Introduction to mathematical modelling of avian influenza in livestock

Julien Arino

April 2023

AI characteristics

Modelling AI

AI characteristics
- Overview
- History of AI outbreaks
- Mechanisms of spread
- Where does one find the virus?

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Avian influenza

- Caused by Influenza A virus
- Adapted to birds but can also stably adapt and sustain P2P transmission

High pathogenicity avian influenza (HPAI)

- HPAI A virus subtype H5N1: emerging avian influenza virus causing global concern as a potential pandemic threat
- H5N1 has killed millions of poultry throughout Asia, Europe, and Africa
- Coexistence of human flu viruses and avian flu viruses (especially H5N1) will provide an opportunity for genetic material to be exchanged between species-specific viruses, possibly creating a new virulent influenza strain that is easily transmissible and lethal to humans
- CFR for humans with H5N1 is 60%

Global concern because it involves multiple bird species, both wild and livestock

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Modelling AI

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Review

The history of avian influenza

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Year	Event
1878	First description of highly pathogenic avian influenza (HPAI) or fowl plague
1880	Differentiation of HPAI from fowl cholera
1901	Identification of HPAI as a virus
1901–1930s	Major outbreaks of HPAI throughout the world
1918	Major human pandemic
1931	First influenza virus isolated (swine)
1941	Recognition of hemagglutination by influenza viruses
1942	HPAI and Newcastle disease virus shown to agglutinate red blood cells and to be different serologically
1955	HPAI virus shown to be a type A influenza virus
1959	Isolation of a HPAI virus serologically different from the classical fowl plague virus in hemagglutination inhibition test
1970s	Intensive surveillance of influenza viruses in wild birds and recognition that wild birds harbor all identified subtypes of influenza viruses
1971	Classification of influenza viruses based on antigenic properties of the NP (type) and HA and NA (subtype) proteins and the species of origin
1977–1981	Recognition that the presence of multiple basic amino acids in the HA cleavage site correlates with tissue spread and virulence of AI strains
1978	Recognition that the 1957 (H2N2) and 1968 (H3N2) pandemic influenza viruses aroused by reassortment with AI viruses
1980	Classification of influenza viruses based on antigenic properties of the NP (type) and HA and NA (subtype) proteins regardless of the species of origin
1981	First International Symposium on Avian Influenza
1981	The name highly pathogenic avian influenza is proposed to substitute fowl plague
1999–2001	H9N2 virus transmission to humans
1997-present	HPAI H5N1 transmission to humans
2000s	H9N2 becomes endemic in Asia
2003-present	HPAI H5N1 spreads through Asia, Europe and Africa and becomes endemic in Asia

Important outbreaks of HPAI documented since 1959*

	HPAI virus	Subtype	Species affected	Approximately number of birds culled
1	A/chicken/Scotland/59	H5N1	Chicken	1 small farm
2	A/tern/South Africa/61	H5N3	Common tern	1300
3	A/turkey/England/63	H7N3	Turkey	29,000
4	A/turkey/Ontario/7732/66	H5N9	Turkey	8000
5	A/chicken/Victoria/76	H7N7	Chickens, ducks	58,000
6	A/chicken/Germany/79	H7N7	Chicken and goose	1chicken and 1 goose farm
7	A/turkey/England/199/79	H7N7	Turkey	9000
8	A/chicken/Pennsylvania/1370/83**	H5N2	Chicken turkey	17,000,000 chickens and turkeys
9	A/turkey/Ireland/1378/83	H5N8	Turkey	307,000, chickens, turkeys and mostly ducks
10	A/chicken/Victoria/85	H7N7	Chicken	240,000
11	A/turkey/England/50-92/91	H5N1	Turkey	8000
12	A/chicken/Victoria/1/92	H7N3	Chicken	18,000 broiler breeders, ducks
13	A/chicken/Queenland/667-6/94	H7N3	Chicken	22,000
14	A/chicken/Mexico/8623-607/94**	H5N2	Chicken	Millions?
15	A/chicken/Pakistan/447/94**	H7N3	Chicken	>6,000,000

