A REVIEW OF POTENTIAL HEALTH HAZARDS TO HUMANS AND LIVESTOCK FROM CANADA GEESE (*Branta canadensis*) AND CACKLING GEESE (*Branta hutchinsii*)

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The Canadian Wildlife Service

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On behalf of
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1. EXECUTIVE SUMMARY

Canada and Cackling geese are protected under Canada's *Migratory Birds Convention Act*[1]. This act prohibits capturing or killing of geese, or damaging, destroying, removing, or disturbing their nests, except as provided for under the *Migratory Birds Regulations*[2]. Canada geese, along with all other migratory birds, are protected and managed by the Canadian Wildlife Service (CWS) of Environment Canada. The *Migratory Birds Regulations* provide for management actions under permit to "remove or eliminate migratory birds or nests where it is necessary to do so to avoid injury …" CWS requested the Canadian Cooperative Wildlife Health Centre (CCWHC) to perform a risk assessment in order to support their decision-making for permit issuance for removing protected geese because of concerns for human or livestock health.

The most significant conclusion of this review was that there is an insufficient basis in available evidence to conduct a reliable and meaningful risk assessment of infectious hazards. Most of the data requirements to make a valid assessment of risks simply could not be met with the available data on pathogens potentially transmitted by geese. There were very large gaps in some of the following key determinants of risk: prevalence of pathogens and parasites in geese, epidemiological information to link the pathogen in geese to cases in people or livestock, goose fecal distribution patterns, and human or livestock exposure patterns (i.e. the nature and extent of contact between geese and humans and geese and livestock). As a result, a reliable, evidence-based risk assessment of health risks to people or livestock from free-ranging geese could not legitimately be performed with the existing availability and quality of data.

Hazard identification

There was evidence that many pathogens of importance to humans and livestock can infect Canada and Cackling geese and be shed into the environment by geese, and vice versa. However, there was a very large gap in our understanding of the ecological and epidemiological factors that may contribute to the transmission from geese to humans or livestock. As discussed above, effective risk analyses (identification, assessment, management and communication) require data inputs from quality surveillance information (Table 1). Although there was a large list of pathogens associated with geese and other members of the Anatidae family, there was scarce serological, microbiological or epidemiological data as evidence of transmission between geese and humans and geese and livestock.

Release assessment

We can conclude that there are several plausible routes of exposure to infectious material from geese, but estimates of the probability of these exposures are not attainable with existing data.

[1] http://www.cws-scf.ec.gc.ca/legislations/laws1_e.cfm
[2] http://laws.justice.gc.ca/en/M-7.01/C.R.C.-c.1035/

The most plausible source of infection for both people and livestock appears to be via contaminated aquatic environments. Another plausible route is through direct contact with infected fecal material (this includes infected airborne particles). These routes provide the opportunity for the widest possible exposure for both people and livestock. In this report we highlight some of the pathogens that we feel should be of greatest concern to CWS and as a result be considered in their risk management and communication strategies. However, it must be noted that none of the pathogens examined had sufficient evidence (i.e. temporality, specificity, consistency, etc) to demonstrate a causal relationship between exposure to a pathogen of goose origin and a resulting infection in either people or livestock.

Due to the fact that Canada and Cackling geese are aquatic birds that aggregate in large numbers and produce a prolific amount of feces, concerns have been raised about their role in water contamination. As a result, there has been an emphasis on research of waterfowl-related pathogens and in particular on *E. coli, Cryptosporidium,* and *Giardia*. This research bias could affect the assessment of risks from waterborne transmission from geese in that more important, but unresearched pathogens are not considered as much as these three pathogens. In addition, the perceptions of waterborne pathogen risks are shifting as a consequence of more recent molecular and epidemiological information that is coming on stream and showing more host specificity than previously thought. Hence, the assessment of pathogens in this report might be biased by the older literature that could not make distinctions between strains that may be highly host-specific and therefore present a diminished risk for interspecies transmission (Hansen et al, 2009; Graczyk et al, 2008).

Consequence assessment

In this report, the pathogens that were categorized as 'high' potential impact to human health had more than one of the following features: 1) are capable of causing severe illness that can result in hospitalization; 2) had the ability to spread epidemically from person-to-person; 3) had high fatality rates; 4) did not have effective treatment or preventive methods; or 5) were nationally reportable. The pathogens that filled more than one of these criteria included: highly pathogenic avian influenza (HPAI), enterotoxigenic E. coli (ETEC), and West Nile virus. Pathogens of medium and low impact are shown in Appendix C. In livestock, the pathogens with a 'high' ranking had more than one of the following features: 1) are capable of causing severe illness; 2) had the ability to spread epidemically within or between herds or flocks; 3) had high fatality rates; 4) did not have effective treatment or preventive methods; or 5) were nationally reportable to the Canadian Food Inspection Agency. The pathogens that filled more than one of these criteria included: highly pathogenic avian influenza, *Mycobacterium avium*, Newcastle disease, and West Nile virus. We found no direct evidence that linked human or livestock health outcomes to geese. Hence, this consequence assessment is the consequence of the pathogens in general and not the pathogen from geese specifically. Almost all of the hazards listed have multiple sources including people, other species and environmental sources.

Synthesis

Major deficits exist in our understanding of the frequency of disease-causing agents in free-ranging geese. Perhaps more importantly, there was virtually no information on the frequency or probability with which pathogens from wild geese are transmitted from geese to people or livestock. We found a fairly extensive list of potential infectious and parasitic pathogens from geese that could infect people, poultry or other livestock and thus the potential for shared diseases exists. We concluded that the nature of the risks are not generic across Canada and are very context specific. The risks are affected by the potential exposure pathway (with waterborne being the route of most concern); people or livestock being exposed (immune compromised people and domestic poultry being of highest risk) and the specific pathogens of concern (enterohemorhagic *E. coli*, avian influenza, waterborne protozoan pathogens *Cryptosporidium* and *Giardia,* West Nile virus and Newcastle disease virus in poultry are the most reasonable to be concerned about).

Recommendations

- Invest in monitoring and research that will provide the CWS with the necessary components to develop an evidence-based risk assessment. Some of the key areas of investigation should be improving our understanding of exposure probabilities (i.e.. nature and extent of goose interactions with people and livestock) but also elucidating the prevalence of specific pathogens for which we have no information for geese, as well as further genetic characterization of strains of pathogens and how they are related to similar human or livestock pathogens. There is a large number of possible hazards widespread over a variety of risk settings in Canada. Investment in such a program of research would need to be substantial and long-term to make significant shifts in our understanding of these determinants of risk.

- Model risks for some of the priority pathogens identified here as a means of accounting for the high degree of uncertainty. Uncertainty can either be modeled using quantitative or qualitative methods. Increasingly in areas in which there are large data gaps or uncertainty, a mixed approach, such as multi-criteria decision making can be used. However, models would need to account for the issue of context which modifies risk (exposure setting, nature of the at risk group, etc).

- Develop and improve risk reduction and communication strategies that focus on specific higher risk settings (i.e. aquatic resources, hospital settings, high livestock density areas).

- Form a working group to develop national standards for the management of peri-urban goose populations. Expertise from public health, regulatory agencies, resource managers, engineers, epidemiologists, and biologists along with input from the public are needed to develop a consensus on goose management practices. A consensus approach has been used to good effect in other situations where there is a high level of uncertainty.

2. REASON FOR REQUEST

The Canadian Cooperative Wildlife Health Centre (CCWHC) was asked by the Canadian Wildlife Service (CWS), Environment Canada to carry out a risk assessment of Cackling (*Branta hutchinsii*) and Canada (*Branta canadensis*) geese on human and livestock health. The CCWHC requested the Centre for Coastal Health (CCH) to complete a risk assessment. The CCH is the Pacific regional node of the CCWHC. The CCH has a strong background and expertise in performing risk assessments on a variety of issues ranging from assessing risks in the translocation of endangered species, to chronic wasting disease, to assessing disease risks in aquaculture settings.

Canada and Cackling geese are protected under Canada's *Migratory Birds Convention Act*[3]. This act prohibits capturing or killing of geese, or damaging, destroying, removing, or disturbing their nests, except as provided for under the *Migratory Birds Regulations*[4]. Canada Geese, along with all other migratory birds, are protected and managed by the Canadian Wildlife Service (CWS), Environment Canada. The *Migratory Birds Regulations* provide for management actions to "remove or eliminate migratory birds or nests where it is necessary to do so to avoid injury …"

CWS requested the Canadian Cooperative Wildlife Health Centre (CCWHC) to perform a risk assessment in order to support their decision-making for permit issuance for removing protected geese because of concerns for human or livestock health. They often receive requests for permits to "destroy eggs, relocate or kill birds (primarily Canada Geese), particularly from urban environments. Among other concerns, the justification accompanying the request is sometimes about concerns for human health and in other situations for livestock health." They have found that they have insufficient information on the actual health risks to humans or livestock on which to base their decisions. Some of the cases presented to them are straightforward in the sense that there is clearly property damage occurring as a result of the presence of geese. It is the cases where the issue of 'risks to humans or livestock health' is the stated reason for the request that the CWS requires a more detailed analysis of the risks to determine whether the requirements of the *Migratory Birds Regulations* would be met and thus support their decision about whether or not to issue a permit (pers. comm., Kathryn Dickson, CWS).

Most goose populations have increased significantly throughout North America over the last several decades[5]. The public has become increasingly concerned with the both the aesthetic of high numbers of geese and the associated contamination of green spaces, beaches, and ponds. In addition, perhaps as a result of recent dissemination of, and associated media attention towards pathogens such as highly pathogenic Avian influenza (HPAI) and West Nile virus (WNV), there is increasing public concern that pathogens carried by Canada geese pose risks to human and livestock health. As a result of these concerns, there has been an increase in the number of

[3] http://www.cws-scf.ec.gc.ca/legislations/laws1_e.cfm
[4] http://laws.justice.gc.ca/en/M-7.01/C.R.C.-c.1035/
[5] http://www.cws-scf.ec.gc.ca/mgbc/trends/index.cfm?lang=e&go=info.bird&speciesid=1720

complaints received by Parks Departments and other agencies, such as the CWS, which are responsible for managing Canada and Cackling geese in Canada.

There are a variety of risks that geese do or might present including risks to public health, agriculture, conservation, economics, animal welfare, or the environment. The types of potential risks to public health from geese could be grouped into the following categories: physical (i.e. trauma), psychological, or infectious. The categories of risk to livestock include: physical harm (i.e. trauma) and infectious disease.

The bulk of public discourse (i.e. media) related to Canada or Cackling geese focus on these species as nuisance animals in public spaces and potential sources of pathogens infectious for humans or livestock. The risk of goose to human trauma or psychological harm is most pronounced in the aviation industry where there have been a number of incidents in which Canada geese have collided with airplanes (Eschenfelder, 2001).

Canada geese are known to be highly protective during nesting season and could frighten people and other animals in their efforts to defend their territory against intruders. However, these types of incidents remain unquantified (Allan et al, 1995). There was also some reference to the risk to humans from slipping in the feces of Canada geese that are abundantly deposited in areas of high goose and human usage (i.e.. parks, golf courses, etc) (Allan et al, 1995; Conover and Chasco, 1985); however, we were unable to locate any data to support this claim or document the extent of this problem.

Potential risks to economic activities (i.e.. parks agencies, golf courses, private property value; not including livestock-related economics and trade), agriculture (crops), conservation (threats to migrant subspecies from overabundant resident goose populations), as well as to the environment (i.e.. overgrazing, eutrophication and algal growth of ponds/lakes) posed by Canada geese have been documented elsewhere and are beyond the scope of this risk assessment (Manny et al, 1994; Conover, 1991; Flegler et al, 1987; Conover and Chasco, 1985).

A variety of reports and assessments have been produced in regards to the management of goose populations throughout North America (USDA, 2004; Clark, 2003; US Fish and Wildlife, 2002; Gabig, 2000; Canada Goose Committee Atlantic Flyway Technical Section, 1999). A common theme in these documents is the reference to the health risks that geese pose to people and livestock. However, it is interesting to note that even though the public discourse around the Canada goose as a nuisance places a strong emphasis on human health risks, the majority of complaints to the USDA-APHIS Wildlife Service in West Virginia were found primarily to be related to damage to property (82%) or agriculture (15%) rather than human health and safety (3%) (USDA, 2004).

As per the request by CWS, the focus of this report is on the role of Canada and Cackling geese in the transport, dissemination, and possible transmission of pathogens of importance to human and livestock health.

3. METHODOLOGY

LITERATURE REVIEW

A literature search for peer-reviewed articles was undertaken using PubMed, Google Scholar, Science Direct and Agricola database systems/search engines. The following terms were used: 'geese', 'Canada goose', 'Anatidae', 'waterfowl, 'migratory birds', 'infection', 'disease', 'management' and specific pathogens known to be associated with birds or waterfowl such as 'Avian Influenza', 'Salmonella', 'West Nile Virus', etc. In addition, we searched for literature, using the same search engines, on the pathogens identified in our search for bird/goose-associated literature, which was associated with human or livestock disease or infection. A stronger emphasis was placed on scientific literature that was published after 1980 due to both accessibility reasons and to the improvement in pathogen detection and elucidation methodologies (i.e. molecular tools) that have the potential to provide more information on the likelihood of transmission from geese to either humans or livestock.

Non peer-reviewed literature, or 'gray literature', was searched using Google using the following terms: 'Canada geese/goose', 'Cackling geese/goose', 'management', 'population', 'migration', biology', 'disease', and 'interaction'. We also carried out specific searches of the following agency websites:

- Canadian Food Inspection Agency (www.inspection.gc.ca)
- Canadian Wildlife Service, Environment Canada (www.cws-scf.ec.gc.ca)
- Canadian municipal parks departments (Kelowna, Kitchener, Montreal, Niagara, Toronto, Vancouver, Victoria)
- Canadian Cooperative Wildlife Health Centre (www.ccwhc.ca)
- United States Department of Agriculture, Animal and Plant Health Inspection Service, Wildlife (USDA/APHIS), National Wildlife Research Centre (www.aphis.usda.gov/wildlife_damage/nwrc)
- United States Fish and Wildlife Service, Migratory Bird Program (www.fws.gov)

Canadian regulations and policies around the management of Canada and Cackling geese populations were reviewed:

- Canada Environmental Protection Act (http://laws.justice.gc.ca/en/C-15.31/)
- Migratory Birds Convention Act (http://laws.justice.gc.ca/eng/M-7.01/index.html)
- Migratory Birds Regulations (http://laws.justice.gc.ca/eng/C.R.C.-c.1035/index.html)

We also obtained additional clinical and epidemiologic information on specific pathogens and disease manifestations through the following online sources: Centre for Disease Control (CDC), World Organization for Animal Health (OIE), and the Merck Veterinary Manual.

For the purposes of this risk assessment, risks posed by Canada and Cackling geese were considered together. None of the literature we encountered on the pathogens associated with geese indicated that there were any differences between Canada or Cackling geese. In fact, Cackling geese, because of its relatively recent status as a separate species (2004), were not mentioned in the pathogen-focused body of literature that we reviewed.

HAZARD IDENTIFICATION, REVIEW, AND PRIORITIZATION

Hazards to humans or livestock from geese were identified using the following criteria:

Step 1: Can the potential hazard infect or be transmitted by Canada or Cackling geese or another member of the Anatidae family that includes geese, ducks, and swans?

Step 2: Can the potential hazard produce negative impacts on humans or livestock?

Step 3: Is the potential hazard present in North America?

If the answer to either the first or second steps was yes or uncertain AND it is present in North America, then the disease or pathogen was identified as a hazard.

