

Toxoplasmosis of Australian mammals

Fact sheet

October 2019

Introductory statement

Toxoplasmosis has been reported as a significant cause of morbidity and mortality in captive marsupials both within Australia and internationally. As such Australian marsupials are considered to be highly susceptible to toxoplasmosis. Although it has been commonly accepted that Australian marsupials are very susceptible to disease impacts of toxoplasmosis, there is little published data that correlates disease with seroprevalence in Australian wild mammals. The available data on seroprevalence in Australian mammals is limited and many studies are opportunistic or limited in their scope. Although clinical toxoplasmosis is recognised as a serious disease in captive Australian mammals, the true impact of toxoplasmosis on free-ranging populations of Australian marsupials, and other native mammals more broadly, is largely unknown. Species specific validation of diagnostic tests, appropriate survey sample sizes, minimisation or elimination of sampling bias, cohort studies and control of confounding variables are required to better understand the impact of this disease on free-ranging populations of Australian mammals ^[1].

Aetiology

Toxoplasma gondii is an obligate intracellular coccidian parasite belonging to the family *Sarcocystidae* within the phylum Apicomplexa and is the causative agent of toxoplasmosis.

Natural hosts

Members of the family *Felidae*, both domestic cats (*Felis catus*) and a broad range of non domestic felines, are the known definitive hosts (DH), while all endothermic vertebrates are thought capable of acting as intermediate hosts (IH).

Within Australia, clinical, serological or pathological evidence of toxoplasmosis has been reported in a wide range of marsupial species, including:

- red and grey kangaroos, wallaroos and wallabies (*Macropus* spp.); pademelons (*Thylogale billardierii*), bridled nailtail wallaby (*Onychogalea fraenata*) and rock wallabies (*Pterogale* spp.)
- bettongs (*Bettongia* sp.), potoroos (*Potorous* spp.) and quokka (*Setonix brachyurus*)
- brushtail and ringtail possums (*Trichosurus* spp. and *Pseudocheirus* spp.) and pygmy possums (*Cercartetus* spp.)
- common wombats (*Vombatus ursinus*)
- bandicoots (*Isodon* spp. and *Perameles* spp.) and bilbies (*Macrotis lagotis*)
- Tasmanian devils (*Sarcophilus harrisii*), quolls (*Dasyurus* spp.), antechinus (*Antechinus* spp.), phascogales (*Phascogale* spp.), kowari (*Dasyuriodes byrnei*) and dunnarts (*Sminthopsis* spp.) ^[1].

Reports of clinical, pathological or serological evidence of infection in eutherian mammals include:

- flying foxes (*Pteropus* spp.) (eWHIS)
- dugongs (*Dugong dugon*) (eWHIS)
- New Zealand fur seal (*Arctocephalus forsteri*)^[2]
- Risso's dolphin (*Grampus griseus*)^[3]
- Indo-Pacific bottlenose dolphin (*Tursiops aduncus*)^[4]
- Indo-Pacific humpbacked dolphins (*Sousa chinensis*)^[4, 5].

Age and sex linked predispositions are generally unknown for most Australian mammals. However, in free-ranging eastern quolls (*Dasyurus viverrinus*) in Tasmania, higher seroprevalence has been observed in males compared with females and probability of infection increased with age^[6]. In contrast female free-ranging western grey kangaroos (*Macropus fuliginosus*) were significantly more likely to be seropositive than males^[7].

World distribution

The disease occurs worldwide, wherever cats are found.

Occurrences in Australia

The disease is presumed to occur Australia wide, due to the extensive range of feral cats. Seroprevalence studies vary widely both between and within Australian states, with high seroprevalence observed in both Qld and Tas. There is insufficient data to determine if there is a difference in prevalence in tropical versus temperate areas of the country.

Epidemiology

The life cycle of *T. gondii* is divided into sexual and asexual replication phases. Sexual replication occurs in the feline intestine following ingestion of either environmental oocysts or tachyzoites and/or bradyzoites present in the tissues of infected intermediate hosts. The intestinal sexual replication phase results in the production of unsporulated oocysts which are passed in faeces and subsequently sporulate within one - five days depending upon environmental conditions. Sporulated oocysts can survive for months to years in wet soil.

Environmental contamination is widespread with cats shedding millions of oocysts following infection. Global *T. gondii* seroprevalence is estimated at 30-40% for domestic cats however oocysts are only shed for a period of one to two weeks following infection and less than 1% of cats are thought to be shedding oocysts at any one time^[8].

