Control of *Toxoplasma gondii* in *Enhydra lutris nereis*, the southern sea otter

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Abstract

Toxoplasma gondii is a protozoan parasite that infects birds and mammals across the globe. The feline definitive host's feces contain oocysts capable of infecting the intermediate host. When these contaminated feces are transported into waterways via surface runoff, *Enhydra lutris nereis*, the southern sea otter, can become infected by ingesting filter-feeding bivalves that concentrate the oocysts from water. Some control measures have been implemented to protect sea otters from toxoplasmosis, but further measures should be put in place due to the devastating effects of this disease on sea otter populations. Through increased monitoring, education, vaccine development, and environmental protection, *T. gondii* can be better controlled and sea otter populations can be better conserved.

Introduction

Toxoplasma gondii is a protozoan parasite that causes toxoplasmosis, a disease that affects all mammals and birds worldwide (Dubey and Beattie 1988). Cats, both domestic and wild, serve as the definitive host, or the host in which the parasite sexually reproduces (Frenkel et al. 1970, Miller et al. 1972, Jewell et al. 1972, Dubey 2008). The intermediate host for *T. gondii*, or the host in which the parasite is not sexually reproducing, is typically a rodent (Niccole and Manceux 1908, Dubey 2008), but virtually any mammal or bird can serve as an intermediate host for the parasite (Dubey and Beattie 1988).

The lifecycle of *T. gondii* consists of several life stages. First, cats produce *T. gondii* oocysts in their feces (Dubey and Beattie 1988, Dubey 2009). These oocysts contain an infective form of the parasite called sporozoites. These feces are then typically consumed by the intermediate host – for example, a rat (Dubey et al. 1998, Dubey 2008, Dubey 2009). Within the rat, oocysts become tachyzoites, or pseudocysts, which then become bradyzoites, or cysts (Dubey et al. 1998, Dubey 2009). The rat containing one of the three infective stages of *T. gondii* (sporozoites, tachyzoites, or bradyzoites) is then consumed by the feline definitive host, completing the life cycle of the parasite (Dubey et al. 1998, Dubey 2009).

Infections in the intermediate host have harmful pathology. When *T. gondii* tachyzoites are present, the intermediate host's intestinal tissues become inflamed and cysts begin to form, especially in the brain (Dubey and Frenkel 1976, Dubey and Beattie 1988, Dubey et al. 1998). When the parasite encysts on the brain, behavioral modifications can occur; rats become sexually aroused by the smell of cat urine, a stench that normally creates repulsion and fear to keep rats safe from predation by felines (Vyas et al. 2007, House et al. 2011). This behavioral modification results in an increased likelihood of the rat being consumed by the feline definitive

host, because instead of staying far away from the smell of cat urine, the rat increases the likelihood that it will be eaten by a cat by going towards the smell (Vyas et al. 2007, House et al. 2011).

Although trophic transmission between cats and rats is the most typical and well documented form of toxoplasmosis, transmission to other intermediate hosts is also possible. When cat feces get washed away via storm water runoff, food and water sources can become contaminated with *T. gondii* (Dubey 2004, Dubey and Jones 2008). The sporulated oocysts are consumed by a variety of birds and mammals, including humans, in this way.

One of the many mammals that is commonly infected with toxoplasmosis is *Enhydra lutris nereis*, the southern sea otter (Cole et al. 2000, Dubey et al. 2003, Conrad et al. 2005). When cat feces are washed into waterways, the oocysts can be delivered into freshwater or marine environments (Miller et al. 2002, Dubey 2004, Fayer et al. 2004). Sea otters become contaminated with *T. gondii* from eating bivalves, which filter the oocysts out of water (Lindsay et al. 2001, Arkush et al. 2003, Miller et al. 2002). Sea otters are three times more likely to be infected with *T. gondii* if they were sampled near freshwater inflows than when sampled further away from these influxes (Miller et al. 2002). When sea otters become infected with *T. gondii*, they experience convulsions, neurological problems, behavioral changes, abortion, and even death (Miller et al. 2002). Additionally, behavioral modifications can occur, causing the sea otter to exhibit more risky behaviors that probably increase the probability of death via shark bite, as sea otters with *T. gondii* are 3.7 times more likely to be bitten (Kreuder et al. 2003).

