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Water-borne transmission drives avian influenza dynamics in wild birds: The case of the 2005–2006 epidemics in the Camargue area

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ABSTRACT

Transmission and persistence of avian influenza viruses (AIV) among wildlife remains an unresolved issue because it depends both on the ecology of the host (*e.g.* population density, migration) and on the environment (*e.g.* AIV persistence in water). We have developed a mathematical model that accounts for both AIV epidemics and bird community dynamics. The model is parameterized using bird counts and AIV prevalence data. Results suggest that the transmission patterns driving the dynamics of infection at our study site (Camargue, South of France) involved both a density-dependent and a water-borne transmission processes. Water-borne transmission is, however, the main determinant of the disease dynamics and observed prevalence level. This pattern of transmission highlights the importance of the persistence of viral particles in water in AIV dynamics in wild birds.

1. Introduction

Ecological issues and human health concerns have been shown to be increasingly connected (Collinge and Ray, 2006; Thomas et al., 2005), therefore, understanding the dynamics of infectious diseases in wildlife has become a central focus in public health science. Disease origin, persistence and transmission among wildlife remain difficult to assess because they depend on a huge variety of factors, including strict host–pathogen interactions (*e.g.* virulence, immunity) and ecological aspects, such as the characteristics of natural environments (Collinge and Ray, 2006; Thomas et al., 2005; Daszak et al., 2000). Thus, the threat of emerging zoonotic diseases cannot be addressed without considering the myriad of environmental factors that influence transmission patterns in field conditions.

Influenza viruses are widespread in the animal kingdom; birds, humans, horses and pigs are often infected, as sometimes are cetaceans, dogs and mustelids. Wild aquatic birds of the orders Anseriformes (*e.g.* ducks, geese, swans) and Charadriiformes (*e.g.* gulls, terns, waders) are traditionally considered natural hosts of most avian influenza viruses (AIV). These pathogens are assumed to be mainly transmitted via the fecal–oral route. In wild birds, infection is caused by low pathogenic (LP) AIV (Olsen et al., 2006; Webster et al., 1992) and is usually asymptomatic. However, recent reports have shown that behavioral modifications brought about by infection are probably more common than previously recognized (*e.g.* van Gils et al., 2007). Conversely, domestic birds, particularly poultry, have experienced recurrent outbreaks of highly pathogenic (HP) AIV of the subtypes H5 and H7 (Alexander, 2000), resulting in high mortality and significant economic loss.

Two main routes of transmission could be involved in AIV transmission: (i) a direct bird-to-bird transmission and (ii) a water-borne transmission. For inter-individual transmission, two transmission functions are considered like usually in infectious diseases modeling. The first one is the "density-dependent" process (McCallum et al., 2001), which is the classical assumption for inter-individual transmission of wildlife diseases. According to this transmission pattern, contact rate between individuals increases when the host community size increases (assuming that host community size is correlated to the host density). For clarity, we consider here that "host community" refers to all bird species within our study area. The second pattern is "frequency-dependent" inter-individual disease transmission. In this case,

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the contact rate between bird individuals remains constant even though the host community size increases. Although density-dependent transmission is often involved heuristically, frequency-dependent process sometimes provide a better model fit to a range of wildlife disease data sets (McCallum et al., 2001; Dobson and Meagher, 1996).

Finally, waterfowl may also acquire AIV by drinking or filtering water while feeding. AIV persistence in water has been suggested as a natural mechanism to maintain influenza viruses in avian species (Hinshaw et al., 1979; Stallknecht et al., 1990a,b; Ito et al., 1995; Brown et al., 2007a). Water-borne transmission is likely to spread the infection without the need of direct contact between birds. This transmission pattern has explained the maintenance of LP AIV in domestic ducks under particular farming conditions (Markwell and Shortridge, 1982). However, the general importance of water-borne transmission in natural ecosystems is, as yet, poorly understood.

In this study, we tried to elucidate the most plausible transmission route of AIV involved in Camague by computing a mathematical model that reproduces AIV dynamics and takes into account host community dynamics. This area is of particular interest because it is at the cross-road of many migratory routes of Palaearctic birds (Berthold, 2001) and is identified as a potential hot-spot for the introduction and transmission of bird-borne pathogens (Jourdain et al., 2007).