For each hazard identified, we reviewed the literature for relevant criteria that could contribute to an estimation of the probability of transmission from geese to humans and geese to livestock (see Appendices A and B). The variables that were included were:

- prevalence of pathogen in wild geese in North American
- prevalence of pathogen in humans or livestock in North America
- detection and persistence of the agent in the environment
- evidence for cross-species transmission
- evidence for goose to human or goose to livestock transmission

The impact on human or livestock health, should a pathogen be transmitted from Canada or Cackling geese, were individually evaluated and rated (Appendices C and D). There was insufficient information to rate the impact of the pathogens quantitatively, so all ratings were carried out qualitatively using the criteria described below. The variables that were considered to assess the impact in either people or livestock for each of the hazards included:

- nature and severity of disease
- morbidity, mortality, hospitalization rates
- availability of suitable treatment and prevention measures
- national disease reporting requirement[6,7]

The categories of impact were determined based on the following criteria:

Negligible	No, rare or sporadic disease resulting from infections. Very effective treatments or control measures available. No impacts on trade. Not reportable.
Low	Disease can occur uncommonly from infection and/or symptoms are generally mild or self limiting. No impacts on livestock herd/flock

[6] List of human reportable diseases in Canada: http://dsol-smed.phac-aspc.gc.ca/dsol-smed/ndis/list_e.html

[7] Animal disease reporting to the CFIA: http://www.inspection.gc.ca/english/anima/disemala/guidee.shtml

Reportable diseases: "[diseases that are] usually of significant importance to human or animal health or to the Canadian economy"

Immediately notifiable diseases (for laboratories only): "diseases [that] are exotic to Canada for which there are not control or eradication programs"

Annually notifiable diseases (for laboratories only): "diseases for which Canada must submit an annual report to the World Organization for Animal Health (OIE) indicating their presence in Canada, but are not classified as reportable or immediately notifiable"

	productivity. Effective treatment or control is available. Hospitalization or death is unexpected. No impacts on trade. Not reportable.
Medium	Resulting disease can cause illness requiring medical attention and sometimes hospitalization. Deaths can occur in individuals and/or some herd/flock impacts are possible. Possible, but low, impacts on trade. The disease is reportable and treatment options are either less effective or limited in availability.
High	Resulting disease can cause severe illness and can spread epidemically, often requiring hospitalization in people. Deaths occur in individuals and/or herd/flock impacts occur. Livestock trade is affected. The disease is reportable. No treatment or prevention methods are available.

For many pathogens there is a range in the nature and severity of disease. There are often extreme manifestations and sequelae of infection that come about for a variety of reasons. Most often these more severe outcomes are due to risk factors in the susceptible species such as: immune status, age, gender, or predisposing conditions. Our final impact assessment is based on the average manifestation of disease but highlights where immune status or other risk factors might escalate severity of disease.

4. UNCERTAINTY

The framework that is commonly used and promoted by the World Organization for Animal Health (OIE) to assess risks in animal populations involves three components: release, exposure and consequence assessments (Zepeda et al, 2001). The release assessment determines whether the disease is present (or potentially present) in either the country of origin or species of interest, which, in this case, are the Canada and Cackling goose. The exposure assessment describes the pathways of exposure and associated probabilities that could cause infection in other populations, which, in this case, are people or livestock (note, for the purposes of this report, we include domestic birds and mammals raised for agricultural purposes as livestock). The consequence assessment describes the biologic (i.e. mortality and morbidity rates) and economic (i.e. trade restrictions, days off work) impacts should the disease occur in the species of concern (people and livestock in this case). Risk is the combination of the likelihood of occurrence of an adverse event and the magnitude of the consequences (Zepeda et al, 2001).

Table 1 outlines the data requirements for risk assessment and uncertainty analyses, adapted from Zepeda (2001). For this risk assessment concerning pathogens in wild geese, most of the data requirements simply cannot be met with the available data on pathogens potentially transmitted by geese. There are very large gaps in some of the following key determinants of risk: prevalence of pathogens and parasites in geese, epidemiological information to link the pathogen in geese to cases in people or livestock, fecal distribution patterns, and human or livestock exposure patterns (i.e.. the nature and extent of contact between geese and humans and geese and livestock). As a

result, a reliable, evidence-based risk assessment of health risks to people or livestock from free-ranging geese cannot legitimately be performed with the existing availability and quality of data.

Unfortunately, this is not very helpful to the CWS as it must make decisions in the face of uncertainty and the absence of critical information on the true nature of risks that geese pose to human and livestock health. Therefore, we have adapted the risk assessment framework described above into a format that provides CWS with current information on any supporting evidence for pathogen transmission from geese and also provides some tools to help prioritize pathogens into areas of potential risk.

Table 1: Information requirements for risk assessment and uncertainty analyses

Risk assessment steps	Epidemiological components	Data/knowledge requirements	Data available for geese
Hazard identification	List of pathogenic agents that could be associated with geese	Pathogens exotic to Canada	Inadequate surveillance
		Emerging pathogens	Inadequate surveillance
		Epidemiology of each endemic, epidemic and emerging pathogens in relation to geese	Limited to isolated surveys for the most part focused on a subset of pathogens
	Knowledge on the presence or absence of pathogen in Canada	Methods to demonstrate absence of pathogen	Iinsufficient testing
Release assessment	Prevalence of pathogen in Canada	Survey and surveillance results	Inadequate information
		Survey methodology	Variable
		Confidence level, precision, expected prevalence	Unknown
		True prevalence	Unknown
	Epidemiological characteristics of the disease and the pathogen	Incubation	Variable
		Carriers	Variable but largely unknown
		Morbidity	Inadequate information
		Mortality	Inadequate information
		Method of spread	Inadequate information
		Pathogenesis	Variable
		Target organs	Variable
		Susceptible species	Variable
	Diagnostic tests	Test sensitivity and specificity	Inadequate information
Exposure assessment	Characteristics of the susceptible populations and environmental factors	Pathways of exposure	Variable and unquantified
		Flock densities	Some information available.
		Flock distributions	Some information available.

Risk assessment steps	Epidemiological components	Data/knowledge requirements	Data available for geese
		Contact rate, and nature of contact, of geese with people or livestock	Inadequate information
		Immune status	Unknown
		Vectors	Variable
		Seasonality	Variable but most known
Consequence assessment	Biologic and economic consequences	Susceptible species	Unknown or variable
		Method of spread	Variable
		Contact rates	Unknown
		Morbidity	Variable
		Mortality	Variable
		Number of animals affected	Mostly unknown
		Direct economic impact	Information available
		Cost of control and eradication	Information available
		Indirect economic impact: interrupted trade, loss of international markets	Variable

In table 1, the term "variable" refers to either (i) that the quantity or quality of information was variable amongst the different pathogens wherein we knew significantly more about a small subset of pathogens than for all identified infectious agents or (ii) that the nature of the data varied with the context in which the pathogen was found (ex. impacts of a pathogen varied with immune status).

5. HAZARD IDENTIFICATION

This section provides a list of the pathogens that could be transmitted between geese and people or geese and livestock (Table 2). This is not a complete list of all pathogens of consequence to geese but narrowed to those that could potentially infect people or livestock. They were selected based on literature review (see methodology section). Due to the scope of the project and the significant number of agents that needed to be considered, only a cursory review of nematodes and helminths of geese was carried out.

Table 2: List of potential hazards that are found in North America in members of the Anatidae family (geese, ducks, swans) and in either people or livestock

Pathogens	Demonstrated infection in people	Demonstrated infection in livestock	References
Bacterial			
Actinobacillis suis	No	Yes, swine	Gottschalk, 2000; Maddux et al, 1987
Arcobacter spp.	Yes	Yes, all species	Collado et al, 2009; Houf et al, 2009; Atabay et al, 2008; Ho et al, 2006: Vandenberg et al, 2004
*Borrelia anserine (*avian borreliosis)	No	Yes, poultry	Lisboa et al, 2009; Ataliba et al, 2007
Borrelia burgdorferi	Yes	Yes, poultry	Ogden et al, 2009; Reed et al, 2003; Piesman et al, 1996; Burgess, 1989
Campylobacter spp.	Yes	Yes, all species	Hughes et al, 2009; Colles et al, 2008; French et al, 2009; Converse, 1999
Chlamydophila psittaci	Yes	Yes, poultry	Olsen, 2009; Laroucau, 2008; Fallacara, 2004; Longbottom and Coulter, 2003; Converse, 1999
Clostridium botulinum Type C (avian botulism)	No	Yes, poultry (note: this is an intoxication from ingestion of spores rather than an infection from *C. botulinum*	Lu et al, 2009; Rocke, 2006; Jean et al, 1995; Wobeser et al, 1987
C. perfringens ('necrotic enteritis')	Yes	Yes, all species	Olsen, 2009; Holtby, 2008; Leal et al, 2008; Turcsan, 2001; Wobeser, 1987
Erysipelothrix rhusiopathiae (Erysipelas)	Yes	Yes, turkeys and pigs	Olsen, 2009
Escherichia coli	Yes	Yes, all species	Edge and Hill, 2007; Kullas et al, 2002; Feare et al, 1999
Helicobacter spp.	Yes	Yes, all species	Tsiodras et al, 2008; Fox et al, 2006
Legionella pneumophilia	Yes	No	Clark, 2003; Fields et al, 2002; Yu et al, 2002; Fabbi et al, 1998
Listeria monocytogenes	Yes	Yes, all species	Lianou and Sofos, 2007; Swaminathan et al, 2007; Converse et al, 1999

Pathogens	Demonstrated infection in people	Demonstrated infection in livestock	References
Mycobacterium avium	Yes	Yes, all species	Olsen, 2009; Inderlied et al, 1993;
Mycoplasma spp.	Yes	Yes, poultry	Dobos-Kovacs et al, 2009; Olsen, 2009; Waites et al, 2004; Rosengarten et al, 2001; Bradbury et al, 1988; Baseman and Tully, 1997; Stipkovits et al, 1993
Pasteurella multocida (Fowl cholera)	Yes	Yes, poultry	Olsen, 2009; Pedersen et al, 2003; Samuel et al, 1997; Botzler, 1991
Salmonella enteric	Yes	Yes, all species	Olsen, 2009; Converse et al, 1999: Feare et al, 1999
Streptococcus spp (Group D) (S. bovis, S. gallolyticus subsp. Pasteurianus)	Yes	Yes, all species	Herrera et al, 2009; Hogg and Pearson, 2009; Onoyama, 2009; Barnett et al, 2008; Devriese et al, 1998
*Vibrio spp. (*non-cholera)	Yes	Yes, all species	Bush et al, 2006 ; Hinz et al, 1999; Buck, 1990; Schlater et al, 1981
Yersinia pseudotuberculosis	Yes	Yes, all species	Tauxe, 2004: Niskanen et al, 2003
Fungal			
Aspergilla spp.	Yes	Yes, poultry	Beytut et al, 2004; Akan et al, 2002; Stroud et al, 1982; Adrian et al, 1978
Candida albicans	Yes	Yes, poultry	Buck, 1990; Beemer et al, 1973
Cryptococcus spp.	Yes	Yes, all species	Duncan et al, 2006; Filion et al, 2006; Soogarun et al, 2006; Baro et al, 1998
Parasitic			
Coccidia spp.	No	Yes, poultry	Converse et al, 1999; Gomis et al, 1996; Chave et al, 1993
Cryptosporidia spp.	Yes	Yes, all species	Schuster et al, 2005; Zhou et al, 2004; Graczyk, 1998;
Giardia spp.	Yes	Yes, all species	Thompson et al, 2008; Graczyk and Lucy, 2007; Schuster et al, 2005; Graczyk, 1998;
Leucocytozoon spp.	No	Yes, poultry	Shutler et al, 2009; Hellgren et al, 2008; Nakamura et al, 2008; Bennett et al, 1982; Herman et al, 1975
Nematodes: *Amidostomum* ssp. *, Epomidiostomum* spp*., Trichostrongylus spp.*	No	Yes, domestic waterfowl	Nowicki et al, 1995
Sarcocystis spp.	Yes	Yes, all species	Dubey et al, 2006; Fayer, 2004; Kutkiene et al, 2008 and 2004
Toxoplasma gondii	Yes	Yes, all species	Dorny et al, 2009; Dubey et al, 2002, 2006 and 2007; Tenter et al, 2000
Trematodes: *Schistosome cercariae* (swimmer's itch)	Yes	No	Brant and Loker, 2009; Skírnisson et al, 2009

Viral			
Arboviruses (Eastern and Western Equine Encephalitis, St. Louis Encephalitis, West Nile Virus)	Yes	Yes, all species	Reimann et al, 2008; Thomas et al, 2007; Wojnarowicz et al, 2007; Austin et al, 2004; Banet-Noach et al, 2003; McLean et al, 2002; Swayne et al, 2001
Avian adenovirus	No	Yes, poultry	Chen et al, 2009
Avian herpesvirus (duck viral enteritis)	No	Yes, domesticated waterfowl	Campagnolo et al, 2001; Converse and Kidd, 2001; Gough and Hansen, 2000
Avian influenza	Yes	Yes, poultry	Brown et al, 2008; Ward et al, 2008; Pasick et al, 2007; Van Reeth et al, 2007; Clark and Hall, 2006; Gill et al, 2006; Olsen et al, 2006
Avian pneumovirus	No	Yes, poultry	Cook, 2000; Shin et al, 2000
Avian pox virus	No	Yes, poultry	Buller and Palumbo, 1991; Cox, 1980
Coronavirus	Yes	Yes, all species	Jonassen et al, 2005; Weiss and Navas-Martin, 2005
Duck Hepatitis Virus	No	Yes, domestic waterfowl	Marion et al, 2005
Goose parvovirus	No	Yes, domestic geese	Yang et al, 2009; Irvine et al, 2008
Newcastle disease virus	No	Yes, poultry	Iowa State, 2008; Alexander et al , 1998 and 1999; Palmer and Trainer, 1970
New-type gosling viral enteritis	No	Yes, domestic geese	Chen et al, 2009
Goose Hemorrhagic Polyomavirus	No	Yes, domestic geese	Pingret, 2008; Lacroux et al, 2004
Reticuloendotheliosis virus	No	Yes, poultry	Lin et al, 2009

6. ADAPTED RISK ASSESSMENT

6.A. RELEASE ASSESSMENT

RISK SETTING

There is a vast body of literature describing the biology of Canada and Cackling geese (Mowbray et al, 2002; Allan et al, 1995; CWS[8]). We include here only a brief overview of some of the aspects of their biology that help us understand the risk setting in which Canada or Cackling geese may contribute to the transport, dissemination, or transmission of pathogens of importance to humans or livestock in Canada.

Canada and Cackling geese range and migrate widely throughout North America[9]. They are largely migratory species that breed in Canada and in the Northern US and overwinter in southern Canada, the US and in northern Mexico. Until recently, all "white-cheeked geese" in North America were considered to comprise several races of a single species, *Branta canadensis*, commonly known as Canada geese. In 2004, the American Ornithologists Union declared that the genetic differences between two groups of races were sufficient that they represent two species, including the species *Branta hutchinsii,* or Cackling geese, in addition to those that comprise the Canada goose species.

In general, geese found nesting or moulting in urban areas of southern Canada during spring and summer in the central and eastern regions of Canada are considered to be a mix of races, perhaps predominantly giant Canada geese (*Branta canadensis maxima*), whereas those found in western regions of Canada are called western Canada geese (predominantly *Branta canadensis moffit*ti) (Smith et al, 1999). Collectively these are referred to as "temperate-breeding" Canada geese. On the other hand, Cackling geese are arctic-nesting birds and are present in southern Canada only during the migration period. Thus the majority of opportunities for conflicts with people occur with temperate breeding populations of Canada geese. However, in some situations migrating Cackling geese may also cause conflicts.

