1 Modular Guide Series
Worm Control in Dogs and Cats

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There is a wide range of helminths, including nematodes, cestodes and trematodes, that can infect dogs and cats in Europe.

Major groups by location in the host are:

Intestinal worms

- Ascarids (Roundworms)
- Tapeworms
- Hookworms
- Whipworm
- Threadworm

Non-intestinal worms

- Heartworm
- Subcutaneous worm
- French heartworm
- Lungworms

The following series of modular guides for veterinary practitioners gives an overview of the most important worm species and suggests control measures in order to prevent animal and/or human infection.

Key companion animal parasites

Diagnosis of helminth infections

Patent infections caused by most of the worms mentioned can be identified by faecal examination. However, there are exceptions. Blood samples can be examined for microfilariae in cases of *D. immitis* and *D. repens*, for antigens of *D. immitis* and *A. vasorum* and for antibodies to D. immitis (in cats only).

Faecal examination for worm eggs or larvae should be carried out with at least 4–5 g faeces. Ascarid, hookworm, whipworm and taeniid eggs are easily recognisable. For the detection of lungworm larvae, the Baermann method should be used.

Since dogs may ingest or eat faeces, care should be taken to identify and eliminate false positive results caused by coprophagia.

Preventive measures

- Parasite infections should be controlled through endoparasite and ectoparasite management i.e. tailored anthelmintic treatment at appropriate intervals and/or faecal examinations[1].
- All common worms, with some exceptions such as *Dirofilaria* species, are transmitted by the passage of eggs or larvae in faeces, therefore hygiene measures, especially cleaning up pet faeces regularly, will reduce environmental contamination with infective parasite stages.
- Feeding commercial diets, cooked food or previously frozen food helps prevent parasite infections transmitted through raw meat. Dogs and cats should not be allowed access to rodents or livestock carcasses, placentae or aborted foetuses. They should also be provided with clean water.
- When recommending a parasite management programme, veterinarians should consider the animal's age, reproductive status, health status, history (including travel), nutrition and environment.

Preventing zoonotic infection

Pet owners should be informed about the potential health risks of parasitic infections, not only to their pets but also to family members, friends and neighbours. Regular deworming or participation in "pet health-check programmes" should be promoted to the general public by veterinary practitioners, veterinary nurses and other animal health professionals. Responsible dog and cat ownership, together with good personal hygiene, can reduce public health concerns.

- Carefully remove dog and cat faeces from yards and pens and dispose of responsibly.
- Practice good personal hygiene e.g. wash hands, keep fingernails short, rinse fruit and vegetables and wear gloves when working in soil.
- Dogs and cats should be dewormed regularly, 1–12 times a year depending on a risk assessment, following veterinary advice.
- Always feed commercial dry or canned diets or cooked food, NOT raw meat (freezing meat at -20°C for 1–2 days inactivates most parasites).
- Keep children away from contaminated areas. Reduce the risk of sandpit contamination by using covers.

[1] See www.esccap.org for links to therapy tables by country or region.

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ESCCAP Secretariat
Malvern Hills Science Park
Geraldine Road, Malvern
WR14 3SZ United Kingdom

Tel: +44 (0) 1684 585135
www.esccap.org

1 Modular Guide Series

1.1a: Dog roundworm (*Toxocara canis*, *Toxascaris leonina*)

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Toxocara canis is a large, intestinal nematode that can cause disease in dogs. It is also zoonotic.

Toxocara canis is a large worm with adults measuring as much as 15 cm in length. Pups can be heavily infected by *T. canis* worms in utero or via their mother's milk and this may cause serious illness in pups before diagnosis of patent infection is possible by faecal examination.

Toxascaris leonina, with adults measuring up to 12 cm long, can be found in both dogs and cats, though less frequently than the *Toxocara* species and generally less pathogenic. In contrast to canine and feline *Toxocara* species, the released third-stage larvae of *T. leonina* do not migrate but return to the intestinal lumen after moulting in the intestinal wall, where they develop into sexually mature stages and excrete eggs after approximately 7–10 weeks. Somatic migration can occur in non-canid/felid hosts (e.g. mice) that can then act as paratenic hosts. Diagnostic procedures and treatment strategies are comparable to those employed for *Toxocara* spp. infections.

Distribution

Toxocara canis is ubiquitous in dog and fox populations throughout the world. Prevalence of patent infections is higher in pups and lower in adolescent and adult dogs. However, there is no immunity at any age and adult dogs may show patent infections.

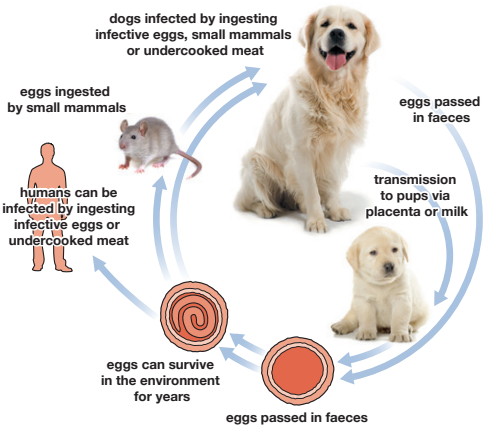
Life Cycle

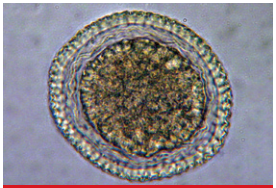
Adult worms inhabit the small intestine where they lay eggs that are then passed in the faeces. The eggs can become infective after several weeks and these can survive in the environment for years. Dogs become infected when they ingest infective eggs from the environment or raw/undercooked meat.

After ingestion of larvae via the predation of paratenic hosts or infective eggs from the environment, the prepatent period for *Toxocara* spp. is a little over four weeks.

The eggs hatch in the intestine, releasing larvae that penetrate the intestinal wall and undergo a hepatotracheal migration. The life cycle is completed when the larvae are coughed up and swallowed, returning to the small intestine to complete their migration.

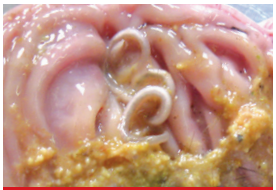
Somatic migration may occur in older canines and non-canid hosts, such as small mammals acting as paratenic hosts. In pups, infection can occur via the passage of larvae across the placenta from about the 42nd day of pregnancy and after birth, through the milk.





Toxocara canis egg

Toxascaris leonina egg



Adult worms live in the small intestine of infected dogs

Dogs can also become infected when they ingest infective eggs from the environment, eat undercooked meat or prey on an infected paratenic host (e.g. rodent). Infection of humans can occur as a result of accidentally ingesting infective eggs or undercooked meat containing larvae.

Clinical Signs

Pups carrying a heavy burden may appear cachexic with a distended abdomen. They may also show pulmonary signs, have loose faeces and may develop an intussusception. Older dogs are extremely unlikely to show clinical signs. Occasionally a worm may be passed in faeces or seen in vomit.

Diagnosis

Toxocara eggs are easily recognisable. Diagnosis is based on identifying eggs in faeces by using the flotation method with 4–5 g faeces (fresh or fixed). *Toxocara cati* eggs can be present in a sample as a result of coprophagia. Commercial coproantigen detection is also possible.

Treatment

Pups should be treated with appropriate anthelmintics, normally starting when they are 14 days old, continuing at fortnightly intervals until two weeks after weaning. Monthly treatments should then be given until six months of age if there is an ongoing increased risk of infection (e.g. puppy playgrounds).

Nursing bitches should be treated concurrently with the first treatment of their offspring, since they may have patent infections.

Infection can occur in adult dogs but is extremely unlikely to be associated with clinical signs. Consequently, it is difficult to determine whether a dog is infected unless regular faecal examinations are conducted. It has been shown that increasing the frequency of treatment effectively reduces the occurrence of *T. canis* and monthly deworming can prevent patent infections.

Based on the prepatent period, monthly treatment with a suitable anthelmintic will minimise the risk of patent infections and can be recommended in high-risk scenarios such as pets living in families with small children and having access to gardens or parks. Deworming 4–12 times per year, depending on risk exposure, is the general recommendation[1]. Alternatively, where an owner chooses not to use regular anthelmintic therapy or where local legislation requires diagnosis or risk assessment prior to treatment, faecal examinations can be performed at the same frequency.

