A REVIEW OF PATHOGENS OF AGRICULTURAL AND HUMAN HEALTH INTEREST FOUND IN CANADA GEESE

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Abstract: The roles that waterfowl in general, and Canada geese in particular, have in the dissemination and transmission of viral and bacterial diseases of human or agricultural importance are covered in this review. In addition to the biological information about the etiology of the disease, economic impacts and zoonotic potential of viral and bacterial pathogens are considered. In most cases existing evidence suggests the importance of waterfowl in disease dissemination and transmission, however, definitive data are often lacking, indicating the need for more directed studies before quantitative risk assessments can be made. Finally, a brief assessment of management options is considered.

Key words: avian influenza, avian pox, bacteria, Campylobacter, Canada goose, E. coli, exotic Newcastle disease, foot and mouth disease, waterfowl, zoonoses

INTRODUCTION

The study of wildlife disease from an animal damage management perspective focuses on four areas: (1) the role that wildlife has in the dissemination and transmission of pathogens with zoonotic potential, (2) the role that wildlife has in the dissemination and transmission of pathogens that affect domesticated animals, e.g., livestock, and poultry, (3) the economic consequences of wildlife disseminated and transmitted diseases, and (4) possible management options to disrupt dissemination and transmission of pathogens. Implicit in this treatment of wildlife disease in the context animal damage management is the process of risk assessment commonly used in epidemiology.

This review is intended as a source of information about common viral and bacterial pathogens of zoonotic and animal health concern. Moreover, this review focuses on pathogens of concern that have been documented to occur within waterfowl in general, and Canada geese, Branta canadensis, in particular. Where the data allow, the review attempts to address the role of waterfowl as host/reservoirs for pathogens of concern, the possibility of transmission to humans, animal stock, or poultry, and the economic or human health consequences of the manifested disease. This review is not intended as a compendium of diseases of geese or waterfowl. That is to say, waterfowl may simply be involved in carriage of the pathogen in some cases. Thus, for the purpose of this review, the etiological agents considered are those that are pathogenic to humans, domestic stock, or poultry, and they may or may not cause disease in the waterfowl or geese.

The special reference to Canada geese is justified because non-migratory Canada
Goose populations have increased eight fold over the past 20 years in North America. One consequence of this population build up has been an increased number of nuisance related complaints due to the geese and their feces. Most people do not come into direct contact with the geese, but they more often encounter Canada Goose feces which had been lying on the ground. Many complaints frequently focus on public health concerns regarding fecal contamination of parks and waterways, and to a lesser extent the problem they may pose to agriculture.

**VIRUSES**

**Avian Influenza**

Avian Influenza (AI) is caused by type A viruses belonging to the Orthomyxovirus group (Easterday et al. 1997). Viruses within this group vary considerably in their virulence. The H5 and H7 strains are extremely virulent and are also highly contagious. Commercially, chickens and turkeys are at risk, with the animal health and economic consequences of outbreaks being considerable (Hahn and Clark 2002). During 1983-84 an outbreak of AI in the poultry flocks of Pennsylvania, Virginia, and Maryland resulted in the destruction of over 17 million birds. This outbreak resulted in costs to producers of $55 million in direct losses, with and additional $8 million in associated clean-up costs. Of the total $63 million in costs, 40 million of those dollars eventually came at taxpayer expense in the form of indemnification to the producers. Direct costs to the consumer, reflected in increased retail prices of poultry food products after the outbreak, were estimated to be $349 million over a 6 month period.

Waterfowl surveys within the Atlantic flyway during the 1983-84 epizootic found 24 strains, including the highly pathogenic H5N2 strain isolated from poultry farms in PA, indicating a spillover from waterfowl to chickens or vice versa (Deibel et al. 1985). Regardless, the detection of virulent AI in migratory waterfowl implicates them as a risk factor in pathogen dissemination.

