Theoretical Ecology
https://doi.org/10.1007/s12080-019-0423-6

ORIGINAL PAPER

Ecological determinants of pathogen transmission in communally roosting species

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Received: 16 July 2018 / Accepted: 6 March 2019

Abstract

Many animals derive benefits from roosting communally but may also face increased risk of infectious disease transmission. In spite of recent high-profile disease outbreaks in roosting animals of conservation and public health concern, we currently lack general theory for how attributes of roosting animals and their pathogens influence pathogen spread among roosts and overall population impacts on roosting species. Here we develop a model to explore how roost size and host site fidelity influence the time for a pathogen to escape from its initial roost, overall infection prevalence, and host population size, for pathogens with density- or frequency-dependent transmission and varying virulence. We find that pathogens spread rapidly to all roosts when animals are distributed among a small number of large roosts, and that roost size more strongly influences spread rate for density-dependent than frequency-dependent transmitted pathogens. However, roosting animals that exhibit high site fidelity and distribute among a large number of small roosts are buffered from population-level impacts of pathogens of both transmission modes. We discuss our results in the context of anthropogenic change that is altering aspects of roosting behavior relevant to emerging pathogen spread.

Keywords Animal aggregations · Communal roost · Pathogen transmission · Roost size · Site fidelity

Introduction

Animal aggregations occur in a wide variety of taxa, at varying spatial and temporal scales, and represent some of the most striking patterns of emergent behavior in natural systems (Parrish and Edelstein-Keshet 1999). Communal roosts, here defined as aggregations of conspecifics during the inactive period of the diurnal cycle (Grether et al. 2014), have been documented in many species of mammals (Kunz 1982; Hamilton 1982; Thompson 1989), fish (Clough and Ladle, 1997), birds (Eiserer 1984; Beauchamp 1999), and invertebrates (Mallet 1986; Grether and Switzer 2000). Roosts can range in size from just a few individuals to spectacular aggregations of millions of animals. Roosts vary substantially within and among species in attributes including their duration and seasonality (Eiserer 1984; O'Shea and Bogan, 2003; Kunz 1982), spatial reliability of their location (Yadon 1956; O'Donnell and Sedgeley 1999), and the degree to which individuals intermix between roosts to form "roost systems" (Leyrer et al. 2006; Grether and Switzer 2000; Laughlin et al. 2014). Given that roosts provide important ecological functions such as "nutrient hotspots" from guano (Duchamp et al. 2010) and ecosystem services such as pest control (Cleveland et al. 2006), understanding how these roost attributes influence the population dynamics of roosting species, and their ecological interactions, is a question of practical importance.

In previous work, we explored how different roost dynamics can emerge from different aggregative behaviors (Laughlin et al. 2014). By changing the strengths of roost-site fidelity (the tendency for an individual to return to its previous roost) and conspecific attraction (the tendency for an individual to

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Published online: 13 March 2019

group together with conspecifics), different roost systems emerged including systems with many small roosts and low individual fidelity, few large roosts and high fidelity to them, and several systems in between these extremes. Roost dynamics can thus be more simply described in two dimensions: the size of the roosts and site fidelity.

Communal roosting can provide benefits such as decreased individual predation risk (Hamilton 1971), information sharing (Ward and Zahavi 1973), and thermoregulation (Eiserer 1984), but these aggregations can also lead to negative outcomes such as facilitating transmission of pathogens and parasites (Krause and Ruxton 2002; Altizer et al. 2006). For example, Phocine distemper virus is spread among Harbor seals (*Phoca vitulina*) at their haulouts where seals aggregate seasonally on sea ice or beaches (Swinton et al. 1998), occasionally leading to large outbreaks. Moreover, roosts have been implicated as transmission hubs for emerging pathogens that cause mass mortality of their hosts, as observed in North American songbirds following the introduction of West Nile virus (Diuk-Wasser et al. 2010; Dawson et al. 2007) and in cave-roosting bats infected with the fungus causing White-nose syndrome (Frick et al. 2010; Langwig et al. 2015; Lorch et al. 2013).

Human activity can profoundly alter local and regional dynamics of roosting species with consequences for infection risk. Human-made structures could provide new roosting habitats or create barriers to inter-roost movement. Food subsidies in the form of crops and exotic plantings could increase the duration of roost site occupancy, roost size, and site fidelity of individuals, increasing the likelihood of spillover to domestic animals and humans. For example, flying foxes (*Pteropus* spp.) in SE Asia are attracted to palm oil cultivation, and contact with their feces is causing human cases of Nipah virus with high fatality (Deka & Morshed 2018). The formation of semi-permanent large roosts of flying foxes in urban and suburban Australia has led to local outbreaks of Hendra virus in horses and humans (Plowright et al. 2011) and has led to calls for these roosts to be forcibly removed. Understanding the consequences of anthropogenic change in roosting behavior is thus a pressing question of public health and conservation concern.