16	A/chicken/NSW/97	H7N4	Chicken	160,000 chickens, emus
17	A/chicken/Hong Kong/97	H5N1	Chicken, duck	1,500,000 chickens and other domestic birds
18	A/chicken/Italy330/97	H5N2	Chicken	8000 chickens, turkeys, guinea-fowl, ducks, quail, pigeons, geese, pheasant
19	A/turkey/Italy/99**	H7N1	Turkey	14,000,000 chickens, turkeys, guinea-fowl, quail, ducks, pheasants, ostriches
20	A/chicken/Chile/02	H7N3	Chicken	700,000 chickens, turkeys
21	A/grey heron/Hong Kong/861.1/02	H5N1	Wild birds	Outbreak in wild birds; over 800,000 domestic birds were culled
22	A/chicken/Netherlands/03**	H7N7	Chicken	>34,000,000
23	A/chicken/Asia, Europe and Africa/03-07**	H5N1	Chicken, duck	100s of millions
24	A/chicken/Texas/04	H5N2	Chicken	6600
25	A/chicken/Canada/04**	H7N3	Chicken	16,000,000
26	A/ostrich/South Africa/04	H5N2	Ostrich	30,000
27	A/chicken/North Korea/05	H7N7	Chicken	219,000
28	A/turkey/England/07	H5N1	Turkey	160,000

Strain	Country/area	Date	Cases (deaths)	Symptoms	Source
H7N7 hp	USA	1959	1	Respiratory	Overseas travel
H7N7 hp	Australia	1977	1	Conjunctivitis	Unknown
H7N7 lp	USA	1978–1979	1	Conjunctivitis	Seals
H7N7 lp	UK	1995	1	Conjunctivitis	Pet ducks
H5N1 hp	Hong Kong SAR	1997	18 (6)	Respiratory	Poultry
H9N2 lp	China (Guangdong)	1998	5	Unknown	Unknown
H9N2 lp	Hong Kong SAR	1999	2	Respiratory	Poultry, unknown
H7N2 lp	Virginia, USA	2002	1	Respiratory	Poultry
H5N1 hp	China	2003	1	Respiratory	Unknown
	Hong Kong SAR		2		Poultry
	Viet Nam		3		Poultry
H7N2 lp	New York	2003	1	Respiratory	Unknown
H7N7 hp	Netherlands, Belgium, Germany	2003	89 (1)	Conjunctivitis (pneumonia, respiratory failure in fatal case)	Poultry
H9N2 lp	Hong Kong SAR	2003	1	Respiratory	Poultry
H5N1 hp	Thailand	2004	17 (12)	Respiratory	Poultry
	Viet Nam		29 (20)		
H7N3 hp	Canada	2004	2	Conjunctivitis	Poultry
H10N7	Egypt	2004	2	Respiratory, fever	Poultry/wild ducks
H5N1 hp	Cambodia	2005	4 (4)	Respiratory	Poultry
	China		8 (5)		
	Indonesia		20 (13)		
	Thailand		5 (2)		
	Viet Nam		61 (19)		
H5N1 hp	Azerbaijan	2006	8 (5)	Respiratory	
	Cambodia		2 (2)		
	China		13 (8)		
	Djibouti		1		
	Egypt		18 (10)		Poultry
	Indonesia		55 (45)		Wild birds
	Iraq		3 (2)		
	Thailand		3 (3)		
	Turkey		12 (4)		
H7N3 lp	United Kingdom	2006	1	Conjunctivitis	Poultry
H5N1 hp	Cambodia	2007	1 (1)	Respiratory	
	China		5 (3)		
	Egypt		25 (9)		
	Indonesia		42 (37)		Poultry
	Lao PDR		2 (2)		Birds
	Myanmar		1		Unknown
	Nigeria		1 (1)		
	Pakistan		3 (1)		
	Viet Nam		8 (5)		
H7N2 lp	United Kingdom	2007	4	Conjunctivitis	Poultry
H5N1 hp	Bangladesh	2008	1	Respiratory	Poultry
	Cambodia		1		
	China		4 (4)		Unknown
	Egypt		8 (4)		
	Indonesia		24 (20)		
	Viet Nam		6 (5)		
H5N1 hp	China	2009	1(1)		
	Egypt		32(4)		
	Vietnam		4(4)		

