February 2004

West Nile Virus: Impact on Crow Populations in the United States

Robert G. McLean
USDA-APHIS-Wildlife Services

West Nile Virus: Impact on Crow Populations in the United States

Robert G. McLean
USDA APHIS Wildlife Services, National Wildlife Research Center, Fort Collins, Colorado

ABSTRACT: Since the introduction of the mosquito-borne West Nile Virus (WNV) into New York City (NYC) in 1999, it has expanded westward across the North American continent in an unprecedented fashion, taking in its wake hundreds of thousands and possibly millions of native and exotic birds. Corvid species, particularly the American crow, are particularly susceptible to this virulent strain of virus and have died dramatically during the summer virus transmission season. Experimental studies have shown that the fatality rate from WNV infection in American crows is nearly 100%. This mortality in crows and other corvids was used as a sensitive sentinel system to detect the presence and movement of the virus through a public reporting and laboratory testing national surveillance program. Crows were also the earliest indicator of virus activity in the majority of locations and were a useful predictor of human cases. Bird mortality from WNV peaks during August-September at the height of the mosquito-transmission period but extends from April to November each year in some states. An impact of WNV on local populations of crows was observed in some localities such as the NYC area, but no significant declines have been detected yet by the regional population trend data. The geographical distribution of WNV activity is not continuous across local landscapes and unexposed crows can then serve as a source to repopulate affected areas when overall populations are high. If WNV transmission continues for years with regular mortality, the resiliency of the regional crow populations to sustain this high mortality rate will diminish.

KEY WORDS: American crow, bird mortality, *Corvus brachyrhynchos*, crow, surveillance, United States, West Nile virus

Proc. 21st Vertebr. Pest Conf. (R. M. Timm and W. P. Gorenzel, Eds.)
Published at Univ. of Calif., Davis. 2004. Pp. 180-184.

INTRODUCTION

The introduction of the exotic West Nile virus (WNV) into the United States (USA) in 1999 from the Middle East (Lanciotti et al. 1999) initiated an epizootic in local birds followed by a human epidemic in the New York City (NYC) area (CDC 1999). Bird deaths, predominantly in American crows (*Corvus brachyrhynchos*), expanded from the epidemic center in Queens in NYC and from the central cluster of WNV-positive birds in the NYC area to a >100-mile-wide (>161-km-wide) area in 22 counties in New York, New Jersey, and Connecticut surrounding NYC (Eidson et al. 2001a). Sightings of dead crows in the region occurred from August to October, peaking in September, and matched the outward expansion of the laboratory-confirmed WNV-positive crows, suggesting that crows were likely responsible for the expansion of WNV out of NYC and that thousands of crows may have died from WNV infection (Eidson et al. 2001b). West Nile virus is a mosquito-transmitted virus infection of birds that has been responsible for human epidemics and equine epizootics within its historical range of Africa, the Middle East, Europe, and western Asia (Petersen and Roehrig 2001). The virus strains circulating in nature there did not cause notable mortality in native birds, including in hooded crows (*Corvus corone*), until 1997-1998 when mortality in domestic geese occurred in Israel (McLean et al. 2002, Swayne et al. 2001). A field study in Egypt (Work et. al. 1955) found an average of 57% of 120 birds of 6 free-ranging species to be WNV-antibody positive (thus survived the infection), including 88% of hooded crows. However, the strain of WNV introduced into the USA, likely from Israel (Lanciotti et al. 1999), was particularly virulent, especially to North American species of Corvidae, and has caused significant avian mortality (McLean 2002).

The American crow emerged as a valuable indicator of WNV presence in the northeastern USA because of its high susceptibility to infection with WNV. Dead crows became an ideal sentinel for public health surveillance because the crow is a conspicuous species even when sick or dead, widely distributed throughout the USA, a relatively local species, and is found in multiple habitats – thus more easily seen and reported by the public (McLean 2002). Enhanced surveillance for the detection of WNV dissemination out of the original focus in the NYC area was established subsequently, utilizing mortality in crows as a sentinel system for WNV activity. In addition, public health departments began using the occurrence and intensity of WNV-positive crows to make public health decisions about human risk.