Standard epidemic theory suggests that locally, within a roost system, roost size could influence pathogen invasion, as well as the size and duration of a local epidemic. How roost size relates to pathogen invasion and persistence depends critically on the transmission mode. Importantly, attributes of roosting behavior and/or roost structure could alter contact structure for close contact-transmitted pathogens. For example, roosting structures such as trees could distribute animals into smaller sub-groups leading to frequency-dependent transmission, in which the probability of a susceptible becoming infected is independent of total roost size, whereas roosts where large numbers of individuals are densely packed, such as large caves, could result in density-dependent transmission. Additionally, the daily rate of movement between roost sites (a function of individual site fidelity and roost size) will determine rate of spatial spread within a roost network. While models have been developed to explore drivers of disease in focal host-pathogen systems (Plowright et al. 2011; Maher et al. 2012), to date we lack general theory about how attributes of roosts (e.g., size), hosts (e.g., site fidelity), and pathogens (e.g., transmission mode and virulence) interact to determine pathogen impacts and spatial spread within a network of roosts.

In this study, we developed simple deterministic and stochastic models to explore different aspects of pathogen dynamics in communally roosting host species. We developed an aggregated deterministic model in which we calculated the time it took for the disease to escape a roost under different roost size and fidelity rate combinations, and under different pathogen transmission modes. Because time and population size are treated as continuous variables in the deterministic model and because such models are known to not accurately predict dynamics in small populations, we developed a stochastic individual-based model to further explore the effects of, and interactions between, population size, roost size, roost-site fidelity, pathogen transmission mode, and virulence on the spread of an infectious disease through a population. The modeling approach we adopt here is structurally similar to other models of disease spread among patches linked by dispersal (e.g., Arino et al. 2005, McCormack and Allen 2007), which are usually applied over multiple host generations to determine conditions for endemic persistence of pathogens. In our analysis, however, we quantify the time until the pathogen establishes in a second roost and the effect of the disease on seasonal survival as well as infection prevalence at the end of the nonbreeding season in order to focus on understanding how roost size and roosting behavior affects the transient dynamics of pathogen colonization following introduction during a single nonbreeding season.

Deterministic model

Model development

Here we develop a model for a roosting species during a nonbreeding period, and for simplicity assume that nonbreeding mortality during this period is negligible in the absence of infection. The total population (size N) distributes roughly equally among r roosts, so that in the absence of infection, the expected population size in each roost is $N_r = N/r$. All individuals of the roosting species have site fidelity, F, defined as the probability that an

individual stays in its original roost for the duration of the nonbreeding season. Thus, if the initial population size at roost j is $N_j(0)$ and the nonbreeding season has length T_{NB}, the expected number of original roost members remaining at the end of the season is:

$$N_j(T_{NB}) = FN_j(0). \quad (1)$$

In a disease-free roosting population distributed across r roosts, we use differential equations to describe the among-roost movement dynamics. Individuals depart roosts at a per capita rate m, and dispersers from each roost redistribute equally among the remaining $r-1$ roosts. This can be expressed by the following equation:

$$\frac{dN_j}{dt} = -mN_j + \frac{m}{r-1}\sum_{k\neq j}N_k \quad (2)$$

We can relate the per capita movement rate, m, to site fidelity, F, by solving Eq. (2) in the absence of new colonizers; the number of original roost members remaining at their roost throughout the nonbreeding season is then $N_j(T_{NB}) = N_j(0)e^{-mT_{NB}}$. Equating this to expression (1) and rearranging yields the following expression for the movement rate:

$$m = -\frac{\ln(F)}{T_{NB}} \quad (3)$$

Now suppose a pathogen invades from which there is no recovery or immunity from infection, so that individuals at roost j can be classified as susceptible to or infected by the pathogen (with respective numbers S_j and I_j). The pathogen has a transmission rate per infected individual β_j, where roost structure and/or pathogen properties determine whether the transmission rate is density dependent ($\beta_j = \beta_d S_j$) or frequency dependent ($\beta_j = \beta_f S_j/N_j$). Pathogen infection results in pathogen disease-induced mortality at rate, so that the expected infectious period of an infected host is $1/v$. The dynamics at each patch ($j = 1. . r$) are described by the following equations:

$$\frac{dS_j}{dt} = -\beta_j I_j - mS_j + \frac{m}{r-1}\sum_{k\neq j}S_k \quad (4a)$$

$$\frac{dI_j}{dt} = \beta_j I_j - vI_j - mI_j + \frac{m}{r-1}\sum_{k\neq j}I_k \quad (4b)$$

Conditions for pathogen invasion and inter-roost spread

Prior to pathogen introduction, each roost is at its disease-free equilibrium size Nr. We make the following heuristic argument to derive an approximate condition for pathogen invasion (i.e., for infection to increase after initial introduction to a wholly susceptible roost). If the pathogen has relatively high virulence compared to the movement rate ($v > m$), then the infectious individual is expected to spend its entire infectious period in the initial roost, so an upper bound for the lifetime number of cases caused by the index case is β_j/v. Alternatively, if the pathogen has relatively low virulence and/or the movement rate is high ($m > v$), the expected number of roosts the index case visits is the ratio of the infectious period ($1/v$) to the expected residence time in each patch ($1/m$) = m/v. In each patch that the infected individual visits, the total number of infections caused by the index case before it departs is β_j/m. The total number of infected cases caused by the index case in the roost network is the product of the expected number of roosts visited and the number of cases per roost, i.e., $=(m/v)\times\ \beta_j/m\ =\ \beta_j/v$. The threshold for sustained transmission is that the number of new cases caused by the index case exceeds one, in other words $\beta_j > v$. This threshold is independent of initial roost size and overall population size for frequency-dependent transmission ($\beta_f > v$), but scales linearly with roost size and overall population size for density-dependent transmission ($\beta_d N_r > v$). Notably, the threshold for pathogen invasion is independent of site fidelity, but site fidelity does influence the rate of spread between patches. To quantify this, we derive a simple approximation for the expected time taken for one infected individual to leave a roost colonized by one infected individual, T_E. Initially, infection within a roost increases approximately exponentially, so that