The asexual replication phase occurs in intermediate hosts (IH), including both herbivorous and carnivorous species, which are infected via different pathways. Herbivorous IHs are exposed following ingestion of vegetation, soil or water contaminated with sporulated oocysts. Following ingestion of oocysts, sporozoites invade intestinal cells within 24 hours and subsequently divide, via an asexual process, to become tachyzoites. The tachyzoite is the infectious stage that multiplies rapidly inside the host's body during the acute phase of the disease. Following dissemination throughout the body, tachyzoites differentiate into slowly replicating bradyzoites and subsequently

form tissue cysts. Tissue cysts can develop in any tissue but are most prevalent in muscular and neural tissues, particularly the central nervous system, the eye and cardiac and skeletal muscle. Tissue cysts may persist for the life of the intermediate host. Evidence exists to support vertical transmission as a potential route of infection in macropods^[9, 10].

Carnivorous intermediate hosts are exposed following ingestion of bradyzoites or tachyzoites in infected prey; bradyzoites invade the intestinal epithelial cells to initiate asexual replication. Experimental studies in eastern barred bandicoots (*Perameles gunnii*) suggest that earthworms, a major dietary component of this species, may act as mechanical vectors of *T. gondii*, passing oocyst contaminated soil through their alimentary tracts^[11].

Exposure pathways have not been definitely identified for Australian marine mammals although infection via contaminated coastal freshwater runoff is considered likely and was identified as a potential risk factor in Indo-Pacific humpbacked dolphins (*Sousa chinensis*), an Indo-Pacific bottlenose dolphin (*Tursiops aduncus*), Risso's dolphins (*Grampus griseus*) and a New Zealand fur seal (*Arctocephalus forsteri*) with toxoplasmosis^[2-5]. Benthic invertebrates and filter feeding fish have been postulated as potential sources of oocysts for Australian marine mammals^[12].

Recent studies in a range of Australian marsupials have demonstrated the presence of non-archetypal type II *T. gondii* genotypes and atypical strains and, in the case of western grey kangaroos, multiple infections in individual animals^[13-15]. Additionally, atypical type II strains have been isolated from a New Zealand fur seal and a Risso's dolphin with toxoplasmosis^[2]. The significance of the predominance of these non-archetypal and atypical strains in clinical cases of toxoplasmosis in free-ranging Australian mammals is unknown but it has been suggested these strains may be more pathogenic.

The incidence of morbidity and mortality in Australian mammals following infection with *T. gondii* is unknown but the presence of antibodies to *T. gondii* in a range of species demonstrates that exposure is not invariably fatal^[6, 7, 16]. The outcome for the host following exposure is likely influenced by the virulence and strain of *T. gondii* involved, inoculation dose and immune status of the host^[1].

There appears to be variation in host species susceptibility to toxoplasmosis, with suggestions that marsupials as a taxon are particularly sensitive to the clinical impacts of *Toxoplasma* infection. Amongst marsupials, macropods, wombats and bandicoots are considered highly susceptible, however there is little robust published data to demonstrate increased species susceptibility other than apparently higher disease prevalence. A longitudinal study on small numbers of wild eastern barred bandicoots (*Perameles gunnii*), concluded that findings suggested that this species was probably highly susceptible to primary *T. gondii* infection^[17] however another study suggested toxoplasma infections in captive eastern barred bandicoots occurred at low prevalence, were chronic in nature and not associated clinical disease^[18].

Clinical signs

A broad range of clinical signs have been reported from Australian mammals consistent with the multiple body systems affected by this disease. The course of the disease can range from peracute

through to chronic and subclinical infections are possible. Death without premonitory signs appears to be a common presentation in captive marsupials. Clinical signs may vary considerably given the wide range of body systems affected but neurological signs predominate across most taxonomic groups and may include blindness, ataxia, circling, incoordination, nystagmus, head tilt, hind limb paralysis, altered mentation and dysphagia^[13, 19-21]. Ocular lesions include keratitis, uveitis, chorioretinitis, endophthalmitis and the development of unilateral or bilateral cataracts in some individuals. Other more generalised signs include dyspnoea, tachypnoea, coughing, depression, anorexia, lymphadenopathy, polydipsia, weight loss, generalised weakness, diurnal activity in nocturnal species and dehydration. Diarrhoea, although not described in other marsupials, occurs relatively frequently in macropod^[19, 22]. Clinical signs in marine mammals include stranding, emaciation, altered mentation and impaired navigation^[2, 3, 5].

