GUIDELINE for Toxoplasma gondii infection

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The Toxoplasma gondii infection in cats guidelines that the present article is updating were published in J Feline Med Surg 2013; 15: 631-637;[44] this update has been compiled by Katrin Hartmann.

Modified October 2023, work for an update is in progress.

Synopsis

Toxoplasma gondii infection is common in cats, but the clinical picture is rare. Up to 50% of cats, especially free-roaming ones, have antibodies indicating infection and the presence of cystic stages. Clinical signs usually appear when cats become immunosuppressed – in these situations, cystic stages can be reactivated. Organs commonly affected are the CNS, muscle, lungs, and eyes. Cats can pose a risk for humans when they shed oocysts. However, this happens only once in their lifetime, usually only for three to ten days after ingestion of tissue cysts. Thus, cats that have antibodies to *T. gondii* do no longer shed oocysts and neither are nor will become a risk for humans.

Agent properties

Toxoplasma (T.) gondii is an obligate intracellular coccidian parasite that can infect virtually all species of warm-blooded animals, including people. Domestic cats and other felids are the natural hosts – non-feline species serve only as intermediates.[1,2]

Three infectious structures can be distinguished: sporozoites in oocysts, tachyzoites (the actively multiplying stage), and bradyzoites (the slowly multiplying stage) enclosed in tissue cysts. Oocysts are excreted in faeces, whereas tachyzoites and bradyzoites are found in tissues and milk.[1,2]

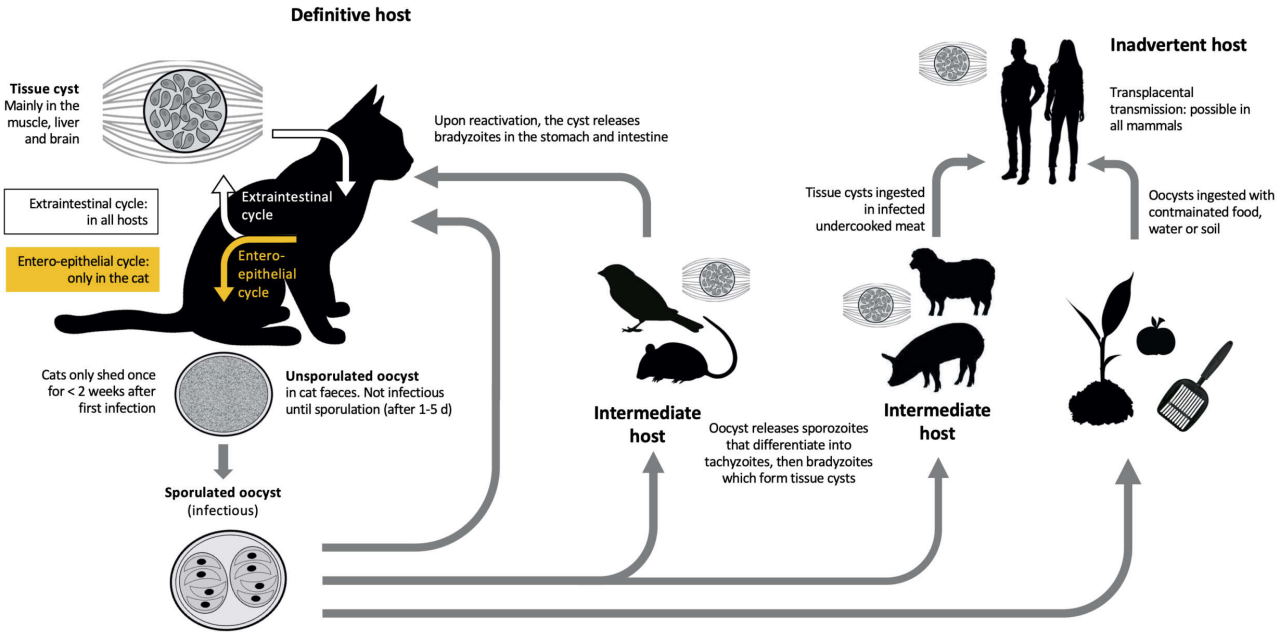

Fig. 1. Toxoplasma life cycle. Cats and humans can be infected by transplacental or lactogenic transmission, or by ingestion of tachyzoites or bradyzoites in tissue (i.e. meat) cysts or oocysts. ©ABCD, Karin de Lange