Control

Dogs should not be fed raw or undercooked meat unless it has been frozen at -20°C for 1–2 days, a process that inactivates most parasites in meat. Measures should also be taken to prevent dogs from hunting. Faeces should be collected and disposed of regularly and areas used by dogs should be kept clean. These are all important preventive measures that should be combined with appropriate anthelmintic treatment or faecal examinations.

[1] See www.esccap.org for links to therapy tables by country or region.

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Malvern Hills Science Park
Geraldine Road, Malvern
WR14 3SZ United Kingdom

Tel: +44 (0) 1684 585135
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1 Modular Guide Series
1.1b: Cat roundworm (*Toxocara cati*)

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Toxocara cati is a large, intestinal nematode that can cause disease in cats. It is also zoonotic.

Toxocara cati is a large worm with adults measuring as much as 10 cm in length. Kittens can become heavily infected by *T. cati* larvae via the milk and these infections may cause serious illness before diagnosis is possible by faecal examination.

Distribution

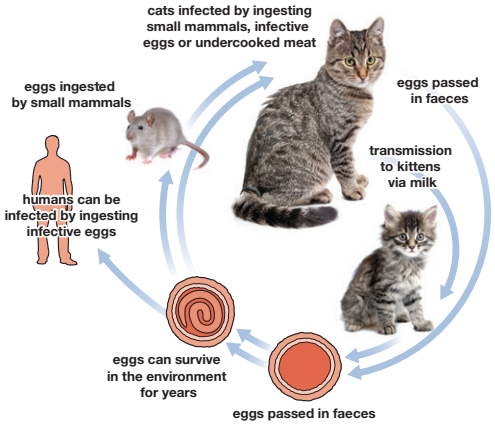
Toxocara cati is ubiquitous in cat populations throughout the world. Prevalence of patent infections is highest in kittens, lower in adolescent cats and lowest in adult cats. However, infection can also occur in adult cats.

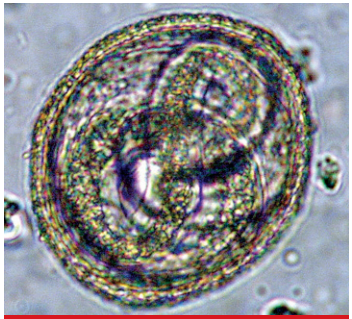
Life Cycle

Adult worms inhabit the small intestine where they lay eggs that are then passed in the faeces. The eggs can become infective after several weeks. Cats are infected when they ingest infective eggs from the environment or through the ingestion of infected paratenic hosts.

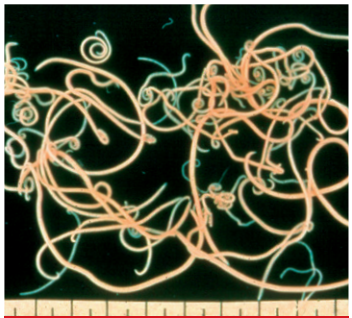
The prepatent period for *Toxocara* spp., after ingestion of larvae via predation of paratenic hosts (rodents) or ingestion of infective eggs from the environment, is a little over four weeks.

The eggs hatch in the intestine, releasing larvae that penetrate the intestinal wall and undergo a hepatotracheal migration. The life cycle is completed when the larvae are coughed up and swallowed,





***Toxocara cati* infective egg**



Adult worms live in the small intestine of infected cats

returning to the small intestine to complete their migration. Somatic migration may occur in older felines and non-feline hosts, such as rodents acting as paratenic hosts. In kittens, infection can occur through the milk.

Cats can also become infected when they eat undercooked meat or an infected paratenic host as prey. Infection of humans can occur as a result of accidentally ingesting infective eggs or undercooked meat containing larvae.

Clinical Signs

Infected kittens may show respiratory signs associated with a heavy burden of migrating larvae. Kittens carrying a heavy burden may appear cachexic with a distended abdomen. Older cats are extremely unlikely to show clinical signs. Occasionally, a worm may be passed in faeces or seen in vomit.

Diagnosis

Toxocara eggs are easily recognisable. Diagnosis is based on identifying eggs in faeces by using the flotation method with 4–5 g faeces (fresh or fixed). Commercial coproantigen detection is also possible.

Treatment

Because prenatal infection does not occur in **kittens**, fortnightly treatment can begin at three weeks of age and be repeated fortnightly until two weeks after weaning. Monthly treatment can then be given for six months if there is an ongoing increased risk of infection (e.g. free roaming).

Pregnant queens can be treated with emodepside spot-on approximately seven days before expected parturition to prevent lactogenic transmission of *Toxocara cati* larvae to the kittens.

Nursing queens should be treated concurrently with the first treatment of their offspring, since they may have patent infections.

Infection can occur in **adult cats** but is extremely unlikely to be associated with clinical signs. Consequently, it is difficult to determine whether a cat is infected unless regular faecal examinations are conducted. It has been shown that increasing the frequency of treatment effectively reduces the occurrence of *T. cati* and monthly deworming can prevent patent infections.

Based on the prepatent period, monthly treatment with a suitable anthelmintic will minimise the risk of patent infections and can be recommended in high-risk scenarios such as the pet living in a family with small children and with access to gardens or parks.

Deworming adult cats and dogs with outdoor access 4–12 times a year against intestinal nematodes is the general recommendation[1]. Animals with no or very limited access to the outdoors should be treated 1–2 times a year. Alternatively, faecal examinations can be carried out at the same frequencies, followed by anthelmintic treatment according to findings.

Control

Cats should not be fed raw or undercooked meat unless it has been frozen at -20°C for 1–2 days, a process that inactivates most parasites in meat. Preventing predation, collecting and disposing of faeces and good hygiene practices (e.g. between litters of kittens) are all important preventive measures that should be combined with appropriate anthelmintic treatment or faecal examinations.

[1] See www.esccap.org for links to therapy tables by country or region.

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1 Modular Guide Series
1.2a: Dog tapeworm (*Echinococcus granulosus*)

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Echinococcus granulosus is a small cestode that inhabits the small intestine of dogs and some other canids, excluding foxes.

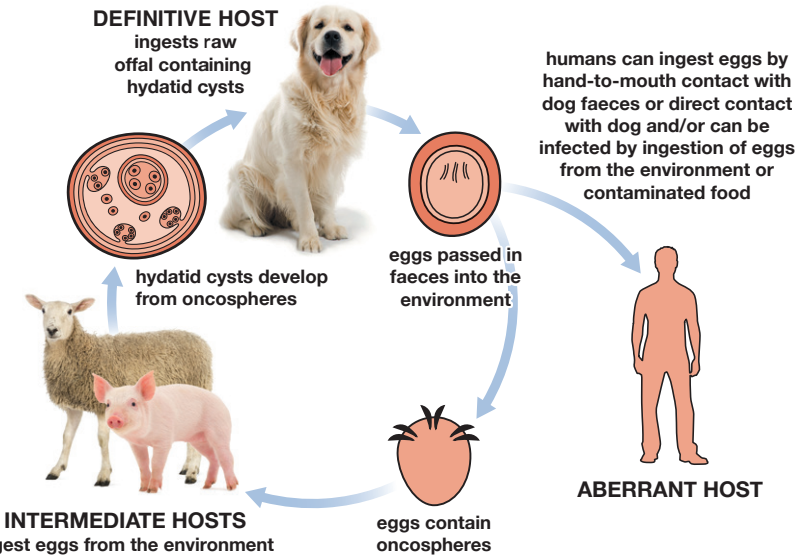
It is the cause of cystic echinococcosis in humans, due to infection with eggs passed in the faeces of infected canids.

Distribution

Echinococcus granulosus in sheep and pigs and associated species, (*Echinococcus equinus* in horses and *Echinococcus ortleppi* in cattle), are endemic in discrete areas of Europe.

Life Cycle

Adult worms inhabit the small intestine of canids, with the terminal proglottid breaking off once it matures. This is passed in the faeces and contains eggs that are immediately infective to the appropriate intermediate host. Within the intermediate host, the immature tapeworm leaves the intestine, normally coming to rest in the liver and lungs where it develops into a cyst containing many immature tapeworms. Definitive hosts become infected when they ingest the cysts within intermediate hosts.



