Parascaris
(helminth: nematode)

Overview

Nematodes are triploblastic pseudocoelomate unsegmented worms that undergo protostomial embryonic cleavage and grow by cuticular moulting (ecdysis). Two groups identified by the presence/absence of sensory phasmids have partly been ratified by molecular studies recognising three subclasses: Enoplia and Dorylaimia (both without phasmids) and Chromadoria (most with phasmids). Many phasmidian parasites of vertebrates are grouped in the chromadorian order Rhabditida; including tylenchinids, rhabditinids and spirurinids. The latter contains the infraorder Ascaridomorpha which includes ascaridoid nematodes (roundworms) characterised by their large size, three prominent anterior lips and the absence of a bursa. They occur in the small intestines of many animals (including humans) and most have simple direct life-cycles involving faecal-oral transmission. Female worms produce numerous eggs which are excreted with host faeces and undergo embryonation to contain infective larvae. When ingested, larvae hatch from the eggs and develop into adult worms in the gut. The larvae of ascaridoid species undergo hepato-pulmonary migration before forming adults, whereas those of heterakoid species do not. Two major ascaridoid families are recognised: ascarids in terrestrial mammals; and anisakids in marine mammals. Infections by *Parascaris* spp. cause enteritis and illthrift in horses worldwide.

Classification:

Domain:	Eukaryota (membrane-bound nucleus)
Supergroup:	Amorphea (unikonts with single flagellum, or nonflagellated amoebae)
Kingdom:	Metazoa (multicellular eukaryotes, heterotrophs, notably animals)
Group:	Protostomia (triploblastic, spiral cleavage)
Subgroup:	Ecdysozoa (cuticle moulted = ecdysis)
Phylum:	Nematoda (unsegmented, pseudocoelomate roundworms, tubular digestive tract, dioecious)
Class:	Chromadorea (spiral amphids, three oesophageal glands, usually annulated bodies, free-living and parasitic)
Order:	Rhabditida (Secernentea, Phasmidea) (secretors, with phasmids, bipartite oesophagus, single testis)
Suborder:	Spirurina (mostly parasitic in vertebrate hosts)
Infraorder:	Ascaridomorpha (large roundworms, mouth surrounded by three large lips, numerous caudal papillae)
Superfamily:	Ascaridoidea (ascarids, eggs thick-shelled, direct cycle but larvae undertake hepato-pulmonary migration)
Family:	Ascarididae (large pale roundworms, in terrestrial mammals)
Genus:	*Parascaris* (parasitic in small intestines of horses)
Species:	several species cause illthrift in foals

Parasite biodiversity and host range: Most Metazoa are multicellular triploblastic animals with differentiated tissues, many being bilaterally symmetrical with a body cavity. Most invertebrate animals are protostomes as their embryonic development involves spiral determinate cleavage. Those that moult their external cuticles during their life-cycles (process known as ecdysis) are grouped together in the unique clade Ecdysozoa, including the nematodes (roundworms), onychophorans (velvet worms), tardigrades (water bears) and arthropods (myriapods, chelicerates, crustaceans and hexapods, all with jointed limbs). Nematodes (roundworms) are unsegmented tubular worms with a fluid-filled body cavity (pseudocoelom) that acts as a hydrostatic skeleton. They have longitudinal muscles and typically exhibit a sideways thrashing motion. They have well developed digestive tracts with various partitions: the foregut comprising the mouth (often with lips and papillae), buccal capsule (sometimes with ridges, rods, plates, spears, stylets or teeth) and oesophagus (glandular, muscular or both); the midgut (nonmuscular absorptive section); and hindgut (rectum) emptying through a subterminal anus (cloaca in males). Most nematodes are dioecious and form separate sexes. Male worms have a single testis (sometimes 2), an elongate vas deferens often equipped with a seminal vesicle and ejaculatory duct (glandular and/or muscular), 1-2 copulatory spicules (sometimes with an accessory gubernaculum), and bursate species with elaborate posterior claspers. Female worms are usually didelphic (some monodelphic or polydelphic) with 2 ovaries, 2 oviducts usually with spermatheca, 2 uteri opening into a common vagina and a vulva often equipped with a muscular ovejector. Female worms are oviparous or viviparous and produce numerous eggs or larvae, respectively. Larval stages undergo several moults (L1-L4) before maturing into adult worms. Some nematodes have direct life-cycles where eggs or larvae infect definitive hosts (per os or per cutaneous), but many have indirect cycles where larvae first develop in invertebrate intermediate hosts before infecting definitive hosts (by ingestion, injection or deposition). Many nematode species are free-living in terrestrial and aquatic habitats, while some species from diverse groups have become plant or animal parasites. Two nematode groups identified by the presence/absence of sensory phasmids have partly been ratified by molecular studies recognising three subclasses: Enoplia and Dorylaimia (both without phasmids) and Chromadoria (most with phasmids). Most Enoplia are free-living marine organisms but some are found in freshwater, and on land as plant parasites. The Dorylaimia comprise numerous freshwater and terrestrial species, including major groups of plant and animal parasites. The Chromadoria is represented by many marine groups as well as a