Kalthoff *et al*/Veterinary Microbiology (2010)

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Modelling AI

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Virus Research

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Natural history of highly pathogenic avian influenza H5N1

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Other wild birds
e.g., passerines,
columbidae,
raptors
?
?
?
Sporadic
transmission
Sustained interspecies
transmission
Endemic
H1–H16
N1–N9
Seeding
Reservoir 1
Wild waterfowl
Sporadic
transmission
Sporadic
transmission
Sporadic interspecies
transmission
Sustained or sporadic
interspecies transmission
?
Sporadic
transmission
Endemic: H1–H3
Endemic: H5, H6, H9
Reservoir 3
Mammals
Reservoir 2
Poultry

a) Stable Period
Wild waterfowl
H1-16
N1-9
LPAI
?
Domestic poultry
Mammals
b) Transition Period (Sporadic)
LPAI
LPAI
LPAI
LPAI
LPAI
Mammals
mutation to HPAI
c) Transition Period (Adaptation)
LPAI
LPAI
LPAI
LPAI
LPAI
Mammals
mutation to HPAI
HPAI
HPAI
HPAI
d) Transition Period (Expansion)
?
HPAI
HPAI
HPAI
HPAI
HPAI
HPAI
Mammals

Fig. 3. Timeline of major events of goose/Guangdong-lineage H5N1 evolution. Shown are times of major changes in the evolution of highly pathogenic H5N1, goose/Guangdong-lineage. Expansion into different geographical areas is depicted, as is status in various hosts in different locations: solid lines depict stable interactions between virus and hosts and dashed lines depict transient interactions. Blue lines represent aquatic poultry hosts, green lines terrestrial poultry hosts, and red lines wild birds hosts. SE Asia – South East Asia, Gs/GD – goose/Guangdong, HK – Hong Kong.

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Avian Pathology (June 2009) **38(3),** 193–200

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REVIEW ARTICLE

Avian influenza viruses in poultry products: a review

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Table 1. *Summary of data available in the literature on the presence of HPAI viruses in poultry commodities*

