

# Toxoplasmosis

## *Toxoplasma Infection*

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## Importance

Toxoplasmosis is a zoonotic disease caused by the protozoal parasite *Toxoplasma gondii*. Felids are the definitive hosts for *T. gondii*, but encysted parasites can survive for very long periods, perhaps lifelong, in the tissues of most or all hosts. Some clinical cases result from new exposures to *T. gondii*; others occur when parasites in tissue cysts become reactivated. Infection with *T. gondii* is common in warm-blooded animals, including humans, and usually causes no illness or mild clinical signs in immunocompetent, non-pregnant individuals. However, infections acquired during pregnancy can result in mild to serious congenital defects in the fetus, and immunocompromised humans or animals can develop severe, life-threatening infections. Recently, serious and life-threatening infections among immunocompetent people in French Guiana and Suriname have raised the possibility that some unusually virulent strains of *T. gondii* may exist in tropical forests.

## Etiology

Toxoplasmosis is caused by *Toxoplasma gondii*, an obligate intracellular protozoan parasite in the order Coccidia and phylum Apicomplexa. Some strains of *T. gondii* are more virulent than others in mice. However, it is not yet clear how widely this applies to other hosts, and strains that are nonvirulent in mice are not necessarily nonvirulent in other species.

## Species Affected

Members of the Felidae, including domesticated cats, are the definitive hosts for *T. gondii*. Most or all mammals and marsupials can serve as intermediate hosts. While the vast majority of infections are subclinical, clinical cases can occur in diverse species. Among domesticated animals, small ruminants and pigs are affected most often, but a few cases have been documented in other hosts including cats, dogs, horses and camelids. Toxoplasmosis seems to be relatively common in captive New World monkeys, some captive Macropodidae (e.g., wallabies), various captive or wild marine mammals, and squirrels. Among felids, sand cats (*Felis margarita*) and Pallas cats (*Otocolobus manul*) may be particularly susceptible. Rare clinical cases have been reported in many other species, such as wild hares (*Lepus europaeus*, *L. timidus*), a wild mink (*Mustela vison*), captive porcupines, a captive red panda (*Ailurus fulgens fulgens*), a captive giant panda (*Ailuropoda melanoleuca*) and even captive fruit bats (*Pteropus conspicillatus*, *P. scapulatus*).

Birds can be intermediate hosts for *T. gondii*, with asymptomatic infections documented in a number of species. Clinical cases or outbreaks have been reported uncommonly in birds from multiple orders, including Columbiformes (pigeons and doves) Passeriformes (passerine birds), Piciformes (woodpeckers), Psittaciformes (parrots and other psittacines), Galliformes (e.g., chickens, turkeys, guinea fowl), Sphenisciformes (captive black-footed penguins, *Spheniscus demersus*) and Apterygiformes (North Island brown kiwi, *Apteryx mantelli*).

*T. gondii* DNA has been detected in captive snakes, and some fish and marine mollusks may act as transport hosts.

## Zoonotic potential

*T. gondii* affects humans.

## Geographic Distribution

*T. gondii* occurs throughout the world. This organism is especially prevalent in warm, humid climates, but significant numbers of animals and humans have been exposed even in very cold regions such as the Arctic.

## Transmission and Life Cycle

### Life cycle

There are four major forms of *T. gondii*: oocysts, which are shed in the feces of the definitive host, (containing sporozoites after sporulation); tachyzoites, rapidly multiplying organisms found in the tissues; bradyzoites, slowly multiplying

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organisms found in the tissues; and tissue cysts, which are walled structures that contain bradyzoites.

*T. gondii* undergoes an asexual reproductive cycle in all species. When the parasite is eaten, the tissue cyst or oocyst wall is dissolved during digestion, and releases bradyzoites or sporozoites, respectively. These organisms enter the lamina propria of the small intestine and begin to multiply as tachyzoites. Tachyzoites can disseminate to extraintestinal tissues within a few hours of infection, via the lymph and blood. They can enter nearly any cell and multiply; the host cell eventually ruptures and the released tachyzoites enter new cells. As host resistance develops, tachyzoites begin to disappear, and form bradyzoites within tissue cysts. Tissue cysts can be found in many organs, but are particularly common in skeletal muscle, myocardium and the central nervous system (CNS). They generally do not cause a host reaction, and can persist for many years, possibly lifelong. While bradyzoites in tissue cysts have traditionally been viewed as "resting," new research suggests that they continue to replicate. Tissue cysts occasionally rupture and release parasites, which are readily controlled by the immune response in immunocompetent individuals, but may multiply and spread if the host becomes immunosuppressed. Toxoplasmosis is often a reactivated rather than a new infection in AIDS patients. Many clinical cases in older or immunosuppressed cats are also thought to result from reactivated infections. Some tissue cysts may eventually die.

