Outbreaks of H5N1 in poultry in Thailand: the relative role of poultry production types in sustaining transmission and the impact of active surveillance in control

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H5N1, highly pathogenic avian influenza, continues to pose a public health risk in the countries of southeast Asia where it has become endemic. However, in Thailand, which experienced two of the largest recorded epidemics in 2004–2005, the disease has been successfully reduced to very low levels. We fitted a spatio-temporal model of the spread of infection to outbreak data collected during the second wave of outbreaks to assess the extent to which different poultry types were responsible for propagating infection. Our estimates suggest that the wave of outbreaks would not have been possible without the contribution of backyard flocks to the susceptibility of a sub-district. However, we also estimated that outbreaks involving commercial poultry, a much larger sector in Thailand than in neighbouring countries, were disproportionately infectious, a factor which was also crucial in sustaining the wave. As a result, implemented measures that aim to reduce the role of commercial farms in the spread of infection, such as the drive to bring aspects of the supply chain ‘in house’, may help to explain the subsequent success in controlling H5N1 in Thailand. We also found that periods of active surveillance substantially improved the rate of outbreak detection.

Keywords: H5N1 avian influenza; poultry production systems; mathematical modelling; Bayesian data augmentation; risk maps

1. INTRODUCTION

The strains of highly pathogenic avian influenza H5N1 currently circulating in various regions of the world, including much of southeast Asia, continue to cross the species barrier from poultry, causing severe disease in humans which often proves fatal [1]. Various strategies aimed at preventing and mitigating infection within poultry have been developed. The application of these measures and the relative success they have had in terms of providing sustained reductions in the prevalence and incidence of outbreaks or human infections have varied sharply between different countries [2–4].

Thailand’s experience of H5N1 is unique within southeast Asia in several ways, with the most immediately apparent difference compared with neighbouring countries being the eventual level of control achieved. During 2004 and 2005, large waves of outbreaks comparable with those occurring in other countries in the region such as Vietnam and China spread throughout Thailand [5,6]. Unlike every other country that experienced outbreaks on such a large scale, outbreaks of H5N1 in Thailand subsequently fell to very low levels and, since 2006, no human cases have occurred [7]. By contrast, in countries such as China, Indonesia, Egypt and Vietnam, the disease remains entrenched despite evolving control strategies, including pro-active vaccination campaigns [8], which have had varying degrees of success [4,9,10]. As a result, in order to contribute to our understanding of the factors that can aid the control of current and future epidemics in the region, it is important to evaluate the factors that contributed to Thailand’s relative success in reducing the transmission of H5N1.

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One important difference between Thailand and the countries mentioned above is the fact that vaccination of poultry against H5N1 has been prohibited in Thailand. Instead, a policy of 'stamping out' was pursued, involving the culling of all birds within a 5 km radius of a reported outbreak and the destruction and disinfection of all contaminated materials and housing, as well as the implementation of stringent movement restrictions and a campaign to restrict chicken production to closed farms [11]. These policies aimed to enhance biosecurity and prevent the circulation of infection in smallholder flocks [12], the main flock type reported as infected in the recorded outbreaks [13].

Another defining feature of the Thai response to the second wave of outbreaks was the deployment of rounds of intensive active surveillance, known as 'X-ray' campaigns, where inspectors throughout the country travelled from door-to-door looking for evidence of infection. This may have increased the extent and the rate to which outbreaks were detected.