Commodity	Species	Strain	Experimental (E) or natural (N) infection	Infecting dose (EID_{50} log_{10})	Titres (log_{10} EID_{50}/g) detected in commodity
Meat	Chickens	A/duck/Anyang/AVL-1/01 (H5N1)		6/0.1 ml	5.3 to 5.5
		A/chicken/Pennsylvania/1370/1983 (H5N2)	E	6/0.1 ml	2.2 to 3.2
		A/tern/South Africa/61 (H5N3)		7/0.1 ml	>4
	Turkeys	A/turkey/Italy/4580/99 (H7N1)	E	7/0.1 ml	4.38
	Ducks	A/duck/Anyang/AVL-1/01 (H5N1)	E and N	6/0.1 ml	3 to 4
		A/goose/Vietnam/3/2005 (H5N1)	E	6/0.1 ml	3
		A/Vietnam/1203/2004, A/ThailandPB/6231/2004, A/crow/Thailand/2004, A/Egret/HK/757.2/2002 (H5N1)	E	5/0.1 ml	4 to 6 (2-week-old birds) 2 to 4 (5-week-old birds)
		A/egret/HK/757.2/2002 (H5N1)	E	8/0.1 ml	7
		A/chicken/Yamaguchi/7/2004 (H5N1)	E	7/0.1 ml	1.5
		A/duck/Vietnam/12/2007 (H5N1)			
Eggs	Turkeys	A/turkey/Ontario/7732/66 (H5N9)	N	Not reported	Not reported
		H5N2 (Virginia/1985)	E	Not reported	Not reported
	Chickens	H5N2 (Virginia/1985)	N	Not reported	Not reported
	Ducks and geese	H5N1 (strain not reported)	N	Not reported	Not reported
	Quail	H5N1 (strain not reported)	N	Not reported	4.6 to 6.2
Feathers	Chickens, turkeys, quail, guinea fowl	A/chicken/Yamaguchi/7/2004, A/chicken/Miyazaki/K11/2007, A/chicken/Hong Kong/220/1997	E	5.8 to 6.2/0.1 ml	Not investigated
	Ducks	A/chicken/Yamaguchi/7/2004, A/chicken/Miyazaki/K11/2007	E	8/0.1 ml	Not investigated
	Turkeys	H5N1 (strain not reported)	N	Not available	Not investigated
Liver	Ducks	A/chicken/Vietnam/12/2005 (H5N1)	E	7/0.1 ml	Not reported
Blood	Chickens	A/tern/South Africa/61 (H5N3)	E	7/0.1 ml	4
		A/chicken/Pennsylvania/1370/1983 (H5N2)		6/0.1 ml	Not reported
	Pigeons, geese	A/turkey/Ontario/7732/66 (H5N9)	E	8/0.5 ml	Not recovered
	Turkeys	A/turkey/Italy/4580/99 (H7N1)	E	6/0.1 ml	1 to 5.8
		A/turkey/Ontario/7732/66 (H5N9)		8.7/0.5 ml	2.7 to 3.7
	Ducks	A/chicken/Vietnam/12/2005 (H5N1)	E	7/0.1 ml	Not reported
		A/turkey/Ontario/7732/66 (H5N9)	E	8/0.5 ml	Not recovered
Skin	Ducks	A/chicken/Yamaguchi/7/2004,	E	8/0.1 ml	2.5 to 4.4
	Geese	A/chicken/Yamaguchi/7/2004	E	8/0.1 ml	3.5
		A/chicken/Miyazaki/K11/2007	E		4.5

Table 2. *Summary of data available in literature on the presence of LPAI viruses in poultry commodities*

Commodity	Species	Strain	Natural (N) or experimental (E) infection	Infecting dose (EID_{50} log_{10})	Titres (log_{10} EID_{50}/g
Meat	Chickens	A/chicken/aq-Y-55/01 (H9N2);	E	7/0.1 ml	1.6 to 2
		A/chicken/aq-Y-135/01(H9N2)	E	7/0.1 ml	1.6 to 2
	Turkeys	A/turkey/Italy/3675/99 (H7N1)	E	6/0.1 ml	
		A/turkey/Virginia/159512/2002 (H7N2)	E	6/0.1 ml	No infectious virus detected
		A/chicken/New York/21586–8/99 (H7N2)	E	6/0.1 ml	
	Ducks	No data available			
Eggs	Turkeys	A/turkey/California/meleagrium/64; A/turkey/California/5142/66	E	2.25/0.2 ml	No infectious virus detected
	Chickens	A/chicken/Alabama/7395/75 (H4N8)	N	Not reported	No infectious virus detected
	Ducks and geese	Not available			
Feathers	Avian species	Not available			
Liver	Avian species	Not available			
Blood	Chickens	A/chicken/aq-Y-55/01 (H9N2)	E (co infection with *S. aureus*)	6/0.1 ml	Not reported
		A/chicken/Beijing/2/97 (H9N2)		6/0.1 ml	
	Turkeys	A/turkey/Italy/3675/99 (H7N1)	E	6/0.1 ml	<1
	Ducks	No data available			

AI characteristics

Modelling AI

- A review of Stegeman *et al*
- A within-host model of Xie *et al*
- A within-host model of Hagenaars *et al*
- A model of Liu *et al*
- Discrete-time model of Ma and Wang
- A model of Barnes *et al* for outbreaks in poultry farms
- Tiensen *et al* on H5N1 in Thailand
- Nickbakhsh *et al* on co-circulating LPAI strains

Mathematical modelling of AI

A lot more popular than FMD!

