

Habitat Specialization by Wildlife Reduces Pathogen Spread in Urbanizing Landscapes

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Submitted January 20, 2021; Accepted September 23, 2021; Electronically published December 23, 2021

Online enhancements: supplemental PDF. Dryad data: <https://doi.org/10.5061/dryad.1jwstqjvg>.

ABSTRACT: Urban areas are expanding globally with far-reaching ecological consequences, including for wildlife-pathogen interactions. Wildlife show tremendous variation in their responses to urbanization; even within a single population, some individuals can specialize on urban or natural habitat types. This specialization could alter pathogen impacts on host populations via changes to wildlife movement and aggregation. Here, we build a mechanistic model to explore how habitat specialization in urban landscapes affects interactions between a mobile host population and a density-dependent specialist pathogen that confers no immunity. We model movement on a network of resource-stable urban sites and resource-fluctuating natural sites, where hosts are urban specialists, natural specialists, or generalists that use both patch types. We find that for generalists, natural and partially urban landscapes produce the highest infection prevalence and mortality, driven by high movement rates at natural sites and high densities at urban sites. However, habitat specialization protects hosts from these negative effects of partially urban landscapes by limiting movement between patch types. These findings suggest that habitat specialization can benefit populations by reducing infectious disease transmission, but by reducing movement between habitat types it could also carry the cost of reducing other movement-related ecosystem functions, such as seed dispersal and pollination.

Keywords: food provisioning, infectious disease, transmission, spatial network, metapopulation, urbanization.

Introduction

As urbanization intensifies and urban areas expand worldwide, wildlife are increasingly using urban environments. Although urbanization extirpates many species (McDonald et al. 2008), others can survive and even thrive in cities (McKinney 2006). For species that are able to adapt to urban environments, living in cities can alter their behavior,

diet, and local abundance (Lowry et al. 2013), which in turn can affect ecological processes at the community and ecosystem scales (Shochat et al. 2006; Jokimäki et al. 2011). One important ecological consequence of urbanization involves changes to animal-parasite interactions (Bradley and Altizer 2007), which can be important for wildlife conservation (Deem et al. 2008).

Urbanization can either amplify or limit the transmission of pathogens, depending on the details of host and pathogen biology (Bradley and Altizer 2007). For example, food resources are often patchily distributed in urban areas, which promotes host aggregation and increases contact rates, thus increasing the rate of pathogen transmission (Farnsworth et al. 2005; McCallum and Dobson 2006). Conversely, barriers to movement in cities can isolate urban subpopulations and limit the spread of disease (Gras et al. 2018). Urban environments can also expose animals to novel pathogens or high densities of pathogen infectious stages, as is the case for woodchucks, which are exposed to *Toxoplasma gondii* when they come into contact with fecal contamination from domestic cats in urban areas (Lehrer et al. 2010). In addition, changes in diet in urban areas can increase host immunity owing to abundant food resources (e.g., bird feeders, dumpsters) or can decrease host immunity via exposure to contaminants (Becker et al. 2015). These mechanisms often operate simultaneously to alter the intensity, timing, and severity of pathogen outbreaks in urban wildlife.

Urbanization also affects wildlife-pathogen dynamics through changes to animal movement. In cities, many animals move less, exhibiting higher site fidelity, smaller home ranges, and/or lower migratory propensity (Partecke and Gwinner 2007; Furst et al. 2018; Tucker et al. 2018). These changes are often associated with stable and abundant resources in urban areas (Shochat et al. 2006; Oro et al. 2013) and can subsequently affect pathogen transmission

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and infection outcomes (McCallum and Dobson 2006). For example, naturally nomadic flying foxes have become more sedentary in urban gardens that provide year-round flowering resources, producing larger but less frequent outbreaks of Hendra virus (Plowright et al. 2011). Similarly, raccoons in urban areas have more stable home ranges than those in suburban and exurban areas, which can limit the spatial spread of rabies by reducing long-distance movements of infectious individuals (Prange et al. 2006; McClure et al. 2020). In contrast, for species such as bobcats and coyotes, for which urbanization increases distances between feeding and resting locations, movements and space use can increase in urban areas (Riley et al. 2003).

Another way that urbanization influences animal movement—and potentially infection dynamics—is through habitat specialization, where some individuals specialize in urban or natural habitats at the landscape scale (Cryan et al. 2012; Navarro et al. 2017; Teitelbaum et al. 2020b). For example, although herring gulls occupy a diversity of habitats, individual gulls specialize in different habitat types (e.g., fishing ports or estuaries), and spatial overlap between individuals is highest in human-dominated habitats, such as fishing ports (Navarro et al. 2017). Infection risk for three different zoonotic bacteria is also high at fishing ports in this species (Navarro et al. 2019), suggesting that individual habitat specialization could amplify within- and cross-species transmission. Habitat specialization is favored in heterogeneous environments with high resource diversity (Araújo et al. 2011; Layman et al. 2015), conditions often created by human development (Layman et al. 2015). Because of the complex interactions among urbanization, specialization, and infection and the importance of urban wildlife pathogens for animal and human health, a general theory that predicts how specialization should impact infection dynamics in urban wildlife is needed.

Here, we investigate the effects of urbanization and habitat specialization on population dynamics and infectious disease dynamics in a wildlife metapopulation. Our general model is motivated by recent work in a population of American white ibis (*Eudocimus albus*) in South Florida. Ibis are generally considered nomadic during the nonbreeding season, moving between wetlands in response to water levels and prey density (Bancroft et al. 2002), but in urban areas they have access to stable food resources, including irrigated lawns and human-provided food such as bread (Hernandez et al. 2016; Murray et al. 2018). This population consists of a mixture of urban habitat specialists, natural habitat specialists, and generalists, and habitat specialization in urban areas reduces connectivity across a network of ~300 sites (Teitelbaum et al. 2020b). Urban-foraging ibis can also maintain fecal-oral transmission of *Salmonella* spp. via the environment (Becker et al. 2018b) and individuals sampled in urban areas differ significantly

in their gut microbial composition compared with those from natural areas (Murray et al. 2020), suggesting that habitat specialization could be related to shifts in symbiont transmission patterns.