Diagnosis

A presumptive ante mortem diagnosis of toxoplasmosis can be made in the live animal by demonstrating either an elevated IgM titre in conjunction with a low or negative IgG titre (acute disease) or rising/elevated IgG and IgM titres (chronic disease) combined with supporting clinical signs and history. A definitive diagnosis can be made post mortem using a combination of histopathology and immunohistochemistry with or without molecular diagnostic techniques.

Serological tests available for the ante mortem diagnosis of toxoplasmosis include the modified (MAT) and direct agglutination tests (DAT), Sabin-Feldman dye test, the latex agglutination test, the indirect haemagglutination test, the immunofluorescent antibody test (IFAT) and various ELISAs. However, given the often acute course of the disease, a serological response may not be evident making ante mortem diagnosis of acute clinical cases challenging. An elevated immunoglobulin M (IgM) titre, as determined by the DAT, with a low or absent immunoglobulin G (IgG) titre, as determined by the MAT, is considered to be indicative of acute toxoplasmosis^[17, 23, 24]. Elevations in both IgM and IgG occur following the acute phase of infection.

Toxoplasma gondii tissue cysts cannot be differentiated from those of other cyst forming coccidia such as *Neospora caninum* or *Hammondia hammondi* on the basis of histopathology alone. Molecular diagnostic and immunohistochemical techniques can be used on tissue samples to confirm toxoplasmosis post mortem. Immunohistochemistry using anti-*Toxoplasma gondii* polyclonal serum can be performed on fixed tissue^[2, 5, 13]. Polymerase chain reaction (PCR) has recently been employed to diagnose toxoplasmosis in common wombats (*Vombatus ursinus*), various macropod species and a New Zealand fur seal^[2, 13-15]. Next generation sequencing based on the 18S rRNA gene has also been used to diagnose and characterise the *T. gondii* genotype involved in a case of toxoplasmosis in a Risso's dolphin^[3].

Laboratory diagnostic specimens and procedures

Submit serum from the live animal for serology. The Tasmanian Department of Primary Industries, Parks, Water and Environment Animal Health Laboratory can perform serology using either the MAT or IFAT on a minimum of 0.2 mL of serum (<https://nre.tas.gov.au/biosecurity-tasmania/animal-biosecurity/animal-health-laboratories/animal-health-laboratory/serology>). Post mortem samples include fresh or frozen tissues for molecular diagnostics and fixed tissue for

histopathology and immunohistochemistry. Tissue cysts are most prevalent in muscular and neural tissues so brain, ocular tissue, cardiac and skeletal muscle are priority tissues.

Detailed information on laboratory procedures required for diagnosis of toxoplasmosis is available at the Australian Registry of Wildlife Health (arwh.org).

Clinical pathology

There is limited information on changes in haematologic and biochemical parameters in affected in Australian mammals. Biochemical abnormalities in a captive red necked wallaby (*Macropus rufogriseus*) with acute systemic toxoplasmosis included azotaemia, hypoglycaemia, hypoalbuminaemia, elevated alanine aminotransferase, hypercholesterolaemia, hyperbilirubinaemia, hyperphosphataemia and hypocalcaemia ^[25]. Haematologic changes included a severe thrombocytopenia and the presence of ovoid intracytoplasmic protozoal inclusions in circulating monocytes and neutrophils. Haematological and biochemical changes reported in a free-ranging Indo-Pacific humpbacked dolphin included a neutrophilia and elevations in fibrinogen, creatine kinase and lactate dehydrogenase ^[5].

Pathology

Often there are no gross findings at necropsy. If present, lesions include pulmonary congestion and oedema, myocardial haemorrhage, splenomegaly, gastric ulceration, lymphadenomegaly and malacia of the brain ^[26]. 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 ^[19, 26-28]. Periodic Acid Schiff (PAS) positive tissue cysts are most commonly found in the brain, muscle and adrenal gland ^[19]. Necrosis, nonsuppurative meningoencephalitis and myocarditis are the predominant histopathological lesions in marine mammals ^[2, 3, 5].

Differential diagnoses

Due to the multiple body systems affected, variable clinical signs and often non-specific gross pathology a broad range of diseases could be considered as potential differential diagnoses.

Treatment

Treatment is unlikely to be practical in free-ranging wildlife and is frequently unrewarding in the captive setting. Some success has been achieved with the anti-protozoal drug atovaquone at 100 mg/kg PO SID for at least 30 days ^[29]. Other potential treatment options include clindamycin and trimethoprim-sulphonamide orally.