SURVEILLANCE FOR WEST NILE VIRUS

During the initial bird surveillance in 1999 in NYC (Eidson et al. 2001a, b), 17,339 dead birds were reported, of which 5,697 (33%) were crows. Of the 671 dead birds tested in 1999, 294 (44%) were laboratory-confirmed WNV positive and 269 (89%) of these positive birds were crows (Figure 1). After the initial expansion of WNV activity in the NYC area in 1999, the virus survived through the temperate winter and reappeared within the epicenter focal area in May 2000 (CDC 2001). A multistate surveillance network was established to track the movement of the virus (CDC 2000). Surveillance consisted of enhanced passive reporting of human and equine clinical cases, mosquito collection and testing, regular antibody testing of sentinel birds, and dead bird reporting and testing. The surveillance data from each state were submitted to a national surveillance data base, ArboNet, and were verified and updated weekly (Marfin et al. 2001). The type and extent of the dead bird surveillance varied, with some states like New York testing any bird species submitted (Bernard et al. 2001) while other states like Connecticut tested only crows (Hadler et al. 2001).

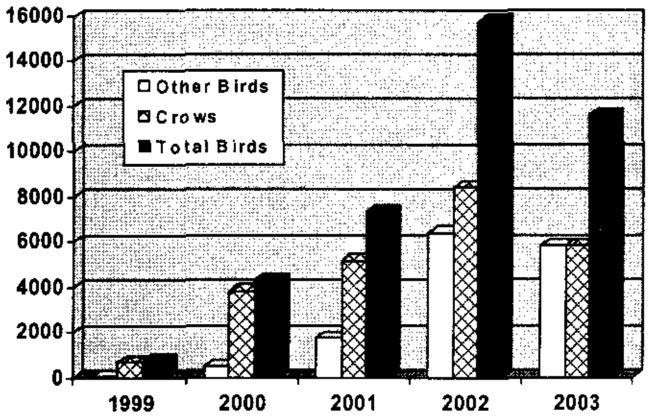

Figure 1. The number of birds reported positive for West Nile virus in the United States, 1999-2003 (Eidson et al. 2001, Marfin et al. 2001, Campbell 2003, Hayes 2004).

In 2000, WNV activity expanded to12 states and the District of Columbia; 104,816 dead birds were reported and 12,961 (12.4%) were submitted for WNV testing, with 4,305 (33.3%) found infected (Marfin et al. 2001). Crows were 58% of the birds tested and 89% of the WNV-positive birds (Figure 1); 50.4% of the 7,580 crows tested were infected. In New York, 68% of the positive birds were crows, and 32% of the positives were among 59 other bird species (Bernard et al. 2001). Of the 1,732 crows tested in New York, 47% were WNV infected compared to 70% of 1,574 crows tested in Connecticut (Hadler et al. 2001, Beckwith et al. 2002). The intensity of infection among crows in Connecticut increased during the transmission season and peaked at 98% during a week in September (Figure 2).

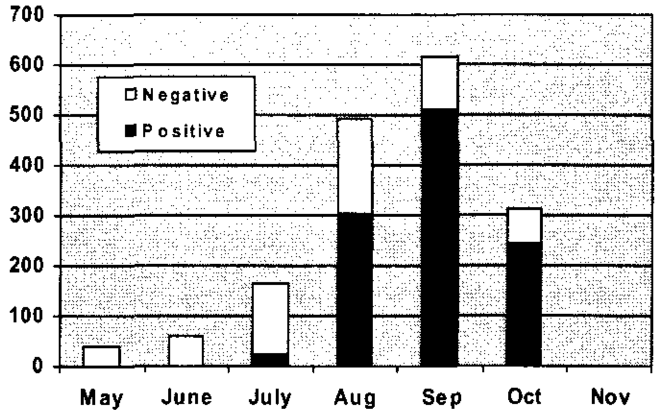

Figure 2. The number of dead crows submitted for laboratory testing and the number reported positive for West Nile virus in Connecticut during 2000 (Beckwith et al. 2002).

