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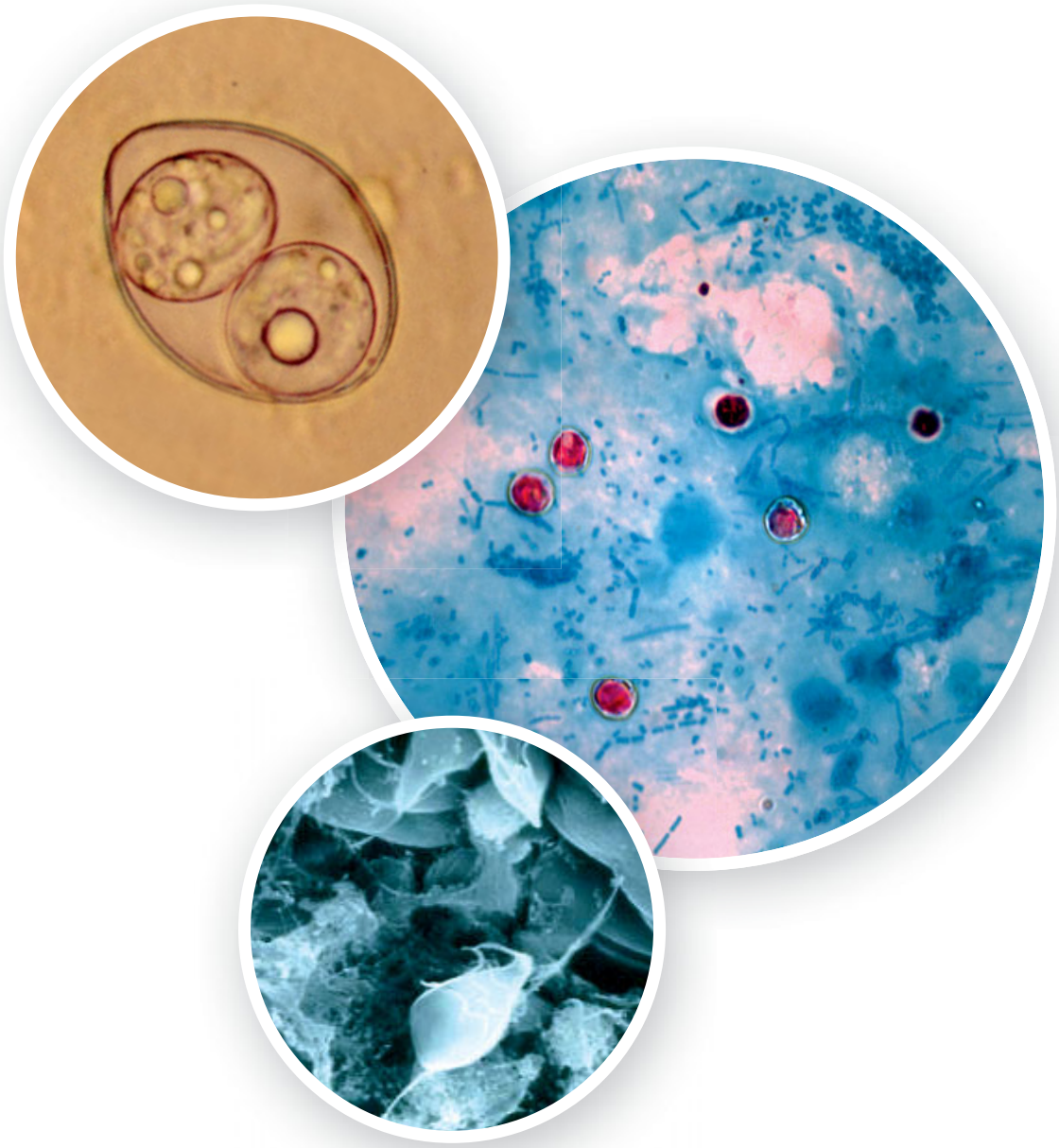

Control of Intestinal Protozoa in Dogs and Cats

ESCCAP Guideline 06 First Edition – August 2011

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A catalogue record for this publication is available from the British Library