terrestrial group of plant and animal parasites. The taxonomic ranks of many nematode assemblages vary considerably depending on which classification system has been followed. Molecular phylogenetic studies, however, have supported the separate classification of most groups, particularly at the level of superfamily. Collectively, species from at least 16 superfamilies are considered to pose serious threats to human and animal health as infectious diseases.

CLASSIFICATION* OF SUPERFAMILIES OF PARASITIC NEMATODES
Class: Enoplea (Aphasmidea, Adenophorea) (gland-bearers, cylindrical oesophagus, no phasmids, setae, two testes)
Subclass: Dorylaimia (five or more oesophageal glands, buccal stylet (odontostyle), free-living or parasitic)[clade I(2)]
Order: Trichinellida (Trichocephalida, Trichurida) (single spicule, stichosome oesophagus, L1 with buccal stylet)
Superfamily: Trichinelloidea (oesophagus with short anterior muscular and long posterior glandular portions)
Class: Chromadorea (spiral amphids, 3 oesophageal glands, usually annulated bodies, free-living and parasitic)
Order: Rhabditida (Secernentea, Phasmidea) (secretors, phasmids present, amphids anterior, bulbous oesophagus)
Suborder: Rhabditina (free-living or parasitic in invertebrates/lower vertebrates)[clade V(9)]
Infraorder: Rhabditomorpha ('rod-shaped' buccal cavity)
Superfamily: Rhabditoidea (open tube stoma, excretory system with lateral canals)
Superfamily: Strongyloidea (bursate males, prominent buccal capsules, parasites of mammals, birds, reptiles)
Suborder: Spirurina (animal parasites, many use invertebrate intermediate hosts (IH))[clade III(8)]
Incertae sedis Superfamily: Dracunculoidea (elongate parasites of vertebrate tissues, freshwater crustacean IH)
Infraorder: Ascaridomorpha (large roundworms, three large lips, numerous caudal papillae)
Superfamily: Ascaridoidea (ascarids, eggs thick-shelled, larvae may undertake hepato-pulmonary migration)
Superfamily: Heterakoidea (preanal sucker anterior to cloaca in males, direct cycle, infection by egg ingestion)
Infraorder: Gnathostomatomorpha ('jaw-mouthed' due to unique bulbous armed heads)
Superfamily: Gnathostomatoidea (first IH copepod, often use paratenic hosts)
Infraorder: Oxyuridomorpha (pinworms, pointed tails, oesophagus with terminal bulb, males with single spicule)
Superfamily: Oxyuroidea (common in mammals, birds, reptiles, amphibians)
Infraorder: Spiruromorpha (enigmatic clade linked by molecular characters, indirect cycles with IHs)
Superfamily: Acuarioidea (small parasites mostly of birds, with cephalic cordons, ptilina or serrated shields)
Superfamily: Camallanoidea (conspicuous phasmids, L1 with dorsal tooth, ovoviviparous, L1-L3 in copepod)
Superfamily: Filarioidea (tissue-dwelling filarial parasites, lack lips, infect tissues/vessels, arthropod IH)
Superfamily: Habronematoidea (unique head structures with small pseudolabia and median lips)
Superfamily: Physalopteroidea (stomach worms in mammals, insect IH)
Superfamily: Spiruroidea (pseudolabia, bipartite oesophagus, infect birds (crop/gizzard), arthropod IHs
Superfamily: Thelazioidea (eye-worms of birds and mammals, transmitted by insects)
Suborder: Tylenchina (fungal, plant and animal parasites)[clade IV(10,11,12)]
Infraorder: Panagrolaimomorpha (free-living or parasitic (insects, reptiles, amphibians, mammals))
Superfamily: Strongyloidoidea (dauer stages, lip region without processes, striated cuticle)

*Contemporary genotypic classification schemes recognize strong monophyletic clades at the level of superfamily and infraorder, while previous phenotypic classification schemes had ranked many as separate orders.

