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Definitions and terms

Veterinary parasitology : It is branch of science which deals with the study of parasites and parasitism including classification , habit and habitat morphological characters of parasites life cycle .

Parasite: the word parasite is divided from the Greek it is made up of two words "para " means beside and "sitos "means food .Parasites are – those organisms which live on or within some other living organisms , which is known as host. For example Ascaris (parasite)in man (host).

Host: It is an organism or animal , which harbours a parasite. It is always larger than parasite

Difinitive host (Final host) : the organism in which the parasite develops to its sexully mature, adult stage.

Intermediate host : An organism in which development , growth , encystment or asexual multiplication of the parasite occurs . usually the stage infective to the final host develops.

Transport host The stage of parasite does not undergo any development and remain in the same stage . ex.earthworms act as transport egg of *Ascaridia galli*

Normal host : In which naturally acquired infection results in rapid and complete development.

Paratenic host : An organism which serves to transfer a larval stage or stages from one host to another .

Prepatent period : The time taken for development from infection until the .production of eggs or larvae by the mature adult worms

Patent period : the Time during which adult worms continue producing and shedding .egg or lavae

.**Moult** : Periodic cuticle replacement in the life cycle of nematodes

Temporary parasites : those which visit the host time to time but do not remain with . it all the time

Example : Fleas, Flies

Permanent parasites : Are those parasites which remain with the host all the time and do not leave it at anytime.Example Lice , helminthes

Ectoparasite or external parasites Are those parasites which are found on the surface of the body of the host .Example lice

Endoparasites or internal parasites: those parasites which are found inside the body .of the host in the gut, body cavity or tissue

Example , helminthes , protozoa

Erratic or aberrant parasites : Endoparasites found in other organs than the norm of . habitat in the host example Fasciola when found in lungs , Kidney

Incidental parasites or accidental parasites : Those endoparasites in a host which . these usually not live. Not found normally

.Example *Ascaris lumbricoides* in sheep

Hyperparasites : those parasites which may themselves be parasitized on the other parasites example . *Histomomnas meleagridis* in the egg and larvae of *Heterakis . gallinae*

Pseudoparasites :those parasites objects , which are mistaken for parasites.ex.Small .white thread for *Trichostrongylus*

. **Hemoparasites** :parasites which are live in blood .ex.Trypanosoma

Protozoan parasites : the members which are included in sub kingdom protozoa . ex. .Trypanosome

. **Metazoan parasites** : multicellular parasites like Tape worms , round worms

Helminthic or worm parasites :parasites included under the phyla platy helminthes , .nemathelminthes ,Acanthceephala

Carriers :Are those hosts Which have a light infection with some parasites but are not harmed by it .usually due to immunity resulting from previous exposure Ex. Adult birds .rarely from coccidiosis

Vector: The vector is an arthropod , mollusk , which transmits the parasite from one . vertebrate host to another .ex, Mosquitoes for Malaria

Parasitism It is an intimate association between two specifically distinct organisms in which one smaller lives on or within the other larger in order to obtain sustenance ..*Toxocara canis* in dog

Symbiosis:The relationship of two individuals in which both are benefied

Ex. Intestinal protozoa provide the habitat and the capable of hydrolyzing the cellulose . for their own

Predator:It is an individual who temporarily attacks and destroy animals or plants in order to obtain food .ex. cats for rats

Infestation :The term mostly used to the parasitism by the external harassing parasites Ex. Lice on the animals and man

Autoinfection : when a infected person is his own direct source of the re-exposure it is . called autoinfection

Ex. *Enterobius vermicularis*

Retroinfection : when the larvae of parasites hatch from eggs around the anus and crowl back through anus into the intestine and mature called retroinfection .Ex.*Enterobius vermicularis*

(Extent of Damage by parasites to the Host (pathogenicity

The parasites produce different types of effects on their hosts ,some are harmless , some are mildly pathogenic or moderately harmful while a few others are highly . pathogenic and fetal

The degree of harmful effects among various parasites on host depends **on number of factors**

Number of parasites gaining enterance habits , invasive power , virulence, biopotentiality , release of toxin substances, degree of adoptation between host and .parasites , localization in the body of their hosts

Extrinsic factors outside of their hosts like general health condition , level of nutrition , age , sex, breed of the host

:The damage and harm caused by parasites are as follows

use of host 's food-١

Especially endoparasitic forms have depletion pf host's nutrients by parasites may have serious consequences .Some of the parasites competed with the host's food either by ingesting the intestinal contents (gastrointestinal nematodes, trematodes)or .(by absorbing them through the body wall(tape worms

use of other substances than food -٢

(Some parasites endo and ectoparasites suck blood (mosquitoes, hook worms

Mechanical interference or obstruction -٣

The parasites may cause mechanical obstruction or interference with host tissue functions for ex. *Ascaris lumbricoides* when present large numbers can easily block .the lumen of intestine of the host

Destruction of host tissues -٤

By A- some parasites injure the host's tissues when they enter the host while others . infect tissue damage after entering into the body

Ex. Cercaria of shistosoma causes extensive damage to cells and during the . penetration of the host skin

b- They may destroy the tissues of the host by using the various organelles like head spins teeth , sucker,ex, tapeworm

c-They destroy the host cells by growing in them ex. Coccidian

d- They may exert extreme pressure on different tissues ex.hydatid cyst

e- they may perforate the intestine of the hosts ex. Ascarids

They may cause distortion displacement ,rupture.or hemorrhages in organs, tissues.ex. coccidia

Transmission of Parasites to the host(Mode of infection

The infection is transmitted from one vertibrate host to another host by one of the :following routs

1- by ingestion infected food and water:

a-ingestion of grass , hay, bedding straw and other contaminated with emberyonated .(eggs (*Ascaris*) , cysts (*Entamoeba histolytica*

b-By drinking contaminated of water containing infective stages by host

.(*Entamoeba histolytica*)

c-by ingesting uncooked or less cooked meat or fishes

infected with larvae stages of parasites ex. Man and carnivorous are infected eating uncooked meat or less cooked of cattle infected with Cysticercus bovis , Sarcocysts of .Sarcocystis .Hydated cysts

٢- By skin penetration or through percutaneous infection .ex. Cercaria of *Schistosoma*, .sp. enter into the host by penetration of the skin of the host

3- by eating Insects harboring infective stage of parasites ex.

Infected snails harboring infective stage larvae of Protostrongylus sp. are swallowed .(by definitive host.(Ruminants

4- by insect transmission : Blood sucking arthropods act as vector for many protozoa.

5- Transmission by coitus:

There are some protozoan parasites Which are transmitted from one host to another .by coitus ex, *Trichomonas foetus* in cattle

6- Transplacental transmission through uterus mother . ex. *Toxoplasma gondi*

٧- Lactogenic transmission or transmammary transmission the transmission through the milk . ex. *Toxocara canis* .

٨- Autoinfection : the infected host itself transmit the infection . ex. *Cryptosporidium sp*

٩- By direct contact.*Ex. lice infestation and mange mites are transmitted from one* to another host by direct contact.

-By inhalation10 - -

. Ex. Eggs *of Enterobius vermicularis* may be transmitted by inhalation

Life cycles

Different parasites have different types of life cycles . Each parasite may have either direct or indirect life cycle

-Life cycle pattern in Digenetic Trematodes 1-

All digenetic trematodes have indirect life cycles . if Trematodes has only one intermediate host in its life cycle then it is essentially a species of snail (molusc) but when two or more intermediate hosts are required to complete the life cycle, then second intermediate host, dragon fly (larva), some crustaceans, snail, frog ex. .*Schistosoma sp*. the final host are sheep , man and the intermediate host is snail

.-life cycle pattern in Tape worms 2

All tapeworms have indirect life cycle except *Hymenolepis nana* in which both direct and indirect life cycle occur

-life cycle pattern in round worms. 3 - -

Nematodes mainly have six successive stages namely five larval stages(L1-L5)and the adult , with growth and moult of cuticle between two stages. In the life cycle of .parasites of man and animals , the infective stage is usually is the third stage larva

Nematodes have direct life cycle such as *Trichostrongylus* ,While the same larval . development takes place inside the intermediate host

Or an insect vector when life cycle is indirect. Ex . Filarid worm transmitted by blood .sucking insects act as vector or intermediate hosts

Immunity to parasites :

Parasitic infections typically stimulate a number of immunological defence mechanisms **both antibody and cell – mediated** and the responses that are most effective depend upon the particular parasites and the stage of infection .In general , **the cell- mediated responses** are **more effective against intracellular protozoa** , while **antibody is more effective against extracellular parasites in blood and tissue fluids.** Whithin single infection different immune responses may act against different .developmental stages of the parasites . ex. Malaria

While the antibody against extracellular forms blocks their capacity to invade new cells, cell-mediated immunity prevents the development of the liver stage within the . hepatocytes

Phylum NEMATHELMINTHES

The phylum Nemathelminthes has six classes only one of these, the nematoda, contains worms of parasitic significance. The nematodes are commonly called roundworms, from their appearance in cross section.