ISBN 978-1-907259-24-1

ESCCAP Guideline No.6

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INTRODUCTION

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

These infections share common characteristics:

- Signs of disease are often associated with developing stages in the intestine and are in most cases nonspecific
- Younger animals are more commonly affected
- Their pathogenicity is variable both within and between genera, and infections are often asymptomatic and usually self-limiting
- The onset of clinical signs usually occurs within several days after infection
- Severe clinical signs are often, but not always related to co-infections with other pathogens, for example viruses and bacteria
- Diagnosis and differential diagnosis are difficult and often require repeated sampling and molecular typing
- Negative faecal findings cannot rule out infection
- Treatment is often complicated due to lack of effective drugs or the need for off-label use of existing drugs
- Several agents are important zoonoses, such as *Giardia*, *Cryptosporidium* and *Toxoplasma*

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

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

Entamoeba histolytica is a human and primate pathogen that infects dogs only sporadically and has not been included because of very limited relevance to our pets.

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

The guideline is divided into five sections:

1. **Consideration of pet health and lifestyle factors**
2. **Lifelong control of major intestinal protozoa**
3. **Environmental control of parasite transmission**
4. **Pet owner considerations in preventing zoonotic diseases**
5. **Staff, pet owner and community education**

1. CONSIDERATION OF PET HEALTH AND LIFESTYLE FACTORS

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

- Animal:
  All the protozoa mentioned infect predominantly young animals, such as puppies and kittens; older animals are mostly immune after previous infections and seldom show signs of disease, with the exceptions of geriatric, chronically sick or immune-compromised animals and perhaps pregnant animals. Old animals, however, may still be a source of infection and thus pass on infections to their offspring. Health status and background of the animal have to be considered.

- Environment:
  Dogs and cats living in kennels/catteries, animal shelters, or in crowded conditions, with perhaps 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.

- Nutrition:
  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. LIFELONG CONTROL OF MAJOR INTESTINAL PROTOZOA

2.1. *Giardia intestinalis*

2.1.1. Basic biology

Species
Giardia intestinalis (syn. *G. duodenalis*, *G. lamblia*) infects a range of vertebrates, including dogs and cats, and is currently classified into assemblages (strains or genotypes) A-G of variable host specificity. Assemblages C and D are commonly found in dogs while F has been isolated from cats and a range of other animals. Assemblage A has been found in both dogs and cats on occasions and assemblage B only rarely. Humans are usually infected with assemblages A and B.

Life Cycle
Giardia has a direct life cycle with repeated, asexual production of trophozoites (i.e. active motile stages) in the small intestine and intermittent production of resistant cysts which are passed in the faeces, initially often in large numbers. Infection is by oral uptake of cysts. Trophozoites attach to the epithelial cells after infection and the prepatent period is 4-16 days. Patency usually persists for several weeks or 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 the most frequent endoparasite in this age group. Excretion of cysts is seen in both healthy and diseased animals. The infection is believed to induce partial immunity resulting in less severe disease and in some cases in elimination of the agent but with limited resistance to re-infection. The transmission is faeco-oral by cysts in water, food or from the environment – only a few cysts are needed to cause infection. Cysts may survive in the environment for months but they are susceptible to desiccation and are greatly reduced in numbers during winter. Other vertebrates, including wildlife and humans (see 2.1.5), may serve as hosts.

2.1.2 Clinical signs

Most often infection remains asymptomatic, but may also cause chronic intermittent pasty diarrhoea rich in mucus, anorexia, vomiting, weight loss and lethargy, particularly in immunocompromised patients or puppies/kittens with coexisting infections.

2.1.3 Diagnosis

In faeces ovoid cysts, measuring 8-15 x 7-10 µm, can be detected by direct wet mounts of faeces or after concentration by sedimentation. Cysts become deformed if salt flotation is attempted. In very fresh faeces from animals with clinical signs motile trophozoites (pear-shaped, 9-21 x 5-12 µm) may be detected. Due to fluctuating excretion, and in order to improve detection, three samples over 3-5 days are recommended. Detection of antigen in faeces is also possible using a range of sensitive, commercially available test kits but these tests do not always yield comparable results, probably due to antigenic variation. Immuno-fluorescent staining is used in diagnostic laboratories.