2. Materials and methods

2.1. Data

We computed data from bird community demography (Jourdain et al., 2007; see online appendix) and AIV prevalence recorded in the Camargue area (Rhône delta, South of France). We sampled wild migratory and resident bird species from September 2005 to July 2006 (11 months). Sampling effort were mainly dependent to bird accessibility and species abundance recorded in the Camargue. During fall and winter (September to February) we mainly sampled ducks with funnel live-traps and freshly killed birds (Lebarbenchon et al., 2007b for details). From March to July, our sampling mainly concerned migratory passerine bird species but also gulls, herons and flamingos (Lebarbenchon et al., 2007a for details). Cloacal swabs were performed for each birds except for gulls for which fresh dropping samples were collected near nests on breeding colonies (Lebarbenchon et al., 2007a for details). All living birds were marked with a ring and identified for species and sex before being release.

During this period we collected and tested 2389 fecal samples (by cloacal swabbing) from 89 free-living avian species belonging to 12 orders. The presence of avian influenza viruses (AIV) was detected by reverse transcription polymerase chain reaction amplification (RT-PCR) targeting the conserved matrix gene segment. Twenty-six samples were positive for AIV (*i.e.* detection of virus in feces), but none were found positive for a HP AIV. All viruses were detected in waterbirds (duck and gull species). Details concerning samples collection and molecular analysis were previously published by Lebarbenchon et al. (2007a,b).

According to other studies performed in Northern Europe (*e.g.* Munster et al., 2007; Wallensten et al., 2007), AIV dynamics show marked annual epidemiological cycles. In our study site, we thus made the assumption that such epidemiological cycles appear every year in the local birds community. Annual cycles has indeed been recently described in the Camargue (Lebarbenchon, 2008), with high prevalences in September, suggesting that the dynamic of infection used in this study is consistent with the annual pattern observed in our study site.

2.2. Model

We simulated a classical SIRS model (Anderson and May, 1991; Grenfell and Dobson, 1995). This model assumes that hosts are divided into three classes according to their immunological status: susceptible (S), which includes the immunologically naive individuals, infectious (I), made up of birds which can infect other individuals, and recovered (R) which consists of birds resistant to the disease.

In this study, we added an additional compartment (B), which represents the aquatic environment or, more precisely, the quantity of viral particles in the aquatic environment. Our model is based on previous mathematical models for human cholera, which is an environmentally persistent water-borne disease (Codeço, 2001; Constantin De Magny et al., 2005). Here, we assume that each bird drinks or filters a similar amount of water while feeding and hence a similar viral load. The following differential equations were used to describe the disease dynamics (see Table 1 for parameters' definition):

$$\frac{dS}{dt} = b_m N - (\lambda + d_m)S + \varepsilon R$$
$$\frac{dI}{dt} = \lambda S - (d_m + \sigma)I$$
$$\frac{dR}{dt} = \sigma I - (d_m + \varepsilon)R$$
$$\frac{dB}{dt} = \gamma I - \pi^* B$$

Parameters b_m and d_m are, respectively, birth and death rates for month m, N is the number of birds in the community. Thanks to these two parameters, we introduced time-forced demographic parameters that allowed us to reproduce the avian community dynamics as observed in our study site. By this way, we assumed that population fluctuations were only due to a birth or death process. The impacts of this assumption has been relaxed thanks to a parameter c which reflects a constant adding in birth and death rates (see online appendix) and which does not change conclusions. λ is the rate of infection for each transmission process, which is assumed constant through time. For inter-individual transmission with a density-dependent process we thus have: $\lambda = \beta I$; for inter-individual transmission with a frequency-dependent process: $\lambda = \phi I/N$; and finally, for water-borne transmission: $\lambda = \omega B/(\theta + B)$ with θ the viral load in water needed to lead an infection. This kind of transmission is assumed to be a saturating function since experimental studies have tested independently different initial viruses load which all lead to an high infection rate (Lu and Castro, 2004; Webster et al., 1978, 1992). In fact, these results suggest a threshold for viral load needed to bring susceptible individuals to an infectious state (θ for the viral load in water).

