Avian Influenza Surveys in Waterfowl Part I: The Role of Wild and Domestic Waterfowl in Avian Influenza Outbreaks in Domestic Poultry Written by Todd Waster DVM. National Surveillance Unit

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It is generally accepted that waterfowl play an important role in the generation, spread, and enzootic transmission of avian influenza (AI). Published surveys indicate that all 15 hemagglutinin (HA) and 9 neuraminidase (NA) subtypes have been isolated from wild waterfowl and aquatic shorebirds.^{1,2,3} Reported overall prevalence estimates vary considerably between studies and study years. For example, recently cited overall AI prevalence rates in waterfowl ranged from 5.9 percent in Ohio, 8.2 percent in Pennsylvania, and 13.9 percent on the eastern shore of Maryland.^{4,5,6}

The prevalence of AI subtypes in waterfowl varies by age, season, and species ^{7,8,9,10} Age appears to be the primary risk factor for AI infection. Prevalence rates in juveniles have been reported to be significantly higher than prevalence rates in adults. Typically, susceptible resident juvenile birds are infected from July through November as migratory waterfowl congregate in staging areas in preparation for migration. Prevalence of AI decreases throughout the fall and winter, with the lowest reported prevalence rates occurring in January through May. Mallards and blue-winged teal have the highest species prevalence rates reported in surveys of wild waterfowl.

Although studies have addressed the potential of waterfowl to harbor influenza subtypes that subsequently appear in poultry, few epidemiological studies have linked AI subtypes found in waterfowl to outbreaks in poultry. A study in Minnesota found that seasonal patterns of AI infection in migratory waterfowl and sentinel ducks occurred 6 to 8 weeks prior to seasonal outbreaks in range-farmed turkeys.¹⁰ Circulation and maintenance of AI among range-farmed turkeys within the same geographic area were ruled out as the cause of infection due to the seasonal nature of turkey production in Minnesota at that time. It was postulated that cooler environmental and surface water temperatures, surface and groundwater fecal contamination, increased waterfowl activity associated with congregation in preparation for migration, and domestic turkeys' adaptation to the virus subtype before detection could have been responsible for the delayed infection in turkeys.

In 1979, domestic mallards in Minnesota were infected with the same AI virus subtype (H10N7) responsible for an AI outbreak in commercial turkeys.¹¹ In 15 flocks on one operation, mortality rates ranged from 5.7 percent to 31.0 percent. The H10N7 subtype had not been isolated previously in the State. It was theorized that domestic mallards inhabiting a pond 500 meters from the premises may have served as a reservoir for the H10N7 subtype. However, turkeys selected to become breeders and raised on-range on the same premises as part of a separate operation were subclinically infected with the same H10N7 subtype. Twenty-six seagulls killed on-range yielded negative serological tests, and attempts to isolate virus were unsuccessful.

Wildlife surveys associated with an outbreak of AI subtype H5N2 during the 1983-84 epizootic in domestic poultry in Pennsylvania, New Jersey, Maryland, and Virginia failed to isolate the responsible virus from wildlife species within the quarantine area.¹² In

another study, isolation attempts were made on 4,466 birds and small rodents within the quarantine area and 1,511 nearby waterfowl.¹³ Wild animals were not shown to be responsible for introducing the virus to domestic poultry during the Pennsylvania outbreak. A separate wildlife survey was conducted from late June through November 1984 within the Pennsylvania and Virginia quarantine zones. Of the 13 AI subtype viruses isolated from waterfowl within the quarantine zones, only one H5N2 subtype was found in a hunter-killed duck Genetic analysis determined that the HA gene could be clearly distinguished from the H5N2 family of viruses that caused the Pennsylvania epidemic. Virus isolates from this study were passaged in chickens; the isolates were found to replicate but did not produce disease.

