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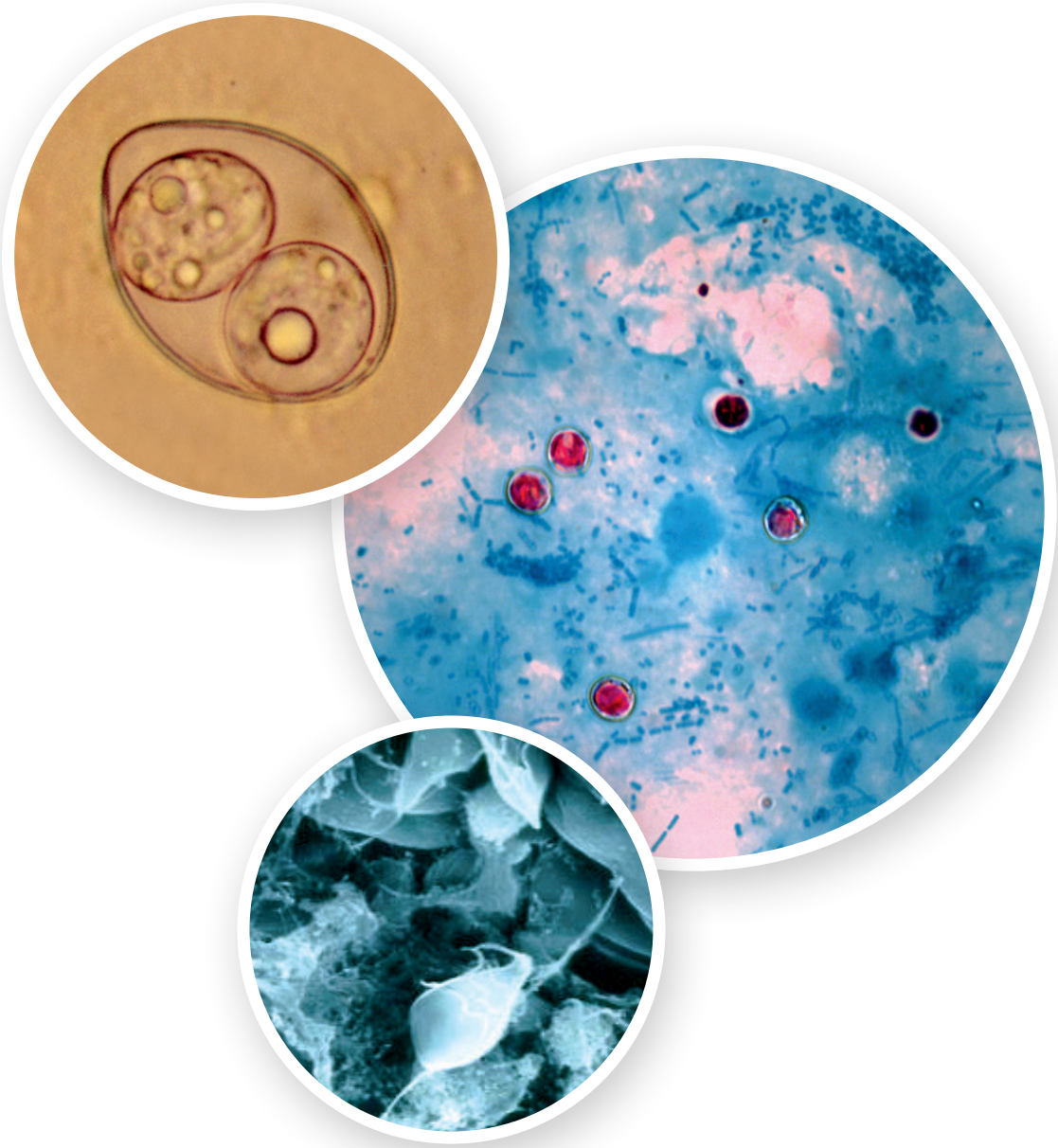

6 Control of Intestinal Protozoa in Dogs and Cats

ESCCAP Guideline 06 Third Edition – March 2025

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INTRODUCTION

A wide range of intestinal protozoa commonly infect dogs and cats throughout Europe; with a few exceptions there seem to be no limitations in geographical distribution. The group covers flagellates (*Giardia* and *Tritrichomonas*) and apicomplexan parasites (*Cystoisospora*, *Cryptosporidium*, *Hammondia*, *Neospora*, *Toxoplasma* and *Sarcocystis*).

These infections share common characteristics:

- Signs of disease are often associated with developmental stages in the intestine and are, in most cases, non-specific. However, for *Toxoplasma* and *Neospora*, disease is usually related to extraintestinal stages.
- Younger animals are most commonly affected.
- Their pathogenicity is variable both within and between genera and infections are often subclinical and can be self-limiting.
- The onset of clinical signs usually occurs several days after infection.
- Severe clinical signs may sometimes be related to co-infections with other pathogens, for example, nematodes, viruses and bacteria.
- Diagnosis and differential diagnosis can be difficult and often require repeated sampling and molecular typing.
- Treatment is often complicated due to the lack of effective drugs or the need for off-label use of existing drugs.
- Several agents are zoonotic, such as *Toxoplasma*, *Cryptosporidium* and potentially *Giardia*.

This guideline focuses on the following common, and often clinically important, intestinal infections:

1. *Giardia duodenalis*
2. *Tritrichomonas foetus*
3. *Cystoisospora* spp.
4. *Cryptosporidium* spp.
5. *Toxoplasma gondii*
6. *Neospora caninum*
7. *Hammondia* spp.
8. *Sarcocystis* spp.

Entamoeba histolytica is primarily a human and primate pathogen that infects dogs only sporadically, and has not been included due to its minimal relevance to our pets. Rarely, *Pentatrichomonas hominis*, an intestinal flagellate of humans, has been reported in dogs and cats.

This guideline aims to provide an overview of intestinal protozoa and their significance and, importantly, to suggest rational control measures for the most important species to prevent animal and/or human infection. In cases where extraintestinal parasite stages can cause disease in dogs or cats (neosporosis, toxoplasmosis), this is specifically mentioned.

The guideline is divided into five sections:

1: Consideration of pet health and lifestyle factors
2: Control of major intestinal protozoa
3: Environmental control of parasite transmission
4: Owner considerations in preventing zoonotic diseases
5: Staff, pet owner, pet caretaker and community education

1: CONSIDERATION OF PET HEALTH AND LIFESTYLE FACTORS

Animals require care tailored to their individual needs. Certain factors may dictate comprehensive monitoring and/or treatment, while others may suggest a less intensive approach. When recommending a parasite management programme, veterinarians should consider the following:

Animal
Clinical signs of infection with the mentioned protozoa are predominantly seen in young animals. Older animals are mostly immune after previous infections and seldom show signs of disease, except for geriatric, chronically sick or immunocompromised animals, severely stressed animals and potentially pregnant animals. Older animals, however, may still be a source of infection and can pass on infections to their offspring. The health status and background of the animal must be considered.

Environment
Dogs and cats living in kennels/catteries, animal shelters or in crowded conditions with poor sanitation may have a high risk of acquiring infections with protozoa that are transmitted directly, for example, *Giardia*, *Tritrichomonas*, *Cryptosporidium* and *Cystoisospora*, and these may require special consideration. Access to the outdoors may also influence the risk of infection.

Diet
Dogs and cats with access to rodents and raw meat, including viscera and/or foetal or placental material, may be at risk of acquiring infections with cyst-forming coccidia, i.e. *Neospora*, *Hammondia*, *Toxoplasma* and *Sarcocystis*.

Location and travel
Most infections are widespread in Europe and travel is not a major risk factor.

