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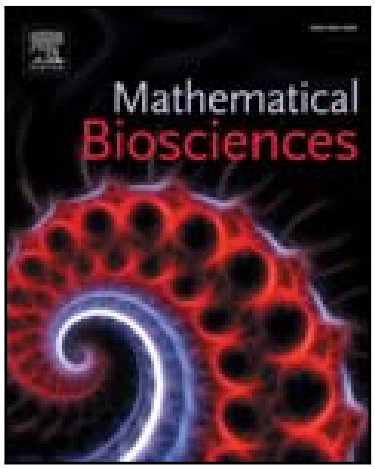

Modelling low pathogenic avian influenza introduction into the commercial poultry industry

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PII: S0025-5564(17)30416-9
DOI: 10.1016/j.mbs.2018.03.021
Reference: MBS 8054

To appear in: *Mathematical Biosciences*

Received date: 31 August 2017
Revised date: 15 March 2018
Accepted date: 19 March 2018

Please cite this article as: Belinda Barnes, Kathryn Glass, Modelling low pathogenic avian influenza introduction into the commercial poultry industry, *Mathematical Biosciences* (2018), doi: 10.1016/j.mbs.2018.03.021

Highlights

- The branching process model formulated for livestock populations, which includes continuous stochastic low-level virus introduction, variable virus transmission rates and distinct housing structures, has general relevance.
- Including variability in virus transmission rates to model heterogeneity in influenza virus characteristics is important
- For similar flock sizes, free-range access is the most influential driver of virus introduction across poultry sectors, while the effect of production cycle length is low
- Flock sizes below 10,000 birds, typical in free-range systems, temper the increase in risk due to free-range access in a highly nonlinear way.
- Outbreaks are less likely in caged systems than in barn systems, and risk decreases rapidly with fewer than 10 birds in a cage.
- The probability the meat sector is virus-free may be lower than for the layer sector, while the probability of no major outbreak is higher than for the layer sector.
- Barn meat sheds have the lowest probability of virus introduction; however, large flock and sector sizes mean the sector can be highly influential on risk at the industry scale.

Modelling low pathogenic avian influenza introduction into the commercial poultry industry

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March 22, 2018

Abstract

Outbreaks of highly pathogenic avian influenza (HPAI) in commercial poultry flocks are rare but highly disruptive to the industry. There is evidence that low pathogenic avian influenza (LPAI) can transfer from wild birds to domestic flocks, where it may mutate to HPAI, and the industry is concerned that an increasing demand for free-range produce may affect the risk of LPAI and HPAI outbreaks. In this paper we focus on LPAI introduction and establishment, and formulate a branching process model to compare risk between sectors and their contribution to overall industry-level risk. Our aim is to determine how heterogeneity in avian influenza viruses and the distinct population structures of each sector — caged, barn and free-range, meat and layer — interact with a continuous risk of virus introduction to affect outbreak probabilities. We show that free-range access is the most influential driver of LPAI outbreaks, with production cycle length having relatively little effect. We demonstrate that variation in virus transmission rates is particularly important when modelling avian influenza introduction to domestic poultry. Virus-free status is of interest for biosecurity and we distinguish how it differs from the usual probability of extinction, and discuss how production cycle length affects this difference. We also use the nonlinear relationship between shed size and risk to identify conditions for which shed size is most influential.

1 Introduction

The poultry industry faces a continuous risk that avian influenza will be introduced from wild bird populations which carry various subtypes of the virus. The outbreaks of avian influenza in poultry in the United States in 2014–15 were described by the US Department of Agriculture as being the "worst animal disease outbreak in US history", affecting over 48 million birds [10]. Highly pathogenic avian influenza (HPAI) kills domestic birds rapidly and is a concern for human health. While HPAI can be transferred directly from wild birds to flocks [12, 13], it is more common for low pathogenic avian influenza (LPAI) to be transferred to domestic flocks, where it may mutate quickly to HPAI [23, 25], or circulate undetected for some time before mutating to HPAI [8, 14, 18, 27]. The increase in demand for free-range eggs and chicken meat has changed typical farming practices, and a current concern for the poultry industry is that this could lead to an increase in avian influenza outbreaks due to increased contact of poultry with the environment.

Stochastic branching processes are ideal for modelling the probability of disease outbreaks in livestock populations. They are particularly robust when assessing mitigation and response strategies aimed at reducing the risk of outbreaks, and when comparing the relative risk of disease outbreaks under different scenarios. Such models can allow for distinct local conditions (such as stock density, housing conditions, and re-stocking practices), thereby incorporating heterogeneity in population structure and associated differences in disease transmission mechanisms. Probability generating functions can be used to calculate the probability of disease outbreaks in different settings, predict the impact of changes in industry structure on outbreak risk, and assess the potential for control measures to reduce this risk.

We formulate a flexible branching process model to assess the relative risk of LPAI outbreaks posed by each sector of a commercial poultry industry. Our purpose is to determine how differences in housing, or population structures, interact with stochastic introduction and the heterogeneous nature of avian influenza viruses to influence LPAI outbreak probabilities, at both the sector and industry scales. In prior work, we developed a multi-scale branching process model to capture the spread of equine influenza in horse populations [15]. However, our model assumed that disease had been initiated by one infected animal and did not include disease introduction. We also did not consider animal management practices within farms. Here we extend that model to include the diversity of LPAI viruses and a stochastic low-level risk of introduction, and explore how they interact with poultry management practices to drive the probability of disease outbreaks. The framework constructed builds on theoretical results for Galton-Watson processes with immigration [1, 2, 19], and although designed for the poultry application it has general relevance to a variety of biosecurity questions related to livestock.

