

Toxoplasmosis: An Overview

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Background on toxoplasmosis and the role of cats

Toxoplasmosis is caused by the parasite *Toxoplasma gondii* (*T. gondii*). The definitive host (DH) for *T. gondii* is cats: domestic and wild, where domestic cats include pets – both indoor and outdoor – and feral, or free-roaming/unowned cats. As the DH, the cat is the ONLY host in which *T. gondii* reproduces. That is, the parasite MUST pass through the cat's digestive system to reproduce and make eggs, or oocysts; these are then shed in cat feces.¹ Birds, rodents and other mammalian species, e.g., pigs, sheep, chickens, are intermediate hosts (IH) in that they become infected by ingesting oocysts in water or soil, or by eating other infected animals. Humans are also infected by *T. gondii*, from ingesting water or soil contaminated with oocysts, or by ingesting tissue cysts in undercooked meat.² Cats often become infected after ingesting the tissue cysts of infected animals.

According to the Centers for Disease Control and Prevention, cats only spread *T. gondii* in their feces for a few weeks following infection with the parasite. Interestingly, Jones and Dubey note, in their 2010 paper, that information on cats shedding oocysts is based on observations of cats under laboratory conditions; they state that how often cats shed oocysts in nature is unknown.³ The parasite does **not** (emphasis added) become infectious until 1 to 5 days after being shed in the infected cat's feces.⁴

Both outdoor pet cats and feral/free-roaming cats deposit infected feces outdoors. In laboratory conditions, domestic cats shed millions of oocysts after becoming infected, but, once again, the amount shed by cats in nature is not known.⁵ Jones and Dubey explain, in their 2010 article, that the reason the amount shed has not been quantified is that there are a number of technical problems in all of the methods used.

Given the uncertainty in the number of oocysts that an infected cat sheds, there have been various estimates made of the quantities of oocysts deposited into the environment. The total quantity deposited also depends upon the number of cats shedding oocysts. Jones and Dubey assumed that 30 percent of domestic and feral cats shed oocysts, at the rate of one million per cat.⁶ Using numbers from a study in

¹ Jones, J.L and Dubey, J.P. (2010) Waterborne toxoplasmosis – Recent Developments. *Experimental Parasitology* 120: 10 - 25

² Ibid

³ Ibid

⁴ http://www.cdc.gov/parasites/toxoplasmosis/gen_info/faqs.html#cat_infected

⁵ Jones, J.L and Dubey, J.P. (2010) Waterborne toxoplasmosis – Recent Developments. *Experimental Parasitology* 120: 10 – 25

⁶ Ibid.

2005 that estimated the number of domestic cats as 78 million, and the number of feral cats at 73 million, they reported that there would be at least 50 million oocysts in the environment.⁷ A study done in 2007 looked at three communities in California that included 7,284 pet cats and 2,046 feral cats. Using a range of reported oocyst production, the researchers estimated that the number of oocysts ranged from 9 to 434 per square foot.⁸ Note that the Humane Society of the United States estimates the number of domestic cats in 2012 as 86.4 million.⁹ The number of feral cats will certainly have increased since the 2005 estimate of 73 million. However, whether that number is in the same ratio as the 2005 estimate, i.e., 73 million as compared to 78 million, or is similar to the ratio found in the three California communities, i.e., 7284 compared to 2046, is not known.

***T. gondii* in the environment**

Given the many millions of oocysts deposited by cats, there are a number of questions that arise regarding the effects that these oocysts may cause: How long do oocysts remain viable in the environment? Do different environments – fresh water, salt water, different types of soils, different temperature and humidity – affect the viability of the oocysts? Do oocysts travel in run-off from places where they are deposited and buried into the ocean? Has *T. gondii* infected and/or caused the death of marine animals?