2.1.4 Control

Treatment
One option is fenbendazole (50 mg/kg once daily for 5 days; in some countries it is only licensed for 3 days), and the treatment can be repeated if clinical signs and cyst excretions persist. Fenbendazole is registered for the treatment of giardiosis in dogs in most European countries and can also be recommended for cats. Another option is to use a combination tablet containing febantel/pyrantel/praziquantel at the standard deworming dose (15.0 mg/kg of febantel, 14.4 mg/kg pyrantel, 5.0 mg/kg praziquantel) repeated once daily for three days. This treatment is licensed in some European countries and countries outside the EU. Metronidazole (25 mg/kg twice daily for 5 days) and tinidazole are also effective but are not licensed for veterinary use. Treatment failure does occur and may be due to re-infections, co-infections or other underlying disease which should be addressed, or by incomplete parasite removal following treatment. Drug resistance has been described in human isolates. Long lasting success of treatment is often hampered by reinfection pressure from the contaminated environment, thus additional measures to reduce infection pressure are critical. Shampooing of dogs (e.g. with a product containing chlorhexidine digluconate) at the beginning and the end of antiprotozoal treatment may assist in reducing re-infections.

Prevention
Cleaning and drying of the environment, the use of clean utensils for feed and water, bathing to remove adhering faeces or cysts and proper disposal of faeces are pre-requisites to avoid animal-to-animal transmission. There are indications that cysts on surfaces are killed by quaternary ammonium compounds but no disinfectants are registered for this purpose. Surfaces should be left to dry completely. Personal hygiene of animal carers to avoid spreading of cysts is mandatory. On site 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.

2.1.5 Public Health Considerations

Assemblages A and B are generally considered zoonotic, and assemblage A is occasionally found in dogs and cats. However, in some environments dogs and humans in the same households may harbour the same genotypes. There is currently no evidence of transmission from cats to humans.

2.2. *Tritrichomonas foetus*

2.2.1. Basic biology

Species
Tritrichomonas foetus has recently been identified as a cause of diarrhoea in cats and other felids. The agent is generally considered almost identical to enteric *T. suis* in pigs and to *T. foetus* in cattle. The latter is a cause of infertility (and occasionally abortion) in cattle and, although mostly eradicated, it remains a notifiable disease in many EU countries. *T. foetus* is occasionally/rarely isolated from dogs.

Life Cycle
The life cycle is direct with trophozoite formation in both the small and the large intestine and there is no 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 14 days and the infection is often long-lasting.

Epidemiology
The infection route is considered to be faeco-oral. Prevalences may be relatively high in restricted environments like catteries and shelters but otherwise can be expected to be low, although surveys are limited in many countries. At present, there is no evidence to suggest any link between feline and bovine *T. foetus* reservoirs.

2.2.2 Clinical signs

Infections are often asymptomatic but typically kittens or otherwise naïve animals may exhibit clinical signs of *T. foetus* infection including semi-formed (“cow-pat”) faeces with blood and/or mucus and faecal incontinence with irritation and pain around the anus. The clinical course often fluctuates with transient remission after therapy (see below). Disease is seldom observed in dogs.

2.2.3 Diagnosis

Pear-shaped trophozoites (10-25 x 3-15 µm) are detected in faeces by direct wet mounts of fresh samples but sensitivity is generally low. The trophozoites are similar to *Giardia* in size but the rapid “jerky” movement and the presence of an undulating membrane in *T. foetus* are different from the sluggish motion and the typical “eyes” (two large nuclei) of *Giardia*. It also needs to be differentiated from the commensal *Pentatrichomonas hominis*, which can be seen in both cats and dogs, and occasionally other trichomonads. In contrast to other protozoans, *T. foetus* can be cultured, e.g. in a commercially available test system (InPouchTF-Feline, BioMed Diagnostics) which will not propagate *P. hominis* and *Giardia*. Direct detection by PCR is another option, which can also be used to provide speciation.

