

THE EMERGENCE OF MAJOR AVIAN DISEASES IN NORTH AMERICA: WEST NILE VIRUS AND MORE

ROBERT G. MCLEAN, USDA, APHIS, Wildlife Services, National Wildlife Research Center, 4101 LaPorte Avenue, Fort Collins, CO 80521, USA

Abstract: Some major diseases of wild birds have emerged dramatically in North America recently due in part to the rapid global movement of animals and pathogens and major changes in the size, quality, and continuity of natural habitats. These diseases include invasive pathogens, emergence of new disease agents or syndromes, and reemergence of some established diseases. A prominent example is West Nile virus that invaded North America, became easily established, and has expanded throughout the continent within 4 years. A number of factors have influenced the frequency of occurrence, the variety of species affected, and the geographical extent of these emergent diseases. Management and control is limited by the transitory nature of many bird species, difficulties in diagnosing the diseases, and the lack of adequate resources.

Key words: avian cholera, avian disease, botulism, conjunctivitis, west nile virus

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INTRODUCTION

Some major diseases of free-ranging wild birds have invaded, emerged or reemerged during the last few decades in North America (Friend et al. 2001). These diseases have increased in frequency of occurrence, prominence and geographical distribution; and are affecting a wide variety of avian species. Diseases in wildlife have changed during the last few decades from sporadic, self-limiting outbreaks with minor losses to that of frequently occurring mortality events with major losses of wildlife. Some zoonotic diseases of wildlife that affect humans have emerged dramatically worldwide during this same time period. Some of the causes of invasive and emergent diseases are the unprecedented worldwide population growth that has resulted in enormous destruction, fragmentation, and deterioration of natural habitats and in increased human encroach-

ment into wilderness areas. At the same time, international travel and worldwide transport of animals, food products, and movement of disease pathogens have increased dramatically. People and animals can travel rapidly between continents during the incubation period of diseases and introduce new pathogens into naïve populations. Changes in animal production and handling and food processing along with the development of resistance to antibiotics and other antimicrobial drugs have allowed bacterial pathogens to expand and affect wildlife. Finally, changes in major ecosystems and climate changes that affect the distribution and population sizes of wildlife species and disease vectors have enhanced transmission of disease agents and influenced wildlife losses from diseases (McLean 2001).

MAJOR AVIAN DISEASES

Some of the important emerging diseases are urban geese associated diseases, antibiotic resistant bacteria, house finch

conjunctivitis, trematode infections of waterbirds, avian botulism, avian cholera, avian vacuolar myelinopathy, and West Nile virus (WNV) (Table 1).

Table 1. Examples of diseases of major concern among North American birds (adapted from Friend et al. 2001).

<u>Disease</u>	<u>Cause</u>	<u>Primar Species Affected</u>	<u>Year First Reported</u>	<u>Location</u>
West Nile	Virus	Corvids	1999	New York City, NY
Pathogenic Bacteria	Bacteria	Canada geese	1999	Fort Collins, CO
Leyogonimus	Trematode Parasite	American coot	1996	Shawano Lake, WI
House finch Conjunctivitis	Bacteria	House finch	1994	Washington D.C.
Avian vacuolar Myelinopathy	Unknown (toxin?)	Bald eagle, American coot, waterfowl	1994	DeGray Lake, AR
Avian Cholera	Bacteria	Waterfowl	1944	North America
Avian botulism	Toxin	Waterfowl	1890's	North America

Conjunctivitis, an infectious disease caused by the bacteria *Mycoplasma gallisepticum*, was identified in house finches (*Carpodacus mexicanus*) in Washington D.C. in 1994 (Ley et al. 1996) and then rapidly spread westward to the Mississippi River within 3 years causing substantial finch mortality throughout their range in the eastern U.S. (Fischer et al. 1997). Avian vacuolar myelinopathy, an emerging neurological disease syndrome, was discovered killing bald eagles (*Haliaeetus leucocephalus*) and American coots (*Fulica americana*) at DeGray Lake, Arkansas, in 1994 (Thomas et al. 1998). The disease is likely caused by a toxin(s) and also affects waterfowl, other avian species, and a few aquatic mammals. The disease distribution has expanded and now occurs at man-made lakes in numerous locations in the southeastern U.S. A trematode parasite, *Leyogonimus polyoon*, known previously in Eastern Europe was

likely introduced in infected snails into Shawano Lake, Wisconsin. The parasite amplified in the intermediate aquatic snail hosts in the lake and was transmitted to American coots causing epizootics in 1996-97 that killed over 12,000 birds (Cole and Friend 1999). The disease appears unique to that lake environment and has not yet spread to other locations. Avian cholera, caused by the bacteria *Pasteurella multocida*, and avian botulism, caused by type C toxin produced by the bacteria *Clostridium botulinum*, are the most common diseases of North American waterfowl. These two diseases have been reported for a number of years but have reemerged as a major problem throughout many areas in the western U.S. causing frequent epizootics with major bird losses. The bird mortality has also affected a large number of species (Friend 1999, Rocke and Friend 1999). An aberrant form of type C avian botulism has emerged at the Salton Sea in southern