Also, the system of poultry production in Thailand differs markedly from that of neighbouring countries. Rather than a system where the majority of poultry are raised extensively by smallholders and consumed locally (both in terms of the number of poultry produced and income generated), production is dominated by relatively few large-scale industrial integrators [14]. These industrial producers were also severely affected by the onset of H5N1 because of the negative effect this had upon Thailand's large export market. As a result, there was an intensification of the ongoing trend towards vertical integration, whereby aspects of the supply chain such as hatching and slaughtering are brought 'in-house' in an effort to reduce the risk of infection and thus improve access to overseas markets. The extent to which the measures to improve biosecurity in either of these sectors were responsible for achieving the level of control that occurred in Thailand can only be assessed accurately if the extent to which they were responsible for sustaining transmission during the initial waves of outbreaks is known. It has previously been estimated that the overall odds of an outbreak being reported within the commercial layer and broiler flocks in many areas of Thailand, particularly in central provinces where the majority of outbreaks were reported [15]. While this estimate may be affected by reporting biases within different poultry production sectors, it does highlight the fact that the available data do not necessarily suggest that the epidemic was entirely driven by infection within smallholder flocks. Moreover, to date, no attempt has been made to quantify the respective roles of these two sectors in propagating the spread of infection directly.

In this paper, we attempt to quantify the spatial dynamics of the risk of transmission during the spread of H5N1 during the second wave of outbreaks in Thailand, occurring between mid-2004 and early 2005, and the respective roles of commercial and backyard chickens, as well as those of ducks and geese. A spatio-temporal mathematical model is fitted to the available outbreak report data, taking into account the distribution of poultry within the country and the uncertainty with regards to the unobserved times at which outbreaks initially occurred. The model uses detailed information on the composition of poultry production at a sub-district (otherwise known as ‘tambon’) level, collected during the X-ray campaigns. The model allows us to construct maps of the spatial distribution of the risk of transmission between tambons and to estimate the roles played by each poultry sector in propagating the wave. Moreover, by assessing whether there were any changes in the estimated duration of time taken to report an outbreak, we investigate whether periods of ‘X-ray’ surveillance were successful in increasing the extent to which outbreaks were detected and discuss the extent to which using outbreak data collected from different surveillance systems may affect our results.

2. MATERIAL AND METHODS

2.1. Description of data

Spatial information in the form of a shape-file with the administrative boundaries at the tambon level was obtained from the Geo-informatics and Space Technology Development Agency and the Department of Land Development, Thailand. Data on the outbreaks and poultry population were obtained from the Department of Livestock Development.

Poultry census data were collected as part of the first ‘X-ray’ survey between October and mid-November 2004. The data included the number of backyard chickens, broilers, layers, quails, turkeys, geese, meat and free-grazing ducks, and the number of flocks of each type within each tambon.

Data on H5N1 outbreaks reported between July 2004 and November 2005 were compiled by the Department of Livestock Development and comprise the date at which the outbreak was reported, the tambon within which the outbreak was detected, and the species and production type of the flock in which disease was detected.

2.2. Models of the spread of infection

In the absence of spatial information about the location of outbreaks within each tambon and the number of poultry involved in each outbreak, the dynamics of infection within the tambon were ignored. Instead, our analysis of the wave focused upon the dynamics of infection between tambons, with the location of each outbreak calculated as the tambon centroid. This infection process was modelled as a space–time survival process [16,17], with the force of infection exerted by an infectious tambon $j$ upon a susceptible tambon $i$ defined as:

$$\lambda_{ij} = s_i y_j k^*(d_{ij}).$$

Here $s_i$ and $y_j$ are, respectively, data-derived measures of the susceptibility of tambon $i$ and of the infectivity of tambon $j$, $k^*(d_{ij})$ is a normalized version of the following spatial kernel:

$$k(d_{ij}) = \left(1 + \frac{d_{ij}}{\rho}\right)^{-a},$$
which describes how the risk of transmission scales with distance, where \( d_{ij} \) is the distance between tambons \( i \) and \( j \), and the power \( \alpha \) and offset \( \rho \) are parameters to be estimated. Fitting a model using a Euclidean spatial kernel such as this to the spread of infection throughout all of Thailand would involve measuring transmissibility between country borders and over long stretches of ocean. Therefore, in order to produce a more convex area, the study region was confined to the 6403 northernmost tambons. As a result, outbreaks and poultry data in Southern Thailand were not included in the analysis.