Clinical Signs

Infected dogs are extremely unlikely to show clinical signs. The segments are too small for them to be evident in faeces.

Diagnosis

Specific diagnosis of *Echinococcus* infections in definitive hosts is difficult because the taeniid eggs cannot be differentiated morphologically and are passed intermittently. Coproantigen tests are not commercially available and polymerase chain reaction tests (PCRs) for species and/or genotype identification are only performed in specialised laboratories.

Therefore, in *Echinococcus* endemic areas, taeniid infections identified by egg detection should be considered potential *Echinococcus* infections.

Treatment

Where animals are infected with *Echinococcus* species, treatment with an anthelmintic containing praziquantel is advisable, carried out under veterinary supervision[1].

Dogs should be shampooed to remove any parasite eggs adhering to the coat.

The faeces of treated dogs should be disposed of appropriately.

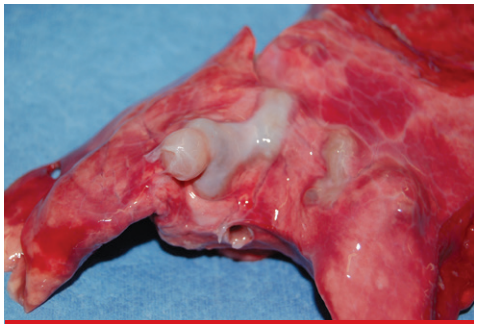
Personnel involved should use suitable protective clothing, including protective gloves.

Control

Dogs that may hunt or have access to offal or carcasses of *Echinococcus* intermediate hosts within an endemic area should be treated at least every six weeks with an effective anthelmintic containing praziquantel.

Dogs should not be fed raw or undercooked meat unless it has been frozen at -20°C for 1–2 days, a process that inactivates most parasites in meat. Care should also be taken to prevent them having access to raw offal and carcasses.

When travelling to the UK, Malta, Ireland, Finland and Norway, dogs must be treated for this infection



Dogs become infected when they ingest sheep or pig offal containing cysts

[1] See www.esccap.org for links to therapy tables by country or region.

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1 Modular Guide Series
1.2b: Fox tapeworm (*Echinococcus multilocularis*)

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Echinococcus multilocularis (fox tapeworm) is a small cestode that inhabits the small intestine of dogs, foxes, some other canids and, less commonly, cats.

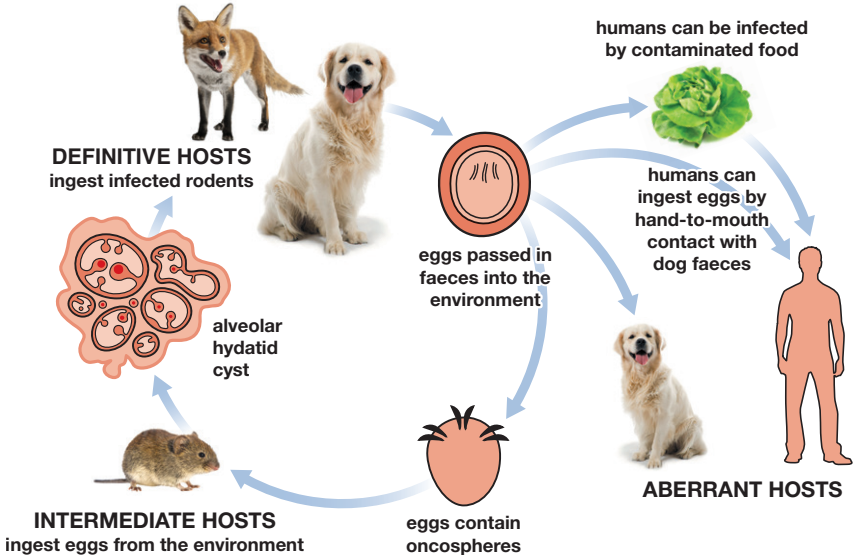
It is the cause of alveolar echinococcosis in humans, due to infection with eggs passed in the faeces by definitive hosts.

Distribution

Echinococcus multilocularis is endemic in a wide area of central and eastern Europe.

Life Cycle

Adult worms inhabit the small intestine of the definitive hosts with the terminal proglottid breaking off once it matures. This is passed in faeces and includes eggs containing the larval stages (oncospheres), which are immediately infective to an intermediate host, normally voles (Arvicolidae). Within the intermediate host the immature tapeworm leaves the intestine, coming to rest in the liver where it develops into a multilocular cyst containing many immature tapeworms. Definitive hosts are infected when they ingest the cysts within the intermediate hosts.



Clinical Signs

Infected dogs are very unlikely to show clinical signs. The segments are too small for them to be evident in faeces.

Although extremely rare, dogs may also act as intermediate hosts and show severe clinical signs.

Diagnosis

Specific diagnosis of *Echinococcus* infections in definitive hosts is difficult because taeniid eggs cannot be differentiated morphologically and are passed intermittently. Coproantigen tests are not commercially available and polymerase chain reaction tests (PCRs) for species and/or genotype identification are only performed in specialised laboratories. Therefore, in *Echinococcus* endemic areas, taeniid infections identified by egg detection should be considered potential *Echinococcus* infections.

Treatment

Where animals are infected with *Echinococcus* species, treatment with an anthelmintic containing praziquantel is advisable, carried out under veterinary supervision[1].

Dogs should be shampooed to remove any parasite eggs adhering to the coat.

The faeces of treated dogs should be disposed of appropriately.

Personnel involved should use suitable protective clothing, including protective gloves.

Cats, in contrast to dogs, are epidemiologically insignificant as sources of egg output because they are poor hosts for this worm. However, they can sporadically acquire infection and occasionally pass eggs. In dogs, eggs are commonly found in the fur of infected animals. However to date, no eggs have been recovered from the coat of an infected cat. As there is a small risk of cats carrying an infection, treatment in high-risk situations is a reasonable precaution, for example, before entry into countries where the infection is not present.

When travelling to the UK, Malta, Ireland, Finland and Norway, dogs must be treated for this infection

Control

Dogs that may hunt and eat small prey should be treated at least every four weeks with an effective anthelmintic containing praziquantel. Animals should not be fed raw or undercooked meat unless it has been frozen at -20°C for 1–2 days, a process that inactivates most parasites in meat. Care should also be taken to prevent them hunting.

[1] See www.esccap.org for links to therapy tables by country or region.

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1 Modular Guide Series
1.3: Flea tapeworm (*Dipylidium caninum*)

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Dipylidium caninum is a tapeworm of dogs and cats.
The flea or the chewing dog louse are intermediate hosts.

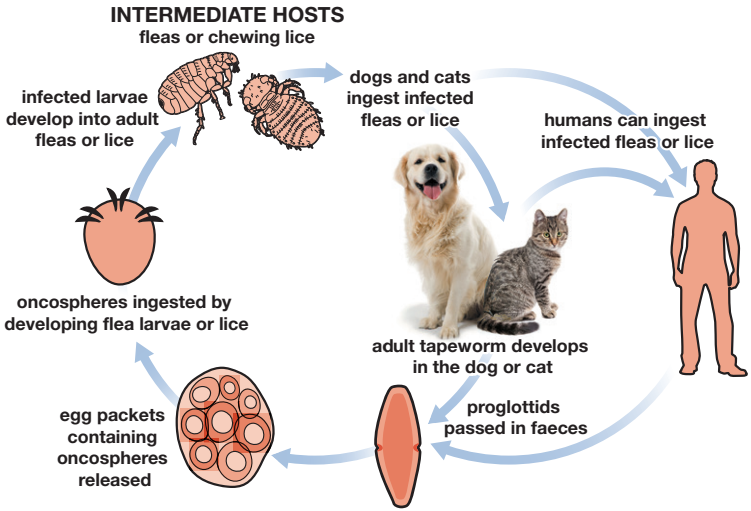
Distribution

The parasite is common throughout Europe.

Life Cycle

The intermediate hosts are fleas or chewing dog lice and dogs and cats become infected when they ingest these infected insects. The adult tapeworm develops within the small intestine of the dog or cat. *Dipylidium caninum* is zoonotic and humans can become infected if they accidentally ingest infected fleas or lice, although this is rare.

The prepatent period is approximately three weeks.



