Strongylus
(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 spirurinids, tylenchinids and rhabditinids. The latter contains the infraorder Rhabditomorpha which includes strongyloid nematodes characterised by an expansion of the tail of the male known as the copulatory bursa (clasper with one dorsal and two lateral lobes with muscular rays). Many families are recognised: including the strongylids which are stout worms with a large buccal capsule often armed with teeth and with a leaf crown around the mouth. Small and large species occur in the intestinal or respiratory tracts of domestic animals and birds. They have direct life-cycles where eggs passed in faeces develop to L3 (L2 cuticle retained as a sheath). Hosts ingest L3 which exsheath, migrate into the mucosa and moult into L4 before returning to the lumen to moult into adults. Extra-intestinal migrations of larvae and/or young adults through host organs sometimes occurs. *Strongylus* spp. are migratory large strongyles in the caecum/colon contributing to diseases associated with colic, diarrhoea and anaemia 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:	Rhabditina (free-living or parasitic in invertebrates/lower vertebrates)
Infraorder:	Rhabditomorpha (‘rod-shaped’ buccal cavity)
Superfamily:	Strongyloidea (bursate males, prominent buccal capsules, parasites of mammals, birds, reptiles)
Family:	Strongylidae (strongyles, large buccal capsules, often with teeth/leaf crown, infection by ingestion of L3, three pairs of branches in dorsal ray, equid hosts,)
Subfamily:	Strongylinae (large strongyles, red-worms, globular buccal capsules)
Genus:	*Strongylus* (parasitic in caecum/colon of equines)
Species:	various species cause diseases associated with colic, diarrhoea and anaemia in horses

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 with 2 ovaries (some monodelphic or polydelphic), 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 His
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 superfamily Strongyloidea comprises a range of worms often with prominent buccal capsules and specialised oral structures well-suited to their feeding habits on host tissues and/or fluids. Adults of most species are parasitic in the gastrointestinal tracts of mammals and some birds, while larval stages feed on bacteria in the external environment, although some larvae may infect invertebrates as intermediate or paratenic hosts. The adult worms are sexually dimorphic, the smaller males characterised by an expansion of the tail (bursa) which is used as a copulatory clasping organ. Many classification schemes group these ‘bursate’ nematodes into one or more superfamilies in the order Strongylida (with suborders containing the strongyles, trichostrongyles, hookworms and lungworms), although the families essentially remain the same. Many families are recognised on the basis of parasite morphology, biology, life-cycle, host specificity and tissue tropism; including the following which contain many notorious parasites of vertebrates.

Representative Strongyloidea (cf. Strongylida) [with bursate males]				
Family	**Characters**	**Definitive Hosts**	**Transmission***	**No. genera**
Strongylina (strongyles)				
Strongylidae (strongyles)	large buccal capsule often armed with teeth, leaf crown around mouth	mammals, reptiles, birds	ingestion of L3	32
Chabertiidae (nodule worms)	large buccal capsules, leaf crown of labial collar, L3 sheathed	artiodactyls, primates	ingestion of L3	22
Syngamidae (gapeworm)	cup-shaped buccal capsule, armed with teeth, male attached to female	birds, mammals	ingestion of L3 or invertebrate PH	7
Stephanurinae (kidneyworm)	buccal capsule armed with teeth, leaf crowns and external epaulettes	suids	transdermal penetration or ingestion of L3 or PH	1
Trichostrongylina (trichostrongyles)				
Trichostrongylidae (trichostrongyles)	reduced buccal capsule, ridged synlophe, oesophagus lacking bulb, thin-shelled eggs	artiodactyls, birds	ingestion of L3	50
Molineidae (stomach/intestinal worms)	reduced buccal capsule, cephalic vesicle, female tail with spine or cusps, oviparous/viviparous	mammals, birds, reptiles	ingestion of L3	61
Heligmonellidae (hookworm-like)	body coiled, cephalic vesicle, ridged synlophe, bursa asymmetrical	mammals, birds	transdermal penetration of L3	56
Ancylostomatina (hookworms)				
Ancylostomatidae (hookworms)	large buccal capsule bent dorsally, armed with teeth/cutting plates	primates, carnivores, artiodactyls	transdermal penetration of L3 (sometimes *per os*)	20
Metastrongylina (lungworms)				
Metastrongylidae (lungworms)	small buccal capsule, 2 trilobed lips, bursa with reduced dorsal lobe	suids	ingestion of IH carrying L3	1
Protostrongylidae (lungworms)	small buccal capsule, bursa with large lobes, gubernaculum	artiodactyls	ingestion of IH carrying L3	17
Angiostrongylidae (lungworms)	no or reduced buccal cavity, short club-shaped oesophagus	carnivores, rodents	ingestion of IH or PH carrying L3	28
Dictylocaulidae (lungworms)	small buccal capsule, bursa with large lobes, short stout spicules	ungulates, reptiles	ingestion of L3	5
Filaroididae (lungworms)	small buccal capsule, reduced male bursa, infective L1	carnivores	ingestion of L1	4

