LINKING ANTHROPOGENIC RESOURCES TO WILDLIFE–PATHOGEN DYNAMICS: FROM THEORY TO VAMPIRE BATS

by

DANIEL JOSEPH BECKER

(Under the Direction of Sonia Altizer and Daniel Streicker)

ABSTRACT

For many wildlife, consumer–resource interactions are altered by human activities that provide supplemental food. As anthropogenic resources are often more abundant and predictable than natural foods, subsidized populations can be larger, aggregated, and better-fed than wild animals. A growing literature suggests anthropogenic resources can have profound impacts on host–pathogen interactions, in some cases increasing cross-species transmission. However, predicting when and where anthropogenic resources will increase such risks is complicated, as supplemental resources can simultaneously affect hosts and pathogens through multiple and opposing mechanisms. Factors enhancing transmission, such as increased aggregation, could be offset by improved immunity due to lower starvation stress. The aim of this dissertation is to develop and test an integrative framework for understanding how anthropogenic resources affect wildlife disease. I first built supplemental resources into mechanistic models to explore the overall effect of provisioning on epidemiological outcomes; this work demonstrated that how resources affect host immunity is critical for understanding if supplemental feeding increases or decreases transmission. I next synthesized the literature on how host–pathogen interactions respond to supplemental feeding to test support for these theoretical mechanisms; this meta-

analysis demonstrated strong support for higher contact rates in supplemented populations increasing infection but variable effects of provisioning on host condition and weak effects on demography. I next extended models to a metapopulation scale to predict how the landscape-level deployment of supplemental food affects infection dynamics; these studies improved existing theory on pathogen dynamics in metapopulations and showed that host occupancy can be maximized and infection can be minimized if landscapes contain heterogeneous mixtures of unprovisioned and provisioned habitats. Lastly, I examined these processes in the context of a natural system by asking how anthropogenic resources interact with immunity, demography, and bacterial infection for vampire bats in Latin America. Results suggested that livestock expansion could stimulate bat demography and innate immunity, in turn having strong negative effects on prevalence of a vector-borne pathogen but no relationship with a directly transmitted pathogen. Collectively, this dissertation provides theoretically informed and testable predictions for how anthropogenic resources affect wildlife disease across taxa and in an important applied system.

INDEX WORDS: *Desmodus rotundus*; ecoimmunology; host–pathogen dynamics; metapopulations; resource provisioning; supplemental feeding

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DEDICATION

For mom, dad, Alex, and Nimbus.

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CHAPTER 1

INTRODUCTION AND LITERAURE REVIEW

The abundance and distribution of food resources affect population and community dynamics, in part by influencing reproduction, dispersal, and trophic interactions [1–3]. Yet for many species throughout the world, these resources are being altered by landscape changes and human activities that provide supplemental food to wildlife [4,5]. Supplemental food is commonly provided intentionally from household bird feeding [6,7], by wildlife tourism [8,9], from conservation programs [10,11], and to manage game species or to limit human–wildlife conflicts [12,13]. Similarly, generalist species within urbanized and agricultural landscapes can capitalize on unintentionally provided but abundant food resources such as crop fields [14], landfills [15,16], backyard gardens [17,18], and even livestock [19,20]. As anthropogenic resources are often more abundant and spatiotemporally predictable than natural foods [21–23], subsidized populations can be larger [24,25], more aggregated [26,27], and better-fed than their wild counterparts [28,29]. Resource provisioning can consequently have cascading ecological effects, such as on food web structure and nutrient cycling [4,19,30].

A growing body of work also suggests that anthropogenic resources can have profound impacts on host–pathogen interactions [31,32], in some cases increasing risks of cross-species transmission [33]. For example, agricultural intensification in Malaysia attracted flying foxes (*Pteropus* spp.) to forage on mango farms, which resulted in greater contact between bats, pigs, and humans and facilitated spillover of Nipah virus from bats [34,35]. Similarly, the mixing of species at bird feeders was implicated in the spillover of *Trichomonas gallinae* from pigeons

(*Columbiformes*) to finches (e.g., *Carduelis chloris*) [36], which has led to dramatic songbird population declines throughout the United Kingdom [37]. The dense aggregations of house finches (*Haemorhous mexicanus*) at bird feeders is thought to contribute to the spread of the similarly virulent *Mycoplasma gallisepticum* [38,39]. Yet while these case studies illustrate that resource provisioning can increase cross-species transmission risks and pathogen transmission within a host species, supplemental feeding can also have the opposite effect of reducing infectious disease burdens. For example, lace monitors (*Varanus varius*) feeding on landfills showed lower haemoparasite intensity than naturally foraging lizards [40]. Likewise, red foxes (*Vulpes vulpes*) in Switzerland that forage on household garbage were less frequently infected with a zoonotic tapeworm (*Echinococcus multilocularis*) compared to rural foxes [41].

Given that resource provisioning can produce contrasting effects on host–pathogen interactions, how can we predict when and where anthropogenic resources will increase or decrease pathogen risks for humans and wildlife? Such forecasting has been limited by a lack of conceptual and mechanistic frameworks that synthesize the multiple mechanisms by which supplemental resources can affect wildlife hosts and their pathogens. At the individual level, greater access to food and improved nutrition can facilitate host resistance to or recovery from infections [42,43]. As one example, *ad libitum* access to food allowed field voles (*Microtus agrestis*) to mount stronger immunological defenses against nematodes [44]. Yet at the population level, supplemental feeding can increase host aggregations and contact rates, increasing transmission opportunities [45,46]. Hosts that become more sedentary owing to reduced foraging activity can be continuously exposed to pathogens shed into the environment [47,48]. Furthermore, greater availability of food could support greater recruitment of susceptible hosts through births or immigration [10,49], and these larger populations could fuel pathogen

transmission via density-dependent processes [50,51]. These different mechanisms can operate simultaneously within provisioned wildlife populations and may have especially strong effects for particular host and pathogen groups and in particular feeding contexts.

The aim of this dissertation is to develop and test an integrative framework for understanding how anthropogenic resources affect infectious disease dynamics in wildlife. Three main objectives underpin this work: *(i)* develop mechanistic and conceptual models that consider how different host and pathogen processes respond to resource provisioning and assess empirical support for these processes (Chapters 2 and 3), *(ii)* extend models to a metapopulation scale to predict how the landscape-level deployment of supplemental food affects infection dynamics (Chapters 4 and 5), and *(iii)* examine processes in the contact of a real-world host–pathogen system by asking how anthropogenic resources interact with host immunity, demography, and bacterial infection for wild vampire bats in Latin America (Chapter 6).

To address Goal 1, I first develop simple theoretical models that build supplemental resources into individual- and population-level parameters to explore the net effect of resource provisioning on epidemiological outcomes such as infection prevalence (Chapter 2). This work provides a novel contribution to epidemiological theory and shows that the extent to which host immune defense improves with resource provisioning is critical for understanding whether supplemental feeding increases or decreases infection prevalence [52]. Weak but positive effects of resource provisioning on host resistance maximize infection prevalence at intermediate and high degrees of food supplementation, whereas strong effects on immunity drive pathogens to extinction with intermediate intensities of resource provisioning. In Chapter 3, I next test empirical support for the mechanisms built into these models (e.g., supplemental feeding increasing contact rates and decreasing host susceptibility) by synthesizing results from the

published literature in a comprehensive meta-analysis. This work demonstrates that higher contact rates in food-supplemented populations increase infection measures, but shows variable effects of resource provisioning on host condition and immune defense and weak to no effects on host demography [33]. The strongest positive effects of provisioning on infection were also detected for bacteria and viruses and for intentional feeding, identifying pathogen types and feeding contexts where infection risk is highest. This synthesis more broadly provides a roadmap for future studies of provisioning and infectious disease and outlines outstanding priorities.

Given that many wildlife display spatial structure [53,54] and resource provisioning often occurs across a network of habitat patches, it is important to consider how the spatial extent of food-supplemented habitats affects disease dynamics. For Goal 2, I thus extend the biological scale of these models to incorporate effects of local food supplementation on patch occupancy time (Chapter 4) and on patch attractiveness and production of dispersers (Chapter 5) to examine whether increasing the fraction of supplemented habitats facilitates or impedes the spatial spread of virulent pathogens. A key finding from Chapter 4 is that although the widespread deployment of resource provisioning across a landscape can facilitate pathogen invasion and spatial spread, host occupancy can be maximized and infection prevalence can be minimized when an intermediate fraction of habitats is provisioned [55]. Chapter 5 further shows that increasing the fraction of provisioned habitats in a landscape can reduce infection prevalence and facilitate the extinction of a pathogen from host metapopulations when supplemental feeding promotes shifts towards sedentary behavior. Both frameworks more broadly demonstrate how the spatial extent of resource provisioning can affect the transmission of pathogens between populations.

Goals 1 and 2 demonstrate that changes in immune defense and dispersal in response to resource provisioning can determine whether pathogen transmission increases or decreases.

However, empirical studies linking food availability with these specific aspects of wildlife biology and infection are rarely conducted in natural systems. To address goal 3, Chapter 6 describes a multi-year study of how bacterial infection prevalence in common vampire bats (*Desmodus rotundus*) correlates with variation in the intensity of livestock rearing practices across a network of field sites in Peru and Belize (Chapter 6). Although vampire bats historically fed on wild mammals, populations residing near humans now preferentially feed on mammalian livestock and poultry owing to their greater accessibility and reliability [20,56]. This greater access to livestock prey increases the frequency of successful feeding [22,57], which could affect both vampire bat immune defense and demography given their extreme physiological sensitivity to starvation [58,59]. I therefore ask how resource provisioning in the form of local livestock biomass correlates with bat diet, demography, immunity, and infection with *Bartonella* spp. and hemotropic *Mycoplasma* spp., two bacterial pathogens. Results show that *Bartonella* prevalence declines with provisioning, which could occur from supplemental food enhancing bat immune defense. In contrast, hemoplasma prevalence does not vary with livestock biomass, which could occur if improved immune defense is offset by transmission-enhancing processes such as greater colony size or higher disperser attraction to livestock-dense habitats.

Collectively, this dissertation provides theoretically informed and testable predictions for how anthropogenic food resources affect host–pathogen interactions. My dissertation both builds and evaluates epidemiological theory for how food resources interact with infectious disease dynamics while examining the overall consequences of resource provisioning for the health status of a widespread bat reservoir host of zoonotic pathogens in Latin America. These contributions are particularly important given that epidemiology has typically ignored the role of host resources [60,61] and that many other bat species can likewise capitalize on anthropogenic

resources in urbanized and agricultural landscapes [34,62]. In terms of applied significance, these findings suggest that the spread of virulent pathogens can be mitigated when supplemental resources improve resistance to infection, do not facilitate large degrees of host dispersal, and are deployed in a limited manner across a landscape.

CHAPTER 2

TOO MUCH OF A GOOD THING: RESOURCE PROVISIONING ALTERS INFECTIOUS DISEASE DYNAMICS IN WILDLIFE[1]

[1] Becker DJ, Hall RJ. 2014 Too much of a good thing: resource provisioning alters infectious disease dynamics in wildlife. *Biology Letters* 10, 20140309. (doi:10.1098/rsbl.2014.0309). Reprinted here with permission of the publisher.

ABSTRACT

Provisioning of abundant food resources in human-altered landscapes can have profound effects on wildlife ecology, with important implications for pathogen transmission. While empirical studies have quantified the effects of provisioning on host behavior and immunology, the net interactive effect of these components on host–pathogen dynamics is unknown. We use simple compartmental models to investigate how provisioning-induced changes to host demography, contact behavior, and immune defense influence pathogen invasion and persistence. We show that pathogen invasion success and equilibrium prevalence depend critically on how provisioning affects host immune defense, and that moderate levels of provisioning can lead to drastically different outcomes of pathogen extinction or maximizing prevalence. These results highlight the need for further empirical studies to fully understand how provisioning affects pathogen transmission in urbanized environments.

Keywords: supplemental feeding; compartmental models; host-pathogen interactions; urbanization; feline leukemia virus

INTRODUCTION

The acquisition of food resources is a central driver of animal ecology [63]. Human activities can present wildlife with novel food resources that are abundant and spatio-temporally predictable. This resource provisioning can be accidental, as when wildlife capitalize on refuse or agricultural byproducts, or intentional, through supplemental feeding for management or recreation [5,64,65]. By increasing individual foraging success and thus fitness, provisioning can result in greater population size, and these subsidized populations can exert disproportionate influence on ecological processes, including pathogen transmission [4,32]. Given that provisioning often results in sustained contact between humans, wildlife, and domestic animals, understanding how provisioning influences host–pathogen interactions is a question of concern to public health, livestock well-being, and conservation [5,32,62].

Provisioning can modify multiple processes influencing pathogen transmission, potentially in opposing directions (Table 2.1). These tensions can be understood by considering their effects on the pathogen basic reproductive number (R_0), a threshold quantity determining pathogen invasion success and outbreak size [66]. For a close-contact pathogen with density-dependent transmission, R_0 is the product of the host population size, infectious period, and transmission rate. Provisioning can elevate R_0 by increasing host population size through improved survival and reproduction and contact rates via aggregation around resources [62,67]. Supplemental feeding can also improve host immune response by reducing nutritional stress and increasing body condition [68,69], which can have contrasting effects on R_0. Provisioning can decrease host susceptibility [61], thereby lowering the transmission rate, but improved nutrition can also increase tolerance to infection, prolonging the infectious period [70]. Practical limitations mean that the net interactive effects of provisioning on disease processes have rarely

been addressed in empirical studies. For example, an observational study of songbirds found highest measures of body condition at sites with intermediate urbanization, possibly due to high availability of bird feeders. These provisioned populations also showed the highest prevalence of antibodies to West Nile virus, but authors were unable to distinguish whether higher seroprevalence was due to increased exposure to urban vectors or recovery from disease via abundant resources [71]. Furthermore, although conceptual and mechanistic models have explored the influence of host resources on individual infection outcomes [72,73] and asked how provisioning will alter select components of R_0 (e.g., contact rate and population size; [61,62]), the interactions between individual- and population-level effects and their net epidemiological outcomes have not been fully explored. Here, we develop a general modeling framework explicitly incorporating provisioning into host demography, contact behavior, and immune defense and ask how their interaction influences resource-dependent thresholds for pathogen invasion and long-term prevalence.

MATERIALS AND METHODS

To explore mechanisms by which provisioning can influence pathogen transmission, we modify simple compartmental models of disease dynamics [66] and allow parameters reflecting host demography, contact behavior, and immune defense to depend on resource availability. Hosts are categorized according to infection status (susceptible, *S* and infected, *I*) and we consider two scenarios reflecting differing assumptions about pathogen clearance. For non-immunizing infections where recovered individuals can be immediately reinfected, disease dynamics are described by the susceptible–infected–susceptible (SIS) framework:

$$dS/dt = \big(b_0 - b_1(S+I)\big)(S+I) - \mu S - \alpha\delta SI + \gamma I$$

$$dI/dt = \alpha\delta SI - (\mu + \nu + \gamma)I$$

If pathogen clearance confers lifelong immunity, we introduce a third variable denoting recovered individuals (R) and use the susceptible–infected–recovered (SIR) framework:

$$dS/dt = \big(b_0 - b_1(S+I+R)\big)(S+I+R) - \mu S - \alpha\delta SI$$

$$dI/dt = \alpha\delta SI - (\mu + \nu + \gamma)I$$

$$dR/dt = \gamma I - \mu R$$

Host demography is described by a natural mortality rate μ and a density-dependent birth rate $b_0 - b_1 N$, where N is the population size and b_0 and b_1 are constants. We assume density-dependent pathogen transmission, but to account for opposing effects of resource-altered aggregation and resistance on transmission, we write the transmission parameter as the product of terms describing contact rate (α) and probability of infection upon encounter (δ). Pathogen clearance and disease-induced mortality occur at rates γ and ν respectively.

We incorporate provisioning through the parameter ρ, where increasing values reflect improved resource abundance and predictability. It takes values between 0 and 1, where $\rho = 0$ corresponds to no provisioning (and parameters take baseline values), while $\rho = 1$ reflects intensive supplemental feeding. The functional dependence of parameters on provisioning is assumed to be monotonic and saturating. If parameter x increases with provisioning, the functional form used is

$$x = x_{max} - (x_{max} - x_{min})e^{-\theta_x \rho}$$

while if x decreases with provisioning, we use

$$x = x_{min} + (x_{max} - x_{min})e^{-\theta_x \rho}$$

where x_{min} and x_{max} are the minimum and maximum values attained and θ_x describes the strength of the response to provisioning, allowing parameters to scale with resource availability in forms that range from a near-linear to quickly saturating effect.

The model was parameterized from published data on feral cats (*Felis catus*) infected with feline leukemia virus, a retrovirus transmitted primarily during aggressive encounters and sharing of food resources [74]. Since feral cats attain high densities in urban centers through supplemental feeding and foraging on human refuse, there is concern that increased aggregation will amplify viral transmission [65]. Following evidence from the published literature and this host–pathogen system, we assumed provisioning simultaneously influences host demography by increasing fecundity (b_0) and decreasing mortality (μ); contact behaviour by increasing encounter rates (α); and immune defence through reducing susceptibility and infectivity (δ) as well as disease-induced mortality (ν). Since reduced susceptibility and infectivity lowers transmission and acts antagonistically with demographic, behavioral, and tolerance effects on R_0 (Table 2.1), we investigate the net effect of this interaction by co-varying the strength of the immune response to provisioning ($\theta_\delta = \theta_\nu = \theta_{\delta\nu}$). Parameterization is detailed in the Supplementary Material (Appendix A), and model schematics and parameter dependence on provisioning are visualized in Figure 2.1.

We quantified the net effect of provisioning on pathogen invasion and long-term infection burdens through calculation of R_0 and equilibrium prevalence. Expressions for R_0 and prevalence were determined analytically (Appendix A) and plotted as functions of ρ under varying assumptions about the response of host immunity to provisioning.

RESULTS

Effects of provisioning on isolated mechanisms influenced R_0 and equilibrium prevalence predictably (Appendix A). Provisioning-increased survival and fecundity made pathogen invasion more likely by increasing the susceptible population size and infectious period. Provisioning increased equilibrium prevalence above baseline levels, but while SIS prevalence increased dramatically, SIR prevalence attained its maximum at intermediate provisioning but decreased at intensive supplementation owing to the increased proportion of immune individuals. Increasing contact rates also increased R_0 and prevalence. In contrast, changes to immunity reduced R_0 below the invasion threshold ($R_0 < 1$) and resulted in pathogen extinction.

When provisioning simultaneously affected demographic rates, contact behavior, and immune defense, various epidemiological outcomes were observed, contingent on how strongly provisioning influenced immunity (Figure 2.2). When provisioning had no influence on transmissibility and virulence, changes in demography and contact exerted a synergistic increase in R_0 eight-fold above baseline values ($\theta_{\delta\nu} = 0$; dashed line) and increased prevalence above maxima observed when provisioning influenced these mechanisms in isolation. Weak effects of provisioning on immunity ($\theta_{\delta\nu} = 1$; thin line) maximized R_0 at intermediate resource levels and dampened the prior increase in prevalence at intensive supplemental feeding. When provisioning

more strongly affected immunity ($\theta_{\delta v} = 2$; medium line), the observed maxima for R_0 and prevalence in both models decreased.

When provisioning induced a strong saturating immune response, equal to the response of demographic parameters ($\theta_{\delta v} = \theta_{b_0 \mu} = 4$; thick line), R_0 remained above one but attained a smaller maximum at lower resource levels, while prevalence patterns diverged between models. SIS prevalence was reduced at intensive provisioning but remained above baseline levels, while SIR prevalence was lower than prevalence in unsupplemented populations.

Inducing a quickly saturating effect of provisioning on immunity ($\theta_{\delta v} = 8$; thickest line) reduced R_0 below one at intermediate resource levels, meaning that pathogen invasion is only possible in unprovisioned or heavily provisioned populations. Equilibrium SIS prevalence at high provisioning exceeded baseline values, while SIR prevalence was lower in heavily-subsidized populations.

DISCUSSION

Provisioning has gained growing attention from ecologists and epidemiologists to better understand infectious disease dynamics in response to environmental change and to improve wildlife, domestic animal, and human health [32,61,62]. Theoretical and empirical studies have demonstrated that provisioning can increase pathogen prevalence and outbreak intensity through increases in aggregation and population size [62,67], while others suggest provisioning improves nutrition and immune defense in ways that lower infection burdens [64,71,75]. We demonstrate through simple, mechanistic models that interactions between provisioning-altered host demography, contact behavior, and immune defense can produce a range of disease outcomes in

urban-foraging wildlife, thereby providing a unifying framework for understanding opposing prevalence patterns from prior studies.

In agreement with previous work, our models suggest provisioning dramatically increases pathogen invasion success and long-term prevalence when primarily influencing demography or aggregation, and that synergistic interactions between them can amplify transmission. Importantly, our study is the first to reveal that this amplifying effect can be modulated or reversed when provisioning substantially reduces susceptibility through improved immune defense. Our model found R_0 to be maximized at intermediate provisioning when effects on resistance were weak, mirroring results from a modeling study where resource quality improved host resistance, fecundity, and pathogen shedding [61]. However, when host susceptibility dropped sharply in response to provisioning, prevalence was minimized or pathogen eradication occurred at intermediate resource levels. This pattern runs opposite of that predicted in other theoretical models [61,62,73] and suggests an important role of immune defense in resolving observed outcomes in which supplemental resources have decreased prevalence or catalyzed epidemics.

Understanding whether pathogen invasion increases with provisioning or attains its maximum or minimum at intermediate provisioning can be explained by the response strength of immune defense to provisioning relative to behavioral and demographic processes. Reduced susceptibility through provisioning initially drives down R_0 relative to unprovisioned populations, but this effect is overcome by greater susceptible density and encounter rates at high provisioning [66]. This has important implications for provisioning as a disease management strategy. First, provisioning will only reduce prevalence if resources strongly improve host condition and immunity. Second, even if provisioning is observed to reduce prevalence, further

supplemental feeding can result in pathogen resurgence and increased prevalence relative to unsubsidized populations.

Our results illuminate areas of empirical study necessary to improve our understanding of host–pathogen dynamics in provisioned environments. Because our model highlighted the key role of resource-mediated immunity for determining disease outcomes, experimental manipulations are needed to determine how provisioning influences susceptibility and infectivity. Additionally, although host recovery was not a function of provisioning in our model (owing to stronger evidence for virulence-related effects of improved nutrition), field studies suggest provisioning may decrease prevalence through improved pathogen clearance [71,75]. Thus laboratory food supplementation experiments quantifying immune defense could incorporate pathogen challenge to better elucidate this relationship [76].

Although we present simple models for directly transmitted pathogens, our framework can be expanded to include alternative transmission pathways, foraging behavior, and within-host infection dynamics. We have examined the case where the probability of infection given contact decreases with provisioning through improved immune defense; however, provisioned animals in better condition may enhance within-host pathogen replication [77], resulting in an increasing or convex relationship between infectivity and resources [73]. Spatial dynamics of disease may also be affected by provisioning; for example, a model of Hendra virus transmission in flying foxes proposed resources from urban gardens could increase aggregation and reduce population connectivity, resulting in explosive outbreaks [62]. Further modeling of interactions between subpopulations experiencing different resource levels and thus altered immunity could have profound implications for pathogen spread and persistence. We also assumed provisioned animals experience resources nutritionally equivalent or superior to naturally occurring

resources. Although realistic in cases of intentional provisioning for management and recreation, this assumption may not hold in situations where wildlife experience dietary simplification, as when foraging at refuse dumps. Poor-quality diets deficient in protein or micronutrients could limit or reverse provisioning-mediated improvements to immune defense [69]. Future work incorporating these ideas is a vital step towards predicting and managing disease outbreaks in humans, wildlife, and domestic animals in provisioned environments.

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Table 2.1. Potential effects of provisioning on parameters reflecting host demography, contact, and immune defense and the predicted effect on R_0. Shading indicates assumptions used in the modeling framework.

Mechanism	**Provisioning impact**	**Effect on parameters**	**Influence on R_0**	
Host demography	↓ nutritional stress	↑ birth rate	+	↑ population size
		↓ mortality rate	+	↑ infectious period
	↑ stress of crowding	↓ birth rate	–	↓ population size
		↑ mortality rate	–	↓ infectious period
Contact behavior	↑ aggregation	↑ encounter rate	+	↑ transmission rate
Immune function	↑ energy allocation toward host defense	↓ infection probability	–	↓ transmission rate
		↓ disease mortality	+	↑ infectious period
		↑ pathogen clearance	–	↓ infectious period
	↑ dietary simplification	↑ infection probability	+	↑ transmission rate
		↑ disease mortality	–	↓ infectious period
	↑ stress of crowding	↓ pathogen clearance	+	↑ infectious period
Pathogen performance	↑ resources for replication within host	↑ infection probability	+	↑ transmission rate
		↑ disease mortality	–	↓ infectious period

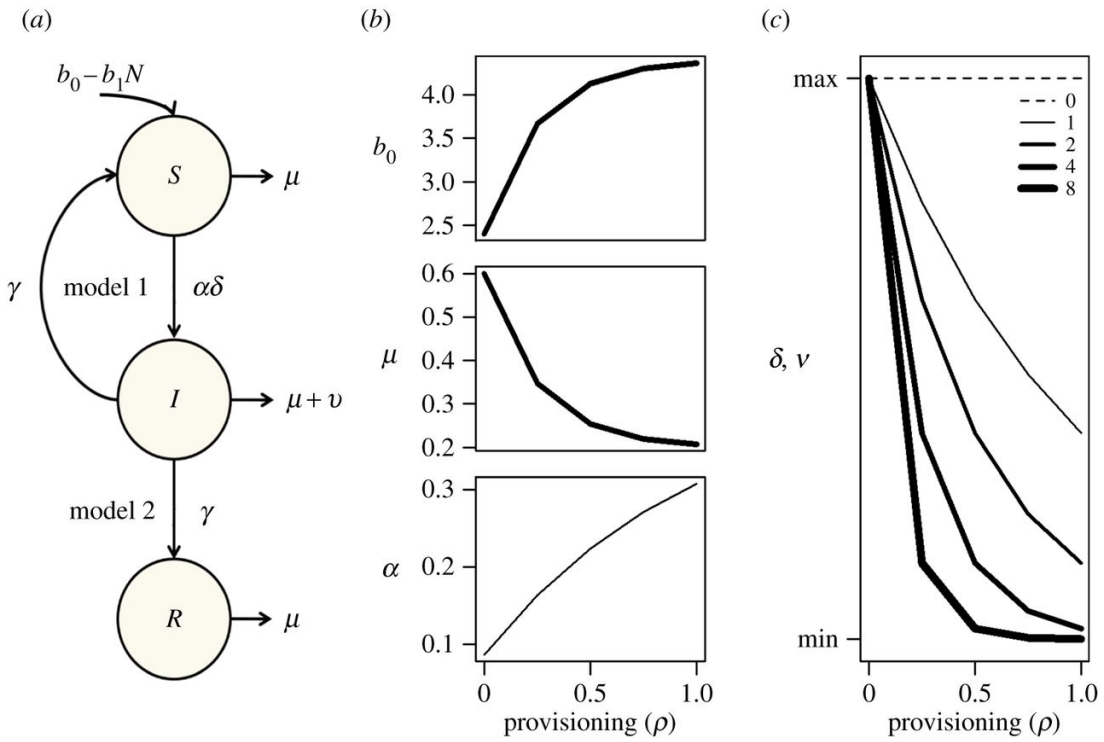

Figure 2.1. (a) Schematic of SIS and SIR models. (b–c) Effects of provisioning (ρ) on host fecundity (b_0), mortality (μ), contact (α), transmissibility (δ), and disease-induced mortality (ν). Line width depicts how strongly provisioning affects immune defence ($\theta_{\delta\nu}$ values shown in legend).

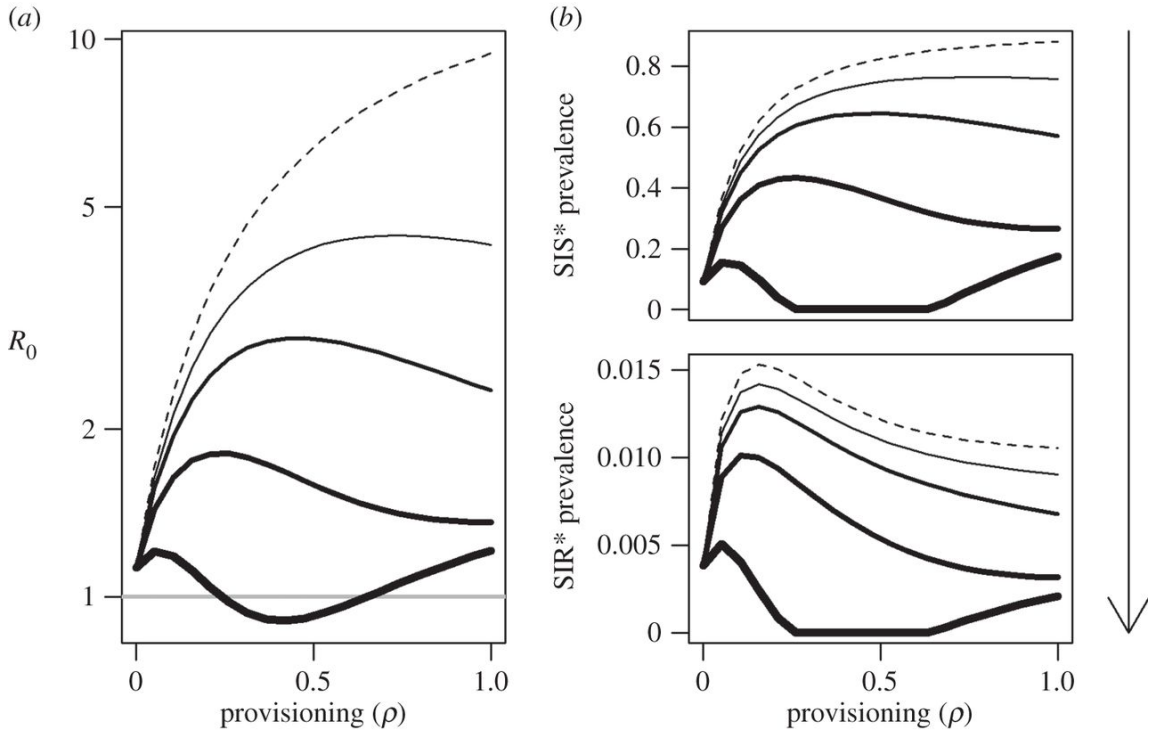

Figure 2.2. Effects of provisioning on (a) pathogen invasion success (R_0) and (b) equilibrium SIS and SIR prevalence. Line widths depict effects of provisioning on immunity as described in Figure 1, and the vertical arrow indicates an increasingly saturating effect on immunity. Grey line shows the pathogen invasion threshold, $R_0 = 1$.

CHAPTER 3

LINKING ANTHROPOGENIC RESOURCES TO WILDLIFE–PATHOGEN DYNAMICS: A REVIEW AND META-ANALYSIS [2]

[2] Becker DJ, Streicker DG, Altizer S. 2015 Linking anthropogenic resources to wildlife–pathogen dynamics: a review and meta-analysis. *Ecology Letters* 18, 483–495. (doi:10.1111/ele.12428). Reprinted here with permission of the publisher.

ABSTRACT

Urbanization and agriculture cause declines for many wildlife, but some species benefit from novel resources, especially food, provided in human-dominated habitats. Resulting shifts in wildlife ecology can alter infectious disease dynamics and create opportunities for cross-species transmission, yet predicting host–pathogen responses to resource provisioning is challenging. Factors enhancing transmission, such as increased aggregation, could be offset by better host immunity due to improved nutrition. Here we conduct a review and meta-analysis to show that food provisioning results in highly heterogeneous infection outcomes that depend on pathogen type and anthropogenic food source. We also find empirical support for behavioral and immune mechanisms through which human-provided resources alter host exposure and tolerance to pathogens. A review of recent theoretical models of resource provisioning and infection dynamics shows that changes in host contact rates and immunity produce strong nonlinear responses in pathogen invasion and prevalence. By integrating results of our meta-analysis back into a theoretical framework, we find provisioning amplifies pathogen invasion under increased host aggregation and tolerance, but reduces transmission if provisioned food decreases dietary exposure to parasites. These results carry implications for wildlife disease management and highlight areas for future work, such as how resource shifts might affect virulence evolution.

Keywords: host–parasite interactions; infectious disease ecology; urbanization; agriculture; supplemental feeding; foraging ecology; immune defense; aggregation; mathematical models

INTRODUCTION

Human activities and changes to the landscape can dramatically alter the types, abundance, and distribution of resources available to wildlife. These changes can affect nesting structures, shelter, and water but are particularly apparent for food resources. Urbanization, agricultural intensification, and overfishing have depleted food abundance for many wildlife through habitat degradation and reduction in prey stocks [78,79]. Many species decline in response to such activities, but some generalists thrive in human-dominated habitats by capitalizing on novel food resources [80,81].