Initially, all heterogeneity in the type of poultry production was ignored and the susceptibility measure of an uninfected tambon was calculated as \( s_i = n_r \) the total number of poultry within the tambon. In a similar manner, the infectivity measure of an infectious tambon was calculated as:

\[
y_i = \psi_f.
\]

This is the product of \( \psi \), a transmissibility parameter to be estimated, and the number of outbreaks reported in an infected tambon \( i \), \( f_i \).

Subsequently, in order to incorporate heterogeneity in the susceptibility and infectivity of different types of poultry, outbreak reports and poultry population data were disaggregated into three separate production types:

(i) commercial chickens;
(ii) ducks and geese (classed together because of their similar methods of production [15]); and
(iii) backyard chickens and any other species including wild birds.

Using \( n^k_i \) to denote the number of poultry within tambon \( i \) for each production type, \( k = 1, 2, 3 \), the susceptibility of tambon \( i \) was calculated as:

\[
s_i = \varphi^1 n^1_i + \varphi^2 n^2_i + n^3_i,
\]

where \( \varphi^1 \) and \( \varphi^2 \) are parameters determining the relative susceptibility of commercial chickens, and ducks and geese with that of backyard chickens. Letting \( \psi_f^k \) and \( f_i^k \) denote, respectively, an infectivity parameter and the number of outbreaks of production type \( k \) within the infected tambon \( i \), the infectivity of this tambon is given by:

\[
y_i = \sum_{k=1}^{3} \psi_f^k = \sum_{k=1}^{3} \psi_f^k f_i^k.
\]

### 2.3. Fitting the model to outbreak report data

To fit the models to the observed outbreak data, we require estimates of the times at which tambons were infected, the times they became infectious and the times at which they were removed from the wave. The removal time of each outbreak, \( R = \{ r_i; r \leq N \} \), was determined by the date on which the outbreak was reported, with outbreaks assumed to have been controlled one day after it had been reported. However, the observed data provide little information about the times at which tambons became infected. Working within a Bayesian framework, these unobserved times of infection, \( U = \{ u_i; u \leq N \} \), were treated as additional nuisance parameters to be imputed according to a Gamma-distributed infection-to-report duration, \( (R - U) \sim \Gamma(\varphi, \gamma) \).

For both the homogeneous and production-type specific models, the overall force of infection experienced by tambon \( i \) at time \( t \) can be calculated as:

\[
\lambda_i(t) = \sum_{j=0}^{N} \lambda_{ij} \mathbb{I}(j \text{ is infectious at } t),
\]

where \( \mathbb{I}(.) \) is the indicator function. Based upon a 24 h incubation period of individual birds [18], tambons were assumed to become infectious on the day following infection. Given the history of the wave and the associated accumulated exposure to infection \( \Lambda(t) = \int_{t_0}^{t} \lambda_i(r) \, dr \), the probability that a tambon escapes infection until time \( u_i \) is:

\[
g_i(u_i) = \exp(-\Lambda(u_i)).
\]

Conditional on having avoided infection to day \( u_i \), the probability that tambon \( j \) becomes infected on day \( u_i \) is then:

\[
p_i(u_i) = 1 - \exp(-\Lambda(u_i)).
\]

Using these probabilities, the likelihood of the overall infection process can be calculated for any set of model parameters and infection times. This likelihood was explored using a two-step Markov chain Monte Carlo (MCMC) algorithm. A first step comprised the imputation of the unknown infection times, followed by a second step updating each of the model parameters to obtain the posterior distribution of each of the model parameters. Full details of the MCMC algorithm are given in the electronic supplementary material.

Using data-augmentation techniques [19,20] to avoid directly integrating the entire likelihood over possible dates of infection makes it difficult to calculate established measures of model fit such as the deviance information criterion [21,22]. Instead, we adopted an approach formulated by Cauchemez et al. [23], comparing the ‘reconstructed’ predicted distribution of the times of infection on each day of the wave with the ‘expected’ next-step-ahead predictions based upon the predicted distribution of infection times prior to that day. This can be used to assess whether, for any of the models, there are any stages of the wave where the level of transmission observed in the data is inconsistent with that predicted by the model. Further details of this method are provided in the electronic supplementary material.