We build a model where animals move among natural patches, defined here to be patches that vary unpredictably in their resource availability over space and time, and urban patches, which provide relatively stable resources year-round. We refer to these patches as urban and natural in reference to the ibis system, but this framework could easily apply to other systems where patches differ in environmental variability (e.g., irrigated vs. nonirrigated agricultural areas, managed vs. unmanaged wetlands; Forbes et al. 2008; Ambika et al. 2016). We then introduce a pathogen and measure its prevalence, rate of spread, and population-wide host survival. Last, we explore how habitat specialization, where some hosts use only urban patches and others use only natural patches, affects survival and infection outcomes. We hypothesize that (1) population-level survival will be higher in landscapes with more urban patches because of greater resource stability; (2) pathogen prevalence will be higher in landscapes with more urban patches because of higher contact rates and/or densities; (3) pathogens will spread more slowly across landscapes with more urban patches because of decreased movement; and (4) habitat specialization will reduce the rate at which pathogens spread across the landscape by reducing mixing among hosts. With this study, we aim to provide predictions for how urbanization affects wildlife-pathogen dynamics and ultimately inform species conservation in urban landscapes.

Model Framework

We model resource dynamics, movement, and infection for a population of mobile animals across a landscape of discrete patches over the course of a single nonbreeding season. Patches are classified as either urban or natural and are distinguished by differences in resource availability and variability. Host movements occur as a response to resource availability and conspecific density. Within each patch, we consider density-dependent transmission of a specialist pathogen (i.e., no other sources of infection) and density-dependent mortality, such that both natural and disease-induced mortality increase with decreasing resource availability. This structure is motivated by the ibis system but is applicable to other long-lived, annually reproducing animals with high movement capacity (e.g., ungulates; Holdo et al. 2009).

Landscape Structure

We model landscapes as fully connected networks similar to a classical metapopulation model (Levins 1969), such that movement is possible between any pair of patches

(Teitelbaum et al. 2020a; fig. 1a). Resource availability in each patch can vary over time between 0 (minimum possible seasonal resource availability) and 1 (maximum possible seasonal resource availability). Patches in the network can be either urban (u) or natural (w); urban and natural patches differ in their mean resource availability and/or their variance in resource availability. At natural patches, resource availability at patch i at time t ($A_i(t)$) is drawn from a uniform distribution: $A_i(t) = U(0, 1)$. We use a weekly time step, so A_i changes weekly. This uniform distribution and weekly time step are designed to represent an environ-

ment where resource availability changes quickly depending on local conditions, such as precipitation, but average resource availability does not change over the nonbreeding season (e.g., in the ibis system; Dorn et al. 2011).

At urban patches, resource availability varies around a mean A_u with range var_u ($\text{var}_u < 1$) and also follows a uniform distribution, such that at urban patches

$$A_i(t) = U\left(A_u - \frac{\text{var}_u}{2}, A_u + \frac{\text{var}_u}{2}\right).$$

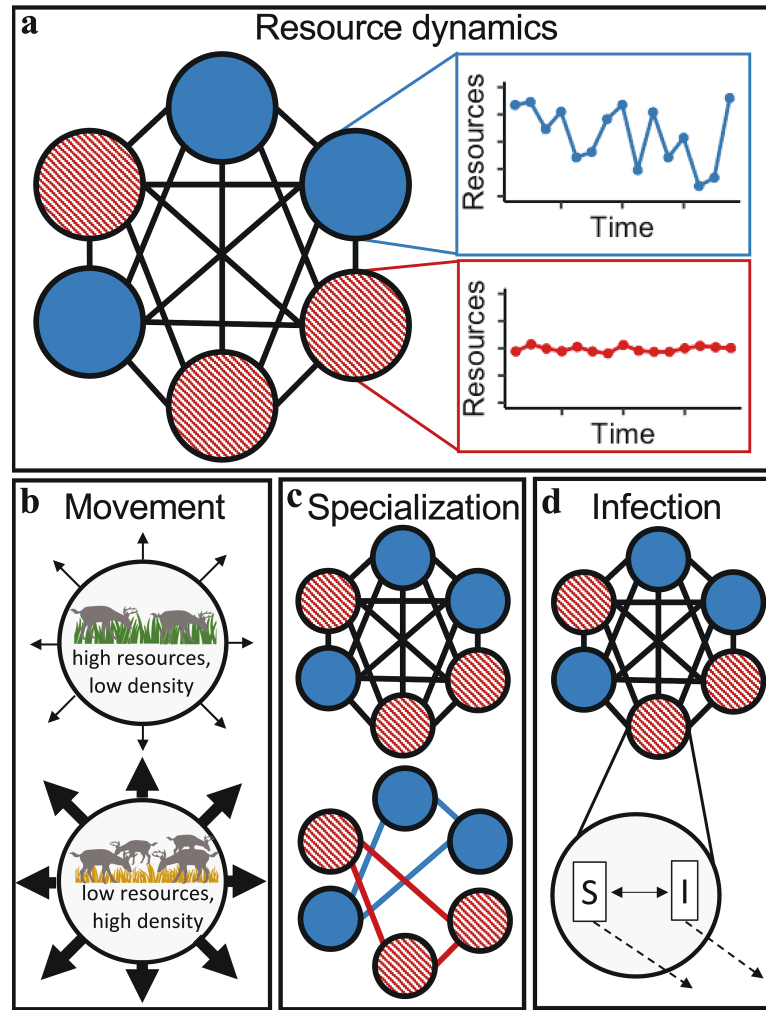


Figure 1: Model framework for interpatch movement and infection dynamics. *a*, The landscape is structured as a fully connected network of patches. Natural sites (blue) have resources that vary over time, and urban sites (red, striped) have more stable resources. Six patches are shown for visualization only; actual networks contained 10 or 50 patches. *b*, Departure from patches is a function of resource availability and host density. Arrow thickness indicates the number of animals that would depart a patch for two example density-resource combinations; in the model, any combination of density and resource availability was possible. *c*, Selection of a destination patch depends on habitat specialization. Generalists (black, *top*) move indiscriminately with respect to habitat type; urban or natural specialists (*bottom*) move only to patches of their own type. *d*, Infection and mortality occur within each patch according to an S-I-S (susceptible-infectious-susceptible) framework.

Therefore, when $\text{var}_u = 0$, urban patches have completely stable resource availability, and urban patches would be equivalent to natural patches when $A_u = 0.5$, $\text{var}_u = 1$.