In Section 2 we develop a stochastic branching process model for the poultry industry which includes continuous introduction and transmission of LPAI. We incorporate diversity in influenza virus characteristics and distinct poultry housing conditions that affect influenza transmission (Sections 3 and 4) and establish an appropriate parameterisation for our application (Section 5). We compare outbreak risks between and across farming sectors at

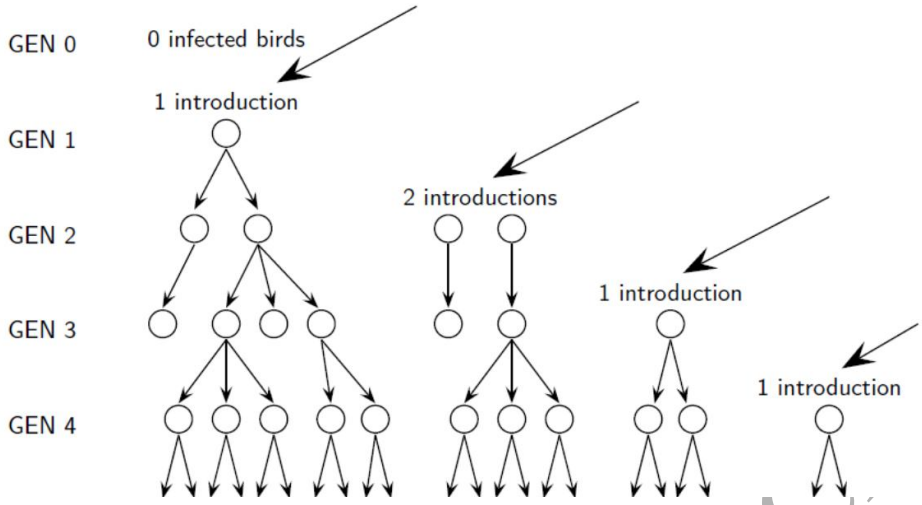

Figure 1: Conceptual diagram of avian influenza introduction and transmission in generations. Each node represents an infected bird and arrows represent transmission.

both the shed and industry levels (Section 6). In Section 7 we summarise our findings and discuss extensions to the model and some alternative applications.

2 Model framework

We adopt a Galton-Walton process with immigration to describe poultry populations in which birds live in close proximity to one another, with introduction of disease from outside the population. The model concept is illustrated in Figure 1 and Table 1 lists variables and probabilities. Let X be a random variable for the number of newly infected birds in a generation caused by a single infected bird, where a generation is the average time between successive infection events. Let $\Phi_{\text{x}}(s)$ be the probability generating function associated with X. Starting with a single infected bird in generation $t = 0$, the probability generating functions for successive generations $\Phi_{\text{x}}^{(t)}(s)$ are:

$$\begin{aligned}
t = 0: &\quad \Phi_{\text{x}}^{(0)}(s) = s \\
t = 1: &\quad \Phi_{\text{x}}^{(1)}(s) = \Phi_{\text{x}}\left(\Phi_{\text{x}}^{(0)}(s)\right) = \Phi_{\text{x}}(s) \\
t = 2: &\quad \Phi_{\text{x}}^{(2)}(s) = \Phi_{\text{x}}\left(\Phi_{\text{x}}^{(1)}(s)\right) = \Phi_{\text{x}}\left(\Phi_{\text{x}}(s)\right) \\
t = 3: &\quad \Phi_{\text{x}}^{(3)}(s) = \Phi_{\text{x}}\left(\Phi_{\text{x}}^{(2)}(s)\right) = \Phi_{\text{x}}\left(\Phi_{\text{x}}\left(\Phi_{\text{x}}(s)\right)\right) \\
\vdots &\quad \vdots
\end{aligned} \tag{1}$$

where $\Phi_{\text{x}}^{(t)}(s)$ is the probability generating function for the number of newly infected birds in generation t, conditional on a single infected bird at time $t = 0$, and is given by the t^{th} iterate of $\Phi_{\text{x}}(s)$.

We now add disease introduction into the above process. Let $\Phi_{\text{B}}(s)$ be the probability generating function for disease introduction, where B is a random variable for the number

symbol	description
t	generation of the process
X	random variable for disease transmission
B	random variable for disease introduction
$Z_i^{(t)}$	random variable for the number of infected birds in generation t arising from a process (processes) initiated in generation i
$N^{(t)}$	random variable for total infected birds in generation t
q_τ	probability that a process initiated in or before generation τ dies out

Table 1: Symbols and descriptions for variables, parameters and probabilities.

of newly infected birds in the population from external sources in a single generation. We assume a stationary introduction process: that is, the risk of disease introduction is the same in each generation.

To simplify analysis of this system, we group infected birds in transmission chains so that we can consider the process as a sum of identically distributed, independent processes. For example, in Figure 1, there are five chains: one initiated in generation 1, two initiated in generation 2, and one initiated in each of generations 3 and 4. Define $N^{(t)}$ to be the total number of newly infected birds in generation t, expressed as a sum of random variables:

$$N^{(t)} = \sum_{i=1}^{t} Z_i^{(t)} = Z_1^{(t)} + Z_2^{(t)} + ... + Z_t^{(t)}, \tag{2}$$

where each $Z_i^{(t)}$ is a random variable for the number of newly infected birds in generation t from the branching processes initiated in generation i through introduction. Random variables $Z_i^{(t)}$ are independent of one another because introduction events are independent and each chain is independent of all others.