When chewing lice or fleas are ingested they can transmit *D. caninum*

Clinical Signs

Dipylidium caninum is rarely associated with clinical signs in dogs or cats, although anal pruritus may occur.

Diagnosis

The white proglottids may be seen in fresh faeces or in the coat around the anus. When dry, they resemble grains of rice and may be visible in the perianal area.

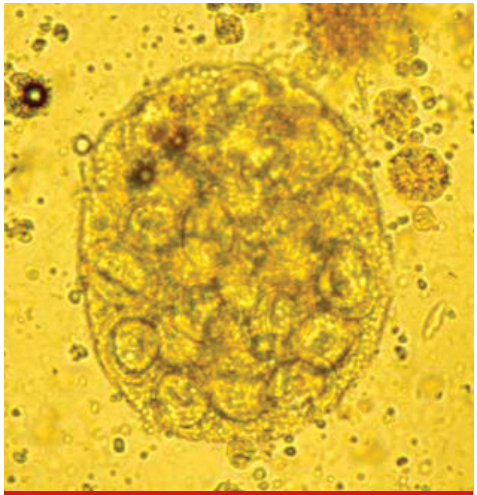
Proglottids may also sometimes be seen emerging from the anus and dried proglottids may be found in samples from the animal's bedding.

Treatment

Treatment involves the administration of an effective anthelmintic containing praziquantel at regular intervals[1].

Control

Dipylidium caninum infection can be prevented by the effective control of fleas and lice.



Egg packets containing oncospheres

Anal pruritus may occur

[1] See www.esccap.org for links to therapy tables by country or region.

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1 Modular Guide Series
1.4: Taeniid tapeworms (*Taenia* spp.)

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Taenia spp. are tapeworms that can infect dogs, cats and foxes by ingestion of intermediate hosts.

Distribution

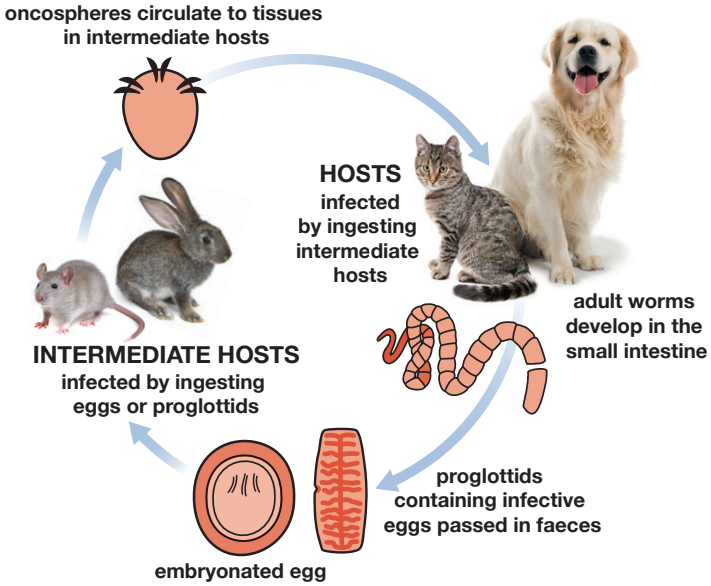
Taenia spp. are common throughout Europe.

Life Cycle

The intermediate hosts are varied and, depending on the *Taenia* spp., range from sheep and cattle (*Taenia multiceps*) to rabbits (*Taenia serialis*, *Taenia pisiformis*), rodents (*Hydatigera taeniaeformis*), ruminants and pigs (*Taenia hydatigena*) and sheep and goats (*Taenia ovis*).

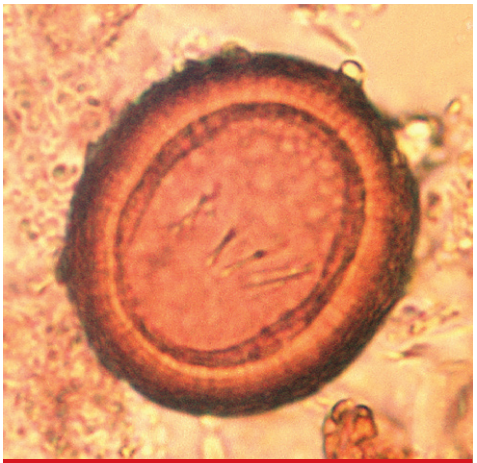
Dogs or cats become infected when they eat the tissues or viscera of infected hosts.

Adult worms can survive in the small intestine for several months up to several years.



Clinical Signs

Taenia spp. are rarely associated with clinical signs, although the mature segments of the adult worm may cause anal irritation, resulting in the animal rubbing its bottom along the ground. Owners may notice segments attached to the animal's coat after the segments have emerged from the anus.



Taeniid egg

Diagnosis

Taeniid eggs may be detected upon faecal examination.

Taenia spp. eggs cannot be differentiated microscopically from *Echinococcus* eggs. Therefore in *Echinococcus* endemic areas, taeniid infections identified by egg detection should be considered potential *Echinococcus* infections.

Macroscopic examination of the faeces may demonstrate the presence of white proglottids. Microscopically, unlike *D. caninum*, each has only one genital pore.

Treatment

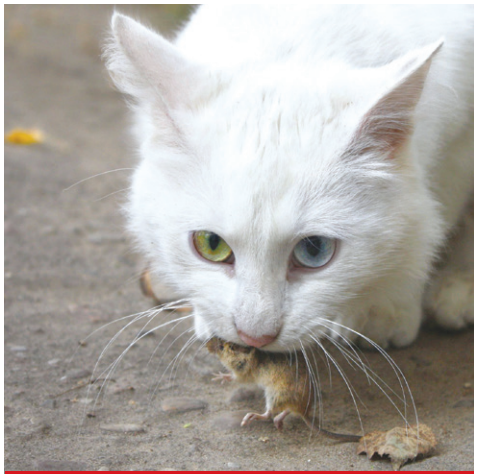
Treatment involves the administration of an effective anthelmintic at suitable intervals, which will most likely depend upon evidence of an existing infection[1].

[1] See www.esccap.org for links to therapy tables by country or region.

Control

Eggs can remain viable in the environment for prolonged periods. Owners should try and prevent dogs and cats having access to the various intermediate hosts.

The feeding of raw meat and viscera should be discouraged.



Hunting dogs and free roaming cats at higher risk of infection

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1 Modular Guide Series
1.5: Heartworm (*Dirofilaria immitis*)

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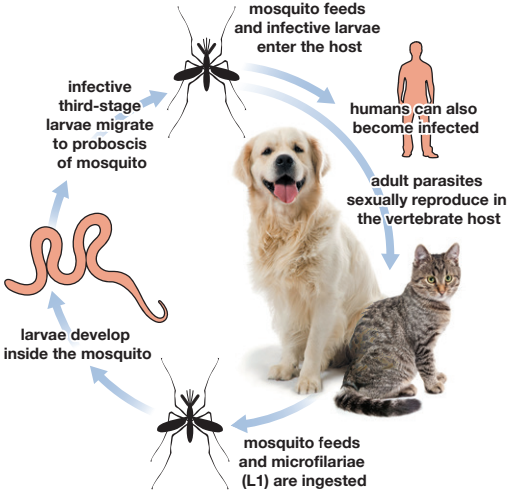
Dirofilaria immitis is a filarial worm that resides in pulmonary arteries of dogs and cats. Also known as heartworm, it is transmitted by intermediate mosquito hosts. It is zoonotic but human infection is rare.

Distribution

Dirofilaria immitis is endemic/hyperendemic in many countries of southern, central and eastern Europe. The prevalence in cats is generally only a tenth of that in dogs.

Life Cycle

Dirofilaria immitis has an indirect life cycle. Dogs and cats are the definitive hosts. The adult parasites sexually reproduce in their vertebrate hosts and the offspring, known as microfilariae, are transferred to the intermediate host, which is usually a mosquito. The larvae develop inside the mosquito and when the mosquito feeds, the infective larvae enter the canine or feline host through the puncture wound. The parasites migrate within the connective tissue for approximately 2–3 months before entering the host's bloodstream and reaching the pulmonary arteries. Mature females release offspring 6–7 months post infection, which become available to blood-sucking mosquitoes.