2: CONTROL OF MAJOR INTESTINAL PROTOZOA

2.1 *Giardia duodenalis*

2.1.1 Basic biology

Species
Giardia duodenalis (syn. *G. intestinalis*, *G. lamblia*) infects a range of vertebrates, including dogs and cats, and is currently classified into assemblages (strains or genotypes) A–H, of variable host specificity. Assemblages C and D are commonly found in dogs, while F has been isolated from cats and, more rarely, from other animals. Assemblage A has been found in both dogs and cats on occasions and assemblage B only rarely. Human infection with *Giardia* is almost always with either assemblage A or B. This indicates that although there is the potential for zoonotic transmission from pets, this is very rare. Animal genotypes detected in humans usually occur in the context of immunosuppression.

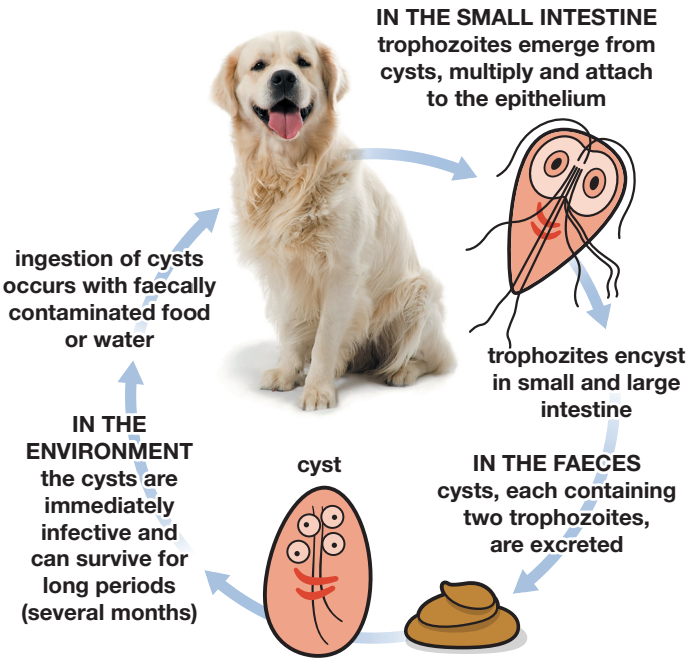

Figure 1: *Giardia duodenalis* life cycle

Life cycle
Giardia has a direct life cycle with repeated, asexual reproduction of trophozoites (i.e. active motile stages) in the small intestine and intermittent production of resistant cysts that are passed in the faeces, initially often in large numbers. Infection occurs through the oral ingestion of cysts. In the small intestine, the cysts release two trophozoites which attach to the epithelial cells and cause reduced absorptive capacity and altered intestinal permeability. The prepatent period is 4–16 days. Patency usually persists for several weeks to months.

Epidemiology
In Europe, the overall prevalence in dogs and cats is around 3–7%. However, this is significantly higher in young animals below one year of age, making it one of the most frequent endoparasites in this age group. Cyst shedding is seen in both healthy and sick animals. The infection is believed to induce partial immunity, resulting in loss of clinical signs and, in some cases, elimination of the agent but with limited resistance to reinfection. The transmission is faecal–oral by cysts in contaminated water or food, or from the environment, and only a few cysts are needed to cause infection. Cysts may survive in the environment for months, but are susceptible to desiccation and significantly reduced in numbers by seasonal freeze–thaw cycles.

2.1.2 Clinical signs

Infection mostly remains subclinical but may also cause chronic intermittent pasty diarrhoea rich in mucus, anorexia, vomiting, weight loss and lethargy, particularly in immunocompromised animals, puppies/kittens with coexisting infections or stressed animals (e.g. working sled dogs), and young animals after weaning or re-homing. In dogs, *Giardia* infections are frequently subclinical; in cats, they usually cause clinical disease.

2.1.3 Diagnosis

In faeces, ovoid translucent cysts measuring 8–15 x 7–10 µm can be detected by direct wet mounts of fresh faeces or after concentration by sedimentation–flotation. Cysts may become deformed if flotation is attempted using hyperosmotic flotation solutions or if the sample is old. This will be prevented if sodium acetate – acetic acid – formalin (SAF) is used for concentration. In very fresh faeces from animals with clinical signs, motile trophozoites (pear-shaped, 9–21 x 5–12 µm) may be detected in rare cases. Due to fluctuating excretion, and to improve detection, examining three samples over 3–5 days is recommended. Sedimentation techniques, in combination with merthiolate iodine formalin (MIF) staining, are also suitable methods for detecting cysts in faecal samples. Detection of *Giardia*-specific coproantigen using commercially available assays (e.g. ELISA, immunochromatography as point-of-care tests) is also possible. Tests may differ in their target; some are often significantly more sensitive than methods for the microscopy-based detection of *Giardia* cysts. This means that a diagnosis is possible also in cases when cyst excretion has temporarily halted. A direct fluorescent antibody test and, in specific cases, PCR (with subsequent sequencing for genotyping) may be used in diagnostic laboratories. Genotyping is recommended in cases where the potential for zoonotic transmission is to be clarified, i.e. in cases of vulnerable in-contact persons.

For further information, see ESCCAP Guideline 4: Parasitological Diagnosis in Cats, Dogs and Equines at www.esccap.org/guidelines/gl4/.

Unfortunately, in the case of a positive *Giardia* test, no general statement can be made regarding a therapy decision (see section 2.1.4) since *Giardia* infections are often subclinical, even over a long period of time. Animals that shed a higher number of cysts (that are then often detectable by microscopy) are more frequently clinically affected.

2.1.4 Control

Treatment
Dogs and cats with gastrointestinal disorders that test positive for *Giardia* (cysts or coproantigen) should be treated. Animals with persistent diarrhoea with no other aetiology identified should be re-tested. It is not generally advisable to treat clinically inconspicuous *Giardia* positive animals. In such cases, a risk assessment is needed before reaching a treatment decision. In high-risk environments like kennels, catteries or breeding establishments, particularly with a high incidence of diarrhoea in puppies, or where there are small children or immunocompromised humans and/or when potentially zoonotic genotypes are detected, treatment is advised. Strict hygiene measures are needed – see prevention.

Fenbendazole and metronidazole are effective against *Giardia*. Treatment focuses on resolving clinical signs and not the complete elimination of the agent. Fenbendazole (50 mg/kg bodyweight orally every 24 hours for at least 3, preferably 5, days) can be used. It is registered for the treatment of giardiosis in dogs in most European countries and can also be recommended for cats. Metronidazole (25 mg/kg bodyweight orally twice daily or 50 mg/kg bodyweight once daily for at least 5, preferably 7 days) is an antimicrobial licensed in most European countries for dogs and cats against *Giardia* and is considered effective; however, to prevent the development of antimicrobial resistance, its application should only be used in cases where fenbendazole does not provide sufficient efficacy. Since neurotoxicity of metronidazole has been reported in dogs, especially with treatment beyond the recommended period, treated animals should be monitored for neurological signs during treatment. Care should be taken when administering metronidazole and gloves worn as nitroimidazoles are mutagenic, although the effect is less pronounced than for ronidazole (see chapter 2.2.4). For the same reason, treatment of pregnant and nursing cats and kittens less than 12 weeks old is not recommended.

Another option is the use of a combination of febantel/pyrantel/praziquantel at the standard deworming dose (15.0 mg/kg of febantel, 14.4 mg/kg pyrantel, 5.0 mg/kg praziquantel for dogs; 12.5 mg/kg febantel, 12.0 mg/kg pyrantel, 4.16 mg/kg praziquantel for cats) repeated once daily for 3 days (dogs) or 5 days (cats). This treatment is licensed for dogs in most European countries and countries outside the EU, but the manufacturer's claim does not include *Giardia* infections.