AI viruses occur widely in wild birds, especially waterfowl, and most strains are characterized by low pathogenicity (Bahl et al. 1977, Alexander 2000). For example, antigenically related H5N2 viruses from geese, replicated in chickens but did not produce disease (Hinshaw et al. 1986). However, even AI viruses of low pathogenicity have the potential to become virulent through mutation and reassortment. This high reassortment capacity for interspecies transmission to terrestrial poultry and mammals and the ability to transform to a virulent form is of concern from a health and economic standpoint (Guan et al. 2002a, 2002b). During 1996-1997 non-pathogenic strains were detected in egg-layer flocks in Lancaster, PA. Because of concern of the virus mutating to a virulent form, 9 flocks were destroyed and a quarantine was imposed by the state (Hahn and Clark 2002). Similarly, a low virulent strain of AI virus was isolated in Virginia in March 2002. The control and containment efforts cost $13 million in destruction of flocks, $50 million in paid indemnities, and an overall cost of $129 million to the industry in an effort to minimize the trade impacts (Hahn and Clark 2002). AI viruses are not only of concern to the poultry industry, but of some concern to human health as well. In 1997, an outbreak of avian flu in humans caused 18 illnesses and 6 deaths. The outbreak was traced to the H5N1 strain whose origin was from a goose at a live bird market in Hong Kong. In 2003, during an outbreak of a virulent strain of AI (H7N7) in the Netherlands, there were 82 confirmed cases of human H7N7 influenza and a veterinarian treating affected flocks died from acute respiratory disease syndrome traced to
the H7N7 strain (Fouchier 2003). More alarming was the observation that there were 3 cases of secondary infection, i.e., from poultry workers to their immediate family, raising concern for pandemic potential.

Waterfowl are an important reservoir for AI viruses (Deibel et al. 1985). Canada geese show variability in the prevalence of AI, but can be considered an important reservoir as well (Easterday et al. 1968, Winkler et al. 1972, Boudreault et al. 1980). Given their capacity for migratory travel and utilization of agricultural areas (pastures and water sources), transmission by direct contact, fecal contact, or indirect contact via farm workers exposed to environmental contamination poses a serious level of risk to human health and the poultry industry (Figure 1, Webster 1998, Webster et al. 2002).

Figure 1. Possible routes of exposure and dissemination of pathogens between geese and poultry.

Newcastle Disease

Newcastle disease virus (NDV) is single a stranded RNA virus belonging to the genus *Paramyxovirus*. Virions are highly contagious and cause respiratory disease in birds (Alexander 2000). The most virulent strains (i.e., velogenic strains, e.g., OIE List A avian paramyxovirus serotype 1 (APMV-1) will cause 100% mortality in chicken flocks and is of critical concern to the poultry industry. Moderately virulent strains (i.e., mesogenic strains) will result in less mortality but severely depress egg production in commercial chickens. The least virulent strains (i.e., lentogenic strains, e.g., APMV-2 to APMV-9 strains) cause little mortality except in young birds, but will result in decreased egg production. Thus, lentogenic strains are of commercial concern for layer hens, but do not represent a significant risk for broilers.

Over 250 species of domestic and wild birds have been infected with various strains of NDV, suggesting that most birds are susceptible to the disease (Kaleta and Baldouf 1988). As stated above, the consequences of infection varies with the strain of virus and the host species. Transmission occurs through the respiratory route via aerosols. The virus also may be fecally shed and acquired via ingestion (Burridge et al. 1975). There is no evidence of vertical transmission.

