AVIAN INFLUENZA VIRUSES IN WHICH INFECTIVITY IS NEUTRALIZED BY ANTISERA LACKING HEMAGGLUTINATION-INHIBITION ACTIVITY

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Hemagglutination of influenza viruses A/duck/Hokkaido/5/77 (H3N2) (Dk/77) and A/duck/Alberta/157/77 (H4N6) (Dk/A1b) was not inhibited by the antisera of mice to each homologous virus, whereas the sera effectively neutralized infectivity of the viruses. This phenomenon was not observed when the antisera to other influenza viruses of 3 avian, 2 human and 1 equine origin were examined. This peculiar finding does not concur with the generally accepted concept that hemagglutination-inhibition (HI) and viral neutralization activities of antisera to influenza viruses are closely correlated.

Surprisingly, anti-Dk/77 mouse sera inhibited hemagglutination of heterologous H3 viruses, A/duck/Hokkaido/8/80 (H3N8) (Dk/80) and A/Aichi/2/68 (H3N2) (Aichi), in addition to hemagglutinin rosettes prepared from Dk/77 virus. Anti-Dk/Alb mouse sera also inhibited hemagglutination of the hemagglutinin rosettes from the homologous virus.

Species-dependent differences among inhibitory activities of anti-Dk/77 sera were observed. Lack of HI activities of anti-Dk/77 sera against the intact Dk/77 virus was noticed in mice and ducks. On the other hand, the sera of mink and chickens infected with DK/77 showed activities of HI as well as neutralization of infectivity of the virus. The sera of ducks infected with DK/77 inhibited hemagglutination of the Dk/77 hemagglutinin rosettes and of the heterologous intact Dk/80 and Aichi viruses as observed in the anti-Dk/77 mouse sera. Anti-Dk/77 duck sera neutralized infectivity of the virus at low titers.

When anti-Dk/77 mouse and duck sera were absorbed with intact Dk/77 virus, the inhibitory activities of the sera against hemagglutination of the Dk/77 hemagglutinin rosettes and heterologous viruses was lost, indicating that the antibodies in the anti-Dk/77 mouse and duck sera bound to intact DK/77 virus. These antibodies may inhibit infectivity by interfering with some later step of viral replication than attachment of the virus to the receptor of host cells.