STRUCTURE AND FUNCTION

Most nematodes have a cylindrical form, tapering at either end, and the body is covered by a colorless, translucent, the cuticle. **The digestive system** is tubular. The mouth of many nematodes is a simple opening which may be Surrounded by two or three lips, and leads directly into the oesophagus. In others, such as the strongyloids, it is large, and opens into a buccal capsule, which may contain teeth; such parasites, when feeding, draw a plug of mucosa into the buccal capsule

Those with very small buccal capsules, like the trichostrongyloids, or simple oral openings, like the ascaridoids, generally feed on mucosal fluid, products of host dioestion and cell debris, while others, such as the oxyuroids, appear to scavenge on the contents of the lower gut. Worms living in the bloodstream or tissue spaces, such as the filarioids, feed exclusively on body fluids.

The oesophagus is usually muscular and pumps food into the intestine. It is of variable form

1-filariform, simple and slightly thickened posteriorly, as in the bursate nematodes:

2-bulb-shaped, with a large posterior swelling, as in the ascaridoids ,

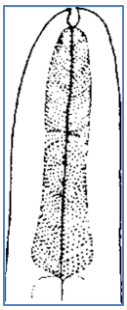
3- double bulb-shaped, as in the oxyuroids.

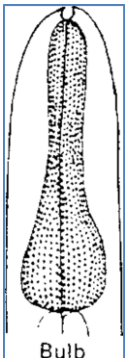
4-filarioids and spiruroids have a **muscular-glandular** oesophagus which is muscular anteriorly, the posterior part being glandular

5-, the trichuroid oesophagus has a **capillary** form, passing through a single column of cells. the whole being known as a stichosome.

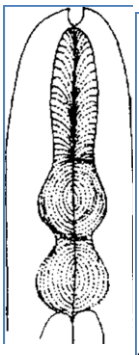
6-A rhabditiform oesophagus, with slight anterior and posterior swellings, is present in the preparasitic larvae of many nematodes, and in adult free-living nematodes.

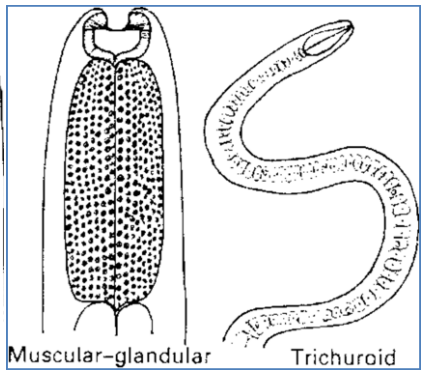
filariform





doubl bulb





The intestine is a tube whose lumen is enclosed by a single layer of cells

In female worms the intestine terminates in an anus while in males there is a cloaca

The so-called `excretory system' is very primitive, consisting of a canal within each lateral cord joining at the excretory pore in the oesophageal regionThe reproductive systems consist of filamentous

tubes.
The female organs comprise ovary, oviduct and uterus, which may be paired, ending in a common short vagina which opens at the vulva. At the junction of uterus and vagina in some species there is a short muscular organ, the ovejector, which assists in egg-laying. A vulva] flap may also be present. The male organs consist of a single continuous testis and a vas deferens terminating in an ejaculatory duct into the cloaca. Accessory male organs are sometimes important in identification, especially of the trichostrongyloids, the two most important being the spicules and gubernaculum

The spicules are Bursa - chitinous organs, usually paired, which are imported during copulation.

The cuticle may be modified to form various structures, the more important (Fig. 7) of which are:
Leaf crowns consisting of rows of papillae occurring as fringes round the rim of the buccal capsule (external leaf crowns) or just inside the rim (internal leaf crowns). They are especially prominent in certain nematodes of horses.
Cervical papillae occur anteriorly in the oesophageal region, and caudal papillae posteriorly at the tail. They are spine-like or finger-like processes, and are usually diametrically placed. Their function may be sensory or supportive.

Cervical and caudal alae are flattened wing-like expansions of the cuticle in the oesophageal and tail regions.

Cephalic and cervical vesicles are inflations of the cuticle around the mouth opening and in the oesophageal region.

The copulatory bursa, which embraces the female during copulation, is important in the identification of certain male nematodes and is derived from much expanded caudal alae, which are supported by elongated caudal papillae called bursal rays. It consists of two lateral lobes and a single small dorsal lobe.(fig1)

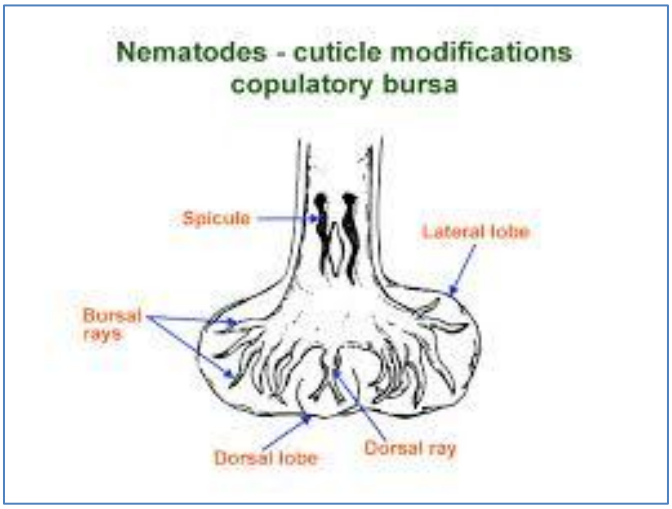


Fig 1

Supcrfamily ASCARIDOIDEA

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

With a few exceptions the genera have the following characters in common.

They are large, white opaque worms which inhabit the small intestine. There is no buccal capsule, the mouth consisting simply of a small opening surrounded by three lips. The common mode of infection is by ingestion of the thick-shelled egg containing the L.,. However, the cycle may involve transport and paratenic hosts.

Genus Ascaris

Host: Pig.

Site:

Small intestine.

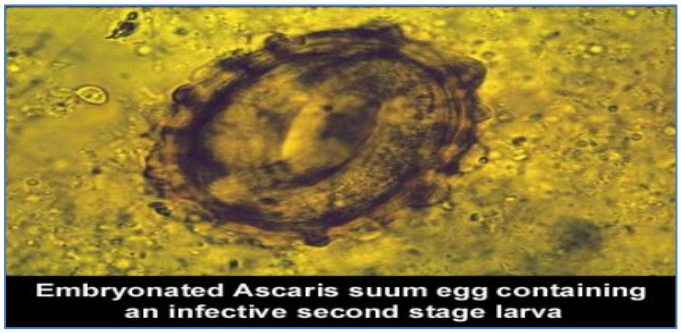
Species: *Ascaris suum.*

Distribution: Worldwide.

IDENTIFICATION

A. suum is by far the largest nematode of the pig: the females are up to 40.Ocm long,.

The egg is ovoid and yellowish, with a thick shell, the outer layer of which is irregularly mamillated fig2



LIFE CYCLE

The life cycle is direct. Though the single preparasitic moult occurs at about three weeks after the egg is passed,. The egg is very resistant to temperature extremes, and is viable for more than four years.

After infection, the egg hatches in the small intestine and the L; travels to the liver, where the first parasitic moult takes place. The L, then passes in the bloodstream to the lungs and thence to the small intestine via the trachea. In the intestine the final two parasitic moults occur.

If the eggs are ingested by an earthworm or dung beetle they will hatch, and the 1, travel to the tissues of these paratenic hosts, where they can remain, fully infective for pigs, for a long period.

The prepatent period is between 6 and 8 weeks, and each female worm is capable of producing more than 200000 eggs per day.

PATHOGENESIS

The migrating larval stages in large numbers may cause a transient pneumonia, but it is now recognized that many cases of so-called *'Ascaris* pneumonia' may be attributable to other infections, or to piglet anaemia.

