- Provoke **thrombus** formation in the involved arteries

S. edentatus → is 2.5 – 4.5cm in length & has no teeth

S. equinus → is from 2.5 – 5.0cm in length & has 3 conical teeth

- One teeth is situated dorsally & is larger than the others & is bifid.



Life cycle

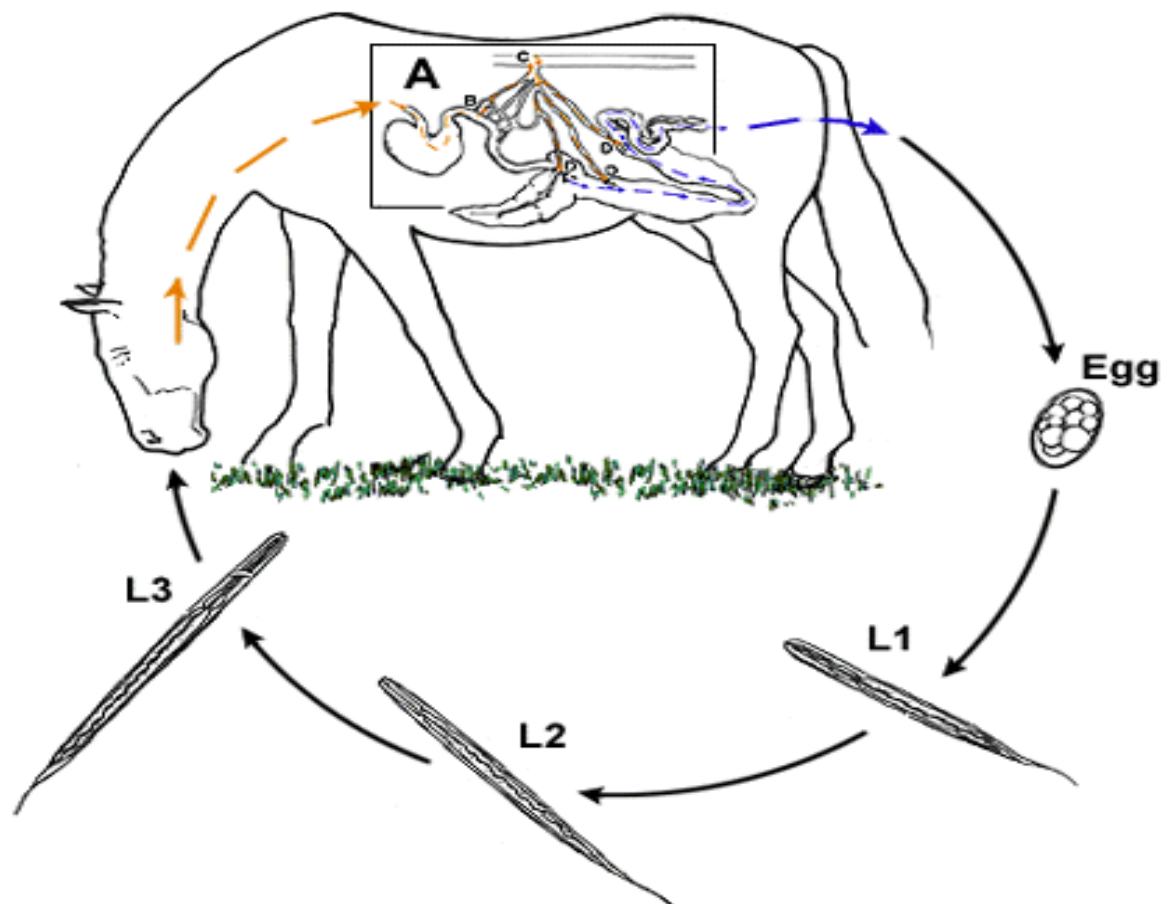
S. vulgaris

- L₃ moult to L₄ in the submucosa then enter small arteries
- Then migrate on the endothelium to cranial mesenteric arteries & its main branches then moult to L₅ & return back to intestinal wall via arterial lumina.
- In the wall of caecum & colon nodules form around L₅
- Has 6 –7 months of prepatency period

S. edentatus

- L₃ via portal system reach liver parenchyma & moult to L₄
- migrate to their predilection site to flanks & hepatic ligaments & moult to L₅
- migrate to subperitoneally to wall of large intestine where large **purulent** nodules is formed around them
- Has 10 –12 months of prepatency period

***Strongylus vulgaris* - Life Cycle**



S. equinus

- L₃ provoke nodule formation in the muscular & subserosal layer of intestines
- L₃ moult to L₄ within these nodules & then travel via peritoneal cavity to liver
- Then L₄ & L₅ are found in & around pancreas before appearance in GIT lumen
- 8 –9 months of prepatency period

Pathogenesis

Larvae

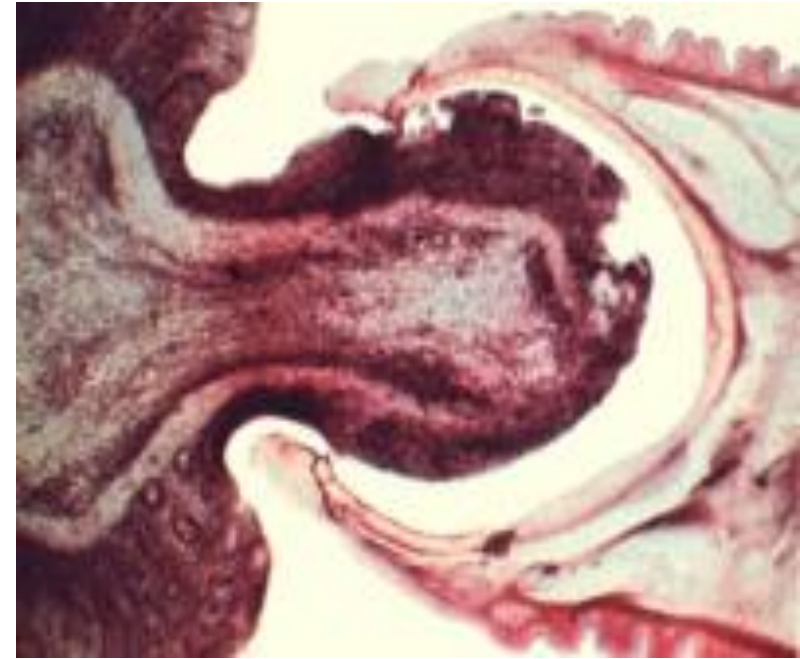
- *S. vulgaris* larvae provoke thrombus formation in the cranial mesenteric artery & its main branches.
- true **aneurysms** with dilatation & thinning of the arterial wall is initiated by it.
- *S. edentatus* there are gross causes haemorrhage in the liver & pancreas
- *S. equinus* there is only little work