The probability generating function for $N^{(t)}$, notated $\Phi_{\text{N}}^{(t)}(s)$, is then:

$$\Phi_{\text{N}}^{(t)}(s) = \prod_{i=1}^{t} \Phi_{z_i}^{(t)}(s),$$

where $\Phi_{z_i}^{(t)}(s)$ are the probability generating functions for $Z_i^{(t)}$. Each process Z_i may be initiated by one or more infected birds. From Equation (1), the probability generating function for the number of newly infected birds in generation t from a chain initiated in by a *single* infected bird in generation i is $\Phi_X^{(t-i)}(s)$. It follows that:

$$\Phi_{z_i}^{(t)}(s) = \sum_b \mathbf{P}\left(B = b\right) \left(\Phi_{\text{X}}^{(t-i)}(s)\right)^b = \Phi_{\text{B}}\left(\Phi_{\text{X}}^{(t-i)}(s)\right).$$

Combining introductions across generations $1 \leq i \leq t$, we have:

$$\Phi_{\text{N}}^{(t)}(s) = \prod_{i=1}^{t} \Phi_{\text{B}}\left(\Phi_{\text{X}}^{(t-i)}(s)\right). \tag{3}$$

This probability generating function defines the distribution for the number of newly infected birds in generation t, with stochastic disease introduction in any generation.

A key use of probability generating functions is the derivation of the probability that a branching process dies out. As our model includes continuous disease introduction, we define a similar concept: the probability (q_τ) that all branching processes initiated by virus introductions in generations up to generation τ die out. We compare this definition with an alternative $\widehat{q}_\tau$, that all branching processes initiated by virus introductions in generations up to generation τ, have died out by generation τ, that is, the population is virus-free in generation τ (Supplementary Material Section A). These two extinction probabilities, q_τ and $\widehat{q}_\tau$, are subtly different but provide distinct insights, with $\widehat{q}_\tau < q_\tau$ for $\tau \geq 1$.

3 Variability in the transmission rate

Influenza viruses in wild birds are known to be diverse, even within sub-types and strains, and thus we expect the virus transmission rate following introduction into poultry to vary between introduction events [13, 16]. We adapt model (3) to incorporate this diversity. We assume within-flock transmission to be a Poisson process with mean rate Λ, where Λ is sampled from an exponential distribution with parameter $\nu = 1/\mathbf{E}(\Lambda) = 1/\lambda$:

$$X|\Lambda \sim \text{Poisson}(\Lambda), \quad \text{with} \quad \Lambda \sim \text{Exponential}\,(\nu)\,.$$

It follows that the probability all virus introductions into a single population in τ generations die out is (Supplementary Material Section B):

$$q_\tau = \left(\Phi_{\text{B}}\left(\Phi_\Lambda\left(q_\Lambda\right)\right)\right)^\tau = \left(\mathrm{e}^{\eta\left(\int_0^\infty q_\ell \lambda^{-1}\mathrm{e}^{-\ell/\lambda}\,d\ell - 1\right)}\right)^\tau, \tag{4}$$

where, for $\Lambda = \ell$, $q_\ell \in (0, 1]$ is the smallest root of $q_\ell = \mathrm{e}^{\ell(q_\ell - 1)}$. Alternatively, for all virus introductions into a single population in τ generations, the probability that the population is virus-free in the τ^{th} generation, is:

$$\widehat{q}_\tau = \prod_{i=1}^{\tau} \Phi_{\text{B}}\left(\Phi_\Lambda\left(q_{\tau-i,\Lambda}\right)\right) = \prod_{i=1}^{\tau} \mathrm{e}^{\eta\left(\int_0^\infty q_{\tau-i,\ell}\,\lambda^{-1}\mathrm{e}^{-\ell/\lambda}\,d\ell - 1\right)}, \tag{5}$$

where, for $\Lambda = \ell$, $q_{\tau-i,\ell}$ is the probability an introduced virus with transmission rate ℓ dies out within $\tau - i$ generations, $q_{0,\ell} = 0$ and $q_{\tau-i,\ell} = \Phi_{X|\Lambda=\ell}(q_{\tau-i-1,\ell})$ for $1 \leq i < \tau$.

4 Population structure

The spread of avian influenza is made more complex by poultry housing conditions, which may allow birds to move freely (as in free-range or barn sheds), or may restrict movement (as in caged sheds).

4.1 Barn and Free-Range sheds

In a barn or free-range shed we assume a Poisson offspring distribution ($X \sim \text{Poisson}(\lambda)$) for within-shed transmission. We define M to be the number of birds in the shed, and assume there are two mechanisms of transmission that occur concurrently and independently. The first mechanism reflects local transmission and is the probability of direct infectious contact with any susceptible bird (p_{L}^{B}). The second mechanism reflects global spread within the shed and is the probability of indirect infectious contact via people, equipment, water, or feed (p_{G}^{B}). Chickens are gregarious animals with defined social order, and mixing and feeding are restricted to groups [22, 26]. We adjust for this local contact population by defining L to be the local-group size within the shed ($0 < L \ll M$), and assuming $\lambda = p_{\text{L}}^{\text{B}} L + p_{\text{G}}^{\text{B}} M$. This structure allows for local spread that is independent of shed size (since $L \ll M$), together with global spread that scales with the number of birds in the shed. The reproduction number is then:

$$R_*^{\text{B}} = p_{\text{L}}^{\text{B}} L + p_{\text{G}}^{\text{B}} M, \tag{6}$$

and the probability generating function is:

$$\Phi_{\text{X}}(s) = \text{e}^{(p_{\text{L}}^{\text{B}} L + p_{\text{G}}^{\text{B}} M)(s-1)}. \tag{7}$$

To accommodate small values of L, embedding a local household-type model within the model structure would improve results and provide greater generality. For our application it has not been included because its effect on our results is likely to be small, with local group sizes relatively large in commercial flocks [22].

