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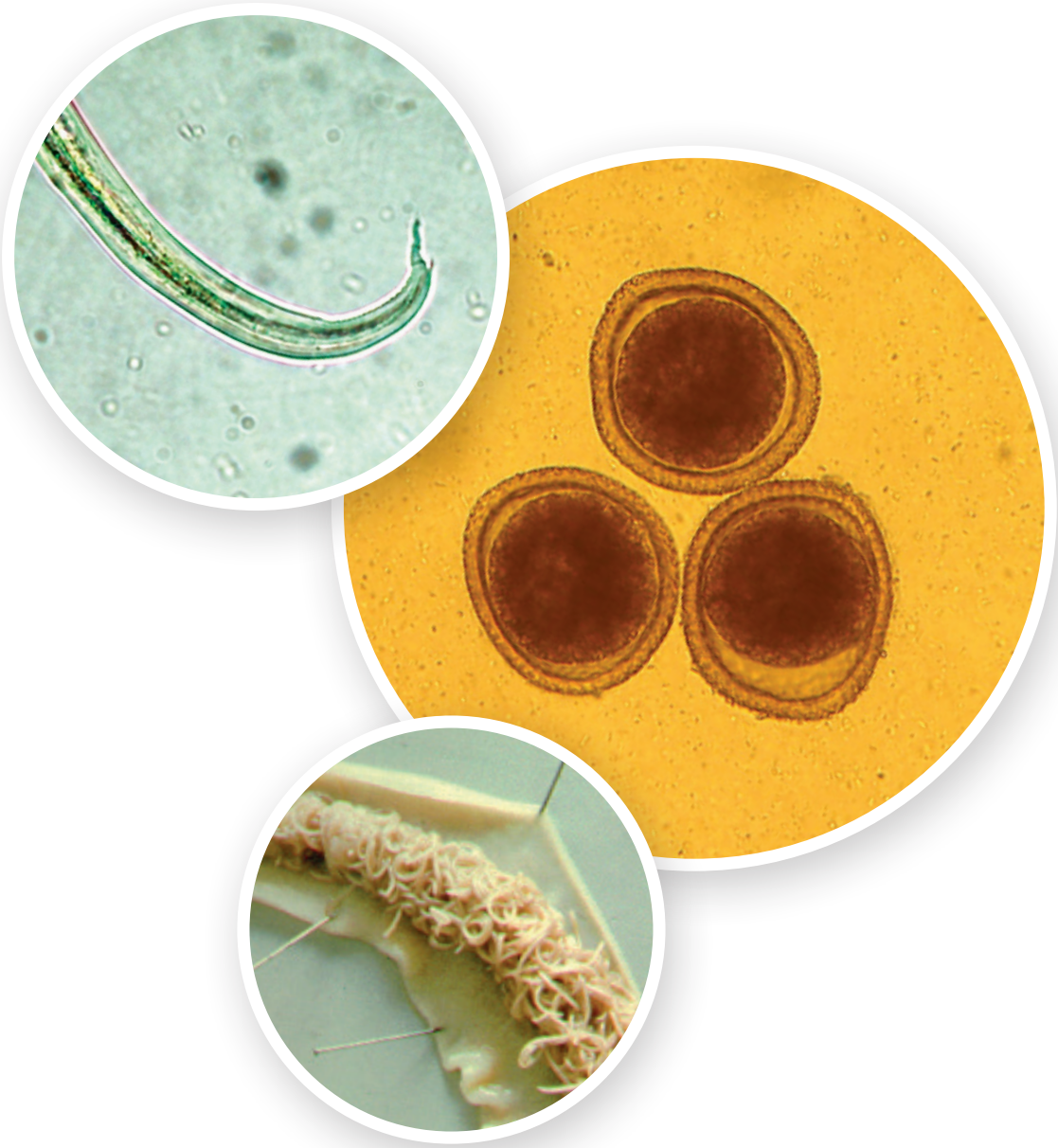
EUROPEAN SCIENTIFIC COUNSEL COMPANION ANIMAL PARASITES®



1 Worm Control in Dogs and Cats

ESCCAP Guideline 01 Seventh Edition – June 2025

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1 Worm Control in Dogs and Cats

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INTRODUCTION

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 (*Toxocara* spp., *Toxascaris leonina*)
- Tapeworms
- Hookworms (*Ancylostoma* and *Uncinaria* spp.)
- Whipworm (*Trichuris vulpis*)
- Threadworm (*Strongyloides stercoralis*)

Non-intestinal worms

- Heartworm (*Dirofilaria immitis*)
- Subcutaneous worm (*Dirofilaria repens*)
- French heartworm (*Angiostrongylus vasorum*[†])
- Lungworms (*Crenosoma vulpis*, *Aelurostrongylus abstrusus*)
- Eye worms (*Thelazia callipaeda*)

These groups are further summarised in Tables 2A, 2B and 2C. Factors affecting the importance of these worms include:

- Prevalence
- Pathogenicity for the host
- Zoonotic potential
- A combination of these factors

This guideline aims to give an overview of these worms and their significance and to suggest control measures for the most important species in order to prevent animal and/or human infection.

For simplicity, the nematodes, cestodes and trematodes mentioned in this guideline will be referred to as "worms", therapeutic compounds as "anthelmintics" and treatments as "dewormings".

[†] *A. vasorum* is also commonly named 'the French Heartworm', to differentiate from *Dirofilaria immitis*: the adults of both parasites are located in the pulmonary arteries and the right heart. However, due to its taxonomic grouping with other metastrongyloid lungworms and due to first-stage larvae in the lungs causing verminous pneumonia, *A. vasorum* is sometimes referred to as a lungworm.

SCOPE

ESCCAP provides research-based, independent advice. It is the aim of ESCCAP to produce a guideline which delivers comprehensive information and support to assist both veterinarians and pet owners to successfully control worm infection in dogs and cats. This guideline concentrates on the most important groups of companion animal worms, both intestinal and non-intestinal. Other canine and feline parasites are addressed in other guidelines; these will be referred to, where appropriate, in the text. For more information on the control of ectoparasites, superficial mycoses, vector-borne diseases and intestinal protozoa, see ESCCAP guidelines at www.esccap.org/guidelines/.

PRESENT SITUATION AND EMERGING THREATS

In Europe, an increase in pet travel plus climatic changes will probably influence the present epidemiological situation of certain endoparasites or may introduce them into different regions. Rare diseases may rise in frequency due to increased importation into presently non-endemic areas. Furthermore, within the European Union, removal of border controls under the Schengen Treaty and implementation of the (pre-Brexit) PETS Travel Scheme in the United Kingdom have led to easy travel between the various countries within continental Europe and, except for the UK, there are no or limited customs controls of pet animals moving from one country to another. Whilst pets travelling with their owners account for the majority of pet movement, a large number of dogs and, to a lesser extent cats, are now being relocated by welfare organisations from, for example, Mediterranean countries to private households all over Europe. This is particularly significant as the Mediterranean is an area where parasites such as *Dirofilaria immitis* are highly prevalent.

Veterinary medicinal products go through a rigorous testing process prior to their approval by European or national authorities and each indication for use has to be scientifically justified. Veterinarians are trained in the appropriate use of these compounds according to current national legislation. Most modern endoparasiticidal compounds for companion animals can be used prophylactically or therapeutically to control endoparasites.

LIFELONG CONTROL OF COMMON WORMS

Parasite infections should be controlled through endoparasite and ectoparasite management and treatment. Few parasite infections are strictly age-related; the risk continues as the animal ages and so consideration should be given to provide each dog and cat with appropriate worm control throughout its lifetime. The routine treatment and prevention of worms depend upon legislation in individual countries. Veterinary professionals will consider local epidemiological circumstances, owner perception and individual risk assessments i.e. hunting pets, previous lungworm exposure, raw meat diets etc. The zoonotic potential and its consequences must also be assessed. **Deworming practices should therefore always be on the advice of a veterinary professional.** See Figures 1 and 2: Schemes for individual worm management in dogs and cats.

Please be advised that:

- In countries or regions where routine treatments are not acceptable for legislative or other reasons, regular faecal examinations are recommended. See specific parasite sections within this guideline for more tailored treatment and control recommendations.
- Feeding commercial diets or cooked food (internal temperature of at least 65°C for 10 minutes) or deep frozen (at least for one week at -17 to -20°C) will prevent raw meat-transmitted parasite infections (see Tables 3 and 5).
- Dogs and cats should not be allowed access to rodents, carcasses, placentae or aborted foetuses of cattle or sheep.
- Coprophagy in dogs and cats should be prevented to avoid aberrant host infection (alveolar echinococcosis).
- Dogs and cats should always be provided with fresh, potable water (drinking from puddles should not be allowed).

Where a specific worm infection is diagnosed, the infection should be appropriately treated and then preventive measures put in place. Symptomatic dogs or cats should have a physical examination (including relevant parasitic diagnostic procedures) and complete history considered, as these are crucial for the diagnosis, treatment and control of parasitic infections.

For healthy dogs and cats, the prevention of worm infections is essential. To simplify preventive measures, ESCCAP has identified three "key" parasite groups that can cause severe disease, pose a zoonotic risk and/or have high prevalence in some or all areas of Europe:

- Ascarids (*Toxocara* spp., *Toxascaris leonina*) (prevalent in all areas)
- *Echinococcus* spp. (see Figures 9 and 10 for distribution)
- Heartworms (*Dirofilaria immitis* see Figure 18 for distribution; *Angiostrongylus vasorum* occurs Europe-wide in endemic spots).

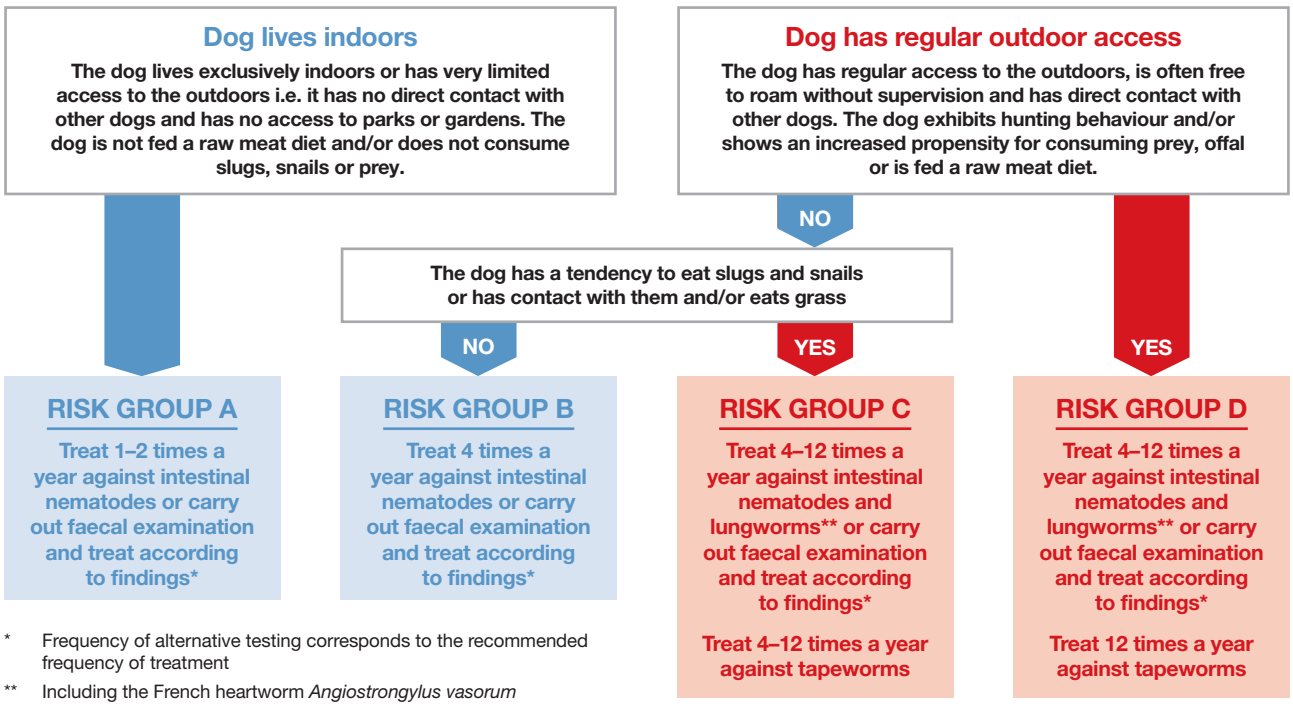
Ascarid infections occur across Europe, whilst the distribution of other infections is geographically related. By adding *Echinococcus* spp. and/or *D. immitis*/*A. vasorum* control to ascarid control measures, basic control plans can be produced for dogs and cats anywhere in Europe.

- In areas endemic for *Echinococcus multilocularis*, dogs that may hunt and eat small prey should be treated monthly with a product effective against this parasite.
- In areas endemic for *Echinococcus granulosus*, dogs with access to offal or livestock carcasses should be treated with a product effective against this parasite at least every six weeks.
- In areas endemic for *Dirofilaria* spp., administration of a monthly preventive or a long-acting injectable preventive during the vector season is recommended. In areas endemic for *Angiostrongylus vasorum*, regular diagnostic controls or monthly anthelmintic treatments against this parasite prevent the onset of important clinical signs.
- In areas where only *Toxocara* spp. is a concern, deworming at least four times a year is recommended if dogs and cats are housed outside or have access to the outdoors.

Control of other parasites, such as hookworms, whipworms, lungworms and other tapeworm species can be added as necessary. Appropriate anthelmintic treatment for all parasites can be identified and the animals treated at suitable intervals.

Responsible ownership of cats and dogs includes regular health controls with faecal diagnostics and other tests (i.e. serology) and deworming. Anthelmintic efficacy testing may be considered.

More detailed considerations for each of the companion animal parasites can be found in the individual parasite sections.



* Frequency of alternative testing corresponds to the recommended frequency of treatment

** Including the French heartworm *Angiostrongylus vasorum*

ADDITIONAL TREATMENTS FOR DOGS	
Roundworms	
Puppies	For the first time on the 14th day after birth, then every 2 weeks until 2 weeks after weaning. If there is an ongoing increased risk of infection (e.g. puppy playgroups), monthly treatments up to six months of age.
Pregnant bitches	To reduce transmission to the puppies, pregnant females can be given macrocyclic lactones around the 40th and 55th day of pregnancy or fenbendazole daily from the 40th day of pregnancy until the 2nd day after delivery.
Lactating bitches	Deworm at the first treatment of puppies (2 weeks after delivery).
Dogs with increased risk of infection i.e. those used in sport, competitions, shows or those kept in kennels etc.	Two treatments: a maximum of 2 weeks before and 2 weeks after the event. For kennels: use planned deworming once a month or examine faecal samples every four weeks and treat according to findings.
Professional dogs i.e. therapy, rescue or police dogs	Depending on the risk assessment, use planned deworming once a month or, when exposure to *Echinococcus* infection is low, examine faecal samples once a month and treat according to findings.
Dogs sharing homes with small children (below 5–6 years), immunocompromised or elderly individuals	Depending on the risk assessment, use planned deworming once a month or examine faecal samples once a month and treat according to findings.
Lungworms/French heartworm	
Dog has a tendency to eat slugs and/or snails or has contact with them, eats grass	Depending on an individual risk assessment e.g. based on the intensity of slug/snail uptake and the epidemiological situation, monthly preventive treatment may be required.
Tapeworms	
Travel or importation into/from areas endemic for *Echinococcus* spp.	Deworm dogs with a high risk of infection 4 weeks after start of travelling and then every 4 weeks, with the last deworming no later than 4 weeks after return. Immediate deworming after importation.
Eats raw meat and/or offal, eats prey	Dogs that are fed raw meat that has not been sufficiently heated (10 minutes, core temperature 65°C) or frozen (one week, -17 to -20°C) should be treated for tapeworms every 4 weeks.
Flea or chewing lice infestation (as a vector for *Dipylidium*)	Once when the infestation is established.
Heartworm (*Dirofilaria immitis*)[1]	
Dogs living in areas endemic for heartworm (see Figure 18)	Treatment against transmitted third-stage larvae with macrocyclic lactones at monthly intervals (or according to the corresponding package insert) during the mosquito season and for a 30-day period after the end of the mosquito season.
Travelling to areas endemic for heartworm	During the mosquito season, prophylactic treatment against transmitted third-stage larvae with macrocyclic lactones within 30 days of arrival into the endemic area, followed by further treatments at monthly intervals until 30 days after return.
Importation from areas endemic for heartworm	Immediately after importation, one-off prophylactic treatment against third-stage larvae and microfilariae with macrocyclic lactones. Preliminary examination for any existing infection at time of importation and retest earliest 6 months later.

- Deworming practices should always be on the advice of a veterinary professional. For intestinal nematodes and lungworms, regular coprological examination of faeces (eventually with subsequent deworming) can be an alternative to standard deworming advice if performed at the same frequency as the suggested treatments.
- If an animal's individual risk of infection with intestinal nematodes cannot be clearly assessed, the dog should be dewormed or faeces examined at least 4 times a year. The same applies in principle to tapeworm infection, although the reliability of detecting tapeworm infections using faecal sample tests is low (with the exception of *Dipylidium*, for which a coproantigen test allows detection with high sensitivity). Therefore, the recommendation is to treat against tapeworms at least 4 times a year. Studies have shown that 1–3 annual dewormings do not provide sufficient protection.

[1] In areas endemic for heartworm, dogs that live indoors but are taken for walks may be exposed to mosquitoes, therefore *Dirofilaria* prevention should be considered. Detailed information about heartworm infection in dogs and cats can be found in ESCCAP Guideline 5: Control of Vector-Borne Diseases in Dogs and Cats

Figure 1: Scheme for individual worm management in dogs

Cat lives indoors

The cat lives exclusively indoors or only has access to a fenced-in patio or garden. The cat is unlikely to eat rodents or other prey.

