



Wildlife pathogens and diseases in Canada

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PREFACE

The Canadian Councils of Resource Ministers developed a Biodiversity Outcomes Framework¹ in 2006 to focus conservation and restoration actions under the *Canadian Biodiversity Strategy*.² *Canadian Biodiversity: Ecosystem Status and Trends 2010*³ was a first report under this framework. It assesses progress towards the framework's goal of "Healthy and Diverse Ecosystems" and the two desired conservation outcomes: i) productive, resilient, diverse ecosystems with the capacity to recover and adapt; and ii) damaged ecosystems restored.

The 22 recurring key findings that are presented in *Canadian Biodiversity: Ecosystem Status and Trends 2010* emerged from synthesis and analysis of technical reports prepared as part of this project. Over 500 experts participated in the writing and review of these foundation documents. This report, *Wildlife pathogens and diseases in Canada*, is one of several reports prepared on the status and trends of national cross-cutting themes. It has been prepared and reviewed by experts in the field of study and reflects the views of its authors.

Contributing Authors

I.K. Barker, Doug Campbell, Pierre-Yves Daoust, Zoe Lucas, John Lumsden, Danna Schock, Helen Schwantje, Kim Taylor, and Gary Wobeser.

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The information in this chapter draws heavily on the program, data and staff of the Canadian Cooperative Wildlife Health Centre (CCWHC), a wildlife health sciences centre that is a partnership among Canada's five colleges of veterinary medicine, federal, provincial and territorial governments in Canada, and several non-government organizations (www.ccwhc.ca). The CCWHC is a research institute that coordinates wildlife disease surveillance in Canada, provides information to government agencies and the public, offers a variety educational programs and is a Collaborating Centre of the World Organization for Animal Health (OIE). I also thank the reviewers of this report.

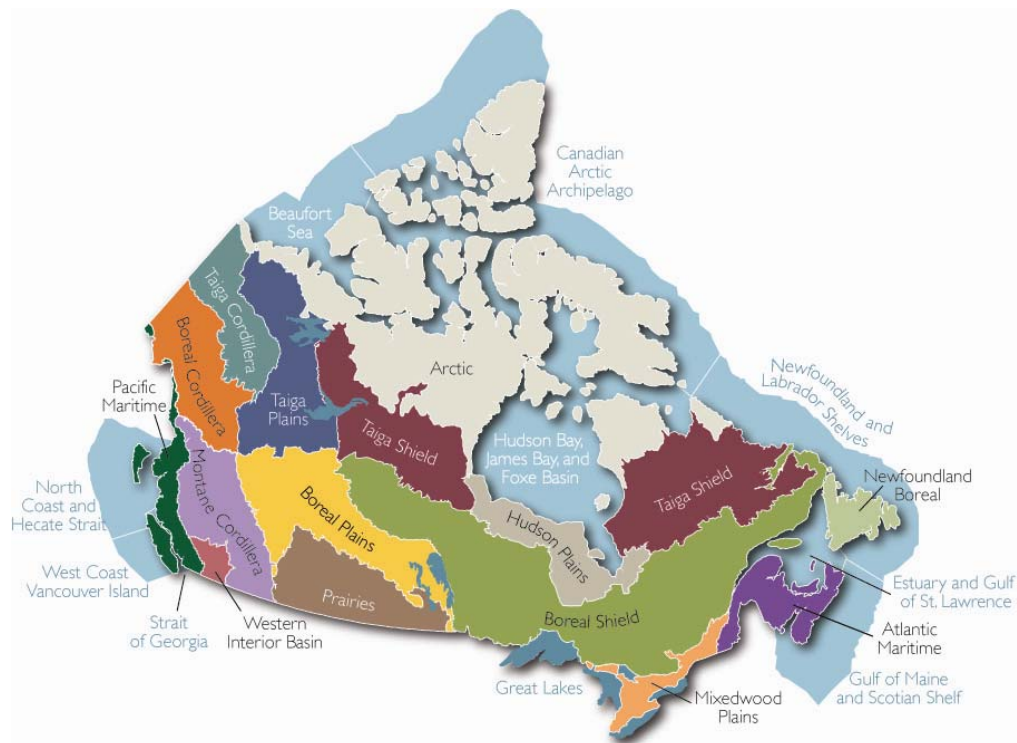
¹ Environment Canada. 2006. Biodiversity outcomes framework for Canada. Canadian Councils of Resource Ministers. Ottawa, ON. 8 p. <http://www.biodivcanada.ca/default.asp?lang=En&n=F14D37B9-1>

² Federal-Provincial-Territorial Biodiversity Working Group. 1995. Canadian biodiversity strategy: Canada's response to the Convention on Biological Diversity. Environment Canada, Biodiversity Convention Office. Ottawa, ON. 86 p. <http://www.biodivcanada.ca/default.asp?lang=En&n=560ED58E-1>

³ Federal, Provincial and Territorial Governments of Canada. 2010. Canadian biodiversity: ecosystem status and trends 2010. Canadian Councils of Resource Ministers. Ottawa, ON. vi + 142 p. <http://www.biodivcanada.ca/default.asp?lang=En&n=83A35E06-1>

Ecological Classification System – Ecozones⁺

A slightly modified version of the Terrestrial Ecozones of Canada, described in the *National Ecological Framework for Canada*,⁴ provided the ecosystem-based units for all reports related to this project. Modifications from the original framework include: adjustments to terrestrial boundaries to reflect improvements from ground-truthing exercises; the combination of three Arctic ecozones into one; the use of two ecoprovinces – Western Interior Basin and Newfoundland Boreal; the addition of nine marine ecosystem-based units; and, the addition of the Great Lakes as a unit. This modified classification system is referred to as “ecozones” throughout these reports to avoid confusion with the more familiar “ecozones” of the original framework.⁵



⁴ Ecological Stratification Working Group. 1995. A national ecological framework for Canada. Agriculture and Agri-Food Canada, Research Branch, Centre for Land and Biological Resources Research and Environment Canada, State of the Environment Directorate, Ecozone Analysis Branch. Ottawa/Hull, ON. 125 p. Report and national map at 1:7 500 000 scale.

⁵ Rankin, R., Austin, M. and Rice, J. 2011. Ecological classification system for the ecosystem status and trends report. Canadian Biodiversity: Ecosystem Status and Trends 2010, Technical Thematic Report No. 1. Canadian Councils of Resource Ministers. Ottawa, ON. <http://www.biodivcanada.ca/default.asp?lang=En&n=137E1147-0>

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OVERVIEW OF PATHOGENS AND DISEASES OF WILD VERTEBRATES

Pathogens are living or non-living things capable of causing disease -- that is, some degree of physiological dysfunction -- in living organisms. Pathogens are normal components of ecosystems and are part of the biological and environmental complexity that lends stability and resilience to ecological function. Living pathogens thus are important components of each ecosystem's biodiversity, and include a wide spectrum of organisms, from worms and arthropods, to fungi, protozoa, bacteria, and viruses. Non-living pathogens include simple chemical elements like lead and mercury, a wide range of industrial and other chemicals produced by humans, complex biological toxins produced by various organisms, and misfolded proteins of normal primary structure as with the prions responsible for the transmissible spongiform encephalopathies such as bovine spongiform encephalopathy (BSE or "mad cow disease") and chronic wasting disease.

Ecologists and epidemiologists recognize that the occurrence, intensity, and importance of disease in an individual or a population is governed by an interplay of influences that include the physiology and population dynamics of the species in question, many details about the pathogens to which that species may be exposed, and a wide range of environmental parameters that determine the conditions under which the host species and pathogens will interact. The patterns of disease occurrence in an ecosystem thus are governed by this triad of host species-pathogen-environment (Hudson et al., 2002; Patz and Confalonieri, 2005). The 20th century has been characterized by profound and escalating environmental changes (Cohen, 1995; McNeill, 2000). It is therefore not surprising that patterns of disease in people, domestic animals, and wild animals also have changed profoundly, particularly in the past six decades. This phenomenon of abruptly changing patterns of disease occurrence has been termed "disease emergence," and has been a central focus of epidemiology and disease ecology in the past two decades (Lederberg et al., 1992; Jones et al., 2008).

Canada initiated a program of national surveillance for diseases in wild vertebrate animals in 1992 with the establishment the Canadian Cooperative Wildlife Health Centre (CCWHC) (Leighton et al., 1997). This Technical Report reviews the status of a selection of diseases in Canadian wildlife based on that surveillance program and additional information. Many of the selected diseases fit the general definition of "emerging diseases," in that they are occurring in new locations, with increased intensity, or are entirely new to Canada. Wildlife disease surveillance is recent in Canada and is carried out at a coarse scale. Thus, trends can be discerned in the patterns of occurrence of some diseases but not in others. Such trends are noted in this report when they can be discerned. The coarse scale of disease surveillance also means that it is not possible to consider each ecozone⁺ separately with respect to disease occurrence. Most of the pathogens covered in this report are distributed across many ecozones⁺ and are therefore organized by pathogen rather than geography. An accounting of the ecozones⁺ included in the range of each pathogen is included within each pathogen section of the report.

Pathogens and diseases that are important or occur in only one or a few ecozones⁺ are described in subsequent sections of the report, organized by ecozone⁺.

Some pathogens of Canadian wild vertebrates are of national and international concern because they can cause disease in people (zoonotic diseases) or in domestic animals, or threaten wild populations around the world. There is a legal obligation to report such disease occurrences to the Canadian Food Inspection Agency (CFIA) under the regulations of the *Health of Animals Act*. Where such a reporting requirement exists, it is indicated in each section heading by the categories Reportable, Immediately Notifiable, or Annually Notifiable. Any suspicion of a Reportable disease must be reported immediately to a CFIA District Veterinarian. Reporting of Immediately Notifiable and Annually Notifiable disease occurrences applies only to disease diagnostic laboratories; reports must be made by these laboratories to the CFIA either immediately or once each year, in February (Canadian Food Inspection Agency, 2009). Occurrence of viral hemorrhagic septicemia of fish must be reported to Fisheries and Oceans Canada.

PATHOGENS DISTRIBUTED ACROSS MULTIPLE ECOZONES⁺

Viruses

Rabies [Reportable]

Rabies is a fatal infection of the central nervous system caused by any of the several different strains of rabies virus, a rhabdovirus. It is transmitted from infected to uninfected animals primarily by bite wounds contaminated with infectious saliva. All mammals appear to be susceptible to all strains of rabies virus, but each strain persists in nature by transmission cycles within a single host species or a very small group of sympatric host species. The strains are named according to the principal maintenance host species, for example, skunk rabies, dog rabies, and raccoon rabies (Rupprecht et al., 2001).

Strains of rabies virus maintained in arctic foxes and in several different species of bats appear to have been present in Canada since prehistoric times. The domestic dog strain circulated in southern Canada from 1907 to 1934. Vaccination of domestic dogs began in the 1940s. The arctic fox strain persists in the Canadian Arctic and has spread southward periodically into southern Canada. A major southward incursion in the 1940s brought this strain of rabies into a large zone from British Columbia to New Brunswick (Tabel et al., 1974). The fox strain virus did not persist in southwestern Canada but became established in red foxes in Ontario and Quebec where it has persisted despite wildlife vaccination programs. There also were incursions of the arctic fox strain of rabies into insular Newfoundland in 1988 and 2002 (Whitney, 2004). In 1959, there was an incursion of a striped skunk rabies strain into southern Manitoba from adjacent North Dakota and Minnesota. This strain spread northwest, reaching Saskatchewan in 1963, and

Alberta in 1970. Rabies persists in skunks in Manitoba and Saskatchewan. Further incursion into Alberta has been prevented thus far by an intensive program of skunk depopulation associated with rabies occurrences along the Saskatchewan-Alberta border. In 1977, an epidemic of the raccoon strain of rabies occurred in West Virginia because of importation of rabid raccoons into the state for hunting (Jenkins and Winkler, 1987). This epidemic swept through the rural and urban zones of the eastern United States and entered Ontario in 1999, New Brunswick in 2000, and Quebec in 2006. Epidemics of fox and raccoon strains of rabies are enormously costly to all affected areas due to the high rate of human exposure to potentially rabid raccoons, the high cost (approximately \$2,000) of each post-exposure treatment, and the cost of the intensive surveillance, vaccination, and population control efforts used to try to stop its spread.

Several different species of bat in Canada carry different strains of rabies virus. Since 1970, all human infections with rabies acquired in Canada have been due to strains carried by bats (24 human cases of rabies have been recorded since 1924). The proportion of bats found dead and tested for rabies that have been found to carry the virus has been in 4-5% range in Canada in the past four decades (Johnstone, 2008).

Dog strain rabies is a conservation concern for several wild African canids, and is a world-wide human scourge, killing 25,000 to 50,00 people each year. Widespread vaccination of companion animals effectively has eliminated this concern in Canada. Control of fox strain rabies in Europe was followed by a pronounced increase in red fox populations with attendant new disease concerns including human echinococcosis (Rupprecht et al., 2001; Hegglin et al., 2003).

No clear trends in the occurrence of rabies in Canada are discernable from the historical record. Two exotic strains of rabies virus have invaded Canada in the past 50 years. Intensive and costly programs aimed at eradicating the fox (native) and raccoon (exotic) strains from southern Canada began 20 years ago and are still in operation, with renewed efforts directed against the newly-arrived raccoon strain.

West Nile virus [Immediately Notifiable]

West Nile virus (WNV), a flavivirus, persists in nature through transmission cycles between a wide range of species of wild birds and a smaller species assemblage of bird-feeding mosquitoes. It was transported to North America from its Afro-Eurasian area of origin and first detected here in 1999. It spread rapidly across North America (McLean and Ubico, 2007). It was first detected in Canada in 2001, and affected all provinces from Nova Scotia to Alberta by 2003 (Figure 1) (Canadian Cooperative Wildlife Health Centre, 2008). The virus was detected in British Columbia for the first time in August 2009 in mosquitoes trapped in the Okanagan Valley. Although maintained in mosquito and bird populations, the virus also can infect and cause disease in a wide range of mammals, including people, and some species of reptiles. Most infected humans experience no illness. Some experience severe fever but readily recover. A small proportion experience severe neurological disease (encephalitis) which may be fatal or cause long-term debility. Despite the small proportion of exposed people who develop severe disease, WNV has caused the largest epidemic of infectious encephalitis in people ever recorded in North America (McLean and Ubico, 2007).

