Old problems on a new playing field: Helminth zoonoses transmitted among dogs, wildlife, and people in a changing northern climate

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ABSTRACT

Although surveillance is limited, indigenous residents at latitudes ranging from 53 to 73°N in Canada appear to have a higher occurrence of infection with some zoonotic parasites than the general population. Conversely, they are relatively naïve to other zoonotic parasites that have previously been unable to establish at northern latitudes. For those parasites that circulate among dogs, wildlife, and people, potential risk factors in the North include limited availability of veterinary services, presence of free-roaming dog populations, and consumption of locally harvested fish and wildlife. These regions are also experiencing some of the greatest impacts of climate change in North America, including increased temperature, precipitation, and frequency and severity of extreme weather. We review the current taxonomy, genetic diversity, host and geographic distributions, epidemiology and risk factors for 3 genera of helminths (*Diphyllobothrium* spp., *Echinococcus* spp., and *Toxocara* sp.) in Canada's North in order to identify climate-sensitive aspects of their ecology. Free-living stages of parasitic zoonoses endemic in the Arctic (such as *Diphyllobothrium dendriticum*, the cervid strain of *Echinococcus granulosus*, and Arctic strains of *Echinococcus multilocularis*) will experience trade-offs between enhanced survival under wetter conditions and increased mortality under warmer conditions. Climate change might also lead to the introduction and establishment in the Arctic of parasitic zoonoses previously restricted to the sub-Arctic, such as *Diphyllobothrium latum*, *Toxocara canis*, and the prairie strain of *E. multilocularis*. Molecular techniques applied in broad geographic surveys are needed to address critical knowledge gaps in the geographic distribution, genetic diversity, and public health significance of zoonotic helminths already in the circumpolar North, and to determine the current barriers to range expansion of temperate-adapted parasites into the North. Dogs will continue to play important roles in the North, including that of a "bridging" host between sylvatic cycles and human communities. In a warming north, increased opportunities for business, agriculture, and tourism favor importation of dogs and their parasites into a newly suitable environment. Collaborations among veterinarians, public health personnel, and policy-makers are needed to enhance surveillance and mitigate for dog-transmitted parasitic zoonoses in a changing North.

1. Introduction

Parasitic diseases in the North remain a challenge for veterinary medicine and public health, even in developed nations such as Canada. In Canada, the North is functionally defined as the area north of the southern limit of the discontinuous permafrost zone, which begins at 53°N in some areas (Fig. 1). This includes the three northern territories and roughly the northern half of the non-Maritime provinces. The term "Arctic" is reserved here for latitudes greater than 60°N. Despite recognition of the problem for at least half a century, indigenous and northern peoples

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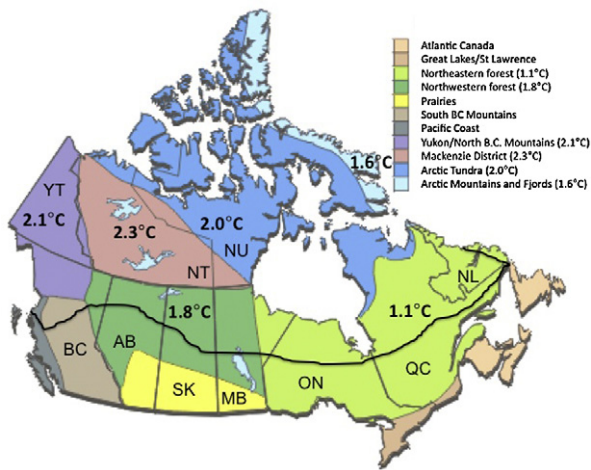 *E-mail address:* Emily.jenkins@usask.ca (E.J. Jenkins).

Fig. 1. Environment Canada climate regions (Climate Trends and Variations Bulletin) showing warming trends in mean annual temperature in the Canadian North over the last 63 years (1948–2010). Black line roughly delineates the southern limit of discontinuous permafrost; areas north of this line are functionally defined as the Canadian North. Grey lines delineate provinces and territories including the Yukon Territory (YT), Northwest Territories (NT), Nunavut (NU), Newfoundland Labrador (NL), British Columbia (BC), Alberta (AB), Saskatchewan (SK), Manitoba (MB), Ontario (ON), and Quebec (QC).

remain disproportionately affected by parasitic zoonoses (Miller, 1953; Cameron and Choquette, 1963; Rausch, 1972; Gyorkos et al., 2003; Hotez, 2010; Gilbert et al., 2010). Socioeconomic factors account for many health disparities observed between indigenous residents and the general Canadian population. Zoonoses might play only a small role in these health disparities, but they likely act synergistically with other infectious and non-infectious diseases. For parasitic zoonoses, risk factors include lack of access to veterinary services, large free-ranging dog populations in close association with people, and consumption of harvested wildlife by both people and dogs (Rausch, 1972; Salb et al., 2008; Brook et al., 2010).

In the past, sled dogs were a critical means of transportation in the Canadian North, but this role has diminished over the last century. However, dogs continue to play important roles in the economy and culture of many northern and indigenous communities, including wildlife deterrence, security, and companionship (Choquette and Moynihan, 1964; Rausch, 1972; Brook et al., 2010). Dogs in these communities are often free ranging, subsist by scavenging and hunting wildlife, and are rarely, if ever, dewormed (Unruh et al., 1973; Salb et al., 2008; Himsworth et al., 2010). In recent studies in Saskatchewan (SK), 19–50% of fecal samples from dogs in northern and indigenous communities were positive for at least one helminth or protozoan parasite (J. Schurer and E. Jenkins, unpublished data), as compared with only 4.4% of dogs in an urban centre in SK (Gaunt and Carr, 2011). People, especially children, are exposed to zoonotic helminths such as *Toxocara canis* and *Echinococcus* spp. through accidental consumption of eggs from the feces of dogs. Both dogs and people are exposed to *Diphyllobothrium* through consumption of

infected fish, with dogs serving as animal sentinels of the risk of human exposure (Salb et al., 2008).

In recent times, the North has become a sentinel for the effects of climate change on transmission of parasites and other pathogens (Bradley et al., 2005; Jenkins et al., 2005b; Kutz et al., 2005, 2009b). Northwestern Canada has already experienced a warming trend of 1.8–2.3 °C (versus 1.6 °C nationally) in mean annual temperature over the last 63 years of data recorded by Environment Canada, with warming in the Northeast proceeding more slowly (Fig. 1) (http://www.ec.gc.ca/adsc-cmda/default.asp?lang=En&n=77842065-1). Precipitation has also increased in the Canadian Arctic over the past 63 years, with mean departures from normal ranging from +2 to +25% (Furgal and Prowse, 2008). In the future, median increases of approximately 2, 4, and 6 °C in mean annual temperature are projected for the Canadian Arctic in the 2020s, 2050s, and 2080s, respectively (based on 7 Global Climate Models and 7 Emissions Scenarios). Temperature increases will be greater in the western Arctic and in the fall and winter. Increases in precipitation of 5–8%, 10–20%, and 15–30% are projected in the 2020s, 2050s, and 2080s, respectively. Precipitation increases will be greater in the extreme north and in the winter (Furgal and Prowse, 2008). Despite this, depth and duration of snow cover, and correspondingly volume of spring run-off, might remain stable or even decline as a result of warmer temperatures for longer durations. Effects on regional hydrology are complex; however, permafrost melting will lead to greater amounts of groundwater, and marked changes in drainage and distribution of surface water (Furgal and Prowse, 2008). Finally, most climate scenarios for northern North America predict an increase in the frequency and severity of extreme weather events such as heavy rainfalls and storms (Delecluse, 2008).

Zoonotic helminths are vulnerable to the effects of climate change due to their complex ecology involving multiple hosts and transmission routes, and often periods of development and survival in the environment (Charron et al., 2008; Polley and Thompson, 2009). Climate change will have variable effects on parasites with different life history strategies, such as directly transmitted saprozoonoses (such as *T. canis*) and indirectly transmitted cyclozoonoses (such as *Echinococcus* and *Diphyllobothrium* spp.) that use at least one intermediate host species. Broad mechanisms by which climate change might affect parasite transmission include altered development, survival, and distribution of parasites in the environment or in poikilothermic intermediate hosts, and host (including human) distribution, abundance, and behaviour (Hoberg et al., 2008b). Direct and indirect effects of climate warming might exacerbate existing health disparities for inhabitants of remote regions (Kutz et al., 2009b; Hotez, 2010; Weaver et al., 2010). The objectives of this paper are to review the current status of parasitic helminths transmitted among dogs, wildlife, and people in the Canadian North, and to identify potential mechanisms by which climate change and other drivers might alter the complex ecology of these zoonoses. We will also identify knowledge gaps that need to be addressed before the net effects of climate change on these complex host-parasite systems can be accurately predicted.

2. *Diphyllobothrium* (broad or fish tapeworm)

2.1. Taxonomy/genetic diversity

Diphyllobothrium spp. are important fish-borne zoonoses worldwide, but our understanding of diversity and distribution of this genus is incomplete, especially in the Canadian North (Curtis and Bylund, 1991; Chai et al., 2005). Identifications reported in the literature are not always well supported by morphological or molecular characterization. The major species of public health importance in northern Canada appear to be *Diphyllobothrium latum*, *Diphyllobothrium dendriticum*, and *Diphyllobothrium ursi* (Lantis, 1981; Curtis and Bylund, 1991). Variability in morphology and morphometrics of *D. latum* in Canada as compared to Eurasia (Dick and Poole, 1985) requires further investigation using molecular characterization. Phylogenetic analyses are also critically needed to resolve controversy over the timing of establishment of *D. latum* in North America, which crossed the Beringian land bridge and/or more recently arrived with immigration from Europe (Dick et al., 2001).

Variability in morphology and genetics among populations of *D. dendriticum* within Canada has been reported (de Vos et al., 1990). The relationship of *D. dendriticum* with *D. ursi* is somewhat unclear and would also benefit from application of molecular techniques. Both have similar morphology (other than larger size of some features of adult *D. ursi*) and it is not possible to rule out host specific effects on morphology (Rausch, 1954; Andersen et al., 1987). *D. ursi* is known to be present in the northern Pacific, and recently *Diphyllobothrium nihonkaiense* has been detected in people who consumed Pacific salmon from northwestern Canada (Margolis et al., 1973; Ching, 1984; Yera et al., 2006; Arizono et al., 2009). A non-zoonotic species, *Diphyllobothrium ditremum*, has also been reported in salmonid fish in Arctic Canada, and often co-occurs with *D. dendriticum* (Andersen et al., 1987; Curtis et al., 1988; Curtis and Bylund, 1991). Morphologically distinct plerocercoids from char in the eastern Canadian Arctic consumed by people and dogs in feeding trials were likely *D. dendriticum* (which established in people and dogs) and *D. ditremum* (which did not establish in people or dogs) (Freeman and Jamieson, 1976). Further molecular work is needed to determine the diversity, ecology, and public health significance of *Diphyllobothrium* spp. in the Canadian North.

2.2. Life cycle/host and geographic distributions

Adult tapeworms of *Diphyllobothrium* spp. inhabit the small intestine of fish-eating vertebrates serving as definitive hosts. Eggs shed in feces hatch to release a coracidium, which is ingested by a copepod first intermediate host. Predatory fish acting as second intermediate hosts ingest an infected copepod and the parasite develops to a plerocercoid larval stage in the viscera or muscles of the fish host. Plerocercoids of *D. latum* are present in the musculature of freshwater fish such as pike, walleye, perch, and burbot, and the definitive hosts are people, dogs, and wild carnivores (Dick et al., 2001; Chai et al., 2005). Peo-ple are certainly an important host for this parasite, but it can be maintained in dog and wildlife reservoir hosts in their absence (Dick and Poole, 1985; Dick et al., 2001). Plerocercoids of *D. dendriticum* are primarily in the viscera of salmonids (char, salmon, trout, grayling, and whitefish), and the normal definitive hosts are fish-eating birds; how-ever, people, dogs, and wild canids also serve as definitive hosts (Curtis and Bylund, 1991). Plerocercoids of *D. ursi* are present in the viscera and those of *D. nihonkaiense* in the skeletal muscles of Pacific salmon, and definitive hosts are bears and occasionally people (Ching, 1984; Curtis and Bylund, 1991; Arizono et al., 2009; Scholz et al., 2009).

