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# Crosstalk between H9N2 avian influenza virus and crypt-derived intestinal organoids

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The spread of Avian influenza virus (AIV) via animal feces makes the virus difficult to prevent, which causes great threat to human health. Therefore, it is imperative to understand the survival and invasion mechanism of H9N2 virus in the intestinal mucosa. In this study, we used mouse three-dimensional intestinal organoids that contained intestinal crypts and villi differentiated from intestinal stem cells (ISCs) to explore interactions between H9N2 avian influenza virus and the intestinal mucosa. The *HA, NA, NP* and *PB1* genes of H9N2 viruses could be detected in intestinal organoids at 1 h, and reached peak levels at 48 h post-infection. Moreover, the *HA* and NP proteins of H9N2 virus could also be detected in organoids via immunofluorescence. Virus invasion caused damage to intestinal organoids with reduced mRNA transcript expression of Wnt3, Axin2, Dll1 and Dll4. The abnormal growth of intestinal organoids may be attributed to the loss of Paneth cells, as indicated by the low mRNA transcript levels of lyz1 and defcr1. This present study demonstrated that H9N2 virus could invade intestinal organoids and then cause damage, as well as affect intestinal stem cells proliferation and differentiation, promoting the loss of Paneth cells.

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