Heartworms are transmitted by different types of mosquitoes

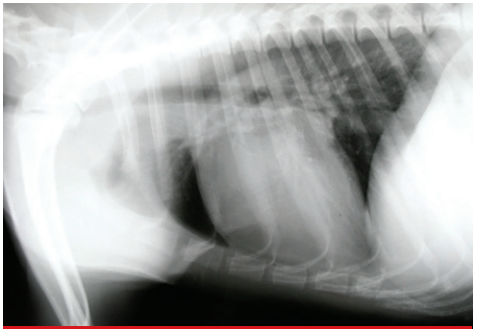
Adult worms live in the pulmonary arteries

Clinical Signs

Infection with *D. immitis* may cause severe and potentially fatal disease in dogs and cats.

In dogs, low worm burdens can be asymptomatic. Increasing worm burdens can cause clinical signs such as loss of condition, weakness, dyspnoea and chronic cough. If untreated, the disease can progress to rightside heart failure and death.

In cats, the disease is often asymptomatic until severe clinical signs (i.e. HARD, heartworm-associated respiratory disease) or sudden death occur.



Heartworm infection causes pulmonary disease

Diagnosis

Diagnosis of *D. immitis* infection is based on blood tests to detect microfilariae and serological tests to detect circulating antigens (in dogs) or antibodies (especially in cats).

Treatment

The organic arsenical compound melarsomine dihydrochloride (2.5 mg/kg bodyweight) is the only effective drug available for treating adult heartworm infections in dogs. The recommended protocol is one deep intramuscular injection, followed by two doses administered at 24-hour intervals at least one month later[1].

Control

Control of heartworm in dogs and cats relies on the use of preventive treatments to kill immature heartworm stages before they migrate to the pulmonary arteries. Monthly administration of topical or oral macrocyclic lactones throughout the transmission season, usually April to November, is effective. Slow-release formulations are available that provide protection for 6 to 12 months[2].

[1] See www.esccap.org for links to therapy tables by country or region.

[2] For more information see: ESCCAP Guideline 05: Control of Vector-Borne Diseases in Dogs and Cats.

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1 Modular Guide Series
1.6: Subcutaneous worm (*Dirofilaria repens*)

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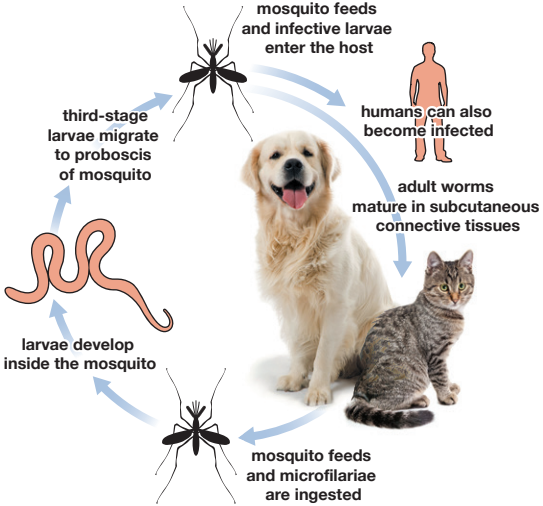
Dirofilaria repens is a subcutaneous filarial worm found in dogs and cats. It is transmitted via intermediate mosquito hosts. Humans can also become infected.

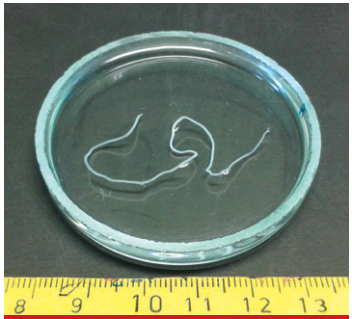
Distribution

In many regions of Europe, areas where *D. repens* is endemic overlap with those where *D. immitis* is also established. *D. repens* is the main species occurring in areas such as northern France and Hungary and is the most important *Dirofilaria* species responsible for zoonotic infections in Europe. Recently, *D. repens* infections have been documented in dogs with no travel history in Germany, the Netherlands, Austria, Portugal, Spain, Estonia and Poland.

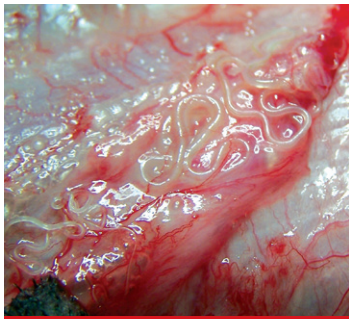
Life Cycle

The microfilariae develop in the uterus of female worms and are released into the bloodstream where they become available to the vectors, which are blood-sucking mosquitoes. Further development occurs within the vector and the infective larvae are transmitted to the definitive host through the vector's saliva during feeding. The adult worm then matures in the subcutaneous connective tissue.



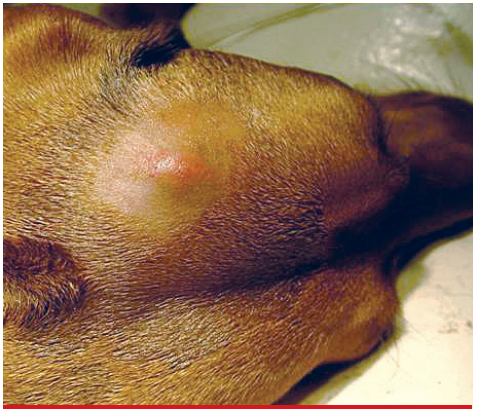


The parasite is approximately 7–12 cm long


Dirofilaria repens resides in the subcutaneous tissue

Clinical Signs

Dirofilaria repens is the species most frequently associated with subcutaneous filariosis in dogs and cats. Most infections are asymptomatic but in some cases, subcutaneous, non-inflammatory nodules containing adult parasites or microfilariae can be observed. Most infected dogs do not show clinical signs for years. In cases of heavy infection, severe dermatitis may occur.



The worm may cause skin nodules and swelling

Diagnosis

In dogs, blood tests can demonstrate the presence of microfilariae and reference should be made to ESCCAP Guidelines 4 and 5[1] for a range of diagnostic options that may be appropriate. In cats, detection of microfilariae in the blood is unlikely to be successful because the density of the microfilariae in the circulation is very low.

Treatment

The combination of moxidectin/imidacloprid is licenced in the EU for adulticidal treatment of *D. repens*. Because of its zoonotic potential, microfilaraemic dogs should be treated monthly for one year with products that are effective against microfilariae. Subcutaneous filariosis can be safely and effectively prevented in both dogs and cats through chemoprophylactic treatment. Monthly treatment with macrocyclic lactones (oral or spot-on formulations) is effective in preventing subcutaneous infection in dogs[2,3].

Control

In Europe, *D. repens* is the most important agent responsible for human filarial infection so control in dogs and cats is essential.

Before and after travelling, dogs and cats should be examined for *D. repens* infection by testing for microfilariae. If microfilariae are detected in a blood sample, dogs and cats should not travel to non-endemic areas without prior microfilaricidal treatment.

Treatment with an appropriate prophylactic will provide protection on entry into an endemic area.

For more information see:

[1] ESCCAP Guideline 04: Parasitological Diagnosis in Cats, Dogs and Equines.
ESCCAP Guideline 05: Control of Vector-Borne Diseases in Dogs and Cats.

[2] ESCCAP Guideline 01: Worm Control in Dogs and Cats.

[3] See www.esccap.org for links to therapy tables by country or region.

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1 Modular Guide Series
1.7: French heartworm (*Angiostrongylus vasorum*)

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Angiostrongylus vasorum (French heartworm) is a nematode that, in its adult stage, resides in the pulmonary arteries and right ventricle of dogs, foxes and some other carnivores. Cats are not affected.

Distribution

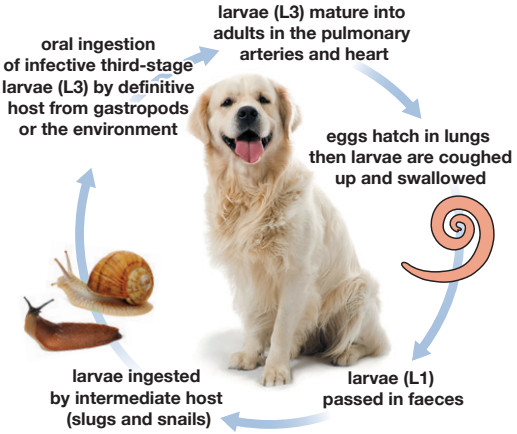
Angiostrongylus vasorum is prevalent in various European countries including the UK, Ireland, Portugal, Spain, France, Switzerland, the Netherlands, Belgium, Denmark, Germany, Italy, Hungary, Slovakia, Poland and Sweden.

