Dynamics of virulent avian influenza viruses: conditions favouring invasion and spread

Sema Nickbakhsh and Rowland R. Kao

Boyd Orr Centre for Population and Ecosystem Health, University of Glasgow, Bearsden Road, Glasgow, G61 1QH, UK

Introduction

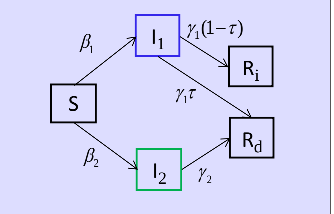
Highly pathogenic avian influenza (HPAI) viruses are known to evolve from **low pathogenic avian influenza (LPAI)** viruses during circulation within commercial poultry flocks[1]. Analytical studies[2] suggest that under complete cross-immunity LPAI should outcompete HPAI within these flocks as high bird mortality drives a relatively lower transmissibility (R_0) for HPAI[3]. However, partial cross-immunity[4] and indirect environmental transmission[5] could enable HPAI to invade and spread in the presence of LPAI. **We explored the dynamics of co-circulating LPAI and HPAI within a poultry flock and identified scenarios that could pose a risk for between-farm spread.**

Within-flock ODE model scenarios

1 = LPAI
2 = HPAI

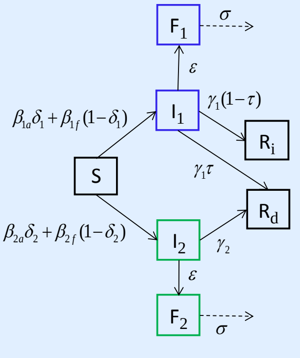
(A) Complete cross-immunity

The number of birds infected with LPAI (**I_1**) and HPAI (**I_2**) are tracked over time. Infection is transmitted between birds directly (via aerosol) at rates β_1 and β_2 respectively. LPAI infected birds either become immune to both strains (**R_i**) at rate $\gamma_1(1\text{-}\tau)$ or die (**R_d**) at rate $\gamma_1\tau$ and all HPAI infected birds eventually die at rate γ_2.



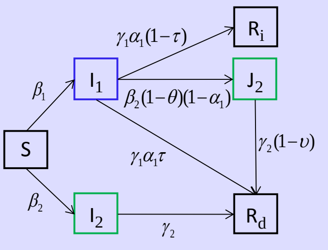
(B) Complete cross-immunity & environmental transmission

Model framework as for **A** but with LPAI (**I_1**) and HPAI (**I_2**) infection transmission by direct (via aerosol, a) and indirect (via infectious faeces, f) mechanisms generating overall rates $\beta_{1a}+\beta_{1f}$ and $\beta_{2a}+\beta_{2f}$ respectively. LPAI and HPAI infectious birds excrete faeces at rate ε and the environmental build-up of infectious faeces is tracked over time (**F_1 and F_2**). Infectious faeces decay at rate σ.



(C) Partial cross-immunity

Model framework as for **A** but with primary LPAI infections (**I_1**) resulting in partial cross-immunity to HPAI. Secondary HPAI infections (**J_2**) occur at rate (β_2 $(1\text{-}\theta)(1\text{-}\alpha)$) where ($\theta$) represents a reduced susceptibility of LPAI infected birds to HPAI. Birds with secondary HPAI infection are assumed to die at a reduced rate (υ).

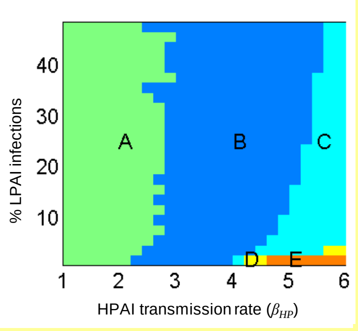


Conditions favouring HPAI invasion

For parameters consistent with recent evidence, where β_{LP}=β_{HP}=2 under frequency-dependent transmission[3], HPAI could not outcompete LPAI and achieve dominance – defined here as a greater relative prevalence – for any model scenario. **HPAI achieved dominance under conditions of relatively high transmission rates (approx. β_{HP}>4) and was more likely for environmental transmission and partial cross-immunity model scenarios.**

Conditions favouring HPAI spread

The risk of between-farm spread of HPAI will likely depend on both the relative **prevalence of HPAI** and the **speed of outbreak detection**. Figure 1 shows how these flock-level characteristics vary with: (i) the HPAI transmission rate (with LPAI transmission rate fixed; β_{LP} = 2) and (ii) the fraction of background LPAI infected birds present at t=0 representing the **time to HPAI emergence**.



A No HPAI dominance, detection >10 days under environmental transmission and partial cross-immunity.

B No HPAI dominance and detection <10 days under all model scenarios.

C HPAI dominance under partial cross-immunity only, detection <10 days under all model scenarios

D HPAI dominance under environmental transmission and partial cross-immunity, detection <10 days under all model scenarios.

E HPAI dominance under all model scenarios, detection >10 days for all model scenarios.

Figure 1. Identifying within-flock conditions that pose a risk of further spread from the index farm in which highly pathogenic avian influenza emerged.

- Environmental transmission and partial cross-immunity can enable HPAI dominance but do not necessarily pose the highest risk as they can result in relatively fast outbreak detection **(C and D).**

- For low β_{HP} HPAI dominance does not occur; under these conditions environmental transmission and partial cross-immunity pose a higher risk through relatively slow outbreak detection **(A)**.

Conclusions

For HPAI to outcompete LPAI within a commercial poultry flock these viruses must transmit at a relatively higher rate than that suggested by recent evidence and is also more likely to occur under environmental transmission and partial cross-immunity. Under these model scenarios outbreak detection can be delayed at relatively low rates of HPAI transmission which also increases the risk of spread through these mechanisms compared to complete cross-immunity.

References

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sema.nickbakhsh@glasgow.ac.uk