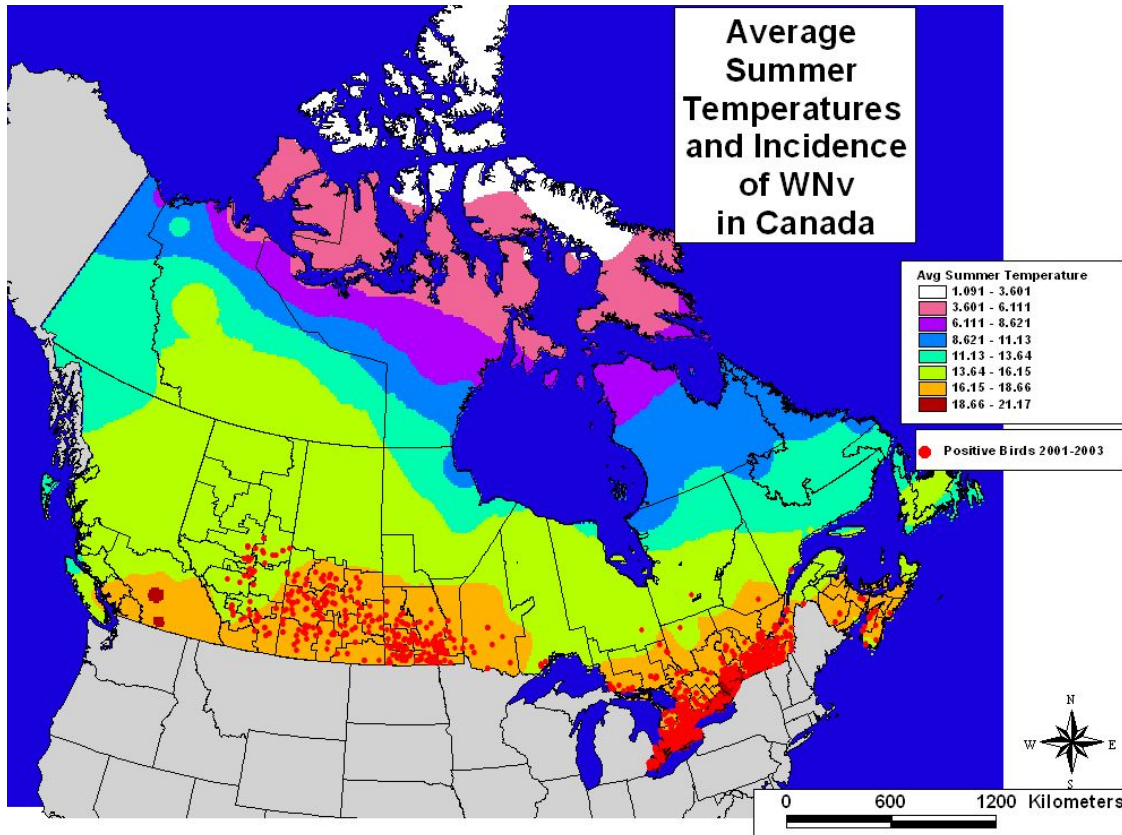


Figure 1. Distribution of birds testing positive for West Nile virus, 2001-2003.
Source: Health Canada (2003)

West Nile virus is not native to Canada and has the potential to produce ecological effects. Some native wild bird species experience high mortality when infected with WNV. Corvids (crows, jays, magpies, and their relatives) are highly susceptible; as are loggerhead shrike and greater sage grouse, two species of conservation concern. Late summer survival of greater sage grouse populations under study in 2003 in western Canada (Prairies Ecozone*) and the United States was reduced by 25% due to WNV infection (Naugle et al., 2004). Another study found declines in continental populations of American crow, blue jay, American robin, eastern bluebird, tufted titmouse, Carolina chickadee, black-capped chickadee, and house wren associated with WNV (LaDeau et al., 2007). Surveillance in Canada, from Nova Scotia to Alberta, has detected WNV as the cause of death in thousands of corvids and over 100 individuals representing 19 non-corvid bird species, including raptors (60%), passerines (15%), greater sage grouse (5%), and gulls (3%), and in two species of squirrel (red and eastern gray) (9%) (Canadian Cooperative Wildlife Health Centre, 2008).

West Nile virus is now firmly established in southern Canada from Alberta to the Maritime Provinces. The intensity of WNV activity has varied greatly among years and appears associated with a range of ecological factors affecting mosquito and bird population parameters. Climate is a key variable, but predictive modeling of likely future scenarios of climate change and WNV activity in Canada has not yet been done.

Avian influenza

Avian influenza (AI) viruses are pathogens of intense current global concern because of the capacity of certain strains of these viruses to kill massive numbers of domestic poultry and the potential of some of these viruses to cause massive global human disease. Influenza viruses in birds all are members of the influenza A group of influenza viruses, and wild birds, particularly wild ducks, are the principal reservoir for influenza A viruses in the biosphere. In wild bird populations, influenza A viruses appear to persist as genetic strains with little capacity to cause disease. However, in other species, these viruses can undergo rapid genetic changes and develop into strains that can, and do, cause diseases, with affects ranging from mild to rapidly fatal. The H5N1 strain of current global concern, as well the H7N3 strains that caused epidemics in Canadian poultry in 2004 and 2007, appear to be recent genetic variants of non-disease causing strains present in wild birds (Capua and Alexander, 2006; Pasick et al., 2007; Berhane et al., 2009; Pasick et al., 2010).

Recent surveys of Canadian wild birds have found AI viruses to be abundant in wild ducks across the country and also to be present to a lesser extent in other species (Parmley et al., 2008; Canadian Cooperative Wildlife Health Centre, 2008). Table 1 shows the percentage of healthy live wild ducks sampled in Canada from 2005 to 2008 that were found to be infected with one or more AI viruses. None of the viruses detected was a highly pathogenic strain. In other species, the infection rate detected has been in the range of 0 to 13%, averaging less than 3%. None of the AI viruses found to date in Canadian wild birds has been a strain of direct concern to the health of people, poultry, or wildlife. However, many different strains of AI viruses have been found, indicating that a very large gene pool of AI virus persists in Canadian wild birds.

Table 1. Percentage of wild duck samples infected with Avian influenza virus.

Survey year	Wild ducks		
	Number tested	Number infected	Percent infected
2005	4,268	1,572	37%
2006	4,035	1,275	32%
2007	5,500	1,340	24%
2008	1,445	33	2%

Source: Canadian Cooperative Wildlife Health Centre (2009)

Globally, AI viruses seldom have been viewed as threats to ecological functions or conservation of endangered species. The currently-circulating H5N1 virus of concern to poultry and human health has also caused mortality in wild birds, including a large number of bar-headed geese in central Asia. This virus strain is a pathogen originating in domestic poultry populations that has been transmitted to wild bird populations, and is not a virus that originated in wild bird populations themselves. It is not known whether or not this or other disease-causing strains of AI viruses are able to persist in wild bird populations without periodic re-introduction from the poultry reservoir. However, such disease-causing strains originating in poultry are a potential risk to wild bird populations. The extraordinarily large global populations of domestic poultry

that have been created in the past few decades are an enormous potential source of disease-causing AI viruses for wild birds (Steinfeld et al., 2006). This situation is unprecedented and the risk posed to wild birds is not known.

Newcastle disease [Reportable]

Newcastle disease (ND) is the internationally accepted name for infection of birds with certain strains of avian paramyxovirus type 1 (APMV-1) that cause high mortality in experimentally-infected domestic chickens, and thus threaten poultry. The many strains of APMV-1 differ markedly in their capacities to cause disease. Newcastle disease is among the most serious epidemic diseases of poultry world-wide. It is highly contagious; any occurrence in any species threatens agricultural economies and the supply of affordable human dietary animal protein in affected regions (Alexander, 2000).

Strains of APMV-1 have been isolated from hundreds of different species of wild birds, particularly waterfowl, in Canada and all over the world. However, only two species of wild bird in Canada carry and maintain Newcastle disease viruses (NDV) strains of APMV-1 capable of causing severe disease in chickens: the double-crested cormorant (*Phalacrocorax auritus*) and the rock pigeon (*Columba livia*). NDV was first detected in wild birds in Canada in double-crested cormorants in the St. Lawrence Estuary in 1975. It was next detected in 1990 in double-crested cormorants in Saskatchewan, and subsequently has been detected regularly, if sporadically, from Alberta to the Atlantic coast. Antibodies to APMV-1 have been found in double-crested cormorant eggs from British Columbia. On one double-crested cormorant colony in Saskatchewan monitored continuously from 1994 to 2008, ND occurred in 1995, 1997, 1999, 2001, 2003, and 2008 (Leighton, F. A., unpublished data; Leighton and Heckert, 2007). NDV in rock pigeons emerged in the Middle East in the 1970s, spread westward across Europe, and reached North America in 1984. It was first detected in Ontario in 1985 and had spread as far west as Saskatchewan by 1990 (Johnston and Key, 1992; Leighton and Heckert, 2007). This rock pigeon virus strain sometimes meets the criteria to be classified as NDV but often does not, and thus is usually referred to as pigeon paramyxovirus to avoid the national and international trade and legal implications of the term "Newcastle disease".

Wild and feral rock pigeon populations appear to suffer high mortality when first infected with pigeon paramyxovirus, but recover quickly despite the persistence of the virus within the affected populations. The discovery in the early 1990s that true NDV is widespread in double-crested cormorant populations across North America was made during a rapid increase of the population of that species, restoring them to historical levels after two centuries of decline associated with human activities (Wires and Cuthbert, 2006). It is likely that mortality events are more readily detected on large colonies with thousands of nests than on the small colonies that were predominant prior to the 1970s. It is not, therefore, known whether NDV in double-crested cormorant is recent or ancient. ND has caused on the order of 40% immediate mortality of late fledgling and fledged hatch-year birds on one closely-studied colony, and additional, ultimately fatal, wing and leg paralysis in numerous surviving birds (Kuiken et al., 1998; Kuiken, 1999). Despite this regular high mortality, no population trends have yet been attributable to ND in double-crested cormorant. Adult double-crested cormorants appear unaffected by ND. NDV in

large and wide-spread double-crested cormorant populations across North America is a constant risk to domestic poultry.

Duck plague [Annually Notifiable]

Duck plague, also known as duck virus enteritis, is caused by anamid herpesvirus 1, a virus native to Eurasia that can infect and cause disease in ducks, geese, and swans. It was first recognized in North America in 1967 when it caused outbreaks of fatal disease in domestic ducks on Long Island, New York and a small outbreak in wild ducks (primarily American black duck and mallard) in adjacent wetlands. In January 1973, a major epidemic of duck plague occurred at Lake Andes Wildlife Refuge in South Dakota; 42,000 of the 100,000 mallards on the refuge died as did a number of Canada geese (Friend, 1999; Hansen and Gough, 2008). This event electrified North American wildlife managers, and the United States immediately established a national wildlife disease laboratory, now the National Wildlife Health Centre in Madison, Wisconsin. It was feared that similar outbreaks might destroy large numbers of wild waterfowl across the continent; regulations were put in place to permit strong and immediate actions to eradicate the virus.

Nevertheless, in the decades since 1973, duck plague has caused no major outbreaks in North America. It has caused small outbreaks, often in semi-domestic urban populations of ducks, in many parts of the United States and in five provinces in Canada: in a private waterfowl collection in Manitoba in 1973 (Prairies Ecozone⁺) (Bernier and Filion, 1975); in a small flock of captive muscovy ducks near Edmonton in 1974 (Prairies Ecozone⁺) (Hanson and Willis, 1976); in a zoo collection in Quebec in 1975 (Mixedwood Plains Ecozone⁺) (Bernier and Filion, 1975); in a single free-ranging mallard from a zoo pond in Saskatoon in 1984 (Prairies Ecozone⁺) (Wobeser and Docherty, 1987); perhaps in three mallards from southwestern Ontario in 1984 (Mixedwood Plains Ecozone⁺) (Wojcinski et al., 1991); and at three separate locations in British Columbia in captive barnacle geese and muscovy ducks in 1993 (Bowes, 1993). One outbreak, of moderate size but of no numerical importance to the affected populations, occurred in American black ducks and mallards in the Finger Lakes region of New York State in February 1994, just south of the Great Lakes and Mixedwood Plains ecozones⁺ (Hansen and Gough, 2008).

This virus now appears to be widespread in wild waterfowl populations across the continent, but does not appear to be the threat to those populations as once feared. Its behaviour and significance in North America now appears similar to the situation in Europe, where duck plague is associated with periodic, small mortality events, often in semi-domestic birds. Whether or not duck plague could again cause large-scale mortality in North America under changing environmental circumstances is not known, nor is it known if the virus now exerts sublethal effects on wild waterfowl populations (Hansen and Gough, 2008).

Hemorrhagic diseases of deer [Reportable/Immediately Notifiable]

Hemorrhagic disease of deer is the general name given to diseases caused by infections of wild ungulates with various strains of two closely-related virus groups: bluetongue (BT) viruses and epizootic hemorrhagic disease (EHD) viruses, both of which belong to the orbivirus family.

Records of diseases likely to have been hemorrhagic disease date back to the 19th century in North America. The viruses are transmitted from animal to animal by small biting flies of the genus *Culicoides* (midges, gnats, no-see-ums). Although white-tailed deer and mule deer are the wild ungulates most affected, these viruses can cause disease in elk, pronghorn, bison, and wild sheep. At the northern edge of their range, where susceptible ungulate populations often have not been previously exposed to the viruses, periodic epidemics of fatal disease, often separated by several years or decades, are the usual expression of hemorrhagic disease. Further south, exposure to these viruses is more constant and a range of expressions, from pockets of epidemic mortality to various sub-lethal conditions, is seen annually (Howerth et al., 2001).

EHD and BT viruses are present on most continents, but none is currently established in Canadian ungulate populations. Both BT and EHD persist in wild and domestic ungulate populations across much of the United States, including states that border Canada, and both have made occasional late summer incursions into western Canada, probably due to wind-borne incursions of infected *Culicoides* sp. (Sellers and Maarouf, 1991). EHD occurred in southeastern Alberta (Prairies Ecozone⁺) in 1962 causing mortality in white-tailed deer, mule deer, and pronghorn, and in the Okanagan Valley of southern British Columbia (Western Interior Basin Ecozone⁺) in 1987 and 1999 causing mortality in wild white-tailed deer and bighorn sheep, and in several other species on a nearby game farm -- mule deer, American bison, elk, and mountain goat (Dulac et al., 1992; Pasick et al., 2001). Antibodies to EHD virus, but not to BT virus, were detected in domestic cattle in southcentral Saskatchewan (Prairies Ecozone⁺) in a survey conducted in 1986/87, but no disease was evident in the cattle or regional wildlife (Shapiro et al., 1991; Dulac et al., 1992). Incursions of BT viruses occurred in the Okanagan Valley in 1975, 1987, and 1998, but were detected only in domestic cattle and sheep (Dulac et al., 1992; Clavijo et al., 2000). Although mortality from these viruses in deer has not had a long-term impact on overall wildlife population sizes, they may pose a threat to small local or regional populations of species of conservation concern such as bighorn sheep and pronghorn.