In the liver, the migrating L, and L_3 can cause `milk spot' which appears as cloudy whitish spots of up to 1.0 cm in diameter,

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

.

CLINICAL SIGNS

1-The main effect of the adult worms is to cause production loss in terms of diminished weight gain.

2-Clinically evident pneumonia

DIAGNOSIS

Diagnosis is based on clinical signs, and in infections with the adult worm, on the presence in faeces of the yellow-brown ovoid eggs, with thick mamillated shells. Being dense, the eggs float more readily in saturated solutions of zinc sulphate or magnesium sulphate than in the saturated sodium chloride solution which is used in most faecal examination techniques.

TREATMENT
the anthelmintics in current use in pigs, and the majority of these, such as the benzimidazoles, levamisole and ivermectin may be more convenient.

[Ascariosis in man: The type species, *Ascaris lurnbricoides,* occurs in man, and at one time it was not differentiated from *A. scuem,* so that the pig was thought to present a zoonotic risk for man. With morphological distinction now possible, *A. lumbricoides is* accepted as specific for man, and is irrelevant to veterinary medicine.]

Toxocara
Though the members of this genus are in many respects typical ascaridoids, their biology is sufficiently varied for it to be necessary to consider each species separately.

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

Host: Dog.
Site:
Small intestine.

Distribution: Worldwide.

IDENTIFICATION
Toxocara canis is a large white worm up to IO.Ocm in length, and in the dog can be confused only with *Toxascaris leonina.* Differentiation of these two species is difficult, as the only useful character, visible with a hand lens, is the presence of a small finger-like process on the tail of the male *TT canis.*
The egg is dark brown and subglobular, with a thick, pitted shell (Fig.3).

LIFE CYCLE

This species has the most complex life cycle in the superfamily, with **four possible modes** of infection.The basic form is typically ascaridoid,

1-the **egg containing the L_2** being infective, at optimal temperatures, four weeks after being passed. After **ingestion,** and hatching in the small intestine, the L_2 travel by the bloodstream via the liver to the lungs, where the second moult occurs, the L3 returning via the trachea to the intestine where the final two moults take place.

This form of infection occurs regularly only in dogs of up to three months old.

2-**prenatal infection** occurs, or intrauterine infection of the developing fetus . larvae becoming mobilized at about three weeks prior to parturition and migrating to the lungs of the foetus where they moult to L_3 just before birth. In the newborn pup the cycle is completed when the larvae travel to the intestine via the trachea, and the final moults occur.

3-**Transmammary transmission The suckling pup**s may also be infected by ingestion of **L2 in the milk** during the first three weeks of lactation. There is no migration in the pup following infection by this route.

4-Paratenic hosts such as rodents or birds may ingest the infective eggs, and the L2 travel to their tissues where they remain until eaten by a dog when subsequent development is apparently confined to the gastrointestinal tract.

PATHOGENESIS

In moderate infections, the larval migratory phase is accomplished without any apparent damage to the tissues, and the adult worms provoke little reaction in the intestine.

In heavy infections the pulmonary phase of larval migration is associated with **pneumonia**, which is sometimes accompanied by **pulmonary oedema;** the adult worms cause a mucoid enteritis, there may be partial or complete occlusion of the gut and, in rare cases, perforation with peritonitis or in some instances blockage of the bile duct.

CLINICAL SIGNS

1-In mild to moderate infections, there are no clinical signs during the pulmonary phase of larval migration.

2- The adults in the intestine may cause pot-belly, and occasional diarrhoea. Entire worms are sometimes vomited or passed in the faeces.

3-The signs in heavy infections during larval migration result from pulmonary damage and include **coughing, increased respiratory rate**, and a frothy nasal discharge.

4-pups which have been heavily infected transplacentally may die within a few days of birth.

5-Nervous convulsions have been attributed by some clinicians to toxocarosis,

EPIDEMIOLOGY

1-The highest prevalences have been recorded in dogs of less than six months of age, The widespread distribution and high intensity of infection with *T. canis* depend essentially on factors.

, the females are extremely fecund, one worm being able to contribute about 700 eggs to each gramme of faeces per day,

2-Second, the eggs are highly resistant to climatic extremes, and can survive for years on the ground.

3-Aconstant reservoir of infection in the somatic tissues of the hitch. and larvae in these sites are insusceptible to most anthelmintics.

DIAGNOSIS

1- diagnosis is possible during the pulmonary phase of heavy infections when the larvae are migrating, and is based on the simultaneous appearance of **pneumonic signs** in a litter, often within two weeks of birth.

2-The eggs in faeces, subglobu(ar and brown with thick pitted shells, are species-diagnostic. The egg production of the worms is so high that there is no need to use flotation methods, and they are readily found in simple faecal smears to which a drop of water has been added.

TREATMENT AND CONTROL

The adult worms are easily removed by anthelmintic treatment. The most popular drug used has been piperazine, benzunidazoles, fenbendazole and mebendazole..

recommended regime for control of toxocarosis in young dogs is as follows.

1-All pups should be dosed at 2 weeks of age, and again 2-3 weeks later, to eliminate prenatally acquired infection. It is also recommended that the bitch should be treated at the same time as the pups.

2-A further dose should be given to the pups at two months old, to eliminate any infection acquired from the milk of the dam or from any increase in faecal egg output by the dam in the weeks following whelping.

Newly purchased pups should be dosed twice at an interval of 14 days.

3-that adult dogs should be treated every 3-6 months throughout their lives.

VISCERAL LARVA MIGRANS

Though this term was originally applied to invasion of tissues of an animal by parasites whose white worm, natural hosts were other animals, it has now, in common usage, come to represent this type of invasion in humans alone and, in particular, by the larvae of *Toxocara canis*. Its complementary term is cutaneous larva migrans, for infections by 'foreign' larvae which are limited to the skin.

The condition occurs most commonly in children who have had close contact with household pets, or who have frequented areas such as public parks where there is contamination of the ground by dog faeces. Surveys of such areas in many countries have almost invariably shown the presence of viable eggs of T. *canis* in around 10% of soil samples.

In many cases larval invasion is limited to the liver, and may give rise to **hepatomegaly a**nd e**osinophilia**, hut on some occasions a larva escapes into the general circulation and arrives in another organ, the most frequently noted being the eye. Here, **a granuloma forms around the larva on the retina, often resembling a retinoblastoma**, and there have been cases of precipitate removal of the eye in children following misdiagnosis. Only in rare cases does the granuloma involve the optic disc, with total loss of vision, and most reports are of partial impairment of vision, with endophthalmitis or granulomatous retinitis. Such cases are currently treated using laser therapy. In a few cases of epilepsy, *T. canis* infection has been identified serologically .

Toxascaris

This genus occurs in domestic carnivores, and though common, is of less significance than *Toxocara* because its parasitic phase is non-migratory.

Hosts:

Dog and cat

Toxocara cati

Host**:** Cat.

Site**:**

Small intestine.

Distribution**:** Worldwide.

IDENTIFICATION

Typically of the superfamily, *Toxocara cati* is a large the visceral often occurring as a mixed infection with the other ascaridoid of carnivores, *Toxascaris leonina.* Differentiation is readily made between the two on gross examination, when the cervical alae of *T. cati* are seen to have an arrow-head form, with the posterior margins almost at a right angle to the body, whereas those of *Toxascaris* taper gradually into the body. The male, like that of T. *canis,* has a small finger-like process at the tip of the tail.

The egg, subglobular, with a thick, pitted shell and almost colourless, is characteristic in cat faeces.

LIFE CYCLE
Like *T. canis*, the life cycle of *T. cati* is migratory

Toxocara (syn. Neoascaris) vittulorm

Hosts:
Cattle and buffalo.

Site:
Small intestine.

Distribution:
Mainly in tropical and warm regions.

IDENTIFICATION
T. vitulorum is the largest intestinal parasite of cattle, the females being up to 30.Ocm long. It is a thick worm, pinkish when fresh, and the cuticle is rather transparent so that the internal organs can be seen.
The egg is subglobular, with a thick pitted shell, and is almost colourless.
LIFE CYCLE
The life cycle of this species resembles that of *T. cati*, in that the most important source of infection is the milk of the dam in which larvae are present for up to 30 days after parturition.

There is no tissue migration in the calf following infection and the prepatent period is 3-4 weeks. The ingestion of larvated eggs by calves over six months old seldom results in patency, the larvae migrating to the tissues where they are stored; in female animals, resumption of development in late pregnancy allows further transmammary transmission.