On the other side, since this model incorporates time-forced demographic parameters (b_m and d_m, see Table S1) and consequently dynamic population size (N_t), abundance of viral particles fluctuates in time. If this model does not incorporate this feature, abundance of viral particles (B) could reach a persistent value at equilibrium and water-borne transmission could be, in this case, assimilated to a continuum between density-dependent and frequency-dependent transmission. Accordingly, we investigated outputs with such an intermediate model, between frequency-dependent and density-dependent. The corresponding transmission mechanism is defined by $\lambda = \varphi I/(N^\eta)$ where φ is the contact rate between individuals and η is the degree of frequency-dependence of transmission ($\eta = 0$ for full density-dependent transmission and $\eta = 1$ for full frequency-dependent transmission).

We estimated β, ϕ, ω, φ and η using fixed the values range of σ (recovery rate per individual), θ (minimal viral load to initiate infection), γ (viral shedding per day per infected individual) and π

Table 1
Parameter values used for estimations.

Parameter	Definition	Used values	Comments
σ	Recovery rate per individual	1/7 days^{-1}	Timing during which the most intensive virus shedding occurs (Webster et al., 1978; Lu and Castro, 2004)
θ	Minimal viral load needed to initiate an infection	1, $10^{1.8}$ and $10^{4.7}$ EID_{50}	$10^{4\ 7}$ EID_{50} is the minimal viral load recorded to initiate an infection with a LP AIV in experimental studies (Lu and Castro, 2004). Small viral load (up to $10^{1.8}$ EID_{50}) have been detected from water samples (Ito et al., 1995), suggesting that it may be enough to initiate an infection in natural conditions. We deliberately over-estimated θ in order to take into account the potential diversity of infectious efficiency of AIV
γ	Viral shedding per day per infected individual	1, $10^{3.5}$ and 10^{7} EID_{50}	High titers of virus (10^{7}–10^{8} EID_{50}) have been detected in experimental infections of ducks with LP AIV (Webster et al., 1978). Recent studies investigated the replication of HP AIV in wild bird species have however recorded smaller titers (Brown et al., 2006, 2007b, 2008)
π	Viral particle inactivation rate in water	28 and 207 days^{-1}	Experimental studies showed that, depending on temperature, salinity and pH, AIV can persist in water up to 207 days (Stallknecht et al., 1990a,b). Recent studies (Brown et al., 2007a) however estimated short persistence time (about 28 days) when the virus face strong environmental constraints (high salinity and temperatures)
ε	Immunity loss	1, 2 or 4 years^{-1}	Rate for immunity loss, which could be due to an invasion of a new dominant strain or an antibody depletion
β	Transmission rate for density-dependent process	0.00005–0.1	Ind. year^{-1}
φ, η	Transmission rate and level of frequency-dependent for continuum model	0.00005–1550 and 0–1	Ind. year^{-1} and no units
ω	Contact rate with water	0.5–50	Drinking volume year^{-1}
ϕ	Transmission rate for frequency-dependent process	0.00005–1550	Ind. year^{-1}
c	Constant birth and death rates	0–20	Ind. year^{-1}

(viral particle inactivation rate in water) presented in Table 1 (evaluation procedure is detailed in online appendix). We also investigated transmission patterns which involved two transmission routes: density-dependent + water-borne transmission, and frequency-dependent + water-borne transmission. Finally, since immune response against AIV in wild birds is poorly understood, the rate of loss of immunity (ε) has been included in the model, and three scenarios were tested: (i) permanent immunity ($\varepsilon = 0$), (ii) a loss of immunity in 1 year ($\varepsilon = 1$ ind. year^{-1}) and (iii) a loss of immunity in 4 years ($\varepsilon = 4$ ind. year^{-1}).

