OVERVIEW

Toxoplasma gondii: How an Amazonian parasite became an Inuit health issue

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Abstract

Toxoplasma gondii is a protozoan parasite that originated in the Amazon. Felids (mammals in the cat family) are the only definitive hosts. These animals shed large numbers of infectious oocysts into the environment, which can subsequently infect many intermediate hosts, including birds, mammals and, possibly, fish. Human *T. gondii* seroprevalence is high in some parts of the Canadian Arctic and is associated with adverse health consequences among Inuit population. Since the range of felids does not extend to the Arctic, it is not immediately obvious how this parasite got from the Amazon to the Arctic. The objectives of this overview are to summarize the health impacts of *T. gondii* infection in Inuit in Canada's North and to consider how this infection could have reach them. This article reviews the prevalence of *T. gondii* infection in terrestrial and marine animals in the Canadian Arctic and discusses their potential role in the foodborne transmission of this parasite to humans. Two distribution factors seem plausible. First, felids in more southern habitats may release infectious oocysts into waterways. As these oocysts remain viable for months, they can be transported northward via rivers and ocean currents and could infect Arctic fish and eventually the marine mammals that prey on the fish. Second, migratory terrestrial and marine intermediate hosts may be responsible for carrying *T. gondii* tissue cysts to the Arctic, where they may then pass on the infection to carnivores. The most likely source of *T. gondii* in Inuit is from the consumption of traditionally-prepared country foods including meat and organs from intermediate hosts, which may be consumed raw. With climate change, northward migration of felids may increase the prevalence of *T. gondii* in Arctic wildlife.

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Introduction

Toxoplasma gondii infection in humans

Toxoplasma gondii is a protozoan parasite that can infect virtually all birds and mammals (1). Although this parasite originally evolved in the Amazon region of South America (2,3), it now infects an estimated two billion people worldwide, with foci of high prevalence in Latin America, Eastern/Central Europe, the Middle East and South-East Asia and Africa, and lower prevalence in many European countries and both Canada and the United States (4). Humans may become infected via three transmission routes:

- Ingestion of tissue cysts by eating fresh raw meat or organs of an infected intermediate host
- Ingestion of sporulated oocysts, which may persist for months or years in soil or water
- Congenitally, from mother to fetus, if a pregnant woman has acute toxoplasmosis (5)

During the initial infection phase of an intermediate host, including in humans, *T. gondii* replicates rapidly and spreads throughout the tissues, including the brain (acute toxoplasmosis). In humans, symptoms may be subtle, and otherwise healthy individuals may not notice that they have become infected. Eventually, parasite replication slows down, and the protozoa cluster together in tissue cysts (latent toxoplasmosis). People with latent toxoplasmosis who become immunocompromised may develop reactivated toxoplasmosis, in which the dormant parasites in the tissue cysts will start replicating again. This reactivation can cause severe flu-like symptoms, blurred vision or toxoplasmic encephalitis. Latent toxoplasmosis has also been linked to changes in cell signaling pathways that may lead to neurological disorders including schizophrenia, epilepsy, Alzheimer's disease and Parkinson's disease (6–11). Furthermore, a positive association has been made between *T. gondii* infection and increased risk-seeking behaviour in humans (12,13).

Congenital transmission may lead to stillbirth or severe neurological complications.

Socioeconomic factors may have a significant impact on human exposure to this parasite. Factors influencing the seroprevalence in humans include proximity to infected domesticated or wild reservoir hosts, access to clean drinking water, urban versus rural lifestyle, types of food consumed, food preparation (raw vs freezing/cooking/drying) and hygiene (washing hands and rinsing fresh produce) (14).