PATHOGENESIS AND CLINICAL SIGNS
The main effects of this infection appear to be caused by the adult worms in the intestines of calves up to six months old. Heavy infections are associated with poor thriving and intermittent diarrhoea, and in buffalo calves particularly, fatalities may occur.

DIAGNOSIS

The subglobular eggs, with thick, pitted shells, are characteristic in bovine faeces.

TREATMENT
The adult worms are susceptible to a wide range of anthelmintics including piperazine, levamisole and the benzimidazoles. All these drugs are also effective against developing stages in the intestine.

CONTROL
The prevalence of infection can be dramatically reduced by treatment of calves at three and six weeks of age preventing developing worms reaching patency.

Genus :Parascaris

Infection with Parascaris throughout the world and is a major cause of unthriftiness in young foals.

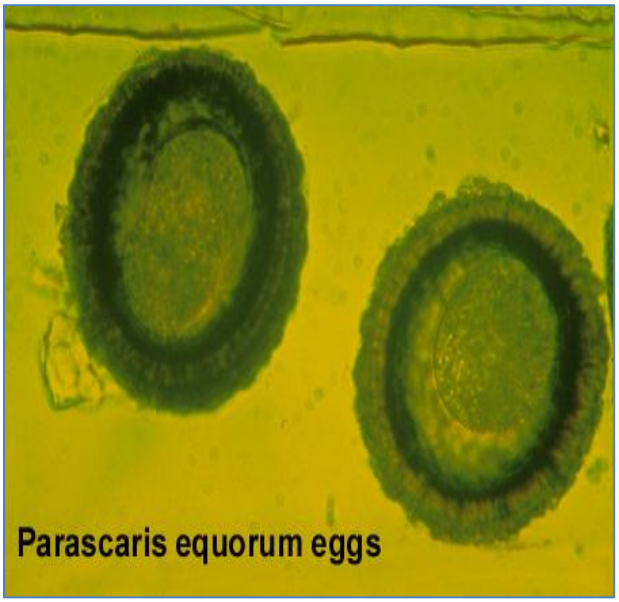
Hosts:
Horses and donkeys.
Site:
Small intestine.

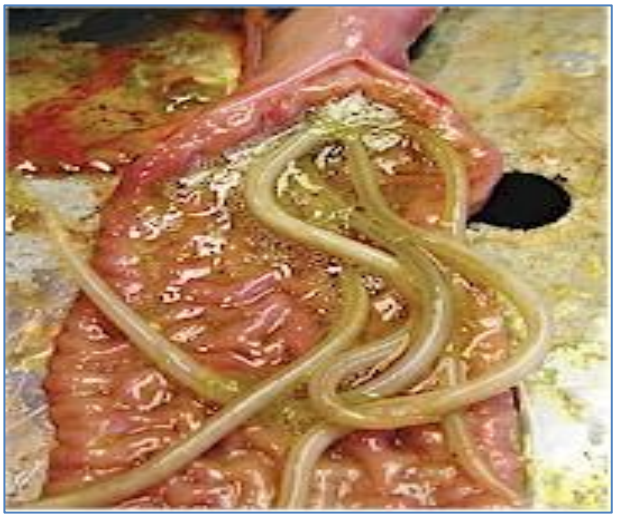
Species:
Parascaris equorum.
Distribution: Worldwide.

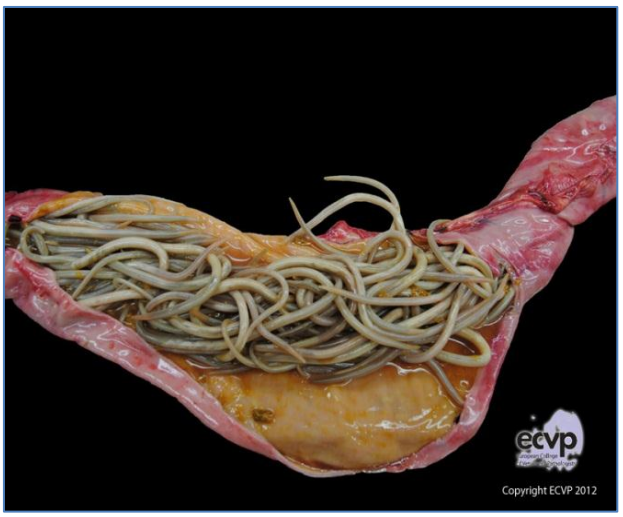
IDENTIFICATION

Gross:
This very large whitish nematode, up to 40 cm in length, cannot be confused with any other intestinal parasite of equines.

Microscopic:
The adult parasites have a simple mouth opening surrounded by three large lips and in the male the tail has small caudal alae.The egg of P. *equorum* is almost spherical, brownish and thick-shelled with an outer pitted coat.

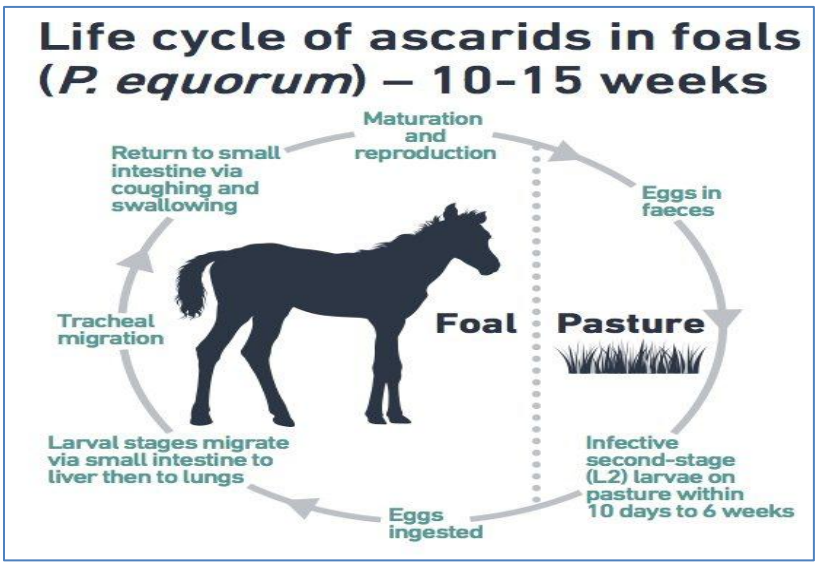






LIFE CYCLE

The life cycle is **direct**. Eggs produced by the adult female worms are passed in the faeces and can reach the infective stage containing the I_2 in as little as 10-14 days, although development may be delayed at low temperatures. After ingestion and hatching the larvae penetrate the intestinal wall and within 48 hours have reached the liver. By two weeks they have arrived in the lungs where they migrate up the bronchi and trachea, are swallowed and return to the small intestine., but it would appear that the moult from $L_2$to L3 occurs between the intestinal mucosa and the liver and the two subsequent moults in the small intestine The minimum prepatent period of *P. equorum* is 10 weeks.



PATHOGENESIS

1-Gross changes are provoked in the liver and lungs by migrating P. equorum larvae. **In the liver**, larvae cause **focal haemorrhages** and **eosinophilic tracts** which resolve leaving **whitish areas of fibrosis**.

2-Larval migration **in the lungs** also leads to **haemorrhage** and infiltration by eosinophils which are later replaced by accumulations of lymphocytes, while sub-pleural greyish-green lymphocytic nodules develop around dead or dying larvae;
3- Although the presence of worms in the small intestine is not associated with any specific lesions, occasionally, heavy infections have been reported as a cause of impaction and perforation leading to peritonitis.

CLINICAL SIGNS

1-the major signs are frequent coughing accompanied in some cases by a greyish nasal discharge although the foals remain bright and alert. 2- unthriftiness in young animals with poor growth rates, dull coats.
3-fever, nervous disturbances and colic have been attributed to field cases of parascariosis,

EPIDEMIOLOGY

There are two important factors.
First, the high fecundity of the adult female parasite, some infected foals passing millions of eggs in the faeces each day.
Secondly, the extreme resistance of the egg in the environment ensures its persistence for several years.

DIAGNOSIS

This depends on 1-clinical signs
2-presence of spherical thick-shelled eggs on faecal examination.