A therapy control should be carried out with one of the above-mentioned methods after the last treatment but before the onset of the patent period, i.e. no longer than 3 days, to enable differentiation between persistent infections and reinfections. If the sample remains positive AND, if the clinical signs persist, treatment should be continued. Reinfection can occur immediately after treatment, therefore it is advisable to inform the pet owner, breeder or caretaker that recurrences are possible, or even likely. Other reasons for apparent "treatment failure" include co-infections or other underlying disease, which should be addressed, or by incomplete parasite removal following treatment. Drug resistance against metronidazole and albendazole (a benzimidazole) has been described in human isolates. Long-lasting treatment success is often hampered by reinfection pressure from the contaminated environment. Thus, additional measures to reduce infection pressure are critical. Shampooing dogs and perhaps cats (e.g. with a product containing chlorhexidine digluconate) at the beginning and the end of antiprotozoal treatment may assist in reducing reinfections. To support the clinical resolution of giardiosis, reduction of carbohydrate intake in the diet is recommended, as excessive carbohydrates may promote growth of potentially pathogenic bacteria, such as clostridia, in the intestinal microbiota. Instead, a diet rich in highly digestible protein may be preferable. Dietary changes should be implemented case by case.

2.1.5 Prevention

Cleaning and drying of the environment (including blankets, bedding etc.), the use of clean utensils for feed and water, bathing with chlorhexidine shampoos to remove adhering faeces or cysts, and proper disposal of faeces are prerequisites to avoid animal–animal transmission. There are indications that cysts on surfaces are killed by hot water high-pressure cleaning (>65°C), but no disinfectants are registered for this purpose. Surfaces should be left to dry completely. Maintaining meticulous personal hygiene is mandatory for all animal carers to avoid the spread of cysts. Food and water containers should be cleaned daily with boiling water. This also applies to litter boxes which should be washed and thoroughly dried before being refilled. Diagnostic tests should be performed on new puppies or kittens when introduced to households with other pets or animals entering breeding establishments. Diarrhoeic animals and carriers should always be quarantined and diagnosed appropriately.

2.1.6 Public health considerations

As mentioned above, humans are rarely infected by dog- or cat-specific assemblages, but potentially human assemblages may circulate in a dog or cat population and be transmitted back. Genotyping (to the assemblage and sub-assemblage level) is necessary to assist in determining the potential for zoonotic transmission. Results must be interpreted with care, considering cyst passage due to coprophagy especially in dogs.

In cases where family members, as well as their dogs or cats, have a *Giardia* infection, the pet owner should be advised to consult their attending physician.

2.2 *Tritrichomonas foetus*

2.2.1 Basic biology

Species
Tritrichomonas foetus has been identified as a cause of chronic large bowel diarrhoea in cats and other felids. *T. foetus* is rarely detected in dogs.

Life cycle
The life cycle is direct, with trophozoite formation in the large intestine (caecum and colon) and ileum, and there is no known cyst stage. Pathogenicity is related to the cytotoxic effects of trophozoites on the intestinal epithelium via secretion/excretion of proteases and other factors; occasionally invasion deeper into the mucosa is seen. Trophozoites can be detected after 7–14 days and the infection is often long-lasting but generally self-limiting.

Epidemiology
The infection route is considered to be faecal–oral. Prevalences may be relatively high in restricted environments like catteries and shelters but otherwise can be expected to be low, although data are limited for most European countries. There are indications that pedigree cats breeding in closed environments are more commonly affected. At present, there is no evidence to suggest any epidemiological link between feline infections and bovine reservoirs with the closely-related bovine genotype of *T. foetus*.

2.2.2 Clinical signs

Infections are often subclinical but typically kittens or otherwise naïve animals may exhibit clinical signs of *T. foetus* infection including semi-formed ("cowpat") faeces, high number of depositions per day (15–20) with blood and/or mucus and faecal incontinence with irritation and pain around the anus. Severe cases of diarrhoea with proctitis have been reported in kittens with chronic disease. The clinical course often fluctuates with transient remission after therapy (see below).

2.2.3 Diagnosis

Pear-shaped trophozoites (10–25 x 3–15 µm) are detected in FRESH, still warm faeces by direct wet mounts but sensitivity is generally low. The trophozoites are of similar size to *Giardia* trophozoites but the rapid "jerky" forward movement and the presence of an undulating membrane in *T. foetus* are different from the "falling leaf" motion and the typical "eyes" (two large nuclei) of *Giardia*. *Tritrichomonas foetus* also needs to be differentiated from *Pentatrichomonas hominis*, which is rarely seen in either cats or dogs, and occasionally other trichomonads. Direct detection by PCR is the preferred option. In contrast to other protozoans, *T. foetus* can be cultured e.g. in a commercially available test system (InPouchTF-Feline™, BioMed Diagnostics), which will propagate *P. hominis* but not *Giardia*.

2.2.4 Control

Treatment
There are no drugs registered for use in cats against *T. foetus* and treatment recommendations are based on case histories. Ronidazole (20–30 mg/kg bodyweight daily for 2 weeks) has been used off-label in severe cases of diarrhoea with some success. Ronidazole is not available as a pre-formulation for cats and a formulation in capsules for release in the large intestines is considered advantageous over powdered in-feed formulation. Care should be taken when administering ronidazole and gloves worn as it is a known mutagenic, carcinogenic and embryotoxic substance. For the same reason, treatment of pregnant and nursing cats and kittens less than 12 weeks old is not recommended. Cats must be closely monitored for drug-induced neurotoxicity (lethargy, ataxia, seizures) and treatment withdrawn immediately should this happen. The signs seem reversible when the drug is withheld. Metronidazole and fenbendazole only cause temporary remission. A change in diet may also alleviate clinical signs, and spontaneous resolution of clinical signs often occurs.

Prevention
As clinical problems are often associated with closed environments where there is a high density of cats, many of the precautions recommended for *Giardia* should be observed. Cases are often chronic and refractory to treatment and will contaminate the environment.

2.2.5 Public health considerations

At present, *T. foetus* has no documented zoonotic potential, although rare human cases have been reported and care must always be taken with immunocompromised individuals. Precautions must be taken when administering ronidazole.

P. hominis is observed in humans and may occasionally cause diarrhoea, but little is known regarding its transmission.

2.3 *Cystoisospora* spp.

2.3.1 Basic biology

Species
The genus *Cystoisospora* is host-specific: *Cystoisospora canis*, *C. ohioensis* and *C. burrowsi* are the common species infecting dogs; the latter two are often referred to as the *C. ohioensis*-complex because they are not readily separated morphologically. *Cystoisospora felis* and *C. rivolta* infect cats.

Life cycle
Infection commonly takes place via the faecal–oral route by the ingestion of sporulated oocysts. Multiplication of the intestinal stages takes place intracellularly throughout the small and large intestines. After a prepatent period of 6–10 days, oocysts are shed in the faeces and then complete their development to the infective stage in the environment usually within several days. Different animals, including rodents and ruminants, can act as paratenic hosts after oral uptake of oocysts and subsequently harbour resting stages (“dormozoites” or “hypnozoites”) in internal organs. After ingestion of dormozoites, the prepatent period is slightly shorter. The excretion period is variable, but most animals shed oocysts for 5–10 days.

Epidemiology
Cystoisospora species of dogs and cats are ubiquitous and oocysts can be found in the faeces of subclinically infected, as well as sick, animals. Primary infections usually take place during the suckling period, between the ages of three and eight weeks. Consequently, most clinical cases are diagnosed in puppies/kittens of 2–4 months old. Oocysts remain infective in the environment for several months and can accumulate in breeding kennels or catteries with a high density of suitable hosts. Dormozoites in paratenic hosts are infective for several years and can be transmitted by ingestion of the raw meat of such hosts. This transmission is probably of less importance than infections during the suckling period (prior to meat consumption).