Surveys of temperate-breeding nesting pairs in Ontario showed an increase from 2,400 pairs in 1970 to over 90,000 pairs in 2006, with an average annual population growth rate of about 13% (Hughes, 2009). These trends are also evident in each of the four North American flyways: Atlantic, Mississippi, Central, and Pacific Flyways (Gabig, 2000; Canada Goose committee, Atlantic Flyway Technical section, 1999). Current estimates of temperate-breeding goose populations in North America are as follows: 1.1 million in the Atlantic Flyway, 1.3 million in the Mississippi Flyway; 1.1 million in the Central Flyway; and 0.1 million in the Pacific Flyway (Coluccy, Ducks Unlimited). Some estimate that temperate-breeding populations now make up over 65% of the overall Canada and Cackling goose population in North America. The majority of the temperate breeding birds

[8] http://wildspace.ec.gc.ca/life.cfm?ID=CAGO&Page=More&Lang=e
[9] http://wildspace.ec.gc.ca/life.cfm?ID=CAGO&Page=RangeMap&Lang=e

geographically nest below 47°N latitude, with a smaller proportion below 49°N latitude (Hughes, 2009).

Temperate-breeding populations have successfully taken hold in North America for a variety of reasons including: the success of captive breeding and re-introduction programs in the 1960s and 1970s; their highly adaptable nature; landscape change and urbanization that has created ideal grazing zones (i.e. golf courses, parks, etc); paucity of predators; and possibly climate change (Conover and Chasko, 1985). The impact of this dramatic and sustained growth in temperate-breeding goose populations has potentially provided increased opportunities for geese to interact with humans and livestock.

Canada and Cackling geese are generally long-lived and have high survival rates. Temperate-breeding geese tend to have higher survival and reproductive rates and are exposed to more favourable weather conditions for breeding than their migrant relatives. Temperate-breeding geese are grazers and generally prefer new-growth sedges and grasses. As a result they are particularly attracted to urban landscapes where recreational areas (parks, golf courses) and private properties are abundant and have wide expanses of manicured grass in close proximity to ponds and lakes. Canada and Cackling geese have very adaptable feeding habits and also graze on agricultural crops when they are available (Mowbray et al, 2002; Conover, 1991).

Canada geese produce copious amounts of feces which are a potential source of exposure to humans or livestock of pathogens carried and disseminated by geese. Estimates of fecal output range from 0.39 to 0.90 kilograms daily (Filion et al, 2006; USDA, 2004). Feare et al (1999) found that the majority of fecal deposits by Canada geese were made within 100 metres of the water's edge, which increases the opportunity for contamination of waterways. Microbial source tracking, combining antimicrobial resistance analysis and DNA fingerprinting methods, has been used to demonstrate the extent of the contribution of Canada geese to elevated fecal counts at several beaches in Ontario (Edge and Hill, 2007).

In urban settings, there are a variety of ways in which humans and geese come into direct or indirect contact with each other. The most plausible urban site for contact is in recreational areas such as parks, beaches, playground, sport fields, and golf courses. Human risk from pathogens from geese is influenced by the way in which people interact directly with geese (i.e.. feeding them, controlling them, surveying them, etc) as well as how people make use of these public spaces (sports, sunbathing, landscape maintenance, picnicking, swimming, etc). For example, those walking in parks will have a different level of risk, than those sunbathing or playing games where the ball might roll through goose feces. The potential risk of contact with goose feces is also influenced by personal mitigation strategies (avoiding direct contact with geese, washing hands, cleaning off shoes, etc).

In rural settings, the impact of geese in agriculture has been noted as a potential cause for concern (Clark, 2003). Historically, the most frequently described impact of geese in agricultural settings was around the issue of crop damage (Fleger et al, 1987). However, more recently the question of the role of wild birds, including geese on the transmission of avian influenza to domestic poultry

and humans has come to the fore (Tsiodras et al, 2008). Goose interactions with livestock might involve some of the following: co-grazing on pastures, sharing ponds, goose contamination of water sources that are consumed by livestock, geese defecating on food sources of cattle (i.e. grains, alfalfa). In 1996, one of the research goals identified by the US National Wildlife Research Centre was to understand the contribution of Canada geese to human and livestock health (see Box 1). Kullas et al (2002) and Clark (2003) have presented several studies in relation to this goal, which we describe further on in this report.

Box 1: Research priorities identified by the US National Wildlife Research Centre in 1996

WATERFOWL AS DISEASE, PARASITE, AND NOXIOUS WEED RESERVOIRS IN URBAN AND AGRICULTURAL LANDSCAPES

Goal: Understand and develop management recommendation related to the contribution and impact of Canada geese as vectors for disease, parasites, and noxious weeds on human health and safety in urban landscapes and on animal health in agricultural landscapes

Research Objectives:

- Determine the prevalence of *Salmonella* spp., *Campyhlobacter* spp., and *Escherichia coli* H:0157 from Canada goose feces derived from urban landscapes, and make recommendations for managing associated risks to human health and safety
- Determine the prevalence of *Salmonella* spp., *Campyhlobacter* spp., and *Escherichia coli* H:0157 from Canada goose feces derived from agricultural pastures
- Determine the prevalence of other bacterial pathogens *(e.g., Streptococcus* spp., *Staphylococcus* spp., *Listeria* spp., *Yersinia* spp., *Shigella* spp.) and parasites (e.g., *Girardia* spp.) in Canada goose feces derived from urban landscapes with the aid of collaborative research laboratories

The livestock species that we consider in this report are those that make up the largest food-animal commodity groups in Canada: chickens, turkeys, cattle, pigs[10]. However, there are also a small proportion of poultry operations in Canada that are rearing domestic geese and ducks. Statistics Canada shows in their alternative livestock census[11] that, in 2006, over a million domestic ducks and 115,000 domestic geese were being raised across the country.

Risk factors associated with potential hosts (i.e.. people or livestock in this case) such as pre-disposing conditions, immune status, age, gender, behavior, etc., can strongly influence the probabilities of exposure and of development of disease as well as the magnitude of impact that might be experienced. For example, if goose populations were congregating and spreading pathogenic feces onto public spaces in close proximity to hospitals where there are likely higher rates of people who might have immune compromised systems, the risks to human health could be higher than if geese were in an area, such as a soccer field, where players are more likely to be healthy and not immunocompromsied. Likewise, the level of risk that the contamination of water resources by geese presents to livestock will vary based on, among other variables, the specific risk factors on individual farms. For example, a cow-calf operation with more young cattle might be more susceptible to infection by a pathogen such as *Cryptosporidium* than a feedlot operation with an older average age of animals as this pathogen is particularly pathogenic to calves. The key message is that risk will vary widely in different settings due to the unique combination of risk

[10] Statistics Canada: http://www40.statcan.gc.ca/l01/ind01/l3_920_2553-eng.htm?hili_none
[11] http://www.statcan.gc.ca/pub/23-502-x/23-502-x2007001-eng.pdf

factors that each presents, and a generic risk assessment for all pathogens and settings is not feasible.

MECHANISMS FOR EXPOSURE TO HUMANS FROM GOOSE PATHOGENS

Pathogens from Canada and Cackling geese could be transmitted to humans by several routes including: direct transmission through airborne transmission, or contact with infected feathers, skin, droppings or external lesions, waterborne or food-borne transmission, occupational exposure to infected materials, transmission via arthropod vectors, and indirect transmission through the same routes if geese transmit the agent to livestock and livestock then are a source of infection for people (see Figure 1) (Tsiodras et al, 2008). Each route of exposure is discussed in more detail in the next section (exposure assessment).

In order for a person to develop a disease from a pathogenic organism in the feces of wild geese, the following steps must take place:

1) an organism that is pathogenic to humans must be present in the feces of geese;
2) the organism in the feces must survive in the environment after their deposition on the ground or in water;
3) geese must deposit feces in an amount and manner where people might come into contact with it,
4) a person must contact the contaminated feces, ingest a sufficient amount (infectious dose), become infected, and produce symptomatic illness (Feare et al, 1999).

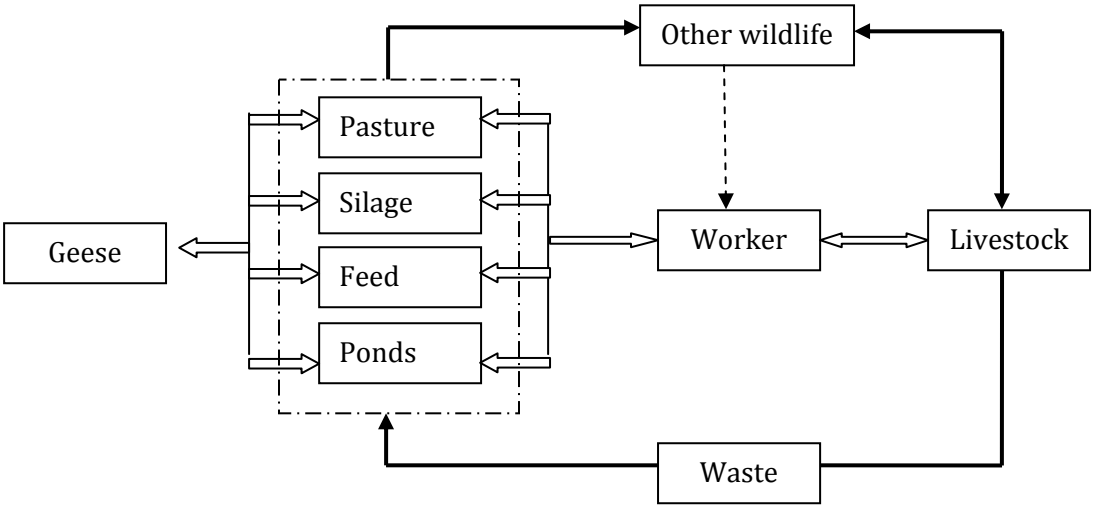
MECHANISMS FOR EXPOSURE TO LIVESTOCK FROM GOOSE PATHOGENS

Figure 1 demonstrates the pathways of exposure for livestock to pathogens disseminated by geese. Direct transmission could occur through aerosol transmission or through direct contact with infected feathers, skin, droppings or external lesions. Infected birds could contaminate water with feces and respiratory secretions that could result in a waterborne transmission through ingestion. Indirect transmission to livestock could also occur through the same routes if geese transmit the agent to humans and humans then transmit the pathogen to livestock. Disease transmission via arthropod vectors is another possible route for livestock infection from geese.

In order for livestock to develop a disease from a pathogenic organism in the feces of wild geese, the following steps must take place:

1) an organism that is pathogenic to livestock must be present in the feces of wild geese;
2) the organism in the feces must survive in the environment after their deposition on the ground or in water;
3) geese must deposit feces in an amount and manner where livestock might come into contact with it,
4) livestock must contact the contaminated feces, ingest a sufficient amount (infectious dose), become infected, and produce symptomatic illness (Feare et al, 1999).

Figure 1: Potential exposure pathway of pathogens between geese, livestock and people (adapted from Clark, 2003)



6.B. EXPOSURE ASSESSMENT

In Appendix A there is an overview of the factors that might affect the probability of transmission between infected geese and humans including: presence in North America, prevalence in wild geese, prevalence in humans in North America, persistence of organism in the environment, evidence for cross-species transmission (of any sort not just humans), evidence for goose to human transmission, and some relevant references. Appendix B presents an overview of the factors that might affect the probability of transmission between infected geese and livestock including: presence in North America, prevalence in wild geese, prevalence in livestock in North America, persistence of organism in the environment, evidence for cross-species transmission, evidence for goose to livestock transmission, and some relevant references. In this section we discuss the plausibility of human and livestock infection from geese taking place by the various routes of transmission described above. We can conclude that there are several plausible routes of exposure to infectious material from geese, but estimates of the probability of these exposures are not attainable with existing data.

Plausibility of transmission by route of infection

Airborne transmission: Airborne transmission generally could occur when there is very close contact between humans or livestock and infected dust, or water droplets, feces or respiratory secretions from infected geese and there is inhalation deep enough to allow for infection. For example, if members of the public were feeding the birds, picking up young birds, or otherwise within very close proximity they could potentially be exposed in this way. This mode of exposure is also plausible for those responsible for handling geese (i.e. occupational exposure of bird control

specialists, wildlife rehabilitation specialists, biologists, veterinarians, etc) and also for landscape maintenance personnel who could be exposed to infected airborne particles through some of the following activities: gardening, cutting grass, moving soil, raking leaves or beaches, etc. In livestock, airborne transmission might occur if geese were in close proximity to the animals (i.e. co-grazing, livestock drinking from pond inhabited by geese.

It is also plausible that wind may play a role in distributing pathogen-carrying particles. Airborne particles, particularly those of smaller size, can be carried over long distances. Some of the factors that affect the occurrence of infections acquired by long-distance airborne transmission include: 1) dilution, 2) the infectious dose, 3) the number of infectious particles, 4) the duration of shedding of the infectious agent, and 5) the persistence of the agent in the environment (Tellier, 2006). The majority of this information is missing for goose-related pathogens.

Direct contact with infected feathers, skin, feces or external lesions: This route, particularly contact with feces, seems most plausible for people in areas of high recreational use of habitats frequented by geese and people, such as urban parks. Livestock must be in close proximity to geese (i.e. co-grazing) in order for transmission via this route to take place. Canada geese have been shown to produce nearly 1 kg of feces per day (range 0.39 – 0.9 kg) (Ilion et al, 2006; USDA, 2004). The range of risk groups that present a reasonable presumption of exposure potential for pathogen transmission might include: children, bathers, picnickers, sunbathers, and landscapers. Of this group young children are probably at greatest risk due to their methods of play and propensity to consume dirt. Stanek and Calabrese (1995) demonstrated that children on average consume 138 mg of dirt daily. Also, bacteria have been shown to persist longer in sand than in water as they adhere to sediment particles, so people, and particularly children, playing in sand might be at greater risk of exposure (Benskin et al, 2009).

Free-ranging livestock or livestock reared in outdoor settings are more at risk for exposure than those kept in closed systems with proper biosecurity, which should prevent transmission of pathogens by direct contact with geese feathers, droppings, skin, or external lesions. The majority (up to 90% in the US) of chickens and turkeys, and about three-quarters (72% in the US) of swine herds are raised in intensive, closed settings (Graham et al, 2008). However, there is a growing consumer demand for free-range animal products that is driving an increase in open-farming systems to meet the demand. It is reasonable to assume that these more open systems where chickens or turkeys spend a portion of their day in outdoor settings would create more opportunity for direct exposure to geese and their pathogens than those reared in entirely enclosed settings.

Water-borne transmission: Infected birds can contaminate water with feces and respiratory secretions that could potentially result in a waterborne transmission through ingestion, or potentially, as a result of swimming (Benskin et al, 2009). A wide range of waterfowl species have been shown to contribute substantially to fecal coliform counts in human drinking water sources and are often suspects in transmission of other waterborne bacterial, parasitic and viral pathogens such as *Campylobacter*, *Cryptosporidium*, *Giardia*, Avian Influenza virus, *Helicobacter*, etc (Graczyk et al, 2008; Tsiodras et al, 2008; Fox et al, 2006; Alderisio and DeLuca, 1999). Waterborne

transmission is potentially one of the most important routes of transmission given the possibility for wide dissemination through municipal or household water sources and the level of access of waterfowl to relevant aquatic environments. It is not unreasonable to assume that livestock might be at greater risk than people from waterborne pathogen transmission as many farms rely on drinking water sources that are often untreated such as rivers, streams and ponds that might be contaminated by pathogens of goose origin.