The infraorder Ascaridomorpha is characterised by large roundworms with poorly developed buccal cavity with 3 large lips sometimes separated by interlabia, an undivided oesophagus, numerous caudal papillae, nonbursate males often with pre-anal suckers, and females with complex ovejectors. Five superfamilies (conventionally assigned to the order Ascaridida) are recognised as parasites in vertebrates: Ascaridoidea (cylindrical oesophagus often terminated by swelling without bulb, coelomyarian, eggs thick-shelled); Heterakoidea (oesophagus cylindrical or with claviform corpus, short isthmus and valved bulb, coelomyarian, pre-anal sucker, eggs thick-shelled); Seuratoidea (lips absent, oesophagus short, platymyarian, eggs with delicate shells or hatch *in utero*); Cosmocercoidea (oesophagus with cylindrical corpus, elongate isthmus and valved bulb, platymyarian, eggs with delicate shells or hatch *in utero*); and Subuluroidea (well-developed buccal capsule without lips, coelomyarian, pre-anal sucker, eggs thick-shelled). Adult worms of the superfamily Ascaridoidea inhabit the gastrointestinal tract of vertebrate hosts and generally consume food ingested by the host. They may have simple monoxenous life cycles involving faecal-oral transmission, or more complicated heteroxenous life-cycles involving larval development in vertebrate intermediate hosts and sometimes larval transport in invertebrate paratenic hosts. Female worms produce unembryonated eggs which are passed in host faeces into the external environment where they embryonate to first-stage larvae (L1) which grow and moult to infective L2 or L3. Aquatic species produce thin-shelled eggs which hatch in water releasing sheathed L2 that are taken up by suitable hosts, while terrestrial species produce thick-shelled eggs which hatch releasing L3 when ingested by suitable hosts. Ascaridoid larval stages then undertake unique journeys: most involving pulmonary or somatic migration in their definitive hosts before maturing in the gut, sometimes including vertical transmission (transplacental and/or transmammary); many migrating into the tissues of intermediate hosts, sometimes involving larval migrans or encapsulation in 'unsuitable' hosts; and a few undergoing precocious development in invertebrate hosts.

Five ascaridoid families are recognised: Ascarididae (lips often with toothed ridge, oesophagus with or without ventriculus, parasites of mammals, birds, reptiles, amphibians, fishes); Anisakidae (lips with tongue-like prolongations with cuticular thickenings, oesophagus with ventriculus with suture-like depressions, parasites of mammals, birds, reptiles and fishes); Crossophoridae (lips semicircular with toothed combs, fimbriated collar, long oesophagus without ventriculus, parasites of hyracoids); Heterocheilidae (lips with tongue-like prolongations with cuticular thickenings, cylindrical oesophagus without ventriculus, parasites of sirenians); and Acanthocheilidae (lips small with teeth or toothed ridges, oesophagus with ventriculus, parasites of elasmobranchs). The family Ascarididae contains 4 subfamilies: Ascaridinae (oesophagus simple, gubernaculum absent, lips hexagonal with anterior region offset from posterior, parasites of terrestrial mammals); Toxocarinae (oesophagus with globular ventriculus without appendices, gubernaculum absent, parasites of terrestrial or marine mammals or birds); Angusticaecinae (oesophagus simple, gubernaculum absent, lips quadrangular and not divided into anterior and posterior regions, parasites of reptiles and amphibians); and Multicaecinae (oesophagus with globular ventriculus usually with appendices, gubernaculum present, parasites of crocodilians or rarely fish). Ascaridid genera of medical and veterinary significance are compared in the following table.