In Felidae, the parasites simultaneously undergo a sexual replication cycle: some parasites multiplying in intestinal epithelial cells initiate a sexual cycle (gametogony), resulting in the formation of an unsporulated oocyst. Oocysts are shed in the feces, with a prepatent period of 3-21 days in domesticated cats. They appear earlier (3-10 days) when cats are infected via tissue cysts than oocysts. The oocyst sporulates in the environment, forming two sporocysts, each with four sporozoites. Sporulation occurs in 1 to 5 days under ideal conditions, but may take up to several weeks. Most cats excrete oocysts for 1-2 weeks, although shedding for up to 3-4 weeks has been reported. Cats usually shed oocysts only on their first exposure to *T. gondii*, and seem to be resistant to reinfection; however, experiments have demonstrated that re-infection and re-shedding are possible under some conditions.

Unsporulated oocysts lose their ability to sporulate and become non-infectious if they are frozen for 7 days at  $-6^{\circ}\text{C}$  ( $21^{\circ}\text{F}$ ), or heated for 1 day at  $37^{\circ}\text{C}$  ( $99^{\circ}\text{F}$ ). Sporulated oocysts are highly resistant to environmental conditions. Under laboratory conditions, they can remain infectious for more than a year in warm, moist soils, and for up to several years in cold ( $4^{\circ}\text{C}$ ) water. They are reported to survive freezing at  $-10^{\circ}\text{C}$  ( $14^{\circ}\text{F}$ ) for nearly 4 months, or heating at  $35^{\circ}\text{C}$  ( $95^{\circ}\text{F}$ ) for 32 days. However, they do not survive well in arid, cool climates. Tissue cysts can remain infectious for weeks in body fluids at room temperature, and in meat for

as long as the meat is edible and uncooked. Tachyzoites are more fragile, although they can survive in body fluids for a day, for as long as a week in goat milk, and up to 50 days in whole blood held at  $4^{\circ}\text{C}$  ( $39^{\circ}\text{F}$ ).

## Transmission

Carnivores and omnivores, including humans, can be infected by eating raw or undercooked tissues containing tissue cysts (or possibly tachyzoites). This is thought to be the more prominent route in cats. All animals, including herbivores, can become infected by ingesting sporulated oocysts from sources such as soil, cat litter, contaminated vegetables/ plants and water. *T. gondii* occurs in semen, and venereal transmission has been demonstrated in some species (e.g., sheep, goats, dogs). Milk-borne infections may be possible, although there is some debate about whether tachyzoites can survive digestion. In a recent study, tachyzoites remained viable in simulated gastric fluid for a time, especially when it was mixed with milk. *T. gondii* may also be present in transplanted organs or transfused blood. Heart transplants are a particularly common source of the parasite.

*T. gondii* is known to cross the placenta in many mammals, including humans. Australian marsupials can transmit this organism to their unfurred young in pouch. Some rodents can infect their progeny repeatedly from tissue cysts alone. In some other species, such as cats and humans, *in utero* transmission usually occurs only if the dam is first infected during that pregnancy. The situation in sheep is not entirely clear, although most studies have suggested that sheep are similar to people and cats.

Arthropods such as flies and cockroaches can act as mechanical vectors for *T. gondii*. It has also been found in several species of ticks, but their role, if any, is still unclear. Some fish and bivalves (mussels, oysters) can concentrate *T. gondii*, and may pass the organism to marine mammals and other species that eat them.

## Disinfection

*T. gondii* oocysts are resistant to most disinfectants, but they can be inactivated by formalin and ammonia. They are destroyed rapidly by temperatures greater than  $66^{\circ}\text{C}$  ( $151^{\circ}\text{F}$ ), and can be killed with boiling water. Oocysts found in water can be eliminated by boiling or filtration (absolute  $1\ \mu\text{m}$  filter), but are resistant to chlorination. Tincture of iodine (2%) can inactivate *T. gondii* with a long exposure time of at least 3 hours.

Tachyzoites and tissue cysts are susceptible to most disinfectants, including 1% sodium hypochlorite and 70% ethanol. Tachyzoites are also inactivated at  $\text{pH} < 4.0$ . Tissue cysts remain viable for approximately 4 minutes at  $60^{\circ}\text{C}$  ( $140^{\circ}\text{F}$ ) or 10 minutes at  $50^{\circ}\text{C}$  ( $122^{\circ}\text{F}$ ). Freezing at  $-12^{\circ}\text{C}$  ( $10^{\circ}\text{F}$ ) for 2-3 days destroys a high percentage of the cysts. Curing meat with salt, sucrose or other solutions may kill tissue cysts, but survival is variable.