2.3.2 Clinical signs

Cystoisosporosis is associated with diarrhoea in puppies and kittens. In severe cases, the faeces can contain blood and cause morbidity or even mortality. Where there have been changes in their diet (e.g. puppies receiving solid food for the first time), animals seem to be more affected by diarrhoea. Diarrhoea occurs around the onset of oocyst excretion. After reinfection, animals usually shed few oocysts and do not show clinical signs. Cross-immunity between *Cystoisospora* species in the same host seems unlikely. Whereas in *C. canis* infection significant oocyst shedding occurs only after primary infection, reinfection with *C. ohioensis*-complex can lead to patent infections but with less severe clinical signs.

2.3.3 Diagnosis

During the patent period, oocysts are shed in the faeces and can be detected by concentration flotation. The morphology of oocysts found in the faeces of infected dogs and cats are described in Table 1. Alternatively, a coproantigen test is available that detects the four species of *Cystoisospora* in dogs, respectively cats.

Table 1: Characteristics of intestinal stages of coccidia detectable in the faeces of dogs and cats

Genus	Species	Host Affected		Average Size (µm)	Shape	Shell
		DOG	CAT			
*Cystoisospora**	*C. burrowsi*	■		21 x 18	round–oval	thin, colourless or brownish
	C. canis	■		39 x 32	round–oval	
	C. ohioensis	■		24 x 20	round–oval	
	C. felis		■	45 x 33	ovoid	
	C. rivolta		■	26 x 24	round–oval	
*Cryptosporidium***	*C. canis*	■		3.5 x 6	round–oval	thin, colourless unless stained
	C. felis		■			
	C. parvum	■	■			
*Toxoplasma****	*T. gondii*		■	12.4 x 10.5	round	thin, colourless
Neospora	*N. caninum*	■		12.0 x 10.5	round	thin, colourless
Hammondia	*H. hammondi*		■	10 x 12	round	thin, colourless
	H. heydorni	■				
*Sarcocystis*****	Oocyst			–	round	very thin, colourless
	Sporocyst	■	■	11 x 8 to 14 x 10	ovoid	thick, colourless

* The oocysts of *Cystoisospora* spp. in fresh faeces contain a large sporoblast; in older faecal samples (> 12 hrs) two round sporocysts may be seen. *Cystoisospora burrowsi* is part of the *C. ohioensis* complex due to the overlap in oocyst size and other biological features.

** Using the modified Ziehl Neelsen technique.

*** *Toxoplasma gondii* can also infect dogs, but the infection leads to the development of extraintestinal stages only, without intestinal development and oocyst shedding.

**** Several species in dogs and cats with morphologically indistinguishable sporocysts; round oocysts with very thin walls, rupture during intestinal passage and release two fully sporulated sporocysts which can be found in the faeces.

2.3.4 Control

Treatment
Due to the rapid replication of the pathogenic intestinal stage and the subsequent excretion of large numbers of oocysts, it is crucial to treat infections early. Litter mates of an affected puppy are at high risk of contracting the infection, even if they are not yet shedding parasites. Hence, treatment should be administered to all susceptible animals, including all litter mates and in-contact puppies.

Toltrazuril and diclazuril are currently the drugs of choice against cystoisosporosis. Toltrazuril (9–20 mg/kg bodyweight) or diclazuril (2.5–5.0 mg/kg bodyweight) in a single application significantly reduce oocyst shedding in excreting animals; application in the pre-patent period largely prevents oocyst excretion and reduces diarrhoea in affected litters. In dogs, a combination of toltrazuril/emodepside (9 mg/0.45 mg/kg bodyweight) is registered for co-infections of coccidia and roundworms. This product is not licensed for use in cats, and it is necessary to use it off-label at an increased dose.

Prevention
Due to the ubiquitous nature of the parasites, eradication is not normally feasible. The risk of acquiring an infection can be reduced by hygiene measures including the daily removal of faeces from kennels and thorough cleaning and disinfection of litter areas in breeding units. Since heat (steam cleaning) and chemical disinfection using cresols are necessary to inactivate oocysts, floors and walls of boarding kennels, animal shelters and larger breeding units etc. should be made of material that is resistant to such treatment. Surfaces should be left to dry completely since this also reduces the survival of oocysts in the environment. Meticulous personal hygiene is very important for all animal handlers to avoid spreading oocysts through faecal material to cats/dogs or paratenic hosts. Transmission via raw meat is of minor importance, but should be considered for control.

2.3.5 Public health considerations

Cystoisosporosis of cats and dogs has no zoonotic implication, as the parasites are strictly host-specific.

2.4 *Cryptosporidium* spp.

2.4.1 Basic biology

Species
Cryptosporidium oocysts are very small and do not allow species differentiation based on morphology (Table 1). *Cryptosporidium canis* and *C. felis* infect dogs and cats, respectively, and these species have only very rarely been found in humans or other animals. *Cryptosporidium parvum* is a species with low host specificity and parasitises mainly calves, along with other young of different ruminants and cervids, and humans, but can also infect a range of other mammals, including, occasionally, dogs and cats. Since species differentiation relies on molecular typing, the exact distribution amongst positive cats and dogs is still unknown.

Life cycle
Infection with *Cryptosporidium* is initiated when oocysts from the environment are ingested and the released sporozoites invade the epithelium of the small intestine and begin intracellular multiplication. Endogenous asexual replication ends with the production of sexual stages that fuse to form an oocyst that sporulates in the intestines and is excreted with the faeces already in the infective form. Autoinfection with ruptured oocysts before excretion can result in the shedding of large parasite numbers within a short period of time. The prepatent period varies from 2–14 days for *C. canis* and 3–7 days for *C. felis*. Excretion lasts from 25–80 days, or even longer if the host is immunosuppressed.

Epidemiology
Cryptosporidium oocysts are immediately infective when excreted with the faeces, so faecal–oral infections are common. They are also very small, do not sediment readily in water and are therefore frequently waterborne. The parasite can remain infective in this environment for several months. Unlike the other apicomplexan species described here, *Cryptosporidium* is strictly monoxenous, and paratenic or intermediate hosts are not described.

2.4.2 Clinical signs

In immunocompetent, previously exposed (immune) adult animals, infection is usually subclinical. Clinical signs appear to be most severe in immunocompromised individuals.

Kittens, and less commonly puppies, can develop watery, sometimes foul-smelling, diarrhoea; this can last for days or sometimes weeks, and is frequently accompanied by abdominal pain, vomiting and an elevated body temperature.

2.4.3 Diagnosis

Direct immunofluorescence antibody test in specialised laboratories is the gold standard for diagnosis. Antibodies are usually labelled with fluorescein isothiocyanate that fluoresces apple green under appropriate (blue) filters on a fluorescence microscope. DAPI (4', 6-diamidino-2-phenylindole) can be used to stain nuclei and can be a useful confirmatory stain if oocyst excretion is low (UV filters necessary for visualisation on a fluorescent microscope). In many diagnostic laboratories, the only 3–6 µm small oocysts are detected by direct coproscopy (Table 1) of a faecal smear after staining (Ziehl-Neelsen, Heine, safranin). Oocysts are presented as small, round, colourless, pink or orange bodies when stained, depending on the staining procedure. As with *Giardia*, coproantigen tests for *C. parvum* are commercially available and in some cases also validated for cats and/or dogs. Molecular detection is both sensitive and specific, and species differentiation by PCR and sequencing can be performed if risk assessment raises concern.

2.4.4 Control

Treatment
There is no registered treatment available for cryptosporidiosis in cats and dogs. Since the infection usually resolves spontaneously, only supportive treatment (fluid replacement, spasmolytic medication) can be considered.

Prevention
Cryptosporidium oocysts are highly persistent in the environment and directly infectious so strict hygiene measures must be taken to avoid the spread of infection (see *Cystoisospora*).