4.2 Caged sheds

To model poultry housed in a caged system, we extend the great-circle model of Ball *et al.* [4]. As before, M is the total number of birds in a shed, and we set m to be the number of birds in each cage, with $1 \leq m \ll M$. To simplify the model structure, we assume a one-dimensional linear arrangement of cages on the circumference of a circle to avoid boundary conditions [3]. We set the probability of direct infectious contact with one bird to be p_{L}^{C}, and for simplicity we assume this applies within the cage and to neighbouring cages (although equations with two transmission probabilities are easily deduced). We assume that the total number of birds infected in a cage initiated by one infected bird is given by the final size of the Reed-Frost model [5], and the probability that a neighbouring cage is infected is determined from this final outbreak size, using the clumped Reed-Frost model [3, 5]. Finally, we assume that any infected bird can infect any other bird in the shed through indirect spread with probability p_{G}^{C}, and that the transmission process is Poisson.

The probability generating function for Y, the final number of *newly* infected birds in a cage given one initial infected bird is:

$$\Phi_{\text{Y}}(s) = \sum_{y=0}^{m-1} \mathbf{P}(Y = y, m-1) s^y. \tag{8}$$

where the probability $\mathbf{P}(Y = y)$ is given by the final size of the Reed-Frost model (Supplementary Material Section C). We model transmission between cages based on this final outbreak size $(1 + y)$. Note that this process does not follow strict time generations, but is nevertheless a branching process that can be used to estimate the probability of disease extinction. The probability that an infected cage infects an uninfected adjacent cage is:

$$\begin{aligned} 1 - \left(\sum_{y=0}^{m-1} (1 - p_{\text{L}}^{\text{c}})^{m(1+y)} \mathbf{P}(Y = y) \right) &= 1 - (1 - p_{\text{L}}^{\text{c}})^m \left(\sum_y (1 - p_{\text{L}}^{\text{c}})^{my} \mathbf{P}(Y = y) \right) \\ &= 1 - (1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{Y}} \left((1 - p_{\text{L}}^{\text{c}})^m \right). \end{aligned} \tag{9}$$

Following [2], we define a k-component to be a chain of k infected cages. The probability, denoted π_k, that the initial infected cage leads to a k-component (see Supplementary Material Section D) is:

$$\pi_k = k \left((1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{Y}} \left((1 - p_{\text{L}}^{\text{c}})^m \right) \right)^2 \left(1 - (1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{Y}} \left((1 - p_{\text{L}}^{\text{c}})^m \right) \right)^{k-1},$$

and thus the probability generating function for components is:

$$\begin{aligned} \Phi_\pi(s) &= \sum_{k \geq 1} \pi_k s^k \\ &= \sum_{k \geq 1} k \left((1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{Y}} \left((1 - p_{\text{L}}^{\text{c}})^m \right) \right)^2 \left(1 - (1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{Y}} \left((1 - p_{\text{L}}^{\text{c}})^m \right) \right)^{k-1} s^k \\ &= \left((1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{X}} \left((1 - p_{\text{L}}^{\text{c}})^m \right) \right)^2 s \sum_{k \geq 1} k \left(1 - (1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{Y}} \left((1 - p_{\text{L}}^{\text{c}})^m \right) \right)^{k-1} s^{k-1} \\ &= \frac{\left((1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{Y}} \left((1 - p_{\text{L}}^{\text{c}})^m \right) \right)^2 s}{\left(1 - (1 - (1 - p_{\text{L}}^{\text{c}})^m \Phi_{\text{Y}} \left((1 - p_{\text{L}}^{\text{c}})^m \right)) s \right)^2}. \end{aligned} \tag{10}$$

It follows that the mean component size is:

$$\Phi'_\pi(s)|_{s=1} = \frac{2}{(1 - \hat{p}_{\text{L}}^{\text{c}})^m \Phi_Y \left((1 - p_{\text{L}}^{\text{c}})^m \right)} - 1 = \frac{1 + \mathbf{P}\left(\text{adjacent pen infected}\right)}{1 - \mathbf{P}\left(\text{adjacent pen infected}\right)}. \tag{11}$$

The number of infected birds in a cage is conditional on the k-component to which the cage belongs, and thus the probability generating function for the total number of infected birds in a k-component is:

$$\mathbf{E}\left[s^{k(1+Y|k)} \right] = \left(s \Phi_{\text{Y}_\text{k}}(s) \right)^k,$$

where $\mathbf{E}$ denotes expectation and the probability generating function for Y, conditional on component size k, is:

$$\Phi_{\text{Y}_\text{k}}(s) = \frac{\sum_y \left(s(1 - \hat{p}_{\text{L}}^{\text{c}})^{2m} \right)^y \left(1 - (1 - \hat{p}_{\text{L}}^{\text{c}})^{m(1+y)} \right)^{k-1} \mathbf{P}(Y)}{\sum_y \left((1 - \hat{p}_{\text{L}}^{\text{c}})^{2m} \right)^y \left(1 - (1 - \hat{p}_{\text{L}}^{\text{c}})^{m(1+y)} \right)^{k-1} \mathbf{P}(Y)}.$$

Combining all k-components, the probability generating function for the total number of infected birds is then:

$$\sum_{k \geq 1} \left(s \Phi_{\text{Y}_\text{k}}(s) \right)^k \pi_k = \Phi_\pi \left(s \Phi_{\text{Y}_\text{k}}(s) \right). \tag{12}$$

To include indirect (global) spread, where an infected bird can infect any other bird in the shed with probability p_{G}^{C}, we define $\Phi_{\text{G}}(s)$ to be the probability generating function for this process. The probability generating function for the model that includes all three mechanisms of spread is given by:

$$\Phi_{\pi}\left(\Phi_{\text{G}}(s)\Phi_{Y_{\text{k}}}\left(\Phi_{\text{G}}(s)\right)\right). \tag{13}$$

It follows that the reproduction number for the spread of avian influenza in caged poultry is (Supplementary Material Section E):

$$R_{*}^{\text{C}} \;=\; p_{\text{G}}^{\text{C}} M \sum_{k\geq 1} k\left(1+\mathbf{E}(Y_{\text{k}})\right)\pi_{k}, \tag{14}$$

where $\mathbf{E}(Y_{\text{k}}) = \Phi'_{Y_{\text{k}}}(s)$ evaluated at $s=1$.