Nodules caused by immature adults of *Strongylus vulgaris*



Large strongyles attached to the mucosa



Strongylus edentatus feeding by drawing a plug of mucosa into its buccal cavity

- hemorrhagic nodules in the small intestine by larvae of *Strongylus equinus*

Adults

- associated with damage to large intestinal mucosa by feeding habits of worms that cause hemorrhages, ulcers & anemia.
- Emergence of young adults into the lumen of intestines

Clinical signs

- Fever, inappetance & dullness
- Accompanied by **colic** due to pressure of cranial mesenteric artery
- aneurysm on associated nerve plexuses
- Unthriftiness, anemia & sometimes diarrhoea
- Impaired general performance in adult animals

Diagnosis

- Grazing history, clinical signs, loss of condition, & anemia all aid diagnosis
- Finding of oval, thin-shelled typical strongyle type eggs in the faeces
- Accurate identification can be made by faecal culture.
- Presence of aneurysm as large pulsating body (6-7cm) by rectal palpation

Family : Trichonematidae

- They are ruminant and pig strongyles

A. Genus *Oesophagostomum*

- Referred to as **Nodular worms** formation of nodules on the intestinal wall.
- Called pimply guts or knotty guts or Oesophagostomosis

Hosts _ ruminant & pigs

Site _ caecum & colon

Species

- *Oe. columbianum*-----→ sheep & goats
- *Oe. venulosum*-----→ sheep & goats
- *Oe. radiatum*-----→ cattle & buffalo
- *Oe. dentatum* & *Oe. quadrispirulatum* -----→ pig

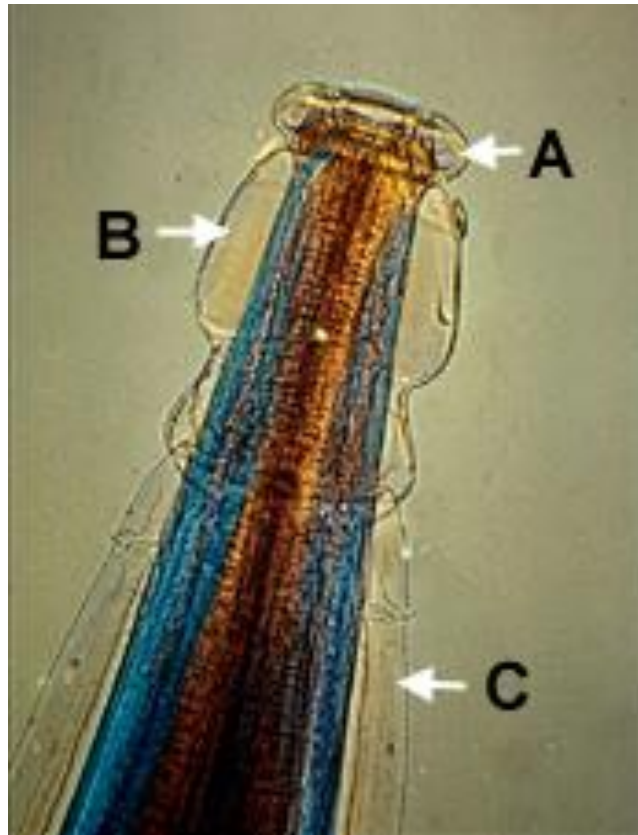
Morphology

Gross

- Stout white up to 2cm in length
- Tapered head distinguishes it from Chabertia

Microscopic

- Have white to grayish white colour
- Have cylindrical narrow buccal capsule surrounded by leaf crowns
- Vulva of females is near posterior end
- Compressed external leaf crowns
- Have hooked-shaped at one extremity due to large cervical alae
- The genus is characterized by having
 - **inflated cuticular cephalic vesicle** terminating in **ventral cervical groove** followed by broad **cervical alae** in some species.



A= cephalic vesicle

B = cervical vesicle

C = cervical alae

Life cycle

- L₃ enter mucosa of intestine & enclosed in obvious nodules & moult to L₄
- Nodules are absent in *Oe. venulosum* unlike in *Oe. Columbianum*, *Oe. radiatum* & *Oe. quadrispirulatum* all causes formation of nodules.
- L₄ emerge onto mucosa & migrate to colon to develop to adult.
- Can be arrested as L₄ for 1 year in the nodules.

Pathogenesis

- All species can cause severe enteritis
- Nodule formation provokes severe inflammatory response
- Nodules containing green puss & L₄ can reach 2cm in diameter
- Emergence of L₄ causes mucosal ulceration & diarrhoea
- Severe infection causes ulcerative colitis leading to debilitation & reduction in production

Clinical signs

- Severe dark green diarrhoea
- Rapid loss of weight & submandibular edema
- Primarily chronic → Inappetance, & emaciation with intermittent diarrhoea & anaemia.

Diagnosis

- Clinical signs, postmortem, eggs in the faeces
- L₃ can be identified after coproculture.

b. Genus *Chabertia*

- **Hosts** _ sheep & goats, rarely cattle, wild ruminants
- **Site** - colon
- **Species** - *C. ovina*

Morphology

- White up to 2 cm **long largest nematode** in colon
- Anterior end appears as if cut off squarely due to large mouth
- Markedly truncated & enlarged anterior end

Life cycle

- L₃ penetrate the wall of the small intestine & moult.
- L₄ appear in the lumen & migrate to caecum to moult to L₅ then migrate to colon



Pathogenesis

- Large buccal capsule of L₅ & adult worms cause haemorrhage as they browse in the mucosa
- Hypoproteinaemia & weight loss due to leakage of plasma in to intestine & albumin loss & its catabolism.
- Anaemia with diarrhoea due to tissue damage is caused in severe infestation

Clinical Signs

- Mass loss, reduction of wool growth & anemia
- Faeces flecked with blood & mucus
- Infected animals usually harbor small numbers of parasites

Diagnosis

- Finding eggs in the feces & specific lesions caused in necropsy
- Identification of L₃ in faecal cultures

Family: Syngamidae

a. Genus *Syngamus*

- Commonly called Gape worm and cause gape worm disease

Hosts - domestic fowl & game birds (poultry)

- **Site** - Trachea
- **Species** - *S. trachea*

Morphology

Gross

- Reddish large female & small male are in permanent copula forming a “Y” shape
- The only parasite inhabiting trachea of domestic birds

Microscopic

- Large shallow buccal capsule with up to 10 triangular teeth at their base
- Ellipsoid thin shelled eggs has operculum at their both ends
- Female vulva is near posterior end