Genus	No. spp.	Definitive Hosts	Location	Adult worms	Eggs	Transmission
Ascaridinae						
Parascaris	3	equids	small intestines	10-50 cm long, 3 large lips each with transverse groove, cuticle striated, males abursate, simple spicules, larvae undergo hepato-pulmonary migration	90-120 µm, spherical, thick-shelled	faecal-oral
Ascaris (roundworm)	2 (+ 150 *nomen dubium*)	primates, suids	small intestines	15-50 cm long, 3 small lips, striated cuticle, males with curved tail, simple spicules, females opisthodelphic, larvae undergo hepato-pulmonary migration	50-87 x 35-60 µm, ovoid, thick-shelled	faecal-oral
Toxascaris	2	carnivores	small intestines	2-15 cm long, 3 lips, long thin cervical alae, oesophageal ventriculus absent, males lack terminal digitiform appendage, larvae do not undergo hepato-pulmonary migration	70-85 x 60-75 µm, ovoid, thick-shelled	faecal-oral (sometimes ingestion of infected PH)
Toxocarinae						
Toxocara	26	carnivores, ruminants, rodents	small intestines	3-30 cm long, 3 lips, long thin cervical alae, oesophageal ventriculus present, males with terminal digitiform appendage, larvae undergo somatic migration, hypobiosis	64-91 µm, spherical, thick-shelled	faecal-oral, ingestion of PH, transplacental, transmammary

The subfamily Ascaridinae contains 5 genera of large roundworms with well-defined lips and a simple oesophagus: *Ascaris* (syn. *Fusaria*, *Lombricoides*, *Stomachida*), *Baylisascaris, Lagochilascaris*, *Parascaris* and *Toxascaris*. The genus *Parascaris* contains worms whose lips are subdivided into anterior and posterior regions by internal transverse grooves forming deep incisions on the lateral margins, small interlabia are present but cervical alae and lateral flanges are absent, and the cuticle has longitudinal bracket-like bars not reaching the surface. Two species, *P. equorum* and *P. univalens,* have been described from equids around the world, mostly in tropical and temperate regions. These species are very similar in morphology, although small differences in spicule structure have been noted (those of *P. equorum* are rounded while those of *P. equorum* are truncated/sharp). Classical and contemporary karyotyping studies differentiated both species, with *P. equorum* having 4 chromosomes with terminal and intercalary heterochromatin, and *P. univalens* having only 2 chromosomes with terminal heterochromatin. More recent molecular characterization studies on nuclear and mitochondrial gene sequences found both species to be closely-related, so close that some authors think they may even be the same species. Irrespective of designation, infections in equids are of clinical significance as a cause of colic, ill-thrift and even death, particularly in foals and yearlings. It is also possible for *P. equorum* eggs ingested by rodents and humans to hatch but the released larvae do not complete their development in these aberrant hosts.

***Parascaris* species**	**Hosts**	**Location [Clinical signs**	**Distribution**
P. equorum (syn. *Ascaris megalocephala bivalens*)	Perissodactyla: equid (horse, Przewalski's horse, donkey, mule, onager, plains zebra, Burchell's zebra, Hartmann's mountain zebra) [plus miscellaneous reports: Artiodactyla: cervid (white-tailed deer); Rodentia: murid (rat); Primates: hominid (human); Diptera: muscid (house fly *Musca domestica*)]	small intestines [illthrift]	worldwide
P. felis	Carnivora: felid (lion)	5 worms passed in faeces	India
P. univalens (syn. *Ascaris megalocephala univalens*)	Perissodactyla: equid (horse, donkey)	small intestines [illthrift]	Europe, Americas

Parasite morphology: *Parascaris* spp. form 3 different types of developmental stages: eggs; larvae; and adult worms. Freshly laid eggs are spherical to subspherical in shape measuring from 90-120 µm in diameter, brown in colour and surrounded by a thick albuminous (sticky) roughened (finely pitted) eggshell and contain a central single-celled morula. Descriptions of the 4 successive larval stages (L1-L4) are limited as embryonation to L1 and the first moult to L2 occur within the eggs and subsequent moults to L3 and L4 occur within host tissues. Larvae recovered from eggs and host tissues were stout cylindrical forms with rounded heads, thin tubular oesophagi, granular intestines and short tapering tails. Larvae (L3/4) recovered from horse lungs 14 days after infection measured 1.6-2.4 mm long, but 0.7-1.0 mm at 23 days, while young worms (sometimes designated L5) recovered from the intestines at 14 days measured 2.0.-2.5 mm long and 4.2-5.1 mm after 23 days. Adults are exceptionally large white worms with stout rigid cylindrical bodies measuring from 10-50 cm in length. They lack cervical alae and have a small buccal capsule surrounded by 3 large hexagonal- to heart-shaped lips, each divided by a transverse groove (notch) on the medial surface, a short tubular oesophagus connected to the intestines, and short conical tails with a subterminal anus/cloaca. Adults are sexually dimorphic, with females being larger than males (150-500 x 3-8 mm cf. 100-300 x 3-6 mm). Mature females are didelphic and have 2 extensive ovaries and uteri connected to a common vulva located just posterior to the midbody. They have rounded tails terminating in a short conical process. Mature males have rounded or bluntly conical tails with small caudal alae (absent in *P. felis*). They lack a gubernaculum but have 2 long stout spicules (2.0-2.5 mm) whose distal tips are rounded (*P. equorum*) or truncated/sharp (*P. univalens*).