This mathematical framework starts from the following hypotheses. (i) Each bird species has the same duration of virus excretion: a 1–2-week duration has been reported for the most common avian species (Webster et al., 1978; Lu and Castro, 2004). (ii) Each bird species excretes the same number of viral particles: we assumed that viral particle production is equal across different host species. Despite that birds from different species could excrete different viral load into the environment (Keawcharoen et al., 2008), this assumption could be applied from a mathematical point of view if a threshold of viral concentration in the environment is reached, which is our case in all our simulations. We also assumed that viral particles produced by different avian species remain infectious for the same duration irrespective of the AIV subtypes. (iii) Water contacts are equal across avian species: we assumed that there are no differences in terms of water contact rate for disease transmission between avian species. This rough approximation allows us to analyze the impact of the mean aquatic behavior of avian species.

For each of the disease transmission mechanisms, we optimized the transmission parameters in our mathematical model to obtain the minimal error against the disease data. More specifically, we maximized the likelihood of all models and applied afterwards a likelihood ratio test (LRT) in order to select the most parsimonious model which shows the best data adequacy. Hence, likelihood values are tested through a χ^2 with one degree of freedom. To distinguish two not nested models, we use the Akaike Information Criterion (AIC) as usually. The evaluation procedure is detailed in online appendix.

We then analyzed how these different transmission patterns may modify the disease dynamics by using optimized transmission parameters for each of the transmission mechanisms we investigated. The most intuitive way to evaluate the effect of transmission was to analyze the synchrony between disease dynamics and host community dynamics. We therefore used the classical cross-correlation method (Tobin and Björnstad, 2003) between AIV and bird community dynamics. With this method, each transmission pattern will result in different "dynamic signatures" (*i.e.* different synchronies between disease dynamics and host community dynamics) which could be compared with the dynamic signature of data.

3. Results

We estimated parameters for different transmission routes (β, ω, ϕ, ρ, η) when all other parameters are fixed (θ, γ, π, ε, see Table 1, and introductory level of infectious and recovered individuals, see online appendix). Because non-transmission parameters are uncertain, transmission parameters have been estimated in 194 different situations, where each of these situations represents a different value for a non-transmission parameters. For each of the six transmission mechanisms we investigated, we choose the one with the best parameter estimation and then compared different model outputs (see Fig. 1 to watch model outputs and observed AIV time series). Results from the LRT (Table 2) allow us to statistically identify the model with the best fit with the infection dynamic data recorded in our study site.

This analysis indicates that the transmission process which drives the dynamics of infection recorded in our study site (Camargue, South of France), involved both a density-dependent and a water-borne transmission component (Fig. 1). The models combining an inter-individual and water-borne transmission are significantly better than all models involving only one transmission route (all the likelihood ratio tests are significant, see Table 2). This first result underlines that a combining transmission route have to be considered. To select the right model between the two remaining possibilities, *i.e.* density-dependent or frequency-

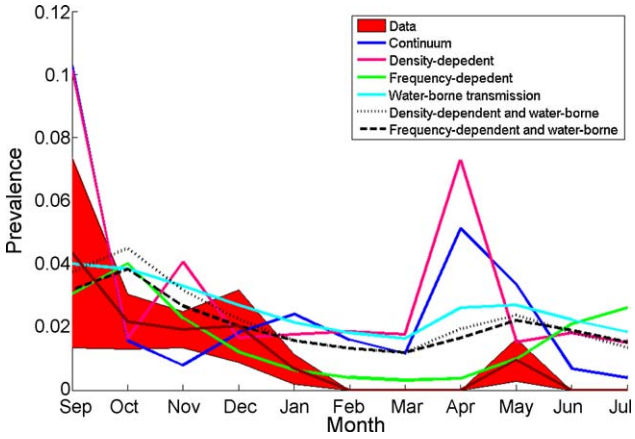

Fig. 1. Model outputs for the best estimation of the model on the basis of the epidemiological data. Dashed red lines are confidence intervals for the observed data. The following parameters were used (i) density-dependent transmission: $\beta = 0.01$, (ii) frequency-dependent transmission: $\phi = 100$, (iii) water-borne transmission: $\omega = 6.73$, (iv) continuum transmission: $\eta = 0.9982$ and $\varphi = 273.6289$ and (v) density-dependent and water-borne transmission: $\beta = 2.85 \times 10^{-4}$, $\omega = 2.34$ and (vi) frequency-dependent and water-borne transmission: $\phi = 100$, $\omega = 1.27$. All these models assume 0% of infectious and recovered newcomers and $c = 0$ (see online appendix for more details).