Some investigators believe that the risk of transmission from waterfowl to poultry is low. Bolte et al. (2001) showed that domestic geese do not readily excrete NDV. Because wild geese are unlikely to come in direct contact with poultry operations and little shedding may occur, the authors conclude that wild geese do not play a major role in the epidemiology of Newcastle disease for poultry. Moreover, waterfowl, including Canada geese, are reservoirs of low pathogenic (lentogenic: APMV-2 to APMV-9) strains of virus (Rosenberger et al.1974, 1975; Ito et al. 1995; Graves 1996), which are generally of lower concern to poultry producers. However, virulent strains (APMV-2) have been isolated from migratory waterfowl and these isolates have been experimentally transmitted to domestic poultry that showed evidence of pathogenicity acquired during passage in the infected chicken population (Takakuwa et al. 1998). Given the high to moderate prevalence of the
viral strains in a variety of waterfowl species (Pearson and McCann 1975, Spalatin and Hanson 1975, Bahl et al. 1977, Deibel et al. 1985, Graves 1996, Takakuwa et al. 1998), vigilance regarding their role is dissemination over long distances and into agricultural situations should be maintained (Pearson and McCann 1975, Hlinak et al. 1998). Finally, the likelihood of mechanical transmission is high. The virus is easily transported by farm workers into poultry flocks (Burridge et al. 1975). Thus, direct contact between waterfowl and poultry may not be needed for waterfowl to be a significant risk factor in disease dissemination and transmission (Figure 1).

There is only minor concern relative to the zoonotic potential of Newcastle disease (Deng et al. 1997). Affected individuals tend to be farm workers in association with poultry houses, and the disease is manifested in the form of mild conjunctivitis.

The poultry industry practices an aggressive vaccination program to control NDV. However, the vaccines are not effective against all strains of NDV. In particular exotic strains have been particularly resistant to vaccination and can severely impact the poultry industry. In 1971, a major outbreak of a APMV-1 velogenic strain (exotic Newcastle disease, END) occurred in California. The outbreak affected over 1,300 flocks and resulted in the destruction of 12 million birds. Eradication efforts cost $56 million, with $275 million (in 1971 $) in clean-up costs. Eradication and clean-up took four years (Hahn and Clark 2002). Adjusting for inflation the control and clean up costs total $1.16 billion in 2003 dollars. These costs do not consider the costs of lost markets, trade embargos, and increased prices to consumers. Using the range of cost ratios (i.e., control:market effect costs) calculated for the avian influenza outbreaks in Pennsylvania and Virginia in 1983 and 1997, the total cost of the 1971 END outbreak is estimated to be $6.4 billion in 2003 dollars. In 2002 and 2003, several outbreaks of END were reported throughout the United States. In California, 22 commercial operations were affected and 3.5 million birds were slaughtered at a cost of $10-15 million. Outbreaks in Nevada, Arizona, New Mexico, and Texas were of limited scope, yet they raised fears about the economic consequences if the containment operations were to have failed.

**Foot and Mouth Disease**

Foot and mouth disease (FMD) is a viral disease of Picorniviridae Apthoviruses with over 7 immunological serotypes and over 60 subtypes. FMD is an economically important disease affecting over 70 mammal species, primarily cloven hoofed domestic mammals. Reptiles and birds are generally resistant, however, birds, including geese, have been experimentally infected (Kaleta 2002). Birds may serve as mechanical vectors for short distances, carrying the virus on plumage or on their feet, thus setting the potential for long distance dissemination (Kaleta 2002). However, the Scottish Executive Rural Affairs Department has considered geese to be very unlikely agents in the dissemination of the virus (Lamont 2001). FMD has low zoonotic potential.

**Avian Pox**

Avian pox is caused by several strains of *Avipoxvirus*. The virus causes warty growths on the feet, legs, base of beak, eye margins, and internal epithelial tissues. This can lead to difficulty breathing, feeding, or perching. Transmission can occur with ingestion of contaminated food or water, contact with contaminated surfaces, or via mechanical vectors such as mosquitoes. Waterfowl are not considered a major reservoir or vector for this disease, though Canada geese have been documented as being
infected. The strain of avian pox virus isolated from the infected Canada geese was successfully transmitted to domestic geese, but not to leghorn chickens or domestic ducks (Cox 1980). Avian pox is not known to be zoonotic. Thus, avian pox from geese does not seem to pose a risk to domestic stock or human health.