2.2.4 Control

Treatment
There are no drugs registered for use in cats against *T. foetus* and treatment recommendations are often based on case histories. Ronidazole (30 mg/kg twice daily for 2 weeks) has been used in severe diarrhoea cases with some success (off-label use). Cats must be closely monitored for drug-induced neurotoxicity (lethargy, ataxia, seizures) during the course of treatment. The signs seem reversible

when the drug is withheld. Metronidazole and fenbendazole only cause temporary remission and should be avoided. A change in diet may also alleviate clinical signs.

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

2.2.5 Public Health Considerations

T. foetus has at present no documented zoonotic potential, although care must always be taken with immuno-compromised individuals. *P. hominis* is observed in humans but little is known regarding its pathogenicity and transmission.

2.3. *Cystoisospora* (syn. *Isospora*) 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 faeco-oral route by 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 which 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) 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 diseased animals. Primary infections usually take place during the suckling period from the third to the eighth week of age. Consequently the majority of clinical cases are diagnosed in puppies/kittens of less than four months old. At that age most infections are acquired via ingestion of oocysts from the environment. Oocysts remain infective in the environment for several months and can accumulate in breeding kennels or catteries with a high density of suitable hosts, i.e. puppies or kittens. Dormozoites in paratenic hosts are infective for several years.

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. Often clinical presentation is associated with viral, helminth or bacterial co-infections; 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. As with many other coccidial infections, diarrhoea often occurs shortly before 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.

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 that can be found in the faeces of infected dogs and cats are described in Table 1.

Table 1: Characteristics of coccidian oocysts found in the faeces of dogs and cats

	Average size (µm)	Shape	Shell
*Cystoisospora**			thin, colourless or brownish
in cats: *C. felis* *C. rivolta*	 45 x 33 26 x 24	 ovoid round-oval	
in dogs: *C. canis* *C. ohioensis* *C. burrowsi*	 39 x 32 24 x 20 21 x 18	 round-oval round-oval round-oval	
Cryptosporidium		round-oval	thin, colourless unless stained
C. parvum *C. canis* *C. felis*	5.0 x 4.5 5.0 x 4.7 3.2-5.0 x 3.0-4.5**		
Toxoplasma gondii (cat)	12.4 x 10.5	round	thin, colourless
Neospora caninum (dog)	12.0 x 10.5	round	thin, colourless
Hammondia			thin, colourless
in cats: *H. hammondi*	 11.4 x 10.6	 round	
in dogs: *H. heydorni*	 11.9 x 11.1	 round	
*Sarcocystis****			
oocyst sporocyst	 11 x 8 (cat), 14 x 10 (dog)	round ovoid	very thin, colourless thick, colourless

* the oocysts of Cystoisospora spp. in fresh faeces contain a large ovum; in older faecal samples (> 12 hrs) two round sporocysts may be seen
** variable information is available
***several species in dogs and cat with morphologically indistinguishable sporocysts; oocyst wall very thin, ruptures during intestinal passage and releases two fully sporulated sporocysts which can be found in the faeces

2.3.4 Control

Treatment
Due to the fast replication of the pathogenic intestinal stage followed by excretion of oocysts in large numbers, it is critical to treat infections at an early stage. Litter mates of an affected puppy have a high risk of being infected even though they may not yet be shedding parasites. Hence, treatment should include all susceptible animals, i.e. all litter mates and in-contact puppies.

Administration of sulphonamides daily for 5-7 days is effective in controlling diarrhoea but not oocyst excretion. Toltrazuril and diclazuril are currently the drugs of choice against feline cystoisosporosis but are not licensed for use in this species. In dogs, a combination of toltrazuril/emodepside (9 mg/0.45 mg /kg bodyweight) is registered for coinfections of coccidia and roundworms. Where it is necessary to use off-label preparations of toltrazuril or diclazuril for dogs or cats, the oral formulations for mammals are suitable but the solution administered in drinking water for poultry is not suitable. Toltrazuril (9-20 mg/kg of body weight) or diclazuril (2.5-5.0 mg/kg of body weight) in a single application significantly reduce oocyst shedding in excreting animals; application in the pre-patent period largely prevents parasite excretion and reduces diarrhoea in affected litters. Available and

licensed treatments for coccidiosis in cats and dogs may vary across Europe and so it must be emphasised that it is the responsibility of the vet to use a locally approved treatment regimen.