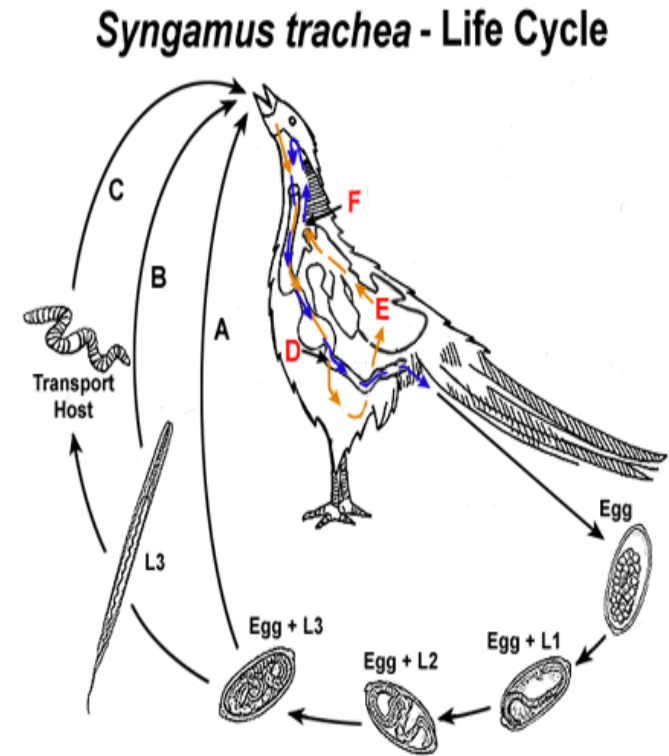


Life cycle

- L_3 develop in the egg infection can be by ingestion of hatched L_3 or unhatched L_3 or by ingesting earthworm, slugs, snails or beetle transport host.
- Then in the FH follows an entero-hepato-pulmonary migration

Pathogenesis

- Especially in young birds migration in the lungs cause pneumonia & death
- Haemorrhagic tracheitis & excess mucus production

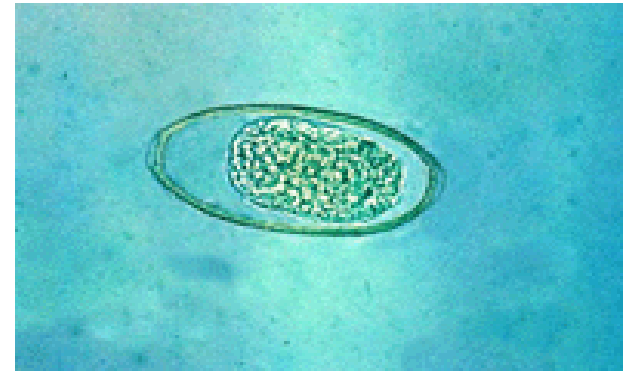


Clinical signs

- Pneumonia, dyspnoea, & depression
- Asphyxia & gasping for air
- Shaking & coughing
- In less severe cases there is weakness, anemia& emaciation

Diagnosis

- Clinical signs & typical eggs in the faeces & adults in the tracheal mucosa



Family: Stephanuridae

Genus *Stephanurus*

- Is a kidney worm of swine which cause Kidney worm diseases of pigs

Hosts - pig

Site - kidney & perirenal tissues

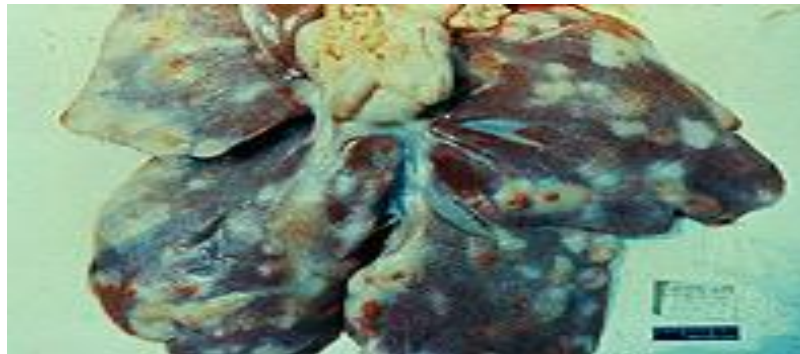
Species - *S. dentatus*

Morphology

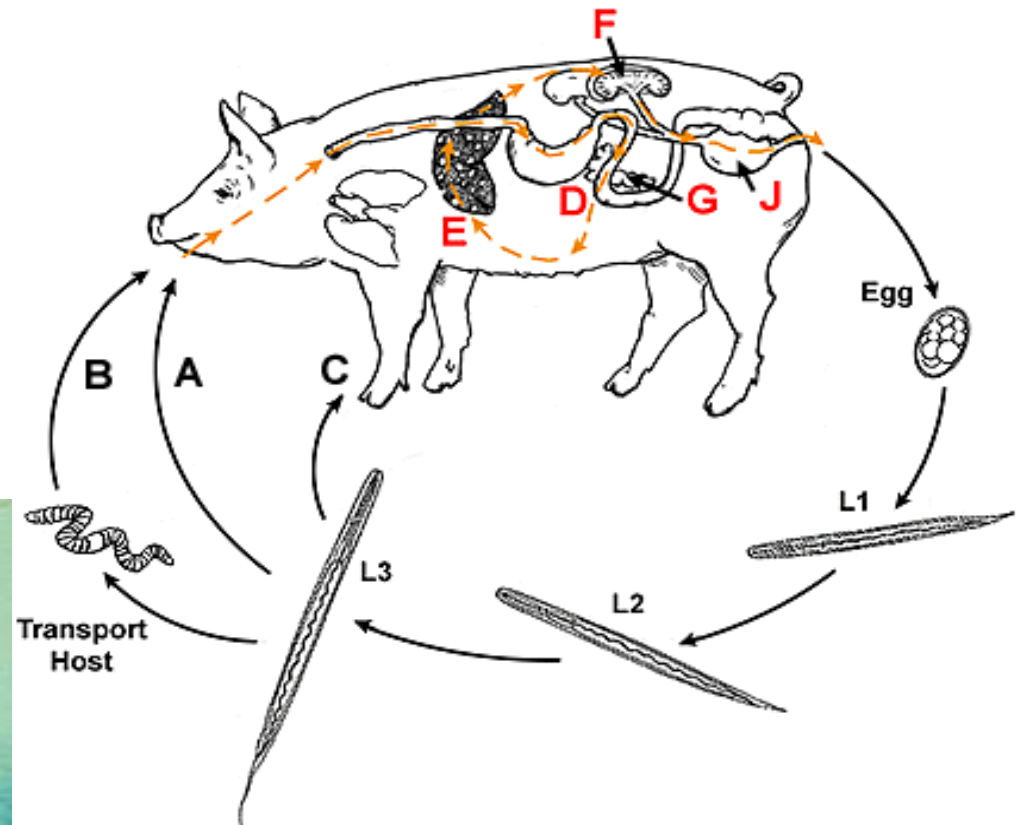
- Pinkish, large, stout 2-4.5 cm in length.
- Have transparent cuticle & large buccal capsule
- Irregular & broad ellipsoidal eggs pass with urine.

Pathogenesis

- Migrating larvae cause cirrhosis, scar formation & hepatic thrombosis, peritonitis, urethritis & cystitis.