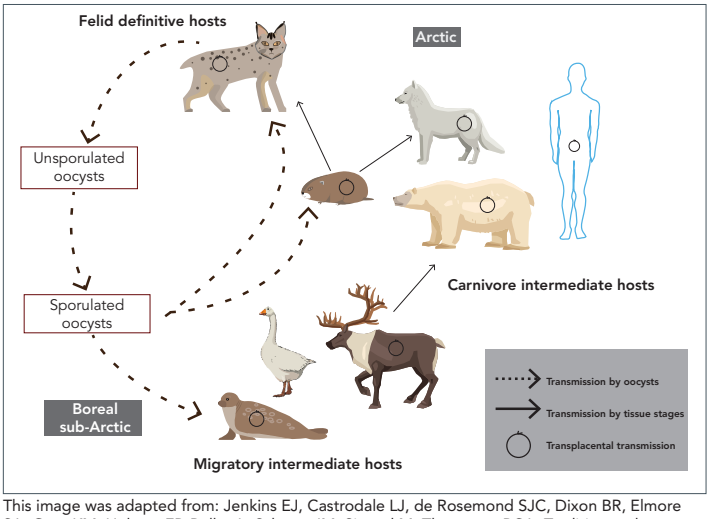
T. gondii from the Amazon to the Arctic

Toxoplasma gondii evolved in the Amazon rainforest (2,3). It is very common in the Amazon region and Indigenous populations of the Amazon River basin have the highest known infection rate worldwide: along the upper Rio Negro, *T. gondii* seroprevalence is greater than 90% (15). Despite its worldwide distribution, only in the Amazon is *T. gondii* characterized by a high level of genetic diversity and the presence of many unique genotypes (3). Analysis of the gene flow of unique genotypes indicated that a small number of ancestral lineages gave rise to the existing diversity of *T. gondii* (2). The primary hypothesis for the worldwide spread of *T. gondii* is that shipping traffic facilitated the travel of domestic cats and infected intermediate hosts to other continents (1). The parasite reproduces in the small intestine of the felid definitive hosts, and millions of oocysts are shed into the environment (5,14). How *T. gondii* spread from the Brazilian rainforest to the Canadian Arctic is not known. In this article, the Arctic boundaries are defined as described by the Conservation of Arctic Flora and Fauna (CAFF), which is the biodiversity group of the Arctic Council. The only wild felid that lives in the Canadian North is the Canada lynx, which has a *T. gondii* seroprevalence of 14% (16); however, the lynx's range does not extend north of the treeline (the boreal forest or subarctic). In addition, there are few domestic cats in Canadian Arctic communities. Thus, while the presence of infected felids may explain the spread of *T. gondii* throughout most of North America, it does not explain the parasite's presence in the Arctic; and, despite the scarcity of potentially infected felids, *T. gondii* is still present in a wide variety of Arctic animals.

To complete the parasite's lifecycle, oocysts that are shed by the felid definitive hosts need to sporulate (**Figure 1**) and be ingested by intermediate hosts, which are potential prey for felids and which include virtually all warm-blooded animals. *Toxoplasma gondii* invades the intermediate host's tissues and disseminates throughout the body, including the brain (1). However, intermediate hosts do not produce oocysts; thus, the mechanism (or mechanisms) of the geographical spread of *T. gondii*, in the absence of a definitive host, is still unknown.

The objective of this review is to highlight the incidence of this parasite in the Canadian Arctic and its impact on Inuit populations, and to consider how this parasite arrived and became endemic in an environment that lacks definitive hosts.

Figure 1: Lifecycle of a *Toxoplasma gondii* in Canada's North



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Toxoplasma gondii in the Arctic

T. gondii infection in Canadian Inuit

Toxoplasma gondii infections were first reported in Inuit in the 1980s (17–19). More recent studies showed that *T. gondii* seroprevalence in Inuit in the Canadian North varies greatly depending upon the region (17). *Toxoplasma gondii* seroprevalence in adults in three Canadian Inuit regions was reported at 8% in Nunatsiavut, 28% in Nunavut and 60% in Nunavik (20–24). There are not enough data to determine whether *T. gondii* prevalence in Inuit is stable or has changed over the decades.