$$I_j(t) \approx e^{(\beta_0 - v)t} \quad (5)$$

The approximate number of infected individuals leaving the roost by time τ is approximately the integral of the departure rate of infecteds, mI_j, from 0 to τ. Hence, we derive T_E as the time at which this integral equals 1:

$$1 = \int_0^{T_E} me^{(\beta_0 - v)t}dt = \frac{m}{\beta_0 - v}\left[e^{(\beta_0 - v)T_E} - 1\right] \quad (6)$$

Note that if the infection rate ($\beta_0 - v$) is low (e.g., because the pathogen is not very contagious, or the contact rate is low, which for density-dependent transmission occurs when the total population size is low and/or the number of roosts is large), a first-order approximation of the exponential yields the expected result that $T_E \approx 1/m$, the average residence time of one infected individual in the roost. We can rearrange the expression above to yield the following approximate expression for the time for the pathogen to escape the initially infected roost site:

$$T_E \approx \frac{1}{\beta_0 - v}\ln\left(\frac{\beta_0 - v}{m} + 1\right) \quad (7)$$

Individual-based stochastic model

Model description

In order to more accurately explore the relationships between roost characteristics and parasite traits in roosts of varying, sometimes small, sizes, we constructed an individual-based, stochastic analogue of the deterministic model that simulates the formation of roosts and subsequent pathogen transmission. Initially, we create r roosts. Each roost has an initial population size drawn from a Poisson distribution with mean $N_r = N/r$, where $N = 1000$ is the approximate total population size. We use three levels of roost sizes; *small* roosts have expected size 40 (spread among 25 roosts), *medium* roosts are size 100 (10 roosts), and *large* roosts are size 333 (3 roosts). Here, roost size refers to initial conditions, and roost sizes naturally fluctuate within the stochastic model. The model simulates roost formation for $T_{NB} = 200$ time steps (approximating the number of days in a typical nonbreeding season for many vertebrate species). Individuals can be in one of two possible states: susceptible (S) or infected (I). At model initiation, all individuals are designated as S except for one randomly selected individual designated as I.

To relate the rates in the deterministic model to daily transition probabilities in the stochastic model, we used the following rules. For transitions with constant per capita rates (movement, m and disease-induced mortality, v) the daily probabilities were obtained by direct integration. The daily probability of not changing roosts, p(stay) is thus e^{-m}, which can be related to season-long site fidelity by Eq. (3) to give

$$\mathrm{p(stay)} = \frac{1}{F^{T_{NB}}} \tag{8}$$

Individuals move with probability $1 - \mathrm{p(stay)}$ and are reassigned randomly to a new roost with equal probability.

The daily probability of surviving infection is related to the per capita disease induced mortality rate, via

$$\mathrm{p(survive)} = e^{-v} \tag{9}$$

and the probability of dying is $1 - \mathrm{p(survive)}$.

After individuals have been assigned a roost for that time step, pathogen transmission occurs. Infection occurs only within roosts; the mean number of infected individuals that a susceptible encounters each night at roost j (such that it would become infected), λ_j, is related to the per susceptible transmission rate in Eqs. (4a) and (4b), so that under density-dependent transmission,

$$\lambda_j = \beta_d I_j \tag{10a}$$

and under frequency-dependent transmission,

$$\lambda_j = \frac{\beta_f I_j}{N_j} \tag{10b}$$

At the end of each day, within each roost, the probability of a susceptible becoming infected is the probability that the susceptible encountered more than zero infected individuals. We assume that the number of infected individuals encountered is drawn from a Poisson distribution with mean λ_j as defined above, so that

$$P(S_j \rightarrow I_j) = 1 - e^{-\lambda_j} \tag{11}$$

A summary of model parameters, their definitions, and default values are given in Table 1.

Model analysis

For each parameter combination and transmission mode, we ran 100 simulations to calculate average and variance. We calculated median and upper/lower quartiles of the daily prevalence of infection over the season (proportion of all infected individuals) for each parameter combination of interest and for each transmission mode. We calculated the number of days it takes for the pathogen to spread from the initial roost to a new roost, analogous to escape time from the deterministic model. We defined this as the number of days it took for a second roost to become infected with at least four individuals to account for stochasticity of infection. Finally, we calculated mean and confidence intervals for population size and prevalence under different levels of virulence.