### 2.4. Calculating the role of each poultry type in sustaining transmission

Once the poultry type-specific transmissibility parameters have been fitted to the outbreak data, the relative roles of each production type in driving transmission during the wave can be investigated by reconstructing the epidemic tree. From a given set of infection times, the probability (conditional on these
infection times, the observed outbreak data and values of the other model parameters) that tambon $i$ is infected by infectious poultry of type $m$ in tambon $j$ is:

$$\mu_{ij}^m = \frac{y_j^m s_1(u_j < u_i)1(r_j \geq u_i)}{\sum_{k=0}^{N} y_k s_1(u_k < u_i)1(r_k \geq u_i)},$$

where $N$ is the number of tambons within the dataset. These type-specific infection probabilities can then be summed-up to obtain the number of tambons infected by tambon $i$, which is the result of infected flocks of production type $m$:

$$T_i^m = \sum_{j=0}^{N} \mu_{ij}^m.$$

Here $\overline{\mu_{ij}^m}$ is the posterior mean of $\mu_{ij}^m$ that can be approximated by repeatedly drawing samples of the model parameters and infection times at regular stages (in this analysis every 50 iterations) of the MCMC sample.

2.5. Calculating risk maps

The local reproduction number of a tambon estimated from an outbreak wave is defined as the number of secondary infections which could have been expected to occur had the first outbreak of the wave occurred in that particular tambon, assuming that all other tambons are susceptible to infection. Within a large population, where it can be assumed that there are no local saturation effects caused by tertiary infection, this can be calculated by summing-up the probabilities that a given tambon infects each other tambon to produce the total expected number of secondary infections. It should be noted that in this analysis, owing to the spatial dependence between infection events, such an assumption is not valid. However, summing-up such probabilities has become an accepted measure of assessing regions most at risk of sustained transmission [24–26]. When the infectivity kernel, the distribution of the infectious period $T$, and the number and composition of outbreaks within an infected tambon are available, such estimates can be obtained analytically [25] using the expression:

$$R_i = E\left[\sum_{j \neq i} p(d_{ij})\right] = \sum_{j \neq i} \{1 - E[e^{-\lambda_j T_i}]\} = \sum_{j \neq i} \{1 - M_T(-\lambda_j)\}.$$

Here $p(d_{ij})$ is the probability that transmission occurs between an infectious and susceptible tambon $d_{ij}$ kilometres apart before the outbreak is removed and $M_T(x)$ is the moment-generating function of the probability distribution of the infectious period. Thus, using the posterior distribution of the model parameters, it would be possible to obtain the posterior distribution of each tambon-level local reproduction number, and plotting these on a map would provide an indication of the geographical areas that are most at risk of experiencing sustained inter-tambon transmission in the event of an outbreak.

However, in this analysis, as the intra-tambon dynamics of infection are not explicitly modelled, it is not possible to generate $\lambda_j$ based solely on the obtained model parameters. Therefore, as a typical outbreak could not be defined if there is heterogeneity in transmission by poultry type, the fitted parameters for the homogeneous model were used to provide an estimate of the spatial distribution of the general risk of onward transmission from a single outbreak of any poultry type within an infected tambon. The fitted parameters from the heterogeneous model were then used to generate poultry type-specific risk maps on the basis of a single outbreak within the poultry type in question. It should be emphasized that these maps only provide an indication of spatial transmissibility in terms of the expected number of secondary tambons infected as a result of the specific outbreak composition being considered and do not take into account the number of outbreaks and types of flock affected when a given tambon becomes infected, which is likely to be determined by various factors including the composition, location and density of poultry production within a tambon and the application of control measures, as well as stochastic effects. As a general rule, the expected number of secondary infections would rise with an increasing number of reported outbreak flocks within a tambon, however, this relationship would be nonlinear and would saturate because of the effects of depletion of local susceptibility.