Table 2
Results of the log-likelihood ratio test (with one degree of freedom, significant threshold >3.84).

	DD	FD	W
DD + W	47.72 ($p < 0.05$)		6.10 ($p < 0.05$)
FD + W		13.41 ($p < 0.05$)	9.03 ($p < 0.05$)

DD: density-dependent transmission, FD: frequency-dependent transmission, and W: water-borne transmission.

dependent transmission with water-borne transmission, we use the AIC. This criterion shows that the combination of density-dependent and water-borne transmission is significantly better than the other one (see Table 3). The associated parameters estimates for this transmission process are: $\theta = 10^{1.8}$, $\gamma = 1$ EID_{50} ind. day^{-1}, $\pi = 28$ $days^{-1}$, $\varepsilon = 1$ ind. $year^{-1}$, 0% of infectious and recovered newcomers and $c = 0$ (for more details on c, see online appendix).

To identify the role of each transmission route in the observed dynamics, and hence to see if one single transmission route may reproduce the dynamic signature observed in our data, we computed a cross-correlation factor between AIV dynamics and bird population dynamics. Cross-correlation factors measures similarity between two dynamics as a function of a time-lag. Results highlight that, for our recorded AIV dynamics on the field, a significant cross-correlation factor was present and corresponded to a lag of zero (Fig. 2A). This means that AIV dynamics are synchronous with host community dynamics (without any delay). Our mathematical model shows that both types of inter-individual transmission considered in isolation (Fig. 2B and C) leads to the loss of synchrony between AIV dynamics and host community dynamics, suggesting that these transmission routes, in isolation, cannot lead to the dynamic signature recorded in our study site. Fig. 2D shows the dynamic signature for a water-borne transmission model. It is different from those obtained for the inter-individual transmission patterns as maximal cross-correlation is centered on lag of zero. This means that the host community dynamics drive the disease transmission (by viral particle production in water and by a high number of susceptible individuals) and the environment constitutes a persistent source of infection through time. Thus, each new susceptible bird (either a juvenile or a migrant) may rapidly become infectious. This process results in a maximal cross-correlation with no delay between host bird community and disease dynamics.

Table 3
Akaike Information Criterion for all different transmission routes.

Transmission route	AIC
Density-dependent transmission	5.1348
Frequency-dependent transmission	5.6036
Continuum transmission	5.8954
Water-borne transmission	6.0589
Density-dependent + water-borne transmission	4.1646
Frequency-dependent + water-borne transmission	3.8826

According to these results, the dynamic signature of our disease data seems to be compatible only with a water-borne transmission pattern. This underlines the important role of water-borne transmission in the processes which drive the dynamics of infection, involving both density-dependent and water-borne transmission components.

We then investigated the implications of each transmission route in this model on the disease dynamics. Hence, we removed, step by step (i) density-dependent transmission and (ii) water-borne transmission, and looked at the consequences on the dynamics of infections (Fig. 3). Removal of density-dependent transmission leads to high prevalence and the correct dynamic behavior is conserved. In contrast, removal of water-borne transmission leads to an increase of prevalence from October to May, but at a lower level than predicted with a water-borne transmission model.

4. Discussion

Density-dependent transmission is classically assumed for directly-transmitted diseases despite some studies showing that frequency-dependent transmission can also explain disease data. In this paper, we investigated if these two transmission functions could be involved in AIV. Because these viruses infect mainly waterbirds, and viral particles can persist in water for a long period, we also considered water-borne transmission in order to determine the role of aquatic ecosystems in AIV epidemiology in wild birds.

This study highlights that the transmission processes which drive the AIV dynamics of infection in our study site (Camargue, South of France), involved both a density-dependent and a water-borne transmission component (Fig. 1, Table 2). Persistence in water has been suggested as a possible way of maintaining AIV in wild birds (Wallensten, 2007). We indeed see the importance of aquatic ecosystems in the persistence and transmission of AIV and further, our research provides evidence that water-borne transmission drives the dynamics of infection in bird communities (Figs. 2 and 3).