Pathogenesis

The entero-epithelial (coccidian) life cycle

This cycle is found only in the feline host. Most cats are infected by ingesting intermediate hosts – typically rodents – infected with tissue cysts. Bradyzoites are released in the stomach and intestine from the tissue cysts when digestive enzymes dissolve their wall. They enter epithelial cells of the small intestine and give rise to schizonts, initiate five types of predetermined asexual stages, and merozoites released from the schizonts eventually form male and female gamonts. After fertilization, a wall is formed around the fertilized macrogamont to form an oocyst (Figs. 2, 3). Oocysts are round to oval, 10 x 12 µm in size, and are still unsporulated (not infectious) when passed in faeces. After exposure to air and moisture for one to five days, they sporulate to contain two sporocysts, each with four sporozoites.[1,2]

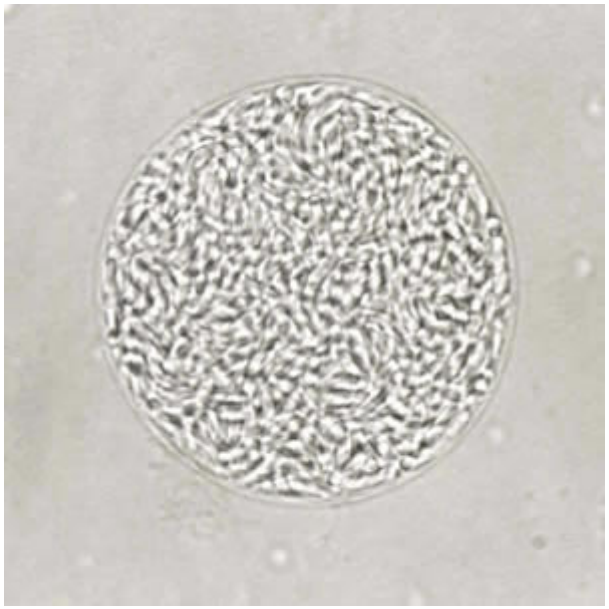

Fig. 2. T. gondii tissue cyst (unstained). Slowly-dividing bradyzoites can be seen inside. From the public domain, Wikipedia USA

The cycle is usually completed within three to ten days after ingestion of tissue cysts, which is the case in up to 97% of naive cats. Only after the rare event in which cats ingest oocysts or tachyzoites, formation of new oocyst is delayed and shedding can occur up to 18 days (rarely more). However, only 20% of cats fed oocysts will shed.[1,2]

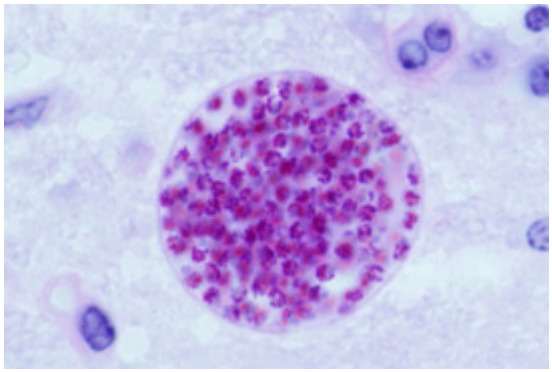

Fig. 3. Cysts develop in the tissues of many vertebrates, here in mouse brain; resting parasites (stained red) are enveloped by a thin cyst wall. Image is in the public domain, originally from the Agricultural Research Service, US Dept of Agriculture

The extra-intestinal life cycle

The extra-intestinal development of *T. gondii* is the same for all hosts, including cats, dogs, and people, whether tissue cysts or oocysts had been ingested. After the ingestion of oocysts, sporozoites hatch in the lumen of the small intestine and enter intestinal cells, including those in the *lamina propria*. Sporozoites divide into two by an asexual process known as endodyogeny, thereby becoming tachyzoites. These are lunate (falciform) in shape, approximately 6 x 2 µm, and multiply in almost any cell of the body. When the cell ruptures, releasing the tachyzoites, these infect new cells. Otherwise, tachyzoites multiply intracellularly for an undetermined period, and eventually encyst. Tissue cysts vary in size from 15 to 60 µm and usually conform to the shape of the parasitized cell. Tissue cysts are formed mainly in the CNS, muscles, and visceral organs, and probably persist for the life of the host. They can be reactivated after immunosuppression, which may then lead to clinical signs.[1,2]