Site of infection: Adults worms infect the small intestines of their equid hosts, being found in the lumen of the duodenum and proximal jejunum, rarely in the stomach. Earlier larval stages undertake hepatic-pulmonary migration from the gut to the liver and then the lungs before being coughed up the trachea and swallowed into the intestines.

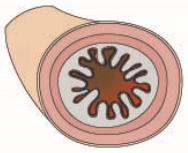
Pathogenesis: Light infections by *P. equorum* and *P. univalens* usually remain asymptomatic, but heavy infections may cause severe disease in suckling and weaning foals as they are highly susceptible due to their immunological immaturity. Foals usually become infected soon after birth and acquire a solid protective immunity over 6-12 months resulting in worm expulsion. Nevertheless, adult horses may harbour some worms and act as carriers, but heavy worm burdens are generally confined to foals and yearlings. Disease develops over 2 phases depending on the stage of infection: developing larvae may cause liver and lung lesions during the prepatent period; while adult worms may cause intestinal disturbances during the patent period. Larvae migrating through the interlobular tissues of the liver form narrow tracts, resulting in traumatic microlesions, haemorrhagic foci, and inflammation (hepatitis) with eosinophilia, later resolving to white fibrotic scars. When they reach the lungs in the pulmonary circulation, they migrate through to alveolar airspaces and ascend the respiratory tree causing pneumonitis, alveolitis, bronchiolitis and bronchitis with interstitial and/or broncho-pneumonia, exacerbated by host immune cells releasing cytotoxic granules which attack worms and damage respiratory tissues (eosinophil infiltrates are later replaced by lymphocytes). Subpleural grey-green lymphocytic nodules develop around dead or dying larvae (nodules more numerous following re-infection). Clinical signs include frequent coughing sometimes with grey nasal discharges, but most foals remain bright and alert although a few may develop fever and anorexia often in association with secondary bacterial infections. Pulmonary signs are most frequent in foals during the spring and autumn. Adult worms living in the intestinal lumen feed on liquid contents competing with the host for food and causing digestive disorders and intestinal disturbances. Gastric motility may be reduced with maldigestion and malabsorption, hypoalbuminaemia, and catarrhal enteritis with abdominal bloating, foul-smelling diarrhoea sometimes alternating with constipation, and colic attacks. Worms and inflammatory responses may also cause intestinal obstruction, impaction, intussusception and sometimes rupture with subsequent peritonitis. Animals may exhibit a variety of clinical signs, mostly involving chronic loss of condition. They develop capricious appetites, sometimes pica, rickets, dull coats, mild anaemia, unthriftiness, reduced growth rates, weight loss, lethargy, weakness, malaise, and emaciation. Aberrant migration of worms can occasionally result in obstruction of the bile and pancreatic ducts which may be life-threatening.