EHD and BT are likely to affect Canadian wild ungulates more often in the future, associated with a warming climate. This is most likely to occur in ecozones⁺ adjacent to areas in the United States where EHD and BT now occur, that is, southern regions of the Western Interior Basin, Montane Cordillera, Prairies, Boreal Shield, and Mixedwood Plains. The distribution of the *Culicoides* species which are competent vectors for these viruses and the rate of virus replication within these vectors both respond to temperature, among other environmental variables (Purse et al., 2008). There have been a few recent occurrences of EHD in the northeastern United States, close to Canada -- Michigan in 2006 and Pennsylvania and New York State in 2007 (including in Niagara County bordering the Mixedwood Plains Ecozone⁺) (New York State Department of Environmental Conservation, 2007; Pennsylvania Game Commission, 2008; Department of Natural Resources, 2009). However, these occurrences are not clearly related to trends in climate change. Recent incursions of BT viruses into Europe give warning of the rapid and massive shift in BT virus distribution that can occur in association with climate change and other factors (Purse et al., 2008). Canada is recognized as free of BT in domestic livestock for purposes of

international trade; establishment of BT in Canada would negatively affect aspects of the Canadian livestock economy.

Ranaviruses of amphibians [Reportable]

Author: Danna Schock

Ranaviruses (family Iridoviridae) infect fish, reptiles, and amphibians, and are of considerable concern in aquaculture (Chinchar, 2002; Williams et al., 2005). Lethal amphibian ranaviruses infect a wide range of species and die-offs have been documented world-wide (Collins et al., 2003). In Canada, ranaviruses have been isolated from amphibian populations in Ontario (Mixedwood Plains and southern edge of Boreal Shield) (Greer et al., 2005; Duffus et al., 2008), Manitoba, Saskatchewan and Alberta (Prairies and Boreal Plains) (Schock et al., 2008; Goater, C., pers. comm.), and the Northwest Territories (Taiga Plains) (Schock, D., unpublished data). In a review of 64 amphibian mortality events in the United States, Green et al. (2002) reported that ranaviruses affected only widespread and abundant amphibian species. However, amphibian ranaviruses isolated from abundant species such as barred tiger salamanders (*Ambystoma mavortium*) and wood frogs (*Rana sylvatica*) can cause lethal infections in multiple amphibian species (Schock et al., 2008). Thus, the potential exists for ranaviruses maintained in resilient populations of abundant species to serve as sources of infection to vulnerable rare species in the same habitat. Further, die-offs caused by ranaviruses can severely reduce numbers of amphibians at breeding sites and may affect amphibian populations in highly fragmented habitats where re-colonization is unlikely (Collins et al., 2003).

Too little is known about ranaviruses and their effects on Canadian amphibian populations to identify trends or future trajectories of amphibian-ranavirus interactions. It is now clear that human activities, particularly the trade in fishing bait, has the potential to transport different strains of ranaviruses among widely-separated habitats (Collins, 2008), and there is strong experimental evidence that virus strains new to local populations can sometimes cause very high mortality (Schock, 2007).

Morbilliviruses of terrestrial and marine carnivores

Morbilliviruses are a large virus group that includes human measles virus, several viruses important to livestock (rinderpest and peste des petites ruminants viruses), and an array of viruses in wild animals that are well-known but of uncertain virus taxonomy. Many morbilliviruses cause severe disease in susceptible wild species, often with high fatality rates. However, a virus that is fatal in one species may infect one or more other host species with little or no clinical disease. Thus, certain species serve as maintenance hosts for these viruses, and sources of infection for other, more susceptible species. For example, harp seals may be sources of infection with phocine distemper virus for harbor seals (Duignean et al., 1995a; Duignean et al., 1997; Barrett et al., 2003). Morbilliviruses have been named according to the animal host in which each was first discovered to cause disease, for example, canine distemper virus (domestic dogs), phocine distemper virus (seals), dolphin morbillivirus. Nevertheless, these names can be misleading; for example, canine distemper viruses cause severe disease in many non-canid

species such as raccoons, skunks, ferrets, and several wild cats, and antibodies indicating exposure to dolphin morbillivirus and to phocine distemper virus have been found in land-locked grizzly bears in the Montane Cordillera Ecozone⁺ (Rossiter et al., 2001; Philippa et al., 2004).

Because they are known to infect such a wide range of widely distributed carnivores in Canada, morbilliviruses must be assumed to occur in Canadian wild carnivores in all ecozones⁺ (Duigneau et al., 1995a; Duigneau et al., 1995b; Duigneau et al., 1997; Rossiter et al., 2001; Philippa et al., 2004; Canadian Cooperative Wildlife Health Centre, 2008). The ecology, reservoirs, and transmission dynamics of these viruses in Canada are largely unknown, however. Morbilliviruses known to be present in Canadian wildlife include various strains of canine distemper virus (wide host range including canids, felids, procyonids, and mustelids, assumed to be in all terrestrial ecozones⁺) (Rossiter et al., 2001; Canadian Cooperative Wildlife Health Centre, 2008), phocine distemper virus (harbour, gray, harp, and hooded seals in the Gulf of Maine and Scotian Shelf and the Estuary and Gulf of St. Lawrence ecozones⁺, and ringed seals in the Canadian Arctic Archipelago, Beaufort Sea, and Hudson Bay, James Bay and Foxe Basin ecozones⁺) (Duigneau et al., 1995b; Duigneau et al., 1997), and porpoise morbillivirus (harbour porpoise) in the Gulf of Maine and Scotian Shelf Ecozone⁺ (Duigneau et al., 1995a).

Morbilliviruses can threaten small populations of susceptible rare species when they are maintained in larger populations of sympatric species. Canine distemper virus was in the process of killing the last known wild population of black-footed ferrets when the last remnant animals were brought into captivity to prevent infection and permit captive breeding and release (Thorne and Williams, 1989). This virus continues to menace the success of restoration programs, including planned restoration to Canada's Grasslands National Park (Prairies Ecozone⁺). Morbilliviruses have had significant impacts on conservation programs for African wild dogs and lions (Roelke-Parker et al., 1996; Van de Bildt et al., 2002), and have caused severe epidemics in harbour seals in the eastern Atlantic (phocine distemper) (Barrett et al., 2003) and seals of the Caspian Sea and Lake Baikal (canine distemper) (Kennedy et al., 2000). Potentially vulnerable populations of species at risk in Canada include the American badger population of the Western Interior Basin and Montane Cordillera ecozones⁺, the swift fox of the Prairies Ecozone⁺, and the beluga population in the St. Lawrence Estuary. Environmental or other changes that result in new range overlaps or intensified interactions among previously separated carnivore populations appear to be significant risk factors for epidemics due to morbilliviruses.

Bacteria

Avian cholera [Immediately Notifiable]

Avian cholera is the name given to infection of birds with disease-causing strains of the bacterium *Pasteurella multocida*. In poultry, the name fowl cholera often is used for this disease.

Despite these names, this disease is not related in any way to human cholera (Samuel et al., 2008).

Avian cholera was recognized as an important cause of mortality in wild birds in North America only since the middle of the last century (Samuel et al., 2008). Large die-offs of wild birds, especially ducks and geese, were observed in California and Texas in winter in the 1940s. Beginning in the 1970s in Canada, avian cholera was recognized as occurring regularly in migrating geese in the Prairies Ecozone⁺, and in common eider nesting in the St. Lawrence Estuary (Wobeser, 1997b). More recently, epidemics also have occurred in double-crested cormorants in the Boreal Plains Ecozone⁺, in Arctic-nesting eiders in the Hudson Bay, James Bay & Foxe Basin Ecozone⁺, and in pelagic and coastal marine birds in winter in the Newfoundland and Labrador Shelves and the Gulf of Maine and Scotian Shelf ecozones⁺ (Canadian Cooperative Wildlife Health Centre, 2008; Buttlar, 2009). In wild geese in Canada, (lesser snow, Canada, cackling, Ross, white-front), the disease has been recognized as a cause of annual but minor mortality during spring and fall migration through the Prairies Ecozone⁺, but occasionally causing larger epidemics among nesting geese in the Taiga Shield and Arctic ecozones⁺ (Canadian Cooperative Wildlife Health Centre, 2008; Samuel et al., 2008).

The strains of *P. mulocida* which cause avian cholera in wild birds are extremely powerful pathogens, rapidly transmissible within populations of birds and often producing very high levels of mortality. Although there often is collateral infection and mortality among other avian species using the same habitat as the main affected species during an epidemic, including avian predators and scavengers, each epidemic seems overwhelmingly to involve only one or a small number of species.

The emergence of avian cholera as an epidemic disease of wild birds in North America has been coincident with, and possibly related to, broad land use changes resulting in loss of wetland habitat, the consequent high densities of birds on refuges and other habitat remnants, and the associated unsanitary conditions and stress conducive to transmission of the bacterium among birds, particularly through contaminated water. However, too little is known about risk factors for these epidemics to attribute outbreaks to specific environmental factors (Samuel et al., 2008). Recognition of large outbreaks in double-crested cormorants also has been coincident with dramatic increases in the size of breeding colonies. Recent outbreaks in Arctic eiders and marine birds of the western Atlantic are unexplained. It is not yet possible to discern whether the record of occurrence of avian cholera in North American wild birds over the past 60 years represents the progressive expansion of an epidemic disease across an ever larger spectrum of species and populations, or a series of unconnected disease events, each with its own set of causal factors. Despite large epidemics affecting many thousands of birds, there is no evidence to date that avian cholera has had a significant impact on continental populations of the most-affected species.

Lyme disease

Clinical disease in people due to infection with the bacterium *Borellia burgdorferi* was first recognized in North America in a cluster of cases in the town of Old Lyme, Connecticut, and

has been called Lyme disease ever since. This is not an important disease of wild animals, but wild animals are the source of infection for people. Lyme disease is a common and severe illness in the northeastern and northcentral United States and is of increasing importance in Canada. Infection is transmitted from the wildlife reservoir of the bacterium in small rodents, such as the white-footed mouse and eastern chipmunk, to people by two species of tick: *Ixodes scapularis* east of the Rocky Mountains and *Ixodes pacificus* in British Columbia. People are at risk of contracting Lyme disease in spring, summer, and fall in areas where the tick vector is a permanent resident and where local small rodents carry the bacterium (Brown and Burgess, 2001). Until very recently, these conditions prevailed only in a small area of the Mixedwood Plains along the north shore of Lake Erie and at the southern margin of the Pacific Maritime Ecozone⁺. In the past decade, however, zones of permanent human risk of Lyme disease in Canada have expanded to include a larger area of the Mixedwood Plains, a small area within the Atlantic Maritime, and small areas within the southern margin of the Boreal Shield and Boreal Plains (Public Health Agency of Canada, 2006).

Ecological models of the effect of climate change on the public health risk of Lyme disease in Canada predict expansion of the range of the vector tick (*I. scapularis*) northward 200 km by 2020, to include the eastern Prairies, southern Boreal Plains, southern Boreal Shield, all of the Mixedwood Plains, and most of the Atlantic Maritime ecozones⁺, and up to 1,000 km by 2080, to include larger segments of the Prairies, Boreal Plains, southern Hudson Plains, Boreal Shield, Atlantic Maritime, and Newfoundland Boreal ecozones⁺ (Ogden et al., 2006). Thus, areas of permanent human risk of Lyme disease are predicted to include a majority of Canada's human population within a few decades. Migratory passerine birds are a constant source of *I. scapularis* ticks to much of Canada. It is estimated that from 50 to 175 million *I. scapularis* are brought to Canada each year by spring migrant song birds (Ogden et al., 2008). Thus, any habitat made suitable to this tick through climate change may quickly be populated. These birds also transport the bacterium, either by transporting infected ticks or because the birds themselves are infected. Sporadic cases of Lyme disease in areas where the vector tick does not complete its life cycle are most likely due to the bites of infected ticks brought into the area by migratory birds. However, introduction and establishment of the bacterium in new populations of the tick may occur less frequently than does dissemination of the tick itself. This may extend the time required for Lyme disease itself to spread northward in association with the expanding range of the tick vector (Ogden et al., 2008). The recent expansion in Canada of the areas at risk of Lyme disease are consistent with these predictions and mechanisms.

***Mycoplasma gallisepticum* [Annually Notifiable]**

The bacterium *Mycoplasma gallisepticum* (MG) is an important pathogen of domestic poultry. In 1994, infection with MG causing severe conjunctivitis (inflammation of the moist membranes of the outer eye and inner eyelid) was first detected in the eastern population of the house finch in Washington, DC. By June 1996, this infection had been recognized in house finches in all states east of the Mississippi River and in Ontario, Quebec, and the Maritime Provinces (Mixedwood Plains, Boreal Shield, and Atlantic Maritime ecozones⁺) (Fischer et al., 1997). Affected house finch populations underwent marked declines; infection has persisted in these populations and

their numbers have not recovered (Dhondt et al., 1998; Nolan et al., 1998; Hochachka and Dhondt, 2000). Infection and disease also have been found in pine grosbeak and evening grosbeak in Quebec (southern Boreal Shield Ecozone*) and in American goldfinch in the United States (Fischer et al., 1997; Mikaelian et al., 2001). Possible effects on populations of these other species have not been studied. Live house finches with severely inflamed conjunctiva typical of MG infection have been observed in the Prairies but the specimens required to confirm MG as the cause have not been available. Infection has been confirmed in adjacent Montana (Duckworth et al., 2003). This is evidence of spread of MG from the eastern to the western population of house finch.