Movement

A landscape consists of P patches, and we model movement and infection across landscapes that vary in the proportion of their patches that are urban. Interpatch movement occurs at the same weekly time step that resource availability changes and as a response to resource availability (i.e., after resources change). Movement is a function of two elements: a departure decision and a selection of destination (fig. 1b, 1c). Departure from a focal patch follows a density-dependent departure rule (see Teitelbaum et al. 2020a), where the proportion of individuals departing a patch decreases with resource availability ($A_i(t)$) and increases with host density ($N_i(t)$). Hosts are defined according to their habitat specialization (q): natural (w), urban (u), and generalist (i.e., no preference; r). The number of hosts of type q departing patch i at time t is denoted as $M_i^q(t)$:

$$M_i^q(t) = \begin{cases} N_i^q \left(1 - \frac{A_i(t)}{N_i(t)} \right) & \text{if } N_i(t) > A_i(t), \\ 0 & \text{if } N_i(t) \leq A_i(t). \end{cases}$$

Next, hosts that depart must select a destination. This destination depends on the host type and the destination patch type. Generalist hosts ($q = r$) distribute equally among all other patches, under the assumption that departing individuals have no knowledge of conditions at other patches. Specialized hosts move only to destination patches of their own type and distribute equally among patches of that type. Hosts do not return to the patch from which they departed unless they are specialized and there is only one patch of that type. Thus, $M_{ij}^q(t)$, the number of hosts of type q moving from patch i to patch j , is

$$M_{ij}^q(t) = \begin{cases} M_i^q(t) \frac{1}{P-1} & \text{if } q = r, \\ M_i^q(t) \frac{1}{P_q-1} & \text{if } q = u, w \text{ and } P_q > 1, \\ M_i^q(t) & \text{if } q = u, w \text{ and } P_q = 1, \end{cases}$$

where P_q is the number of patches of type q and P is the total number of patches in the system. In all cases, the initial conditions are such that the number of habitat specialists of each type is proportional to the frequency of the corresponding patch type on the landscape; for example, if the

landscape is 30% urban, then 30% of specialized hosts will be urban specialists and 70% of specialized hosts will be natural specialists.

We assume that movement is instantaneous, has no mortality cost, occurs immediately following changes in resource availability, and is independent of host infection status. Thus, following departure and immigration, the number of hosts of type q at a patch is

$$N_i^q(t+1) = N_i^q(t) - M_i^q(t) + \sum_j M_{ji}^q(t).$$

Population and Infection Dynamics

We model demography and infection dynamics on a faster timescale than movement or changes in resource availability, so that pathogen transmission and host mortality at each patch occur in continuous time between weekly bouts of movement. These processes occur after arrival at the destination patch but before the subsequent change in resource availability. Thus, for infection and population dynamics within each weeklong period of residency, we use τ in $[0, \tau_{\max}]$ to represent time.

Because we model dynamics for the nonbreeding season, the within-patch equations exclude a term for host births. Natural mortality (μ) has both density-dependent and density-independent components (μ_0 and μ_1 , respectively). We assume no adaptive benefit of specialization (i.e., survival rates are independent of host type, patch type, or their interaction). A patch's carrying capacity is equal to its resource availability, and additional density-dependent mortality occurs only when the population exceeds carrying capacity (i.e., when $N_i(t) > A_i(t)$):

$$\mu_i(t) = \begin{cases} \mu_0 + \mu_1 \left(\frac{N_i(t)}{A_i(t)} \right) & \text{if } N_i > A_i \text{ and } A_i > 0, \\ \mu_0 & \text{otherwise.} \end{cases}$$

We model the dynamics of an infection where individuals transition between susceptible (S) and infectious (I) classes and recovered hosts have no immunity and can be reinfected (i.e., an S-I-S compartmental model of infection; fig. 1d). We assume that all transmission occurs within patches rather than during interpatch movement. Transmission occurs at the density-dependent transmission rate β between susceptible and infectious individuals; β is constant across time at each patch but can differ between patch types, for example, if urban patches have higher transmission rates due to aggregation around resources (Plowright et al. 2011). This increase in transmission rates at urban sites is controlled by the parameter $\Delta\beta$ ($\Delta\beta \geq 0$). Infectious individuals recover and become susceptible at rate γ . Infected

individuals experience an additional infection-induced mortality rate ν , which scales linearly with resource availability such that infection-induced mortality is 0 when $A_i(t) = 1$ and attains a maximum of ν when $A_i(t) = 0$. The parameters γ and ν are constant across patches and across time. The infection dynamics are described by the following equations:

$$\begin{aligned}\frac{dS_i^q}{d\tau} &= -S_i^q\mu_i(t) - \beta S_i^q I_i + \gamma I_i^q, \\ \frac{dI_i^q}{d\tau} &= -I_i^q\mu_i(t) + \beta S_i^q I_i - \gamma I_i^q - I_i^q\nu(1 - A_i(t)).\end{aligned}$$

Methods and Results

We ran four sets of models to understand how the proportion of urban patches and specialized hosts affect population dynamics and infection outcomes: models with and without infection, and models with and without habitat specialization. For each combination of parameters (table 1), we ran 20 replicate simulations of the model, each with a different simulated time series of resource availability (except for cases where $\text{var}_u = 0$ and all patches were urban, where we ran only one simulation because the model was deterministic). Across all sets of models, we ran

Table 1: Parameter descriptions and values

Parameter	Description	Units	Tested values	Default values	Varies in ^a
Time and landscape structure:					
$t_{\max}$	Simulation duration	Weeks	40, 20	40	1, 2, 3, 4
$\tau_{\max}$	Timescale on which resources are updated and movement occurs	Days	7	7	...
P	Number of patches in the system	...	10, 50	10	1
Proportion urban	Proportion of patches that are urban	...	0, .1, .2, ..., 1	...	1, 2, 3, 4
A_u	Resource availability at urban patches	...	.3, .5, .7	.5	1, 2
var_u	Range of resource availability at urban patches	...	0, .01, .1, .2, .5, .8	.1	1, 2
Demography:					
μ_0	Density-independent mortality: natural mortality independent of resources or infection	Per day	.000, .001, .002	.001	1
μ_1	Density-dependent mortality: depends on resource availability and patch density	Unit resource availability per individual-day	.000, .002, .004	.002	1
Infection:					
β	Contact/transmission rate	Per infected-day	0, .03, .06, .09, .15	0, .15	2
γ	Recovery rate	Per day	0, .03, .06, .09	0, .03	2
ν	Maximum disease-induced mortality (when $A_i = 0$)	Per unit resource availability-day	0, .01, .1	0, .01	2
$\Delta\beta$	Augmentation of transmission rate at urban relative to natural sites	Per infected-day	0, .01, .06, .09	0	2
Habitat specialization:					
Proportion specialized	Proportion of hosts in the population that are either urban or natural specialists ($q = u, q = w$)	...	0, .1, .2, ..., 1	0	3, 4

Note: The “Tested values” column shows all values used at any point in the study. The “Default values” column shows the value of each parameter in portions of the study where it was held constant. The “Varies in” column shows in which of the four sections of the study the parameter was explored; in all other experiments, the parameter value was that shown in the “Default values” column.