## Infections in Animals

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### Incubation Period

The incubation period in animals is probably similar to the 5-23 day incubation period in humans. Experimentally infected kittens developed diarrhea 5-6 days after inoculation. Reactivation can occur years after an animal was infected.

### Clinical Signs

Toxoplasmosis is a significant cause of reproductive losses in sheep and goats. Rare to occasional clinical cases and outbreaks occur in other species. However, most infections, including infections in non-pregnant small ruminants, are subclinical.

#### *Cats and other felids*

The vast majority of infections in domesticated cats are asymptomatic. Most cases of toxoplasmosis seem to occur in young or immunocompromised cats, although older, apparently immunocompetent, animals have also been affected. Common early, nonspecific clinical signs in acute toxoplasmosis include lethargy, persistent fever despite treatment with some antibiotics (i.e., those ineffective against *T. gondii*), and anorexia. Many cats develop respiratory signs, including dyspnea. Severe respiratory involvement is often fatal. Some cats primarily have signs of an acute abdominal condition such as hepatitis (e.g., hepatomegaly, abdominal tenderness, diarrhea, occasional vomiting) or pancreatitis, or develop a nonspecific systemic illness. Self-limited diarrhea has been a component of some cases, and in rare instances, it may be accompanied by a palpable intestinal mass. Neurological signs may be prominent, especially in older cats. The specific signs depend on the site(s) affected in the brain or spinal cord, and may include convulsions, changes in mentation (e.g., restlessness, somnolence, personality changes), hyperesthesia, incoordination, paralysis and depressed reflexes. Kittens with encephalitis may sleep excessively or cry constantly. Other syndromes reported in individual cases include myocarditis, myositis and rare cutaneous lesions ranging from a single large nodule to multiple ulcers and/or alopecic, hyperemic skin nodules. Most, but not all, cats with cutaneous lesions have also had systemic signs. Eosinophilic fibrosing gastritis was reported in an immunocompetent cat that also had diarrhea and CNS signs, and one kidney transplant patient developed a localized mass (necrotizing pyogranulomatous cystitis) in the urinary bladder at the transplant site. Abortions, stillbirths and the birth of premature, weak or deformed kittens may be seen if cats are infected for the first time during pregnancy. Metritis and/or fatal systemic toxoplasmosis have been reported in a few of these dams.

Ocular signs are common in cats with toxoplasmosis, either as the primary sign or in conjunction with systemic illness. They may include generalized retinitis or irregular

reddish, dark or pale retinal foci; in some cases, the retina is partially or completely detached. The retinal vessels may be congested, and hemorrhages and exudates may cloud the vitreous humor. The iris, ciliary body and aqueous humor can also be involved, but the conjunctiva and nictitating membranes are rarely affected. Chronic, low-grade infections may cause glaucoma, corneal opacity and panophthalmitis.

Clinical cases in other felids have resembled the illness in domesticated cats. Congenital toxoplasmosis has caused high newborn mortality in sand cats, and clinical cases have been seen in some adult sand cats. Toxoplasmosis is also reported to be common in Pallas cats. Disseminated, fatal toxoplasmosis has been reported rarely in young felids of other species, including a 4-month-old cheetah (*Acinonyx jubatus*) cub, a juvenile cheetah with concurrent feline infectious peritonitis, and two juvenile lions (*Panthera leo*).

#### *Sheep, goats and cattle*

*T. gondii* usually infects adult sheep and goats without clinical signs; however, infections acquired during pregnancy can cause abortions, stillbirths, and mummification or resorption of the fetus. The consequences are influenced by the timing of the infection. Fetuses infected early in gestation are affected the most severely, and deaths are common. Infections at mid-gestation are more likely to result in stillbirths or the birth of a weak lamb, often accompanied by a small mummified fetus. Congenitally infected lambs may be incoordinated, weak and unable to nurse, and often die. Some animals may have signs of disseminated disease, such as fever and dyspnea. Lambs infected late in gestation are less likely to be affected, and may be asymptomatic. Reproductive losses are generally not thought to recur during subsequent pregnancies; however, some recent studies have questioned whether this is always the case.

Toxoplasmosis seems to be very rare or absent in cattle, but fever, respiratory distress, nasal discharge and conjunctival hyperemia were described in experimentally infected calves.

#### *Other intermediate hosts*

Outbreaks of toxoplasmosis, with abortions, stillbirths, mummified fetuses, neonatal mortality and/or generalized illnesses, are occasionally reported in swine. While some outbreaks are mainly characterized by reproductive losses, illnesses and deaths sometimes occur in older animals, including feeder pigs and pregnant sows. These animals may have nonspecific signs such as fever, depression and lethargy, as well as vomiting, signs of interstitial pneumonia (e.g., dyspnea, coughing), diarrhea, chorioretinitis, neurological signs, lymphadenopathy and signs attributed to the involvement of other organs, such as the liver.