*IH = intermediate host, PH = paratenic (transport) host, L1 = first-stage larva, L3 = third-stage larva

Strongyle worms are characterised by their prominent buccal capsules with lips, leaf crowns or labial collars at the mouth opening, sometimes with teeth or cutting plates. They are parasitic in a variety of organ systems in a range of animals, including the large intestines of mammals (ruminants, suids, elephants, perissodactyls, rodents, marsupials and primates), tortoises and ratite birds, the forestomach of marsupials, the trachea of birds and mammals, and the renal system of suids. Four families are recognised: Strongylidae (buccal capsule globular or cylindrical, oral opening circular, dorsal ray with 6 terminal branchlets, 32 genera in large intestines of mammals, reptiles and birds); Chabertiidae (buccal capsule globular or cylindrical, oral opening circular or oval, with leaf crown or labial collar, with up to 3 teeth at base of buccal capsule, dorsal ray with 4 terminal branchlets, 22 genera in gastrointestinal tract of mammals); Syngamidae (buccal capsule subglobular, oral opening hexagonal, numerous teeth at base of buccal capsule, dorsal ray with 4 terminal branchlets, 7 genera in respiratory, urinary and digestive tracts of mammals and birds); and Deletrocephalidae (oral opening hexagonal, dorsal ray with 6 terminal branches, 2 genera in large intestines of birds). The family Strongylidae contains 2 subfamilies conventionally differentiated on worm size and buccal capsule morphology: namely, Strongylinae ('large' strongyles (or strongylins) with globular or funnel-shaped buccal capsules) parasitic in the large intestines of perissodactyls, elephants and ratite birds (sometimes undergoing extra-intestinal migration); and Cyathostominae ('small' strongyles (or trichonemes, cyathostomes, cyathostomines, cyathostomins) with cylindrical or ring-shaped buccal capsules) found in the large intestines of perissodactyls, elephants, hyracoids, tortoises and occasionally some artiodactyls. Contemporary molecular characterization studies, however, do not fully support the separation of these subfamilies as several 'large' strongyles group together with the 'small' strongyles.

Genus	No. spp.	Definitive Hosts	Location	Adult worms	Worm eggs
Strongylinae (‘large’ strongyles or strongylins)					
Strongylus (redworms)	13	ungulates, esp. equids	large intestines	10-50 mm long, red colouration, large globular buccal capsule often armed with teeth, leaf crowns, L3 with long filamentous sheath	64-99 x 36-58 µm, ellipsoidal, thin-shelled
Triodontophorus, Oesophagodontus, Craterostomum, Bidentostomum	11	equids	large intestines	6-25 mm long, globular or funnel-shaped buccal capsule often with teeth, leaf crowns [*despite their large strongyle features, molecular studies indicate that they are genetically closer to small strongyles]	66-130 x 33-68 µm. ellipsoidal, thin-shelled
Cyathostominae (‘small’ strongyles* or cyathostomins)					
Cyathostomum, etc.	6	equids	large intestines	5-30 mm long, red-grey colouration, cylindrical buccal capsule, with labial collar, some with dorsal gutter, external and internal leaf crowns variable	35-150 x 17-68 µm, ellipsoidal, thin-shelled