Stephanurus dentatus - Life Cycle



B. Hook Worms

1. Hook Worms of Ruminants

a. Genus *Bunostomum*

- **Hosts** - ruminants
- **Site** - small intestine
- **Species** - *B. phlebotomum* → cattle
- *B. trigonocephalum* → sheep & goats

Morphology

Gross

- Largest nematode in the small intestine up to 3 cm long
- Hooked at anterior end

Microscopic

- Anterior end bent dorsally so buccal capsule opens anterodorsally
- Large buccal capsule bears marginal pair of chitinous cutting plates
- internally it has large dorsal cone carrying the duct of esophageal gland.



Life cycle

- L_3 infects percutaneous or orally
- Infection by percutaneous route is followed by pulmonary migration where L_3 ----- $\rightarrow L_4$

Pathogenesis

- 100-500 worms can cause anemia & hypoalbuminaemia

Clinical signs

- Anemia, loss of weight, occasional diarrhoea, foot stumping & signs of itching

Diagnosis

- Eggs are more bluntly rounded & usually debris found adherent to the thick sticky shells & lower epg than Haemonchosis.
- Accurate diagnosis requires L_3 identification by coproculture

2.1.3. Superfamily Ancylostomatoidea

Family: Ancylostomatidae

- Are hook worms of dog and cats

A. Genus *Ancylostoma*

Hosts - dog, cat & fox

Site - small intestine

Species

- *A. caninum* -----→ dog & fox
- *A. tubaeforme* ----→ cat
- *A. braziliense* ----→ dog & cat

Morphology

Gross

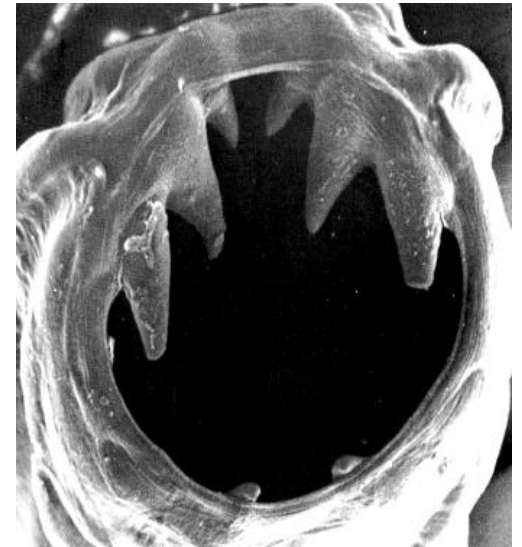
- Smaller size of 1-2cm in the small intestine
- Characteristic anterior hook posture

Microscopic

- Anterior end is bent dorsal & oral aperture is directed anterodorsally
- Large & deep buccal capsule with marginal teeth
- *A. caninum* & *A. tubaeforme* has 3 pairs of teeth
- Where as *A. braziliense* has 2 pairs of teeth



A. braziliense



A. caninum

Life cycle

- Infection can be by either skin penetration or ingestion
- In percutaneous infection L_3 travel via blood to lungs & moult to L_4 in the bronchi & trachea then swallowed & pass to small intestine to moult to L_5
- if infection is by ingestion L_3 penetrate buccal mucosa to undergo pulmonary migration or pass directly to the intestine to develop to L_5
- In susceptible bitches portions of L_3 that reach lungs migrate to skeletal muscles to remain in state of dormancy & later infect suckling young pups for 3 weeks for 3 consecutive litters

- Dormant L_3 can also resume (recommence) migration months or years later to become L_5 in the intestine.
- In *A. tubaeform* of cat & *A. braziliense* of dog & cat there is no evidence of transmammary transmission
- The major importance of *A. braziliense* of dog & cat is it is the primary cause of **cutaneous larval migrans** in Human.
- **Cutaneous larval migrans** is a tortuous erythematous inflammatory tracts within the dermis & severe pruritis by L_3
 - L_3 penetrates skin & wanders in the dermis for weeks but don't develop to L_5 .

Pathogenesis

- Acute or chronic haemorrhagic anemia & Fe deficiency
- Each worm removes about 0.1 ml of blood per day
- Skin reactions like eczema & ulceration

Clinical signs

- Anemia, lassitude - respiratory embarrassment
- Diarrhoea containing blood & mucus
- Loss of weight, poor coat, loss of appetite & pica
- Skin lesions & lameness

Diagnosis

- Clinical signs, history & faecal examination especially high epg

Treatment

- Bezimidazoles
- Parenteral Fe administration & supply of protein rich diet in severe cases

Control

- Regular anthelmintic therapy every 3 months & hygiene



Order Ascaridida

2.1.4. Superfamily Asacaridoidea

Family Ascarididae

Common characteristics

- Are most common & largest nematodes
- Have simple morphology
- Inhabit small intestine
- Cause significant economic loss
- Both larvae & adults stages have **deleterious** effect

Common morphology are

- Large, white opaque worms
- Not attach to the intestinal mucosa
- Lack buccal capsule
- Mouth is surrounded by 3 lips (1 dorsal & 2 subventral)
- Esophagus usually lacks posterior bulb
- Aren't bloodsuckers or plug feeders but are feed on gut contents.
- Males lack copulatory bursa but has 2 spicules
- Females are oviparous & highly prolific
- Tail of female is blunt but in male is frequently coiled



- Usually direct life cycle but can involve Paratenic or transport hosts
- Infection is by sticky thick-shelled egg containing L₂, prenatal, lactogenic or paratenic host.
- Eggs are highly resistant
- *A. suum* up to 40cm
- *T. canis*, *T. cati* & *T. leonina* all are up to 10cm
- *T. vitulorum* is up to 30 cm
- *P. equorum* is up to 50cm
- Ascaridia are up to 12 cm
- *H. gallinurum* is up to 1.5 cm in length

A. Genus *Ascaris*

Host: Pig

Site: Small intestine

Species: *A. suum*

Distribution: World wide

Identification

- Largest nematode of Pig
- Female up to 40 cm length
- The egg is ovoid and yellowish with a thick shell
- the outer layer is irregularly mamillated

Life cycle

- The egg is very resistant to temperature extremes and is viable for more than 4 years
- the egg hatches in the SI and L2 travel to the liver
- L3 then pass to blood stream to the lung and to the small intestine
- If eggs ingested by paratenic hosts/earth worms or dung beetle they will hatch in to L2 remain for years.
- The PPP is b/n 6-8 wks and each female worm is capable of producing more than 200,000 egg/ day

Pathogenesis

- Transient pneumonia/Ascaris pneumonia/
- In the liver migrating larvae can cause “milk spot” of up to 1.0cm in diameter
- If large number of adult worms present in the intestine they may obstruct the passage

Clinical signs

- Production loss (diminished weight gain)
- pneumonic signs and also obstructive problems of intestine and biliary system
- Occasionally infect cattle, causing an acute, atypical, pneumonia which may provoke fatal.

Diagnosis

- Clinical sign
- presence of yellow- brown ovoid eggs, with thick mamillated shells
- Being dense, the egg float more readily saturated solutions of zinc sulfate (MgSO_4) than NaCl.