Prevention
Due to the ubiquitous nature of the parasites, eradication is not normally feasible. The risk of acquiring an infection can be reduced by hygienic measures including 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 areas intended for keeping animals in boarding kennels, animal shelters and larger breeding units should be chosen to resist such treatment. Surfaces should be left to dry completely since this also reduces the survival of oocysts in the environment. Personal hygiene of the animal carers to avoid spreading oocysts with faecal material is very important.

2.3.5 Public Health Considerations

Isosporosis 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. In dogs and cats the following species are described:

Cryptosporidium parvum is a species with low host specificity and parasitizes mainly calves but can also infect a range of other mammals, including humans and occasionally dogs and cats. *C. canis* has been reported primarily in dogs and *C. felis* infects primarily cats but both have also been found in calves and humans. Since species differentiation relies on molecular typing the exact distribution amongst positive cats and dogs is 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 replication ends with the production of sexual stages which 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 is common and 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.

Epidemiology
Cryptosporidium oocysts are immediately infective when excreted with the faeces, so faeco-oral infections are common. They are also very small and do not sediment readily in water; they are therefore frequently water-borne and the parasite can remain infective in this environment for several months. Unlike the other coccidial species described here, *Cryptosporidium* is strictly homoxenic, and paratenic or intermediate hosts are not described.

2.4.2 Clinical signs

Immunocompetent adult animals are usually subclinically infected. Kittens and less commonly puppies can develop watery, sometimes foul-smelling diarrhoea; this can last days or occasionally weeks, and is frequently accompanied by abdominal pain, vomiting and an elevated body temperature. Diarrhoea usually starts several days after the onset of oocyst excretion. Clinical signs appear to be most severe in immuno-compromised individuals.

2.4.3 Diagnosis

Oocysts can be detected by coproscopy (see Table 1). The method of choice is faecal smear and staining (Ziehl-Neelsen, Heine, safranin). Oocysts are presented as small, round, and red or orange bodies when stained. As with *Giardia*, copro-antigen tests are commercially available and can detect infections even if the number of excreted oocysts is low. Molecular detection is both sensitive and specific but PCR tests are not commercially available.

2.4.4 Control

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

Prevention
Cryptosporidium oocysts are highly resistant so strict hygienic measures must be taken to avoid the spread of the 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 restricted to immunocompromised individuals. Owners of young animals should generally be advised to adhere to effective hygiene protocols and immunocompromised patients should not be in close contact with cats and 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 and dogs) as well as birds can act as intermediate hosts. *T. gondii* is globally present in at least three genotypes and multiple mixed forms thereof.

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. Although felids can become infected with oocysts via the faeco-oral route, this appears to be a less common route of infection for the definitive host. 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, but 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.

Epidemiology
Cats can excrete an impressive number of oocysts for a few days but subsequently they excrete few or no parasites, even after reinfection, except if the cat is immunocompromised. Due to the ubiquitous nature of the parasite, the distribution of *T. gondii* is broad in free-ranging intermediate hosts. 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; in contrast, 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

Acute toxoplasmosis 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) may play a role. Affected animals show signs of systemic infection including fever, anorexia, abdominal pain, dyspnoea, ocular inflammation, and rarely central nervous disorders. Clinical signs are seldom related to the enteric stage of development. Occasionally acute disease that may be accompanied by neuromuscular signs is observed in dogs infected with *T. gondii*.

2.5.3 Diagnosis

Excreting animals shed the small oocysts in high numbers, but due to the short patent period and infrequent re-shedding, infection is usually not detected by faecal examination. The oocysts are morphologically similar to *Hammondia* oocysts (Table 1).

Diagnosis is based on clinical signs and by the detection of specific serum antibodies in the blood. Many, but not all cats with subclinical infections may display antibody titres, so positive results only indicate previous infection. Clinical toxoplasmosis in dogs is diagnosed by serology complemented by PCR on cerebrospinal fluid.