Finally, we introduce the continuous low-level risk of virus introduction with probability generating function $\Phi_{\text{B}}(s)$. The probability generating function for the full model including virus introduction in any of τ generations is given by:

$$\left(\Phi_{\text{B}}\left(\Phi_{\pi}\left(\Phi_{\text{G}}(s)\Phi_{Y_{\text{k}}}\left(\Phi_{\text{G}}(s)\right)\right)\right)\right)^{\tau}, \tag{15}$$

It follows that the probability that no major outbreaks occur as a result of virus introduction over τ generations is:

$$q_{\tau} = \left(\Phi_{\text{B}}(q)\right)^{\tau},$$

where q is the smallest root of $q = \Phi_{\pi}(\Phi_{\text{G}}(q)\Phi_{Y_{\text{k}}}(\Phi_{\text{G}}(q)))$.

For low expected transmission rates and small cage sizes the above model can be estimated by assuming cage outbreak-size to be independent of component size. In this case the probability generating functions for: (a) the total number of birds infected locally; (b) the total number infected; and (c) the reproduction number, have a more tractable form:

$$\Phi_{\pi}\left(s\Phi_{\text{Y}}(s)\right) = \frac{\left((1-\hat{p}_{\text{L}}^{\text{C}})^{m}\Phi_{\text{Y}}\left((1-\hat{p}_{\text{L}}^{\text{C}})^{m}\right)\right)^{2} s\Phi_{\text{Y}}(s)}{\left(1-\left(1-(1-\hat{p}_{\text{L}}^{\text{C}})^{m}\Phi_{\text{Y}}\left((1-\hat{p}_{\text{L}}^{\text{C}})^{m}\right)\right)s\Phi_{\text{Y}}(s)\right)^{2}}; \tag{16a}$$

$$\Phi_{\pi}\left(\Phi_{\text{G}}(s)\Phi_{\text{Y}}\left(\Phi_{\text{G}}(s)\right)\right) = \frac{\left((1-p_{\text{L}}^{\text{C}})^{m}\Phi_{\text{Y}}\left((1-p_{\text{L}}^{\text{C}})^{m}\right)\right)^{2} \mathrm{e}^{p_{\text{G}}^{\text{C}}M(s-1)}\Phi_{\text{Y}}\left(\mathrm{e}^{p_{\text{G}}^{\text{C}}M(s-1)}\right)}{\left(1-\left(1-(1-p_{\text{L}}^{\text{C}})^{m}\Phi_{\text{Y}}\left((1-p_{\text{L}}^{\text{C}})^{m}\right)\right)\mathrm{e}^{p_{\text{G}}^{\text{C}}M(s-1)}\Phi_{\text{Y}}\left(\mathrm{e}^{p_{\text{G}}^{\text{C}}M(s-1)}\right)\right)^{2}}; \tag{16b}$$

$$R_{*}^{\text{C}} = p_{\text{G}}^{\text{C}} M\left(1+\mathbf{E}(Y)\right)\left(\frac{2}{(1-p_{\text{L}}^{\text{C}})^{m}\Phi_{\text{Y}}\left((1-p_{\text{L}}^{\text{C}})^{m}\right)}-1\right). \tag{16c}$$

Numerical solutions for our application show that this approximation slightly overestimates the probability of an outbreak in most cases. Full expressions (13)–(15) have been used for all results presented.

We note that the above model does not allow for infection to re-enter a cage. Inclusion would increase model complexity while changes to outbreak probabilities in our application are likely small; nevertheless, allowance for re-infection would improve insight into the spread dynamics and risk associated with caged systems.

Symbol	Value	Description and source
η	0–0.00001	Avian influenza introduction rate (per generation) [11, 17]
g	3.5	Generation interval for avian influenza (days) [7, 9, 16]
$\mathbf{E}(\Lambda)$	0.5–0.9	Expected within-shed transmission rate per generation [16]
M	10 000–20 000	Number of birds in a shed [24]
m	1–10	Number of birds in a cage [24]
L	80–100	Number of birds in a 'mixing group' within a large shed [22]
$p_{\mathrm{L}}^{\mathrm{B}}$	$0.95\lambda/L$	Probability of direct contact within a barn
$p_{\mathrm{G}}^{\mathrm{B}}$	$0.05\lambda/M$	Probability of indirect contact within a barn
$p_{\mathrm{L}}^{\mathrm{C}}$	$\approx 0.95\lambda/m^*$	Probability of direct contact within a cage
$p_{\mathrm{G}}^{\mathrm{C}}$	$0.05\lambda/M$	Probability of indirect contact within a caged shed
$\hat{p}_{\mathrm{G}}^{\mathrm{C}}$	$\approx 0.95\lambda/m^*$	Probability of direct contact between birds in adjacent cages

Table 2: Parameter values and plausible intervals for commercial chicken farms in Australia. *This probability is bounded above by one.