^a Numbers refer to results subsections in the order in which they appear: (1) “Model 1: Urbanization, Movement, and Survival” (no infection or specialization); (2) “Model 2: Urbanization and Infection” (no specialization); (3) “Model 3: Habitat Specialization and Survival” (no infection); and (4) “Model 4: Habitat Specialization and Infection.”

51,367 simulations representing 2,651 parameter combinations. Each simulation started with each patch at its average carrying capacity (i.e., 0.5 for natural patches and A_u for urban patches), under the assumption that the population is well adapted to its current environment. These numbers do not represent whole individuals but are scaled relative to the hypothetical maximum population size if all patches in the system were at their maximum resource availability. For each time step, we simulated a change in resource availability, applied the movement equations, and then, after animals had arrived at their destination patch, solved the mortality and infection equations numerically using the deSolve package in R (ver. 4.0.1; Soetaert et al. 2010; R Development Core Team 2020). We repeated this process for 40 weekly time steps.

For each simulation, we calculated multiple outcome metrics (described below) and then summarized the mean and standard deviation of each of these outcomes across the 20 simulations for each parameter combination.

Model 1: Urbanization, Movement, and Survival

To explore the effect of urbanization alone on host population dynamics, we ran models with no infection or specialization (i.e., $\beta = 0$, proportion specialized = 0, $N(0) = S(0)$, $I(0) = 0$). To measure population outcomes, we calculated the proportion of individuals surviving the entire nonbreeding season for each simulation (end population size/starting population size). We explored the effects of parameters for demography (μ_0 , μ_1) and landscape structure (P , A_u , var_u) on these outcomes (table 1), and we also explored the effects of our main focal variable, that is, the proportion of urban patches in the landscape.

Across the urbanization gradient (i.e., 0% to 100% urban patches), between 51.2% and 74.3% of hosts survived a full nonbreeding season ($\mu_0 = 0.001$, $\mu_1 = 0.002$, $P = 10$, $A_u = 0.5$, $\text{var}_u = 0.1$; table 1; fig. 2a). Survival (i.e., the proportion of animals surviving a full season) was lowest when the landscape was <50% urban and increased monotonically from that point. When both urban and natural patches were present, urban patches persisted at or above carrying capacity (fig. 2b, 2c), which produced high mortality rates (fig. S1; figs. S1–S12 are available online). The relationship between urbanization and survival was magnified when density-dependent mortality was stronger (μ_1 ; fig. S2). These patterns were consistent for a larger network ($P = 10$ vs. $P = 50$; fig. S3) and for a shorter nonbreeding season (20 vs. 40 weeks; fig. S4a, S4b).

Changing resource variability (var_u) or resource availability (A_u) at urban patches did not change the direction of the relationship between urbanization and survival, but these parameters altered the strength and/or shape of this

relationship (figs. 3a, 3b, S5). When resources at urban sites were more variable (higher var_u), survival was lower in highly urban landscapes (i.e., >50% urban). For example, 74.3% of hosts survived a full nonbreeding season in a mostly stable, 100% urban landscape ($\text{var}_u = 0.1$), but only 55.9% survived when urban patches were half as variable as natural patches ($\text{var}_u = 0.5$; fig. 3a). Higher resource availability at urban patches (A_u) accelerated the positive relationship between survival and urbanization (fig. 3b). For instance, in landscapes where urban patches were subsidized ($A_u = 0.7$), survival began to exceed that of a fully natural landscape when about 40% of patches were urban. In contrast, when urban patches were degraded ($A_u = 0.3$), survival did not exceed that of a fully natural landscape until about 65% of patches were urban. Population sizes were always larger with higher urban resource availability, since urban patches supported larger populations.

We also quantified the cumulative number of movements that occurred across all patches for the entire simulation. Movement tended to decline as urbanization increased (fig. S6), declining to near zero in highly urban landscapes unless urban resource variability was high ($\text{var}_u \geq 0.5$). This pattern occurred because movement depended on resource availability; while resource availability could drop to near zero at natural sites, urban sites never dropped below $A_u - \text{var}_u$, meaning that departure from urban sites was less frequent than departure from natural sites. Movements were relatively unaffected by urban resource availability (fig. S6).

Model 2: Urbanization and Infection

Next, we included infection to analyze relationships among urbanization, infection, and population dynamics. We seeded infection by initializing each simulation with 1% of hosts at one patch infected (i.e., 0.1% of the metapopulation in a 10-patch system). All other individuals started in the susceptible class. When the landscape contained both urban and natural patches, we performed half of the simulations ($N = 10$) with infection seeded an urban patch and half with infection seeded at a natural patch. For this set of simulations, we explored the effects of the three infection-related parameters (β , λ , and v ; table 1) and parameters for urban resource dynamics (A_u and var_u).

In addition to quantifying survival and movement as described above, we calculated end-of-season host infection prevalence, that is, the proportion of hosts infected at the end of the simulation. As a metric of transient dynamics, we also calculated the maximum infection prevalence over the course of the simulation ($\text{prev}_{\max}$), the time to reach maximum prevalence (t_{prev} in weeks), and the rate of infection spread ($[\text{prev}_{\max} - \text{prev}_0]/t_{\text{prev}}$). To measure spatial spread, we calculated the spatial variation in end prevalence as the