Treatment

- Benzimidazoles given in the feed
- Levamisole and Ivermectin in injectable form

Control

- The chief problem is the survival of eggs
- In housed Pigs, strict hygiene in feeding and bedding with hosing of walls or floors
- Discontinue the use of paddocks for several years

B. Genus *Toxocara*

- In many respects typical ascaridoids, their biology is sufficiently varied for it

Toxocara canis

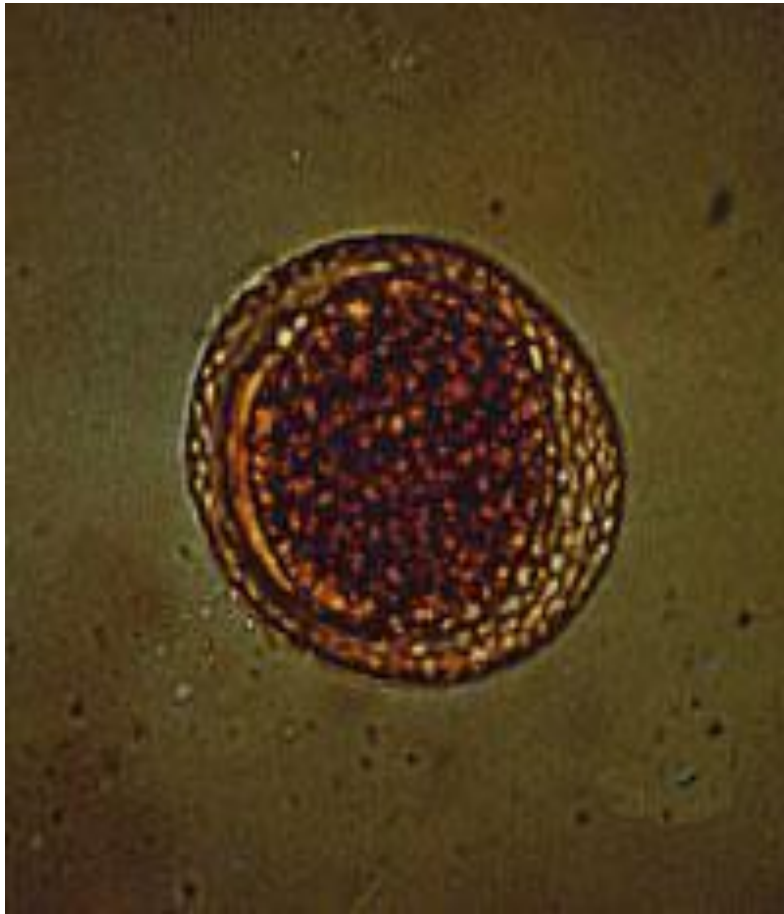
Host: dog

Distribution: World wide

Site: Small intestine

Identification:

- Large white worm up to 10 cm in length
- Difficult to differentiate from *T. leonina*
- The only useful character
 - a small finger- like process on the tail of the male *T. canis*.



Life cycle

- Complex life cycle with four modes of infection
- The basic form is typically ascaridoid
 - egg containing the L2 being infective four weeks after being passed.
- Ingestion → SI hatching to L2 → travel by the bloodstream via the liver → lungs where 2nd moult occurs, L3 returning via the trachea → Intestine where final two moults occur → occurs regularly only in dogs of up to 3 months old.
- In dogs over 3 months of age, the hepatic tracheal migration occurs less frequently at six months it has almost ceased.
- Instead, the L2 travel to wide range of tissue.

- In the pregnant bitch **prenatal infection** occurs, larvae becoming mobilized at about 3 weeks prior to parturition
 - and migrating to the lungs of the foetus where they moult to L3 just before birth
- A bitch, once infected, harbor sufficient larvae to infect all her subsequent litters, even if she never again encounters the infection
- The suckling pup may also be infected by ingestion of L3 in the milk during the 1st 3 weeks of lactation and no migration in the pup following infection by this route.
- Paratenic hosts such as rodents or birds may ingest the infective eggs, and L2 travel to their tissues.

Pathogenesis

- Adult worms provoke little reaction in the intestine
- In heavy infections
 - Pneumonia and Pulmonary edema
 - Mucoid enteritis
 - Partial or complete occlusion of the gut
 - Peritonitis and total blockage of the bile duct

Clinical signs

- No in mild to moderate infections
- In heavy infections
 - Coughing, Increased respiratory rate and FORTHY nasal discharge
 - Fatality in larval phase in puppies borne infected with transplacentally
 - Nervous convulsion

Diagnosis

- Tentative diagnosis during the pulmonary phase
- Eggs in faeces, subglobular and brown with thick pitted shells

Treatment and control

- Anthelmintics: piperazine superseded by the benzimidazole, Fenbendazole etc

control in young dogs;

- all pups should be treated at two weeks of age and again 2-3 weeks later to eliminate prenatal infection
- the bitch should be treated at the same time as pups

- Adult dogs should be treated 3-6 months interval throughout their lives
- pre- partum and post-partum treatment has largely eliminate transmammary and prenatal infection of the pups

Visceral Larva Migrants

- represent in human alone in particular the larvae of *T. canis*
- occurs most commonly in children who have had close contact with household pets or public parks contaminated by dog faeces

Control

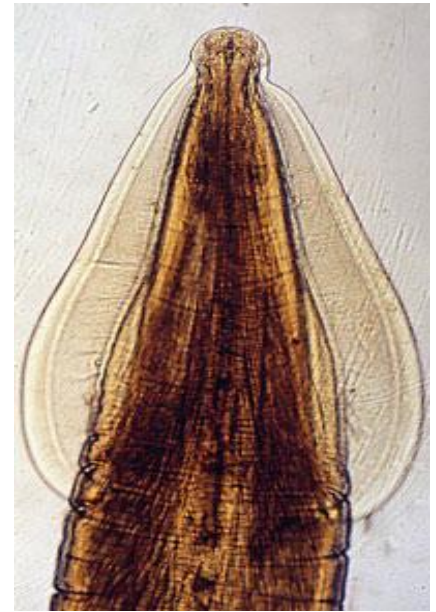
- In addition to anthelmintic regimen
- rate disposal of dog faeces and limitation of access by dogs to areas where children plays

Toxocara cati

- Host: cat
- Site: Small intestine
- Distribution: World wide

Identification:

- Large white worm often occur in mixed infection to *Toxascaris leonia*
- cervical alae of *T. cati* are seen to have **arrow-head** form with the posterior margins almost at a right angle to the body whereas in *Toxascaris* taper gradually into the body.