The subfamily Strongylinae conventionally contains 11 genera: 7 typified as large strongyles (*Strongylus* (syn. *Sclerostoma*, *Sclerostomum*, *Alfortia*, *Delafondia*) in equids; *Choniangium, Decrusia, Equinurbia* in elephants; *Codiostomum* in ostriches; *Macropicola* and *Hypodontus* in macropodid marsupials); and 4 other genera (*Triodontophorus* (syn. *Triodontus*)*, Bidentostomum, Craterostomum,* and *Oesophagodontus* (syn. *Pseudosclerostomum*) in equids, originally considered to be large strongyles. However, recent molecular-genetic studies have grouped them together with the small strongyles. The genus *Strongylus* contains large worms with deep buccal capsules, high collars, corona radiata with fine needle-like elements, buccal teeth if present with rounded points, males with 3 pairs of branches in the dorsal ray of the bursa, and straight spicule tips. Early studies recognised 5 *Strongylus* subgenera on the basis of host occurrence and the variable possession of spicule accessory pieces and buccal teeth: namely *S.* (*Strongylus*)*, S.* (*Alfortia*) and *S.* (*Delafondia*) in equids; *S.* (*Decrusia*) in elephants; and *S.* (*Dicerocola*) in rhinoceros. In contrast, other biotypic studies promoted many subgenera to generic status, but recent genotypic studies did not find significant differences between the subgenera in horses and thus collapsed them into the single genus *Strongylus*. Parasites in equids are commonly known as bloodworms (also redworms, palisade worms or migratory strongyles) and have been associated with thromboembolic colic (verminous arteritis) due to migrating larvae, as well as colonic ulceration and/or diarrhoea due to adult worms. Infections were prevalent in horses around the world until the introduction and widespread application of ‘modern’ anthelminthics in the 1970s.

***Strongylus* species**	**Definitive Hosts**	**Location [Clinical signs]**	**Distribution**
S. additictus (now *Decrusia*)	Proboscidea: elephantid (Indian elephant)		India
S. asini (syn. *S.* (*Delafondia*))	Perissodactyla: equid (donkey, horse, Burchell’s zebra, Hartmann’s mountain zebra, Grevy’s zebra)	large intestines, liver [cysts]	Africa
S. auricularis	Anura: bufonid (common toad), ranid (edible frog), pelobatid (common spadefoot)		Eurasia
S. edentatus (syn. *S.* (*Alfortia*))	Perissodactyla: equid (horse, Przewalski’s horse, donkey, mule, tarpan); Proboscidea: elephantid (African bush elephant); Rodentia: nesomyid (Gambian pouched rat)	large intestines [colic, anaemia]	worldwide
S. equinus (syn. *Sclerostonum armatum p.p.*) (syn. *S.* (*Strongylus*))	Perissodactyla: equid (horse, Przewalski’s horse, donkey, mule, onager, Burchell’s zebra)	large intestines [colic, anaemia]	worldwide
S. hypostoma (syn. *Sclerostoma*)	Artiodactyla: bovid (sheep)		
S. micrurus	Perissodactyla: equid (horse, onager)		Asia
S. myoxi	Rodentia: glirid (edible dormouse)		Europe
S. nodularis	Gruiformes: rallid (Eurasian coot)		Europe
S. paradoxus (syn. *S.* (*Alfortia*))	Artiodactyla: suid (pig)		Europe
S. sagittatus (*Varestrongylus*?)	Artiodactyla: cervid (red deer)		Europe
S. tremletti (now *Dicerocola*)	Perissodactyla: rhinocerotid (black rhinoceros)	colon	Africa

S. ventricosus	Artiodactyla: cervid (red deer)		Europe
S. vulgaris (syn. *Sclerostonum armatum p.p.*) (syn. *S.* (*Delafondia*))	Perissodactyla: equid (horse, Przewalski's horse, donkey, mule, Burchell's zebra)	large intestines, mesenteries [colic, anaemia]	worldwide

Many early descriptions of enteric bursate nematodes in mammals, reptiles and amphibians recorded species within the genus *Strongylus s.l.* (*sensu latu*, in the broad sense), but most have since been reclassified to other strongylid genera: e.g., species in sheep included *Strongylus contortus* (now *Haemonchus contortus*), *Strongylus hypostomum* (now *Chabertia ovina*), and *Strongylus filaria* (now *Dictyocaulus filaria*). There are therefore many historical records of *Strongylus* spp. which are no longer valid as they have been synonymized elsewhere or are considered *nomen dubium* or *species inquirenda*.