Human provisioning of wildlife with food is geographically widespread, occurs at local and landscape scales, and can be intentional or accidental [4]. Bird feeders, supplemental feeding stations, and wildlife tourism are examples of intentional provisioning [5,9,46], whereas accidental food can be provided through agriculture, household waste, and landfills [24,82,83]. The high abundance and predictability of these resources across space and time can make them accessible components of wildlife diets, potentially resulting in populations that are larger, more aggregated, and better-fed [4,84]. Subsidized wildlife populations can in turn influence ecological processes ranging from trophic cascades to alternative stable states [19,30].

A growing number of studies indicate that anthropogenic resources can alter host–pathogen interactions, leading to either increased or decreased infection risk for wildlife and humans depending on the nature of provisioning and the particular host–pathogen interaction (Table 3.1). Heterogeneity in infection outcomes observed to date underscores the need for conceptual frameworks to reconcile these divergent consequences. This is especially important given that provisioning frequently brings different host species into contact and could facilitate host shifts and novel pathogen emergence, with consequences for wildlife conservation and

human health [32]. For example, bird feeders have been implicated in the spread of several songbird pathogens, including mycoplasmal conjunctivitis and a virulent strain of trichomoniasis, in part owing to transmission opportunities created by the close proximity and large aggregations of birds around human-provided food sources [36,85]. In Malaysia, the planting of fruit trees near pigsties is known to attract fruit bats to forage nearby, providing opportunities for the cross-species transmission of Nipah virus from bats to pigs and leading to human exposures [86].

Here we provide a conceptual framework for understanding how provisioning affects infection dynamics in wildlife and consider the practical implications for pathogen emergence and control. We start by reviewing empirical support for three mechanisms through which provisioning can affect host–pathogen interactions by altering (i) host immune defenses, (ii) host contact and movement behaviors, and (iii) host demography (Figure 3.1). These mechanisms can operate simultaneously and might have divergent effects on population-level disease outcomes. We next conduct a meta-analysis of empirical studies to characterize the range of outcomes observed in response to provisioning and assess the importance of host, pathogen, and environmental factors in determining whether infections increase or decrease in response to anthropogenic resources. Our analyses provide support for behavioral and immunological processes by which exposure, resistance, and tolerance are altered by provisioning. These analyses also identify pathogen type and food source as determinants of infection outcomes. To synthesize these findings, we review theoretical models examining the effect of provisioning on pathogen dynamics and integrate our meta-analysis results back into a mechanistic and predictive framework using the basic reproductive number R_0, a threshold quantity determining whether a pathogen can invade a host population, as a measure of pathogen fitness [87]. We conclude by

highlighting the management implications of our analyses and suggest avenues for future research on how wildlife–pathogen interactions respond to anthropogenic resources.

MECHANISTIC LINKS BETWEEN PROVISIONING AND PATHOGEN INFECTION

Feeding a fever: how resources alter immune defense

A host's ability to mount defenses and recover from infection depends on its nutritional state, which is influenced by both food quantity and quality [88]. Studies in humans, mice, and poultry show that energy and protein deficiencies can weaken immune cell function and complement proteins [69,89]. Moreover, nutrient deficiencies (zinc; iron; beta-carotene; Vitamins B6, B12, C, D, and E; and folic acid) can impair immune defense, especially in young and old individuals [90,91]. Malnourished wildlife can become immunosuppressed, which can increase pathogen replication and lead to higher host morbidity and mortality [92,93]. Therefore, by providing reliable food resources, provisioning could boost wildlife body condition and increase immune defenses, and could also allow wildlife to spend less time foraging and budget more time towards behavioral defenses such as grooming. This in turn could reduce pathogen fitness by decreasing individual susceptibility to pathogens and shortening the time to recovery following infection (Figure 3.1).

In support of this idea, work on kit foxes in California showed animals in residential areas to be in better condition compared to animals occupying a reserve, which weighed less and showed signs of dehydration and tissue catabolism [43]. Although exposure to three canine viruses was similar between the two groups, hematology suggested better immune status in urban foxes, likely owing to improved access to water and food and in turn reduced risk of starvation. Other studies of lace monitors foraging on human refuse found that resource-mediated increases

in body condition were associated with lower intensity of blood parasites compared to unprovisioned animals [40].

Under some conditions, supplemental resources could have the opposite effect of increasing host susceptibility to infection, which should increase rather than reduce R_0. Abundant but poor-quality anthropogenic food sources that are low in protein or high in fat could impair immune function, especially antibody-mediated defenses [94,95]. Although direct support for dietary shifts causing increased disease susceptibility in wildlife is rare, several case studies suggest this could occur. For example, supplemental feeding of rock iguanas by tourists in the Bahamas with carbohydrate-rich foods such as cereals and grapes was associated with altered nutritional status and increased hookworm burdens [96]. Similarly, southern stingrays fed by tourist boat operators in the Cayman Islands experienced impaired physiology resulting from poor nutrition and stress arising from crowding [97]. In addition, some forms of provisioning could enhance pathogen transmission by improving host tolerance to infection, thus allowing heavily infected animals to better survive and shed infectious stages [98,99].

Stay awhile and eat: resource-driven changes in host aggregation and dispersal

By providing concentrated and reliable resources, provisioning can reduce host foraging ranges, promote aggregation, and might favor more sedentary behavior as animals move less in search of food [84]. Such changes in response to greater resources have been observed in urban feral cats, which show more localized foraging with greater territory overlap around supplemental feeding stations compared to rural cats [100]. Higher aggregation and local host density could increase host contact rates, which should increase R_0 [51] (Figure 3.1). In support of this idea, wild raccoons experimentally provisioned with concentrated food resources had greater contact rates,

resulting in higher prevalence of endoparasite infections [67]. Additionally, elevated contact rates from flocking at bird feeders were suggested to cause greater spread of mycoplasma conjunctivitis in house finches [38]. Importantly, a positive response of pathogens to host aggregation requires that contact rates and pathogen transmission scale positively with local host density [101]. As one example, studies of vampire bats in Latin America suggest that the growing availability of blood meals from livestock rearing has facilitated range expansions and population growth of this host, which serves as the key reservoir for rabies virus [102]. However, despite a weak positive relationship between livestock density and bat colony size, rabies virus exposure was not associated with the latter, indicating that contact rates between susceptible and infected bats might not increase with host density [103].

Stable food sources might decrease host foraging movements and could encourage migratory or nomadic species to form sedentary populations [104]. For example, Spanish white storks in recent years have abandoned long-distance migration to Africa and instead now overwinter on urban landfills close to their breeding range [82]. Reduced host movement could increase pathogen transmission by allowing year-round exposure to pathogens that accumulate in the environment [104,105]. Importantly, sedentary populations could also lose connectivity with other groups, as has been suggested by work on urbanized flying foxes in Australia, leading to local viral extinction over short timescales and setting the stage for larger outbreaks following pathogen reintroduction [62].

Some host behavioral responses to provisioning could decrease infection risk, especially for parasites commonly encountered in the course of wildlife foraging activity, such as those with complex life cycles involving intermediate hosts. Work on Balinese long-tailed macaques suggested that increased feeding on tourist-provided food decreased the prevalence and intensity

of several gastrointestinal protozoa [42], possibly because provisioned habitats and food were relatively free of infectious stages found in natural environments. Similarly, provisioning decreased the prevalence of helminths recovered from subsidized raccoons and red foxes, possibly because hosts switched diets away from feeding on naturally infected intermediate hosts [41,106].

Food for the masses: how resources influence wildlife demography

Pathogen invasion and persistence rely on the supply of new susceptible hosts through births or immigration. Supplemental feeding has been shown to increase fecundity or shorten the time to first reproduction across a range of animal taxa [2,84,107]. Since offspring are typically born immunologically naïve (or become so after waning of maternal antibodies), heightened reproduction can increase the number of susceptible individuals and thereby elevate R_0 (Figure 3.1). Additionally, provisioning can reduce juvenile mortality rates by reducing starvation and improving overall condition, further contributing to the pool of susceptible hosts [108].

If novel resources increase local carrying capacities for wildlife, this could favor pathogen transmission by two well-known processes: the critical community size (a threshold population size at which stochastic extinction of pathogens becomes unlikely) and density-dependent transmission (in which pathogen prevalence scales positively with host density; [51]. Evidence to date for pathogen responses to provisioning-altered host demography is primarily indirect. For example, supplemental feeding of white-tailed deer and red deer elevates host densities, which has been suggested to increase the prevalence of bovine tuberculosis [109,110]. Similarly, urban gardens in Scotland had greater bumblebee densities and higher prevalence of

multiple pathogens [111], although direct links between population size and infection were not examined.

Although effects of provisioning on demographic processes are generally expected to increase transmission, complex patterns could arise for immunizing pathogens. If provisioning prolongs the survival of previously-exposed immune individuals more than it stimulates fecundity, this could increase herd immunity and reduce pathogen transmission. Thus, understanding precisely which demographic processes respond to anthropogenic resources and how this affects the age, sex, and immunological structure of populations is critical to anticipate the consequences of provisioning for host–pathogen dynamics.

META-ANALYSIS OF PROVISIONING EFFECTS ON INFECTION OUTCOMES

The examples and mechanisms noted above suggest that resource provisioning can generate wide variation in pathogen fitness. To better characterize the range of infection outcomes and to identify key predictors of this variation, we conducted a meta-analysis of empirical studies of microparasites (viruses, bacteria, protozoa, fungi) and macroparasites (helminths and ectoparasites). We focused on studies that recorded either pathogen prevalence (proportion of individuals infected), seroprevalence (proportion displaying a pathogen-specific immune response), or intensity of infection (average number of parasites per infected host) in provisioned and unprovisioned wildlife populations. Our specific goals were to i) characterize the breadth of studies in the provisioning–disease literature; ii) identify the range and average responses of infection; iii) identify host, parasite, and environmental factors that best explain variation in observed infection outcomes; and iv) test empirical support for our proposed mechanisms of immunological, behavioral, and demographic changes.

Literature survey and statistical approach

Scholarly articles were identified through Web of Science, Google Scholar, CAB Abstracts, and PubMed searches using strings of terms relevant to anthropogenic resources, wildlife ecology, and pathogen transmission. Our systematic search identified 144 studies meeting criteria for inclusion, of which 23 provided infection measures (prevalence, intensity, or seroprevalence). From each study, we recorded the relationship between provisioning and infection measures (effect size and directionality) along with the source and intention of provisioning, host and pathogen type, and transmission mode of the pathogen. Because many studies reported data for multiple pathogens or hosts, our data set included 132 records, where each record consisted of a particular host–pathogen combination. Further details on search procedures, criteria for study inclusion, categorical assignments, descriptive analyses, and tests of publication bias are provided in the Supplementary Material (Appendix B Figure S3.1).

To test support for mechanisms described above, we recorded whether studies quantified host condition or immune defense, contact behavior, or demography as well as how these measures covaried with provisioning. For the first mechanism, we considered studies that included body condition indices (e.g., mass~length residuals or subjective scoring) or quantified immune function (e.g., humoral or cellular components). Behavioral measures included group size, time animals spent foraging, dietary complexity, and contact rates. Demographic variables included host density and population size (as birth and death rates were generally not reported). Of the 23 studies, 52% (n=12) quantified host condition or immune defense, 43% (n=10) quantified behavioral changes, and, 26% (n=6) quantified demography (Appendix B Figure S3.3).

We collected standardized effect sizes from reported test statistics (e.g., r^2, odds ratios, χ^2) and sample sizes for each provisioning–infection outcome. When authors did not report test statistics, we derived effect sizes by simplifying data to a contingency table. If comparisons were made between several provisioned and unprovisioned groups, samples were pooled to calculate χ^2 statistics with Yates correction for prevalence or Hedges g for intensity [112]. If comparisons were made between different categories of provisioning, measures were compared between the most extreme levels [113]. We converted effect sizes into the correlation-based r [112,114] and assigned a negative value to cases where provisioning significantly reduced infection. Directional r effect sizes were transformed using Fisher's Z to stabilize variance [115].

Our analysis used random-effects models (REM) to infer the average effect of provisioning on infection. Next, we used mixed-effects models (MEM) to explain variation in infection according to pathogen type, transmission mode, host taxonomy, and provisioning type and source. Model simplification used backward removal of the least significant variable using Wald-type chi-square tests followed by nested likelihood ratio tests [116]. We calculated contrasts for our best-fit MEM to test if coefficients differed significantly from zero after adjusting for the potentially inflated false-discovery rate associated with multiple comparisons, using the Benjamini and Hochberg correction and the *multcomp* package in R [117–119]. Finally, we used MEM to test support for effects of resource-altered host condition, behavior, and demography on infection in each data subset reporting these variables. We used the R package *metafor* for r-to-Z effect size conversions and REM and MEM analyses [119,120].

Drivers of infection outcomes following provisioning

Our meta-analysis demonstrated that provisioning is associated with a wide range of infection outcomes in wildlife (Figure 3.2a). Of the 132 wildlife–pathogen interactions identified, most showed no relationship between provisioning and infection measures (65%, n=86), with 24% (n=31) identifying positive and 11% (n=15) identifying negative effects of anthropogenic resources. After adjusting for missing data due to suppression of extreme or non-significant results (Appendix B Figure S3.4), there was significant heterogeneity in infection outcomes (τ^2=0.18; Q=16902, df=176, p<0.001) but no net directional effect of provisioning in the REM (z=–1.79, p=0.07; Figure 3.2a).

MEM analysis of individual covariates demonstrated that pathogen type, transmission mode, provisioning type and source, and host taxonomy explained significant variation in infection outcomes (Appendix B Table S3.2 & Figure S3.5). Stepwise model selection and AIC further identified pathogen type and provisioning source as the strongest predictors (LRT=25.54, df=2, p=0.001; Table 3.2). Univariate MEMs of these covariates showed that hosts provisioned intentionally by wildlife management and recreational resources had higher infection measures, whereas hosts foraging on unintentionally provided sources in urban areas experienced reduced infection (Figure 3.2b). Additionally, infection measures for helminths and viruses generally increased with provisioning, whereas ectoparasites, bacteria, and protozoa showed no general response (Figure 3.2b). Our additive MEM integrating food source and pathogen type predicted infection with bacteria, helminths, and viruses to be significantly increased in recreational feeding areas (bacterium: z=2.54, p=0.04, helminth: z=3.44, p=0.01; virus: z=3.34, p=0.02), whereas infection with helminths and protozoa was predicted to be significantly reduced in hosts feeding on urban waste (helminth: z=–2.82, p=0.02, protozoan: z=–4.23, p=0.001; Figure 3.3).

Low sample sizes for agricultural sources of provisioning and fungi prevented detailed analysis of their relative effects.

We found mixed support for effects of resource-altered host immunity, behavior, and demography on infection outcomes (Figure 3.2b). Studies quantifying host condition or immune function showed roughly even evidence for positive, negative, and no responses of these variables to provisioning (Appendix B Figure S3.3). MEM analysis indicated that responses of condition to provisioning predicted changes in infection (Q=24.8, df=3, p<0.001). Somewhat surprisingly, both greater (μ=0.13, z=2.97, p=0.003) and poorer (μ=0.12, z=2. 75, p=0.006) host condition in provisioned wildlife were associated with greater infection measures (Figure 3.2b). Behavioral responses to provisioning also explained variation in infection (Q=45.3, df=2, p<0.001). Studies quantifying host behavior primarily found contact and aggregation to increase with provisioning (Appendix B Figure S3.3), which was associated with greater infection measures (μ=0.098, z=3.02, p=0.03). A subset of studies also found dietary diversity to decrease in provisioned populations, which was associated with lower infection measures (Figure 3.2b; μ=–0.8435, z=–6.01, p<0.001). This pattern could arise if provisioning reduces parasite exposure through decreased consumption of intermediate hosts or infectious stages in natural food. Lastly, demographic variables (abundance, density) showed either no effect or positive responses to provisioning (Appendix B Figure S3.3), but these differences did not predict infection outcomes (Q=3.58, df=2, p=0.16; Figure 3.2b).

Our meta-analysis demonstrates that wildlife–pathogen responses to provisioning vary widely, with pathogen type and food source explaining the greatest variation in infection. Some pathogens that increased in response to provisioning, such as *Mycobacterium bovis* in deer and *herpesvirus* in raptors, are spread through close contact, while others such as *Cryptosporidium* in

possums are transmitted through environmental infectious stages. Both transmission routes could be favored if provisioning increases host aggregation and encourages sedentary behavior, increasing exposure to infected conspecifics and to pathogens shed into the environment. The source of provisioning also predicted variation in infection outcomes, with intentionally managed and recreational resources generally increasing infection. For example, feeding stations to manage elk in the greater Yellowstone area during the winter months attract high densities of hosts, support sedentary behavior and allow for the buildup of environmentally transmitted parasites, and in turn increase exposure to bacterial pathogens and helminths [46,48]. In another study, feeder station density was associated with greater nematode prevalence and intensity in wild boar [121]. Accordingly, our best model predicted infection with such pathogens to be highest in hosts foraging at managed and recreational resources (Figure 3.3), lending support to provisioning amplifying transmission by creating hubs of high host contact and pathogen shedding in supplemented feeding environments.

Another mechanism to explain increased helminth transmission in provisioned habitats could be that well-fed hosts constitute a better reproductive environment for macroparasites [77], as supported through our finding that resource-improved condition predicts greater infection. Yet our analysis also found that provisioning can reduce body condition and immune function in some wildlife species, which was similarly associated with increased infection. Two non-exclusive mechanisms could underlie this pattern. First, some provisioned resources might be of low quality and lack nutrition, especially protein, needed for mounting immune defenses [92,94]. Food provided to wildlife with good intentions could also contain contaminants that hamper immune defense. For example, one study in our analysis found supplemental food used to improve breeding success of imperial eagles contained pharmaceuticals, which depressed

immune function and elevated infection by multiple pathogens [122]. Second, crowding and high intra-specific competition around resources could function as a stressor that impairs host condition [123]. One study of tourism in our analysis suggested this process, as provisioned stingrays intensively competed for food and in turn showed lower condition, higher injury rates, and increased ectoparasites burdens [124]. From a broader perspective, these results demonstrate negative fitness consequences of anthropogenic resources, suggesting that some provisioned habitats function as ecological traps for wildlife [125].

Altogether, our findings provide support for several processes by which provisioning elevates host exposure and susceptibility to pathogens. However, our analyses also support pathways by which pathogen transmission is lowered in response to novel resources. Our best-supported model showed that hosts foraging on resources unintentionally provided in urban habitats experienced reduced infection with protozoa and helminths (Figure 3.3). This result may be driven by dependence on trophic transmission, for which shifts towards easily accessible anthropogenic food could reduce the consumption of natural intermediate hosts. In one study included in our analysis, reduced dietary breadth of ring-billed gulls foraging in urban areas was associated with lower helminth burdens, as birds fed more on urban waste and less on naturally infected intermediate hosts such as snails and crustaceans [126].

Surprisingly, despite support for increases in host population size and density following provisioning, we found no effects of these demographic responses on infection outcomes. The failure of infection outcomes to scale with demographic patterns might reflect a dominance of frequency-dependent rather than density-dependent transmission in the studies analyzed [51]. The particular demographic process (birth or survival) that is affected by provisioning might have a stronger impact than change in population size. In particular, if provisioning increases

survival more than reproduction, this could decrease transmission through a buildup of herd immunity. This highlights the importance of measuring not just population size, but also the underlying demographic process generating larger population sizes in provisioned populations.

INTEGRATING RESOURCES INTO EPIDEMIOLOGICAL MODELS

Our review and meta-analysis suggest multiple processes through which provisioning can alter infectious disease dynamics. Because these mechanisms can act simultaneously and with potentially opposing directional effects, modeling approaches are critical for predicting the overall effect of provisioning on pathogen invasion and spread. Below, we review several recent studies that used empirically informed mechanistic models to better understand how host resources affect pathogen dynamics. We then integrate the best-supported relationships from our meta-analysis back into a mechanistic framework to gain a deeper understanding of processes underlying the observed variation in infection outcomes.

Review of resource-dependent modeling approaches

Mathematical models that examine food provisioning and infectious disease dynamics include both system-specific and general theoretical approaches. Motivated by field observations showing that prevalence of fungal pathogens of *Daphnia* increased when lake resources were poor and declined when resources improved, one study integrated experimental resource manipulation with a mechanistic modeling approach [61]. Experiments showed positive relationships between resource quality and both host fecundity and fungal spore production, which likely favored transmission; however, greater resources also lowered host susceptibility to infection, slowing down transmission. When these empirical relationships were integrated into a

compartmental model tracking the density of susceptible and infected hosts as well as free-living spores, simulations showed that R_0 was maximized at intermediate resources and declined at both high and low resource levels.

A more general modeling approach by Becker and Hall [52] examined how resource-modified host demography, contact behavior, and immune defense alter the transmission of close-contact microparasites (Box 3.1). By coupling functional responses of parameters including host birth and death rates, infection probability, tolerance of infection, and contact rates to provisioning, this model generated a range of pathogen invasion outcomes (Figure 3.4). In particular, host immune defense emerged as critical to predicting the net effect of provisioning on R_0. When provisioning had minimal effects on host immunity, the positive effects of provisioning on host density and contact rates resulted in higher pathogen invasion. Yet when immune defense increased with provisioning in a saturating response, pathogen extinction occurred at intermediate resource levels and invasion was only possible at low and high resources (Figure 3.4), a pattern opposite of the modeling outcomes of [61]. In the latter model, low immunity at low resource levels allows pathogen invasion despite relatively low contact rates. At intermediate resources, high resistance to pathogens leads to low prevalence or pathogen extinction. At even greater resource levels, the continued increase in host contact rates overcomes host resistance and allows the pathogen to reach high prevalence (Figure 3.4). By demonstrating that R_0 changes along a gradient of provisioning and by showing how this depends on underlying individual-level effects of resources, this approach provides a useful reference point for understanding the divergent patterns in disease outcomes observed in prior empirical work.

Some recent studies have extended models of local-scale dynamics to account for spatial heterogeneity in resource provisioning. As one example, a spatial model of Hendra virus dynamics in flying foxes examined how the combination of host aggregation around urban resources (and resulting sedentary behavior and loss of connectivity) influenced viral invasion and persistence [62]. In particular, decreasing connectivity associated with urban areas increased epidemic size by increasing the duration of time between viral introductions, allowing subpopulations to recruit more susceptibles in the absence of infection and permitting the local loss of herd immunity. When decreased connectivity was modeled alongside urban aggregation, simulations produced the largest viral outbreaks in urban bat colonies, likely increasing the risk of spillover infections to other host species [62].

Modeling work on resource-driven infection dynamics to date has generally focused on microparasites, ignoring the heterogeneities in infection intensity and external transmission stages that characterize most macroparasites [127,128]. Importantly, expressions for R_0 in macroparasite models depend on several parameters not represented in microparasite models, including the rate of production of free-living stages by adult worms, host uptake of infectious stages from the environment, and the mortality rate of adult parasites within their hosts, all of which could be influenced by provisioned resources. For example, well-fed hosts might provide better environments for macroparasite reproduction and survival, translating into greater R_0 [77]. Shifts away from natural food sources could also reduce exposure to infective stages, with the opposite effect of lowering R_0 [126]. Future work that builds these resource-dependent relationships into macroparasite models will offer important advances for understanding divergent infection outcomes of resource provisioning.

Predicting the effects of provisioning on R_0 of a microparasite

We integrated modeling and empirical work by building the best-supported relationships from our meta-analysis into a mathematical model to examine effects on pathogen invasion (R_0). Following the framework of [52], we set parameters for a susceptible–infected–recovered model to depend on resource levels (Box 3.1 and Appendix B) and examined two different transmission scenarios (close contact versus dietary exposure). Because our meta-analysis suggests that anthropogenic provisioning might generally increase host susceptibility to infection (Figure 3.2b), we assume an increasing per-contact probability of infection (δ) with provisioning. We also assume that host tolerance increases with provisioning, by modeling the disease-induced mortality rate (ν) as a negative function of resources. Together, these two processes elevate R_0. For pathogens transmitted by close contact, our analysis supported greater aggregation of hosts around resources, which likely increases contact rates (α). For pathogens transmitted through dietary exposure, studies indicated that provisioned diets could bypass parasite infectious stages (especially intermediate hosts). Finally, although our analysis found no significant support for resource-altered demography in driving infection, over half of the studies examined here found that provisioning affected demographic variables. We therefore follow [52] in assuming host birth (b_0) increases and background mortality (μ) decreases with provisioning, but vary the strength of how these parameters respond to provisioning (as described in Box 3.1). Thus, our revised modeling framework includes increased host susceptibility and tolerance to infection alongside a range of weak to strong positive effects on host fecundity and survival. To account for different scenarios in which provisioning could (a) increase host contact or (b) decrease dietary exposure, we perform two simulations that capture these processes separately (through positive versus negative associations between provisioning and host exposure, α).

Our new simulations show that when provisioning increases host contact rates, the net outcome is an increase in R_0 (Figure 3.5a). Even when host birth and background mortality remain unchanged, greater provisioning elevates R_0 far above baseline levels due to the combined effects of increased contact, higher susceptibility, and improved host tolerance. When provisioning increases host birth rates and survival, we observe an even stronger increase in R_0 similar to that found by [52]. These interactive processes would predict a net positive influence of provisioning on pathogen fitness, consistent with some studies in our analysis but counter to the average trend (Figure 3.2a).

Importantly, modifying the model to assume that provisioning reduces dietary exposure to pathogens predicts different outcomes for R_0 (Figure 3.5b). Under this scenario, when host fecundity and lifespan are unaffected by provisioning, reduced dietary exposure drives the pathogen to extinction, despite greater host susceptibility and tolerance to infection. If host survival and fecundity increase with provisioning, the pathogen can invade and persist at low to moderate resource levels. At high levels of provisioning, resource-altered dietary exposure dominates the overall effect on R_0, driving the pathogen below the invasion threshold. This model prediction might explain cases in our analysis where supplemental resources decreased infection measures or had no net effect. The sensitivity of our model both to pathogen exposure routes and to demographic processes further highlights the need for detailed empirical studies of underlying mechanisms to understand outcomes for different wildlife–pathogen interactions.

MANAGEMENT IMPLICATIONS, FUTURE DIRECTIONS, AND CONCLUDING REMARKS

Given the diverse responses of wildlife behavior, immunity, and demography to dietary provisioning, and the potential for these changes to alter pathogen transmission within and between species, an important question is whether and how to manage pathogen risks to humans and wildlife arising from provisioning. Our analyses suggest that focusing on specific food sources and pathogen groups could improve disease management, as these together explained substantial variation in infection outcomes (Table 3.2). In cases where microparasites are spread through close contact, solutions might involve spacing apart feeding stations to limit host aggregation, maintaining natural food sources, or preventing access to anthropogenic food altogether. As one example of this approach, in Uganda, better management of livestock grazing and encouraging the conservation of natural forest habitats have been proposed to mitigate enteric bacterial transmission between humans, domestic animals, and wild primates, the latter of which frequently forage in agricultural fields [129]. For helminths or environmentally transmitted microparasites, solutions might involve periodic rotation or cleaning of feeding stations to limit the buildup of persistent infectious stages [130]. When anthropogenic resources are found to lower host immune defenses, food could be fortified to make wildlife diets more nutritionally balanced [96]. Moreover, wildlife managers could use supplemented food sources to distribute vaccines or treatment to wildlife, taking advantage of oral bait vaccines such as those used for rabies and bovine tuberculosis [131,132]. Finally, public outreach to promote awareness of how supplemental feeding affects the spread of wildlife pathogens or poses risks for human exposures might reduce transmission opportunities and limit human–wildlife contacts that allow pathogens to move in either direction.

Understanding how wildlife–pathogen dynamics respond to provisioning offers exciting challenges for new work (Box 3.2). Future studies could focus on systems where supplemental

feeding is already known to affect host population dynamics or community interactions, but for which direct effects of feeding on pathogen transmission have not yet been quantified. For example, despite the popularity of recreational bird feeding [5], our review identified only four studies in which avian disease was explicitly quantified in the context of supplemental food (Appendix B Figure S3.2). Work in these tractable systems would benefit from longitudinal and experimental approaches, especially necessary to test how provisioning affects host immune defenses, demography, and rates of recovery and pathogen shedding. To this end, researchers might capitalize on the human–wildlife connection inherent in provisioning by involving the public through citizen science projects and engaging with civic and recreational organizations during the design of wildlife surveillance programs. Similarly, collaboration with sociologists, anthropologists, and geographers can elucidate behavioral and socioeconomic drivers of provisioning and quantify human–wildlife interactions around these resources to better understand risks of human exposures and guide control strategies [133].

Our analysis demonstrates the utility of mathematical modeling to predict how anthropogenic resources affect host–pathogen dynamics. Such theoretical approaches have mainly focused on microparasite transmission, and a need remains to develop macroparasite models that capture effects of food provisioning. Building on established frameworks for helminth dynamics [127], future models could examine how resource dependence influences adult parasite survival and egg production, parasite impacts on host survival and fecundity, and parasite encounter rates through host foraging [128]. Another important step for mathematical models is the development of spatial frameworks that capture local- and regional-scale heterogeneity in provisioning and allow for host movement between provisioned and unprovisioned patches [62,134]. Finally, future modeling studies can borrow from community

ecology to understand more complex interactions between hosts, pathogens, and resources, including multiple host or pathogen species or predators and their prey.

From an evolutionary perspective, an exciting avenue for future work is to ask how provisioning might affect host resistance and pathogen virulence evolution. Increased opportunities for pathogen transmission are expected to favor the evolution of more virulent pathogen strains [135,136], but empirical data to test this prediction in the context of transmission opportunities arising from provisioning are lacking. From a different perspective, our analysis suggests anthropogenic resources can in some cases allow wildlife to better tolerate infection. This association is corroborated by laboratory evidence demonstrating improved nutrition prolongs the survival of infected animals and increases the duration of pathogen shedding [61,70,99]. More tolerant hosts could further select for faster-replicating pathogen strains that cause greater virulence [137]. Thus, although resource-improved condition could reduce disease-induced mortality in the short-term, provisioning could favor the evolution of more harmful pathogen strains in the longer term [138]. Evolutionary models exploring the impact of improved tolerance within the context of other immune, behavioral, and demographic effects are needed to predict the long-term consequences of provisioning for wildlife and human health.

As human populations expand, natural habitats and food sources for many wildlife species will continue to be replaced by human-dominated landscapes and anthropogenic resources. These changes will have profound effects on the spatial and temporal distribution of wildlife and on their interactions with parasites and pathogens. Our review underscores the need to better understand how food resources affect wildlife physiology and behavior and how changes at individual- and local-scales alter landscape-level pathogen dynamics. Our synthesis of

evidence to date highlights the fact that provisioning effects on infection depend crucially on details of the host–pathogen interaction; however, some generalities nevertheless arise based on pathogen type, transmission mode, sources of novel food, and the relative impacts on host behavior and immunity. Future integration of long-term field studies, experimental approaches, and mathematical models of provisioning are needed to define more robust mechanistic frameworks and to guide efforts to mitigate infection risks for wildlife, domesticated animals, and humans.

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Table 3.1. Examples of host–pathogen interactions that respond to provisioning, including the anthropogenic resources provided, pathogens affected, and observed impacts on the host. Shading indicates mechanisms suggested to underlie observed infection outcome in the main text (grey = condition and immune defense, blue = contact behavior or pathogen exposure, green = host demography, orange = inter-species mixing). Symbols $(+/-/0)$ indicate whether infection measures generally increased or decreased with food provisioning. Images are from Wikimedia Commons.

Host species		**Food resource**	**Pathogens affected**	**Provisioning impact**	**Refs**
	Lace monitor (*Varanus varius*)	Accidental Urban waste	*Haemogregarina varanicola* (–)	Improved nutrition and condition, lower parasite intensity	[40]
	Elk (*Cervus elaphus*)	Intentional Management	*Brucella abortis* (+)	Increased contact rates, higher pathogen exposure	[46]

	Common vampire bat (*Desmodus rotundus)*	Accidental Agriculture	*Rabies virus* (0)	Larger colonies in livestock regions, but no effect on viral exposure	[103]
	European greenfinch (*Chloris chloris)*	Intentional Recreation	*Trichomonas gallinae* (+)	Aggregation and species mixing around feeders could increase transmission	[36]
	Long-tailed macaque (*Macaca fascicularis*)	Intentional Recreation	*Giardia* (–) *Endolimax* (–)	Decreased infection likely from reduced exposure and better nutrition	[42]

	Red fox (*Vulpes vulpes*)	Accidental Urban waste	*Echinococcus multilocularis* (–)	Decreased consumption of intermediate hosts reduces exposure	[41]
	White-tailed deer (*Odocoileus virginianus*)	Intentional Management	*Mycobacterium bovis* (+)	Supplemental feeding increases transmission via density dependence	[109]
	Indian flying fox (*Pteropus giganteus*)	Accidental Agriculture	*Nipah virus* (+)	Overlap between mango and pig farms increases viral spillover	[86]

Table 3.2. MEM rankings for predicting infection outcomes of provisioning, including the R^2 derived from likelihood ratio tests against the base REM.