2.4.5 Public health considerations

Due to the rather low host-specificity of *C. parvum*, this parasite is infectious to humans, while zoonotic infections with *C. felis* or *C. canis* are usually, but not always, restricted to immunocompromised individuals. Owners of young animals should generally be advised to adhere to effective hygiene protocols and immunocompromised individuals should not be in close contact with sick cats and/or dogs.

2.5 *Toxoplasma gondii*

2.5.1 Basic biology

Species
Toxoplasma gondii is the only valid species in the genus *Toxoplasma*. It infects only cats and a few other felids as definitive hosts, while probably all mammals (including humans, cats and dogs) as well as birds, can act as intermediate hosts. *T. gondii* is globally present in at least three clonal genotypes and multiple mixed forms thereof. *T. gondii* is a major cause of abortion in small ruminants.

Life cycle

Cats usually acquire the infection by ingestion of tissue cysts, most commonly by predation on rodents and birds, by feeding on raw or undercooked meat from infected livestock or, less commonly, on aborted material or by ingestion of sporulated oocysts (Figure 2). The prepatent period is 3–10 days after ingestion of tissue cysts and 18–36 days after uptake of oocysts. Excretion of oocysts can last up to 20 days and is most intensive 2–5 days after the onset of shedding. Oocysts are not infective immediately after excretion, but require at least 24 hours and usually 2–5 days for sporulation in the environment, depending on temperature. Therefore, thorough daily cleaning of the litter box (see 2.5.4 Control) minimises the development of sporulated (infectious) oocysts.

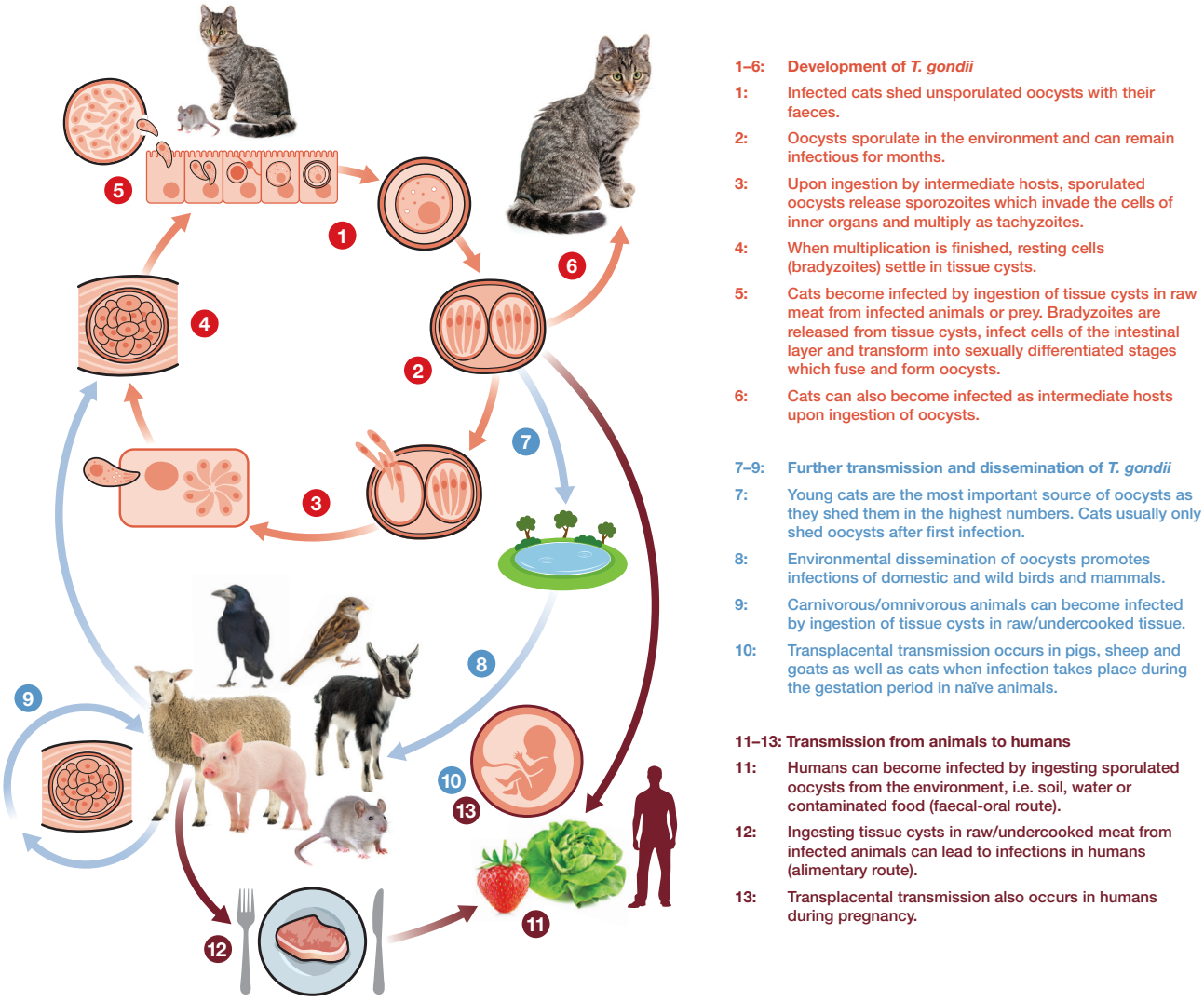

Figure 2: *Toxoplasma gondii* life cycle

Epidemiology

Cats can excrete a large number of oocysts (hundreds of thousands to millions) for a few days after primary infection. However, after this initial period, they excrete few or no oocysts, even after reinfection or if immunosuppressed. After excretion, the stages rapidly sporulate (within 1-5 days depending on ambient temperature) and are infective. The small oocysts can be distributed easily and get into surface water where they can survive for several months, making water, as well as humid soil or feedstuffs contaminated with cat faeces, the prime source of infection for herbivorous intermediate hosts. Due to the ubiquitous nature of the parasite, the distribution of *T. gondii* is broad in intermediate hosts. Carnivorous hosts most often acquire the infection via ingestion of tissue cysts in meat from infected hosts. Rodents, particularly mice, act as very efficient reservoir hosts.

2.5.2 Clinical signs

Clinical signs (diarrhoea) related to the enteral stage of infection in cats can be observed following initial primary infection, especially in kittens.

Acute toxoplasmosis due to extraintestinal parasite development (as in other intermediate hosts) is rare in cats. Kittens infected *in utero* can show signs of infection after birth and prenatal infections of kittens are frequently fatal. The reasons for clinical manifestations in adult cats are unclear; it is presumed that immunosuppression by viral pathogens (FeLV, FIV, FIP) may play a role.

In general, cats and dogs affected as intermediate hosts show signs of systemic infection with fever, anorexia, abdominal pain, dyspnoea, ocular inflammation (uveitis), and, rarely, central nervous or neuromuscular disorders. In cats, transplacental transmission can lead to foetal infection with stillbirths and congenital disease.

2.5.3 Diagnosis

Excreting cats shed the small oocysts in high numbers, but due to the short patent period and absence of re-shedding, infection is usually not detected by faecal examination. The oocysts cannot be reliably distinguished from those of *Hammondia hammondi* based on morphology (Table 1).

Clinical toxoplasmosis in dogs and cats is diagnosed by serology, supported by PCR on cerebrospinal fluid.

2.5.4 Control

Treatment
Cats with clinical disease can be treated with clindamycin (oral treatment: clindamycin hydrochloride 10–12 mg/kg bodyweight, twice daily for four weeks; animals should be offered feed and water immediately after treatment to avoid complications such as oesophagitis). Alternatively, oral treatment with trimethoprim/sulfonamide (15 mg/kg bodyweight twice daily for 4 weeks) is recommended. Treatment of cats after infection has not been shown to prevent oocyst excretion. Sick dogs may also be treated with clindamycin.