The geographical expansion of WNV in North America continued during the next 3 years, reaching all but one of the 48 continental states (Figure 3) as well as 7 provinces in Canada, Mexico, and countries in the Caribbean and Central America (Hayes 2004, Galvan 2004). During the 5 years that WNV spread rapidly throughout the USA, 39,280 birds of 232 native and exotic bird species, both free-ranging and captive, have been found infected with WNV (Hayes 2004). Crows (23,466, 60% of total) were the dominant species found positive for the first 3 years, while blue jays and other Corvid species became prominent as the virus moved westward from the original introduction site (Figure 1). Virus-positive crows were the first indication of WNV in an area and were the earliest seasonal surveillance event, 4-8 weeks before any other surveillance indicator. In those USA counties detecting the presence of the virus in 2002, WNV-positive dead birds were the first to be reported in 62% of positive counties, before sentinel chickens, mosquitoes, or other methods detected the disease (Campbell 2003), and finding a WNV-positive bird before August 1 was a good predictor of subsequent human cases (Guptill et al. 2003).

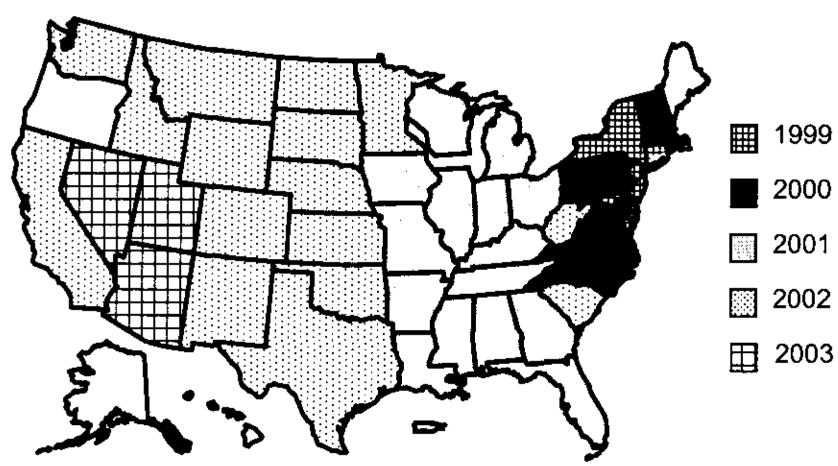

Figure 3. States reported positive for West Nile virus by year of reporting, 1999-2003.

EXPERIMENTAL INFECTION STUDIES

Experimental studies were conducted in biological containment animal facilities (BSL-3) on various species of birds, including Corvid species, to determine their susceptibility to and reservoir competence for WNV. The fatality rate (number dying of those infected) was 100% for American crows in two separate experiments (McLean et al. 2001, Komar et al. 2003) and 100% in black-billed magpies (*Pica hudsonia*), 83% in blue jays (*Cyanocitta cristata*), and 64% in fish crows (*Corvus ossifragus*) (Table 1). American crows died between days 4-8 days post infection and exhibited progressive clinical signs of lethargy, ataxia, unusual posture, inability to perch or stand, recumbency, and death. A further indication of the high mortality rate in crows is the low WNV antibody prevalence detected in free-ranging populations exposed to the virus. Sampling of 175 free-ranging crows in the NYC area in 1999 found only 1.1% to be antibody positive (McLean et al., unpubl. data), and 3.2% of 156 crows in central Illinois were positive (Yaremych et al. 2004), for a total of 2.1% of 331 crows overall.

Table 1. Mortality of species of Corvidae following experimental infection with West Nile virus (from Komar et al. 2003).

Species	Number Infected	Percentage Mortality
American crow	20	100%
Fish crow	11	64%
Black-billed magpie	5	100%
Blue jay	6	83%

Direct contact transmission between infected and uninfected crows and other species occurred during these experiments, and the clinical signs and fatality rate was similar in the inoculated or exposed birds and the birds infected by contact transmission. Oral transmission of WNV was demonstrated in 5 bird species, and American crows became infected after ingesting the carcass of a WNV-infected house sparrow (Komar et al. 2003) and WNV-infected white mice (McLean et al., unpubl. data). It is not known if direct contact or oral transmission occurs in nature, nor if these are important methods of transmission beyond the normal mosquito transmission route.