There are *many* mathematical models

However, most models look at zoonotic aspects, i.e., include a human component

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AVIAN DISEASES 54:707–712, 2010

Invited Review—

Use of Epidemiologic Models in the Control of Highly Pathogenic Avian Influenza

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SUMMARY. In the past decades, mathematical models have become more and more accepted as a tool to develop surveillance programs and to evaluate the efficacy of intervention measures for the control of infectious diseases such as highly pathogenic avian influenza. Predictive models are used to simulate the effect of various control measures on the course of an epidemic; analytical models are used to analyze data from outbreaks or from experiments. A key parameter in both types of models is the reproductive ratio, which indicates whether virus can be transmitted in the population, resulting in an epidemic, or not. Parameters obtained from real data using the analytical models can subsequently be used in predictive models to evaluate control strategies or surveillance programs. Examples of the use of these models are described here.

	Analytical models	Simulation models
Advantages	Contain few but measurable parameters	Contain a large number of parameters, which take into account many factors that are supposed to affect the spread of these infections in the population of interest. They are often not measured but based on expert opinions
	Can be used to unravel the complex behavior of pathogens in the population	Demonstrate the effect of changes in parameter values on the outcome of the model
	Can be used to quantify the parameters that determine the spread of infections between animals or between farms	
	Helpful for policy makers as they evaluate the efficacy of control and surveillance programs	Helpful for policy makers as they demonstrate more clearly the effect of various scenarios on the outcome of an epidemic
	Provide parameters necessary to run predictive models	Can be used without carrying out experiments or collecting field data or for situations that cannot be examined experimentally or in the field if the pathogen is absent
Disadvantages	Simplification of reality and do not describe the transmission mechanism in much detail	These models often lack real data, as the majority of these parameters cannot be quantified
	Parameters have to be quantified using real data from outbreaks or experiments	The estimates are often based on expert opinions, and the models contain several (implicit) assumptions

Table 2. Estimates of transmission of various AI virus strains between birds of various species.

Pathogenicity	Strain	Bird type	β (day^{-1})	R_0	Study type[A]
HP	H5N1	Chicken		2	Exp
	H5N1	Chicken	1 d^{-1}	1.6	Exp
	H5N1	Duck		>1.5	Exp
	H5N1	Turkey	1.26	7.8	Exp
	H5N1	Poultry		2.6	Field
	H5N2	Chicken		>1.3	Exp
	H7N7	Teal		>1.5	Exp
	H7N7	Pheasant		>1.5	Exp
	H7N7	Chicken		10	Exp
	H7N7	Chicken	4.5 d^{-1}		Field
LP	H5N2	Chicken		>1.3	Exp

[A]Exp = experiment.

Table 3. Estimates of transmission of various AI virus strains between flocks with various species based on field studies.

Pathogenicity	Strain	Bird type	β (week^{-1})	R_0	Reference
HP	H5N1	Poultry		2–2.6	51
	H7N7	Poultry	2	6.5	37
	HxNx	Poultry		1.1–2.4	19
LP	H7N1	Poultry	0.04–0.07	0.6–0.8	30

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A within-host mathematical model of H9N2 avian influenza infection and type-I interferon response pathways in chickens

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Refractory cells
IFN Pathway 1
Susceptible cells
Type-I interferons
Eclipse-phase cells
Infected cells
IFN Pathway 2
Virus
IFN Pathway 3

The effect of three previously described type-I interferon (IFN) pathways and an eclipse phase on cloacal shedding of H9N2 avian influenza infection in chickens. The associated pathways, Sum of Squared Errors (SSE) and corrected Akaike Information Criterion (AICc) of each tested within-host deterministic model is shown. A model schematic can be found in Fig. 1.