Sea otters are an important animal to protect. The southern sea otter is listed as threatened according to the Environmental Protection Act (EPA; Doroff and Burdin 2015). Although their population has been relatively stable over the past few years, the population was only 2,941

otters in 2013, a small number compared to the 16,000 otters that existed before fur trading began (Doroff and Burdin 2015). Sea otters are also important to protect because they serve as a keystone predator in the marine ecosystem. Sea otters are predators of sea urchins, and without sea otters, sea urchin populations would dominate the ecosystem (Estes and Palmisano 1974). This phenomenon would lead to a depletion of kelp populations and would drastically change kelp bed ecosystem structure, affecting the provision of a variety of ecosystem services, such as shoreline buffering (Estes and Palmisano 1974). In addition to their ecological importance, sea otters hold an economic value. They are charismatic marine mammals that many people care about and willingly travel and pay to see, benefiting tourism industries (Loomis 2006).

Because sea otters are such an integral part of the marine ecosystem, control of *T. gondii* in otter populations is important. A study conducted by Miller et al. (2002) found that 42% of live sea otters and 62% of dead sea otters along the coast of California had *T. gondii* infections. A study by Kreuder et al. (2003) on necropsied southern sea otters in California between 1998 and 2001 found that 16.2% of sea otter deaths were primarily caused by *T. gondii*, and 13.3% died via trauma from a shark inflicted wound. Additionally, sea otter deaths by shark attack have been increasing in recent years and this is now the primary cause of mortality in southern sea otters (Doroff and Burdin 2015, Tinker et al. 2016).

Although desirable, complete elimination or eradication of toxoplasmosis is a nearly impossible task, because the disease is so widespread and is found in every mammal and bird on Earth (Dubey and Beattie 1988). However, little control has been implemented for sea otters. Additional control programs need to be deployed for toxoplasmosis in the southern sea otter, including an increase in monitoring programs, education of the public, vaccine development, and environmental protection.

General Steps Toward Control That Are Already in Place

One important step toward controlling a parasite is research. Through research and experimentation, much has been discovered about the host, life cycle, and pathology of *T. gondii*. Studies on the viability of oocysts have shown that oocysts remain viable in seawater for up to months at a time (Lindsay et al. 2003). Research has been done on detecting and identifying *T. gondii* molecularly in individual hosts (Su et al. 2010). This research is important for the control of *T. gondii* because understanding how the parasite interacts with both the environment and its host can inform policy and control efforts.

Another step towards general control of *T. gondii* that has been implemented is the start of vaccine development. A vaccine against *T. gondii* has been developed in mice (Yang et al. 2017). After vaccination, the number of new cysts in the mice decreased, meaning the vaccine can provide at least partial immunity for mice (Yang et al. 2017). Additionally, a vaccine has also been developed for pregnant sheep, by infecting the sheep with a strain of *T. gondii* that is living, but does not produce cysts (Buxton and Innes 1995, Dubey 2008). After receiving the vaccination, the sheep had a decrease in the number of deaths of their offspring; however, this vaccine is not available for commercial uses (Buxton and Innes 1995, Dubey 2008). Most relevantly, vaccines have been tested to prevent felines from shedding oocysts into the environment (Frenkel et al. 1991, Innes et al. 2009). These vaccines have been created using a few different strains and mutations of *T. gondii* and have overall proved to be effective in reducing transmission (Innes et al. 2009). However, the vaccines involve using a live form of the parasite, so they can be dangerous to administer, difficult to use on a bigger scale, and will not last long once prepared because the cysts can only stay living for so long (Innes et al. 2009).

Suggestions for Improved Control

Although steps have been made toward the general control of *T. gondii*, increased control is needed to protect southern sea otter populations specifically. A program should be implemented to control the parasite in sea otters, and that program should include an increase in monitoring, education programs for cat owners, vaccine development, and environmental protection.