5 Model parameters

The commercial chicken industry has five main sectors (barn meat, free-range meat, barn layer, free-range layer and caged layer sectors), each with different housing systems. Using data from the literature and the Australian industry, we compare the effect of these distinct population structures on the probability of a LPAI outbreak. Layer sheds are assumed to have a production cycle length greater than 360 days, which means that sheds are not emptied and completely cleaned during that period. Meat sheds, in contrast, have a cycle length of 49 days. Birds are kept for 7 weeks, after which the shed is fully cleaned and remains empty for 2 weeks before new stock arrive. In Australia, approximately 64% of farms are barn meat, 14% are free-range meat, 2% are barn layer, 9% are free-range layer, and 11% are caged layer [20]. We adopt the same model (Section 4.1) for free-range and barn sheds and assume that wild birds have contact with both barn and free-range flocks, but that free-range sheds typically have a higher risk of disease introduction [6, 24]. Model parameters are listed in Table 2.

LPAI viruses are prevalent in Australian wild birds, but rates of LPAI introduction and transmission have not been measured for Australian commercial flocks. We model introduction as $B \sim \text{Poisson}(\eta)$, where introduction is rare with an extreme upper bound for η based on data from sentinel free-range layer flocks in Australian wetlands [11]. Since introduction is rare, a first order approximation provides a reasonable estimate of how introduction affects results. Free-range sheds are assumed to have higher LPAI introduction probabilities due to an increased probability of direct contact between poultry and wild birds. The extent of this increase in free-range sheds has been estimated (measured) at 10–fold for the Netherlands [17]; a 3.5–fold increase is inferred from an Australian survey [24]; and a 2–fold increase reported for New Zealand [21] and Belgium [28]. Here we assume a 3.5–fold increase, although we recognise this may vary seasonally and by location, and explore variation. We assume a mean reproduction number for LPAI viruses that is less than one [11, 13, 16, 17, 18, 27], but model a distribution about this mean (Section

3) and provide results for a range of values. We assume that indirect transmission within sheds accounts for, on average, 5% of total within shed transmission for all shed types, but that the probability of direct transmission between birds in the same cage is greater than between two birds in the same local group within a barn or free-range shed (Table 2).

6 Results

Figure 2 demonstrates the importance of incorporating virus heterogeneity into the model, particularly for H5 and H7 LPAI viruses which, more often than not, are unable to spread in domestic poultry, and thus the expected transmission rate of introduced viruses is likely below one [13, 16, 18]. Modelling 1000 barn-layer sheds over one year, there is a relatively slow increase in the probability of an outbreak as the mean transmission rate increases in the model that includes a distribution (Figure 2, black curve), compared with a sharp increase in this probability when there is no underlying distribution for the transmission rate (Figure 2, grey curve). For LPAI introduction to commercial poultry, where viruses are highly diverse with an expected transmission rate below 1, this fundamental difference in the effect of the transmission rate is particularly pertinent.

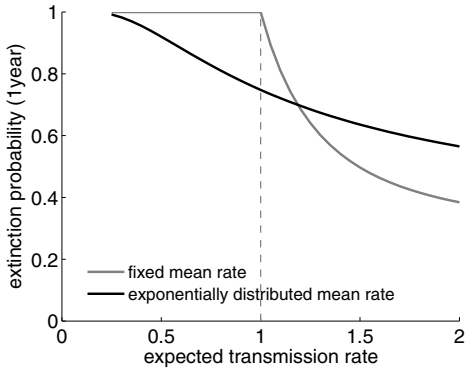

Figure 2: The probability there is no LPAI outbreak across 1000 barn-layer sheds over 1 year for two scenarios: a fixed within-flock transmission rate (grey curve), or a transmission rate sampled from an exponential distribution with the same mean (black curve).

Small shed sizes are a specific characteristic of particular sectors. We investigate their impact, together with a variable transmission rate, on the probability of a LPAI outbreak as the expected transmission rate varies (Figure 3(a)) and as the probability of infectious contact with wild birds increases (Figure 3(b)). We model 1000 sheds over one year with stochastic virus introduction. These results illustrate the nonlinear effect of shed size, which is most pronounced when shed sizes are reduced from 20 000 to below 10 000, with smaller enterprises having a greatly reduced outbreak probability. Most shed sizes in the Australian industry are marginally above 20 000 individuals, with the exception of free-range layer sheds which, typically, house around 10 000 birds. This result demonstrates that, for the Australian industry, the increase in range access for free-range layers may be offset by smaller shed sizes — particularly when these fall below 10 000 birds.