Parasitaemia during pregnancy of the host can cause placentitis and spread of tachyzoites to the foetus. Many kittens born to queens infected with *T. gondii* during gestation become infected transplacentally or when suckling. Clinical signs are common in them, varying with the stage of gestation at the time of infection; some of these newborn kittens shed oocysts.[1,2]

Epidemiology

Antibody prevalence to *T. gondii* varies geographically; in Portugal, 24% of the cats had antibodies[3], in the USA 16% to 40% were seropositive, depending on the state[2]. However, only 3 of 326 faecal samples from cats in California and 1 out of 252 in Switzerland contained *T. gondii* oocysts.[4] The annual burden in the environment is about 90 to 5,000 oocysts/square meter.[5] The age of the cat does not play a role in the frequency of *T. gondii* shedding, but the season does: shedding is more common between July and December.[6]

The three major modes of transmission of *T. gondii* in all host species are congenital infection, ingestion of infected tissue, and ingestion of oocyst-contaminated food or water.[2] Less important are blood transfusions and organ transplantations.[1,2] Lactogenic transmission is suspected because the organism has been detected in queens milk.[7]

T. gondii blocks the innate aversion of rats for cat urine, instead makes them attracted by the feline pheromone, which may increase the likelihood of a cat capturing an infected rat. This reflects adaptive, "behavioural manipulation" by *T. gondii* in optimizing the chances for completing the parasite's life cycle: it reproduces only in the feline intestine. The behavioural manipulation hypothesis postulates that a parasite will specifically manipulate host conduct essential for its transmission. However, the neural circuits for innate fear, anxiety, and acquired fright all overlap, raising the possibility that *T. gondii* may disrupt all of these non-specifically.[8] Experimental infections have shown that *T. gondii* may change chemical messages in the CNS that affect rodent behaviour; the infection may lead to cyst formation in the CNS with production of tyrosine hydroxylase, resulting in a lack of dopamine.[9-14]

Meat contaminated with *T. gondii* cysts has been the primary source of infection in persons, and antibody prevalence in human beings is relatively high. Exposure from oocyst-contaminated soil or water is common. Indeed, water-borne outbreaks of toxoplasmosis have been reported worldwide and support the theory that exposure to environmental oocysts poses a significant health risk.[15]

Fig. 4. Cat with toxoplasmosis suffering from myositis caused by T. gondii cysts. The cat presented in lateral recumbency, was unable to get up, and showed severe muscle hyperesthesia (courtesy of Katrin Hartmann, Medizinische Kleintierklinik, Ludwig-Maximilians-Universitaet Muenchen, Germany).

Clinical signs

Clinical signs develop due to inflammation and tissue necrosis caused by intracellular growth of tachyzoites.[2] Congenital infection tends to be more serious than infection of the adult cat.[2]

Clinical toxoplasmosis develops during dissemination and intracellular replication of tachyzoites. It usually originates from reactivation of a latent infection rather than after a newly acquired infection. If a carrier cat is immunosuppressed, bradyzoites in tissue cysts replicate rapidly and disseminate again as tachyzoites. Clinical toxoplasmosis has been documented in cats infected with feline

immunodeficiency virus (FIV) or feline leukaemia virus (FeLV).[16] Commonly used doses of glucocorticoids do predispose to reactivation.[17] However, administration of cyclosporine to cats with renal transplants or dermatologic disease has been associated with clinically manifestations.[18-20]

The most commonly affected tissues are the CNS, muscles (Fig. 4), lungs (Fig. 5) and eyes. Hepatic and pancreatic involvement is less likely. Cats with toxoplasmosis show neurologic signs (e.g., seizures, ataxia), muscle hyperesthesia, dyspnoea, uveitis, icterus, diarrhoea, fever, depression, anorexia, and weight loss.[2] Transplacentally or lactogenically infected kittens develop more severe signs and frequently die of pulmonary or hepatic disease.[21] Immune complex formation[22] and deposition in tissues as well as delayed hypersensitivity reactions may be involved in chronic forms of toxoplasmosis. Since *T. gondii* is not cleared from the body, neither naturally nor through treatment, toxoplasmosis may recur.

