

A bovine H5N1 virus efficiently replicates in differentiated human nasal epithelial cells at the temperature of the upper airways

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Highly pathogenic avian influenza (H5N1) viruses of clade 2.3.4.4b have caused significant losses in bird populations worldwide and repeatedly infected mammals, including humans, without sustained human to human transmission. Here we show that an H5N1 virus (H5N1-Tex/24) isolated from bovine milk in Texas in 2024 replicates just as efficiently in differentiated human nasal epithelial cells as a pandemic H1N1 virus strain from 2009 (H1N1), at both 37 °C and 33 °C. The adaptive mutations PB2 M631L and PA K497R promoted replication at 33 °C but had no effect on replication at 37 °C. An H5N1 virus (H5N1-BE/22) isolated from a pelican in 2022, which lacked these mutations, replicated efficiently at 37 °C but poorly at 33 °C, and this limitation was not overcome by the introduction of the PB2 M631L and PA K497R mutations. The differentiated nasal epithelial cell cultures expressed receptors for both human and avian influenza viruses. Accordingly, no HA mutations associated with altered receptor specificity were detected. H5N1-Tex/24 was able to effectively suppress the production of interferon-lambda, yet remained sensitive to the antiviral effects of this cytokine. These findings suggest that H5N1-Tex/24 possesses intrinsic traits supporting efficient replication in differentiated human upper airway cell cultures.

Keywords

avian influenza; zoonosis; pandemic preparedness; virus transmission; species barrier; receptor usage; tropism; adaptation; innate immunity

Professional status of the speaker

Senior Scientist

Junior scientist status

No, I am not a junior scientist.

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