Model	Type-I Interferon Pathway(s)	SSE	Model AICc
7E	Eclipse Pathways 2 + 3	1.01e+07	54.58
3	Pathway 3	6.05e+07	58.21
7	Pathways 2 + 3	4.40e+07	59.95
3E	Eclipse Pathway 3	5.38e+07	60.96
1E	Eclipse Pathway 1	4.01e+07	62.82
2E	Eclipse Pathway 2	9.57e+07	63.84
5E	Eclipse Pathways 1 + 2 + 3	4.01e+07	64.49
2	Pathway 2	2.29e+08	64.86
6	Pathways 1 + 3	7.02e+07	65.62
5	Pathways 1 + 2 + 3	6.63e+07	66.29
4	Pathways 1 + 2	1.03e+08	67.55
6E	Eclipse Pathways 1 + 3	1.05e+08	68.58
4E	Eclipse Pathways 1 + 2	1.27e+08	69.54
1	Pathway 1	3.43e+08	72.22

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RESEARCH ARTICLE

Modelling the Innate Immune Response against Avian Influenza Virus in Chicken

T. J. Hagenaars[1]*, E. A. J. Fischer[1¤a], C. A. Jansen[2], J. M. J. Rebel[1¤b], D. Spekreijse[3¤c], L. Vervelde[2¤d], J. A. Backer[1¤e], M. C. M. de Jong[4], A. P. Koets[1,2,3]

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Fig 1. Model structure. Diagrammatic representation of the model, featuring pulmonary cells, virus, interferons and (non-resident) NK cells. Full-line arrows with v-shaped head denote flows between model compartments, corresponding to cell transition rates. Dotted arrows denote "influences" on transition rates. Out-flows from compartments indicated by full-line arrows with triangular head: mortality. In-flows into compartments indicated by full-line arrows with bullet-shaped base: production.

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Spread Pattern Formation of H5N1-Avian Influenza and its Implications for Control Strategies

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3 types of birds + environment

- Poultry (mainly chicken), c
- Wild birds who die after H5N1 infection, w
- Wild birds who survive after H5N1 infection, d
- V virus density in the environment

α_c
d_{ic}
S_c
I_c
r_c
β_c
r_{ew}
d_{iw}
α_{ew}, α_{iw}
δ
S_w
E_w
μ_w
I_w
r_{iw}
V
β_w
d_v
r_d
α_d
γ_d
S_d
I_d
R_d
β_d
η_d

AI characteristics

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A discrete model of avian influenza with seasonal reproduction and transmission

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In this paper, we formulate a discrete-time model with the reproductive and overwintering periods to assess the impact of avian influenza transmission in poultry. It is shown that the disease is extinct if the basic reproduction number is less than one and is persistent if the basic reproductive number is greater than one. Furthermore, the model admits a closed invariant cycle, which means that avian influenza fluctuates in poultry.

Keywords: poultry; reproduction number; global stability; permanence; Hopf bifurcation

Divide year in two periods

- During the *reproductive period*, denoted p, poultry can reproduce

- During the *overwintering period*, denoted w, poultry does not reproduce and AI emerges. Further subdivided
  - infection phase
  - disease-control phase

Individuals infected with AI do not recover

$$
\begin{aligned}
S^p_{n+1} &= f_r(N^w_n)(S^w_n + vI^w_n) + u_p S^w_n \\
I^p_{n+1} &= u_p \sigma_p I^w_n \\
S_{n+1} &= S^p_{n+1} \Phi\left(\beta \frac{I_n 1^p}{N^p_{n+1}}\right) \\
I_{n+1} &= S^p_{n+1}\left(1 - \Phi\left(\beta \frac{I_n 1^p}{N^p_{n+1}}\right)\right) + I^p_{n+1} \\
S^w_{n+1} &= u_w S_{n+1} \\
I^w_{n+1} &= u_w \sigma_w I_{n+1}
\end{aligned}
$$

where

$$
\begin{aligned}
N^p_n &= S^p_n + I^p_n \\
N^w_n &= S^w_n + I^w_n \\
N_n &= S_n + I_n
\end{aligned}
$$