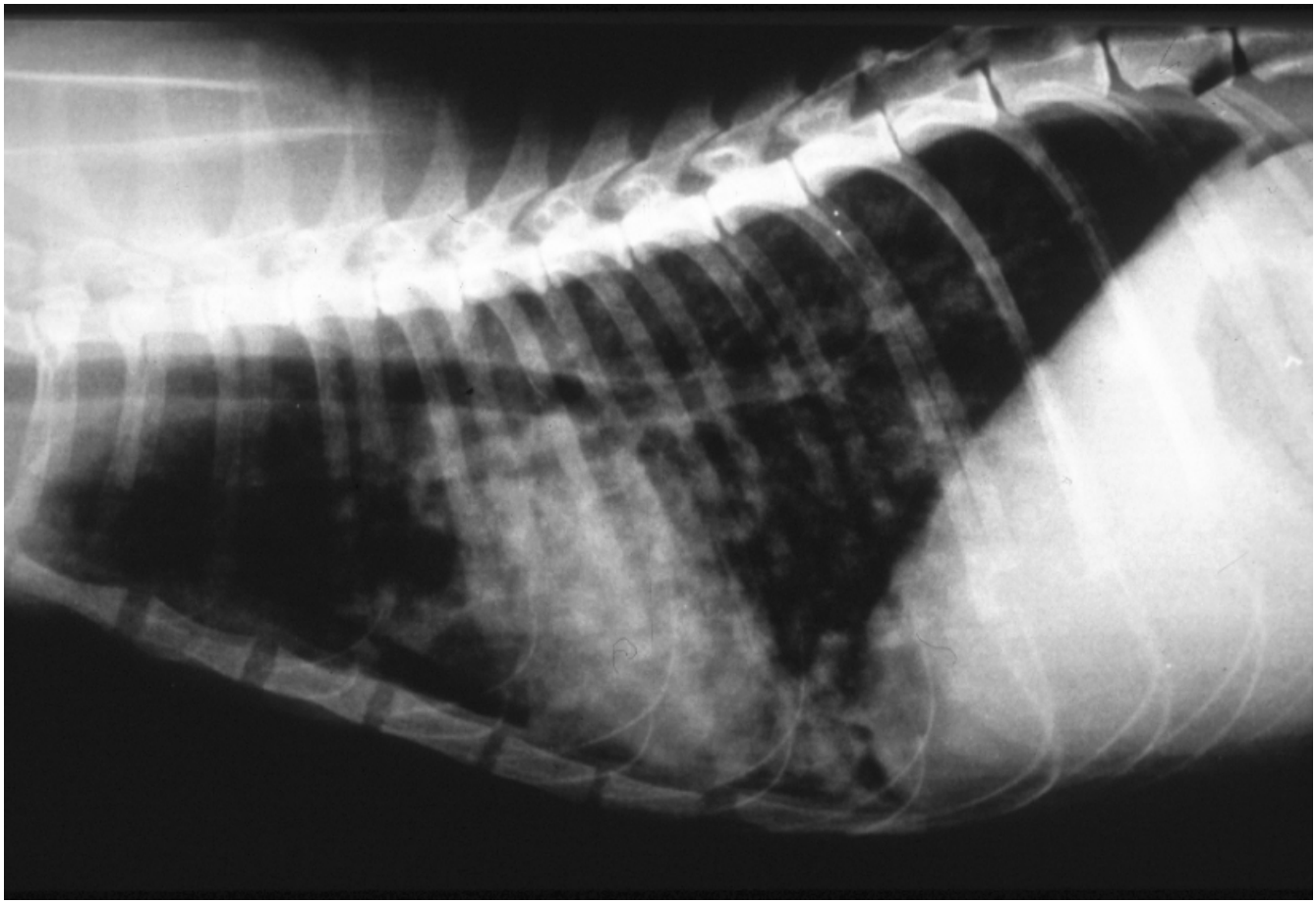

Fig. 5. Thoracic radiographs (latero-lateral view) of a cat with pulmonary toxoplasmosis (courtesy of Katrin Hartmann, Medizinische Kleintierklinik, Ludwig-Maximilians-Universitaet Muenchen, Germany).