Traditionally prepared "country foods" have great cultural significance for Inuit and in general are regarded as safe and nutritious for most people. However, it appears that *T. gondii* infection is related to the harvest and consumption of "country foods", especially meat and organs, which may be consumed raw (19,25). A correlation between *T. gondii* seroprevalence and different hunting practices and dietary habits has been debated (26–29). In contrast to Inuit communities, neighboring Cree communities, who usually cook their meat, were found to have a *T. gondii* seroprevalence of only 5% (29). It has been demonstrated that either thoroughly cooking meat, or freezing meat for several days, kills the pathogens present in the tissue cysts (30).

While toxoplasmosis is often asymptomatic in healthy individuals, pregnant women with acute toxoplasmosis are at risk of transmitting the parasite to the developing fetus. In 1987, an outbreak of toxoplasmosis was reported in pregnant women in Nunavik (19). Infection was associated with skinning of animals and consumption of raw caribou meat (19).

T. gondii in the absence of definitive hosts

A study from Svalbard, Norway suggested that the role of oocysts in the transmission of *T. gondii* to Arctic terrestrial animals has been overemphasized (31). The Svalbard archipelago is free from any wild or domestic cats, which eliminates the spread of infectious *T. gondii* oocysts into the environment (31). The absence of *T. gondii* oocysts in Svalbard is supported by findings that non-migratory birds and herbivores were seronegative for *T. gondii* (31). However, carnivores (foxes) were found to be *T. gondii*-positive. Thus, migrating birds may have introduced *T. gondii* to Svalbard, and local carnivores were subsequently infected by eating infected prey. Thus, it is possible for *T. gondii* to be transmitted from one intermediate host to another (e.g. bird to carnivore) without the need of sexual reproduction of the parasite in a felid definitive host. This transmission cycle between multiple intermediate hosts may explain the prevalence of *T. gondii* in the Arctic, including the Canadian Arctic, especially in non-felid carnivores. This hypothesis is supported by findings that all tested migratory birds and local carnivores in Svalbard were *T. gondii*-positive (31).

Canadian Arctic terrestrial animals

Regardless of the source of infection (environmental oocysts vs tissue cysts from infected prey), numerous mammals and birds in Canada's North have been reported to have tested positive for *T. gondii* (**Table 1**). Birds worldwide have been shown to be susceptible to *T. gondii* infection (31) and in Canada, migratory birds, such as geese, overwinter in areas where felids are common and where infectious *T. gondii* oocysts are likely to be found in high numbers in the environment (32–34). *Toxoplasma gondii* has been detected in the three tested geese species, with the highest prevalence reported in Ross's geese (34.5%) and the lowest in Canada geese (5.8%). Of the ptarmigan species tested, only one rock ptarmigan was found to be *T. gondii*-positive, possibly due to low exposure to oocysts in their arctic, subarctic and alpine tundra habitats.

Canadian Arctic rodents and lagomorphs showed no prevalence for *T. gondii*. Nearctic brown lemmings were negative, as were Arctic hares and snowshoe hares (Table 1). The only route of *T. gondii* transmission for non-migratory herbivores would be via ingestion of soil, plants or water contaminated with infectious oocysts. The absence of *T. gondii* prevalence in rodents and lagomorphs in the Canadian Arctic support the hypothesis that non-migratory Arctic herbivores have little to no exposure to infectious *T. gondii* oocysts (31).

The *T. gondii* exposure of ungulates varied between species. Caribou had a *T. gondii* prevalence of 11.3%, while the subspecies barren-ground caribou had a prevalence of 36.8%. It is unclear why barren-ground caribou were found to have such a high *T. gondii* prevalence. Muskox had a *T. gondii* prevalence of only 4.6% (Table 1).

