



TOXOPLASMOSIS FACT SHEET

Introductory statement

Toxoplasmosis is caused by the protozoan parasite *Toxoplasma gondii*. Cats, including non-domestic species, are the definitive host. Australian marsupials can act as an intermediate host. Although toxoplasmosis is a significant cause of morbidity and mortality in wild marsupials throughout Australia, its impact at a population level is not known.

Aetiology

Toxoplasmosis is caused by the protozoan parasite *Toxoplasma gondii*, which belongs to the Family Sarcocystidae.

Natural hosts

Cats, including non-domestic species, are the definitive host. All other mammals and birds can act as an intermediate host.

World distribution

The disease occurs world wide, wherever cats are found.

Occurrences in Australia

The disease occurs Australia wide.

Epidemiology

Cats become infected by consuming oocysts or infected intermediate hosts. Parasite development occurs in the intestine and faecal shedding begins one to three weeks after infection. Shedding continues for one to two weeks after which the cat becomes a carrier. Disease or immunosuppression may result in a reoccurrence of oocyst shedding. While a large number of cats are seropositive for *T. gondii* it is estimated that only 1% are shedding oocysts at any given time (Soulsby 1982).

While infection appears to be common in mammals and birds relatively few individuals develop overt disease, with the exception of marsupials that seem to be much more sensitive. Infection is contracted through exposure to cat faeces, food or water that has been contaminated by cat faeces, or by eating the flesh of another animal that contains the parasite encysted in its muscles. Oocysts can also be mechanically transmitted by infected earthworms, flies and cockroaches (Bettioli et al 2000b).

Clinical signs

Clinical signs are variable and include sudden death, blindness, keratitis, chorioretinitis, neurological signs, depression, anorexia, fever and dyspnoea.

Diagnosis

Toxoplasmosis should be high on the list of differential diagnoses for any marsupial that is displaying neurological signs, found dead, or showing vague symptoms with no obvious cause. Definitive diagnosis in the live animal depends on serological testing. In the dead animal diagnosis relies on the presence of tissue cysts associated with an inflammatory response.

Pathology

Often there are no gross findings at necropsy. If present, lesions include pulmonary congestion, myocardial haemorrhage, splenomegaly and gastric ulceration. Histologically multiple foci of necrosis with lymphoid infiltrates may be found in the central nervous system, lung, myocardium, skeletal muscle, lymphoid tissue, adrenal, pancreas and liver. Periodic Acid Schiff (PAS) positive tissue cysts are most commonly found in the brain, muscle and adrenal (Canfield et al 1990).

Differential diagnoses

Because of the extremely variable presentation toxoplasmosis should be on the differential diagnosis list for almost any clinically abnormal marsupial.

Laboratory diagnostic specimens

Submit serum from the live animal for serology.

Laboratory procedures

Diagnosis can be made in the live animal by submitting serum for testing with the direct agglutination test (DAT), modified agglutination test (MAT), latex agglutination test or indirect fluorescent antibody test. MAT measures only IgG and records minimal levels early in the disease. DAT measures IgG and IgM and the detection of antibodies with this test, along with a low or negative MAT result, is considered to be indicative of acute toxoplasmosis (Vogelnest and Portas 2008, Bettiol et al 2000a). Repeat testing of an infected animal shows both DAT and MAT titres rising.

Treatment

Treatment is usually unsuccessful but can be attempted using atovaquone 100 mg/kg SID for 30 days, clindamycin 10 mg/kg BID for four weeks, or trimethoprim-sulphonamide 15 mg/kg SID.

Prevention and control

Control of the disease focuses on preventing access to cat faeces. Oocysts are very resistant and can survive up to 18 months in the environment. Meat should be cooked to at least 66°C or frozen for at least two weeks prior to feeding. Equipment can be sterilised by autoclaving or heating to 70°C for at least ten minutes. *T. gondii* is killed by soap and water.

Surveillance and management

There is no targeted surveillance program for *T. gondii*. There is no AUSVETPLAN or Import Risk Analysis for *T. gondii*. Limited serological testing of wild Victorian brush-tailed rock wallabies (*Petrogale penicillata*) in Gippsland has not identified any seropositive individuals. A study in Tasmania found 6.7% seropositive eastern barred bandicoots (*Perameles gunnii*) out of a sample of 150 animals (Obendorf et al 1996).

Statistics

Limited information is available in the National Wildlife Health Surveillance Database (eWHIS – See <http://www.wildlifehealth.org.au/AWHN/home.aspx>). Cases reported in eWHIS include Tasmanian pademelons (*Thylogale billardierii*), common wombats (*Vombatus ursinus*) and Bennett's wallabies (*Macropus rufogriseus*) from Tasmania, western grey kangaroos (*Macropus fuliginosus*) and southern brown bandicoots (*Isodon obesulus*) from Western Australia, tawny frogmouths (*Podargus strigoides*) from New South Wales, agile wallabies (*Macropus agilis*), and a dugong (*Dugong dugon*) from Queensland, and a common wombat from Victoria.

Research

Previous research has investigated the possibility of developing a vaccine against *T. gondii*. A commercial *T. gondii* vaccine, developed for use in sheep, caused fatal toxoplasmosis when administered to tammar wallabies (*Macropus eugenii*). An oral vaccine consisting of *Hammondia hammondi*, a related protozoal organism, provided partial protection with three of five vaccinated tammar wallabies dying of toxoplasmosis when challenged with infection (Lynch et al 1993, Reddacliff et al 1993).

Current research is investigating the possible role of *T. gondii* in woylie (*Bettongia penicillata*) decline in Western Australia.

Human health implications

Toxoplasmosis is a zoonosis. *T. gondii* infection can cause abortion and foetal abnormalities in pregnant women and encephalitis in immunocompromised people.

Conclusions

While toxoplasmosis has the potential to cause significant mortality of wild marsupials its impact on wild populations is unknown. It has been cited as a cause of decline of the endangered Victorian brush-tailed rock wallaby but no evidence has been found to support this theory. Successful treatment of the condition remains elusive and, as long as feral cats are spread throughout the country, prevention will not be possible.

References and other information

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Updated: 23 March 2009

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