Occupational exposure: Exposure to infected tissues, blood and feces is another source of direct transmission. Veterinarians, hunters, biologists, wildlife control officers, wildlife rehabilitators are some of the risk groups for occupational exposure. These groups, perhaps more than any other, may be more inclined to take some preventive measures to mitigate against pathogen exposure such as those listed in the Public Health Agency of Canada's "*Fact Sheet: Guidance on Precautions for the Handling of Wild Birds*[12]". This could be an indirect transmission route for livestock if their handlers or veterinarians were in contact with infected goose tissues and pass it on to them.

Food-borne transmission: Food-borne infections in humans could develop as a result of the consumption of contaminated geese, particularly through the ingestion of raw or undercooked meat, blood, organs, etc (Tsiodras et al, 2008). Indirect transmission to humans could also occur through the same routes if geese transmit the agent to livestock (see Figure 1). Livestock could acquire pathogens from geese through their food-stuffs that were contaminated with pathogens from geese during the production, harvesting, processing or distribution process.

Arthropod-borne transmission: Disease transmission via arthropod vectors is another possible route for infection from geese. Humans or livestock may be affected by arthropods either directly by bites, stings, or infestation of tissues, or indirectly through pathogen transmission. Ticks and mosquitoes are the most important genera for pathogen transmission. The most significant mode of vector-borne disease transmission is by biological transmission by blood-feeding arthropods. The pathogen multiplies within the arthropod vector, and the pathogen is transmitted when the arthropod takes a blood meal from the person or animal. Arthropods are also capable of infecting people or livestock indirectly through mechanical transmission of the agent when they physically carry pathogens from one place or host to another, usually on body parts. Key determinants of arthropod-borne diseases include the:

- abundance of vectors and intermediate and reservoir hosts;
- prevalence of pathogens suitably adapted to the vectors and the host;
- local environmental conditions such as temperature and humidity; and
- behaviour and immune status of the human population.

In summary, there are a number of biologically plausible routes of exposure to the hazards listed in Table 2. However, there are insufficient data to quantify the probability or average exposure rates for any of the pathogens. The exposure route of greatest concern, based on the extent of coverage in the peer-reviewed literature as well as in the lay media, is direct contact of people (contact in parks

[12] http://www.phac-aspc.gc.ca/influenza/fs-hwb-fr-mos-eng.php#4

or from water sources) and livestock (on-farm contact or from water sources) to infected goose feces. Airborne transmission of pathogens from geese to poultry (i.e. Avian influenza, Newcastle disease) is also widely discussed in the literature and media, but less so than for direct transmission from contact with feces. If feces are the major concern, high concentrations of geese with high rates of fecal deposition would increase the likelihood of exposure to hazards. This likelihood increases if fecal deposition is occurring in areas of high human use (density) or livestock density. This might suggest that exposure risk mitigation strategies to contain goose fecal matter is an appropriate area of focus (i.e. (1) dispersing birds to decrease the density of fecal contamination in areas of high human or livestock density; (2) habitat modification to make areas less attractive to Canada Geese; (3) waste management via removal of feces from affected areas; and (4) aquatic resource management) (Smith et al, 1999; Allan et al, 1995).

6.C. CONSEQUENCE ASSESSMENT

This section covers the types of consequences, or impact, that could arise as a result of human or livestock infection from geese. Appendix C presents an overview of the factors that affect the impact of each pathogen should it be transmitted to humans from geese including: the route of transmission, clinical manifestation (what does the agent do?), severity of the illness (is it treatable? how often are people hospitalized? how often do they die from the disease?), whether or not the disease is nationally reportable, and some relevant references. Appendix D presents an overview of the factors that affect the impact of each pathogen should it be transmitted to livestock from geese including: the route of transmission, clinical manifestation (what does the agent do?), severity of the illness (is it treatable?, how severe are the signs? how often do livestock die from the disease?), national disease reporting requirements, and some relevant references. In each of these tables the hazards are each given an impact category (negligible, low, medium or high) based on the criteria described in the methods section.

In humans, the pathogens with a 'high' ranking had more than one of the following features: 1) caused severe illness, often resulting in hospitalization; 2) had the ability to spread epidemically from person-to-person; 3) had high fatality rates; 4) did not have effective treatment or preventive methods; and 5) and were nationally reportable. The pathogens that filled more than one of these criteria included: highly pathogenic avian influenza (HPAI) and enterotoxigenic E. coli (ETEC). Pathogens of medium and low impact are shown in Appendix C.

In livestock, the pathogens with a 'high' ranking had more than one of the following features: 1) caused severe illness; 2) had the ability to spread epidemically within or between herds or flocks; 3) had high fatality rates; 4) did not have effective treatment or preventive methods; and 5) and were nationally reportable to the Canadian Food Inspection Agency. The pathogens that filled more than one of these criteria included: highly pathogenic avian influenza (poultry), *Mycobacterium avium*, and Newcastle disease (poultry). Once again, we found no direct evidence that linked human or livestock health outcomes to geese. Hence, this consequence assessment is the

consequence of the pathogens in general and not the pathogen from geese. Almost all of the hazards listed have multiple sources including people, other species and environmental sources.

7. SYNTHESIS

There is evidence that many pathogens of importance to humans and livestock can infect Canada and Cackling geese and be shed into the environment by geese, and vice versa. However, there is a very large gap in our understanding of the ecological and epidemiological factors that may contribute to the transmission from geese to humans or livestock. As discussed above, effective risk analyses (identification, assessment, management and communication) require data inputs from quality surveillance information (Table 1). Although there was a large list of pathogens associated with geese and other members of the Anatidae family, there was scarce serological, microbiological or epidemiological data as evidence of transmission between geese and humans and geese and livestock.

In a traditional risk assessment, this section would involve making overall risk estimations for the transmission of each identified hazard by combining the results of the release/exposure (probability estimates) and consequence (magnitude of impact) assessments. But in this case, we were not able to estimate probability so we are left with identifying some of the priority pathogens and routes of exposures that CWS should consider in its risk management strategies. These pathogens were identified based on the level of potential impact to human health and livestock health and trade and the plausibility and related evidence for transmission.

What type of evidence is needed to conclude that pathogens of goose origin are a cause of adverse health outcomes in people or livestock? The Bradford- Hill criteria that are used as guidelines to evaluate evidence of disease causation are relevant in this scenario (See Box 2). The evidence of transmission from geese to either humans or livestock must at a minimum be: 1) plausible, 2) specific (i.e.. same species, strain, serotype), ideally to the molecular level, and 3) associated by time and geographic location.

BOX 2: Bradford-Hill criteria for causation (Wikipedia, adapted from Hill, 1965)

Strength: A small association does not mean that there is not a causal effect.

Consistency: Consistent findings observed by different persons in different places with different samples strengthen the likelihood of an effect.

Specificity: Causation is likely if a very specific population at a specific site and disease with no other likely explanation. The more specific an association between a factor and an effect is, the bigger the probability of a causal relationship.

Temporality: The effect has to occur after the cause (and if there is an expected delay between the cause and expected effect, then the effect must occur after that delay).

Biological gradient: Greater exposure should generally lead to greater incidence of the effect. However, in some cases, the mere presence of the factor can trigger the effect. In other cases, an inverse proportion is observed: greater exposure leads to lower incidence.

Plausibility: A plausible mechanism between cause and effect is helpful.

Coherence: Coherence between epidemiological and laboratory findings increases the likelihood of an effect. However, Hill noted that "... lack of such [laboratory] evidence cannot nullify the epidemiological effect on associations"

Experiment: "Occasionally it is possible to appeal to experimental evidence".

Analogy: The effect of similar factors may be considered

The most plausible source of infection for both people and livestock appears to be via contaminated aquatic environments. Another plausible route is through direct contact with infected fecal material (this includes infected airborne particles). These routes provide the opportunity for the widest possible exposure for both people and livestock. Below we discuss some of the pathogens that we feel should be of greatest concern to CWS and as a result be considered in their risk management and communication strategies. However, it must be noted that the only Bradford-Hill criteria that any of the geese-related pathogens meet are plausibility and some meet the 'consistency' criteria due to geo-spatial associations. Therefore, all hazards fail to meet Bradford-Hill's criteria so no conclusions about causal linkages can be made. Even though we highlight several pathogens here, some of them have high species specificity and low virulence capacity so potentially present a low to negligible risk of transmission.

Due to the fact that Canada and Cackling geese are aquatic birds that aggregate in large numbers and produce a prolific amount of feces, concerns have been raised about their role in water contamination. As a result, there has been an emphasis on research of waterfowl-related pathogens and in particular for *E. coli, Cryptosporidium,* and *Giardia*. This research bias could affect the assessment of risks from waterborne transmission from geese in that more important, but un- or under-researched pathogens are not considered as much as these three pathogens. In addition, the perceptions of waterborne pathogen risks are shifting as a consequence of more recent molecular

and epidemiological information that is coming on stream and showing more host specificity than previously thought. Hence, the assessment of pathogens in this report might be biased by the older literature that could not make distinctions between strains that may be highly host-specific and therefore present a diminished risk for interspecies transmission (Hansen et al, 2009; Graczyk et al, 2008).

Of all the potential hazards presented in this report, *E. coli* is the pathogen for which there exists the most amount of information and research. It is most commonly looked for in fecal surveys and enumerated in water quality tests; it is also often top of mind by the public as a potential risk from goose feces. Alderisio and DeLuca (1999) found that Canada geese contributed on average 1.28 x 10^5 fecal coliforms per fecal deposit to surface water. Although *E. coli* has over 200 specific serological types, the majority of which are harmless to humans or livestock, there are several virulent forms that can cause severe illness (Edge and Hill, 2007; Clark, 2003; Kullas et al, 2002; Roscoe, 2001; Feare et al, 1999). Virulent strains, such as Enterohaemorrhagic *E. coli* (EHEC), only infrequently cause disease in animals; ruminants, and cattle in particular, are the primary reservoir. Poultry and pigs are not considered to be a source of EHEC (Caprioli et al, 2005). It is not unreasonable to assume that Canada or Cackling geese could potentially acquire EHEC, or other serotypes, from on-farm sources such as infected pastures, manure piles or slurry and then mechanically transfer to other locals and contaminate aquatic environments used by people or livestock (Kudva et al, 1998). However, this possible route of exposure and transfer, to the best of our knowledge, has not been demonstrated. This again speaks to the importance of the unique risk settings that must be considered when assessing health risks such as goose feeding ecology, migration patterns, farming practices (i.e. fertilization of fields with manure), and the distribution and types of farms and their relationship with goose ecology.

Improved water-quality indicators are needed, and are currently being developed, that move beyond simple coliform counts as indicators of risk to methodologies that track specific host sources of pathogens (Hansen et al, 2009; Hamilton et al, 2006). These tools will contribute essential information for the assessment of risks from goose fecal contamination in aquatic environments that currently cannot be ascertained. Significant data gaps remain on the determinants of risk such as the prevalence of pathogens that are shed by geese and the exposure to these pathogens through aquatic environments (i.e. drinking water sources for both people and livestock and exposure patterns during recreational activities such as swimming).

Cryptosporidium and *Giardia* are considered to be the most common causes of human waterborne outbreaks and gastroenteritis world-wide and are the most important parasitic pathogen in livestock (non-poultry) (Plutzer et al, 2009; Thompson et al, 2008). Oocysts (the transmissive stage) of *Cryptosporidium* and *Giardia* are very robust and can survive in water for prolonged periods in adverse conditions; they are considered to be ubiquitous in aquatic environments (Gracyzk et al, 2008). Canada geese can distribute *Cryptosporidium* strains that are pathogenic to people and ruminants but the majority of the strains they shed have been shown to be goose-specific and not pathogenic to other species (Gracyzk et al, 1998). However, temperate-breeding Canada geese have been shown to acquire *C. hominis* (human-adapted species) oocysts from

garbage and other contaminated sites (Zhou et al, 2004) so they have the potential to mechanically transfer this agent. Cattle are considered to be the primary reservoir of *C. parvum*, so increased opportunities for geese to contact contaminated feces from cattle (i.e. co-grazing, field fertilization with manure) has the potential to increase goose exposure and then possible transmission from geese back to aquatic environments of high human or livestock use. Zhou et al (2004) found that only 10% of recovered *Cryptosporidium* sp. from goose feces were *C. parvum* and *C. hominis*, the species that are pathogenic to humans, and concluded that Canada geese "might only serve as accidental carriers of cryptosporidia infectious to humans and probably play a minor role in the animal-to-human transmission cycle of the pathogen**".**

HPAI can cause devastating illness in poultry flocks, as seen in recent outbreaks in British Columbia and Saskatchewan (Berhane et al, 2009; Pasick et al, 2007). Avian influenza virus infection is endemic in a wide range of wild bird species, particularly species associated with water such as Anseriformes and Charadriiformes (Ward et al, 2009). There are conflicting theories and evidence in the literature about the role of wild birds in the transmission cycle of highly pathogenic avian influenza (HPAI) (i.e. H5N1) of importance to humans (Berhane et al, 2009; Ward et al, 2009; Tsiodras et al, 2008; Clark and Hall, 2006; Olsen et al, 2006; FAO media centre[13]). However on balance, wild birds do not appear to have a dominant role in the transmission cycle of HPAI as it has rarely been isolated from wild birds and is thought to result from local transmission from poultry rather than from de novo generation in wild birds (Tsiodras et al, 2008). Wild waterfowl are often infected with low pathogenic avian influenza (LPAI) viruses, and the theory is that wild birds could transmit LPAI to poultry flocks, which then act as mixing vessels for new, and potentially highly pathogenic, strains of influenza that could then infect people. Waterfowl, particularly ducks, can excrete LPAI in relatively high concentrations in their feces (Parmley et al, 2007; Pasick et al, 2007; Olsen et al, 2006). Influenza virus can remain infectious in freshwater for up to 4 days at 22°C and more than 30 days at 0°C. The relatively high virus prevalence in waterfowl might be due, in part, to efficient transmission through the waterborne fecal-oral route of transmission (Olsen et al, 2006). However, factors such as species, sex, age, and season have been shown to influence prevalence rates in ducks (Pasick et al, 2007; Halvorson et al, 1985).

In response to the HPAI outbreak in domestic poultry in British Columbia in 2004, a Canadian surveillance program for influenza A viruses in wild aquatic birds was initiated the following year to address information gaps on the role of wild birds in the pathogen transmission. The Canadian Cooperative Wildlife Health Centre was tasked with coordinating the Inter-agency Wild Bird Influenza Survey[14] to meet the following objectives:

1) "*create an inventory of influenza A viruses that occur in wild birds in different areas across Canada;*

[13] FAO media centre. March 23,2010. *On the trail of avian influenza: International task force concerned over declining support for H5N1 monitoring, despite disease persistence and spread.* http://www.fao.org/news/story/en/item/40827/icode/

[14] http://www.cws-scf.ec.gc.ca/nwrc-cnrf/default.asp?lang=en&n=00DF74F2-1

2) *to characterize these viruses to a sufficient degree that it may be possible to determine whether they may be the source, in whole or in part, of any future outbreaks in domestic animals or humans;*
3) *to monitor for the presence of particular influenza A viruses or their genetic components in the Canadian wild bird population;*
4) *to establish an archive of influenza A virus strains that would permit rapid retrospective analysis in response to disease outbreaks; and*
5) *to build and maintain an integrated, multi-agency field, laboratory, regulatory, and communications capacity within Canada to carry out influenza A virus sampling, identification, and molecular characterization of large volumes of samples."* (Pasick et al, 2007)

Results from this survey have show that "on average, 30% of all live ducks sampled, 5% of other species of live birds and 3% of birds found dead have tested positive for avian influenza, all of North American lineage and of low pathogenicity" (Parmley et al, 2009). Geese were sampled in this survey in only 2006 and 2007. Of all geese sampled, including Snow geese, Ross' geese, Greater White-fronted geese, Canada and Cackling geese, 5.5% (207/3738) were positive for Avian Influenza during this period; Canada or Cackling geese accounted for 32% (66/207) of the positive geese samples. Between 2005 and 2009, there were nine dead goose submissions (4 Snow geese, 5 Canada geese) that were positive for Avian Influenza. In 2007, the year of the H7N3 BC outbreak, an H7N3 LPAI strain was detected in a single Canada goose that was submitted to the CCWHC as part of the Inter-agency Wild Bird Influenza Survey. The relationship between this finding and the poultry outbreak in the Fraser Valley are unknown but could potentially be due to spill-over from infected poultry to the goose. Berhane et al (2009) report on an HPAI outbreak in the Saskatchewan poultry industry where they indicate that wild birds played a role in seeding the outbreak with a low pathogenic avian influenza strain that converted into a more highly virulent strain and caused significant losses to the industry. In this case, phylogenetic analysis demonstrated a close relationship of Saskatchewan/2007 H7N3 with recent viruses of free-flying waterfowl in North America. In summary, the exposure setting is of paramount importance in determining risk; the proximity of geese to poultry operations and the nature of their contact with domestic flocks will be a key determinant of risk for development and transmission of HPAI.