Immunity

Immunity to *T. gondii* in the cat is poorly understood. In the mouse and in humans, it is highly dependent on cell-mediated effector responses.[23]

All infected cats develop IgG and IgA antibodies, about 80% also have IgM antibodies. IgG can take four to six weeks to appear, and maximal antibody titres are achieved within two to three weeks after first appearance.[2]

Diagnosis

Clinical toxoplasmosis is ideally diagnosed by detection of the organism in muscle biopsies or bronchoalveolar lavage, or by PCR in CSF

or humor aqueous. During acute illness, tachyzoites can be detected in tissues and body fluids by cytology. They are rarely found in blood, but occasionally in CSF, fine-needle aspirates of organs (e.g., lymph nodes), and transtracheal or bronchoalveolar washings, and are common in the peritoneal and thoracic fluids of animals developing thoracic effusions or ascites. Detection of tachyzoites confirms the diagnosis.

A tentative diagnosis may be based on increasing IgM titers, exclusion of other causes of the clinical signs, and a beneficial clinical response to an anti-toxoplasma drug.[1,2]

Oocyst shedding is diagnosed by microscopy of faecal samples. Diagnosis of the disease is only confirmed when the organism is found in body fluids or tissue. If suitable samples cannot be taken, a tentative diagnosis is sometimes based on rising IgM titers, exclusion of other causes for the clinical signs, and a favourable clinical response to anti-*T. gondii* drugs.[1,2]

Detection of oocysts in faeces

T. gondii oocysts are 10 µm in size and best demonstrated by centrifugation using Sheather's sugar solution (saccharose solution with a specific gravity of 1.27 g/ml) during the shedding period (Fig. 6). *T. gondii* oocysts are morphologically indistinguishable from those of *Hammondia hammondi*, *Besnoitia orcytofelisi,* and *Besnoitia darling*.[2] A CsCl method for easy purification of *T. gondii* oocysts from faeces of infected cats has been described.[15]

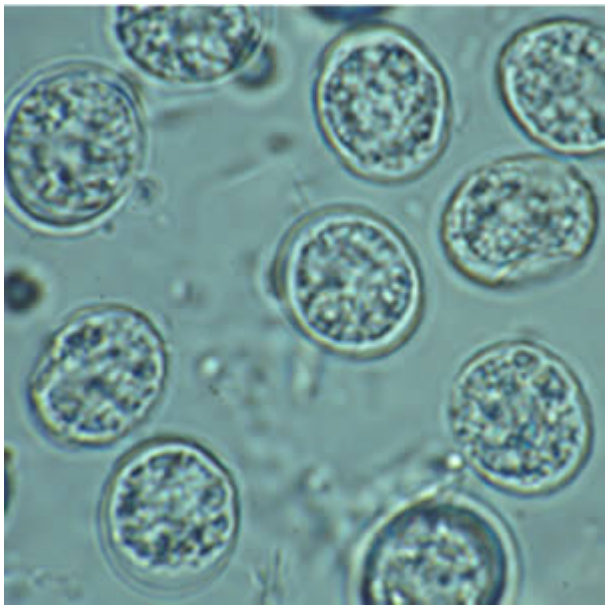

Fig. 6. T. gondii oocysts in fecal flotation. Source http://dpd.cdc.gov/dpdx/HTML/ImageLibrary/Toxoplasmosis_il.htm; US Center for Disease Control and Prevention

Detection of tachyzoites

Ante-mortem diagnosis of clinical toxoplasmosis ideally is based on the detection of the organism by cytology or PCR. Tachyzoites may be detected in various tissues and body fluids during acute illness (Fig. 7). They are rarely found in blood, but occasionally in CSF or aqueous humour, fine-needle aspirates of organs (e. g. lymph nodes), and transtracheal or bronchoalveolar washings. Detection of tachyzoites results in a definitive diagnosis. Alternatively, a PCR can be performed using CSF, aqueous humour, or bronchoalveolar lavage fluid.

