

# TOXOPLASMA GONDII

## Aetiology Epidemiology Diagnosis Prevention and Control Potential Impacts of Disease Agent Beyond Clinical Illness References

### **AETIOLOGY**

#### **Classification of the causative agent**

*Toxoplasma gondii* (*T. gondii*), the causative agent of toxoplasmosis, is an opportunistic, zoonotic, obligate intracellular protozoan parasite that has the capacity to infect all endotherms including birds. While infection does not cause clinical illness in most animal species, it can cause acute life-threatening disease in some. In others (particularly sheep and goats, but occasionally pigs), it multiplies in the placenta and foetus to cause reproductive disease during pregnancy.

*T. gondii* has a sexual cycle in Felidae and a two-stage asexual cycle in all other warm blooded animals. In the acute phase of infection, tachyzoites multiply in cells to cause varying degrees of tissue destruction and, in fatal cases, tachyzoites may be demonstrated in ascitic fluid or in lung impression smears. With the onset of an immune response, tachyzoites are transformed into bradyzoites that multiply slowly in cells to produce tissue cysts.

It should also be noted that many wild animals have presented with serum antibodies against both Toxoplasmosis and canine distemper. This toxoplasmosis-distemper complex arises from a distemper viral infection that ultimately lowers resistance, which promotes the development of latent toxoplasmosis into an aggressive disease process.

#### **Resistance to physical and chemical action**

Temperature:	Oocysts are inactivated by temperatures >66° C; tissue cysts are inactivated at 0°C.
pH:	Tachyzoites are inactivated at pH <4.0
Chemicals/Disinfectants:	Oocysts are susceptible to iodine and formalin but resistant to most disinfectants; tachyzoites and tissue cysts are susceptible to most disinfectants; 70% ethanol, 1% sodium hypochlorite; cysts do not survive in >6% NaCl solution.
Survival:	Oocysts are very stable and can survive up to 1 year in water or moist soil; tachyzoites can survive in body fluids up to 1 day, in whole blood for up to 50 days at 4°C; tissue cysts can survive for weeks at room temperature in body fluids.

### **EPIDEMIOLOGY**

#### **Definitive Hosts**

- Felidae (wild & domestic)

#### **Intermediate Hosts**

- Has been demonstrated in virtually every species of mammal and many species of birds; commonly:
  - Sheep (*Ovis aries*)

- Calves (*Bos taurus*)
- Coyotes (*Canis latrans*)
- Red foxes (*Vulpes vulpes*)
- White-tailed deer (*Odocoileus virginianus*)
- Canada geese (*Branta canadensis*)
- European Rabbits (*Oryctolagus cuniculus*)
- Pigs (*Sus*)
- Wild Boar (*Sus scrofa*)
- Sea Otters (*Enhydra lutris*)

### **Transmission**

- Occurs predominantly via ingestion of tissue cysts, ingestion of oocysts from contaminated food or water, or congenital transmission
  - The sexual life-cycle of the parasite takes place exclusively in epithelial cells of the feline intestine and can result in the excretion of large numbers of oocysts in the faeces.
  - Once an intermediate host consumes the oocyst or tachyzoite, it undergoes replication and infects the intestine. Adult immunocompetent animals elicit a strong immune response, controlling the infection and encapsulating the pathogen into cysts (bradyzoites).
  - Oocysts may remain viable in the environment for many months.

### **Sources**

- Ingestion of animal tissues that contain cysts, contamination of the environment or food with oocysts (fecal-oral route), etc.

### **Occurrence**

Toxoplasmosis has a worldwide distribution within endothermic animals. Areas of high prevalence exist in Latin America, parts of Eastern/Central Europe, the Middle East, and parts of Southeast Asia and Africa. Infection is common in food animals such as pigs, sheep, and rabbits. Where feral cat populations are most out of control there is likely to be a higher risk of exposure.

