



TOXOPLASMOSIS (GENERAL)

ANIMAL GROUP AFFECTED	TRANSMISSION	CLINICAL SIGNS	FATAL DISEASE ?	TREATMENT	PREVENTION & CONTROL
All mammalian and avian species are susceptible	Congenital, carnivorous, faecal-oral. Felidae (domestic and wild) are the only definitive hosts	Depending on the species and organs affected. Lymphadenopathy, abortion, acute death	Yes – in New World monkeys, marsupials, meerkats, gazelles	Clindamycin. Pyrimethamine + sulfadiazine + Folic acid. Primary prophylaxis with trimethoprim/sulfamethoxazole	<p><i>In houses</i> Meat of any animal should be cooked to 70°C before consumption. Hands should be washed with soap after handling meat. All cutting materials coming in contact with uncooked meat should be washed with soap + water</p> <p><i>in zoos</i> reduce exposure to feline faeces and uncooked meat. Preventing feral cats from entering exhibits. Frozen meat is preferred to fresh meat</p>

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<p>Susceptible animal groups In most species infection may be common, but clinical disease is rare. Fatal acute toxoplasmosis has been reported in Australian marsupials (especially macropods but also in possums, dasyurids, bandicoots, wombats, and koalas), New World primates (especially squirrel monkeys and callitrichids), Prosimians (particularly lemurs), viverrids (Slender-tailed meerkat), and in some nondomestic ruminants (including different gazelle species, deers and gerenuk).</p>	
<p>Causative organism <i>Toxoplasma gondii</i> is an obligate intracellular parasite being felids the only definitive hosts. Felidae excrete nonsporulated (therefore noninfectious) <i>T. gondii</i> oocysts in their faeces. Sporulation (development of infectious sporozoites inside the oocysts) may take 1 to 5 days after defecation. After ingestion of oocysts by a warm-blooded animal, oocysts rupture in the intestine, releasing 8 sporozoites that multiply intracellularly in the intestines and in associated lymph nodes, and tachyzoites or trophozoites (rapidly multiplying forms) are formed. Tachyzoites then spread to the rest of the body via blood and lymph and eventually encyst in the brain, skeletal and cardiac muscles, and liver. Encysted <i>T. gondii</i> are called bradyzoites or cystozoites (slowly multiplying forms).</p>	
<p>Zoonotic potential Results of several studies indicate that the cat is involved in the epidemiologic aspects of toxoplasmosis. Moreover epidemiologic data suggest that the ingestion of tissue cysts in undercooked or uncooked meat is an</p>	



important source of infection of people in the United States. Pre-natal infection is serious coursing with abortion, encephalitis, blindness or mental retardation of the foetus. Immuno-compromised people can develop a fatal reactivation of a latent *T. gondii* infection.

Distribution

World-wide-

Transmission

This parasite is transmitted via 3 primary ways: 1) Congenital: Transplacental infection can occur when a previously non-infected host becomes infected during pregnancy. *T gondii* multiplies in the placenta and then spread to foetal tissues. Although transplacental infection can occur at any stage of gestation, the foetus is affected more severely if the dam becomes infected during the first half of gestation. 2) Faecal-oral: Ingestion of feline faecal matter contaminated with sporulated oocysts. 3) Carnivorism: After ingestion of infected tissues, the cysts wall is dissolved and bradyzoites are released which infect the host. After entry into host cells, bradyzoites transform into tachyzoites and those may undergo repeated divisions, ultimately encysting in tissues. The cycle is completed when tissue cysts are ingested by the cat.

Incubation period

Clinical symptoms

Clinical manifestations of toxoplasmosis usually include lymphadenopathy, headache, and muscle aches; however any organ may be affected so clinical findings may vary. Signs in humans as well as in animals may be localised as in ocular involvement (retinitis, uveitis), CNS involvement or pneumonia, or they may be generalised. Respiratory insufficiency, neurologic signs and incoordination. However, the most common clinical sign of toxoplasmosis in sheep and goats is abortion. Usually acute death occurs when disseminated toxoplasmosis affects highly susceptible species.

Post mortem findings

Where present, common necropsy findings include: pulmonary congestion, oedema and consolidation, adrenal enlargement and reddening, haemorrhage and ulceration of stomach and small intestine, and lymphadenomegaly and splenomegaly. Protozoan organisms consistent with *T. gondii* tachyzoites and specifically immunostained with a *T. gondii* polyclonal antibody can be seen in many lesions.

Diagnosis

Difficult based on clinical signs alone. Toxoplasmosis should be considered if major organ systems are affected (lung, liver, CNS), and domestic or wild cats are present in the area. Definitive diagnosis requires the demonstration of a rising serum antibody titer or identification of the organism in biopsy or necropsy samples. The detection of antibodies in serum suggests previous or current infection by *T. gondii*. Serologic tests available include: methylene blue dye binding (MBD), indirect immunofluorescent antibody (IFA), indirect haemagglutination, enzyme linked immunosorbent assay (ELISA), direct and modified agglutination (DAT, MAT) and latex agglutination (LA).

Material required for laboratory analysis

Serum for antibodies determination.

Tissues for light and electron microscopy, immunohistochemical staining, bioassay, tissue antigen ELISA or PCR analysis.