RISK GROUP A

Treat 1–2 times a year against intestinal nematodes or carry out faecal examination and treat according to findings

Cat is free to roam

The cat has unrestricted access to the outdoors and has direct contact with other cats. Hunting behaviour and the potential ingestion of rodents or other prey cannot be ruled out.

RISK GROUP B

Treat 4–12 times a year against intestinal nematodes or carry out faecal examination and treat according to findings

Treat 4–12 times a year against tapeworms

ADDITIONAL TREATMENTS FOR CATS	
Roundworms	
Kittens	For the first time at 3 weeks of age, then every 2 weeks after weaning. If there is an ongoing increased risk of infection (e.g. free roam): monthly treatments up to 6 months of age.
Pregnant queens	A single treatment with emodepside spot-on approximately seven days before expected parturition prevents lactogenic transmission of *Toxocara cati* larvae to the kittens.
Lactating queens	Deworm at the first treatment of kittens (3 weeks after delivery).
Cats with increased risk of infection i.e. those used in competitions, shows or those kept in catteries etc.	Two treatments: 2 weeks before and 2–4 weeks after the event. For catteries: use planned deworming once a month or examine faecal samples every four weeks and treat according to findings.
Cats sharing homes with small children (below 5–6 years), immunocompromised or elderly individuals	Depending on the risk assessment, use planned deworming once a month or examine faecal samples once a month and treat according to findings.
Tapeworms	
Eats raw meat and/or offal, eats prey or goes hunting	Cats should be tested at least 4 times a year by faecal examination and treated according to findings, or dewormed at least 4 times a year. Infections with *Hydatigera taeniaeformis* (formerly *Taenia taeniaeformis*) predominate among tapeworm infections in cats. In areas endemic for *Echinococcus multilocularis* (the fox tapeworm), rodent-eating cats may shed infective eggs posing a risk to humans. However, compared to dogs, the risk of egg excretion is significantly lower. To shift the residual risk towards zero, higher treatment frequencies can be implemented. Monthly treatments (12 times a year) prevent egg excretion.
Flea infestation (as a vector for *Dipylidium*)	Once when the infestation is established.
Lungworms (*Aelurostrongylus abstrusus*, *Troglostrongylus* spp.)	
In highly endemic areas, cats with outdoor access that may eat slugs and snails or hunt paratenic hosts such as birds, reptiles or mice	Treat preventively against lungworms at monthly intervals all year round.
Heartworm (*Dirofilaria immitis*)**	
Cats living in areas endemic for heartworm (see Figure 18)	Treatment against transmitted third-stage larvae with macrocyclic lactones at monthly intervals during the mosquito season and for a 30-day period after the end of the mosquito season.
Travelling to areas endemic for heartworm	During the mosquito season, prophylactic treatment against transmitted third-stage larvae with macrocyclic lactones within 30 days of arrival into the endemic area, followed by further treatments at monthly intervals until 30 days after return.
Importation from areas endemic for heartworm	Immediately after importation, one-off prophylactic treatment against third-stage larvae and microfilariae with macrocyclic lactones. Preliminary examination for any existing infection at the time of importation and retest earliest 6 months later.

- Deworming practices should always be on the advice of a veterinary professional. Regular coprological examination of faeces (eventually with subsequent deworming), as suggested in Groups A and B can be a good alternative to standard deworming advice, if performed in the same frequency as the suggested treatments.
- If an animal's individual risk of infection with intestinal nematodes cannot be clearly assessed, the cat should be dewormed or faeces examined at least 4 times a year. The same applies in principle to tapeworm infections, although the reliability of detecting tapeworm infections using faecal sample tests is low (with the exception of *Dipylidium*, for which a coproantigen test allows detection with high sensitivity). Therefore, the recommendation in this case is to treat against tapeworms at least 4 times a year. Studies have shown that 1–3 annual dewormings do not provide sufficient protection. Deworming every 3 months does not necessarily prevent patent infections.

** Detailed information about heartworm infection in dogs and cats can be found in ESCCAP Guideline 5: Control of Vector-Borne Diseases in ESCCAP Guideline 5: Control of Vector-Borne Diseases in Dogs and Cats

Figure 2: Scheme for individual worm management in cats

BIOLOGY, DIAGNOSIS AND CONTROL OF WORMS

1. Roundworms (*Toxocara* spp., *Toxascaris leonina*)

Toxocara canis is a large, intestinal nematode, with adults measuring as much as 15 cm in length that can cause disease in young dogs. Similarly, *Toxocara cati*, an intestinal nematode with adults measuring up to 10 cm in length, can cause disease in young cats. In contrast to the two aforementioned canine and feline-specific roundworms, *Toxascaris leonina* occurs in both dogs and cats and is also mutually transmissible. This parasite, which can be up to 12 cm long, occurs less frequently than the *Toxocara* species and is also less pathogenic.

Toxocara spp. infection can occur in puppies and kittens but also in older dogs and cats. Infection of humans can occur as a result of accidentally ingesting infective eggs or eating undercooked meat containing larvae.

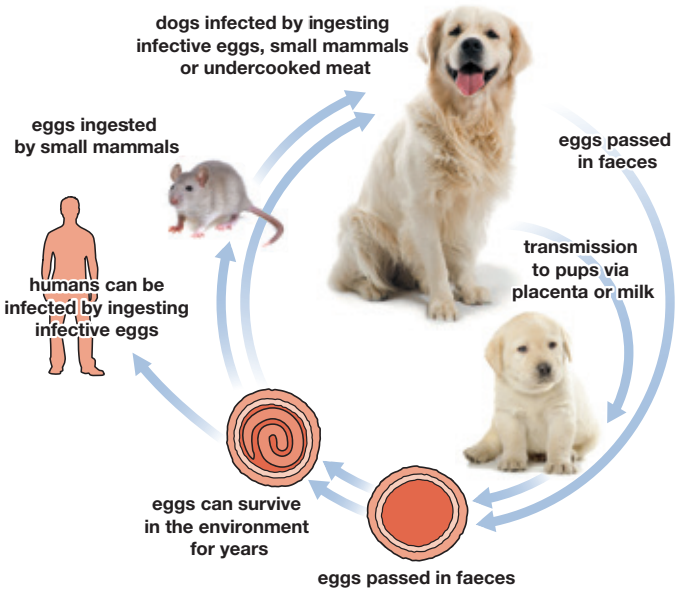


Figure 3: *Toxocara canis* life cycle

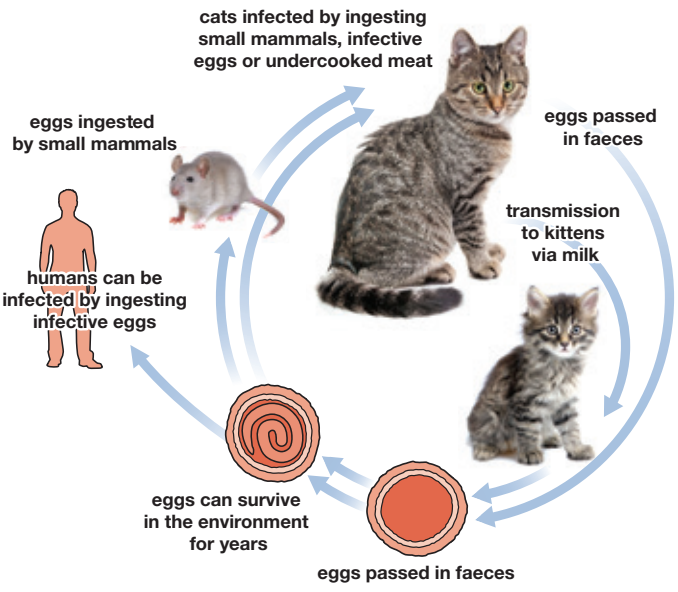


Figure 4: *Toxocara cati* life cycle

Adult worms inhabit the small intestine (Figure 5) where they lay numerous eggs that are then passed in the faeces. Within the eggs, an infective larva will develop under suitable environmental conditions (warm, moist) within ca. two weeks and these can survive in the environment for years. Dogs and cats become infected when they ingest infective eggs from the environment (Figure 6). Dogs and cats can also become infected when they eat undercooked meat or prey on an infected paratenic host (e.g. rodents).

The eggs hatch in the intestine releasing larvae that penetrate the small intestinal wall. The *Toxocara* larvae undergo a hepato-tracheal migration, with the life cycle completed when larvae are coughed up and swallowed, returning to the small intestine to complete their migration (Figure 3 and Figure 4). In puppies, infection can occur by the passage of larvae across the placenta from about the 42nd day of pregnancy and later through the milk (Figure 3). Kittens can be infected through the milk (Figure 4).

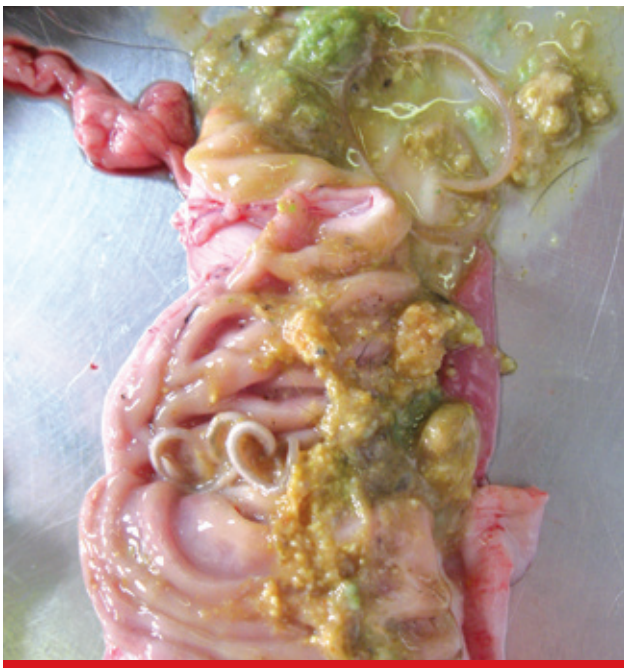


Figure 5: Adult worms live in the small intestine of infected dogs and cats

In the case of infection with *T. leonina* eggs, the released larvae 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 that can then act as paratenic hosts.

Patent *Toxocara* infections also frequently occur in adult dogs and cats. In *T. canis* infections, the eggs are excreted approximately 4–8 weeks after the ingestion of infective eggs (young dogs: 4–6 weeks; approximately one-year-old dogs: 6–8 weeks), but from the 16th day after birth at the earliest in intrauterine-infected puppies. After lactogenic transmission to the kittens, prepatency is at least four, but usually eight weeks. After ingestion of paratenic hosts, it takes up to approximately one month before *T. canis* or *T. cati* eggs are excreted in the faeces.

In adult animals, infections are extremely unlikely to be associated with clinical signs therefore it is difficult to determine whether a dog is infected unless regular faecal examinations are conducted. Puppies can be heavily infected by *T. canis* worms in utero or via nursing and these may cause serious illness before diagnosis is possible by faecal examination. In addition, these parasites are prolific egg-layers and just a few worms can produce large numbers of eggs which are able to survive for a long time in the environment.

Regular faecal examinations can determine whether an animal is infected with mature stages of *Toxocara*. Flotation methods are used to detect the characteristic roundworm eggs. For some time now, commercial coproantigen methods (ELISA) have also been available. These have been proven to detect a *T. canis* infection, even during pre- or postpatency, and apparently do not lead to false-positive results when *Toxocara* eggs ingested coprophagically pass through the intestine.

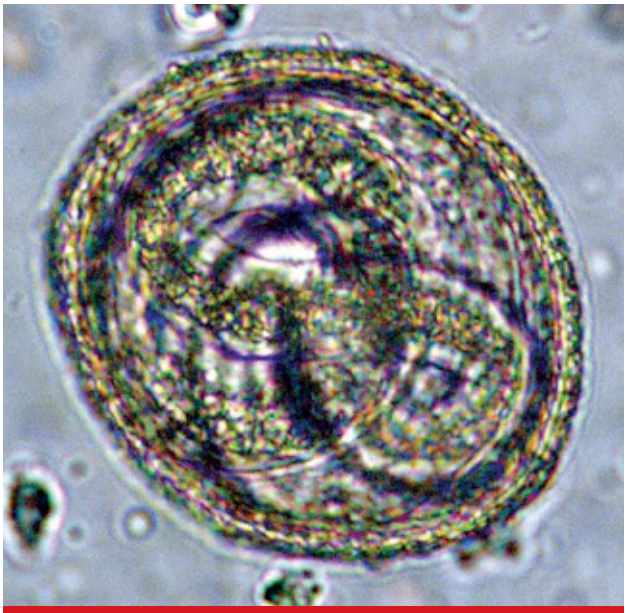


Figure 6: *Toxocara cati* infective egg

Toxocara roundworms have an elevated zoonotic potential. After oral intake of infective roundworm eggs, the larvae may begin somatic migration (larva migrans complex). This can have serious consequences on human health (see chapter on **OWNER CONSIDERATIONS IN PREVENTING ZOONOTIC DISEASES**). For these reasons, *Toxocara* spp. infections in dogs and cats of all ages merit consideration.

- **Puppies** should be treated with appropriate anthelmintics from 14 days old. The treatment should then be repeated fortnightly until two weeks after weaning and, depending on infection risks, monthly treatments carried out up to six months of age.
- 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. If there is an ongoing increased risk of infection (e.g. free roam), monthly treatments should be carried out up to 6 months of age.
- To reduce transmission to the puppies, **pregnant bitches** can be given macrocyclic lactones on the 40th and 55th day of pregnancy, or fenbendazole daily from the 40th day of pregnancy continuing until two days postpartum.
- **Pregnant queens** should be treated with emodepside spot-on approximately seven days before expected parturition to prevent lactogenic transmission of *Toxocara cati* larvae to the kittens.
- **Nursing bitches and queens** should be treated concurrently with the first treatment of their offspring, as they often develop patent infections at this time.
- For **adult dogs and cats**, ESCCAP recommends an individual risk assessment for each animal to determine whether anthelmintic treatment is necessary, and how often. There is surprisingly little information about the impact of treatment intervals on parasite burdens and environmental contamination. Accordingly, maximum re-treatment intervals may vary under different epidemiological conditions. Current information suggests that annual or twice-yearly treatments do not have a significant impact on preventing patent infection within a population. Therefore, a treatment frequency of at least four times per year is a general recommendation if the risk of infection is unknown or if infections cannot be excluded through diagnostic tests.
- Without diagnostics, the individual situation of an animal and the resulting recommended deworming frequency can only be estimated.
- False-positive coproscopic findings are relatively common in dogs that consume cat or dog faeces containing eggs of *T. cati*/*T. canis*: coprophagy may cause false-positive results and suggests drug resistance. Such cases can be identified with extended analyses, i.e. coproantigen test (or by avoiding coprophagy prior to sampling).
- So far, there is no evidence of anthelmintic resistance in *Toxocara*; in case of suspicion, coprophagy should be ruled out first.
- As the pre-patent period for *Toxocara* spp. after ingestion of larvae via predation of paratenic hosts (rodents) or infective eggs from the environment is a little over four weeks, monthly treatment will minimise the risk of patent infections and is recommended in risk scenarios, for example when the pet shares a house with small children and has frequent risk of infection (free-roaming, access to garden).
- As an alternative to repeated treatments, faecal examinations can be performed at suitable intervals followed by anthelmintic treatment where positive results are found (see chapter on **DIAGNOSIS OF HELMINTH INFECTIONS**). This approach should be adopted in countries where routine treatments are not acceptable for legislative reasons. Nevertheless, between faecal examinations, the excretion of infective eggs is still possible and cannot be prevented. Caution must be taken in cases of negative results following faecal examination: it cannot be assumed with certitude that an animal is not infected with roundworms in case of prepatent infections or when the number of excreted eggs is under the detection limit of the analysis.

For further information on *Toxocara* spp. and *Toxascaris leonina* characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2A and 3–7.

2. Tapeworms

Echinococcus granulosus sensu lato and *Echinococcus multilocularis*

Echinococcus granulosus (dog tapeworm) is a small cestode that inhabits the small intestine of dogs and some other canids, excluding foxes. *Echinococcus multilocularis* (fox tapeworm) is a small cestode that inhabits the small intestine of foxes, raccoon dogs, some other canids and rarely dogs and very seldom cats. See Figures 7 and 8 for life cycles.

Both tapeworms, *E. granulosus* and *E. multilocularis*, induce extra-intestinal metacestode stages in intermediate hosts and both are zoonoses of major public health concern. In humans, the species of the *E. granulosus* group cause cystic echinococcosis and *E. multilocularis* causes alveolar echinococcosis, which if untreated can have potentially fatal consequences. Both infections result in the formation of cysts, most commonly in the liver (*E. multilocularis*, *E. granulosus*) or in the lung (*E. granulosus* group, within this mainly *E. ortleppi*, the so-called bovine strain). These infections occur following the oral ingestion of eggs or proglottids excreted in the faeces of definitive hosts. Eggs are immediately infective to intermediate hosts and humans (aberrant host). Also, dogs can become infected through the ingestion of eggs and develop alveolar echinococcosis.