In Arctic regions of Canada, *D. dendriticum* is widely distributed, with *D. ursi* (and possibly *D. nihonkaiense*) restricted to the north Pacific region. The presence of *D. latum* in people or other definitive hosts in Arctic regions of Canada is not well documented. Reports of *Diphyllobothrium* in people are seldom identified to species level, and it is possible that many diagnoses in northern and Inuit Cana-dians assumed to be *D. latum* were in fact *D. dendriticum* (Dick et al., 2001; Scholz et al., 2009). Plerocercoids of *D. latum* have been reported in fish in the Northwest Territo-ries (NT), as well as in northern Alberta, Saskatchewan, and Manitoba (Ching, 1984; Dick and Poole, 1985; Dick et al., 2001). *D. latum* appears to be largely restricted to subarctic regions of North America, possibly due to the distribution of fish intermediate hosts and environmental tolerances of free-living stages (Freeman and Jamieson, 1976; Andersen et al., 1987; Curtis and Bylund, 1991).

2.3. Epidemiology and risk factors

Diphyllobothriosis was historically common among the Canadian Inuit (prevalence generally around 30%, as high as 83%) but appears to have declined in the North over the last 60 years (Arh, 1960; Cameron and Choquette, 1963; Freeman and Jamieson, 1976; Lantis, 1981; Curtis et al., 1988). Diphyllobothriosis is not reportable to pub-lic health authorities at the national level in Canada. In the Northwest Territories (NT), only 46 cases were reported from the NT and what is now Nunavut between 1969 and 1978, and no cases have been reported in the NT since the early 1990s (K. Kandola, Medical Officer of Health, personal communication). In Igloolik in the eastern Cana-dian Arctic, prevalence dropped from 33% (of 97 fecal samples examined) in 1949 to 4% (of 247 fecal samples examined) in 1970–1971 (Freeman and Jamieson, 1976). Overall, incidence appears to be decreasing in the general North American population, although in some regions of Canada and elsewhere it might be increasing due to chang-ing socioeconomic status and cultural preferences for raw fish (Ching, 1984; Dick et al., 2001; Chai et al., 2005).

While *D. latum* has been associated with pernicious ane-mia in people in Eurasia, other species of *Diphyllobothrium* cause only mild, transient gastrointestinal and hemato-logical signs; however, all species may cause considerable emotional distress in infected people (and presumably owners of infected dogs) (Curtis and Bylund, 1991; Arizono et al., 2009; Scholz et al., 2009). Duration of infection with *D. dendriticum* is generally only 4–6 months, with egg shed-ding in people peaking in late summer and fall (Curtis

et al., 1988; Curtis and Bylund, 1991). In addition to short, seasonal, and generally subclinical infection, factors hindering detection through laboratory submissions include reticence to report passing of tapeworms to medical personnel and to submit fecal samples for testing. As a result, dogs may act as excellent sentinels for the presence of *D. latum* and *D. dendriticum* across the Canadian North. In two communities in northern Quebec (Nunavik), only two of 87 people were positive, while 44% of 80 dogs were shedding eggs of *Diphyllobothrium* spp. (Curtis et al., 1988).

Risk factors for transmission to people and dogs in the Canadian North include the ingestion of undercooked fish (especially viscera for *D. dendriticum*), and possibly cold-smoked or salted fish. There are also demographic risk factors including occupation, age, and gender (Chai et al., 2005). Some protective behaviours include cooking to temperatures greater than 55 °C for 10–30 min and gutting fish immediately after harvest, thereby reducing the time available for plerocercoids of *D. dendriticum* to migrate from viscera to flesh (Curtis et al., 1988). Freezing at −18 °C for 24 h also kills plerocercoids, historically restricting the transmission of *Diphyllobothrium* to the summer months in the Canadian Arctic (Curtis and Bylund, 1991). Management of *D. latum* has been accomplished in some areas through treatment of people and improved sewage management (Curtis and Bylund, 1991). Management of other *Diphyllobothrium* spp. with wildlife reservoirs in northern regions is significantly more challenging.

2.4. Potential effects of climate change

Environmental change (such as El Niño events), management practices (such as protection of marine mammal hosts), and global translocation of fish and people have altered the distribution and abundance of *Diphyllobothrium* spp. worldwide (Chai et al., 2005). Climate change is likely to have marked effects on the distribution and abundance of freshwater fish intermediate hosts in North America, including the local extirpation (but not likely mass extinction) of Arctic-adapted species, and the northward movement of species currently limited to the sub-Arctic (Marcogliese, 2001; Furgal and Prowse, 2008). This, in combination with warmer temperatures, might allow *D. latum* (currently restricted to the sub-Arctic) to become better established in Arctic Canada. *D. latum* is more pathogenic, human-specific, and longer-lived in people than *D. dendriticum*. As well, protective knowledge and preparation techniques developed to prevent transmission of *D. dendriticum*, where the plerocercoids are primarily in the viscera, would not prevent transmission of *D. latum*, where the plerocercoids are primarily in the skeletal musculature.

Climate change might also alter the transmission and effects of *D. dendriticum*, currently endemic in the Canadian North, through effects on trophic interactions of hosts. Experimental manipulations have reduced the prevalence of *D. dendriticum* in the Arctic by eliminating gull definitive hosts and, in another study, providing fish with food other than copepods (reviewed in Curtis and Bylund, 1991). Under laboratory conditions, warmer temperatures accelerate embryonation and hatching of eggs, while decreasing survival of coracidia (Hilliard, 1960). Plerocercoids of *D.

dendriticum* within fish intermediate hosts are more active and grow larger at warmer temperatures, probably causing more pathology in the fish (reviewed in Marcogliese, 2001). Thus, at warmer temperatures, there will be a trade-off between accelerated development of parasite stages in the environment and poikilothermic intermediate hosts, and decreased survival of both free-living coracidia and stages within fish hosts.

Transmission of *D. dendriticum* relies on seasonal synchronicity between hatching of eggs and availability of copepod intermediate hosts (Marcogliese, 2001). Therefore, the life cycle might well be disrupted by climate change, especially if hatching is more influenced by photoperiod while copepod abundance is more tied to temperature (Hilliard, 1960). Transmission of *D. dendriticum* to people is highly seasonal in the Canadian North, in part due to freezing susceptibility of plerocercoids (Curtis et al., 1988). Therefore, climate change might extend the season of transmission, as fresh fish might be consumed by people or fed to dogs for a longer portion of the year. Finally, climate change might alter the location of traditional fishing grounds. If people transport fish for longer distances before processing, this could allow more time for migration of greater numbers of plerocercoids of *D. dendriticum* from the viscera to the musculature. Therefore, transmission of this cyclozoonoses will be influenced by climate change at multiple levels, not the least of which is human behaviour.

3. *Echinococcus granulosus*

3.1. Taxonomy/genetic diversity

E. granulosus is a species complex of at least 8 strains and 3 proposed species (Jenkins et al., 2005a). The cervid strain of *E. granulosus* is the only species endemic in Canada, and is comprised of the G8 and G10 genotypes. The cervid strain is considered distinct from other species and genotypes of *Echinococcus*, with unique biology, host specificity, development patterns, and pathogenicity (Thompson et al., 2006); however, recent phylogenetic analysis suggests that the G8 cervid strain may be grouped with the G6/camel and the G7/pig strains as a single species (Nakao et al., 2006). The cervid strain is thought to have reduced pathogenicity in people, especially indigenous North Americans (Rausch, 2003; McManus and Thompson, 2004). As compared to the domestic sheep or pastoral strain, the cervid strain is less likely to cause anaphylaxis or secondary seeding upon rupture, and might best be managed through a "watch and wait" approach (Lamy et al., 1993; Brunetti et al., 2010). However, such an approach relies on repeated medical imaging (Finlay and Speert, 1992), often unavailable in remote areas. Symptoms depend on cyst localization and can include chest pain, coughing, dyspnea, abdominal pain, and neurological deficits (Moore et al., 1994; Somily et al., 2005). Two severe cases of cystic hydatid disease of the liver in Alaska suggest that the G8 cervid strain might not be as benign as once thought (Castrodale et al., 2002; McManus et al., 2002). In 2008, a child from an indigenous community in northern Saskatchewan was diagnosed with a neural hydatid cyst, most likely the G10 cervid strain based

on genotyping of *E. granulosus* in dogs in the community (Himsworth et al., 2010).

Recent molecular work suggests that the two genotypes (G8 and G10) present in cervids in Canada are most appropriately unified and designated as *E. canadensis* (Nakao et al., 2006; Thompson et al., 2006). However, if these strains are maintained in sympatry in natural sylvatic cycles, as observed in captive elk in western Canada (Thompson et al., 2006), there might be some validity to their distinct status. It is possible that the G10 strain, established in Fennoscandia (Lavikainen et al., 2003), was introduced into Canada with the importation of reindeer from Siberia and Fennoscandia to the Northwest Territories in the early 20th century (reviewed in Rausch, 2003; Thompson et al., 2006). However, this strain is now present in Alberta, Saskatchewan, and Manitoba (Thompson et al., 2006; Himsworth et al., 2010), suggesting a rapid rate of spread (~3000 km in 80 years). The broad distribution of *E. granulosus* in cervids in Canada, the cryptic diversity demonstrated within this species complex, and the relatively few numbers of Canadian isolates that have been sequenced, indicate a need for morphological and genetic characterization of additional specimens.

3.2. Life cycle/host and geographic distributions

Adult tapeworms are present in the small intestine of definitive hosts, which are large carnivores such as wolves (*Canis lupus*), coyotes (*Canis latrans*) and domestic dogs (*C. lupus familiaris*) (Sweatman, 1952; Jones and Pybus, 2001). Eggs shed in feces of the carnivore are immediately infective and environmentally resistant. Temperature and humidity affect survival of eggs of *E. granulosus* but are not thought to limit the global distribution of this parasite, with eggs surviving temperatures ranging from −30 to +30 °C (Eckert and Deplazes, 2004; Mas-Coma et al., 2008). However, this does not take into account the possibility of local adaptation and genetic differences in the environmental tolerances of eggs of *E. granulosus*.

Intermediate hosts (including people) harbour discrete, unilocular hydatid cysts (generally in the lungs and liver). Cysts containing viable protoscolices are infective when consumed by the definitive host. The proportion of cysts that are fertile is likely influenced by host factors (i.e. species, immune status, and age), and intensity and chronicity of infection. People are generally considered atypical intermediate hosts and might not develop fertile cysts (Rausch, 2003). In the Canadian Arctic, primary sylvatic intermediate hosts are cervids such as caribou (*Rangifer tarandus*) and moose (*Alces alces*). Heavily infected moose appear to be at increased risk of predation by wolves (Joly and Messier, 2004), likely as a result of compromised lung function when escaping from these chase predators. In the Canadian sub-Arctic, wapiti (*Cervus elaphus* or *Cervus canadensis*) might also play a role in maintenance of this parasite; however, relatively few fertile cysts developed in experimentally infected red deer (*C. elaphus*) (Sweatman and Williams, 1963). Other wild ungulates, including deer (*Odocoileus* spp.) and muskoxen (*Ovibos moschatus*), occasionally harbour hydatid cysts, but likely represent spillover hosts when sympatric with moose or caribou (Sweatman, 1952; Miller, 1953; Gibbs and Tener, 1958; Jones and Pybus, 2001).

The cervid strain of *E. granulosus* is present across Canada except where wolves have been historically absent: i.e. the Maritime provinces (east of the St. Lawrence River), the island of Newfoundland, and some High Arctic islands (Sweatman, 1952; Miller, 1953). Interestingly, coyotes have invaded the island of Newfoundland in the last 15 years, where naïve moose and caribou populations are present; this represents an opportunity for range expansion of the cervid strain (H. Whitney, Chief Veterinary Officer, NL, personal communication). The distribution map of *E. granulosus* in Canada from Sweatman (1952), based on the overlap of the ranges of moose and wolves, lacked the eastern Canadian Arctic, where caribou fill the ecological role filled by moose elsewhere. A more recent distribution map corrects this by including the eastern Canadian Arctic, but also the Maritime provinces and Newfoundland (Eckert et al., 2001), which is not supported by the literature or personal communications with wildlife managers in these regions. Therefore, an updated distribution of the cervid strain in Canada, ideally incorporating genotype data (G8 and G10), is needed to better understand the origins and drivers of transmission of the cervid strain of *E. granulosus* in North America.