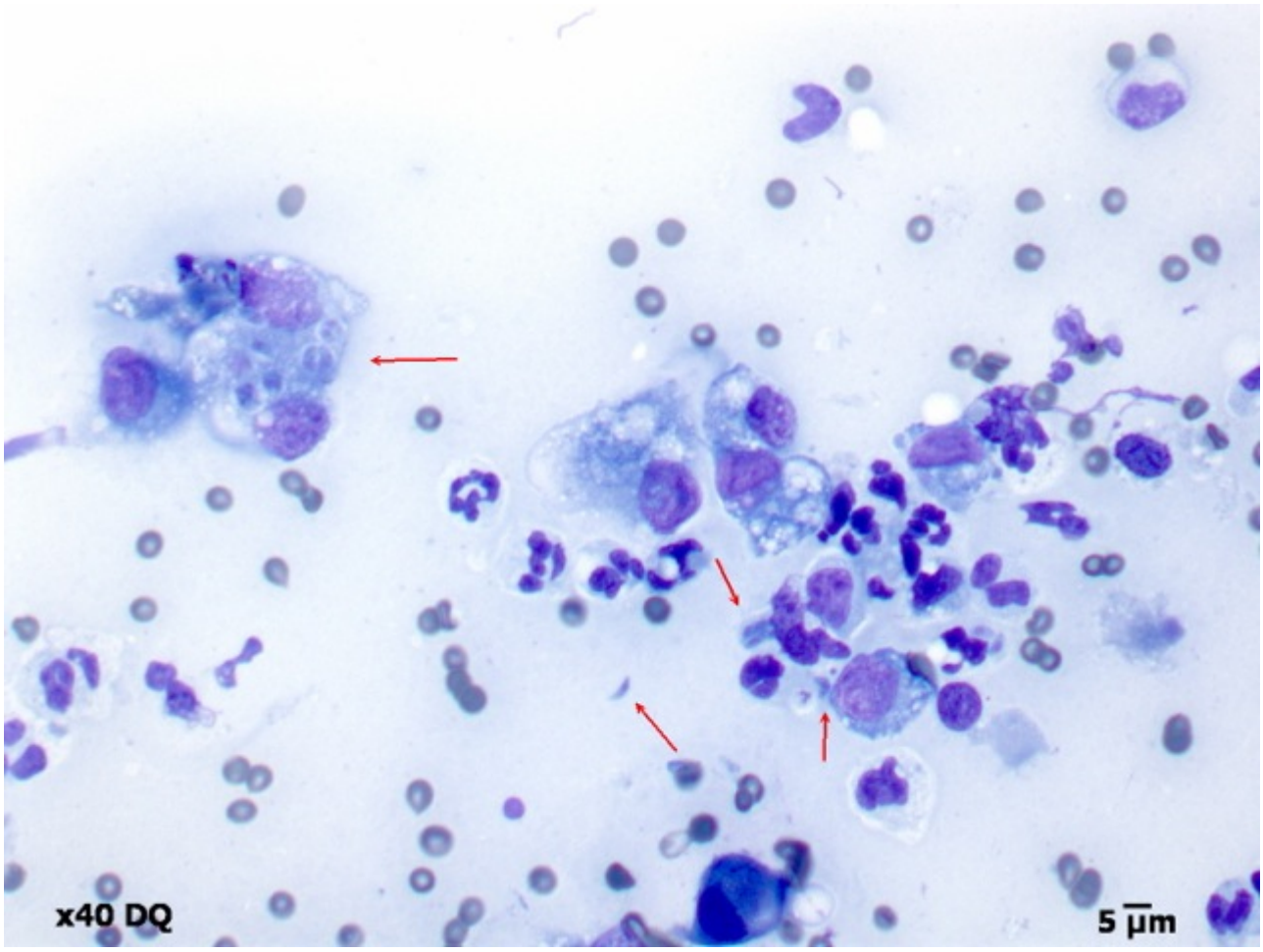

Fig. 7. Cytology of a fine needle aspirate of a cat with pulmonary toxoplasmosis and lung consolidation with numerous intracellular and extracellular T. gondii tachyzoites and cysts (arrows). Courtesy of George Reppas, Vetnostics, Australia.