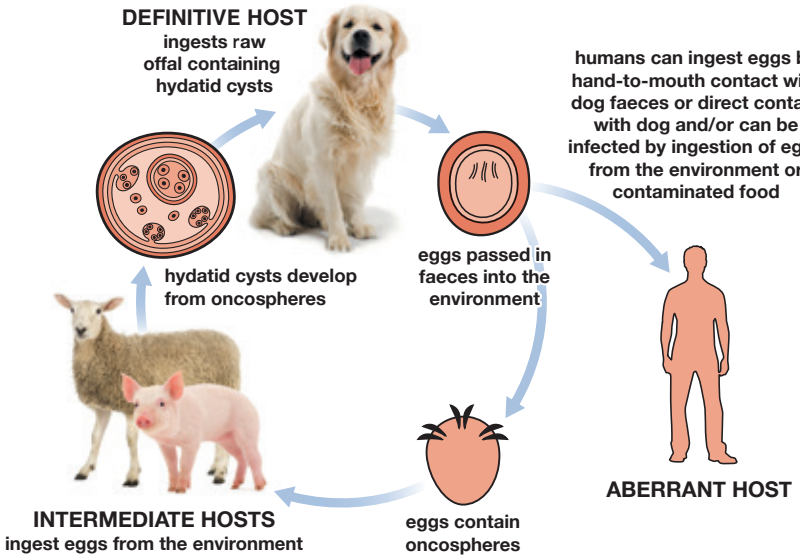


Figure 7: *Echinococcus granulosus* life cycle

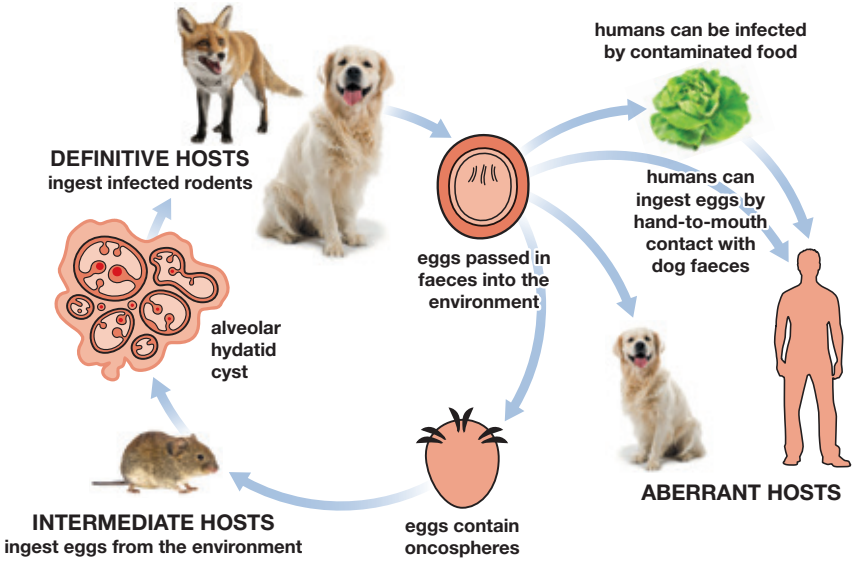


Figure 8: *Echinococcus multilocularis* life cycle

In areas where members of the *E. granulosus* group are endemic (Figure 9), care should be taken to prevent dogs having access to raw offal and carcasses. The prepatent period varies between 5–8 weeks, depending on the species. Where dogs may have access to carcasses or raw viscera especially from sheep, pigs, cattle or horses (depending on the *Echinococcus* genotypes present locally) they should be treated at least every six weeks with an effective anthelmintic containing praziquantel.

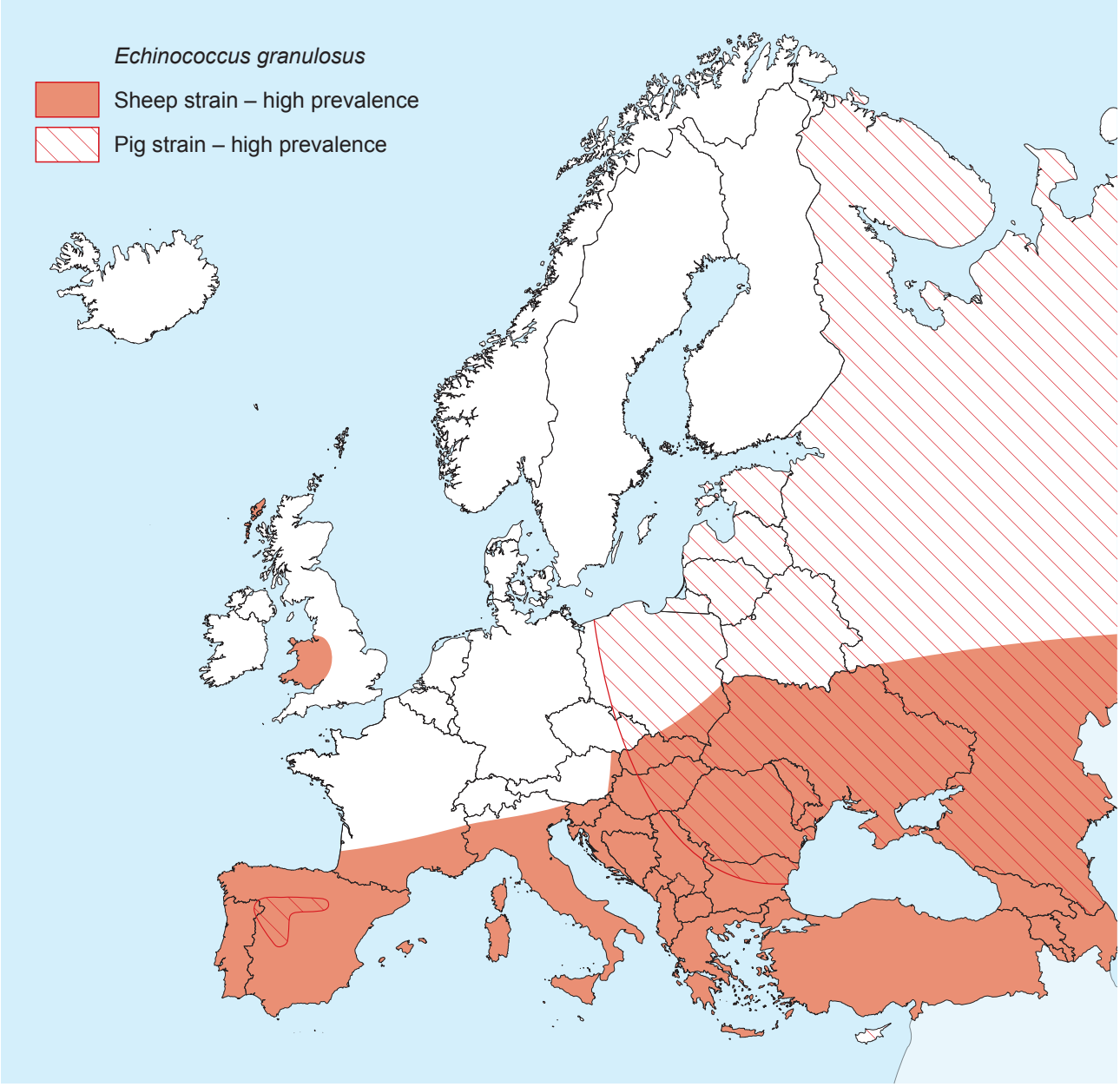


Figure 9: Approximate overview of the occurrence of *Echinococcus granulosus* strains in Europe (© ESCCAP)

In the central and Eastern European endemic area of *E. multilocularis* (Figure 10) red foxes are the main definitive hosts and voles act as intermediate hosts. Dogs and cats are infected by eating wild rodents that carry metacestodes of *E. multilocularis*. The prepatent period is just under four weeks. Dogs that have access to rodents should be treated at four-week intervals with an effective anthelmintic containing praziquantel. Cats, in contrast to dogs, are epidemiologically insignificant as sources of egg excretion. Whilst in dogs it is common to find eggs in the fur of infected animals, no eggs have been recovered to date from the coat of infected cats and their zoonotic potential is also probably limited because there is only a small risk of cats excreting large numbers of eggs.

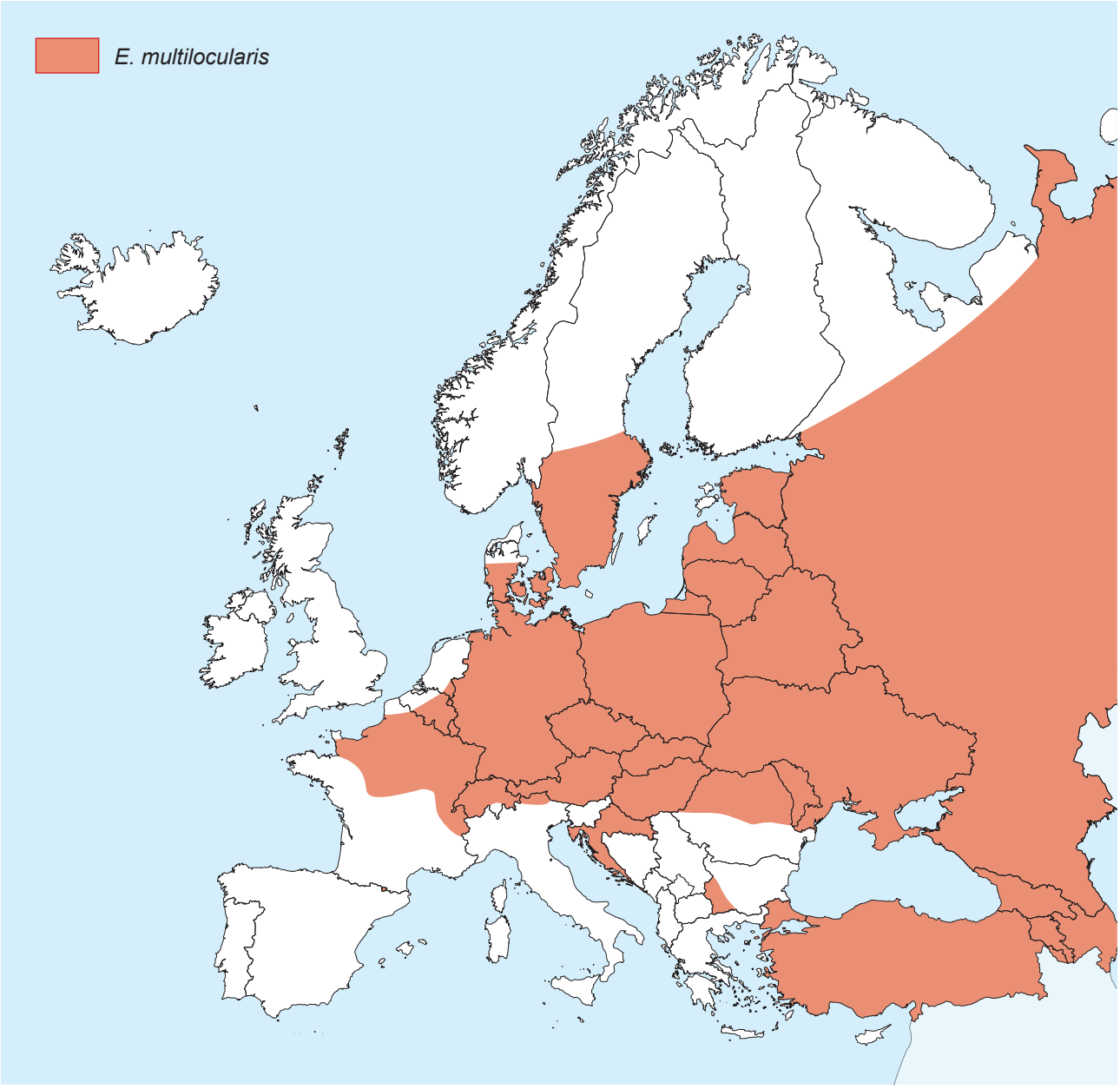


Figure 10: Approximate occurrence of *Echinococcus multilocularis* in the fox in Europe (© ESCCAP)

Specific diagnosis of *Echinococcus* infections in definitive hosts is challenging. The *Echinococcus* proglottids are so small that they are practically invisible to the naked eye in faeces. The microscopic detection of taeniid eggs (Figure 13) using the flotation method has low sensitivity as they are passed intermittently in faeces (flotation solutions with higher specific gravity are recommended, see ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines). Alternatively, the perianal adhesive tape method can be used with better success. Importantly, when taeniid eggs (including *Echinococcus* spp. and *Taenia* spp.) are detected, they cannot be differentiated morphologically. Thus, it is compulsory that DNA-based tests for species and/or genotype identification are performed. Generally, in areas endemic for *Echinococcus*, since eggs are directly infective, taeniid infections based on egg detection should be handled as potential *Echinococcus* infections.

Anthelmintic treatment and accompanying measures

When infection with an *Echinococcus* species is confirmed, the animal owner should be informed about the previous risk of infection and instructed on how to proceed (e.g. serological follow-up implemented through a medical check).

Positive dogs should be treated twice with praziquantel at 24-hour intervals, and bathed after each treatment to remove any parasite eggs adhering to the coat. Personnel should wear suitable protective clothing, including gloves and a mask. The excreted faeces must be appropriately disposed of, ideally through incineration, up to three days after anthelmintic treatment. The dog must be bathed again before being handed over to the owners. Therapeutic success is evaluated after 7–14 days by faecal examination using flotation and PCR.

Dogs can also become accidental aberrant hosts for *E. multilocularis*, without egg excretion, by ingesting eggs from the environment or by coprophagic behaviour. This leads to the development of larval stages (metacestodes) in their organs, primarily the liver. However, such cases are rare and may be underestimated. The presence of alveolar echinococcosis in dogs is usually suspected following the use of imaging procedures and can be clarified using serological, histological and molecular methods.

Prevention of *Echinococcus* infection is achieved through the following recommendations:

- If possible, dogs should not have access to wild rodents.
- Coprophagy in dogs should be prevented to avoid alveolar echinococcosis.
- Dogs and cats should not be given slaughter waste or raw meat but only commercial food or meat that has been heated for 10 minutes (inner temperature: 65°C) or frozen for one week at -17 to -20°C.
- For dogs with a high risk of infection with *Echinococcus* spp., ESCCAP promotes monthly treatments with an appropriate anthelmintic containing praziquantel.
- Dogs travelling into areas with a high risk of *Echinococcus* spp. infections should be treated four weeks after starting the trip and for four weeks after returning with an appropriate anthelmintic containing praziquantel.
- Dogs imported from endemic areas should be promptly seen by a veterinarian and treated with an appropriate anthelmintic containing praziquantel.
- Cats face similar tapeworm infection risks as dogs, potentially even higher due to their more frequent rodent hunting behaviour. However, cats are comparatively unsuitable hosts for *Echinococcus multilocularis* and only rarely excrete eggs, and in this case, in small numbers. In addition, eggs excreted by cats are not infective to rodents according to an experimental study. However, while very rare, recent French studies have detected sometimes substantial numbers of presumably infective eggs in cat faeces. Thus, considerable differences have been observed in individual susceptibility of cats to *E. multilocularis* infection. Based on current knowledge, zoonotic risk cannot be definitively ruled out. Therefore, the decision of whether to carry out diagnostic checks or monthly anthelmintic treatment should be at the discretion of the responsible veterinarian in consultation with the animal owner, considering individual risk factors (e.g. small children in the household, very close human–animal contact).

For further information on *Echinococcus* spp. characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2B and 3–7.

Dipylidium caninum

Dipylidium caninum is a tapeworm of dogs and cats, common throughout Europe. The intermediate hosts are fleas or chewing dog lice, and dogs and cats become infected when they ingest the infected insects. The adult tapeworm develops within the dog or cat in the small intestine (Figure 11). *D. caninum* is zoonotic and if humans ingest infected fleas or lice, they can become infected, although this is rare. The prepatent period is approximately three weeks.

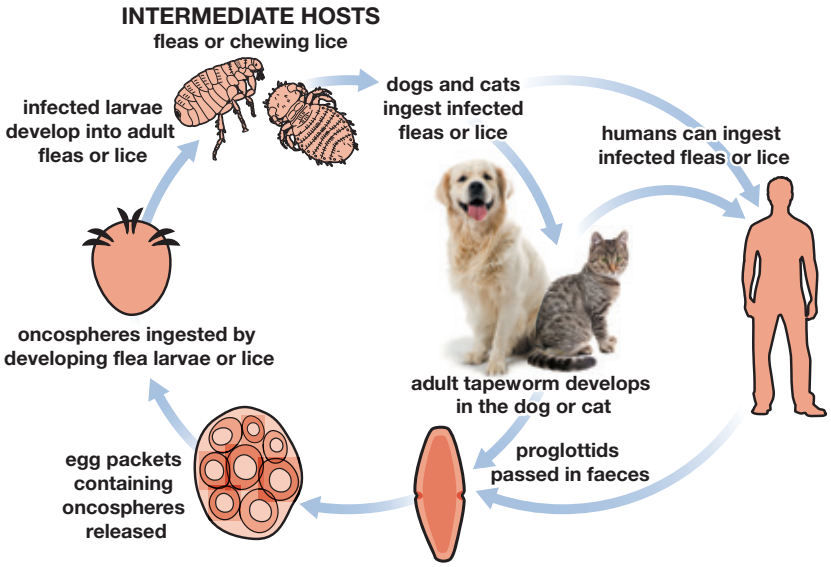


Figure 11: *Dipylidium caninum* life cycle

Infection with *D. caninum* is rarely associated with clinical signs in dogs and cats. The mature segments leaving the anus may result in anal irritation (pruritus) causing an animal to rub its bottom along the ground, accompanied by restlessness. However, massive worm burdens can also lead to small intestinal obstructions and, particularly in young animals, to inappetence or diarrhoea.

Coproscopic examination, i.e. flotation, to detect egg packages typical for *D. caninum*, is very uncertain. Occasionally, the approximately 1 cm long, white proglottids can actively migrate out of the anal opening, otherwise they are excreted in the faeces. Consequently, the white proglottids may be seen in fresh faeces or in the coat around the anus. When dry, these are shaped like rice grains and may be evident around the perianal area and in samples from the animal's bedding. A commercial coproantigen test has also become available for the detection of *D. caninum* infection, which has a significantly higher sensitivity than coproscopic worm detection.

Treatment is performed with praziquantel and control management is achieved by additional control of fleas and lice. Single reports of anthelmintic resistance have been reported.

For further information on *D. caninum* characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2B and 3–7.

Taenia spp. and *Hydatigera taeniaeformis*

Taenia spp. and *Hydatigera* (syn. *Taenia*) *taeniaeformis* are "large" (i.e. 20–250 cm long) tapeworms that can infect dogs, cats and foxes by the ingestion of intermediate hosts. They are common throughout Europe. The intermediate hosts are varied and, depending on the *Taenia* species, 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*) (Table 1). Infection of the intermediate host occurs by ingestion of tapeworm eggs in proglottids passed in the faeces of the definitive host (Figure 12). Dogs and cats become infected when they eat the tissue or viscera of infected intermediate hosts. The effects on the intermediate host may be more profound than on the definitive host.