Toxocara cati



Toxascaris

Control:

- infection is 1st acquired during suckling complete control will be based on removal of kitten from the dam
(artificial rearing)
- early and repeated anthelmintics treatment

Toxocara vitulorum

- Host: cattle or buffalo
- Site: small intestine

Distribution: In tropical and warm regions

Identification

- Largest intestinal parasite of cattle the females being up to 30.0 cm long pinkish transparent cuticle
- Subglobular, thick pitted shell and colorless egg

Life cycle

- Resemble to *T. canis*.
- The larvae may present up to 30 days after parturition
- There is no tissue migration in the calf following infection
- larvae migrating to the tissues are stored in female , development in late pregnancy allows further transmammary transmission

Pathogenesis and clinical signs

- The main effects of by adult worms calves
- Heavy infections
 - poor thriving intermittent diarrhea
 - in buffalo calves fatalities may occur

Diagnosis: Faecal examination findings are characteristic

Treatment: Anthelmintics: - Piperazine, Levamisole and Benzimidazole

Control:

- treatment of calves at three and six weeks of age
 - preventing developing worms reaching patency.

C. Genus *Parascaris*

- It is a major cause of unthriftiness in young foals

Hosts: Horses and donkeys

Site: Small intestine

Species: *P. equorum*

Distribution: World wide

Identification

Gross:

- Large whitish nematode up to 40cm length can't confused with other parasites

Microscopic

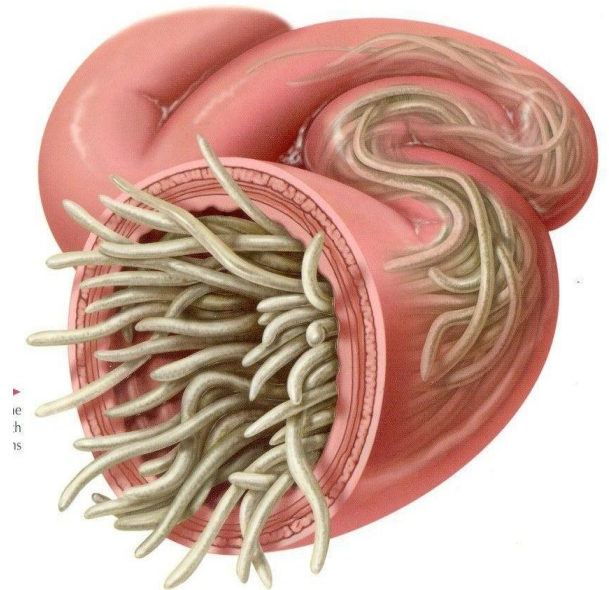
- Simple mouth opening surrounded by 3 large lips
- The male have small caudal alae in the tail and egg is spherical, brownish thick shelled with pitted coat

Life cycle

- L2 is an infective stage with 10-14 days
- After ingestion and hatching the larvae penetrate the intestinal walls reach to liver
- reached to the lungs → bronchi and trachea → swallowed return to SI

Pathogenesis

- Gross changes in the liver and lung resulting haemorrhages and eosinophilic infiltration.
- intestine heavy infection cause impaction and perforation leading peritonitis.



Clinical signs

- Frequent coughing
- grayish nasal discharge
- unthriftiness in young animals with poor growth rates
- dull coats and lassitude
- Fever, nervous disturbances and colic have been attributed to field cases of parascariosis

Diagnosis

- clinical signs
- spherical thick-shelled eggs on faecal examination.

Control

- Anthelmintic prophylaxis for the horse Strongyle will effectively control *P. equorum* infection
- Avoid using the same paddocks for nursing mares and their foals in successive years.

General clinical signs

- Weight loss, coughing, poor appetite, unthriftiness & poor growth
- Immunosuppression due to poor food conversion & slow weight gain
- Colic, evil smelling faeces & abdominal pain
- Diarrhoea alternating with constipation & dull coat.
- Marked abdominal enlargement (pot belly)
- Vomition so worms in the vomitus (due to their wandering about behavior) or stool
- Enzootic virus pneumonia in pigs

General Pathogenesis of Ascaridiod

- Migrating larvae cause destruction of tissue & hemorrhages in the liver.
- In the lungs pneumonic lesions, petechiae, bronchitis due to numerous haemorrhages in alveoli & bronchi
- Catarrhal enteritis or obstructive jaundice
- Robbing of food
- Partial or complete obstruction of intestine or bile lumen

Family : Heterakidae

Genera: *Ascaridia* , *Heterakis*

A. Genus *Ascaridia*

- a non-migratory ascaridoid

Hosts: Domestic and wild birds

Site: small intestine

Species:

- *Ascaridia galli* in chicken
- *A. dissimilis* in turkeys
- *A. columbae* in pigeons

Distribution: world wide

Identification:

- stout and densely white
- Females measuring up to 12.0cm length
- The largest nematode of poultry
- Oval with a smooth shell egg
- can not easily distinguished from poultry Heterakis

Life cycle

- Egg → infective with in three weeks
- The parasitic phase is non-migratory
- Egg is sometimes ingested by earthworm which act as transport hosts
- PPP 5-6 weeks and in chicks 8 weeks
- The worms live for about one year.

Pathogenesis and clinical signs

- not a highly pathogenic worm
- The prepatent phase
 - catarrhal enteritis usually
 - hemorrhagic in severe infection
- Large numbers
 - intestinal occlusion and death

Diagnosis

- Faecal examination
 - difficult to distinguish from heterakis eggs
- conformation
 - postmortem examination white worms will be found.
- In the PPP, larvae
 - in the intestinal contents
 - scraping of the mucosa



Organ : Intestine.

Disease : Adult ascarids.

Macro : Intestinal perforation

B. Genus *Heterakis*

- a small size worms
- Located in the LI is exceptional

Hosts: Domestic and wild birds

Site: caecum

Species:

- *H. gallinarum*
- *H. isolonche* in pheasants (game birds)

Distribution: World wide

Identification

- whitish worm up to 1.5 cm long with pointed tail
 - Unequal spicules length in *H. gallinarum*
 - equal length *H. isolonche*
- The egg is ovoid and smooth-shelled

Lifecycle

- Egg pass through the gut or paratenic hosts in which the egg hatches
- Then 3 moults occur in the caecal lumen. PPP is about 4 weeks.

Pathogenesis and clinical signs

- usually non-pathogenic
- Severe inflammation of the caeca
 - causes diarrhea with progressive
- emaciation and high mortality in *H. isolonche*

Diagnosis

- eggs in faeces or worms at necropsy
- *H. gallinarum* nodules containing adult worms
- Examination of spicules in *H. isolonche*

2.1.5. Superfamily Oxyuroidea

Family: Oxyuridae

- Commonly called pinworms
- Inhabit large intestine
- Have direct life cycle

A. Genus Oxyuris

- **Hosts** - horses & donkeys
- **Site** - caecum, colon & rectum
- **Species** - *O. equi*

Morphology

Gross

- thick anteriorly, narrowing abruptly to a thin tail.
- Females are up to 10cm in length & have pointed tails that is more than 3 times as long as the rest of the body
- mature males are less than 1cm.

Microscopic

- Double esophageal bulb
- Tiny males have caudal alae & a single pin-shaped spicule
- Females are larger than males & their vulva is situated near the anterior end.
- Eggs are ovoid, yellow & asymmetrical slightly flattened on one side with a mucoid plug (operculum) at on end.

