Executive summary – the effects of *Toxoplasma gondii* on New Zealand wildlife: implications for conservation and management

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Toxoplasma gondii (hereafter toxoplasma) is a globally widespread protozoan parasite that can cause the potentially fatal disease toxoplasmosis. This review summarises the research on:

- the effects of toxoplasma on New Zealand and global wildlife species
- the current understanding of pathways of toxoplasma infection
- potential risk factors for infection and morbidity of host species.

The review then identifies some relevant management options for reducing the transmission of toxoplasma, and for minimising risks to New Zealand wildlife.

Cat species, including the domestic cat, are the only known definitive hosts for toxoplasma, and cats typically shed millions of oocysts in their faeces after infection. The oocysts can remain infective for long periods (many months) in soil, freshwater and saltwater. Infection also occurs through other pathways; i.e. ingestion of toxoplasma cysts in tissue of prey species, or (for mammals only) toxoplasma can cross the placenta to infect the foetus. These characteristics have led to the widespread distribution of toxoplasma, and high infection rates across a multitude of terrestrial, freshwater and marine bird and mammal species worldwide.

A review of the international literature suggests that risk factors for infection with toxoplasma are related to exposure to toxoplasma, host and toxoplasma genotype and immunosuppression. Unsurprisingly, spatial overlap with felids is a significant risk factor for infection of wildlife populations; with carnivores, omnivores and apex predators being particularly susceptible to infection through eating prey containing tissue cysts. Ground-dwelling species are also prone to infection, through incidentally ingesting oocysts while foraging for food. Perhaps more surprising is the infection of aquatic species, and the high rates of infection for some coastal species, which is related to the transport of toxoplasma oocysts in freshwater runoff. Toxoplasmosis has been identified as a cause of death for multiple marine mammal species, including sea otters, phocid and otariid seals, dolphins, porpoises, and large cetaceans. Clinical toxoplasmosis is likely to be a population risk for multiple marine mammal populations of the Pacific Region, including sea otters along the west coast of North America, Hawai'ian monk seals, and Hector's and Māui dolphins around New Zealand.

Toxoplasma virulence in a host is strongly influenced by the interaction of host and parasite genetics, particularly in geographically incoherent host-parasite combinations. For example, toxoplasma strains common in Eastern Asia are virulent for laboratory mice, which are predominantly Western European in genetic composition, but are not virulent for the southeastern Asian house mouse. Previous studies have noted the increased susceptibility of certain mammals and birds to morbidity and mortality from toxoplasmosis, including some marsupials and New World monkeys. Pigeons and true finches may have increased susceptibility to clinical toxoplasmosis, and cases of fatal toxoplasmosis have been reported in the Hawai'ian crow/'alala, parrots, wild turkey, and various penguin species, including little blue penguin (in Australia).

Infected individuals require an effective immune response to contain the subsequent dissemination of the parasite. Immunosuppression can therefore increase the likelihood of active toxoplasmosis. Relevant mechanisms known to cause immunosuppression in vertebrates include adverse nutritional status, genetic abnormalities, viral diseases, and high levels of certain toxic agents within the body (immunotoxicity), as well as natural life history processes such as pregnancy and ageing. In marine mammals, infection with morbillivirus is known to impair immune function in a number of species, particularly in conjunction with elevated tissue concentrations of contaminants such as polychlorinated biphenyls (PCBs). In a number of mammal species, including humans and mice, females can be more susceptible to toxoplasmosis, which appears to relate to sex-differences in the immune response to infection. Predictable changes in the female immune system occur throughout gestation, including

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the temporary suppression of interferon-gamma, which is essential to prevent the reactivation of latent toxoplasma infection. However, the effects of gestation on maternal susceptibility to toxoplasmosis are poorly understood, particularly for mammals other than humans and mice.

New Zealand lacks any wild felids, so toxoplasma should have been absent from the environment and native fauna prior to the introduction of the domestic cat in the late 18th Century. Although the effects of toxoplasmosis on livestock are well-understood in New Zealand, and information on the human health implications of the disease is widely available, toxoplasma has only recently been identified as a threat to New Zealand native wildlife.

Cat ownership in New Zealand is high compared with many other countries. In 2015, 44% of households had at least one cat and the owned (pet) cat population was estimated to be 1.13 million individuals. Estimates for unowned cat populations in New Zealand are much more uncertain but could range between c. 160,000 to 760,000 cats. Thus, the total New Zealand cat population could be between 1.3 and 1.9 million individuals. Cats primarily acquire toxoplasma via the predation of infected prey, and seroprevalence is generally higher in feral and stray cats than in owned cats due to an elevated dietary intake and access to infected wild prey. Owned cats with outdoor access to wild prey tend to have higher toxoplasma seroprevalence than indoor cats. Approximately 90% of New Zealand's owned cats have outdoor access, which is significantly higher than in many other countries, meaning that they are more likely to capture prey and defecate outdoors.