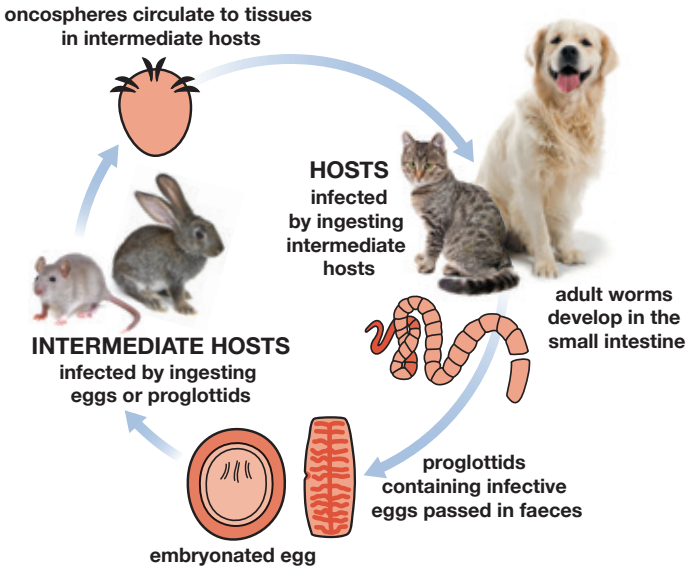


Figure 12: *Taenia* spp. and *Hydatigera taeniaeformis* life cycle

The *Taenia* and *Hydatigera* species found in canids and felids have different intermediate hosts, depending on the species (see Table 1): *Hydatigera* (syn. *Taenia*) *taeniaeformis* (rodents: liver), *Taenia crassiceps* (rodents: subcutis, body cavities), *Taenia pisiformis* (lagomorphs and rodents: liver), *Taenia hydatigena* (small ruminants, cattle, pigs: subserosal on the mesentery and liver), *Taenia ovis*, *Taenia cervi* (sheep, goats, cervids: musculature), *Taenia multiceps* (sheep, goats, cattle: brain and spinal cord), *Taenia serialis* (hares: connective tissue).

The prepatent period for *Taenia* spp. ranges from about 4–9 weeks in dogs (depending on the species) and is approximately 5–8 weeks for *Hydatigera taeniaeformis* in cats. Patency can last for several months up to several years, for example *T. ovis*, a *Taenia* species infecting dogs, can be patent for up to five years.

Taenia spp. infections are rarely associated with clinical signs in dogs or cats. The mature segments leaving the anus may result in anal pruritus causing an animal to rub its bottom along the ground. Owners may also notice motile segments crawling on the animal's coat after leaving the anus.

Taenia crassiceps is rarely the cause of exogenous budding that leads to the formation of subcutaneous or abdominal cysts.

Coproscopic examination for the diagnosis of a *Taenia* or *Hydatigera* infection is rather uncertain, due to intermittent egg excretion and the proglottids (unlike *Dipylidium* having only one genital pore) may be only observed macroscopically by chance. Therefore, the microscopic detection of taeniid eggs (Figure 13) using the flotation method or the combined sedimentation-flotation method has limited sensitivity (see *Echinococcus*), especially when using standard flotation solutions. Accordingly, flotation solutions with higher specific gravity are recommended (see ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines). Alternatively, the perianal adhesive tape method can be used, with better success. However, microscopically, *Taenia* or *Hydatigera* eggs cannot be distinguished from *Echinococcus* eggs. Therefore DNA-based tests for species and/or genotype identification should be carried out for clarification.

Generally, in *Echinococcus* endemic areas, since eggs are directly infective, taeniid infections based on egg detection should be handled as potential *Echinococcus* infections.

Treatment is by the administration of an effective anthelmintic at suitable intervals which will most likely depend upon evidence of an existing infection. Eggs can remain viable for lengthy periods in the environment. For prevention, owners should try and prevent dogs and cats having access to the various intermediate hosts. The feeding of viscera and raw meat that has not been frozen for a sufficiently long time (one week, -17 to -20°C) should be discouraged.

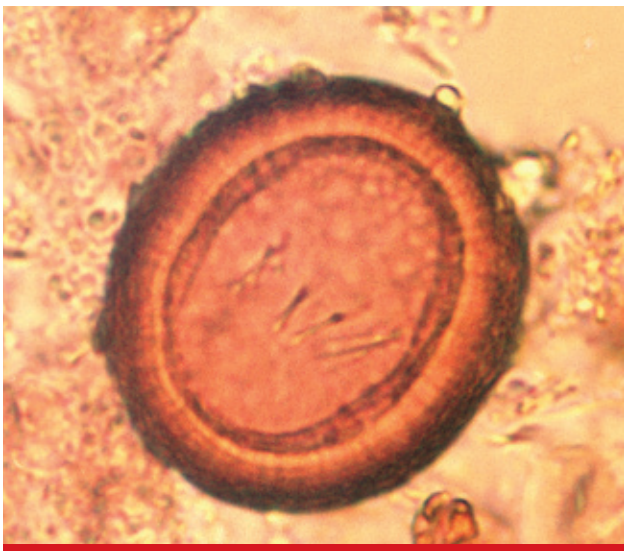


Figure 13: Taeniid egg

Table 1: Summary of *Taenia* spp. and *Hydatigera* spp. found in dogs and cats

Definitive hosts	**DOGS**						**CATS**
Species	***Taenia multiceps***	***Taenia serialis***	***Taenia crassiceps****	***Taenia pisiformis***	***Taenia hydatigena***	***Taenia ovis***	***Hydatigera* (syn. *Taenia*) *taeniaeformis***
Prepatent period (approx. in weeks)	6	5–6	4–6	6–8	7–10	6–8	5–10
Intermediate host	Sheep, goats and cattle	Rabbits (and rodents), primates, roe deer	Rodents	Rabbits/hares (and rodents)	Sheep, goats, cattle and pigs	Sheep and goats	Rodents
Intermediate stage and site	Coenurus larvae in brain and spinal cord	Coenurus larvae in connective tissue	Cysticercus larvae in body cavities or subcutaneous tissue	Cysticercus larvae in abdomen or liver	Cysticercus larvae in abdomen or liver	Cysticercus larvae in muscles	Strobilocercus larvae in liver and abdomen

* much more frequently found in red foxes

For further information on *Taenia* spp. and *Hydatigera taeniaeformis* characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2B and 3–7.

3. Heartworm and Subcutaneous Worms

Dirofilaria immitis

Dirofilaria immitis, also known as 'heartworm', is a thick filarioid worm, measuring up to 30 cm long and 1–3 mm thick, that resides in the pulmonary arteries of dogs and cats (Figure 14). It has an indirect life cycle, transmitted through intermediate mosquito hosts (Figure 15), with dogs and cats acting as the definitive hosts. The adult parasites (macrofilariae) mate in the definitive host, after which the offspring (microfilariae) are ingested by mosquitoes during blood meals. The larvae mature to the infective third-stage larvae (L3) in the mosquitoes, which are then transferred to a new mammalian host during the next blood meal. During the first two to four months, the larvae moult twice and migrate through the mammal's connective tissues to finally reach the heart or pulmonary artery via the bloodstream. Six to seven months after infection, at the earliest, the female worms begin to produce microfilariae (when both male and female worms are present), which are released into the circulating blood. Heartworm infection (*D. immitis*) is endemic in many southern and southeastern European countries (Figure 18).

Climatic changes, favourable to parasite development, and the increasing number of imported and travelling pets have increased the risk of infection for dogs, cats and pet ferrets.

Although cats and ferrets are potential hosts for heartworm, their relevance as definitive host is clearly reduced compared to dogs.

Infection with *D. immitis* may cause severe and potentially fatal disease in dogs and cats. Low worm burdens can be subclinical. Larger worm burdens can cause clinical signs such as loss of condition, weakness, dyspnoea and chronic cough. If untreated, the disease can progress to right side heart failure and death. In cats, the disease passes mostly unnoticed until potential sudden death. Clinical signs are described more in detail in ESCCAP Guideline 5: Control of Vector-Borne Diseases in Dogs and Cats.

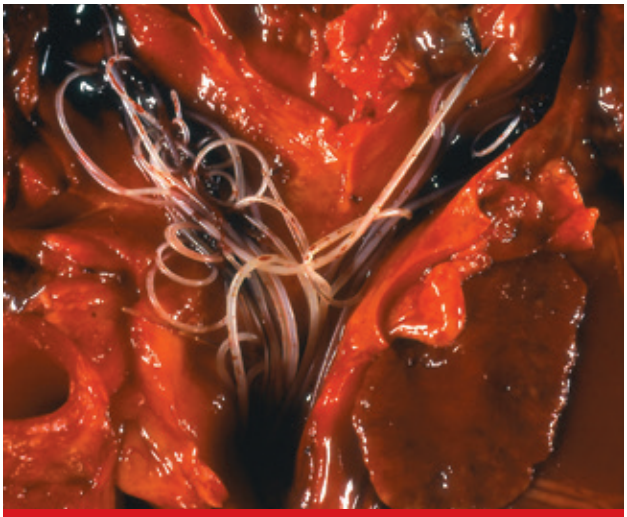


Figure 14: Adult worms live in the pulmonary arteries

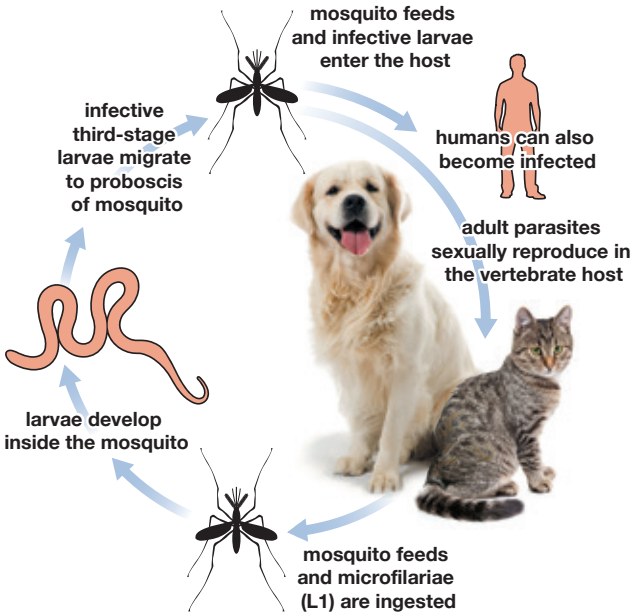


Figure 15: *Dirofilaria immitis* life cycle

In most parts of Europe where infection is endemic, the transmission season of heartworm lasts from April to October (depending on the climate). Yearlong transmission of *D. immitis* is actually only reported for the Canary Islands (Spain).

D. immitis infection is diagnosed by detecting circulating antigens in the blood. In addition, microfilariae can be detected by microscopic blood examination: blood samples should be further examined after concentration by the Knott's test or with a filter method (see ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines). The detection of microfilariae is less sensitive and less effective in cats due to microfilariae being rarely present. *Dirofilaria immitis* microfilariae can be differentiated from other microfilariae by different procedures (specified in Guideline 4). In cats, the detection of a *D. immitis* infection is generally more difficult than in dogs and should include imaging of the thorax in addition to laboratory diagnostics. Antibody detection is a complementary method to demonstrate infection.

The organic arsenical compound, melarsomine dihydrochloride, is the only effective drug available for treating adult heartworm infections in **dogs**. The currently-accepted regimen is a two-step treatment to reduce the risk of pulmonary thromboembolism: after one initial treatment of 2.5 mg/kg bodyweight, given by deep intramuscular injection in the lumbar muscles, the recommended follow-up treatment is administered 30–60 days later (2.5 mg/kg bodyweight twice at an interval of 24 hours). Complications, due to pulmonary thromboembolism, should be reduced by the restriction of exercise following treatment and by the administration of heparin and a corticosteroid (e.g. week one: prednisone at 0.5 mg/kg BID, week two: 0.5 mg/kg SID, weeks three and four: 0.5 mg/kg EOD) after melarsomine dihydrochloride injections. *Wolbachia* (obligate, intracellular, gram-negative, endo-symbiotic bacteria) has been implicated as crucial in the pathogenesis of filarial diseases. Doxycycline reduces the *Wolbachia* burden in all stages of heartworms. Thus, administration of doxycycline at 10 mg/kg daily for four weeks, before the administration of melarsomine dihydrochloride, is strongly recommended. Surgical intervention is advised when multiple worms have been displaced into the right cardiac chambers producing sudden onset caval syndrome. Generally, exercise restriction for the duration of the treatment and recovery period is essential. Controlled, short walks on a leash are recommended to reduce the risk of pulmonary embolism by dead or dying worms.

There is no registered adulticide drug for **cats**. Decreasing doses of prednisolone are advised in cats, in order to relieve respiratory distress, with an initial dose of 2 mg/kg bodyweight per day. If a cat presents with severe signs of HARD (heartworm associated respiratory disease), high doses of oral prednisolone (1–2 mg/kg bodyweight 3 times a day) are recommended.

Control strategies for dogs

Topical or oral macrocyclic lactones, administered monthly throughout the transmission season, are effective against *D. immitis* L3 and fourth-stage larvae (L4) which have developed within the previous 30 days, thus preventing disease caused by adult worms. Several compounds alone, or in combination with other parasiticides, are available for oral administration or topical application. An injectable, sustained-release macrocyclic lactone has been approved in some European countries for use only in dogs older than six months and is registered to give protection for one year.

Prevention, through monthly administration of macrocyclic lactones (or according to the corresponding package insert), should start before the mosquito season in spring and continue until late autumn. Topical administration of permethrin has demonstrated repellent efficacy against mosquitoes on dogs for at least four weeks. In southern Europe, protection against heartworm should be carried out from May until the end of November. In hyperendemic areas, year-round preventive therapy is recommended.

Currently, preventative drugs are fully effective against *D. immitis* larvae but reports from the USA suggest that drug resistance is emerging. Even though there have been no reports of autochthonous resistance in Europe, in view of the fact that the maintenance of macrocyclic lactone efficacy is critical for *Dirofilaria* control, there are some recommendations which may assist in decreasing the risk of resistance selection.

1. Dogs should be checked for both circulating antigens and blood microfilariae at the beginning of each preventative annual treatment.
2. Although *Dirofilaria* does not appear to be entirely dependent on its bacterial symbiont *Wolbachia*, which can be killed by prolonged antibiotic treatment, the clearing of bacteria from circulating microfilariae seems to prevent the onset of infective larvae which may develop in mosquitoes afterwards.
3. The combination of heartworm preventatives with products designed to prevent mosquito blood-feeding activity (repellents) during the heartworm transmission season, could be useful in protecting dogs from infection and from ectoparasite infestations that often occur in the same season.

Control strategies for cats

Prophylactic larval treatments in cats follow the same regimen as in dogs, with monthly dosing regimens (see www.esccap.org for links to tables of approved compounds in individual countries). Pyrethroid-based products are highly toxic for cats and no other compound has indications for repellent activity.

In endemic areas, puppies and kittens need to be placed on preventive heartworm treatment as soon as possible after birth (consistent with label recommendations). Most preventive anthelmintics effective against heartworm also control a range of other worms, therefore a product should be chosen to control all relevant worms. In addition, treatment can be extended throughout the year to ensure the continued control of non-seasonal parasites such as *Echinococcus* spp. and *Toxocara* spp., where necessary. The use of such products should commence within the first four weeks after the start of a potential transmission and maintained monthly (or according to the product indications) until 30 days after the last potential date of an infection. As a principle, all dogs previously exposed to the risk of *D. immitis* infection should receive a complete clinical check-up, including blood tests to detect microfilariae and/or serology to detect circulating antigens or antibodies for the diagnosis of heartworm infections. Similarly, clinical examination and the appropriate laboratory tests should also be carried out on cats.

Detailed information about heartworm infection in dogs and cats can be found in ESCCAP Guideline 5: Control of Vector-Borne Diseases in Dogs and Cats and on the website of the European Society of Dirofilariosis and Angiostrongylosis (www.esda.vet/index.php/guidelines).

Dirofilaria repens

Dirofilaria repens is a nematode, reaching up to 17 cm in length, which can infect both dogs and cats and is also transmitted by mosquitoes (Figure 17). *D. repens* is the species most frequently associated with subcutaneous filarioidosis of dogs and cats. Female worms release microfilariae that circulate in the bloodstream for many months. These are ingested by mosquitoes during a blood meal where they further develop into infective third-stage larvae. They are then transferred back to a new mammalian host, via saliva, during the next blood meal. The infective larvae of *D. repens* migrate into the subcutaneous connective tissues where they reach maturity. Adult worms are found between subcutaneous and deep connective tissue layers in most body organs. Adults can live for several years.

Endemic areas for *D. repens* overlap with endemic *D. immitis* areas in many regions of Europe. *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, *Dirofilaria repens* infections have been documented in dogs with no travel history from Germany, the Netherlands, Austria, Portugal, Spain, Estonia and Poland. The distribution of *D. repens* is shown in Figure 18.

Most infections are subclinical, though cold, painless nodules (unique or multiple) containing the adult parasites and microfilariae can be found under the skin of infected animals (Figure 16). In cases of heavy infection or in sensitised animals, a mild to severe dermatitis can sometimes be observed.

Despite infections of *D. repens* being mostly subclinical, therapy is recommended because of the zoonotic potential of the parasite. The nodules can be eliminated by surgery but it is preferable to extract the adult worms by aspiration with a catheter because it is much less invasive.