The 2007 study by Dabritz, et al. stated that the level of land contamination they determined is likely to be high enough to reach waters where mammals such as sea otters can be infected.¹⁰ In fact, the Dabritz, et al. study was attempting to determine the origin of cat fecal contamination thought to be responsible for the deaths of sea otters in the Morro Bay region of California: the three communities studied were coastal communities near that bay.¹¹

Dubey, et al., in a 2003 study, looked at parasitic infections, including *T. gondii*, of marine mammals. They noted that the mechanism of infection of marine mammals was “intriguing” because most of these animals feed on fish or invertebrates – which

⁷ Conrad, P.A., et al. (2005) Transmission of *Toxoplasma*: Clues from the study of sea otters as sentinels of *Toxoplasma gondii* flow into the marine environment. *International Journal for Parasitology* 35: 1155 - 1168

⁸ Dabritz, H.A., et al. (2007) Detection of *Toxoplasma gondii*-like oocysts in cat feces and estimates of the environmental burden. *Journal of the American Veterinary Medical Association* 231: 1676 - 1684

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http://www.humanesociety.org/issues/pet_overpopulation/facts/pet_ownership_statistics.html

¹⁰ Dabritz, H.A., et al. (2007) Detection of *Toxoplasma gondii*-like oocysts in cat feces and estimates of the environmental burden. *Journal of the American Veterinary Medical Association* 231: 1676 - 1684

¹¹ Jessup, D. A., et al. (2007) Sea otters in a dirty ocean. *Journal of the American Veterinary Association* 231 (11): 1648 - 1652

are cold-blooded and not intermediate hosts – or are exclusively herbivorous.¹² When a 2002 study suggested that land-based surface runoff was a risk for *T. gondii* infection in California sea otters,¹³ researchers began examining the survivability of the parasite in sea water, as well as the role of marine invertebrates – mussels, mollusks, etc. – in infecting marine mammals. Mollusks can filter large quantities of water, and in the laboratory were able to concentrate the oocysts.¹⁴

It is important to note here what the Miller, et al. 2002 study did and did not state. This important study found that otters sampled near areas of maximal freshwater runoff were approximately three times more likely to be seropositive – positive in antibody testing – to *T. gondii* than otters sampled in areas of low flow. The authors also state: “The marine source of *T. gondii* exposure for sea otters is not known.” They add that one possible route is direct ingestion of seawater with infective oocysts, but that infection might also result from ingesting clams and mussels that concentrate the oocysts. The authors concluding remarks in the article are that their study provides “compelling evidence” implicating land-based runoff as a source of infection for sea otters.¹⁵ At no time do the authors state that runoff containing *T. gondii* oocysts is the cause of infection in otters. And, in fact, a recent 2013 article on *T. gondii* oocysts in the environment states “... otter infection [with *T. gondii*] **supports** (emphasis added) land-to-sea transmission of this parasite through contaminated freshwater runoff.”¹⁶ There is no disputing that California sea otters have died from *T. gondii* infections, but **proof** (emphasis added) that the oocysts have come from runoff contaminated by cat feces is not currently available.

In terms of marine mammals, the one of importance to Hawai'i, and specifically, Kaua'i, is the Hawai'ian monk seal. The Hawai'ian monk seal is endangered, and in January 2004, an adult male was found dead in the surf zone on the beach at Kapa'a. This seal was the first reported infection with and death from *T. gondii*. Upon detailed necropsy, the seal was found to have multiple visceral and cerebral (brain)

¹² Dubey, J.P., et al. (2003) *Toxoplasma gondii*, *Neospora caninum*, *Sarcocystis neurona* and *Sarcocystis canis*-like infections in marine mammals. *Veterinary Parasitology* 116: 275 - 296

¹³ Miller, M.A., et al. (2002) Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*) *International Journal for Parasitology* 32: 997 - 1006

¹⁴ Dubey, J.P., et al. (2003) *Toxoplasma gondii*, *Neospora caninum*, *Sarcocystis neurona* and *Sarcocystis canis*-like infections in marine mammals. *Veterinary Parasitology* 116: 275 - 296

¹⁵ Miller, M.A., et al. (2002) Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*) *International Journal for Parasitology* 32: 997 - 1006

¹⁶ VanWormer, E., et al. (2013) Molecules to modeling: *Toxoplasma gondii* oocysts at the human-animal-environment interface. *Comparative Immunology, Microbiology and Infectious Diseases* 36: 217 - 231

lesions, considered typical of disseminated toxoplasmosis. The diagnosis of *T. gondii* was confirmed by three methods, including detailed DNA analysis.¹⁷

Two studies published in 2007 provided survey information on disease risks for monk seals: one in the northwestern Hawai'ian Islands (NWHI) and one in the main Hawai'ian Islands (MHI).