MEM models	weight	ΔAIC	R^2
~ pathogen + source	0.50	0.00	17.85
~ pathogen + source + host	0.40	0.44	20.77
~ pathogen + source + host + transmission	0.04	5.04	21.62
~ source	0.03	5.36	7.58
~ pathogen + source + host + transmission + type	0.02	7.01	21.64
~ type	0.01	8.12	2.51
~ pathogen	0.00	10.48	6.77
~ host	0.00	11.86	2.59
~ transmission	0.00	14.55	0.61

Box 3.1. A compartmental model of microparasite dynamics in response to provisioning.

Because resource provisioning simultaneously affects individual- and population-level processes that can interact in opposing ways, mechanistic models can help resolve the net outcome for host–pathogen dynamics. In a modeling framework describing the effects of provisioning on microparasite systems outlined by Becker and Hall [52], hosts were categorized according to infection status (susceptible, *S*; infected, *I*; and recovered, *R*, where recovered hosts retained lifelong immunity), with susceptible hosts infected at the density-dependent rate $\alpha\delta SI$. Increasing provisioning, tracked by the parameter ρ, reflects improved resource abundance and predictability, where $\rho = 0$ corresponds to no supplemental feeding and $\rho = 1$ reflects intensive provisioning. Provisioning here was assumed to be nutritionally complete, and parameter functional dependence on resources was assumed to be monotonic and saturating. If parameter x increased with provisioning, the functional form used was

$$x = x_{max} - (x_{max} - x_{min})e^{-\theta_x \rho}$$

and if x decreased with provisioning, the relationship was described by

$$x = x_{min} + (x_{max} - x_{min})e^{-\theta_x \rho}$$

where x_{min} and x_{max} are the minimum and maximum values attained and θ_x describes the strength of the effect of provisioning. Through the shape parameter θ_x, model parameters could

scale with provisioning in forms that assume a weak but continuously increasing relationship to those assuming a strong, quickly saturating response.

Following expectations from the literature on the behavioral and physiological response of wildlife to provisioning, model parameters describing demographic rates (birth, b_0 and mortality, μ), contact behavior (encounter rate, α), and immune defense (susceptibility, δ and tolerance, ν) were set to depend on ρ, with birth, contact, and tolerance increasing with provisioning and mortality and susceptibility decreasing with provisioning (Figure 4a). Since reduced susceptibility due to improved immune function would reduce transmission rates and therefore counter other changes that could increase pathogen spread (larger host population size, increased contact rates), the net effect of this interaction on infection dynamics was examined by co-varying the strength of the responses of both susceptibility and tolerance to provisioning ($\theta_\delta = \theta_\upsilon = \theta_{\delta\upsilon}$; an increasingly saturating effect is shown through line width in Figure 4b). The net effects of provisioning on pathogen invasion and outbreak capacity were inferred from analytic derivation of R_0 in the SIR system. Further details, model parameterization, and long-term epidemiological consequences of provisioning (equilibrium prevalence) are provided by Becker and Hall [52]. Ordinary differential equations of the SIR model and the analytic expression for R_0 are given in the Supplemental Material (Appendix B).

Simulations generated a range of R_0 outcomes based on specific effects of provisioning (Figure 3.4c). When provisioning affected host demography and contact behavior but not susceptibility and tolerance (dashed line, Figure 3.4c), this resulted in a dramatic increase in R_0. However, this effect was modulated or even reversed when provisioning increased host immune response (increasing line width, Figure 3.4c). In this case of strong effects on host susceptibility and tolerance, R_0 was minimized below the invasion threshold ($R_0 = 1$) at intermediate levels of

provisioning, indicating that provisioning can terminate epidemics. Hence depending on the response of host immune defense and the magnitude of provisioning, anthropogenic resources might result in explosive outbreaks and enhance pathogen fitness or could minimize prevalence and allow for pathogen extinction.

Box 3.2. Outstanding needs for future work at the interface of provisioning and wildlife–pathogen dynamics.

1. **Moving beyond associational field studies**
    - More intensive longitudinal and spatial monitoring of provisioned populations are needed to capture different resource levels and seasonality in responses.
    - Need to better quantify underlying mechanisms (immune defense, contact behavior, dietary avoidance, birth and death rates) in the field.
    - Experimental manipulations of food sources and pathogen infection (i.e., pathogen removal studies) are needed to move beyond correlational outcomes.
2. **Examining within-host responses to resource quality and quantity**
    - Experimental studies of ecologically relevant field systems could test how diet quality (protein and energy content) and quantity (abundance and distribution) influence immune defense (including innate and adaptive immune pathways) and susceptibility and tolerance to specific infections.
    - Field and experimental studies are needed to ask how dietary shifts through provisioning affect the host microbiome and the resulting consequences for host condition and individual susceptibility to infection.
3. **Developing new modeling approaches**
    - Macroparasite models are needed to develop a mechanistic understanding of how environmentally transmitted and complex life cycle parasites respond to novel resources.
    - Spatial models that account for effects of resource heterogeneity on local dynamics and movement–connectivity will be essential for understanding the persistence and spatial spread of infection.

4. **Community context and host–pathogen evolution**

- Multi-host modeling frameworks could explore how differential species contributions to parasite fitness are altered by the presence of novel resources, including broader potential for dilution or amplification effects on disease risk.
- Predator–prey–pathogen models could ask how provisioned resources for predators and prey alter dynamical interactions.
- Field and modeling studies are needed to understand whether and how supplemental feeding could influence the evolution of pathogen virulence and host resistance/tolerance to infection.

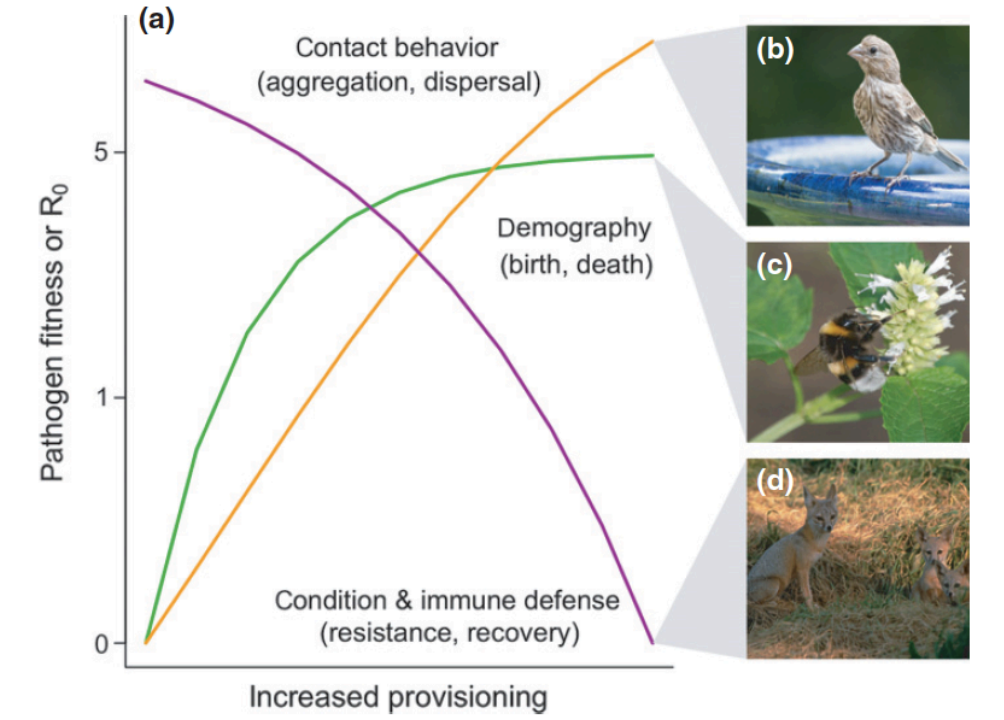

Figure 3.1. Predicted relationships between provisioning and R_0 (where $R_0 = 1$ is the pathogen invasion threshold). Aggregation around resources could increase host contact rates and infectious stage buildup in the environment (a; orange), an effect illustrated by increased flocking of house finches at bird feeders and associated increases in conjunctivitis prevalence [38]. Provisioning can also improve host vital rates and increase host population sizes (a; green), which was suggested to explain higher pathogen prevalence among bumblebees in urban versus rural gardens [111]. Positive effects of provisioning on R_0 could be countered by improved host condition and immune defense (a; purple). Such an effect is suggested by kit foxes showing lower nutritional stress, higher body condition, and improved immune function in urban areas where food and water was more plentiful [43]. Images are provided by Wikimedia Commons.

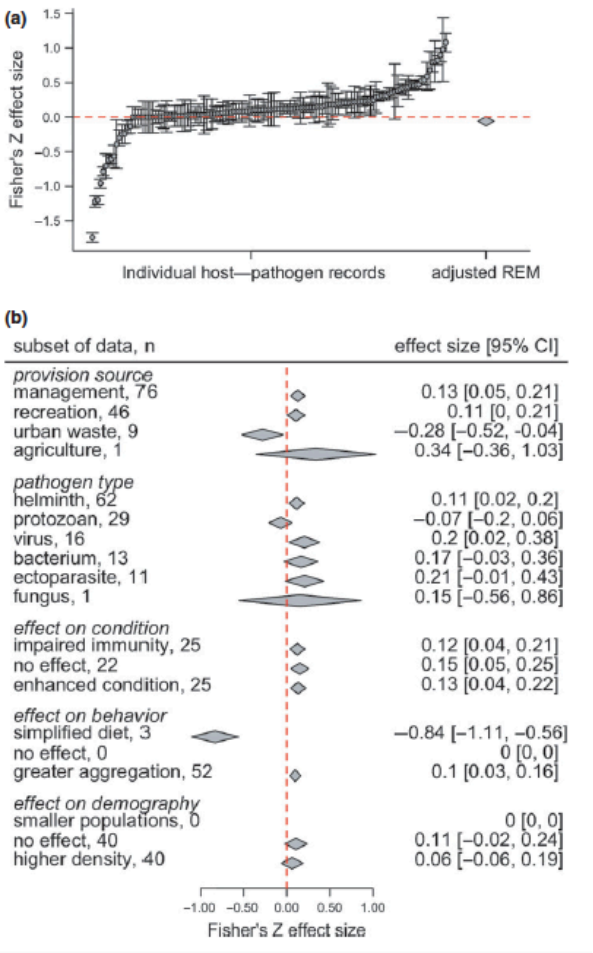

Figure 3.2. Distribution of effect sizes for observed relationships between provisioning and infection outcomes (points ± 95% confidence intervals) alongside the mean effect size estimate (diamond) from the bias-corrected REM (a). Each point is a particular host–pathogen interaction. Points above the horizontal line demonstrate cases where provisioning increased infection prevalence, intensity, or seroprevalence; points below the horizontal line demonstrate reduced infection outcomes. (b) Estimated mean effect size of predictors on infection outcomes, denoted through diamonds alongside 95% confidence intervals. Sample size (n) refers to the number of host–pathogen interactions corresponding to each level. Positive effect sizes indicate increases in infection outcomes (measures of prevalence, seroprevalence, and intensity are pooled).

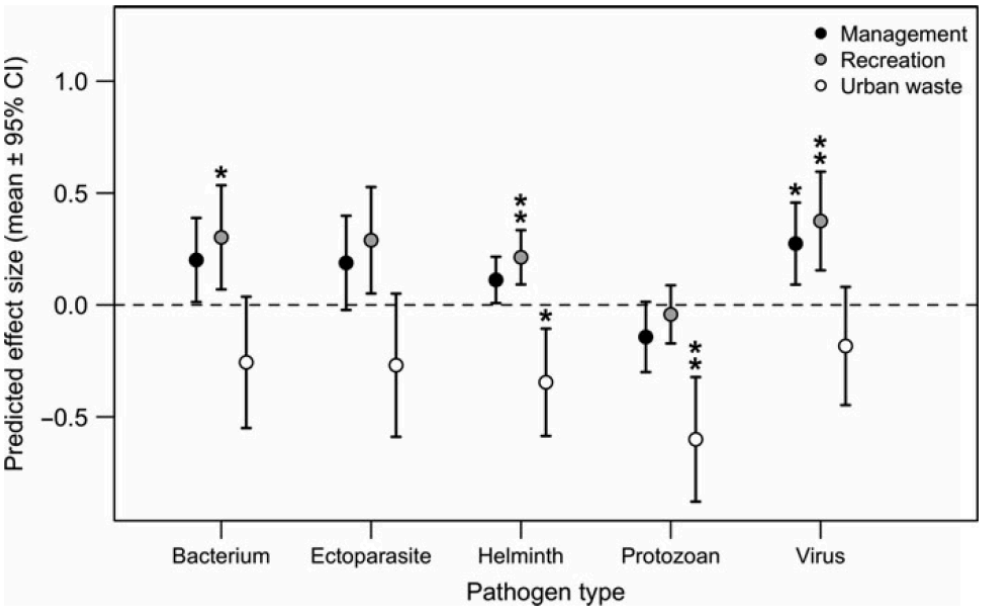

Figure 3.3. Visualization of the MEM explaining the most variation in infection outcomes from the meta-analysis. Data points represent the predicted outcome of provisioning for each combination of food source (see legend) and pathogen type, where the horizontal line represents no influence of supplemental feeding on infection. Asterisks represent means significantly different from zero after adjusting for multiple comparisons (*p<0.05, **p<0.01). Effects based on agricultural food and fungal pathogens not shown owing to limited data.

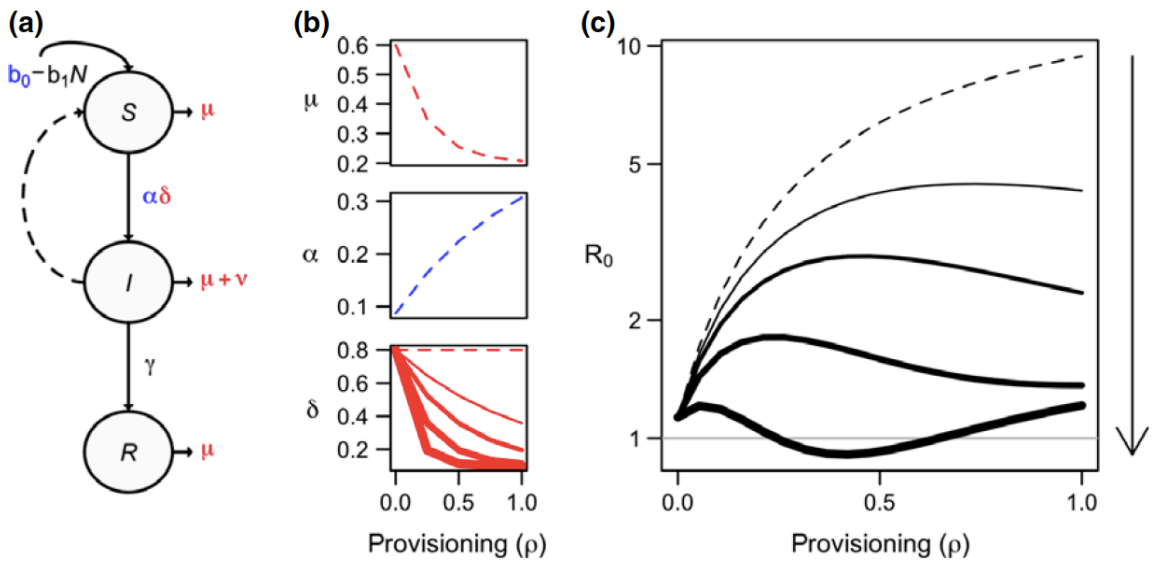

Figure 3.4. General modeling framework for how provisioning affects infectious disease dynamics of a microparasite (Box 3.1). In this compartmental framework (a–b), provisioning causes key parameters to increase (shown in blue) or decrease (shown in red). Varying the response of immune parameters to provisioning generates a range of outcomes on R_0 (c). An increasingly saturating effect of provisioning is shown through line width (dashed indicates no effect on immunity), and this approach can generate outcomes ranging from amplifying prevalence to driving R_0 below the invasion threshold (grey line). Figure is adapted from Becker and Hall [52], and further model details and parameter definitions are provided in Box 3.1.

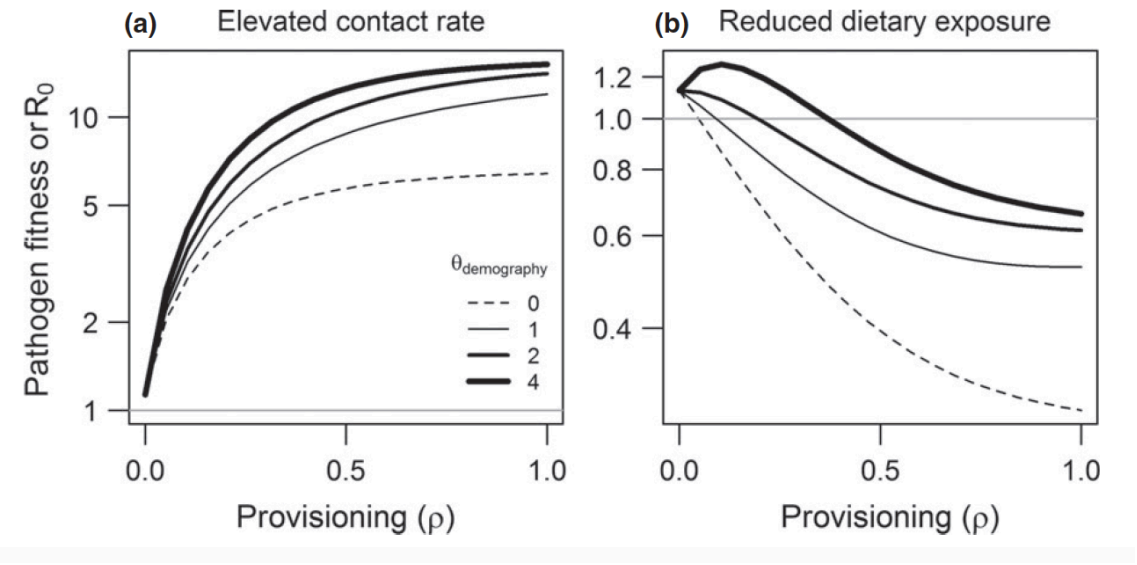

Figure 3.5. Meta-analysis–guided re-assessment of provisioning effects on pathogen invasion via mathematical models. Simulations examine net effects of resource-mediated processes on R_0 by considering two independent behavioral mechanisms supported by our analysis, in which provisioning either elevates contact rates (a) or decreases dietary exposure to pathogens (b). Along with incorporating the above effects and those of resource-altered resistance and tolerance, the model includes potential influence of resource-altered demography, where line width indicates how strongly these parameters respond to provisioning (shown in the legend). Simulations follow parameterization given in [52], and the analytic expression for R_0 is provided in the Supplemental Material (Appendix B).

CHAPTER 4

HETEROGENEITY IN PATCH QUALITY BUFFERS METAPOPULATIONS FROM PATHOGEN IMPACTS [3]

[3] Becker DJ, Hall RJ. 2016 Heterogeneity in patch quality buffers metapopulations from pathogen impacts. *Theoretical Ecology* 9, 197–205. Reprinted here with permission of the publisher.

ABSTRACT

Many wildlife species persist on a network of ephemerally occupied habitat patches connected by dispersal. Provisioning of food and other resources for conservation management or recreation is frequently used to improve local habitat quality and attract wildlife. Resource improvement can also facilitate local pathogen transmission, but the landscape-level consequences of provisioning for pathogen spread and habitat occupancy are poorly understood. Here we develop a simple metapopulation model to investigate how heterogeneity in patch quality resulting from resource improvement influences long-term metapopulation occupancy in the presence of a virulent pathogen. We derive expressions for equilibrium host–pathogen outcomes in terms of provisioning effects on individual patches (through decreased patch extinction rates) and at the landscape level (the fraction of high-quality, provisioned patches), and highlight two cases of practical concern. First, if occupancy in the unprovisioned metapopulation is sufficiently low, a local maximum in occupancy occurs for mixtures of high- and low-quality patches, such that further increasing the number of high-quality patches both lowers occupancy and allows pathogen invasion. Second, if the pathogen persists in the unprovisioned metapopulation, further provisioning can result in all patches becoming infected and in a global minimum in occupancy. This work highlights the need for more empirical research on landscape-level impacts of local resource provisioning on pathogen dynamics.

Keywords: conservation biology; infectious disease; habitat management; metapopulation; resource provisioning; supplemental feeding; mathematical modeling; source–sink dynamics

INTRODUCTION

Metapopulation dynamics, in which local populations connected through dispersal persist through frequent extinction and colonization events, have been demonstrated in a wide variety of taxa including insects specializing on patchily distributed host plants [139], amphibians and other aquatic species inhabiting spatially discrete wetlands [140], or species limited by availability of colonial breeding sites including bats [141]. Metapopulation dynamics can also result from habitat fragmentation and degradation [142,143]. Human activities such as agriculture and urbanization can produce a network of fragmented, low-quality habitat patches across a landscape [144]. Reductions in breeding habitat and inter-patch dispersal can result in loss of genetic diversity and elevated local extinction risk [145,146], threatening metapopulation persistence [147].

As conservation resources are often limited, cost-effective management of patchily distributed populations has benefited from insights provided by theory [148]. Consideration of classic [53] and spatially realistic [149] metapopulation models suggests persistence can be enhanced by landscape-scale efforts to increase connectivity, such as by creating corridors between isolated patches, and to prioritize conservation of large, well-connected patches [150]. At local scales, improving patch quality by enhancing food or breeding site availability or by removing predators and competitors can increase metapopulation persistence by enhancing local fecundity and survival and by disproportionally attracting immigrants, therefore reducing extinction risk [151–153]. Improving patch quality is especially appealing as a management option when the number, size, and spatial configuration of patches are fixed, as in urbanized landscapes or networks of wetlands connected by flood events.

Patch improvement through the provisioning of spatially and temporally predictable food resources is a frequently used management strategy that has enhanced breeding success and survival in imperiled and range-restricted wildlife [154,155], including a wide variety of species exhibiting metapopulation structure (examples are listed in Appendix C Table S4.1). Similarly, resource subsidies provided recreationally or unintentionally in urban and agricultural habitat, such as bird feeders and crop fields, can elevate local population densities [6,49]. Yet this resource improvement can have negative consequences, including skewed sex ratios and increased predation [156,157]. Greater resource availability can also facilitate local pathogen transmission by aggregating hosts and amplifying pathogen replication and shedding [45,137]. Resource provisioning has consequently been implicated in the emergence of pathogens such as *Trichomonas gallinae* and subsequent population declines [37].

Management or unintentional actions that improve connectivity and patch quality could likewise influence the spread of pathogens through fragmented landscapes. A body of theory based on extending the Levins model [53] suggests that increasing inter-patch colonization rates can increase risk of pathogen invasion [158–160]. A more detailed model simulating viral dynamics in flying foxes suggested that increased resource availability in urban habitat results in decreased connectivity of bat colonies, reducing outbreak frequency across the landscape but in turn increasing the size of local outbreaks due to loss of herd immunity in isolated subpopulations [62].

Although existing theory elucidates how connectivity influences pathogen spread in patchily distributed landscapes, the role of heterogeneity in habitat quality in promoting or limiting pathogen spread is poorly understood. This is particularly important for guiding conservation or recreational practices related to resource improvement and supplemental feeding

in pathogen-susceptible or imperiled metapopulations. Here we develop a simple metapopulation model to explore how patch occupancy, pathogen invasion, and infection prevalence are influenced by resource improvement at two spatial scales: local improvement to patch quality and the landscape-level deployment of patch improvement across the metapopulation. Contrary to expectation, we find a non-monotonic relationship between equilibrium occupancy and the number and quality of provisioned patches, such that habitat improvement instigated at all patches can result in lower occupancy than when a mixture of low- and high-quality patches is maintained. In addition to informing conservation and management strategies based in resource improvement, these findings suggest more generally that low-quality habitat patches can inhibit pathogen spread across landscapes if they act as sinks for infected subpopulations.

MODEL AND METHODS

Resource improvement and patch quality

To describe the dynamics of a metapopulation subject to improvement in patch quality, we extend the Levins model describing changes in the fraction of occupied patches resulting from colonization and extinction [53,147] to include two patch types: low-quality (unprovisioned) and high-quality (provisioned) habitat, where N_L and N_H denote the respective proportions (not absolute numbers) of occupied patches. Effects of resource improvement are reflected in two model parameters. The parameter f represents the fraction of patches provisioned, while the parameter q represents the improvement in habitat quality, defined as the proportionate increase in expected occupancy time in provisioned versus unprovisioned patches (i.e., $q = x_L/x_H$, where x_L and x_H are the extinction rates of low- and high-quality patches). Since these changes focus on improving local habitat quality, for simplicity we assume the colonization rate (c) from

any occupied patch to an empty patch is the same for provisioned and unprovisioned patches. The total colonization rate for low-quality patches is the product of the species-specific colonization rate (c), the fraction of empty low-quality patches (the difference between the total fraction of low-quality patches, $1 - f$, and the fraction currently occupied, N_L), and the fraction of low- and high-quality patches currently occupied, $N_H + N_L$; a similar expression is readily derived for the colonization of high-quality patches. Therefore, the metapopulation dynamics can be described by the following differential equations:

$$dN_L/dt = c(1 - f - N_L)(N_H + N_L) - x_L N_L \quad (1a)$$

$$dN_H/dt = c(f - N_H)(N_H + N_L) - (x_L/q)N_H \quad (1b)$$

Pathogen dynamics

Following [158], we model pathogen dynamics by categorizing the fraction of occupied patches as susceptible (S) or infected (I), with the subscripts L and H denoting low or high quality. For simplicity, we assume that infection status does not affect the colonization parameter c; that empty patches colonized by infected patches become infected, while colonized, disease-free patches become infected by infected patches with probability $\delta \leq 1$. We assume that once infected, patches remain infected until local extinction occurs. Infection increases the extinction rate of low- and high-quality patches by ν_L and ν_H respectively; we refer to these as the disease-induced extinction rates. We assume patch improvement reduces disease-induced extinction in the same way as it reduces disease-free extinction ($\nu_H = \nu_L/q$); an alternative scenario where disease-induced extinction is independent of improvement ($\nu_H = \nu_L$) is considered in Appendix C. The dynamics are now described by:

$$dS_L/dt = c(1 - f - S_L - I_L)(S_H + S_L) - x_L S_L - \delta c S_L(I_H + I_L) \quad (2a)$$

$$dI_L/dt = c(1 - f - S_L - I_L)(I_H + I_L) + \delta c S_L(I_H + I_L) - (x_L + \nu_L) I_L \quad (2b)$$

$$dS_H/dt = c(f - S_H - I_H)(S_H + S_L) - (x_L/q) S_H - \delta c S_H(I_H + I_L) \quad (2c)$$

$$dI_H/dt = c(f - S_H - I_H)(I_H + I_L) + \delta c S_H(I_H + I_L) - \big((x_L/q) + \nu_H\big) I_H \quad (2d)$$

A visualization of the model formulation can be seen in Figure 4.1.

Modeling approach

To assess the effects of local patch improvement and its landscape-level deployment on metapopulation and disease dynamics, we co-vary the increase in patch quality resulting from resource provisioning (q) and the fraction of patches provisioned (f) and calculate the equilibrium metapopulation occupancy ($N^* = S_L + I_L + S_H + I_H$) and infection prevalence (fraction of occupied patches infected, $P^* = (I_L + I_H)/N^*$). Where possible, we derive analytical expressions for metapopulation persistence and pathogen invasion (Appendix C); otherwise, the model is numerically solved using the *deSolve* package in R [119,161]. For initial conditions, we set the initial occupancy of low- and high-quality patches to their disease free equilibrium for the particular provisioning scenario (specified by f and q*;* analytical expressions are given in Appendix C), and introduced a small fraction of infected patches; note that the equilibrium outcomes were not influenced by the specific choice of initial conditions. We illustrate our results for for two scenarios of practical importance describing the nature of resource improvement and the state of the metapopopulation prior to improvement: (A) an imperiled metapopulation declining prior to management ($c < x_L$) that is rescued via resource

supplementation and (B) a persistent metapopulation ($c > x_L$) subject to improved patch quality via recreational feeding or unintentional subsidies in human-modified habitats. In both scenarios, the metapopulation is challenged by a highly transmissible, virulent pathogen; parameter definitions and default values are given in Table 4.1.

RESULTS

Dynamics

Numerical solution of the ordinary differential equations over a wide range of parameter values and initial conditions confirmed that dynamics converge on a point equilibrium after a relatively short transient period (typically 30–60 "generations" as measured by the expected persistence time of low-quality patches, $1/x_L$). If occupancy in the unprovisioned metapopulation is too low to allow pathogen invasion, provisioning has the expected effect of increasing occupancy. However, once occupancy is high enough to permit pathogen invasion, equilibrium occupancy declines as the fraction of infected patches increases (example dynamics are shown in Figure 4.2).

Equilibrium analysis

Across all possible parameter ranges, the model has four equilibrium states: metapopulation extinction, disease-free persistence of the host, pathogen invasion and persistence at an endemic equilibrium (i.e., a constant fraction of patches are infected), and pandemic equilibrium (where all occupied patches are infected). Stability of these successive states is increasingly likely at higher degrees of local patch improvement (i.e., increasing values of q) and landscape

deployment of provisioning (i.e., increasing values of f). Analytical expressions for these equilibria as functions of the model parameters are derived in Appendix C.

The threshold conditions for disease-free, endemic, and pandemic stability are described by approximately inverse relationships between the local and landscape provisioning parameters q and f (Table 4.2 and illustrated for Scenarios A and B in the left column of Figure 4.3). Thus, we use f as our bifurcation parameter to understand how increasing provisioning influences host occupancy and pathogen prevalence, but note that qualitatively similar results would be obtained by fixing f and increasing q. We note the following general patterns between equilibrium occupancy and the fraction of patches provisioned.

- When $f_{df} > 0$, provisioning is necessary for metapopulation persistence.
- When $f_{df} < f < f_e$, equilibrium occupancy is an increasing function of f. Therefore, provisioning is always beneficial if the pathogen is unable to invade when all patches are provisioned (i.e., when $f_e > 1$).
- When $f_e < f < f_p$, occupancy declines with increasing f. Therefore, metapopulation occupancy is maximized for mixtures of high- and low-quality patches whenever the pathogen can invade the fully provisioned metapopulation, but does not reach pandemic equilibrium (i.e., when $f_e < 1$ and $f_p > 1$).
- When $f > f_p$ (i.e., all occupied patches are infected), occupancy again increases with f due to increased persistence time of infected provisioned patches relative to unprovisioned patches. If the pathogen is endemic in the unprovisioned metapopulation ($f_e < 0$), equilibrium occupancy is minimized at $f = f_p$. If additionally the pathogen is endemic but not pandemic in a fully provisioned metapopulation ($f_p > 1$), provisioning is always detrimental to metapopulation occupancy.

Provisioning impacts on host occupancy and pathogen prevalence

The practical significance of these results is best illustrated with our two scenarios of (A) declining ($c < x_L$) versus (B) persistent ($c > x_L$) metapopulations prior to supplementation (i.e., when $f = 0$). For each scenario, we plot equilibrium occupancy and pathogen prevalence as a function of the fraction of patches provisioned (f) for two different levels of provisioning: "moderate" provisioning ($q = 2$, meaning that provisioned patches remain occupied for twice as long as unprovisioned patches in disease-free populations; Figure 4.3 center panel) and "high" provisioning ($q = 4$, provisioned patches remain occupied four times as long as unprovisioned patches; Figure 4.3 right panel). Although the specific values of q chosen are arbitrary, they illustrate qualitatively different relationships between occupancy and provisioning (unimodal versus non-monotonic increasing).

For a declining, managed metapopulation (scenario A) and subject to "moderate" provisioning, pathogen invasion is only possible when most patches are managed, and pandemic equilibrium is impossible even when all patches are provisioned (Figure 4.3A, center). In this case, maintaining a mixture of high- and low-quality patches can result in higher occupancy than if all patches receive provisioning. Under "high" provisioning ($q = 4$, Figure 4.3A, right), the local maximum in occupancy is attained when a smaller fraction of patches are treated. Here maximum occupancy is achieved when all patches are provisioned, but all patches are infected. Therefore, maintaining some unmanaged, low-quality patches may be optimal both for increasing occupancy and minimizing pathogen spread.