3.3. Epidemiology and risk factors

Cystic hydatid disease caused by *E. granulosus* has been recognized in Canada since 1882 (Table 1). Detection of hydatid cysts in Canadians increased upon implementation of routine chest radiographs for tuberculosis in the 1950s. This led to efforts to determine the prevalence of infection with *E. granulosus* with the Casoni skin test using antigen from reindeer in the northern Yukon; initial attempts using antigen from hydatid cysts in sheep from Australia had proven unsuccessful. In the 1950s, positive Casoni tests were detected in 9–43% of people in communities in the Yukon (145/333), BC (95/1098), northern AB (155/458), NT (100/659), and what is now NU (43/249) (Cameron, 1960). Of the cases of cystic hydatid disease reported in people in Canada in the 1950s, 139 of 141 were in indigenous people (Miller, 1953). Similarly, almost all recorded cases of cystic hydatid disease in Alaska (>300) have occurred in indigenous people (Rausch, 2003). With the passing of the sled dog as a primary means of transportation, interest in the disease (and presumably prevalence) in North America has decreased (Rausch, 2003), as has awareness in the medical community (Lamy et al., 1993).

In Canada, this disease is not nationally notifiable to public health so significance and trends are difficult to determine in the general population. Nonetheless, cystic hydatid disease remains endemic in this country, with northern and indigenous people disproportionately affected by this disease. Between 1991 and 2001, 41% of 42 hospitalized cases of cystic hydatid disease (suspected or confirmed) from northern Alberta and the Northwest Territories occurred in people self-identifying as aboriginal; only 3% of the population of Canada self-identified as aboriginal at this time (Somily et al., 2005). A recent Canada-wide study based on hospitalization data

Table 1

Published cases of autochthonous cystic hydatid disease in Canadians; abbreviations of provinces and territories as for Fig. 1.

Location	Dates	Number of cases	Reference
Central Canada	Prior to 1883	8–10[a]	Cameron (1960)
ON, MB, SK, AB, BC, YT, NT	1948–1955	At least 180	Meltzer et al. (1956) and Miller (1953)
MB and NW ON	1987–1997	17	Al Saghier et al. (2001)
Southern BC	1987–1991	5	Finlay and Speert (1992)
NT, Northern AB	1970–1992	14	Moore et al. (1994)
NT, Northern AB	1991–2001	22 (+20 possible)	Somily et al. (2005)
Canada (hospitalization)	2001–2005	108	Gilbert et al. (2010)
SK	2008	1	Himsworth et al. (2010)

[a] Some may not have been autochthonous.

demonstrated that the highest incidence (3.59 cases/1,000,000 people) and relative risk (4.9) occurred in people at latitudes exceeding 55°N, including a high proportion of indigenous people (Gilbert et al., 2010).

In people, serology (an Enzyme-Linked ImmunoSorbent Assay (ELISA) for immunoglobulin G) lacks both sensitivity and specificity for the cervid strain of *E. granulosus*, which is not immunogenic in the early and late stages of infection, and cross-reacts with other helminths (including *Echinococcus multilocularis*) (Finlay and Speert, 1992; Brunetti et al., 2010). IgG ELISA was positive in only 40–54% of radiologically confirmed cases of cystic hydatid disease (Finlay and Speert, 1992; Somily et al., 2005). Current serological tests (which may be followed by confirmatory immunoblot assay) employed in Canada are not optimized for the cervid strain. Only 9% of clinically suspected cases of hydatid disease in Canadians submitted to the National Reference Centre for Parasitology are seropositive (M. Ndao, Laboratory Director, personal communication). This may be due to the antigen source, which is likely the camel/G6 strain of *E. granulosus*, although recent phylogenetic analyses indicate that this may be closely related to the G8 cervid strain (Nakao et al., 2006).

Despite the low sensitivity, serological testing suggests that exposure to *Echinococcus* continues in northern and indigenous communities, with 2–4% of 264 residents in two communities in northern Quebec (Nunavik, 58°N and 62°N) seropositive in 1983–1986 (Curtis et al., 1988), and 8% of 917 Nunavik Inuit sampled in 2004 (Lévesque et al., 2007b). In an indigenous community in northern Saskatchewan (∼53°N), 11% of 106 residents were seropositive for *Echinococcus* in 2008 (Himsworth et al., 2010); however, only two residents had clinical lesions upon medical imaging (S. Skinner, Infectious Disease Specialist, personal communication). Potential risk factors in this community include subsistence hunting of cervids and large numbers of free-ranging, untreated dogs, which serve as an important "bridging host" between sylvatic cycles and people (Rausch, 2003; Himsworth et al., 2010). People ingest eggs of *Echinococcus* in contaminated food, soil, and surface water, but the relative significance of these routes of infection in Canada is not known. Eggs are immediately infective and adhere to the coats of dogs, so zoonotic transmission through direct contact is a possibility. Control of the cervid strain of cystic hydatid disease in the Canadian North is challenging due to its presence in wildlife reservoirs, continued reliance on subsistence hunting, large numbers of free-roaming dogs, and lack of veterinary services.

3.4. Potential effects of climate change

In eastern North America, the cervid strain appears to have a southern limit roughly corresponding to the Canadian/USA border (Eckert et al., 2001), which might reflect absence of intact predator-prey food webs in the USA, or environmental tolerances of eggs of the northern-adapted cervid strain. If the latter, warming temperatures might push the current southern distributional limit of the cervid strain further north. Increased precipitation in the north would favor survival of eggs of *E. granulosus* in currently moisture-limited environments, and raises the possibility of water-borne outbreaks if eggs are washed into drinking reservoirs. Excessively wet conditions, however, might reduce availability of eggs for ingestion by grazing intermediate hosts (Schwabe, 1984).

Populations of cervid intermediate hosts for *E. granulosus* are already being influenced by climate change. Barrenground and woodland caribou populations in Canada are declining as a result of "knock-on" effects of climate and landscape change (McLoughlin et al., 2003; Vors and Boyce, 2009). In the future, increased winter precipitation and frequency of extreme weather events will likely contribute to further declines in northern caribou populations (Miller and Gunn, 2003; Vors and Boyce, 2009). Prevalence of *E. granulosus* is directly linked to density of populations of intermediate hosts (Joly and Messier, 2004), and therefore decreased abundance and even local extirpation of caribou populations would reduce parasite transmission in Arctic regions. However, several cervid species in Canada are competent intermediate hosts for *E. granulosus*. Wapiti and moose moving further north in response to climate and habitat changes could fill the current role of caribou in the life cycle of *E. granulosus* at Arctic latitudes.

4. *E. multilocularis*

4.1. Taxonomy/genetic diversity

Genetic diversity across the Holarctic distribution of *E. multilocularis*, while not of the same magnitude as that observed in *E. granulosus*, has only recently been recognized at mitochondrial and microsatellite loci variable
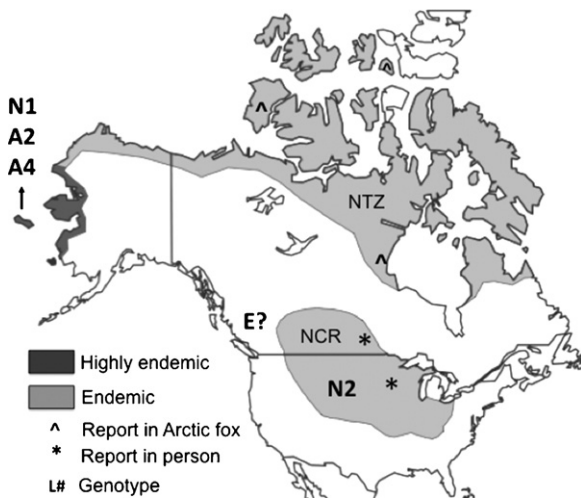
Fig. 2. Genotypes and distribution of *E. multilocularis* in North America, with the Northern Tundra Zone (NTZ) corresponding to the distribution of Arctic fox and the North Central Region (NCR) including the southern half of the 3 Canadian prairie provinces and 13 American states. 'N' denotes North American genotypes detected in the United States. 'A' denotes Asian genotypes detected on St. Lawrence Island, Alaska (Nakao et al., 2009). 'E?' denotes a European-type genotype detected in a dog with an alveolar hydatid cyst and no history of travel outside Canada (Peregrine et al., 2010). Map adapted from Eckert et al. (2001).

enough to detect strain differences (Casulli et al., 2009; Knapp et al., 2009; Nakao et al., 2009). Two North American mitochondrial haplotypes have been recognized on St. Lawrence Island, Alaska (N1) and in the central USA (N2) (Fig. 2). N2 haplotypes have also been identified in prairie Canada (E. Jenkins, K. Gesy, C. Somers, J. Hill, unpublished data). Molecular clocks suggest that the N1 and N2 haplotypes have been isolated for as long as 75,000 years (Nakao et al., 2009). Asian haplotypes (A2 and A4) are also present on St Lawrence Island (Nakao et al., 2009). These recent observations of genetic differences might account for observed differences in development of *E. multilocularis* from Alaska and Montana in intermediate hosts (Bartel et al., 1992). Haplotypes present on the mainland of Alaska and the Canadian Arctic have not yet been determined. Recently, a European-type strain (E? in Fig. 2) was detected in a dog in north-central British Columbia, not previously thought to be an endemic region (Peregrine et al., 2010).

4.2. Life cycle/host and geographic distribution

Definitive hosts for *E. multilocularis* in northern Canada include canids such as Arctic fox (*Vulpes lagopus*, previously *Alopex lagopus*), red fox (*Vulpes vulpes*), coyote, and domestic dogs. Felids also serve as (less suitable) definitive hosts, although domestic cats are not common in the Canadian north and lynx (*Lynx canadensis*) are not present north of the taiga (northern boreal forest). As for *E. granulosus*, eggs of *E. multilocularis* shed in carnivore feces are immediately infective and environmentally resistant; however, eggs of *E. multilocularis* are more susceptible to warm temperatures and dry conditions, restricting *E. multilocularis* to the northern hemisphere (Mas-Coma et al., 2008). Eggs from Germany lost infectivity within 2–4 h at temperatures

of 43–45 °C and at a relative humidity of 15% (Veit et al., 1995). These eggs were, however, freeze tolerant, surviving temperatures of −18 °C for 8 months, and eggs from Alaska remained infective even after exposure to temperatures as low as −51 °C (Schiller, 1955). The possibility of strain differences in environmental resistance of eggs of *E. multilocularis* requires further investigation.

Rodent intermediate hosts in the Canadian north include arvicoline and neotomine rodents, such as deer mice, voles and lemmings (Jones and Pybus, 2001). In intermediate hosts, multilocular alveolar hydatid cysts stemming from a germinal membrane often originate in the liver and can fill the entire abdominal cavity. People serve as aberrant intermediate hosts, in which protoscolices are generally absent. In people, the germinal membrane of *E. multilocularis* behaves like a metastatic tumor, invading the affected organ (often liver) and abdominal cavity. Treatment requires aggressive surgical resection and long-term chemotherapy, and success is strongly linked to early detection (Eckert and Deplazes, 2004). Interestingly, dogs also occasionally serve as intermediate hosts, harbouring fertile alveolar hydatid cysts, although this appears to be more common with European strains of *E. multilocularis* (Eckert and Deplazes, 2004; Peregrine et al., 2010).

The distribution of *E. multilocularis* in North America is divided into two distinct populations, corresponding to the Northern Tundra Zone (NTZ) and North Central Region (NCR) (the Canadian portion of the NCR in Fig. 2 corresponds to the "prairies" region in Fig. 1) (Schantz et al., 1995; Eckert et al., 2000). Insufficient density of suitable rodent intermediate hosts in the intervening boreal forest region (Northwestern Forest region in Fig. 1) might account for this disjunct distribution (Schantz et al., 1995). In the NCR, *E. multilocularis* is thought to be undergoing range expansion (Wilson et al., 1995; Storandt et al., 2002). The distribution of *E. multilocularis* in Arctic Canada (NTZ) is thought to correspond with that of the Arctic fox; however, actual reports are limited to a few morphological identifications, one on the mainland and two on high Arctic islands (Choquette et al., 1962; Eaton and Secord, 1979; Wilson et al., 1995) (Fig. 2). Further work is needed to characterize the distribution (based on confirmed molecular or morphological reports) and genetic diversity of *E. multilocularis* in Canada.