Table 1: Birds and terrestrial mammals that have been tested for *Toxoplasma gondii* in the Canadian Arctic[a]

Common name (References)	Latin name	Number tested	Number positive	Percent positive
Birds				
Rock ptarmigan (35)	*Lagopus muta*	25	1	4.0%
Willow ptarmigan (35)	*Lagopus lagopus*	24	0	0.0%
Ross's goose (36,37)	*Chen rossii*	357	123	34.5%
Lesser snow goose (36,37)	*Chen caerulescens*	354	110	31.1%
Canada goose (35,38)	*Branta canadensis*	240	14	5.8%
Mammals				
Rodents				
Nearctic brown lemming (37)	*Lemmus trimucronatus*	84	0	0.0%
Lagomorphs				
Snowshoe hare (35)	*Lepus americanus*	8	0	0.0%
Arctic hare (35)	*Lepus arcticus*	2	0	0.0%
Ungulates				
Barren-ground caribou (39)	*Rangifer tarandus groenlandicus*	117	43	36.8%
Caribou (35)	*Rangifer tarandus*	97	11	11.3%
Muskox (35,40)	*Ovibus moschatus*	348	16	4.6%
Carnivores				
Arctic fox (41)	*Vulpes lagopus*	39	17	43.6%
Canada lynx (16,35)	*Lynx canadensis*	173	44	25.4%
Wolverine (42)	*Gulo gulo*	41	17	41.5%
Grey wolf (35)	*Canis lupus*	37	7	18.9%
Black bear (35,43)	*Ursus americanus*	43	16	37.2%

[a] including seasonally Arctic animals

The prevalence of *T. gondii* in carnivores was high in all species tested, as is to be expected even if the parasite's prevalence in their prey is relatively low. In Canada, *T. gondii* prevalence was found to be 43.6% in Arctic foxes, 25.4% in Canada lynxes, 41.5% in wolverines, 18.9% in grey wolves and 37.2% in black bears (Table 1).

Canadian Arctic marine mammals

Most pinnipeds in the Canadian Arctic were positive for *T. gondii*, including harbour seals (16.4%), ringed seals (10.7%), bearded seals (10.0%), hooded seals (1.7%) and walrus (14.7%) (**Table 2**).

Toxoplasma gondii was not detected in harp seals and more research may be required to determine if different feeding habits protect them from exposure to infected prey.

Table 2: Marine mammals that have been tested for *Toxoplasma gondii* in the Canadian Arctic[a,b]

Common name (References)	Latin name	Number tested	Number positive	Percent positive
Pinnipeds				
Harbour seal (26)	*Phoca vitulina*	311	51	16.4%
Ringed seal (26,35)	*Phoca hispida*	896	96	10.7%
Harp seal (35,44)	*Phoca groenlandica*	113	0	0.0%
Bearded seal (26)	*Erignathus barbatus*	20	2	10.0%
Hooded seal (44)	*Cystophora cristata*	60	1	1.7%
Walrus (35)	*Odobenus rosmarus*	34	5	14.7%
Bears				
Polar Bear (35,44–47)	*Ursus maritimus*	599	67	11.2%
Cetaceans				
Beluga (35,48)	*Delphinapterus leucas*	69	13	18.8%
Bowhead whale (35)	*Balaena mysticetus*	2	1	50.0%

[a] including seasonally Arctic animals
[b] including Amundsen Gulf, the Gulf of St. Lawrence, Hudson Bay, Labrador Sea and Beaufort Sea

Polar bears are the only ursines that are considered to be marine mammals because of their dependency on the ocean for food and habitat. *Toxoplasma gondii* has been detected in polar bears on the Canadian mainland and the Beaufort Sea, with an overall prevalence of 11.2%.

Two Arctic cetacean species have been tested for *T. gondii*: belugas and bowhead whales (Table 2). *Toxoplasma gondii* prevalence in belugas in the western Canadian Arctic was found to be 18.8% (Table 2). Of the two bowhead whales tested, one animal was *T. gondii*. gondii-positive (35).

T.gondii in Arctic waters

Toxoplasma gondii DNA has been detected in up to 77% of samples of treated and untreated surface water and well water worldwide (49,50). In some regions of Canada, increased rainfall has been associated with elevated numbers of *T. gondii* oocysts in surface waters (51). Most of Canada's rivers flow northward; 39% of Canada's freshwater drains into Hudson Bay and 36% drains into the Arctic Ocean (52). Oocysts that are washed into seawater are known to remain infective for up to two years and may be disseminated with the ocean currents (20,53–55).