Parasite morphology: *Strongylus* spp. form 3 different types of developmental stages: eggs; larvae (L1-L4); and adult worms. Freshly-laid eggs are elliptical to ovoid in shape, ranging in size from 64-99 x 36-58 µm depending on species (e.g., *S. vulgaris* eggs: 64-75 × 36-46 µm, *S. asini* eggs: 75 × 45 µm, *S. equinus* eggs: 71-90 × 42-58 µm, *S. edentatus* eggs: 90-99 × 43-51 µm). The eggs are surrounded by a smooth thin eggshell and contain a morula at the 4-8 cell (blastomere) stage of development. The eggs release first-stage larvae (L1) which undergo 3 moults, forming L2, L3 and L4 before moulting to form young adults. L1 and L2 are free-living stages measuring up to 750 µm in length and they have a rounded head, a rhabditiform (bulbed) oesophagus, and a long tapering tail. L3 begin as free-living ensheathed stages but when they become infective and are ingested by a suitable host, they exsheath and become parasitic. The sheath is formed by retention of the L2 cuticle as a protective covering and it characteristically has a long filamentous tail (260-300 µm). Including the sheath, L3 range from 740-1,090 µm in length, but they do exhibit some species-specific variation with respect to size as well as gut structure. *S. edentatus* form short thin L3 (740-840 µm long) with the gut (alimentary tract) comprising 18-20 poorly-defined elongated cells, *S. equinus* form long thin L3 (920–1,020 µm) with the gut comprising 16 poorly-defined rectangular cells, and *S. vulgaris* form long broad L3 (930-1,090 µm) with the gut comprising 28-32 well-defined rectangular cells. All L3 have conical heads with small buccal cavities and a long thin strongyliform (non-divided) oesophagus. L4 are parasitic stages that are 2-5 times the size of L3 and whose genital primordia have begun to display gender-specific organs (notably male bursa and female vulva structures). Adults are large stout tubular worms measuring from 10-50 mm in length by 1.0-2.3 mm in width. They are often dark red to light black in colour due to their uptake of host blood (hence commonly known as red-worms or blood worms). They are bound by a tough cuticle with circular striations but lacking longitudinal ridges (synlophe absent) and lacking an anterior inflation (cephalic vesicle absent). The anterior oral opening is circular and surrounded by well-developed corona radiata with the external leaf crown containing longer but fewer needle-like elements than the internal leaf crown (40-75 cf. 42-80). The buccal capsule is large and subglobular (deeper than wide) and possesses a dorsal gutter (containing the duct of the dorsal oesophageal gland) and a variable number of teeth (e.g. no teeth for *S. edentatus,* 2 rounded teeth for *S. vulgaris,* 2 lobar teeth for *S. asini,* and 3 pointed teeth for *S. equinus*). Adult worms are sexually dimorphic, with females being larger than males (e.g. *S. vulgaris* females 13-25 x 1.0-1.4 mm cf. males 10-19 x 1.0-1.4 mm; *S. edentatus* 28-45 x 1.3-2.2 mm cf. 19-28 x 1.3-2.2 mm; *S. equinus* 35-50 x 1.1-2.3 mm cf. 24-36 x 1.1-2.3 mm. *S. asini* 30-42 mm cf. 18-33 mm). Mature males have a distinctive copulatory bursa comprising 2 medium lateral lobes supported by rays (comprising muscular elements following nerve channels to terminal papillae) arranged in a unique configuration (2 short ventral rays closely adjoined, 3 lateral rays radiating from a common stem, and single externodorsal ray arising from the base of the dorsal ray) and a well-developed rounded dorsal lobe supported by a dorsal ray with 3 pairs of branches. Males have a short conical genital cone, a small gubernaculum and 2 elongate spicules ranging in length from 1.9-3.2 mm with straight or curved tips (fused in the case of *S. vulgaris*). Mature females are didelphic with 2 ovaries and uteri connected to a vagina by paired muscular ovejectors (with a T-shape configuration) which open into a common vulva located in the posterior third of the body but anterior to the anus. The tail of female worms is short and tapers to a fine point. Females are oviparous and they lay numerous eggs.

Site of infection: Adult worms are luminal but attach to the mucosa of the large intestines (caecum and colon); while larval stages (L3 and L4) undergo extensive extra-intestinal migration (varying for each species) before returning to the gut and maturing to adults. Worm eggs deposited on pastures in faecal pats give rise to 2 free-living larval stages (L1-L2) before moulting to infective L3.