2.6. Investigating the effects of ‘X-ray’ surveillance

The stated aim of ‘X-ray’ surveillance is to provide a cross-sectional view of the H5N1 status of poultry throughout Thailand. This included sampling all sick poultry and assessing every household for poultrydie-offs to increase the likelihood that cases were detected. As a result, one measure of whether this policy was effective is likely to be the speed at which outbreaks were reported following the introduction of infection. To investigate this, the models were additionally fit using two separate Gamma-distributed infection-to-report distributions, one to outbreaks reported during periods, where ‘X-ray’ surveillance was taking place (1–31 October 2004 and 1–28 February 2005) and a second to outbreaks reported outside these periods.

3. RESULTS

Within the region analysed, outbreaks were predominately concentrated in the centre of Thailand in the vicinity of, and within a strip running north from, Bangkok (figure 1a). These are the areas of the country where the majority of poultry production takes place [13]. There was also some sporadic infection, generally involving backyard chickens, reported within the eastern Khon Kaen plateau. In total, the wave lasted for approximately 10 months, with the majority of reported outbreaks occurring between September and December 2004 (figure 1b).

When the models were fitted to these data, both the homogeneous and production type-specific models
estimated a mean duration of approximately 8.5 days between a tambon becoming infected and the initial report of an outbreak (table 1). The risk map of local reproductive numbers calculated from a single infected flock obtained using the fitted parameters of the homogenous model demonstrates that it was possible to qualitatively reproduce the observed patterns of transmission (figure 2a). Moreover, as the average number of reported outbreaks within an infected tambon during the outbreak wave was 1.7 and could be as many as 14, in many cases, the number of secondary between-tambon infections arising from an infected tambon could be expected to be greater than those illustrated in this map, which only shows the expected number of secondary infection arising from a single outbreak. However, the qualitative pattern of transmission risk estimated by the model would only appear to be likely to change in the seemingly counterintuitive case that the number of outbreaks in an infected sub-district increases in areas with a low density of poultry.

The estimates of transmissibility parameters obtained from fitting the model which incorporated heterogeneity in poultry production-type suggest that, per bird, ducks and geese and, in particular, commercial chickens contribute much less to the susceptibility of a tambon to infection (although the respective distribution of flock sizes would mean that the flock-level contribution to the susceptibility of infection of a tambon was still higher for many larger commercial and duck or goose flocks (figure 1c)). This result was robust to the choice of relative susceptibility used before (see the electronic supplementary material, table S2). By contrast, outbreaks involving commercial chicken flocks within a tambon were estimated to be approximately twice as transmissible to neighbouring tambons as those involving backyard chickens (table 2). As a result, according to the reconstructed epidemic tree, although the majority of between-tambon transmission occurred as the result of outbreaks involving backyard chickens, the number of transmissions per outbreak was higher for outbreaks involving commercial chickens. Commercial chickens were also the only production type where the number of transmissions per outbreak was greater than unity and,
when considered on the basis of the total number of flocks within the region, by far, the production type most likely to transmit infection (table 3).

The estimated disproportionate role of commercial chickens can also be observed when examining the production type-specific risk maps which suggests that outbreaks involving commercial chickens in Central Thailand, the region of Thailand where the majority of large-scale commercial production takes place, were highly likely to result in the transmission of infection to neighbouring tambons (figure 2c). This map also highlights the Eastern region as an area at high risk of infection, however, as very few of the outbreaks occurring in this region involved commercial poultry (figure 1a), the level of risk of transmission from such an outbreak is likely to have had little bearing upon the progress of the wave.