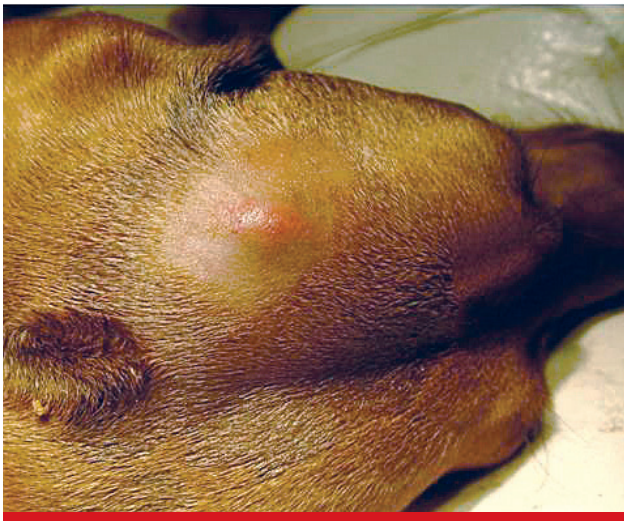


Figure 16: The worm may cause skin nodules and swelling

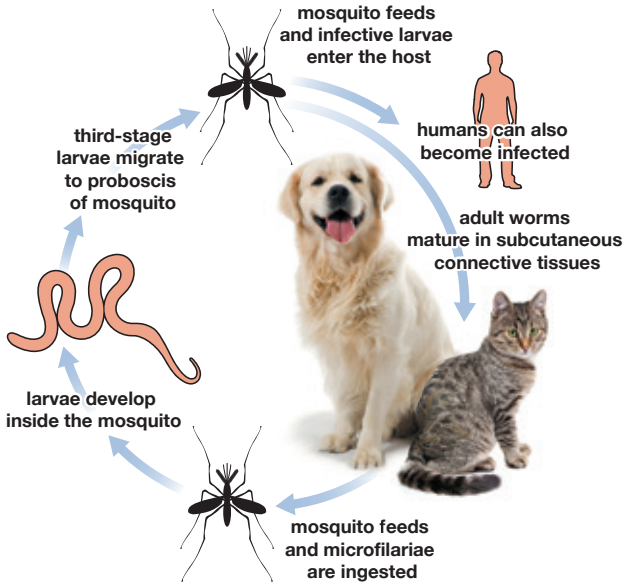


Figure 17: *Dirofilaria repens* life cycle

Spot-on moxidectin is licenced in some European countries as an effective adulticide therapy for *D. repens* infection in dogs (also for prevention and to reduce microfilaria burden). Due to the zoonotic potential, dogs in endemic areas should be treated monthly with a macrocyclic lactone during the transmission season (usually from April to November). If microfilaraemia is detected, a repellent insecticide should be used in addition to monthly treatment with moxidectin in order to prevent further spread of the parasite or endemisation in previously non-endemic regions.

Before and after travelling, dogs and cats should be examined for infection by *D. repens* microfilariae. In dogs, blood tests can demonstrate the presence of microfilariae. Information on further diagnostic details can be found in ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines.

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.

Monthly treatment with a macrocyclic lactone during the transmission season provides protection against a patent skin worm infection. When travelling to endemic areas for less than four weeks, treatment should be given immediately after the return journey. For longer journeys, treatment should be given monthly and once again, within four weeks of returning (see also information on *D. immitis*). If an animal is to be transported from an endemic area to a non-endemic area, it is advisable to test its blood for microfilariae. If positive, treatment against microfilariae should be carried out in the endemic area and a repellent insecticide applied no later than 30 days after arrival in the non-endemic area.

See ESCCAP Guideline 5: Control of Vector-Borne Diseases in Dogs and Cats for a range of diagnostic and treatment options that may be appropriate.

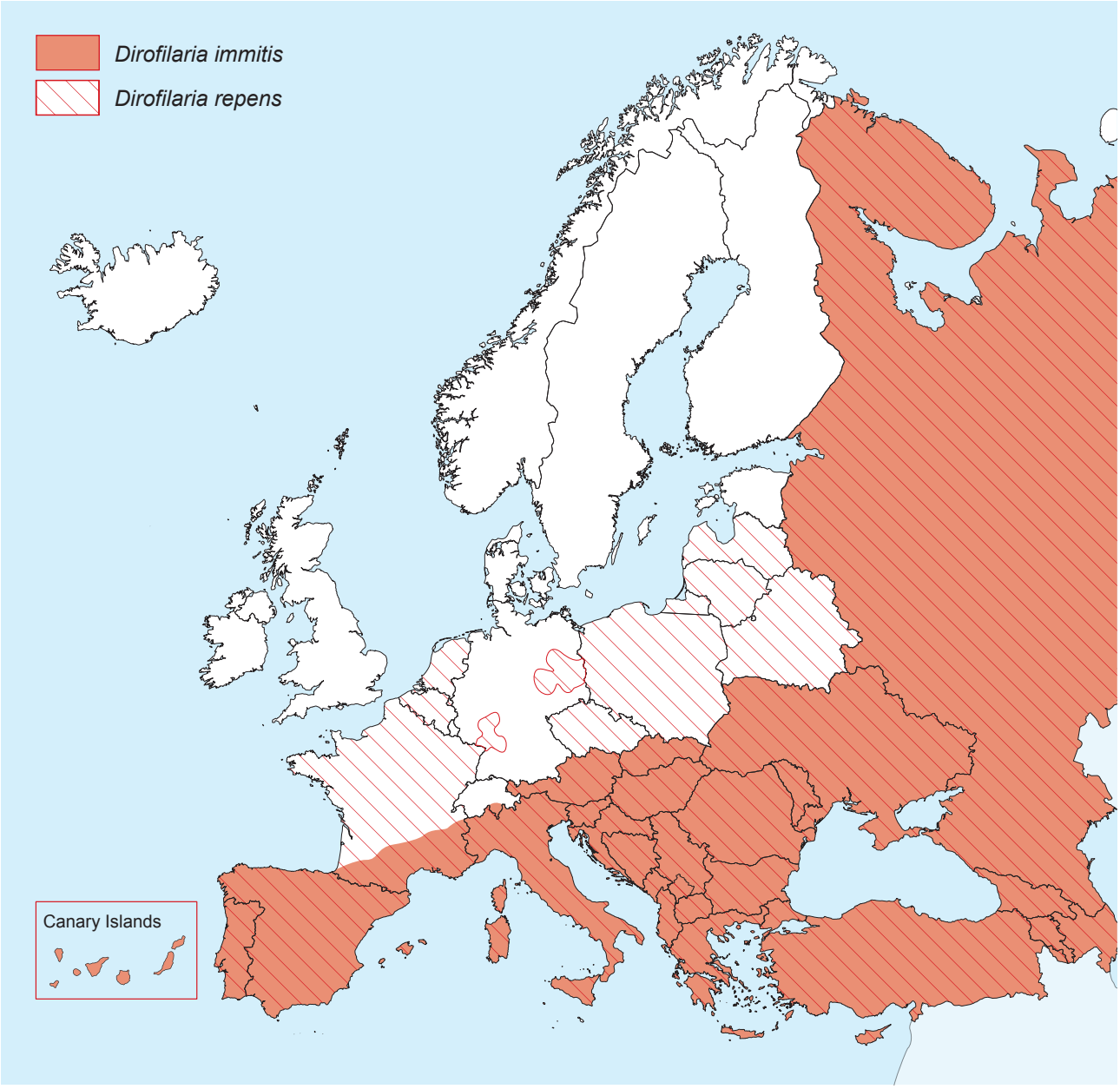


Figure 18: Approximate distribution of *Dirofilaria immitis* and *Dirofilaria repens* in Europe (© ESCCAP)

Zoonotic potential of *D. immitis* and *D. repens*

Most cases of zoonotic *Dirofilaria* infections in Europe are caused by *D. repens*. After being bitten by a mosquito infected with *D. repens,* the most common findings are subcutaneous nodules and nodules under the conjunctiva of the eye. *D. immitis* can develop into granulomas in different organs (mainly the lungs), which nevertheless remain mostly without clinical relevance. Due to the zoonotic potential of *D. repens*, microfilaraemic dogs should be treated monthly with preventative drugs able to kill microfilariae, combined with a repellent insecticide. Administering moxidectin once a month for six consecutive months also eliminates the adult worms in dogs.

For further information on *Dirofilaria* spp. characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2C and 3–7 and ESCCAP Guideline 5: Control of Vector-Borne Diseases in Dogs and Cats.

4. French Heartworm (*Angiostrongylus vasorum*)

Angiostrongylus vasorum is a nematode that resides as the adult stage in the pulmonary arteries and the right side of the heart in dogs and other carnivores (excluding cats).

The distribution of *A. vasorum* includes endemic areas in several European countries. However, former reports of isolated endemic foci are being increasingly replaced by the description of larger endemic areas, involving dogs and wildlife. Foxes in particular are considered an important reservoir, with wolves, coyotes and jackals being further potential sources of infection.

Figure 19: *A. vasorum* first-stage larvae measure approximately 345 µm and are characterised by a wavy tail with a dorsal notch

Like other metastrongylids, the life cycle of *A. vasorum* includes some species of slugs and snails as intermediate hosts. Dogs acquire infection through the ingestion of intermediate hosts or frogs or possibly birds acting as paratenic hosts (Figure 20). Another route of infection to be considered, is the direct oral ingestion of third-stage larvae released into the environment by snails (e.g. by drinking water from puddles or by eating grass). Under experimental conditions, it has been shown that third-stage larvae of *A. vasorum* and *C. vulpis* can remain infective for at least eight weeks after being excreted by snails. Following the ingestion of infective L3 by the definitive host, the larvae moult twice and migrate to the right side of the heart and pulmonary artery, where they mature into adult stages. Female worms begin to produce larvated eggs 6–8 weeks after infection (prepatency). The first-stage larvae, which hatch rapidly from the eggs, migrate through the vessels into the lung parenchyma and after passing through the lung capillaries, are then transported retrogradely via the tracheal ciliated epithelium into the oral cavity or coughed up, then swallowed and excreted with the faeces (see Figure 20). Without anthelmintic treatment, lifelong infections can persist.

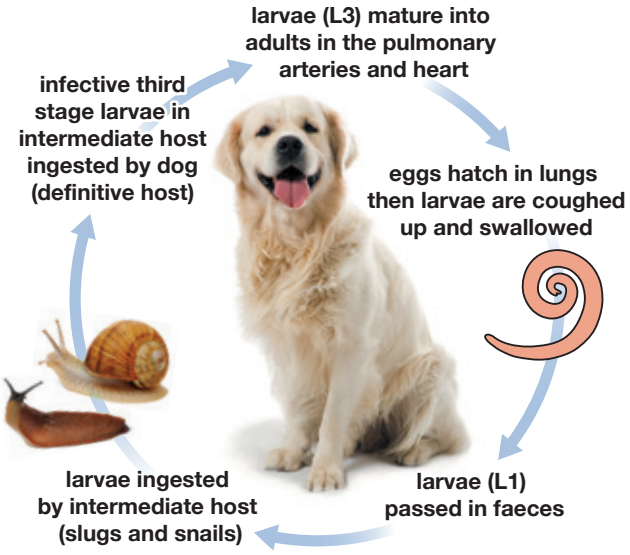


Figure 20: *Angiostrongylus vasorum* life cycle

Clinical manifestations of *A. vasorum* infection in dogs are variable. Naturally-infected subclinical dogs are reported but respiratory signs such as coughing and dyspnoea induced by verminous pneumonia are frequently observed, complemented by bleeding disorders, neurological, gastrointestinal or non-specific signs. In chronic infections, anorexia, anaemia, weight loss, depression, pulmonary hypertension and signs of coagulopathy (e.g. melaena, haemoptysis, prolonged bleeding from minor injuries and subcutaneous haematomas) can be seen. Occasionally, sudden death may occur.

In rare cases, both larvae and, less commonly, adult stages of *A. vasorum* can be found in ectopic locations such as the brain, bladder, kidney or anterior chamber of the eye. This may result in clinical signs relating to the invasion of these organs.

Diagnosis can be performed by detecting first-stage larvae from (at least) 4 g of fresh faeces using the Baermann method. Faeces are preferentially sampled from three consecutive defecations (collected over 1–2 days) due to large variation in larval excretion. However, samples should not be pooled and should be as fresh as possible when examined. Alternatively, microscopic detection of first-stage larvae in bronchial lavage material can be used, if carried out for additional investigations. In cases of severe *A. vasorum* infection, first-stage larvae can even be detected in a rectal faecal smear. Furthermore, serology, in particular a commercial serological test for detection of circulating antigen with high specificity, is available.

Material obtained through broncho-alveolar lavage can be examined by microscopy for larval detection and possibly confirmed by genetic methods. In cases of suspected pulmonary inflammation, imaging (thoracic radiography, computed tomography) should be performed as a preliminary and complementary investigation.

Anthelmintic therapy includes the use of a macrocyclic lactone-based anthelmintic with varying treatment protocols or repeated daily administration of a benzimidazole-based anthelmintic (for three weeks). In severe cases, supportive treatment, including glucocorticoid-based products, blood substitute fluids, measures to control bleeding, antibiotics and oxygen may be needed and the animal should be rested during the treatment period (at least two to three days).

In local areas of high endemicity and/or if the dog is exposed, e.g. used for hunting or eats grass, slugs or snails ("hoovers"), prevention can be achieved with the monthly administration of macrocyclic lactones.

For further information on *A. vasorum* characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2C, 3 and 6.

5. Lungworms (*Crenosoma vulpis*, *Aelurostrongylus abstrusus*)

Infection with *Crenosoma vulpis* is common in dogs in many European countries and red foxes, in particular, serve as reservoir hosts, along with other wild canids. *Aelurostrongylus abstrusus* occurs commonly in cats. Less frequently encountered are infections caused by *Angiostrongylus chabaudi* and *Troglostrongylus brevior*, for which wild cats serve as the reservoir hosts. *T. brevior* infections are more severe and mostly fatal in kittens.

Crenosoma vulpis – the fox lungworm

The adult worms of the nematode *Crenosoma vulpis* reside in the trachea and bronchi of foxes and other wild canids and domestic **dogs**. The parasite is endemic in wild canid populations in Europe, with foxes representing an important reservoir.

The life cycle is similar to other metastrongyloids such as *A. vasorum*, with slugs and snails acting as intermediate hosts. Infection may occur through oral ingestion of infective L3 in gastropods but may also occur through L3 being released into the environment by infected slugs and snails.

Adult *C. vulpis* nematodes are 4–15 mm in length and possess a distinctive anterior end characterised by a cuticula with approximately 20 overlapping folds bearing small spines. The female worm is ovo-viviparous, i.e. first-stage larvae (L1) develop quickly within a thin eggshell and hatch within the host while being coughed up and excreted in faeces. The prepatent period is about three weeks. L1 measure approximately 300 µm with a straight tail, ending in a simple spike. Adult worms live for about 10 months. Dogs infected with *C. vulpis* show catarrhal bronchitis with eosinophilia and a broncho-interstitial pattern. The main clinical signs are coughing and tachypnoea, dyspnoea and sneezing, accompanied by unspecific signs such as a poor general condition and fever.

As with *A. vasorum*, diagnosis can be performed by detecting L1 in at least 4 g (5–10 g) of fresh faeces using the Baermann method. Due to variation in larval excretion, samples should be preferentially collected from three consecutive defecations. Alternatively, microscopic detection of L1 in bronchial lavage material can be used and confirmed by genetic methods.

Several anthelmintic compounds can be used to treat *Crenosoma* infections e.g. macrocyclic lactones as well as fenbendazole for 3–5 consecutive days are used. Following treatment, the prognosis is generally very good. To prevent infection, controlling intermediate hosts by collecting and removing gastropods from yards and preventing dogs from eating slugs and snails are recommended preventative measures.

For further information on *C. vulpis* characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2C, 3 and 6.

Aelurostrongylus abstrusus – the cat lungworm

Aelurostrongylus abstrusus is a metastrongylid nematode that inhabits the respiratory bronchioles, alveolar ducts and alveoli as the adult stage. It may also be found in subpleural nodules up to 10 mm in size in the lung parenchyma.

A. abstrusus is up to 10 mm long and very thin (less than 100 µm). In Europe *A. abstrusus* is endemic, with a reported prevalence of up to 30% in some countries. It is the most prevalent lungworm in domestic cats, considered to be species-specific and referred to as the "cat lungworm". *A. abstrusus* is a threat to all domestic cats that have regular outdoor access, irrespective of their age and gender. Feral and stray cats are at a higher risk of infection. The nematode may also infect wild felids (e.g. European wildcats, *Felis silvestris silvestris*) that prey on rodents and birds and live in sympatry with domestic cats.

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 eating molluscs infected with L3. Cats become infected after the ingestion of infective larvae (L3) in paratenic hosts or by direct ingestion of infected molluscs, i.e. during grooming. The prepatency period is 5–6 weeks.

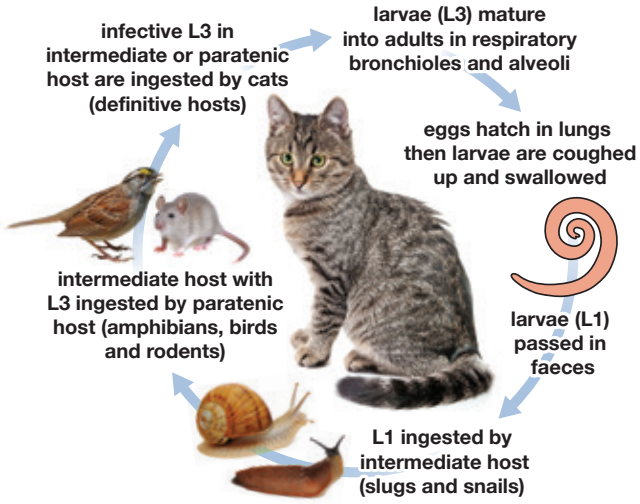


Figure 21: *Aelurostrongylus abstrusus* life cycle

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 have subclinical infections. In severe cases, signs of bronchitis and pneumonia may develop. If left untreated, the infection can be fatal.