For a persistent but subsidized metapopulation (scenario B) subject to "moderate" provisioning (Figure 4.3B, center), provisioning always increases occupancy relative to the unprovisioned metapopulation. Yet the magnitude of this improvement declines over a large

range of f, and pathogen invasion can occur when a relatively small fraction of patches is provisioned. However, under "high" provisioning (Figure 4.3B, right), occupancy can decline below that in the unprovisioned metapopulation, and all patches become infected. Therefore, subsidizing persistent metapopulations tends to favor pathogen spread, and in some cases is also detrimental to host occupancy.

Sensitivity of results to pathogen traits

To better understand whether the non-monotonic relationships observed between provisioning and occupancy in our parameterization hold more generally, we performed extensive sensitivity analyses on pathogen parameters (Appendix C). Our model assumes that improvements to patch quality (increasing q) reduce both natural (x_H) and disease-induced (ν_H) extinction rates. Alternatively, if we assume that disease-induced extinction is unaffected by patch improvement (i.e., $\nu_H = \nu_L$), we obtain qualitatively similar results (Appendix C); however, for the same parameterization used in the main text, pathogen invasion occurs in a much smaller region of provisioning parameter space (when most patches receive high levels of provisioning). This is because the extinction rate of provisioned patches can never decrease below ν_L, and so the positive effects of provisioning on infected patch persistence saturate.

The two cases of most practical interest in our provisioning scenarios are 1) when maintaining some low-quality patches results in higher occupancy than a fully provisioned metapopulation (i.e., when $N^*(f_e) > N^*(1)$ for $0 < f_e < 1$) and 2) when provisioning causes reductions in occupancy relative to the unprovisioned population (i.e., when $N^*(f_p) < N^*(0)$ for $f_p > 0$). We varied pathogen transmissibility (probability of a colonized patch becoming infected, δ) and virulence (disease-induced extinction rate, ν_L) to see whether cases 1 and 2

occurred more generally (Appendix C; Figures S4.3 and S4.4). Due to complex interactions between provisioning and pathogen parameters in the expressions for equilibrium occupancy, no clear mechanistic patterns emerged; however, we observed global maxima and minima in occupancy in provisioned, heterogeneous populations over a range of pathogen traits, suggesting that too much provisioning can be detrimental to metapopulations exposed to diverse pathogens.

DISCUSSION

Variation in habitat quality has long been recognized as an important regulator of population dynamics [151,162]. In source–sink dynamics, sink habitats (in which local population growth rates are negative) can play an important role in metapopulation persistence if they increase connectivity between source patches or encourage local adaptation [163]. Here we outline conditions under which metapopulations containing some lower quality patches can attain higher occupancy than those with uniformly high-quality patches in the presence of virulent pathogens. A necessary, but not sufficient, condition for heterogeneity in patch quality to be beneficial is that low-quality patches occupied by infected host subpopulations have high extinction rates and thus act as sinks for the pathogen.

Our modeling approach has analogs in the theory of multi-host pathogen interactions, in which high- and low-quality patches are equivalent to competent reservoir and less-competent spillover host species [164,165]. Comparable to the dilution effect hypothesis describing how host community composition shapes pathogen dynamics, our model suggests heterogeneity in patch quality, particularly in the frequency of low-quality patches with reduced occupancy times of hosts and pathogens, can mediate landscape-level infection prevalence. A key difference that results from treating patches rather than individual hosts as the base unit of infection is that all

occupied patches can become infected if the proportion of high-quality patches is sufficiently large (whereas typically only a fraction of individual hosts in a population are infected). Existence of this pandemic equilibrium has been demonstrated as a consequence of high connectivity in homogeneous metapopulations [158,160], but this state has not been investigated in the context of habitat heterogeneity.

Resource improvement for conservation, recreation, or as a consequence of urbanization can increase local population growth and thus create source patches. However, provisioning may allow local epidemics of virulent pathogens to persist for longer (for example, due to larger host population sizes; [52], promoting landscape-scale pathogen spread and reduction in overall occupancy. Thus if the fraction of high-quality patches is too high, provisioned patches may in fact function as ecological traps [166].

Our analyses have practical importance for the conservation of imperiled metapopulations. If conservation management primarily relies on local habitat improvement, maintaining mixtures of unmanaged and managed patches can achieve two desirable goals: maximizing patch occupancy while minimizing risk of pathogen invasion. This could be particularly important for small subpopulations with low genetic variation, which are frequently highly susceptible to mass mortality following pathogen outbreaks [167]. In contrast, for non-threatened metapopulations subject to resource subsidies in urban or agricultural landscapes, subsidized habitat patches can lead to global declines in occupancy and to pandemic spread of infection, even when these patches are scarce relative to unsubsidized patches. Although pathogens are unlikely to cause catastrophic population declines in this case, high infection prevalence across the landscape can have negative consequences, including increased risk of spillover to wild, domestic, or human hosts or increased morbidity and mortality. For example,

widespread bird feeding in the continental United States was likely a key driver in the emergence and spatial spread of mycoplasmal conjunctivitis in house finches [38]. Severe infections can cause disfiguring lesions and individual mortality, which threaten population declines and are likewise distressing to citizens who feed birds [168].

Our model makes several simplifying assumptions that may not hold in real systems. We focused on effects of resource improvement on patch extinction rates, and following previous models [158], we assumed that the colonization rate of all empty patches was equal irrespective of patch quality and the infection status of the donor patch. If more infected individuals (or propagules) disperse from high-quality patches, and lower quality patches produce fewer infected dispersers or propagules, we predict this would exacerbate the advantageous effects of patch heterogeneity on equilibrium occupancy by reducing disease spread relative to metapopulations with uniformly high patch quality. Our assumption of uniform colonization rates is most appropriate for highly mobile species such as birds and species dispersed passively by air or water currents or human activity (e.g., seeds or pelagic larvae). However, large inter-patch distances and geographical barriers will cause heterogeneity in movement patterns for less-mobile species. Although beyond the scope of this study, the effects of landscape heterogeneity and dispersal limitation could be investigated using spatially realistic metapopulation models [169]. For example, our modeling approach could be extended to include patch isolation and to modify the attractiveness of each patch to depend on quality (resource availability) as well as patch size.

Our model only investigated susceptible–infected dynamics (i.e., infected patches remain infected until extinction), while infected subpopulations may be able clear infection prior to extinction (susceptible–infected–susceptible dynamics). Allowing recovery from infection makes

pathogen invasion more difficult in homogeneous metapopulations by reducing the infectious period of patches [160]. If lower quality patches support fewer hosts, local pathogen extinction may occur well before host extinction, which would again support our hypothesis that low-quality patches can minimize pathogen impacts on occupancy. The role of patch heterogeneity in determining landscape infection prevalence may be reduced if the pathogen has an alternative, more abundant host [159] or a persistent environmental reservoir [170]. Thus our results apply primarily to specialist pathogens transmitted by direct contacts between hosts, by biting arthropod vectors, or by short-lived environmental stages (e.g., those transmitted through fecal–oral pathways). Finally, since our model tracks only patch occupancy and infection status, it cannot directly account for resource-mediated effects on individual hosts (e.g., body condition, immunity, and pathogen virulence), host population size, and subsequent infection processes. Mechanistically linking within-patch processes to between-patch spread in provisioned landscapes is worthy of further theoretical exploration, but will necessitate explicit modeling of within-patch host–pathogen dynamics [52].

Resource improvement is increasingly recognized to facilitate pathogen transmission [33,62], and changes to the spatial distribution and quality of resources influence population dynamics and behavior in ways that can exacerbate disease spread [47,171]. We have developed a simple model to demonstrate how unmanaged resource subsidies for wildlife as well as conservation based in resource improvement can disproportionally contribute to the spatial dynamics of infectious disease and potentially lead to declines in occupancy of habitat patches across the landscape. To develop predictive models for the landscape-level consequences of local resource provisioning on host–pathogen dynamics, further empirical work at the landscape scale is urgently required. Estimation of local parameters relating to the intensity of provisioning (e.g.,

the amount and frequency of food supplements) and patch infection status (e.g., the presence, prevalence, or peak abundance of infectious hosts) will be especially important to quantify relative differences in persistence time and colonization rates between landscapes with varying configurations of low- and high-quality patches [172,173]. In particular, studies quantifying how the spatial deployment of resource subsidies influences movement between patches and pathogen spread will be vital for parameterizing predictive models and in turn guiding spatially coordinated conservation and management practices related to resource improvement and supplemental feeding.

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Table 4.1. Parameterization of the metapopulation model, where grey shading indicates parameters related to resource improvement.

Parameter	Definition	Value	Interpretation
x_L	Extinction rate of low-quality patches	1	Time is measured in units corresponding to the expected occupancy time of low-quality
c	Colonization rate	A: 0.9 B: 1.4	When $c < x_L$, the metapopulation declines in absence of provisioning (scenario A); when $c > x_L$
q	Quality difference	1–5	High-quality provisioned patches persist for 1 to 5 times as long as low-quality patches
f	Proportion of high-quality habitat	0–1	0 to 100 percent of patches are provisioned by resource improvement
δ	Pathogen transmissibility	0.75	High probability of infection in a patch upon arrival of diseased immigrant
ν_L	Disease-induced extinction rate for	0.5	Infected low-quality patches go extinct 1.5 times faster than uninfected counterparts
ν_H	Disease-induced extinction rate for	ν_L/q	Disease-induced extinction rate is inversely proportional to patch quality improvement

Table 4.2. Model equilibria and conditions for their stability, expressed using the fraction of high-quality patches (f) as a bifurcation parameter.

Equilibrium	**Stability criteria**
Metapopulation extinction $(S_L^*, I_L^*, S_H^*, I_H^*) = (0,0,0,0)$	$f < f_{df} = \frac{1}{q-1}\left(\frac{x_L - c}{c}\right)$
Disease-free (df) $(S_L(df), 0, S_H(df), 0)$	$f_{df} < f < f_e = \frac{1}{q-1}\left(\frac{x_L - c}{c} + \frac{\nu_L}{\delta c}\right)$
Endemic (e) $(S_L^*(e), I_L^*(e), S_H^*(e), I_H^*(e))$	$f_e < f < f_p = \frac{Z^2 - Y^2}{4(1-Y)}$, where $Y = 1 - (\theta + \phi)\left(1 - \frac{1}{q}\right)$, $Z = \left(1 + \frac{1}{q}\right)\left(\frac{\delta\theta + (1+\delta)\phi - \delta - \sqrt{W}}{1+\delta}\right)$ and $W = \left(1 - \theta\left(1 + \frac{1}{q}\right)\right)^2 + \frac{4\theta\phi(1+\delta)}{q\delta}$
Pandemic (p) $(0, I_L(p), 0, I_H(p))$	$f > f_p$

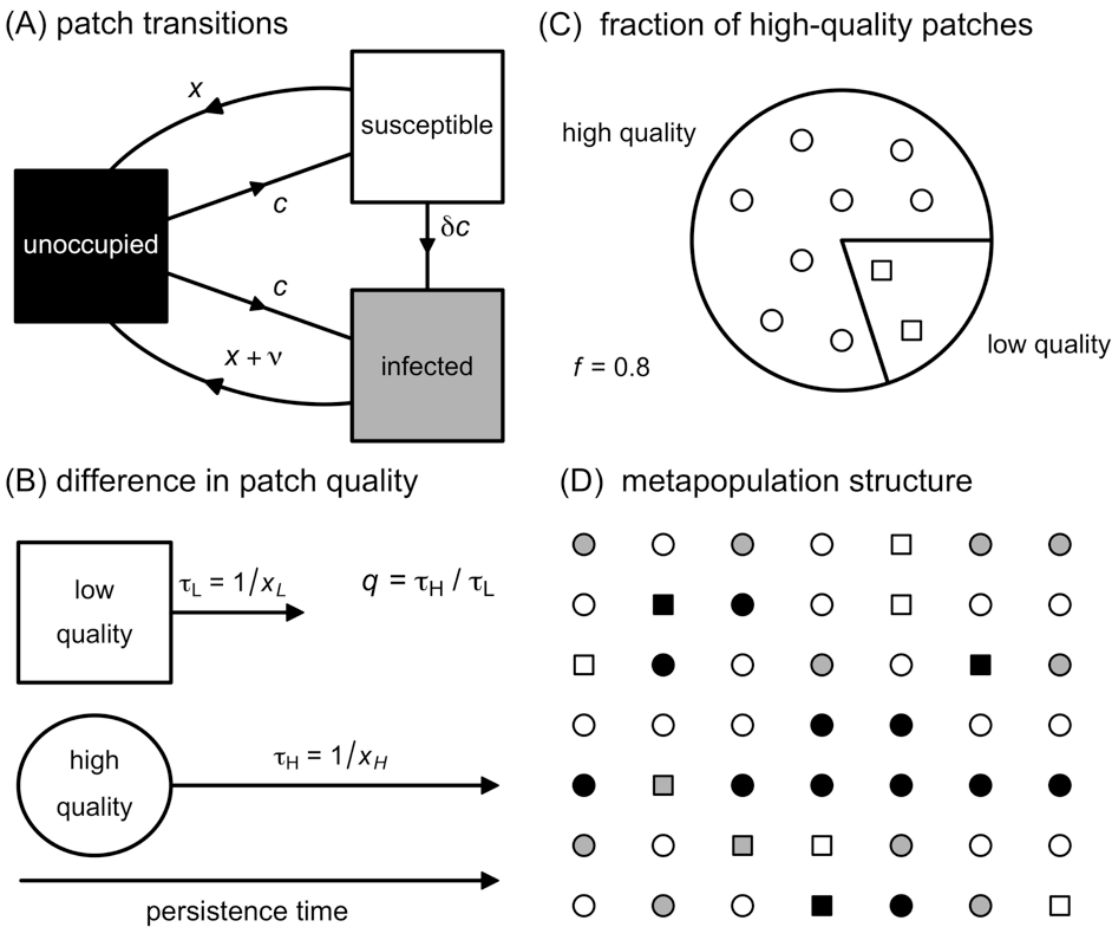

Figure 4.1. Conceptual framework for the quality-dependent metapopulation model. (A) Transitions between patch types follow a Hess-type epidemic model of unoccupied, susceptible, and infected patches, where model parameters are defined as in Table 4.1. (B) We consider two patch types, where unprovisioned patches are of low quality (square) and resource-improved patches are of high quality (circle) and are occupied for longer. Arrow length indicates the expected persistence time for each patch type, and the quality difference parameter (q) is defined as the ratio of persistence times in patches of high (circle) versus low (square) quality. (C) Heterogeneity in patch quality in the metapopulation is defined by the proportion of patches at which resource improvement occurs (f). (D) A snapshot of the metapopulation structure in which patch status, type, and the proportion improved are as defined in parts A–C.

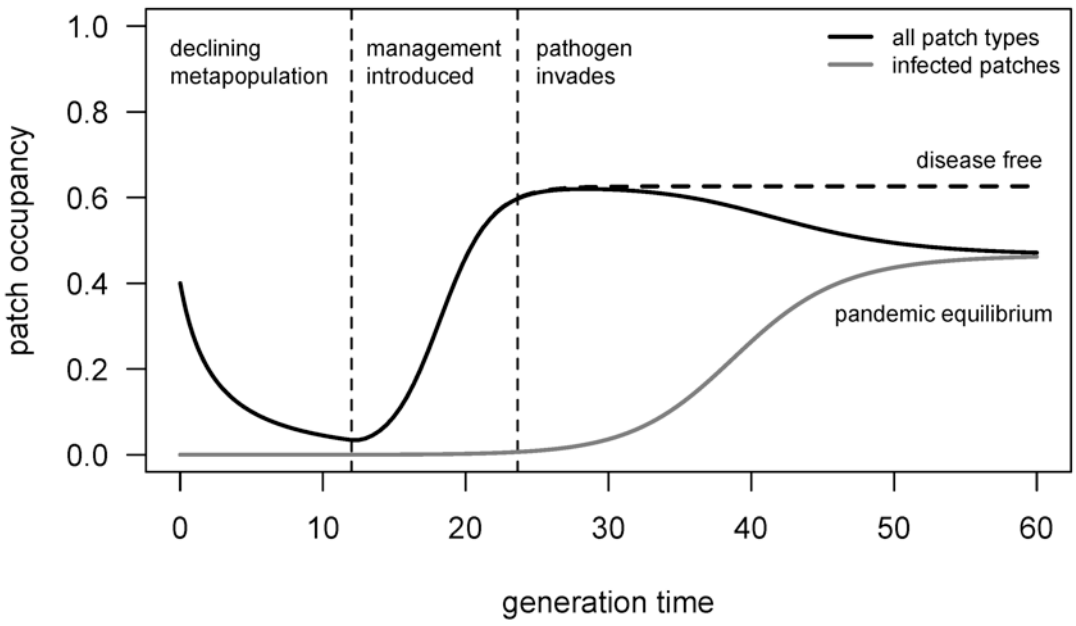

Figure 4.2. Example dynamics showing how total patch occupancy (bold line) and the fraction of infected patches (gray line) change through time, for the case of a declining metapopulation in which management is introduced at time $t = 12$. In the absence of the pathogen, equilibrium occupancy of 0.63 would be attained (dashed line). However, at time $t = 23$, occupancy becomes high enough to permit pathogen invasion and eventually become pandemic (i.e., all occupied patches are infected), resulting in a reduced occupancy of 0.47. Parameters were selected so that resource improvement results in a four-fold increase in the persistence time of provisioned over unprovisioned patches ($q = 4$) and that 80% of patches in the metapopulation are improved ($f = 0.8$); other parameters describing host metapopulation and pathogen traits are given in Table 4.1.

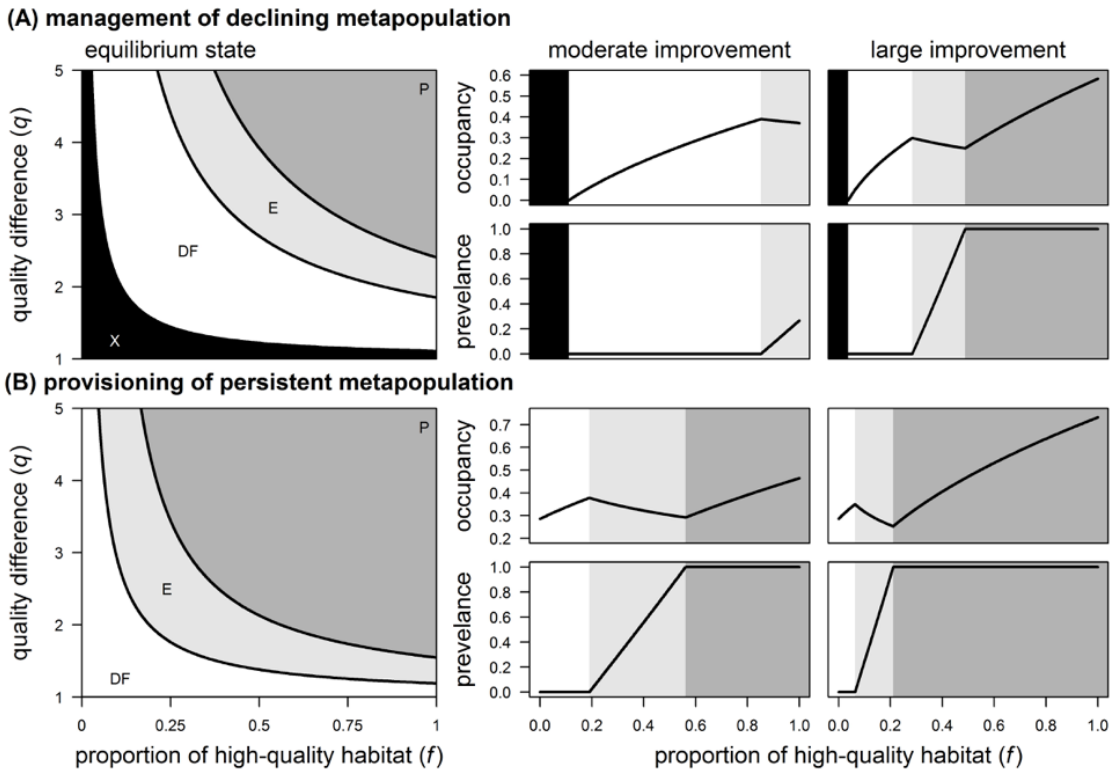

Figure 4.3. Left: effect of local patch quality improvement (q) and the fraction of high-quality improved patches (f) on metapopulation occupancy and pathogen colonization. Shaded regions depict where the metapopulation is extinct (X, black), persists in a disease-free state (DF, white), and where the pathogen invades and becomes endemic (E, light grey) or pandemic (P, dark grey). Right: equilibrium occupancy and infection prevalence as a function of the fraction of high-quality patches (f) for moderate ($q = 2$) and large ($q = 4$) differences in patch quality. Row A illustrates results for the case of a metapopulation in decline prior to management through supplemental feeding ($c = 0.9$); row B illustrates results for the case of a persistent metapopulation where hosts are provisioned through resource subsidies ($c = 1.4$).

CHAPTER 5

HOST MOVEMENT RESPONSES TO RESOURCE SUPPLEMENTATION DETERMINE PATHOGEN SPREAD IN WILDLIFE METAPOPULATIONS [4]

[4] Becker DJ, Snedden C, Altizer S, Hall RJ. To be submitted to *American Naturalist*.

ABSTRACT

As natural landscapes are increasingly modified by anthropogenic activity, many wildlife are capitalizing on supplemental food resources, which can have profound consequences for animal distributions and movement patterns. The high densities of individuals within these provisioned habitats can promote the local transmission of pathogens, including those of concern for human, wildlife, and domestic animal health. However, given that many wildlife show spatial structure and provisioned habitats are distributed across landscapes, understanding how host movement processes are affected by the availability of these resource-dense habitats is important to predict consequences for the spread of infections in wildlife metapopulations. We here develop simple, generalizable theory for pathogen spread in a metapopulation of natural and provisioned patches and determine the consequences of increasing the abundance of these subsidized habitats for host occupancy and infection prevalence. We demonstrate infection outcomes at the landscape scale depend critically on how local resource abundance alters the preference and net production of dispersers and on whether infection has costs for inter-patch movement. When provisioning has strong effects on patch attraction and disperser production and infection has minimal costs for movement, these high contributions to colonization facilitate pathogen invasion and infection can spread completely through highly provisioned landscapes. Yet if animal movement responds to provisioning with site fidelity or more sedentary behavior, increasing the fraction of subsidized habitats can reduce infection prevalence and permit pathogen extinction while also maximizing host occupancy. This work highlights the importance of considering how host movement can differentially respond to resource availability in the face of variable pathogens to understand and predict how changing resource distributions can promote or limit the spatial spread of infection.

Keywords: colonization; dispersal; infectious disease; resource provisioning; urbanization

INTRODUCTION

The abundance and distribution of food is a major determinant of animal fitness and movement [174]. The dietary landscapes of many wildlife are being modified by human activities that provide food subsidies unintentionally (e.g., rubbish dumps, agricultural fields) or deliberately for management or recreation (e.g., backyard bird feeders) [4,5]. While these anthropogenic food subsidies, henceforth referred to as resource provisioning, can provide accessible and predictable sources of nutrition that benefit species facing seasonal food shortages and habitat loss [49,175], supplemental feeding can negatively affect wildlife by enhancing pathogen transmission [31,33]. For example, bird feeders have been associated with outbreaks of *Mycoplasma gallisepticum* in house finches (*Haemorhous mexicanus*) [38,39], the spatial expansion of which has driven range-wide host population declines [168]. Provisioning can also facilitate pathogen spillover from wildlife reservoirs into human populations. For example, red foxes (*Vulpes vulpes*) and raccoon dogs (*Nyctereutes procyonoides*) in Europe have experienced dramatic population growth owing to anthropogenic foods in urban habitats, which could increase cross-species transmission of zoonoses such as rabies virus and sarcoptic mange [21,176]. Mitigating these risks to wildlife, domestic animals, and humans requires synthetic approaches that integrate complex effects of provisioning on hosts from local transmission to landscape-level movement between habitat [177].

Supplemental resources are often deployed across a network of habitat patches within a landscape [55,178]. For example, bird feeders are distributed in a heterogeneous fashion across urbanized landscapes [179,180]. Similarly, anthropogenic food in the form of livestock functions as a patchily distributed but preferred prey source for vampire bats (*Desmodus rotundus*) [20,181]. Accordingly, the landscape-level availability of provisioned habitat can influence host

movement patterns and the colonization of currently unoccupied habitats (Table 5.1). Provisioned patches can foremost be more attractive to potential colonists; in the case of vampire bats, livestock-rich habitats likely determine patterns of bat roost selection [182,183]. High densities of flowering resources also increase immigration of butterflies (*Parnassius smintheus* and *Boloria eunomia*) into provisioned habitats [184,185]. Provisioning could also increase the local production of emmigrants within a patch by increasing fecundity and offspring dispersal success. For example, food supplementation experiments have increased clutch size, offspring survival, and fledging success in birds such as Florida scrub jays (*Aphelocoma coerulescens*) and black-billed magpies (*Pica pica*) [186,187], an effect mirrored in many wild vertebrates [84]. However, supplemental feeding can also promote site fidelity and reduce the number of dispersers that emigrate from a habitat patch [15,171,188]. For example, raccoons (*Procyon lotor*) in urban habitats with abundant anthropogenic food display smaller home ranges and more aggregated spatial distributions than rural counterparts [189]. Similarly, flying foxes (*Pteropus* spp.) in Australia have transitioned from nomadic foraging on ephemeral fruit and nectar resources to forming sedentary camps in urban gardens [190,191]. In the extreme cases that provisioning drastically reduces the number of dispersers leaving a patch or produces fewer dispersers if anthropogenic food is of poor quality and reduces fitness [96,192], provisioned habitats could also act as ecological traps for wildlife species that rely on colonization–extinction dynamics for their long-term persistence [193,194].

Importantly, these changes in host movement patterns owing to provisioning could influence the spatial spread of pathogens but in potentially in opposing directions. The greater production of emmigrants from provisioned habitats could seed infection into occupied and unoccupied patches and promote pathogen spread [158,195,196]. Provisioned habitats that attract

more infectious dispersers could also promote the between-patch transmission of pathogens. Attraction towards provisioned habitats likely facilitated shifts in flying fox foraging toward mango plantations in Malaysia and the spillover of Nipah virus from bats into pigs and humans [34,35]. However, promotion of site fidelity in provisioned patches could limit or reduce landscape-level infection prevalence by reducing the movement of infectious dispersers between habitats. For example, a mechanistic model of Hendra virus dynamics in urbanized flying foxes demonstrated that reducing colony connectivity from more sedentary behavior decreased epidemic frequency and the net number of infected flying foxes over time [62]. Infection could further complicate dynamics if infected hosts are less likely to survive during inter-patch dispersal (Table 5.1). For example, monarch butterflies (*Danaus plexippus*) infected with a protozoan parasite fly shorter distances and are less likely to make successful migrations [197]. Dynamics could accordingly differ for systems in which infection has weak effect on dispersal; as one example, the virulent Nipah virus shows no disease in flying foxes [198] and likely does not affect bat movement [199]. As provisioning and infection can play important but contrasting roles in shaping host dispersal, predicting relationships between the distribution of provisioned habitat and the spatial dynamics of hosts and pathogens thus requires integrating these sources of heterogeneity in colonization.

Mathematical models are a powerful tool for exploring mechanisms driving infectious disease dynamics [200,201] and have provided important insights into how provisioning influences pathogen invasion and persistence within closed populations [52,61,202]. At the landscape level, metapopulation models have been used to explore pathogen dynamics among homogeneous patches [158–160,170] and patches that differ in their extinction rates [55] and in their attractiveness and production of emmigrants [203]; however, the joint effects of

provisioning and infection status on colonization processes has not been explored. We here provide a novel contribution to ecological and epidemiological theory by developing a simple metapopulation model that accounts for these colonization heterogeneities in a landscape composed of unprovisioned and provisioned patches. We derive key threshold parameters for understanding how host occupancy and infection prevalence depend on the extent of provisioning, how habitat quality affects patch attractiveness and disperser production, and the cost of infection to dispersers. We lastly define regions of parameter space corresponding to ecological conditions under which provisioning is most beneficial or detrimental to host occupancy and pathogen spread. Our general framework remains flexible enough to accommodate assumptions and biological realism across a range of host–pathogen systems where resource availability varies across a landscape (Table 5.1).

MATERIALS AND METHODS

Provisioning and metapopulation dynamics

To describe occupancy dynamics of a provisioned metapopulation, we extend the classic Levins model in which the proportion of habitat patches occupied by a focal species is governed by a colonization rate c and extinction rate x [53]. Despite its simplicity, the Levins model captures metapopulation dynamics for a range of wildlife species and can provide accurate predictions of the fraction of patches occupied in a landscape [54]. We classify patches as unprovisioned or provisioned, where N_U and N_P denote the respective proportions of occupied patches. Following our prior extension of the Levins metapopulation model [55], the spatial extent of provisioning is described by the fraction of provisioned patches across the landscape, f. We assume

provisioning modifies the extinction rate of patches by a factor α, so that provisioned patches persist for $1/\alpha$ times the typical occupancy time of an occupied unprovisioned patch [55].

We next consider two mechanisms by which provisioning introduces heterogeneity into the colonization rate. First, provisioning can alter the rate at which dispersers are produced relative to an unprovisioned patch by a factor θ. When θ is greater than one, provisioned patches produce relatively more dispersers per unit time than unprovisioned patches, while θ less than one reflects cases where individuals remain more site faithful to provisioned habitat or where the production of dispersers is reduced due to lower quality of provisioned food. Provisioned patches can also be relatively more attractive to potential colonists than unprovisioned patches, which we model by a factor ϕ; when ϕ is greater than 1, a provisioned patch is more likely to be chosen than an unprovisioned patch. We assume that dispersers distribute among provisioned and unprovisioned patches dependent on the relative frequency of each patch type ($1-f$ or f, respectively) and the relative attractiveness of provisioned patches (1 or ϕ). The colonization rate for each patch type is thus the product of the relative number of dispersers produced per time by the donor patch, the probability that a disperser chooses the recipient patch according to its relative abundance and attractiveness, and the probability the recipient patch is unoccupied (Appendix D). Metapopulation dynamics are described by the following differential equations:

$$\frac{dN_U}{dt} = \frac{c}{1+(\phi-1)f}(1-f-N_U)(N_U+\theta N_P) - xN_U \quad (1a)$$

$$\frac{dN_P}{dt} = \frac{\phi c}{1+(\phi-1)f}(f-N_P)(N_U+\theta N_P) - \alpha xN_U \quad (1b)$$

Incorporating pathogen dynamics

We introduce pathogen dynamics following Hess [158], where occupied patches are categorized according to whether the pathogen is absent (susceptible, S) or present (infected, I); subscripts U and P again denote unprovisioned and provisioned patches. We assume empty patches become infected if colonized from an infected patch, while infected dispersers seed infection in occupied patches with probability δ [158]. Patches remain infected until local extinction, and infection increases the extinction rate by an additive factor v for unprovisioned patches and αv for provisioned patches [55]. To account for the effects of infection on colonization, we assume infection reduces the number of dispersers produced by a factor $\psi \leq 1$ relative to uninfected patches. For simplicity, we consider effects of provisioning and infection to be independent, so that their combined effects on colonization and extinction rates are multiplicative. The full system of differential equations for infection dynamics in the metapopulation are as follows:

$$\frac{dS_U}{dt} = \frac{c}{1+(\phi-1)f}\left((1-f-S_U-I_U)(S_U+\theta S_P) - \delta\psi S_U(I_U+\theta I_P)\right) - xS_U \quad (2a)$$

$$\frac{dI_U}{dt} = \frac{\psi c}{1+(\phi-1)f}(1-f-S_U-I_U+\delta S_U)(I_U+\theta I_P) - (x+v)I_U \quad (2b)$$

$$\frac{dS_P}{dt} = \frac{\phi c}{1+(\phi-1)f}\left((f-S_P-I_P)(S_U+\theta S_P) - \delta\psi S_P(I_U+\theta I_P)\right) - \alpha x S_P \quad (2c)$$

$$\frac{dI_P}{dt} = \frac{\psi\phi c}{1+(\phi-1)f}(f-S_P-I_P+\delta S_P)(I_U+\theta I_P) - \alpha(x+v)I_P \quad (2d)$$

Modeling approach

To assess how the spatial extent of provisioning (f) and colonization heterogeneities induced by resource supplementation (θ, ϕ) and infection (ψ) jointly affect metapopulation dynamics, we first derived threshold conditions for metapopulation persistence, for pathogen invasion, and for all occupied patches to become infected (Appendix D). We next determined how these colonization heterogeneities affect equilibrium host occupancy (i.e., the fraction of all patches occupied; $N^* = S_U^* + I_U^* + S_P^* + I_P^*$) and infection prevalence (i.e., the fraction of occupied patches in which infection is present; $P^* = (I_U^* + I_P^*)/N^*$) by numerically solving the model using the *deSolve* package in R [119,161]. We set initial conditions so that 5% of each patch type were occupied and uninfected and 1% were occupied and infected; however, model equilibria were not influenced by initial conditions given some patches were first occupied by uninfected and infected hosts. We ran all simulations for 1000 generations, although these models generally converge on point equilibria after a relatively shorter period (e.g., under 100 generations; [55]).