When animals die from infection, it was thought that they were dead-end hosts for the virus and would not contribute to virus transmission. However, particularly crows and other corvid species, circulated virus in their blood (viremia) in sufficient titers for 3-5 days prior to their death to contribute to transmission (reservoir competent) by infecting mosquitoes that feed upon them (Table 2, Komar et al. 2003). Sick and viremic crows would also be a more receptive host for mosquito feeding and thus contribute even more to transmission than healthy birds. In addition, crows and other species shed WNV at high titers through oral and cloacal exudates for days. For some corvid species, the virus could be detected on oral and cloacal swabs for days after death (Komar et al. 2002). A rapid antigen capture wicking assay (VecTest, Medical Analysis Systems, Camarillo, CA) was found useful in testing dead corvids, particularly American crows, for WNV infection, and this simple test could be used for rapid field evaluation in surveillance programs (Lindsay et al. 2003).

Table 2. Experimental infection of species of Corvidae with West Nile virus (from Komar et al. 2003).

Species	Number Tested	Days Infectious[1]	Mean Peak Viremia[2]
Blue jay	4	4	11.0
American crow	8	3.2	10.1
Black-billed magpie	3	3.0	8.7
Fish crow	8	2.8	6.8

[1] Number of days that the infected bird has viremia of 5.0 log^{10}
[2] Mean peak titer of virus (log^{10}) per ml of serum

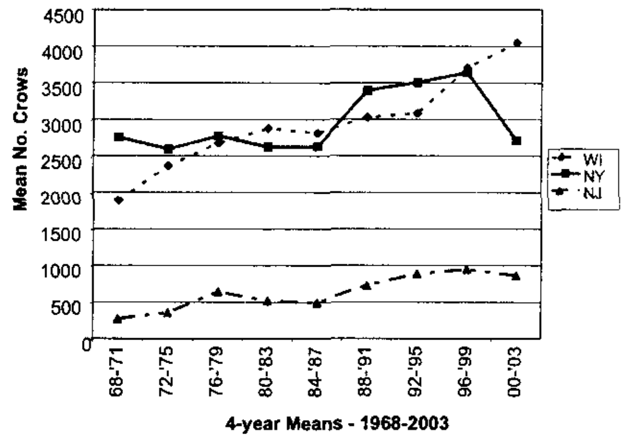

Figure 4. Four-year mean transect counts of American crows from the Breeding Bird Survey for Wisconsin, New Jersey, and New York, from 1968-2003 (Sauer et al 2003).

IMPACT OF WNV ON BIRD POPULATIONS

American crows and other highly susceptible bird species that have a high fatality rate from WNV infection could be suffering enough mortality to impact populations. Data from the breeding bird survey show that American crow densities in Midwestern and northeastern states increased during the last several decades and were in record numbers at certain sites in the NYC area prior to the invasion of WNV (Figure 4, Sauer et al. 2003). A recent decline of crows in New York is evident from survey trend data. The regional abundance of such a susceptible host species likely improved the chances for the introduced virus to survive and rapidly amplify. Analysis of Christmas bird count data from the NYC area showed a local decline in the number of crows in the affected zone after the epizootic in 1999 compared to 1998 data (Eidson et al. 2001a). Crow populations in some localities there continued to decline by as much as 90%, but adjacent areas to the east on Long Island showed no detectable declines (Chu et al. 2003). Studies of local American crow populations infected with WNV in 2002 showed an overall estimated mortality rate from the virus at 43% of 216 birds observed in 3 different states. The highest mortality rate occurred in central Illinois, where 68% of 28 radio-tagged crows died from confirmed WNV infection during the summer transmission season (Yaremych et al. 2004). Confirmed crow mortality from WNV infection in a local New York crow population was 37% of 68 birds observed during the year (McGowan et al. 2003) and estimated mortality in an Oklahoma population was about 40% of 120 crows (Caffrey et al. 2003).

Regional or national impacts on bird populations are more difficult to determine, and the only large-scale bird population data available are trend data from citizen monitoring surveys. National/regional annual surveys include Christmas Bird Counts (CBC), Breeding Bird Survey, Project Feeder Watch (PFW), Great Backyard Bird Count, Neighborhood Nest Watch, and Bird Conservation Network. Two separate winter bird monitoring efforts are CBC and PFW (Wells et al. 1998, Link and Sauer 1999) and analyses of these data bases have been conducted to examine potential impacts on populations of birds.