As in other countries, toxoplasmosis is likely to be a significant public health issue in New Zealand. Although fatal cases are typically associated with immunosuppression, sublethal effects (including foetal abnormalities and ocular toxoplasmosis causing blindness) are more common. Internationally, seroprevalence varies from 0 to 100% across different countries, regions and groups of people. Seroprevalence studies in New Zealand are limited, although testing of blood donors from the Waikato region indicated that seroprevalence was 42.9% ± 8.12%.

The importance of toxoplasmosis in New Zealand livestock was first recognised in the 1950s, when it was identified as an important cause of lamb mortality. The only commercially licensed toxoplasma vaccine in the world, Toxovax®, was developed in New Zealand and made commercially available in 1988 for use in prevention of abortion and congenital infections in sheep and goats. However, toxoplasmosis continues to be a major economic burden to the New Zealand sheep industry, due to lost production and the cost of vaccinating ewes.

Relative to captive animals, few full diagnostic investigations have been completed on wild-living populations in New Zealand, meaning that our understanding of toxoplasma infection rates and toxoplasmosis-related morbidity and mortality in wildlife is dependent on opportunistic studies. There are few reports of fatal toxoplasmosis in native avian species in the peer-reviewed literature. Six avian cases in five different endemic New Zealand bird species were reported between 2009 and 2014, including two kererū, one kākā, one brown kiwi, one paradise shelduck and one red-crowned kākāriki. Other sources of information (e.g. Massey University School of Veterinary Science Pathology Database) contain several more cases in kiwi, kererū and kākā. Based on the total number of records in the database, however, fatal toxoplasmosis seems to be uncommon in native avian species, and there have been no recorded cases of clinical toxoplasmosis amongst the 67 necropsies conducted on native bat species.

As for terrestrial wildlife, information with respect to toxoplasma infection and associated morbidity in the majority of marine wildlife is extremely limited, or entirely lacking. However, there are known to be high rates of toxoplasma infection and subsequent mortality from toxoplasmosis in Hector's and Māui dolphin. One study found 17 out of 28 (61%) of Hector's and Māui dolphins were infected, and a total of nine out of 38 (24%) deaths recorded in the Massey University SoVS Pathology Database between 2007 and 2018 were determined to have been caused by toxoplasmosis, excluding neonates. Two of the mortalities were from the west coast of the North Island (the habitat of Māui dolphin subspecies) and the others were Hector's dolphins, including five from the east coast of the South Island and two from the west coast, which indicates that toxoplasma infections of this species are geographically widespread. Seven out of the nine were females, of which six were deemed to be mature. Although the sample size is small, this tentatively suggests an increased susceptibility of reproductive females to toxoplasmosis. The mechanisms for a potential sex-bias are not known, though they could include natural or nutrition-related changes in immune function in late pregnancy (all reported Hector's and Māui dolphin toxoplasmosis deaths to date have been in September to November, immediately prior to the main calving period).

There were no recorded cases of toxoplasmosis in the more than 100 individuals of other cetacean species in the SoVS Pathology Database, although full histological examinations were not conducted in most cases, increasing the possibility of false negatives. The seroprevalence in mainland New Zealand sea lions indicates a moderate level of exposure to toxoplasma for the mainland population only. Apart from a single case in 2012 for which toxoplasmosis was a contributing cause of death, no other

cases of clinical toxoplasmosis were found in 27 necropsies of New Zealand sea lions recovered on the mainland. There is very limited or no data on other native marine mammals and bird species, including the New Zealand fur seal and most New Zealand penguin populations – all of which breed on land and forage in coastal waters, so should be subjected to some degree of exposure to infection with toxoplasma.

Most New Zealand native bird and mammal species have not been screened for toxoplasma infection and only a limited range of species are routinely necropsied using methods that could detect active toxoplasmosis. As such, we currently have a very basic understanding of the conservation threat of toxoplasma to New Zealand wildlife. The preliminary evidence suggests that fatal toxoplasmosis may be more frequent in Hector's and Māui dolphins relative to other native cetaceans and avifauna, but further information on the prevalence of toxoplasma infection and the population risk of clinical and fatal toxoplasmosis in other species is required. Investigations of this nature should target those species with a demonstrated susceptibility to fatal toxoplasmosis (such as kiwi, kererū and kākā), potential taxonomic susceptibility (e.g. pigeon and parrot species), species with a high risk of exposure to toxoplasma (e.g. estuarine, wetland and ground-dwelling species), or species with an adverse threat classification status coupled with moderate-to-high risk of exposure to toxoplasma infection.

Management of toxoplasma infection in wildlife presents a significant challenge due to the widespread distribution of both the domestic house cat (the only definitive host in New Zealand) and species that can act as intermediate hosts (i.e. all bird and mammal species), coupled with the transmission of oocysts via hydrological networks, and the longevity of the parasite in hosts. Management of cat populations can be contentious and challenging, involving conflicts in human values, as well as obvious resourcing issues relating to the large scale of the problem. Nevertheless, in order to protect New Zealand wildlife, management strategies need to be aimed either at cats (owned or unowned), or at limiting the transport of toxoplasma through the environment, or a combination of both.