**BACTERIA**

**Campylobacter**

Infections by *Campylobacter* spp. are leading causes of human enteritis (Meade 2000). Food animals are the major reservoir for organisms with human infection occurring after consumption of contaminated food. However, up to 20% of *Campylobacter* enteritis cases are attributable to infections via exposure to environmental contaminants (Meade et al. 1999), for which domestic and wild animals are implicated as the source of the pathogen. Migratory waterfowl, and in particular Canada geese, should be considered high risk species for environmental contamination by *Campylobacter* (Pacha et al. 1988, Aydin et al. 2001). However, the prevalence for *Campylobacter* spp. found in goose feces varies widely among studies. Converse et al. (2001) did not isolate *Campylobacter* in fecal samples from Massachusetts, New Jersey, and Virginia, while two studies centered in Ohio obtained 52.0 and 38.9% prevalences (Fallacara et al. 2001). In a national survey for the prevalence of Campylobacter in Canada goose feces, Clark et al. (unpublished data) found the following: California (15.4% in spring and 58.3% in fall); Colorado (11.1% in spring), New York (11.5% in spring), Oregon (0% in spring and fall), Washington (8% in spring), Wisconsin (20% in spring), where the sample sizes for each season and state were, n = 25, and spring samples were taken in April-May, while the fall samples were collected in September - November.

The contribution of wildlife to the carriage and transmission of drug resistant strains of bacteria also is of concern for disease management in agricultural settings. All strains of *C. jejuni* (n=12) isolated from domestic free-ranging geese were resistant to penicillin G and cephalothin; 92% were resistant to sodium deuroxime, and 67% were resistant to cloxacillin, ampicillin, and colistin sulphate; 25% were resistant to tetracycline, and 8% were resistant to sulfamethoxazole/trimethoprim and kanamycin (Aydin et al. 2001).

*Campylobacter* does not survive well in the environment. Thus, human health risks associated with contact with feces, or contamination of turf, are presumed to be low. Nonetheless, our surveillance shows that *Campylobacter* survival is adequate in fecal samples up to 24 hrs post deposition, suggesting some moderate level of environmental risk exposure may occur.

**Coliform bacteria**

Coliform bacteria are often benign, but some strains may adversely affect disease and mortality risks. In the public health arena, coliform counts in water supplies and food samples are used as a correlative index for human health risk. Hussong et al. (1979) examined the impact of migratory geese and swans on the water quality of the Chesapeake Bay. They found that overwintering migrants were a source of human pathogenic *E. coli* and caused increased coliform counts in the esturine waters. In London parks, the prevalence of human pathogenic strains of *E. coli* in Canada goose feces was 55% (Feare et al. 1999). More detailed studies of *E. coli* in Canada goose feces by Kullas et al. (2002) showed that the prevalence of human pathogenic serogroups was 25% in Colorado: 12% of the strains were consistent with Enterotoxic human pathogenic serogroups;
6% were consistent with Enterohemorrhagic human pathogenic serogroups; 5% of the strains were consistent with Enteroaggregative human pathogenic serogroups, and; the remaining 2% were consistent with other human pathogenic serogroups of *E. coli*. In their national survey, Clark et al. (unpublished data) showed the prevalence for the virulence determinants Sta, Stb, and K1 capsular antigen to range between 2 and 4% of fecal samples. Neither Kullas et al. (2002) nor Clark et al. (unpublished data) found evidence for the human virulence determinants: *eae*, *Hly-A*, shiga-like toxins 1 or 2, or cell necrotic factors 1 or 2. No study has isolated the highly virulent strain O157:H7 from goose feces (Converse et al. 2001, Feare et al. 1999, Roscoe 2001, Fallacara et al. 2001).