Christmas Bird Counts occur on a traditional site or route during 1 day within a 2-week period around Christmas and involve about 50,000 participants on approximately 2,000 sites or counts each year for more than 100 continuous years. Statistical and graphical analyses of CBC data for 10 bird species from 6 northeastern states could not determine if WNV caused any significant declines in bird populations, even when comparing counts in counties with and without WNV detected (Caffrey and Peterson 2003). The analyses did find a weak but not persuasive association only for American crows and Great-horned owls (*Bubo virginianus*); however, two insensitive sets of data were used, i.e., CBC and presence or absence of WNV in a county, to do the analyses. The intensity and variability of WNV transmission to birds through the summer season was not reflected in these analyses, and WNV-positive dead birds are reported nationally only by county (USGS 2001). This reporting does not reflect the true focal and patchy distribution of WNV activity, as was evident in Connecticut in 2000 (Beckwith et al. 2002).

Project Feeder Watch is a winter long (Nov-April) counting of birds in residential yards (2 consecutive days once every 2 weeks), and there are about 17,000 observers throughout the USA and Canada. The main limitation of the PFW monitoring project is that it has only been operating continent-wide since 1988, but the counts are much more densely distributed for a longer period of time during the winter than CBC. Analyses of PFW data from 800-1,400 sites for 6 species were conducted to determine if there were unusual increases or declines in bird abundance between the winters of 2001-2002 and 2002-2003 (Bonter and Hochachka 2003). Observed PFW declines were compared to CBC data from 28 geographically similar sites in upper Midwestern states to confirm these declines. The only notable declines observed were local declines with a patchy regional distribution for chickadees (*Parus atricapillus* and *P. carolinensis*), tufted titmouse (*P. bicolor*), and American crows; no large scale regional declines could be documented, whereas blue jay (*Cyanocitta cristata*), northern cardinal (*Cardinalis cardinalis*), and house sparrow (*Passer domesticus*) numbers remained stable. These affected species experienced population fluctuations in the past, but the recent local declines were synchronized across a broad area. West Nile virus was suspected as the cause of the declines.

CONCLUSION

West Nile virus is an emerging and significant cause of mortality in birds in North America. The disease continues to expand its distribution and affect a wide variety of bird species, particularly corvid species such as the American crow. The significance of the mortality is unknown because birds die singly and not in groups, so dead birds, especially the smaller species, are less likely to be observed. Many more birds die than are observed or tested; therefore, numbers are significantly higher than are reported. American crows and other Corvidae seem to be impacted the most but other species are less likely to be found. The national trend data on bird population monitoring are either too insensitive to detect regional effects on crow populations, or the effects are compensated for by immigration of unaffected birds from surrounding localities because of the patchy distribution of WNV. The extent of mortality in regional and national crow populations and the overall significance and impact to this species are unknown, but recent evidence suggests there are some significant local impacts and possible long-term effects.

LITERATURE CITED

BECKWITH, W. H., S. SIRPENSKI, R. A. FRENCH, R. NELSON, AND D. MAYO. 2002. Isolation of eastern equine encephalitis virus and West Nile virus from crows during increased arbovirus surveillance in Connecticut, 2000. Amer. J. Trop. Med. Hyg. 66:422-426.

BERNARD, K. A., J. G. MAFFEI, S. A. JONES, E. B. KAUFFMAN, G. D. EBEL, A. P. DUPUIS II, K. A. NGO, D. C. NICHOLAS, D. M. YOUNG, P-Y SHI, V. L. KULASEKERA, M. EIDSON, D. J. WHITE, W. B. STONE, NY STATE WEST NILE VIRUS SURVEILLANCE TEAM, AND L. D. KRAMER. 2001. West Nile virus infection in birds and mosquitoes, New York State, 2000. Emerg. Inf. Dis. 7:679-685.

BONTER, D. N., AND W. M. HOCHACHKA. 2003. Declines of chickadees and corvids: possible impacts of West Nile virus. Pp. 22-25 *in*: Amer. Birds, The 103rd Christmas Bird Count.

CAFFREY, C., AND C. C. PETERSON. 2003. West Nile virus may not be a conservation issue in northeastern United States. Pp. 14-21 *in*: Amer. Birds, The 103rd Christmas Bird Count.

