Protection of Chickens Against Challenge with Virulent Influenza A Viruses of Hav5 Subtype Conferred by Prior Infection with Influenza A Viruses of Hsw1 Subtype

Brief Report

By

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Summary

Prior infection of six-week-old chickens with influenza A viruses of Hsw1 haemagglutinin subtype and irrelevant neuraminidase subtypes reduced the deaths and sickness in groups of those birds challenged with A/tcrn/S.Africa/61 (Hav5 Nav2/3) and A/chicken/Scotland/59 (Hav5 N1).

On the recommendations of the WHO Expert Committee (13) influenza viruses are classified into types A, B or C on the basis of the ribonucleoprotein antigen and influenza A viruses into subtypes on the basis of the haemagglutinin (H) or neuraminidase (N) antigens. However, relationships have been revealed amongst H subtypes originally thought to be distinct by serological tests (6, 8, 10) and, in the case of the relationship between Heq1 and Hav1 subtypes, by protection studies in chickens (9). More recently immuno-double-diffusion tests with antisera against isolated antigens have suggested several interrelationships between subtypes and a reorganization of the classification system for influenza A viruses has been recommended (11, 14, 15). Although SCHILD et al. (11) do not report any relationship between Hav5 (proposed H5) and Hsw1 (proposed H1), low-level cross relationships between these subtypes have been reported (2, 12). In the present study we have examined the ability of viruses of Hsw1 subtype to protect chickens against virulent Hav5 viruses with irrelevant N subtypes.

The viruses and their sources have been described (1, 2) with the exception of A/duck/Alberta/35/76 (Hsw1N1) (7) and A/duck/Hong Kong/196/77 (Hsw1N2) which were received from Dr. K. F. Shorridge, Hong Kong University, Hong Kong. In protection studies six-week-old chickens were infected by intramuscular inoculations of about 10^8 EID_{50} of primary virus and reinfected by the same route with a similar dose three weeks later. Two weeks after the second dose birds were bled and challenged with 0.1 ml of diluted infectious allantoic fluid contain-
ing about $10^6$ EID$_{50}$ of challenge virus by intramuscular injection. Birds were examined twice daily for signs of disease. Those alive but too sick to eat or drink were killed and recorded as dead at the next observation. Experiments were restricted to those in which the primary infecting virus and the challenge virus had dissimilar N subtypes as antibodies to this antigen also afford protection (4, 9). Uninfected fully susceptible controls were also challenged and A/equine/Prague/1/56 (Heq 1 Neq 1) and A/turkey/England/63 (Hav 1 Nav 2/3) were used for primary infection and challenge as controls for susceptibility and protection. A/turkey/England/N28/73 (Hav 5 N2), which is of low virulence for chickens, was used to demonstrate protection by an Hav 5 virus.

The serological responses seen after primary infection and challenge and the signs of disease and deaths are shown in Table 1. All susceptible birds challenged with A/tern/S.Africa/61 became sick and died with a mean death time (MDT) of 5.1 days. Primary infection with dk/H.K./196, ty/Eng/250 and dk/Alb/35 conferred considerable resistance to challenge with tn/S.A./61, only 2/10 birds dying from each group infected with dk/H.K./196 and ty/Eng/250 and 1/10 with dk/Alb/35. While 5/10, 3/10 and 3/10 respectively showed signs of disease. Protection by A/swine/Cambridge/39 (Hsw 1 N1) was not so marked, 9/10 birds showing signs of disease but only 6/10 dying. Prior infection with the virus of Hav 5 sub-type, ty/Eng/N28, induced complete protection to challenge with tn/S.A./61. One bird primary infected with eq/Prague survived challenge with tn/S.A./61, although the low post challenge haemagglutination inhibition (HI) titre to tn/S.A./61 in this bird may indicate that infection was never established. The other nine eq/Prague infected birds all became sick and died in a noticeably shorter time than susceptible controls. With the exception of ty/Eng/N28 infected birds, none had shown prechallenge HI titres to tn/S.A./61. All birds surviving challenge were positive by HI tests to tn/S.A./61 and all individual birds in the ty/Eng/250, dk/H.K./196 and dk/Alb/76 groups showed increased HI titres to the primary infecting virus after challenge. Although earlier work indicated that ek/Scot/59 was as virulent as tn/S.A./61 (5) in the present study only 8/10 susceptible controls were sick and 7/10 died after challenge with ek/Scot/59. All three surviving birds showed high HI titres to ek/Scot/59 indicating that they had been infected. Prior infection with dk/H.K./196 produced considerable protection to challenge with ek/Scot/59. One bird was found dead on day 4 after challenge but this was the only bird to show any signs of disease. Birds infected with ty/Eng/N28 were fully protected against challenge with ek/Scot/59. As a further control, selected viruses were used as primary infecting viruses prior to challenge with turkey/Eng/63 (Hav 1 Nav 2/3). Birds were not protected against this virus by sw/Camb/39 and, as seen with eq/Prague/56 and tn/S.A./61, deaths and onset of sickness occurred noticeably sooner than with challenged susceptible birds. One bird primary infected with dk/H.K./196 survived challenge with ty/Eng/63, the other nine becoming sick and dying at about the same time as in susceptible controls. The surviving bird showed a high HI titre to ty/Eng/63. Eq/Prague/56 conferred a high level of protection to challenge with ty/Eng/63, only one bird showing signs of disease and dying. Calculation of pathogenicity indices (Table 1) for the challenged birds gave a good indication of the virulence of the challenge viruses and the degree of protection conferred by the primary viruses.