For all model simulations, we varied the fraction of provisioned patches and calculated equilibrium occupancy (N^*) and infection prevalence (P^*). We varied the relative productivity of provisioned patches (θ, defined as the ratio of dispersers produced per unit time of provisioned vs unprovisioned patches) to account for two qualitatively different scenarios: the case where provisioning increases dispersal success ($\theta > 1$) and the case where provisioned patches promote site fidelity ($\theta < 1$). Within these simulations, we also considered the scenarios where dispersers show no preference for colonizing provisioned or unprovisioned patches ($\phi = 1$) or are more attracted ($\phi > 1$) to provisioned patches and where infection has no effect on ($\psi = 1$) or reduces ($\psi < 1$) dispersal success. We held the probability of colonizers infecting an occupied patch (δ), the effects of infection (v) and provisioning (α) on patch extinction rates, and the colonization rate in an unprovisioned metapopulation (c) constant, assuming that the pathogen is

highly transmissible ($\delta = 1$), infection increases patch extinction rates ($v > 0$), provisioning increases patch persistence time ($\alpha > 0$), and the metapopulation can persist in the absence of provisioning ($c > x$); detailed sensitivity analyses of these parameters have been performed previously [55]. We consider provisioning to benefit host metapopulations in regions of parameter space where increasing f increases equilibrium occupancy N^* ($\partial N^*/\partial f > 0$) while reducing equilibrium prevalence P^* or preventing pathogen invasion ($\partial P^*/\partial f \leq 0$). The model parameterization is described in Table 5.2 and visualized in Figure 5.1.

RESULTS

Stability analysis

By evaluating the Jacobian matrix in the absence of infection (equation 1), we derived an analytic expression for the lifetime colonization success of the metapopulation (ρ), defined as the proportion of patches in an empty metapopulation that would be colonized by each patch type over its occupancy time, weighted by the relative frequency of that patch type in the landscape.

$$\rho = \frac{c}{x}\left(1 + \frac{\phi\left(\frac{\theta}{\alpha} - 1\right)}{\frac{1}{f} + \phi - 1}\right) \quad (3)$$

The parameter ρ is a monotonic function of the fraction of provisioned patches (f), and metapopulation persistence requires that $\rho \geq 1$. When provisioned patches produce more dispersers over their lifetime than unprovisioned patches (i.e., $\theta/\alpha \geq 1$), increasing f, improves the likelihood of metapopulation persistence. However, if provisioning promotes site fidelity or reduces the total number of dispersers produced (i.e., $\theta/\alpha < 1$), ρ is a decreasing function of f and occupancy declines under increase provisioning. The threshold condition for disease-free

stability is described by an approximately positive relationship between f and θ (Figure 5.2); an expression for the fraction of provisioned patches necessary to permit persistence (f_{df}) is provided in Appendix D. Given that the metapopulation persists in the absence of provisioning (i.e., $c \geq x$), metapopulation extinction can only occur with provisioning when such patches produce fewer dispersers over their lifetime than unprovisioned patches ($\theta/\alpha < 1$). In relation to colonization parameters, provisioning is more likely to result in metapopulation extinction when disperser production from provisioned patches is low ($\theta/\alpha < 1$) and particularly when provisioned patches are much more attractive than unprovisioned patches ($\phi = 10$; Figure 5.2).

Use of the next-generation matrix method to derive R_0 revealed that lifetime colonization success (ρ) is also key for anticipating pathogen invasion into the provisioned metapopulation:

$$R_0 = \frac{\psi x}{x+v}(1 + (\rho - 1)\delta) \quad (4)$$

R_0 is a linear function of ρ, and pathogen invasion ($R_0 \geq 1$) is more likely in metapopulations with a larger lifetime colonization success. Given that the unprovisioned metapopulation can persist ($c \geq x$) and that infection imposes no cost for dispersal ($\psi = 1$), pathogen invasion is possible across much of parameter space (Figure 5.2, left column). For our parameterization, pandemic equilibrium (i.e., in which all occupied patches contain the pathogen) can occur in the absence of provisioning ($P^* = 1$ when $f = 0$). When provisioning increases the lifetime production of dispersers ($\theta/\alpha \geq 1$), infection remains endemic or pandemic as the landscape becomes fully provisioned (i.e., as $f \to 1$). However, if provisioning promotes site fidelity or provisioned patches otherwise produce fewer dispersers ($\theta < 1$), R_0 declines with f, facilitating pathogen extinction and even host extinction when most of the landscape is provisioned. The

fractions of provisioned patches necessary for pathogen invasion (f_e) and pandemic equilibrium (f_p) increase as provisioning promotes dispersal (i.e., as $\theta \to \infty$). These thresholds also decrease if provisioned patches are more attractive ($\phi = 10$); however, pandemics can be obtained at lower f_p when provisioning also greatly increases lifetime dispersal success (i.e., $\theta/\alpha \gg 1$).

The transitions between these equilibria with provisioning differ markedly when infection reduces dispersal success ($\psi = 0.5$; Figure 5.2, right column). If provisioned patches produce more dispersers over their lifetime ($\theta/\alpha \geq 1$), pathogen invasion requires a smaller fraction of provisioned patches; thus f_e decreases as provisioning promotes dispersal (i.e., as $\theta \to \infty$). The threshold for the pandemic state is described by an inverse relationship between the fraction of provisioned patches and the effect of provisioning on the production of dispersers. Pandemics occur when provisioning is widespread (i.e., as $f \to 1$) and promotes dispersal (i.e., as $\theta \to \infty$).

Effects of provisioning on occupancy and infection

Colonization heterogeneities induced by provisioning and infection determine whether resource supplementation benefits the host metapopulation. When effects of infection on dispersal are low ($\psi = 1$), transmissible pathogens are pandemic in the unprovisioned metapopulation ($P^* = 1$ when $f = 0$). The consequences of increasing f for occupancy and infection prevalence depend on ρ and how provisioning affects lifetime colonization success. When provisioned patches produce fewer dispersers over their lifetime than unprovisioned patches ($\theta/\alpha < 1$) and are equally attractive ($\phi = 1$), increasing f reduces infection prevalence and increases host occupancy until pathogen extinction occurs (Figure 5.3A, grey line); provisioning is most beneficial to the metapopulation when $f_p < f < f_e$ (Table 5.3). Similar patterns are generated when provisioned patches are far more attractive ($\phi = 10$); however, the decline in infection

prevalence occurs at a lower spatial extent of provisioning but is mirrored by a non-monotonic increase in occupancy while $f_p < f < f_e$ (Figure 5.3A, black line). In contrast, increasing f does not benefit the metapopulation when provisioned patches produce more dispersers over their lifetime ($\theta/\alpha \geq 1$). When provisioning has no effect on patch attraction ($\phi = 1$), provisioning increases occupancy at the cost of infection being present in all occupied patches (i.e., $P^* = 1$ across all f; Figure 5.3B, grey line). Increasing the attraction of provisioned patches ($\phi = 10$) shows identical patterns in infection prevalence; however, the increase in occupancy with provisioning is not monotonic, as occupancy is minimized at small f (Figure 5.3B, black line).

For pathogens where infection reduces dispersal success ($\psi = 0.5$), greater spatial extents of provisioning are generally detrimental to the host species (Table 5.3). If provisioned patches produce fewer dispersers over their lifetime ($\theta/\alpha < 1$), increasing f restricts pathogen invasion (i.e., $P^* = 0$ across all f) but at the cost of minimizing occupancy (Figure 5.4A). Host occupancy is maximized in the unprovisioned metapopulation (i.e., $f = 0$), even if provisioned patches are more attractive. Provisioning can be beneficial when provisioned patches produce more dispersers ($\theta/\alpha \geq 1$) if provisioning also has weak effects on patch attractiveness ($\phi = 1$); here increasing f results in a local maximum in occupancy while $f < f_e$ (Figure 5.4B, grey line). Further increasing f facilitates pathogen invasion and pandemics. However, patterns differ when provisioned patches are more attractive ($\phi = 10$). While the criterion for provisioning being beneficial is here identical ($f < f_e$), pathogen invasion is prevented only when a very small fraction of the landscape is provisioned (Figure 5.4B, black line). Consequently, provisioning has approximately negative effects on occupancy under such parameterization (Table 5.3).

DISCUSSION

Many wildlife display metapopulation dynamics [204,205], and previous theory has shown how increases in colonization can enhance pathogen spread in homogenous landscapes [158–160]. However, processes that reduce colonization have rarely been addressed in such frameworks, particularly when considering the influence of both patch quality and infection status. We here develop a simple, generalizable model that integrates these sources of colonization heterogeneity in the important context of resource provisioning and the changing distributions of subsidized habitats across human-modified landscapes. We demonstrate that how local resource abundance affects the preference and net production of dispersers and how infection modifies inter-patch movement can explain whether provisioned landscapes facilitate or limit pathogen invasion. In agreement with previous theory [55,203], our model illustrates that greater abundance of high-quality patches can increase occupancy when provisioning increases the production of dispersers but only at the expense of allowing infection to spread across the landscape. Yet our framework also shows that if host movement responds with site fidelity or sedentary behavior, increased provisioning can reduce infection and cause pathogen extinction while maximizing occupancy. This outcome has not been predicted from previous metapopulation models but could resolve empirical work in which high resource availability correlates with minimal infection.

Increasing the spatial extent of provisioned habitats most benefits host metapopulations when three conditions are met: (*i*) provisioned patches produce fewer dispersers over their lifetime, which could occur owing to greater site fidelity or sedentary behavior; (*ii*) provisioned patches are no more attractive to immigrants than natural habitats; and (*iii*) infection has low costs for dispersal success. Given these requisites and that infection is already pandemic in the unprovisioned landscape, increasing the proportion of high-quality habitats reduces facilitates a decline in infection prevalence when the landscape contains a mixture of both patch types. More

widespread provisioned habitat is beneficial because these regions function as sinks that limit the movement of infectious dispersers between patches. Occupancy is maximized at the threshold for pathogen extinction, at which point enough of the landscape is composed of such patch types that lower disperser production reduces colonization and occupancy declines. Such predictions may therefore be most applicable to nomadic or naturally wide-ranging species for which food subsidies restrict home range or promote greater site fidelity but for which infection has limited effects on movement patterns. For example, nomadic black (*Pteropus alecto*) and grey-headed flying foxes (*Pteropus poliocephalus*) have formed numerous resident camps around consistently available fruit and nectar resources in urban habitats [17,206,207], which could reduce the relative production of dispersers from sedentary behavior and from poorer quality of anthropogenic food. Hendra virus presents asymptomatically in flying foxes [86,208] and thus infection prevalence could be expected to decline over long timescales given our model outputs. Data-driven and spatially explicit modeling efforts support these movement responses to provisioned habitats are linked to less frequent Hendra virus epidemics [62] and hence lower equilibrium prevalence; however, across short timescales these processes could still produce larger epidemics that facilitate spillover [209]. These movement responses in landscapes with relatively high abundance of provisioned habitat could likewise explain positive trends in flying fox occupancy [209,210].

In contrast, when provisioned patches not only produce fewer dispersers but also are more attractive to immigrants, landscapes with more high-quality habitats show low infection prevalence and low occupancy. Attracting and retaining many dispersers could pull in colonizers from the surrounding matrix without contributing to emigration, suggesting provisioned patches could function as ecological traps and net sinks for hosts and pathogens [193,194]. Although we

defined this outcome as generally negative for wildlife, lower occupancy and infection could be deemed beneficial if considering how provisioning affects reservoir hosts and risks of pathogen spillover [211]. For example, raccoons (*Procyon lotor*) are reservoirs for zoonoses like *Salmonella* spp. and *Baylisascaris procyonis* [212–214], and urban habitats with abundant anthropogenic foods attract raccoons and promote more sedentary behavior [189,215]. As raccoons are asymptomatic hosts of these pathogens [212–214], infection could have limited costs for host dispersal. Greater frequency of provisioned habitats might therefore minimize both human–raccoon conflict and spillover risks. However, these processes could still harm threatened species that display similar movement responses to provisioning but for which zoonoses are also asymptomatic, such as Malayan flying foxes (*Pteropus vampyrus*), known reservoirs of Nipah virus [34,216].

Across both these movement contexts, our models also suggest greater attraction to and retention within provisioned habitats could have especially negative effects on occupancy for host–pathogen systems where infection has strong costs for dispersal. In these simulations, landscapes with more provisioned habitats always showed monotonic declines in occupancy. These predictions could be particularly applicable for pathogens such as *Salmonella* spp. and *Mycoplasma gallisepticum* in passerines [217,218], and our model could provide a mechanistic explanation for the population declines of avian hosts in response to these epizootics [168]. For such pathogens where infection reduces dispersal success, provisioning only had positive effects on host occupancy when supplemented patches produced more dispersers over their lifetime. Under these simulations, provisioning can be beneficial when (*i*) provisioned patches produce more dispersers and (*ii*) provisioned patches are similarly as attractive to dispersers. When these conditions are met, some deployment of provisioned habitats can increase occupancy; however, a

substantially provisioned fraction of the landscape facilitates pathogen invasion and pandemics owing to the general increase in colonization [158–160]. Pandemic spread of infection is even more likely when provisioned patches are more attractive and when infection has no costs for dispersal, suggesting that overall resource supplementation is detrimental to host species for which high local food abundance increases the production and attraction of dispersers. One empirical system amenable for future model validation could include the dynamics of vampire bats and rabies virus in relation to greater availability of livestock-dense habitat, given observed roost selection for such environments and their positive demographic responses [20,22,181–183,219]. Work on other wildlife for which provisioning has positive effects on demographic parameters (e.g., survival, fecundity, immigration, emigration), such as many rodents, could also provide tests of these model predictions for pathogens with variable impacts on host movement [220,221].

The distribution of food resources for many wildlife are changing in response to resource provisioning. Our work here provides a simple but unified framework for considering how shifts in host movement influence the spatial dynamics of hosts and pathogens and how infection feeds back into these processes at the landscape scale. We highlight that increasing the availability of provisioned habitats most benefits wildlife when supplemental food resources restrict inter-patch movement in the presence of infection, whereas greater food availability can primarily negative effects on facilitating pathogen spread across landscapes when it stimulates host demography. Future work to obtain empirical estimates of how these colonization heterogeneities affect host movement will strengthen theory presented here and improve our ability to both understand and predict how changing resource distributions can promote or limit the spatial spread of infection.

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COMPETING INTERESTS

We have no competing interests.

AUTHOR CONTRIBUTIONS

DJB, CES, SA, and RJH conceptualized the project; DJB and CES wrote code and ran numerical simulations; CES and RJH derived analytic results; and DJB wrote the manuscript, with all authors contributing to revisions.

Table 5.1. Empirical examples of provisioning- and infection-induced heterogeneities in wildlife movement and the colonization process.

Mechanism	Host species	Effect on colonization process		Ref
Provisioning increases patch attractiveness	*Parnassius smintheus*	↑	Patches with more nectar flowers have more immigration	[184]
	Boloria eunomia	↑	Most movement observed from low- to high-quality habitat	[185]
	Spermophilus beecheyi	↑	More individuals moved into the food-supplemented colony	[222]
	Poecile atricapillus	↑	Feeder habitat receives influx of new birds throughout the year	[223]
Provisioning increases the number of dispersers	*Dendroica caerulescens*	↑	Provisioned female birds produce more second broods	[107]
	Pica pica	↑	Magpies using feeders have higher survival of nest contents and improved fledgling success	[187]
	Aphelocoma coerulescens	↑	Supplemental food increases jay clutch size and offspring survival	[186]
Provisioning reduces the number of dispersers	*Procyon lotor*	↓	Subsidized urban raccoons have smaller home ranges	[189]
	Pteropus poliocephalus	↓	Loss of nomadic behavior as flying foxes develop a permeant camp around urban resources	[190]

	Accipiter gentilis	↓	Control juvenile goshawks disperse while supplemented birds remain in study area	[224]
Infection reduces dispersal success	*Danaus plexippus*	↓	Infection with a protozoan parasite reduces monarch butterfly flight performance	[197]
	Anas platyrhynchos	↓	Mallards infected with avian influenza virus have less regional movement	[225]
	Paramecium caudatum	↓	Bacterial infection lowers the proportion of hosts dispersing	[226]
	Parus major	↓	Birds from flea-infested nests disperse shorter distances	[227]

Table 5.2. Metapopulation model parameters, values or ranges, and interpretations; parameters related to provisioning are presented in grey shading.

Parameter definition		**Value**	**Interpretation**
x	Extinction rate of unprovisioned patches	1	Time is measured in units corresponding to occupancy time of unprovisioned patches
c	Colonization rate of unprovisioned patches	2	The metapopulation persists in the absence of provisioning given that $c > x$
f	Fraction of provisioned patches	0–1	0 to 100% of patches are provisioned
α	Quality difference	0.5	Provisioned patches have a 50% lower extinction rate and persist twice as long as unprovisioned patches
ϕ	Relative attractiveness of provisioned patches	1–10	Provisioned patches are 1–10 times as attractive to dispersers compared to unprovisioned patches
θ	Relative productivity of provisioned patches	0–2	When greater than 1, provisioned patches produce relatively more dispersers compared to unprovisioned patches. When less than 1, provisioned patches produce relatively fewer dispersers compared to unprovisioned patches (e.g., greater site fidelity)
δ	Pathogen transmissibility	1	Represents a very high probability of infection establishment upon the arrival of a diseased colonizer
v	Infection-induced extinction rate	0.3	Infected unprovisioned patches go extinct faster than their uninfected counterparts
ψ	Proportional reduction in	0–1	Infection reduces the proportion of dispersers by 0 to

	dispersal from infection		100%

Table 5.3. Conditions of parameter space under which increasing the spatial extent of provisioning (f) benefits host metapopulations ($\partial N^*/\partial f > 0$ and $\partial P^*/\partial f \leq 0$).

	provisioned patches produce more dispersers over their lifetime ($\theta/\alpha > 1$)		provisioned patches produce fewer dispersers over their lifetime ($\theta/\alpha < 1$)	
	provisioned patches are equally attractive ($\phi = 1$)	provisioned patches are more attractive ($\phi = 10$)	provisioned patches are equally attractive ($\phi = 1$)	provisioned patches are more attractive ($\phi = 10$)
infection has no cost for dispersal ($\psi = 1$)	$f_p < f < f_e$	$f_p \lesssim f \lesssim f_e$	$f = 0$	$f = 0$
infection has high costs for dispersal ($\psi = 0.5$)	$f = 0$	$f = 0$	$f < f_e$	$f \approx 0$

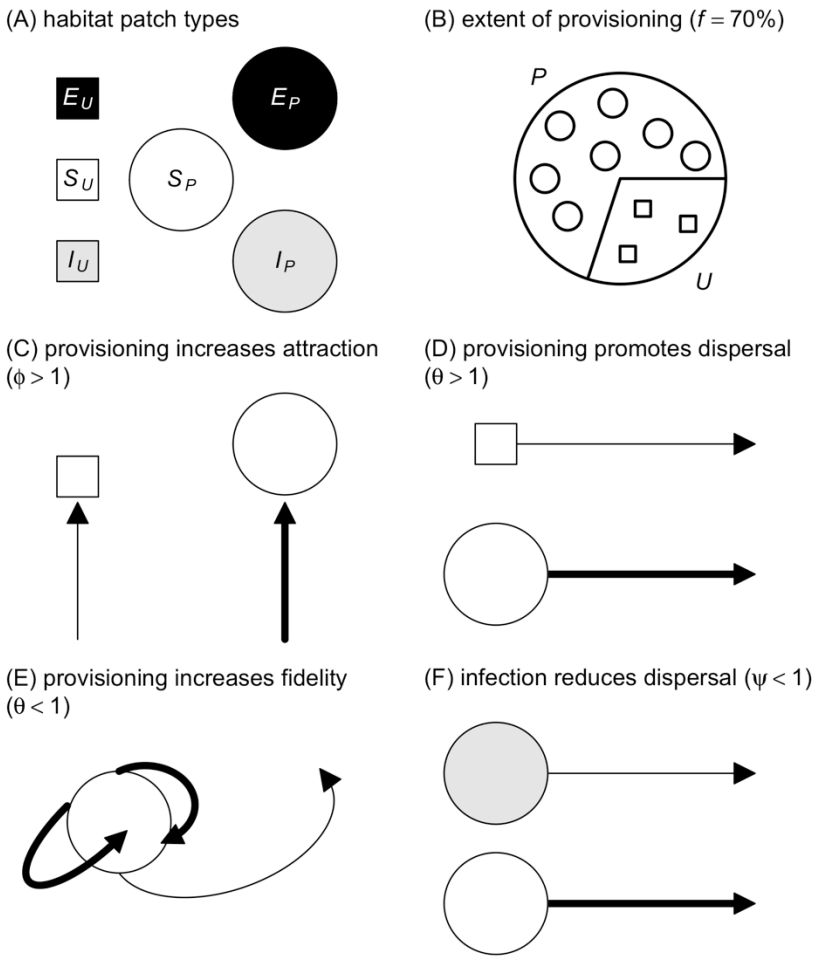

Figure 5.1. Conceptualization of how provisioning and infection affect the between-patch colonization process. (A) The metapopulation consists of habitat patches that are unoccupied and empty (*E*, black), susceptible (*S*, white), or infected (*I*, grey), with subscripts denoting whether patches are unprovisioned (*U*, squares) or provisioned (*P*, large circles). (B) The spatial extent of provisioning across the landscape is given by f, denoting the proportion of provisioned habitat. (C) Provisioned patches can attract more dispersers than unprovisioned habitats (when $\phi > 1$). Provisioned patches can next either (D) produce more dispersers than unprovisioned patches (i.e., $\theta > 1$) or (E) retain more dispersers by promoting site fidelity (i.e., $\theta < 1$). (F) Infection also reduces the proportion of dispersers that successfully move between habitats (when $\psi < 1$).

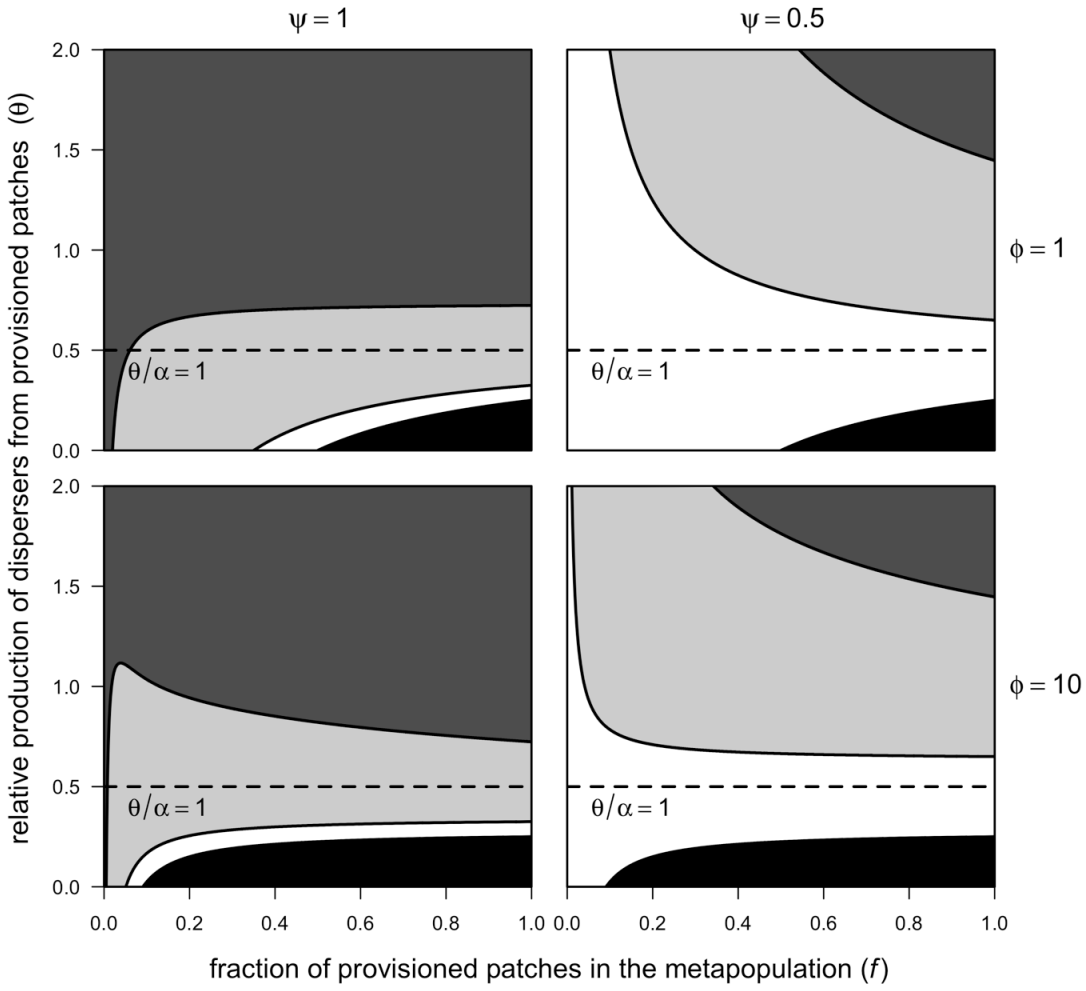

Figure 5.2. Effects of varying the fraction of provisioned patches (f) and how provisioning affects host dispersal (θ) on model equilibria and stability. Shading depicts regions of parameter space where the metapopulation is extinct ($\rho \leq 1$, black), where the host persists in a disease-free state (white), where the pathogen can invade ($R_0 \geq 1$) and become endemic (gray), and where the pathogen obtains pandemic equilibrium (dark grey). Conditions are shown for cases where infection has no ($\psi = 1$) or high ($\psi = 0.5$) costs for dispersal and where provisioned patches are equally ($\phi = 1$) or more ($\phi = 10$) attractive to dispersers. Dashed lines show θ for which provisioned patches produce an equal quantity of dispersers over their lifetime ($\theta/\alpha = 1$).

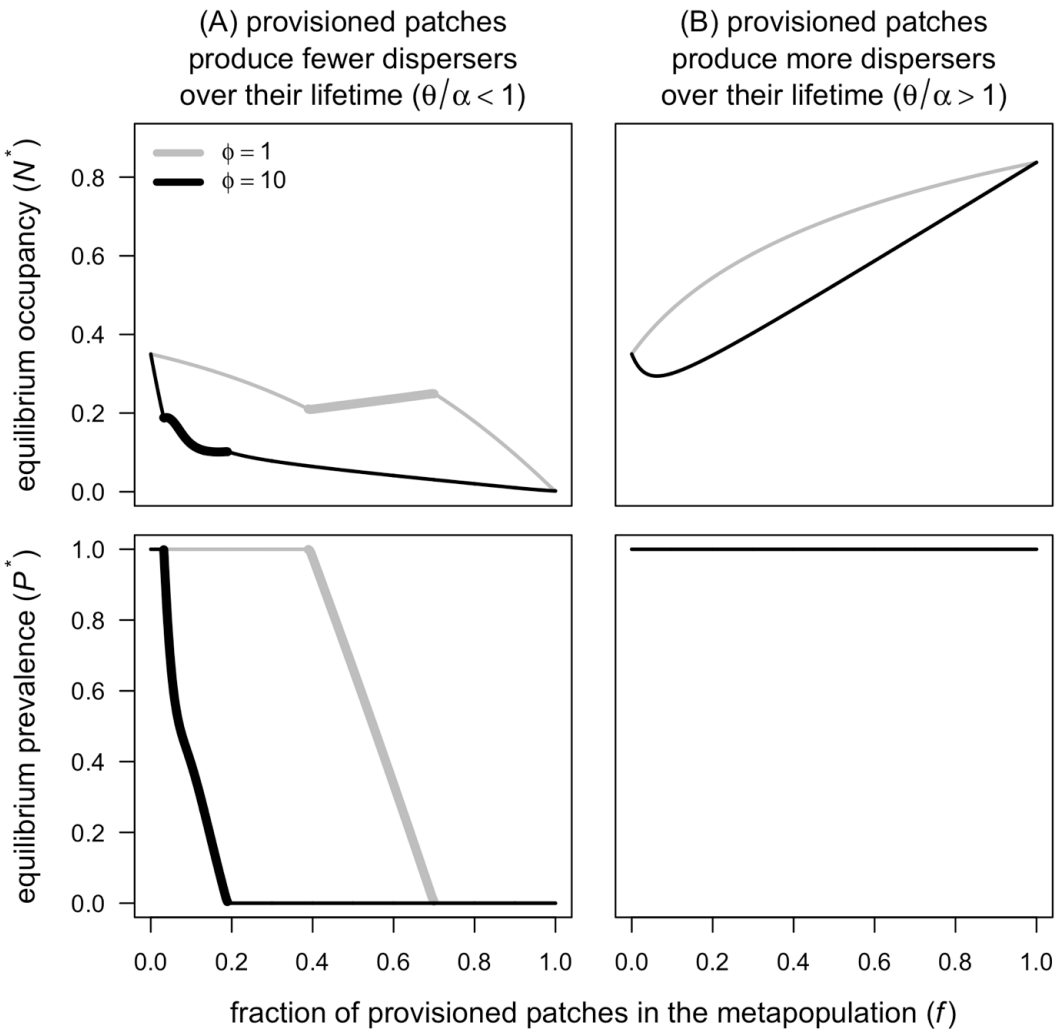

Figure 5.3. Effects of varying the fraction of provisioned patches (f) on equilibrium occupancy (N^*, top) and infection prevalence (P^*, bottom) for cases where provisioned patches produce fewer ($\theta/\alpha < 1$; A) and more ($\theta/\alpha > 1$; B) dispersers over their lifetime relative to unprovisioned patches. Conditions are shown for parameterization where provisioned patches are equally ($\phi = 1$, grey line) or more ($\phi = 10$, black line) attractive to dispersers. Simulations are shown for pathogens where infection has no costs for dispersal ($\psi = 1$). Thick lines show where provisioning is most beneficial ($\partial N^*/\partial f > 0$ and $\partial P^*/\partial f \leq 0$).

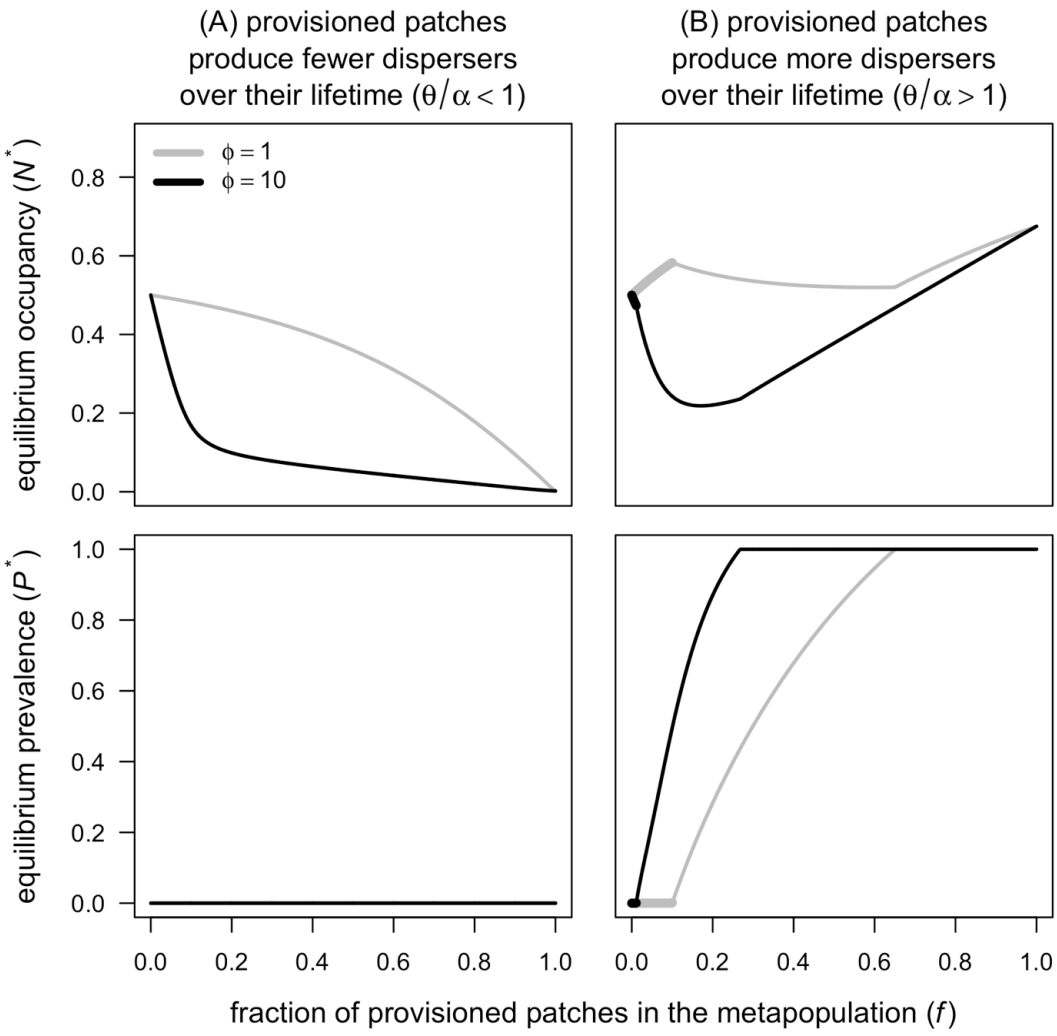

Figure 5.4. Effects of varying the fraction of provisioned patches (f) on equilibrium occupancy (N^*, top) and infection prevalence (P^*, bottom) for cases where provisioned patches produce fewer ($\theta/\alpha < 1$; A) and more ($\theta/\alpha > 1$; B) dispersers over their lifetime relative to unprovisioned patches. Conditions are shown for parameterization where provisioned patches are equally ($\phi = 1$, grey line) or more ($\phi = 10$, black line) attractive to dispersers. Simulations are shown for pathogens where infection has high costs for dispersal ($\psi = 0.5$). Thick lines show where provisioning is most beneficial ($\partial N^*/\partial f > 0$ and $\partial P^*/\partial f \leq 0$).