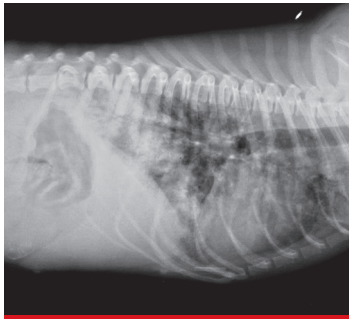
Foxes are important reservoir hosts and the distribution of infection in dogs may, to some extent, mirror that seen in foxes.

Life Cycle

Slugs and snails serve as intermediate hosts. Dogs may also become infected by ingesting frogs, which act as paratenic hosts. Following ingestion, larvae develop and migrate to the right ventricle and pulmonary arteries. Female worms begin laying eggs from 6–8 weeks after infection. Eggs hatch rapidly and larvae penetrate the alveoli before being coughed up and passed in the faeces as first-stage larvae.

Once an infection is established, patency may be very long; up to five years.





A lateral radiographic view of an infected dog

A. vasorum **first-stage larvae measure approximately 345 µm and are characterised by a wavy tail with a dorsal notch[A]**

[A] Photo courtesy Rolf Nijsse, ESCCAP Benelux.

Clinical Signs

Early or light infection	No clinical signs
Significant infection	Heavy productive cough Dyspnoea Anaemia Depression Anorexia Signs of coagulopathy
Severe infection	Right-sided heart failure Sudden death Neurological signs
Chronic infection	Verminous pneumonia leading to anorexia, weight loss, bleeding, emaciation and pulmonary hypertension, coagulopathy with bleeding, neuropathies associated with haematoma in the CNS.
Ectopic infection	Occasionally, larvae and rarely adult stages of *A. vasorum* are located in ectopic locations such as the brain, bladder, kidney or the anterior chamber of the eye. Clinical signs relating to invasion of these organs can occur.

[1] See www.esccap.org for links to therapy tables by country or region.

Diagnosis

Live larvae can be detected in at least 4 g (5–10 g) of fresh faeces using the Baermann method. Due to substantial daily variation in larval excretion, faecal samples should be collected from three consecutive defecations over 1–2 days. Alternatively, larvae can be detected microscopically in bronchial lavage material. A commercial serological test for detection of the circulating antigen is also available.

Treatment

Anthelmintic therapy includes the use of adequate macrocyclic lactone-based anthelmintics (some of which require repeated administration) or daily administration of benzimidazole-based anthelmintics for three weeks[1]. Supportive treatment i.e. glucocorticoid-based products and fluid substitution therapy, may be needed in severe clinical cases and the animal should be rested during the treatment period (at least 2–3 days).

Control

The prophylactic use of adequate macrocyclic lactone-based products has been shown to be effective.

When possible, dogs should be prevented from ingesting slugs or snails.

Apparatus used for the Baermann method

Faecal samples soaked overnight in gauze

Sediment removed and examined microscopically

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1 Modular Guide Series
1.8a: The fox lungworm (*Crenosoma vulpis*)

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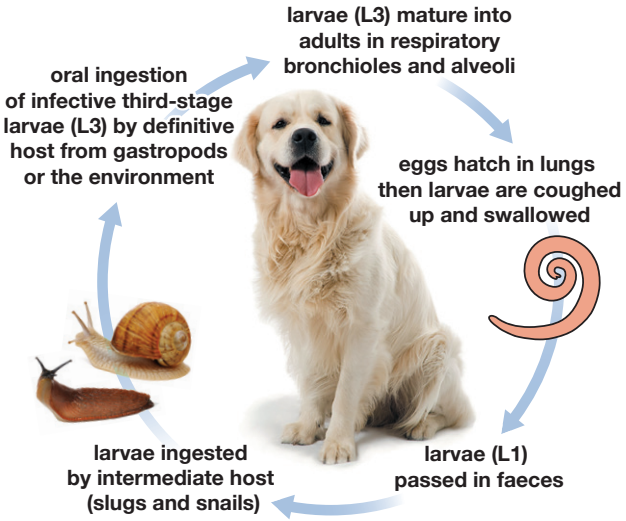
Crenosoma vulpis is common in dogs in many European countries. The adult worms reside in the trachea and bronchi.

Distribution

Crenosoma vulpis is endemic in wild canid populations in Europe, with red foxes serving as reservoir hosts.

Life cycle

The life cycle is similar to that of other metastrongyloids, such as *A. vasorum*, with slugs and snails acting as intermediate hosts. Infection may occur through the oral ingestion of infective L3 in gastropods but also through the ingestion of L3 larvae released into the environment by infected slugs and snails. Adult *C. vulpis* nematodes are 4–15 mm in length. The female is ovo-viviparous, i.e. first-stage larvae (L1) develop quickly within a thin eggshell and hatch within the host before being coughed up and excreted in faeces. The prepatent period is approximately three weeks.



L1 from fresh faeces isolated by the Baermann method[B]

The adult *C. vulpis* reside in the trachea and bronchi[B]

Clinical signs

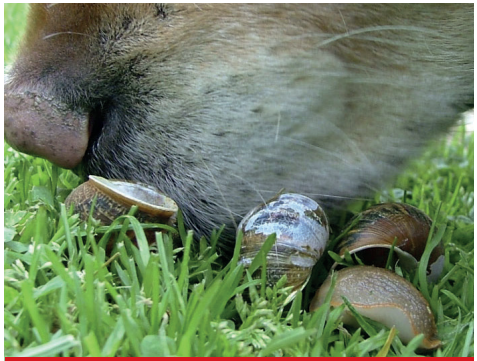
Infected dogs may develop catarrhal bronchitis with eosinophilia and a broncho-interstitial pattern. The main clinical signs are coughing, tachypnoea, dyspnoea and sneezing, accompanied by non-specific signs such as a poor general condition and fever.

Diagnosis

Diagnosis is based on detecting L1 in at least 4 g (5–10 g) of fresh faeces using the Baermann method. Due to substantial variation in larval excretion, faecal samples should be collected from three consecutive defecations over 1–2 days. Alternatively, L1 can be detected microscopically in bronchial lavage material and subsequently confirmed using genetic methods.

Treatment

Several anthelmintic compounds can be used to treat *Crenosoma* infections e.g. macrocyclic lactones as well as fenbendazole for 3–5 consecutive days.



Slugs and snails act as intermediate hosts

Control

Preventing infection involves controlling intermediate hosts by removing gastropods from yards and ensuring dogs do not eat slugs or snails.

[B] Photos courtesy John McGarry, ESCCAP UK & Ireland.

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1 Modular Guide Series
1.8b: Cat lungworm (*Aelurostrongylus abstrusus*)

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Aelurostrongylus abstrusus is the most prevalent lungworm in domestic cats. It is considered to be species-specific and is commonly referred to as the "cat lungworm".

Distribution

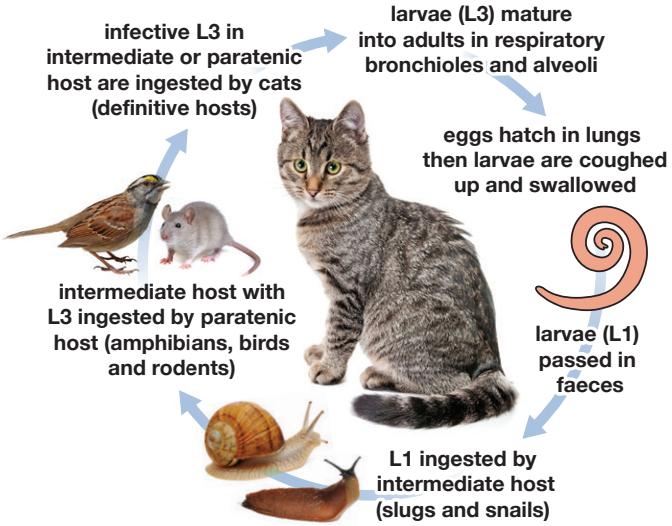
Aelurostrongylus abstrusus is a metastrongylid nematode that, in its adult stage, inhabits the respiratory bronchioles, alveolar ducts and alveoli. It may also be found in subpleural nodules in the lung parenchyma. *A. abstrusus* can measure up to 10 mm in length and is very thin (less than 100 µm). In Europe, *A. abstrusus* is endemic, with a reported prevalence of up to 30% in some countries.