4.3. Epidemiology and risk factors

Historically, autochthonous human cases of alveolar hydatid disease in North America have been largely confined to focal outbreaks in western Alaska (the highly endemic region in Fig. 2). Only two autochthonous cases have been confirmed in the NCR region, in Manitoba, Canada, and Minnesota, USA (James and Boyd, 1937; Gamble et al., 1979) (* in Fig. 2). In general, zoonotic transmission in North America outside of western Alaska appears to be rare, especially compared to Europe, where 53 cases were confirmed in 2008 alone (European Food Safety Authority and European Centre for Disease Prevention and Control, 2010). It is not clear whether the absence of human cases in North America is due to the genotype(s) present, absence of risk factors for human

exposure, or simply misdiagnosis of the disease, which mimics hepatic carcinoma (Webster and Cameron, 1967; Hildreth et al., 2000). Inadequate surveillance may also play a role, as alveolar hydatid disease is not nationally notifiable to public health authorities in Canada and hospital records/coding do not always distinguish cystic from alveolar hydatid disease (Gilbert et al., 2010).

The presence of Asian genotypes, high rodent abundance, and cultural practices might account for enhanced zoonotic transmission of *E. multilocularis* in the highly endemic region in western Alaska. Prevalence was as high as 80% of 1579 Arctic fox on St. Lawrence Island, whereas only 4% of 94 Arctic fox on the mainland of Alaska were positive (Rausch, 1956; Rausch et al., 1990b). In Arctic fox in Canada, prevalence is also low, with only 1 infected Arctic fox of 50 examined from Banks Island (Eaton and Secord, 1979). Stability and abundance of rodent populations might account for greater prevalence and enhanced transmission of *E. multilocularis* on St. Lawrence Island, as compared to highly fluctuating rodent populations on the mainland (Rausch, 1956). In northwestern Alaska, risk factors include long-term pet (versus sled) dog ownership, tethering dogs near the house, and living in houses built directly on the tundra (Stehr-Green et al., 1988). In Alaska and in the central USA, close association with foxes did not increase risk of acquiring *E. multilocularis* (Hildreth et al., 2000).

4.4. Potential effects of climate change

E. multilocularis is the only cestode thus far identified as being susceptible to the effects of climate change, partly mediated through effects of temperature and moisture on the survival of eggs (Mas-Coma et al., 2008). In North America, climate change might have different effects on the survival of eggs of Arctic and prairie (N2) strains. Northern-adapted Arctic strains might disappear at the southern distributional limit due to increased egg mortality at warmer temperatures and following increased freeze–thaw cycles. In the NCR, the N2 strain of *E. multilocularis* (presumably tolerant of warmer temperatures) might further expand its range due to increased survival of eggs in wetter microhabitats.

Climate change will also influence transmission of *E. multilocularis* through effects on the abundance of rodent intermediate hosts. In temperate regions of North America, prevalence of *E. multilocularis* was higher at sites where deer mice were locally abundant (Holmes et al., 1971). On the one hand, climate change is predicted to increase precipitation overall, which might lead to increased stability and density of rodent populations and therefore facilitate transmission of *E. multilocularis*. On the other hand, climate change will also lead to increased frequency of severe weather events, increasing both amplitude and frequency of oscillations in rodent populations, and thereby decreasing overall transmission of *E. multilocularis*. Climate change is occurring concomitantly with other drivers of pathogen emergence, including landscape change and global translocation of hosts/pathogens. The fine-scale distribution and transmission ecology of *E. multilocularis* in Europe is highly sensitive to landscape variables, and is

favored by deforestation and agricultural practices that artificially increase rodent and fox populations (Viel et al., 1999; Giraudoux et al., 2004; Romig et al., 2006). Deforestation due to anthropogenic activities and climate warming might well open new habitat for neotomine rodents in North America. *E. multilocularis* recently established on Svalbard in the Norwegian Arctic following introduction of a suitable intermediate host, likely from shipping (Henttonen et al., 2001). Most recently, *E. multilocularis* was detected for the first time in red fox in Sweden, possibly as a result of movements of infected dogs (Osterman Lind et al., 2011). The source of the European strain detected in a dog in western Canada has not yet been identified, but might have occurred as a result of translocation of domestic dogs (Peregrine et al., 2010). Increased opportunities for introduction of infected rodents (through international shipping through the Northwest passage) and dogs (through increased tourism and agricultural development) will likely arise in a warming Canadian North.

Climate change will also result in changes in the distribution and abundance of sylvatic definitive hosts for *E. multilocularis* in Arctic Canada, namely red and Arctic fox. During the early 20th century, red fox moved north and successfully outcompeted Arctic fox for food and den sites (Hersteinsson and MacDonald, 1992). Arctic fox might be more suitable hosts for Arctic strain(s) of *E. multilocularis*, while red fox might be more suitable for the prairie strain. It is possible that mixing of the Arctic and prairie strains has already occurred; if not, this might well occur in a warming Arctic, where red fox will presumably continue to flourish. Recent detection of a European strain of *E. multilocularis* in an area of Canada where the Holarctic clade of red fox is established (Aubry et al., 2009) raises concerns that this strain will establish in native wildlife.

5. *T. canis*

5.1. Taxonomy/genetic diversity

Two morphologically and genetically distinct ascarids are present in dogs in northern Canada: *T. canis* and *Toxascaris leonina*. Of the two, only *T. canis* is traditionally considered a zoonosis. There is no evidence to support biological or genetic differences within *T. canis* in the Canadian North. However, morphological identifications of *T.* cf. *canis* elsewhere have proved to be new species of *Toxocara* upon molecular characterization (Gasser, 2006). Therefore, unrecognized genetic diversity might exist within the ascarid identified as *T. canis* across its broad distribution and host range.

5.2. Life cycle/host and geographic distributions

T. canis undergoes a direct life cycle, with canids (e.g. dog, fox, coyote, and wolf) as the normal definitive hosts. Canids become infected through consumption of embryonated eggs from the environment or larvae encysted in prey species serving as paratenic hosts. Within the canid, larval migration results either in establishment of patent infections in the intestinal tract, or somatic larvae that

reactivate in pregnant females and infect pups *in utero* or, less commonly, through the milk. Thus, both paratenic hosts and vertical transmission to some extent minimize the vulnerability of the life cycle of *T. canis* to environmental conditions. *Toxascaris leonina* also undergoes a direct life cycle (with or without paratenic hosts), although prenatal and transmammary transmission are not thought to occur, and larvae within the definitive host undergo only a simple mucosal migration.

Eggs of *T. canis* shed in feces of dogs are not immediately infective and undergo development in the environment at a rate that varies directly with temperature and humidity (Gamboa, 2005). Eggs are environmentally resistant; under laboratory conditions, 50% of fertilized eggs held at 4 °C in 2% formalin for 21 months could fully embryonate and were infective for mice (Chung et al., 2004). Survival is influenced by microhabitat and microclimate under field conditions, with survival decreasing at lower soil humidity and oxygenation (Mizgajska, 1997). Upper temperature tolerances for egg survival are not well defined, but eggs begin to deform and die at temperatures higher than 34–45 °C (Uga and Kataoka, 1995; Gamboa, 2005). At the lower end of the temperature range, embryonated egg viability decreased from 95% to 20–30% when eggs were held at freezing temperatures (−7 to −20 °C, mean −13 °C) for 15–30 days under laboratory conditions (O'Lorcain, 1995). In a similar experiment, embryonated eggs of *T. canis* held at −15 °C for 5 days were non-viable, while those of *T. leonina* displayed much greater freeze tolerance (Okoshi and Usui, 1968). This likely accounts for the differences observed in the northern limits of the distribution of *T. canis* and *T. leonina* in Canada.

T. canis is present at temperate and subarctic latitudes in northern Canada; eggs were present in 6% of 360 fecal samples from dogs at multiple sites in northwestern Canada at 53–55°N in 1971 (Unruh et al., 1973) (Fig. 3). It has historically been absent from latitudes north of 60°N in Canada, despite its presence elsewhere in the circumpolar north and in Alaska (Schantz, 1989). Adults of *T. canis* were not detected in necropsies of 50 Arctic fox on Banks Island (73°N) (Eaton and Secord, 1979) or in 14 dogs in Kuujjuaq (58°N) (Desrochers and Curtis, 1987). Eggs of *T. canis* were largely absent in dog feces sampled in the past 30–70 years across Canada at latitudes ranging from 55 to 70°N, although eggs of *T. canis* were not always distinguished from those of *T. leonina* (Cameron et al., 1940; Unruh et al., 1973). The absence of *T. canis* at Arctic latitudes is not likely due to lack of sampling effort, diagnostic test sensitivity, or suitable hosts, as eggs and adults of *T. leonina* have been detected in wild and domestic carnivore populations examined in the Canadian North at latitudes between 53 and 73°N (Cameron et al., 1940; Unruh et al., 1973; Eaton and Secord, 1979; Desrochers and Curtis, 1987). Interestingly, no ascarid eggs were detected in dogs sampled between 1933 and 1939 on the High Arctic Islands of Victoria, Ellesmere, Somerset, or the northern part of Baffin Island, suggesting that even *T. leonina* might have an as yet undetermined northern distributional limit (Cameron et al., 1940). Further work is needed to determine the effects of microclimate on the development and survival of eggs of *T. canis* and *T. leonina* under laboratory and field
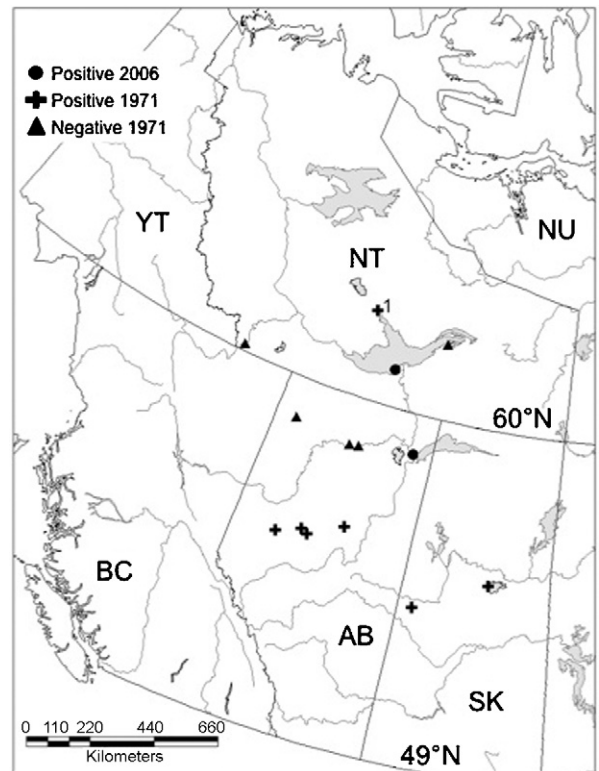

Fig. 3. Sampling for *Toxocara* eggs in dog feces in 1971 (Unruh et al., 1973) and in 2006 (Salb et al., 2008). Eggs of *T. canis* were not present in 598 dogs at locations ranging from 58 to 62°N in 1971 ([1] one positive of 143 dogs examined at this location in 1971), but were present at locations at 58 and 61°N in 2006. Abbreviations of provinces and territories as for Fig. 1.

conditions, and on the current host and geographic distributions of these ascarids in northern Canada.