As in other species, toxoplasmosis in dogs can cause reproductive losses, sometimes with stillbirths and

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apparently healthy pups in the same litter. Generalized toxoplasmosis mainly occurs in young dogs (< 1 year); common clinical signs include fever, tonsillitis, dyspnea, diarrhea and vomiting. Liver or respiratory involvement can be rapidly fatal. Myocardial involvement is usually subclinical in young dogs, but arrhythmias and heart failure may be evident in some older animals. Some dogs primarily have neurological signs, which may last for weeks, can be either focal or multifocal, and may include paraparesis or tetraparesis. Myositis, with initial signs of an abnormal gait, muscle wasting and/or stiffness, may also be seen. Ocular signs are usually similar to those in other species, but localized keratitis or conjunctivitis has been reported rarely in dogs with pre-existing ocular conditions that were treated with immunosuppressive drugs. Cutaneous lesions have been described rarely, either with or without concurrent systemic signs. Skin lesions were characterized by alopecic pustular dermatitis; multiple raised, alopecic, and ulcerated nodules; or a single raised dermal nodule and plaque.

Toxoplasmosis has also been reported sporadically in many other species of domesticated or wild mammals and marsupials. In most reported cases, the animals had disseminated disease. Some were found dead, or died after a brief illness with nonspecific signs. Other cases were characterized by various combinations of respiratory, hepatic and ocular signs. Neurological signs were the main presentation in some animals. Gastrointestinal signs, myocarditis or myositis were sometimes seen. Some captive wallabies had severe enteritis, in addition to other organ involvement. Reproductive losses have occasionally been reported.

Only rare clinical cases have been described in birds. Birds with systemic toxoplasmosis often have few or no clinical signs before death, and any signs are frequently nonspecific (e.g., lethargy, anorexia, fluffed feathers). However, neurological signs were prominent in some birds, and others had respiratory signs (e.g., dyspnea) and/ or diarrhea. Affected birds often die very quickly. Chickens seem to be resistant to experimental infection by ingestion, but occasional clinical cases and outbreaks have been described in nature. In one recent outbreak, affected chickens had peripheral neuritis; in another, 3 of 14 chickens died rapidly after developing torticollis, lateral recumbency and an inability to stand.

## Post Mortem Lesions [Click to view images](#)

Toxoplasmosis lesions are related to parasite migration through the tissues and organs, and the accompanying necrosis. In cats, there may be pulmonary edema and small pale foci, often with red centers, in the lungs. Pleural effusion has been reported in a few cats. The liver may be enlarged, with small red or yellow foci or a mottled appearance; hepatitis may be accompanied by icterus. The spleen is sometimes enlarged, with pale or hemorrhagic foci, and may be covered in fibrin. The lymph nodes, particularly in the thorax and abdomen, are variably

enlarged and reddened. Hemorrhages and focal pallor in the myocardium, pericardial effusion and edema have been reported in cases with cardiac involvement. Extensive involvement of the pancreas may appear as an abdominal mass. Gastrointestinal lesions are uncommon in cats, but granulomas, usually associated with areas of chronic enteritis, are occasionally seen. Hemorrhages, necrosis, ulcers, desquamation of the mucosa and eosinophilic fibrosing gastritis have been noted in the stomach. CNS lesions are usually limited to microscopic abnormalities, but visible areas of necrosis are occasionally found. Ocular lesions of the retina, choroid and other structures may also be observed. Mural hemorrhages of the urinary bladder and kidney involvement are uncommon. Esophagitis and skin nodules are rare. Similar lesions, especially involving the lungs, spleen and liver, are reported in other species. Gastrointestinal mucosal hemorrhage, as well as respiratory lesions, were prominent in a giant panda. In *Toxoplasma* abortions of sheep and goats, characteristic 1-3 mm gray-white necrotic foci are found on the cotyledons of the placenta. The intercotyledonary region is usually normal or slightly edematous.

In birds, there may be pulmonary consolidation, pneumonia, splenomegaly and hepatomegaly, and necrotic foci may be found in the liver, lymph nodes, lungs, spleen, heart, kidneys, air sacs and other organs. Additional lesions (e.g., encephalitis, myocarditis, peritonitis, enlarged kidneys) have been reported in some cases.