Results

Escape time (time until inter-roost spread)

The deterministic model predicts that under density-dependent transmission, the time taken for an infected individual to move from the roost where initial infection occurred (T_E) increases faster than linearly with increasing roost site fidelity and decreases with roost size (Fig. 1a). Under frequency-dependent transmission, however, T_E only increases with site fidelity, and roost size is not predicted to have an effect (Fig. 1b). Analogously in the simulation model, with density-dependent transmission, the disease was able to spread to another roost within 200 days in 80–100% of runs except when fidelity was high ($F = 0.95$) and roosts were small (in this case, we saw colonization of a second roost in only 43% of runs; Fig. 2c). Roost size influenced the time it took for infection of a second roost. Larger roosts facilitated faster spread of the disease, and this effect was exaggerated as fidelity increased (Fig. 2a–c). Under frequency-dependent transmission with low or medium fidelity, roost size had no effect on the mean time to infect a second roost (Fig. 2d, e). However, as fidelity increased to nearly 100% ($F = 0.95$), roost size appeared to also influence the spread rate in a

Table 1 Parameters used in the analytical and simulation models

Parameter	Description	Value
Roosting species attributes		
N	Total population size (individuals)	~1000
N_r	Roost size (expected number of individuals at each roost)	Small (~25 at 40 roosts), medium (~100 at 10 roosts), large (~333 at 3 roosts)
F	Site fidelity (probability that individual stays in one roost for entire season)	Low (0.05), medium (0.5), high (0.95)
m	Movement rate (among roosts) used in deterministic model	Calculated from F: $m = -\ln(F)/T_{NB}$
T_{NB}	Length of roosting season	200 days
Pathogen attributes		
β_d	Transmission rate (density dependent)	0.002
β_f	Transmission rate (frequency dependent)	0.2
v	Disease-induced mortality rate	Default 0.01 (range 10^{-3} to 1)

frequency-dependent transmission mode, counter to the deterministic model predictions (Figs. 1b and 2f).

Infection prevalence and system-wide spread

Roost size greatly influenced the rate of disease transmission at the population-level under density-dependent transmission (Fig. 3a–c). In *large* roosts, (average of 333 individuals in a population of 1000), the disease spread throughout the population within 25 days, whereas in *medium* roosts (average roost size of 100) it took on average 75 days. In *small* roosts (average roost size of 40), the median prevalence rarely reached 100% of the population in 200 days. At higher fidelity to roost sites, $F = 0.5, 0.95$, under density-dependent transmission, the spread of the disease slowed under all scenarios, but more so in medium and small roosts, similar to the deterministic model predictions. Under frequency-dependent transmission, roost size did not influence the rate of pathogen transmission when fidelity was low ($F = 0.05$; Fig. 3d). As fidelity increased, however, roost size did influence transmission dynamics: in smaller roosts, transmission rate decreased more than in larger roosts (Fig. 3e, f). In both transmission modes under high rates of fidelity ($F = 0.95$), prevalence of the pathogen first saturated at the initially infected roost and the rate of increase in overall prevalence dropped until another roost became infected, as evidenced by the stair-step pattern in large roosts in Fig. 3c, f. Under these high rates of fidelity, transmission mode had a smaller influence than fidelity in influencing the rate of disease spread, whereas under lower fidelity, transmission mode dominated the disease dynamics.

Effect of pathogen virulence on pathogen invasion and impacts on the host population

As pathogen virulence varied over orders of magnitude, mean infection prevalence decreased monotonically to zero across all combinations of roost size, site fidelity, and transmission mode (Fig. 4). For density-dependent transmission, overall prevalence increased with roost size and decreased with site fidelity, such that small and medium roosts were unable to sustain transmission of highly virulent pathogens when fidelity was high (Fig. 4a–c). By contrast, for frequency-dependent transmission, roost size had a weaker effect on prevalence of moderately virulent pathogens, but analogous to density dependence, high site fidelity prevented invasion of virulent pathogens in small- and medium-size roosts (Fig. 4d–f).

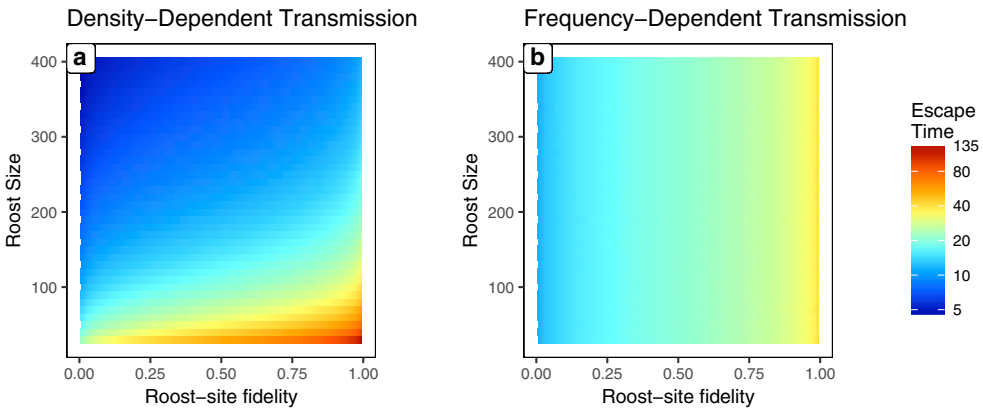

Fig. 1 Heatmap showing the "escape time" in days, i.e., the expected time taken for one infected individual to leave a roost colonized by one infected individual, T_E, from the deterministic model