These results depend both on the bird community dynamics and AIV prevalence recorded in our study site. AIV prevalence is known to vary not only by region, but also by species, in which case differences are likely to be a result of feeding behavior (Garamszegi and Moller, 2007), susceptibility or social behavior. Our mathematical model follows the assumptions that all species have the same duration of virus excretion, immunity, number of viral particles produced and water contact rate. More ecological data concerning these aspects are required in order to refine such a mathematical model. Along the same idea, identifying habitat heterogeneity (*e.g.* salinity, pH, temperature) is particularly important in order to identify potential hot-spots of aquatic transmission risks. We are also aware that our model simplifies the impacts of bird migrations in term of introduction and dispersion of influenza viruses. From a conceptual point of view, it seems

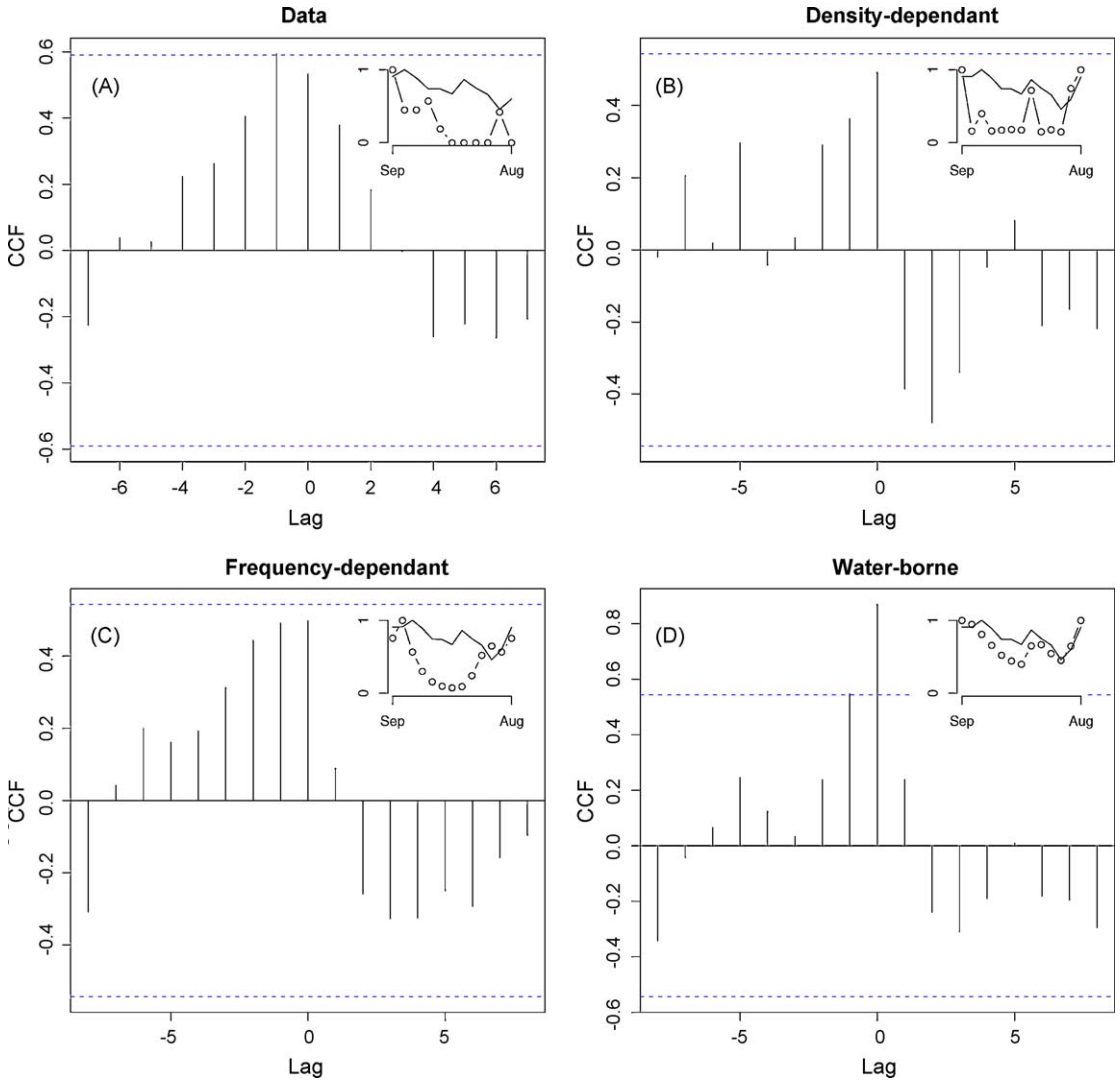

Fig. 2. Cross-correlation factors (CCF) between disease dynamics and host community dynamics for the three different transmission processes. (A) Field data; (B) density-dependent transmission; (C) frequency-dependent transmission; and (D) water-borne disease transmission. Inserted plots represent bird community dynamics (solid lines) and AIV dynamics observed (A) or predicted (B–D) (dotted lines).

plausible that migrations may explain alone the observed disease dynamics. However, investigations of such aspects would require precise datasets concerning birds' movements, according to the species and periods of the year. Brown et al. (2009) recently showed that physical characteristics of water could affect dramatically persistence of viral particles in water. In this study, we assume a constant degradation rate of these viral particles because of the lack of precise and quantitative data. However, integrating fluctuations in environmental persistence will only improve quantitatively our predictions.

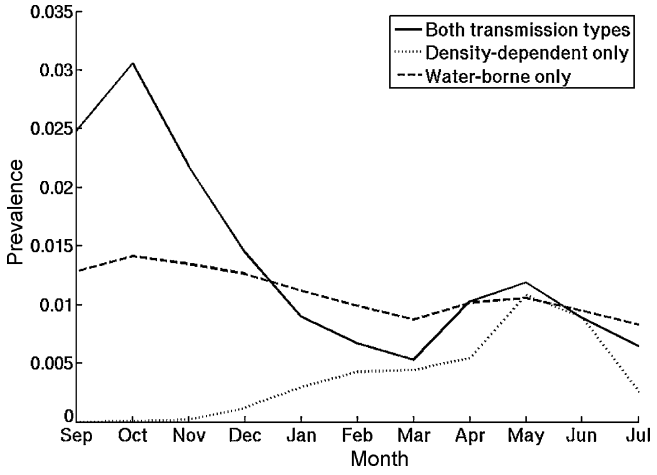

Fig. 3. Impact of transmission route removal in the model which involves density-dependent and water-borne transmission.