Pathogenesis: Strongylin infections (particularly *S. vulgaris*) can cause serious disease in domestic equids. In the past, the prevalence of disease (strongylosis/strongyliasids) was substantial, but the widespread introduction (from the 1970s onwards) and application of anthelmintic drugs led to a significant reduction in prevalence over time. Nonetheless, animals are still at risk of disease, depending on the pathogenicity of the parasite species involved (*S. vulgaris* being the most pathogenic), the intensity of infection (number of parasites), the stage of infection (migrating larvae cause more serious pathology than feeding adult worms) and host susceptibility (young and naïve animals are most at risk). Adult worms are tissue-plug feeders as they draw a plug of mucosa into their large buccal capsules, where it is broken down by teeth/plates and enzymes secreted via the dorsal gutter. This feeding also causes blood loss as they frequently rupture capillaries ingesting some blood as well as causing petechial haemorrhages and leakage. Worms are aggressive feeders and they frequently move to new feeding sites leaving behind small bleeding ulcers. Heavy feeding may result in significant traumatic damage to the intestinal mucosa, colonic ulceration, mild anaemia (normochromic

normocytic), hypoproteinaemia, fluid and protein loss, dehydration, depression, anorexia, weight loss, emaciation, poor condition and performance, and sometimes diarrhoea, fever and/or colic. However, most of the pathogenic effects are associated with the earlier extra-intestinal migration of developing larval stages which cause mechanical damage, inflammation (particularly linked to arteritis), intravascular thrombus formation, emboli, fibrosis and/or colic. The route of migration, and pathological consequences, varies for each *Strongylus* species, but it involves intra-abdominal organs and blood vessels over a period of 6-12 months. *S. vulgaris* larvae migrate along the walls of mesenteric arteries, *S. edentatus* larvae migrate to the liver and then the abdominal walls, and *S. equinus* larvae migrate through the intestinal mucosa to the liver and then the pancreas. For most species, late-stage larvae returning to the caecum/colon form nodules in the intestinal walls which subsequently rupture releasing young adults into the lumen. Encysted larvae provoke minimal inflammatory responses, but the synchronous emergence of large numbers may cause diffuse inflammation. *S. vulgaris* is the most pathogenic species, as the intravascular migration of larvae can cause a severe verminous arteritis with marked intimal thickening, cellular infiltrates, thrombotic lesions and ensuing emboli. Thromboemboli released from the arterial wall can partially or completely block vessels downstream in the large intestines, resulting in ischaemia (as a consequence of restricted blood flow or blockage), infarction, necrosis (sometimes with ensuing gangrene) and associated clinical signs including anorexia, constipation/diarrhoea, marked pyrexia, and a rapid onset of severe colic (intermittent or persistent pain in the abdominal cavity), substantially elevated respiratory and heart rates, and sudden death (sometimes due to rupture of aneurisms close to the posterior aorta). Thromboemboli released from the arterial wall (of heavily infected equids) can also travel to and intermittently or completely occlude the femoral artery (uni- or bi-laterally – usually unilateral), resulting in hypoxia or ischaemia in the musculature of the rear leg(s). Affected horses exhibit an 'intermittent lameness', as the emboli can dislodge within the artery during exercise (enabling blood to flow and oxygen to get to muscles). The severity of disease usually depends on the number of migrating larvae, although moderate infections by 600 larvae can be fatal in foals with low levels of immunity. Acute disease is often observed in older foals/yearlings when grazed on heavily contaminated pastures, while chronic sporadic disease can become apparent over months to years. *S. equinus* and *S. edentatus* larvae may also cause haemorrhages, oedema, inflammation, fibrosis and/or nodule formation in the tissues through which they move (liver, pancreas, peritoneal cavity), but the lesions not as severe as those caused by *S. vulgaris* larvae. Aberrant larval migrations have been associated with haemorrhages and granulomatous lesions (sometimes with adhesions or 'tags') in the abdominal muscles, omentum, ileum wall (haemomelasma ilei), kidneys, testes, diaphragm or lungs. Changes to clinical parameters during larval migration have included reduced haematocrits, hypoproteinaemia (particularly hypoalbuminaemia), hypergammaglobulinaemia, and leucocytosis (including eosinophilia).