Prevention
Control measures aim at the prevention of oocyst shedding to reduce the infection of humans and livestock with *T. gondii*. Cats should be discouraged from eating prey animals. Where possible, cat faeces should not be introduced to the environment and the contents of litter boxes should be incinerated as regular household waste, not composted.

2.5.5 Public health considerations

T. gondii is one of the most prevalent parasitic zoonoses worldwide. Although there are differences in virulence associated with genotype, healthy adults generally have a low risk of developing severe toxoplasmosis after infection. However, immunocompromised individuals or children infected *in utero* can suffer from severe, or even fatal, local (mostly ocular or cerebral) or generalised toxoplasmosis. Prenatal infections occur as a result of a primary infection of the mother during pregnancy.

In humans, the infection can be acquired either by the ingestion of raw or undercooked meat from infected animals, or of sporulated oocysts from the environment (ingesting soil or sand from sandpits or water, fruit or vegetables contaminated by the faeces of infected cats). It is therefore recommended (especially for high-risk individuals e.g. previously unexposed pregnant women or the immunocompromised) that meat is consumed only after thorough cooking or freezing (-20°C for at least two days) and personal hygiene measures are observed whilst handling meat or meat products. Pregnant women should avoid contact with expectant ewes or nannies. They should not assist in lambing or kidding due to the risk of hand–mouth contamination by contact with recently-infected dams during delivery. Neither should they handle lambs or kids. Working in the meat industry (abattoir, cutting plant) is significantly associated with acquiring infection (occupational disease). Similarly, drinking unfiltered surface water or accidental ingestion of soil as well as contact with cat faeces in general must be avoided. All fruit and vegetables (especially from gardens) should be washed thoroughly before consumption, and gloves should be worn when gardening or handling soil or sand from sandpits.

Litter trays should be thoroughly cleaned every day so that, in case of defaecation by shedding cats, oocysts do not have time to sporulate. This task should not be performed by pregnant women or other persons at risk.

2.6 *Neospora caninum*

2.6.1 Basic biology

Species

Neospora caninum is the type species of the genus. In Europe, dogs are currently the only identified definitive hosts and they also act as intermediate hosts. Wild canids, such as the grey wolf and golden jackal, but not the red fox, can also act as definitive hosts. Cattle, sheep, goats and other domestic and wild ungulates, as well as rodents and birds, are natural intermediate hosts of the parasite, harbouring tachyzoites and cysts with bradyzoites in various tissues. *N. caninum* is a major cause of reproductive disease and infertility in cattle.

Life cycle

Dogs acquire the infection mainly by ingesting cysts containing bradyzoites located in tissues of infected intermediate hosts, in particular cattle (Figure 3). In the intestinal epithelium of the definitive host, sexual development leads to the production of oocysts that are excreted in faeces and sporulate in the environment. The prepatent period is 5–9 days and patency generally lasts 11–20 days. Oocysts are not immediately infective for other hosts after excretion in the faeces but require sporulation for 1–3 days in the environment. Repeated transplacental transmission of tissue-dwelling parasites from chronically infected dams to the foetus is possible, although highly variable. It has been reported, however, that up to 50% of pups of *N. caninum*-positive dams might become infected transplacentally, with 25% developing clinical signs (neonatal neosporosis).

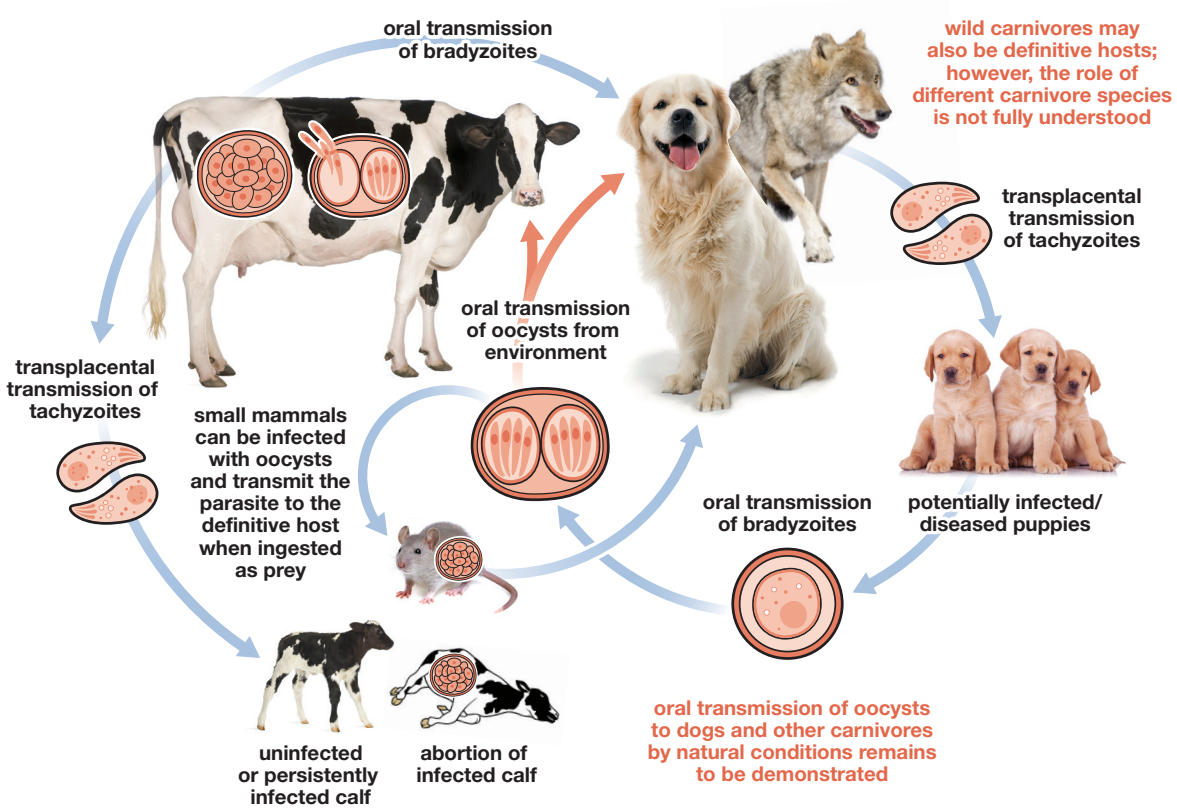

Figure 3: *Neospora caninum* life cycle

Epidemiology
Age-related seroprevalence data indicate that the majority of dogs become infected after birth. Higher prevalences have been documented in older dogs than in younger ones. It has been reported that placentas from aborting cattle as well as infected small mammals/rodents are the main source of infection for dogs and feeding of raw beef has also been identified as a risk factor for canine neosporosis. Not surprisingly, hunting dogs fed raw meat from cattle or ungulate game and dogs preying on small mammals have high seroprevalence rates. *N. caninum* oocysts have been found in faeces from dogs ranging in age from 45 days to 13 years and the number of oocysts per gram of faeces varies from only a few to over 100,000. Transplacental transmission can take place repeatedly in dams of any age and can cause disease with variable clinical signs in the offspring (see 2.6.2).

2.6.2 Clinical signs

The systemic phase can cause clinical disease whereas there are no signs associated with enteric development. Most cases of clinical neosporosis are reported in puppies of at least 5–7 weeks old, but less than six months old (neonatal neosporosis) that were infected transplacentally. However, primary infection with *N. caninum* can cause illness in dogs of any age. Clinical signs which should arouse suspicion of neosporosis include hind limb paresis and ataxia that becomes progressively more severe. Muscle atrophy, quadriceps contracture, hyperextension of forelimbs, signs of pain on palpation of the lumbar and/or quadriceps muscles and later signs of head and neck involvement (head tilt); ocular abnormalities and dysphagia may all be signs of neosporosis. Other neurological conditions not readily attributable to other causes should also be investigated as possible neosporosis, particularly in mature dogs. In older dogs, ulcerative dermatitis, myocarditis, pneumonia and pancreatitis have been reported. In puppies, the ascending paralysis caused by *Neospora* can often be fatal and several litter mates may be affected, although not necessarily simultaneously.