5.3. Epidemiology and risk factors

In people, clinical syndromes associated with migrating or dormant larvae of *T. canis* include covert, ocular, and visceral larval migrans (Schantz, 1989; Despommier, 2003). Surveillance for this zoonosis in people in Canada is limited to periodic case reviews (often triggered by a clinical case) and serosurveys (Table 2), as larval migrans is not nationally notifiable to public health authorities. In temperate regions, rural residents appear to have higher seroprevalence than urban, and veterinary clinic personnel did not appear to be at higher risk than the general public (likely reflecting environmental transmission of eggs versus direct transmission from handling animals) (Yang et al., 1982; Embil et al., 1988). Risk factors in temperate and urban regions of Canada are likely similar to those found in the USA, which include age, ethnicity, socioeconomic status, education, pet ownership, and blood lead levels (as an indicator of pica) (Won et al., 2008). In 321 fecal samples collected from dogs in five indigenous communities in north-central SK in the past 2 years, prevalence of *T. canis* was 11% (J. Schurer and E. Jenkins, unpublished data), as compared to 0.2% of 457 owned dogs in 2009 and 3% of 132 shelter dogs in 1969 in Saskatoon, a nearby urban

Table 2
Published prevalence of *Toxocara* [seropositives unless indicated as visceral larval migrans (VLM) or ocular larval migrans (OLM)] in Canadians, in increasing order of latitude. Abbreviations of provinces and territories as for Fig. 1.

Location	Dates	Number of cases (overall prevalence)	Reference
Toronto, ON (43°N)	1952–1978	18 VLM; 3 OLM	Fanning et al. (1981)
Toronto, ON (43°N)	1970s	10/113 vet personnel 11/114 controls (9%)	Yang et al. (1982)
Halifax and rural Nova Scotia (44°N)	1982–1984	102/524 rural children 63/449 urban children (17%)	Embil et al. (1988)
Montreal, QC (45°N)	1977–1979	7/43 children OLM 5/80 adults OLM (10%)	Viens et al. (1981)
Northcentral QC (48–62°N)	1980s	44/436 Cree 83/759 Inuit (11%)	Tanner et al. (1987)
Mistissini, QC (50°N)	2005	2/48 Cree (4%)	Lévesque et al. (2007a)
James Bay, QC (52°N)	2007	8/250 Cree (3%)	Campagna (2009)
Nunavik, QC (55–62°N)	2004	37/917 adult Inuit (4%)	Lévesque et al. (2007b)

centre (Anvik et al., 1974; Gaunt and Carr, 2011). This might in part reflect a very young demographic of dogs in indigenous communities, as free-ranging dogs are periodically culled; the mean age of dogs brought to remote animal health clinics in these communities was 1–2 years. As these dogs are seldom treated, people in these indigenous communities in the Sub-Arctic likely have higher risk of human toxocariasis than those in urban or Arctic regions of Canada.

In large urban centres in Canada, seroprevalence for antibodies to *T. canis* in people ranged from 9 to 14%, similar to the prevalence of 14% reported in a national serosurvey in the USA (Won et al., 2008). Seroprevalence for *Toxocara* appears to be lower (3–4%) in indigenous people in northern communities in Canada (Table 2). This is consistent with a lower prevalence of *T. canis* in dogs and wild canids at more northerly latitudes. However, 7% and 20% of 264 people tested in two communities in Northern Quebec (58 and 62°N) were seropositive for *Toxocara* in 1983–1986 (Curtis et al., 1988). *T. canis* was not detected in fecal samples from 80 dogs nor in the intestines of 14 necropsied dogs in the more southern of these communities at this time (Desrochers and Curtis, 1987). *Toxocara cati* was likely not present as cats were (and remain) extremely rare in Inuit communities (Messier et al., 2009). It is possible that there was cross-reaction with other ascarids, such as *Toxascaris leonina*, which was present in both dogs and wild carnivores and might have greater zoonotic potential than previously thought.

5.4. Potential effects of climate change

While *T. canis* might not currently be a significant parasitic zoonosis in Arctic regions of Canada, climate change might already be altering its distribution in northwestern Canada (Fig. 3). Eggs of *T. canis* were absent in feces from 598/599 dogs sampled at latitudes north of 55°N in northwestern Canada in 1971 (Unruh et al., 1973). In 2006, sampling in the same region at latitudes from 58 to 61°N

revealed eggs of *T. canis* in 5% of 129 dogs (Salb et al., 2008). This might reflect a northward shift in distribution in response to lifting climate restraints; these regions have experienced a warming trend of 1.8–2.3 °C in mean annual temperature in the last 60 years (Fig. 1). As the climate continues to become warmer and wetter in the northwestern Arctic, it is likely that *T. canis* will continue to move north and establish in naïve wildlife, dog, and human populations. Mechanisms of introduction of *T. canis* include translocation of domestic dogs from the south to northern communities by transient employees working in oil and gas extraction, and natural movements of wildlife populations. Arctic fox disperse over long distances and the distribution of red fox appears to be expanding northwards in Canada (Hersteinsson and MacDonald, 1992; Dalen et al., 2005).

Once introduced, climate will likely determine the success of establishment of *T. canis* at Arctic latitudes. Under past climate conditions, prenatal transmission was not sufficient for maintenance of *T. canis* at Arctic latitudes, i.e. somatically infected females imported from the South did not go on to transmit *T. canis* to their pups in sufficient numbers to maintain *T. canis* in the absence of environmental egg transmission. Therefore, the limiting factor is likely the vulnerability of eggs of *T. canis* to freezing temperatures. In a warmer future, there will likely be trade-offs among accelerated embryonation of eggs, increased over-winter survival of eggs, and decreased survival of eggs in summer. In the Canadian Arctic, winter temperatures are predicted to warm more dramatically than summer temperatures, so the net effect will likely favor establishment and transmission of *T. canis* at higher latitudes.

6. Future of dog-transmitted parasitic zoonoses in the North

Climate, along with other drivers, is an important determinant of the distribution and abundance of parasitic zoonoses, through effects on parasite survival and

development, seasonal and spatial patterns of parasite transmission, as well as host distribution, abundance, resistance and behaviour. To make meaningful predictions, uncertainties about the magnitude and direction of climate change on a regional level and unknowns about the local ecology of parasitic zoonoses still need to be addressed. Nonetheless, through examination of three genera of zoonotic helminths, including one saprozoonosis and two cyclozoonoses, common themes can be detected. Climate change will alter the ecology of cyclozoonoses currently established in the Canadian Arctic, including *D. dendriticum*, Arctic strain(s) of *E. multilocularis*, and the cervid strain of *E. granulosus*. With climate change, these northern-adapted parasites might move farther north along with their hosts. Intermediate hosts will likely be more sensitive to the effects of climate change than wild and domestic carnivore definitive hosts, which have a broad distribution across Canada. In decreasing order of vulnerability, climate change will influence the ecology of the intermediate hosts of *D. dendriticum* (copepods and fish), *E. multilocularis* (rodents), and *E. granulosus* (cervids). However, all of these parasites are fairly non-specific and will likely be able to colonize new intermediate host species moving north and playing similar ecological roles. Therefore, these parasites will likely maintain most of their current distribution and possibly expand northwards.

Of the three parasites, the life cycle of *D. dendriticum* includes more stages vulnerable to the direct effects of climate change on development and survival (egg, coracidium, and stages within poikilothermic intermediate hosts). For this parasite, warming temperatures will likely generate a trade-off between accelerated development and decreased survival of free-living stages. In contrast, eggs are the only free-living stage of *Echinococcus* and do not undergo development in the environment. For eggs of *Echinococcus* spp., warming temperatures and increased number of freeze–thaw cycles will lead to decreased survival, but this might well be offset by enhanced survival under wetter conditions. Climate and landscape change will likely have greater impacts on *E. multilocularis* than *E. granulosus*, because of its focal distribution, decreased genetic plasticity, marked spatial and temporal variation in abundance of intermediate hosts, and increased vulnerability of eggs to warmer temperatures.

Climate change could lead to the introduction and establishment in the Arctic of parasitic zoonoses restricted to the sub-Arctic, such as *D. latum*, *T. canis*, and the prairie strain of *E. multilocularis*. For *D. latum*, current limitations most likely reflect distribution of suitable fish intermediate hosts as well as environmental tolerances of free-living stages, whereas for *T. canis*, the limitation is most likely due to susceptibility of the egg to freezing. The distribution of *T. canis* might already be shifting further north in western Canada under warming conditions in the last 40 years. The current barrier to northward expansion of the prairie strain of *E. multilocularis* appears to be the absence of suitable habitat for rodent intermediate hosts in the boreal region. This strain might shift north in response to changing climate and landscape conditions (natural or anthropogenic) that favor more stable and abundant rodent populations.

For all of these helminths, dogs serve as reservoir and/or amplifying hosts. For *Echinococcus* and *Toxocara* spp., dogs are the most important source of human infection, while for zoonotic *Diphyllobothrium* spp., dogs may amplify local transmission and serve as sentinels of the risk of human infection. It is likely that dogs will continue to play important roles in the North, including that of a "bridging" host between sylvatic cycles and human communities. In a warming north, increased opportunities for resource extraction, agriculture, and tourism might increase the importation of dogs from southern areas of Canada and elsewhere in the world. Current regulations for importation and translocation of pets do not address the risk posed by introduction of foreign strains or species of parasites, including zoonoses newly able to establish in a changing climate.

7. Control of dog-transmitted parasitic zoonoses in the North

With the exception of *T. canis* in prenatally infected pups, none of these helminth zoonoses have a significant impact on canine health. Therefore, motivation for control is almost entirely from public health, whereas means for control depend largely on veterinary medicine. Various measures have been used to prevent and control parasitic zoonoses transmitted among dogs, wildlife, and people. Where success has been observed, a combination of surveillance, regulation, education, and community involvement appear to be key (Schwabe, 1984; Kamiya, 2007). A cornerstone of management is anthelmintic treatment. For the helminths discussed in this review, highly effective drugs are available for treatment of dogs. In localized regions, anthelmintic baiting has reduced prevalence in wildlife (Kamiya, 2007). Generally, however, veterinary treatment efforts are focused on dogs (versus wildlife) as the most important source of human exposure to pathogens such as *T. canis* and *Echinococcus* spp. In a 10-year field trial with monthly deworming and strict confinement of dogs, the prevalence of *E. multilocularis* in voles (as an index of human risk) on St Lawrence Island was reduced from 53% to 5% (Rausch et al., 1990a). Although highly effective treatments are available, in practice, delivery of these treatments remains a challenge in the Canadian North, as many communities do not have access to veterinary services, including vaccination, sterilization, and anthelmintic treatment (Brook et al., 2010).

In two recent Canadian studies, less than a third of dogs in the Northwest Territories had been previously dewormed, and in an indigenous community in northern Saskatchewan, only 1 of 22 dog-owning households had ever dewormed their dog(s); this community was 80 km away from the nearest veterinary clinic (Brook et al., 2010; Himsworth et al., 2010). Mandates to control dog populations and to reduce zoonotic risks from companion animals in northern and indigenous communities in Canada are not well defined. Laws pertaining to dogs are largely community specific, and include strategies such as confining dogs, shooting dogs, and creating wildlife buffer zones; however, such efforts have not been effective over the long-term and are culturally unacceptable in some

Table 3
Assessment of 3 policies for control of dog-transmitted parasitic zoonoses in northern and indigenous communities in Canada, using weighted criteria technique.

Criteria for success	Weight (%)	Strategy 1 Remove dogs	Strategy 2 CAHWs[a]	Strategy 3 Remote vet services
Decrease human exposure to zoonotic parasites	**40**	Parasites shed in dog feces would decrease, but exposure to parasites shed by wild carnivores might increase. **25**	Reduction of parasites shed by dogs and consumption of parasites in wildlife by dogs and people. **30**	Current strategy in a few communities – not regular enough to reduce risks to people, no game inspection. **10**
Keep costs manageable	**20**	Implementation by local Environmental Officers. **20**	Training and CE[b] for CAHWs will incur costs. Communities can reduce costs by local diagnosis and bulk purchasing. **15**	Parasitological analyses by clinics or diagnostic laboratories and anthelmintic drug dispensing fees prohibitive for most owners. **10**
Keep compliance high	**20**	Compliance unlikely where dogs considered part of community. Compliance higher in communities with dog attacks. **5**	CAHWs are trusted as local stakeholders. Compliance high if services local and free to individuals. **20**	Some residents opposed to providing samples to outsiders or to self-identify as pet owners. **10**
Respect perceived rights	**10**	Right to equal treatment not respected if urban residents can own pets but northern and indigenous residents cannot. **0**	CAHWs are sensitive to local beliefs and traditions and can work within them. **10**	Some communities do not support interference with wildlife/pet populations. **5**
Legality	**10**	Band Councils are able to ban detrimental elements (e.g. alcohol). **10**	Legislation needed to avoid conflict with Veterinary Medical Associations. **0**	Potential conflict with Veterinary Medical Associations and nearby veterinary clinics. **5**
Total score	**100**	**60**	**75**	**40**

[a] Community Animal Health Workers.
[b] Continuing Education.

communities (Choquette and Moynihan, 1964; Brook et al., 2010). Educational programs can and have been delivered by regular public health personnel, but many experience high turnover in northern and indigenous communities and are unwilling or unable to handle animals to deliver preventative treatments. Similarly, strategies used in urban areas for dog population control (i.e. leash, license, and "poop and scoop" bylaws) and promotion of responsible pet ownership are often ineffective in northern and indigenous communities due to socioeconomic factors, cultural beliefs (which might not support concepts of ownership and interference with animals) and the role that free-roaming dogs play in home security and deterrence of wildlife such as bears and wolves.