Developmental cycle and mode of transmission: *Parascaris* spp. have direct monoxenous life cycles involving faecal-oral transmission of eggs containing infective larvae. Female worms lay millions of eggs (up to 200,000 eggs per day) which are excreted with host faeces. The eggs embryonate to contain L1 which then moult within the egg to form sheathed L2 over 7-14 days (longer in cooler conditions). The eggs are extremely resistant to external environmental conditions (desiccation, warmth and sunlight) and can survive for up to 10 years in favourable cool climates. Egg development may be arrested over cold winter periods, but they do not survive hot summers unless protected from desiccation in organic matter (such as manure). The eggs are dispersed in the environment by wind and water action and their sticky coats allow them to adhere to many substrates (horses, stables, vegetation, small insects) thus providing many potential sources of infection for foals. Equids become infected by ingesting eggs containing infective L2. Ingested eggs hatch in small intestine and the released larvae penetrate the gut wall and undertake undergo hepato-pulmonary migration. They move to the liver within 2 days via the portal system moulting along the way to L3 which then migrate through the liver overt the next 7 days (leaving white fibrotic tracts). L3 then re-enter the blood stream through the hepatic vein to the caudal vena cava and subsequently travel through the heart to the pulmonary artery and the pulmonary capillaries to eventually reach the alveoli by 14-23 days. Some workers think the L3 moult to L4 here, while others think it occurs later in intestines. The larvae ascend the mucociliary escalator to the trachea where they are swallowed and return to the small intestine. Here they moult (once or twice) and mature to adults over 7-10 days. The prepatent period (time from infection to first egg excretion) ranges from 63-115 days and the patent period (duration of egg excretion) may last up to 2 years but often declines within several months as acquired immune responses may lead to worm expulsion. There is no evidence of transplacental or transmammary transmission. Infections are abundant in horse populations around the world due to the high fecundity of the females and the extreme resistance of the eggs to external environmental conditions. Infections often demonstrate a seasonal cycle coinciding with the arrival of new susceptible foals in herds with high rates of infections in yearlings (rather than adults).

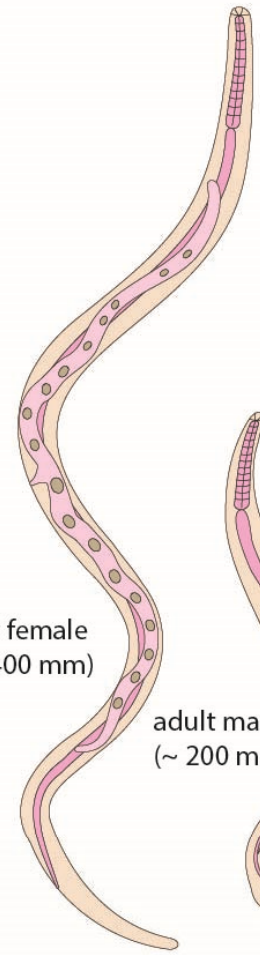
Differential diagnosis: Clinical signs in foals (cough, intermittent diarrhoea and/or colic) and yearlings (loss of condition) may be suggestive of infection, but a variety of other conditions may cause similar signs. Haematological examinations often reveal elevated white blood cell counts with marked eosinophilia and blood biochemistry may sometimes reveal elevated liver enzymes and hypoalbuminaemia. Medical imaging techniques (thoracic or abdominal radiography) may show inflammation of the airways and/or air sacs and help locate gut obstructions. Endoscopic examination (bronchoscopy) has occasionally been used to detect larvae in the respiratory tract in association with increased mucus production. Infections are conventionally diagnosed by the microscopic detection of characteristic eggs (round with thick pitted walls) in faecal samples, usually following their concentration by sedimentation in water and centrifugal floatation in saturated sugar or heavy metal salt solutions. Infections may also be diagnosed at necropsy by the detection of large white worms inside the small intestines. A range of genotypic characterization techniques have been applied to the identification of parasite species. Karyotyping studies found *P. equorum* to have 4 chromosomes with terminal and intercalary heterochromatin, and *P. univalens* to have 2 chromosomes with terminal heterochromatin. Molecular biological techniques have revealed further differences between these species by polymerase chain reaction (PCR) amplification and restriction fragment length polymorphism (RFLP) analyses or sequencing of nuclear genes (internal transcribed spacer 2 of ribosomal RNA, beta-tubulin) or the entire mitochondrial genome.