## Diagnostic Tests

Toxoplasmosis can be difficult to diagnose, due to the high percentage of subclinical infections and the persistence of this parasite in tissues, which complicates tests such as PCR. Serology is often used for diagnosis. In animals, antibodies to *T. gondii* are often detected with agglutination tests (e.g., indirect hemagglutination, latex agglutination, modified agglutination/ MAT), although some additional tests, such as ELISAs and the indirect fluorescent antibody (IFA) test, are available for certain species. A limited number of IgM tests have been validated for veterinary use. The Sabin-Feldman dye test, which is considered a “gold standard” test, is no longer performed in most veterinary (or human) diagnostic laboratories because it requires live tachyzoites. IgM titers or rising IgG titers can help distinguish recent from older infections, and are suggestive of toxoplasmosis if the clinical signs are consistent. Test interpretation can be complicated by the occasional persistence of *Toxoplasma*-specific IgM for months or years, for instance in some healthy cats. In immunosuppressed cats, IgG titers often do not increase when the illness results from the reactivation of tissue cysts. Single high IgG titers can be seen in healthy, asymptomatic cats. Antibodies in cerebrospinal fluid (CSF) or aqueous humor, especially IgM, can be helpful in the diagnosis of ocular or CNS disease.



Direct detection of *T. gondii* can be attempted in sick animals. It can also be used to identify healthy felids that may be shedding oocysts. *Toxoplasma* oocysts, which are ovoid and approximately 10-12 µm in diameter, can be found by fecal flotation. They are morphologically indistinguishable from *Hammondia* and *Besnoitia*, but can be distinguished from these organisms by PCR. Oocysts are not necessarily present in cats with clinical signs. In sick animals of any species, tachyzoites or bradyzoites may be detected in the aqueous humor, CSF, bronchoalveolar lavage fluid, or biopsy/ necropsy samples from other tissues. However, parasites may be sparse and difficult to find antemortem. In aborted fetuses, organisms are most likely to be detected in the placenta and brain. Immunostaining and/or cytocentrifugation may make them easier to find. The tissue forms of *Toxoplasma* appear similar to *Neospora caninum* and *Sarcocystis* species, but can be identified by PCR, tissue immunocytochemistry or ultrastructural studies. PCR can also be used directly on tissues and fluids; however, the persistence of *T. gondii* in healthy animals complicates interpretation. *T. gondii* may also be detected by mouse bioassay. Isolation in cell cultures is now rarely used.

## Treatment

Clinical cases are treated with antibiotics. Only certain drugs, such as clindamycin, trimethoprim-sulfonamide, azithromycin and pyrimethamine, used alone or in various combinations, are effective. Corticosteroids may be administered concurrently in ocular disease, to reduce inflammation. While antibiotics can suppress actively dividing parasites, they cannot destroy tissue cysts and are unlikely to completely eliminate *T. gondii* from the body. Intensive supportive treatment may be necessary in animals with disseminated disease.

## Prevention

### Disease reporting

Veterinarians who encounter or suspect *Toxoplasma gondii* should follow their national and/or local guidelines for disease reporting. However, this organism is ubiquitous, and infections in animals are generally not reportable.

### Prevention

After an abortion, the placenta and abortion products should be removed, and the area cleaned and disinfected. A modified live vaccine is available for sheep in New Zealand and some European countries. Prophylactic treatment with some antibiotics (e.g., monensin) reduced fetal losses in experimentally infected sheep. Felids should be kept away from the environments of pigs, pregnant small ruminants, and other species that are particularly susceptible to illness. However, cats with *Toxoplasma* - specific IgG have already been infected with this organism and are unlikely to shed oocysts in the future.

To prevent cats from becoming infected, they should not be fed raw or undercooked meat, allowed to hunt and eat intermediate hosts (e.g., rodents), or exposed to possible transport hosts, such as cockroaches. Such measures are particularly important for cats on cyclosporine or other intensive immunosuppression, but they also reduce the risk that a healthy cat will shed oocysts and infect humans or other animals. Seropositive feline renal transplant recipients on cyclosporine may be prescribed prophylactic antibiotics. To prevent transplant-associated toxoplasmosis, seronegative donors are usually used for kidney transplants in cats. Some zoos have reported using prophylactic clindamycin for asymptomatic Pallas cat kittens at risk of developing toxoplasmosis.

## Morbidity and Mortality

Asymptomatic *T. gondii* infections are common in animals. The frequency of exposure differs with the animal's environmental niche, but even non-carnivorous species that spend little or no time on the ground, such as bats, can be infected. Management factors (e.g., access to pasture), water and feed sources, and rodent control may affect prevalence. Exposure is uncommon in pigs or chickens housed indoors, but more common in outdoor pigs (seroprevalence of 10-50%) and free-range chickens (up to 100%). Antibodies to *T. gondii* are also reported to be relatively common in some free-living wildlife, including marine mammals, and in some zoos. Cats and wild felids are important in maintaining *T. gondii*; however, this parasite is also common at some isolated terrestrial sites where there are no felids. At these locations, it might be transmitted from animal to animal by predation and vertical transmission. Seroprevalence rates vary widely in domesticated cats, but they are commonly 10- 40%, and can be as high as 80-90% in some areas. Infections are particularly common in strays, and less prevalent in pets. Similar exposure rates have been reported in dogs.