Developmental cycle and mode of transmission: *Strongylus* spp. have direct monoxenous life-cycles involving the faecal-oral transmission of infective larvae (L3) from pastures. Gravid female worms lay many eggs which are excreted with host faeces contaminating the external environment. Eggs embryonate and hatch in faecal pats releasing free-living rhabditiform L1 which feed on bacteria. These larvae moult to free-living L2 which also feed on bacteria. Eventually they moult to form L3 which are ensheathed (encased in L2 cuticle) and therefore do not feed. Development to L3 may be completed in as little as 3 days under optimal conditions (warm and moist), but usually takes 1-2 weeks in suboptimal conditions (cool and/or dry). L3 can survive for months in faecal pats or in the underlying soil, even surviving cold winters under protective snow cover. Given adequate moisture (particularly following rainfall), L3 exhibit horizontal migration (dispersing from faecal pats) and vertical migration (ascending vegetation). Hosts become infected when they ingest infective L3 when grazing. The L3 exsheath in the gastrointestinal tract and undergo extensive somatic (intra-abdominal) migration before returning to the large intestines to mature as adults. The long duration of larval migration accounts for very long prepatent periods (5-12 months) before worms mature and produce eggs. The route of migration also varies for each species. *S. vulgaris* L3 penetrate the intestinal mucosa and moult to L4 in the submucosa over 5-7 days. These larvae penetrate blood vessels and migrate along the endothelium of the mesenteric arterial network for several months before moulting and migrating to the intestinal wall and form nodules which eventually rupture releasing young adult worms into the lumen. The prepatent period (time from infection to first egg production) is 5-7 months and the patent period (duration of egg production) can last up to 18 months. *S. edentatus* L3 migrate via the portal system to the liver where moult to L4 (some within granulomata). Weeks later, they emerge and enter the hepatic ligaments and migrate retroperitoneally through the subperitoneum, mesenteries and abdominal wall (predilection for the flanks). Here they moult to L5 which migrate back to the large intestines forming purulent nodules which rupture releasing young adults into the lumen. The prepatent period is 10-11 months and patency may last for up to 18 months. *S. equinus* L3 form haemorrhagic nodules in the muscular and subserosal layers of the intestines before moulting to L4 and migrating across the peritoneal cavity to the liver where migration continues for several weeks. Then, L4 enter the pancreas and develop and migrate to the gut lumen via the connective tissue adhesion of the pancreas and the head of the caecum. The prepatent period is 8-9 months and patency may last for 18 months. Infections in equids typically involve large mixed populations of numerous strongyle species, so that the epidemiological and clinical pictures can become confounded. Although worms may persist year-round, many infections exhibit annual seasonal cycles, with egg production and pasture larvae development and survival varying markedly depending on climatic conditions and geographic location (higher in spring in temperate regions or in autumn in subtropical regions).

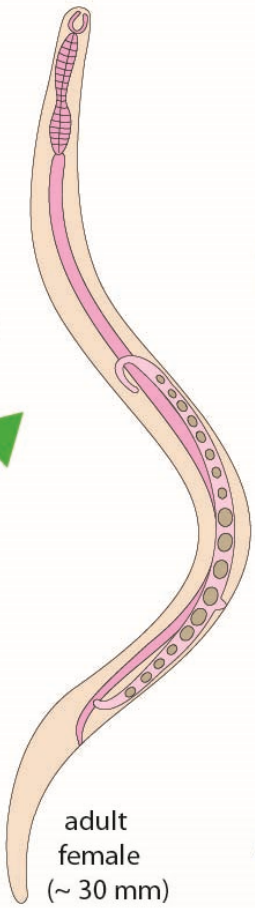
Differential diagnosis: While infections may be suspected on the basis of clinical signs (acute or chronic colic) and appropriate history (e.g., young animals, climate and season), other aetiological agents may be implicated. If verminous arteritis is considered as a cause of colic, physical examination (palpation per rectum) may reveal a painful enlargement of the root of the mesentery. Various clinical parameters are also indicative but not definitive, including changes in serum biochemistry (hypoproteinaemia, hypoalbuminaemia, increased alkaline phosphatase, increased gamma-glutamyl transpeptidase), haematology (anaemia,