Treatment and control: A range of anthelmintic drugs have been used to treat clinical infections in equids, including benzimidazoles (fenbendazole, febantel, oxfendazole, oxibendazole, albendazole, thiabendazole), macrocyclic lactones (ivermectin, abamectin, moxidectin), tetrahydropyrimidines (pyrantel), diethylenediamines (piperazine) and even organophosphonates (dichlorvos, trichlorfon). Most treatments were effective against adult worms in the gut, but care should be taken treating individuals with high worm burdens to avoid subsequent gut obstructions. Few anthelmintics were effective against larval stages in the tissues, therefore repeated treatments are mandated (often monthly from 2-12 months of age). Regrettably, the widespread adoption of regular prophylactic treatment in the horse industries has led to the development of drug resistance in parasite populations, particularly to macrocyclic lactones and pyrantel. It is now recommended by many veterinary authorities that prophylactic treatment not be carried out without diagnosis, that faecal egg count reduction tests be conducted to select the most efficacious drug, that drenches be rotated on a cyclic basis, and that animals be treated systematically (foals and yearlings) or selectively (as required). In order for animals to develop a solid protective immunity against disease, they must still be exposed to low levels of natural infection, so the objective of treatment programmes should be control rather than eradication. Various preventive strategies may also be applied at the farm level to reduce transmission rates; mostly by improved sanitation and hygiene (frequent removal of faeces from pastures, regular cleaning and disinfection of stables and yards, provision of clean food and water), stock management (quarantine and isolation, reducing stocking rates, treating pregnant horses, separating foals and yearlings, stabling or moving to clean pastures after treatment) and pasture management (pasture rotation or spelling, but remembering that worm eggs may remain viable for years).

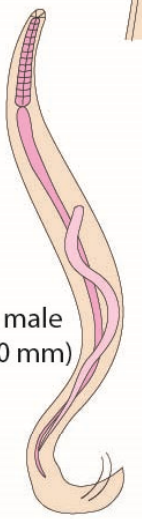
Parascaris



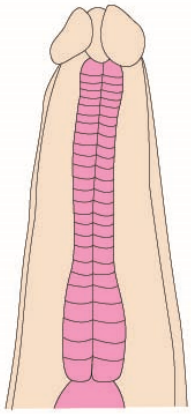
intestines
(hepatitis, pneumonitis,
enteritis, illthrift)



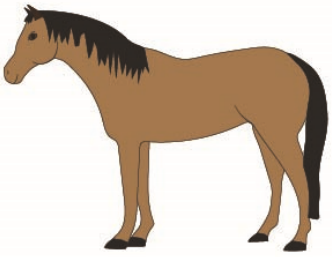
adult female
(~ 400 mm)



adult male
(~ 200 mm)

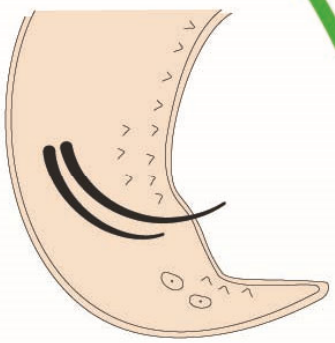


head

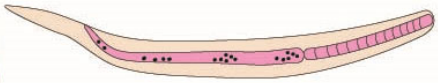


Definitive Hosts
(equids)

hepato-
pulmonary
migration



male tail
(lateral)

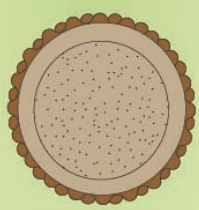


second-stage larvae (L2)
(~ 2 mm)

eggs
ingested

eggs
excreted
in faeces

larva develop
within eggs



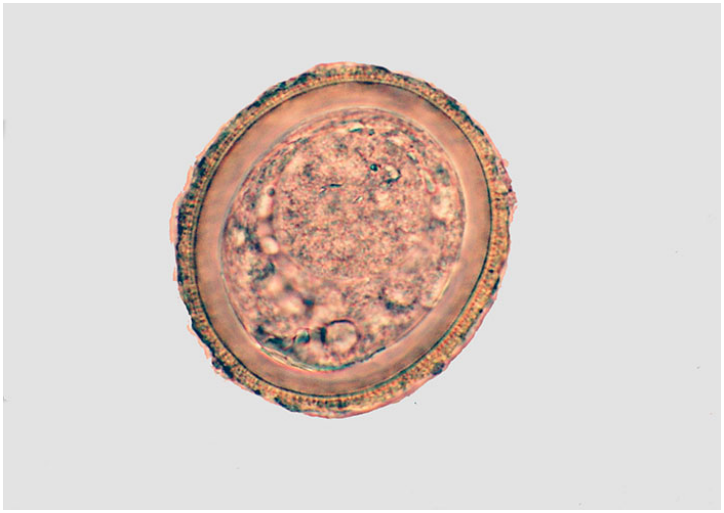
eggs
(~ 110 µm)

external
environment

faecal-oral transmission

Parascaris adult worms

Parascaris adult worms



Parascaris worm egg