Relevant diagnostic laboratories

Treatment

No treatment is effective in eliminating infection with *T. gondii*. Clindamicin is efficacious in small domestic animals. Another treatment proposed is based on the combination of sulfonamides (30-60 mg/kg PO q12 hours) with pyrimethamine (0.25-0.5 mg/kg PO q12 hours); Folic acid must be added during this therapy. Other drugs less effective but with fewer side effects are chloramphenicol, tetracycline and doxyciline.

Prevention and control in zoos

Basic strategies are:

- Reduce exposure of zoo animals to feline faeces and uncooked meat;
- Preventing feral cats from entering exhibits;
- Meat that is fed to zoo felids should be frozen for at least 3 days at -12°C to kill *Toxoplasma* tissue cysts;
- Dissemination may be controlled through strict sanitation and by controlling rodent and cat populations in the feed storage areas;
- Keepers working with highly susceptible species should not enter felids exhibits;
- Cat proof hay storage areas should be made;
- Feline faeces should be removed daily to prevent sporulation of oocysts.
- In outbreak situation, although chemoprophylaxis is not practical at the present, antibiotic therapy can be advisable.



Suggested disinfectant for housing facilities The parasite is destroyed by low (freezing at -12°C for 3 days) or elevated temperatures (cooking). No disinfectant is applicable.
Notification List C of OIE.
Guarantees required under EU Legislation
Guarantees required by EAZA Zoos
Measures required under the Animal Disease Surveillance Plan
Measures required for introducing animals from non-approved sources
Measures to be taken in case of disease outbreak or positive laboratory findings
Conditions for restoring disease-free status after an outbreak
Contacts for further information
References <ol style="list-style-type: none">1. Acha, P. N. 1997. Zoonosis y enfermedades transmisibles comunes al hombre y a los animales. Organización Panamericana de la salud, Washington, USA. Pp.646-657.2. Borst, G. H. A., and F. van Knapen. 1984. Acute acquired toxoplasmosis in primates in a zoo. J. Zoo Anim. Med. 15: 60-62.3. Briscoe, N., J. G. Humphreys, and J. P. Dubey. 1993. Prevalence of <i>Toxoplasma gondii</i> infections in Pennsylvania black bears, <i>Ursus americanus</i>. J. Wildl. Dis. 29: 599-601.4. Bulmer, W. S. 1971. Toxoplasmosis in captive Saiga antelope. J. Wildl. Dis. 7: 310-316.5. Canfield, P. J., W. J. Hartley, and J. P. Dubey. 1990. Lesions of toxoplasmosis in Australian macropods. J. Comp. Pathol. 103: 159-167.6. Dresden, D. W. 1990. <i>Toxoplasma gondii</i> infections in wildlife. J. Am. Vet. Med. Assoc. 196: 274-276.7. Dubey, J. P. 1986. Toxoplasmosis. J. Am. Vet. Med. Assoc. 189: 166-170.8. Dubey, J. P. 1986. A review of toxoplasmosis in cattle. Vet. Parasitol. 22: 177-202.9. Dubey, J. P. 1987. Toxoplasmosis in goats. Agri-Practice 8: 43-52.10. Fernandez-Moran, J., C. Enseñat, B. Moreno-Burgos, G. Aduriz, and R. Juste. An outbreak of toxoplasmosis in dorcas gazelles (<i>Gazella dorcas</i>) in Spain. Infect. Dis. Rev. 1(4): 270-273.11. Frenkel, J. K. 1990. Transmission of toxoplasmosis and the role of immunity in limiting transmission and illness. J. Am. Vet. Med. Assoc. 196: 233-240.12. Garell, D. M. Toxoplasmosis in zoo animals. In M. Fowler, and E. Miller (eds.): Zoo and Wild Animal medicine Current Therapy 4. W B Saunders, Philadelphia, USA. Pp. 131-135.13. Gilbert, D., R. Moellering, M. Sande. 2000. The Standford Guide to Antimicrobial Therapy, 30th ed, Antimicrobial Therapy, Inc. VT, USA.14. Gorman, T. R., V. Riveros, H. A. Alcaino, D. R. Salas, and E. R. Thiermann. 1986. Helminthiasis and toxoplasmosis among exotic mammals at the Santiago National Zoo. J. Am. Vet. Med. Assoc. 189: 1068-107015. Juan-Sallès, C., Prats, N., López, S., Domingo, M., Marco, J. A., Fernández-Morán, J., 1997. Epizootic Disseminated Toxoplasmosis in captive Slender-Tailed Meerkats (<i>Suricata suricatta</i>). Vet. Pathol. 34:1-716. Juan Salles, C., N. Prats, A. Marco, J. Ramos-Vara, D. Borrás, and J. Fernandez-Moran. 1998. Fatal acute toxoplasmosis in three golden lion tamarins (<i>Leontopithecus rosalia</i>). J. Zoo Wildl. Med. 29: 55-60.17. Oertley, K. D., K. W. Walls. 1980. Prevalence of antibodies to <i>Toxoplasma gondii</i> among bobcats of West Virginia and Georgia. J. Am. Vet. Med. Assoc. 177: 852-853.18. Patton, S. 1993. Toxoplasmosis in the zoological park. Proc. Am. Assoc. Zoo Vet. Saint Louis, USA. Pp. 189-192.19. Stover, J., E. R. Jacobson, J. Lukas, M. R. Lappin, and C. D. Buergelt. 1990. <i>Toxoplasma gondii</i> in a collection of nondomestic ruminants. J. Zoo Wildl. Med. 21: 295.