The recent spread of the HP H5N1 AIV is a matter of concern for public health authorities. Despite the fact that the relative contribution of wild water birds in the spread of the disease remains controversial (Gauthier-Clerc et al., 2007), the dynamics and pathways of virus dispersion has started to become clearer (Kilpatrick et al., 2006). Recent studies reporting the effects of wild bird infections with HP H5N1 AIV highlighted that the virus replicates more (and for a longer time) in the host bird trachea rather than in the digestive tract (Sturm-Ramirez et al., 2005; Brown et al., 2006; Keawcharoen et al., 2008) suggesting that airborne transmission may be more significant for HP than for LP AIV. In artificial conditions (*i.e.* intensive farming), HP AIV are likely to be transmitted by density- or frequency-dependent mechanisms, because of the confined environment and high contact rate among bird. Nevertheless, in natural conditions, the mechanistic acquisition of AIV in water is probably similar both for LP and HP AIV. Brown et al. (2007a) recently provided evidence that HP H5N1 does not persist as long as LP AIV in water, at least under

experimental conditions. Because the persistence of viral particles into water seems to be the key parameter of AIV dynamics in wild birds, these results are of primarily importance in understanding HP transmission in aquatic ecosystems.

Water-borne transmission could be a major determinant in the epidemiology of AIV. Increased persistence of multiple strains concurrently in the environment is likely to translate into an increased risk of infection for animals and humans, but could also enhance the probability of host co-infection and viral re-assortment (Spackman et al., 2005). This underlines the potential role of the environment for AIV emergence and dispersion, and the urgent need of a thorough consideration of ecological factors to properly assess the risk of emerging infectious diseases.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.meegid.2009.04.009.

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