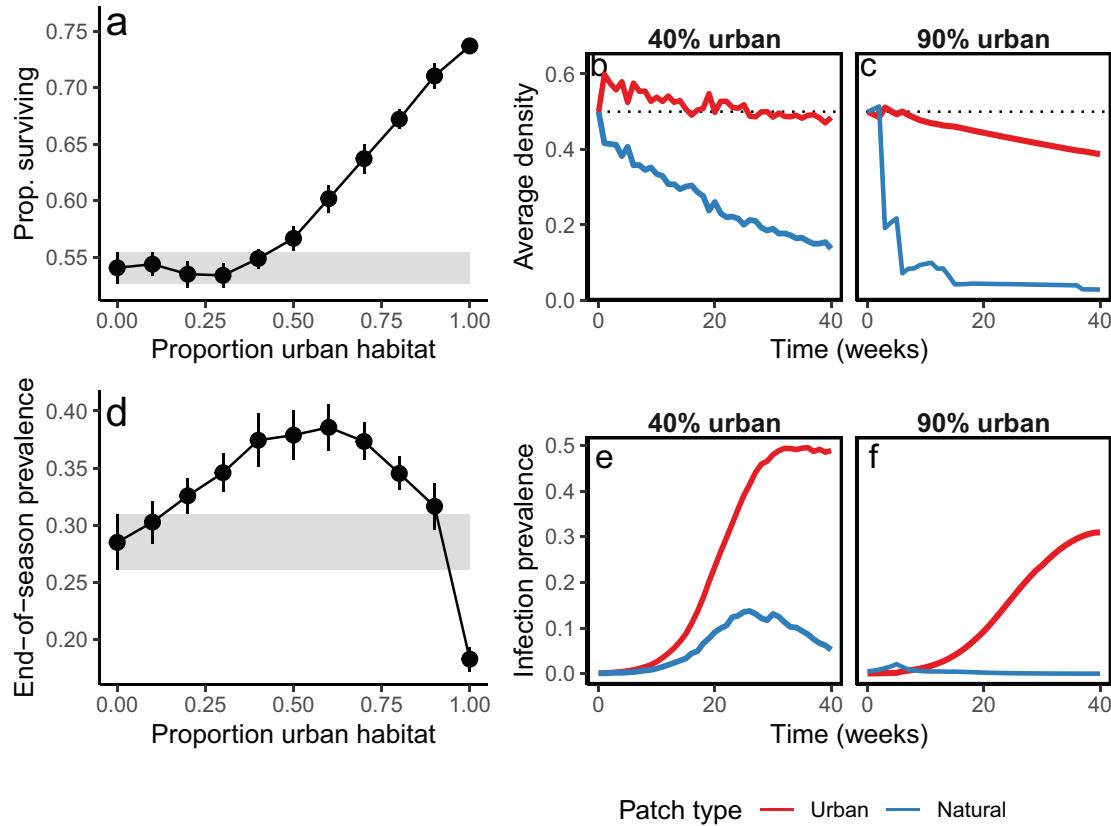


Figure 2: Effects of urbanization on the proportion of individuals surviving and mean infection prevalence when there is no habitat specialization. In *a* and *d*, error bars show the standard deviation of the mean across 20 simulations. The horizontal gray bar shows the mean and standard deviation of values for a 100% natural landscape. *a*, The fewest individuals survive in natural and partially urban landscapes. These simulations had no infection present; other parameter values are shown in table 1. *b*, *c*, Population density across time for each of the 20 simulations; the density shown is the mean population size across all patches of a given type. Population sizes at urban patches are always higher than those at natural patches. The horizontal line at 0.5 is the carrying capacity of an urban site for this parameter set ($A_u = 0.5$). *d*, End-of-season infection prevalence (proportion of the population that is infected at $t = 40$ weeks) is highest in 30%–80% urban landscapes. *e*, *f*, Prevalence is higher at urban than at natural patches, except for a few weeks in highly urban landscapes. Prevalence displayed is the mean prevalence at a site of a given type.

coefficient of variation in end-of-season infection prevalence across patches (i.e., the standard deviation of prevalence across patches divided by the mean prevalence across the whole landscape).

Infection prevalence had a hump-shaped relationship with urbanization, such that prevalence was highest in landscapes that were 30%–80% urban and was lowest 100% urban landscapes (fig. 2*d*). These patterns were magnified when contact rates were higher and when pathogen-induced mortality rates were intermediate (fig. S7). This nonlinear relationship between urbanization and infection resulted from changes in host movement and density. In predominantly urban landscapes, low movement reduced pathogen spread between patches, resulting in minimal transmission. In mostly natural landscapes, high movement promoted pathogen

spread, but low population sizes limited transmission. In partially urban landscapes, host movement from natural patches allowed the pathogen to spread, and high densities at urban patches allowed the pathogen to transmit efficiently. In support of this mechanism, in partially urban landscapes, host density and infection prevalence were higher at urban patches than at natural patches (figs. 2*e*, 2*f*, S8).

The shape of the relationship between urbanization and infection prevalence also depended on urban resource dynamics. Higher resource variability (var_u) at urban patches decreased infection prevalence, particularly in partially urban landscapes (fig. 3*c*). In addition, when urban patches provided more resources than natural patches, infection prevalence increased with urbanization, and when urban patches were degraded, infection prevalence decreased with