Three management options for reducing risks of exposure of northern and indigenous residents to dog-transmitted parasitic zoonoses were considered as part of a weighted criteria policy analysis (Patton and Sawicki, 1986; Bardach, 2009). Essentially, decision criteria important in solving the problem were identified and weighted, and three policy alternatives were subjectively evaluated for each criterion (Table 3).

(1) Remove dogs from northern and indigenous communities.
(2) Train and support Community-based Animal Health Workers (CAHW) to deliver basic veterinary procedures and to inspect wild fish and game, with costs of parasite surveillance and treatment covered by communities or government agencies.
(3) Sporadic, remote delivery of veterinary services, with costs of parasite surveillance and treatment covered by individuals (existing model).

Of these, creation of Community-based Animal Health Workers (CAHW) programs taking a "herd health" approach was considered the best option, capitalizing on existing models in Canada such as the rabies lay-vaccinator program in the Northwest Territories and the Trichinellosis Prevention Program in Nunavik (Proulx et al., 2002; Brook et al., 2010). The World Organization for Animal Health has demonstrated success in the development of programs that support CAHWs, who are local residents given training in routine veterinary procedures, such as administration of vaccines and anthelmintics. Given appropriate equipment and training, CAHWs could perform diagnostic fecal flotations directly in northern and indigenous communities in Canada, allowing dog owners and community members to visualize parasites – a more powerful public health message than a written diagnostic report. CAHWs remain under the supervision of and report to veterinarians, enhancing disease surveillance and reporting from remote areas. Not only have these programs decreased animal morbidity and mortality, they have also decreased the risk of parasite transmission from animals to people, and increased willingness to pay for veterinary services (Catley et al., 2004).

8. Knowledge gaps and the way forward

At present, parasitic helminths are relatively rare in people in developed countries such as Canada. However, indigenous and northern residents are disproportionately affected by endemic helminth zoonoses, some of which have the potential to emerge as a result of climate and landscape change, and are naïve to another subset of zoonotic pathogens moving up from temperate regions. Challenges to understanding the effects of climate change and other drivers on the ecology of parasitic zoonoses in the North include the complexity of life cycles involving multiple routes of transmission and wildlife hosts. Basic information about northern wildlife is often lacking, making it difficult to predict how climate change might alter their ecology, behaviour, and resistance to infection and disease. For many parasitic zoonoses, the relative significance of food, water, and soil borne routes for transmission to people is unknown; it is likely that water-borne diseases will be particularly influenced by climate change (Charron et al., 2004). Since the life cycles of zoonoses also involve people, the effects of climate change on human behaviour, overall health, and globalization of agriculture and aquaculture relevant to the North need to be incorporated into risk analyses from a public health perspective. A critical first step is surveillance to determine the prevalence and significance of parasitic zoonoses in people in indigenous and northern communities, and cost-benefit analyses to determine the need for and feasibility of risk management for these zoonoses.

Application of molecular techniques holds great promise to further our understanding of the magnitude and significance of genetic diversity of parasitic zoonoses (Polley and Thompson, 2009), especially those previously considered to be genetically uniform across their circumpolar range (such as *E. multilocularis*). Veterinary and medical diagnoses rarely include genotyping necessary to determine zoonotic potential and public health significance, as the cost is difficult to justify on a case-by-case basis. Finally, molecular techniques applied in broad geographic surveys can be used to better define the northern distributional limits of parasitic zoonoses (i.e. for *D. latum* and *T. canis*). This, in combination with experimental work, can help determine current ecological and intrinsic barriers to range expansion into the Arctic.

Future experimental work on development and survival of free-living parasite stages will facilitate more accurate predictions of the net effects of climate change on the trade-off between parasite development and survival. There are few experimental models (and almost no recent studies) on development and survival of infective stages of zoonotic helminths. As well, determining the effects of temperature and humidity on viability of infective stages traditionally requires experimentally infecting an animal host, which poses ethical and logistical challenges. Rodent and domestic animal models are suitable for some of these zoonoses, but not, for example, for the cervid strain of *E. granulosus*. Models are useful for projecting the effects of climate change on host–parasite systems, but such models must be parameterized with real numbers, from laboratory and field-validated empirical studies (Jenkins et al., 2005b; Kutz et al., 2005; Hoberg et al., 2008a).

Relative to the physical and biological impacts of climate change in the North, little attention has been given to the effects on parasite ecology, despite evidence that it is already fundamentally altered across the Canadian North (Kutz et al., 2009a). While we have a growing understanding of how climate change is altering (or will alter) the ecology of parasites of wild ungulates in northern North America (Jenkins et al., 2005b; Kutz et al., 2005, 2009b; Hoberg et al., 2008b), parasitic zoonoses remain a more uncertain area (Bradley et al., 2005; Mas-Coma et al., 2008; Polley and Thompson, 2009). Understanding the effects of climate change on the ecology of zoonoses transmitted among wildlife, people, and domestic animals requires expertise in climatology, wildlife biology, ecology, parasitology, veterinary medicine, epidemiology of infectious disease, public health, and sociology. Research funding and regulatory mandates seldom encompass such a broad focus. Interdisciplinary teams are needed to explore these pressing issues, and indeed there is a growing movement bringing such diverse interests together to jointly address this challenge, especially as it pertains to human health (Parkinson and Berner, 2009). Collaboration at all levels is needed to determine the significance of current and emerging parasitic zoonoses in a changing North, and to mitigate the synergistic effects of these preventable infections in perpetuating disparities in the health of indigenous and northern peoples.

Conflict of interest

There are no known conflicts of interest.

References

Al Saghier, M., Taylor, M.C., Greenberg, H.M., 2001. Canadian-acquired hydatid disease: a case report. Can. J. Infect. Dis. Med. 12, 178–182.

Andersen, K., Ching, H.L., Vik, R., 1987. A review of freshwater species of *Diphyllobothrium* with redescriptions and the distribution of *D. dendriticum* (Nitzsch, 1824) and *D. ditremum* (Creplin, 1825) from North America. Can. J. Zool. 65, 2216–2228.

Anvik, J.O., Hague, A.E., Rahaman, A., 1974. A method of estimating urban dog populations and its application to the assessment of canine fecal pollution and endoparasitism in Saskatchewan. Can. Vet. J. 15, 219.

Arh, I., 1960. Fish tapeworm in Eskimos in the Port Harrison area, Canada. Can. J. Public Health 51, 268–271.

Arizono, N., Yamada, M., Nakamura-Uchiyama, F., Ohnishi, K., 2009. Diphyllobothriasis associated with eating raw pacific salmon. Emerg. Infect. Dis. 15, 866.

Aubry, K.B., Statham, M.J., Sacks, B.N., Perrine, J.D., Wisely, S.M., 2009. Phylogeography of the North American red fox: vicariance in Pleistocene forest refugia. Mol. Ecol. 18, 2668–2686.

Bardach, E., 2009. A Practical Guide for Policy Analysis: The Eightfold Path to More Effective Problem Solving. CQ Press, Washington, DC, USA.

Bartel, M.H., Seesee, F.M., Worley, D.E., 1992. Comparison of Montana and Alaska isolates of *Echinococcus multilocularis* in gerbils with observations on the cyst growth, hook characteristics, and host response. J. Parasitol. 78, 529–532.

Bradley, M.J., Kutz, S.J., Jenkins, E., O Hara, T.M., 2005. The potential impact of climate change on infectious diseases of Arctic fauna. Int. J. Circumpolar Health 64, 468–477.

Brook, R.K., Kutz, S., Millins, C., Veitch, A.M., Elkin, B.T., Leighton, F.A., 2010. Evaluation and delivery of domestic animal health services in remote communities in the Northwest Territories: a case study of status and needs. Can. Vet. J. 51, 1115–1122.

Brunetti, E., Kern, P., Vuitton, D.A., Writing Panel for the WHO-IWGE, 2010. Expert consensus for the diagnosis and treatment of cystic and alveolar echinococcosis in humans. Acta Trop. 114, 1–16.

Cameron, T.W.M., 1960. The incidence and diagnosis of hydatid cyst in Canada. *Echinococcus granulosus* var. *canadensis*. Parasitologia 2, 381–390.

Cameron, T.W.M., Choquette, L.P.E., 1963. Parasitological problems in high northern latitudes, with particular reference to Canada. Polar Rec. 11, 567–577.

Cameron, T.W.M., Parnell, I.W., Lyster, L.L., 1940. The helminth parasites of sledge-dogs in northern Canada and Newfoundland. Can. J. Res. 18, 325–332.

Campagna, S., 2009. Prevalence and environmental risk factors for ten zoonoses in two Cree communities of James Bay (Canada). MEnv, University of Sherbrooke, Longueuil, Quebec, Canada.

Castrodale, L.J., Beller, M., Wilson, J.F., Schantz, P.M., McManus, D.P., Zhang, L.H., Fallico, F.G., Sacco, F.D., 2002. Two atypical cases of cystic echinococcosis (*Echinococcus granulosus*) in Alaska, 1999. Am. J. Trop. Med. Hyg. 66, 325–327.

Casulli, A., Bart, J.M., Knapp, J., La Rosa, G., Dusher, G., Gottstein, B., Di Cerbo, A., Manfredi, M.T., Genchi, C., Piarroux, R., Pozio, E., 2009. Multi-locus microsatellite analysis supports the hypothesis of an autochthonous focus of *Echinococcus multilocularis* in northern Italy. Int. J. Parasitol. 39, 837–842.

Catley, A., Leyland, T., Mariner, J.C., Akabwai, D.M.O., Admassu, B., Asfaw, W., Bekele, G., Hassan, H.S., 2004. Para-veterinary professionals and the development of quality, self-sustaining community-based services. Rev. Sci. Tech. OIE 23, 225–252.

Chai, J.Y., Darwin Murrell, K., Lymbery, A.J., 2005. Fish-borne parasitic zoonoses: status and issues. Int. J. Parasitol. 35, 1233–1254.

Charron, D.F., Thomas, M.K., Waltner-Toews, D., Aramini, J.J., Edge, T., Kent, R.A., Maarouf, A.R., Wilson, J., 2004. Vulnerability of waterborne diseases to climate change in Canada: a review. J. Toxicol. Environ. Health A 67, 1667–1677.

Charron, D., Fleury, M., Lindsay, R., Ogden, N.H., Schuster, C., 2008. The impacts of climate change on water-, food-, vector-, and rodent-borne diseases. In: Seguin, J. (Ed.), Human Health in a Changing Climate: A Canadian Assessment of Vulnerabilities and Adaptive Capacity. Health Canada, Ottawa, Ontario, pp. 171–210.

Ching, H.L., 1984. Fish tapeworm infections (diphyllobothriasis) in Canada, particularly British Columbia. Can. Med. Assoc. J. 130, 1125–1127.

Choquette, L.P.E., Macpherson, A.H., Cousineau, J.G., 1962. Note on the occurrence of *Echinococcus multilocularis* Leuckart, 1863 in the arctic fox in Canada. Can. J. Zool. 40, 1167.

Choquette, L.P.E., Moynihan, W.A., 1964. Control of disease in dogs in the Canadian north. Can. Vet. J. 5, 262–267.