California where fish are affected and serve as a source of toxin for fish-eating birds. An epizootic in these birds occurred there in 1996 and was responsible for a major loss of 15 to 20% of the western population of white pelicans (*Pelicanus erythrorhynchos*) and of a large number of the endangered brown pelican (*Pelicanus occidentalis*) (Friend et al. 2001). Avian botulism now causes annual mortality of pelicans at the Salton Sea. The rapidly increasing populations of urban Canada geese (*Branta canadensis*) are contaminating recreational waters and land use areas used by the public with pathogenic bacteria that may pose some human health risks (Clark 2003). Birds may also be the source of and disseminators of emerging pathogenic bacteria that affect livestock at confined animal feeding operations and dairies.

WEST NILE VIRUS

West Nile virus was previously known only from Africa, the Middle East, Europe, and western Asia until it was introduced into the U.S. in New York City (NYC) in 1999. The virus quickly became established causing a regional epizootic in the bird population, mostly in American crows (*Corvus brachyrhynchos*), followed by a localized human epidemic of 62 cases and seven deaths (CDC 1999). The strain of WNV introduced was especially virulent for many North American bird species and caused significant mortality in corvids (crows, jays, magpies, and related species) (McLean 2002a, Komar et al. 2003). Mortality in the avian host species for the other arboviruses in the U.S. (St. Louis encephalitis, eastern equine encephalitis, and western equine encephalitis viruses) is unusual (McLean et al. 2002), but the significant bird mortality from WNV quickly became useful to public health officials to detect the presence of the virus (Eidson et al. 2001).

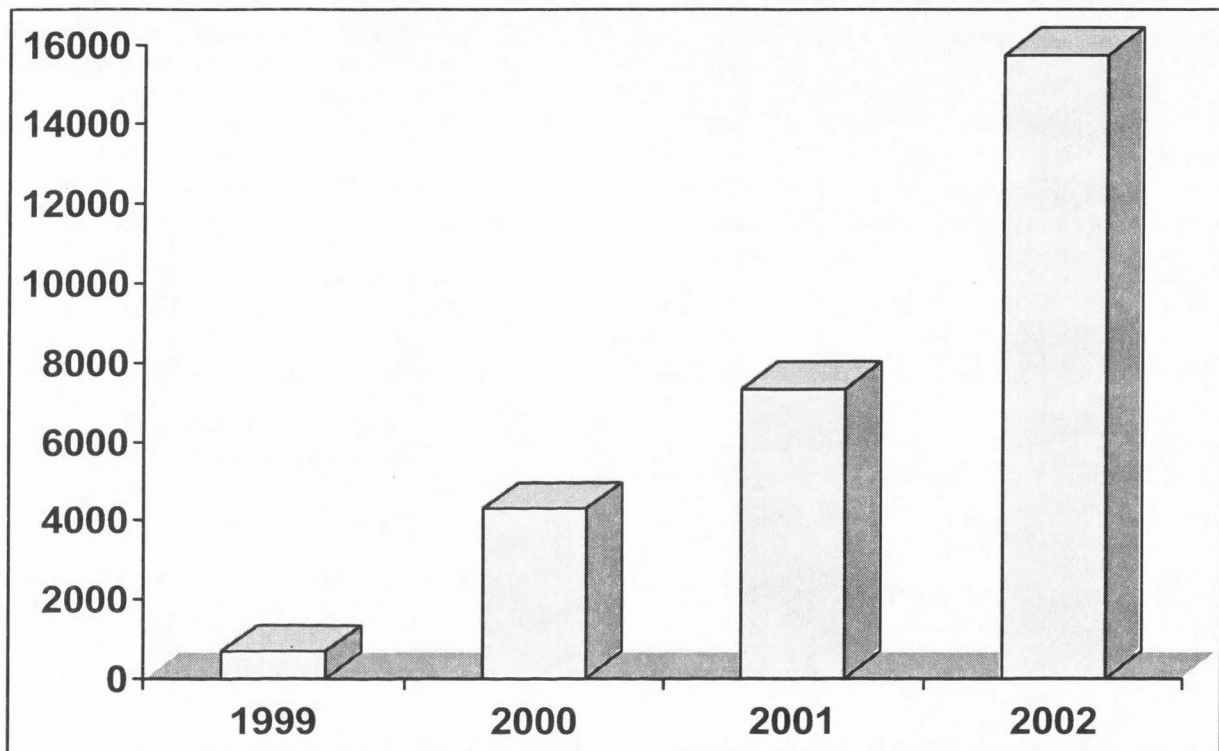
State-wide surveillance was started in 2000 in the Atlantic and Gulf coastal states by the Centers for Disease Control and Prevention (CDC) to track the expansion of WNV from the affected area in the northeast. The surveillance system used multiple techniques: dead bird surveillance (virus testing of dead wild birds), monitoring antibody conversions in sentinel chickens, mosquito capture and testing, and veterinary and human surveillance (CDC 2001a). New molecular technology to detect virus/antigen in specimens (polymerase chain reaction, PCR) provided rapid testing of birds and mosquitoes and aided in tracking the movement of the virus, and sensitive and specific serologic testing enable the rapid detection and subsequent confirmation of human and equine cases (Lanciotti et al. 2001). A rapid field diagnostic assay (VecTest) was developed to test mosquitoes for WNV antigen (Ryan et al. 2003) and is being evaluated as a rapid field test for corvids who excrete large amounts of virus when infected (Komar et al. 2002). If successful, this field testing capability will allow field biologists to obtain rapid information about WNV infections in dead birds. A national system (ArboNET) for surveillance data was established and maintained by CDC to allow weekly reporting and updating of WNV surveillance information by all states involved in the surveillance and maps of the reported information were prepared weekly by the U.S. Geological Survey (Marfin et al. 2001).