leucocytosis, eosinophilia) and discolouration of peritoneal fluid (dark yellow). Patent infections are usually diagnosed by the detection of worm eggs in faecal samples, usually following their concentration by sedimentation in water and/or flotation in saturated sugar or salt solutions (with some techniques being quantitative and estimating the number of eggs per gram of faeces (epg)). However, coprological techniques cannot diagnose prepatent clinical infections due to migrating larval stages (as egg production has not yet commenced). The differential diagnosis of infections on the basis of egg morphology is not possible as most strongyle eggs are similar in size, shape and appearance. Instead, faecal samples may be incubated for 10-14 days to harvest L3 for microscopic identification (*Strongylus* spp. form ensheathed L3 with long filamentous tails and intestines comprising 16-32 elongated or rectangular cells, whereas those of small strongyles have intestines comprising 8-16 triangular or pentagonal cells). Serological techniques (enzyme immunoassays) have also been developed to detect host antibodies against specific parasite antigens, but their test specificity and sensitivity remains to be critically validated. Molecular biological techniques have also been used to characterize eggs, larvae and adults of various species by the polymerase chain reaction (PCR) amplification of specific nuclear genes (ribosomal DNA sequences, including internal transcribed spacer regions 1 and 2, and the intergenic spacer).

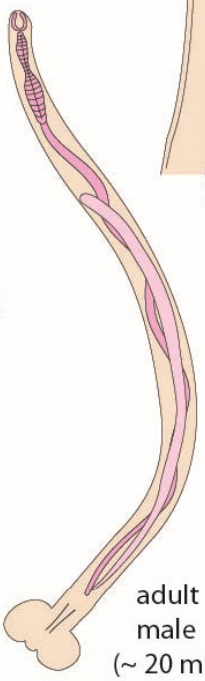
Treatment and control: Infections have been successfully treated with a range of anthelmintic drugs, with the macrocyclic lactones (ivermectin, moxidectin, abamectin) and benzimidazoles (fenbendazole, thiabendazole, cambendazole, oxibendazole) proving effective against larvae and adults, with tetrahydropyrimidines (pyrantel) being effective against adult worms only. The clinical treatment of severe cases of enteropathy may benefit from the administration of non-steroidal anti-inflammatory drugs. The widespread use of these drugs in domestic and performance horses has dramatically reduced the incidence of strongylosis worldwide, but sporadic outbreaks may occur in untreated feral and wild equid populations. It is recommended that foals be treated regularly due to their high susceptibility, and that drenches be used in cyclic rotation to forestall the emergence of drug resistance (which regrettably has occurred with many small strongyles or cyathostomins). Control programmes (suppressive, strategic and integrated) should include pasture management strategies to reduce faecal contamination by worm eggs, and limit the development and survival of infective larvae on pastures; including stock rotation, mixed grazing, removing and composting faeces, and periodically spelling pastures. Several studies have also demonstrated the utility of biological control using nematophagous fungi to reduce larval contamination of pastures.

Strongylus

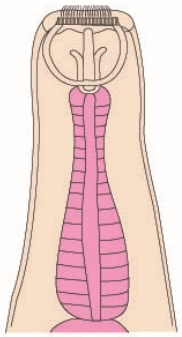
intestines
(diarrhoea, anaemia,
verminous arteritis, colic)



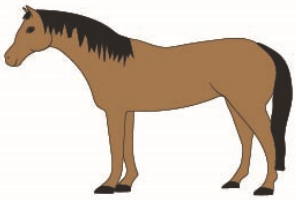
adult
female
(~ 30 mm)



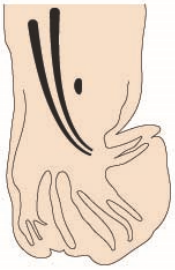
adult
male
(~ 20 mm)



head



Definitive Hosts
(perissodactyls)

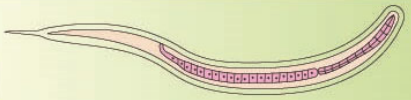


male tail
(lateral)

some undergo
extra-intestinal
migration

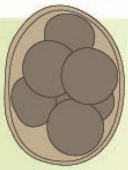
L3
ingested

eggs
excreted
in faeces



filariform third-
stage larvae (L3)
(~ 1 mm)