Assumptions on Φ

Φ satisfies

- $\Phi : [0, \infty) \to [0, 1]$
- $\Phi(0) = 1$, $\lim_{x \to \infty} \Phi(x) = 0$
- $\Phi'(x) < 0$ and $\Phi''(x) \geq 0$ for all $x \in [0, \infty)$
- $-\Phi'(x)x < 1$ for all $x \in [0, \infty)$

They then conduct a thorough analysis of the system using

$$\mathcal{R}^\star = \frac{a u_w}{1 - u_p u_w}$$

and

$$\mathcal{R}_0 = \frac{-\beta u_w u_p \sigma_p \sigma_w \Phi'(0)}{1 - u_p \sigma_p u_w \sigma_w}$$

Proposition 1

Let $\mathcal{R}^\star > 1$. If $\mathcal{R}_0 < 1$, DFE is GAS; if $\mathcal{R}_0 > 1$, DFE is unstable and system is uniformly persistent

Additional results (flip bifurcation, Hopf bifurcation, etc.) based on another $\hat{\mathcal{R}}_0$

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Modelling high pathogenic avian influenza outbreaks in the commercial poultry industry

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A branching process model

X r.v. "number of newly infected birds in a generation"

Generation time: average time in days between successive generations in infection process

Assume Poisson branching process for transmission, mean transmission rate $\lambda = p_L^B L + p_G^B M$

Local contact probability p_L^B between birds within social group of size L and smaller global component between all birds within the flock of size M with lower infectious contact probability p_G^B

Then probability generating function for number of newly infected birds in each generation is

$$\Phi_X(s) = \exp\Big((p_L^B L + p_G^B M)(s-1)\Big)$$

and probability of extinction q the smallest root in $(0,1]$ of

$$q = \Phi_X(q)$$

Reproduction number is

$$R_\star = p_L^B L + p_G^B M$$

They then derive a model for infection within a cage

And models for emergence of HPAI from LPAI

Then look at various population structures (barn and free-range layer flocks, caged layer flocks, barn and free-range meat flocks, LPAI introductions)

Production sector characteristics for commercial chicken production farms in Australia. Data are from a survey of Australian farms and expert opinion, where the number of birds per shed, and sheds per farm, are mean values, and the relative contact probabilities are weighted medians. Further details are provided in the text.

Enterprise characteristic	Enterprise type				
	B meat	FR meat	B layer	C layer	FR layer
Birds/cage	–	–	–	6	–
Birds/shed	20,500	22,500	10,500	26,500	10,500
Sheds/farm	4	7	4	4	4
Farms	≈ 700	≈ 160	≈ 25	≈ 125	≈ 100
Cycle length	49 days	49 days	87 weeks	87 weeks	87 weeks
Cleaning time	14 days	14 days	–	–	–
Range access	N	Y	N	N	Y
Relative probability of infectious contact with wildbirds	1	1.12	1	1	3.62

B meat – barn meat; FR meat – free-range meat; B layer – barn layer; C layer – caged layer; FR layer – free-range layer.

Fig. 2. Contours for the probability of an avian influenza outbreak across the Australian industry (1110 farms) in a year: (a) for an LPAI outbreak; (b) for an HPAI outbreak with mutation probability per transmission event $\gamma = 0.001$; and (c) for an HPAI outbreak with mutation probability per transmission event $\gamma = 0.1$. Details for each sector, and their relative size within the industry, are provided in Table 2.