Diagnosis relies upon detecting L1 from (at least) 10 g of fresh faeces using the Baermann method. The diagnostic sensitivity through larval detection decreases in cases of chronic or repeated infections, due to interrupted larval shedding. However, quantitative information on the number of excreted larvae correlates with the severity of the alterations observed by diagnostic imaging. First-stage larvae can also be detected in bronchial lavage fluid. However, this diagnostic method is less sensitive and should only be considered if BAL is being carried out for additional investigations. In cases of suspected pulmonary inflammation, imaging (thoracic radiography, computed tomography) should be performed as a preliminary and complementary investigation. This may reveal disseminated interstitial and peribronchial changes, although not differentiating from other feline lungworm infections or other bronchopulmonary diseases.

Treatment options to control a diagnosed infection of *A. abstrusus* include the use of macrocyclic lactones and emodepside-containing preparations, some of which must be used repeatedly.

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

For further information on *A. abstrusus* characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 4, 5 and 7.

6. Hookworms (*Ancylostoma* spp. and *Uncinaria* spp.)

Hookworms are small nematodes (ca. 0.5–1.5 cm) characterised by large mouthparts that are at an angle to the rest of the worm, hence the common name. There are three significant species in Europe: *Ancylostoma caninum* (dogs), *Ancylostoma tubaeforme* (cats) and *Uncinaria stenocephala* (dogs and rarely cats).

U. stenocephala, known as the northern hookworm, tolerates colder climates than *A. caninum* and is found throughout Europe. *A. caninum* is found predominantly in central and southern Europe and *A. tubaeforme* is found throughout continental Europe.

The adult worms (Figure 22) inhabit the small intestine and have a direct life cycle with eggs passed in the faeces developing to third-stage larvae (L3) in the environment. When these are ingested, they develop within two to four weeks to adult, egg shedding worms (Figure 23).

Hookworms, most notably *Ancylostoma* spp. larvae, can be transmitted through milk from the lactating mother to the puppies and are also capable of penetrating skin and thus making their way to the intestine. Lactogenic transmission does not occur in *U. stenocephala*; percutaneous infection is possible, but at a lower rate compared to *Ancylostoma*.

All species feed by grasping the intestinal mucosa with their mouthparts and damaging the surface to obtain nutrients: largely blood in the case of *Ancylostoma* spp., as they require oxygen from the blood, whilst *U. stenocephala* worms mostly obtain nourishment from tissue components on the surface of the intestine.

Diarrhoea, weight loss and anaemia are the common clinical signs, and in the case of *A. caninum* and *A. tubaeforme*, 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. *Ancylostoma* species can cause significant anaemia when present in high numbers or over a period of time. Lactogenic transmission of larvae by *A. caninum* can result in acute anaemia and even the death of young pups. *U. stenocephala* is less pathogenic.

Figure 22: Hookworms are small nematodes that live in the intestine of infected dogs and cats

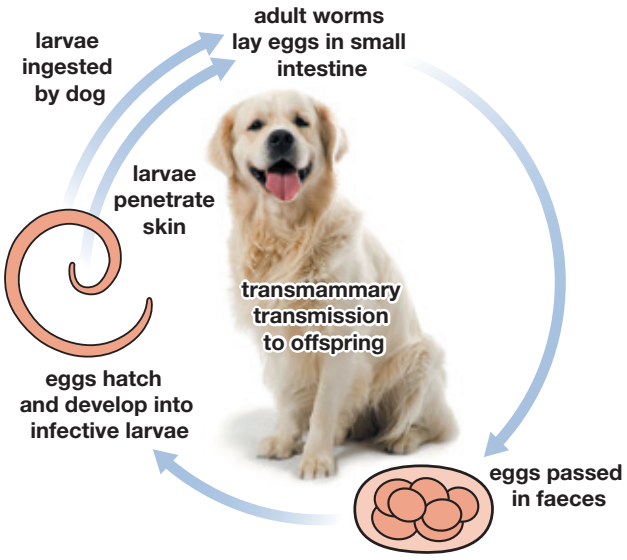


Figure 23: Hookworm life cycle

Immunity develops after exposure, but is unlikely to be absolute. Infection thrives best where animals have access to outdoor environments such as kennel runs. Diagnosis is based on identifying hookworm eggs in fresh or fixed faecal samples using a flotation method, although the eggs of the two genera are indistinguishable (Figure 24). A coproantigen ELISA, based on the detection of hookworm antigen, is also available and is usually positive before egg excretion (see ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines).

When hookworms are detected, anthelmintic treatment should be administered. Care must be taken to ensure that the treatment has a sufficient effect on the hookworm species identified, as there can be significant differences in efficacy for the various active ingredients. Diagnosis in young puppies can be complicated by signs of disease occurring before infection is patent, i.e. before eggs are passed in faeces (but coproantigen detection may be positive). Where young animals are clinically affected by the infection, supportive therapy may be necessary in addition to anthelmintic treatment. After exposure, immunity develops, but this is not complete. For this reason, animals that are exposed to a high infection pressure (e.g. puppies and young animals in breeding centres) should be dewormed regularly. If possible, animals should be removed from contaminated environments during decontamination.

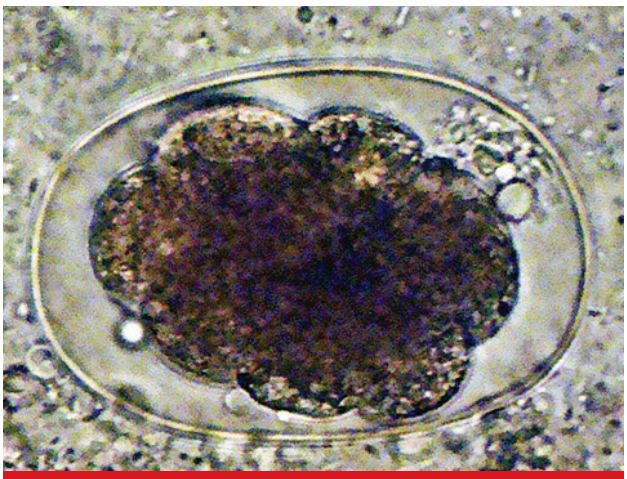


Figure 24: Infection can be diagnosed by faecal examination and identification of eggs, but only at the family level (as Ancylostomatidae)

For further information on hookworm characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2A and 3–7.

7. Whipworm (*Trichuris vulpis*)

Trichuris vulpis, a nematode reaching lengths of up to 8 cm, inhabits the large intestine of dogs. Its posterior end is enlarged giving it a distinctive 'whip-like' shape (Figure 25). *T. vulpis* is most likely to occur in central and southern parts of Europe where temperatures are suitable for the environmental development of eggs and in specific premises, such as kennels and animal shelters. Considerable and persistent contamination of the environment with infective eggs can occur. Control can therefore be difficult, as dogs may become repeatedly re-infected if they remain in the same environment.

Figure 25: *Trichuris vulpis* worm

The lemon-shaped eggs are passed in the faeces of infected dogs and the infective L1 develops within the egg in one to two months at temperatures above 4°C. The larvae are protected by the eggshell and can survive in the environment for years. Dogs become infected when they ingest infective eggs (Figure 26). The prepatent period is 9–10 weeks, after which infected dogs may continue to shed eggs for up to a year.

The worms are anchored, by their thinner anterior part, to the mucosa of the large intestine, where they feed on mucosal cells and blood. A heavy infection (Figure 27) will result in diarrhoeic, bloody, mucus-filled faeces accompanied by weight loss and ultimately, the animal will no longer be able to compensate and will develop electrolyte imbalances including hyponatraemia, also defined as 'pseudo-addison disease'.

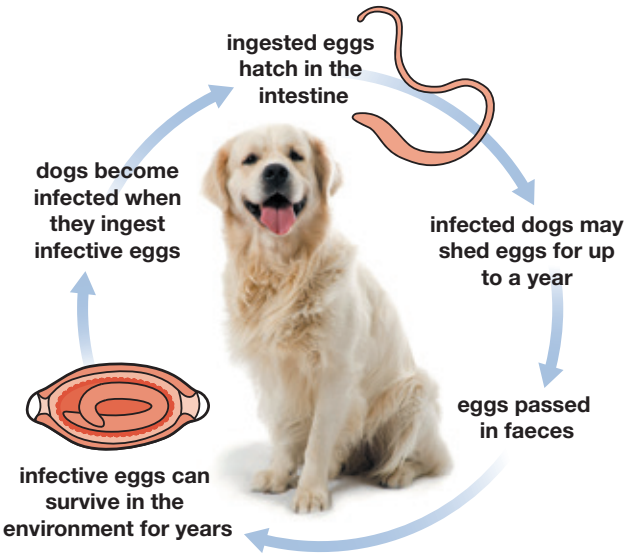


Figure 26: *Trichuris vulpis* life cycle

Infection can be diagnosed by finding characteristic "lemon-shaped" eggs (Figure 28) on examination of 4–10 g of faecal samples using a suitable flotation technique. A coproantigen immunoassay for the detection of *Trichuris* antigen has also been available for some time. This enables the detection of a whipworm infection three weeks after infection and therefore up to six weeks before the onset of patency.

Most modern anthelmintics are effective against *T. vulpis* but to be effective, repeated deworming (3–5 days, according to package leaflet) is required. Where possible, dogs should be removed from contaminated areas and put on repeated anthelmintic treatment. 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, e.g. flame burning. Rotavating and reseeding may also help to eliminate contamination.

For further information on *T. vulpis* characteristics, risk factors, clinical signs, diagnosis and treatments, see Tables 2A, 3 and 6.

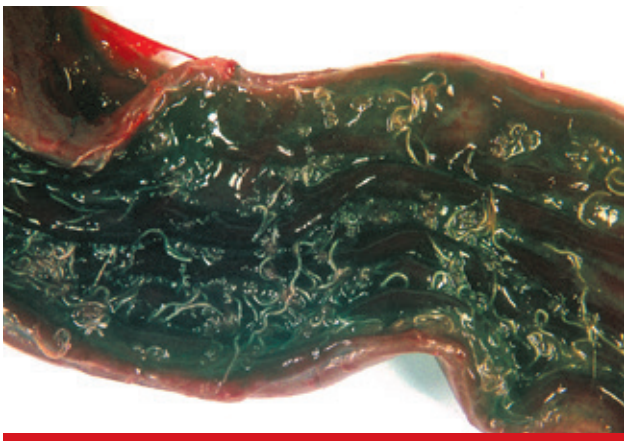


Figure 27: A heavy infection of *Trichuris vulpis* in the large intestine of a dog

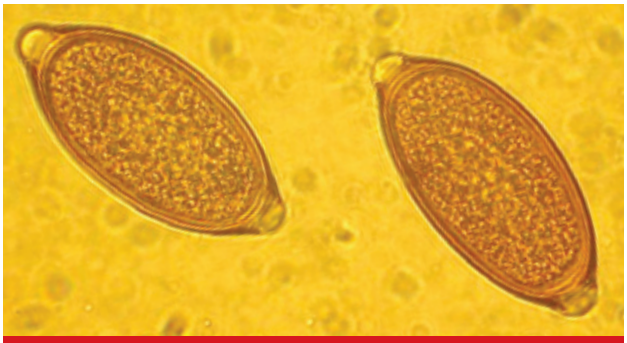


Figure 28: *Trichuris vulpis* eggs

8. Threadworm (*Strongyloides stercoralis*)

Strongyloides stercoralis is a worldwide zoonotic nematode of canids and primates. The slender adult worms (3–8 mm) reside in the small intestine. It is most commonly found in tropical and subtropical regions but may be diagnosed also in temperate regions or in Northern Europe. Cats become infected with *S. stercoralis* less commonly than dogs. In its complex life cycle, only female worms are parasitic. The adult females are embedded in the intestinal mucosa and reproduce asexually by parthenogenesis, releasing eggs into the gut lumen. These eggs may quickly hatch and the resulting L1 (rhabditiform larvae) are then shed with the faeces. The L1 may then develop into L3 in the soil within 24 hours, or mature into non-parasitic female and male worms capable of reproducing sexually in the environment. This process can lead to massive multiplication and contamination of the surrounding area. The L3 infect the host via percutaneous, oral or lactogenic transmission. Autoinfection, where L1 larvae develop into L3 within the same dog, seldom occurs.

Strongyloides infections are often moderate and subclinical, with disease occurring mainly in massively challenged neonates and nurslings. Severe infections in sick dogs can lead to pneumonia and watery to mucous 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 early stages of the disease. Infections are usually associated with warm, wet, crowded, unsanitary housing.

Diagnosis of *S. stercoralis* in dogs relies upon the detection of L1 in faeces by the Baermann method. Samples must be as fresh as possible, and multiple samples increase diagnostic sensitivity. Larvae may only occasionally be observed by flotation. Specificity is linked to the morphological identification of L1 (e.g. long rhabditiform oesophagus and straight tail, buccal capsule and genital primordium), differentiating them from other parasitic or free-living species. Occasionally, larvated eggs (50–60 × 30–35 µm) may be identified by flotation of fresh faeces. Faecal PCR assay is also offered by some diagnostic laboratories.

Breeding kennels and animal trade seem to play an important epidemiological role in the dissemination of *S. stercoralis*. Since routine faecal flotation methods have low sensitivity for detection of L1, and shedding of eggs is uncommon, the prevalence, as well as the clinical significance of *S. stercoralis* in dogs may be underestimated. Poor sanitation and mixing of susceptible and infected dogs can lead to a rapid buildup of the infection in all dogs in a kennel or pen. Dogs with diarrhoea should be promptly isolated from dogs 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.

In recent years, molecular and epidemiological studies have suggested that both dog-adapted and zoonotic populations of *S. stercoralis* may exist. Because the disease in humans can be serious, caution should be exercised when handling infected dogs. The disease in humans (as in dogs) is much more likely to be severe if the patient is immunosuppressed.

Infections in dogs can be tentatively treated with ivermectin (0.2 mg/kg/day orally for two consecutive days, off-label, definitively not recommended in ivermectin-sensitive dogs, to be tested prior to treatment) or fenbendazole (50 mg/kg/day orally for five days, repeated four weeks later). In isolated cases, higher doses, intramuscular administration of ivermectin, repeated treatments or even a combination of ivermectin and fenbendazole have been used to eliminate infection. In cats, fenbendazole (50 mg/kg/day orally for three days) can be administered. These are not approved regimens in either cats or dogs. In all animals, faeces should be examined regularly for at least six months after treatment to confirm efficacy.

DIAGNOSIS OF HELMINTH INFECTIONS

Patent infections of all worms mentioned can be identified by faecal examination, except for *D. immitis* and *D. repens* where a blood sample is examined for microfilariae and antigens (dogs). Faecal examination for worm eggs should be carried out with at least 5–10 g of fresh faeces and can be conducted using flotation techniques with solutions of appropriate density (Tables 6 and 7 and ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines). The analysis of faecal samples collected over different days increases the sensitivity of the employed methods. Coproantigen ELISAs have also been available for some time for the detection of *Toxocara* spp., *Trichuris vulpis* and hookworms.

Eggs of ascarids, hookworms, whipworm and most tapeworms are easily recognisable. In some cases, worm burden can be crudely estimated from the number of eggs present in the sample. However, it should be noted that for ascarids such as *Toxocara*, a negative correlation between fecundity per worm and number of adult worms has been reported. Furthermore, there is poor correlation between taeniid infection and the detection of eggs in faeces. Since dogs may ingest or eat faeces, care should be taken to identify and eliminate false-positive results caused by coprophagia, which can be ruled out by repeated faecal examinations for eggs or adding helminth coproantigen testing (if available) into the examination protocol.

Where larvae (L1) are produced (lungworms, *A. vasorum*, *Strongyloides stercoralis*), faecal samples should be examined using the Baermann technique (Tables 6 and 7 and ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines). If possible, faeces should be sampled on three consecutive defecations due to variation in larval excretion. Faeces should be collected from a fresh sample and not from the ground in a kennel or run. Differentiation of the metastrongylid L1 is based on size and morphology of the tail. Re-testing is recommended approximately three weeks after starting the anthelmintic treatment(s) to check that treatment has resulted in the removal of the adult worms. Alternatively, a commercially-available test for the serological detection of circulating antigens of *A. vasorum* can be used for suspected clinical cases. Dogs, clinically affected by angiostrongylosis, should be further investigated to evaluate pulmonary and circulatory status and clotting parameters. In suspected cases, several methods are needed to increase sensitivity (e.g. serology, Baermann, BAL including PCR).

In principle, a thorough diagnostic examination should be carried out at least once a year with the aim of determining the status, irrespective of any deworming.

IMPACT OF PET HEALTH AND LIFESTYLE FACTORS

The type and frequency of diagnostic, preventive and therapeutic measures need to be tailored to suit individual needs based upon where and how the animal is kept. When recommending a parasite management programme, veterinarians should consider the following (see Tables 3 and 5 for more details).

The animal

Age: puppies, kittens and geriatric animals are at greater risk than healthy adults.
Reproductive status: pregnant bitches may pass *T. canis* larvae to the foetus in utero.
Lactation: lactating bitches may pass *T. canis* to their suckling pups via milk (lactating bitches often have patent *T. canis* infections as they become infected by their offspring). Lactating queens can pass *T. cati* to their sucking kittens via milk. *A. caninum* infections can also be transmitted to pups via milk.
Health status: e.g., ectoparasite infestation.

Environment/use of the animal

Shared accommodation: animals kept in kennels, shelters or breeding stations or those living with other dogs or cats are at greater risk of acquiring parasites and may require special consideration.
Roaming: dogs and cats who live outdoors or those with unrestricted access to the outdoors are at greater risk of acquiring parasites.
Working dogs: hunting and working dogs may also be at a greater risk.