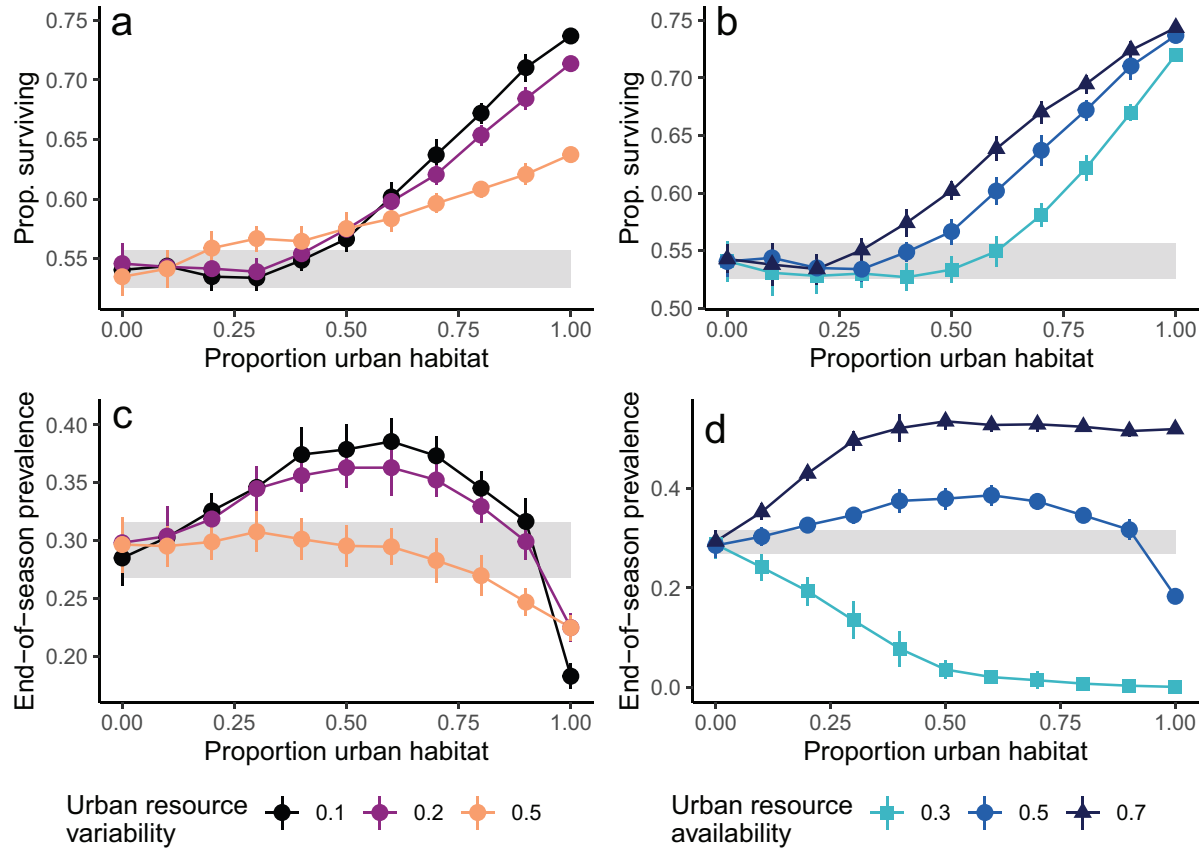


Figure 3: Effects of urban resource variability (*a*, *c*) and urban resource availability (*b*, *d*) on survival and infection prevalence. Error bars show the standard deviation of the mean across 20 simulations. The horizontal gray bar shows the mean and standard deviation of values for a 100% natural landscape. In *a* and *b*, no infection was present. *a*, Urbanization increases survival, but its effect is smaller when resource availability at urban patches is more variable. For more values of var_u , see figure S5. *b*, Survival increases more quickly with urbanization when urban habitats are subsidized ($A_u > 0.5$) and declines with urbanization when urban habitats are degraded ($A_u < 0.5$). *c*, Infection prevalence peaks in partially urban landscapes, but only when urban patches are less than half as variable as natural patches ($\text{var}_u < 0.5$). *d*, Infection prevalence decreases with decreasing resource availability at urban patches. Note the different y-axis scales in each panel.

urbanization (fig. 3*d*). End-of-season infection prevalence was correlated with other metrics of infection outcomes (table S1), meaning that infection spread more quickly and reached higher peak prevalence in landscapes with higher end-of-season infection prevalence.

We also explored the scenario where urban sites have higher transmission rates (β) than natural sites, to model differences in aggregation (e.g., clustering around point sources of food in urban landscapes). Increasing transmission rates at urban sites increased end-of-season infection prevalence except in 100% natural landscapes. When contact rates at urban sites were substantially higher than those at natural sites (~ 1.4 times), the hump-shaped relationship disappeared and infection prevalence increased monotonically with urbanization or plateaued after about 50% of patches were urbanized (fig. S9). In these cases, urban-

ization provided little or no survival benefit, even in fully urbanized landscapes (fig. S9).

Model 3: Habitat Specialization and Survival

Next, we explored how habitat specialization affects population dynamics by running models that included specialization but no infection (i.e., proportion specialized > 0 , $\beta = 0$). Here, we measured the same outcomes as in simulations without specialization (model 1) but also calculated the difference in survival between urban specialist, natural specialist, and generalist hosts.

Habitat specialization, where some or all hosts moved preferentially to urban or natural habitats, increased survival, especially when the proportion of urban patches was between 10% and 80% (fig. 4*a*). When all hosts were

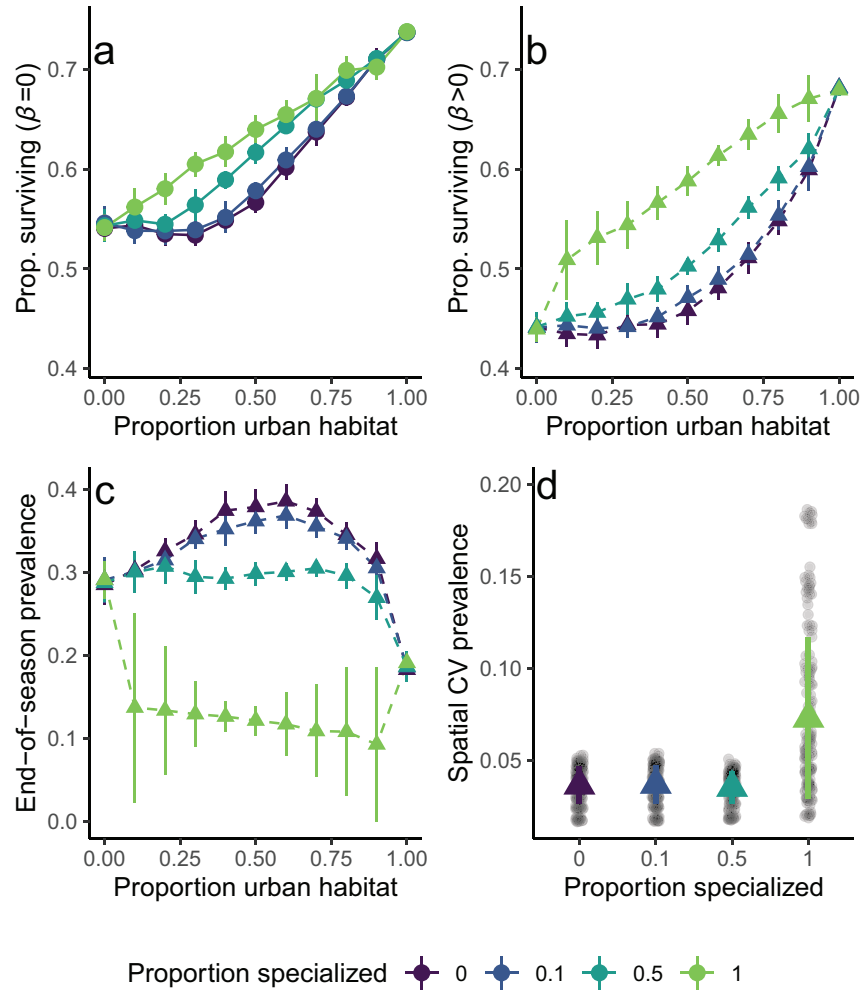


Figure 4: Effects of host habitat specialization on population-level survival (a, b) and infection prevalence (c, d). “Proportion specialized” refers to the sum of urban and natural habitat specialists in the population. a, Proportion of the population surviving in a case where infection is absent. Specialization (blue and green lines) increases survival relative to full generalism. b, Proportion of the population surviving when infection is present. c, Specialization reduces infection prevalence, especially in partially urban landscapes. Full specialization has a larger effect than partial specialization. In a–c, error bars show the standard deviation of the mean across 20 simulations. d, Specialization increases spatial variation in infection prevalence, measured as the coefficient of variation (CV) in infection prevalence across patches. Each bar shows the mean and standard deviation of the CV of prevalence across all partially urban landscapes (i.e., $0 < \text{proportion urban} < 1$). Partially transparent points are data from individual simulations.

specialized, the relationship between urbanization and survival was positive and linear (fig. 4a). These patterns occurred because specialization prevented urban patches from accumulating individuals above their carrying capacity, which increased survival for both urban and natural hosts. Survival was higher for urban specialists than for natural specialists when the proportion of specialized hosts and/or urban patches was high (>80% specialists; fig. S10). Otherwise, survival was higher for natural specialists because the movements of generalist individuals increased densities and reduced survival at urban sites. Specialization had relatively small effects on movement at the metapopulation scale but

slightly increased movement when all individuals were specialized (fig. S11) because larger population sizes in the presence of specialization meant a higher number of cumulative movements.

Model 4: Habitat Specialization and Infection

Last, we modeled the interactions among urbanization, specialization, and infection. We measured the same outcomes as in simulations without specialization as well as the difference in end-of-season prevalence between urban and natural hosts.

Host habitat specialization decreased infection prevalence relative to a generalist host population, measured using both end-of-season prevalence (fig. 4c) and peak infection prevalence (fig. S12). The effect of specialization was nonlinear; a population with 50% specialists was more similar to a generalist population than it was to a fully specialized population (fig. 4b, 4c). Specialization reduced infection prevalence by reducing densities at urban patches and, in the case of complete specialization, because specialized populations had lower effective population sizes (i.e., a 10-patch network became two separate 5-patch networks). Moreover, prevalence was more variable across space when all hosts were specialized (fig. 4d) because there was limited movement between urban and natural patches. This pattern also increased the variability across simulations, since in the presence of specialization, prevalence depended on whether the initial infection began at an urban or a natural patch. These effects also appeared quickly; even when considering a shorter nonbreeding season (20 vs. 40 weeks) specialization reduced prevalence, although its effect was smaller and more variable (fig. S4c, S4d).

Discussion

Relationships between urbanization and wildlife population or infection dynamics have been observed across a number of empirical studies, but often in contrasting directions (Bradley and Altizer 2007; Brearley et al. 2013). For example, wildlife in urban areas can have higher (Murray et al. 2016) or lower (Fischer et al. 2005) infection prevalence than in natural areas. By linking resource dynamics, infection, and movement in a single framework, our model clarifies that changes in host movement patterns in urban areas can link landscape-scale resource variability to pathogen transmission and host population dynamics. Furthermore, these results highlight that the effects of urbanization on infection depend not only on how urban landscapes affect transmission within patches but also on how individual hosts respond to urbanization. Host responses to urbanization depend on both individual host characteristics (in this case, habitat specialization) and resource dynamics at urban patches. Some degree of habitat specialization has been reported across taxa, from insects to birds to mammals (Araújo et al. 2011). Our results show that host landscape-level habitat affinity can have clear downstream effects on population-level survival and infection prevalence.

In our model, urbanization had nonlinear effects on survival and infection prevalence in an urban-adapted host population, such that survival was lowest and infection prevalence peaked in natural or partially urban landscapes. In these partially urban landscapes, urban and natural patches played different roles in driving population and infection dynamics. When they provided at least as many resources

as natural sites, urban patches persisted at high densities, which produced population sinks and high mortality rates. This pattern was especially prominent when urban sites provided stable resources. High host densities at urban sites also increased pathogen prevalence by promoting transmission, even when our model did not assume that contact rates would be inherently higher at urban patches (which is common because of aggregation in fragmented habitats or around human-provided food sources; Bradley and Altizer 2007; Murray et al. 2016). These results are consistent with field studies that report higher *Salmonella* infection prevalence in urban flocks of American white ibis (Murray et al. 2021) and higher risk of disease-induced mortality in urban raccoons (Prange et al. 2003). At the same time, our models showed the highest survival and lowest infection prevalence in 100% urban landscapes. In these fully urban landscapes, low interpatch movement limited pathogen spread. It is important to note, however, that this scenario—where the entire landscape is homogeneously urbanized—is unlikely to occur frequently in the real world, since most cities include some natural areas (e.g., large parks or undeveloped sections) and would be best represented by a partially urban landscape in our model. These results were also robust to the size of the network, suggesting that as long as animals are able to move freely among patches, the effects of urbanization might be independent of habitat availability on the landscape.

These general relationships between infection prevalence and urbanization align with results from other models of infection, movement, and resources. Mechanistic models have confirmed the importance of movement characteristics for infection dynamics; for example, models have shown that the effect of long-distance nomadic movements on outbreaks depends on movement frequency (Craft et al. 2011) and that outbreaks are more intense when hosts can detect suitable habitat over longer distances (White et al. 2018). Similar to our result that urban sites promoted transmission in partially urban landscapes, one metapopulation model showed that food provisioning tends to decrease infection prevalence when it promotes site fidelity (Becker et al. 2018a). Another individual-based model found that disease transmission was fastest at intermediate levels of landscape permeability (Tracey et al. 2014). In contrast, in another metapopulation model, toxicant-contaminated urban patches reduced landscape-level infection transmission by decreasing survival (Sánchez et al. 2020). This result aligns with our finding that urbanization decreased infection prevalence when resource availability was lower at urban patches than at natural patches. Together, these results indicate that the effect of urbanization on infection prevalence depends on whether a species is adapted to urban areas (i.e., whether urban patches increase or decrease survival and movement) and therefore that the direction of the urbanization-infection

relationship depends on a focal species' movement and survival responses to urbanization.

In our model, habitat specialization increased population-level survival, and this effect was particularly strong in partially urban landscapes. When some or all individuals used only urban or only natural patches, host densities at urban patches were lower, which reduced density-dependent mortality rates. This pattern emerged despite the fact that parameters for individual-level traits (e.g., mortality rates μ_0 and μ_1) were equal across host types, which suggests that habitat specialization could increase host success even if it provides no a priori fitness benefit. Previous work has shown that niche specialization should affect fitness (Bolnick et al. 2003) and could potentially increase the range of habitats or geographic space over which a species can persist (Holt 1985); our results show that it can have further benefits by preventing source-sink dynamics in heterogeneous landscapes.

