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Pigeons and influenza viruses

Pigeons have been experimentally inoculated with avian influenza virus (AIV) in a few studies. These studies demonstrate that pigeons are quite resistant to infection. Pigeons were inoculated with AIV isolates obtained from chickens and one from emus. In 8-32 mo. old pigeons inoculated IV or intranasally with AIV, no antibody titers were detected after 21 days nor was any virus isolated from choanal or cloacal swabs or from internal organs. Inoculated pigeons did not spread virus to susceptible chickens in direct contact. Both low and high pathogenicity viruses were used in this study (Panigrahy et al., 1996). Perkins et al., (2002) inoculated 4 week old pigeons (concurrently infected with pigeon circovirus) with the H5N1 Hong Kong isolate intranasally. No virus was isolated from inoculated pigeons nor were any lesions detected. Serology was not performed in this study. Slemons and Easterday (1972) demonstrated that the highly pathogenic turkey isolate A/Tk/Ontario H5N9 AIV could be isolated from 2/19 experimentally inoculated pigeons however, it could not be determined if the virus isolated was directly from the inoculum or if the pigeons became infected. Pigeons inoculated oculonasally with 10e6 of A/Anhui/1/13 (H7N9) or A/turkey/Germany/AR534/13 (H7N7) were infected with both viruses and shed low levels between days 2 and 7 post inoculation. Most shedding was from the respiratory tree although A/turkey/Germany/AR534/13 (H7N7) was also detected in feces. No viral antigen was detected in internal organs (Kalthoff et al., 2014). The only study conducted using a virus recovered from a naturally infected pigeon (H5N1, clade 2.2.1/C, Egypt) caused neurological signs and

greenish diarrhea in experimentally infected birds. Interestingly, infected birds did not appear to shed much virus with titers ranging from $10^{1.6}$ - $10^{3.16}$ from experimental studies (Shimaa et al., 2014).

Surveys of wildlife including 480 pigeons and doves after the 1983 PA AI outbreak resulted in no isolations of AIV. The Ck/PA H5N2 virus neither replicated nor elicited antibodies in experimentally inoculated pigeons (Nettles, et al., 1985). An additional 309 pigeons surveyed during the outbreak had no antibodies to H5N2 virus (Tudor, 1991). Recent surveys of birds in live bird markets in NY have never revealed any pigeons with antibodies to AIV (Trock, personal communication).

A few isolations have been made from dropping boards under pigeon cages in the Hong Kong markets. In genetic comparisons, these viruses are very similar to those isolated from silkie chickens, quail and other chickens in the live bird markets from Hong Kong (Guan et al., 2000, Cauthen et al., 2000, Guan et al., 2002, Peiris et al., 2001). No experimental inoculations of pigeons have been done with these isolates.

A sublineage of H5 viruses (clade 2.2.1/C) did cause an outbreak of HPAI in pigeons in Egypt. This virus was similar to viruses concurrently circulating in backyard poultry flocks and ducks and causing human infections. Two squabs (40 days old) from a flock of 50 of various ages in a backyard free-ranging flock were submitted and diagnosed with HPAI. The flock had experienced sudden death (50% mortality), neurologic signs and greenish diarrhea (Shimaa, et al., 2014).

In conclusion, pigeons are probably quite resistant to infection with AIV normally. However, it may be possible for AIV to become adapted to pigeons. The naturally occurring infections that have been documented have been limited in numbers not caused outbreaks. Pigeons have been infected with several H5 sublineage viruses indicating that they are a potential host. When infected, clinical signs have primarily been neurologic and greenish diarrhea.

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