CHAPTER 6

LIVESTOCK EXPANSION PREDICTS VAMPIRE BAT DEMOGRAPHY, IMMUNE PROFILES, AND BACTERIAL INFECTION RISK [5]

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[5] Becker DJ, Czirják GÁ, Volokhov DV, Bentz AB, Carrera JE, Camus MS, Navara KJ, Chizhikov VE, Fenton MB, Simmons NB, Gilbert AT, Recuenco SE, Altizer S, Streicker DG. To be submitted to *Philosophical Transactions of the Royal Society B: Biological Sciences*.

ABSTRACT

Human activities such as agriculture create abundant and predictable food resources that can alter wildlife–pathogen interactions. How these resources affect host immune defense can determine whether resource provisioning amplifies or dampens pathogen transmission. Yet studies linking food availability, immunity, and infection are rare in natural systems, where resource provisioning can simultaneously also affect host demography and behavior. We tested these relationships through a four-year study of 389 vampire bats (*Desmodus rotundus*) across 10 sites in Peru and Belize that differ in the availability of livestock, an important anthropogenic food source for vampire bats. We quantified innate and adaptive immune measures from bats and predicted greater feeding on livestock could reduce starvation stress and energy spent foraging, allowing bats to invest more in immune defenses. Results showed that bats from high-livestock sites had higher proportions of neutrophils and microbicidal activity but lower immunoglobulin G and proportions of lymphocytes, suggesting greater chronic stress and investment in innate immunity relative to adaptive immunity. This relationship was most pronounced in reproductive bats, which were also more common in livestock-dense habitats, suggesting a negative feedback between fitness benefits of provisioning and immune investment. Prevalence of *Bartonella* spp. declined with investment in innate immunity and was accordingly lowest in bats from high-livestock sites. Prevalence of hemoplasmas was also negatively related to investment in innate immunity yet was unrelated to livestock biomass. Differences in how provisioning correlates with bacterial infection might reflect different mechanisms of infection clearance or different transmission modes. Our results highlight the challenges in predicting how resource provisioning alters host–pathogen interactions and emphasize the need to consider how both within-host processes and between-host transmission respond to anthropogenic shifts in food resources.

Keywords: agricultural intensification; *Bartonella*; demography; *Desmodus rotundus*; ecoimmunology; hemoplasmas; livestock density; resource provisioning; supplemental feeding

INTRODUCTION

Human activities such as agriculture, urbanization, and recreational feeding can create abundant, predictable food resources for many wildlife [4,5]. While supplemental resources can benefit species facing seasonal food shortages [49], they can also have negative conseuqneces for human and animal health [33,228]. Resource provisioning can facilitate pathogen spillover owing to novel assemblages of host species at anthropogenic resources. For example, increased spatial overlap between mango plantations and pig farms in Malaysia attracted flying foxes (*Pteropus* spp.) to abundant fruit, resulting in spillover of Nipah virus from bats to pigs and humans [34]. However, supplemental food can have the opposite effect of reducing pathogen transmission; for example, red foxes (*Vulpes vulpes*) in Switzerland foraging on urban waste were less frequently infected with a zoonotic tapeworm compared to rural foxes [41]. Understanding when provisioning increases or decreases infection in wildlife is thus important to manage emerging disease risks.

Increased infection from provisioning is partly explained by changes in host demography and behavior such as increased fecundity and aggregation [25,26], which can amplify pathogen transmission from density dependence and increased contact with conspecifics and infectious stages [45,48,229]. At the individual host level, these processes could be offset and pathogen transmission can decline if improved nutrition enhances host resistance to or recovery from infection [52,61]. Because immune defenses are energetically costly [230], supplemental feeding can alleviate tradeoffs between immunity and other physiological processes (e.g., growth rate [231]) or between different arms of the immune system [232]. Reduced starvation stress from provisioning can also improve host immunity; *ad libitum* access to food increased antibody production in deer mice (*Peromyscus maniculatus*) [233] and allowed voles (*Microtus agrestis*)

to mount stronger defenses against nematodes [44]. Yet a broad understanding of the links between food availability, immune defense, and infection remains elusive in natural systems, where resource provisioning can simultaneously also affect host demography and behavior.

The common vampire bat (*Desmodus rotundus*) has experienced major ecological changes due to provisioning throughout its range in Latin America [234]. Although uncommon to rare in undisturbed habitats [235], vampire bats can become abundant where their ranges overlap with agricultural lands [22,234]. While vampire bats historically fed on wild mammals in forested habitats, populations residing near humans now preferentially feed on livestock and poultry owing to their greater accessibility and reliability [20,56]. This feeding preference appears driven by predictability and quantity rather than quality, because mammalian livestock and wildlife have similar blood composition (i.e., hematocrit) [236] and vampire bats do not select blood based on taste [237]. Greater access to livestock increases the probability of feeding success [22,57,238], which could improve vampire bat immune defense given their physiological sensitivity to starvation [58,59]. Bats occupying livestock-dense habitat could thus show lower physiological stress and improved immune measures. However, livestock-dense habitat could also represent an ecological disturbance that could suppress bat immunity [239]. Greater food could also increase local bat density from more reproduction and immigration [22,181], which could contribute to a larger susceptible pool and shift allocation of immune defenses [240,241]. Thus, diet shifts towards livestock or occupancy in agricultural habitat could increase or decrease pathogen transmission.

Here we conducted a four-year field study of vampire bats across 10 sites in Peru and Belize that differ in livestock densities to ask how resource provisioning predicts changes in host demography, immunity, and infection. To test the prediction that provisioned bats shift foraging

toward livestock prey, we first assessed relationships between livestock density and bat feeding patterns as revealed by isotopic analysis of bat hair samples. Second, to test the prediction that greater availability of livestock stimulates bat demography, we examined associations between livestock density and the reproductive activity and sex of bats in each population. Third, we assessed the relative importance of diet (inferred from isotopic analysis) and local livestock density for eight measures of bat immunity, including humoral and cellular effectors of the innate and adaptive immune system [242]; we expected that bats in livestock-dense habitats and with a diet dominated by livestock would show lower chronic stress and more robust humoral measures of immunity. We lastly tested if and how provisioning-mediated variation in immunity was linked to infections with two sympatric bacteria common in Neotropical bats, *Bartonella* spp. and hemotropic *Mycoplasma* spp. (i.e., hemoplasmas) [243,244]. Although their transmission routes in bats are poorly understood, *Bartonella* are generally spread by arthropod vectors [245–247], while hemoplasmas transmit primarily from direct (i.e., blood and saliva) and potentially vector-borne exposure [248–250]. Differential responses of these bacteria to provisioning could therefore reflect contrasting transmission modes or different immune defenses. We therefore used statistical tools for assessing hypothesized causal relationships to assess the potential for effects of provisioning on infection to be mediated through immunological mechanisms.

MATERIALS AND METHODS

Field sites and livestock density

Between July 2013 and September 2016, we sampled 389 vampire bats across 10 sites in Peru (Departments of Cajamarca, Amazonas, and Loreto) and Belize (Orange Walk District; Figure

6.1A). Sampling consisted of capture–recapture over 2–5 nights per site. During 2013–2014, we sampled regions in distinct years (Amazonas and Cajamarca in 2013, Belize and Loreto in 2014). All sites were then repeatedly sampled 1–2 times annually between 2015–2016.

Capture sites consisted of broadleaf deciduous, upland, or flooded forest and spanned a range of agricultural intensities. Sites in Belize were located near agricultural habitat [251,252]. Sites in Peru included intact forest and areas with small- to intermediate-scale cattle farming [103,253,254]. Half our capture sites (n=4) included structures (trees, caves, cistern, Mayan ruins) known to be inhabited by vampire bats. Other sites (n=6) included capture near livestock corrals and chicken coops where bat bites had been reported. Sites were separated by at least 8 km, as movement between roosts occurs over lesser distances [182]. Exceptions were made for several sites in Amazonas and Loreto, which were 0.12–2.85 km apart; however, no recapture between these sites was detected, whereas several bats were recaptured consistently in the same site.

We quantified livestock density as the total biomass of mammalian livestock (cattle, pigs) and poultry (chickens) [hereafter livestock biomass] within a 5 km radius of each site using 2005 modelled livestock density estimates of the Food and Agriculture Organization (FAO) [20,255] and average species mass (kg) from the AnAge Database [256]. FAO data were provided in 3 minutes of arc resolution and were processed and assigned to sites (Figure 6.1B and Appendix E Table S6.1) using the *raster* package in R [119]; livestock biomass was normalized with a log transformation.

Capture and sampling of vampire bats

Vampire bats were captured in mist nets or harp traps placed at roost exits, along flight paths, or outside livestock corrals from 19:00–05:00. Upon capture, bats were placed in individual holding bags and issued a uniquely coded incoloy wing band (3.5 mm, Porzana Inc.). We classified age as juvenile, sub-adult, or adult based on fusion of phalangeal epiphyses [257]. Reproductive activity was indicated by the presence of scrotal testes in males and by evidence of pregnancy or lactation in females. For isotopic analysis of diet, we trimmed <5 mg hair from the back of each bat [20]. To quantify bat immune measures, we obtained up to 150 μL blood by lancing the propatagial vein with a sterile 23-gauge needle, followed by collection with heparinized capillary tubes. Thin blood smears were prepared on glass slides and stained with buffered Wright–Giemsa (Camco Quik Stain II). Plasma was obtained by centrifuging blood in serum separator tubes and stored on cold packs until freezing at –20°C and long-term storage at –80°C. Up to 30 μL blood was stored on Whatman FTA cards to preserve bacterial DNA [258]. Except for 14 bats that were humanely sacrificed for other studies, all bats were released at their capture site.

Stable isotope analysis

Hair samples were dried at 60°C for 72 hours and cut into 1 mm fragments [20]. Stable carbon (^{13}C) and nitrogen (^{15}N) isotope signatures were determined using a Thermo Delta V isotope ratio mass spectrometer at the UGA Center for Applied Isotope Studies. Isotope values were expressed in standard δ notation, where $\delta^{13}C$ or $\delta^{15}N = [(R_{sample}/R_{standard}) - 1] \times 1000$, and R is the ratio of $^{13}C/^{12}C$ or $^{15}N/^{14}N$. Analyses used two standards per 12 samples for $\delta^{13}C$ and $\delta^{15}N$: bovine (σ=0.05, 0.30 and μ=–21.75, 7.44) or 1577c (σ=0.08, 0.10 and μ=–17.52, 8.12) and spinach (σ=0.23, 0.42 and μ=–27.39, –0.48).

Vampire bat feeding on livestock has been differentiated from feeding on wildlife using $\delta^{13}C$ [20,236], as most grasses consumed by livestock use the C4 pathway and most forest plants consumed by wildlife use the C3 pathway [259]. $\delta^{15}N$ also provides inference into trophic level, as consumer $\delta^{15}N$ is enriched by 3−4‰ relative to its diet [260]. Samples were opportunistically collected from potential prey in each study region to quantify differences in bat feeding patterns while accounting for different isotopic baselines. Prey included cattle (*Bos* spp.), horses (*Equus caballus*), chickens (*Gallus domesticus*), pigs (*Sus scrofa domesticus*), goats (*Capra aegagrus hircus*), tapir (*Tapirus bairdii*), red brocket (*Mazama americana*) and white-tailed deer (*Odocoileus virginianus*), peccaries (*Tayassu* spp.), and lowland paca (*Cuniculus paca*); means and standard deviations of prey groups are presented in Figure S1. Within each study region, we calculated the minimum distance in isotopic space between each bat and the mean positions of mammalian livestock and poultry to estimate consumption of provisioned food [261]. We did not use mixing models given that prey coverage was uneven between study regions.

Quantifying bat immune components

We used leukocyte profiles from blood smears to measure investment in cellular immunity [262] and chronic stress, given that high ratios of neutrophils to lymphocytes can indicate elevated blood glucocorticoid hormones [263]. We estimated total white blood cells (WBCs) as the average number of leukocytes from 10 fields of view at 400X magnification with manual light microscopy [264]; quantitative counts were not performed owing to limited blood volumes and remote field conditions. Nucleated cell differentials recorded the percentage of neutrophils, lymphocytes, monocytes, eosinophils, and basophils by counting 100 leukocytes at 1000X magnification. Total WBC estimates were normalized with a quarter-root transformation.

We assessed humoral innate immunity by quantifying the *ex vivo* bacterial killing ability (BKA) of plasma against *Escherichia coli* ATCC 8739 [265], which is mediated mostly through complement proteins [266]. We used the microplate reader method [267], using 1:8 dilutions of plasma to phosphate buffered saline (PBS) run in 22 μL duplicates and challenged with 5 μL of a 10^4 bacteria/mL solution in PBS (E power Microorganisms #0483E7, Microbiologics Inc.) [251]. We prepared tryptic soy broth (TSB; Bacto, BD) two days before assays [268] and added 125 μL TSB to each well. Optical density (OD) was measured at 340 nm to determine background OD before and after incubation for 12 hours at 37°C. We subtracted background OD from OD at 12 hours and calculated BKA as one minus mean OD per sample, divided by mean OD of positive controls. To quantify humoral adaptive immunity, we measured immunoglobulin G (IgG) antibody in plasma with a protein G ELISA [269], which binds IgG from many wildlife including bats [270]. We diluted 3 μL of each sample to 1:30,000 with 50mM NaHCO3 buffer (pH 9.5) and ran 100 μL of each sample in duplicate using protein G–horseradish peroxidase conjugate (P21041, Life Technologies) [251]. We included human IgG (MP Biomedicals, LLC) as a positive control. According to the Beer–Lambert law, antibody concentration is proportional to OD (measured at 450 nm); we therefore analyzed mean IgG OD of duplicates.

Pathogen detection

Blood smears were screened for extracellular haemoparasites (trypanosomes and microfilariae) by microscopically reviewing 100 fields of view at 400X magnification [271]. For detection of bacteremia, genomic DNA was isolated from blood on Whatman FTA cards using QIAamp DNA Investigator Kits (Qiagen, Hilden, Germany) following the manufacturer's instructions. For *Bartonella* spp*.,* we used nested PCR to amplify a region of the citrate synthase gene (*gltA*),

which has high discriminatory power for differentiating among *Bartonella* species [272]. External primers were 443f and 1210r, followed by internal primers 781f and 1137r [273]. For hemoplasmas, we amplified the partial 16S rRNA hemoplasma genes with the universal primers UNI_16S_mycF and UNI_16S_mycR [274], and all strong amplicons were sequenced using the same primers (Volokhov et al., in review).

Statistical analysis

We first used a permutation multivariate analysis of variance (PERMANOVA) to test whether livestock biomass predicts bat isotopic position (matrix of hair $\delta^{13}C$ and $\delta^{15}N$; n=315). We then used a linear mixed model (LMM) fit with maximum likelihood (ML) to test if the minimum isotopic distance of bats from either livestock or poultry prey varied with livestock biomass [275]. Bat identification number (ID) was included a random effect to account for multiple sampling of individuals over time (n=13). We fit LMMs with linear and quadratic terms and compared these with a likelihood ratio test. LMMs were then refit with restricted ML (REML) to calculate marginal r^2 (r^2_m) and conditional r^2 (r^2_c) [276]. We also used generalized linear mixed models (GLMMs) with binomial errors, a logit link, and bat ID (random effect) to test if livestock biomass was associated with bat reproduction (n=382) and sex (n=384).

To analyze immunological data, we used a principal component analysis (PCA) to collapse eight measures (Table S6.2; $|\rho|$ ranged from 0.01–0.98, $|\mu|$=0.18) into one axis [277]. The PCA included the proportion of each WBC type, estimated WBCs, BKA, and IgG, with variables centered and scaled to have unit variance (n=166; Figure S6.2). PC1 accounted for 30% of the variance and was loaded positively by neutrophils (0.60), basophils (0.01), estimated WBCs (0.10), and BKA (0.27) and negatively by lymphocytes (–0.58), monocytes (–0.33),

eosinophils (–0.27), and IgG (–0.20). Larger PC1values represents more investment in innate immunity (neutrophils, BKA, total WBCs) and less investment in adaptive immunity (lymphocytes, IgG).

We first tested relationships between provisioning and bat immunity using another PERMANOVA that evaluated how all measures correlate with livestock biomass and bat diet. To next assess the relative contribution of livestock biomass and bat diet on immunity, we used ML to fit LMMs with PC1 as the response variable, bat ID as a random effect, and livestock biomass, minimum isotopic distance from livestock and poultry, bat age, sex, and reproductive status as fixed effects with appropriate interactions (Table S6.3); for example, sample sizes were insufficient to consider a three-way interaction between livestock biomass, sex, and reproductive status. We generated a candidate set of all possible additive LMMs limited to a maximum of six covariates each to keep the number of models low (R=93) relative to our sample excluding missing values (n=157) [278]. We compared LMMs with Akaike information criterion corrected for small sample size and refit models with REML to calculate r^2_m and r^2_c. We used model averaging to estimate mean effect sizes and 95% confidence intervals for how livestock biomass and isotopic distance correlate with the immune component PC1. Averaging was performed across LMMs whose cumulative Akaike weight (w_i) summed to 95%, and mean coefficients were standardized with partial standard deviation [279]. We used the *MuMIn* and *lme4* packages for model selection and averaging [280,281].

We used causal mediation analysis (CMA) to assess associations between provisioning, bat immune profiles, and bacterial infection. CMA estimates how much of a direct relationship between two variables (i.e., outcome model) is mediated indirectly through a third variable (i.e., mediator model) [282]. The mediator model was given by the most competitive LMM for the

relationship between provisioning and immunity identified above. For the outcome model, we fit GLMMs with binomial errors and a logit link separately for infection with *Bartonella* and hemoplasmas. Each GLMM included the provisioning covariate in the mediator model and our immunity axis (PC1). Models were fit to reduced datasets (n=128 for *Bartonella* and n=112 for hemoplasmas) to accommodate missing data; results from univariate GLMMs are provided in the Supplemental Material. We performed CMA with 10000 Monte Carlo draws using the *mediation* package to estimate the proportion of the relationship between provisioning and infection mediated through the immune component PC1 [283].

RESULTS

Livestock biomass, bat diet, and demography

We detected broad variation in vampire bat feeding strategies across sites (Figure 6.1E). Bats in Loreto, where livestock biomass was generally lowest, had lower $\delta^{13}C$ and higher $\delta^{15}N$ than bats in Amazonas, Cajamarca, and Belize, where livestock biomass was greater (Table S6.1, Figure S6.1). PERMANOVA confirmed bat $\delta^{13}C$ and $\delta^{15}N$ were significantly associated with livestock biomass ($F_{1,314}$=67.71, p<0.001), although this measure only explained 18% of the variation in bat isotopic space. Comparison of isotope values from bats and their potential prey suggested bats in low-livestock sites foraged on poultry and wildlife (e.g., LR1 and LR3), while bats in high-livestock sites fed primarily on mammal livestock and poultry (e.g., CA1 and OW2). A likelihood ratio test found that a linear LMM of livestock biomass was only weakly supported over a quadratic LMM (X^2=2.71, p=0.10); yet we found no linear relationship between livestock biomass and bat isotopic distance to livestock and poultry (X^2=0.38, p=0.54, r^2_m=0; Figure S6.3A). The quadratic LMM instead showed that bat isotopes were further from livestock and

poultry in sites with low livestock biomass, whereas bats in sites with high livestock biomass displayed isotopes more closely aligned with all livestock prey (X^2=2.70, p=0.10, r^2_m=0.01; Figure S6.3B).

Bat demography also varied with livestock biomass. The proportion of reproductive bats per site ranged from 0–76% and was positively associated with livestock biomass (X^2=6.73, p<0.01, r^2_m=0.03; Figure 6.2A). The proportion of male bats per site ranged from 24–86% and was similarly positively associated with livestock biomass (X^2=11.81, p<0.001, r^2_m=0.05; Figure 6.2B).

Immunological correlates of provisioning

Measures of provisioning predicted differences in bat immunity; livestock biomass explained 10% of the variation in immune profiles (PERMANOVA; $F_{1,160}$=20.19, p<0.001), while isotopic distance from livestock explained 8% of this variation ($F_{1,160}$=16.46, p<0.001). When averaging was applied across the 95% confidence set of LMMs (Figure 6.3A; Table S6.3), immune component PC1 positively correlated with livestock biomass (β=0.42, 95% CI=0.22 to 0.63) but showed no relationship with isotopic distance from provisioned food (β=0.13, 95% CI=–0.10 to 0.35). Stronger effects of livestock biomass were also reflected in this covariate having greater relative importance (1.00) than isotopic distance (0.17). The most competitive LMMs (ΔAICc≤2) contained livestock biomass, reproductive status, age, and isotopic distance; the former two terms appeared in all LMMs (Table 6.1). The top model with livestock biomass and reproductive status (w_i=0.13, r^2_m=0.35) was the most parsimonious (k=3). Applying this LMM to a complete dataset (n=163) showed that the immune component PC1 was greatest for bats in

high-livestock sites ($F_{1,160}$=15.41, p<0.001; Figure 6.3B) and for reproductive bats ($F_{1,160}$=42.60, p<0.001; Figure 6.3B).

As many immune parameters can reflect the acute stress of capture and handling [284], we assessed if bat immunity correlated with the time between capture and blood sampling. Holding time varied from 5–983 minutes and was positively associated with the immune component PC1 across our sample (X^2=14.21, p<0.001; n=136); however, including holding time in our LMMs did not affect mean relationships between the immune component PC1 and livestock biomass (β=0.40, 95% CI=0.19 to 0.61) or isotopic distance (β=0.02, 95% CI=–0.20 to 0.23; Figure S6.4A). Analyses were also robust to excluding bats held for more than four hours [285] (Figure S6.4B).

Given the relationship between livestock biomass and bat reproduction on immunity, we tested whether the greater proportions of reproductive and male bats associated with provisioning (Figure 6.2) were linked to variation in bat immune profiles. Linear regressions weighted by log sample size showed that the proportion of reproductive bats (r^2=0.51, p=0.01) and male bats (r^2=0.56, p<0.01) were positively related to mean immune component PC1 per site (Figure S6.5).

Links between provisioning, immunity, and bacterial infection

Prevalence of *Bartonella* and hemoplasmas in 192 bats as assessed by PCR was 67%, ranging from 44–100% for *Bartonella* and 48–86% for hemoplasmas by site; neither bacteria were detected microscopically. Coinfection prevalence was 51% (95% CI=0.43 to 0.58; n=188) and infection was positively associated; bats positive for *Bartonella* had higher odds of infection with hemoplasmas (OR=2.92, p<0.01). BLAST search in GenBank showed that sequences identified

in these populations showed phylogenetic similarity to to hemoplasmas from other non-vampire bats, non-human primates, rodents, and humans (Volokhov et al., in review). Among the 290 bats for which hemoparasites were assessed microscopically, we detected no trypanosomes, and microfilariae were detected in only one bat from AM3.

Univariate GLMMs showed that the odds of both bacterial infections tended to decline with livestock biomass (Figure S6.6), though the effect size for *Bartonella* was stronger (OR=0.64, $p<0.001$) than that for hemoplasmas (OR=0.84, $p=0.07$). Our multivariable GLMMs with both livestock biomass and the immune component PC1 also showed that prevalence of *Bartonella* tended to decrease with livestock biomass (OR=0.78, $p=0.06$; Figure 6.4A) but that of hemoplasmas showed no response (OR=0.99, $p=0.94$; Figure 6.4B). Greater investment in innate immunity was weakly negatively associated with *Bartonella* infection (OR=0.74, $p=0.06$; Figure 6.4C) but strongly negatively associated with hemoplasma infection (OR=0.61, $p<0.01$; Figure 6.4D). Accordingly, CMA showed that while 22% of the direct relationship between livestock biomass and *Bartonella* infection was mediated by the relationship between livestock biomass and immune profiles ($p=0.06$), no significant mediation was detected for hemoplasma infection ($p=0.30$).

DISCUSSION

Our study shows that resource provisioning in the form of livestock expansion predicts changes in vampire bat demography, immune profiles, and bacterial infections, which we hypothesize acts through multiple mechanisms (Figure 6.5). Initial results showed livestock biomass predicted isotope indicators of provisioned food in bat diets through a roughly saturating relationship, which could suggest that even small introductions of livestock prey shift bat feeding

toward a domestic animal–dominated diet [20,236]. Bat satiation with provisioning (e.g., when livestock are locally common and easily accessible) could also explain the positive relationships between livestock biomass and the proportion of reproductive and male bats per site. As vampire bats are highly susceptible to starvation [59], the reliable and abundant resources provided by livestock could facilitate greater survival and opportunities for bat reproduction [257]. The higher frequency of males in provisioned sites could result from improved maternal condition biasing sex ratios towards males [156,183,286] or from greater immigration of males from neighboring sites [181–183].

Livestock density and bat immunity

Bats in high-livestock habitats had a greater proportion of neutrophils in blood, higher BKA, and more leukocytes but lower levels of plasma IgG and proportions of lymphocytes. This indicates that abundant livestock might contribute to a shift from adaptive immunity to innate immunity. Livestock biomass was a stronger predictor of this relationship than individual bat diet (Figure 6.5), suggesting an indirect relationship between provisioning and bat immunity and that consistency of feeding on livestock cannot explain these observations. One explanation could be that bats in high-livestock habitats experience greater chronic stress, consistent with higher neutrophil-to-lymphocyte (NL) ratios of bats from these sites [263]. High chronic stress in provisioned wildlife is somewhat unexpected [29,287] but could arise if anthropogenic foods are of poor nutritional quality [96], contain contaminants that compromise immunity [122], or if supplemental resources are accompanied by habitat degradation [239]. Alternatively, the shift towards innate immunity could reflect greater testosterone production, given that higher proportions of reproductive and male bats were captured in livestock-dense habitats and that sites

with more male bats displayed immune profiles oriented towards innate immunity. This mechanism is consistent with prior work showing that testosterone can enhance investment in innate immunity [262,288] and could be supported by future work quantifying testosterone in tissue with long turnover such as hair.

Another mechanism that could cause immunological shifts in response to provisioning relates to changes in bat density and associated interactions (Figure 6.5). Increased births and immigration with supplemental feeding could facilitate crowding and food competition [123,228]. For example, tourist-fed southern stingrays (*Dasyatis americana*) displayed more aggressive interactions and higher stress levels [97,124]. While we could not quantify bat density owing to limited recaptures, reproductive activity was more common in high-livestock sites and predicted inflammatory immune profiles at the site level, supporting physiological costs to demographic benefits of provisioning. The observational nature of our study does not allow us to exclude that shifts towards innate immunity could also represent a response to higher pathogen pressure in provisioned habitats where bat density is greatest [29,289]. However, we note that while high NL ratios can also reflect acute infection, PC1 was also negatively loaded by the proportion of eosinophils, for which declines are more consistent with elevated glucocorticoid levels [290].

Infection correlates of differential immunity

Shifts towards innate immunity associated with higher livestock biomass correlated with lower odds of bat infection with *Bartonella* and hemoplasmas (Figure 6.5). The pathogenesis of bacterial infection in bats is poorly understood [291,292]. Both *Bartonella* and hemoplasmas are typically intracellular infections, with the former typically producing asymptomatic infection in

reservoir hosts [293]. Experimental studies of *Bartonella* infection in mice and in cats have identified a role for adaptive immunity (e.g., B cells, T cells, IFN-γ) in bacterial clearance [294,295]. Patterns of infection in vampire bats partly agree with these findings, as bats with more leukocytes had lower odds of infection; however, our data suggest resistance or clearance of infection depends more on innate than adaptive immune pathways in bats. Importantly, *in vitro* studies confirm that complement proteins, which mediate BKA in bat plasma [266], are important in defenses against *Bartonella* [296]. Findings here thus lend some support to the hypothesis that innate immunity may operate distinctly in bats than in other mammals [297,298]. Further work including experimental infection trials, additional longitudinal studies, and mathematical models will be necessary to understand whether innate immune components manifest in bacterial resistance or clearance in bats and to predict the consequences are for epidemiological dynamics [299].

Theoretical insights into bacterial prevalence

Despite the association between innate-oriented immune profiles and lower odds of bacterial infection at the level of individual bats, only prevalence of *Bartonella* decreased with livestock biomass. We found that 22% of this association was mediated by the relationship between provisioning and immune profiles, supporting a key role of resource-mediated immune variation for shaping differences in prevalence [52,61]. For a pathogen likely transmitted via frequency-dependent contact (e.g., bat flies or arthropod vectors [245–247]), *Bartonella* transmission may not increase with the higher bat densities that are predicted to inhabit livestock-dense habitats. In the absence of greater exposure to infection, higher resistance to or recovery from infection should decrease prevalence as we observed here [52]. Such processes could explain similar

patterns of vector-borne disease responses to supplemental food, such as West Nile virus in songbirds [71]. Alternatively, supplemental food could allow bats to spend less time foraging and more time grooming [300], which could lower ectoparasitism and reduce vector-borne transmission [301].

Hemoplasma prevalence showed no relationship with livestock biomass, which could arise if transmission-enhancing effects of provisioning on bat density and immigration increase contact rates and pathogen transmission [22,52,181,183,229]. The negative relationship between innate immunity and infection was stronger for hemoplasmas than for *Bartonella*, suggesting that transmission-enhancing processes are required to offset the expected declines in prevalence [52]. Direct transmission of hemoplasmas via saliva and blood is likely [249,250], particularly given the food-sharing habits of vampire bats [302]; this supports the idea that hemoplasma transmission could increase in response to provisioning within contexts where innate immunity is suppressed.

Conclusions

Resource provisioning in the form of livestock expansion predicted important differences in vampire bat demography and immune defense that could interact to affect infection dynamics in complex ways. Understanding how greater reproduction and investment in innate immunity in bats living in livestock-dense habitats affects infection dynamics is complicated owing to multi-scale factors, although our data suggest the prevalence of vector-borne bacterial pathogens such as *Bartonella* could decline with provisioning from strong effects on bat susceptibility and recovery. For pathogens with direct or more complex transmission routes (e.g., hemoplasmas), an important next step is to disentangle the contribution of resource-altered demography and

immunology with a combination of field studies and mechanistic modeling approaches. Such advances would also be important for predicting how shifts in bat demography and immunity affect viral dynamics. As bats in high-livestock sites also showed lower measures of adaptive immunity (e.g., lymphocytes, IgG) that play key roles in the defense against viruses [303], weakened antiviral defenses could result in greater susceptibility to pathogens such as rabies. Determining if such findings reflect impaired adaptive immunity or reduced viral exposure in livestock-dense habitats, and how these patterns interact with demographic and behavioral processes, will be critical to anticipate how agricultural change will affect risks of pathogen spillover from vampire bats. More broadly, this work demonstrates that considering how food availability affects multiple host mechanisms can enhance our understanding of how population-level infection dynamics respond to landscape changes. Given the diversity of ways in which anthropogenic activities subsidize wildlife [4,5], ranging from urban gardens and agriculture to conservation programs and tourism [34,97,156,191], integrative and multi-scale approaches in other natural systems could enhance our ability to predict and manage emerging disease risks.