CONTROL

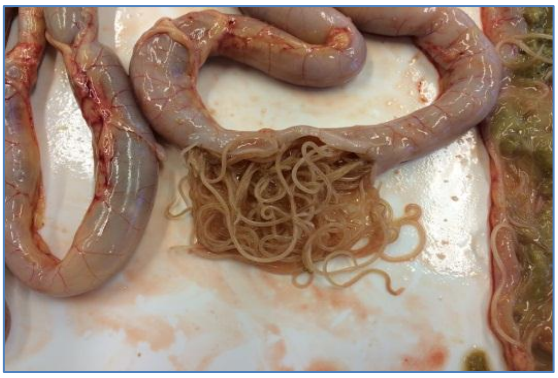
Anthelmintic prophylaxis for the horse strongyles will effectively control P. equonun infection.

Genus Ascaridia

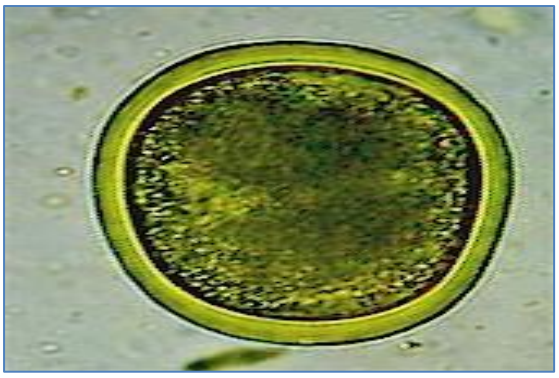
This is a non-migratory
Hosts:
Domestic and wild birds.
Site:
Small intestine.
Species:
Ascaridia galli.
Distribution: Worldwide.

IDENTIFICATION

The worms are stout and densely white, the females measuring up to 12.0cm in length. Ascaridia is by far the largest nematode of poultry.The egg is distinctly oval, with a smooth shell, and cannot easily be distinguished from that of the other common poultry



ascaridoid, Heterakis. Adult worm

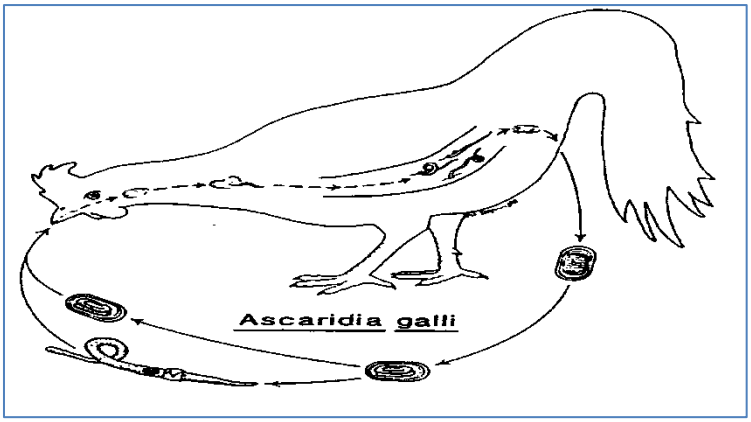


egg of *Ascaridia galli*

LIFE CYCLE

Is direct,this is a non-migratory

. The egg is sometimes ingested by earthworms, which may act as transport hosts. The prepatent period ranges from 5-6 weeks in chicks to eight weeks or more in adult birds



PATHOGENESIS AND CLINICAL SIGNS

Ascaridia is not a highly pathogenic worm, and any effects are seen in young birds, adults appearing rela tively unaffected. The main effect is seen during the prepatent phase, when the larvae are in the mucosa. There they cause an enteritis which is usually catarrhal, but in very heavy infections may be haemorrhagic. In moderate infections the adult worms are tolerated without clinical signs, but when considerable numbers are present the large size of theseworms may cause intestinal occlusion and death

EPIDEMIOLOGY

Adult birds are symptomless carriers, and the reservoir of infection is on the ground, either as free eggs or in earthworm transport hosts.

DIAGNOSIS

In infections with adult worms, the eggs will be found in faeces, but since it is difficult to distinguish these from Heterakis eggs, confirmation must be made by post-mortem examination of a casualty when the large white worms will be found. In the prepatent period, larvae will be found in the intestinal contents and in scrapings of the mucosa.

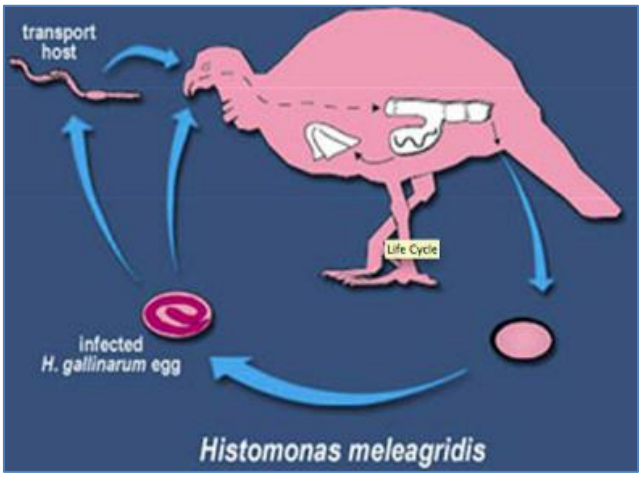
TREATMENT AND CONTROL
Treatment with piperazine salts levamisole or a benzimidazole, such as flubendazole, can be administered either in the drinking water or the feed. Capsules containing fenbendazole for use in pigeons are also highly effective.

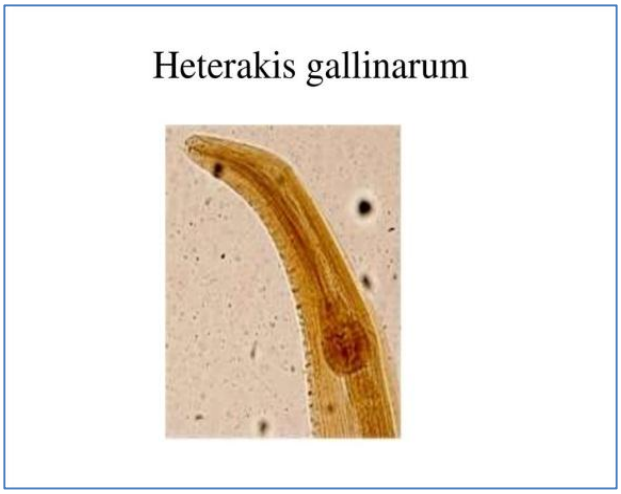
Genus :Heterakis

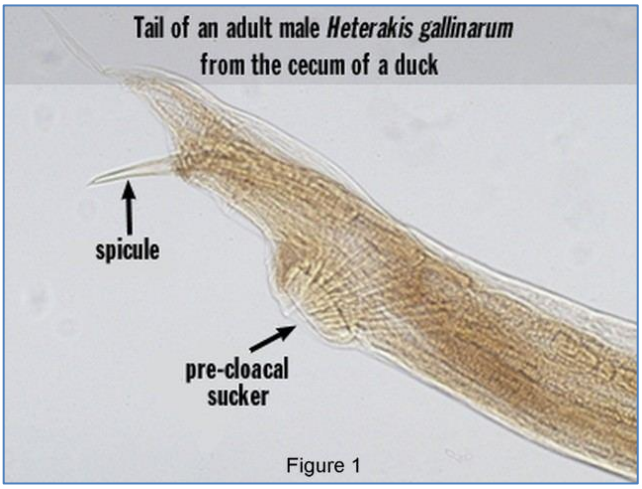
This genus is exceptional in its small size and in its location in the large intestine, in contrast to Ascaridia which is large and inhabits the small intestine

Hosts:
Domestic and wild birds
Site: Caeca.
Species:
Heterakis gallinarum. Another species, *H. isolonche*,
occurs in game birds

Distribution: WorldwideIDENTIFICATION
Whitish worms up to 1.5cm long, with elongated pointed tails. Gross examination readily indicates the genus, but for specific identification microscopic examination is necessary to demonstrate the spicules, which are **unequal in length** in ***H. gallinarum***, but of equal length in *H. isolonche*. Microscopically,
also, generic identity may be confirmed by the presence of a large precloacal sucker in the male, and prominent caudal alae supported by large caudal papillae. The egg is ovoid and smooth-shelled, and is difficult to distinguish from that of Ascaridia.