8. RECOMMENDATIONS

For this risk assessment concerning pathogens in wild geese, most of the data requirements simply cannot be met with the available data on pathogens potentially transmitted by geese. There are very large gaps in some of the following key determinants of risk: prevalence of pathogens and parasites in geese, epidemiological information to link the pathogen in geese to cases in people or livestock, fecal distribution patterns, and human or livestock exposure patterns (i.e. the nature and extent of contact between geese and humans and geese and livestock). As a result, a reliable, evidence-based risk assessment of health risks to people or livestock from free-ranging geese cannot legitimately be performed with the existing availability and quality of data.

The Canadian Wildlife Service is in a challenging position to manage this issue in which there are rising populations of Canada and Cackling geese, especially of the temperate breeding variety, that are increasingly coming into conflict with human and agricultural activities. In addition, the CWS is faced with very large information gaps on the risk of geese for human and livestock to help it with its decision-making processes. Some strategies for the CWS to consider include:

- Investing in monitoring and research that will provide the CWS with the necessary components to develop an evidence-based risk assessment. Some of the key areas of investigation should be improving our understanding of exposure probabilities (i.e.. nature and extent of goose interactions with people and livestock) but also elucidating the prevalence of specific pathogens for which we have no information for geese, as well as further genetic characterization of strains of pathogens and how they are related to similar human or livestock pathogens. In the last several years, there has been an increasing development and application of advanced diagnostic and survey tools that are helping to elucidate the relationship between goose, human and livestock pathogens. Some of the methods that are increasingly being used include: molecular methods such as sequence analysis (i.e. *Mycobacterium* spp.), phylogenetic analyses, pulsed-field gel electrophoresis, polymerase chain reaction (i.e. E. coli), serologic methods (i.e. influenza, psittacosis), and epidemiological methods (i.e. *Salmonella*, West Nile virus). These pathogen detection methodologies coming on stream give us hope that these gaps can be narrowed over the coming years with molecular-level data that can demonstrate strain overlaps between geese and other species and help track the source of human and livestock infections and outbreaks, particularly those with waterborne origin and for which waterfowl are suggested as a possible source. However, there is a large number of possible hazards widespread over a variety of risk settings in Canada. Investment in such a program of research would need to be large and long-term to make significant shifts in our understanding of these determinants of risk.

- Modeling of risks for some of the priority pathogens identified here as a means of accounting for the high degree of uncertainty. Uncertainty can either be modeled using quantitative or qualitative methods. Increasingly in areas in which there are large data gaps or uncertainty, a mixed approach, such as multi-criteria decision making can be used. However, models would need to account for the issue of context which modifies risk (exposure setting, nature of the at risk group etc). Models are also only as good as the data available hence any modeling attempts would be opinion based or would need to use analytical methods to account for data uncertainty.

- Developing and improving risk reduction and communication strategies that focus on specific higher risk settings (i.e. aquatic resources, hospital settings, and high livestock density areas).

- Forming a working group to develop national standards for the management of peri-urban goose populations. Expertise from public health, regulatory agencies, resource managers, engineers, epidemiologists, and biologists along with input from the public are needed to

develop a consensus on goose management practices. A consensus approach has been used to good effect in other situations where there is a high level of uncertainty. For example, the Northeast Wildlife Damage Management Research and Outreach Cooperative[15] was formed in the US "to enhance coordination and collaboration among wildlife agencies, universities, and other cooperators to promote consistent, multi-state approaches for resolving wildlife-related concerns". This group has examined management issues associated with Canada geese, white-tailed deer, black bears, and beavers. Community-based approaches have also been shown to be effective in developing location-specific methods to address nuisance wildlife issues (Curtis et al, 2005).

[15] http://wildlifecontrol.info/newdm/Pages/default.aspx

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APPENDIX A: OVERVIEW OF PATHOGEN-SPECIFIC CRITERIA RELATED TO POSSIBLE TRANSMISSION FROM GEESE TO HUMANS IN NORTH AMERICA.

Hazard	Prevalence in wild geese in N. America	Prevalence in humans in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to human transmission	References
Bacterial						
Arcobacter spp.	Unknown in wild geese. (European-based study showed 18% (16/90) prevalence in domestic geese)	Low-moderate. Few studies available. (0.15% of diarrheic samples)	Aerotolerant; can persistent for long periods in the environment.	*Similar species (A. cryaerophilus*, *A. skirrowii* and *A. butzleri).* Detected in water reservoir.	None found. No reports of *Arcobacter* in wild geese. But newly identified pathogen so few studies available.	Atabay et al, 2008; Ho et al, 2006; Vandenberg et al, 2004
Borrelia burgdorferi	No specific reports found for geese. (10% of surveyed birds from endemic areas carry agent).	Low Canada: 0.1 per 100,000 reported. US: 8 cases per 100,000 reported (CDC)	Vector borne: distinct endemic areas in Canada. However, distribution of host ticks is expanding.	Yes. Humans are infected via ticks carrying the agent.	None found.	Ogden et al, 2009; CDC, 2007[16]

[16] CDC. Morbidity and Mortality Weekly Report: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5623a1.htm

Hazard	Prevalence in wild geese in N. America	Prevalence in humans in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to human transmission	References
Campylobacter spp.	Variable prevalence in fecal samples: 0-100%.	High. Most common reportable enteric disease. Annually, 30-40 cases per 100,000 in Canada.[17]	Generally poor survival in open air, can survive in cool water. Microaerophilic, sensitive to heat and UV. Higher rates in cooler temps with lower light.	Well-known zoonosis, commonly acquired from poultry meat. Little evidence of cross-contamination genotypes among geese, chickens, starlings (Colles).	High degree of host specificity in goose isolates (i.e. high number of unique clonal complexes noted in geese). Several shared clonal complexes (at low prevalence <1%) between geese and humans (ST-21, 45 and 1034) (not causally related). Probable link between waterborne campylobacter outbreak in humans and pink-footed geese in Norway.	CCDR, 2009[18]; Ogden et al, 2009; Colles et al, 2008; French et al, 2008; Murphy et al, 2006; Clark, 2003; Converse et al, 1999

[17] Canadian Integrated Surveillance Report: http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/09vol35/35s3/
[18] CCDR,2009. http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/09pdf/35s3-eng.pdf

Hazard	Prevalence in wild geese in N. America	Prevalence in humans in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to human transmission	References
Chlamydophila psittaci	No reports found for geese. 10-30% of surveyed avian populations have been found positive.	Rare[19] (<1 case per 100,000).	Resistant to drying. Can persist in feces or respiratory discharge for up to several months.	Many examples of transmission. Human outbreak linked to domestic duck farm (both samples) (both isolates exhibited the same PCR-RFLP restriction pattern).	Several cases of *C. psittaci* reported from occupational exposure during wild bird necropsies, including, among other species, Canada geese and lesser snow geese.	Longbottom and Coulter, 2003; Wobeser and Brand, 1982
C. perfringens	Outbreaks in geese reported in Canada.	Low (1.8 per 100,000/year of all Clostridium spp. 40% of these are C. perfringens) food-borne related cases.	Widely distributed in the environment.	Food-borne infections from uncooked poultry have been documented (no causal link established with infected chickens).	No evidence found.	Holtby et al, 2008; Leal et al, 2008; Wobeser et al, 1987
Erysipelothrix rhusiopathiae (Erysipelas)	Unknown but one report of outbreak on goose farm noted. Worldwide distribution.	Rare.	Can survive for up to a month in soil.	Transmission from pigs most common cause of human infections.	No evidence found.	Brooke and Riley, 1999; Gunning and Morton, 1998

[19] http://www.ccohs.ca/oshanswers/diseases/psittacosis.html

Hazard	Prevalence in wild geese in N. America	Prevalence in humans in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to human transmission	References
Escherichia coli[20]	High in fecal samples (2-100% depending on season). ETEC: 13% EHEC: 6% (no O157:H7 detected in geese). EIEC: 4.6% EAEC: 1.3% Goose feces with human virulence factors: 2%.	~ 5 cases per 100,000 reported.	Prevalence increases with temperature and humidity (higher rates in spring/summer). Recovery rates do not seem to relate to fecal density. Persists for up to one month in water and for much longer on soil or other substrate.	Low level of virulence factors detected in goose feces (2%) (STa and K1 factors).	No direct evidence found. *E.coli* from geese detected in areas of high human use (beaches, parks). 19-38% of water samples from Ontario beaches shown to be contaminated by goose feces (using PCR signal tests).	Edge and Hill, 2007; Kullas et al, 2002; Feare et al, 1999; Mead et al, 1999
Helicobacter spp.	40% of surveyed Canada geese had helicobacter spp in their feces. (PCR). 28% prevalence by fecal culture. Goose -specific species detected (*H. anseris* and *H. brantae). Also H. canadensis.*	Unknown for species shared by geese and people. But one survey found a prevalence of *H. pullorum* of 4%.	Yes. Survives well in fresh water.	*H. pullorum* shown to infect both chickens and humans (disease-causing for both but not causally linked).	No direct evidence. *H. canadensis* detected in diarrheic people and in geese in Europe (not causally linked).	Tsiodras et al, 2008; Fox et al, 2006; Adams et al, 2003; Waldenstrom et al, 2003

[20] ETEC = enterotoxogenic *E. coli*; EHEC = enterohemorrhagic *E.coli* ; EIEC = enteroinvasive *E. coli*; EAEC = enteroagglomerative *E. coli*

Hazard	Prevalence in wild geese in N. America	Prevalence in humans in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to human transmission	References
Legionella pneumophilia	No reports for geese in N. America. 6-23% prevalence rate in domestic geese in China.	Low (<1 case per 100,000). Most cases linked with specific risk factor (i.e. exposure to infected air cooling system).	Yes. Survives well in freshwater (i.e. streams) and in water and cooling systems (i.e. air conditioners).	No animal to animal or animal to human transfer has been demonstrated. But could be source of environmental contamination (i.e. saprozoonsis).	No evidence found.	Clark, 2003; Fields et al, 2002; Steinert et al, 2002
Listeria mono cytogenes	7% of fecal samples carried *Listeria* of which 42% were *L. monocytogenes.* Authors suggest sampling technique might falsely inflate the prevalence rate.	Low (<1 case per 100,000).	Survives well in soil, feces, water, etc. can survive for more than 2 years.	Yes. Livestock, wildlife, and human *L. monocytogenes* strains have overlapping but distinct populations.	One sample had the same genetic fingerprint as a strain involved in human disease. (not casually linked).	Bueno et al, 2010; Lyautey et al, 2007; Swaminathan and Gerner-Smidt, 2007; Converse et al, 1999
Mycobacterium avium	Unknown in geese populations specifically. One case noted in CCWHC database in 2002. <1% of wild Anatidae submitted for necropsy had avian tuberculosis.	Low (1 to 10/100,000 annually). Most common in immune-compromised people.	Can survive in soil and water for prolonged periods.	Broad host range. Experimental cross-species transmission reported. Mycobacterium avium is primarily considered opportunistic saprophyte (i.e. environmental pathogen).	No evidence found.	Soler et al, 2009; Biet et al, 2005; CDC factsheet. Inderlied et al, 1993
Mycoplasma spp.	Unknown in wild geese. Isolated case reports of *Mycoplasma* in domesticated geese.	Low (<1/100,000)	Rapidly inactivated in the environment.	Person-to-person transmission of *M pneumoniae* most common cause of infection.	No evidence found. Different species found in geese than in humans. In geese: *M aneris, M gallinarium, M cloacale.*	Waites and Talkington, 2004; Bunz et al, 1986

Hazard	Prevalence in wild geese in N. America	Prevalence in humans in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to human transmission	References
Salmonella enterica	Low. 0 to 2.5% prevalence in Canada geese.	2nd most common reportable enteric disease. 10-20 cases per 100,000.	Can survive in the environment for at least nine months.	Commonly documented transmission from animals and their product (i.e. eggs) to humans.	No evidence found.	CCDR, 2009[21]; Voetsch et al, 2004; Clark, 2003; Roscoe, 2001; Feare et al, 1999; Mead et al, 1999
Streptococcus spp (Group D) (S. bovis, S. gallolyticus subsp. Pasteurianus)	Unknown. One report of *S. galloyticus* in farmed geese.	Uncommon in people.	Short-lived survival in environment (hours to several days).	Similar strains of *S. gallolyticus* shown in humans and pigeons (not casually linked).	No evidence found.	Herrera et al, 2009; Barnett et al, 2008; Devriese et al, 1998;
Vibrio spp.(non-cholera)	Low. Only one study found 6% prevalence in Canada geese (low sample size (16)). Non-01 vibrio demonstrated in farmed geese.	Low. (<1 / 100,000). Rates increased by 80% between 1996 and 2001 in the US.	Primarily survives in marine environment.	Different species detected in geese vs humans.	No evidence found.	FDA[22]; Dechet et al, 2008; Buck et al, 1990; Schlater et al, 1981
Yersinia pseudotuberculosis	Low. Two studies did not recover *Yersinia* spp. from 'resident' goose fecal samples.	Considered to be rare in people.	Survives well in the environment and in water.	Unknown but animal source(s) suspected.	No evidence found.	Tauxe, 2004; Roscoe, 2001Feare et al, 1999;

[21] CCDR,2009. http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/09pdf/35s3-eng.pdf

[22] FDA, Bad Bug Book: Foodborne Pathogenic Microorganisms and Natural Toxins Handbook *Vibrio cholerae* Serogroup non-01 http://www.fda.gov/Food/FoodSafety/FoodborneIllness/FoodborneIllnessFoodbornePathogensNaturalToxins/BadBugBook/ucm070419.htm

Fungal						
Aspergilla spp.	Reports of die-offs in wild waterfowl, including geese, from feeding on contaminated crops. Commonly found in captive geese. 15 cases noted in CCWHC database (passive surveillance) from 1990 to 20009.	Low. Most common in immuno-compromised patients.	Commonly found in the environment.	Common mold in soil and fungus so environment exposure most important route.	No evidence found.	Latge, 1999; Bowes, 1990; Adrian et al, 1978; McDougal and Vaught, 1968
Candida albicans	Unknown. One report found of disease in farmed geese.	Fourth most commonly recovered blood culture isolate in the US.	Normal flora of humans and animals.	This is a commensal organism; part of the normal GI flora of humans and animals. No direct transfer.	No evidence found.	Leroy et al, 2009; Buck et al, 1990; Beemer et al, 1973
Cryptococcus spp.	*C laurentii* was found in 5 samples from Canada geese in NY state.	Uncommon. Recent outbreak in people and animals of *C. gattii* in Pacific Northwest. *C. laurentii* recently shown to cause several cases of human disease in immune-compromised patients.	Survives in the specialized niches. Ie. pigeon guano (*C. neoformans*), in the tropics and sub-tropics, and now Vancouver Island (*C. gattii*).	Pigeon guano is known to be a source of spores for *C. neoformans.* Few cases of bird to human transfer have been reported.	No evidence found.	Filion et al, 2006; Hoang et al, 2004;