MG in wild finches is a potential conservation concern for two reasons. The causative bacterium appears to be a strain of a poultry pathogen now adapted to and maintained within the wild house finch population. As such, it is an example of a pathogen transmitted from a domestic animal source to a wild species that has significantly reduced the population of the affected wild population. The effect of MG on house finch populations also appears to be significantly affected by climate. The prevalence of diseased birds was approximately three times higher in the southern than it was in central and northern areas of the eastern United States and prevalence appeared associated with minimum winter temperature (Altizer et al., 2004). Therefore, the impact of MG on house finch or other susceptible bird populations in Canada may increase with predicted climatic warming. On the other hand, the most affected population has been the eastern population of the house finch, which originated from a small number of birds taken from the normal range of this species in the western United States and released on the east coast in 1940 (Hill, 2008). Recent studies have shown that the eastern population may have reduced genetic variability due to its foundation on a small number of individuals and also that house finches with greater genetic heterozygosity are more resistant to the effects of MG infection (Hawley et al., 2005; Hawley et al., 2006).

Bovine tuberculosis [Reportable]

Bovine tuberculosis (BTb) is caused by infection with the bacterium *Mycobacterium bovis*. It readily infects domestic cattle, and in people it causes a disease indistinguishable from human tuberculosis (infection with *M. tuberculosis*). Infection generally is permanent if untreated, and disease is prolonged and debilitating or fatal (Clifton-Hadley et al., 2001). Infected animals, meat products, and milk are significant health hazards for people, and for public health reasons, BTb was successfully eradicated from Canada's domestic animal population through a long and costly program of testing all herds and slaughtering entire herds in which any infected animals were detected. Herd slaughter is necessary because the tests for BTb in live animals are unreliable and easily fail to detect infected individuals. These tests are quite accurate, however, when used to identify infected herds. BTb can infect and cause disease in a wide range of mammalian species. However, maintenance of infection in a population appears to require gregarious behaviour which affords the necessary rates of contact to achieve inter-generational infections. These maintenance populations also can provide a constant source of infection for susceptible scavenger species, such as wild or feral pigs, as occurs when these are sympatric with an infected maintenance population (Connelly et al., 1990; Clifton-Hadley et al., 2001).

Bison in Wood Buffalo National Park and adjacent areas (Boreal Plains, Taiga Plains, and Taiga Shield ecozones⁺) became infected with BTb in the 1920s when an infected herd of over 6,000 plains bison were translocated from the former Buffalo National Park in eastcentral Alberta (Nishi et al., 2006). Infection has persisted in this herd and surveys between 1997 and 1999 found that approximately 49% of these bison were infected (Joly and Messier, 2004a). (These bison also carry bovine brucellosis – see page 15) In the past two decades, other populations of wild bison, apparently free of infection with BTb, have become established in the Taiga Plains, Taiga Cordillera, Boreal Cordillera, and Boreal Plains ecozones⁺ to the north, west, and south of the range of the infected herds (Gates et al., 2001) (Figure 2). Effective measures to prevent the spread of BTb to these infection-free herds are not in place (Nishi et al., 2006). Thus, the potential spread of BTb from infected to non-infected wild bison, all of which are assessed as Threatened by the Committee on the Status of Endangered Wildlife in Canada, and also to livestock, is a major conservation and socio-economic issue.

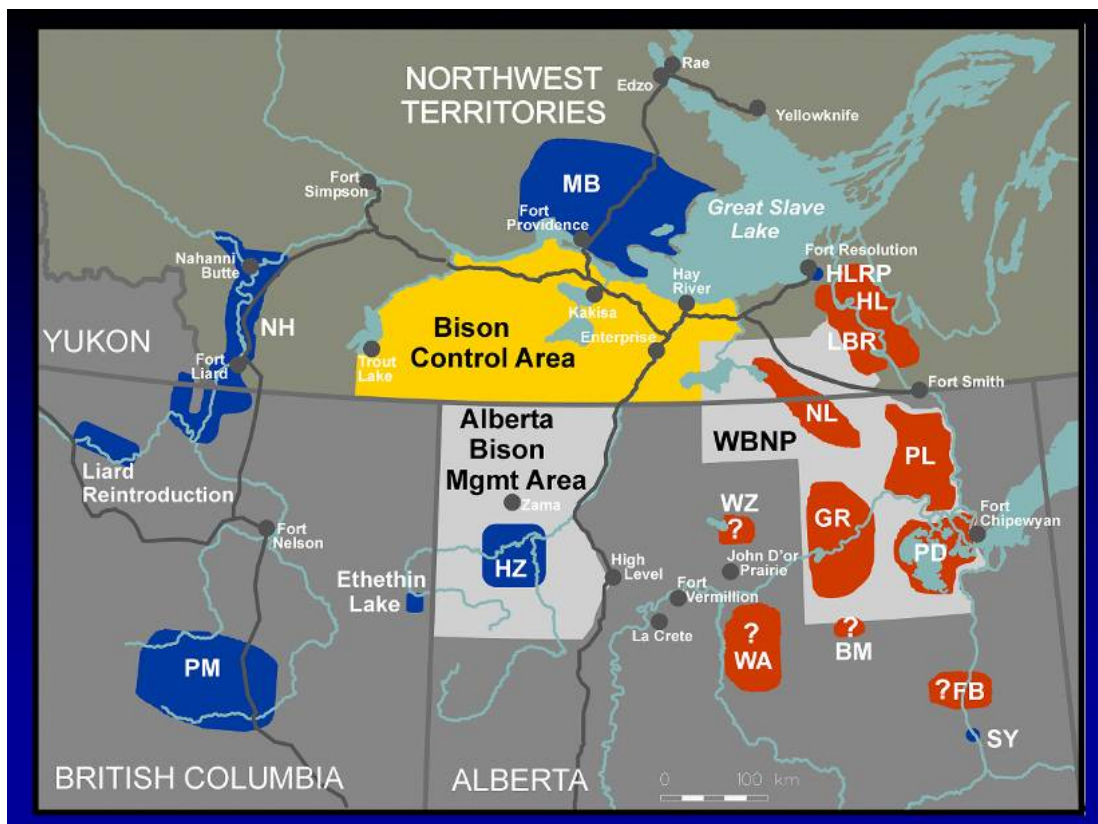


Figure 2. Distribution of bovine tuberculosis-free and diseased free-ranging bison herds in northwestern Canada.

Herds in blue are considered disease-free. Herds in red have had disease presence confirmed or are assumed to be diseased due to movement patterns into areas of confirmed disease status.

WBNP = Wood Buffalo National Park, NH = Nahanni Herd, PM = Pink Mountain, HZ = Hay Zama, GR = Garden River Herd, HLRP = Hook Lake Recovery Project, LBR = Little Buffalo River, MB = Mackenzie Bison Herd, WZ = Wentzel, WA = Wabasca Herd, NL = Nyarling, PL = Pine Lake, PD = Peace Delta, BM = Birch Mountains, FB = Firebag, SY = Syncrude/Fort Mckay, Liard Reintroduction = Norquist herd
Source: Elkin (2008)

The most extensive research on whether or not BTb (and brucellosis) have an impact on the demography of the infected bison herds found evidence that there is a negative impact (the disease alters the predation rate resulting in the predator having a larger impact than would be the case without the disease) and proposed a biological mechanism through which the impact will persist and keep the population well below the carrying capacity of the available habitat (Joly and Messier, 2004b). Others have disputed this interpretation (Bradley and Wilmshurst, 2004). The current rise in the population of infected bison in the area is compatible with either interpretation.

There is agreement among many scientists that BTb (and brucellosis) could be eliminated from wild bison through complete eradication of the infected herds and re-population with bison free of BTb and brucellosis, and also that such eradication is technically possible (Shury et al., 2006). However, the governments of Canada, Alberta, and the Northwest Territories have not yet resolved this issue since it was first fully articulated in 1990 (Connelly et al., 1990; Nishi et al., 2006). It seems certain that without effective intervention of some form, BTb (and brucellosis) will spread to non-infected wild bison herds progressively over time, and that the vast majority of all wild bison in Canada will become infected (Gates et al., 2001). As recent controversies associated with the movement of diseased bison out of Yellowstone National Park in the United States illustrate, infection of wild bison with diseases of major public health and socioeconomic concern can limit the conservation options for this species (Brown, 2008).

BTb was discovered in elk, domestic cattle, and white-tailed deer in the area of Riding Mountain National Park in 1991 (Lees et al., 2003; Nishi et al., 2006). It appears that the bacterium had persisted undetected in this area in cattle and/or elk herds during the eradication program. In 2009, it was estimated that 3.5% of elk and 1% of white-tailed deer in the Riding Mountain National Park area are infected (Shury, T., unpublished data). There is evidence that BTb is maintained in populations of white-tailed deer only under conditions of unusually high population density, such as the large-scale deer feeding programs associated with BTb in deer in Michigan (Miller et al., 2003). This may be a factor in the maintenance of BTb in elk in the National Park area (Lees et al., 2003). Currently, a multi-stakeholder Task Group for Bovine Tuberculosis is taking a range of actions to reduce transmission of BTb among elk and from elk to cattle (Nishi et al., 2006).

Brucellosis [Reportable]

Brucellosis is the name given to all diseases caused by infection with any of the several different species of the bacterial genus *Brucella*. The clinical manifestations of brucellosis are many, but the most common are infection and inflammation of the female and male reproductive tracts with resulting abortion and male infertility, and infection of joints and tendon sheaths resulting in progressive lameness. Infection persists, often for the lifetime of the animal. People are similarly susceptible to infection with *Brucella* sp., and brucellosis in animals with which people have contact is a public health risk (Chan et al., 1989; Forbes, 1991; Thorne, 2001).

In Canadian wildlife, infection with *Brucella* sp. is widespread and of potential ecological and public health significance in three areas: 1) in bison in and around Wood Buffalo National Park,

where the bison populations infected with bovine tuberculosis are co-infected with bovine brucellosis caused by *Brucella abortus* (Boreal Plains, Taiga Plains and Taiga Shield ecozones⁺) (see bovine tuberculosis, above) (Tessaro, 1986); 2) in barren ground caribou populations, and one herd of reindeer near Tuktoyaktuk, Northwest Territories, which are infected with *Brucella suis* biotype 4 across the Arctic, Taiga Cordillera, Taiga Plains, Taiga Shield, and the northern edges of the Boreal Plains, Boreal Shield, and Hudson Plains ecozones⁺; (Forbes, 1991); and 3) in seals (harbour, harp, hooded, gray, and ringed seals, and walrus) and whales (beluga, narwal) in the Gulf of Maine and Scotian Shelf, Estuary and Gulf of St. Lawrence, Newfoundland and Labrador Shelves, Canadian Arctic Archipelago, Hudson Bay, James Bay and Foxe Basin, and West Coast Vancouver Island ecozones⁺ (Forbes et al., 2000; Nielsen et al., 2001).

Bovine brucellosis was eradicated from the Canadian cattle herd in 1985 and now is an issue for bison conservation, human health, and agricultural economies that parallels that posed by bovine tuberculosis (Connelly et al., 1990). Approximately 30% of bison in the Wood Buffalo National Park area are infected (Joly and Messier, 2004a). Brucellosis is widespread in arctic caribou, with 20 to 50% of animals in various herds infected (Leighton, F. A., unpublished data; Koller-Jones, M., pers. comm.). However, its ecological impact, if any, on infected populations is not known. Infection of northern people with this bacterium occurs and is associated with consumption of caribou (Chan et al., 1989; Forbes, 1991). Whether or not *B. suis* biotype 4 is a naturally occurring pathogen in North America or a pathogen introduced from Europe in imported reindeer also is not known. There are no records of this infection in woodland caribou, including the George River herd of northern Quebec. *Brucella* infection in marine mammals was discovered only in 1994 (Forbes et al., 2000) and its importance to wild populations and to human health have not been assessed.

As noted for bovine tuberculosis, it seems certain that without effective intervention of some form bovine brucellosis will spread to non-infected wild bison herds progressively over time, and that the vast majority of wild bison in Canada then will be infected (Gates et al., 2001). This will place bison recovery efforts further at odds with livestock economies and public health interests. Too little is known about the ecology of *Brucella* in caribou or in marine mammals to identify current trends or predict future trajectories. A serological survey of a large herd of reindeer in the western edge of the Arctic Ecozone⁺ and of a barren ground caribou herd (Kaminuriak) in the Taiga Shield and adjacent Arctic ecozones⁺ of Manitoba and Nunavut in the 1960s found only 9% of reindeer and 4% of caribou infected (Broughton et al., 1970). The more recent infection rates of 20 to 50% may represent a trend of increasing prevalence. Any environmental changes that increase the overlap of barren ground caribou with woodland caribou carry the risk that *Brucella suis* biotype 4 may become established in woodland caribou populations.

Anthrax [Reportable]

Anthrax is the name given to all forms of disease caused by infection with the bacterium *Bacillus anthracis*. It is most typically a disease of wild and domestic ungulates, in which it usually is rapidly fatal. Mammalian predators and scavengers also die regularly during anthrax outbreaks in ungulates. Humans are susceptible to anthrax and disease in people ranges from a self-

limiting infection of the skin to rapidly fatal disease following ingestion, inhalation, or contamination of wounds. Ungulates generally become infected from bacterial spores in soil. Environmental conditions that cause these spores to persist for decades or even centuries in soil and to concentrate on the soil surface, such as high-calcium soil chemistry for spore persistence, and flooding followed by dry periods for spore concentration, appear to be major risk factors in outbreaks of anthrax in wild and domestic ungulates. Animal to animal transmission of the bacterium plays only a minor role. Anthrax probably was introduced to North America by European exploration and settlement (Dragon and Rennie, 1995; Dragon et al., 1999; Hugh-Jones and de Vos, 2002).

In Canadian wildlife, anthrax has been recognized most often in bison in and around Wood Buffalo National Park. The first recognized outbreak was in 1962 and sporadic outbreaks have occurred ever since, often with inter-outbreak time spans of many years. Anthrax was recognized in bison in the Mackenzie Bison Sanctuary in 1993 (Taiga Plains Ecozone⁺) and in a bison herd associated with Prince Albert National Park (Saskatchewan, Boreal Plains Ecozone⁺) in 2008 (Dragon and Rennie, 1995; Dragon et al., 1999). Wild ungulates also have died in association with outbreaks of anthrax in domestic cattle in Canada. For example, small numbers of white-tailed deer and moose were found dead of anthrax in such an outbreak in central Saskatchewan in 2007 (Canadian Cooperative Wildlife Health Centre, 2008).