2.5.4 Control

Treatment
Cats with clinical disease can be treated with clindamycin (oral treatment: 10-12 mg clindamycin hydrochloride/kg body weight, twice daily for four weeks; parenteral treatment: 12.5-25 mg clindamycin phosphate/kg body weight by intramuscular injection (i.m.), twice daily for four weeks). Treatment of cats after infection has not been shown to prevent oocyst excretion. Affected dogs may be treated with clindamycin or sulphonamide/trimethoprim.

Prevention
Control measures aim at the prevention of oocyst shedding in order to reduce the infection of humans with *T. gondii*. Cats should not be fed raw meat or allowed to catch and eat prey animals. However, since animals with outdoor access presumably prey on mice and other potential hosts of *T. gondii*, accidental infections cannot be completely avoided.

2.5.5 Public Health Considerations

T. gondii is one of the most prevalent parasitic zoonoses worldwide. While healthy adults have a low risk of developing severe toxoplasmosis after infection, immunocompromised individuals or children infected in utero can suffer from severe or even fatal local (mostly ocular or cerebral) or generalised toxoplasmosis. Pre-natal infections occur during a primary infection of the mother during pregnancy.

In humans the infection can be acquired either by ingestion of infected raw or undercooked meat or by ingestion of sporulated oocysts from the contaminated environment. It is therefore recommended (especially for high-risk individuals, e.g. previously unexposed pregnant women) that meat is generally consumed only after thorough cooking or freezing (-20°C for two or more days) and personal hygiene in handling meat is mandatory. Pregnant women should not lamb sheep or kid goats due to risk of hand-mouth contamination by contact with recently infected dams during delivery. 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. Within a household, cats themselves are not a risk factor for transmission to family members. A litter tray should be thoroughly cleaned every day so that any potential oocysts do not have time to sporulate.

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. It is likely that other wild canids such as wolves can also act as definitive hosts. Cattle, sheep, goats and other domestic and wild ungulates are natural intermediate hosts of the parasite, harbouring tachyzoites and cysts with bradyzoites in various tissues. *N. caninum* is a major cause of abortion in cattle.

Another species, *Neospora hughesi*, has been recognised as a cause of equine protozoal myeloencephalitis in North and South America.

Life Cycle

Dogs acquire the infection mainly by ingesting cysts containing bradyzoites located in tissues of infected intermediate hosts, in particular cattle (Fig.1). In natural infection, the pre-patent 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, even though 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.

Epidemiology

Age-related prevalence data indicate that the majority of dogs become infected after birth. Higher prevalences have been documented in old compared with young dogs. It has been reported that placentas from aborting cattle 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 bovine meat 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.

