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EFFECT OF DOMESTIC POULTRY FARM TO COMMERCIAL POULTRY FARMS TO INCREASE RISK OF INFLUENZA TO SOCIETY

Pro. Maidhura Thomson[1]; Dr. Grey Smith[2].

[1]faculty in Department of vet nary in University in Denver, Colorado, united state.

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Corresponding Author:
Pro. Maidhura Thomson[1]
[1]faculty In Department Of Geography In University In Denver,Colorado
madhu.df@gmail.com

ABSTRACT

This paper summarizes the evidence that the contribution of backyard poultry flocks to the continued transmission dynamics of an avian influenza epidemic in commercial flocks is modest at the best. Nevertheless, while disease control strategies needn't involve the backyard flocks, an analysis of the contribution of every element of the subsequent generation matrix to the essential reproduction number indicates that models which ignore the contribution of backyard flocks in estimating the hassle required of strategies focused on one host type (e.g. commercial flocks only) necessarily underestimate the amount of effort to an extent that will interest policymaker.

KEYWORDS: Avian; influenza; Model; Backyard; flocks;

INTRODUCTION

The 2003 avian influenza (H7N7) outbreak within the Netherlands involved large commercial flocks and a few backyard flocks (Stegeman et al., 2004, Thomas et al., 2005, Le Menach et al., 2006, Boender et al., 2007, Bavinck et al., 2009). However, Thomas et al. (2005) argued that the contact structure and also the small size of the backyard flocks meant that their role during this epidemic was "probably negligible". This conclusion was buttressed by a later analysis of the next-generation matrix for a two-type SEI model of a little of the outbreak, which led Bavinck et al. (2009) to conclude that "from an epidemiological perspective" backyard flocks played only a marginal role. At face value, this has obvious implications for control of avian influenza in Europe and North America and, indeed, for the architecture and data requirements of future models of this disease. as an example, Bavinck et al. (2009) suggest that "if during a future epidemic, backyard flocks appear to be less susceptible than commercial flocks, as shown in our study, preemptive culling won't be necessarily applied to backyard poultry flocks, because the probability of becoming infected appears to be much lower." While not necessarily disagreeing with Bavinck et al. (2009), it'll be argued here that it might be unwise to further conclude that we are able to ignore the contribution of backyard flocks to future epidemics whether or not they will be shown to be less susceptible than commercial flocks. This is often very true after we are trying to estimate the trouble required to curtail a pestilence using strategies directed at only 1 type (sensu Roberts and Heester beek, 2003, Diekmann et al., 2010) of the host.

This paper is going to be begun as follows. First, we shall consider the rather difficult problem of defining what we mean by "backyard flocks" within the context of business poultry operations in Western Europe and North America. Next, we shall summarize the evidence for and against the notion that backyard flocks contribute rather little to the transmission of avian influenza virus in commercial operations. Finally, we shall compare the analysis of Bavinck et al. (2009) with our own analysis of the 2004 highly pathogenic avian influenza (H7N3) outbreak that occurred within the Fraser Valley of British Columbia, Canada (Anon, 2004), and, specifically, address the question of how we will use such analyses to estimate the trouble required for targeted interventions (that is, strategies directed against commercial flocks only).

Backyard flock — definitions

One of the difficulties we've in addressing backyard flocks is that the difficulty of defining what we mean by a "backyard flock". The phrase is in common use but because the OIE (World Health Organization for Animals) points outs, there's no accepted definition (Anon, 2009). Common criteria include the quantity of birds within the flock frequently conflated with whether or not the flock is included in some register of business flocks: for instance, the Dutch Ministry of Agriculture, Nature and Food Quality defines a backyard flock as consisting of fewer than 500 birds or as not having a singular farm number (Bavinck et al., 2009); the National Animal Health Monitoring System "Poultry '04" study within the USA defined backyard flocks as residences with fewer than 1000 birds apart from pet birds (Garber et al., 2007); the Canadian Food Inspection Agency defines backyard flocks as flocks that are smaller than 1000 birds that aren't registered as commercial poultry operations (Anon, 2004). Sometimes, by default, backyard flocks are simply those flocks which due to their small size don't seem to be obligated to be recorded in national databases: models of avian influenza in Britain don't include flocks but 50 birds because these flocks are nor reported within the Great Britain Poultry Register (Truscott et al., 2007, Sharkey et al., 2008, Dent et al., 2008). Capua et al. (2002) suggest that backyard flocks should be defined as those having no functional connections with industrial establishments. If we understand the word functional to point any contact or process that would plausibly cause transmission between commercial and backyard flocks (as Capua et al. clearly intend), then obviously, by definition, flocks with no functional connection to industrial premises don't have any role within the epidemiology of transmission. But setting aside the tautology inherent the definition how would we all know that there was no functional connection? A recent study within the USA found that only 3.5% of all backyard flocks (range 0.9–8.5%) had someone within the household who worked for an advertisement poultry operation, only 2.5% of backyard flocks received veterinary care and only 2.8% of backyard flocks were vaccinated (Anon, 2005). But even for those flocks within which there have been no obvious commercial or social contacts (e.g. shared personnel, equipment, or breeding birds) we could never make sure that wind-blown virus (for example) failed to constitute a functional connection between industrial and backyard premises. within the 2004 highly pathogenic avian influenza (H7N3) outbreak that occurred within the Fraser Valley of British Columbia, Canada, not only was there epidemiological evidence of wind-borne spread of the virus but also air sampling techniques detected small quantities of wind-borne virus up to 800 m from infected premises (Power, 2005, Schofield et al., 2005).