LIFE CYCLE

The egg is infective on the ground in about two weeks at optimal temperatures. Earthworms may be transport hosts, the eggs simply passing through the gut, In *H. gallinarum* all three parasitic moults appear to occur in the caecal lumen, but in *H. isolonche* infection the hatched larvae enter the caecal mucosa, and develop to maturity in nodules. Each nodule has an opening into the gut through which the eggs reach the lumen. The prepatent period of the genus is about four weeks

PATHOGENESIS AND CLINICAL SIGNS

H. gallinarum is the commonest nematode parasite of poultry, and is usually regarded as being non-pathogenic. Its chief pathogenic importance is as a vector of the protozoan, ***Histomonas meleagridis*****,** the causal agent **of blackhead' in turkeys**. The organism can be transmitted from fowl to fowl in the egg of Heterakis and in earthworms containing hatched larvae of the worm.

H. isolonche of game birds is in itself pathogenic, causing a severe inflammation of the caeca with nodules projecting from both peritoneal and mucosal surfaces. These cause diarrhoea with progressive emaciation and there may be high mortality in heavily infected flocks.

EPIDEMIOLOGY

H. gallinarum is widespread in most poultry flocks and is of little pathogenic significance in itself, but is of great importance in the epidemiology of Histomonas. In contrast H. isolonche in game birds occurs as a clinical entity.

DIAGNOSIS

H. gallinarurn infection is usually only diagnosed accidentally, by the finding of eggs in faeces or the presence of worms at necropsy. H. isolonche infection is diagnosed at necropsy by the finding of caecal nodules containing adult worms, and if necessary confirmed microscopically by examination of the spicules.

TREATMENT

Like Asccrridia, Neterrrkis is susceptible to piperazine, levamisole and a number of benzimidazoles

CONTROL

Control of *H. gallinarum* is only necessary when histomonosis is a problem in turkeys. It is largely based on hygiene, and in backyard flocks two main points are:

١- the segregation of turkeys from other domestic poultry, and the removal and disposal of litter from poultry houses. Where the problem is serious and continuous

٢- Administer either piperazine or levamisole intermittently in the feed or water.

Family : Subuliridae

Genus : *Subulura brumpti*

Species of this genus, found in birds, are small worms located in the caecae. They are similar to *Heterakis*, although the tail is not as pointed. The males measure around 8–10 mm and the females up to 14–18 mm in length.

Life cycle: Eggs passed in faeces are ingested by the intermediate host where they develop to the infective L3 stage after about 2 weeks. Following ingestion by the final host the larvae migrate to the lumen of the caeca. The prepatent period is 6–8 weeks.

***Subulura* species**

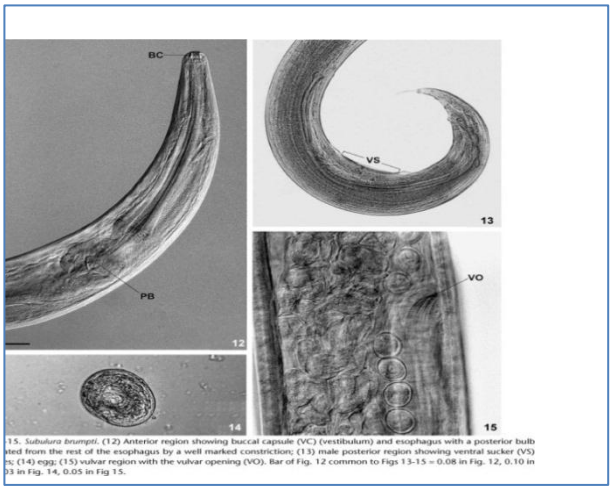
Intermediate hosts: Beetles, cockroaches

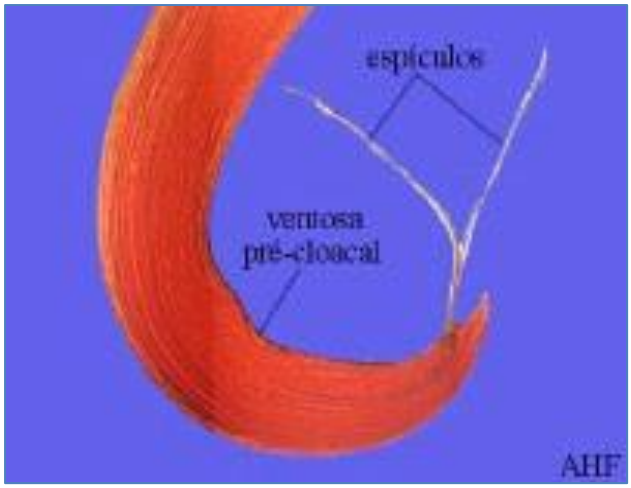
Site: Caeca

Species :Subulura suctoria
(syn. *Subulura brumpti*,
Subulura differens,)
Hosts :Chicken, turkey, guinea fowl, quail, grouse, pheasant, duck

Synonyms: *Subulura brumpti*, *Subulura differens*, *Allodapa suctoria*

Description: The buccal capsule is small and possesses three teeth at its base. The oesophagus is dilated posteriorly, followed by a bulb. The tail of the male has large lateral alae and is curved ventrad. Two long thin curved spicules are present. A slit-like pre-cloacal sucker is present, surrounded by radiating muscle fibres. In the female, the vulva is situated just anterior to the middle of the body





PATHOGENESIS AND CLINICAL SIGNS

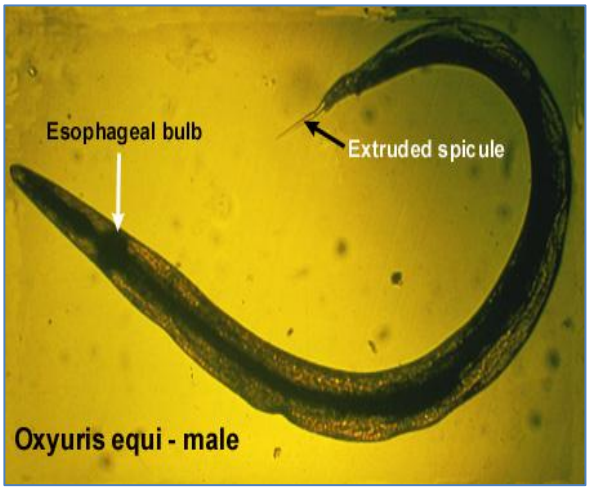
the affected birds are dull and depressed with ruffled feathers. In the postmortem examination of dead birds, they observed severe inflammation of caecal mucosa and caecum was filled with haemorrhagic contents, in which thousands of tiny worms were found moving actively

Superfamily OXYUROIDEA

Adult oxyuroids of animals inhabit the large intestine and are commonly called ‘pinworms’ because of the long pointed tail of the female parasite. The ventrolateral papillae are often absent and where present are very much reduced. The number of spicules can vary in the males from zero, one depending on the species. They have a double bulb oesophagus, the posterior bulb being well developed. The life cycle is direct.

Genus :Oxyuris

Infection with the horse pinworm, Oxyuris equi, is extremely common and, although of limited pathogenic significance in the intestine, the female parasites may cause an intense anal pruritis during the process of egg laying.



egg

Hosts:
Horses and donkeys.
Site:
Caecum, colon and rectum.
Species: Oxyuris equi.
Distribution: Worldwide.

IDENTIFICATION

Gross:

The mature females are large white worms with pointed tails which may reach 10.0cm in length ,whereas the mature males are generally less than 1.0cm long. O. *equi* L4, from 5 to 10 mm in length, have tapering tails and are often attached orally to the intestinal mucosa.

Microscopic:

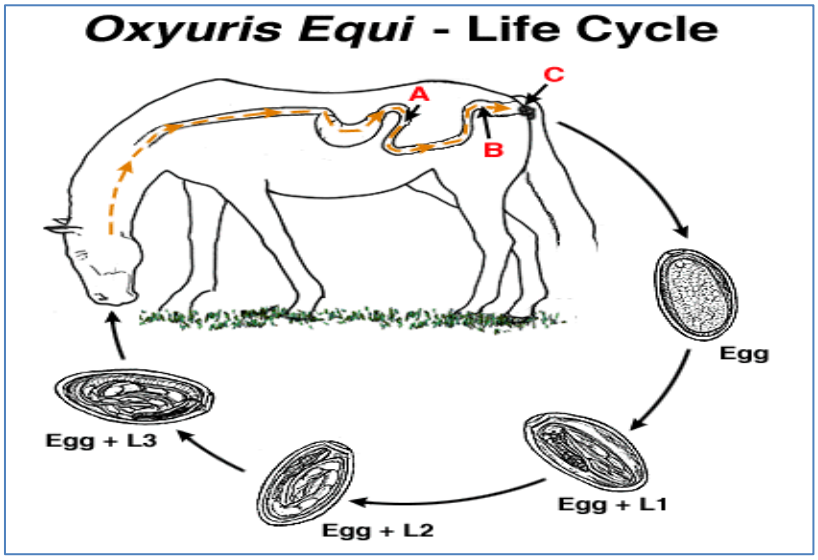
There is a double oesophageal bulb and the tinv males have caudal alae and **a single spicule**. In the female the vulva is situated anteriorly.