Habitat specialization also lowered infection prevalence by reducing host densities at urban patches and limiting movement between patch types. This reduced movement effectively created a spatially structured population; in structured populations, pathogens must have a higher transmission probability or longer infectious period in order to spread, since they must persist within a group long enough for movement to occur between groups (Cross et al. 2007). These findings coincide with those from studies of bumblebees, in which strong individual specialization in flower use can affect foraging network structure (Tur et al. 2014), with potential effects on pathogen prevalence (Ellner et al. 2020). However, it is important to note that the relative timescales of contact and the transmissibility of a pathogen are important for the relationships between interpatch movements and the spread of infection. For example, feline immunodeficiency virus shows less genetic isolation than do its mountain lion hosts, possibly because the threshold for virus transmission is lower than that for gene flow among mountain lions (Lee et al. 2012). These patterns suggest that habitat specialization could eventually drive the evolution of pathogen traits, such as recovery rates and virulence, that would allow them to persist in a spatially structured population (Haraguchi and Sasaki 2000; Boots et al. 2004).

In our model, reduced movement in stable urban landscapes limited pathogen spread. However, this pattern also indicates that provision of beneficial ecosystem services by mobile wildlife could be at risk in urban landscapes that provide stable and abundant resources. For example, nomadic movements of trumpeter hornbills more than double the probability of long-distance seed dispersal in a fragmented forested landscape (Mueller et al. 2014), and reductions in movement would erode this transport service. Increasing resource variability at urban sites—for example, by planting species with varied phenology or by limiting wildlife feeding—might promote movement and restore these

services (Jones et al. 2014; Satterfield et al. 2015). Counter-intuitively, specialization actually increased movement in many cases, both because it increased population sizes (and thus the potential number of individuals moving) and because it prevented natural-specialized hosts from getting “stuck” in urban patches. These effects were relatively small, but they indicate that specialization could provide some rescue from the negative effects of urbanization on movement. Moreover, since urban habitats can be sources of pollutants and invasive species (Von Der Lippe and Kowarik 2008; Blitzler et al. 2012), specialization could prevent the export of these potentially harmful materials from urban sites (Blais et al. 2007).

Beyond their importance for understanding host-pathogen dynamics, our results imply that effectively managing infections in urban wildlife requires understanding how individuals respond to resource stabilization in urban areas. For example, individual American white ibis vary in their use of urban and wetland habitats (Teitelbaum et al. 2020b), and ibis in urban areas have high prevalence of *Salmonella* infection (Hernandez et al. 2016). Similarly, transmission of *Mycoplasma gallisepticum* in house finches centers around domestic bird feeders (Adelman et al. 2015). If only some individuals specialize on or dominate use of feeders (e.g., as in house sparrows; Galbraith et al. 2017), population-wide infection spread will be limited. In each of these examples, using human-dominated landscapes (i.e., urban parks or bird feeders) to monitor population health would provide a biased sample in which infection prevalence is higher than in the population as a whole. In addition, our results reveal a potential trade-off in the outcomes of habitat restoration. Restoration is beneficial because it can increase population sizes (Wiegand et al. 2005), especially for non-urban-adapted species. It can also reduce the potential for spillover of zoonotic pathogens such as *Salmonella* and Hendra virus by drawing wildlife away from urban areas, where contact between humans and wildlife is more likely (Plowright et al. 2014). However, in our model partially urban landscapes had the highest prevalence of infection, so restoration could have unintended consequences for wildlife health, especially if individuals move frequently between urban and restored habitats. Monitoring and managing wildlife infectious diseases in urban areas will be more effective with a better understanding of how individuals vary in their responses to landscape heterogeneity.

These results motivate future investigations of the interactions among resource fluctuations, wildlife movement, and health. Our model provides predictions for how resource stabilization should affect infection; integrating these predictions with observational data on resources, movement, survival, and infection in a real-world system would help understand the generality and condition dependence of the model results. This general conceptual model could

also be interpreted for entirely nonurban systems where the magnitude of resource fluctuations differs across space. For example, diurnal temperature fluctuations are larger in the tree canopy than in the understory (Rapp and Silman 2012). For small ectotherms such as insects or snails that move at the scale of meters rather than kilometers, these small-scale differences in temperature variability could influence movement patterns, conspecific interactions, population dynamics, and infection dynamics in similar ways as we showed here.

This flexible framework also provides several opportunities for exploring additional interactions between movement and infection. First, infection can change movement patterns and habitat use; for example, coyotes are more likely to forage in urban areas when infected because human resources are easier to access (Murray et al. 2015). Incorporating feedbacks between infection and movement could reveal different dynamics and applications to more systems. In addition, we used a simple susceptible-infected framework to model transmission, which can apply to bacteria, arthropods, or other pathogens in which immunity is minimal or nonexistent. Future models could incorporate different assumptions about immunity and transmission modes, for example, seeking to model the Hendra virus–flying fox system (Plowright et al. 2011), in which immunity is important, or the white ibis–*Salmonella* system, where bacteria can be transmitted via the environment (Becker et al. 2018b). Finally, specialization can have fitness consequences on its own (Bolnick et al. 2003), and future models could help understand the how pathogen transmission could affect the evolution of specialization as well as how specialization could affect pathogen evolution.

Acknowledgments

This work was supported by a National Science Foundation (NSF) Graduate Research Fellowship to C.S.T., NSF DEB-1754392 and NSF Ecology and Evolution of Infectious Diseases (EEID) grant DEB-1518611 to S.A. and R.J.H., and NSF EEID grant DEB-1911925 to R.J.H. We thank the Hall and Altizer labs for helpful feedback on early versions of this model.

Statement of Authorship

All authors contributed to study conceptualization and funding acquisition. C.S.T. and R.J.H. developed the model. C.S.T. wrote model code, implemented simulations, visualized results, and wrote the first draft of the manuscript. R.J.H. provided computational resources. All authors contributed to interpretation of results and manuscript editing.

Data and Code Availability

Data and code are available in the Dryad Digital Repository (<https://doi.org/10.5061/dryad.1jwstqjvg>; Teitelbaum et al. 2021).

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Associate Editor: Sébastien Lion
Editor: Daniel I. Bolnick



American white ibis (*Eudocimus albus*) eat bread at an urban site in South Florida. White ibis are abundant in urban parks and residential areas, where they often feed on provided food and irrigated lawns and may be exposed to pathogens, such as *Salmonella*. Photo by Richard Hall.