If we were to shift to regulate strategies for avian influenza within which infected backyard flocks and their dangerous contacts were depopulated but all the remainder of the control effort (depopulation or vaccination) were focused on the commercial flocks would models that omitted backyard flocks altogether fairly represent the control effort required to curtail the outbreak? How big must be the contribution of backyard flocks to transmission before we feel obligated to incorporate them in our models? This question is addressed within the next section.

Pro. Maidhura Thomson[1; 1] *faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com

Epidemic model of the 2004 Abbotsford (H7N3) avian influenza outbreak

The first recorded outbreak of highly pathogenic avian influenza in Canada occurred near Abbotsford within the Fraser Valley, Canadian province. During the course of 91 days, 42 out of 410 commercial flocks and 11 out of 553 backyard flocks were infected (Pasik et al., 2009). Control measures included movement bans, active surveillance, and an increasingly draconian program of depopulation beginning with the depopulation of known infected farms about to the pre-emptive culling of all farms within 3, 5 or 10 km of the infected farms (depending on the date) and culminating during a strategy (beginning about 20–22 days into the outbreak) intended to depopulate all the flocks (whether infected or not) within the affected area (Anon, 2004).

Most infected flocks were detected because the results of active surveillance; some were detected because the results of responses to reports of sick birds or increased rates of mortality in flocks. The epidemic data comprise the date that the samples were taken (for PCR), the date of positive diagnosis and therefore the date of depopulation (Anon, 2004). In formulating our model, we focused on the transmission of avian influenza virus between the distinct sets of premises housing the birds. Each set of premises often contains several flocks but, for easy expression, we shall use the word "flock" to represent the premises as a full and, in doing so, we shall follow the standard convention of relating the flocks as being in "susceptible", "latent" or "infectious" states (although, of course, it's the condition of the birds that confer these properties on the flocks). We assumed that an infected flock more established a latent (infected but not yet infectious) period that lasted 2 days and was thereafter infectious until depopulation (Boender et al., 2007). We followed the traditional assumption that takes no account of temporal changes within the threat presented by any given infectious flock that may plausibly be attributed to the changing number of infected birds within the flock or the imposition of a quarantine following the confirmation of infection (Stegeman et al., 2004, Le Menach et al., 2006, Bavinck et al., 2009). We divided the host population into two host types (sensu Roberts and Heesterbeek, 2003, Diekmann et al., 2010). the 2 host types are commercial and backyard flocks denoted by the subscripts 1 and a couple of respectively. We further subdivided each type into susceptible (Si) flocks, latently infected flocks (Ei) and infectious flocks (Ii).

$$\frac{dS_1}{dt} = -\beta_{11} I_1 S_1 - \beta_{12} I_2 S_1 - \mu_1 S$$
$$\frac{dE_1}{dt} = \beta_{11} I_1 S_1 + \beta_{12} I_2 S_1 - \delta E_1 - \mu_1 E$$
$$\frac{dI_1}{dt} = \delta E_1 - \alpha I_1$$

$$\frac{dS_2}{dt} = -\beta_{21} I_1 S_2 - \beta_{22} I_2 S_2 - \mu_2 S_2$$
$$\frac{dE_1}{dt} = \beta_{21} I_1 S_2 + \beta_{22} I_2 S_2 - \delta E_2 - \mu_2 E_2$$
$$\frac{dI_1}{dt} = \delta E_2 - \alpha I_2 .$$