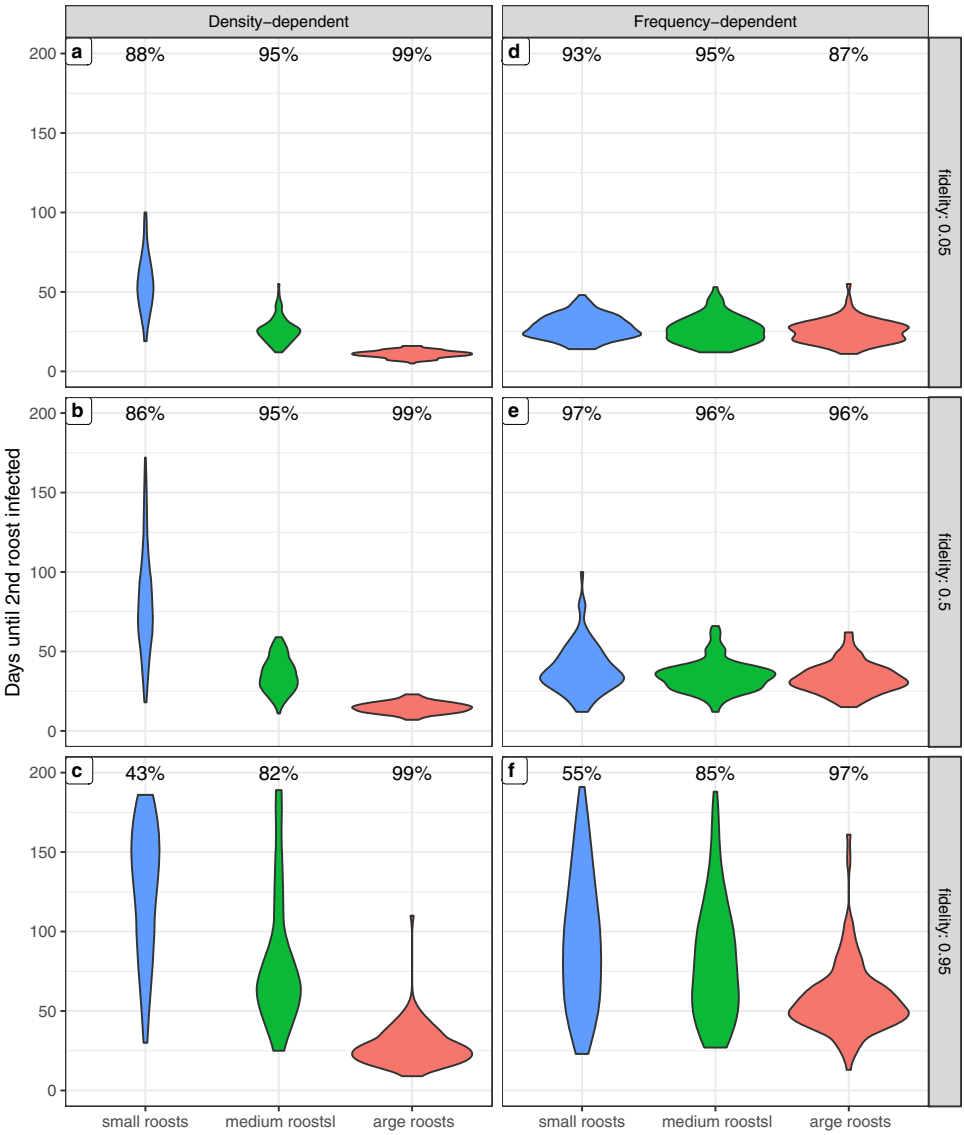

Fig. 2 Violin plots showing the distributions of number of days before a second roost becomes infected under different roost sizes and transmission modes, generated from 100 simulations for each parameter combination in the individual-based stochastic model. The number above each plot is the percentage of simulations in which infection of a second roost occurred within 200 days

These patterns were also reflected in the population impacts of pathogen invasion, where all populations showed a *U*-shaped response to infection of pathogens with increasing virulence. The largest reductions in population sizes occurred at intermediate virulence and in large roosts (Fig. 5). High site fidelity lowered the maximum impact of the pathogen on overall population size for medium and large roosts; however, large roosts always experienced high mortality for pathogens with virulence between 0.01 and 0.1 (corresponding to infectious periods of approximately 100 and 10 days, respectively).

Discussion

Motivated by recent high-profile disease outbreaks in a variety of communally roosting species, we investigated how properties of roosting hosts (roost size and site fidelity) and pathogens (transmission mode and virulence) influenced infection spread and impact through a system of roosts connected by dispersal. Our deterministic model predicted that increasing site fidelity of hosts should result in faster than linear increase in the escape time of the pathogen from the initially infected roost, and that roost size should affect the rate of inter-roost spread for density-dependent, but not frequency-dependent pathogen transmission. These results were largely borne out in a simulation model; however, interactions between demographic stochasticity and roost size resulted in a lower probability of inter-roost pathogen spread for both transmission modes in highly site faithful roosting species. Furthermore, high site fidelity reduced the invasion probability and population-level impacts of virulent pathogens except when hosts were distributed among a small number of large roosts. These results suggest that animals that