The NWHI study surveyed seals from all six breeding sites from 1997 – 2001, using 332 captured animals.¹⁸ *T. gondii* antibodies were found in two adult females. The authors used antibody testing to look for potential pathogens and found antibodies to 12 different pathogens, including *T. gondii*. However, antibody testing is not very specific, and the authors recommended additional studies to examine these pathogens as disease sources. The authors also concluded that the absence of clinical signs of toxoplasmosis, coupled with low antibody titers, provided no reason to think that *T. gondii* was a health problem for monk seals.¹⁹

The survey in the MHI looked at 12 monk seals.²⁰ The authors collected blood and tissue samples from the seals and also attached tracking devices. Seals were tracked for anywhere between 32 and 167 days. Using the tracking data, the authors determined that the seals foraged, traveled and rested in near-shore waters close to human population centers, sources of land-based water runoff and sewage dispersal. Thus, the authors state that seals are at **risk** (emphasis added) of exposure to infectious diseases associated with terrestrial animals that contaminate marine habitats. The specific disease-causing pathogens mentioned were: *T. gondii*, *Sarcocystis canis*, *Leptospira spp.*, *Brucella*, *Salmonella*. However, the tested seals had antibody titers to only two of these pathogens: two seals had *T. gondii* antibody titers, and two seals had antibody titers to *Sarcocystis canis*.²¹ The authors make note of the monk seal that was found dead in Kauai in early 2004, adding that it was apparently resident there and known to visit harbors and estuaries. The authors then state that it “... was **apparently** (emphasis added) infected by ingesting free-floating oocytes directly or secondarily through consumption of oocyte-contaminated prey.”²² The authors add that the most “likely” source of the infective states is from cat feces being washed into the ocean along natural watershed

¹⁷ Honnold, S.P., et al. (2005) Toxoplasmosis in a Hawaiian Monk Seal (*Monachus schauinslandi*). *Journal for Parasitology* 91(3): 695 - 697

¹⁸ Aguirre, A. A., et al. (2007) Infectious Disease Monitoring of the Endangered Hawaiian Monk Seal *Journal of Wildlife Diseases* 43(2): 229 -241

¹⁹ Ibid.

²⁰ Littnan, C.L., et al. (2007) Survey for Selected Pathogens and valuation of Disease Risk Factors for Endangered Hawaiian Monk Seals in the Main Hawaiian Islands *EcoHealth* 3: 232 -244

²¹ Ibid.

²² Ibid

drainages or sewage pipes.”²³ (NB: The authors cite the Conrad, et al. 2005 article and the Miller, et al. 2002 article as the basis of their statements on the cause of the fatal 2004 *T. gondii* infection. However, BOTH of these articles, on the deaths of California sea otters, state very clearly that the source of infection of the otters is unknown.^{24,25})

There is also anecdotal reporting that an additional five or more monk seals have died from *T. gondii* over the past ten years.²⁶ Specifically, this December 2010 article cites Charles Littnan’s work on *T. gondii* in monk seals. Littnan noted that he suspected that *T. gondii* had caused the death of a monk seal found on the beach in Kapa’a in the spring of 2010, but that he was awaiting confirmatory results. Extensive literature searches have not been able to find any reporting on this, or the other monk seal deaths mentioned in the Scientific American article. A telephone conversation with Dr. Littnan, the director of the NOAA Pacific Islands Fisheries Science Center’s Hawai’ian Monk Seal Research Program on 10 September 2013 did not provide additional details. Dr. Littnan referred the author of this paper to Dr. Michelle Barbieri, the veterinarian for the monk seal program. Because the team was getting ready for a three-week long research trip to the NWHI, Dr. Barbieri said that she was unable to provide any details about the reported monk seals. She did say that she was certain that *T. gondii* had been confirmed as the cause of death, and that the reason that no literature on the subject had been uncovered was because the paper was still being written. Dr. Barbieri promised to thoroughly research the monk seal deaths reported as being caused by *T. gondii* upon her return, and to contact the author of this paper.