Here β11, β12, β21 and β22 are the transmission parameters whose values were to be estimated from the epidemic data. We assumed that until day 21, the turnover rates (μ1 and μ2) of the susceptible commercial and backyard flocks were best represented by those rates normally commensurate with the arena to which they belonged. We assumed that the 410 depopulated commercial flocks consisted of 96 that produced commercial table eggs, 61 that produced broiler hatching eggs and 47 that produced turkey meat. the info in Anon (2004) are consistent and unequivocal with regard to the numbers of flocks in each of those sectors. However, the document contains conflicting reports of what percentage "chicken meat" flocks there have been within the Fraser Valley (Anon, 2004). The quoted figures ranged between 235 and 286. only if not all flocks within the Valley were depopulated we simply assumed that the 206 flocks of the 410 flocks not yet assigned to a sector were "chicken meat" flocks. Average production cycle time for all of those sectors combined was estimated as $(63 * 96 / 410) + (43 * 61 / 410) + (14 * 47 / 410) + (7 * 206 / 410) = 26.39$ weeks. Converting this average cycle time to days (185

Pro. Maidhura Thomson[1; 1] *faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com

days), and following standard reasoning, we estimated μ1 = 1/185 = 0.0054/flock/day. Backyard flocks don't undergo regular cycles of depopulation and replacement (Anon, 2005) so we conservatively estimated the turnover of backyard flocks as μ2 = 0.001/flock/day. We rather crudely mimicked the increasing pace of pre-emptive culling of susceptible flocks by replacing μ1 and μ2 with a relentless value ρ/flock/day from day 21 of the outbreak. Day 21 was chosen to most closely mimic the date on which the increased pre-emptive culling began (Anon, 2004); we estimated the worth of ρ from the epidemic data. the speed at which latently infected flocks became infectious was given by δ = 0.5/flock/day. Two connected, problematic issues remained. First, the epidemic data indicate only the day on which the flock was sampled. Sometimes, sampling was administered because the flock was experiencing a greater than expected mortality. within the face of a deadly disease it's likely that producers are going to be rather sensitive to any increases in mortality then it seems reasonable to assume that these flocks taken away of the latent phase 6–7 days previously (Bos et al., 2007). However, most infected flocks were detected because the results of active surveillance suggesting that the move from latency to infectiousness had occurred at some unknown time but 6–7 days before the sampling date. We therefore constructed a brand new data set from the epidemic data within which the move from latency to infectiousness for all detected flocks was set by subtracting a random number between 1 and seven from the sampling date. Given our assumptions, the date of infection was 2 days before that.

A ≈ 1 / (13.00 − 0.17x)/flock/day. The utmost likelihood algorithms available in Berkeley Madonna (version 8.3.9) were wont to fit the model to the info. Two models were fitted to the information. Within the full model it had been assumed initially that transmission was possible within and between host types (i.e. β11, β12, β21 and β22 were all greater than zero). Within the reduced model, it absolutely was assumed that infected backyard flocks were an example of "spill over" which the backyard flocks played no part in transmission (i.e. β12 = β22 = 0). the simplest fit as judged by the basis mean square deviation was obtained employing a model during which β11 = 0.000505, β12 = 0.00238, β21 = 0.000166 and β22 = 0 (Fig. 1b).

