### Animal Group(s) Affected

All vertebrates (primarily birds and mammals)

### Transmission

- Ingestion of oocysts from felid feces
- Ingestion of tissue cysts
- Transplacental
- Transmammary

### Clinical Signs

Variable, depending on species and organs affected. Can range from asymptomatic to sudden death.

### Severity

Variable depending on species. Causes severe disease in Australian marsupials, New World primates, and lemurs. Usually asymptomatic in most felids.

### Treatment

- Atovaquone, clindamycin, sulfonamide

### Prevention and Control

Prevent exposure to felid feces. Control intermediate hosts in environment.

### Zoonotic Potential

Yes

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**Fact Sheet compiled by:** Joseph A. Smith  
**Sheet completed on:** 30 June 2011; updated 15 July 2013  
**Fact Sheet Reviewed by:** Charles Faulkner; Jitender P. Dubey

**Susceptible animal groups:** Felids are the only definitive host for *Toxoplasma gondii*. Australian marsupials, lemurs, New World primates, brown hares, southern sea otters, and pronghorn antelope are reported to be highly susceptible. Cattle, rats, horses, Old World monkeys, and turkeys are reported as relatively resistant to clinical disease. Pallas’ cats are an exception to most felids in that a positive queen’s immune response does not prevent congenital transmission.

**Causative organism:** Toxoplasmosis is caused by the obligate intracellular coccidian *Toxoplasma gondii*. Felids are the definitive host and are the only taxa known to transmit infective oocysts in feces. Other species are most frequently infected by ingestion of oocysts from felid feces, which may survive for months to years in the environment. Once ingested by an intermediate host, the organism forms tachyzoites that rapidly reproduce in host tissues. Tachyzoites are the cause of most clinical signs. Tachyzoites can then transform into thin-walled tissue cysts containing bradyzoites. The life cycle is completed when felids ingest the tissue cysts from prey species. Other non-felid carnivorous species may also become infected from ingestion of tissue cysts, but are unable to complete the life cycle and do not produce infective oocysts in feces; however, they – as prey species – can become carrier hosts which are infective, and usually termed intermediate hosts in the literature, although they are not required to complete the life cycle.

**Zoonotic potential:** In the US, it is estimated that 22.5% of the population has been infected with toxoplasmosis with this number approaching 95% in some other parts of the world. Transmission can occur from ingestion of oocysts passed in cat feces (e.g. cleaning pet litter boxes, gardening/contact with contaminated soil, contaminated produce), ingestion of undercooked meat, transplacentally, or rarely through blood transfusions and organ transplants. Most infections are asymptomatic or cause mild self-limiting flu-like symptoms. Infections acquired during pregnancy can cause abortion, congenital defects, or more severe disease in the child. Clinical signs in the child, including ocular disease, seizures, and mental disability, may not be present until later in life. Infections in immunocompromised persons may be severe.
**Immunosuppression** may also cause a recrudescence of an infection that was acquired earlier in life.