Pathogenesis

- due to the feeding habit of the L_4
 - browse on the mucosa causes erosions of the mucosa
 - causing inflammatory response due to their large buccal capsule.
- Adult females during egg laying process cause intense anal pruritis (irritation)

Clinical signs

- Rubbing against any suitable object causes broken hair, bare patches & inflammation of the skin over the rump & the tail

Pathogenesis

- due to the feeding habit of the L₄
 - browse on the mucosa causes erosions of the mucosa
 - causing inflammatory response due to their large buccal capsule.
- Adult females during egg laying process cause intense anal pruritis (irritation)

Clinical signs

- Rubbing against any suitable object causes broken hair, bare patches & inflammation of the skin over the rump & the tail



Diagnosis

- Eggs are rarely found on faecal examination of sample taken from the rectum
- From perineal area by a method called Adhesive tape(scotch method, Anal swab method or Anal scrapping method) .

Order : Spirurida

2.1.6. Superfamily Spiruroidea

Family : Thelaziidae

- Have tight spirally coiled tail in males.
- Indirect lifecycle

A. Genus Spirocerca

- **Hosts** - dogs, rarely cat
- **Intermediate** - coprophagous beetles
- **Paratenic hosts** - reptiles, insectivores, rodents, birds that ingest infected beetles
- **Site** - granulomatous lesions of up to 4cm in size in the wall of esophagus----->adult
_ in the wall of aorta----->migrating larvae
- **Species** - *S. lupi*

Pathogenesis

- Larvae produce scarring of the internal wall of aorta resulting in stenosis, aneurysma or rupture.
- Cause complications
 - highly invasive esophageal osteosarcoma

Clinical signs

- Dysphagia, vomiting



Organ : Esophagus.
Disease: Spirocercu lupi.
Macro : Fibrotic nodules
are seen in the
esophageal mucosa

B. Genus Thelazia

- FH _ Cattle, other domestic animals, rarely man
- IH- muscid flies like *Musca*, *Fannia* & *Morellia*
- Site - conjunctival sac & lachrymal duct

Species

- *Th. Lacrymalis* ----->mainly equine
- *Th. rhodesi*, *Th. gulosa* & *Th. skrjabni* ----->cattle, buffalo, bison, shoats
- *Th. Californiensis* -----> dog, cat rarely sheep

Morphology

Gross

- Small thin white worms up to 2 cm long

Microscopic

- Mouth capsule is present
- Cuticle has prominent transverse striations at the anterior end
- Males have unequal spicules (left longer)
- Vulva is near posterior end

Life cycle

- Females are viviparous so muscid flies visiting the eyes ingest L_1 from lachrymal secretions that develop to L_3

Pathogenesis

- Serrated cuticle & active adult worm movement causes lesions like lachrymation then conjunctivitis

Clinical signs

- Lachrymation, conjunctivitis, & photophobia
- In severe cases opacity of cornea

Epidemiology

- Maximum fly activity

Treatment

- Manual removal under local anesthesia
- Levamisole or ivermectin or boric acid topically
- Diethylcarbamazine

2.1.7. Superfamily Trichuroidea

Family: Trichuridae

A. Genus *Trichuris*

- Commonly called whip worms due to their appearance

Species & Hosts

- *T. ovis*-----→sheep & goat
- *T. globulosa*-----→cattle
- *T. suis*-----→pig
- *T. vulpis*-----→dog
- *T. trichuria*-----→man
- **Site** - large intestine especially caecum

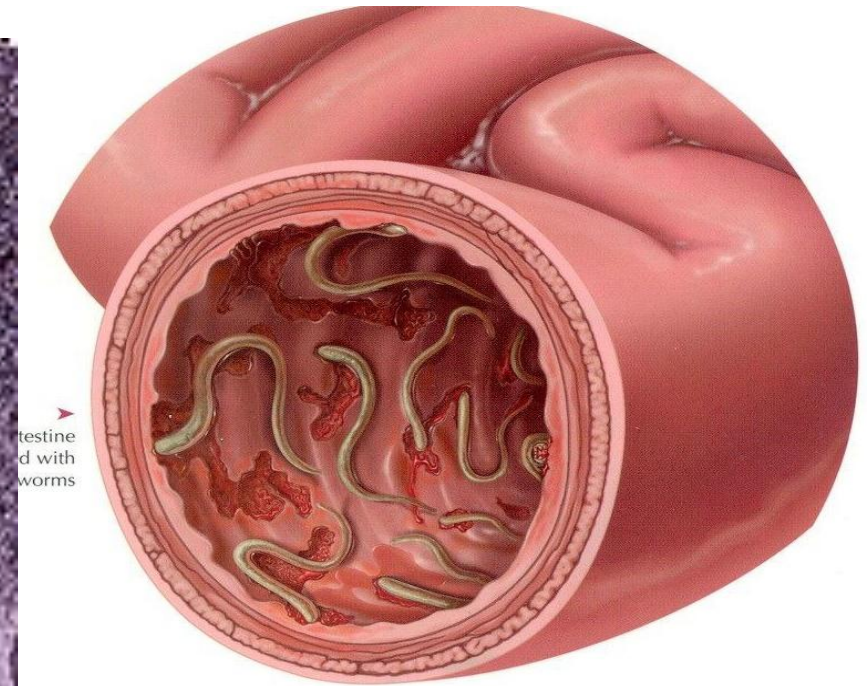
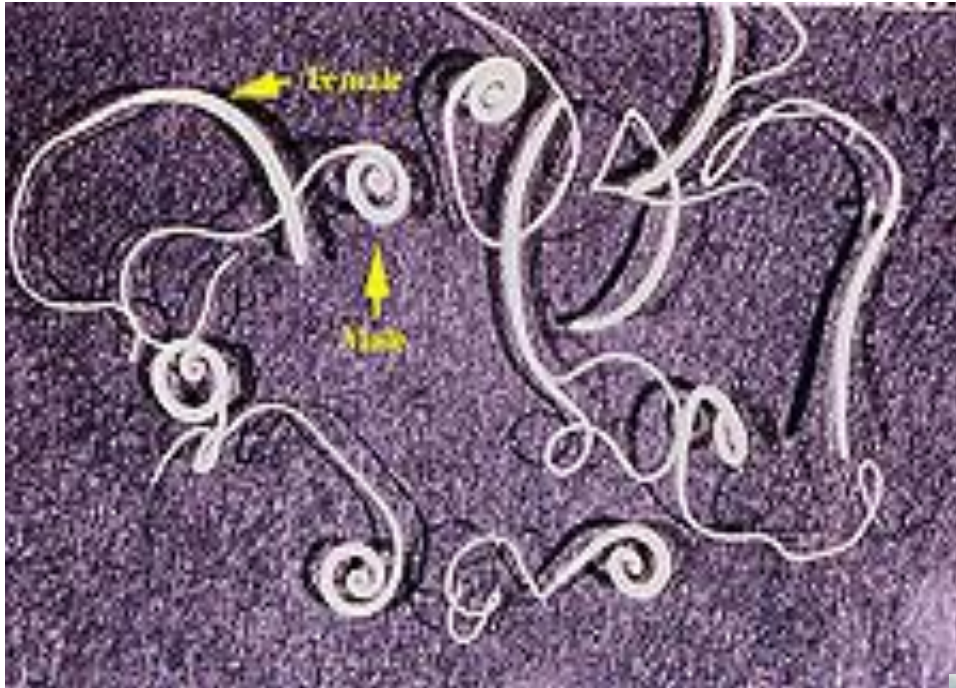
Morphology

Gross

- Have thick posterior end
- Tapering rapidly to a long filamentous anterior end found embedded in the mucosa
- The slender anterior end section takes $\frac{2}{3}$ & $\frac{3}{4}$ of the body length in males & females respectively.