Elimination of *T. gondii*

In terms of information that is available on *T. gondii* oocysts, Jones and Dubey’s 2010 article on waterborne toxoplasmosis has many details on the resistance of oocysts to various environmental influences. Oocysts are not killed by chemical and physical treatments applied in water treatment plants; however, most normally operating municipal water filtering systems in developed countries that use coagulation, flocculation and settling prior to filtration.²⁷ In the environment,

²³ Littnan, C.L., et al. (2007) Survey for Selected Pathogens and valuation of Disease Risk Factors for Endangered Hawaiian Monk Seals in the Main Hawaiian Islands *EcoHealth* 3: 232 -244

²⁴ Conrad, P.A., et al. (2005) Transmission of *Toxoplasma*: Clues from the study of sea otters as sentinels of *Toxoplasma gondii* flow into the marine environment. *International Journal for Parasitology* 35: 1155 – 1168

²⁵ Miller, M.A., et al. (2002) Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*) *International Journal for Parasitology* 32: 997 – 1006

²⁶ <http://scientificamerican.com/article.cfm?id=cat-disease-threatens-endangered-monk-seals> 7 December 2010

²⁷ Jones, J.L and Dubey, J.P. (2010) Waterborne toxoplasmosis – Recent Developments. *Experimental Parasitology* 120: 10 - 25

oocysts survived for up to 54 months in cold water, and for up to three days in water at a temperature of 45 degrees Centigrade (113 degrees Fahrenheit), as well as in soil for over a year.²⁸ Oocysts have also survived in seawater for more than six months.²⁹

Because of the long-term survival of *T. gondii* oocysts in the environment and the fact that cats – both domestic and feral and wild – continually defecate outside and deposit oocysts that infect other cats and other intermediate hosts that can then infect other cats and other intermediate hosts, it would be difficult to eliminate *T. gondii* from the environment. Although *T. gondii* cannot exist without cats, the only situation in which *T. gondii* could theoretically be eliminated from the environment would be in a location that could be totally controlled, in which all cats could be eliminated with absolute certainty. In addition, there is no research that provides information on how long it would take, in such a situation, for *T. gondii* to no longer be present.³⁰

It is therefore interesting to note that in Svalbard, Norway, above the Arctic Circle, where there are no felids and domestic cats are prohibited, *T. gondii* was isolated in 2007 from the arctic fox.³¹ With the total absence of any type of cat, the source of *T. gondii* is thought to be migratory birds.

²⁸ Ibid.

²⁹ Lindsay, D.S. and Dubey, J.P. (2009) Long-term survival of *Toxoplasma gondii* sporulated oocysts in seawater. *Journal for Parasitology* 95(4): 1019 - 1020

³⁰ Personal communication, J.P. Dubey, 19 August 2013

³¹ Prestrud, K.W., et al. (2008) First isolate of *Toxoplasma gondii* from arctic ox (*Vulpes lagopus*) from Svalbard. *Veterinary Parasitology* 151(2 - 4): 110 - 114

Diagram of transport of *Toxoplasma gondii* oocysts. Domestic (pets and feral/free-roaming) and wild felids are the only source of oocysts (red arrow). Freshwater runoff (blue arrow) likely plays a significant role in environmental transmission of oocysts in terrestrial and aquatic systems. Light gray arrows indicate possible routes of *T. gondii* transmission by exposure directly to oocysts or indirectly through food sources. (From: VanWormer, E., et al. (2013) Molecules to modeling: *Toxoplasma gondii* oocysts at the human-animal-environment interface. *Comparative Immunology, Microbiology and Infectious Diseases* 36: 217 – 231)