<table>
<thead>
<tr>
<th><strong>Distribution:</strong></th>
<th>Worldwide anywhere felids are present or have been introduced. Runoff water infected with oocysts can introduce the organism to ocean environments.</th>
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<tbody>
<tr>
<td><strong>Incubation period:</strong></td>
<td>Infections acquired from ingestion of tissue cysts have a 3-10 day prepatent period in felids. Infections acquired from ingestion of oocysts have a 19-48 day prepatent period in felids. Oocysts passed in feces become infective after 1-5 days in the environment. Felids can shed millions of oocysts over 1-3 weeks. Tissue cysts can remain present for years.</td>
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<td><strong>Clinical signs:</strong></td>
<td>Infections in felids are usually subclinical, although a transient mild diarrhea may occur. If species sensitive to the disease, animals are often found dead with no clinical signs observed prior to death. If present, clinical signs may vary depending on the organs affected. Reported clinical signs include respiratory signs (dyspnea, tachypnea, coughing), gastrointestinal signs (diarrhea), general signs (depression, anorexia, behavioral changes), lymphadenopathy, muscle weakness, neurologic signs (blindness, ataxia, dysphagia), ocular disease (keratitis, uveitis, chorioretinitis, endophthalmitis, cataracts), and abortion. Serum biochemical abnormalities may include elevated muscle and liver enzymes.</td>
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<tr>
<td><strong>Post mortem, gross, or histologic findings:</strong></td>
<td>Gross—Affected animals may have no gross lesions. If present, gross lesions may include congestion, hemorrhage, organomegaly, or necrosis of any affected organs. Histologic—Multifocal, multi-organ necrosis is often associated with acute toxoplasmosis. Focal necrosis of affected organs may be associated with free and intracellular tachyzoites (2µm x 6µm crescent-shaped structures with pointed anterior and rounded posterior). Brain (encephalitis with microglial nodules and perivascular cuffing), myocardium (myocarditis), and lung (interstitial pneumonia) are frequently affected. Tissue cysts measuring 5-100µm in diameter can be found in any tissue, but frequently occur in the brain, eye, and muscle. Cysts have thin (&lt;0.5µm) elastic walls and contain up to hundreds of 7µm x 1.5µm crescent-shaped bradyzoites.</td>
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<td><strong>Diagnosis:</strong></td>
<td>Definitive diagnosis can be achieved by observation of tachyzoites or bradyzoites in affected tissues with cytology or histopathology. Multiple serologic testing modalities capable of detecting IgG and IgM antibodies are available including ELISA, Western blot, direct agglutination test (DAT), modified agglutination test (MAT), latex agglutination test (LAT), and indirect hemagglutination test (IHAT). A single positive serologic test indicates exposure to the organism. In young animals, transfer of maternal antibodies can produce positive serology results. Active infections are generally characterized by a high positive IgM titer with subsequent seroconversion and development of an IgG antibody titer &gt; 32 or by a 4-fold increase in paired IgG titers taken 2-4 weeks apart. PCR and immunohistochemical staining can also be used to detect <em>Toxoplasma</em> antigen in tissues.</td>
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<td><strong>Material required for laboratory analysis:</strong></td>
<td>Formalin-fixed affected tissues can be used for histopathology and immunohistochemical staining. Fresh and frozen tissue can be used for PCR. DNA denatures in formalin so PCR becomes less accurate in tissues that have been fixated in formalin for long periods. Serum is needed for the serologic tests. Aqueous humour, cerebrospinal fluid, and plasma can also be assayed for IgG antibodies by the MAT.</td>
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<td><strong>Relevant diagnostic laboratories:</strong></td>
<td>Clinical Parasitology Diagnostic Service Laboratory (immunoassay by MAT) Room A233 University of Tennessee College of Veterinary Medicine 2407 River Drive Knoxville, TN 37996-4500 865-974-5645</td>
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</tbody>
</table>
TOXOPLASMOSIS

American Association of Zoo Veterinarians
Infectious Disease Committee Manual 2013

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**Treatment:** Atovaquone has shown the most promise in treating toxoplasmosis in multiple species. Administration with a high fat meal (e.g. canola oil) has been suggested to increase absorption of the drug. However, the efficacy of this practice is unknown for foregut fermenters. Other drugs including sulfa drugs, clindamycin, spiramycin, ponazuril, and pyrimethamine have also been used alone or in combination with variable success. General supportive care is also usually needed for active cases of toxoplasmosis.

**Prevention and control:** Controlling exposure to cat feces is an important part of toxoplasmosis prevention. Feral cats are a common source of infective oocysts in the environment. Contamination of food and bedding materials with cat feces may be a source of infection in situations where felids are not known to be present near the affected animal. A live attenuated vaccine has been developed for livestock, but efficacy is variable in other species. Meat containing tissue cysts can be rendered non-infective by cooking to 67 °C or freezing to -12 °C for at least 24 hours. Control of intermediate hosts (e.g. rodents) in the environment can help prevent transmission to carnivores. Prophylactic treatment of queens or kittens has been recommended to reduce morbidity and mortality in Pallas' cats.

**Suggested disinfectant for housing facilities:** *Toxoplasma* is resistant to most disinfectants but is usually susceptible to boiling water, formalin, and iodine.

**Notification:** Not a reportable disease.

**Measures required under the Animal Disease Surveillance Plan:** None.

**Measures required for introducing animals to infected animal:** Animals introduced to the environment of an infected felid are at risk of contracting toxoplasmosis. Non-felid species that are infected with toxoplasmosis do not pose a risk to other individuals in the environment unless their tissue is ingested. Vertical transmission between females and their offspring is possible in all mammalian species when the infection occurs during gestation.

**Conditions for restoring disease-free status after an outbreak:** Once an individual becomes infected with toxoplasmosis, it can remain infected for life. *Toxoplasma* organisms can remain dormant in tissue cysts where they are protected from the host's immune response. Episodes of immunosuppression can result in a recrudescence of clinical disease. Serologic testing and removal of positive individuals is a possible way of reaching disease-free status provided that there is not continued exposure to infective oocysts in the environment.

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## TOXOPLASMOSIS

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### References: