Seasonality and wildlife disease: how seasonal birth, aggregation and variation in immunity affect the dynamics of *Mycoplasma gallisepticum* in house finches

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We examine the role of host seasonal breeding, host seasonal social aggregation and partial immunity in affecting wildlife disease dynamics, focusing on the dynamics of house finch conjunctivitis (*Mycoplasma gallisepticum* (MG) in *Carpodacus mexicanus*). This case study of an unmanaged emerging infectious disease provides useful insight into the important role of seasonal factors in driving ongoing disease dynamics. Seasonal breeding can force recurrent epidemics through the input of fresh susceptibles, which will clearly affect a wide variety of wildlife disease dynamics. Seasonal patterns of social aggregation and foraging behaviour could change transmission dynamics. We use latitudinal variation in the timing of breeding, and social systems to model seasonal dynamics of house finch conjunctivitis across eastern North America. We quantify the patterns of seasonal breeding, and social aggregation across a latitudinal gradient in the eastern range of the house finch, supplemented with known field and laboratory information on immunity to MG in finches. We then examine the interactions of these factors in a theoretical model of disease dynamics. We find that both forms of seasonality could explain the dynamics of the house finch–MG system, and that these factors could have important effects on the dynamics of wildlife diseases generally. In particular, while either alone is sufficient to create recurrent cycles of prevalence in a population with an endemic disease, both are required to produce the specific semi-annual pattern of disease prevalence seen in the house finch conjunctivitis system.

Keywords: *Carpodacus mexicanus*; *Mycoplasma gallisepticum*; disease; dynamics; partial immunity; seasonality

1. INTRODUCTION

The emergence of new infectious diseases in wildlife, their potential threat as zoonotic diseases of humans and livestock, or their potential conservation implications, has become an important component of concerns over anthropogenic effects on the global environment (Daszak *et al.* 2000; Harvell *et al.* 2002). This concern has renewed interest in the potential for diseases to regulate populations, cause extinctions and spread geographically and across species. The emergence of *Mycoplasma gallisepticum* (MG) in house finches provides an interesting case study because it provides an opportunity to study the unmanaged emergence of a wildlife disease (Friend *et al.* 2001). This disease appears to have entered a robust regime of semi-annual cycles of which the magnitude and form vary latitudinally (figure 1; Altizer *et al.* 2004*a*). Hence, we focus on the dynamics of this novel host–pathogen interaction, and use latitudinal variation in the seasonal aspects of host life history to examine how these dynamics could arise.

Although several aspects of wildlife biology have strong seasonal components, which vary with latitude (Cartron *et al.* 2000; Cooper *et al.* 2004), only a few disease models

have examined the implications of seasonal aspects of biology on wildlife disease dynamics (Swinton *et al.* 1998). Yet from studies on human diseases, such as measles (Bolker & Grenfell 1993; Bjørnstad *et al.* 2002; Keeling & Grenfell 2002) and cholera (Rodo *et al.* 2002), it has become clear that seasonal changes can have important effects on the dynamics of disease.

The purpose of this paper is to explore how seasonal patterns and latitudinal gradients in avian behaviour and reproduction could affect annual outbreaks of infection. We explore the combinations of a set of mechanisms that produce the semi-annual patterns of prevalence similar to those seen in wild populations of house finches experiencing mycoplasmal conjunctivitis (figure 1; Altizer *et al.* 2004*a*). In particular, we investigate the consequences of seasonality in breeding and sociality, together with variation in immune function.

Seasonal breeding in birds, including house finches, often varies with latitude, owing to climatic constraints (Cartron *et al.* 2000; Cooper *et al.* 2004). Both the initiation and end of reproduction in multi-brooded species are limited by temperature and food conditions. Thus, seasonal breeding begins earlier in the south, but often also ends slightly earlier. Thus, latitudinal variation in length of the breeding season is one of the key features that we model.
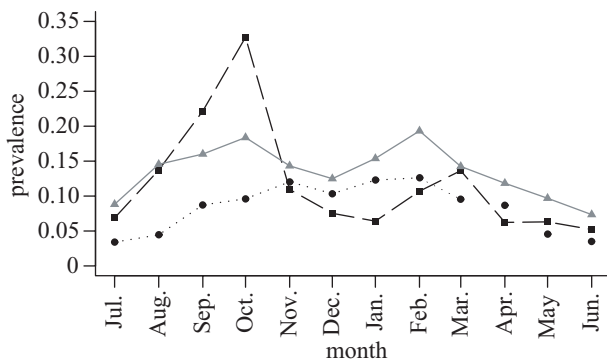
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Figure 1. Seasonal component of annual variation in prevalence from empirical observations, based on Altizer *et al.* (2004*a*). Dashed line with squares represents southern region of the eastern United States, grey line with triangles represents central region, and dotted line with circles represents northern region.

In house finches, as in many bird species, social organization varies seasonally. During the breeding season groups are small (typically pairs or small breeding aggregations), while during the non-breeding seasons birds aggregate into larger feeding flocks (Hill 1993). Thus, behavioural affiliations between individuals and the transmissibility of disease in their social networks may change with seasons, and therefore vary with latitude, potentially in nonlinear ways. Although much behavioural ecology research has focused on these patterns of social organization, they are rarely viewed in light of how they would affect disease dynamics (but see Gremillion-Smith & Woolf 1988; Altizer *et al.* 2004*a*). Variation in social structure could, and probably does, interact with variations in immune function and the endocrine system (Balm 1999). Although our group has only preliminary data on these factors in house finches (K. M. Lindström, D. Hawley, A. Davis and M. Wikelski, unpublished data), we do know that finches can recover from MG (Kollias *et al.* 2004), but continue to remain somewhat susceptible to the disease, and may even recrudesce on occasion (K. V. Sydenstricker, A. A. Dhondt, D. H. Ley and K. V. Kollias, unpublished data).

We begin by presenting the essential natural histories of house finches and MG, the causative agent of the conjunctivitis. We then focus on three key biological factors in this system: immune response to MG, seasonal breeding and seasonal social aggregation. These factors are incorporated into a model framework, and the model is examined and compared with empirical patterns.

2. HOUSE FINCHES AND *MYCOPLASMA GALLISEPTICUM*

House finches are native to southwest North America, and have spread throughout the eastern United States after an introduction in 1940 (Bock & Lepthien 1976; Hill 1993). House finches in the eastern range depend on human-settled areas, particularly suburban areas, for survival, although facultative use of such habitat has also expanded the western range of the house finch (Hill 1993). The previously increasing expansion of house finches in the eastern range has slowed, and abundances declined in some areas through the recent emergence and rapid spread of a novel strain of the bacterium *Mycoplasma gallisepticum*, a

common pathogen of domestic poultry (Ley *et al.* 1996; Luttrell *et al.* 1996; Dhondt *et al.* 1998; Hochachka & Dhondt 2000).

(a) *Disease progression and immunity*

House finches that are infected with MG develop mild to severe conjunctivitis (Kollias *et al.* 2004), although asymptomatic carriers may exist (A. A. Dhondt, unpublished data). The physical signs of disease in the eyes allow description of prevalence through observational studies. House finches recover from disease both in captivity (Roberts *et al.* 2001; Kollias *et al.* 2004) and in the field (Faustino *et al.* 2004). Experimental studies in the laboratory indicate that house finches gain partial immunity to MG, so that reinfection causes less severe disease of reduced duration (K. V. Sydenstricker, A. A. Dhondt, D. H. Ley and K. V. Kollias, unpublished data). Therefore, we modelled the system not just as a susceptible–infected–recovered model (Anderson & May 1979; Anderson 1982), but divided the infected class into novel infections (I_1) and reinfections (I_2), birds that become infected from the recovered stage.

The simplest model for duration of infection and duration of immunity is an exponential distribution, which appears accurate for the duration of infection of MG in house finches (Kollias *et al.* 2004). The duration of immunity, however, lasts a relatively long time in all recovered birds (more than 14 months), although there are some indications that it might wane to some degree with time and that there is heterogeneity in immunity among individuals (K. V. Sydenstricker, A. A. Dhondt, D. H. Ley and K. V. Kollias, unpublished data). Therefore, we initially investigated the effects of a gamma-distributed time to loss of immunity. This provides a simple model of both heterogeneity and non-exponentially distributed immunity. However, we concluded that permanent, partial immunity is a sufficient model, even if rarely a few long-lived house finches may lose immunity.

(b) *Seasonal breeding*

House finches are a non-colonial, non-territorial, multi-brooded species that often relocate between nesting attempts. House finches take *ca.* 33 days from clutch initiation until fledging (Hill 1993). There can be more than six nest attempts over the season though three sets of successfully fledged young is probably maximal. In Hawaii, the average number of young fledged per nest (including unsuccessful nests) is 1.91 (Hirai 1975), close to our own initial estimates of 2.07 young fledged per nest in Ithaca, NY (M. J. Driscoll and A. A. Dhondt, unpublished data). The breeding season is consistently from April to July in Ithaca, NY, but can shift substantially earlier in the south, with start dates in early March (Hill 1993; S. Altizer, personal communication).

(c) *Seasonal aggregation*

House finches have a seasonally dynamic social structure: birds occur as nesting pairs over the summer, often clustered in certain areas, probably because of habitat suitability, but are neither particularly hostile to, nor particularly gregarious with, neighbours. Thus, social contacts are diffuse, except within the immediate families. As autumn progresses, first juveniles, then adults begin to form foraging flocks, with the size of these flocks peaking in

Table 1. Model parameters.

parameter	description	value
d	background mortality of healthy finches	0.5
K	density-dependent mortality control	50.0
α	disease induced mortality	0.5
λ	density-dependent transmission rate	2.0
ϕ	frequency-dependent transmission factor	15.0
γ	recovery rates	24.0
ξ	increase factor for I_2 finches (> 1)	2.0
v	number of recovery stages	8
χ	reduction factor for recovered finches ꓱ inicode range 22, hex 220A, decimal 8714(0, 1)	0.6

January, and in northern latitudes (Altizer *et al.* 2004*a*). In late February and March, these flocks break up, and breeding pairs form. These differences in social aggregation across seasons are incorporated as seasonal forcing in the transmission term of the model.

3. THE MODEL

We have used the susceptible–infected–recovered (SIR) model framework (Anderson & May 1979; Anderson 1982). This class of models has generally been used for epidemic modelling, as the typical dynamical behaviour is a brief excursion to high disease prevalence upon introduction of a few infected individuals into a susceptible population, followed by a stable endemic disease phase. Although it has been shown that this class of models can exhibit multi-annual cycles when seasonal forcing in the contact rate occurs (Bjørnstad *et al.* 2002; Keeling & Grenfell 2002), less research has focused on how dynamic breeding and seasonal aggregation (i.e. seasonal forcing in the contact rate) in wildlife would change disease dynamics. We incorporate our knowledge of immunity and disease progression by accounting for novel and reinfections separately. We incorporate seasonal effects by adding forcing functions based on empirical statistical models of bird breeding and aggregation.

The equations that we developed as a model of the system, incorporating this biological knowledge are given here:

$$\frac{dS}{dt} = b(t,l)N - \beta(t,l)(I_1 + I_2)S - d\left(1 + \frac{N}{K}\right)S, \quad (3.1a)$$

$$\frac{dI_1}{dt} = \beta(t,l)(I_1 + I_2)S - (d + \alpha)\left(1 + \frac{N}{K}\right)I_1 - \gamma I_1, \quad (3.1b)$$

$$\frac{dR}{dt} = \gamma(I_1 + \xi I_2) - d\left(1 + \frac{N}{K}\right)R - \chi\beta(t,l)(I_1 + I_2)R, \quad (3.1c)$$

$$\frac{dI_2}{dt} = \chi\beta(t,l)(I_1 + I_2)R - (d + \alpha)\left(1 + \frac{N}{K}\right)I_2 - \gamma\xi I_2, \quad (3.1d)$$

$$N = S + I_1 + R + I_2. \quad (3.1e)$$

N represents the total population; S, susceptibles; I_1, birds infected for the first time; I_2, birds infected for the second (or more) time; and R, birds recovered from an infection. t symbolizes time, in decimal years; and l is latitude in decimal degrees North. $b(t,l)$ is seasonal birth rate, and $\beta(t,l)$ is seasonal disease transmission as a function of seasonal aggregation. The details of these forcing functions are

described in § 3*a*. Scalar parameters are defined in table 1. In this model, we ignore the sub-stages and sub-categories of infection, and presume that symptomatic birds are at least a constantly proportionate index of the number of actually infectious birds (Rodo *et al.* 2002).

(a) *Seasonal breeding*

Seasonal breeding is the first forcing function, where $b(t,l)$ represents the seasonally forced birth rate into the susceptible pool (equation (3.1*a*)). To define geographical variation in the duration of breeding, we used breeding data for Eastern bluebirds, *Sialia sialis* (Cooper *et al.* 2004), because detailed data on geographical variation in breeding are unavailable for house finches. Bluebirds, like house finches, are multi-brooded and begin breeding early; more crucially, the estimates for the earliest and last 1% of clutch initiations of bluebirds match those for house finches (Cooper *et al.* 2004; M. J. Driscoll, E. C. Swarthout and A. A. Dhondt, unpublished data)—clutch initiation in early April around Ithaca, NY, and clutch initiation as early as early March in the south, with less change in the end of season. Because house finches are very prolific and relatively constant breeders, as a first approximation we use a simple birth-rate model over the breeding season, to represent the input of fledglings into the population. Thus seasonal breeding is defined as follows:

$$b(t,l) = 2.5 \left(\frac{1}{1 + e^{400(s(l)-t)}}\right)\left(\frac{e^{400(c(l)-t)}}{1 + e^{400(c(l)-t)}}\right)$$

$$s(l) = \frac{-3.56 + 3.05l}{365.25}$$

$$c(l) = \frac{214.8 + 0.75l}{365.25}, \quad (3.2)$$

where $s(l)$ represents the initial date fledglings enter the population, and $c(l)$ represents the last date fledglings enter the population. We use an arbitrarily steep double-logit function to avoid non-smoothness issues in the numerical integration. Figure 2*b* shows these curves for 42.5° N (Ithaca, NY) and 33.8° N (Atlanta, GA).

(b) *Statistically modelling seasonal aggregation*

To model the effects of aggregation at bird feeders during the winter months, we used Project FeederWatch data, which consist of maximum counts of birds of all species at bird feeders observed over a two day period, at repeated intervals from November to March, by citizen science volunteers (Cornell Laboratory of Ornithology 2002; Wells *et al.* 1998). We only examined the data for
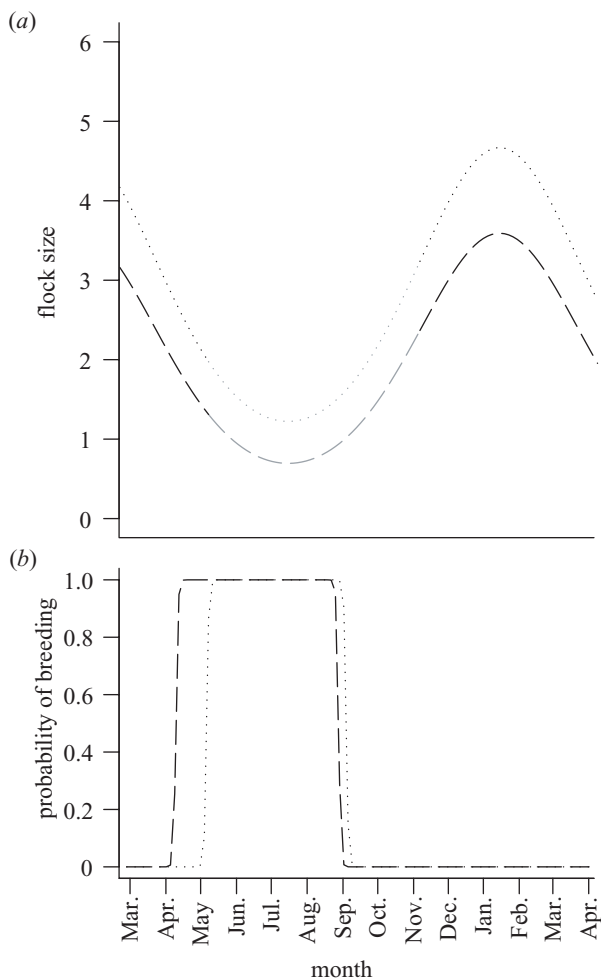
Figure 2. (*a*) Seasonal aggregation (flock size) statistical model based on equation (3.3), with parameters in table 3. (*b*) Seasonal breeding statistical model, adapted from Cooper *et al.* (2004). Dotted line represents fits for Ithaca, NY ($l = 42.5°$ N), dashed line Atlanta, GA ($l = 33.8°$ N).

the eastern population of house finches. Additionally we limited the data to observers who watched over the entire two day period, for between 1 and 4 h. This category of data comprises *ca.* 25% of observers, and prevents the necessity of statistically modelling observer effort. Because we are interested specifically in within-season variation, we limited ourselves to data from observers who recorded observations for more than three months of a given winter. We limited the data geographically to observations east of 100° W longitude, and between 30° N and 45° N latitude, to restrict our analysis to the introduced eastern population, and to avoid latitudinal effects being confounded by longitudinal and regional effects at extreme latitudes where data were sparse. We also removed from analysis observers with a known history of miscounting.

Because we were interested in within-season variation and latitudinal variation, we chose to fit a cosine function to the data, as the simplest smooth function to represent seasonal variation. We used a nonlinear mixed-effects model framework that controls for the effect of observer and 'winter within observer' (Pinherio & Bates 2000; R Development Core Team 2003). The full model is described in equation (3.3), where Φ is the fitted

aggregation function, l represents latitude, t represents decimal year since 1 January 1998, m-coefficients represent fixed effects on the magnitude of the within season fluctuation, b-coefficients represent fixed linear effects, τ represents an offset exactly when the mid-winter peak occurs relative to 1 January, and σ represents random effects, m for those in the magnitude of the cosine function, b for a linear effect and ε for pure error:

$$\log(\Phi(t,l)) = (m_0 + m_1 \cdot l + \sigma_m)\cos(t + \tau)$$
$$+ b_0 + b_1 \cdot l + \sigma_b + \sigma_\varepsilon. \quad (3.3)$$

Ten versions of the fixed effects of this model were fitted, from a simple mean plus linear random effects model, to a model with latitudinal gradients in both magnitude and linear portions of the model (table 2). Random effects were either estimated for the magnitude portion (σ_m) or the linear portion (σ_b), but not both, because models that attempted both were singular. Information theory methods and the AIC criteria were used for model selection (Burnham & Anderson 2002), and because we were comparing among fixed-effects portions, we used unrestricted maximum-likelihood methods to fit the mixed-effects models.

Although we fitted models that used either just observer or just winter for random effects, these models all had AIC-values greater than 500 units higher than the models presented with both observer and winter within observer effects. Therefore, we do not present these relatively uninformative models. However, these simpler models were used to provide starting coefficients for the final model fits. The final model set, including both random effects, and their ΔAIC values and weights are provided in table 2.

This statistical work yielded two models with essentially all of the AIC-weight. The most complex model, number 2, with random effects in the magnitude of the cosine function received 34% of the weight, and the same model without a latitudinal effect in the magnitude of the cosine, model no. 1, received 66% of the weight. Given the closeness of these models, and the fact that they both have the same random effects terms, weighted model averaging was used to produce the final coefficients used in our aggregation model (table 3). Because we examine scenarios without seasonal variation, we will also use model no. 9, without seasonal variation, where $b_0 = 0.64$ and $b_1 = 0.023$.

Interestingly, the random effects are clearly most useful in the cosine term, which means that the magnitude of within-season fluctuation at any given feeder may vary substantially, and is clearly more useful than a simple variation in abundance for any given feeder. This position for the random effect in the fluctuation term suggests that the number of finches seen at a bird feeder differs most at times of peak aggregation across observers and year of observation, but there is less variation in the numbers of birds seen at the beginning and end of the season, when aggregation is low. Additionally, the latitudinal effect is strongest as a simple linear effect, but that may not be its only effect. This emphasis of the latitudinal effect in the linear term instead of the fluctuation term suggests that latitude is affecting average numbers, perhaps through climate, but that it may also have some effect on maximal aggregation, either through winter climate or through day length cues to social behaviour.

Table 2. *Information theory summary of fitted models with both observer and winter within observer random effects.*

number	model	ΔAIC	likelihood ratio	AIC weight
1	$(m_0 + \sigma_m)\cos(t + \tau) + b_0 + b_1 l + \sigma_\varepsilon$	0	1.0000	0.6598
2	$(m_0 + m_1 l + \sigma_m)\cos(t + \tau) + b_0 + b_1 l + \sigma_\varepsilon$	1.32	0.5155	0.3402
3	$(m_0 + m_1 l + \sigma_m)\cos(t + \tau) + b_0 + \sigma_\varepsilon$	20.03	$< 10^{-4}$	$< 10^{-4}$
4	$(m_0 + \sigma_m)\cos(t + \tau) + b_0 + \sigma_\varepsilon$	27.31	$< 10^{-4}$	$< 10^{-4}$
5	$(m_0)\cos(t + \tau) + b_0 + b_1 l + \sigma_b + \sigma_\varepsilon$	79.35	$< 10^{-4}$	$< 10^{-4}$
6	$(m_0 + m_1 l)\cos(t + \tau) + b_0 + b_1 l + \sigma_b + \sigma_\varepsilon$	79.61	$< 10^{-4}$	$< 10^{-4}$
7	$(m_0)\cos(t + \tau) + b_0 \sigma_b + \sigma_\varepsilon$	88.76	$< 10^{-4}$	$< 10^{-4}$
8	$(m_0 + m_1 l)\cos(t + \tau) + b_0 + \sigma_b + \sigma_\varepsilon$	89.48	$< 10^{-4}$	$< 10^{-4}$
9	$b_0 + b_1 l + \sigma_b + \sigma_\varepsilon$	580.9	$< 10^{-4}$	$< 10^{-4}$
10	$b_0 + \sigma_b + \sigma_\varepsilon$	590.0	$< 10^{-4}$	$< 10^{-4}$

Table 3. *Model averaged coefficients for models 1 and 2 from table 2.*

		coefficient	s.e.m.	z-value	p-value
magnitude of seasonal fluctuation	m_0	0.626	0.281	2.19	0.0285
slope for this by latitude	m_1	-0.00349	0.00695	-0.502	0.6157
offset of seasonal peak	τ	-0.241	0.00993	-24.249	0.0000
linear intercept	b_0	0.0890	0.230	0.387	0.6985
linear slope	b_1	0.0277	0.00572	4.845	$< 10^{-4}$

Although these curves were fitted to data collected from November until March, there are no other directly comparable sources of data for the other portions of the year. Although we chose the cosine function for mathematical simplicity in describing seasonal variation, the extrapolation of the cosine function into the summer months does agree with anecdotal information, because house finches exist as mated pairs throughout the summer and do not feed as intensively at bird feeders. We will use the extrapolation, acknowledging that we do this in the interests of a case-motivated examination of the implications of the effects of social interaction on disease dynamics, and not as a final version model fitted to data nor necessarily the best choice for empirical representation of social aggregation. Figure 2*a* shows the fitted curve for 42.5° N (Ithaca, NY) and 33.8° N (Atlanta, GA).

(c) *Seasonal aggregation and disease transmission*

Because seasonal aggregation at bird feeders increases during the winter, well away from the breeding season, it seems clear that this aggregation is not responding directly to population abundance, so we include this aggregation in the model as a seasonally forced frequency-dependent transmission. However, we also believe that the influx of susceptibles at the end of the breeding season plays an important role in the transmission of the disease, which suggests that there is also a density-dependent transmission component. Rather than trying to use a complex form that compromises between the two types of transmission (Pascual *et al.* 2002; Roy & Pascual 2003), we have chosen to regard the two as additive. Biologically, we interpret this to mean that transmission at feeders depends mostly on how many birds aggregate at such a site, which depends more on behaviour than population abundance, thus we model it as a frequency-dependent process. In addition to transmission at feeders, however, there is transmission in the general environment, which does respond to

population abundance. Equation (3.4) outlines this transmission term, $\beta(t,l)$:

$$\beta(t,l)S(I_1 + I_2) = \lambda S(I_1 + I_2) + \phi(\Phi(t,l) - 1)\frac{(I_1 + I_2)}{N}S$$

$$\beta(t,l) = \lambda + \phi\frac{\Phi(t,l) - 1}{N}. \qquad (3.4)$$

λ represents the constant density-dependent transmission rate. This is the rate of contact between susceptibles and infectious birds in the general environment. ϕ is the fitted social aggregation function, explained above. We subtract one in order to not count oneself in the contact rate, times a constant of proportionality (ϕ), times prevalence, times the total abundance of susceptibles. Thus, these factors all together represent the rate of contact between susceptible and infectious birds at the bird feeder and, in fact, both forms of transmission are necessary if the model is to respond to both seasonal drivers.

4. RESULTS

Figure 3 shows the model output for seasonal variation in prevalence across 2 years, analogous to the data in figure 1. Although it is not an exact fit, the key qualitative features are captured. There is an early autumn peak, caused by the influx of susceptibles from the seasonal breeding. A mid-winter trough follows this peak, as the infected birds recover. Then, a late-winter resurgence occurs as the seasonal aggregation increases the transmission rate sufficiently to create a second epidemic among the recovered birds. Prevalence is higher in the south than the north, owing to the longer breeding season, and thus there is a larger pool of susceptibles in the south. Additionally, the autumn peak occurs earlier in the south than in the north. These observations are true for both the empirical observations (figure 1) and the model output (figure 3). To distinguish what is causing this pattern in the model, and thus what could be causing the pattern in the
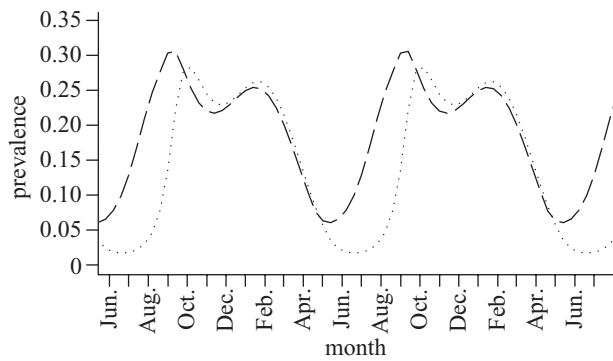
Figure 3. Model output prevalence over time at equilibrium for full model, producing semi-annual pattern. Dotted line represents fits for Ithaca, NY ($l = 42.5°$ N), dashed line Atlanta, GA ($l = 33.8°$ N).

wild population, we will examine what happens as we subtract mechanisms from the model.

Figure 4 shows the effect of each seasonal component operating separately. We replace $b(t,l)$ with a temporally constant $b(l)$, or postulate constant breeding (figure 4a), thus we only see an annual cycle directly caused by the seasonal aggregation, with only a winter peak in prevalence. If we reverse the procedure, and replace $\beta(t,l)$ with a temporally constant $\beta(l)$, or postulate constant transmission, this implies no seasonal variation in aggregation (figure 4b). In this case, we only see an annual cycle caused by the influx of susceptibles, with a late summer–early autumn peak. In neither case do we obtain the semi-annual pattern seen in the full model as well as the data, implying that both forms of seasonal variation are necessary.

Figure 5 shows the effect of latitudinal variation operating separately on each process. We replace $b(t,l)$, with a function $b(t)$ with putative latitude fixed at a central value (figure 5a), demonstrating that latitudinal variation in the timing and length of the breeding season drives the timing of the autumn peak in prevalence, because without this variation there is no change in the timing of the autumn peak. By contrast, when we replace $\beta(t,l)$, with a function $\beta(t)$ with putative latitude fixed at a central value (figure 5b), leaving the $b(t,l)$ function in place, we can see that latitudinal variation in aggregation largely affects the second, early spring peak. Without latitudinal variation in social aggregation, the spring peak barely exists in the north, and is too dramatic in the south.

Finally, figure 6 shows the effect of varying levels of immunological efficacy, with both seasonal breeding and aggregation in the model. Here, we see that partial immunity is key for the late winter resurgence of the disease. Full immunity reduces to a single annual peak caused by annual births, although with no contribution to transmission from the recovered class the peak is delayed until there is some interaction with the increased transmission from seasonal aggregation (figure 5a). Without any immunity, the increase in transmission from seasonal aggregation dominates the disease dynamics, although there is a shoulder on the peak from the influx of susceptibles because of seasonal breeding (figure 5c). Thus, partial immunity moderates the effects of seasonal aggregation, which allows the effects of seasonal breeding to be more prominent. Additionally, the presence of seasonal breeding, combined with the
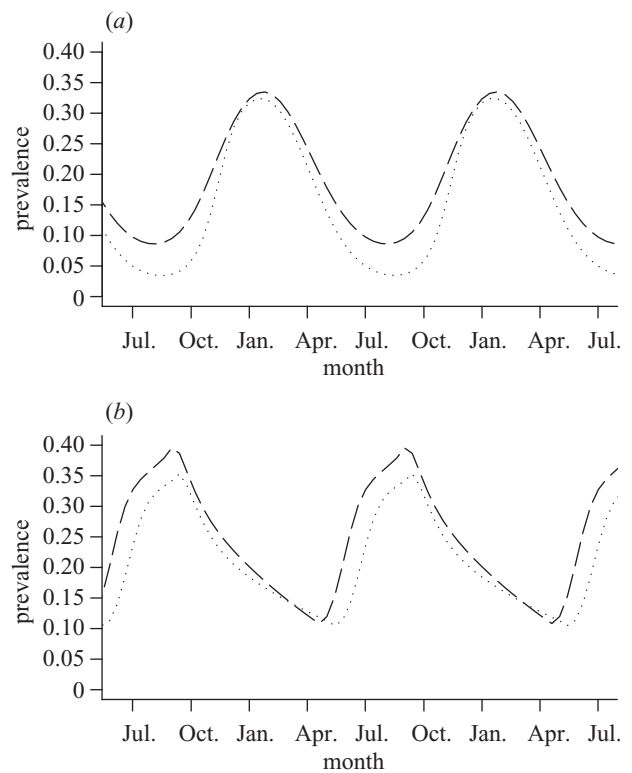

Figure 4. (a) Time-series of prevalence against month of year after transient, for model with only seasonal aggregation (constant breeding). (b) Time-series of prevalence against month of year after transient for model without aggregation (constant transmission). Dotted line represents fits for Ithaca, NY ($l = 42.5°$ N), dashed line Atlanta, GA ($l = 33.8°$ N).

moderation from partial immunity, delays the second peak from mid-winter to late winter.

To clarify how these factors interact in the dynamics of the disease, we can look beyond prevalence to the abundance output in figure 7. Here, we see that the initial peak in prevalence is launched by the influx of new susceptibles becoming infected for the first time (I_1). These susceptibles then spread the disease throughout the population. The late winter peak in prevalence, by contrast, is entirely composed of I_2 individuals, and is caused by the upsurge in transmission owing to seasonal aggregation.

5. DISCUSSION

Consistent with empirical patterns described from long-term citizen science monitoring studies of the house finch–MG system (figure 1; Altizer *et al.* 2004a), our model has captured the major features of the seasonal and latitudinal variation in dynamics of the house finch–MG system. Three main features of house finch biology drive these sustained fluctuations in prevalence: seasonality of breeding, annual changes in social aggregation and the partial immunity of finches to MG. The two seasonal processes can each cause a single annual peak, but only together can they create the semi-annual peaks because the two processes are out of phase. However, full immunity to MG would dampen these cycles too much to allow both seasonal processes to affect the disease dynamics sufficiently to cause the empirically observed semi-annual pattern. Matching this observed pattern at both northern and southern latitudes requires the
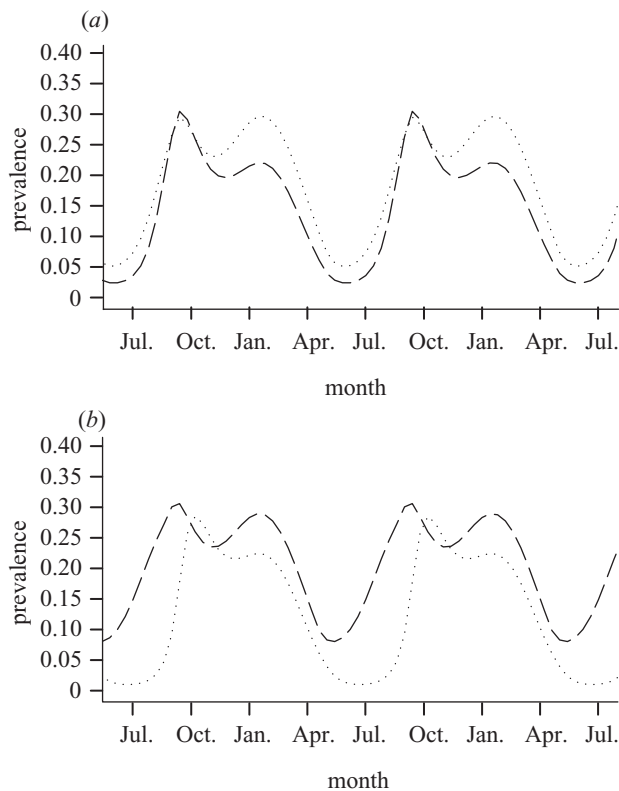
Figure 5. (*a*) Time-series of prevalence against month of year after transient for model with latitudinal variation in social aggregation function, and not in breeding (fixed *l* = 38.15). (*b*) Time-series of prevalence against month of year after transient, for model with latitudinal variation in birth function, and not in social aggregation (fixed *l* = 38.15). Dotted line represents fits for Ithaca, NY (*l* = 42.5° N), dashed line Atlanta, GA (*l* = 33.8° N).

Figure 6. Time-series of prevalence against month of year after transient for with (*a*) full immunity ($\chi = 0$), (*b*) partial immunity ($\chi = 0.6$), (*c*) without immunity ($\chi = 1$). Dotted line represents Ithaca, NY (*l* = 42.5° N), dashed line Atlanta, GA (*l* = 33.8° N).

incorporation of latitudinal variation in these seasonal processes.

Thus our model suggests that the seasonal pulse of new susceptibles that enter the population as the breeding season ends cause the initial autumn peak; whereas seasonal aggregation of partly immune individuals creates a second late winter peak (figure 1). This implies that host behaviour could be extremely important for understanding the seasonality of wildlife disease dynamics. Bird feeders, a key resource that allows the over-winter survival of house finches, amplify the 'natural' seasonal aggregation that makes the birds more prone to disease transmission. The latitudinal variation in social aggregation suggests that this is more important in the more severe winters of northern latitudes than in southern latitudes. Seasonal breeding, the norm rather than the exception for wildlife, can be a key driver of transmission as it causes the main influx of new susceptibles into the host population. Here, latitudinal variation in climate can affect how the timing of seasonal breeding drives the timing of seasonal variation in disease. Of course, if vertical transmission plays a larger role in disease transmission, seasonal breeding may still have profound, but substantially different dynamical effects.

In this system, 'out of phase' seasonality maintains a regime of persistent, regular disease outbreaks, which would reduce to a low, constant endemic level of disease
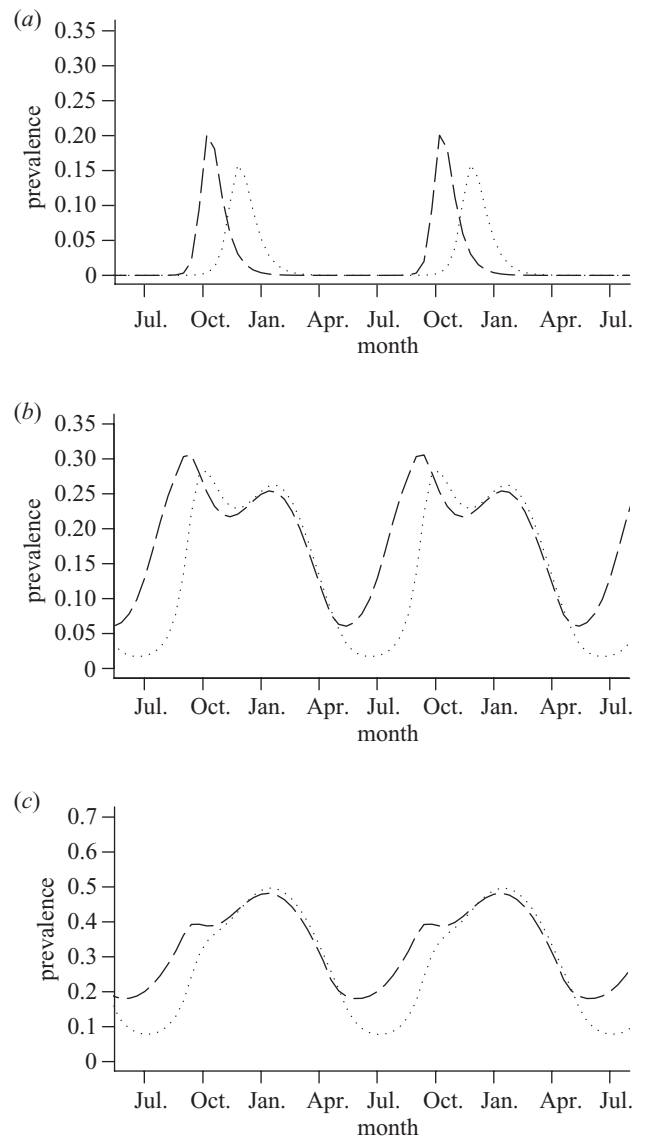
without seasonality, similar to standard SIR models (Anderson & May 1979). By contrast, Swinton *et al.* (1998) find that 'in phase' seasonal aggregation, such as occurs when seals haul out to mate and give birth, reduces the ability of phocine distemper to persist in seal populations. In addition to differences in seasonality, however, seals live much longer, and appear to retain a more complete immunity to phocine distemper for longer, than house finches do concerning MG. If the finches had full immunity to MG, it would be doubtful that the disease would persist, at least without local fadeouts (figure 6*a*). Thus, it is the interaction between forms of seasonality and immunity that drives the nature of both of these disease dynamics.