Serology

Using the immunofluorescence test (IFA), antibodies of the IgM, IgG, and IgA isotypes can be detected. For assessing human health risks, test results from healthy cats are useful. A seronegative cat may be shedding oocysts (early during infection, before antibodies had time to develop) and will likely shed oocysts if exposed for the first time. An antibody-positive cat is unlikely to shed oocysts: antibodies need two to three weeks to develop, by that time cats usually do no longer shed, and it sheds only once in its lifetime. It is also unlikely to shed oocysts if re-exposed or immunosuppressed.[1]

Antibodies are commonly found in both healthy and sick cats. Thus, their presence does not prove clinical toxoplasmosis. Antibodies of the IgM class are commonly detected in healthy cats and do not correlate with clinical signs.

Antibody test results from healthy cats are useful to assess the health risk for humans. An antibody-negative cat could be shedding oocysts (early after infection, before antibodies have developed) or will shed oocysts if exposed; this cat poses the greatest public health risk.

An antibody-positive cat is unlikely to shed oocysts, because antibodies need two to three weeks to develop, and by that time, the infection has been controlled; also, shedding usually occurs only once in its lifetime. A seropositive cat is also unlikely to shed oocysts when re-exposed or immunosuppressed.[1] In one study, cats inoculated with *T gondii* tissue cysts were orally re-challenged several years later, and a few of them did shed oocysts after this second challenge – although only low amounts and over a short time.[38] This, however, has never been shown to occur in naturally infected cats. Thus, the risk of shedding by an antibody-positive cat is low.

Antibodies are common in both healthy and diseased cats and therefore do not prove clinical toxoplasmosis. Not only IgG antibodies, but also antibodies of the IgM class are commonly detected in healthy cats and stay high over long periods; thus their detection is of no

use either for diagnosing toxoplasmosis. *T gondii*-specific IgM is detected in the serum of cats with latent or reactivated infection and titers therefore do not indicate recent exposure. If increasing IgM titers are detected, however, this may raise the suspicion of clinical toxoplasmosis.

DRUG	ABCD RECOMMENDATION	COMMENT	EVIDENCE-BASED GRADE
Antiparasitic therapy Clindamycin	10-12 mg/kg PO q12h		III
Symptomatic therapy Prednisolone acetate (1%)	Use in addition to systemic antibiotic treatment Apply topically to the eye q6-8h	For cats with toxoplasma-induced uveitis (to avoid secondary glaucoma and lens luxation)	IV

Table 1. Treatment of Toxoplasmosis

Treatment

Clindamycin is the treatment of choice[24] and should be administered at 10 – 12 mg/kg orally q 12 h for four weeks (Table 1). Cats with systemic disease and uveitis should be treated with clindamycin in combination with topical, oral, or parenteral glucocorticoids, to avoid secondary glaucoma and lens luxation.[25] Prednisolone acetate (1% solution) applied topically to the eye three to four times daily is generally sufficient.

Clinical signs not involving the eyes or the CNS usually begin to resolve within the first two to three days of clindamycin administration. CNS and ocular toxoplasmosis tend to respond more slowly. In cases of pulmonary toxoplasmosis, radiographic abnormalities might not resolve for several weeks. Prognosis is usually poor in pulmonary or hepatic disease, particularly in immunocompromised animals.[26]

Prevention

Prevention of infection

Preventing toxoplasmosis in cats involves measures intended to reduce the incidence of infections and the shedding of oocysts into the environment. Cats should preferably be fed commercially available, processed food. Prevalence of feline *T. gondii* infection is higher in countries where raw meat is fed. Freezing or irradiation can kill tissue cysts without affecting meat quality. Pets should be prevented from hunting and eating intermediate hosts (rodents) or mechanical vectors, such as cockroaches and earthworms. If meat is fed, it should be thoroughly cooked, even if frozen. Cats should be prevented from entering buildings where food-producing animals are housed or where feed storage areas are located.[1]

Box 1

- Ingestion of meat containing tissue cysts is the most common route of infection. Thorough cooking or freezing for several days will kill tissue cysts.[29-32]
- Ingestion of sporulated oocysts, either from the environment, e. g., through contact with contaminated soil, or from faeces of shedder cats is the second most common route. This may also happen when eating unwashed fruit or vegetables. Infection *via* the environment is more common than from directly through cat contacts