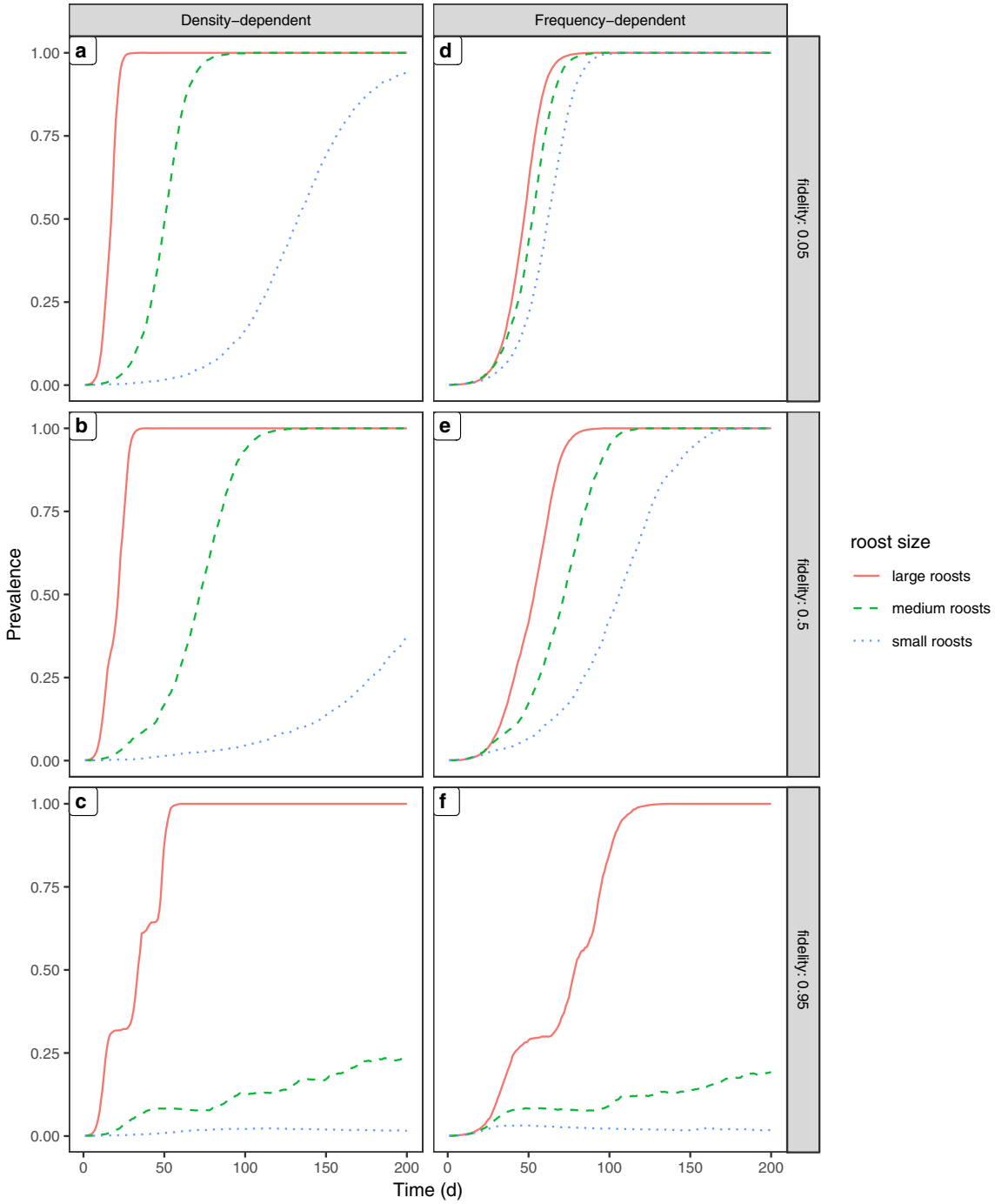

Fig. 3 Median and upper/lower quartiles of prevalence of infection over time under different roost sizes, rates of fidelity, and transmission modes from the individual-based stochastic model. Solid line is large roosts, dashed line is medium roosts, and dotted line is small roosts

form smaller roosts and have high site fidelity to these roosts may be buffered against the spread and impact of emerging pathogens.

Our finding that large roosts promote pathogen spread even when site fidelity is high is especially relevant in the context of ongoing anthropogenic change that alters roosting behaviors. Loss of natural roost sites, creation of large artificial roosting structures such as bridges, and attraction to abundant and seasonally stable resources associated with agriculture and cities cause some species to aggregate into super-roosts (Neubaum et al. 2007, Daoud-Opit & Jones 2016). This is especially concerning for multi-host pathogens, where proximity of roost sites to humans and domestic animals has resulted in disease outbreaks in wildlife (Daszak et al. 2000) as well as zoonotic spillover into humans (Plowright et al. 2011). Thus, anthropogenic changes to roosting ecology could erode