O. equi eggs are ovoid, yellow and slightly flattened on one side with a mucoid plug at one end

LIFE CYCLE

The adult worms are found in the lumen of the colon. After fertilization the gravid female migrates to the anus, extrudes her anterior end and lays her eggs in clumps, seen grossly as yellowish white gelatinous streaks on the perineal skin. Development

is rapid and within 4-5 days the egg contains the infective L_3. Infection is by ingestion of the eggs and the larvae are released in the small intestine, move into the large intestine and migrate into the mucosal crypts of the caecum and colon where development to L4 takes place within 10 days. The L4 then emerge and feed on the mucosa before maturing to adult stages which feed on intestinal contents. The prepatent period of O. *equi* is 5 months.



PATHOGENESIS

Most of the pathogenic effects of O. *equi* in the intestine are due to the feeding habits of the L4 which result in small erosions of the mucosa and, in heavy infections, these may be widespread and accompanied by an inflammatory response. Normally, a more important effect is the perineal irritation caused by the adult females during egg laying.

CLINICAL SIGNS

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

DIAGNOSIS

This is based on signs of anal pruritis and the finding of greyish-yellow egg masses on the perineal skin. The large white long-tailed female worms are often seen in the faeces, having been dislodged while laying their eggs. *O. 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 AND CONTROL

1-O. equi is susceptible to many broad spectrum anthelmintics and should be controlled by routine chemotherapy for the more important horse parasites
2-tail should be frequently cleaned using a disposable cloth, in addition to anthelmintic treatment. A high standard of stable hygiene should be observed.

[Pinworms in man: *Ercterobius vermicularis*, the human pinworm, is common throughout the world and although it occurs in all age groups it is most prevalent in children. It is important to note that *E. vermicurlaris*, like other oxyuroids, is highly host-specific .

Superfamily RHABDITOIDEA
This is a primitive group of nematodes which are mostly free-living, or parasitic. Although a few normally free-living genera such as *Micronema* and *Rhabditis* occasionally cause problems in animals, the only important genus from the veterinary point of view is *Strongyloides.*

Genus :Strongyloides

Hosts:
Most animals.
Site:
Small intestine; also caecum in poultry.

Species:
Strongyloides westeri Horses and donkeys
S. papillosus Ruminants
S. stercoralis Dogs and cats: man
S. avium Poultry.

Distribution: Worldwide.

IDENTIFICATION

Gross:

Slender, hair-like worms generally less than 1.0 cm long.

Microscopic:

Only females are parasitic. The long oesophagus may occupy up to one third of the body length and the uterus is intertwined with the intestine giving the appearance of twisted thread. Unlike other intestinal parasites of similar size the tail has a blunt point

Strongyloides eggs are oval, thin-shelled and small, being half the size of typical strongyle eggs . In herbivores it is the larvated egg which is passed out in the faeces but in other animals it is the hatched L1.

LIFE CYCLE

Strongyloides is unique among the nematodes of veterinary importance, being capable of both parasitic and free-living reproductive cycles.

The parasitic phase is composed entirely of female worms in the small intestine and these produce larvated eggs by parthenogenesis, ie. development from an unfertilized egg. After hatching, larvae may develop through four larval stages into free-living adult male and female worms and this can be followed by a succession of free-living generations.

However under certain conditions, possibly related to temperature and moisture, the L3 can **become parasitic, infecting the** host by skin penetration or ingestion and migrating via the venous system, the lungs and trachea to develop into **adult female** worms in the small intestine.

Foals, lambs and piglets may acquire infection immediately after birth from the mobilization of arrested larvae in the tissues of the ventral abdominal wall of the dam which are subsequently excreted in the milk.

The prepatent period is from 8 to 14 days.

PATHOGENESIS

Skin penetration by infective larvae may cause an erythematous reaction which in sheep can allow the entry of the causative organisms of foot-rot. Passage of larvae through the lungs has been shown

multiple small haemorrhages visible over most of the lung surfaces.

Mature parasites are found in the duodenum and proximal jejunum and if present in large numbers may cause inflammation with oedema and erosion of the epithelium. This results in a catarrhal enteritis with impairment of digestion and absorption.

CLINICAL SIGNS

The common clinical signs usually seen only in very young animals are diarrhoea, anorexia, dullness, loss of weight or reduced growth rate.

EPIDEMIOLOGY

Strongyloides infective larvae are not ensheathed and are susceptible to extreme climatic conditions. However warmth and moisture favour development and allow the accumulation of large numbers of infective stages..

A second major source of infection for the very young animal is the reservoir of larvae in the tissues of their dams and this may lead to clinical strongyloidosis in foals in the first few weeks of life.

DIAGNOSIS
1-The clinical signs in very young animals,
2-finding of large numbers of the characteristic eggs or larvae in the faeces
.
TREATMENT AND CONTROL
The benzimidazoles and the avermectins/milbemycins may be used for the treatment of clinical.

Genus :Rhabditis
Several members of this free-living genus of nematodes may become casual parasites, invading the skin and causing an intense pruritus. Cases have been reported in dogs

. The verv small worms 1.0-2.8mm in length with a rhabditiform oesophagus may be recovered from skin scrapings.
Treatment is symptomatic and the condition can be prevented by housing animals on clean, dry bedding
.
**otitis externa associated with *Rhabditis* infection has been reported in cattle.

Strongyles

The strongyles are parasitic in the large intestine and the important genera are Strongylus, Triodontophorus, Trichonema (cyathostomes),
Chabertia and Oesophagostonuum.
Hookworms are parasites of the small intestine and the three genera of veterinary importance are
Anacylostoma, Uncinaria and Bunostornum.
STRONGYLES OF HORSES
Genus :Strongylus
Members of this genus live in the large intestine of horses and donkeys and. with Triodontophorus, are commonly known as the **large strongyles.**
Hosts: Horses and donkeys.

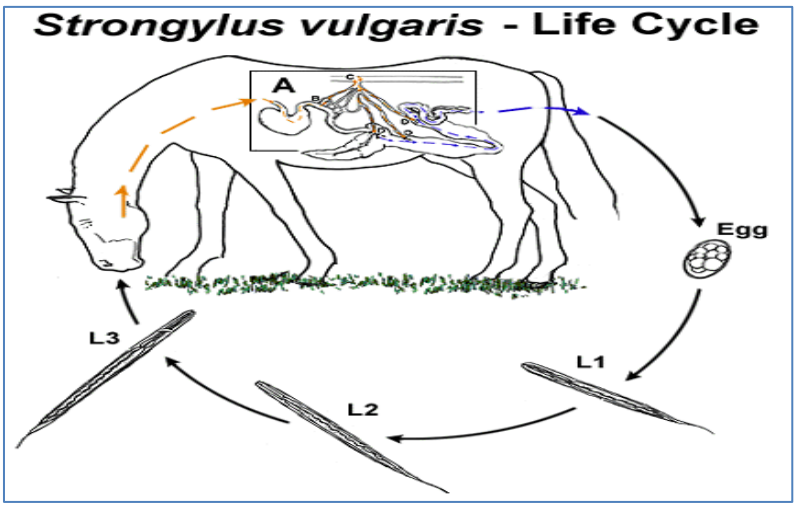
Site: Caecum and colon.
Species: *Strongyltcs vulgaris S. edentatus S. equinus.*
Distribution: Worldwide.
IDENTIFICATION

Gross: 

Robust dark-red worms which are easily seen against the intestinal mucosa. The well developed buccal capsule of the adult parasite is prominent as is the bursa of the male.
Microscopic:

Strongylus vulgaris

Species differentiation is based **on size and the presence and shape of the teeth in the base of the buccal capsule** .



the adult parasites live in the caecum and colon . eggs are passed in the faeces and development from egg to the L3 . Infection is by ingestion of the L3 . in S. vulgaris the L3 penetrated the intestinal mucosa and moult to L4 in the submucosa then enter small arteries and migrate on the endothelium to their predilection site in the cranial mesenteric artery and its main branches.the L4----- L5and return to the intestinal wallvia the arterial lumina. Nodules are formed around the larvae mainly in the wall of the caecum and colon . subsequent ruptue of these nodules release the young adult parasites into the lumen of the intestine