A. abstrusus poses a risk to all domestic cats with regular outdoor access, irrespective of their age and gender. Feral and stray cats are at a higher risk of infection.

Life cycle

The life cycle of *A. abstrusus* includes some mollusc species (slugs and snails) as intermediate hosts.

Amphibians, reptiles, birds and rodents act as paratenic hosts after ingesting molluscs infected with infective third-stage larvae (L3). Cats become infected by ingesting L3 in paratenic hosts or by directly ingesting infected molluscs, i.e. during grooming. The prepatent period is 5–6 weeks.



L1 from fresh faeces isolated by the Baermann method[C]

Adult worms within the lung tissue[B]

Clinical signs

A. abstrusus infections most frequently manifest as mild to intense coughing, sneezing, tachypnoea, open-mouthed abdominal breathing, mucopurulent nasal discharge and fatigue. Cats may also develop subclinical infections. In severe cases, signs of bronchitis and pneumonia may occur. If left untreated, the infection can be fatal. In particular, cats undergoing anaesthesia are at increased risk of death.

Diagnosis

Diagnosis relies upon detecting L1 in at least 10 g of fresh faeces using the Baermann method. Diagnostic sensitivity based on larval detection decreases in cases of chronic or repeated infections, due to interrupted larval shedding. In cases of suspected pulmonary inflammation, imaging (thoracic radiography, computed tomography) should be performed as a preliminary and complementary investigation.

Treatment

Treatment options include the use of macrocyclic lactones and emodepside-containing preparations, some of which require repeated administration.

Control

In highly endemic local areas and when there is a high risk of *A. abstrusus* infection, for example, if the cat preys on rodents or birds or eats slugs or snails, preventive treatment with monthly administration of macrocyclic lactones or emodepside is advised. Appropriate prevention reduces the risk of chronic pulmonary changes resulting from undiagnosed aelurostrongylosis.

[B] Photo courtesy John McGarry, ESCCAP UK & Ireland.

[C] Photo courtesy Anja Joachim, ESCCAP Germany & Austria.

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1 Modular Guide Series
1.9: Hookworms (*Ancylostoma* spp. and *Uncinaria* spp.)

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Hookworms are nematodes of the small intestine that can cause disease in dogs, cats and foxes.

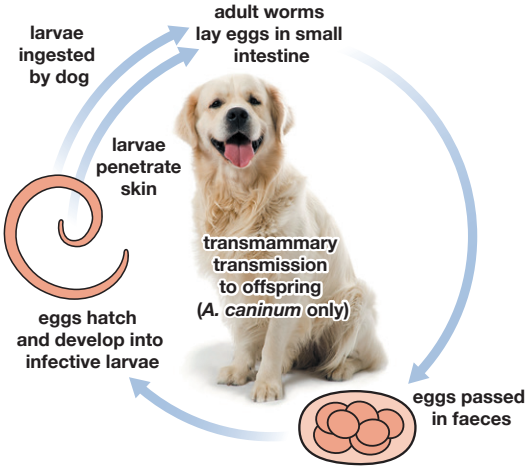
Their common name is derived from their large mouthparts, which are set at an angle to the rest of the worm. All species feed by grasping and removing plugs of intestinal mucosa with their mouthparts, thereby damaging the mucosal surface. Infection is most common where animals have access to outdoor environments such as runs and kennels.

Distribution

There are three significant species in Europe: *Ancylostoma caninum* (dogs), *Ancylostoma tubaeforme* (cats) and *Uncinaria stenocephala* (dogs and rarely cats). *Ancylostoma caninum* is predominantly found in central and southern Europe, while *A. tubaeforme* is found throughout continental Europe. *Uncinaria stenocephala* is known as the northern hookworm because it tolerates colder climates and is found throughout Europe.

Life Cycle

Adult worms inhabit the small intestine where they lay eggs that are subsequently passed in the faeces.



Pups can become infected while suckling (*A. caninum* only)

Hookworms are small nematodes that live in the intestine of infected dogs and cats

The eggs hatch, releasing larvae that develop to infective third-stage larvae in the environment. These larvae are then ingested and develop into adult worms within 2–4 weeks.

Hookworms, most notably *Ancylostoma* spp. larvae, are also capable of penetrating the skin and migrating to the intestine. However, it is unlikely that this route of infection contributes significantly to the life cycle of *U. stenocephala*.

Suckling pups can become infected with *A. caninum* through the lactogenic transmission of larvae.

Clinical Signs

Diarrhoea, weight loss and anaemia are common clinical signs. In cases of *A. caninum* and *A. tubaeforme* infection, the diarrhoea may contain blood.

Skin lesions can appear on the foot pads of dogs and cats caused by larvae burrowing into and along the skin.

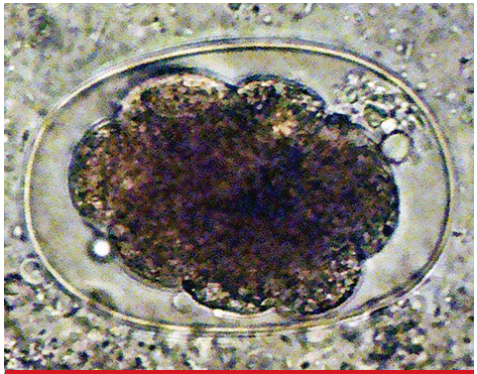
Transmission of *A. caninum* larvae through the milk can result in acute anaemia, which may prove fatal in young pups.

Diagnosis

Diagnosis is based on identifying hookworm eggs in fresh or fixed faecal samples using a flotation method. Diagnosis in young pups can be complicated by the onset of clinical signs before the infection becomes patent i.e. before eggs are passed in the faeces. Commercial coproantigen detection tests (ELISA) are also available.

Treatment

Immunity does develop after exposure but is unlikely to be absolute, therefore animals in heavily infected environments may require regular anthelmintic therapy to control hookworm infections. Where young animals are clinically affected by the infection, supportive therapy may be necessary in addition to anthelmintic treatment[1].



Infection can be diagnosed by faecal examination and identification of eggs

Control

A sustained programme of anthelmintic treatment and environmental management (hygiene) should be implemented for dogs and cats with access to contaminated areas such as runs and kennels. Potential anthelmintic resistance, particularly in the USA and Australia, may necessitate treatment with anthelmintics of demonstrated efficacy, alongside temporarily relocating animals to a clean environment while the affected area is decontaminated.

[1] See www.esccap.org for links to therapy tables by country or region.

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1 Modular Guide Series
2.0: Whipworm (*Trichuris vulpis*)

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Trichuris vulpis (whipworm) is an intestinal nematode that can cause disease in dogs.

Distribution

Infection with *Trichuris vulpis* occurs throughout Europe but is most likely to occur in central and southern Europe where temperatures are most suitable for the environmental development of eggs (no development occurs below 4°C).

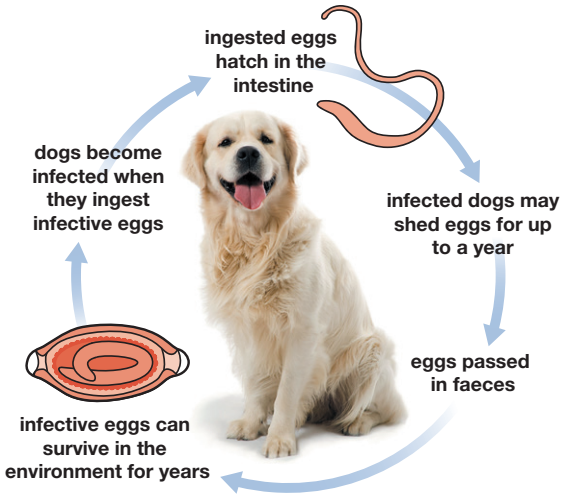
Life Cycle

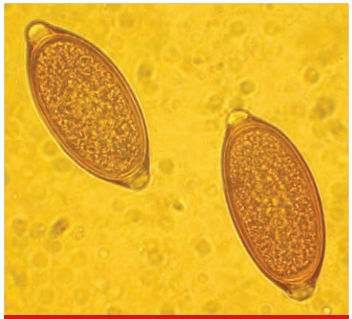
Eggs are passed in the faeces of infected dogs. The infective first-stage larvae (L1) develop within the eggs in 1–2 months. These infective stages can survive in the environment for years. Dogs become infected when they ingest infective eggs. The prepatent period is 9–10 weeks and infected dogs may continue to shed eggs for up to a year.