By contrast, the risk map for outbreaks involving backyard chickens suggests that, with the exception of a single hotspot in Central Thailand, the number of expected transmissions from such an outbreak did not exceed unity in any region (figure 2b) and the risk map for outbreaks involving ducks and geese did not contain a single tambon, where the expected number

Table 1. Posterior mean and 95% credible intervals for each of the fitted models. (For production type-specific model, values shown are from the model fitted using baseline priors.)

<table>
<thead>
<tr>
<th>parameter</th>
<th>homogenous model</th>
<th>production-type model</th>
</tr>
</thead>
<tbody>
<tr>
<td>kernel power</td>
<td>2.2 (2.0–2.5)</td>
<td>2.7 (2.5–3.0)</td>
</tr>
<tr>
<td>kernel offset</td>
<td>6.6 (3.6–10.8)</td>
<td>8.5 (5.1–13.1)</td>
</tr>
<tr>
<td>infectivity</td>
<td>23.9 (21.5–26.7)</td>
<td>see table 2</td>
</tr>
<tr>
<td>mean infection to report duration</td>
<td>8.6 (6.8–10.1)</td>
<td>8.5 (7.6–9.4)</td>
</tr>
<tr>
<td>infection to period s.d.</td>
<td>5.0 (4.4–5.8)</td>
<td>5.3 (4.6–5.9)</td>
</tr>
</tbody>
</table>
of tambons infected during such an outbreak exceeded 0.5 (data not shown), suggesting that, in the absence of outbreaks involving commercial poultry, the ability of the wave to spread would have been severely limited. Similarly, preventing backyard poultry from contributing to the susceptibility of a tambon would also appear likely to be effective at averting sustained between-tambon transmission (figure 2d).

Considering the number of days tested and the fact that, for each day, the reconstructed number of infections fall within the 75 per cent interval of the predictive distribution, the imputed epidemic curves from both fitted models appear to be well matched to the next-step-ahead distribution (figure 3). Moreover, looking at the reconstructed chain of transmission throughout the wave, it is interesting to note that, both before and during the first ‘X-ray’ campaign, commercial chickens are the only production type estimated to be consistently causing more sub-district level infections than they are involved in (figure 4). This measure of onward transmission does, however, peak immediately before the beginning of the first period of active surveillance, which may be an indication that the model does have to overcompensate for the level of infection reported during this period (figure 4b).

The results from fitting a separate infection-to-report distribution to outbreaks reported during periods of ‘X-ray’ surveillance suggest that such measures were successful in enhancing the reporting of outbreaks. When carried out for the poultry type-specific model, this resulted in an estimated infection-to-report duration of 5.8 days for outbreaks reported during an ‘X-ray’ period and 10.8 days for outbreaks reported outside this (figure 5). This would suggest that the reporting of outbreaks was improved as a result of such active surveillance, probably as a result of a combination of detecting outbreaks more rapidly than those reported as a result of the existing surveillance structure and by combating any under-reporting of outbreaks. The latter factor would mean, however, that it is not possible to make the simple conclusion that ‘X-ray’ surveillance reduced the mean time between infection and report by 5 days as the under-reporting occurring outside ‘X-ray’ is likely to lead to an overestimation of the time to report during these periods. This is because, when fitting such a model to an epidemic where outbreaks have been under-reported, the fitting algorithm is forced to join outbreaks occurring further apart in time [27].

### Table 2. Production type-specific transmissibility parameters.

<table>
<thead>
<tr>
<th>parameter</th>
<th>description</th>
<th>posterior mean (95% credible intervals)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \phi^1 )</td>
<td>infectivity of commercial chicken outbreaks</td>
<td>101.9 (75.7–131.0)</td>
</tr>
<tr>
<td>( \phi^2 )</td>
<td>infectivity of duck and goose outbreaks</td>
<td>9.5 (4.0–16.6)</td>
</tr>
<tr>
<td>( \phi^3 )</td>
<td>infectivity of non-commercial chicken outbreaks</td>
<td>44.3 (38.1–51.1)</td>
</tr>
<tr>
<td>( \psi^1 )</td>
<td>susceptibility of commercial chickens relative to non-commercial chickens</td>
<td>0.005 (0.002–0.01)</td>
</tr>
<tr>
<td>( \psi^2 )</td>
<td>susceptibility of ducks and geese to non-commercial chickens</td>
<td>0.16 (0.12–0.22)</td>
</tr>
</tbody>
</table>

