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# Host immunity and viral genotype modulate HPAIV feather replication and shedding

## Inhalt

H5Nx highly pathogenic avian influenza viruses (HPAIV) represent a major threat to animal and public health. In birds, HPAIV shedding mainly occurs via respiratory and digestive routes. These viruses also replicate in vascularized immature feathers, thus representing an additional shedding route. Understanding virus-feather interactions is crucial to limit viral spread and adapt biosafety. Our study aims to characterize feather infection dynamics by several HPAIV, and assess the impact of immunity on viral replication in feathers.
An in ovo model highlighted early feather infection events by Gs/Gd viruses with evidence of sequential infection from feather endothelium to pulp, and epithelium. To assess feather susceptibility independently of systemic dissemination, hatched duck-derived feather explants were infected. All HPAIV and low pathogenicity avian influenza viruses tested were able to infect explants. Upon in vivo challenge, HPAIV replication in naïve animal feathers resulted in necrosis and epithelial debris while replication was blocked in feathers after anti-H5 vaccination of ducks. Interestingly, endothelial infection was more readily detected in ovo than in vivo.
Collectively, our data support a model in which HPAIV infect immature feathers through viremia, leading to epithelial infection and likely contributing to environmental shedding. While many AIV are able to replicate in feather, virus-dependent viremia and host immune status may modulate access to feathers.

## Keywords

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## Professional status of the speaker

PhD student

## Junior scientist status

Yes, I am a junior scientist.

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