Monitoring is an effective way to find disease hot spots and determine where control should be most heavily implemented. Sea otter monitoring programs already exist; these programs consist of responding to dead otters that wash up on beaches and performing a necropsy (Kreuder et al. 2003). This information can be used to determine how many sea otters are dying of *T. gondii* infection and where they are located. Tracking and monitoring sea otters is important because this information can be used to map which geographic and environmental areas are more heavily affected by *T. gondii* and where control should be implemented. Sea otter monitoring should also be paired with shellfish monitoring programs. Shellfish from various locations around the coast of California should be collected and analyzed for *T. gondii* oocysts (Putignani 2011). This information can also help map the prevalence of the disease and inform management decisions. Big cat monitoring programs should also be executed, as big cats such as pumas and bobcats have shown higher prevalence of *T. gondii* than domestic cats (Bevins et al. 2012), meaning they are contributing to the oocysts ending up in waterways. Increasing tracking and monitoring programs of these large felines can also aid in determining where control measures should be most strongly implemented, as areas with more wild, big cats are more likely to have higher *T. gondii* prevalence.

An increase in education on *T. gondii* and its effects is an important step in further control of the parasite. Cat owners may help to decrease the number of *T. gondii* oocysts in the ocean if they keep their domestic cats indoors. This practice will decrease the likelihood of their pet ingesting tachyzoites or bradyzoites by preying on animals such as rodents (Elmore et al. 2010). A simple way to implement an education campaign would be to provide informational brochures on *T. gondii* and its effects on sea otters at veterinary clinics, pet adoption facilities, and pet stores. Additionally, outreach through social media could inform cat owners of the danger to sea otters of allowing a cat outdoors. Education campaigns have been implemented in the past to encourage cat owners to keep their cats inside to protect bird diversity (Fiore and Sullivan 2000, Dauphiné and Cooper 2009). Cat owners who were educated on how their pet could be impacting wildlife were more motivated to take the proper steps to protect wildlife (Fiore and Sullivan 2000, Dauphiné and Cooper 2009). If cat owners become aware of the problem, they will be more likely to avoid behaviors that could increase *T. gondii* transmission.

Effort and money should be invested in further development of a *T. gondii* vaccine for felines. A vaccine exists (Frenkel et al. 1991, Innes et al. 2009), but it requires significant improvement before it can become marketable. The current vaccine is not ideal, since it contains a live form of the parasite (Innes et al. 2009), so further research should be done to develop a killed-parasite vaccine. If an improved vaccine became marketable, domestic cats could be immunized by their veterinarian to prevent oocysts shedding. Additionally, studies have shown that feral cats are more highly infected with *T. gondii* than are domestic cats (Levy and Crawford 2004), so feral cats should also be vaccinated. This vaccination could be combined with a capture and spay/neuter program, so that the overall population of feral cats would be reduced. If more time and money is put into vaccine development, toxoplasmosis in sea otters could be

greatly reduced, as a vaccine would result in fewer oocysts being shed into the environment by both domestic and feral cats.

Finally, environmental protection and restoration should be implemented to decrease *T. gondii* transmission to sea otters. Degradation of wetlands has been linked to an increase in *T. gondii*, with complete degradation of these ecosystems leading to an increase of over 6 orders of magnitude in *T. gondii* transport (Shapiro et al. 2010). By protecting wetlands from further degradation through conservation efforts, the overall transport of *T. gondii* can be greatly reduced, leading to fewer infections in sea otters. Seagrass beds should also be conserved, as they can be beneficial for reducing marine disease due to their bioremediation qualities (Lamb et al. 2017). Also, because storm water runoff is responsible for washing *T. gondii* oocytes into waterways, forested areas should be conserved. Forested areas absorb storm water as opposed to developed impermeable surfaces, which increase runoff and allow more oocysts to be washed into waterways (Shuster et al. 2005). A final form of environmental conservation that should be used is protecting and restoring abalone populations. Studies have shown that sea otters who eat more abalone are less likely to be infected with *T. gondii* (Johnson et al. 2009). By preventing abalone harvest in areas where *T. gondii* is prevalent and increasing management and protection of abalone species, sea otters will ingest fewer *T. gondii* oocysts, leading to decreased toxoplasmosis in sea otters.

Conclusion

Toxoplasma gondii is an extremely widespread disease with a large variety of hosts. Control of this parasite in the threatened *Enhydra lutris nereis*, southern sea otter, is important, as few control mechanisms exist, leaving the sea otter greatly affected. As marine mammals are

sentinels for the health of the environment and ecological risks for humans (Bossart 2006), it is very important to protect these organisms from deadly diseases such as toxoplasmosis. Through these four methods of control – monitoring, public outreach and education, vaccine development, and environmental protection – *T. gondii* can be better controlled in sea otters, leading to greater protection of this important species.

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