To date, outbreaks of anthrax in wildlife in Canada have affected relatively few individuals. The total number of bison and other species to have died of anthrax is unknown, but a minimum of 1,309 bison died of the disease in outbreaks between 1962 and 1993. Clearly, anthrax contributes to the overall effects of infectious diseases on these bison populations but has not, by itself, posed a significant risk to the survival of these populations (Joly and Messier, 2004b). Anthrax in bison does represent a potential health risk to people who hunt or handle infected bison or their tissues. The occurrence of outbreaks in wild bison and in livestock appear linked to climatic factors, particularly intense precipitation followed by drought. To date, no predictive models have been published with respect to outbreaks of anthrax in Canada and predicted climate change.

Plague

Plague is the name given to all forms of disease caused by infection with the bacterium *Yersinia pestis*. Synonyms for the disease in people include 'bubonic plague' and 'the black death.' The bacterium probably evolved quite recently (2 to 20,000 years ago) in central Asia as a pathogen of small rodent communities transmitted by fleas. Plague caused wide spread human disease in the Near East and Europe in the 6th to 8th centuries and again in the 14th to 19th centuries. In the late 1800s, an epidemic of plague spread from China around the world by newly available fast steam ship traffic, arriving in San Francisco in 1900. The bacterium became established in local wild rodent and flea communities and spread in wildlife east, north, and south over the ensuing three to four decades. It now is present in Canada in an area of uncertain size in the southern Prairies and Western Interior Basin ecozones⁺, and possibly the Montane Cordillera. Surveys conducted in the 1930s and 1990s detected *Yersinia pestis* in southern Saskatchewan and Alberta. Two bushy-tailed woodrats were found dead of plague in the Lillooet area of British Columbia

in 1988 (Gibbons, 1939; Gibbons and Humphreys, 1941; Humphreys and Campbell, 1947; Barnes, 1982; Lewis, 1989b; Gage et al., 1995; Leighton et al., 2001).

Plague persists in communities of small rodents that are sufficiently resistant to severe disease that the bacterium can persist in their rodent-flea communities. However, populations of species such as colonial ground squirrels and prairie dogs suffer severe epidemics when plague is introduced into their colonies, with mortalities as high as 99% (Gage and Kosoy, 2005). These highly susceptible rodent populations are able to recover in one to three years. Nevertheless, the introduction of plague to North America is thought to have been a significant factor in driving the black-footed ferret to extinction in the wild by the late 1980s because of this animal's dependence on prairie dogs for food and also its own susceptibility to plague (Biggins and Godbey, 2003). Plague in prairie dogs is now a significant factor affecting the long-term success of the black-footed ferret recovery program based on re-introduction of captive-bred animals in the United States and Canada (Grasslands National Park, Prairies Ecozone⁺). Only one case of plague in people derived from a wildlife source is on record in Canada (Gibbons and Humphreys, 1941), but there are 10 to 15 such cases each year in the United States (Division of Vector-Borne Infectious Disease, 2009). Plague in North American wildlife appears most prevalent in dry habitat at medium elevations in the American southwest (Gage et al., 1995). Currently, southwestern Canada is the northern limit of the geographic distribution of plague. It seems likely that a warming climate will change this distribution, with extensions northward toward major urban centers such as Calgary, Edmonton, Regina, and Saskatoon.

Fungi

Chytrid fungus of amphibians [Reportable]

Author: Danna Schock

The amphibian pathogen *Batrachochytrium dendrobatidis* (*Bd*), a chytrid fungus which infects the skin of amphibians, has been linked to catastrophic amphibian declines around the world since the 1990s (Skerratt et al., 2007). The origin of *Bd* is unclear (Rachowicz et al., 2005; Morgan et al., 2007). In many regions of the world, patterns of disease and declines in affected species suggest *Bd* is a newly-introduced pathogen that has swept through susceptible host species, decimating amphibian diversity, and has then become established in suitable reservoir species (Retallick et al., 2004). Alternatively, *Bd* may be a historically widely distributed organism and recent declines associated with *Bd* may be linked to large-scale environmental changes that increase the impact of *Bd* on host populations (Ouellet et al., 2005; Pounds et al., 2006). Regardless of the ultimate source of *Bd*, this pathogen now is widespread and causing declines in many parts of the world.

Bd has been isolated from several amphibians across Canada. In a large study of museum specimens, *Bd* was detected microscopically in amphibians collected in various parts of British Columbia, Ontario, Quebec, New Brunswick, and Nova Scotia (Ouellet et al., 2005). The majority of these specimens were from Quebec, especially along the St. Lawrence River

(Mixedwood Plains, Atlantic Maritime, and Boreal Shield ecozones⁺). The authors argue that *Bd* is not linked with recent amphibian declines in eastern Canada because they detected high percentages of *Bd*-infected specimens collected as far back as 1960 from species that have not declined. However, there is strong evidence linking *Bd* to the declines of species in western North America (Briggs et al., 2005; Schlaepfer et al., 2007). The timing of the collapse of northern leopard frog (*Rana pipiens*) populations across western North America, including in Canada's Prairies Ecozone⁺, in the 1970s and 80s, is consistent with *Bd*-related declines elsewhere in the west. However, the absence of necessary data makes it unlikely that the cause(s) of the declines will be conclusively determined. *Bd* has been isolated from multiple amphibian species in British Columbia (Pacific Maritime Ecozone⁺) and Alaska (Reeves and Green, 2006; Adams et al., 2007), Northwest Territories (Taiga Plains Ecozone⁺) (Schock, D., unpublished data), Alberta (Prairies and Boreal Plains ecozones⁺) (Kendell, K., pers. comm.), and Saskatchewan (Prairies Ecozone⁺) (Canadian Cooperative Wildlife Health Centre, 2008).

Linking specific pathogens to declines in amphibian species is problematic. Most amphibians in Canada have boom-and-bust population cycles. Many species live ten or more years and may forego breeding activities altogether in years when rain and other environmental triggers are insufficient or occur too late in the year. Detection of steady downward declines, rather than fluctuations associated with expected boom-bust cycles, may require decades of monitoring (Alford and Richards, 1999). It is clear that *Bd* is causing significant declines in amphibian biodiversity in many parts of the world. In some instances, changing environmental conditions appear to enhance mortality due to *Bd*. It is not possible to predict whether or not similar negative impacts on amphibians in Canada will be triggered by impending changes in climate and land use.

White nose syndrome in bats

Authors: I.K. Barker, Kim Taylor and F.A. Leighton

White nose syndrome (WNS) is a new disease that was first recognized in bats in New York State in the winter of 2006 and has since been found in bats in hibernation caves in the United States from New Hampshire to Tennessee and Missouri, and in Canada in southern Quebec, in the general regions of Ottawa-Gatineau and south of Sherbrooke, and in eastern Ontario, as far north as Kirkland Lake (Figure 3). These locations include the Mixedwood Plains, Boreal Shield and Atlantic Maritime ecozones⁺. The name of the disease refers to a ring of white fungus around the muzzles and elsewhere on the bodies of affected bats (Figure 4). The cause of this disease is not fully understood, but it is associated with the growth on the bats of a particular species of fungus, *Geomyces destructans*, which occurs during hibernation (Blehert et al., 2008; Gargas et al., 2009; Meteyer et al., 2009). The first occurrences of WNS in Canada were detected in the late winter of 2010 during active surveys of bat hibernacula in Ontario and Quebec; similar surveys in 2009 did not detect the disease.

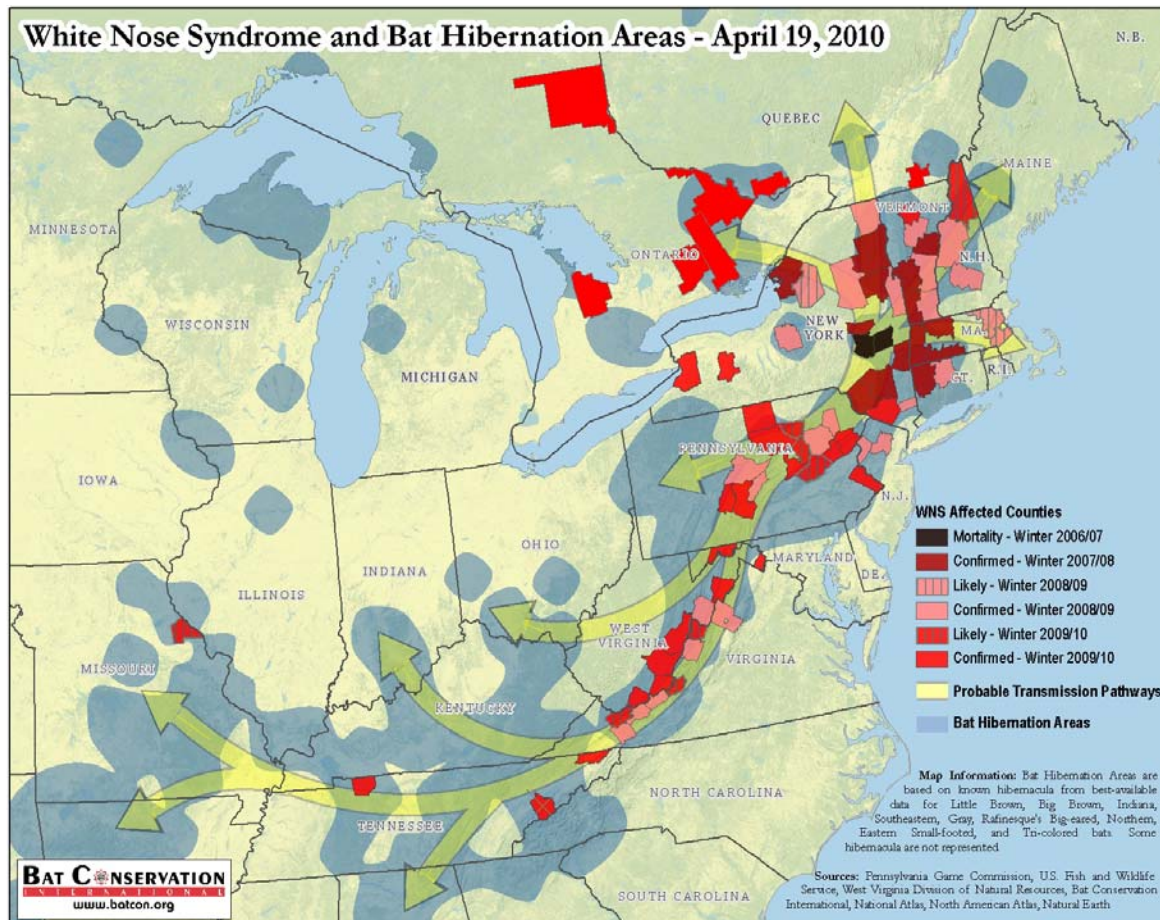


Figure 3. Spread of white nose disease in bats.

Source: Cal Butchkoski, Pennsylvania Game Commission, updated from Szymanski et al. (2009)



Figure 4. Bats with white nose syndrome, Craigmont Mine, Ontario.

Photo credit: Lesley Hale, Ontario Ministry of Natural Resources, Peterborough

Since its discovery in one hibernation cave in New York State in 2006, WNS has killed more than a million bats in the northeastern United States (Puechmaille et al., 2010). Deaths from WNS often exceed 75% of the bats in infected hibernacula, but in some hibernacula, 80 to 99% of the bats have been killed (Boyles and Willis, 2010; U.S. Fish and Wildlife Service, 2010; Puechmaille et al., 2010). Affected individuals suffer severe weight loss, and dead and dying emaciated bats have been found outside of major hibernacula during late winter, presumably searching for food when none is available and the bats should be hibernating. The bat species which have been affected as of 2010 include little brown bat (*Myotis lucifugus*), northern long-eared bat (*Myotis septentrionalis*), big brown bat (*Eptesicus fuscus*), Indiana bat (*M. sodalis*), and tri-colored bat (*Perimyotis subflavus*). Scientists are uncertain about the origin of the associated fungus, *Geomyces destructans*. The fungus, but not the disease, has been found in surveys of hibernating bats in Europe, and it has been suggested that European bats may be resistant to the disease due to a long evolutionary association with the fungus. This also suggests that the fungus may have been brought to North America quite recently (Wibbelt et al., 2010).

White nose syndrome is having an enormous impact on bat populations in North America and is likely to have a significant and long-term impact on the ecology of affected regions. The small insectivorous bats affected by the disease are long-lived and reproduce slowly. Therefore, the high mortality of adult bats associated with this disease has the potential to produce long-term population declines and extinctions. These bats consume large quantities of insects, including species damaging to agricultural crops and forests, and may play other important, if poorly understood, ecological roles (Boyles and Willis, 2010).

Parasites

Besnoitiosis

Besnoitia is a genus of protozoan parasite which develops pin-head sized firm cysts in the skin and connective tissues of its herbivore intermediate host and typical coccidial forms in the intestines of its carnivore definitive hosts. No disease due to *Besnoitia* sp. has been recognized in definitive hosts, but intermediate hosts sometimes develop disease conditions associated with severe infections (Leighton and Gajadhar, 2001). In Canada, *Besnoitia tarandi* infects caribou and reindeer, and probably muskoxen, across their ranges in the Arctic, Taiga Cordillera, Taiga Plains, Taiga Shield, Hudson Plains, Boreal Plains, and Boreal Shield ecozones⁺. Infection is very common in barren ground caribou and has been described in woodland caribou. Infection rates in muskoxen are not known (Choquette et al., 1967; Wobeser, 1976; Gunn et al., 1991; Ayroud et al., 1995). Although occasional severe manifestations of infection on the skin have been seen, most infections appear to have little or no health consequences for these species.

Besnoitia sp. (assumed to be *B. tarandi*) caused severe disease in mule deer in separate outbreaks in two zoos in the Prairies Ecozone⁺ in the 1980s (Glover et al., 1990; Leighton and Gajadhar, 2001). Based on experimental studies in cattle, it is assumed that *Besnoitia* was transmitted between infected caribou in the zoo collections and the zoos' mule deer by biting flies (Bigalke,

1967). These disease occurrences in zoos indicated that transmission between infected caribou or reindeer and mule deer is possible and they occurred at a time when the farming of native deer species, including reindeer secured from the Canadian Arctic, was developing quickly in the Prairies. Regulations were put in place to prevent the importation and maintenance of infected caribou or reindeer within the geographic range of wild mule deer. As of 2008, *Besnoitia* has not been recognized in wild cervids in Canada other than caribou and reindeer (Lewis, 1989a; Canadian Cooperative Wildlife Health Centre, 2008).