Clinical toxoplasmosis is an important cause of reproductive losses in sheep; congenital transmission is estimated to occur in 1-2% of animals. Outbreaks of congenital disease or systemic illness have been reported occasionally in pigs, with morbidity rates as high as 60% and mortality rates up to 10-42% in some fattening pigs. Toxoplasmosis is a significant cause of deaths among sea otters (*Enhydra lutris nereis*), which are often infected with an unusual genotype (type X). Other animals reported to be affected fairly often include captive wallabies and New World primates, and a number of cases have been reported in squirrels. In one instance, toxoplasmosis was partially responsible for an event in the Netherlands where hundreds of wild Eurasian red squirrels (*Sciurus vulgaris*) may have died. In most other species, toxoplasmosis tends to be a sporadic, uncommon disease, predominantly affecting animals that are immunosuppressed, affected by other diseases (e.g., feline immunodeficiency virus in cats), or young. Cats and dogs have developed disseminated

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toxoplasmosis after receiving strongly immunosuppressive drugs such as cyclosporine or azathioprine; however, the usual therapeutic doses of glucocorticoids have not been associated with recrudescence.

Disseminated toxoplasmosis affecting the lungs, liver and/or CNS is often fatal, even when treated. Between 2008 and 2011, toxoplasmosis was the cause of death in 3% of 193 cats submitted to a university in Finland for necropsy. Mortality rates up to 100% have been seen in week-old kittens, and perinatal mortality in infected lambs, kids and piglets can be as high as 50%.

## Infections in Humans

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### Incubation Period

In humans, the incubation period is estimated to be 5-23 days.

### Clinical Signs

Most immunocompetent, non-pregnant people infected with *T. gondii* do not have any symptoms. Approximately 10-20% develop lymphadenitis or a mild, flu-like syndrome characterized by fever, malaise, myalgia, headache, sore throat and lymphadenopathy. Some patients may also have gastrointestinal signs and/or a rash, and in some cases, the disease may mimic infectious mononucleosis. Most people recover without treatment within weeks to months, although illnesses lasting up to a year have been reported. Some studies have suggested possible associations between exposure to *T. gondii* and various neurological conditions such as schizophrenia or epilepsy; however, definitive evidence is still lacking.

Ocular toxoplasmosis tends to be seen in adolescents and young adults. Some cases are a delayed consequence of congenital infection, but some result from postnatal infections, including ones that were recently acquired. The lesions can be unilateral or bilateral. The typical presentation is chorioretinitis, which is often accompanied by varying degrees of vitreous inflammatory reaction, and sometimes by minimal to significant involvement of the anterior chamber. Chorioretinitis often resolves within a few weeks to a few months in immunocompetent patients, leaving a retinal scar. Active lesions may recur in some untreated cases, especially along the borders of the lesion. Complications such as fibrous bands, retinal detachments, and optic neuritis are possible, especially in severe cases. Macular involvement, which may be more common in congenitally acquired cases, can lead to blindness.

Serious disseminated illness or organ involvement is possible in immunocompetent people, but rare. Some reported syndromes include myositis, myocarditis, hepatitis, pneumonitis and focal or disseminated neurological signs. An unusually large number of serious cases have been reported in immunocompetent adults in French Guiana and Suriname, possibly due to an unusually virulent tropical strain of *T. gondii*. The clinical signs have included high

prolonged fever, and in many cases, weight loss, hepatomegaly, splenomegaly, lymphadenopathy, headache, and a dry cough with chest pain, often progressing to dyspnea. Rash, nausea, vomiting, ocular signs, myocarditis/cardiac abnormalities, myositis and neurological signs were occasionally reported. Approximately one third of the patients developed respiratory distress requiring intensive care. Sepsis-like syndromes were also seen.

Toxoplasmosis can be a severe disease in immunosuppressed people. HIV-infected patients with a low CD4+ T cell count tend to develop CNS signs, especially when the illness is caused by reactivated tissue cysts from an earlier infection. Persistent, severe headaches are often the initial sign. Encephalitis may lead to coma and death. Abscesses in nervous tissue can cause the symptoms of a mass lesion. Ocular lesions such as chorioretinitis are similar to those in immunocompetent patients, but unusual syndromes such as scleritis may be seen concurrently, and ocular lesions are more likely to recur. The lungs, heart and/or other organs may also be involved, and some patients develop disseminated disease. In transplant patients, disseminated illness and pulmonary toxoplasmosis seem to be more common than encephalitis. These syndromes can progress rapidly. Cutaneous involvement, with disseminated erythematous, nodular cutaneous lesions; maculopapular, papulopustular, lichenoid, or vegetative dermatitis; or erythema multiforme-like eruptions may be seen occasionally.