Chung, L.Y., Fang, B.H., Chang, J.H., Chye, S.M., Yen, C.M., 2004. The infectivity and antigenicity of *Toxocara canis* eggs can be retained after long-term preservation. Ann. Trop. Med. Parasitol. 98, 251–260.

Curtis, M.A., Bylund, G., 1991. Diphyllobothriasis: fish tapeworm disease in the circumpolar north. Arctic Med. Res. 50, 18–24.

Curtis, M.A., Rau, M.E., Tanner, C.E., Prichard, R.K., Faubert, G.M., Olpinski, S., Trudeau, C., 1988. Parasitic zoonoses in relation to fish and wildlife harvesting by Inuit communities in northern Quebec, Canada. Arctic Med. Res. 47, 693–696.

Dalen, L., Fuglei, E.V.A., Hersteinsson, P., Kapel, C.M.O., Roth, J.D., Samelius, G., Tannerfeldt, M., Angerbjorn, A., 2005. Population history and genetic structure of a circumpolar species: the arctic fox. Biol. J. Linn. Soc. 84, 79–89.

de Vos, T., Szalai, A.J., Dick, T.A., 1990. Genetic and morphological variability in a population of *Diphyllobothrium dendriticum* (Nitzsch, 1824). Syst. Parasitol. 16, 99–105.

Delecluse, P., 2008. The origin of climate changes. Rev. Sci. Tech. OIE 27, 309–317.

Despommier, D., 2003. Toxocariasis: clinical aspects, epidemiology, medical ecology, and molecular aspects. Clin. Microbiol. Rev. 16, 265–272.

Desrochers, F., Curtis, M.A., 1987. The occurrence of gastrointestinal helminths in dogs from Kuujjuaq (Fort Chimo), Quebec, Canada. Can. J. Public Health 78, 403–406.

Dick, T.A., Nelson, P.A., Choudhury, A., 2001. Diphyllobothriasis: update on human cases, foci, patterns and sources of human infections and future considerations. SE Asian J. Trop. Med. 32, 59–76.

Dick, T.A., Poole, B.C., 1985. Identification of *Diphyllobothrium dendriticum* and *Diphyllobothrium latum* from some freshwater fishes of central Canada. Can. J. Zool. 63, 196–201.

Eaton, R.D., Secord, D.C., 1979. Some intestinal parasites of Arctic fox, Banks Island, NWT. Can. J. Comp. Med. 43, 229–230.

Eckert, J., Conraths, F.J., Tackmann, K., 2000. Echinococcosis: an emerging or re-emerging zoonosis? Int. J. Parasitol. 30, 1283–1294.

Eckert, J., Schantz, P.M., Gasser, R.B., Torgerson, P.R., Bessonov, A.S., Movsessian, S.O., Thakur, A., Grimm, F., Nikogossian, M.A., 2001. Geographic distribution and prevalence. In: Eckert, J., Gemmell, M.A., Meslin, F.X., Pawlowski, Z.S. (Eds.), WHO/OIE Manual on Echinococcosis in Humans and Animals: A Public Health Problem of Global Concern. World Organisation for Animal Health (OIE) and World Health Organization (WHO), Paris, France, pp. 100–142.

Eckert, J., Deplazes, P., 2004. Biological, epidemiological, and clinical aspects of echinococcosis, a zoonosis of increasing concern. Clin. Microbiol. Rev. 17, 107–135.

Embil, J.A., Tanner, C.E., Pereira, L.H., Staudt, M., Morrison, E.G., Gualazzi, D.A., 1988. Seroepidemiologic survey of *Toxocara canis* infection in urban and rural children. Public Health 102, 129–133.

European Food Safety Authority and European Centre for Disease Prevention and Control, 2010. The Community Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Food-borne Outbreaks in the European Union in 2008. Parma, Italy, 288 pp.

Fanning, M., Hill, A., Langer, H.M., Keystone, J.S., 1981. Visceral larva migrans (toxocariasis) in Toronto. Can. Med. Assoc. J. 124, 21–26.

Finlay, J.C., Speert, D.P., 1992. Sylvatic hydatid disease in children: case reports and review of endemic *Echinococcus granulosus* infection in Canada and Alaska. Pediatr. Infect. Dis. J. 11, 322–326.

Freeman, R., Jamieson, J., 1976. Parasites of Eskimos at Igloolik and Hall Beach, Northwest Territories. In: Proceedings of the 3rd International Symposium on Circumpolar Health. U of Toronto Press, Toronto, Ontario, pp. 306–315.

Furgal, C., Prowse, T.D., 2008. Northern Canada. In: Lemmen, D.S., Warren, F.J., Lacroix, J., Bush, E. (Eds.), From Impacts to Adaptation: Canada in a Changing Climate. Government of Canada, Ottawa, Ontario, pp. 57–118.

Gamble, W.G., Segal, M., Schantz, P.M., Rausch, R.L., 1979. Alveolar hydatid disease in Minnesota: first human case acquired in the contiguous United States. J. Am. Med. Assoc. 241, 904–907.

Gamboa, M.I., 2005. Effects of temperature and humidity on the development of eggs of *Toxocara canis* under laboratory conditions. J. Helminthol. 79, 327–331.

Gasser, R.B., 2006. Molecular tools – advances, opportunities and prospects. Vet. Parasitol. 136, 69–89.

Gaunt, M.C., Carr, A.P., 2011. A survey of intestinal parasites in dogs from Saskatoon, Saskatchewan. Can. Vet. J. 52, 497–500.

Gibbs, H.C., Tener, J.S., 1958. On some helminth parasites collected from the musk ox (*Ovibos moschatus*) in the Thelon Game Sanctuary, Northwest Territories. Can. J. Zool. 36, 529–532.

Gilbert, N.L., Libman, M.D., Oluwayemisi, K.D., Muchaal, P.K., Ogden, N.H., 2010. Hospitalization for trichinellosis and echinococcosis in the Canadian Arctic, 2001–2005: the tip of the iceberg? Can. J. Public Health 101, 337–340.

Giraudoux, P., Craig, P.S., Delattre, P., Bao, G., Bartholomot, B., Harraga, S., Quere, J.P., Raoul, F., Wang, Y., Shi, D., Vuitton, D.A., 2004. Interactions between landscape changes and host communities can regulate *Echinococcus multilocularis* transmission. Parasitology 127, 121–131.

Gyorkos, T.W., MacLean, J.D., Serhir, B., Ward, B., 2003. Prevalence of parasites in Canada and Alaska. In: Akuffo, H., Linder, E., Ljungstrom, I., Wahlgren, M. (Eds.), Parasites of the Colder Climates. Taylor and Francis, New York, pp. 77–88.

Henttonen, H., Fuglei, E., Gower, C.N., Haukisalmi, V., Ims, R.A., Niemimaa, J., Yoccoz, N.G., 2001. *Echinococcus multilocularis* on Svalbard: introduction of an intermediate host has enabled the local life-cycle. Parasitology 123, 547–552.

Hersteinsson, P., MacDonald, D.W., 1992. Interspecific competition and the geographical distribution of red and arctic foxes *Vulpes vulpes* and *Alopex lagopus*. Oikos 64, 505–515.

Hildreth, M.B., Sriram, S., Gottstein, B., Wilson, M., Schantz, P.M., 2000. Failure to identify alveolar echinococcosis in trappers from South Dakota in spite of high prevalence of *Echinococcus multilocularis* in wild canids. J. Parasitol. 86, 75–77.

Hilliard, D.K., 1960. Studies on the helminth fauna of Alaska. XXXVIII. The taxonomic significance of eggs and coracidia of some diphyllobothriid cestodes. J. Parasitol. 46, 703–716.

Himsworth, C.G., Jenkins, E., Hill, J.E., Nsungu, M., Ndao, M., Andrew Thompson, R.C., Covacin, C., Ash, A., Wagner, B.A., McConnell, A., Leighton, F.A.L., Skinner, S., 2010. Emergence of sylvatic *Echinococcus granulosus* as a parasitic zoonosis of public health concern in an Indigenous community in Canada. Am. J. Trop. Med. Hyg. 82, 643–645.

Hoberg, E.P., Polley, L., Jenkins, E.J., Kutz, S.J., Veitch, A.M., Elkin, B.T., 2008a. Integrated approaches and empirical models for investigation of parasitic diseases in northern wildlife. Emerg. Infect. Dis. 14, 10–17.

Hoberg, E.P., Polley, L., Jenkins, E.J., Kutz, S.J., 2008b. Pathogens of domestic and free-ranging ungulates: global climate change in temperate to boreal latitudes across North America. Rev. Sci. Tech. OIE 27, 511–528.

Holmes, J.C., Mahrt, J.L., Samuel, W.M., 1971. The occurrence of *Echinococcus multilocularis* Leuckart, 1863 in Alberta. Can. J. Zool. 49, 575–576.

Hotez, P.J., 2010. Neglected infections of poverty among the indigenous peoples of the arctic. PLoS Neglect. Trop. D. 4, e606.

James, E., Boyd, W., 1937. *Echinococcus alveolaris* with the report of a case. Can. Med. Assoc. J. 36, 354–356.

Jenkins, D.J., Romig, T., Thompson, R.C.A., 2005a. Emergence/re-emergence of Echinococcus spp.—a global update. Int. J. Parasitol. 35, 1205–1219.

Jenkins, E.J., Veitch, A.M., Kutz, S.J., Hoberg, E., Polley, L., 2005b. Climate change and the epidemiology of protostrongylid nematodes in northern ecosystems: *Parelaphostrongylus odocoilei* and *Protostrongylus stilesi* in Dall's sheep (*Ovis d. dalli*). Parasitology 132, 387–401.

Joly, D.O., Messier, F., 2004. The distribution of *Echinococcus granulosus* in moose: evidence for parasite-induced vulnerability to predation by wolves? Oecologia 140, 586–590.

Jones, A., Pybus, M.J., 2001. Taeniasis and echinococcosis. In: Samuel, W.M., Pybus, M.J., Kocan, A.A. (Eds.), Parasitic Diseases of Wild Mammals. , 2nd ed. Iowa State University Press, Ames, Iowa, pp. 150–192.

Kamiya, M., 2007. Collaborative control initiatives targeting zoonotic agents of alveolar echinococcosis in the northern hemisphere. J. Vet. Sci. 8, 313–321.

Knapp, J., Bart, J.M., Maillard, S., Gottstein, B., Piarroux, R., 2009. The genomic Echinococcus microsatellite EmsB sequences: from a molecular marker to the epidemiological tool. Parasitology 137, 439–449.

Kutz, S.J., Dobson, A., Hoberg, E.P., 2009a. Where are the parasites? Science 326, 1187–1188.

Kutz, S.J., Jenkins, E.J., Veitch, A.M., Ducrocq, J., Polley, L., Elkin, B., Lair, S., 2009b. The Arctic as a model for anticipating, preventing, and mitigating climate change impacts on host–parasite interactions. Vet. Parasitol. 163, 217–228.

Kutz, S.J., Hoberg, E.P., Polley, L., Jenkins, E.J., 2005. Global warming is changing the dynamics of Arctic host–parasite systems. Proc. R. Soc. B: Biol. Sci. 272, 2571–2576.

Lamy, A.L., Cameron, B.H., LeBlanc, J.G., Gordon Culham, J.A., Blair, G.K., Taylor, G.P., 1993. Giant hydatid lung cysts in the Canadian northwest: outcome of conservative treatment in three children. J. Pediatr. Surg. 28, 1140–1143.

Lantis, M., 1981. Zoonotic diseases in the Canadian and Alaskan North. Inuit Studies 5, 83–106.

Lavikainen, A., Lehtinen, M.J., Meri, T., Hirvelä-Koski, V., Meri, S., 2003. Molecular genetic characterization of the Fennoscandian cervid strain, a new genotypic group (G10) of *Echinococcus granulosus*. Parasitology 127, 207–215.

Lévesque, B., Messier, V., Bonnier-Viger, Y., Couillard, M., Côté, S., Ward, B.J., Libman, M.D., Gingras, S., Dick, D., Dewailly, É., 2007a. Seroprevalence of zoonoses in a Cree community (Canada). Diagn. Microbiol. Infect. Dis. 59, 283–286.

Lévesque, B., Messier, V., Proulx, J., Ward, B.J., Libman, M.D., Serhir, B., 2007b. Seroprevalence of zoonoses in Nunavik (Canada). Epidemiology 18, doi:10.1097/01.ede.0000276514.49851.70.