Brain worm of white-tailed deer (Parelaphostrongylus tenuis)

P. tenuis, the brain worm of white-tailed deer, is a parasitic nematode with a life cycle that involves two very different animal hosts: the white-tailed deer and several different species of terrestrial snails and slugs. The adult worms live in the connective tissue membranes on the outside of the brain (the meninges) of the deer. Here they mate and female worms deposit eggs into large veins. The eggs are carried to the lungs where larvae hatch (L₁ phase), move up the airways to the throat, are swallowed, and then expelled in the feces. These larvae burrow into snails and slugs attracted to the deer feces and develop within the snail to a new larval stage (L₃) capable of infecting deer. Deer ingest these larvae inadvertently with vegetation. The ingested larvae penetrate the intestinal wall, follow large nerves up to the spinal cord, develop briefly in the cord, and then move anteriorly along the spinal cord to their final destination on the surface of the brain, developing into adults as they go (Lankester, 2001).

In white-tailed deer, this life cycle is completed and the adult worms live on the surface of the brain for the lifetime of the deer without causing significant damage or disease. However, in moose, caribou, mule deer, elk, and domestic sheep and goats, *P. tenuis* can produce fatal disease. The larvae do more damage by remaining longer and growing larger within the cord, and produce abnormal behaviour, paralysis, and death of the host in the process. *P. tenuis* does not complete its life cycle in any of these other hosts except elk and rarely moose. In elk, infection with small numbers of L₃ larvae can result in non-lethal infection and shedding of L₁ larvae in elk feces, while infection with larger numbers of L₃ can produce fatal infection before the worms mature and produce eggs. The few moose that pass larvae are likely short-lived. Thus, *P. tenuis* acts like a biological weapon of white-tailed deer, causing fatal disease in native and some non-native ungulates that compete for food resources with white-tails.

White-tailed deer have expanded their geographical range and numbers remarkably since historic times (Banfield, 1974). In eastern Canada, this expansion has been associated with decreases in moose populations and extirpation of caribou populations, for example in Nova Scotia and Maine. Efforts to re-establish caribou on their historic ranges newly inhabited by white-tailed deer have failed because of fatal infections with *P. tenuis* (Dauphine, 1975). However, although white-tailed deer have moved westward, arriving in Manitoba in the very early 1900s and are now present across the Prairies, in western British Columbia, and north to the southern Yukon and Northwest Territories, *P. tenuis* has not moved west beyond approximately the Manitoba-Saskatchewan border in the Prairies and southern margin of the Boreal Plains ecozones*. An array of biological or climatic factors currently appears to prevent the westward spread of *P. tenuis*. These include the drier conditions of the more western parts

of these ecozones⁺ which are less favourable for terrestrial snails and slugs, the parasite's intermediate hosts.

P. tenuis is a conservation concern for native cervid species other than white-tailed deer. The rapid expansion of white-tailed deer across much of the continent is thought to be associated with human transformation of the landscape, including forestry, agriculture, removal of important predators, and some direct translocation of white-tails themselves (Banfield, 1974; Benson and Dodds, 1980; Hoefs, 2001). Translocation of white-tailed deer or elk infected with *P. tenuis* west of the Manitoba-Saskatchewan border has the potential to introduce the parasite into habitat in which it may be able to survive and complete its life cycle. For example, a related parasite with nearly identical life cycle, *P. odocoilei*, is widely distributed in western Canada, suggesting that these western environments, now inhabited with white-tailed deer, might support *P. tenuis*, if this parasite were transported across the ecological barrier that otherwise is preventing its spread. Large populations of mule deer, moose, elk, woodland caribou, and possibly wild sheep would then be at risk of significant reductions due to *P. tenuis*. Similarly, changes in climate or other ecological factors could change the distribution of *P. tenuis*, increasing or reducing its current range and impact depending on the nature of the change.

Winter tick (*Dermacentor albipictus*)

Throughout most of their range in North America, moose suffer periodic events of high mortality in late winter associated with severe infestations with winter tick, *Dermacentor albipictis*. This tick is native to North America and infests a variety of other hosts including white-tailed deer, mule deer, elk, woodland caribou, bison, and also domestic horses and cattle. However, severe infestations frequently resulting in death are common only in moose. Winter ticks occur south of approximately 60° N latitude throughout the Atlantic Maritime, Boreal Shield, southern Hudson Plains, Boreal Plains, Prairies, Montane Cordillera, Western Interior Basin, southern Boreal Cordillera, and southern Taiga Plains ecozones⁺, and severe effects on moose have been recorded over most of this range in the past 100 years (Samuel, 2004).

The winter tick hatches from eggs laid on the ground in summer. Larvae climb vegetation and attach to passing ungulate hosts in the fall. They remain on the same individual host animal, maturing from larva to nymph to adult, and mated, blood-engorged adult females drop off the host in March-April. Females lay eggs on the ground and die. Moose are not clinically affected by the ticks until March, when adults develop and begin feeding. At this point, moose suffer significant blood loss, spend abnormally large amounts of time grooming which produces significant hair loss, and spend relatively little time feeding. The consequence is rapid deterioration in nutritional condition as moose expend large amounts of energy and take in very little; this is especially the case for young moose in their first year. On average, infested moose in western Canada have had on the order of 33,000 ticks; 6% had burdens of 80,000 and as many as 150,000 ticks have been found on some individuals, 5 to 8 ticks per square centimetre of skin. By comparison, numbers on elk, white-tailed deer, and bison have been on the order of 1,000, 500, and 100 ticks per animal (Samuel, 2004).

The winter tick was first identified by western science in 1869 and has been on record as a potentially deadly parasite of moose ever since. Large-scale mortality events often are described in association with severe cold, snow, and other challenging conditions for moose in March and April, including inadequate nutrition from poor habitat. Losses on the order of 1,000 out of a total population of 5,000 moose were reported in Riding Mountain National Park in 1999, for example (Samuel, 2004). Often, these events occur over a very large area, for example simultaneously across the entire Boreal Plains, Prairies, and western half of the Boreal Shield ecozones⁺ in 1999. It is clear that weather events affect the abundance of the ticks, particularly conditions in April when gravid adult female ticks drop to the ground and either do or do not survive to lay eggs, thus affecting the numbers of larvae available to infest moose the following fall. Environmental conditions also affect the resilience of the moose, particularly conditions in late winter and early spring the following year when infested moose must endure the ticks. The historical record does not provide data sufficient to determine any temporal trend in the effect on moose populations by the winter tick. Neither is it possible to make predictions based on climate change scenarios and existing knowledge of host and tick ecology. Winter ticks have been imported into southwestern Yukon (Boreal Cordillera Ecozone⁺) by way of importation and release of infested elk and there is reason to believe that translocation of the tick further west into Alaska, where currently it does not exist, would result in establishment of the tick in many of Alaska's moose populations (Zarnke et al., 1990; Samuel, 2004; Merchant, P., pers. comm.). Hunters along the MacKenzie River in the Northwest Territories recently have reported moose in spring with severe hair loss typical of winter tick infestation, a phenomenon new to the traditional knowledge of local First Nations (Elkin, B.T., pers. comm.).

PATHOGENS WITH LIMITED DISTRIBUTION

Gulf of Maine and Scotian Shelf and West Coast Vancouver Island ecozones⁺

Plastic ingestion by marine birds

Authors: Pierre-Yves Daoust and Zoe Lucas

Ingestion of waste plastic by pelagic marine birds has been studied both to assess the impact on the birds themselves and as an index of pollution of the seas with plastic debris (Sievert and Sileo, 1993; Blight and Burger, 1997; Van Franeker, 2008). In the eastern Atlantic/North Sea, the number of avian species documented to have ingested plastic increased from two in the 1960s to more than 109 by the late 1980s. At the same time, global production of plastic increased from 30 million tonnes per year in the 1970s to 150 million tonnes per year in 2000 (Vlietstra and Parga, 2002).

Birds of the order Procellariiformes, which includes albatross, shearwaters, and northern fulmars, appear to ingest the largest amounts of plastic. Of 50 carcasses of northern fulmars collected between 2001 and 2005 during beached bird surveys on Sable Island (200 km east of Halifax, Nova Scotia), the stomachs of 49 contained plastic and/or styrofoam which varied in total weight from 0.015 to 13 g per bird (average, 1.7 g) (Walther et al., 2008) (Figure 5).



Figure 5. Indigestible material from the stomach of one northern fulmar from Sable Island, Nova Scotia. Source: Walther et al. (2008)

In 1987, plastic was found in the stomachs of 75 to 100% of eight different species of Procellariiformes caught in pelagic drift nets off the coasts of British Columbia (West Coast Vancouver Island Ecozone⁺), Washington, and Oregon (Blight and Burger, 1997). It is not clear to what extent plastic ingestion is harmful to these birds, although some evidence of nutritional problems has been reported (Sievert and Sileo, 1993). There is a clear trend for increasing pollution of the seas with plastic debris that can be and is ingested by marine birds (Sievert and Sileo, 1993; Blight and Burger, 1997).

Prairies Ecozone⁺

Chronic wasting disease [Reportable]

Chronic wasting disease (CWD) is a fatal disease of members of the deer family (cervids) resulting from ingestion of a misfolded version of a normal body protein called the prion protein (Pruisner, 1982; Williams et al., 2001). Such diseases are classified as transmissible spongiform encephalopathies; other diseases in this same class include scrapie in sheep, bovine spongiform encephalopathy in cattle, and Creutzfeldt-Jacob disease in people. These diseases result in progressive deterioration of the brain associated with accumulation in the brain of misfolded prion protein. All typically have long intervals (years) between exposure to the misfolded prion protein and the first clinical signs of disease. There are no conclusive medical tests to identify people or animals affected by these diseases prior to the onset of clinical signs, and a definitive diagnosis generally only occurs at autopsy. The abnormally-folded prion proteins which cause these diseases resist most available forms of deactivation such as formaldehyde, disinfectants, heat, and UV light. No effective vaccines or drugs currently are available to prevent or treat these diseases (Williams et al., 2001).

CWD was first recognized as a clinical disease in 1967 among mule deer housed at a research station in Colorado (Williams et al., 2001). It was first recognized to be a form of spongiform encephalopathy in 1978. At about this same time, it was found in captive mule deer in Wyoming and captive elk in Colorado, and then in wild elk, mule deer, and white-tailed deer in these same states. Since these first discoveries, the disease has spread widely in the United States and Canada (Figure 6), mainly in the Prairies Ecozone⁺ but also in the Boreal Plains Ecozone⁺, often in association with sale and transport of farmed cervids.

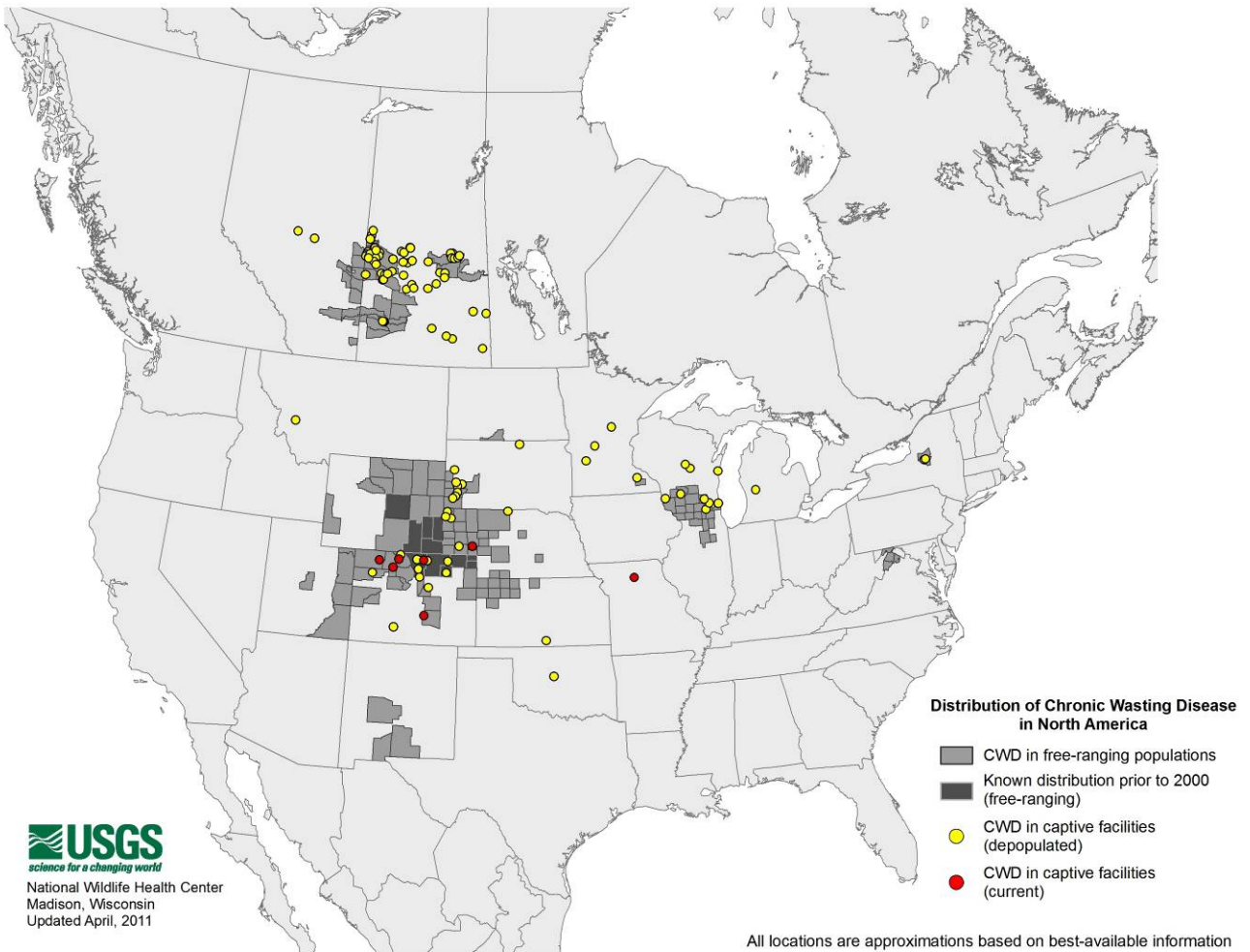


Figure 6. Distribution of chronic wasting disease in North America, 2011.

Depopulated means that all cervids on the farm were killed by government authorities as per North American response to CWD.