ETHICS

All bat sampling was conducted in accordance with accepted guidelines for humane wildlife research as outlined by the American Society of Mammalogists [304]. Field procedures were approved by the UGA Animal Care and Use Committee (AUP A2009-10003-0 and A2014 04-016-Y3-A5). Sample collection was authorized by the Belize Forest Department under permits CD/60/3/14(27), CD/60/3/15(21), and WL/1/1/16(17) and by the Peruvian Government under permits RD-273-2012-SERFOR-DGGSPFFS, RD-009-2015-SERFOR-DGGSPFFS, RD-264-

2015-SERFOR-DGGSPFFS, and RD-142-2015-SERFOR-DGGSPFFS. Access to genetic resources from Peru was granted under permit RD-054-2016-SERFOR-DGGSPFFS.

AUTHOR CONTRIBUTIONS

DJB conceived and designed the study, conducted field and labwork (stable isotope analyses, immunological assays, *Bartonella* PCR), analyzed data, and drafted the paper; GAC helped design the study, provided immunological protocols, and contributed to data analysis; DVV and VEC performed hemoplasma PCR and sequencing; ABB performed IgG assays and conducted fieldwork; JEC, MBF, SER and NBS facilitated field logistics and conducted fieldwork; MSC performed extracellular haemoparasite screening; KJN provided laboratory space and reagents for IgG assays and contributed to data analysis; ATG helped design the study and contributed to data analysis; SA helped design the study, secure funding, and contributed to data analysis; DGS helped design the study, secure funding, facilitated field logistics, and contributed to data analysis. All authors contributed to manuscript revisions and gave final approval for publication.

COMPETING INTERESTS

We have no competing interests.

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Table 6.1. Subset of candidate LMMs predicting bat immune component PC1 and including a random effect of bat ID. LMMs are ranked by ΔAICc with the Akaike weights (w_i) from the full model comparison, number of coefficients (k), and marginal and conditional r^2. Only LMMs within 2 ΔAICc are shown; the full 95% confidence set is listed in Table S3.

Immune function LMM	***k***	**ΔAICc**	w_i	r^2_m	r^2_c
PC1 ~ livestock biomass + reproduction	3	0.00	0.13	0.35	0.49
PC1 ~ livestock biomass + reproduction + isotope distance	4	0.11	0.12	0.35	0.51
PC1 ~ livestock biomass + reproduction * isotope distance	5	1.46	0.06	0.36	0.52
PC1 ~ livestock biomass + reproduction + age	4	1.87	0.05	0.35	0.49
PC1 ~ livestock biomass + reproduction + age + isotope distance	5	1.87	0.05	0.35	0.51

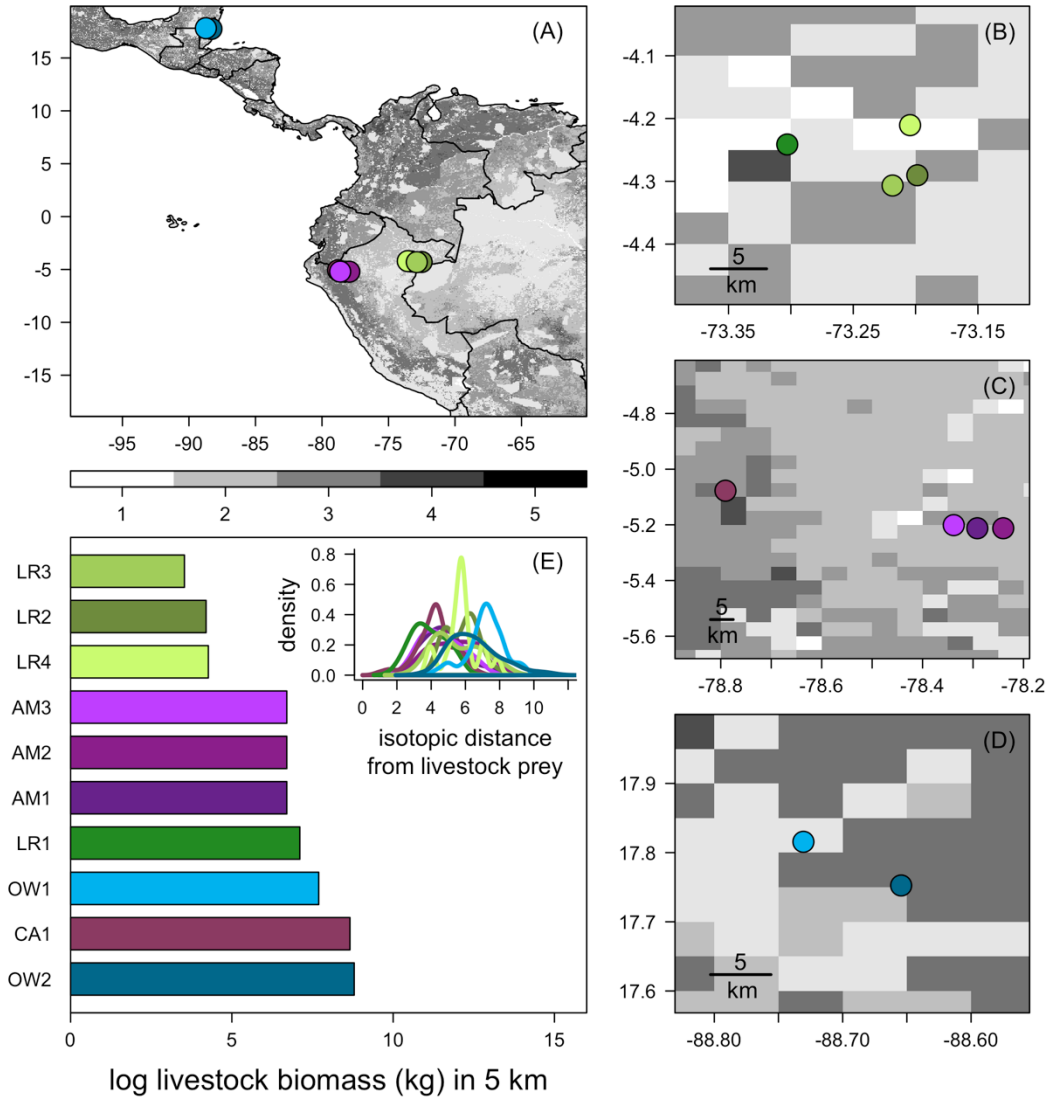

Figure 6.1. Vampire bat sampling sites in Peru and Belize (A), where shading and color strip represent the log biomass (kg) of cows, pigs, and chickens from the FAO and AnAge databases [255,256]. Fine-scale patterns in livestock biomass are shown in B (Loreto), C (Amazonas and Cajamarca), and D (Belize); site coordinates are jittered to reduce overlap. Log-transformed livestock biomass within 5 km of each capture location (E); the inset shows the distribution (kernel density) of individual bat isotopic distance from livestock prey per site. Colors correspond to sampling region (green=Loreto, purple=Amazonas and Cajamarca, blue=Belize).

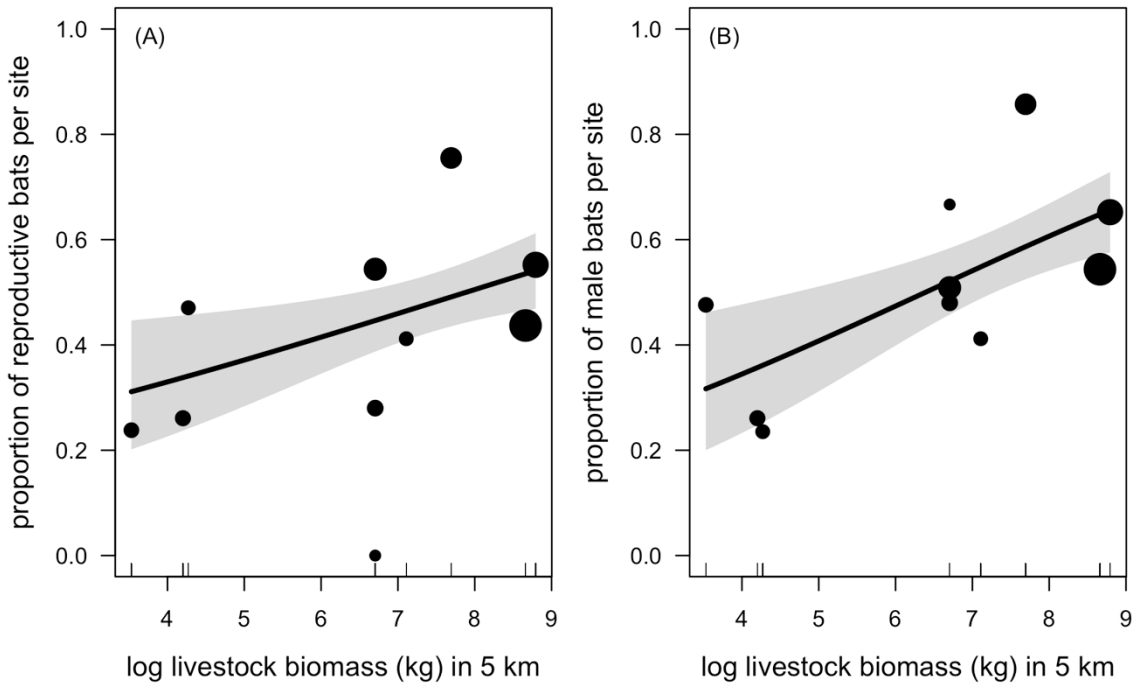

Figure 6.2. Relationships between livestock biomass and vampire bat demography. Livestock biomass predicts increases in the proportions of (A) reproductive and (B) male bats. Curves and grey shading display the fit and 95% confidence interval from GLMMs. Overlaid are the proportion of reproductive and male bats per site, with point size scaled by log sample size.

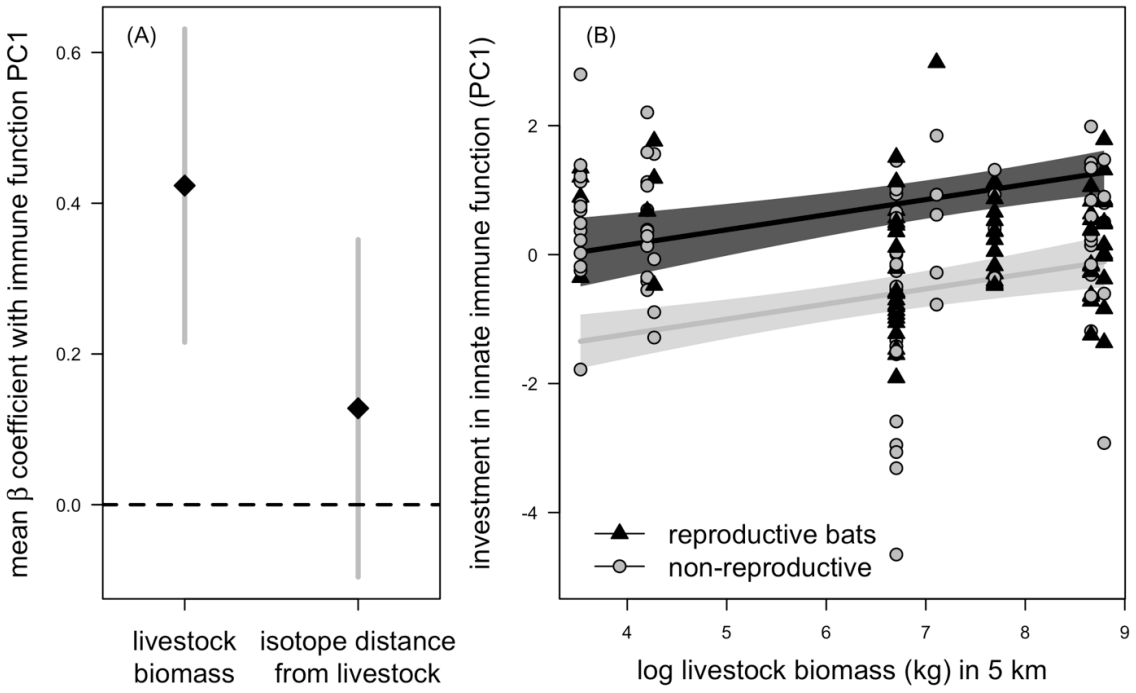

Figure 6.3. Relationships between provisioning covariates and bat immune profiles (PC1); PC1 loads positively with innate immunity and negatively with adaptive immunity. Model averaging results across the 95% confidence set of LMMs (A), with 95% confidence intervals for estimates shown in grey and mean coefficients shown in black diamonds. The dashed line represents no correlation between covariates and immune profiles (β=0). Results from the top LMM (B); points, model fit, and 95% confidence intervals are shaped and colored by bat reproduction.

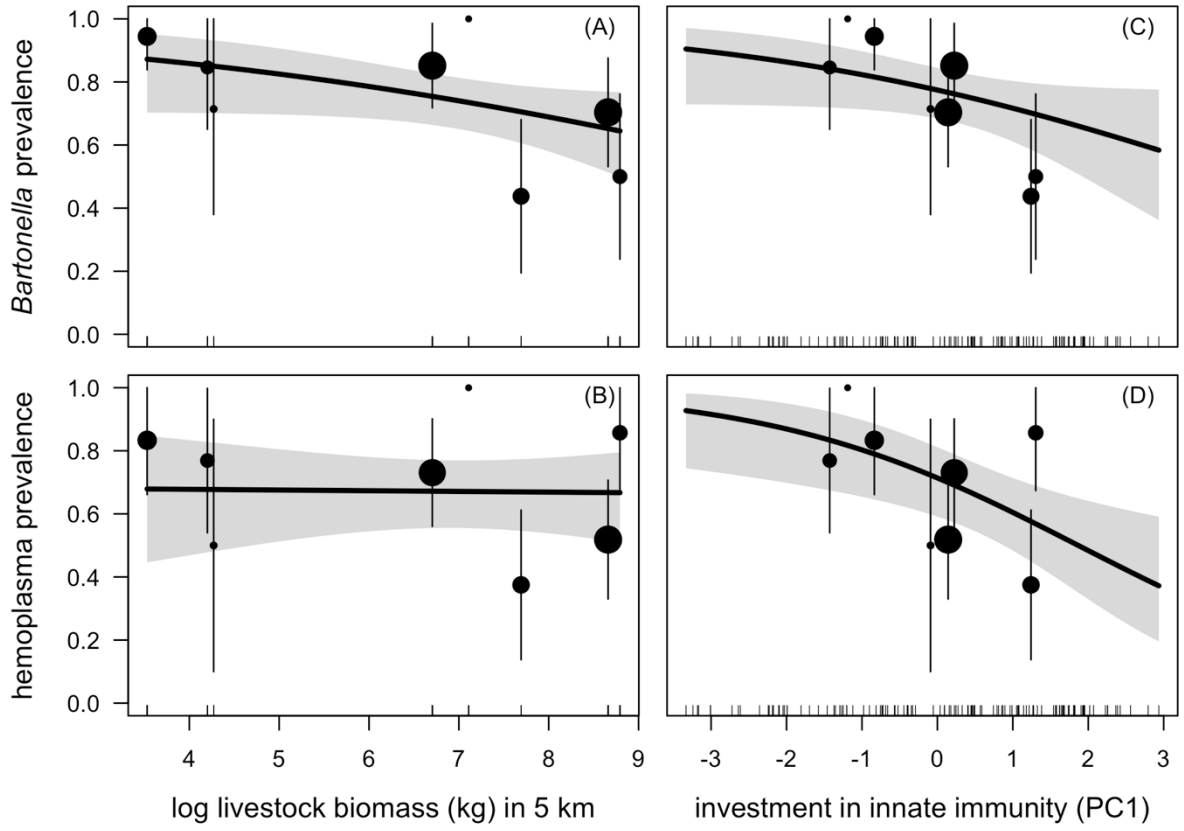

Figure 6.4. Relationships between provisioning, bat immunity, and bacterial infection. Modeled relationships between livestock biomass and individual infection with *Bartonella* (A) and hemoplasmas (B). Modeled relationships between the immune function PC1 and individual infection with *Bartonella* (C) and hemoplasmas (D) from the same GLMMs. Predictions are overlaid with prevalence and 95% confidence intervals per site obtained with the Wald method.

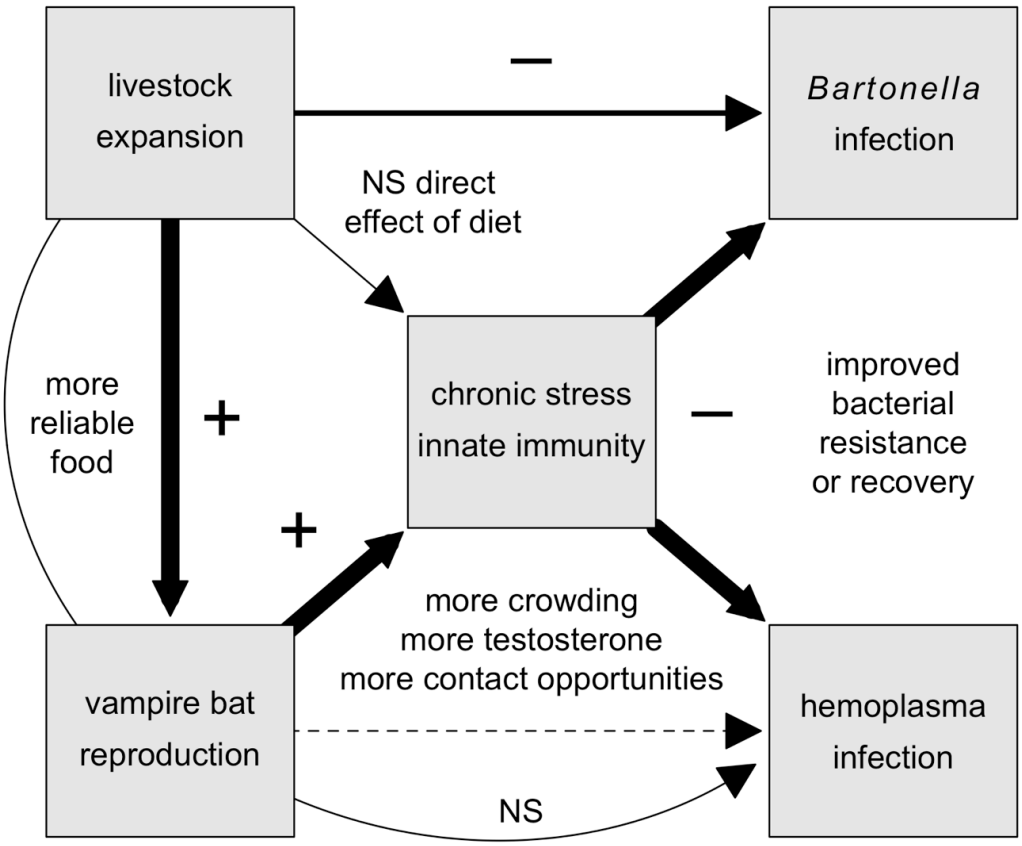

Figure 6.5. Hypothesized mechanisms affecting bacterial infection prevalence in vampire bats in relation to livestock expansion. Signs summarize observational relationships, arrow width displays the magnitude of the association, and dashed lines display unobserved mechanisms; NS=not significant.

CHAPTER 7

CONCLUSIONS AND FUTURE DIRECTIONS

The overarching goal of this dissertation was to develop and test conceptual and mechanistic frameworks for how resource provisioning affects infectious disease dynamics in wildlife. Given that resource provisioning can simultaneously affect multiple and potentially opposing processes important to epidemiological outcomes, such frameworks are needed to understand and predict the contexts where anthropogenic resources will increase or decrease pathogen transmission. Because foraging on supplemental resources can promote host aggregation and contact opportunities leading to cross-species transmission [20,34,36,62], this body of research represents an important applied contribution for considering when and where supplemental food might increase disease risks for wildlife species. This work also makes important contributions to the ecology of infectious disease, which increasingly considers how natural and anthropogenic changes in food availability affect infectious disease dynamics. However, the majority of such studies have been restricted to single host–parasite interactions and to experimental systems where the effects of resource availability or quality on host immunity are decoupled from effects of supplemental food on demography and behavior observed in natural systems [44,60,61,202]. Therefore, syntheses of empirical work across taxa alongside field and theoretical studies that integrate effects of food resources across biological scales of organization can provide insights into how the abundance and distribution of resource affect infection dynamics.

My dissertation was structured around three main goals: *(i)* developing epidemiological models that consider how different mechanisms respond to resource provisioning (Chapter 2)

and assessing empirical support for the resource-dependent processes (Chapter 3), *(ii)* extending these mechanistic frameworks to larger spatial scales to develop testable hypotheses for how the landscape-level deployment of supplemental food affects infection dynamics in metapopulations (Chapters 4 and 5), and *(iii)* examining how the abundance of anthropogenic resources interacts with host immunity, demography, and infection outcomes in a tractable and epidemiologically relevant natural host–pathogen system (Chapter 6). Across these goals, I integrated theory and empiricism to provide novel but ecologically grounded insights into how resource provisioning affects epidemiology and to better interpret complex field data.

In Chapter 2, I developed simple susceptible–infected–recovered (SIR) models that built supplemental resources into model parameters. This work showed that how host immune defense responds to resource provisioning determines if supplemental feeding increases or decreases pathogen invasion potential and prevalence [52], which motivates work to quantify the empirical work in Chapter 6. Unlike prior compartmental models that also consider resource dependence [61,62,73], this mechanistic framework was the first to show how increasing resource availability can decrease infection outcomes. While weak but positive effects of resource supplementation on host resistance maximized R_0 at intermediate and high resource levels, strong effects on immune defense drove pathogens to extinction with intermediate intensities of resource provisioning. This interaction between supplemental food, immune defense, and infection could accordingly explain empirical case studies where resource provisioning is associated with lower infection. However, given that this model is restricted to microparasite dynamics, an important future area for theory will be to consider how parameters controlling macroparasite dynamics respond to resource provisioning.

Chapter 3 synthesized empirical findings from the literature on how host–pathogen interactions respond to supplemental feeding to test support for the mechanisms proposed in Chapter 2 [33]. This comprehensive meta-analysis demonstrated strong support for higher contact rates in food-supplemented populations increasing infection; however, I found variable effects of resource provisioning on host condition and immune defense and weak to no effects on demography. Building these empirical findings back into the modeling frameworks produced in Chapter 2 provided mechanistic support for our more general finding that supplemental feeding causes greater infection, particularly for microparasites like viruses and bacteria, given that resource provisioning is associated with greater contact and weakened host resistance. Furthermore, this chapter provided evidence that in real-world systems, intentional feeding (e.g., supplemental feeding for recreation, tourism, management, and conservation) is associated with greater infection. This has led to additional work to better understand how these feeding contexts can negatively affect wildlife health and how feeding practices within these contexts can be improved [177]. Another research priority stemming from this work has been to better explain the broad variation in how infection responds to resource provisioning; the R^2 of only 20% suggests further work is needed to better identify when and where supplemental feeding will most increase infection risks. This has encouraged continued collection of data for an ongoing meta-analysis to identify host traits that might explain if certain groups of wildlife are more prone to greater infection risks across different parasite taxa to resource provisioning.

Work that addressed Goal 2 directly examined spatial heterogeneity in wildlife resources and how provisioning is distributed across landscapes. Key contributions of Chapters 4 and 5 were to build metapopulation models that accounted for the distribution of provisioned habitats and their impacts on host colonization and extinction rates. Chapter 4 can be considered an in-

depth exploration of model parameter space [55] to ask how the spatial extent of resource provisioning and impacts on patch persistence time influenced pathogen invasion and complete spatial spread (i.e., pandemic equilibrium [158]). Chapter 5 extended this framework to include effects of resource provisioning on the host colonization process, including the production of dispersers and their attraction to habitats with food supplementation. In contrast to Chapters 2 and 3, this work focused on assessing the degree to which increasing the relative frequency of provisioned habitats improves patch occupancy while minimizing infection prevalence. These models extend existing theory on pathogen dynamics in metapopulations [159,160,170,203], such as showing that host occupancy can be maximized and infection prevalence can be minimized if landscapes contain heterogeneous mixtures of unprovisioned and provisioned habitat patches. An important goal for future work will be to couple within-population models from Chapters 2 and 3 with metapopulation frameworks of Chapters 4 and 5, which will require spatially realistic metapopulations models and explicit modeling of within-patch dynamics [169]. Such models would treat infectious disease in response to resource provisioning as a complex system, which could produce novel theoretical predictions or insights into field data.

Chapter 6 used theoretical insights from Chapters 2, 3, 4, and 5 to guide interpretation of how resource provisioning affects multiple mechanisms important to epidemiology within the context of vampire bats and livestock expansion in Latin America. Results showed that (i) livestock-dense habitats likely enhance vampire bat reproduction and attract more individuals, (ii) bats in such habitats show higher inflammation and investment in innate immunity at the expense of adaptive immunity, and (iii) bats that show these innate-oriented immune profiles have lower odds of infection with both *Bartonella* and hemoplasmas. Yet despite both pathogens responding in a similar manner to differences in bat immune systems, only *Bartonella* showed a

negative relationship between resource provisioning and infection prevalence. As *Bartonella* is most likely a vector-borne pathogen in bats [246,247], transmission could be divorced from greater bat density and aggregation expected in provisioned habitats. These data therefore suggest that much of this relationship can be explained by livestock biomass enhancing bat antibacterial defenses, akin to the key theoretical result from Chapter 2. Given that infection with hemoplasmas did not respond to supplemental feeding, my theoretical chapters would suggest that transmission-enhancing processes such as greater bat density, attraction to provisioned sites, or higher contact rates from competition for roost space or prey are all plausible explanations.

Chapter 6 ultimately illustrates how theory can guide interpretations of complex field data when considering the myriad of mechanisms by which supplemental feeding can affect host–pathogen interactions. I hope that this work will encourage future field studies to take a similar approach; only a handful of publications assessed in Chapter 3 quantified multiple mechanisms, and none of these used insights from theory to aid their interpretations. Future work could be particularly useful for understanding how other bat–pathogen systems respond to resource provisioning, as many frugivorous and nectarivorous bat species capitalize on anthropogenic resources such as urban gardens, agricultural fields, and backyard fruit trees [17,34,35,62,191,305,306]; however, surprisingly little empirical work has addressed how these unintentional supplemental feeding activities correlate with infection status despite the clear risks for emerging disease risks for humans, domestic animals, and wildlife. Within the vampire bat system itself, Chapter 6 opens the door for several important research directions. Vampire bats are the primary hosts of rabies virus throughout Latin America [253,307,308], yet it remains unclear how ongoing livestock intensification will affect rabies transmission dynamics and spillover risk [20,103,309]. Given that vampire bats in livestock-dense habitats presented with

lower measures of adaptive immunity that can play key roles in the defense against viruses [303,310], weakened antiviral defenses could result in greater susceptibility to rabies virus [311]. An outstanding direction for future work in this system is to examine how livestock density affects rabies-specific antiviral function and between-patch movement patterns. This ongoing work will integrate the within-patch and metapopulation modeling approaches produced by this dissertation and will help predict how agricultural expansion will affect future rabies risks.

Collectively, this dissertation provides theoretically informed and testable predictions for how anthropogenic food resources affect host–pathogen interactions and demonstrates that considering how food availability affects multiple underlying host mechanisms can enhance our understanding of how population-level infection dynamics respond to landscape changes. Given the diversity of ways in which anthropogenic activities subsidize wildlife, these mechanistic frameworks and multi-scale approaches within the context of natural systems are important to inform and enhance our ability to predict and manage emerging disease risks from provisioning. Regarding applied significance, these findings suggest that supplemental feeding and foraging on anthropogenic resources can most benefit wildlife and limit spillover to humans and domestic animals when food resources improve resistance to infection (e.g., by providing appropriate nutrition), do not facilitate large degrees of host dispersal (i.e., limiting immigration and mixing of populations), are deployed in a limited manner across a landscape (i.e., to prevent facilitating spatial spread of infection), and do not enhance contact rates (e.g., vector-borne pathogens).

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APPENDIX A

CHAPTER 2 SUPPLEMENTAL INFORMATION

AS1 Derivation of pathogen invasion threshold (R_0) and equilibrium prevalence under resource provisioning

For both the SIS and SIR models with a density-dependent host growth rate, the basic reproductive number of a pathogen (R_0) is the product of the disease-free equilibrium host population size (i.e., host carrying capacity), the transmission rate, and the infectious period (the inverse of the rate at which infected individuals leave the infectious class). A derivation of this result can be found in [312].For our system, this expression is:

$$R_0 = \frac{\alpha\delta}{\mu+\upsilon+\gamma}\left(\frac{b_0-\mu}{b_1}\right) \quad \text{(S1)}$$

Under resource provisioning, the host contact rate (α), density-independent per capita birth rate (b_0), and rate of recovery from infection (γ) are increasing functions of ρ, the provisioning parameter. For parameter x, we choose the functional form

$$x = x_{max} - (x_{max} - x_{min})e^{-\theta_x\rho}. \quad \text{(S2)}$$

In contrast, the probability of transmission given contact (δ) and natural host mortality (μ) are decreasing functions of provisioning, described by the functional form

$$x = x_{min} + (x_{max} - x_{min})e^{-\theta_x\rho}. \quad \text{(S3)}$$

For each of these parameters ($x = \alpha, b_0, \gamma, \delta, \mu$), the extreme values of the parameter (x_{min} or x_{max}) and the strength of the response to provisioning (θ_x) are specific to each

parameter and listed in Table S1. The provisioning-dependent expression for R_0 is attained by substituting expressions (S2) and (S3) for each provisioning-dependent parameter into (S1).

The SIS model is

$$dS/dt = \big(b_0 - b_1(S+I)\big)(S+I) - \mu S - \alpha\delta SI + \gamma I$$
$$dI/dt = \alpha\delta SI - (\mu + \nu + \gamma)I \,. \qquad \text{(S4)}$$

To calculate the equilibrium pathogen prevalence, we set each of the equations in (S4) equal to zero. From the second equation, the equilibrium number of susceptibles is

$$S^* = \frac{\mu+\nu+\gamma}{\alpha\delta} \,. \qquad \text{(S5)}$$

Substituting this into the expression $dS/dt = 0$ and rearranging yields a quadratic expression for the equilibrium infected density of the form $AI^2 + BI + C = 0$, where

$$A = b_1$$
$$B = (2b_1 + \alpha\delta)S^* - b_0 - \gamma$$
$$C = -(b_0 - b_1 S^*)S^* + \mu S^* . \qquad \text{(S6)}$$

This quadratic has two roots:

$$I^* = \frac{-B \pm \sqrt{B^2 - 4AC}}{2A} . \quad \text{(S7)}$$

Clearly $A > 0$, and since $-C$ is the population growth rate of susceptibles, $C < 0$ whenever S is less than the carrying capacity, $K = (b_0 - \mu)/b_1$. This means that $-4AC > 0$, so therefore $\sqrt{B^2 - 4AC} > |B|$. For $I^* > 0$, the top half of the fraction must be positive, so the root we require is

$$I^* = \frac{-B + \sqrt{B^2 - 4AC}}{2A} . \quad \text{(S8)}$$

The expression for equilibrium pathogen prevalence is too complex to write out in full, but is calculated by substituting the provisioning-dependent functional forms (S2) and (S3) into our demographic, contact, and immune defense parameters; substituting these into (S5), (S6), and (S8); and calculating the prevalence as

$$p^* = \frac{I^*}{S^* + I^*} . \quad \text{(S9)}$$

The SIR model takes the form

$$\begin{aligned} dS/dt &= \big(b_0 - b_1(S + I + R)\big)(S + I + R) - \mu S - \alpha\delta SI \\ dI/dt &= \alpha\delta SI - (\mu + \nu + \gamma)I \\ dR/dt &= \gamma I - \mu R . \end{aligned} \quad \text{(S10)}$$

Solving this model for equilibrium yields the same expression (S5) for S^*, and by setting $dR/dt = 0$, the equilibrium number of recovered individuals can be expressed simply in terms of I^* as

$$R^* = \frac{\gamma}{\mu} I^* . \tag{S11}$$

By substituting expressions (S5) and (S11) into the $dS/dt = 0$ equation, we again derive a quadratic equation for I^*, where now the coefficients are

$$\begin{aligned} A &= b_1 \left(1 + \frac{\gamma}{\mu}\right)^2 > 0 \\ B &= \left(2b_1 \left(1 + \frac{\gamma}{\mu}\right) + \alpha\delta\right) S^* - b_0 \left(1 + \frac{\gamma}{\mu}\right) \\ C &= -(b_0 - b_1 S^*) S^* + \mu S^* < 0 . \end{aligned} \tag{S12}$$

By the same arguments as before, since $\sqrt{B^2 - 4AC} > |B|$, we need the positive root of (S7) to obtain the equilibrium number of infected individuals:

$$I^* = \frac{-B + \sqrt{B^2 - 4AC}}{2A} . \tag{S13}$$

As before, to calculate infection prevalence for the SIR model, we substitute our provisioning-dependent parameters into expressions (S5), (S12), (S13), and (S11), and substitute these into the expression

$$p^* = \frac{I^*}{S^*+I^*+R^*}\,. \tag{S14}$$

AS2 Model parameterization

Our model was parameterized from published data on feral cats (*Felis catus*) infected with feline leukemia virus (FeLV), a retrovirus transmitted primarily during aggressive encounters and sharing of food resources [74]. The majority of infected hosts mount a successful immune response and clear infection, while those that fail to develop immunity become viremic until death, a period that lasts an average of two years [313,314].