Parasitic						
Cryptosporidium spp.	Low to moderate. 10 and 23% prevalence in cloacal and fecal samples, respectively. In the study (Zhou, 2004) with 23% prevalence, 92% of specimens were goose-adapted genotypes that so far have not been shown to be infectious to people. Infectious cysts of *C. hominis* and of the zoonotic genotype of *C. parvum* have been detected in geese but at low levels (2.4%).	1.85 per 100,000 in Canada (2004 stats). Adult population has a moderate level of seropositivity (~30%). *Cryptosporidium* spp. responsible for half of waterborne disease outbreaks worldwide.	Survives in 15-20°C water: 3-12 months.	Geese can act as mechanical carriers of infectious oocysts of *C. parvum* and distribute into the environment and into water. Geese shown to carry cryptosporidia of goose, ruminant, duck, and human origins. More recent molecular evidence indicates that cryptosporidia are more strongly host-adapted that previously thought.	Source tracking of *C. parvum* in a drinking water supply linked to Canada geese, but oocysts in this case were non-goose host-adapted species shown to be infectious to people. Another study detected goose-adapted strain not known to be infectious in people. No human cases have been directly linked to geese.	Jellison et al, 2009; Pintar et al, 2009; Graczyk et al, 2008; Ruecker et al, 2007; Hunter and Thompson, 2005; Zhou et al, 2004; Roscoe, 2001; Fayer et al, 2000; Graczyk et al, 1998
Giardia spp.	15% prevalence in cloacal samples (Roscoe). Detected in pooled fecal samples from each of 9 nine sites sampled (Graczyk).	13.08 cases per 100,000 in Canada (2004 stats). Giardia spp. responsible for ~40% of waterborne disease outbreaks worldwide.	Survives in 15-20°C water: ~2 months.	Avian isolates of Giardia have caused significant infections in mice. More recent work demonstrates a more significant role of intra-human transmission and places a lower significance on zoonotic transmission.	Direct transmission from geese to people has not been demonstrated.	PHAC, 2009[23]; Graczyk et al, 2008; Yoder and Beach, 2007; Karanis et al, 2007; Hunter and Thompson, 2005; Roscoe, 2001; Graczyk et al, 1998

[23] Public Health Agency of Canada: Notifiable disease surveillance. http://dsol-smed.phac-aspc.gc.ca/dsol-smed/ndis/index-eng.php

Sarcocystis spp.	Unknown. *S. rileyi* 65% prevalence found in the white-fronted goose (Anser albifrons) in Europe.	Low. Case reports only. Did not find any population level studies.	Various stages of the lifecycle take place in the environment.	Different species detected. *S. hominis* or *S. suihominis* pathogenic for humans. *S. rileyi* infect but generally not pathogenic for birds.	No evidence found.	Kutkiene et al, 2008; Fayer, 2004
Schistosome cercariae (swimmer's itch)	Low in Canada geese (< 3% prevalence). Three cases noted in CCWHC database (passive surveillance). Primarily carried by ducks.	6.8 episodes per 100 water-exposure days.	Cycles between waterfowl (primarily ducks) and snails (intermediate stage).	Literature indicates that 'waterfowl' (and intermediate hosts) source of infection for people. Literature focuses on snails.	Only found one study that detected low rate of infection in Canada geese. Another study in BC found 0% (0/122) fecal samples with *S. cercariae.* No direct evidence of transmission from geese.	Verbrugge et al, 2004; Leighton et al, 2000; Loken et al, 1995;
Toxoplasma gondii	First report in 2004 of detection of *T. gondii* in a single Canada goose (type III) from Mississippi. Seroprevalence of waterfowl <16% Several reports of clinical toxoplasmosis in small number of geese.	0-100% seroprevalence in varied human populations.	Sporulated oocysts are very persistent in the environment (up to 18 months). Tissue cysts can survive in refrigerated (1-4°C) meat for up to 3 weeks and can survive freezing (up to -8°C) for longer than a week.	Low genetic variability (Types I, II, and III) among *T. gondi* isolates. Types I and II are thought to be more pathogenic for humans than Type III. Chickens thought to be an important source of infection for humans (Dubey in press).	No direct evidence found.	Yan et al, 2009; Dubey et al, 2007 and 2002;Tenter et al, 2000
Viral						

Arboviruses (Eastern and Western Equine Encephalitis, St. Louis Encephalitis, West Nile Virus)	Reports of outbreaks in domestic geese in Manitoba (25% mortality) and domestic ducks in Saskatchewan (39% mortality). One case of WNV in a Canada goose in CCWHC database. No other reports found.	Low. Average of 700 cases in Canada. Very low rates reported of human infection by SLE, EEE, WEE.	WNV able to survive in overwintering Culex mosquitoes in temperate climates.	Yes. Similar WNV strains between humans and birds. Highest mortality rates shown in corvids.	The WNV circulating in North America has shown similarities to strains from infected geese in Israel. Experimentally, geese had viremia titre sufficient to infect mosquitoes and act as a reservoir host. Transmission between geese by direct contact has been demonstrated.	Wojnarowicz et al, 2007; MacLean, 2006; Austin et al, 2004
Avian influenza	Waterfowl, primarily dabbling ducks, are a natural reservoir for AIV. Detected in 0.8% of Canada geese. Dominant strains in waterfowl are Low Pathogenic and include H3N8, H4N6, H4N8, H6N2, H6N8, and H9N2. One case of H7N3 (low pathogenic strain) detected in a dead Canada goose from British Columbia in 2007.	Very low. Two cases of human infection (conjunctivitis) linked to poultry outbreak of type H5N2 in British Columbia in 2005.	AIV can survive for prolonged periods in water.	Yes. Transmission from domestic poultry to humans has been demonstrated. The only evidence of direct transmission from wild birds to people was between H5N1- infected swans and people plucking their feathers (Azerbaijan). Contradicted by a British example of humans exposed to H5N1- infected swans did not develop disease or seroconvert.	Serologic evidence (H11/N9) of transmission between waterfowl and a duck hunter and two wildlife professionals with many years of exposure to waterfowl has been shown.	Wallenstein et al, 2010; Berhane et al, 2009; Tsiodras et al, 2008; Parmley et al, 2008 ; Pasick et al, 2007; Clark and Hall, 2006; Olsen et al, 2006; Gill et al, 2006

Coronavirus (SARS and non-SARS variants)	Unknown in Canada goose. Detected by cloacal swab in Graylag geese in Norway.	SARS: negligible (previous outbreak contained). Non-SARS: low (<4% prevalence).	Can survive for several days on a variety of surfaces but converts to a non-infectious form after about 90 minutes.	Some molecular evidence is available but not conclusive (civet cats in Asia to humans).	No evidence found.	Jonassen et al, 2005; Weiss and Navas-Martin, 2005

APPENDIX B: OVERVIEW OF PATHOGEN-SPECIFIC CRITERIA RELATED TO POSSIBLE TRANSMISSION FROM GEESE TO LIVESTOCK IN NORTH AMERICA.

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Bacterial						
Actinobacillus suis	Rare. Only found one report about a single case in a Canada goose from a wildlife refuge.	Swine: fairly common, 16-94% herd-prevalence (Ontario study).	Common commensal organism in pigs.	A. suis isolated from horses, cats, and dogs. A few isolates found with similar genotypes, pheno-types as *A. suis* strains from pigs.	No evidence found.	MacInnes et al, 2008; Jeannotte et al, 2002; Maddux et al, 1987
Arcobacter spp.	Unknown in wild geese. (European based study showed 18% prevalence in clinically normal domestic geese).	Prevalence in fecal samples: 44% of porcine, 39% of bovine, 16% of ovine and 15 of equine samples (Belgian study). Poultry: some studies detected no Arcobacter, others up to 15% prevalence.	Aerotolerant, can persistent for long periods in the environment.	*Similar species (A. cryaerophilus*, *A. skirrowii* and *A. butzleri). A. cibarius* detected in poultry and effluent from pig farms. Detected in water reservoir. Found in feces from both clinically healthy and ill livestock animals.	None found. No reports of Arcobacter in wild geese. But newly identified pathogen so few studies available.	Atabay et al, 2008; Ho et al, 2006; Vandenberg et al, 2004; Van Driessche et al, 2003

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Borrelia anserina	Unknown in wild geese in North America.	Low Historical significance.	Primarily found in tropical and subtropical regions where it's transmitted by ticks in the *Argas* spp.	Poultry susceptible to same species.	No evidence found.	Buckland and Guy, 2002
Borrelia burgdorferi	No specific reports found for geese. (10% of surveyed birds from endemic areas carry agent). Mallard ducks are susceptible to infection; become persistently infected for at least 43 days, develop spirochete-emia, and shed in feces for up to 29 d.	Negligible in non-endemic areas. Seroprevalence in dairy cattle in endemic areas: 17%.	Vector borne: distinct endemic areas in Canada. Prevalence of nymphs in endemic areas: 25%. However, distribution of host ticks is expanding.	Yes. Wide range of mammalian, avian and reptile hosts are infected via Ixodid ticks carrying the agent.	No evidence found.	Ogden et al, 2009; Morshed et al, 2006; Barker and Lindsay, 2000; Ji and Collins, 1994
Campylobacter spp.	Variable prevalence in fecal samples: 0-100%.	Campylobacter often found in the intestines of most domestic species. Prevalence in feces in the 20% range for cattle, sheep, goats. Higher in poultry (40%).	Generally poor survival in open air, can survive in cool water. Micro-aerophilic, sensitive to heat and UV. Higher rates in cooler temps with lower light.	Little evidence of cross-contamination genotypes among geese, chickens, starlings (Colles).	High degree of host specificity in goose isolates (i.e. high number of unique clonal complexes noted in geese). No other evidence found.	Ogden et al, 2009; Colles et al, 2008; Murphy et al, 2006; Clark, 2003; Converse et al, 1999

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Chlamydophila psittaci	No reports found for geese. 10-30% of surveyed avian populations.	Outbreaks reported in domestic turkeys and ducks. Chickens have been shown to be quite resistant to *C. psittaci.*	Resistant to drying. Can persist in feces or respiratory discharge for up to several months.	Yes. Demonstrated transmission to humans.	No evidence found.	Zang et al, 2008; Longbottom and Coulter, 2003
Clostridium botulinum	Large scale outbreaks, in waterfowl throughout North America, with very high mortality rates.	Rare. Cluster outbreaks have occurred in cattle and sheep that have ingested contaminated poultry litter.	*C. botulinum* is a strict anaerobe that forms dormant spores in adverse conditions. Spores are very resistant to environmental decay.	Most exposure is due to presence of spores in environment. However, spores also carried in tissues of birds and distributed to new environments in their feces.	No evidence found.	Otter et al, 2006; Rocke, 2006; Wobeser and Rannie, 1987
Clostridium perfringens	Outbreaks in geese reported in Canada. Usually from 'grain overload' after grazing on crops following periods of low nutritional intake (i.e. migration).	Uncommon.	Widely distributed in the environment.	Human food-borne infections from uncooked poultry to have been documented (no causal link established with infected chickens).	No evidence found.	Leal et al, 2008; Wobeser et al, 1987
Erysipelothrix rhusiopathiae (Erysipelas)	Unknown but one report of outbreak on goose farm noted. Worldwide distribution.	Present on most pig farms. Commonly vaccinated against in swine.	Can survive for up to a month in soil.	Transmission from pigs most common cause of human infections.	No evidence found.	Gunning and Morton, 1998

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Escherichia coli[24]	**High in fecal samples** (2-100% depending on season). ETEC: 13% EHEC: 6% (no O157:H7 detected in geese). EIEC: 4.6% EAEC: 1.3% Goose feces with human virulence factors: 2%.	Common cause of neonatal disease in calves and piglets. 3-40% of diarrheic calves are positive for enterotoxigenic E. coli.	Prevalence increases with temp. and humidity (higher rates in spring/summer). Recovery rates relate poorly to fecal density. Can persist for 1 month in water and much longer on soil or other substrate.	Low level of human virulence factors detected in goose feces (2%) (STa and K1 factors).	No direct evidence found. However, serotypes of importance to humans and human virulence factors were detected at low levels in geese.	Edge and Hill, 2007; Kullas et al, 2002; Feare et al, 1999; Mead et al, 1999; Radostits et al, 1994
Helicobacter spp.	40% of surveyed Canada geese had *Helicobacter* spp in their feces. (PCR). 28% prevalence by fecal culture. Goose -specific species detected (*H. anseris* and *H. brantae). Also H. canadensis.*	Unknown for species indentified in geese. H. pullorum relatively common in poultry. Species specificity (pigs carry *H. suis*, cattle carry *H. bovis*).	Yes. Survives well in fresh water.	The pathogenic potential of *H. anseris* and *H. brantae* are unknown. *H. pullorum* shown to infect both chickens and humans (disease-causing for both but not- causally linked).	No evidence found. Couldn't find evidence of same species in geese and livestock species.	Haesebroucket al, 2009; Tsiodras et al, 2008; Fox et al, 2006; Adams et al, 2003; Waldenstrom et al, 2003; Atabay et al, 1998

[24] ETEC = enterotoxogenic *E. coli*; EHEC = enterohemorrhagic *E.coli* ; EIEC = enteroinvasive *E. coli*; EAEC = enteroagglomerative *E. coli*

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Listeria monocytogenes	0-7% of fecal samples carried Listeria of which 42% were *L. monocytogenes.* Authors suggest sampling technique might falsely inflate the prevalence rate.	13% prevalence in cattle. Subclinical carriage is common.	Saprophytic organism so survives well in soil, feces, water, etc. Can survive for more than 2 years.	Yes. Livestock, wildlife, and human *L. monocytogenes* strains have overlapping but distinct populations.	No evidence found.	Bueno et al, 2010; Lyautey et al, 2007; Converse et al, 1999
Mycobacterium avium	Unknown in geese populations specifically. One case noted in CCWHC database in 2002. <1% of wild Anatidae submitted for necropsy had avian tuberculosis.	Infects a wide-range of domestic species.	Can survive in soil and water for prolonged periods.	Broad host range. Experimental cross-species transmission reported. *Mycobacterium avium* is primarily considered opportunistic saprophyte (i.e. environmental pathogen).	No evidence found. Transmission between wildlife and livestock difficult to demonstrate because of long incubation period and difficulty excluding other sources of infection.	Soler et al, 2009; Biet et al, 2005; Inderlied et al, 1993
Mycoplasma spp.	Unknown in wild geese. Case reports of *Mycoplasma* in domesticated geese. 3% of live wild ducks were positive for Mycoplasma spp. in North America.	*Mycoplasma* is generally a cofactor for avian respiratory disease. Ie. synergistic with other pathogens such as avian influenza.	Rapidly inactivated in the environment.	Unknown.	No evidence found. Different species found in geese (*M aneris, M cloacale*) and poultry (M gallisepticum). *M gallinarium* in common but only *M. gallisepticum* considered significant in poultry.	Bercic et al, 2008; Boettger and Dohms, 2006; Goldberg et al, 1995; Stipkovits et al, 1993; Bunz et al, 1986