Source: USGS National Wildlife Health Centre (2011)

CWD was first reported in Canada in 1996 and again in 1998 in captive elk on game farms in Saskatchewan (Kahn et al., 2004). An eradication program for CWD in farmed cervids was carried out between 2000 and 2004, during which approximately 18,000 animals were destroyed on 42 farms in Saskatchewan and Alberta (Figure 6). However, in 2000, CWD was detected in a wild mule deer on the Saskatchewan side of the Saskatchewan-Alberta border. Since this first detection in wild populations, CWD has been detected in wild cervids progressively in four separate geographical areas of the Prairies Ecozone⁺ in mule deer, white-tailed deer, and elk (Figure 7). These affected areas have grown in size and the proportion of hunter-killed animals found affected with CWD within these areas has increased slowly but steadily since 2000 (Canadian Cooperative Wildlife Health Centre, 2008). Table 2 shows the data for Saskatchewan from 1997 to March 2009.

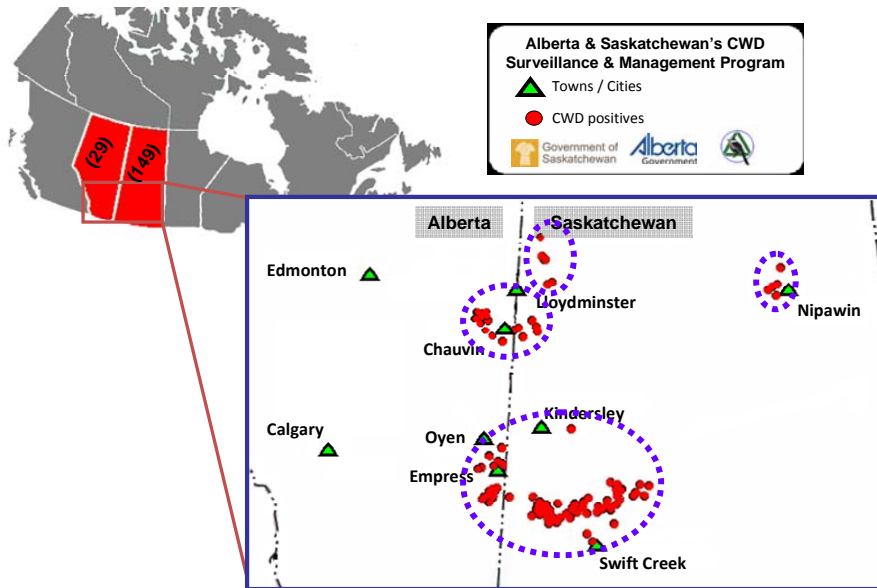


Figure 7. Detections of chronic wasting disease in wild deer in the Prairies.
 Source: Canadian Cooperative Wildlife Health Centre (2008); Bollinger, T., pers. comm.

Table 2. Summary of chronic wasting disease in wild cervids in Saskatchewan, 1997-March 2009.

Year	Mule Deer		White-tailed Deer		Elk		Moose		Total	
	Pos	Neg	Pos	Neg	Pos	Neg	Pos	Neg	Pos	Neg
1997 ¹	0	3	0	36	0	0	0	0	0	39
1998 ¹	0	91	0	18	0	2	0	0	0	111
1999 ¹	0	79	0	57	0	44	0	0	0	180
2000 ¹	1	184	0	726	0	89	0	0	1	999
2001 Spring ¹	1	154	0	58	0	0	0	0	1	212
2001 Fall	0	1,077	0	2,236	0	340	0	0	0	3,653
2002 Spring	1	134	0	24	0	0	0	0	1	158
2002 Fall	7	3,244	2	2,413	0	163	0	0	9	5,820
2003 Fall	22	2,830	0	1,922	0	153	0	0	22	4,905
2004 Fall	30	5,265	2	1,439	0	9	0	0	32	6,713
2005 Fall ²	25	2,635	10	938	0	47	0	4	35	3,624
2006 Fall ²	27	2,343	22	1,497	0	164	0	10	49	4,014
2007 Fall ²	32	2,561	13	1,729	2	90	0	4	47	4,384
2008 Fall ²	43	3,668	5	828	1	214	0	27	49	4,793
Total	189	24,268	54	13,921	3	1,315	0	45	246	39,549

Notes: This table does not include those specimens that were deemed unsuitable for testing.

1- Prior to the fall of 2000, only the brain section was used to diagnose CWD. Starting in 2001, tonsils and/or retropharyngeal lymph nodes were also examined.

2- Starting in the fall of 2005, animals under one year of age were no longer tested under the program because detectable infection is rare in a young animal and therefore not cost effective in terms of surveillance.

Source: Canadian Cooperative Wildlife Health Centre, Western and Northern Regional Centre, unpublished data

CWD is a serious ecological and economic concern to Canada. The approximately 1.8 million white-tailed deer, 350,000 mule deer, 100,000 elk and 900,000 moose in Canada are susceptible to CWD and there are no natural barriers to prevent its spread from its current locations to the rest of the country (Bollinger et al., 2004). It is not known whether or not Canada's 4 million caribou also are susceptible to CWD. CWD is transmitted animal to animal through body secretions such as saliva, but also can be transmitted through exposure of susceptible animals to environments contaminated by the misfolded CWD prion protein, which persists for exceptionally long periods (Miller and Williams, 2003; Mathiason et al., 2006).

CWD is a new disease, caused by a whole new class of disease-causing agent, and thus predictions of its potential effects on cervid populations can only be approached through modelling. Such models are uncertain because large data sets and well-documented model parameters such as transmission rates for CWD are not available. However, as explored through models, a negative impact of CWD on wild cervid populations in the long term (100 years), moderate to extreme in extent, appears the most reasonable expectation (Gross and Miller, 2001). Wild cervids support a variety of commercial and subsistence human economies in Canada which form a large portion of the approximately \$12 billion of Canada's gross domestic product (1996) associated with wildlife (Federal-Provincial-Territorial Task Force on the Importance of Nature to Canadians, 2000). These species also are ecologically important herbivores across all terrestrial ecozones[†]. Thus, unless effective means of controlling the disease are developed and implemented, CWD is likely to reshape landscapes and economies in Canada in the 21st century.

Botulism type-C

Botulism is a form of food poisoning associated with ingestion of powerful toxins produced by various strains of the bacterium *Clostridium botulinum*, designated types A, B, C and so on, according to the type of toxin each produces. Although it has occurred in other ecozones[†], type-C botulism, from ingestion of type-C botulinum toxin, has caused large and recurrent epidemics only in the Prairies Ecozone[†]. Waterfowl, especially ducks, regularly are affected by type-C botulism, with peak occurrence in July and August. Type-C botulism in ducks is a disease of complex ecology (Wobeser, 1997a; Rocke and Bollinger, 2007). *C. botulinum* is a decomposing bacterium which persists for long periods in soil and sediments as resistant spores and which proliferates in nutrient-rich environments such as animal carcasses. The bacterium carries no gene for botulinum toxin but can receive this gene when infected by a carrier bacteriophage virus. Epidemic mortality from type-C botulism occurs when toxin is produced and then ingested by susceptible animals. In the context of waterfowl on prairie wetlands, fly larvae (maggots) growing in carcasses of dead animals are the principal agents of toxin transfer from the dead to the living. Maggots are highly attractive food for birds of all kinds and are eaten voraciously. Maggots absorb but are unaffected by type-C toxin. Thus, the risk of epidemics of type-C botulism is highest when *C. botulinum* and toxin gene-carrying bacteriophage viruses are present in the environment, temperatures are within the range of flesh fly activity (above about 15C) and toxin production, a source of animal carcasses is present, and many susceptible ducks or other birds are present to find and ingest any toxin-bearing maggots that develop. Under

these conditions, a cycle of positive feedback occurs in which the first toxin-bearing maggots are eaten by birds which then die and are substrate for putrefaction and further production of toxin-bearing maggots. Under these conditions, epidemics can grow logarithmically with each cycle of toxin production, transfer to living birds and consequent mortality adding to the biomass of putrefying carcasses producing toxin and maggots. Factors that reduce risk are high scavenging rates that remove carcasses, especially early in the season when only a few carcasses may be present, ambient temperatures below about 15° C, and a low density of susceptible live birds (Wobeser, 1997a; Rocke and Bollinger, 2007)

The alkaline wetlands of the Prairies Ecozone⁺ are habitats favourable to type-C botulism. It is likely that some mortality from botulism occurs every summer in this ecozone⁺, but often the outbreaks are small and go undetected. Some very large epidemics have been documented, however. In the mid-1990s, repeated years of high mortality occurred in southern Alberta, Saskatchewan, and Manitoba. For example, over 100,000 dead ducks were counted in late fall at Old Wives' Lake in southern Saskatchewan in 1996, and a season-long study at this site in 1997 found total mortality from June to October to have been approximately one million birds (Rocke and Bollinger, 2007; Canadian Cooperative Wildlife Health Centre, 2008). These outbreaks were associated with drought conditions during which many of the small wetlands used by waterfowl for nesting were dry and large numbers of birds were concentrated on a small number of large wetlands where suitable habitat remained available. There was a marked reduction in mortality from botulism in subsequent years when precipitation relieved drought conditions (Canadian Cooperative Wildlife Health Centre, 2008).

C. botulinum and their bacteriophages are persistent residents of most wetlands in the Prairies Ecozone⁺. Thus, type-C botulism remains a potential source of mortality of wetland birds (virtually all species are susceptible to lethal intoxication). Occurrence of epidemics has been associated with factors such as low rainfall and high temperatures, both of which are predicted trends for the Prairies Ecozone⁺ in the coming decades (Ogden et al., 2006). The draining and other destruction of wetlands in the past 50 years already has greatly reduced the amount of habitat available to aquatic birds, and, by concentrating birds on remnant wetlands, this also may contribute to an increased risk of type-C botulism.

Newfoundland Boreal Ecozone⁺

Muscle worm of caribou (Elaphostrongylus rangiferi) [Immediately Notifiable]

The nematode parasite *Elaphostrongylus rangiferi* was brought to the island of Newfoundland in 1908 in infected reindeer, from which it spread to native caribou. It is now found in all major caribou populations on the island (Ball et al., 2001). The majority of infections appear to produce very little clinical disease, but infection with large numbers of worms can result in severe disease affecting the brain, a condition known as cerebrospinal elaphostrongylosis or CSE. The life cycle of this parasite is similar to that of the brain worm of white-tailed deer,

Parelaphostrongylus tenuis (see page 22), although the adult worms come to rest in the connective tissue surrounding muscles, especially of the limbs, after undergoing a developmental stage on the surface of the brain (Lankester, 2001).

Two outbreaks of high mortality due to CSE in Newfoundland caribou have been recorded: in central Newfoundland in the mid 1980s and on the Avalon Peninsula beginning in 1996. In the latter outbreak, the affected caribou population was reduced from 7,000 to 2,500 animals over a three year period (Lankester and Fong, 1989). CSE in caribou in Newfoundland is associated with ingestion of large numbers of infective larvae and this, in turn, appears associated with moist conditions and moderate summer temperatures combined with mild winters which extend the period during which the intermediate hosts of the parasite, terrestrial snails and slugs, remain active in the fall and early winter and thus available to be eaten by grazing animals.

E. rangiferi in Newfoundland poses several different conservation and economic risks. Other cervids, as well as domestic goats, are susceptible to infection and to CSE. Moose are abundant and economically valuable in Newfoundland. Thus the parasite represents a potential threat to both caribou and moose populations on the island. Outbreaks of CSE, the severe disease associated with intense infections with *E. rangiferi*, appear related to climate in ways that may result in more frequent and intense outbreaks if winters become warmer. In North America, *E. rangiferi* exists only on the island of Newfoundland. Any translocation of infected animals from this island to the continent risks the introduction of a parasite that may readily infect and cause disease in most or all native cervids and some domestic livestock species. One such translocation of Newfoundland caribou already has taken place, fortunately from a herd that, at the time, probably was not infected with *E. rangiferi* (McCollough and Connery, 1991).

Montane Cordillera Ecozone⁺

Adenovirus hemorrhagic disease of deer

Author: Gary Wobeser

Adenovirus hemorrhagic disease (AHD) is a severe virus disease of cervids (deer family) that has been recognized only in North America, and only since 1987. Knowledge of the geographic range and host species range of this virus has increased in the past two decades and AHD was detected for the first time in Canadian wildlife in 2006.

AHD was first identified during an outbreak that killed thousands of deer in California in 1993 (Woods et al., 1996). Subsequently, examination of archived tissues revealed that adenovirus had caused an earlier outbreak in California in 1987 (Woods, 2001). AHD occurred in wild black-tailed deer in Oregon in 2001 and killed an estimated 400 or more black-tailed deer in one area of Oregon in 2002 (Oregon Department of Fish and Wildlife, 2002). AHD also has been diagnosed in captive deer: black-tailed deer in California, white-tailed deer in Iowa, and moose at the Toronto zoo (Boyce et al., 2000; Sorden et al., 2000; Shilton et al., 2002).

In 2006, AHD was detected for the first time in wild deer in Canada, in Waterton Lakes National Park in southwestern Alberta (Montane Cordillera Ecozone⁺) (Canadian Cooperative Wildlife Health Centre, 2008). In July and August, nine mule deer fawns died in the park town site (mule deer and black-tailed deer are the same biological species). The virus of AHD was identified in tissues taken from two of these fawns, as were pathological changes typical of this disease (Woods et al., 1999; Wobeser, 2006).

A factor that complicates understanding the distribution and significance of AHD is its close resemblance to the more common hemorrhagic diseases of North American deer caused by orbiviruses (EHD and BT, see hemorrhage diseases of deer on page 7). Only detailed laboratory analysis can distinguish AHD from EHD or BT; their clinical characteristics are identical. The apparent increase in the geographic and host range of AHD since 1987 may reflect no more than improved mechanisms of detection and identification. Several more decades of careful disease surveillance will be required to document any trends in its occurrence or its significance to host animal populations in Canada.

Western Interior Basin and Montane Cordillera ecozones⁺

Wild sheep pneumonia syndrome

Author: Helen Schwantje

The historic decline of Rocky Mountain bighorn sheep in western North America is due, at least in part, to mortality from pneumonia. This disease syndrome has occurred in the Western Interior Basin and Montane Cordillera ecozones⁺ of British Columbia and Alberta, and in Rocky Mountain and California bighorn sheep ecotypes. The syndrome occurs as epidemics which are characterized by sudden or more gradual mortality at rates ranging from 10 to over 80% of affected populations. Low survival rates of lambs and periodic mortality of adults from pneumonia can persist for decades in sheep populations following these epidemics. Die-offs due to this syndrome have occurred in several herds constituting a metapopulation in southeastern British Columbia (Montane Cordillera Ecozone⁺) every 20 years from the early 1920s to, most recently, the 1980s. A second metapopulation in the Okanagan region (Western Interior Basin Ecozone⁺) was affected by an all-age die-off in 1999-2000. Die-offs in Alberta appear to occur sporadically in single herds on the eastern slopes of the Rocky Mountains (eastern Montane Cordillera Ecozone⁺). Die-offs have not been reported to occur in thimhorn sheep, but pneumonia in individuals has been reported sporadically (Schwantje, H., unpublished data).