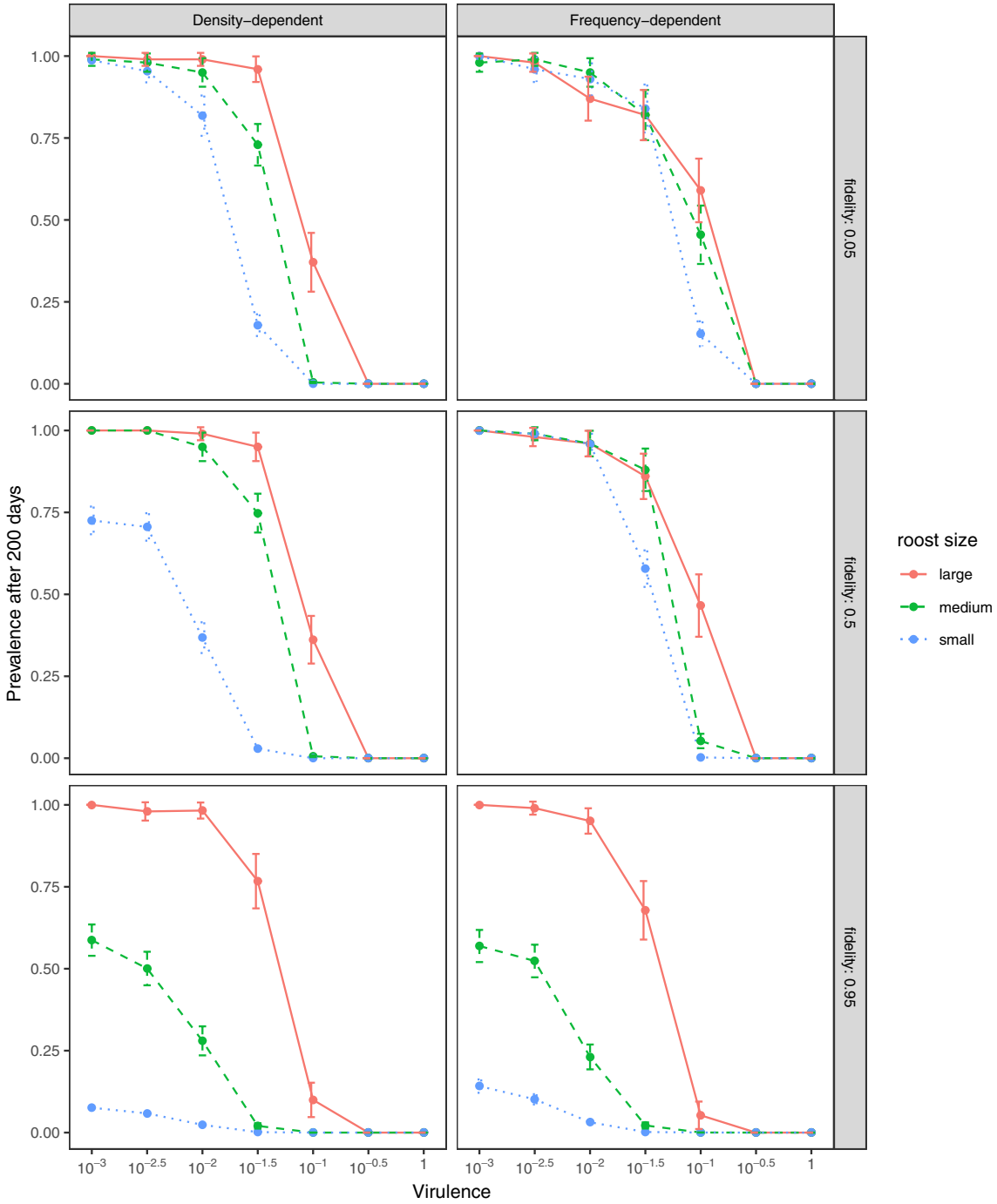

Fig. 4 Prevalence of infection after 200 days under different levels of pathogen virulence roost sizes, and transmission modes from the individual-based stochastic model

benefits of natural roosts such as size limitation or short duration of roost occupancy that reduce disease impacts and spillover risk.

The influence of roost size and site fidelity on pathogen spread could also have important implications for the control of emerging pathogens. Our results highlight the importance of surveillance in large roosts and suggest that vaccine deployment could be targeted at large roosts to effectively reduce roost size and the risk of inter-roost spread. Restricting disruption to disturbance-sensitive seasonal roost sites such as beaches for seals and shorebirds, or crop fields for migratory songbirds (Allen 1984; Lilleyman et al. 2016), could reduce the risk of inter-roost movement. Culling at large roosts could hypothetically reduce both local transmission and the risk of infection escape; however, culling campaigns that fail to account for the ecological determinants of transmission such as