Environmental transmission of toxoplasma oocysts is the major transmission pathway for infection of many wildlife species. Therefore, measures to reduce toxoplasma oocyst loading to the environment (e.g. a reduction in the number of cats with access to the outdoors) should be effective in reducing the threat to wildlife species. For owned cats this would mean encouraging cat owner behaviour change with respect to keeping cats indoors or mandating the physical containment of pet cats through policy or regulation. In New Zealand, widespread support and adoption of such an action would be challenging, as a large proportion (c. 90%) of pet cats currently have access to the outdoors, and cat owners are generally not supportive of cat containment or regulation. An alternative to the containment of pet cats could be widespread vaccination of domestic cats against toxoplasmosis. Several experimental cat-specific vaccines have been developed, but their implementation has been limited by high costs, short shelf-life and limited interest or incentives for cat owners to use them, since toxoplasmosis is rarely fatal to the cat. Consequently, no commercially produced vaccine is currently available either locally or internationally.

The control of unowned (feral and stray) cats is an additional management option for reducing the oocyst loading to the environment. Compared with owned cats, oocyst loading is likely to be higher from unowned cats (on an individual basis) for two reasons: 1) toxoplasma seroprevalence in unowned cats tends to be higher than in pet cats, and 2) unowned cats have shorter life spans, breed more rapidly and have a greater population turnover. There are many complexities and issues associated with cat management in New Zealand, with inconsistencies in legislation, approach and commitment at the local and central government level. Management of owned and unowned cats is currently carried out in a piecemeal manner under various pieces of legislation and there is no legislation giving government departments the authority to control stray cats. A draft National Cat Management Strategy that called for a consistent, national approach for cat management was developed in 2017, and a plan such as this is likely to be critically important in enabling effective management of the risks to wildlife posed by toxoplasma.

Aside from cat management, other management strategies could aim to limit the transport of toxoplasma oocysts through hydrological networks and into the marine environment. Management of such a diffuse (and hardy) environmental contaminant is extremely difficult, although research overseas has shown that wetlands can enhance sedimentation of oocysts or trap oocysts in vegetation. However, the efficacy of wetlands at processing any type of contaminant is dependent on multiple factors, including hydrology, wetland size, location, type of vegetation and contaminant loading. The currently available management options with regards to treatment of stormwater and wastewater are also limited. Oocysts appear to be resistant to standard disinfection processes, including ultraviolet treatment, and so are unlikely to be removed by the wastewater treatment process. Urban stormwater is potentially a significant source of oocysts to waterways, as rainfall runoff is likely to transport cat faeces into stormwater drains, which tend to discharge directly into streams, rivers or the marine environment. Design of green infrastructure, such as vegetated areas or constructed

wetlands in stormwater and wastewater treatment plants, may be able to reduce oocyst transmission into waterways. The efficacy of these interventions for this particular purpose are unknown, however, and new technologies would likely need to be retrofitted into existing infrastructure.

A further complication when assessing or designing management interventions is the poorly developed methodology for measuring toxoplasma oocyst concentration in water samples. This makes it difficult to assess the relative contribution of wastewater treatment plants, stormwater systems or particular catchments in the loading of toxoplasma oocysts to aquatic environments, and then to monitor or assess the efficacy of any mitigation measures that are implemented.

The management of conservation risks posed by toxoplasma to New Zealand wildlife is currently difficult for multiple reasons. These include a limited understanding of the scale of the issue (both in terms of species that are susceptible to infection or mortality associated with toxoplasmosis, and population-level effects), limited understanding of virulent toxoplasma strain(s) in New Zealand, and whether these are associated with certain cat populations and spatial patterns of loading. Furthermore, although there are many potential management actions that could reduce toxoplasma contamination in the environment, there are also major knowledge gaps in terms of the targeting, efficacy, practicalities and costs of these actions. There is a critical need for targeted research to inform effective management.

This review suggests that priority research needs are:

- To assess the prevalence of toxoplasma infection in species that have a demonstrated susceptibility to developing fatal toxoplasmosis (including Hector's and Māui dolphin and certain native bird species), and to collect information for assessing the population risk of toxoplasma disease and mortality for these species.
- Spatial analyses to identify hotspots of contamination in marine and freshwater environments, focussing on catchments used by priority wildlife species.
- Genotyping of toxoplasma strains of different cat populations (i.e. by location or ownership status) to identify cat populations shedding toxoplasma strains that are known to be lethal to Hector's and Māui dolphins and other native species.

There is also a need to assess the efficacy of cat management options and potential treatment options for stormwater, wastewater and different types of wetland, monitoring and improving understanding of co-factors (e.g. environmental contaminants) that may compromise species' immune function and susceptibility to toxoplasmosis, and consideration of integrated management approaches to mitigate toxoplasmosis effects on human health and livestock, in addition to effects on wildlife.