Toxoplasmosis can have serious consequences in pregnant women, including those who are healthy. Congenital toxoplasmosis typically occurs when the mother becomes infected during (or, rarely, just before) pregnancy. The mother usually remains asymptomatic, but the organism can affect the developing fetal brain and/or retina. The incidence and severity of the abnormalities are highly variable, depending on the stage of pregnancy; infections are least likely to cross the placenta during the first trimester but are most severe when they do. Fetal abnormalities range from chorioretinitis, hydrocephalus, convulsions and intracerebral calcifications to only mild effects, such as slightly diminished vision. Strabismus, nystagmus and microphthalmia can also be seen, usually in infants infected early in development. Infants infected late in gestation may have a fever, rash, hepatomegaly, splenomegaly, pneumonia, chorioretinitis or a generalized infection. Many infected infants are asymptomatic at birth; however, some will develop learning and visual disabilities or even severe, life-threatening infections later in life, if left untreated. Abortions and stillbirths can also be seen, particularly if the mother is infected during the first trimester.

### Diagnostic Tests

Tests used to diagnose toxoplasmosis in humans are similar to those employed in animals. Direct detection of the parasite, especially by PCR or mouse bioassay, is useful for diagnosing congenital infections *in utero*, with a sample

of amniotic fluid. Direct detection is also particularly helpful in immunosuppressed patients, who may have delayed serological responses and/or low antibody titers. Various tissue samples, blood, bronchoalveolar lavage material, CSF or other body fluids may be employed. The possibility of a pre-existing, subclinical infection (i.e., tissue cysts) complicates test interpretation in postnatally acquired disease. Computed tomography techniques and magnetic resonance imaging (MRI) are sometimes helpful in cases of cerebral toxoplasmosis, and ultrasound may be used to detect abnormalities in the fetus.

Postnatal cases are often diagnosed with serological tests. Many types of assays, including those used in animals, are available for humans. IgM-specific tests and/or paired IgG titers are performed when it is important to know the time of infection, for instance in a pregnant woman. A negative IgM test suggests that the infection was not recent; however, a positive test can be difficult to interpret. While it generally suggests recent exposure or ongoing active infection, IgM titers occasionally persist for more than 2 years in healthy individuals. Conversely, IgM may be absent, especially during reactivated infections in immunosuppressed people. Because misinterpretation of tests can result in unnecessary treatment, tests in pregnant women are often referred to a reference laboratory. In the U.S., the Centers for Disease Control and Prevention (CDC) recommends that all IgM positives in pregnant women be verified by a *Toxoplasma* reference laboratory such as CDC or the Toxoplasmosis Serology Lab, Palo Alto Medical Foundation, before treatment is started. An IgG avidity test, available at reference laboratories, can estimate the time of infection in the mother. High avidity antibodies take months to develop, and indicate that the infection was not recent.

Newborn infants are usually for tested for IgM and IgG, and sometimes for IgA, using ELISAs or immunosorbent agglutination assays (ISAGAs). The placenta, cord blood and/or infant are often tested at birth, but repeated samples may be needed to exclude false positives or negatives. IgM and IgA contamination from the mother usually disappears shortly after birth. IgG maternal antibodies to *T. gondii* can persist for many months. Test sensitivity can be affected by whether the mother received treatment during the pregnancy, and by when the fetus was infected. Immunoblotting (Western blotting) can compare the patterns of antibodies in mother and infant soon after birth, to help distinguish the source of an infant's antibodies. However, this test is expensive, and is generally used only in specific circumstances. PCR may detect nucleic acids in the CSF, urine or blood of newborns.

Ocular toxoplasmosis is often diagnosed by clinical observation and the response to treatment. PCR of aqueous humor and a comparison of local vs. systemic antibody production may be helpful in some cases. Ocular antibody production can be unpredictable in immunocompromised patients.

## Treatment

People with serious systemic signs and immunocompromised patients are treated with antibiotics. As in animals, antibiotics may not eliminate *T. gondii* completely from the body. Toxoplasmosis may not need to be treated in healthy non-pregnant individuals, because the illness is typically self-limiting and mild. The approach to ocular disease can differ between physicians, and treatment may depend on the location and severity of the lesion. Corticosteroids are usually administered concurrently with antibiotics in eye disease.

Women who become infected during pregnancy may be given spiramycin, which may reduce the risk of fetal infection. Other antibiotics (e.g., pyrimethamine/sulfonamide) can treat an infected fetus *in utero*, or a newborn. Infants may need to be treated for prolonged periods.

