Influenza A virus infection of pigeons

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Lecture given at the 1st World Congress of the IVPA, 6-7 March 2020, Warsaw, Poland

Influenza A viruses of all subtypes, in their low pathogenic state, form part of the natural microbiota of water bird species. When low pathogenic (LPAI) H5Nx or H7Nx subtype viruses circulate for extended periods in susceptible poultry flocks, the highly pathogenic forms (HPAI) can arise through mutational events in the viral genome. In 1996 an H5N1 HPAI virus emerged from the live bird markets in China, and its descendants became known as the Goose/Guangdong H5N1 lineage. Four intercontinental transmission waves of Goose/Guangdong H5 lineage viruses swept the globe since 2005, each characterized by epidemics in poultry that resulted in high mortalities, mass culling, contact transmission to humans (with high fatality rates) and severe economic losses for the affected regions, with resulting trade restrictions. A significant shift in ecology of avian influenza had occurred: not only had HPAI strains adapted to enable sub-clinical infections of some highly mobile water bird species, but an HPAI reservoir had been established in the wild for the first time, from which new variants could seasonally arise and disperse as the birds migrated between their northern breeding and southern overwintering sites.

Host species differ in their innate susceptibility to avian influenza viruses, as determined by the types and distribution of cell surface receptors that the virus targets for entry, as well as the species' unique cellular immune defence capabilities. Thus, between bird orders there exists a spectrum of natural susceptibility towards infection and the ability to transmit influenza A viruses, which is also influenced by the age and immune status of the individual. On the far left of the spectrum are water birds, the natural host and reservoir. Ducks, geese and shorebirds become infected with influenza A virus but show few clinical signs, excrete virus in high quantities into the environment, but are not capable of facilitating the switch from LPAI to HPAI. In the middle, ratites such as ostriches are susceptible to infection with wild bird viruses, excrete reasonable amounts of the virus to facilitate transmission within the flock, and are capable of facilitating the mutation from LPAI to HPAI strains. At the far right of the spectrum are gallinaceous birds such as chickens, turkeys and quail. Upon infection these birds can facilitate the

mutation from LPAI to HPAI, shed great quantities of virus and are thus highly efficient transmitters, but they are highly susceptible to the disease, with lethal consequences. Where then within this spectrum are columbids positioned? Some have argued that the close association of feral pigeons with humans in urban habitats and on poultry farms, and the international movements of racing pigeons for competition events places columbids in a high risk category for the introduction and transmission of avian influenza viruses. The risks that columbids pose in the ecology and epidemiology of AI have been thoroughly investigated over the course of several decades.

The collective findings of all studies on free-living pigeons, market pigeons and those experimentally infected with LPAI and HPAI in studies from 1944 to 2013 established that, in the spectrum of susceptibility and ability to transmit the virus, pigeons are biologically at the farthest left. They usually do not show clinical signs when infected with HPAI viruses, are inefficient propagators and transmitters of the virus (especially to poultry), and do not facilitate the mutation from LPAI to HPAI. Deaths in free-living pigeons during HPAI outbreaks were probably due to their exposure to excessive virus levels in the heavily contaminated environments of infected poultry farms, once again highlighting the importance of on-farm biosecurity and preventing access of wild birds into houses, as any wild bird can act as mechanical vectors for spread. Biologically, the longstanding status of columbids as ineffective propagators and disseminators of HPAI and LPAI viruses prevails: the pigeon has no epidemiological significance in the maintenance and spread of avian influenza.

References:

Abolnik C. A current review of avian influenza in pigeons and doves (Columbidae). Vet Microbiol. 2014; 170(3-4): 181-196.

Abolnik C, Stutchbury S, Hartman MJ. Experimental infection of racing pigeons (*Columba livia domestica*) with highly pathogenic Clade 2.3.4.4 sub-group B H5N8 avian influenza virus. Vet Microbiol. 2018; 227: 127-132.

Bosco-Lauth AM, Marlenee NL, Hartwig AE, Bowen RA, Root JJ. Shedding of clade 2.3.4.4 H5N8 and H5N2 highly pathogenic avian influenza viruses in peridomestic wild birds in the U.S. Transbound Emerg Dis. 2019 ;66(3): 1301-1305.

Hemida MG, Chu D, Abdelaziz A, Alnaeem A, Chan SMS, Peiris M. Molecular characterisation of an avian influenza (H5N8) outbreak in backyard flocks in Al Ahsa, Eastern Saudi Arabia, 2017-2018. Vet Rec Open. 2019; 6(1):e000362. doi: 10.1136/vetreco-2019-000362.

Kwon JH, Noh YK, Lee DH, Yuk SS, Erdene-Ochir TO, Noh JY, Hong WT, Jeong JH, Jeong S, Gwon GB, Song CS, Nahm SS. Experimental infection with highly pathogenic H5N8 avian influenza viruses in the Mandarin duck (*Aix galericulata*) and domestic pigeon (*Columba livia domestica*). Vet Microbiol. 2017; 203: 95-102.

Lee DH, Criado MF, Swayne DE. Pathobiological Origins and Evolutionary History of Highly Pathogenic Avian Influenza Viruses. Cold Spring Harb Perspect Med. 2020 Jan 21. pii: a038679. doi: 10.1101/cshperspect.a038679.

Xiang B, Liang J, You R, Han L, Mei K, Chen L, Chen R, Zhang Y, Dai X, Gao P, Liao M, Xiao C, Ren T. Pathogenicity and transmissibility of a highly pathogenic avian influenza virus H5N6 isolated from a domestic goose in Southern China. Vet Microbiol. 2017; 212: 16-21.