Nutrition

Dogs and cats with access to the following may be at risk of acquiring specific parasites:
Rodents
Slugs and snails
Raw fish
Raw meat including viscera without appropriate heating or freezing
Carcasses, placenta or aborted foetuses

Feeding raw meat (barfing)

Various parasites can be transmitted to dogs and cats via raw meat and offal (e.g. liver, lungs). These include the dangerous dog tapeworm (*Echinococcus granulosus* group), various *Taenia* species, roundworms (*Toxocara* spp.), the pathogen causing toxoplasmosis (*Toxoplasma gondii*) and other single-celled parasites such as *Sarcocystis* spp. and *Neospora caninum*. The risk of dogs and cats becoming infected with these and other pathogens via raw meat is still unknown. The only certainty is that it is possible and that it happens repeatedly. However, this does not mean that dogs and cats should never be fed raw meat from a parasitological point of view but it is important that raw meat is frozen thoroughly and for a sufficient length of time to kill any parasite stages. It is recommended that meat is kept frozen at -17°C to -20°C for at least one week. In addition, meat should be sourced locally, as importing it may introduce pathogens not endemic to the region (e.g. *Echinococcus granulosus* is largely absent from northern Europe). If the source and freezing status of acquired raw meat is unknown, routine faecal testing every four weeks or prophylactic deworming with a roundworm-specific product is recommended. Due to the low certainty of detection of tapeworm infections using faecal tests, treatment against tapeworms is always advisable. Even if the risk of infection via raw meat is not particularly high, the health consequences of infections for animals and humans can be considerable. It is not possible to protect against protozoa transmitted via barfing by using antiparasiticides.

Location and travel

Dogs and cats living in or travelling to specific geographical areas (e.g. for holidays, relocation, boarding facilities, shows and field trials) may be at increased risk of acquiring infections that occur in those areas. Non-endemic diseases can be a diagnostic challenge for veterinarians who are unfamiliar with them. Dogs imported from areas endemic for particular parasites (e.g. *E. multilocularis*) should be promptly visited by a veterinarian and treated with an appropriate anthelmintic.

In each case, diagnostic methods can be used to verify the success of the prevention measures taken and medication chosen.

RESISTANCE TO ANTHELMINTICS

To date, there have been virtually no proven cases of anthelmintic resistance to intestinal and extraintestinal nematodes in dogs and cats in Europe. However, in the USA, anthelmintic resistance of *D. immitis* larvae is recognised and there are several studies suggesting that drug resistance is present in *Ancylostoma caninum* populations in Australia and the USA. Recent studies also report on single resistant *Toxocara canis* and *Dipylidium caninum* worm populations in the USA. In a single dog from Spain imported to Switzerland, apparent *Dipylidium caninum* praziquantel resistance was observed.

Traditional anthelmintic treatment of dogs and cats has always left many parasite stages outside the definitive host that are unselected for resistance by treatment. Based on experience from large animal practice, where resistance has been widely proven to exist, the likelihood of resistance developing is presumably higher in larger dog and cat populations such as in animal shelters, large breeding establishments or similarly intensive forms of husbandry. Higher frequency of anthelmintic treatment could increase the selection pressure for resistance, particularly in kennel settings where groups of dogs or cats are treated simultaneously with the same product. The aforementioned resistance in hookworm populations in the USA also occurred in intensively-kept, frequently-treated (and partially-insufficiently-dosed) greyhounds but also in dogs of other breeds.

It is therefore recommended that worm control in larger dog and cat populations such as in animal shelters, large kennels or similarly intensive forms of husbandry should be carefully planned and accompanied by the regular examination of faecal samples. The aim is to diagnose the worm species present and to continuously monitor the effectiveness of the measures taken. This also includes carrying out random faecal sample tests following deworming treatments, in order to confirm the success of the adopted treatments, and thus the absence of reduced anthelmintic efficacy or anthelmintic resistance. In addition, it is essential that accompanying measures, particularly regarding hygiene, are implemented to limit deworming frequency to an appropriate level for preventive healthcare purposes.

ENVIRONMENTAL CONTROL OF PARASITE TRANSMISSION

For parasites whose eggs or larvae are passed in the faeces, the control of parasite stages in the environment is essential to minimise the infection risk to other animals or humans (zoonotic parasites).

Parasitic contamination of the environment can occur in several ways, including the excretion of parasitic eggs or larvae in the faeces and the release of cestode proglottids.

Environmental infection pressure of dog-transmitted parasites can be maintained by wild foxes and stray dogs in both rural and urban areas. Similarly, feral and wild cats can form a reservoir for feline infections.

The infection of intermediate or paratenic hosts (i.e. birds, rodents, slugs and snails) can contribute to a longer survival time of parasitic stages in the environment.

Most environmental parasite stages are highly resistant to environmental degradation (from months to years). Freshly-excreted stages of many parasites can be directly infective (e.g. *Taenia* spp., *Hydatigera* spp. and *Echinococcus* spp. eggs). Other parasites, such as nematode eggs, require anything from a few days to a few weeks at appropriate temperatures, usually above 16°C, to reach the infective stage. It is therefore important to prevent initial parasite environmental contamination by implementing comprehensive parasite control programmes based on local epidemiological knowledge.

- The safe disposal of animal faeces is essential. This should be on a daily basis and faeces should not be flushed down the toilet or disposed of in compost intended for edible crops. In countries or regions where legislation permits, faeces can be disposed of in household waste collections or dedicated “poo bins”.
- Measures to facilitate faecal removal, such as the provision of disposal bins and bags should be encouraged. As it is difficult to control where outdoor cats defecate, particular attention should be given to worm control in cats.
- Leash-control and faecal clean-up laws should be enforced by local authorities, especially in urban areas.
- Legislation to control stray dogs and feral cat populations should also be enforced by the appropriate authorities.
- Parasitised animals should be treated to minimise environmental contamination. In justified cases, animals should be monitored by faecal examination (e.g. animals with persistent clinical signs or suspected resistance).
- Because eggs may persist in the soil for months or years in heavily contaminated areas, such as in highly populated kennels, extreme measures are needed for decontamination, including the removal of sand/soil or covering the soil with concrete or asphalt.
- In kennels or multi-animal households, the strict treatment and quarantine of new entrants is essential to avoid the introduction of infected animals.
- Children’s playgrounds should be well fenced to prevent entry of animals, especially cats. Sandboxes should be covered when not in use. Sand, particularly if it is uncovered, is likely to have been contaminated with faeces, and should therefore be replaced regularly e.g. at least once or twice a year.
- Desiccation and ultraviolet light are highly detrimental to worm eggs, so allowing exposure to sunlight and drying of contaminated areas can assist in reducing the level of contamination.

OWNER CONSIDERATIONS IN PREVENTING ZOONOTIC DISEASES

Since some dog and cat parasites can also potentially cause infection in humans, veterinarians have an additional responsibility for human health. A particular zoonotic risk comes from the widely-present *Toxocara* spp. roundworms: after oral ingestion of infective eggs, the larvae can perform a somatic migration (larva migrans complex). If larvae become blocked in the human eye, nerve tract and/or brain during migration, serious health problems can occur.

After infection with *E. multilocularis* or *E. granulosus* group species, humans develop alveolar or cystic echinococcosis, respectively, with formation of cysts in the liver and/or other organs, with potentially fatal outcomes. Alveolar echinococcosis is a carcinoma-like disease, which without treatment can have fatal consequences. Human infection occurs as a result of oral ingestion of worm eggs. The main source of contamination of the environment is the fox. Infection can also occur by the ingestion of eggs found on a dog’s fur or of eggs that have been excreted in dog faeces.

Important preventive measures for pet owners include:

- Practicing good personal hygiene, particularly washing hands after handling pets and before eating food.
- Minimising the exposure of children in particular to potentially-contaminated environments and teaching them good personal hygiene. Keeping nails short. Teaching children the importance of such practices.
- Wearing gloves when gardening.
- Washing raw fruit, vegetables and mushrooms before eating.
- Controlling pet parasite infections through repeated treatments and/or regular diagnostic testing.
- Preventing infection by reducing, where possible, the risk of the pet acquiring infection.
- Cleaning up pet faeces regularly to reduce environmental contamination with infective parasite stages. Not disposing of faeces or cat litter in recyclable waste or compost.
- Grooming dogs regularly to minimise the risk of coat contamination with worm eggs.
- Changing shoes to prevent contamination of domestic areas.

People who are in regular contact with animals that may potentially transmit zoonotic parasites should be made aware of the risks and advised that these health risks are greater for pregnant women and those suffering from underlying illnesses or immunosuppression. This information should be made available through physicians and veterinarians, without the need for a medical history of the client and his/her family.

With this in mind, special care should be taken in the case of:

- Immunocompromised individuals such as the elderly, diabetics, people with HIV-infection and those undergoing immunosuppressive chemotherapy, organ transplantation or treatment for autoimmune diseases.
- Other susceptible groups such as pregnant women, babies, toddlers and those with learning disabilities.
- People with occupational risks such as farmers, kennel workers and hunters.

STAFF, PET OWNER AND COMMUNITY EDUCATION

Protocols and recommendations for the control of parasitic infection should be communicated clearly to veterinary and para-veterinary staff and consistently applied.

Cooperation between the medical and veterinary professions should be encouraged wherever possible and its benefits underlined in the case of zoonoses. Pet owners should be made aware of the potential health risks of parasitic infection, not only to their pets but also to themselves and their family and friends. Professional brochures and posters placed in veterinary practices and pet shops are useful tools to facilitate this, as are websites.

The importance of regular anthelmintic treatment or joining a “pet health-check programme” should be made clear to the general public by veterinary surgeons, veterinary nurses and other animal health professionals and promoted consistently. Responsible dog and cat ownership can ease public health concerns and encourage the acceptance of dogs and cats as human companions.

Additional information and resource materials can be obtained from www.esccap.org

Table 2A: Characteristics of worms of dogs in Europe: intestinal nematodes

Worm species	Prepatent period	Patent period	Infective stages and route of infection	Distribution in Europe	Definitive hosts
Roundworms or ascarids					
Toxocara canis	Variable, typically 16–21 days after prenatal infection; 27–35 days after lactogenic infection; 32–39 days after ingestion of eggs	4–6 months, can be longer depending on immune status, e.g. in puppies	Ingestion of embryonated eggs from soil or on fur, larvae in milk or paratenic hosts In utero from dam	Everywhere	Dogs and foxes
Toxascaris leonina	About 8 weeks	4–6 months	Ingestion of embryonated eggs from soil or larvae from paratenic hosts	Everywhere	Dogs, cats and foxes
Hookworms					
Ancylostoma caninum	2–3 weeks	6 months, can be prolonged depending on immune status (7 months to 2 years)	Ingestion of L3 from environment, larvae in bitches' milk or paratenic hosts Percutaneous infection by larvae	Predominantly southern Europe, sporadic in other parts of Europe	Dogs and foxes
Uncinaria stenocephala	3–4 weeks	4–6 months, can be prolonged depending on immune status	L3 orally (minor: subcutaneously) from environment	Predominantly central and northern Europe	Dogs and foxes (and cats)
Threadworms (*Strongyloides*)					
Strongyloides stercoralis	Variable, from 9 days	Several months (3–15 months)	L3 orally from environment or through milk Percutaneously Auto-infections (rarely in dogs)	Rarely everywhere but more predominant in southern and eastern Europe	Dogs (and humans and cats)
Whipworm					
Trichuris vulpis	At least 8 weeks	Up to 18 months	Ingestion of embryonated eggs from the environment	Everywhere	Dogs and foxes

Table 2B: Characteristics of worms of dogs in Europe: tapeworms (cestodes)

Worm species	Prepatent period	Patent period	Infective stages and route of infection	Distribution in Europe	Definitive hosts
Tapeworms					
***Taenia* spp.**	4–10 weeks	Months up to several years	Ingestion of larval stages (cysticercus or coenurus type) in intermediate hosts	Everywhere, with differences depending on the species	Dogs and foxes (and cats)
***Mesocestoides* spp.**	4–10 weeks	Several years	Ingestion of larval stages in meat or tissues of prey	Everywhere	Dogs, cats and foxes (humans)
Dipylidium caninum	3 weeks	Several months, up to three years	Ingestion of larval stages in fleas or lice	Everywhere	Dogs, cats and foxes (humans)
Echinococcus granulosus* complex	5–8 weeks	Several months	Ingestion of larval stages in intermediate hosts (herbivores and omnivores)	See map (Figure 9)	Dogs (foxes)
Echinococcus multilocularis	28 days	Several months	Ingestion of larval stages in intermediate hosts (rodents)	See map (Figure 10)	Foxes, dogs, racoon dogs (and cats)

* There are different species and strains: *E. ortleppi* (cattle), *E. equinus* (horse), sheep-, pig-, cervid- and other strains, see Figure 9 for distribution.

Table 2C: Characteristics of worms of dogs in Europe: non-intestinal nematodes

Worm species	Prepatent period	Patent period	Infective stages and route of infection	Distribution in Europe	Definitive hosts
Heartworm					
Dirofilaria immitis	6–7 months	Several years	L3 transmitted by mosquito vector (intermediate host)	Southern Europe and parts of Central Europe, see map (Figure 18)	Dogs (and cats) and ferrets
French heartworm					
Angiostrongylus vasorum	6–8 weeks	Up to 5 years	L3 within mollusc or paratenic host, infection orally	Everywhere in endemic foci	Foxes and dogs
Lungworms					
Oslerus osleri	10 weeks	Unknown	Direct oral transmission from bitch to pups mostly by coprophagia	Everywhere sporadically	Foxes and dogs
***Filaroides* spp. (*F. hirthi, F. milksi*)**	10–18 weeks	Unknown	Direct oral transmission from bitch to pups mostly by coprophagia	Everywhere sporadically	Dogs
***Eucoleus aerophilus* (syn. *Capillaria aerophila*)**	4 weeks	10–11 months	Ingestion of larvae or infective eggs from environment or via earthworms	Everywhere	Foxes, dogs and cats
Crenosoma vulpis	3 weeks	10 months and longer	L3 within mollusc or paratenic hosts, infection orally	Everywhere	Dogs and foxes
Subcutaneous worms					
Dirofilaria repens	6–8 months	Several years	L3 transmitted by mosquito vectors (intermediate hosts)	Southern Europe and parts of Central Europe, see map (Figure 18)	Dogs (cats, humans)
Eye worms					
Thelazia callipaeda	About 3 weeks	Months to years	Arthropod dipteran vectors (intermediate hosts) while feeding lachrymal fluids	Italy, France (Dordogne), southern Switzerland, Spain, Portugal, Balkan area and Hungary	Dogs, cats and foxes (humans)
***Spirocerca lupi* (oesophagus worm)**	6 months	Several months	Ingestion of infective larvae in intermediate hosts (coprophagus insects) and paratenic hosts (rodents, lizards)	Everywhere (rare)	Dogs (cats)

Table 3: Risk factors for worms of dogs in Europe. Shaded boxes indicate increased risk.

Some dogs are more likely to have parasite infections than others, although the difference is rarely absolute. This table highlights those factors that are likely to increase the probability of dogs carrying specific parasites. It has been drawn up on the basis of available understanding but is not the result of a formal risk assessment. Shaded boxes indicate increased risk.

Worm species	Dog type			Health	Environment		Nutrition			Location and travel
	Pup	Lactating bitch	Stray	Fleas or lice	In kennels	Outdoors	Rodents/ amphibians/ reptiles	Molluscs	Raw meat/ viscera	
INTESTINAL WORMS										
Ascarids										
Toxocara canis	■	■	■		■	■	■		■	
Toxascaris leonina			■		■	■	■			
Hookworms										
Ancylostoma caninum	■	■	■		■	■	■			More in southern Europe
Uncinaria stenocephala			■		■	■	■			Central and northern Europe
Threadworms (*Strongyloides*)										
Strongyloides stercoralis	■	■			■	■				More in southern and eastern Europe
Whipworm										
Trichuris vulpis			■		■	■				
Tapeworms										
***Taenia* spp.**			■			■	■		■	
***Mesocestoides* spp.**			■			■	■			
Dipylidium caninum			■	■	■					
Echinococcus granulosus*			■						■	Central, southern and eastern Europe, see map (Figure 9)
Echinococcus multilocularis			■			■	■			Central, eastern and northern Europe, see map (Figure 10)
NON-INTESTINAL WORMS										
Heartworm										
Dirofilaria immitis			■			■				See map (Figure 18)
French heartworm										
Angiostrongylus vasorum			■				■	■		
Lungworms										
Oslerus osleri	■	■			■					
***Filaroides* spp.**	■				■					
***Eucoleus aerophilus* (syn. *Capillaria aerophila*)**			■			■				
Crenosoma vulpis			■			■		■		
Subcutaneous worms										
Dirofilaria repens			■			■				See map (Figure 18)
Eyeworms										
Thelazia callipaeda			■			■				Italy, France (Dordogne), southern Switzerland, Spain, Portugal, Balkan area and Hungary

* There are different species and strains: *E. ortleppi* (cattle), *E. equinus* (horse), sheep-, pig-, cervid- and other strains, see Figure 9 for distribution.