2.6.3 Diagnosis

Unsporulated oocysts in faeces measure on average 12 x 10.5 µm and are morphologically identical to *Hammondia heydorni* oocysts (Table 1). Differential recognition can be obtained by molecular techniques such as end-point PCR and amplicon sequencing or species-specific PCR. Because clinical disease is caused by the tissue-dwelling forms of the parasite, faecal examination for oocyst detection does not play a role in the diagnosis of canine neosporosis. Clinical suspicion of canine neosporosis in any age group can be confirmed by demonstrating the presence of the parasite through molecular methods: PCR can be carried out on cerebrospinal fluid or muscle biopsies. Most cases, however, are diagnosed through serology. Testing of paired serum samples (2–4 weeks apart) to demonstrate seroconversion is recommended. Puppies usually seroconvert about 2–3 weeks following infection and antibody levels are usually (but not always) high in clinically affected animals. Therefore, diagnosis of canine neosporosis can be based on clinical signs and positive serology with seroconversion (ELISA, IFAT) and/or seroreversion after specific treatment in positive dogs.

2.6.4 Control

Treatment
Treatment of clinical neosporosis in dogs is difficult and only partially effective; it tends to be most effective in the early stages, before muscular contraction has occurred. Indeed, when clinical signs are suggestive of *N. caninum*, it is recommended to initiate treatment immediately rather than wait for serological results. Treatment with clindamycin (20 mg/kg bodyweight twice daily for 30–60 days) has been reported to improve clinical recovery in naturally infected dogs with neurological signs. Alternatively, sulfonamide/trimethoprim and/or pyrimethamine may be used.

Prevention
As mentioned above, seropositive dams can transmit *N. caninum* to puppies. It is therefore recommended that chronically infected females are excluded from a breeding programme. Furthermore, farm dogs should not be fed raw meat or given access to calving pens and areas on farms with aborted calves, placentas, amniotic fluid or raw meat offal. Ingestion of small prey animals should be prevented. Contamination of water, cattle feedstuff and pasture with dog faeces should be avoided.

2.6.5 Public health considerations

As *Neospora* is not known to infect humans, there is no zoonotic potential although antibodies have been reported in some individuals.

2.7 *Hammondia* spp.

2.7.1 Basic biology

Species
Two species of *Hammondia* parasitise cats and dogs, i.e. *H. hammondi* and *H. heydorni* respectively.

Life cycle
The life cycle resembles that of other cyst-forming coccidia (*Sarcocystis*, *Neospora*, *Toxoplasma*). Dogs and cats are the definitive hosts and acquire the infection after ingestion of infected prey; they shed oocysts after a prepatent period of 5–13 days (*H. hammondi*) or 7–17 days (*H. heydorni*). Shedding periods are variable but usually limited to around 20 days and sporulation takes place in the environment. Intermediate hosts (mostly rodents and ruminants) ingest oocysts and subsequently develop tissue cysts, predominantly in muscle and brain tissue.

Epidemiology
Very little is known about the geographical distribution of *Hammondia* but it is found sporadically in the faeces of cats and dogs in Europe. Since the differentiation from *Toxoplasma* (in cats and occasionally in dogs after coprophagia) or *Neospora* (in dogs) is only possible with molecular methods, the true prevalence of these parasites is unknown.

2.7.2 Clinical signs

Hammondia infections in the definitive hosts usually take a subclinical course. Very rarely, anorexia and severe diarrhoea, unresponsive to antibacterial therapy, have been described in infected puppies.

2.7.3 Diagnosis

During the patent stage of infection, the small oocysts can be found in the faeces. Morphological differentiation from *Toxoplasma* or *Neospora* is not possible (see Table 1), but differentiation can be achieved with PCR.

2.7.4 Control

Treatment is not necessary. Prevention of infection can be achieved by discouraging the ingestion of raw food or prey containing tissue cysts from intermediate hosts (warm-blooded animals).

2.7.5 Public health considerations

As *Hammondia* is not known to infect humans, there is no zoonotic potential. However, since their oocysts are indistinguishable from those of *T. gondii*, care should be taken in cases of oocyst-positive animals. PCR confirmation can be used to allay owner fear.

2.8 *Sarcocystis* spp.

2.8.1 Basic biology

Species
Within the genus *Sarcocystis*, several species parasitise cats or dogs as definitive hosts. The faecal stages, sporocysts, are morphologically indistinguishable. Differentiation is based on tissue cyst morphology in the different intermediate hosts (omnivorous or herbivorous animals) and molecular methods. This genus can be responsible for the condemnation of meat from infected cattle.

Life cycle
Carnivorous animals become infected by the ingestion of meat containing tissue cysts. In the intestinal epithelium of the definitive host, sexual development takes place and leads to the production of an oocyst that sporulates before excretion. The oocyst wall is very thin and ruptures during passage through the intestine, so that usually fully infective sporocysts can be found in the faeces; these are then ingested by the intermediate host and develop extraintestinally into tissue cysts. The prepatent period is 8–33 days in dogs and 10–14 days in cats. Patency is long (several months) due to the slow release of parasites from the epithelium.

Epidemiology
The sporocysts in the faeces are infective on excretion and remain so for months, even years, since they have a prolonged survival rate in the environment. Prevalence rates in the intermediate hosts (sheep, cattle, pigs with outdoor access) are up to 100% due to the ubiquitous distribution of the parasites.

2.8.2 Clinical signs

In the definitive host, the development of the parasite is restricted to the final stages and does not cause clinical signs under natural conditions. The clinical and hygienic importance of infection with *Sarcocystis* is restricted to the intermediate host where outbreaks due to faecal contamination of feedstuff or water have been reported and can result in mild and transient clinical signs. Cysts in carcasses can lead to meat condemnation. After infection, dogs and cats usually develop some degree of immunity which is species-specific.

2.8.3 Diagnosis

Sporocysts can be found in faeces in low numbers (see Table 1).

2.8.4 Control

Treatment
Treatment of dogs or cats is not necessary.

Prevention
Since *Sarcocystis* is strictly heteroxenous, the infection can be avoided by feeding meat that has been either previously frozen (-20°C for at least 4 days) or cooked. Appropriate disposal of faeces from cats and dogs reduces the likelihood of propagation of this lifecycle (see Chapter 3).

2.8.5 Public health considerations

None of the *Sarcocystis* spp. involving dogs and cats are zoonotic. Humans serve as definitive hosts for some *Sarcocystis* spp. with cattle and pigs as intermediate hosts, and infection takes place via ingestion of beef or pork. Humans can also be infected as intermediate hosts for *Sarcocystis* species for which reptiles are definitive hosts (e.g. *S. nesbitti*), and has been responsible for various outbreaks.

3: ENVIRONMENTAL CONTROL OF PARASITE TRANSMISSION

A number of actions that will assist in the environmental control of intestinal protozoan infections of dogs and cats have been suggested in the relevant sections above.