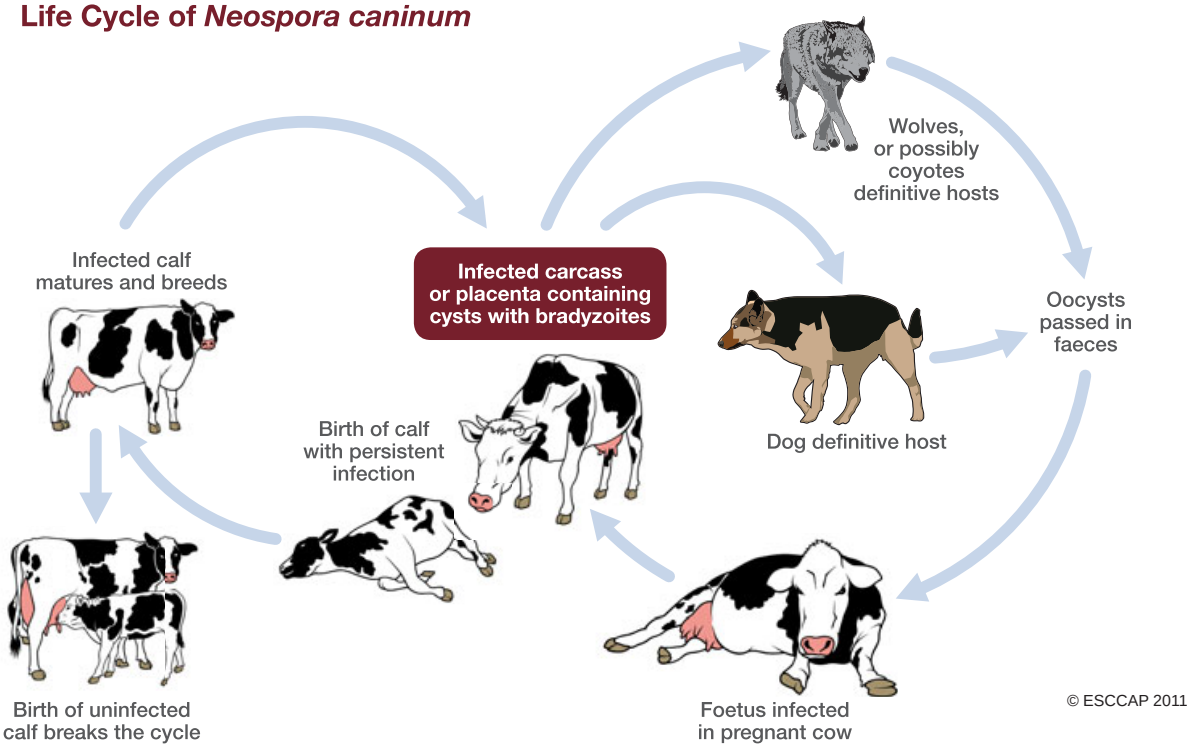

Fig. 1

In most cases of neonatal neosporosis, clinical signs are not apparent until 5 to 7 weeks after birth. This suggests that *N. caninum* is transmitted from the dam to the pups towards the end of gestation.

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 less than six months old (neonatal neosporosis) which were infected transplacentally, but *N. caninum* can cause illness in dogs of any age. Clinical signs which should arouse suspicion of neosporosis include hind limb paresis and ataxia which becomes progressively more severe. Muscle atrophy, quadriceps contracture, 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 cases of 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 can be affected, although not necessarily simultaneously.

2.6.3 Diagnosis

Unsporulated oocysts in faeces measure on average, 12 x 10.5 µm and are microscopically identical to *Hammondia* oocysts (Table 1). Differential recognition can be obtained by 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. A clinical suspicion of canine neosporosis 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. 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 (ELISA, IFAT).

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 results of serology. Treatment with clindamycin (20 mg/kg twice daily for 30-60 days) has been reported to improve clinical recovery in naturally infected dogs with neurological signs. Alternatively sulphonamide/trimethoprim 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 any breeding programme. Furthermore, farm dogs should be fed cooked meat and prevented access to raw meat offal, and faecal contamination of water and cattle feedstuff avoided.

2.6.5 Public Health Considerations

There is no known zoonotic potential, although antibodies have been reported in humans.

2.7. *Hammondia* spp.

2.7.1. Basic biology

Species
Two species of *Hammondia* parasitize 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*). Excretion 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 geographic 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, has 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 avoiding the ingestion of tissue cysts with tissues 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.

2.8. *Sarcocystis* spp.

2.8.1. Basic biology

Species
Within the genus *Sarcocystis* several species parasitize cats or dogs as definitive hosts. The faecal stages, so-called sporocysts, are morphologically indistinguishable. Differentiation is based on tissue cyst morphology in the different intermediate hosts (omnivorous or herbivorous animals) and experimentally, on molecular methods.

Life Cycle
Carnivorous animals become infected by ingestion of meat containing tissue cysts. In the intestinal epithelium of the definitive host sexual development takes place which results in the production of an oocyst which 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 extra-intestinally into tissue cysts. The pre-patent 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. The 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 clinical signs. Cysts in carcasses can lead to meat condemnation. After reinfection dogs and cats usually develop some degree of immunity which is species-specific.

2.8.3 Diagnosis

Sporocysts (see Table 1) can be found in faeces in low numbers. Tools for species diagnosis are not available.

2.8.4 Control

Treatment
Treatment of dogs or cats is not necessary.