Less common routes of infection:

- Ingestion of sporulated oocysts through contact with contaminated water
- Ingestion of raw (unpasteurised) goat milk
- Inhalation of sporulated oocysts on dust particles (rare)

Public health considerations

Public heath issues have come into focus, partially because of the increasing number of immunocompromised persons (e.g. after infection with human immunodeficiency virus). Also, recent research linking psychological and cognitive disorders (e.g. reduced IQ, as evidenced by psychomotor and verbal intelligence tests) to *T. gondii* infection may have contributed (Box 1). A recent survey amongst obstetrician-gynaecologists in the USA to determine their knowledge and practices about toxoplasmosis prevention and testing found that most overestimated the risk of cat ownership *vs* environmental risk factors.[27] A systematic review of risk factors for pregnant women is available;[28] it reports a relatively low risk of cat ownership.

Box 2

Recommendations to reduce the risk of parasite transmission from cat to owner:

- Litter trays should be emptied daily so that oocysts do not have sufficient time (24 hours) to sporulate
- Gloves should be worn when handling cat litter, and hands should be washed thoroughly after cleaning of litter trays
- Litter tray liners should be used if possible, and the tray cleaned regularly with detergent and scalding water
- Cat litter should be disposed in sealed plastic bags
- Children's sandpits should be covered when not in use, to prevent cats from using them
- Only properly cooked food or commercial cat food should be fed
- Hands should be washed after contact with a cat (especially before eating).

Veterinarians commonly get questions from clients whether or not to get rid of their cat. If hygiene recommendations are followed (Box 2, 3), the risk of transmission is low (Box 4)

Box 3

For households with immunocompromised persons or pregnant women, the following additional advice is given:

- Immunosuppressed persons and pregnant women should avoid contact with cat litter
- Cats should be kept indoors to prevent hunting and eating intermediate hosts such as voles and mice
- Cats should not be fed raw or partially cooked meat
- Cats should be discouraged to eat insects (e. g., cockroaches).
- Cats should be tested for *T. gondii* antibodies; their presence indicates past infection. These cats will not be a source of infection as they have completed their period of oocyst shedding.
- Cats without antibody had not been infected earlier and, when newly infected, will shed oocysts in their faeces for a short time. They should therefore be kept indoors during the phase of immunosuppression or pregnancy of the owner.

Box 4

Contact with cats does not increase the risk of *T. gondii* infection.[33] (see: http://www.fabcats.org/owners/toxoplasmosis/info.html):

- Cats shedding oocysts in faeces are rare.[34] In one study, only about 1/ 250 cats shed oocysts.[4]
- Contact with cats has no influence on the probability of people developing antibodies to *T. gondii*, whereas consuming raw meat significantly increases the risk of acquiring the infection.[38]
- Veterinarians working with cats are not more likely to become infected with *T. gondii* or to suffer from toxoplasmosis than the general population, including people without cat contacts.[35-37]
- Stroking a cat will not spread the infection. Even when cats are shedding oocysts in their faeces, they cannot be found

on their coat.[39] Studies in dogs have shown that oocysts do not sporulate on their fur and the same is probably true for cats.[40]

- Cat ownership does not increase the risk of toxoplasmosis in persons with an HIV infection. Although toxoplasmosis is more common in HIV-infected persons, the disease results from reactivation of a previous infection rather than from acquiring a new infection.
- Most people are infected with *T. gondii* through ingestion of undercooked meat, especially goat, mutton, and pork. The risk of infection from cats is low, except for young children playing in soil contaminated with sporulated oocysts.[41]
- Bites or scratches from an infected cat do not transmit the infection.
- Infected cats under treatment with immunosuppressive drugs at standard doses do not start shedding oocysts in their faeces.[17]
- Infected cats also do not re-shed oocysts in their faeces when they become immunosuppressed due to infection with FIV or FeLV.[42] Cats infected with FIV or FeLV that are subsequently infected with *T. gondii* do not shed oocysts for any longer or in any greater numbers than other cats.[2,43]
- Newly identified strains of *T. gondii* are highly infectious for species other than cats; thus, cats might actually become less important in the spread of this infection.

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