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Tropism of an H5N1 virus for bovine cells is determined by the HA2 subunit regulating membrane fusion

Inhalt

In 2024, H5N1 highly pathogenic avian influenza (HPAI) viruses were first detected in dairy cows in Texas, USA. Unlike typical influenza infections, the virus replicated predominantly in mammary gland tissue, causing a mastitis-like disease associated with high virus titers in milk.
To identify viral determinants of bovine cell tropism, we compared the bovine H5N1Tex/24, with the avian H5N1Yam/04 and H5N1Hok/04 viruses in bovine macrophage (BoMac), bovine kidney (MDBK), canine kidney (MDCK), and human lung epithelial (Calu-3) cells. The polybasic HA cleavage sites of H5N1Tex/24 and H5N1Yam/04 were mutated into monobasic motifs, and reassortant viruses were generated by reverse genetics.
H5N1Tex/24 and H5N1Yam/04 replicated efficiently in BoMac and MDBK cells, whereas H5N1Hok/04 did not. However, all three viruses reached high titers in MDCK and Calu-3 cells. Replacement of genome segments 1, 2, 3, 5, and 8 of H5N1Hok/04 with those of H5N1Tex/24 failed to enhance replication in bovine cells. In contrast, replacement of segment 4 significantly increased viral replication, identifying HA as a major determinant of bovine cell tropism.
Serial passaging of H5N1Hok/04 in BoMac cells increased replication capacity and selected mutations in the HA2 subunit. These mutations enhanced HA-mediated membrane fusion, indicating that HA fusion activity is a key factor influencing influenza virus cell tropism.

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No, I am not a junior scientist.

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