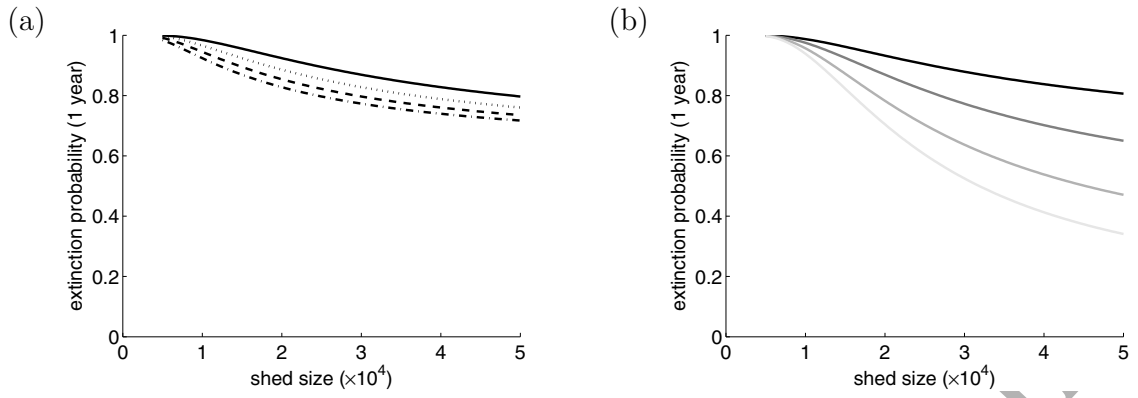

Figure 3: The effect of shed size on the probability there is no LPAI outbreak across 1000 sheds over 1 year in a barn-layer system, with transmission rates sampled from an exponential distribution and including stochastic introduction ($\eta = 0.5 \times 10^{-5}$):
(a) As expected transmission rate varies, with values of the mean probability of direct contact in the barn 2.5×10^{-5} (solid lines), 3.75×10^{-5} (dotted lines), 5.0×10^{-5} (dashed lines) or 6.25×10^{-5} (dash-dotted lines);
(b) As outdoor access, as in free-range layer systems, increases the probability of infectious contact with wild birds by a factor of 2 (dark-grey), by a factor of 3.5 (mid-grey) and by a factor of 5 (light grey), with $\mathbf{E}(\Lambda) = 0.7$.

Figure 4 directly compares the effect of caged-layer and barn-layer population structures on the probability of extinction, assuming a single initial infected bird and a fixed transmission rate. As expected, results indicate that housing poultry in cages with up to 10 individuals in each reduces both the probability of a major outbreak (Figure 4(a)) and the reproduction number (Figure 4(b)). Although the probability of an outbreak increases with cage size, the difference is most dramatic for small cage sizes. Nevertheless, a comparison between cages with 7 and 9 birds shows, approximately, a 3% difference in the probability of a major outbreak. The reproduction numbers for the two models are both threshold parameters, and show that outbreaks can occur for lower values of the transmission rate in the case of barn sheds. However, we note that the caged-layer reproduction number is based on transmission between adjacent clusters of infected cages while the barn reproduction number is based on transmission between infected birds, and thus the two reproduction numbers are not directly comparable, except at this threshold value.

An interesting phenomenon for biosecurity is whether a shed or sector is virus-free, particularly because LPAI outbreaks in commercial poultry may have little discernable effect on the number of eggs laid or the growth of meat birds, and so are often not easily detected. Figure 5 compares the probability of avoiding a major outbreak (Equation (4), solid curve), and the probability a shed is virus-free (Equation (5), dashed curve), for a barn-layer shed over one year for three expected transmission rates. The difference is greatest during the early weeks (5–7 weeks) and over time these differences become negligible. This follows because, initially ($\tau = 1$), $q_\tau = \Phi_B(q) > \Phi_B(0) = \widehat{q}_\tau$. Subsequently, as τ increases, both probabilities can be expressed as products, with new terms for $\widehat{q}_\tau$ quickly approaching those for q_τ (Supplementary Material Section A, Equation (3)). As a consequence, for meat sheds with a short production cycle length of 7 weeks, the difference between q_τ and

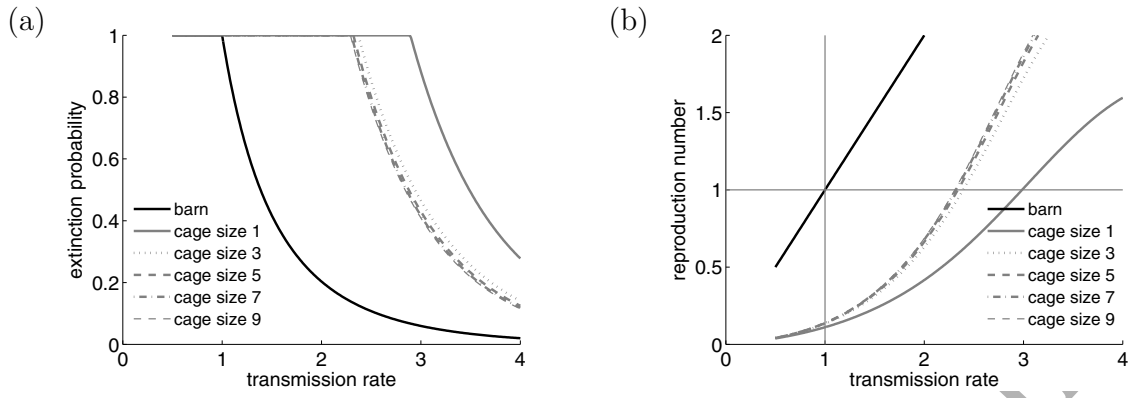

Figure 4: Comparison of a barn-layer (black) and caged-layer (grey) shed with one initial infected bird, a fixed transmission rate and no introduction, for a range of cage sizes provided in the legend.
(a) Probability of extinction as the transmission rate (λ) increases;
(b) Reproduction number as the transmission rate (λ) increases. Note that the barn and cage reproduction numbers are threshold parameters so are directly comparable at one, but are calculated differently so cannot be compared elsewhere (see text).

$\widehat{q}_\tau$ is relatively large. It is conceivable that, across an industry with more meat than layer sheds, there may be a greater probability of virus being present in the meat sector than the layer sector, while a major LPAI outbreak is more likely in the layer than the meat sector. We note that the curves in Figure 5 flatten and differences are less pronounced as introduction rate (η) decreases, but qualitative results remain unchanged.

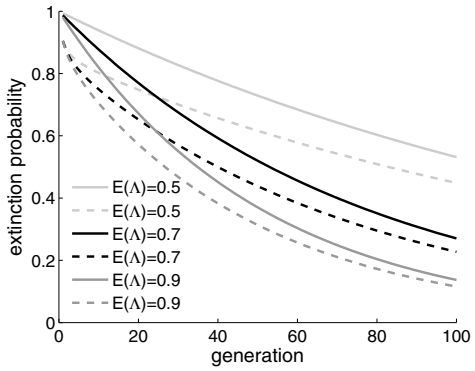

Figure 5: Comparison between the probability of extinction (q_τ — solid curves) and the probability a shed is virus-free ($\hat{q}_\tau$ — dashed curves), for a range of expected transmission values $\mathbf{E}(\Lambda) = 0.5$, 0.7 or 0.9, and introduction rate $\eta = 0.0001$.