CAFFREY, C., T. J. WESTON, AND S. C. R. SMITH. 2003. High mortality among marked crows subsequent to the arrival of West Nile virus. Wildl. Soc. Bull. 31:870-872.

CAMPBELL, R. 2003. Summary of West Nile virus activity, United States 2002. Fourth National Conference on West Nile virus in the United States, Centers for Disease Control and Prevention. Web Page: http://www.cdc.gov/ncidod/dvbid/westnile/conf/index.htm

CDC (CENTERS FOR DISEASE CONTROL AND PREVENTION). 1999. Outbreak of West Nile-like viral encephalitis – New York, 1999. MMWR Morb. Mortal. Wkly. Rep. 48:845-849.

CDC (CENTERS FOR DISEASE CONTROL AND PREVENTION). 2000. Guidelines for Surveillance, Prevention, and Control of West Nile virus infection – United States. MMWR Morb. Mortal. Wkly. Rep. 49:25-28.

CDC (CENTERS FOR DISEASE CONTROL AND PREVENTION). 2001. West Nile virus activity – Eastern United States, 2001. MMWR Morb. Mortal. Wkly. Rep. 50:617-619

CHU, M., W. STONE, K. J. MCGOWAN, A. A. DHONDT, W. M. HOCHACHKA, AND J. E. THERRIEN. 2003. West Nile file. Pp. 10-11 *in*: Birdscope (Winter 2003).

EIDSON, M., N. KOMAR, F. SORHAGE, R. NELSON, T. TALBOT, F. MOSTASHARI, R. MCLEAN, AND WEST NILE VIRUS AVIAN MORTALITY SURVEILLANCE GROUP. 2001a. Crow deaths as a sentinel surveillance system for West Nile virus in the Northeastern United States, 1999. Emerg. Inf. Dis. 7:615-620.

EIDSON, M., L. KRAMER, W. STONE, Y. HAGIWARA, K. SCHMIT, AND WEST NILE VIRUS AVIAN MORTALITY SURVEILLANCE GROUP. 2001b. Dead bird surveillance as an early warning system for West Nile virus. Emerg. Inf. Dis. 7:631-635.

GALVAN, J. M. 2004. West Nile virus in Latin America. Fifth National Conference on West Nile virus in the United States, Centers for Disease Control and Prevention. Web Page: http://www.cdc.gov/ncidod/dvbid/westnile/conf/index .htm

GUPTILL, S. C., K. G. JULIAN, G. L. CAMPBELL, S. D. PRICE, AND A. A. MARFIN. 2003. Early-season avian deaths from West Nile virus as warnings of human infection. Emerg. Inf. Dis. 9:483-484.

HADLER, J., R. NELSON, T. MCCARTHY, T. ANDREADIS, M. J. LIS, R. FRENCH, W. BECKWITH, D. MAYO, G. ARCHAMBAULT, AND M. CARTTER. 2001. West Nile Virus surveillance in Connecticut in 2000: an intense epizootic without high risk for severe human disease. Emerg. Inf. Dis. 7:636-642.

HAYES, N. 2004. Summary of West Nile virus activity, United States 2003. Fifth National Conference on West Nile virus in the United States, Centers for Disease Control and Prevention. Web Page: http://www.cdc.gov/ncidod/dvbid/westnile/conf/index.htm

KOMAR, N., R. LANCIOTTI, R. BOWEN, S. LANGEVIN, AND M. BUNNING. 2002. Detection of West Nile virus in oral and cloacal swabs collected from bird carcasses. Emerg. Infect. Dis. 8:741-742.

KOMAR, N., S. LANGEVIN, S. HINTEN, N. NEMETH, E. EDWARDS, D. HETTLER, B. DAVIS, R. BOWEN, AND M. BUNTING. 2003. Experimental infection of North American birds with the New York 1999 strain of West Nile virus. Emerg. Infect. Dis. 9:311-323.

LANCIOTTI, R. S., J. T. ROEHRIG, V. DEUBEL, J. SMITH, M. PARKER, K. STEELE, B. CRISE, K. E. VOLPE, M. B. CRABTREE, J. H. SCHERRET, R. A. HALL, J. S. MACKENZIE, C. B. CROPP, B. PANIGRAHY, E. OSTLUND, B. SCHMITT, M. MALKINSON, C. BANET, J. WEISSMAN, N. KOMAR, H. M. SAVAGE, W. STONE, T. MCNAMARA, AND D. J. GUBLER. 1999. Origin of the West Nile virus responsible for an outbreak of encephalitis in the Northeastern United States. Science 286:2333-2337.