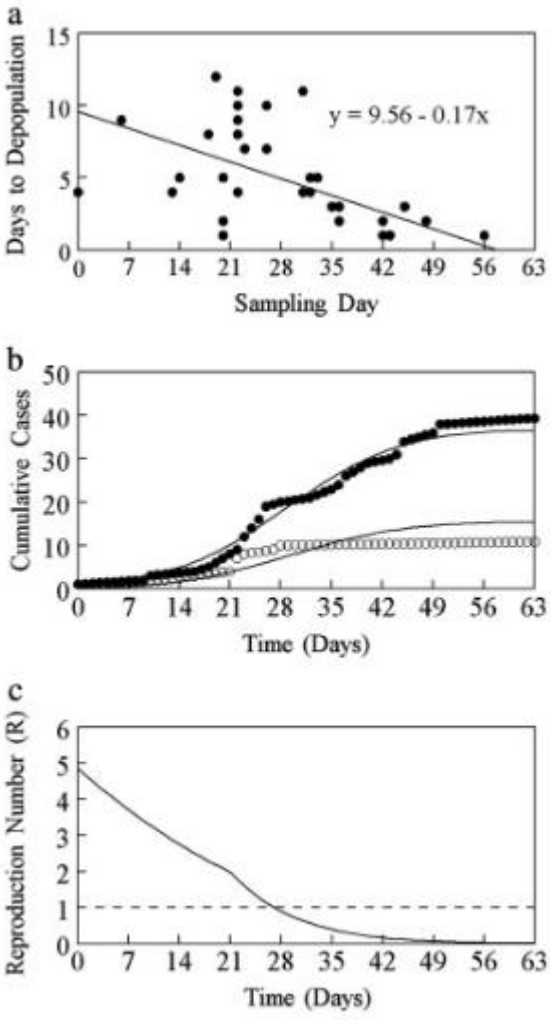

Fig. 1. The 2004 highly pathogenic avian influenza (H7N3) outbreak within the Fraser Valley of British Columbia, Canada. a. Number of days (y) between the date of sampling and therefore the date on which the flock was depopulated plotted by sampling day (x); b. Best fit model (solid lines), the cumulative number of latest cases in commercial flocks (solid circles), and also the cumulative number of recent cases in backyard flocks (open circles); c. Changes within the reproduction number during the course of the epidemic.The basic reproduction number and therefore the effort required to render R0 < 1 The next-generation matrix (K) for the Abbotsford outbreak model is

Pro. Maidhura Thomson[1; 1] *faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com

$$K = \begin{bmatrix} \frac{\delta}{(\delta+\mu_1)}\frac{\beta_{11}N_1}{\alpha_0} & \frac{\delta}{(\delta+\mu_1)}\frac{\beta_{12}N_2}{\alpha_0} \\ \frac{\delta}{(\delta+\mu_2)}\frac{\beta_{21}N_1}{\alpha_0} & \frac{\delta}{(\delta+\mu_2)}\frac{\beta_{22}N_2}{\alpha_0} \end{bmatrix}.$$

Here, N1, N2, and α0 are respectively the quantity of susceptible commercial and backyard flocks at the beginning and also the initial value of the infectious period. Flocks that were depopulated either because they were infected or as a part of the pre-emptive culling processes weren't repopulated until the epidemic was over. Furthermore, the speed of preemptive culling increased over time. Thus, because the epidemic progressed, the amount of susceptible flocks decreased. Additionally, the infectious period (1/α) decreased. As a result, the reproduction number (calculated because the dominant eigenvalue of the matrix, K, Diekmann, et al., 2010) also decreased over time (Fig. 1c). the fundamental reproduction number (calculated using the initial values for all parameters) was given by R0 = 4.8, which is above a previous estimate (Garske et al., 2006) and usually more than most estimates for the essential reproduction number of avian influenza (Garske et al., 2006, Bavinck et al., 2009). This arises because the infectious period is typically assumed to be about 7 days (thus α = 1/7 = 0.142/flock/day even at the start). However, like Stegeman et al. (2004), we found that, initially, the infectious period was about 13 days and decreased once the outbreak had been recognized and surveillance and detection became more efficient.

We now consider the question of targeted control. Bavinck et al. (2009) suggested that if backyard flocks appear to be less susceptible than commercial flocks it'd be sufficient to pre-emptively cull only the commercial flocks. We shall consider this proposition first for the 2004 H7N3 Abbotsford outbreak and so for the 2003 H7N7 outbreak within the Netherlands. Like Bavinck et al. (2009) we acknowledge so ignore the actual fact that the infectious period decreased during the course of the outbreak; for the arguments that follow, we shall set the worth of α to its overall average value (0.143/flock/day). The length of the infectious period reflects the efficiency detection and depopulation. We shall imagine a situation during which all infected flocks (commercial and backyard flocks) will be detected and depopulated (without repopulation) with equal efficiency. this is often the default response but it's often not sufficient to curtail the outbreak as rapidly as policymakers would love and pre-emptive depopulation strategies are frequently implemented additionally. In what follows we shall investigate what fraction of the flocks must be depopulated to curtail the epidemic. The next-generation matrix for the Abbotsford outbreak with the initial flock numbers is thus

$$K = \begin{bmatrix} k_{11} & k_{12} \\ k_{21} & k_{22} \end{bmatrix} = \begin{bmatrix} 1.46 & 0.87 \\ 0.48 & 0.0 \end{bmatrix}.$$

The basic reproduction number R0 = 1.70.