Seroprevalence studies in healthy captive waterfowl at the Baltimore zoo indicated exposure to AI.¹⁴ A survey of Louisiana waterfowl showed that AI virus is transmitted to resident mottled ducks and circulates within duck populations throughout the winter.⁹ In all published waterfowl AI surveys, subtype diversity has been extensive. Subtypes of H5 (0.4 percent), H7 (0.7 percent), and H9 (0.4 percent), which are associated with highly pathogenic influenza outbreaks in poultry or recent human infections, are reported to be isolated less commonly (over 3,100 isolates reported from surveys) than H3, H4, or H6 subtypes (63.8 percent of isolates).¹ One recent study was an exception, where H5, H7, and H9 isolates accounted for 21.5 percent of isolates.¹ Spatial and temporal differences in AI isolate subtypes exist.

In the next edition of NAHSS *Outlook*, the usefulness of waterfowl surveys as part of an ongoing AI surveillance program will be discussed.

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² Alfonso C. P., Cowen B. S., Van Campen H., 1995. Influenza A Viruses Isolated From Waterfowl in Two Wildlife Management Areas of Pennsylvania. *Journal of Wildlife Diseases* 31(2), pp. 179-185.

³ Stallknecht D. E. 1997. Ecology and Epidemiology of Avian Influenza Viruses in Wild Bird Populations: Waterfowl, Shorebirds, Pelicans, Cormorants, etc. In: Proc. 4th International Symposium on Avian Influenza, U.S. Animal Health Association, Athens, GA. Pp. 61-69.

⁴ Slemmons R. D., Shieldcastle M. C., Heyman L. D., Bednarik K. E., and Senne D. A. 1991. Type A Influenza Viruses in Waterfowl in Ohio and Implications for Domestic Turkeys. *Avian Diseases* 35:165-173.

⁵ Alfonso C. P., Cowen B. S., Van Campen H., 1995. Influenza A Viruses Isolated From Waterfowl in Two Wildlife Management Areas of Pennsylvania. *Journal of Wildlife Diseases* 31(2), pp. 179-185.

⁶ Slemons R. D., Hansen W. R., Converse K. A., Senne D. A. 2003. Type A Influenza Virus Surveillance in Fee-Flying, Nonmigratory Ducks Residing on the Eastern Shore of Maryland. *Avian Diseases* 47:1107-1110.

⁷ Stallknecht D. E., and Shane S.M. 1988. Host range of avian influenza virus in free-living birds. *Veterinary Research Communications*, 12, 125-141.

⁸ Hinshaw V. S., Wood J.M., Webster R.G., Deibel R., Turner B. 1985. Circulation of influenza viruses and pramyxoviruses in waterfowl originating from two different areas of North America. *Bulletin of the World Health Organization*, 63 (4): 711-719.

⁹ Stallknecht, D. E., Shane S. M., Zwank P. J., Senne D. A., and Kearney M. T. 1990. Avian Influenza Viruses from Migratory and Resident Ducks of Coastal Louisiana. *Avian Diseases* 34:398-405.

¹⁰ Halvorson D. A., Kelleher C. J., and Senne D. A. 1985. Epizootiology of Avian Influenza: Effect of Season on Incidence in Sentinel Ducks and Domestic Turkeys in Minnesota. *Applied and Environmental Microbiology*, Apr. 1985, P. 914-919.

¹¹ Karunakaran D., Hinshaw V., Poss P., Newman J., Halvorson D. 1983. Influenza A Outbreaks in Minnesota Turkeys Due to Subtype H10N7 and Possible Transmission by Waterfowl. *Avian Diseases* 27: 357-365.

¹² Nettles V. F., Wood J. M., Webster R. G. 1985. Wildlife Surveillance Associated with an Outbreak of Lethal H5N2 Avian Influenza in Domestic Poultry. *Avian Diseases* 29:733-741.

¹³ Hinshaw V. S., Nettles V. F., Shorr L. F., Wood J. M. and Webster R. G., 1986. Influenza Virus Surveillance in Waterfowl in Pennsylvania after the H5N2 Avian Outbreak. *Avian Diseases* 30:207-212.

¹⁴ Walker L. T., Cranfield M. R., Arnold I. D. 1985. Evidence of Exposure of Captive Waterfowl to Avian Influenza Virus and Duck Adenovirus. *Zoo Animal Medicine*, 16:109-112.