Table 4: Characteristics of worms of cats in Europe: nematodes and tapeworms (cestodes)

Worm species	Prepatent period	Patent period	Infective stages and route of infection	Distribution in Europe	Definitive hosts
INTESTINAL WORMS					
Roundworms or ascarids					
Toxocara cati	Variable, usually around six weeks after ingestion of eggs	4–6 months	Ingestion of embryonated eggs from soil, larvae in milk or paratenic hosts	Everywhere	Cats
Toxascaris leonina	8–10 weeks	4–6 months	Ingestion of embryonated eggs from soil, larvae from paratenic hosts	Everywhere	Dogs, cats and foxes
Hookworms					
Ancylostoma tubaeforme	2–3 weeks	18–24 months, can be prolonged depending on immune status	Primarily ingestion of larvae from soil Some percutaneous infection	Continental Europe	Cats
Uncinaria stenocephala	3–4 weeks	4–6 months, can be prolonged depending on immune status	Ingestion of larvae from soil	Predominantly northern and central Europe	Dogs, foxes (and cats)
Other worms					
Ollulanus tricuspis **(stomach worm)**	5 weeks	33–37 days, can sustain due to endogenus autoinfection	Ingestion of larvae or adults in vomitus	Everywhere (rare)	Cats
Tapeworms					
Hydatigera **(syn. *Taenia*)** ***taeniaeformis***	5–10 weeks	Several years	Ingestion of larvae in rodents	Everywhere	Cats
Mesocestoides **spp.**	4–10 weeks	Several years	Ingestion of larval stages in meat or tissues	Everywhere (rare)	Cats, dogs and foxes (humans)
Dipylidium caninum	3 weeks	Several months	Ingestion of larval stages in fleas or lice	Everywhere	Dogs, cats and foxes
Joyeuxiella pasqualei	3–4 weeks	Several months	Ingestion of larval stages in beetles, reptiles and small mammals	Everywhere, predominantly Mediterranean countries	Cats
Echinococcus multilocularis	28 days	Several months	Ingestion of larval stages in intermediate hosts (rodents)	See map (Figure 10)	Foxes, dogs, racoon dogs (and cats)
Liver trematodes					
Opisthorchis felineus	3–4 weeks	Several months	Larval stages (metacercariae) in freshwater fish	North-eastern Germany, locally in central Europe	Cats, foxes, dogs, (humans rarely)

Table 4: Characteristics of worms of cats in Europe: nematodes and tapeworms (cestodes) (continued)

Worm species	Prepatent period	Patent period	Infective stages and route of infection	Distribution in Europe	Definitive hosts
NON-INTESTINAL WORMS					
Heartworm					
Dirofilaria immitis	about 6 months	Rarely occurs with cats, and usually short	L3 transmitted by mosquito vectors (intermediate host)	See map (Figure 18)	Dogs (and cats)
Lungworms					
Aelurostrongylus abstrusus	7–9 weeks	Several years	L3 in mollusc or paratenic host	Everywhere	Cats
***Troglostrongylus* spp.**			L3 in mollusc or paratenic host (and transplacentally)	Italy, Spain, Greece, Portugal	Cats
***Eucoleus aerophilus* (syn. *Capillaria aerophila*)**	4 weeks	10–11 months	Ingestion of larvae or infective eggs from environment or via earthworms	Everywhere	Foxes, dogs and cats
Subcutaneous worms					
Dirofilaria repens	6–8 months	Several years	L3 transmitted by mosquito vectors (intermediate host)	See map (Figure 18)	Dogs (and cats)
Eye worms					
Thelazia callipaeda	About 3 weeks	Several months	Dipteran vectors (intermediate hosts) while feeding lachrymal fluids	Italy, France (Dordogne), southern Switzerland, Spain, Portugal, Balkan area	Dogs, cats and foxes (humans)

Table 5: Risk factors for worm infections in cats in Europe

Some cats are more likely to have parasite infections than others, although the difference is rarely absolute. This table highlights those factors that increase the likelihood of cats carrying specific parasites. It has been drawn up on the basis of available understanding but is not the result of a formal risk assessment. Shaded boxes indicate increased risk.

Worm species	Cat type			Health	Environment		Nutrition			Location and travel
	Kitten	Lactating queen	Stray	Fleas or lice	In cattery	Outdoors	Rodents/ amphibians/ reptiles	Molluscs	Raw meat/ viscera	
INTESTINAL WORMS										
Roundworms or ascarids										
Toxocara cati	■	■	■		■	■	■		■	
Toxascaris leonina			■		■	■				
Hookworms										
Ancylostoma tubaeforme	■		■		■	■	■			Continental Europe
Uncinaria stenocephala			■		■	■	■			
Stomach worm										
Ollulanus tricuspis			■		■	■				■
Tapeworms										
Hydatigera* (syn. *Taenia*) *taeniaeformis			■			■	■			
***Mesocestoides* spp.**			■			■	■			
Dipylidium caninum			■	■	■					
Joyeuxiella pasqualei			■	■	■		■			
Echinococcus multilocularis			■			■	■			Central Europe
Liver trematodes										
Opisthorchis felineus			■			■	Fish		■	North-eastern Germany
NON-INTESTINAL WORMS										
Heartworm										
Dirofilaria immitis			■			■				See map (Figure 18)
Lungworms										
Aelurostrongylus abstrusus			■			■	■	■		
***Troglostrongylus* spp.**			■			■	■	■		Italy, Spain, Greece, Portugal
***Eucoleus aerophilus* (syn. *Capillaria aerophila*)**			■			■	■			■
Subcutaneous worms										
Dirofilaria repens			■			■				See map (Figure 18)

Table 6: Worm infection of dogs: main clinical signs and diagnosis
(for diagnosis: see also ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines)

Worm species	Clinical signs	Material	Diagnosis
INTESTINAL WORMS			
Roundworm or ascarids			
Toxocara canis	Low burden asymptomatic, higher burden may appear as cachexia and pot-bellied appearance in pups; occasionally pneumonia. Large numbers of worms may cause intestinal blockage or intussusceptions.	At least 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Toxascaris leonina	Mostly asymptomatic.	At least 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Hookworms			
Ancylostoma caninum	Diarrhoea, bloody diarrhoea, weight loss and anaemia.	May be acute or chronic signs At least 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Uncinaria stenocephala	Clinical signs rarely occur. In rare cases: diarrhoea, protein loss, weight loss and anaemia.	At least 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Threadworms (*Strongyloides*)			
Strongyloides stercoralis	Heavy infections: watery diarrhoea and occasionally bronchopneumonia.	At least 10 g faeces (fresh or fixed)	First-stage larvae by Baermann funnel method.
Whipworm			
Trichuris vulpis	Asymptomatic but heavy infections associated with anaemia, diarrhoea and weight loss.	At least 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Tapeworms			
***Taenia* spp.**	Asymptomatic, sometimes anal pruritus.	At least 10 g fresh faeces or separate proglottids in faeces, sampling on 3 consecutive days	Proglottids grossly visible with only one genital pore. Taeniid eggs in faeces (see *Echinococcus* below for methods of distinguishing taeniid eggs).
Dipylidium caninum	Mostly asymptomatic, anal pruritus, restlessness.	At least 10 g fresh faeces or separate proglottids in faeces, sampling on 3 consecutive days	Proglottids similar in size to *Taenia* spp. proglottids but morphologically distinct as they have two genital pores. Eggs within proglottids are grouped in egg packets. These can be seen microscopically in faecal samples. Antigen test.
Echinococcus granulosus	Asymptomatic.	At least 10 g faeces, sampling on 3 consecutive days	Morphology and size of proglottids. Egg detection with flotation, sedimentation or combined techniques (not very sensitive and taeniid eggs cannot be differentiated morphologically). PCR/sequencing allows species identification (from isolated eggs or proglottids)*.
Echinococcus multilocularis	Asymptomatic.	At least 10 g faeces, sampling on 3 consecutive days	Morphology and size of proglottids. Egg detection with flotation, sedimentation or combined techniques (not very sensitive and taeniid eggs cannot be differentiated morphologically). PCR/sequencing allows species identification (from isolated eggs or proglottids)*.

* In specialised laboratories only
p.i. post infection

Table 6: Worm infection of dogs: main clinical signs and diagnosis (continued)
(for diagnosis: see also ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines)

Worm species	Clinical signs	Material	Diagnosis
NON-INTESTINAL WORMS			
Heartworm			
Dirofilaria immitis	Low worm burdens asymptomatic. First clinical manifestation 5–7 months p.i.: loss of condition, dyspnoea, cough. Chronic disease: cough, tachycardia, "Caval syndrome", tachypnoea, exercise intolerance, asthenia.	2–4 ml EDTA** blood 1 ml serum or plasma	Circulating antigens* (from 5 months p.i.) (sensitivity around 90% if 1 female worm or approximately 100% if more are present). Detection of microfilariae from 6–7 months p.i. Detection improved by concentration of microfilariae with Difil-Test or Knott's Test complemented by suitable PCR protocols. Microfilariae can be identified to species level using morphological, biochemical or molecular species identification. Thoracic radiography and echocardiography are complementary diagnostic measures.
French heartworm			
Angiostrongylus vasorum	Highly variable: from asymptomatic to respiratory and cardiovascular signs: cough, dyspnoea; coagulopathy (e.g. subcutaneous haematomas); neurological signs.	At least 10 g fresh faeces, sampling on 3 consecutive days, bronchial lavage fluid 1 ml serum or plasma	Detection of live larvae from fresh faeces using the Baermann method, direct smear or microscopic detection of larvae in bronchial lavage material (PCR, less sensitive), detection of circulating antigens in serum or plasma with a commercially-available kit.
Lungworms			
Crenosoma vulpis	Respiratory signs such as coughing, dyspnoea and possibly exercise intolerance.	Fresh faeces (at least 10 g) or bronchial lavage fluid	Detection of live larvae from fresh faeces using the Baermann method, or microscopic detection of larvae in bronchial lavage material (PCR, less sensitive).
Oslerus osleri	Respiratory signs such as coughing, dyspnoea and possibly exercise intolerance.	Fresh faeces (at least 10 g) or bronchial lavage fluid	Detection of live larvae from fresh faeces using the Baermann method, or microscopic detection of larvae in bronchial lavage material (bronchoscopy (nodules with worms at the bifurcatio tracheae less sensitive).
***Filaroides* spp.**	Respiratory signs such as coughing, dyspnoea and possibly exercise intolerance.	Fresh faeces (at least 10 g) or bronchial lavage fluid	Detection of live larvae from fresh faeces using the Baermann method, or microscopic detection of larvae in bronchial lavage material (less sensitive).
***Capillaria* spp.**	Respiratory signs such as coughing, dyspnoea and possibly exercise intolerance.	Fresh faeces (at least 10 g) or bronchial lavage fluid	Egg detection by flotation.
Subcutaneous worms			
Dirofilaria repens	Mostly asymptomatic, subcutaneous lesions. Sometimes skin irritation.	2–4 ml EDTA** blood	Detection of microfilariae from 6 months p.i. Detection improved by concentration of microfilariae with Difil-Test or Knott's Test. Microfilariae can be identified to species level using morphological, biochemical or molecular species identification*.
Eye worms			
Thelazia callipaeda	Blepharospasm and epiphora.	Material from the surface of the eye or under the nictitating membrane	Detection of adult or larval stages from samples of the tear film from the surface of the conjunctiva or from the conjunctival sac.

* In specialised laboratories only
** acid
p.i. post infection

Table 7: Worm infection of cats: main clinical signs and diagnosis

Worm species	Clinical signs	Material	Diagnosis
INTESTINAL WORMS			
Roundworms or ascarids			
Toxocara cati	Low burden asymptomatic, higher burden may appear as cachexia and pot-bellied appearance in kittens. Large number of worms may cause intestinal blockage or intussusceptions. Occasional pneumonia in kittens.	If possible 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Toxascaris leonina	Mostly asymptomatic.	If possible 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Hookworms			
Ancylostoma tubaeforme	Diarrhoea, bloody diarrhoea, weight loss and anaemia. May be acute or chronic signs.	If possible 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Uncinaria stenocephala	Clinical signs rarely occur. In rare cases: diarrhoea, weight loss and anaemia.	If possible 10 g faeces (fresh or fixed)	Egg detection by centrifugation-flotation or antigen test.
Tapeworms			
Taenia taeniaeformis	Asymptomatic.	If possible 10 g faeces (fresh or fixed), sampling on 3 consecutive days, proglottids in faeces	Proglottids grossly visible: morphology of proglottids, particularly that each proglottid has a single genital pore. Taeniid eggs in faecal sample (see *Echinococcus* section for methods to differentiate taeniid eggs).
Dipylidium caninum	Mostly asymptomatic.	If possible 10 g faeces (fresh or fixed), sampling on 3 consecutive days, proglottids or eggs in faeces	Proglottids similar in size but morphologically distinct to proglottids of *Taenia* spp., as each proglottid has two genital pores. Eggs within proglottids are grouped within egg packets which can be seen microscopically within faecal samples. Antigen test.
Echinococcus multilocularis	Asymptomatic.	If possible10 g faeces, sampling on 3 consecutive days	Morphology and size of proglottids. Egg detection with flotation, sedimentation or combined techniques (not very sensitive and taeniid eggs cannot be differentiated morphologically). PCR/ sequencing allows species identification (from isolated eggs or proglottids)*.
Stomach worm			
Ollulanus tricuspis	Gastritis, vomitus.	Vomitus	Detection of larvae or adult worms.
Liver trematodes			
Opisthorchis felineus	Vomitus, anorexia, digestive problems.	If possible 10 g faeces (fresh or fixed)	Egg detection through sedimentation or other special procedures.

Table 7: Worm infection of cats: main clinical signs and diagnosis (continued)

Worm species	Clinical signs	Material	Diagnosis
NON-INTESTINAL WORMS			
Heartworm			
Dirofilaria immitis	Often asymptomatic. Initial signs as the worms reach the heart. Later disease: acute signs associated with worm death including cough, vomiting, tachycardia, tachypnoea, sudden death (HARD, heartworm-associated respiratory disease).	2–4 ml EDTA** blood, 1 ml serum or plasma	Antibody combined with antigen detection. Detection of microfilariae from 8 months p.i. (low sensitivity). Detection may be improved by concentration of microfilariae with Difil-Test or Knott's Test. Microfilariae can be identified to species level using morphological, biochemical or molecular species identification*. Often a definite diagnosis of heartworm infection can only be obtained by haematological tests in conjunction with thoracic radiography and echocardiography.
Lungworms			
Aelurostrongylus abstrusus	Respiratory signs, coughing and possibly exercise intolerance.	Fresh faeces (at least 4 g) or bronchial lavage material	Detection of live larvae from fresh faeces using the Baermann method or microscopic detection of larvae in bronchial lavage material (less sensitive).
***Troglostrongylus* spp.**	Respiratory signs, coughing and possibly exercise intolerance.	Fresh faeces (at least 4 g) or bronchial lavage material	Detection of live larvae from fresh faeces using the Baermann method or microscopic detection of larvae in bronchial lavage material (less sensitive).
Subcutaneous worms			
Dirofilaria repens	Mostly asymptomatic, subcutaneous lesions.	2–4 ml EDTA** blood	Detection of microfilariae from 6 months p.i. (low sensitivity). Detection improved by concentration of microfilariae with Difil-Test or Knott's Test. Microfilariae can be identified to species level using morphological, biochemical or molecular species identification.*
Eye worms			
Thelazia callipaeda	Blepharospasm and epiphora.	Material from the surface of the eye or under the nictitating membrane	Detection of adult or larval stages from samples of the tear film from the surface of the conjunctiva or subconjunctival sac.

* In specialised laboratories only
** acid
p.i. post infection

APPENDIX 1 – GLOSSARY

Application	Like treatment, but describing the various forms of veterinary medicinal products which can be given (applied) to animals, such as spot-ons, pour-ons, oral products, injectables etc.
Autochthonous	Refers to organisms that are indigenous to a specific region, rather than being introduced from another location.
Control	General term comprising 'therapy' (treatment) and 'prevention' (prophylaxis).
Endemic	Refers to an organism/disease that is regularly found and consistently present in a particular geographical area. Describes a species that is native to, and restricted to, a particular area.
Endoparasiticide	Compound developed for the animal. Use as a therapeutic agent to eliminate any existing endoparasite infection and prevent reinfection.
Integrated control	The use of several measures to control different parasites or parasite stages present in the animal and stages present in the environment.
Pesticide	Compound developed for the elimination of different stages of parasites in the environment.
Prevention	Measures taken prior to any infection of the pet animal with endoparasites, to prevent the establishment of an infection. Prevention for an extended period may be achieved by the use of a product with persistent activity for certain periods of time following treatment.
Therapy	Any medical intervention to cure a disease; this includes the use of veterinary medicinal products (treatment), to eliminate an existing parasite infection.
Treatment	Administration of veterinary medicinal products (medication) as deemed necessary based on any given diagnosis.

APPENDIX 2 – BACKGROUND

ESCCAP (European Scientific Counsel Companion Animal Parasites) is an independent, not-for-profit organisation that creates guidelines based on up-to-date scientific information and promotes good practice for the control and treatment of parasites in companion animals. With the application of the proper advice, the risk of diseases and parasitic transmission between animals and humans can be minimised. ESCCAP aspires to see a Europe where companion animal parasites no longer threaten the health and well-being of animals and humans.

There is a great diversity in the range of parasites and their relative importance across Europe and the ESCCAP guidelines summarise and highlight important differences which exist in different parts of Europe and, where necessary, specific control measures are recommended.

ESCCAP believes that:

- Veterinarians and pet owners must take measures to protect their pets from parasitic infections.
- Veterinarians and pet owners must take measures to protect the pet population from risks associated with travel and its consequent potential to change local parasite epidemiological situations through the export or import of non-endemic parasite species.
- Veterinarians, pet owners and physicians should work together to reduce the risks associated with zoonotic transmission of parasitic diseases.
- Veterinarians should be able to give guidance to pet owners regarding risks of parasite infection and diseases and measures which can be taken to minimise these risks.
- Veterinarians should attempt to educate pet owners about parasites to enable them to act responsibly not only for their own pet's health but for the health of other pet animals and people in their communities.
- Veterinarians should, wherever appropriate, utilise diagnostic tests to establish parasite infection status in order to provide the best possible advice.

To achieve these objectives, ESCCAP produces guidelines in different formats:

- A detailed guideline for veterinary surgeons and veterinary parasitologists.
- Translations, extracts, adaptations and summarised versions of guidelines which address the varied requirements of European countries and regions.

Versions of ESCCAP guidelines can be found at www.esccap.org

Disclaimer:

Every effort has been taken to ensure that the information in the guideline, which is based on the authors' experience, is accurate. However, the authors and publishers take no responsibility for any consequence arising from the misinterpretation of the information herein nor is any condition or warranty implied. ESCCAP emphasises that national, regional and local regulations must be borne in mind at all times before following ESCCAP advice. All dosages and indications are provided for guidance. However, vets should consult individual data sheets for details of locally-approved treatment regimens.

ESCCAP
EUROPEAN SCIENTIFIC COUNSEL COMPANION ANIMAL PARASITES®

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1 Worm Control in Dogs and Cats

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