### Table 3. Number of transmissions by production type according to the reconstructed epidemic tree.

<table>
<thead>
<tr>
<th>source of infection</th>
<th>commercial chickens</th>
<th>ducks and geese</th>
<th>backyard</th>
</tr>
</thead>
<tbody>
<tr>
<td>total number of transmissions*</td>
<td>282.92</td>
<td>86.20</td>
<td>578.9</td>
</tr>
<tr>
<td>transmissions per outbreak</td>
<td>1.63</td>
<td>0.20</td>
<td>0.60</td>
</tr>
<tr>
<td>transmissions per flock (( \times 10^3 ))</td>
<td>3.33</td>
<td>0.13</td>
<td>0.16</td>
</tr>
</tbody>
</table>

*Values calculated using the ‘X-ray’-dependent time-to-infection distribution.

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Figure 3. Model consistency throughout the wave. Black lines show \( P^C \) the posterior probability that the ‘reconstructed’ number of infections per day are less than or equal to the number of infections predicted by the next-step ahead distribution. Grey lines show \( P^C \), the posterior probability the equivalent values are greater than or equal to the next-step ahead prediction. (a) Model where transmissibility is homogeneous with respect to poultry production type. (b) Model with production type-specific transmissibility parameters. (NB: Both lines include \( P^C \) which can be substantial when the number of infections are low.)

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The findings of this analysis demonstrate that the nature of poultry production and differences in surveillance infrastructure and policy can be important determinants for the spread of H5N1. The risk maps generated from our fitted model highlight the Central region of Thailand as an area of sustained between-tambon transmission with sporadic infection occurring in Northern and Eastern regions. However, when heterogeneity in the transmissibility of different production types was incorporated, it was found that although, per bird, backyard chickens contribute more to the susceptibility of a tambon to infection, outbreaks involving commercial chickens were substantially more infectious to neighbouring tambons. This resulted in a wave where, as these were the predominant type of poultry involved in outbreaks, the majority of between-tambon transmission occurred as a result of outbreaks in backyard chickens but where commercial chickens played a disproportionate role in the spread of infection. This finding may support the notion that once infected, large-scale commercial operations can spread infection more effectively over longer distances [28,29] and supplements previous analyses which have found that the overall odds of infection are higher within commercial premises [15].

Moreover, the poultry type-specific risk maps obtained from this analysis appear to suggest that the interaction between poultry production types was crucial in propagating the spread of infection, suggesting that outbreaks in backyard chickens, ducks and geese would be unable to sustain between-tambon transmission without outbreaks in commercial chickens. Similarly, if backyard poultry were prevented from contributing to the susceptibility of a tambon to infection, the wave would not have been able to spread.

The finding that, per bird, ducks and geese contribute less to the susceptibility of a tambon to infection than backyard chickens, contrasts with other studies which have found that the presence of free-grazing ducks is a stronger predictor of the likelihood that a tambon became infected during the wave [6,30]. This could be the result of our combining free-grazing and meat ducks, as these two production types have distinct geographical patterns but could not be distinguished within the outbreak data itself. In contrast to free-grazing ducks and backyard chickens, meat ducks have not been found to be significantly associated with the overall presence of H5N1 outbreaks in Thailand, although they have been associated with outbreaks involving only ducks and have been identified as a risk factor for infection at the farm-level in Vietnam [30,31]. This may suggest that there are further heterogeneities in the tambon-level susceptibility of infection which were not captured by the model.

