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Increased pathogenicity in mice of a mouse-adapted influenza H7N9 virus was associated with delayed host innate immune responses

Content

An avian influenza virus A/Anhui/1/2013 (Anhui; A/H7N9) was adapted in mice (Anhui-M), which showed higher pathogenicity than did the original strain (Anhui-E). Fifty % of lethal doses of each virus were 2.5 x 104 pfu/50 L (Anhui-E) and 50 pfu/50 L (Anhui-M), respectively. When mice were inoculated nasally with 4 l (40xLD50) of each virus, which volume allows the inoculated virus spread restricted to the upper respiratory tract (URT), Anhui-E induced little weight loss in the mice while the animals infected with Anhui-M exhibited marked weight loss. Next, the expression of type I IFN-associated genes and IFN- production after 10xLD50/50L of each virus infection in the mice lungs were compared between the two viruses. On day 1 after infection, Anhui-M induced these responses at a lower level than did Anhui-E, but no significant differences of them were shown in 3 days after infection. Furthermore, when mice received poly(I:C) pre-treatment to prepare for type I IFN induction in the respiratory tract, the mice recovered earlier from the fatal lung infections than in non-treated mice. These results suggested that the increased lung pathogenicity of Anhui-M was associated with the delayed host innate immune responses to a small dose of the virus. Six virus clones from Anhui-M had two amino acid substitutions in the PA protein (T97I and L268F) or in the HA protein (A143T and A196E). Further study for the relationship between the pathogenicity and the virus mutations of Anhui-M is in progress in regard to the suppression of host innate immunity.

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Pathogenesis

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Innate Immunity

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