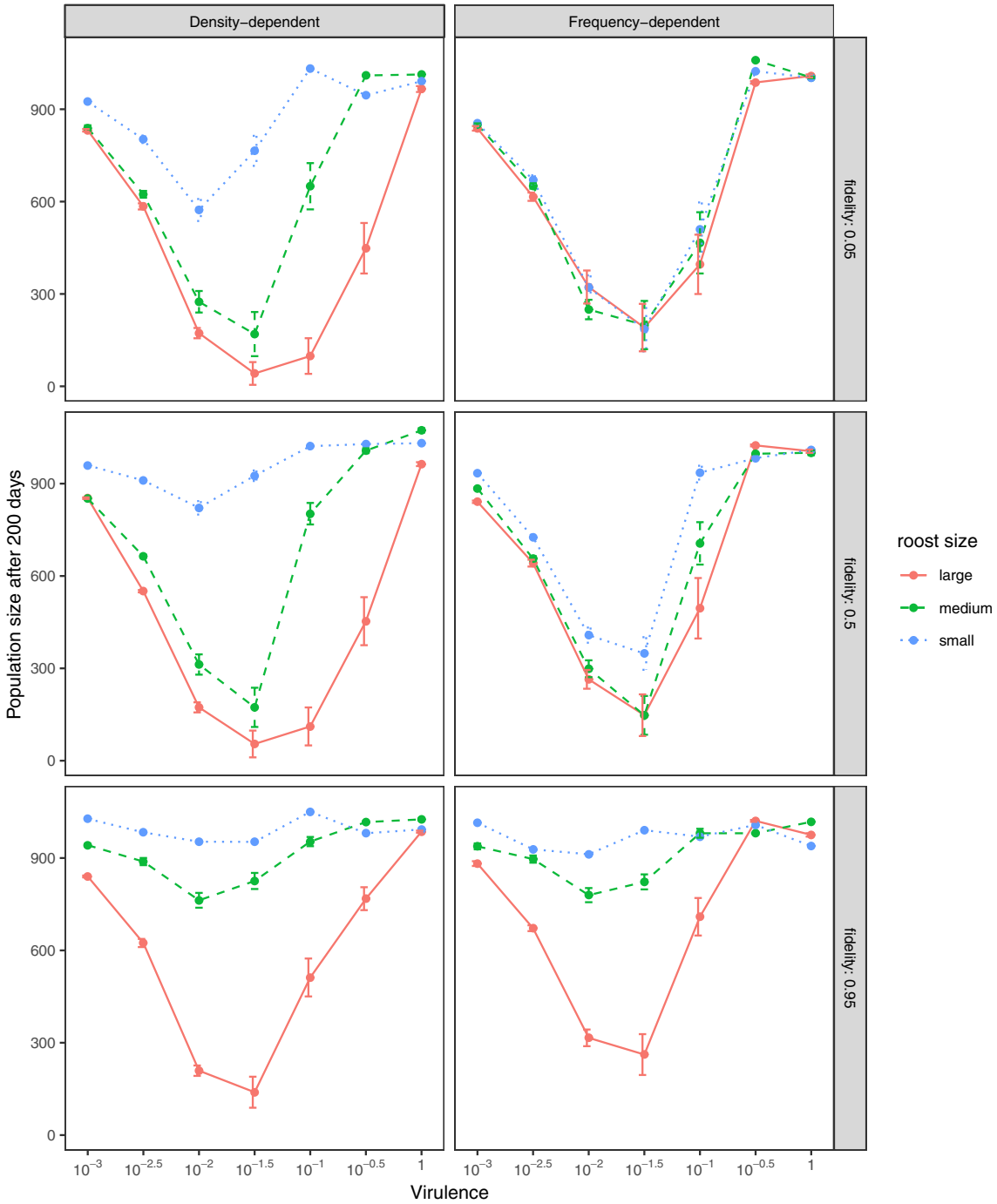

Fig. 5 Population size after 200 days under different levels of pathogen virulence, roost sizes, and transmission modes from the individual-based stochastic model

age-dependent susceptibility and movement can be counterproductive to disease management (Streicker et al. 2012).

Our results also highlight that pathogen properties such as transmission mode and virulence interact with roost attributes to determine spatial spread and pathogen impacts. Pathogens whose transmission mode is density dependent, such as airborne respiratory viruses, are more likely to spread rapidly among large roosts than frequency-dependent transmitted pathogens such as sexually transmitted infections. This result is not unexpected but does highlight that roost structure could also be an important factor influencing pathogen spread since many pathogens' transmission dynamics lie along a spectrum from density to frequency dependence (Ryder et al. 2007). Roost structures where individuals are densely aggregated, or where roosting hosts change position frequently, may promote density-dependent transmission and thus increased inter-

roost spread rates, than roosts where the number of animals in close contact is constrained by roost size or architecture. Furthermore, large, crowded roosts that support higher transmission could select for more virulent pathogen forms that can cause substantial population-level mortality even when site fidelity is relatively high.

Although our simulation model accounted for stochastic variation in some individual and roost characteristics, we did not account for larger-scale heterogeneity in roost size and individual site fidelity that could influence pathogen transmission. Many species' roost systems contain mixtures of large and small roosts, potentially reflecting seasonal or spatial variation in resource availability; this heterogeneity could potentially slow the spread of emerging pathogens if small roosts are frequent enough on the landscape to act as pathogen sinks (Becker and Hall 2016). Additionally, to simplify and make our stochastic model more generalizable, we did not vary our dispersal or fidelity rates by infection status but infection can result in lethargy (Hawley et al. 2007) or increased energetic costs of movement (Bradley and Altizer 2005). This could increase roost site fidelity of infected individuals, leading to high infection prevalence in some roosts but reduced infection spread between roosts.

While our study focused on directly transmitted pathogens, many pathogens of public health or conservation concern are spread via other transmission routes, such as biting vectors or via persistent environmental stages. Indeed, transmission potential of West Nile Virus has been negatively associated with roost size in American Robins (*Turdus migratorius*) due to a reduced biting rate per host in large roosts (Janousek et al. 2014). Other attributes of roosting environments, such as temperature and relative humidity in bat hibernacula, may also scale with roost size in ways that enhance transmission (Langwig et al. 2012). Future research is needed that further elucidates the roles of individual and roost-level heterogeneity, as well as alternative transmission routes, on pathogen dynamics in communal roosts.

Acknowledgements We wish to thank the three anonymous reviewers whose suggestions helped improve the clarity of the manuscript.

Funding information This material is based upon work supported by the National Science Foundation under grant no. DEB-1754392 (CMT, RJH) and DEB-1518611(RJH), a Tulane University Department of Ecology and Evolutionary Biology one-term Dissertation Fellowship (AJL) and by a scholar award from the James S. McDonnell Foundation (CMT).

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