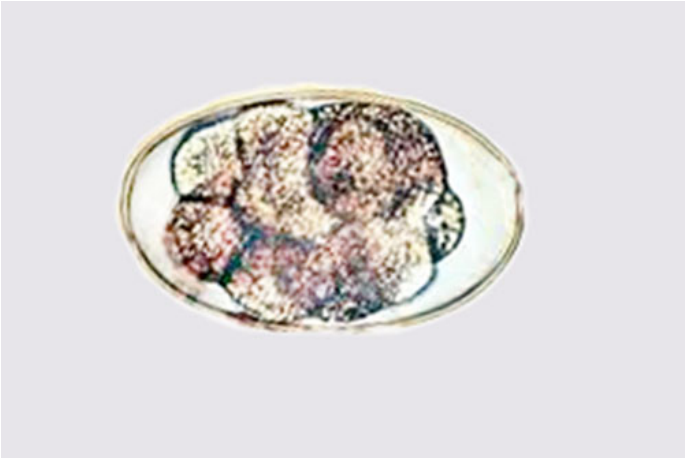
external
environment



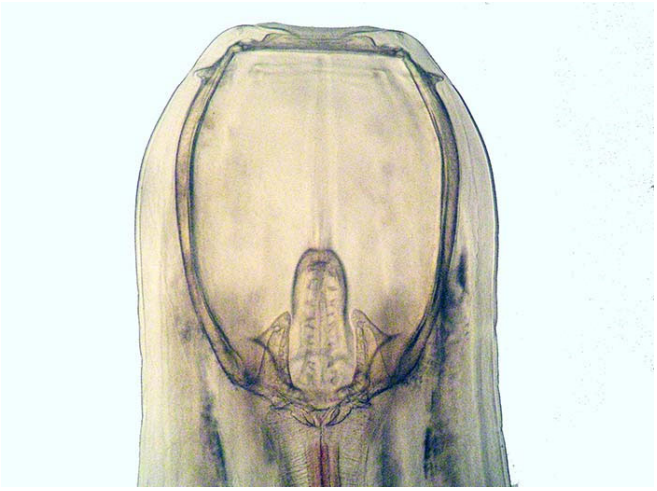
eggs
(~ 80 µm)

rhabditiform first- and
second-stage larvae (L1, L2)
(~ 750 µm)

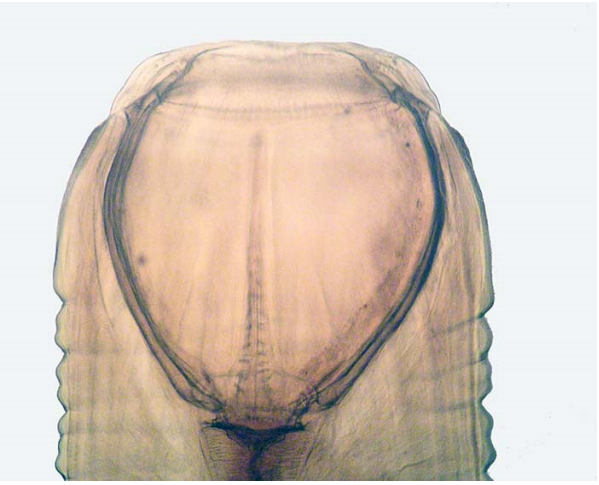
Strongylus adult worms



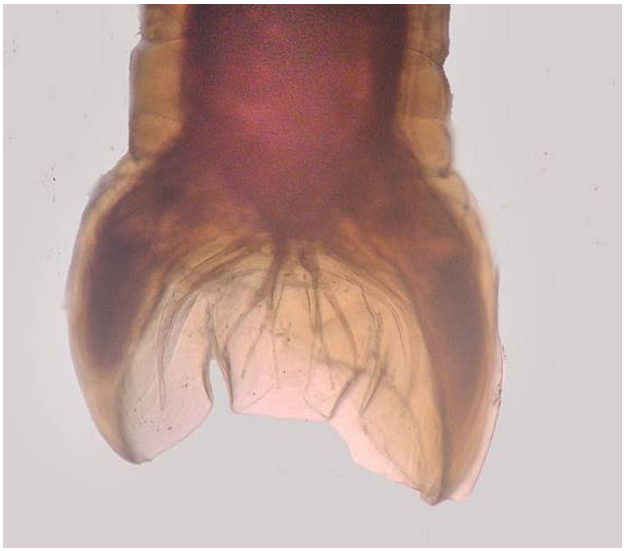
Strongylus worm egg



Strongylus equinus adult worm, head



Strongylus edentatus adult worm, head



Strongylus adult worm, male bursa