In a single host type model, the proportion (p) of susceptible flocks that has got to be pre-emotively depopulated to confirm that infectious flocks bring about to but one new infected flock each would be

$$p = \left(1 - \frac{1}{1.70}\right) = 0.41.$$

If pre-emptive culling were applied equally to commercial and backyard flocks alike specified the numbers of both host types were reduced to 59% of their starting values, the following generation matrix would be

$$K = \begin{bmatrix} 0.86 & 0.51 \\ 0.28 & 0.0 \end{bmatrix}$$

and the reproduction number within the presence of control measures would be R = 1.0, which is that the required and expected result. However, the intention is to pre-emotively depopulate only the commercial flocks. If we reduce only the numbers of economic flocks to 59% of their starting

Pro. Maidhura Thomson[1; 1] *faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com

values, the following generation matrix becomes

$$K = \begin{bmatrix} 0.86 & 0.87 \\ 0.48 & 0.0 \end{bmatrix}$$

And the reproduction number is R = 1.2, which isn't sufficient to eliminate transmission. Even when the backyard flocks don't constitute a reservoir host population (and during this case, they clearly don't because k22 = 0), they'll contribute to overall virus transmission provided k12 > 0 and k21 > 0. An infectious commercial flock can infect other commercial flocks either directly or indirectly via spillover to backyard flocks. Roberts and Heesterbeek, 2003, Heesterbeek and Roberts, 2007 have noted when there's over one host type, and targeted control involves just one of them, the reproduction number will always result in underestimates of the trouble required to curtail the epidemic. Roberts and Heesterbeek, 2003, Hill and Longini, 2003, Heesterbeek and Roberts, 2007 have each described methods for calculating the relevant statistic (Tc, Heesterbeek and Roberts, 2007). For a two host type model,

$$T_c = k_{11} + \frac{k_{12} \cdot k_{21}}{(1 - k_{22})}.$$

When k11 = 1.46, k12 = 0.87, k21 = 0.48 and k22 = 0, then Tc = 1.88. Using this value to estimate the proportion (p) of susceptible commercial flocks that has got to be depopulated to make sure that infectious flocks make to but one new infected flock gives

$$p = \left(1 - \frac{1}{1.88}\right) = 0.47.$$

Recall that the worth of p calculated using R was 0.41. the rise in p that resulted from using Tc instead of R doesn't seem very big but given the quantity of susceptible commercial flocks near Abbotsford at the beginning of the outbreak (410), this represents a further 25 flocks that has got to be pre-emptively depopulated to curtail the epidemic. this can be not trivial. If selective vaccination of economic flocks were the chosen strategy instead of pre-emptive culling, the identical calculations apply. Using the entire number of economic birds killed during the Abbotsford outbreak to estimate the general average number of birds per flock (Hudson and Elwell, 2004) suggests that a price for p of 0.47 instead of 0.41 represents an extra million doses of vaccine.

We can apply the identical arguments to the H7N7 avian influenza outbreak studied by Bavinck et al. (2009). The next-generation matrix for the 2 host type model, in this case, was

$$K = \begin{bmatrix} 1.33 & 0.067 \\ 0.44 & 0.23 \end{bmatrix}$$

for which the basic reproduction number was given by R0 = 1.33. If we were mistakenly to use this number to calculate p, we'd get a price of p = 0.27. The worth of Tc for this model is

$$T_c = 1.33 + \frac{0.067.0.44}{(1 - 0.23)} = 1.37$$

and

$$p = \left(1 - \frac{1}{1.37}\right) = 0.27$$

Given the amount of economic farms (984) within the outbreak studied by Bavinck et al. (2009), this represents an extra 10 commercial farms.

DISCUSSION

One of the more important contributions models can make to the decision-making process is to produce estimates of the trouble required to attain specific results. One methodology for reducing control effort is to concentrate on only 1 host type. within the context of the commercial poultry industry, especially in countries that there's little information

Pro. Maidhura Thomson[1; 1] *faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com

about the placement of backyard flocks — and access is difficult, depopulating or vaccinating only the commercial flocks is an appealing prospect. this concept is buttressed by expert opinion (Capua et al., 2002, Akey, 2003) and modeling studies (Bavinck et al., 2009, and also the work presented here) both of which suggest that the contribution of backyard flocks to the continuing transmission dynamics of an endemic is modest at the best. However, as we've got shown, even this modest contribution could also be sufficient to compromise the calculated effort required for the targeted control strategies. what proportion this can bear on decision-makers will rely on how risk-averse they're, but they must a minimum of be made aware that effort estimates that don't understand of backyard flocks will probably be underestimated.

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Pro. Maidhura Thomson[1; 1] *faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com

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Pro. Maidhura Thomson[1; 1] *faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com

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Pro. Maidhura Thomson[1; 1] *faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com

Pro. Maidhura Thomson[1; 1]*faculty In Department Of Geography In University In Denver,Colorado*
madhu.df@gmail.com