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Risk assessment of fifth-wave H7N9 influenza A viruses in mammalian models

Content

The fifth-wave of the H7N9 influenza epidemic in China was distinguished by a sudden increase in human infections, an extended geographic distribution, and the emergence of highly pathogenic avian influenza (HPAI) viruses. Genetically, some H7N9 viruses from the fifth-wave have acquired novel amino acid changes at positions involved in mammalian adaptation, antigenicity, and HA cleavability. In our study, several low pathogenic avian influenza (LPAI) and HPAI H7N9 human isolates from the fifth epidemic wave were assessed for their pathogenicity and transmissibility in mammalian models, as well as their ability to replicate in human airway epithelial cells. We found that a LPAI virus exhibited a similar capacity to replicate and cause disease in two animal species as viruses from previous waves. In contrast, HPAI H7N9 viruses possessed enhanced virulence, causing greater lethargy and mortality, with an extended tropism for brain tissues in both ferret and mouse models. These HPAI viruses also showed signs of adaptation to mammalian hosts by acquiring the ability to fuse at a lower pH threshold compared with other H7N9 viruses. All of the fifth-wave H7N9 viruses were able to transmit among cohoused ferrets but exhibited a limited capacity to transmit by respiratory droplets. Furthermore, deep sequencing analysis revealed that the H7N9 viruses sampled after transmission showed a reduced amount of minor variants, suggesting a potential purifying selection may take place during H7N9 transmission in ferrets. Taken together, we conclude that the fifth-wave HPAI H7N9 viruses have gained the ability to cause enhanced disease in mammalian models, and with further adaptation may acquire the ability to cause an H7N9 pandemic.

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