Environment
Generally, it is always important to ensure appropriate disposal of pet faeces. Environmental stages of protozoa (oocysts, cysts, sporocysts) may survive in a contaminated environment for prolonged periods. Regular intensive cleaning of potentially contaminated areas will reduce the number of infective stages. Premises such as breeding facilities or sanctuaries should be equipped with surfaces that are easy to decontaminate (smooth, chemically resistant) and should be generally kept as dry as possible. Efficient chemical disinfection depends on dry and clean surfaces. Effective cleaning prior to disinfection is essential for disinfection efficacy. A number of products have been certified in the EU as active against environmental parasite stages based on results of highly standardised tests e.g. in Germany (www.dvg.net). Most of these commercial products contain cresols and all should be handled with care, according to national legislation. Manufacturers' instructions should be strictly followed to ensure maximum efficacy and to minimise environmental and health risks.

Efficient chemical disinfection in households where pets live in close proximity to people is challenging because effective products often contain rather aggressive chemicals so choosing an appropriate product with sufficient efficacy that does not destroy surfaces is mandatory. Prevention of patent infections in pets (see previous chapters) will avoid environmental contamination with infective stages of parasites. Transmission of zoonotic parasites to persons living in the same household is avoidable by observance of reasonable hygiene (including food hygiene in the case of *T. gondii*).

Disinfection of bare ground or grassed areas is not possible. To prevent contamination of such areas, faeces should be collected, properly disposed of in firmly-closed plastic bags and incinerated as regular household waste. Faecal disposal as compost material is not appropriate as the faecal parasite stage will not be sufficiently inactivated. For restricted areas, e.g. exercise yards, removal of topsoil, or replacing with a solid surface like concrete or covering with tiles, could be an option.

Depending on the quality of surfaces, physical disinfection (heat) is the most effective means of inactivating infective parasite stages. This however will not be appropriate in many circumstances. In addition, desiccation can be attempted.

Quarantine
New dogs/cats introduced into kennels/catteries should be quarantined whenever possible. As the prepatent period of protozoa is usually shorter than that of helminths, it is realistic to quarantine cat/dogs in shelters or catteries to control the possible onset of parasite excretion. This can aid the prevention of those infections that are directly transmitted via environmental stages. Faecal samples should be taken every other day and measures undertaken for adequate isolation, environmental hygiene and, in indicated cases, treatment to prevent parasite spreading.

Comment on feeding raw meat to pets
Diets for dogs and cats containing raw meat products, e.g. BARF, are becoming increasingly popular. Feeding fresh, raw meat increases the risk of meat-borne protozoan parasites; i.e. *Toxoplasma*, *Neospora*, *Sarcocystis* and, less importantly, *Cystoisospora*. Freezing at -20°C for 7–10 days prior to use can inactivate stages in raw meat and reduce the risk of transmission. Meat should be of the same quality as intended for human consumption.

4: OWNER CONSIDERATIONS IN PREVENTING ZOONOTIC DISEASES

The most important advice for the prevention of transmission of zoonotic agents, including certain intestinal protozoa, is personal hygiene. Washing hands after contact with dogs, cats and other animals should be routine, as should the appropriate disposal of faeces. Since many of the intestinal protozoan infections mentioned do little or no harm to dogs and cats (especially adult animals) and, in many cases, to pet owners, these infections go unnoticed. The majority of intestinal protozoa of dogs and cats are host-specific. Human infections with *Toxoplasma* are mainly either food, water or soil-borne. Contact with cat faeces and contaminated food/water are the most important risk factors and should be avoided by persons at risk. Human infections with *Sarcocystis* in Europe are almost exclusively transmitted between human–cattle and human–pig respectively. There is no known association with dogs or cats. Although *Cryptosporidium* and *Giardia* are also largely species-specific, some genotypes are zoonotic. Consequently, strict hygiene is the only way to prevent transmission. This is particularly important for individuals with immune deficiency disorders or those undergoing immunosuppressive treatments. In these patients, opportunistic species or rare genotypes of otherwise non-zoonotic parasites can occasionally establish, and these, as well as other zoonotic pathogens, frequently cause severe or even fatal diseases that would otherwise resolve in immunocompetent individuals.

5: STAFF, PET OWNER, PET CARETAKER AND COMMUNITY EDUCATION

Cryptosporidium and *Giardia* are potentially zoonotic (see specific sections above), but only molecular analyses (usually PCR and sequencing) can give definitive information. *Toxoplasma* is a well-known zoonotic agent and can be transmitted to humans by several routes, including via infective oocysts from the faeces of shedding cats. It must be kept in mind, however, that while the stages of *Giardia* and *Cryptosporidium* are immediately infective in fresh faeces, *Toxoplasma* oocysts require external sporulation that takes at least 24 hours, so fresh faecal material does not contain infective oocysts of *Toxoplasma*. Thorough daily cleaning of litter boxes with hot water therefore minimises the risk of transmission via cat faeces. Garden soil or sand areas where cats bury their faeces may represent a greater infection risk than litter boxes that are cleaned daily. Compared with human *Toxoplasma* infections acquired from food (mainly meat, fresh produce and, more rarely, raw goats' milk) or the environment (water/soil) the risk of infections directly from a litter box seems minor if it is cleaned daily.

The information in this guideline deserves to be widely disseminated in veterinary practices including to all auxiliary personnel. Veterinarians working with cats do not have a higher risk of acquiring *Toxoplasma* infections than other professions. Since tachyzoites can be present in the placental tissues of small ruminants, veterinarians should exercise caution during birth assistance and handle abortions appropriately to avoid zoonotic infections and transmission to scavenging carnivores. Correct knowledge of protozoan infections is a prerequisite for proper understanding, which, in turn, will help allay unjustified fear in pet owners and the general public. As in other parasitic, bacterial or viral infections, personal hygiene is the most effective preventive measure and emphasis of this fact should be given a very high priority in all educational programmes dealing with zoonotic disease.

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

APPENDIX 1 – 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 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 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, undertake diagnostic tests to establish parasite infection status in order to provide the best possible advice.

To achieve these objectives, ESCCAP produces:

- Detailed guidelines 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 each guideline 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.

APPENDIX 2 – GLOSSARY

Asexual reproduction	multiplication of parasite stages by binary or multicellular fission without production of sexually differentiated stages
Bradyzoite	slow-dividing tissue stage contained within a pseudocyst or maturating tissue cyst
Cyst	a) environmentally-resistant stage of *Giardia* excreted with faeces able to survive outside the host;
	b) mature stage of heteroxenous protozoa in the extraintestinal tissues (= tissue cysts)
Definitive/final host	a host in which the sexual development (production of sexually differentiated stages) is completed (in contrast to intermediate hosts)
Dormozoites	sleeping cells – non-dividing tissue stages until they are transmitted to a carnivorous host (typically in *Cystoisospora*)
Excystation	escape of parasite stages from the multilayered shell which covers the environmental stages (see cyst, oocyst)
Heteroxenous	infecting several host species in the life cycle
Monoxenous	infecting only one host species in the whole life cycle
Hypnozoites	see dormozoites
Intermediate host	a host in which asexual reproduction or development occurs
Oocyst	a robust transmission stage produced by sexual reproduction in the apicomplexan and capable of surviving outside the host
Paratenic host	a host which serves to maintain the life cycle of the parasite; no parasite development or reproduction takes place, but there may be reasons why paratenic hosts serve to further disseminate the parasites (e.g. large numbers may accumulate in one paratenic host, or the paratenic host may be a particularly relevant prey species for the definitive host)
Sporocyst	a multilayered stage within oocysts that contains the sporozoites
Sporozoite	the cellular infective unit that emerges during excystation of oocysts and sporocysts
Sporulation	development of sporozoites from the stages of sexual development
Tachyzoite	fast-reproducing parasite stages within the host cell
Tissue cyst	see cyst
Trophozoite	motile, active stage in the host e.g. within the life cycle of *Giardia*, *Tritrichomonas* and other protozoa
Zoonosis	any infectious disease that can be transmitted between animals (usually vertebrates) and humans
Zoonotic	transmissible between animals (usually vertebrates) and humans

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6 Control of Intestinal Protozoa in Dogs and Cats

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