**For more recent, detailed information on the occurrence of this disease worldwide, see the OIE World Animal Health Information System - Wild (WAHIS-Wild) Interface [[http://www.oie.int/wahis\\_2/public/wahidwild.php/Index](http://www.oie.int/wahis_2/public/wahidwild.php/Index)].**

### **DIAGNOSIS**

The incubation period of *T. gondii* is 5-20 days for felids. The tachyzoite stage is responsible for tissue damage, and thus clinical signs depend on the number of tachyzoites released, the ability of the host immune system to limit their spread, and the organs they damage.

In some, it causes acute life-threatening disease and in others, particularly sheep and goats, it may manifest itself as a disease of pregnancy by multiplying in the placenta and foetus. Pregnancy complications may include abortion, weak lambs/kids, and foetal mummification.

### **Clinical diagnosis**

Infection in pigs is usually mild and unnoticed, but may cause severe foetal losses in pregnant sows.

## **Lesions**

- 1-3 mm in diameter, necrotic white foci on placental cotyledons
- Focal microgliosis in brain
  - Focal leukomalacia in cerebral white matter

## **Differential diagnoses**

- Encephalopathies
- *Erythroblastosis fetalis*
- Rubella

## **Laboratory diagnosis**

### **Samples**

*For isolation of agent*

- Tissue sections of brain and placenta

*Serological tests*

- Serum

### **Procedures**

*Identification of the agent*

- Immunohistochemistry is the gold standard for diagnosing *T. gondii*.
- Polymerase chain reaction (PCR) may be used to identify parasite DNA in tissues.
- Histopathology of lymphoid tissues or cysts in tissues
- Immunoblotting (Western blotting)
- Enzyme-linked immunosorbent assays (ELISA)

*Serological tests*

- Indirect fluorescent antibody (IFA) test is more reliable and can be used to differentiate IgM and IgG antibodies.
  - A rising antibody titre or detection of DNA can be used for diagnosis via modified agglutination test (MAT)
- Direct agglutination test and the latex agglutination test are both relatively rapid and neither require complex laboratory facilities.
- Enzyme-linked immunosorbent assays require more sophisticated laboratory equipment, but can process large numbers of samples
- =

## **PREVENTION AND CONTROL**

### **Sanitary prophylaxis**

- In areas where risk factors for exposure are high, limiting contact with feral cats or wild felid populations is important.
  - Clean and disinfect where wild felids have defecated

- Pregnant women should be especially cautious due to the risk of transplacental transmission and birth defects.

### **Medical prophylaxis**

- A vaccine composed of live *T. gondii* tachyzoites is available commercially for use in sheep in the UK, Ireland, France, Portugal, Spain and New Zealand.
  - The vaccine is supplied as a concentrated suspension of tachyzoites with an approved diluent and delivery system.
  - The vaccine must be maintained and handled strictly according to the manufacturers' instructions as it has a very short shelf life.
- Treatment with sulfadiazine and pyrimethamine has been widely used for toxoplasmosis.

## **POTENTIAL IMPACTS OF DISEASE AGENT BEYOND CLINICAL ILLNESS**

### **Risks to public health**

- *Toxoplasma gondii* readily infects human beings, and although infection is relatively common (approximately 30% of the population depending on age and environment), clinical illness is relatively uncommon.
- Those particularly at risk of developing clinical illness include:
  - Pregnant women, as the parasite can pose a serious threat to the unborn child if the mother becomes infected for the first time while pregnant.
  - Individuals who are immunosuppressed, such as tissue transplant patients, AIDS patients, patients with certain types of cancer and those undergoing certain forms of cancer therapy. These individuals are at risk of developing acute lethal infection if left untreated.
- The very young and very old may also be more susceptible.
- The most likely sources of human infection are ingestion of raw or lightly cooked meat containing live *T. gondii* tissue cysts, ingestion of raw or lightly cooked vegetables contaminated with oocysts, or exposure to oocysts derived from cat faeces, such as may be encountered in gardens and children's sand pits.
- From a public health viewpoint, it is necessary to distinguish *T. gondii* oocysts from oocysts of a related coccidium, *Hammondia hammondi*, which is also present in cat faeces. *Hammondia hammondi* is non-pathogenic.
  - Bioassays are currently the only definitive way to detect viable oocysts of these parasites but expensive and only a few laboratories in the world have the facilities to do them.
  - Although DNA detection is considered highly specific, cross reactivity has been observed between *T. gondii* and *H. hammondi*.