Marcogliese, D.J., 2001. Implications of climate change for parasitism of animals in the aquatic environment. Can. J. Zool. 79, 1331–1352.

Margolis, L., Rausch, R.L., Robertson, E., 1973. *Diphyllobothrium ursi* from man in British Columbia—first report of this tapeworm in Canada. Can. J. Public Health 64, 588–589.

Mas-Coma, S., Valero, M.A., Bargues, M.D., 2008. Effects of climate change on animal and zoonotic helminthiases. Rev. Sci. Tech. OIE 27, 443–457.

McLoughlin, P.D., Dzus, E., Wynes, B., Boutin, S., 2003. Declines in populations of woodland caribou. J. Wildlife Manage. 67, 755–761.

McManus, D.P., Zhang, L., Castrodale, L.J., Le, T.H., Pearson, M., Blair, D., 2002. Short report: molecular genetic characterization of an unusually severe case of hydatid disease in Alaska caused by the cervid strain of *Echinococcus granulosus*. Am. J. Trop. Med. Hyg. 67, 296–298.

McManus, D.P., Thompson, R.C.A., 2004. Molecular epidemiology of cystic echinococcosis. Parasitology 127, 37–51.

Meltzer, H., Kovacs, L., Orford, T., Matas, M., 1956. Echinococcosis in North American Indians and Eskimos. Can. Med. Assoc. J. 75, 121–128.

Messier, V., Levesque, B., Proulx, J.F., Rochette, L., Libman, M.D., Ward, B.J., Serhir, B., Couillard, M., Ogden, N.H., Dewailly, E., Hubert, B., Dery, S., Barthe, C., Murphy, D., Dixon, B., 2009. Seroprevalence of *Toxoplasma gondii* among Nunavik Inuit (Canada). Zoonoses Public Health 56, 188–197.

Miller, F.L., Gunn, A., 2003. Catastrophic die-off of Peary caribou on the western queen Elizabeth Islands, Canadian High Arctic. Arctic 56, 381.

Miller, M.J., 1953. Hydatid infection in Canada. Can. Med. Assoc. J. 68, 423–434.

Mizgajska, H., 1997. The role of some environmental factors in the contamination of soil with *Toxocara* spp. and other geohelminth eggs. Parasitol. Int. 46, 67–72.

Moore, R.D., Urschel, J.D., Fraser, R.E., Nakai, S.S., Geeraert, A.J., 1994. Cystic hydatid lung disease in northwest Canada. Can. J. Surg. 37, 20–22.

Nakao, M., McManus, D.P., Schantz, P.M., Craig, P.S., Ito, A., 2006. A molecular phylogeny of the genus *Echinococcus* inferred from complete mitochondrial genomes. Parasitology 134, 713–722.

Nakao, M., Xiao, N., Okamoto, M., Yanagida, T., Sako, Y., Ito, A., 2009. Geographic pattern of genetic variation in the fox tapeworm *Echinococcus multilocularis*. Parasitol. Int. 58, 384–389.

O'Lorcain, P., 1995. The effects of freezing on the viability of *Toxocara canis* and *T. cati* embryonated eggs. J. Helminthol. 69, 169–171.

Okoshi, S., Usui, M., 1968. Experimental studies on *Toxascaris leonina*. IV. Development of eggs of three ascarids, *T. leonina*, *Toxocara canis* and *T. cati* in dogs and cats. Jpn. J. Vet. Sci. 30, 29–38.

Osterman Lind, E., Juremalm, M., Christensson, D., Widgren, S., Hallgren, G., Agren, E.O., Uhlhorn, H., Lindberg, A., Cedersmyg, M., Wahlstrom, H., 2011. First detection of *Echinococcus multilocularis* in Sweden, February to March 2011. Eurosurveillance 16.

Parkinson, A.J., Berner, J., 2009. Climate change and impacts on human health in the Arctic: an international workshop on emerging threats and the response of Arctic communities to climate change. Int. J. Circumpolar Health 68, 84–91.

Patton, C., Sawicki, D., 1986. Basic Methods of Policy Analysis and Planning. Prentice-Hall, New Jersey, 496 pp.

Peregrine, A.S., Jenkins, E.J., Barnes, B., Johnson, S., Hill, J., Polley, L., Barker, I.K., Gottstein, B., 2010. Hepatic alveolar echinococcosis in a dog in British Columbia, Canada. In: American Association of Veterinary Parasitologists Annual Meeting, Atlanta, GA.

Polley, L., Thompson, R.C., 2009. Parasite zoonoses and climate change: molecular tools for tracking shifting boundaries. Trends Parasitol. 25, 285–291.

Proulx, J.F., MacLean, J.D., Gyorkos, T.W., Leclair, D., Richter, A.K., Serhir, B., Forbes, L., Gajadhar, A.A., 2002. Novel prevention program for trichinellosis in Inuit communities. Clin. Infect. Dis. 34, 1508–1514.

Rausch, R., 1954. Studies on the helminth fauna of Alaska. XXI. Taxonomy, morphological variation, and ecology of *Diphyllobothrium ursi* n. sp. provis. on Kodiak Island. J. Parasitol. 40, 540–563.

Rausch, R., 1956. Studies on the helminth fauna of Alaska. XXX. The occurrence of *Echinococcus multilocularis* Leuckart, 1863, on the mainland of Alaska. Am. J. Trop. Med. Hyg. 5, 1086–1092.

Rausch, R.L., 1972. Tropical problems in the Arctic: infectious and parasitic diseases, a common denominator. Ind. Trop. Health 7, 63–70.

Rausch, R.L., 2003. Cystic echinococcosis in the Arctic and Sub-Arctic. Parasitology 127, 73–85.

Rausch, R.L., Wilson, J.F., Schantz, P.M., 1990a. A programme to reduce the risk of infection by *Echinococcus multilocularis*: the use of praziquantel to control the cestode in a village in the hyperendemic region of Alaska. Ann. Trop. Med. Parasitol. 84, 239–250.

Rausch, R.L., Fay, F.H., Williamson, F.S., 1990b. The ecology of *Echinococcus multilocularis* (Cestoda: Taeniidae) on St. Lawrence Island, Alaska. II. Helminth populations in the definitive host. Ann. Parasitol. Hum. Comp. 65, 131–140.

Romig, T., Thoma, D., Weible, A.K., 2006. *Echinococcus multilocularis*—a zoonosis of anthropogenic environments? J. Helminthol. 80, 207–212.

Salb, A.L., Barkema, H.W., Elkin, B.T., Thompson, R.C.A., Whiteside, D.P., Black, S.R., Dubey, J.P., Kutz, S.J., 2008. Dogs as sources and sentinels of parasites in humans and wildlife, northern Canada. Emerg. Infect. Dis. 14, 60–63.

Schantz, P.M., 1989. *Toxocara* larva migrans now. Am. J. Trop. Med. Hyg. 41, 21–34.

Schantz, P.M., Chai, J., Craig, P.S., Eckert, J., Jenkins, D.J., Macpherson, C.N.L., Thakur, A., 1995. Epidemiology and control of hydatid disease. In: Thompson, R.C.A., Lymbery, A.J. (Eds.), *Echinococcus* and Hydatid Disease. CAB International, Wallingford, UK, pp. 233–332.

Schiller, E.L., 1955. Studies on the helminth fauna of Alaska. XXVI. Some observations on the cold-resistance of eggs of *Echinococcus sibiricensis* Rausch and Schiller, 1954. J. Parasitol. 41, 578–582.

Scholz, T., Garcia, H.H., Kuchta, R., Wicht, B., 2009. Update on the human broad tapeworm (genus *Diphyllobothrium*), including clinical relevance. Clin. Microbiol. Rev. 22, 146–160.

Schwabe, C.W., 1984. Veterinary Medicine and Human Health, 3rd ed. Williams and Wilkins, Baltimore, MD, 680 pp.

Somily, A., Robinson, J.L., Miedzinski, L.J., Bhargava, R., Marrie, T.J., 2005. Echinococcal disease in Alberta, Canada: more than a calcified opacity. BMC Infect. Dis. 5, doi:10.1186/1471-2334-5-34.

Stehr-Green, J.K., Stehr-Green, P.A., Schantz, P.M., Wilson, J.F., Lanier, A., 1988. Risk factors for infection with *Echinococcus multilocularis* in Alaska. Am. J. Trop. Med. Hyg. 38, 380–385.

Storandt, S.T., Virchow, D.R., Dryden, M.W., Hygnstrom, S.E., Kazacos, K.R., 2002. Distribution and prevalence of *Echinococcus multilocularis* in wild predators in Nebraska, Kansas, and Wyoming. J. Parasitol. 88, 420–422.

Sweatman, G.K., 1952. Distribution and incidence of *Echinococcus granulosus* in man and other animals with special reference to Canada. Can. J. Public Health 43, 480–485.

Sweatman, G.K., Williams, R.J., 1963. Comparative studies on the biology and morphology of *Echinococcus granulosus* from domestic livestock, moose and reindeer. Parasitology 53, 339–390.

Tanner, C.E., Staudt, M., Adamowski, R., Lussier, M., Bertrand, S., Prichard, R.K., 1987. Seroepidemiological study for five different zoonotic parasites in northern Quebec. Can. J. Public Health 78, 262.

Thompson, R.C.A., Boxell, A.C., Ralston, B.J., Constantine, C.C., Hobbs, R.P., Shury, T., Olson, M.E., 2006. Molecular and morphological characterization of *Echinococcus* in cervids from North America. Parasitology 132, 439–447.

Uga, S., Kataoka, N., 1995. Measures to control *Toxocara* egg contamination in sandpits of public parks. Am. J. Trop. Med. Hyg. 52, 21–24.

Unruh, D.H.A., King, J.E., Allen, J.R., Eaton, R.D.P., 1973. Parasites of dogs from Indian settlements in northwestern Canada: a survey with public health implications. Can. J. Comp. Med. 37, 25–32.

Veit, P., Bilger, B., Schad, V., Schäfer, J., Frank, W., Lucius, R., 1995. Influence of environmental factors on the infectivity of *Echinococcus multilocularis* eggs. Parasitology 110, 79–86.

Viel, J.F., Giraudoux, P., Abrial, V., Bresson-Hadni, S., 1999. Water vole (*Arvicola terrestris scherman*) density as risk factor for human alveolar echinococcosis. Am. J. Trop. Med. Hyg. 61, 559–565.

Viens, P., Milot, J., Jacob, J.L., Little, J., Simard, M., Claveau, R., Deschênes, A., Panisset, A., Portnoy, J., Brouillette, G., Dagenais, R., 1981. Parasitic uveitis in Quebec. Unión Méd. Can. 110, 893–898.

Vors, L.I.V.S., Boyce, M.S., 2009. Global declines of caribou and reindeer. Global Change Biol. 15, 2626–2633.

Weaver, H.J., Hawdon, J.M., Hoberg, E.P., 2010. Soil-transmitted helminthiases: implications of climate change and human behavior. Trends Parasitol. 26, 574–581.

Webster, G.A., Cameron, T.W.M., 1967. Epidemiology and diagnosis of echinococcosis in Canada. Can. Med. Assoc. J. 96, 600–607.

Wilson, J.F., Rausch, R.L., Wilson, F.R., 1995. Alveolar hydatid disease: review of the surgical experience in 42 cases of active disease among Alaskan Eskimos. Ann. Surg. 221, 315–323.

Won, K.Y., Kruszon-Moran, D., Schantz, P.M., Jones, J.L., 2008. National seroprevalence and risk factors for zoonotic *Toxocara* spp. infection. Am. J. Trop. Med. Hyg. 79, 552–557.

Yang, J., Keystone, J.S., McIntyre, L., Spence, H., 1982. *Toxocara* antibodies in veterinary personnel. Can. Vet. J. 23, 126–128.

Yera, H., Estran, C., Delaunay, P., Gari-Toussaint, M., Dupouy-Camet, J., Marty, P., 2006. Putative *Diphyllobothrium nihonkaiense* acquired from a Pacific salmon (*Oncorhynchus keta*) eaten in France; genomic identification and case report. Parasitol. Int. 55, 45–49.