LINDSAY, R., I. BARKER, G. NAYAR, M. DREBOT, S. CALVIN, C. SCAMMELL, C. SACHVIE, T. SCAMMELL-LA FLEUR, A. DIBERNARDO, M. ANDONOVA, AND H. ARTSOB. 2003. Rapid antigen-capture assay to detect West Nile virus in dead Corvids. Emerg. Infect. Dis. 9:1406-1410.

LINK, W. A., AND J. R. SAUER. 1999. Controlling for varying effort in count surveys – an analysis of Christmas Bird Count data. J. Agric. Biol. Environ. Statistics 4(2):116-125.

MARFIN A. A., L. R. PETERSEN, M. EIDSON, J. MILLER, J. HADLER, C. FARELLO, B. WERNER, G. L. CAMPBELL, M. LAYTON, P. SMITH, E. BRESNITZ, M. CARTTER, J. SCALETTA, G. OBIRI, M. BUNNING, R. C. CRAVEN, J. T. ROEHRIG, K. G. JULIAN, S. R. HINTEN, D. J. GUBLER, AND THE ARBONET COOPERATIVE SURVEILLANCE GROUP. 2001. Widespread West Nile virus activity, eastern United States, 2000. Emerg. Infect. Dis. 7:730-735.

MCGOWAN, K. J., A. B. CLARK, AND D. A. ROBINSON. *Quoted in*: CAFFREY, C., T. J. WESTON, AND S. C. R. SMITH. 2003. (*see above*)

MCLEAN, R. G. 2002. West Nile virus. A threat to North American avian species. Trans. No. Amer. Wildl. Nat. Resource Conf. 67:62-74.

MCLEAN, R., S. R. UBICO, D. BOURNE, AND N. KOMAR. 2002. West Nile virus in livestock and wildlife. Current Topics in Microbiology and Immunology 267:271-308.

MCLEAN, R. G., S. R. UBICO, D. E. DOCHERTY, W. R. HANSEN, L. SILEO, AND T. S. MCNAMARA. 2001. West Nile virus transmission and ecology in birds. West Nile Virus: Detection, Surveillance, and Control, Annals NY Acad. Sci. 951:54-57.

PETERSEN, L. R., AND J. T. ROEHRIG. 2001. West Nile virus: a reemerging global pathogen. Emerg. Infect. Dis. 7:611-614.

SAUER, J. R., J. E. HINES, AND J. FALLON. 2003. The North American breeding bird survey, results and analysis 1966 - 2002. Version 2003.1, USGS Patuxent Wildlife Research Center, Laurel, MD.

SWAYNE, D. E., J. R. BECK, C. S. SMITH, W.-J. SHIEH, AND S. R. ZAKI. 2001. Fatal encephalitis and myocarditis in young domestic geese (*Anser anser domesticus*) caused by West Nile virus. Emerg. Inf. Dis. 7:751-753.

USGS (UNITED STATES GEOLOGICAL SURVEY). 2001. West Nile virus maps, 2001. Center for Integration of Natural Disaster Information. Web Page: http://cindi.usgs.gov/hazard/event/west_nile/west_nile.html.

WELLS, J. V., K. V. ROSENBERG, E. H. DUNN, D. L. TESSAGLIA-HYMES, AND A. A. DHONDT. 1998. Feeder counts as indicators of spatial and temporal variation in winter abundance of resident birds. J. Field. Ornith. 69:577-586.

WORK, T. H., H. S. HERBERT, AND R. M. TAYLOR. 1955. Indigenous wild birds of the Nile Delta as potential West Nile virus circulating reservoirs. Amer. J. Trop. Med. Hyg. 4:872-888.

YAREMYCH, S. A, R. E. WARNER, P. C. MANKIN, J. D. BRAWN, A. RAIM, AND R. NOVAK. 2004. West Nile virus and high death rate in American crows. Emerg. Infect. Dis. 10:709-711.