Our finding that ducks and geese play only a relatively minor role in spreading infection between tambons is, however, primarily a consequence of the estimated lower between-tambon infectiousness of outbreaks involving these birds, of which there are no comparable estimates within the literature. It should also be noted that field studies have shown that ducks can act as undetected carriers of infection [32,33], which could result in their contribution in spreading the wave being underestimated. Such silent spread of infection would also affect our estimates of the spatial kernel and time-to-report distribution. There may also be issues with our decision,
because of our lack of denominator data, to include all types of poultry other than commercial chickens and ducks and geese within the ‘backyard’ category and we were not able to take into consideration any contribution of wild birds during the wave, aside from those outbreaks which were detected and included as backyard outbreaks.

Our estimates of the relative roles of the other production types may similarly be sensitive to differences in the proportion of outbreaks reported between production types. The nature and extent of any such ascertainment bias are difficult to assess. For example, the shorter infection-to-report fitted during ‘X-ray’ surveillance, where officials go door-to-door looking for signs of infection, gives a strong indication that a proportion of outbreaks in backyard flocks went undetected outside of ‘X-ray’, whereas the presence of highly pathogenic avian influenza may be more easily detected in large-scale commercial operations [29]. Looking at the extent to which our estimates change throughout the wave appears to suggest that outbreaks in commercial flocks were causing a disproportionate level of infection both before and during the first round of active surveillance, however, the fact this peaked prior to the beginning of ‘X-ray’ may be an indication that this estimate has been affected by under-reporting at this stage of the wave.

In order to retrospectively fit models which allow for the possibility that some outbreaks go unreported during an outbreak wave, data augmentation techniques can often be extended to impute unobserved infection events [19,20,34] rather than just unobserved infection times. However, for this study, the mixture of data from different surveillance mechanisms makes this challenging. First, it may be the case that the relative reporting rates in backyard and commercial operations change during such periods of active surveillance. Moreover, active surveillance of the type implemented during the ‘X-ray’ periods may also result in clusters of outbreak reports, correlated in both space and time, which could bias estimates of the infectious period and the contribution of outbreaks to other spatial infectivity parameters. Unfortunately, in our data, outbreaks detected as a direct result of this active surveillance and those reported through the existing veterinary infrastructure are not distinguishable, making it impossible to either validate any of these hypotheses or to further quantify the extent and effect of any such under-reporting. On the other hand, from the perspective of evaluating control policies, the finding of a shorter infection-to-report distribution for outbreaks reported during ‘X-ray’ surveillance does suggest that such campaigns are effective at providing surveillance capacity in excess of that achieved by relying upon the existing passive surveillance infrastructure alone.

In conclusion, the different surveillance strategies implemented during the second wave of outbreaks in Thailand mean the extent and impact of any reporting biases that are difficult to determine. This issue is likely to be a factor in any analysis conducted using these data and highlights the epidemiological importance of monitoring surveillance effort in addition to investigating for signs of disease during waves of outbreaks, which can be carried out using standardized metrics [35]. However, the results of fitting a transmission model to the available outbreak data do not support the notion that outbreaks of H5N1 were solely small-holder-driven. By contrast, our results support the notion that both commercial and backyard poultry sectors played their own distinct role in propagating the initial large-scale waves of outbreaks of H5N1 in Thailand. According to our model, such widespread infection could not be sustained without either the contribution of backyard flocks to the susceptibility of tambons or the existence of highly infectious outbreaks within commercial poultry. As a result, any successful efforts to upgrade the biosecurity of backyard poultry are likely to have had a major impact in reducing disease spread. At the same time, however, measures to prevent infection within such premises, such as the move towards vertical integration, also appear likely to have reduced transmission substantially. Although there are various other potentially significant differences between Thailand and other affected countries including the application of subsequent control measures such as vaccination, this latter factor may also partly explain why, in contrast to neighbouring countries such as Vietnam and China where intensive poultry production occurs on a much smaller scale and outbreaks continue to be regularly reported, Thailand has been able to control the spread of the disease very successfully.

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