At the present time there is no direct epidemiological evidence to link human or livestock illness to *E. coli* derived from waterfowl. However, increasingly studies are documenting the virulence determinants that waterfowl may carry that will allow a quantitative risk assessment. Such assessments will determine whether management policies should also include human health.

**Salmonella**

Although *Salmonella* infection of domestic poultry is widespread, prevalence in Canada geese, as indicated by fecal sampling is low. No *Salmonella* spp. were isolated by Hussong et al. (1979), Roscoe (2001), and Fallacara et al. (2001), while prevalences of 2.5%, 0.4%, and 1.0%, were found by Feare et al. (1999), Converse et al. (2001), and Kullas et al. (2002), respectively. However, Salmonella infection of cattle, which can cause abortion, has been linked to a variety of management practices such as contact of wild geese with cattle or their feed (Warnick et al. 2001).

**Other bacteria**

The role wild waterfowl play in the carriage and transmission of other pathogenic bacteria has not been systematically documented. In their surveys of goose feces (n > 6,000), Clark et al. (unpublished data) found several isolates of *Aeromonas hydrophila* and *Vibrio tubiashi*, both are of concern for the health and production of the shellfish industry and can have human health consequences. Feare et al. (1999) found higher prevalences of *A. hydrophila* (12%), underscoring the observation that geese may largely reflect local environmental contamination as well as acting as disseminators of pathogenic agents. Other investigators have also isolated various *Vibrio* species from goose feces (Buck 1990, Schlater et al. 1981).

*Bordetella avium* causes respiratory disease of poultry. Three strains of *B. avium* have been isolated from Canada geese, two of which were indistinguishable from clinical specimens isolated from domesticated turkeys (Raffel et al. 2002). Thus, Canada geese can act as carriers, and possibly reservoirs for this pathogen. These findings underscore the need to ensure that farm biosecurity measures include physical and procedural barriers between pastures, where geese may be present, to poultry houses.

*Legionella pneumophila* is a serious pathogen for respiratory illness. In one study, *L. pneumophila* was isolated from 6-23% of geese (Liu et al. 1989). Thus, geese may be of general epidemiological concern as a source of environmental contamination.

Toxoplasmosis is a serious disease of the respiratory system caused by *Toxoplasma gondii*. Sixl et al (1978) found an epidemiological risk association for pregnant women who had been exposed to waterfowl.

**SUMMARY**

Populations of Canada goose (*Branta*...
canadensis) have dramatically increased in North America over the past 40 years (Sauer et al. 2001). Increasingly, these geese are utilizing urban parks, recreation areas, and corporate and residential lawns to the point that they frequently are classified as nuisance animals. Because geese produce prodigious quantities of feces (Bedard 1986) there has been concern that the geese may pose human health risks (Conover and Chasko 1985, Cooper and Keefe 1997). While no direct link between contact with goose feces and human illness has been made, there is increasing evidence that human virulence determinants are present in goose feces. Despite the growing concern about the role Canada geese and their feces may play in human health risks, the data on prevalence of disease organisms are few. More studies are needed in order to better assess what risks and exposures the public encounters when using landscapes inhabited by geese.

Similar observations apply to the agricultural landscape. Here the issues revolve around the role of waterfowl as host/reservoir species for pathogens of agricultural concern, the patterns and use of pastures and farm ponds by waterfowl, the degree of environmental contamination by pathogens, and how those pathogens might make their way to livestock and poultry. This review illustrates that geese and other waterfowl have the potential to act as reservoirs and carriers of agricultural diseases. What is needed at this point is a risk assessment for how important these wildlife species are to the transmission of pathogens to animal stock and poultry.

**LITERATURE CITED**


jejuni. Canadian Journal of Microbiology 34:80-82.


SIXL, W., D. STUNZNER, AND H. WITHALM. 1978. Epidemiologic and serologic study of listeriosis in man and domestic and wild animals in Austria. Journal of Hygiene, Epidemiology, Microbiology and Immunology 22:460-469.