Prevention and control

The wide spread distribution of feral cats across Australia and the persistence of infective cysts means that environmental contamination of infective oocysts is widespread, and probably ubiquitous in areas where feral cats are present. Attempts to limit the disease that focus solely on control or management of feral cats may have limited success. In the captive setting, exposure can be minimised by preventing cats from accessing enclosures or preventing faecal contamination of

food and water sources. As juvenile cats have been shown to be more likely to shed the organism, it has been suggested that feral cat sterilisation programs may reduce the environmental load of infective cysts.

Attempts to vaccinate tammar wallabies (*Macropus eugenii*) with a mouse adapted strain of *T. gondii* administered intramuscularly and oral administration with oocysts of *Hammondia hammondi*, a related protozoal organism, were both unsuccessful [24, 30]. Limitations to the development of an effective vaccine for *T. gondii* include the existence of multiple genetic lineages with variable strain virulence and stage specific expression of *T. gondii* proteins [31]. Until advances in vaccine technology and delivery options for *T. gondii* vaccination the options for prevention of toxoplasmosis in Australian wildlife are limited.

Research

Research priorities for *T. gondii* include species specific validation of serological tests, robust serological surveys to better establish the prevalence of the disease, better characterisation of the genotypes present in Australian wildlife and their pathogenicity, establishment of potential predisposing factors for the development of disease, a better understanding of the role of toxoplasmosis in species' declines and the development of an effective and practical vaccine for wildlife where toxoplasmosis is identified as a threatening process.

Surveillance and management

There is no targeted surveillance program for *T. gondii* and it is not a nationally notifiable animal disease. Seroprevalence surveys have been conducted in a range of free-ranging Australian wildlife species including macropods, wombats, bandicoots, possums and dasyurids, using a range of diagnostic tests [6, 7, 16, 17, 32, 33]. Study sample sizes range from very small to large and seroprevalence rates have ranged from 0% to 100%. The variability of findings, even within the same taxonomic group, makes it difficult to draw conclusions on the susceptibility of Australian mammals to this disease. Evidence correlating higher seroprevalence but not clinical disease in Tasmanian pademelons (*Thylogale billardierii*) and eastern quolls with higher feral cat densities has been presented [6, 34]. Other than cat control, there are no active management strategies for toxoplasmosis in Australian wildlife.

Wildlife Health Australia administers Australia's general wildlife health surveillance system, in partnership with government and non-government agencies. Wildlife health data is collected into a national database, the electronic Wildlife Health Information System (eWHIS). Information is reported by a variety of sources including government agencies, zoo based wildlife hospitals, sentinel veterinary clinics, universities, wildlife rehabilitators, and a range of other organisations and individuals. Targeted surveillance data is also collected by WHA. See the WHA website for more information <https://wildlifehealthaustralia.com.au/Our-Work/Surveillance> and <https://wildlifehealthaustralia.com.au/Our-Work/Surveillance/eWHIS-Wildlife-Health-Information-System>

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife

Health Information System. Negative data are also valuable. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

Statistics

There are over 100 cases of toxoplasmosis in native mammals reported in the National Wildlife Health Surveillance Database, from all areas of Australia other than the NT. Cases reported in eWHIS are predominantly from macropods and wombats, and include bandicoots, tawny frogmouths (*Podargus strigoides*) and a range of other avian species.

Human health implications

Toxoplasmosis is a zoonotic disease. Humans are exposed following ingestion of soil, water, fruits and vegetables contaminated with oocysts, or ingestion of tissue cysts present in undercooked meat from IH. *T. gondii* infection can cause abortion and foetal abnormalities in pregnant women and encephalitis in immunocompromised people. Infections in immunocompetent people are generally asymptomatic.

Conclusions

Toxoplasmosis has been documented to cause significant mortality of captive marsupials the evidence for its impact on wild populations is currently equivocal. It is considered an emerging infectious disease of Australian wildlife due to reports of disease in an increasing range of native mammal species. Ongoing studies are recommended to improve understanding of this disease in the Australian context.

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Wildlife Health Australia recognises the Traditional Custodians of Country throughout Australia. We respectfully acknowledge Aboriginal and Torres Strait Islander peoples' continuing connection to land, sea, wildlife and community. We pay our respects to them and their cultures, and to their Elders past and present.

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