Although understanding seasonal dynamics in human population has proven key to understanding multi-annual dynamics (Bjørnstad *et al.* 2002; Keeling & Grenfell 2002), perhaps because the social forcing mechanism (school
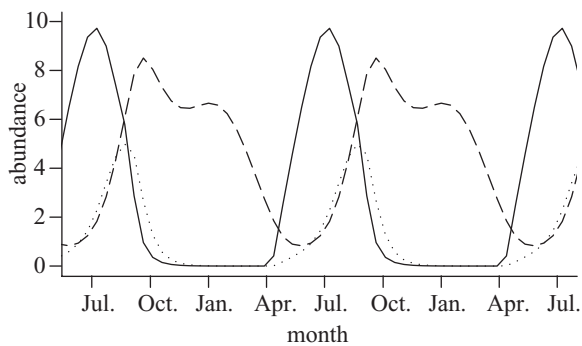
Figure 7. Time-series of abundance against month of year after transient, for Atlanta, GA ($l = 33.8°$ N), corresponds exactly to Atlanta prevalence from figure 3. Solid line for S (susceptibles), dotted line for I_1 (initial infecteds), and dashed line for I_2 (reinfecteds).

terms) seems so unbiological, the implication of seasonal variation in social habits of wildlife have generally been missed. Although, clearly seasonal drivers will have very strong effects on disease dynamics. Because disease-causing organisms usually reproduce on much faster time-scales than their hosts, the seasonal variation in the host behaviour will influence the dynamics of the disease. Although we have not investigated multi-annual dynamics here, the data suggest (Altizer *et al.* 2004*a*), and model can produce dynamics with some regular inter-annual variability. However, a longer time-series may be necessary than is currently available, given the recent emergence of the disease, to fully understand the nature, role and causes of multi-annual dynamics.

Factors that vary with latitude, such as climate, can be expected to influence seasonal drivers of disease dynamics. Because we were able to include latitudinal variation from our statistical models of seasonal drivers in our dynamical model, we were able to demonstrate that latitudinal variation in these seasonal drivers could lead to the latitudinal variation we empirically observe in the disease dynamics. This ability to make a qualitative match across this latitudinal variation strengthens our hypotheses about how seasonality affects house finch–MG system, but also demonstrates more generally how latitudinal variation of host life history can affect disease dynamics.

The mechanisms presented here might not be the only ones driving the pattern of dynamics in the house finch–MG system. In particular, seasonal variation in immunocompetence could be very important. Although our group is investigating this in house finches (K. M. Lindström, D. Hawley, A. Davis and M. Wikelski, unpublished data), there are currently few data available for this species. However, there is some general evidence that immunocompetence can be higher in the breeding season than in the non-breeding season in birds (Hasselquist *et al.* 1999; Møller *et al.* 2003). Yet, this factor is likely to be seen in the transmission term, similar to the social aggregation model we did present. Thus, although we assume that social aggregation is the most parsimonious mechanism driving the winter peak, its amplitude may also be attributed to and enhanced by seasonal variation in immunocompetence. Thus poor food quality, harsh weather or other forms of stress during winter could increase the probability of a

susceptible or partly immune individual catching the disease from infected individuals, or in the case of MG recrudescing. All of these mechanisms will interact with higher contact rates at times of greater social interaction in winter.

We did vary the immunity sub-model, and examined the role of a temporary, but long-lasting gamma-distributed immunity (Lloyd 2001). We did not find any effect of such a loss of immunity on the dynamics, because the relatively short lifespan of house finches does not allow for a sufficient portion of the population to return to the susceptible class. Similarly, for simplicity, we presumed a closed population, although this is not the case for local finch populations. Eastern house finches are partial seasonal migrants (Able & Belthoff 1998), and a consistent portion of our field sample seems to consist of true transients (Faustino *et al.* 2004). However, an open population model would require a good understanding of the disease status of the immigrants, which we lacked. Linking a series of the models presented here into a meta-population may provide additional insights into how the disease works at different spatial scales.

Our modelling work has also helped drive the field studies. Collection of data on summer breeding was begun precisely because early versions of this model suggested that seasonal breeding was such an important driver. Although perhaps intuitive in hindsight, the idea that breeding data would be important to disease dynamics was highlighted by our modelling efforts. We are planning efforts at understanding the social networks of house finches in local field populations to gain a better understanding of how social behaviour changes over the season, and may involve important heterogeneities between individuals.

Altizer *et al.* (2004*b*) have demonstrated that juvenile (hatch year) house finches are more likely to be found diseased than birds that survived the previous winter during the autumn. This agrees with what our model would predict from figure 7, that adult finches, assuming almost all have already had the disease and recovered, would be more resistant to the disease on re-exposure.

In conclusion, our model captures essential features of the dynamics of the house finch–MG system, and identifies major mechanisms underlying seasonality of this and potentially other wildlife diseases. Although additional refinements, particularly a better understanding of seasonal immunity, could improve our model and our understanding of the disease, the importance of seasonal factors in driving the dynamics of this and other wildlife disease seems clear. Because seasonal factors appear to be so important in disease dynamics, it appears plausible that they may also play an important role in disease emergence.

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