Pathogenesis
Larvae
Many horses having lesions in the arterial system of the intestine caused by this species. Lesions are most common in the cranial mesenteric artery and its main branches, and consist of thrombus formation provoked by larval damage to the endothelium together with a marked inflammation and thickening of the arterial wall True **aneurysms** with dilatation and thinning of the arterial wall, although uncommon, may be found especially in animals which have experienced repeated infection.
In *S. edentatus* infection there are gross changes in the liver associated with early larval migration,
, the haemorrhages and fluid-filled nodules which accompany later larval development in subperitoneal tissues
Adults
The pathogenesis of infection with adult Strongylus spp. is associated with damage to the large intestinal mucosa due to the feeding habits of the worms
.Ulcers which result from these bites eventually heal, leaving small circular scars.
Clinical signs
but the gross damage and subsequent loss of blood and tissue fluids is certainly partly responsible for the unthriftiness and anaemia associated with intestinal helminthosis in the horse

DIAGNOSIS
This is based on the grazing history and clinical signs of loss of condition and anaemia. eggs on faecal examination

TREATMENT

There are a number of broad spectrum antheImintics including the benzimidazoles, pyrantel and the avermectins/milbemccins, which are effective in removing lumen-dwelling adult and larval strongyles .

Trichojtentct/Cyathostontes This genus embraces over 40 species, popularly known as T**richonemes, Cyathostomes or small strongyles**. These parasites are found in the large intestine of horses and their effects on the host range from poor performance to clinical signs of severe enteritis.

Hosts:
Horses and donkeys
Site: Caecum and colon.

Distribution: Worldwide.

IDENTIFICATION
Gross: Small to medium sized (< 1.5 cm in length) bursate nematodes ranging in colour from white to dark red, the majority being visible on close inspection of the large intestinal mucosa or contents.

Microscopic:
The well developed buccal capsule is cylindrical and species differentiation is based on characteristics of the buccal capsule, and the internal and external leaf crowns.

LIFE CYCLE
Hatching of eggs and development to L3 is complete within two weeks during the summer in temperate areas, after which the larvae migrate from the faeces on to the surrounding herbage. After ingestion, the L3 exsheath and invade the wall of the large intestine where they develop to L4 and moulting to become young adult worms.

PATHOGENESIS
Parasitic larval development of most species takes place provokes an inflammatory response together with marked goblet cell hypertrophy. with a massive infiltration of the gut mucosa with eosinophils. haernorrhagic enteritis, with thickening and oedema of the mucosa,

HOOKWORMS OF DOGS AND CATS
The family Ancylostomidae, whose members are commonly called hookworms **because of the characteristic hook posture of their anterior ends,** are responsible for widespread morbidity and mortality in animals primarily due to their blood-sucking activities in the intestine.
Genus :Ancylostoma
Hosts: Dog, cat and fox.

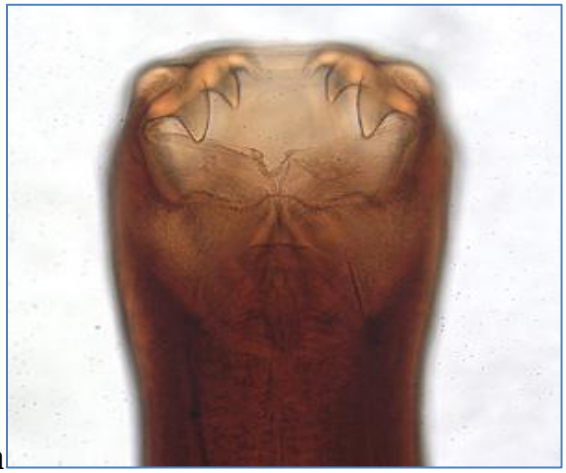
Site:Small intestine.

Species:
Ancylostorna caninum dog and fox
A. tubaeforme cat
A. braziliense dog and cat.

IDENTIFICATION
Gross:
They are readily recognized on the basis of size (1.- 2.0 cm), being much smaller than the common ascarid nematodes which are also found in the small intestine. and by their characteristic `hook' posture.

Microscopic:
The buccal capsule is large with marginal teeth there being three pairs in A. caninum and A. tubaeforme and two pairs in A. braziliense

Genus : Ancylostoma caninum



LIFE CYCLE

The life cycle is direct and given optimal conditions the eggs may hatch and develop to L3, in as little as five days **Infection is by skin penetration or by ingestion**, both methods being equally successful. In percutaneous infection, larvae migrate via the blood stream to the lungs where they moult to L4, in the bronchi and trachea, and are then swallowed and pass to the small intestine where the final moult occurs. If infection is by ingestion the larvae may either penetrate the buccal mucosa and undergo the pulmonary migration described above or pass direct to the intestine and develop to patency. Whichever route is taken the prepatent period is 14-21 days. The worms are prolific egg layers and an infected dog may pass millions of eggs daily for several weeks.
An important feature of A. caraintun infection is that, in susceptible bitches, a proportion of the L3, which reach the lungs migrate to the skeletal muscles where they remain dormant until the bitch is pregnant. They are then reactivated and, still as L3, are passed in the milk of the bitch for a period of about three weeks after whelping.

PATHOGENESIS
An acute or chronic haemorrhagic anaemia. The disease is most commonly seen in dogs under one year old and young pups, infected by the transmammary route, are particularly susceptible due to their low iron reserves.
CLINICAL SIGNS
In acute infections, there is **anaemia and lassitude** and occasionally **respiratory embarrassment**. In suckled pups the anaemia is often severe and is accompanied by **diarrhoea** which may contain blood and mucus. **Respiratory signs may be due to larval damage in the lungs**.
In more chronic infections, the animal is usually underweight, the coat is poor, and there is loss of appetite and perhaps pica.
DIAGNOSIS
This depends on the clinical signs and history supplemented by haematological and faecal examination. High faecal worm egg counts are valuable confirmation of diagnosis.,
TREATMENT
Affected dogs should be treated with an anthelmintic, such as mebendazole, fenbendazole and nitroscanate.

Genus : Chabertia

Hosts:
Sheep, goats and occasionally cattle.
Site: Colon.
Species: *Chabertia ovina*.
IDENTIFICATION
Gross:
The adults are 1.5-2.0 cm in length and are the largest nematodes found in the colon of ruminants. They are white with a markedly truncated and enlarged anterior end due to the presence of the very large buccal capsule.

Microscopic:
The buccal capsule, which is bell shaped, has a double row of small papillae around the rim. There are no teeth

LIFE CYCLE
This is direct and the preparasitic phase is similar to that of the trichostrongyles of ruminants. In the parasitic phase the L3, enter the mucosa of the small intestine and occasionally that of the caecum and colon; after a week they moult, the L4 emerge on to the mucosal surface and migrate to congregate in the caecum where development to the L_5 is completed about 25 days after infection. The young adults then travel to the colon.

PATHOGENESIS
The major pathogenic effect is caused by the L_S and by mature adults which feed by ingesting large plugs of mucosa resulting in local haemorrhage and loss of protein through the damaged mucosa.

CLINICAL SIGNS
In severe infections, diarrhoea, which may contain blood and in which worms may be found.
DIAGNOSIS
the faecal egg count may be very low. However, during the diarrhoeic phase, the worms may be expelled

Genus :Oesophagostomum

Hosts: Ruminants, pigs.
Site:
Caecum and colon.
Species:
Oesophagostorntun columbiantun sheep and goat
IDENTIFICATION
Gross:
Stout white worm 1.0-2.0 cm long. Readily differentiated by its tapered head from Chabertia.

Microscope:
The buccal capsule is small. In many species it is surrounded by leaf crowns.

LIFE CYCLE
The preparasitic phase is typically strongyloid and infection is by ingestion of L3 although there is limited evidence that skin penetration is possible. The L3 enter the mucosa of any part of the small or large intestine then moult to L4,These L4, then emerge on to the mucosal surface, migrate to the colon, and develop to the adult stage.

PATHOGENESIS: the nodules reaching 2.Ocm in diameter and containing greenish eosinophilic pus and an L4,. When the L4, emerge there may be ulceration of the mucosa.
CLINICAL SIGNS
In acute infections of ruminants, severe dark green diarrhoea is the main clinical signs