Clinical Signs

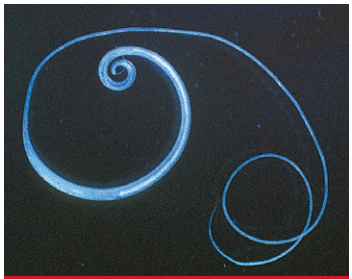
Heavy infection will result in diarrhoeic, bloody, mucoid faeces accompanied by weight loss.

Ultimately, the animal will no longer be able to compensate and will become acutely ill. Metabolic disturbances, including anaemia and hyponatraemia, may also occur.





***Trichuris vulpis* eggs**[D]



***Trichuris vulpis* worm**

[D] Photo courtesy Jakub Gawor, ESCCAP Poland.

Diagnosis

Infection can be diagnosed by detecting characteristic “lemon-shaped” eggs in 4–10 g of faeces using a suitable flotation technique.

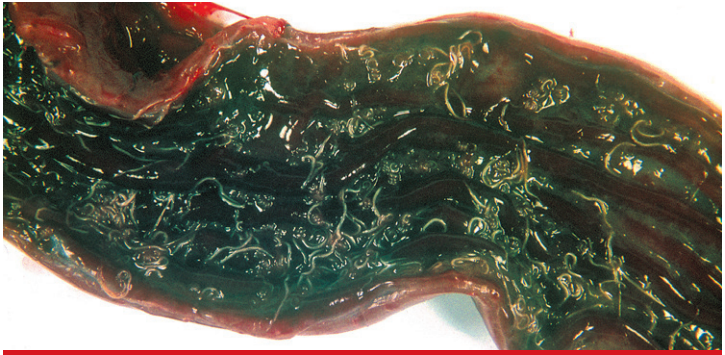
Treatment

Most modern anthelmintics are effective against *T. vulpis*. To be effective, repeated deworming is often required[1].

Control

Clinical cases tend to occur in certain localised geographical areas or on specific premises, such as kennels. Considerable and persistent environmental contamination is common, making control difficult because dogs can easily become re-infected if they remain in the same environment.

Where possible, dogs should be removed from contaminated areas. Since the eggs are difficult to eliminate from the environment, it may be necessary to consider resurfacing kennel flooring (e.g. by paving or laying concrete) to facilitate thorough cleaning. Rotavating and reseeding may also help to reduce environmental contamination.



A heavy infection of *Trichuris vulpis* in the large intestine of a dog

Increased risk of heavy infection in kennels with earth or straw runs

This kennel can be easily cleaned which reduces the risk of infection

[1] See www.esccap.org for links to therapy tables by country or region.

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ESCCAP Secretariat
Malvern Hills Science Park
Geraldine Road, Malvern
WR14 3SZ United Kingdom

Tel: +44 (0) 1684 585135
www.esccap.org

1 Modular Guide Series
2.1: Threadworm (*Strongyloides stercoralis*)

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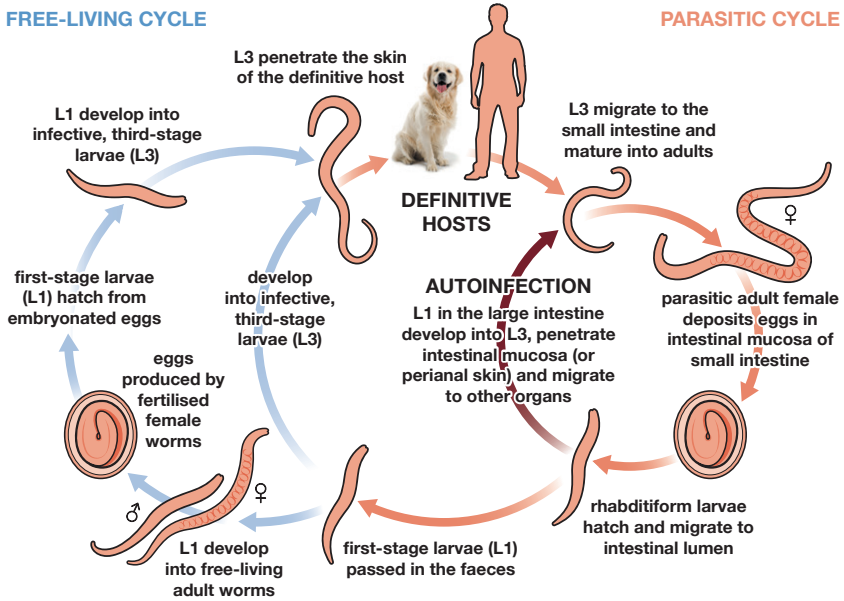
Strongyloides stercoralis is a worldwide zoonotic nematode of canids and primates. The slender adult worms (3–8 mm in length) reside in the small intestine.

Distribution

It is most commonly found in tropical and subtropical regions but may also be diagnosed in temperate regions and in Northern Europe. Cats become infected with *S. stercoralis* less commonly than dogs.

Life cycle

In its complex life cycle, only female worms are parasitic. Adult females are embedded in the intestinal mucosa and reproduce asexually by parthenogenesis, releasing eggs into the gut lumen. These eggs may hatch rapidly and the resulting L1 (rhabditiform larvae) are then shed in the faeces. Within 24 hours, L1 may develop into L3 in the soil or mature into non-parasitic male and female worms capable of reproducing sexually in the environment. This process can lead to extensive multiplication and contamination of the surrounding environment. L3 larvae infect the host via percutaneous, oral or lactogenic transmission. Autoinfection, whereby L1 develop into L3 within the same dog, seldom occurs.



Clinical signs

Strongyloides infections are often moderate and subclinical, with disease occurring mainly in neonates and nurslings with a heavy infection. Severe infections in sick dogs can lead to pneumonia and watery to mucoid diarrhoea. Emaciation is often evident and decreased growth rate may be one of the earliest clinical signs. Appetite is usually good and the dog (or cat) is normally active during the early stages of the disease.

Diagnosis

Diagnosis of *S. stercoralis* in dogs relies upon the detection of L1 in fresh faeces by the Baermann method. Larvae may only occasionally be observed by flotation. Specificity is linked to the morphological identification of L1 and differentiation from other parasitic or free-living species. Faecal PCR assays are also available from some diagnostic laboratories.

Breeding kennels and the animal trade appear to play an important epidemiological role in the dissemination of *S. stercoralis*. Since routine faecal flotation methods have low sensitivity for detecting L1, and egg shedding is uncommon, the prevalence and clinical significance of *S. stercoralis* in dogs may be underestimated.

Treatment

There are no approved regimens for either cats or dogs. Infections in dogs can be tentatively treated with ivermectin (off-label, definitively not recommended in ivermectin-sensitive dogs) or fenbendazole. In isolated cases, higher doses, repeated treatments or a combination of anthelmintics is required. In cats, fenbendazole can be administered.

Control

Infections are usually associated with warm, wet, crowded, insanitary housing. Poor sanitation and mixing susceptible and infected dogs can lead to a rapid build-up of infection in all dogs in a kennel or pen. Dogs with diarrhoea should be promptly isolated from those that appear healthy. Direct sunlight, increased soil or surface temperatures and desiccation are deleterious to all free larval stages.

Thorough washing of wooden and impervious surfaces with steam or concentrated salt or lime solutions, followed by rinsing with hot water, effectively destroys the parasite.

Zoonotic populations of *S. stercoralis* may exist. The disease in humans can be serious, so caution should be exercised when handling infected dogs.

L1 detected in fresh faeces by the Baermann method[E]

Shared kennel accommodation may facilitate rapid transmission of infection

[E] Photo courtesy Barbara Hinney, University of Veterinary Medicine Vienna.

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