It has been hypothesized that fish could be the missing link between oocysts that end up in the watersheds and infection in marine mammals (56). *Toxoplasma gondii* oocysts have been found in the alimentary tract of a wild fish (57) and it was shown that oocysts can remain infectious inside a fish's alimentary tract for several hours (58), thereby providing a possible source of infection for apex predators. To date, experimental infection of fish with *T. gondii* tissue cysts has only been reported in zebrafish and only under tightly controlled conditions (57). *Toxoplasma gondii* has also been reported in a variety of shellfish worldwide (59), and this may provide another source of infection in marine mammals and humans, although this has not yet been documented and confirmed in the Arctic.

To determine if Arctic fish are a potential source of *T. gondii*, we tested muscle tissues of 121 freshwater and euryhaline fish from Nunavik for the presence of *T. gondii* DNA. Fifteen fish (12.4%) tested positive for *T. gondii* using polymerase chain reaction for DNA amplification, followed by Sanger sequencing. Atlantic salmon and Arctic char had a *T. gondii* prevalence of 26.7% and 12.0%, respectively. Other fish species that tested positive for *T. gondii* DNA were lake trout (2.9%) and brook trout (16.7%). *Toxoplasma gondii* was detected in one sculpin (n=1) but it was not found in pike or lake whitefish, possibly due to low sample size (n=2 and 6, respectively) (*Reiling SJ, Boone R, Merks H, Dixon BR. Unpublished data, 2018*). While these are preliminary findings, more fish from the Canadian Arctic are currently being analyzed in our laboratory for the presence of *T. gondii*.

Discussion

There are a number of mechanisms by which *T. gondii* may have been introduced into the Canadian Arctic. *Toxoplasma gondii* may have been introduced via migratory birds and mammals that became infected by ingestion of oocysts (which may persist in soil and water in geographical regions where felids are present), or infected prey, in their southern habitats and carried the infection with them to the North. The parasite could then be transmitted from one intermediate host to another in the Arctic, even in the absence of definitive hosts. In addition, predators, such as Arctic foxes, wolverines and grey wolves, showed high *T. gondii* prevalence, suggesting that carnivory may also be an important route of transmission in the Arctic. Oocysts shed by felids in the south and transported northwards through waterways may be another source of infection in aquatic animals in the Arctic. Until recently, fish had not been known as a potential source for *T. gondii* infection. However, our preliminary findings suggest that *T. gondii* may be present in fish in the Canadian Arctic and could be another source of infection in humans and fish-eating mammals.

Environmental factors that increase *T. gondii* prevalence in animals that are hunted by Inuit for subsistence may pose a growing health threat to Inuit in the Arctic regions of Canada. More research is needed to determine how environmental and socioeconomic changes influence *T. gondii* prevalence in animals and humans in the Canadian Arctic.

Climate change and warmer temperatures may promote forest growth in regions that were previously too cold (60–62). The increasing forest cover could expand the habitat of wild felids, thereby augmenting the release of *T. gondii* oocysts into the environment (20). Higher numbers of oocysts combined with warming temperatures may increase the potential for infection of intermediate hosts, including birds and mammals not yet known to be hosts for *T. gondii* in the Canadian Arctic. This, in turn, may open up new transmission routes to humans who eat traditionally prepared country foods.

Conclusion

Toxoplasmosis has now spread throughout much of North and South America primarily through felids. Despite the absence of felids, *T. gondii* has now extended into Canada's Arctic, and has posed a health risk to Inuit, especially in pregnant women and those with weakened immune systems. The most likely source of *T. gondii* infection in Inuit is through infected intermediate hosts and the consumption of traditionally prepared country foods including meat and organs which may be consumed raw. Preventing infection by cooking or thoroughly freezing fish, meat, and organs and a better understanding of ongoing zoonotic transmission patterns will help to address this risk.

Authors' statement

SJR collected and analyzed the data. SJR and BRD wrote, proofread and approved the manuscript.

Conflict of interest

None.

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