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Pasteurella multocida	Unknown. < 5% seroprevalence in Greater White-fronted geese (*Anser albifrons frontalis*) in Alaska. Epizootics, with significant mortalities, more common in wintering grounds in US than in Canada. "Outbreaks have been happening regularly on breeding grounds in the eastern arctic (annually since 2004) and Saint Lawrence Estuary (not annually but commonly, for decades) in common eider colonies, but a few geese including Canada geese (in eastern arctic) have also died during these outbreaks. Not sweeping numbers for geese compared to the eiders." (pers. comm., Catherine Soos)	Commonly occurs in respiratory tracks of domestic species (sheep, cattle, poultry).	Reasonably short-lived in the environment. Shown to survive less than seven weeks in wetlands following avian cholera outbreaks.	Reported in a wide variety of birds and mammals. Identical strains (using PFGE) noted between poultry flocks and wild birds (Eiders). Experimental transmission demonstrated between Lesser Snow geese and Pekin ducklings.	No direct evidence found. Only one report found of outbreak in Canada geese (Windingstad et al, 1983).	Blanchong et al, 2006; Samuel et al, 2005; Pedersen et al, 2003; Samuel et al, 1997; Botzler, 1991; Windingstad et al, 1983

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Salmonella enterica	Low. 0 to 2.5% prevalence in Canada geese.	30% in dairy herds, 50% in chicken flocks, 54% in turkey flocks.	Can survive in the environment for at least nine months.	Nearly 3% of the isolates from domestically raised ducks and geese were 'wild bird' strains of *Salmonella.* Not necessarily from wild geese and no casual link established.	No evidence found.	Arsenault et al, 2007; Pennycott et al, 2006; Feare et al, 1999
Streptococcus spp (Group D) (S. bovis, S. gallolyticus subsp. Pasteurianus)	Unknown. One report of *S. galloyticus* in farmed geese.	Thought be part of 'normal flora' in mammals.	Short-lived survival in environment (hrs to several days).	Similar strains of *S. gallolyticus* shown in humans and pigeons (not casually linked).	No evidence found.	Herrera et al, 2009; Hogg and Pearson, 2009; Barnett et al, 2008; Devriese et al, 1998;
Vibrio spp.(non-cholera)	Low. Only one study. 6% prevalence in Canada geese (low sample size (16)). Non-01 vibrio cholera and *V. metschnicovii* demonstrated in farmed geese.	Rare. Isolated reports in livestock (cattle, sheep, goats, pigs).	Primarily survives in marine environment.	Similar species detected in livestock species. *V. metschnicovii* detected in chickens and turkeys.	No evidence found.	Bush et al, 2006; Hinz et al, 1999; Buck et al, 1990; Schlater et al, 1981
Yersinia pseudotuberculosis	Low. Two studies did not recover *Yersinia* spp. from 'resident' goose fecal samples.	Common in sheep, pigs, and cattle.	Survives well in the environment and in water.	Unknown but animal source suspected.	No evidence found.	Tauxe, 2004; Roscoe, 2001; Feare et al, 1999;
Fungal						

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Aspergilla spp.	Reports of die-offs in wild waterfowl, including geese, from feeding on contaminated crops. Commonly found in captive geese. 15 cases noted in CCWHC database (passive surveillance) from 1990 to 20009.	Outbreaks are common in chicks and poults.	Commonly found in the environment. Inhalation of fungal spores dispersed from mouldy feed, lit- ter, meal, grains, etc. primary route of infection.	Common mold in soil and fungus so environment exposure most important route.	No evidence found.	Akan et al, 2002; Latge, 1999; Bowes, 1990; Adrian et al, 1978
Candida albicans	Unknown. One report found of disease in farmed geese. 1/16 fecal samples positive in wild Canada geese.	Natural commensal that can occasionally cause opportunistic infection.	Normal flora of humans and animals.	This is a commensal organism; part of the normal GI flora of humans and animals. No direct transfer.	No evidence found.	Gaudie et al, 2009; Buck et al, 1990; Beemer et al, 1978
Cryptococcus spp.	*C. laurentii* was found in five samples from Canada geese in NY state.	Rare. Cryptococcal mastitis report in cattle and goats. No reports of *C. laurentii* in livestock.	Survives in the specialized niches. Ie. pigeon guano (*C. neoformans*), in the tropics and sub-tropics (*C. gattii*), Vancouver Island (C. gattii).	Pigeon guano is known to be a source of spores for *C. neoformans.* Environmental pathogen.	No evidence found.	Filion et al, 2006; Duncan et al, 2006
Parasitic						
Coccidia sp.	*Eimeria truncata* considered to be a significant pathogen of waterfowl. 65% prevalence in lesser snow geese.	Different species of coccidia commonly found in all livestock species.	Sporulated oocysts are resistant to environmental extremes; their sporozoites are infectious for months.	All reports with E. truncata were limited to geese.	No evidence found.	USGS, 1999; Gomis et al, 1996; Clinchy et al, 1994

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Cryptosporidium spp.	Low to moderate. 10 and 23% prevalence in cloacal and fecal samples, respectively. Zhou et al (2004) found 23% prevalence, 92% of specimens were goose-adapted genotypes. Infectious cysts of *C. hominis* and of the zoonotic genotype of *C. parvum* have also been detected in geese but at low levels (2.4%).	Common in cattle, particularly in calves. C. parvum and C. andersoni are the most common species.	Survives in 15-20°C water: 3-12 months.	Geese can act as mechanical carriers of infectious oocysts of *C. parvum* and distribute into the environment and into water. More recent molecular evidence indicates that cryptosporidia are more strongly host-adapted that previously thought.	Source tracking of *C. parvum* in a drinking water supply linked to Canada geese. Geese shown to carry *Cryptosporidia* spp. of goose, ruminant, duck, and human origins.	Jellison et al, 2009; Pintar et al, 2009; Graczyk et al, 2008; Ruecker et al, 2007; Hunter and Thompson, 2005; Zhou et al, 2004; Roscoe, 2001; Fayer et al, 2000; Graczyk et al, 1998
Giardia spp.	15% prevalence in cloacal samples (Roscoe). Detected in pooled fecal samples from each of 9 nine sites sampled (Graczyk).	Common in livestock.	Survives in 15-20°C water: ~2 months.	Avian isolates of Giardia have caused significant infections in mice. More recent work demonstrates a more significant role of intra-human transmission and places a lower significance on zoonotic transmission.	High concentrations of cysts in Canada geese suggest infection rather than mechanical carriage of Giardia (i.e. increased transmission rate).	Graczyk et al, 2008; Olsen et al, 2004; Yoder and Beach, 2007; Karanis et al, 2007; Hunter and Thompson, 2005; Roscoe, 2001; Graczyk et al, 1998

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Sarcocystis spp.	Unknown. *S. rileyi* 65% prevalence found in the white-fronted goose (Anser albifrons) in Europe.	Low.	Various stages of the lifecycle take place in the environment.	Different species detected. *S. hominis* or *S. suihominis* pathogenic for humans. *S. rileyi* infect but generally not pathogenic for birds.	No evidence found.	Kutkiene et al, 2008; Fayer, 2004
Toxoplasma gondii	First report in 2004 of detection of *T. gondii* in a single Canada goose (type III) from Mississippi. Seroprevalence of waterfowl <16%. Several reports of clinical toxoplasmosis in small number of geese.	Low.	Sporulated oocysts are very persistent in the environment (up to 18 months). Tissue cysts can survive in refrigerated (1-4°C) meat for up to 3 weeks and can survive freezing (up to -8°C) for longer than a week.	Low genetic variability (Types I, II, and III) among *T. gondi* isolates. Types I and II are thought to be more pathogenic for humans than Type III.	No evidence found.	Yan et al, 2009; Dubey et al, 2007 and 2002;Tenter et al, 2000
Viral						

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Arboviruses (Eastern and Western Equine Encephalitis, St. Louis Encephalitis, West Nile Virus)	WNV: Reports of outbreaks in domestic geese in Manitoba (25% mortality) and domestic ducks in Saskatchewan (39% mortality). No reports of WNV in Canada geese.	WNV: low in chickens and turkeys. Chickens and turkeys develop very low virus titres and more limited clinical signs than geese.	WNV able to survive in overwintering Culex mosquitoes in temperate climates.	Yes. Similar WNV strains between humans and birds. Highest mortality rates shown in corvids.	The WNV circulating in North America has shown similarities to strains from infected geese in Israel. Experimentally, geese had virus titre sufficient to infect mosquitoes and act as a reservoir host. Transmission between geese by direct contact has been demonstrated.	Wojnarowicz et al, 2007; MacLean, 2006; Austin et al, 2004; Banet-Noach et al, 2003; Swayne et al, 2001; Senne et al, 2000
Avian adenovirus	Antibodies in geese and ducks are widespread. Outbreak in domestic geese in Canada reported. In this case, no contact between domestic geese and wild geese had been noted.	Distributed widely throughout the world. High seroprevalence.	In general, short-lived in the environment. Inactivated after 30 minutes in aqueous solutions.	Only experimental evidence of transmission whereby goose strains grow in vitro on poultry cells.	No evidence found.	Chen et al, 2009; Saif, 2003; Ivanics et al, 2001; Riddell, 1984

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Avian influenza	Waterfowl, primarily dabbling ducks, are a natural reservoir for AIV. Detected in 0.8% of Canada geese. Dominant strains in waterfowl are Low Pathogenic and include H3N8, H4N6, H4N8, H6N2, H6N8, and H9N2. One case of H7N3 (low pathogenic strain) detected in a dead Canada goose from British Columbia in 2007.	Of importance only to poultry. Several major outbreaks of HPAI have occurred in Canada in BC and Saskatchewan.	Remains infectious in freshwater for up to 4 days at 22^0C and more than 30 days at 0^0C.	Yes. AIV zoonotic transmission has been demonstrated. In BC outbreak, two poultry handlers developed conjunctivitis.	No evidence found.	Wallenstein et al, 2010; Tsiodras et al, 2008; Pasick et al, 2007; Clark and Hall, 2006; Gill et al, 2006
Avian pneumovirus	Single study found. Virus detected in Canada geese. Geese and ducks shown to be generally refractory to virus.	No reports of pneumovirus in Canada (likely due to difficulties with virus detection).	Can remain viable for 7 days at room temperature.	Suggestive findings that waterfowl, including geese, can spread APV to turkeys.	No evidence found.	Safi, 2003; Cook et al, 2000; Shin et al, 2000;

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Avian pox	Isolated cases detected. Geese not considered to be a major reservoir.	More common in warmer, humid climates.	High environmental stability, remain contagious for up to several months in an ambient environment. Highly resistance to drying. Especially when released into the environment in dermal crusts, serum, blood residues and other excretions.	Attempts to experimentally transmit pox virus from goose to chickens and ducks failed. Fairly host specific.	No evidence found.	Clark, 2003; Cox 1980
Coronavirus	Unknown in Canada goose. Detected by cloacal swab in Graylag geese in Norway.	Common in livestock (up to 70%). Sub-clinical infections frequently occur.	Can survive for several days on a variety of surfaces but converts to a non-infectious form after about 90 minutes.	Some molecular evidence is available but not conclusive (civet cats in Asia to humans).	No evidence found.	Jonassen et al, 2005; Weiss and Navas-Martin, 2005; Crouch and Acres, 1984.

Hazard	Prevalence in wild geese in N. America	Prevalence in livestock species in N. America	Detection and persistence of agent in environment	Evidence for cross-species transmission	Evidence for goose to livestock transmission	References
Newcastle disease virus (NDV)	High seroprevalence (44% in adults) and geographically widespread in Canada. Waterfowl considered natural reservoir, particularly of mild form of virus.	Highly virulent form has been eradicated from developed countries.	Prolonged survival on eggshells and in feces.	Wild birds suspected in transmission to poultry in Europe, but not proven. Virulent strains isolated from migratory waterfowl have been experimentally transmitted to domestic poultry that showed evidence of pathogenicity acquired during passage in the infected chicken population.	No evidence found.	Dai et al, 2008; Clark, 2003; Alexander et al, 1999; Palmer and Trainer, 1970

APPENDIX C: CHARACTERISTICS OF IDENTIFIED HAZARDS THAT COULD IMPACT HUMANS (MAGNITUDE OF POTENTIAL IMPACT)

Hazard	Route of exposure	What does it do?	Severity of illness				Nationally Reportable (humans)[25]	References	Magnitude ranking
			Treatable	Severity of symptoms	Frequency of hospitalization[26]	Frequency of fatality			
Bacterial									
Arcobacter spp.	Ingestion	Enteritis, bacteremia	yes	Mild to moderate	Low	Low	No	Ho et al, 2006; Vandenberg et al, 2004	Low
Borrelia burgdorferi	Vector	Multisystemic: general malaise, skin, cardiac, musculoskeletal, and neurological symptoms	yes	Mild (when detected early) Moderate-severe if left undiagnosed **Morbidity:** low Canada: 0.1 per 100,000 reported. In US: 8 cases per 100,000	Rare	Rare	No	Ogden et al, 2009; CDC, 2007[27]	Medium

[25] List of human reportable diseases in Canada: http://dsol-smed.phac-aspc.gc.ca/dsol-smed/ndis/list_e.html

[26] Note: hospitalization rates documented here likely overestimate the true hospitalization rate due to inherent biases in the data and reporting methods. Ie. under-reporting, hospital-based studies, etc.

[27] CDC. Morbidity and Mortality Weekly Report: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5623a1.htm

Hazard	Route of exposure	What does it do?	Severity of illness				Nationally Reportable (humans)[25]	References	Magnitude ranking
			Treatable	Severity of symptoms	Frequency of hospitalization[26]	Frequency of fatality			
Campylobacter spp.	Ingestion	Enteritis, arthritis, sepsis	Yes	Mild-moderate	Low 25-100 cases per year[2]	Low 0.07 per 1000 cases[2]	Yes	CCDR, 2009[28] **Morbidity:** Most common reportable enteric disease. Annually, 30-40 cases per 100,000 in Canada[29]	Medium
Chlamydophila psittaci	Aerosol	General malaise, pneumonia	Yes	Mild	Rare	Rare	No	Longbottom and Coulter, 2003	Negligible
C. perfringens	Ingestion	Enteritis, gas gangrene, sepsis	Yes, but resistance an issue	Moderate to severe depending on underlying conditions (e.g. age, cancer, kidney disease)	Often for hospital-acquired cases and patients at risk	High, dependent on study design (i.e. hospital-based vs community) (25-50%)	No	Leal et al, 2008	Medium

[28] CCDR,2009. http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/09pdf/35s3-eng.pdf
[29] Canadian Integrated Surveillance Report: http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/09vol35/35s3/

Hazard	Route of exposure	What does it do?	Severity of illness				Nationally Reportable (humans)[25]	References	Magnitude ranking
			Treatable	Severity of symptoms	Frequency of hospitalization[26]	Frequency of fatality			
Erysipelothrix rhusiopathiae (Erysipelas)	Direct contact	Cutaneous infection, endocarditis	Yes	Mild (cutaneous form) to severe (systemic form)	low	Low (cutaneous form to high (systemic form)	No	Brooke and Riley, 1999	Low
Escherichia coli	Ingestion	Enteritis, hemolytic uremic syndrome (with enterotoxigenic strain)	Yes	Mild to severe depending on strain	Low to high depending on strain	Low to moderate depending on strain	Yes (ETEC)	Karmali et al, 2010; OIE, 2008	High (ETEC, EHEC). Otherwise low
Helicobacter spp.	Ingestion	Chronic gastritis, peptic ulcer disease, recently linked to gastric adenocarcinoma and lymphoma	Not always	Mild to severe	Low	Rare	No	Tsiodras et al, 2008; Fox et al, 2006	Low
Legionella pneumophilia	Aerosol	Pneumonia	Yes	Moderate	Moderate	Moderate (7-24%), highest in immune compromised and elderly	Yes	Fields et al, 2002; Steinert et al, 2002	Medium
Listeria monocytogenes	Ingestion	Enteritis, meningitis, sepsis, fetal infection	Yes	Mild to severe	Moderate	Moderate (20-30%)	Yes	Swaminathan and Gerner-Smidt, 2007	Medium