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Transmission of the Highly Pathogenic Avian Influenza Virus H5N1 within Flocks during the 2004 Epidemic in Thailand

Thanawat Tiensin,[1,5] Mirjam Nielen,[5] Hans Vernooij,[5] Thaweesak Songserm,[4] Wantanee Kalpravidh,[2] Sirikan Chotiprasatintara,[1] Arunee Chaisingh,[3] Surapong Wongkasemjit,[3] Karoon Chanachai,[1] Weerapong Thanapongtham,[1] Thinnarat Srisuvan,[3] and Arjan Stegeman[5]

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B

$$E(C) = \frac{\beta SI}{N}\Delta t$$

Figure 1. Stages in the SIR model used to estimate avian influenza A subtype H5N1's dynamics of transmission between individual birds within a flock. *A*, SIR model: a bird is first susceptible (S), becomes infected (according to transmission-rate parameter [β]), and stays for a time (t) in the infectious stage (I) before it dies of H5N1 (R, removed). *B*, Equation for the expected value for newly infected cases, used for statistical analysis [12, 18].

Figure 2. Characteristics of 139 chicken flocks infected with the highly pathogenic avian influenza A virus subtype H5N1 during the 2004 Thai epidemic that are used in the present study: Number and percentage of flock type *(A)*, flock size *(B)*, cumulative mortality *(C)*, and average daily mortality *(D)*.

Table 2. Estimates of transmission-rate parameter (β) and basic reproduction number (R_0), with 95% confidence interval in parentheses, based on a generalized linear model (GLM) of data on 139 chicken flocks infected with the highly pathogenic avian influenza A virus subtype H5N1 during the 2004 Thai epidemic, with flock as a random effect and with results from the GLM with 2 groups of flock types added.

	Infectious period, days			
Flock type and values	1	2	3	4
All				
β, per day	2.26 (2.01–2.55)	1.23 (1.11–1.36)	0.87 (0.75–1.02)	0.66 (0.50–0.87)
R_0	2.26 (2.01–2.55)	2.46 (2.23–2.72)	2.62 (2.25–3.07)	2.64 (2.02–3.47)
Laying hens and broiler chickens				
β, per day	2.30 (1.92–2.76)	1.43 (1.20–1.71)	1.16 (0.90–1.50)	0.79 (0.50–1.25)
R_0	2.30 (1.92–2.76)	2.86 (2.41–3.41)	3.49 (2.70–4.50)	3.17 (2.01–5.00)
Backyard chickens and fighting cocks				
β, per day	2.18 (1.94–2.46)	1.15 (1.02–1.30)	0.75 (0.63–0.91)	0.60 (0.43–0.84)
R_0	2.18 (1.94–2.46)	2.31 (2.05–2.60)	2.26 (1.88–2.72)	2.40 (1.71–3.36)

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Modelling the impact of co-circulating low pathogenic avian influenza viruses on epidemics of highly pathogenic avian influenza in poultry

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Biological assumptions:

- The LPAI prevalence on day one corresponds with time to emergence of a "fit" HPAI mutant within a single bird
- Transmission occurs via aerosol and infectious faeces in the environment with a 10-fold greater rate via faeces
- LPAI infected birds are temporarily insusceptible to HPAI reflecting the action of innate immunity
- LPAI infected birds acquire a secondary HPAI infection at a rate dependent on the level of immunological cross-protection
- No secondary HPAI infections occur under 100% immunological cross-protection
- Secondary HPAI infections occur at the same rate as primary HPAI infections under 0% immunological cross-protection
- No interference between the two viral strains occurs during co-infection i.e. LPAI and HPAI are equally transmitted
- Birds singly infected with LPAI eventually acquire long-term immunity to both strains
- Birds singly infected with HPAI eventually die as a consequence of avian influenza disease
- Co-infected birds die as a consequence of avian influenza at a rate dependent on the level of cross-protection

S
E1
I1
F1
E3
I3
R
E2
I2
F2
δ
ε
σ
γ1
γ2
β1aI′1+β1fF′1
β2a(1 − φ)I′2+β2f(1 − φ)F′2
β2aI′1+β2fF′2