The surveillance network detected the early reappearance of the virus in crows in May 2000 within the area previous affected by the virus (epicenter) indicating its survival through the winter of 1999-2000. By the end of the summer transmission season in 2000, WNV in birds was detected in 12 states and a total of 12,961 dead birds were submitted for virus testing and 4,305

(33%) were virus positive. American crows comprised 58% of the birds tested and 89% (3,824) of the positive birds (Marfin et al. 2001). In 2001, WNV reappeared even earlier in the spring in a larger area in the northeastern states and a new infected area was detected in northern Florida (CDC 2001b) that was likely established there in the fall of 2000 by southward migrating birds traveling from the infected zone in the northeast. The virus distribution quickly expanded to 27 eastern states and Ontario,

Canada accompanied by increases in human and equine cases, dead birds (Figure 1), and positive mosquitoes (CDC 2002a). The rapid geographical expansion of WNV to the southeastern and mid-western states in 2001 was likely aided by the seasonal movement of migratory birds that move along four major migratory corridors in North America each season between breeding areas in the north and wintering areas in the south (McLean et al. 2001).

Figure 1. The number of dead wild birds laboratory confirmed with West Nile virus in the United States, 1999-2002.



The increased disease intensity and dramatic geographical expansion of WNV in 2002 was remarkable. Virus activity began early in the season in Louisiana and was detected subsequently in a number of the previously affected eastern states and Canada. New foci of WNV transmission were soon discovered in Texas followed by states in the Great Plains and Rocky

Mountain region, and in the central provinces of Canada. Virus activity became diffused across a large region of the North American continent affecting 44 states, Washington D.C., and five Canadian provinces, and caused a more than fifty-fold increase in the number of human cases (58 to >4100) and nearly twenty-fold increase in equine cases (738 to >14,000) in the U.S. as

a consequence of similar increases in wild bird infections and mortality (Figure 1) (CDC 2002b). In 2002, new methods of direct transmission between humans through organ transplants, blood transfusions, infant nursing, and intrauterine infection became evident (CDC 2002b). The large number of vertebrate host species affected increased and included captive rocky mountain goats, sheep, reindeer, additional exotic bird species, and domestic pets. A large die-off of owls and hawks may have been caused by WNV and captive-reared alligators died from WNV infection (Miller et al. 2003). Dissemination of WNV by migratory birds appeared more apparent as the virus spread throughout the Mississippi Flyway and the Central Flyway to the west in 2002. Further evidence of the role of migratory birds in disseminating WNV is the recent discovery of WNV transmission on Caribbean Islands and in Mexico (Dupuis et al. 2003, Loronopino et al. 2003).

The extensive wild bird mortality associated with WNV and its occurrence within backyards in suburban/urban neighborhoods is unique among most avian diseases (except for house finch conjunctivitis) and is alarming to the public. There is also a raising concern among avian biologists as a possible serious threat to populations of some migratory and non-migratory avian species.

CONCLUSION

The geographical extent of the emergent diseases of wild birds and their rapid expansion, variety of species affected, novel hosts involved, transitory nature of many bird species, difficulties in diagnosing diseases, and the lack of adequate resources for wildlife are some of the factors limiting the management and control of these diseases. The need for surveillance, monitoring, diagnosis, and timely reporting of wildlife diseases, particularly those with

domestic animal and human health risks, has frequently been expressed (Friend et al. 2001; McLean 2002b). The recent establishment of a surveillance, monitoring and research program within Wildlife Services of APHIS, USDA, will address this need and should provide better information in the future to appropriately manage some of the major wildlife diseases.

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