### **Risks to agriculture**

- Agriculture can be drastically impacted via food animal infection with *T. gondii*.
  - Livestock become infected by the same routes, yielding meat products that contain tissue cysts which can then infect consumers.
  - Biosecurity measures such as confinement housing play an important role in reducing the levels of infection in animals destined for human consumption.

## REFERENCES AND OTHER INFORMATION

- Aubert, D., Ajzenberg, D., Richomme, C., Gilot-Fromont, E., Terrier, M.E., de Gevigney, C., Game, Y., Maillard, D., Gibert, P., Dardé, M.L., Villena, I. (2010). Molecular and biological characteristics of *Toxoplasma gondii* isolates from wildlife in France. *Vet. Parasitol.* 171:346–349.
- Buxton, D. (1993). Toxoplasmosis: the first commercial vaccine. *Parasitol. Today*, 9, 335–337.
- Canfield, P.J., Hartley, W.J., and Dubey, J.P. (1990). Lesions of toxoplasmosis in Australian marsupials. *J. Comp. Pathol.*, 103, 159–167.
- Cunningham, A.A., Buxton, D., and Thomson, K.M. (1992). An epidemic of toxoplasmosis in a captive colony of squirrel monkeys (*Saimiri sciureus*). *J. Comp. Pathol.*, 107, 207–219.
- Diters, R. W., & Nielsen, S. W. (1978). Toxoplasmosis, Distemper, And Herpesvirus Infection in skunk (*Mephitis mephitis*). *Journal of Wildlife Diseases*, 14(1), 132–136. doi: 10.7589/0090-3558-14.1.132
- Dubey, J.P., Desmonts, G. (1987). Serological responses of equids fed *Toxoplasma gondii* oocysts. *Equine Vet. J.* 19:337–339.
- Hill, D.E., Chirukandoth, S., et al. (2005). Biology and epidemiology of *Toxoplasma gondii* in man and animals. *Anim Health Res Rev.* 6(1):41–61.
- Pappas, G., Roussos, N., et al. (2009). Toxoplasmosis snapshots: global status of *Toxoplasma gondii* seroprevalence and implications for pregnancy and congenital toxoplasmosis. *Int J Parasit.* 39(12):1385–1394.
- “Toxoplasmosis.” *T. gondii*. Cornell University College of Veterinary Medicine Wildlife Health Lab. (2017). Accessed in 2019: [cwhl.vet.cornell.edu/disease/toxoplasmosis#collapse7](http://cwhl.vet.cornell.edu/disease/toxoplasmosis#collapse7)
- Walzer, K.A., Wier, G.M., Dam, R.A., Srinivasan, A.R., Borges, A.L., English, E.D., Herrmann, D.C., Schares, G., Dubey, J.P., and Boyle, J.P. (2014). *Hammondia hammondi* harbors functional orthologs of the host-modulating effectors GRA15 and ROP16 but is distinguished from *Toxoplasma gondii* by a unique transcriptional profile. *Eukaryot. Cell*, 13, 1507–1518.
- World Organisation for Animal Health (2017). Terrestrial Manual. OIE, Paris.

\*

\* \*

The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department ([scientific.dept@oie.int](mailto:scientific.dept@oie.int)). Last updated 2019. Written by Marie Bucko and Samantha Gieger with assistance from the USGS National Wildlife Health Center.