In Figure 6 we consolidate our results and consider the probability of an outbreak by sector and across an entire industry. Stochastic introduction is incorporated, as is a distribution for the reproduction number and the specific population structures and shed sizes of each sector. Results are provided over a year so that the effect of production-cycle length, shed size and free-range access can be appropriately assessed. In Figure 6(a), we compare 1000 sheds of the same size (20 000 birds) and of each type to isolate the effect of housing

characteristics. As expected, free-range access is, by far, the most influential driver of LPAI introduction and circulation, and barn meat farms show a lower risk than barn layer farms, reflecting the effect of a reduced production cycle length on LPAI risk. Figure 6(b) includes shed size (10 000 for free-range layers, 20 000 for the others [24]) and the relative size of different sectors (Section 5). Here, the barn meat sector has a greater risk of LPAI introduction than other sectors, despite lower risk per individual shed than either barn-layer or free-range layer. Our investigations show that these results are qualitatively unchanged as the probability of infectious contact with wild birds in free-range systems increases to 5-fold that for barn systems, which is the upper 90% confidence limit reported in the Australian survey [24].

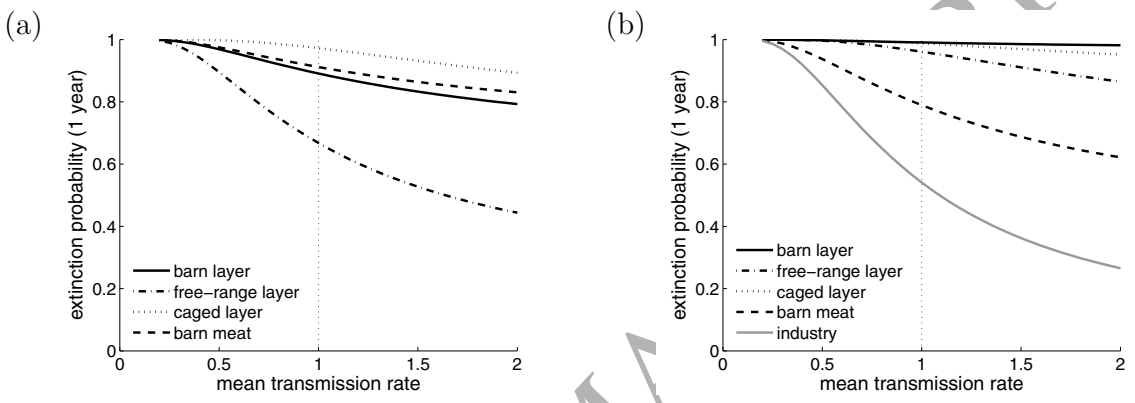

Figure 6: Comparison of LPAI extinction probabilities by sector and their relative contribution to industry-scale risk, assuming stochastic introduction with $\eta = 0.5 \times 10^{-5}$. (a) Probability that there is no LPAI outbreak in 1000 sheds of each type over one year. (b) Probability that there is no LPAI outbreak in 1000 sheds chosen according to the proportion of each type and size in the Australian industry (grey curve) together with the individual components of this probability by shed type. Sector proportions and parameter values are given in Section 5.

7 Conclusions

We present a stochastic model, motivated by current biosecurity concerns and appropriate for assessing the relative risks of low pathogenic avian influenza outbreaks in poultry sectors, and the industry. This model includes several novel features: distinct housing structures together with variation in virus transmission rates and a continuous low-level risk of virus introduction. Our findings show that free-range access is the most influential driver of LPAI introduction and circulation, that production cycle lengths (meat versus layer) have relatively little effect on risk, and that large numbers of barn meat sheds in the Australian industry make this sector the most likely to experience LPAI introduction. We also show how virus heterogeneity affects LPAI outbreak probabilities, and its importance to this application in particular. We establish conditions for which shed size can temper the effect of an increased proportion of free-range farms on risk, even though the probability of LPAI introduction increases with outdoor access. Further, we provide a means of assessing

when a shed or farm is virus-free and establish how this differs from the probability of avoiding an outbreak.

In future work we will extend the models presented here to include virus mutation with the aim of understanding risks of highly pathogenic avian influenza (HPAI) outbreaks in poultry. We expect several factors — including production cycle length — to influence these risks, so that risks associated with LPAI outbreaks identified here are not necessarily those associated with HPAI outbreaks.

Biosecurity guidelines are often required in situations where data are few, and robust distributions for influential parameters are unavailable, as is the case for low pathogenic avian influenza. By formulating explicit and tractable statistical distributions across appropriate parameter intervals, our modelling approach provides an overview of the interaction between key drivers and facilitates a broad interpretation. This is particularly appropriate when establishing general biosecurity principles for commercial industries.

The model framework formulated is also highly relevant to other industries. For example, disease introduction in the pork industry, where there is also an increasing demand for free-range produce, can occur via feral pigs and goats. Pet boarding kennels house animals in adjacent pens with a continuous low-level risk of disease introduction through the arrival of new pets each day. By developing models that capture disease introduction, we can identify broad characteristics of populations — such as rates of introduction, population turn-over, and internal population structures — that drive disease risks.

Acknowledgements

We would like to acknowledge valuable suggestions and guidance from the project Steering Committee, including representatives from the Australian poultry industries and the Australian Wildlife Health Network, during the preparation of this manuscript. This project was supported financially by the Poultry CRC.

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