Prevention
Since *Sarcocystis* is strictly heteroxenic, infection can be avoided by feeding meat that has been either previously frozen (-20 °C for at least 4 days) or cooked. To interrupt transmission, dogs and if possible also cats should not be allowed to defaecate on animal feed or pastures.

2.8.5 Public Health Considerations

None of the *Sarcocystis* spp. involving dogs and cats are zoonotic. Human infections with *Sarcocystis* take place via ingestion of infected beef or pork.

3. ENVIRONMENTAL CONTROL OF PARASITE TRANSMISSION

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

4. OWNER CONSIDERATIONS IN PREVENTING ZOONOTIC DISEASES

The most important advice for prevention of transmission of zoonotic agents including certain intestinal protozoa discussed in this guideline is personal hygiene. Washing hands after contact with dogs, cats and other animals should become normal behaviour. Since many of the intestinal protozoan infections mentioned do little or no harm to dogs and cats (especially adult animals) or, in many cases, to pet owners, these infections go unnoticed. Fortunately the majority of intestinal protozoan infections in dogs and cats are species-specific. Human infections with *Toxoplasma* are mainly either food, water or soil-borne. Direct contact with cats is not a risk factor, however contact with cat faeces and contaminated food/water are risk factors. Human infections with *Sarcocystis* are exclusively transmitted between human-cattle and human-pig respectively. There is no relation with dogs/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 true for individuals with immune deficiency disorders or individuals 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 which would otherwise resolve in immunocompetent individuals.

5. STAFF, PET OWNER AND COMMUNITY EDUCATION

Even within the veterinary profession, the knowledge of intestinal protozoan infection in cats and dogs is limited. *Giardia* and *Cryptosporidium* are the only recognised potential zoonoses, and *Toxoplasma* is often mistakenly considered as exclusively transmitted between cats and humans (pregnant women). It has to be emphasized that only sporulated oocysts of *T. gondii* are infective and if litter boxes are thoroughly cleaned daily the infection risk is minimized. The vast majority of *Toxoplasma* infections are acquired from food or the environment (water/soil). The information in this guideline therefore, deserves to be widely disseminated in veterinary practices including all auxiliary personnel. Correct knowledge of protozoan infections is a pre-requisite 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 at www.esccap.org

APPENDIX 1 - BACKGROUND

ESCCAP (European Scientific Counsel Companion Animal Parasites) is an independent, not-for-profit organisation that develops guidelines and promotes good practice for the control and treatment of parasites in companion animals. With 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 wellbeing 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 infestations***

- ***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 infestation 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 infestation 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 dose-rates 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
Bradyzoites	slow-dividing tissue stages contained within a pseudocyst or maturating tissue cyst
Cysts	a) environmental resistant stage of *Giardia* excreted with faeces able to survive outside the host; b) mature stage of heteroxenic 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
Excystation	escape of parasite stages from the multilayered shell which cover the environmental stages (see cyst, oocyst)
Heteroxenic	infecting several host species in the life cycle
Homoxenic	infecting only one host species in the whole life cycle
Intermediate host	a host in which asexual reproduction or development is completed
Oocyst	a tenacious spore stage 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
Schizogony	see asexual reproduction
Sporocysts	a multilayered stage within oocysts that contains the sporozoites
Sporozoites	the cellular infective units that evolve from excystation of oocysts and sporocysts
Sporulation	development of sporozoites from the stages of sexual development
Tachyzoites	fast-reproducing parasite stages within the host cell
Tissue cyst	see cysts
Trophozoites	motile, active stages in the host e.g. within the life cycle of *Giardia* and other protozoa
Zoonosis	any infectious disease that can be transmitted between humans and non-human vertebrates
Zoonotic	transmissible between non-human vertebrate hosts and humans stages (see cyst, oocyst)

ESCCAP

EUROPEAN SCIENTIFIC COUNSEL COMPANION ANIMAL PARASITES

Control of Intestinal Protozoa in Dogs and Cats

ESCCAP Guideline 06 First Edition – August 2011

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ISBN 978-1-907259-24-1

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