Microscope

- Male tail is coiled (curled) & possesses a single spicule
- female tail is merely curved
- Vulva of females is situated at beginning of the wide part of the body
- Eggs are lemon shaped with conspicuous plug at both ends
- In the faeces eggs appear yellow or brown in colour



Pathogenesis

- Usually light & asymptomatic
- *T. vulpis* & others are blood feeders

Epidemiology

- Longevity of eggs for up to 3-4years

Clinical signs

- Finding eggs in the faeces
- In pigs anemia, dehydration, anorexia, dysentery & loss of weight

Treatment

- Pro & Modern benzimidazoles, avermectins or levamisole
- In dogs some of benzimidazoles & milbemycins
 - Are less effective against larval stages

Family: Trichinellidae

A. Genus : *Trichinella*

- Has a very wide host range
- Cause important worldwide zoonosis
- **Hosts** - most mammals, especially pig & man.
- **Sites** **adults** - small intestine
 _ **Larvae** - striated muscles like diaphragm, intercostals & master
- **Species** - *T. spiralis*

Morphology

Gross

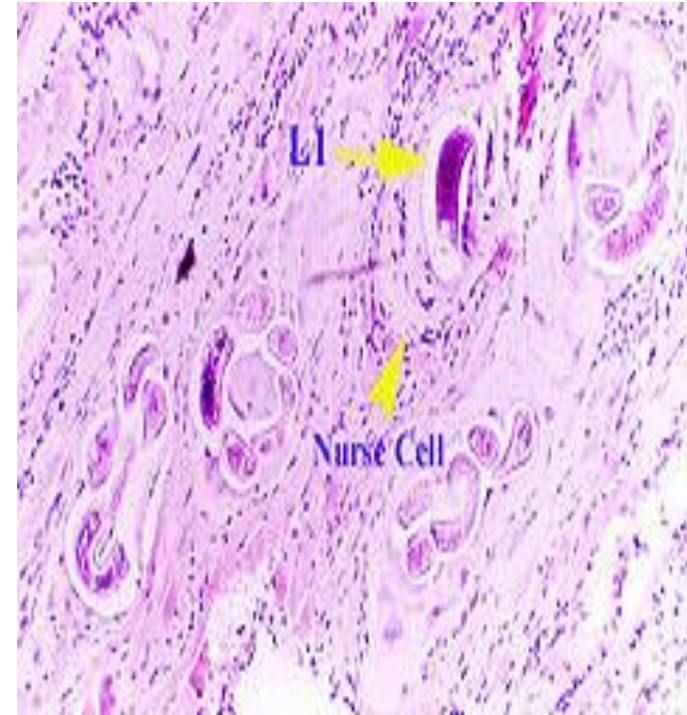
- Adult worms are rarely found in natural infections
- Are up to 3mm in length

Microscopic

- Esophagus is at least one third of the total length
- Tail has small cloacal flaps but no spicule
- In females uterus contain developing larvae

Diagnosis

- Not relevant in live animals
- At meat inspection larvae can be seen with naked eye as grayish white spots
- 1 gm of pig muscles are squeezed between microscopic slides & directly examined under a microscope for presence of larvae the apparatus called trichinoscope or a compressorium.



2.1.8. Superfamily Filarioidea

A. Genus *Parafilaria*

- **Species** - *P. bovicola*-----→cattle & buffalo
- _ *P. multipapillosa*----→horse
- **Intermediate hosts** _ Muscid flies in *P. bovicola*
 _ haematobia in *P. multipapillosa*
- **Site** - subcutaneous & intermuscular connective tissue

Morphology

Microscopic

- Anteriorly has numerous papillae & circular ridges
- Vulva of females is situated anteriorly near mouth
- Lay embryonated eggs that hatch to release microfilariae (L₁)

Pathogenesis

- Gravid females puncture skin while laying eggs & cause haemorrhagic exudates or bleeding points that streaks & mats surrounding hair & attract flies.

Clinical signs

- Active bleeding lesions are pathognomonic

Diagnosis

- Examination of exudates from bleeding points to demonstrate eggs or L₁

Treatment

- Ivermectin or Nitroxynil

4.1. Anthelmintics (Parasiticide)

- A drug that is effective in killing parasites of one form or another
- The drug will not kill all parasites, merely that it will kill at least one species of parasite.
- Most antiparasitic drugs are usually effective in killing several related species of parasites (have broad spectrum activity)
- Control programs for parasites in all our domestic animals rely heavily on the use of **antiparasitic drugs**

- **The properties of an ideal anthelmintic**
 - efficient against all parasitic stages of a particular species
 - non-toxic to the host
 - rapidly cleared and excreted by the host
 - easily administered
 - cost should be reasonable

Use

- **Therapeutics**- to treat existing infections or clinical out breaks
- **Prophylactics**- treatment is based on knowledge of the epidemiology

Therapeutic usage:

1. If the drug is not active to all stages, it must be at least effective against the pathogenic stage of the parasite
2. There should be a marked clinical improvement and rapid recovery after treatment

Prophylactic usage

1. The cost should be justifiable economically
2. The cost-benefit analysis should be comparable to other methods such as pasture management or vaccination (dictyocaulosis)

3. Should not interfere with the development of an acquired immunity
4. Prolonged prophylactic use of one drug should be avoided as this may encourage the development of **anthelmintic** resistance

The major groups of anthelmintics

Chemical group	Drugs	Susceptible parasite
Piperazines	Piperazine salts, Diethylcarbamizine	nematode
Imidazoles	Tetramisole, Levamisole, morantel , pyrantel	nematode
Benzimidazole	Thiabendazole, Menbendzole, Fenbendazole, albendazole, oxibendazole e.t.c	nematode
Avermectins/ milbemycins	Ivermectin, Abamectin, Milbemycin	nematode
Organophosphates	Dichlorvos, haloxon, trichlofon	nematode

salicylanilides	nitroscanate, closantel	nematode
Salicylanilides	nitroxynil, rafoxanide, oxyclozanide, closantel etc	trematode
Benzimidazoles/pro- benzimidazoles	triclabendazole, albendazole	trematode
Others	praziquantel, arecoline	Cestode