## Prevention

The risk of acquiring *T. gondii* can be reduced by proper food preparation. Meats should be cooked to an internal temperature sufficient to kill this organism. Consumers should be aware that it could be present in shellfish and fish, as well as in other meats, and in untreated water sources, such as lakes and streams. Freezing, salting, pickling and smoking do not reliably destroy *T. gondii*, although some techniques (e.g., freezing) destroy a high percentage of cysts. Fruits and vegetables should be peeled or washed well to remove oocysts. Good hygiene, including the use of hot, soapy water on items that touched potentially contaminated food (e.g., meats, raw vegetables), is important. Hands should be washed after contact with raw meat, soil or sand, and before eating or touching the face. Pregnant women and others at risk should wear gloves during soil or sand contact. Care should be taken when handling the live vaccine for sheep.

Highly susceptible individuals should be aware that seronegative cats are likely to shed *T. gondii* on first exposure. Regardless of an animal's antibody status, litter boxes should be cleaned daily to reduce the risk of oocyst sporulation, and rinsed with boiling water. Pregnant women and immunosuppressed individuals should avoid cleaning the litter box; if this is unavoidable, they should use gloves, then wash their hands. Sporulated oocysts are unlikely to be found on fur, and direct contact with cats is not expected to be a risk. Prophylaxis and/or screening for toxoplasmosis may be employed in immunosuppressed patients. Recommendations on prenatal and/or neonatal screening differ between countries. Early treatment of congenitally infected fetuses may reduce some complications.

## Morbidity and Mortality

*T. gondii* infections are common in humans. Seroprevalence rates are highly variable, but they are typically 10-30% in North America, northern Europe and Southeast Asia; 30-50% in Central and Southern Europe;

and higher in Latin America and tropical regions of Africa. Small epidemics are seen occasionally, usually associated with contaminated food or water. Immunity is thought to be lifelong against most strains.

The consequences of infection are most serious in pregnant women and immunocompromised people. Estimates of the rate of congenital toxoplasmosis range from approximately 1 in 3000 births to 1 in 10,000 births. Most women pass *T. gondii* to the fetus only if they are first exposed during the pregnancy. However, a few immunosuppressed women have apparently transmitted reactivated organisms from previous infections. In rare cases, a seropositive, immunocompetent woman has also produced a congenitally infected fetus, possibly either from a recrudescence infection or after she was infected by a different strain of the organism. The risk of transmission from an infected mother is estimated to be 25% during the first trimester, with the majority of these fetuses developing severe clinical signs. The fetal mortality rate at this time is high. Conversely, approximately 50-65% of infants are thought to become infected during the third trimester and 70-90% are asymptomatic at birth, although some will later develop clinical signs if they are not treated.

Immunocompromised persons can become ill either on first exposure to *T. gondii*, or from tissue cysts that already exist in the body (or in a transplanted organ). The illness can be life-threatening. Before highly effective antiviral drugs became available for HIV, toxoplasmosis was common in AIDS patients. In one study, *Toxoplasma* encephalitis occurred in 25% of AIDS patients and was fatal in 84%. Toxoplasmosis is also a significant concern in people taking immunosuppressive drugs to prevent transplant rejection or treat cancer, especially hematological malignancies.

Approximately 80-90% of non-pregnant, immunocompetent people infected with *T. gondii* have no symptoms, and most of the rest have mild illnesses. Deaths are normally rare. However, serious illnesses have been reported recently in French Guiana and Suriname. At least 65 cases were diagnosed in immunocompetent adults between 1998 and 2012, and approximately one third required intensive care. In one report, one of 44 patients died. This syndrome might be linked to a *T. gondii* strain found in a sylvatic cycle, as all patients had links to tropical forests, such as eating undercooked game or drinking untreated water. However, it is possible that other factors, such as an unusually high dose of parasites, might also be involved. A few similar cases have been reported in Europe, in people who might have been infected via sources such as undercooked imported meat.

## Internet Resources

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- Centers for Disease Control and Prevention (CDC), United States  
<http://www.cdc.gov/ncidod/dpd/parasites/toxoplasmosis/default.htm>
- European Centre for Disease Control and Prevention. Toxoplasmosis  
<http://ecdc.europa.eu/en/healthtopics/toxoplasmosis/Pages/index.aspx>
- Public Health Agency of Canada (PHAC). Pathogen Safety Data Sheets  
<http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/index-eng.php>
- The Merck Manual  
<http://www.merckmanuals.com/professional>
- The Merck Veterinary Manual  
<http://www.merckvetmanual.com/mvm/index.html>
- Toxoplasma Serology Laboratory, Palo Alto, CA (U.S.)  
<http://www.pamf.org/serology/>

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