Bacteria of the genera *Pasteurella* and *Mannheimia* have been the most commonly identified causes of pneumonia in bighorn sheep. However, other pathogens are often present as well, including viruses, other bacteria, and lungworm parasites, and combinations of these pathogens are thought to affect the occurrence and severity of disease outbreaks (Miller, 2001; Garde et al., 2005). Other factors that have been associated with the occurrence of bighorn all-age die-offs in Canada include reduction, fragmentation, and overall poor quality of habitat, inter- and intra-

specific competition on winter ranges, human-related stressors such as intense disturbance of critical winter range habitat, inclement weather, and close contact with domestic sheep (Miller, 2001; Garde et al., 2005). Wild sheep are susceptible to a number of strains of *Pasteurella* and *Mannheimia* that may be carried by domestic sheep and goats, and disease outbreaks have occurred following close contact between these species (Rudolph et al., 2003; Garde et al., 2005; George et al., 2008).

Decline in bighorn sheep in Canada is a trend of the past several decades. Wild sheep pneumonia syndrome appears to be an outcome of multiple stressors imposed on wild sheep populations by human activities. One of the most important issues for wild sheep survival in Canada is contact with domestic sheep and goats, and the virulent pathogens these domestic species can carry and transmit to wild sheep. Habitat conservation, restoration and management, and exclusion of domestic sheep and goats from wild sheep habitat are considered the best approaches to reducing the impact of wild sheep pneumonia.

Great Lakes and Mixedwood Plains ecozones⁺

Botulism type-E

Author: D. Campbell

Botulism is a form of food poisoning associated with ingestion of powerful toxins produced by various strains of the bacterium *Clostridium botulinum*, designated types A, B, C and so on, according to the type of toxin each produces. The strains of *C. botulinum* that produce type-E toxin are most common in aquatic and marine environments. The toxin can affect mammals, birds, amphibians, and fish. In 1963 to 1968, epidemic mortality due to type-E botulism occurred in fish-eating birds on Lake Michigan (U.S.). Thousands of loons, gulls, grebes, and mergansers were found dead on the lakeshore. This epidemic was coincident with a major change in the fish community of Lake Michigan in which native species declined and the invasive alewife (*Alosa pseudoharengus*) became the predominant fish in the lake (Fay, 1966; Fay, 1969). From the 1970s to the 1990s, type-E botulism occurred only sporadically in wild birds in North America. However, in 1998, an outbreak on Lake Huron became the first in a series of annual outbreaks on the Great Lakes that has expanded to include all of lakes Erie and Ontario and has affected at least 22 different species of wild birds (Figure 8) (Rocke and Bollinger, 2007; Leighton, 2007; Campbell, 2008; Canadian Cooperative Wildlife Health Centre, 2008).

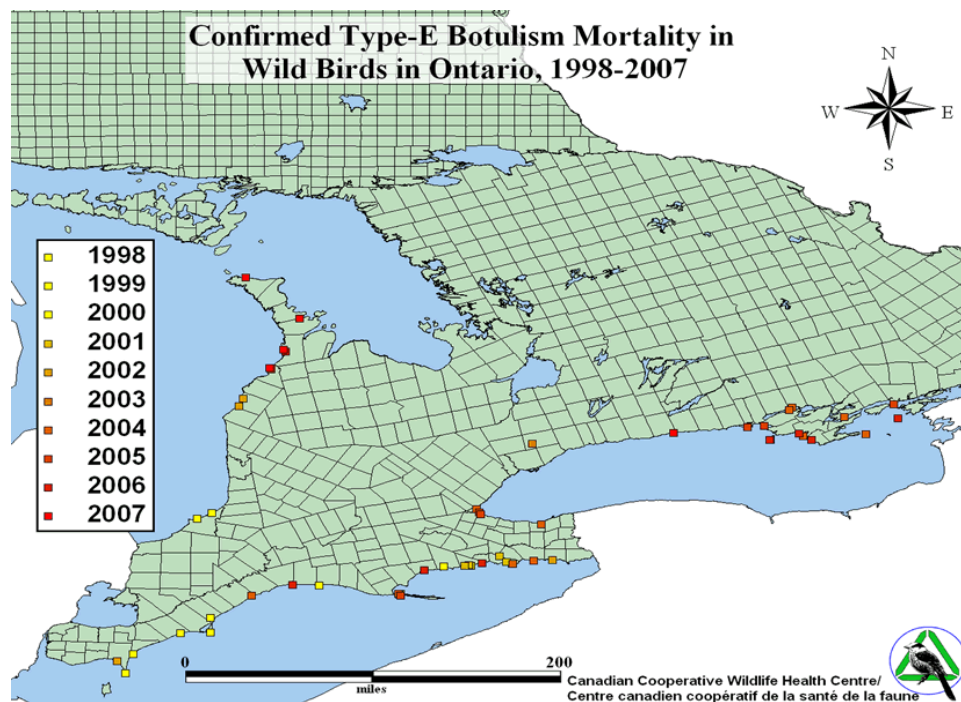


Figure 8. Distribution of confirmed type-E botulism mortality in wild birds in the Mixedwood Plains and Great Lakes ecozones⁺, 1998-2007.

Source: Canadian Cooperative Wildlife Health Centre (2007)

The first outbreak occurred on the southeastern shore of Lake Huron in autumn 1998, when type-E botulism killed hundreds of common loons. In 1999 and 2002, outbreaks in this same area killed gulls (*Larus* spp.) and grebes (*Podiceps* spp.) as well as loons. Lake Erie was first affected in 1999; dead gulls were found on the southern shore, at Presque Isle Pennsylvania, in the summer, and in autumn, type-E botulism killed about 6,000 red-breasted mergansers, loons, and grebes along the north shore of the west basin of Lake Erie. Over the next four years, the epidemic on Lake Erie acquired a regular pattern of small-scale mortality events (tens to hundreds affected in each) in summer of resident gulls, terns, double-crested cormorants, and shorebirds (Scolopacidae), and larger outbreaks (many hundreds to many thousands affected in each) in autumn of southbound migrant fish-eating birds (mainly red-breasted mergansers, common loons, grebes) and diving ducks (mainly long-tailed ducks). Fish-eating birds and diving ducks generally died off-shore and carcasses would be found on the leeward shore (Leighton, 2007; Canadian Cooperative Wildlife Health Centre, 2008).

The location of major outbreaks on Lake Erie shifted from the west basin (1999) to both the central basin and east basin (2000-2004), and then mostly the east basin (2002-2004). In 2000, about 6,000 fish-eating birds washed onto the New York shore at the east end of the lake; in 2001, 3,000 gulls, fish-eating birds and long-tailed ducks died along the New York shore; in 2002, over 3,000 ring-billed gulls died near Buffalo New York, and 12,600 long-tailed ducks and over 3,000 fish-eating birds came ashore on the New York coast; in 2003, 2,000 loons and hundreds of gulls and long-tailed ducks died on both sides of the east basin; in 2004, about

2,800 loons, 2,700 long-tailed ducks, and hundreds of birds of other species were found on the New York shore (Leighton, 2007; Canadian Cooperative Wildlife Health Centre, 2008).

Type-E botulism was first confirmed on Lake Ontario in 2002, when it occurred in gulls and affected about 675 long-tailed ducks along the New York shore. About 1,500 deaths attributed to botulism occurred in gulls, diving ducks, cormorants, and loons on the New York side of Lake Ontario in 2003, and botulism also occurred in great black-backed gulls at the east end of the lake on the Canadian side. In 2004, over 1,750 carcasses were counted on breeding colonies and beaches at the east and west ends of Lake Ontario in late summer and fall, mainly of double-crested cormorants, great black-backed gulls, long-tailed ducks, and white-winged scoters. On the New York shore, about 1,700 birds died, including long-tailed ducks, ring-billed gulls, cormorants, and common loons. Since 2004, botulism has occurred annually on Lake Ontario, following this same general pattern of incidents involving gulls, terns, and cormorants during the summer months, and diving ducks, loons, and grebes in the autumn (Leighton, 2007; Canadian Cooperative Wildlife Health Centre, 2008).

The source of the toxin in these epidemics and the ecology of type-E botulism in the Great Lakes has yet to be fully determined. Identification of the stomach contents of birds dying during botulism events indicates that a substantial proportion of affected fish-eating birds had fed recently on round gobies (*Neogobius melanostomus*). This fish is an invasive species, having arrived within the last 15 years from the Black Sea area, likely in the ballast water of ocean-going ships. This goby is a predator of the zebra and quagga mussels (*Dreissena polymorpha* and *Dreissena rostriformis bugensis*), which also are newly-introduced non-native species originating from the Black Sea area (zebra mussel) and Dnieper River in the Ukraine (quagga mussel). These mussels themselves have been found in the stomachs of many of the diving ducks found dead during botulism episodes. Thus, there appears to be a link between these invasive mussels and gobies, and type-E botulism in the Great Lakes Ecozone[†]. It is possible that the mussels are dying in large numbers due to an unknown cause. Their tissue, in an anoxic environment within their closed shells, could provide the substrate for growth of *C. botulinum* and toxin production. Consumption of these dead mussels by either ducks or fish would move toxin, respectively, directly to mussel-eating ducks or, via gobies, upwards in the food chain to fish-eating birds (Rocke and Bollinger, 2007; Leighton, 2007).

Therefore, based on current evidence, this epidemic of type-E botulism in birds on the Great Lakes appears linked to the changed ecology of the lakes associated with at least three introduced species. While the scale of mortality in birds is locally impressive and alarming, it may not be high enough at present to have a significant impact on continental or global populations of the species suffering the highest mortality (for example, common loon, population of 500,000 to 700,000; red-breasted merganser, 249,000 in Canada and Alaska; double-crested cormorant, over 226,000 breeding pairs (Canadian Wildlife Service, 1996; Rose and Scott, 1997; Wires and Cuthbert, 2006). The epidemic has shown a rapid eastward expansion but also a slower expansion northward along the Bruce Peninsula and into Georgian Bay (Figure 8). A better understanding of the ecology of type-E botulism on the Great Lakes is required before any possibly useful predictions of its future extent or behaviour can be made.

Viral hemorrhagic septicemia virus type-IV 'b'

Author: J. Lumsden

Viral hemorrhagic septicemia (VHS) viruses infect freshwater and marine fish. Infection may result in severe and fatal disease or have little effect on the fish, depending on the virus strain and the species of fish involved. However, VHS viruses are important pathogens in aquaculture and are therefore listed by the World Organization for Animal Health as pathogens against which trade restrictions can be declared by importing nations. VHS viruses have been classified into four different genotypes (I, II, III, and IV), of which only genotype-IV currently occurs on the Atlantic and Pacific coasts of North America (Gagne et al., 2007). A new strain of VHSV type-IV, referred to as type-IV 'b', was discovered in the Great Lakes Ecozone⁺ in Lake Ontario in 2005 associated with a large mortality event in freshwater drum (*Aplodinotus grunniens*) (Lumsden et al., 2007) and was subsequently identified in Lakes Erie, St. Clair, Huron, and Michigan, and in inland fresh water bodies in New York, Michigan, Wisconsin, and in the Thames River in Ontario. From archived samples of muskellunge from Lake St. Clair, this virus is now known to have been present in the ecozone⁺ at least since 2003 (Elsayed et al., 2006). The western Atlantic strains of type-IV are most closely related to the newly recognized Great Lakes strain (Gagne et al., 2007) but the origin of this type-IV 'b' strain is not known. All of the strains of type-IV 'b' isolated from the Great Lakes have been virtually identical (Garver, K., pers. comm.), which suggests either a recent evolution or recent arrival of this virus strain.

The most important pathogenic feature of this type-IV 'b' strain is its very broad host range; dozens of different fish species now are known to be susceptible to infection. To date, freshwater drum, yellow perch, muskellunge, gizzard shad, and the introduced round goby seem to have experienced the most significant mortalities. Assessment of the true impact of the virus will require the sustained efforts of fisheries agencies on both sides of the border, as population surveys are performed and year-class recruitment numbers are documented, particularly for high profile or commercially important species like muskellunge and yellow perch. Economically, the virus already has had a substantial effect (Garver, K., pers. comm.) on the day-to-day operations of baitfish harvesters, aquaculture and fish enhancement activities, on anglers, and on fish health personnel around the Great Lakes, all due to the restrictions imposed in Canada and the United States on trade in Great Lakes fish when this virus was discovered.

INTEGRATED SUMMARY

Disease occurrence patterns in Canadian wild vertebrates in the past two decades appear to conform to the general global pattern of dynamic change and progressive emergence of disease (Jones et al., 2008). At least 14 of the 27 pathogens discussed in this report cause diseases in Canadian wildlife that fit the definition of emerging diseases. Seven pathogens – the raccoon strain of rabies virus, West Nile virus, the house finch variant of *Mycoplasma gallisepticum*, chronic wasting disease prion, adenovirus of deer, and the Great Lakes variant of viral hemorrhagic septicemia – are new to Canada and appeared during the preceding two decades. Seven others – Newcastle disease virus, *Pasteurella multocida* (avian cholera), Lyme disease, bovine tuberculosis, chytrid fungus, type-E botulism and *Elaphostrongylus rangiferi* – have changed their patterns of occurrence significantly during the same period. There are plausible explanations for the emergence of some of these diseases: for example, type-E botulism occurring in association with invasive species causing radical change in the ecology of the Great Lakes; the range of Lyme disease expanding as a function of climate warming and its effect on the tick vector; the introduction of West Nile virus to North America in 1999 and of CWD prion to Canada in the 1980s. For others, the factors responsible for the evident changes in patterns of occurrence have not been identified and may be both multiple and complex (Plowright et al., 2008). The unprecedented scale and speed of environmental change which has characterized the 20th century is likely to be maintained or accelerated in the 21st century (McNeill, 2000; Diamond, 2005). Disease emergence and changing patterns of disease occurrence in Canada's wild vertebrates are likely to continue and perhaps intensify in the coming decades in response to these intense environmental variables and their effects on the interactions among animals and their pathogens.

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