Hazard	Route of exposure	What does it do?	Severity of illness				Nationally Reportable (humans)[25]	References	Magnitude ranking
			Treatable	Severity of symptoms	Frequency of hospitalization[26]	Frequency of fatality			
Mycobacterium avium	Ingestion (water borne) and aerosol	Pneumonia	Yes	Mild to moderate	Low (primarily in HIV patients)	Low (~10% in HIV patients and much lower in non-HIV)	Yes	Inderlied et al, 1993	Low
Mycoplasma spp.	Aerosol	Tracheobronchitis, pneumonia, CNS and skin manifestations	Yes	Mild to moderate	Low	Low	No	Waites and Talkington, 2004	Low
Salmonella enterica	Ingestion	Enteritis	Yes	Mild to moderate	Low (<20%)	Low (<1%)	Yes	CCDR, 2009[30]; Voetsch et al, 2004	Medium
Streptococcus spp (Group D) (S. bovis, S. gallolyticus subsp. Pasteurianus)	Ingestion	Endocarditis, enteritis, implicated in colorectal neoplasia (*S. bovis*)	Yes	Moderate to severe	High	High for S. bovis endocarditis (45%) Moderate for non S. bovis endocarditis (25%)	No	Herrera et al, 2009;	Medium

[30] CCDR,2009. http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/09pdf/35s3-eng.pdf

Hazard	Route of exposure	What does it do?	Severity of illness				Nationally Reportable (humans)[25]	References	Magnitude ranking
			Treatable	Severity of symptoms	Frequency of hospitalization[26]	Frequency of fatality			
Vibrio spp.(non-cholera)	Water-borne agent: Ingestion or direct contact	Enteritis, septicemia, wound infection	Yes	Mild to moderate	Moderate to high depending on Vibrio sp. (20-80%)	Low (10-20%)	No	FDA[31]; Dechet et al, 2008	Medium
Yersinia pseudotuberculosis	Ingestion	Enteritis	Yes	Mild	Moderate (30%)	Low	No	Nuorti et al, 2004; Hannu et al, 2003	Low
Fungal									
Aspergilla spp.	Aerosol Ingestion	Pneumonia	Not always	Moderate to severe	High	Moderate	No	Latge, 1999	Medium
Candida albicans	Commensal org	Oropharyngeal, genital, and systemic manifestations	Yes	Negligible to moderate	Low – moderate depending on underlying conditions	Low-moderate depending on underlying conditions	No	Leroy et al, 2009	Low

[31] FDA, Bad Bug Book: Foodborne Pathogenic Microorganisms and Natural Toxins Handbook *Vibrio cholerae* Serogroup non-01 http://www.fda.gov/Food/FoodSafety/FoodborneIllness/FoodborneIllnessFoodbornePathogensNaturalToxins/BadBugBook/ucm070419.htm

Hazard	Route of exposure	What does it do?	Severity of illness				Nationally Reportable (humans)[25]	References	Magnitude ranking
			Treatable	Severity of symptoms	Frequency of hospitalization[26]	Frequency of fatality			
Cryptococcus spp.	Aerosol	Cutaneous, respiratory and neurologic manifestations	Yes	Negligible to severe	Low	Low	No	Hoang et al, 2004	Low
Parasitic									
Cryptosporidium spp.	Ingestion	Enteritis	Yes	Low to moderate (in immune-compromised patients)	Low	Low	Yes	Pintar et al, 2009; Fayer et al, 2000	Medium
Giardia spp.	Ingestion	Enteritis	Yes	Mild	Low	Low	Yes	Yoder and Beach, 2007	Medium
Sarcocystis spp.	Ingestion	Enteritis	No. Only supportive therapy	Negligible to mild	Rare	Rare	No	Fayer, 2004	Low
Schistosome cercariae (swimmer's itch)	Direct contact (water)	Dermatitis	Supportive	Low	Rare	Rare	No	Leighton et al, 2000	Low

Hazard	Route of exposure	What does it do?	Severity of illness				Nationally Reportable (humans)[25]	References	Magnitude ranking
			Treatable	Severity of symptoms	Frequency of hospitalization[26]	Frequency of fatality			
Toxoplasma gondii	Ingestion	Lymphadenitis, encephalitis, myocarditis, retinochoroiditis, neonatal death	Yes	Negligible to severe	Low	Low to moderate (in immunocompromised)	No	Tenter et al, 2000	Medium
Viral									
Arboviruses (Eastern and Western Equine Encephalitis, St. Louis Encephalitis, West Nile Virus)	Vector	Encephalitis, meningitis, general malaise	No. Only supportive therapy	Negligible – Severe	Low	Low	Yes	Petersen and Marfin, 2002; Petersen and Hayes, 2008	High (West Nile)
Avian influenza	Aerosol, waterborne	General malaise, pneumonia [antivirals are only effective if therapy started within 1st 48hrs after clinical onset]	Yes. (see column on left)	Low to severe	Low-high depending on variant	Low-high depending on variant	Yes	Clark and Hall, 2006	High (HPAI)
Coronavirus (SARS and non-SARS variants)	Aerosol	General malaise, pneumonia, respiratory failure (SARS variant)	No. Supportive therapy.	Low to severe depending on variant	Low to high depending on variant	Low to high depending on variant	No	Talbot et al, 2009; Weiss et al, 2005	Low (non-SARS) High (SARS)

APPENDIX D: CHARACTERISTICS OF IDENTIFIED HAZARDS THAT COULD IMPACT LIVESTOCK (MAGNITUDE OF POTENTIAL IMPACT)

Hazard	Livestock species	Route of exposure	What does it do?	Treatable	Severity of signs	Fatality rate	Vaccine preventable	Reporting requirements [32]	References	Magnitude ranking
Bacterial										
Actinobacillis suis	Swine	Direct, normal flora in oral cavity	Arthritis, pneumonia, pericarditis, nephritis	Yes	Negligible to severe	High		None	Radostits et al, 1994	medium
Arcobacter spp.	Cattle, pigs, sheep	Ingestion (possibly direct contact)	Reproductive disorders, enteritis	Yes	Negligible to moderate	Low	No	None	Houf et al, 2009; Ho et al, 2006	Low
*Borrelia anseria (*avian borreliosis)	Poultry	Vector	Septicemia	Yes	Mild to severe	High	Yes	Annually notifiable	Lisboa et al, 2009	High
Borrelia burgdorferi	Cattle and sheep	Vector	Arthritis, decreased milk production	Yes	negligible to mild	Low		None	Radostits et al, 1994	Low

[32] Animal disease reporting to the CFIA: http://www.inspection.gc.ca/english/anima/disemala/guidee.shtml
Reportable diseases: "[diseases that are] usually of significant importance to human or animal health or to the Canadian economy"
Immediately notifiable diseases (for laboratories only): "diseases [that] are exotic to Canada for which there are not control or eradication programs"
Annually notifiable diseases (for laboratories only): "diseases for which Canada must submit an annual report to the World Organization for Animal Health (OIE) indicating their presence in Canada, but are not classified as reportable or immediately notifiable"

Campylobacter spp.	Cattle, swine, poultry	Ingestion	Enteritis in young animals, abortions in cattle, swine, sheep	Yes	Negligible to moderate	Low	No	None	Radostits et al, 1994	Low
Chlamydophila psittaci	Poultry and domestic waterfowl	Aerosol, ingestion, vector	Pneumonia, septicemia	Yes	Negligible to moderate	low		Immediately notifiable	Coulter and Longbottom, 2003	Medium
Clostridium botulinum Type C (avian botulism)	Poultry and domestic waterfowl	Ingestion	Flaccid paralysis	No	Moderate to severe	Moderate to high	Yes	Annually notifiable	Merck, 2008	Medium
C. perfringens ('necrotic enteritis')	Cattle, sheep, pigs, goats	Ingestion	Enteritis	Often not treatable	Moderate to severe	Moderate to high	Yes	Annually notifiable	Radostits et al, 1994	Medium
Erysipelothrix rhusiopathiae (Erysipelas)	Swine, sheep	Ingestion	Cutaneous infection, arthritis, septicemia	Yes in acute cases	Moderate to severe	Moderate to high	Yes	Annually notifiable	Radostits et al, 1994	Medium
Escherichia coli	All	Ingestion	Enteritis	Yes	Mild	Mild	Yes	Annually notifiable (STEC)	Karmali et al, 2010	Medium
Helicobacter spp.	Poultry, swine	Ingestion	Enteritis Hepatitis	Yes	Negligible to moderate	Low	No	None	Tsiodras et al, 2008; Ceelen et al, 2007; Fox et al, 2006 ; Atabay et al, 1998	Medium
Listeria monocytogenes	All	Ingestion	Septicemia, encephalitis, reproducti	Yes	Mild to severe	Moderate		Annually notifiable	Merck, 2008; Radostits et al, 1994	Medium

			ve disorders							
Mycobacterium avium	Cattle, pigs, Poultry	Aerosol or ingestion	Chronic weight loss, multi-systemic disease	No	Severe	High		Annually notifiable	Radostits et al, 1994	High
Mycoplasma sp	All	Aerosol or direct transmission	Pneumonia, arthritis, reproductive disorders, peritonitis, air-sacculitis, salpingitis	Varies	Mild to severe	Low to high		Annually notifiable	Merck, 2008; Stipkovits et al, 1993	Medium
Pasteurella multocida (Fowl cholera)	All	Ingestion	Septicemia	Yes	Severe	High	Yes	Immediately notifiable	Pedersen et al, 2003	Medium
Salmonella enterica	All	Ingestion	Enteritis, septicemia	Yes if started early	Low to moderate depending on serovar	Low to moderate depending on serovar		Annually notifiable	Radostits et al, 1994	Medium
Streptococcus spp (Group D) (S. bovis, S. gallolyticus subsp. Pasteurianus)	Cattle, poultry	Ingestion	Cattle: bloat, rumen acidosis, mastitis Poultry: septicemia, endocarditis	Yes	Moderate	Low to moderate		None	Herrera et al, 2009; Sekizaki et al, 2008	Low

*Vibrio spp. (*non-cholera)	All	Ingestion	Enteritis, encephalitis	Unknown	Moderate	Moderate		Annually notifiable	Bush et al, 2006; Hinz et al, 1999; Visser et al, 1999	Medium
Yersinia pseudotuberculosis	Cattle, pigs, sheep	Oral	Enteritis	Yes	Low	Low		None	Radostits et al, 1994	Low
Fungal										
Aspergilla spp.	Cattle, pigs, poultry, sheep	Aerosol Ingestion	Pneumonia, encephalitis, reproductive disorders,	No	Low to moderate	Low to moderate	No	None	Akan et al, 2002; Radostits et al, 1994	Medium
Candida albicans	Cattle, pigs, poultry	Commensal	Pneumonia, mastitis	Varies	Low to moderate	Moderate	No	None	Gaudie et al, 2009; Merck, 2008; Radostits et al, 1994	Medium
Cryptococcus sp	Goats, cattle	Aerosol	Pneumonia, encephalitis	Unknown. But likely not	Severe (limited info available)	Unknown	No	None	Baro et al, 1998	Medium
Parasitic										
Coccidia spp.	All	Ingestion	Enteritis	Yes	Low	Low		Annually notifiable	Radostits et al, 1994	Low
Cryptosporidia spp.	All	Ingestion	Enteritis	No. supportive	Low to moderate depending on underlying conditions	Low		None	Thompson et al, 2008	Medium
Giardia spp.	All	Ingestion	Enteritis	Yes	Low	Low		None	Thompson et al, 2008	Low
Leucocytozoon	Poultry	Vector	Multi-	No	Negligi	Moderat		None	Hellgren et al,	Medi

spp.			systemic		ble to moderate	e			2008; Merck, 2008; Bennett et al, 1982	um
Nematodes: *Amidostomum* ssp. , *Epomidiostomum* spp., *Trichostrongylus spp.*	Domestic waterfowl	Ingestion	Lethargy	Yes	Low	Low		None	Nowicki et al, 1995	Low
Sarcocystis spp.	Cattle, goats, sheep, poultry	Ingestion	Weight loss and unthriftiness, abortions, encephalitis	No	Low to moderate	Low		None	Dubey et al, 2006; Mutalib et al, 1995	Medium
Toxoplasma gondii	Pigs, goats, sheep, poultry (rare)	Ingestion	Abortions, pneumonia, encephalitis, endocarditis	Only supportive therapy	Moderate to high	Moderate		Annually notifiable	Dubey et al, 2006 and 2007	Medium
Viral										
Arboviruses (Eastern and Western Equine Encephalitis, St. Louis Encephalitis, West Nile Virus)	Poultry, domestic waterfowl	Vector	Encephalitis	No	Negligible to high	Negligible to high		Immediately notifiable	Austin et al, 2004; Senne et al, 2000	High
Avian adenovirus	Poultry, domestic waterfowl	Ingestion	Decreased egg production, poor eggshell quality, enteritis,	No	Moderate	Low		Immediately notifiable	Merck, 2008; Ivanics et al, 2001; Raj et al, 2001; Hess, 2000	Medium

			pneumonia							
Avian herpesvirus (duck viral enteritis)	Domestic waterfowl	Direct, Ingestion (water)	Multi-systemic	No	Moderate	Low to high		Annually notifiable	Merck, 2008; Campagnolo et al, 2001; Kaleta et al, 1990	Medium
Avian influenza	Pigs, poultry	Ingestion, Aerosol	Pneumonia, decreased egg production, multisystemic disease	No	Variable depending on strain	Variable depending on strain		Reportable (Highly pathogenic)	Lager et al, 2009; Pannwitz et al, 2009; Olsen et al, 2006	High
Avian pneumovirus	Poultry, turkeys in particular	Direct	Respiratory disease	No	Moderate to high	Low to moderate	Yes	Immediately notifiable	Cook, 2000; Shin et al, 2000; Merck, 2008	Medium
Avian pox virus	Poultry	Direct, inhalation, mechanical	Cutaneous lesions, systemic disease	No	Moderate	Low (cutaneous form) High for (diptheric/systemic form)		None	Merck, 2008; Buller et al, 1991	Low
Coronavirus	Group I and II: pigs and cattle Group III: poultry	Aerosol, Ingestion	Respirator disease, enteritis, systemic	No	Moderate to high	Moderate to high		None	Merck, 2008; Jonassen et al, 2005	Medium
Duck hepatitis virus	Domestic waterfowl	Oral	Hepatitis, Sudden death	No	High in young	Low in adults, high in young	Yes	Immediately notifiable	Merck, 2008: Kim et al, 2007	Medium
Goose parvovirus (Derzsy's	Domestic geese	Ingestion	Multisystemic	No	High	High	Yes	Immediately notifiable	Yang et al, 2009; Jansson et al, 2007	Medium

disease)										
Newcastle disease virus	Poultry	Aerosol, Ingestion	Respiratory disease, neurologic signs, enteritis	No	variable depending on strain	variable depending on strain		Reportable	OIE Technical Disease Card	High
New-type gosling viral enteritis	Domestic geese	Ingestion	Enteritis	No	High	High		None	Chen et al, 2009	Medium
Goose Hemorrhagic Polyomavirus	Domestic geese	Ingestion	Nephritis	No	High	High	Yes	None	Lacroux et al, 2004; Guerrin et al, 2000	Medium
Reticuloendotheliosis virus	Poultry	Direct	Neoplasia	No	High in young	High in young	Yes	None	Lin et al, 2009; Cheng et al, 2006	Medium