Large differences in feral cat population size and density between rural and urban habitats are suggested to be a consequence of provisioning, which in urban environments takes the form of supplemental feeding stations and widespread resources such as household refuse [100,315]. Provisioning can increase feral cat reproductive output and decrease natural mortality [316,317], and thus we assumed a positive relationship between provisioning (ρ) and density-dependent birth (b_0) and a negative relationship between ρ and natural mortality (μ). For simplicity, and since demographic rates are often coupled [318], the strength of the saturating response of birth and mortality rates to provisioning was assumed to be equal ($\theta_{b_0} = \theta_{\mu} = \theta_{b_0\mu} = 4$). Parameter estimates were provided from comparative data on feral cats in urban and rural environments and experiencing different levels of supplemental feeding [100,315,319].

As smaller home ranges and group living dynamics in feral cats have been associated with supplemental feeding and abundant anthropogenic resources [100], we assumed a positive relationship between ρ and encounter rates (α). Because reductions in home range and associated aggregation around provisioned resources increase host contact [27,67], we also assumed a non-saturating, continuous increase in encounter rates with increases in resources ($\theta_{\alpha} = 1$).

Food deprivation of cats decreases the total number and proliferation capacity of lymphocytes [320], indicators of immunosuppression and components of cellular immune

defense against FeLV [321]. Provisioning likely buffers against this immunosuppression, as provisioned feral cats display greater measures of body condition and lower levels of physiological stress [316]. Thus we assumed that improved immune function from reduced starvation stress produces a negative relationship between ρ and the susceptibility and infectivity (δ) as well as the disease-induced mortality rate (ν). To investigate the role of provisioning-altered immune defense on disease dynamics, we co-varied the strength of the responses of susceptibility and tolerance to provisioning ($\theta_\delta = \theta_\nu = \theta_{\delta\nu} = 0\text{–}8$). Parameter estimates for δ were derived from laboratory studies of FeLV susceptibility, where experimental increases in stress hormones resulted in 80% of challenged animals developing disease compared to only 10% of controls [322]. For ν, we assumed that provisioning would allow infected cats to survive as long as uninfected counterparts ($\nu_{min} = \mu_{min}$). Lastly, the recovery rate (γ) was taken from estimates of the FeLV infectious period, which on average spans three weeks [315,321].

Mounting a successful immune response is energetically expensive [323,324]. Therefore, successful pathogen clearance often comes at a cost to fitness through reduced fecundity [325,326], which may be alleviated through provisioning [231]. This effect would manifest in unprovisioned recovered cats having lower birth rates (b_0) than provisioned individuals, resulting in a larger carrying capacity (K) for provisioned cats. This reduction in fitness costs of pathogen resistance has a similar qualitative effect on model dynamics and pathogen invasion as our assumptions that birth rates (b_0) are a positive function of provisioning and mortality (μ) is a negative function of provisioning; namely, both of these mechanisms result in an increased K in provisioned wildlife.

The transmission rate ($\beta = \alpha\delta$) in prior studies of FeLV has been estimated from the duration between contact and development of infection in experimental studies [315,327];

however, high artificial aggregation of hosts in such environments (e.g., catteries) can inflate estimates of R_0. We thus scaled β to reflect prior R_0 estimates (1.13) through equation S1. Given baseline estimates of δ from cats experiencing physiological stress [322], we defined α_{min} and α_{max} as β/δ_{max} and $5(\alpha_{min})$, respectively, with α_{max} reflecting the five-fold difference in feral cat densities observed in rural versus provisioned and urbanized populations [315].

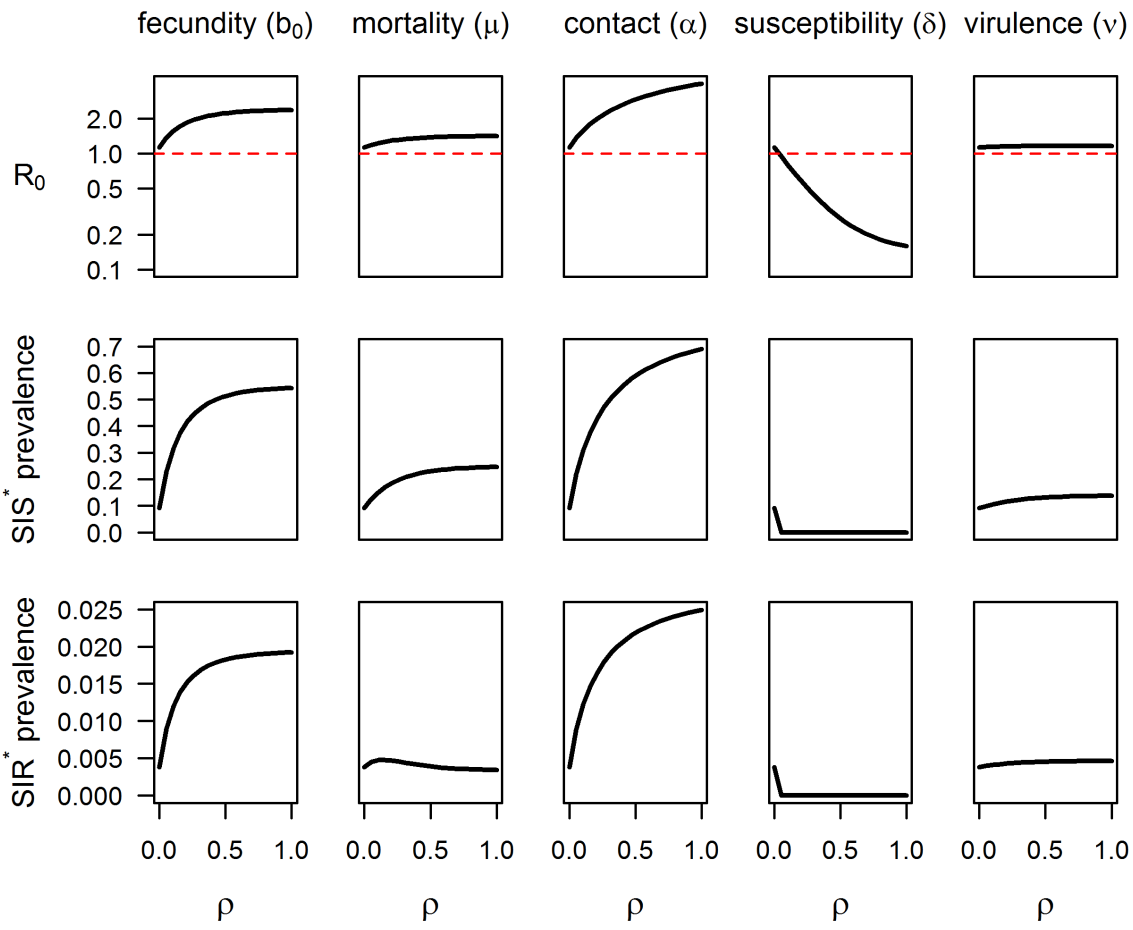
Sources of all baseline and provisioning-altered parameter estimates are provided in Table S2.1. Given baseline unsupplemented parameter estimated, the SIR and SIS models produced equilibrium prevalence values of <1% and 9%, respectively, within the range of FeLV prevalence (0–15%) observed across urban environments [315,328]

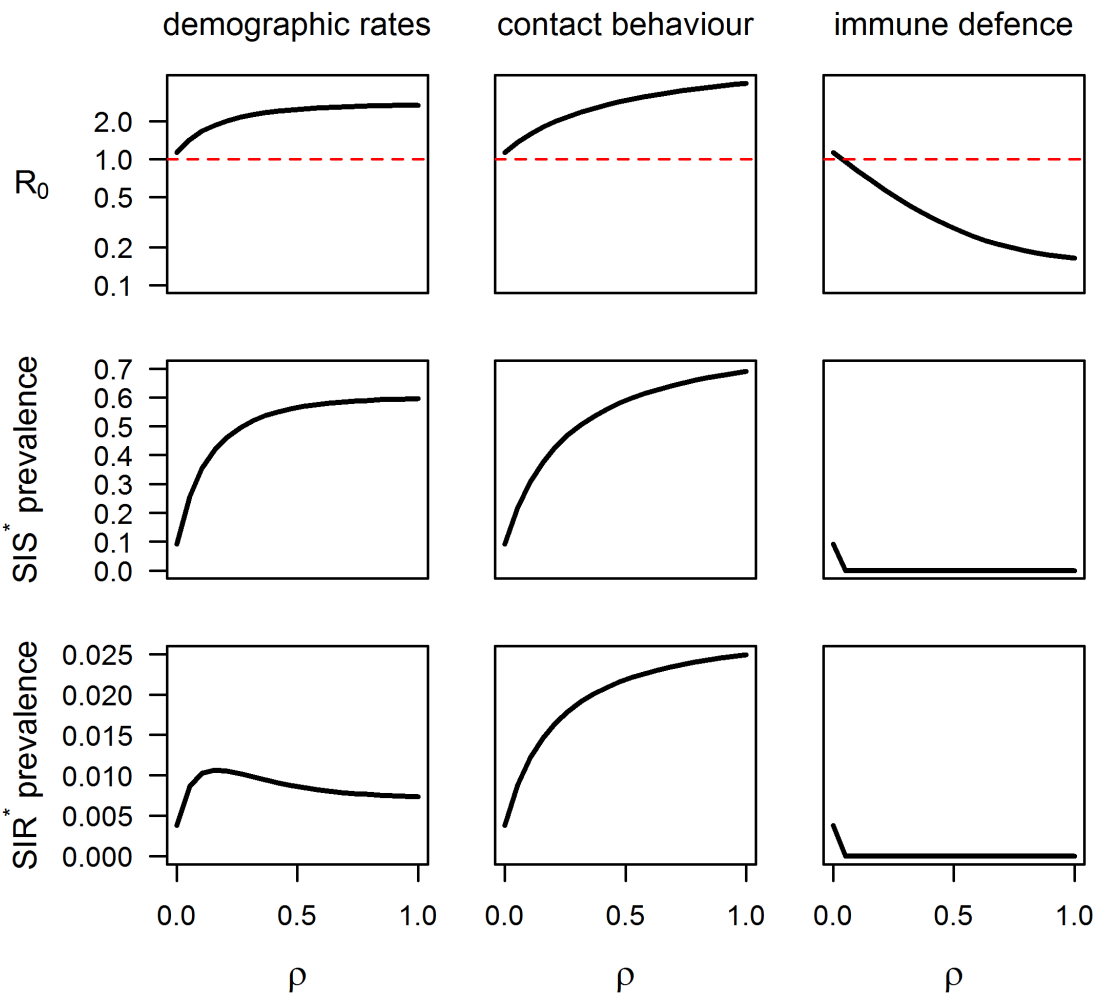
Table S2.1. Parameter estimates and sources for provisioned and unprovisioned values. Asterisks denote baseline unprovisioned estimates.

Parameter	x	Effect of ρ	x_{min}	x_{max}	θ_x	Source
Birth rate (density dependent)	b_0	+	2.4 *	4.4	4	[315,319,319]
Birth rate (density independent)	b_1	NA	0.006	0.006	NA	[315]
Natural mortality rate	μ	–	0.2	0.6 *	4	[315,329]
Encounter rate	α	+	0.087 *	0.436	1	[315,322]
Disease mortality rate	ν	–	0	0.5 *	0–8	[315]
Recovery rate	γ	NA	17.38	17.38	NA	[315,321]
Infection probability	δ	–	0.8	0.1 *	0–8	[322]

AS3 Independent contributions of provisioning-altered demography, contact behavior, and immune defense to disease outcomes

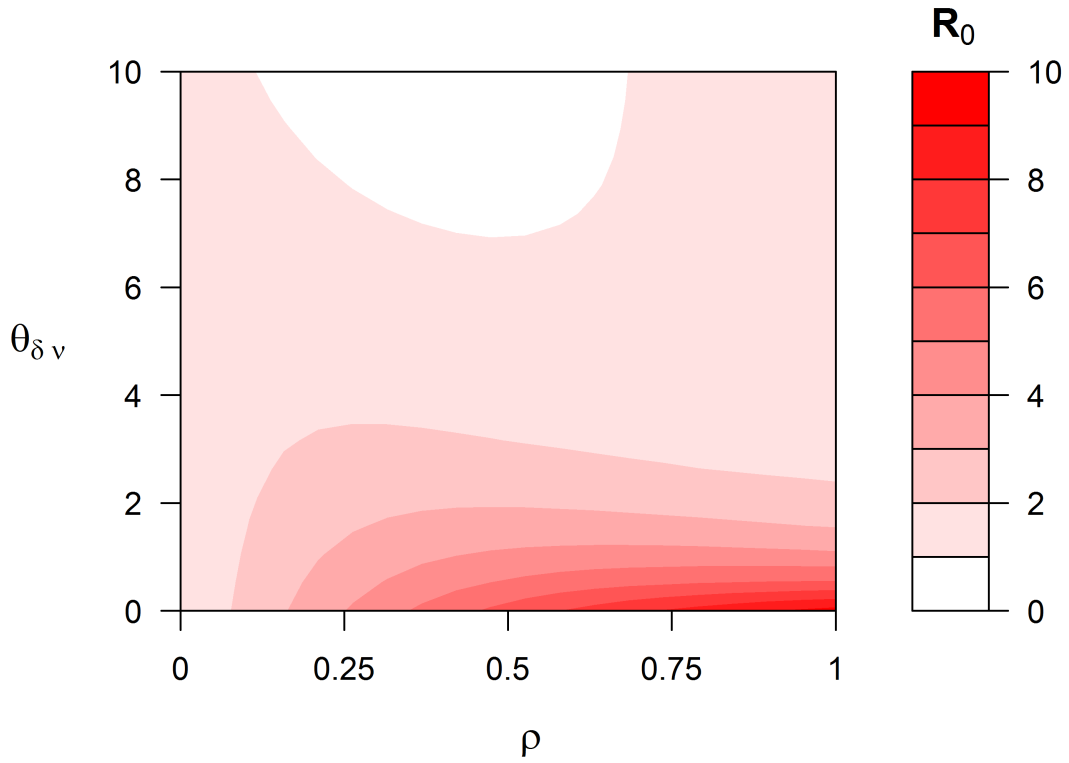
To demonstrate the influence of individual parameters and grouped mechanisms on pathogen invasion capacity and long-term infection burdens, we present model results in which parameters describing demographic rates (b_0 and μ), contact behavior (α), and immune defense (δ and ν) are altered by provisioning in isolation. We performed simulations of the SIS and SIR model in which provisioning affected each parameter and mechanism separately, holding values for remaining parameters at their baseline values. We then plotted outputs of these simulations (R_0, equilibrium prevalence) against the provisioning parameter (ρ) for each parameter and coupled mechanism. All model simulations were conducted in R version 3.0.2 [119].




demographic rates
contact behaviour
immune defence
R0
2.0
1.0
0.5
0.2
0.1
SIS* prevalence
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
SIR* prevalence
0.025
0.020
0.015
0.010
0.005
0.000
0.0
0.5
1.0
ρ

AS4 Pathogen invasion capacity as a function of the strength of immune response to provisioning ($\theta_{\delta v}$)

To reinforce the importance of understanding how provisioning affects host immune defense, we present outcomes of provisioning on the pathogen basic reproductive number, R_0, by varying the strength of the immune response to provisioning across a range of $\theta_{\delta v}$, from no effect of supplemental feeding on immunity to a rapidly saturating response. R_0 was maximized when provisioning has little effect on immune defense, but the pathogen was still able to invade the host population at high levels of ρ regardless of the effect on immune defence, as contact and demographic rates were still influenced by provisioning ($\theta_{b_0\mu} = 4$ and $\theta_{\alpha} = 1$; see Table S2.1 above). Thus the pathogen was least likely to invade at intermediate levels of provisioning and when effects on immune defense are quickly saturating.



APPENDIX B

CHAPTER 3 SUPPLEMENTAL INFORMATION

BS1 Data collection procedure

We here present additional detail on the collection and distribution of data included in our meta-analysis of the relationship between resource provisioning and infection outcomes in wildlife. Systematic searches were performed in Web of Science, Google Scholar, CAB Abstracts, and PubMed. Because the search interfaces are similar, we used the same strings of search terms in Web of Science, CAB Abstracts, and PubMed.

("anthropogenic food" OR "anthropogenic resource" OR provisioning OR provisioned OR "supplemental feed" OR "supplemental resource" OR "resource supplementation" OR "resource subsidies" OR "anthropogenic subsidies" OR "human-provided subsidies" OR "human-provided resource" OR "host resource")
AND
(disease* OR infect* OR pathogen* OR bacteria* OR parasite* OR virus* OR helminth* OR protozoa* OR epidemic* OR transmission*)
AND (wildlife OR wild)

The Web of Science search was restricted by research domain (SCIENCE TECHNOLOGY) and by research area (ENVIRONMENTAL SCIENCES ECOLOGY OR BIODIVERSITY CONSERVATION OR PATHOLOGY OR ZOOLOGY OR EVOLUTIONARY BIOLOGY OR IMMUNOLOGY OR HEMATOLOGY OR INFECTIOUS DISEASES OR MICROBIOLOGY OR VETERINARY SCIENCES OR PARASITOLOGY OR VIROLOGY).

Similarly, our Google Scholar search contained the following search string.

("anthropogenic food" OR provisioning OR "supplemental feed*" OR "supplemental resource*" OR "resource subsidies*" OR supplementation) AND (disease* OR infect* OR pathogen* OR transm*) AND wildlife

We restricted searches from 2014 back to 1990, when the last comprehensive review of wildlife supplementation experiments was published [84].

Together these searches resulted in 2216 records. We then followed the PRIMSA documentation procedure to record the results and exclusion process for the systematic search [330]. Each title and abstract was first screened for duplication and basic criteria for inclusion in our analysis of wildlife provisioning. The majority of records were initially removed as they consisted of human studies, toxicological studies, review papers, theoretical studies, and antibiotic and probiotic supplement experiments. Furthermore, studies were retained during this phase only if they examined effects of provisioning in wild or free-ranging populations. Together these screening criteria narrowed our search to 231 articles that were assessed for eligibility in the full-text.

This in-depth process excluded an additional 87 studies owing either to the above criteria or to a lack of comparison between provisioned and unprovisioned groups. Therefore we have included 144 studies in our overall qualitative synthesis, of which 23 (16%) recorded measures of disease and are included in the meta-analysis. The complete inclusion process is documented below in flow diagram form (Figure S3.1).

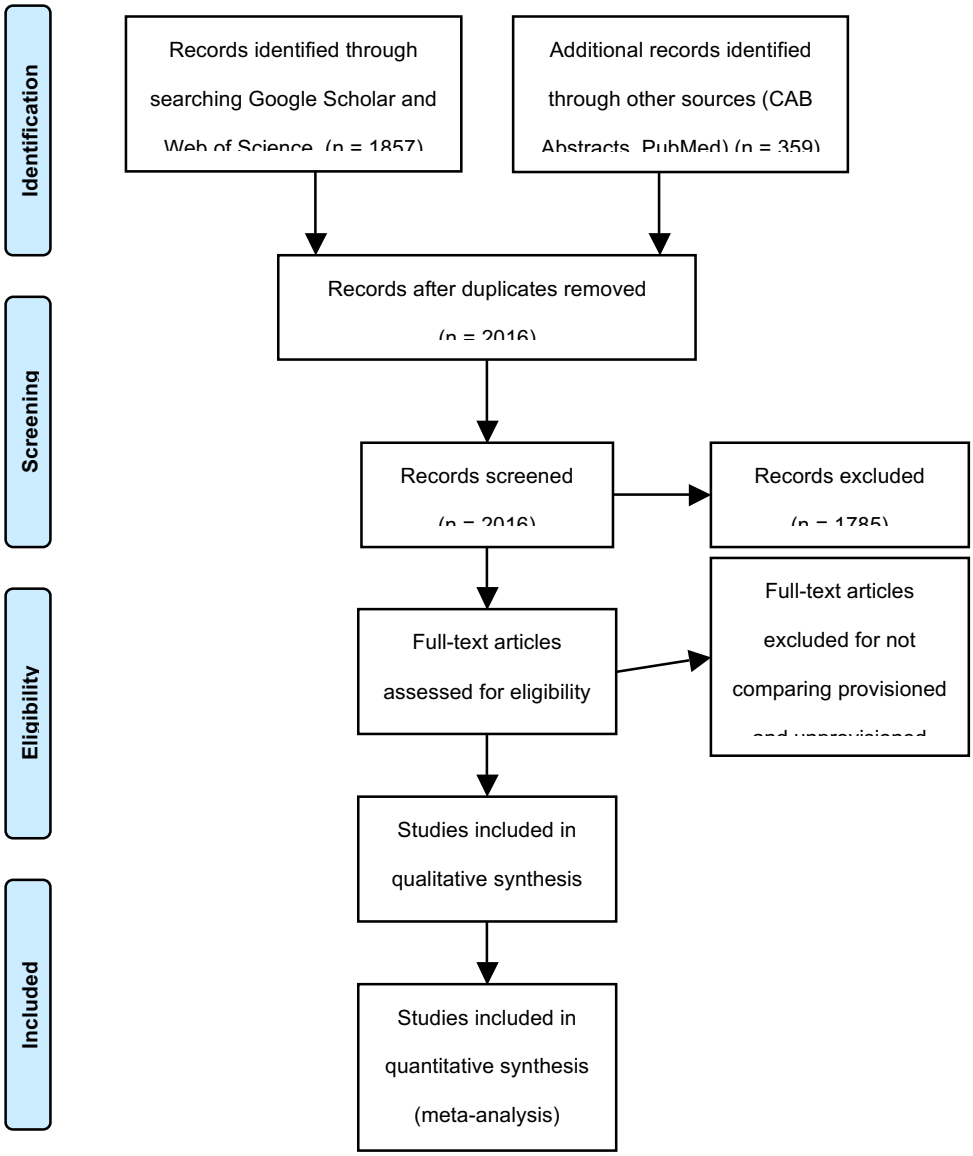


Figure S3.1. PRIMSA diagram documenting the data collection and inclusion process.

For each line of data in the 23 studies documenting infection differences between provisioned and unprovisioned wildlife, we recorded host and pathogen type, pathogen transmission mode, if provisioning was accidental or intentional, and the reported source of provisioning. Host type was recorded as phylogenetic class, and pathogen type was classified as bacterium, virus, fungus, protozoan, ectoparasite, or helminth. We determined transmission mode by matching our parasite species or genus records to those of the Global Mammal Parasite Database (GMPD), the most comprehensive collection of published records of parasitic organisms from free-living mammals [331]. Transmission mode was defined using the GMPD classifications of close contact, non-close contact, vector-borne, and complex life cycle (following Pedersen et al. 2005). However, as 21% (n = 28) of our data were from non-mammals (23 lines Aves, 2 lines Chondrichthyes, 1 line Reptilia), we verified transmission mode from a random sample (n = 9 lines, 30%) of these other host–pathogen interactions using [333]. When transmission mode for a particular pathogen species was not included in this reference, we matched transmission to that reported for the genus or family. If transmission mode was not listed at this level, we matched GMPD classifications to the mode of transmission listed in the study (e.g., Aegidae parasites; [124]. We found 100% identify between the GMPD and these references for our sample.

For each study, we also recorded the reported source of provisioned resources, and the majority of studies directly included provisioning as categorical (e.g., provisioned habitat type or population) or continuous (e.g., quantity of supplemental feed) variables. The remaining studies indirectly incorporated provisioning through explicit mention of anthropogenic resources in habitat type (e.g., urban environments having more abundant food compared to rural counterparts; [43,334] or through proximal measures (e.g., urbanization score as an indirect measure of bird feeder abundance; [71]. We then classified provisioning sources into categories

of agriculture (crop fields), urban waste (landfills, household trash), management (feeding grounds, bait piles), and recreation (tourism, bird feeders). Studies directly including provisioning represented 83% (n = 19) of our dataset, while proximal measures were used in 17% (n = 4) of included studies (Table S3.1).

Table S3.1: Mode of inclusion of anthropogenic resources in studies of host–pathogen interactions and classification of provisioning type and wildlife food source.

study	resource inclusion	type of provisioning	reported source	category of resource
Aponte *et al.* 2014	direct	accidental	urban waste	urban waste
Blanco *et al.* 2011	direct	intentional	management	management
Bradley *et al.* 2008	proxy	intentional	bird feeders	recreation
Cypher & Frost 1999	proxy	accidental	urban waste	urban waste
Hegglin *et al.* 2007	direct	accidental	urban waste	urban waste
Hill *et al.* 2008	proxy	accidental	urban waste	urban waste
Hines *et al.* 2007	direct	intentional	feed grounds	management
Knapp *et al.* 2013	direct	intentional	tourism	recreation
Lane *et al.* 2011	direct	intentional	tourism	recreation
Lonsdorf *et al.* 2011	direct	intentional	banana feeding	management
Luong *et al.* 2014	direct	intentional	bait piles	management
Miller *et al.* 2003	direct	intentional	feed grounds	management
Monello & Gompper 2010	direct	Intentional	bait piles	management
Monello & Gompper 2011	direct	intentional	bait piles	management
Navarro-Gonzalez *et al.* 2013	direct	intentional	hunting estate	management
Page *et al.* 2008	direct	accidental	urban waste	urban waste
Robardet *et al.* 2008	direct	accidental	urban waste	urban waste
Semeniuk & Rothley 2008	direct	intentional	tourism	recreation
Vicente *et al.* 2007a	direct	intentional	feed grounds	management

Vicente *et al.* 2007b	direct	intentional	feed grounds	management
Wenz-Mucke *et al.* 2013	direct	accidental	picnic raiding	recreation
Wright & Gompper 2005	direct	intentional	bait piles	management
Zylberberg *et al.* 2013	proxy	accidental	crop fields	agriculture

BS2 Descriptive meta-analysis

Although the study of provisioning impacts on wildlife populations has increased over time ($F_{1,18}$ = 28.14, p < 0.001), such a trend has not followed in regards to the proportion of studies quantifying disease outcomes ($F_{1,18}$= 3.26, p = 0.09). Out of 144 studies of provisioning identified since 1990, only 23 quantified infection outcomes between provisioned and unprovisioned populations (Figure S3.2a). For studies quantifying disease, we found a strong tendency toward studying mammals, which constituted 79% (n = 104) of our dataset (Figure S3.2b). Protozoa and helminths were the most common pathogens studied (69%, n = 91), with the remaining host–pathogen interactions represented by viruses (12%, n = 16), bacteria (10%, n = 13), ectoparasites (8%, n = 11), and fungi (1%, n = 1). Most studies (65%, n = 15) observed relationships between intentional forms of provisioning (management practices, recreation) and disease outcomes, with 8 studies examining accidental provisioning (Figure S3.2c).

In terms of quantifying potential mechanisms driving effects of provisioning on disease, 52% (n = 12) of studies quantified host condition or immune defense (Figure S3.3a). Of the measures of condition or immune function quantified, calculation of body condition was the most common variable recorded. 43% (n = 10) of studies quantified a behavioral response of hosts, in which aggregated group size around anthropogenic resources and measures of dietary breadth were the most common measures. Additionally, 26% (n = 6) of studies in our dataset quantified demographic factors in response to provisioning, with host density and population size being measured. When recording the response of these measures to provisioning, we observed a roughly even division between condition and immune measures increasing, decreasing, or showing no effect (Figure S3.3b). In contrast measures of behavioral change only showed

positive or negative effects, with positive denoting increases in aggregation or contact while negative denotes simplification of host diet. Demographic variables tended to either increase or not be affected by anthropogenic resources (Figure S3.3b).

Our analysis of this dataset used random (REM) and mixed-effects (MEM) models to infer the average effect of provisioning. The REM estimates the average true effect (μ) and total heterogeneity among true effects (τ^2), while the MEM includes study-level variables to explain variation in outcomes [335]. We estimated μ and τ^2 using maximum likelihood with sample size incorporated into the calculation of weights [336] and test against the null hypotheses of μ=0 and τ^2=0 using the Wald and Cochran's Q test, respectively [337]. We then used MEM to explain variation in infection according to covariates related to pathogen traits and host foraging ecology. We identified the strongest predictors of infection through a model simplification procedure in which the full MEM included pathogen type, transmission mode, host taxonomy, and provisioning type and source. Model simplification used backward removal of the least significant variable using Wald-type chi-square tests followed by nested likelihood ratio tests [116].

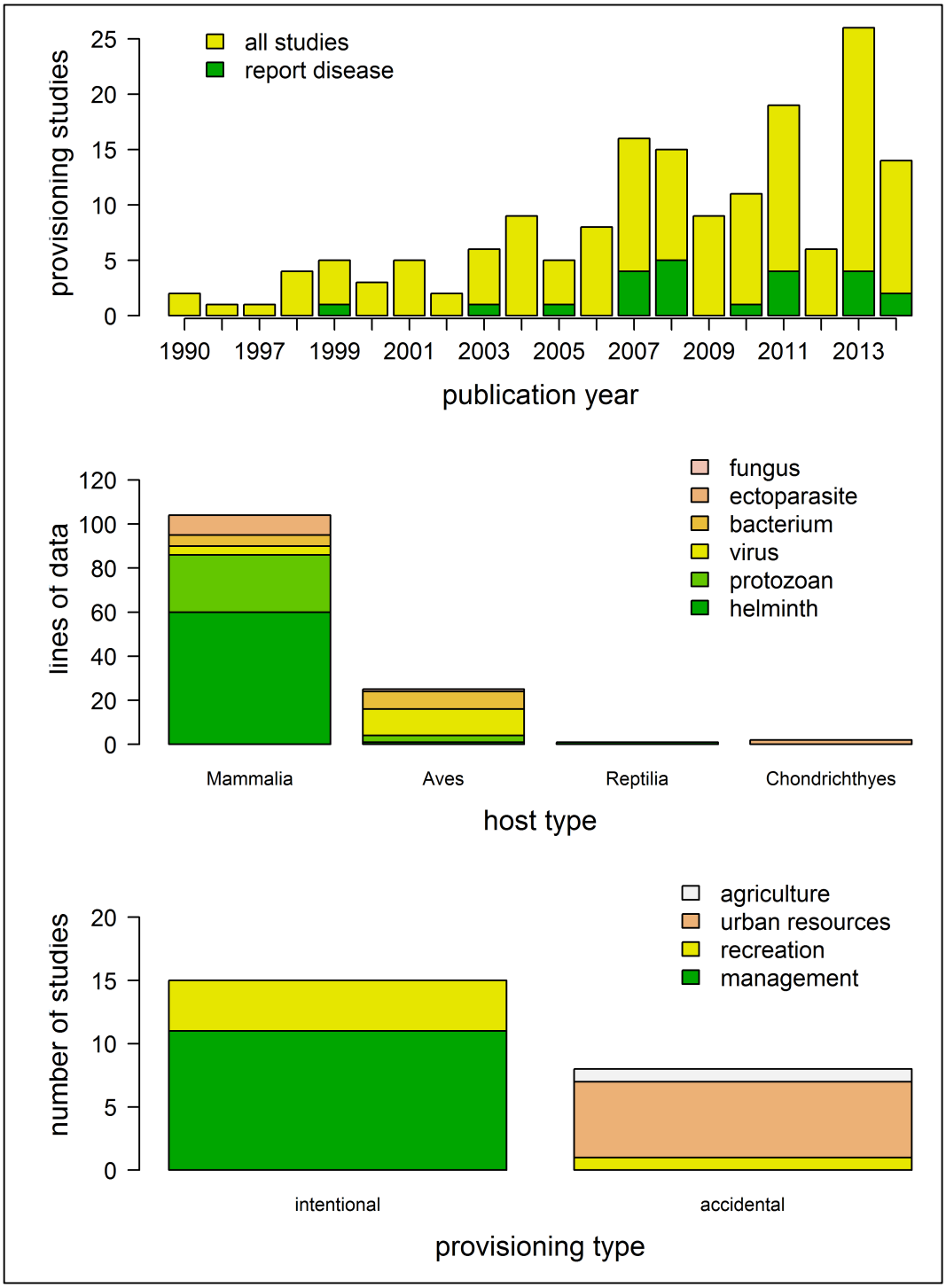


Figure S3.2. Distribution of meta-analysis data by (a) publication year, (b) host and pathogen type, and (c) provisioning intentionality and anthropogenic food source.

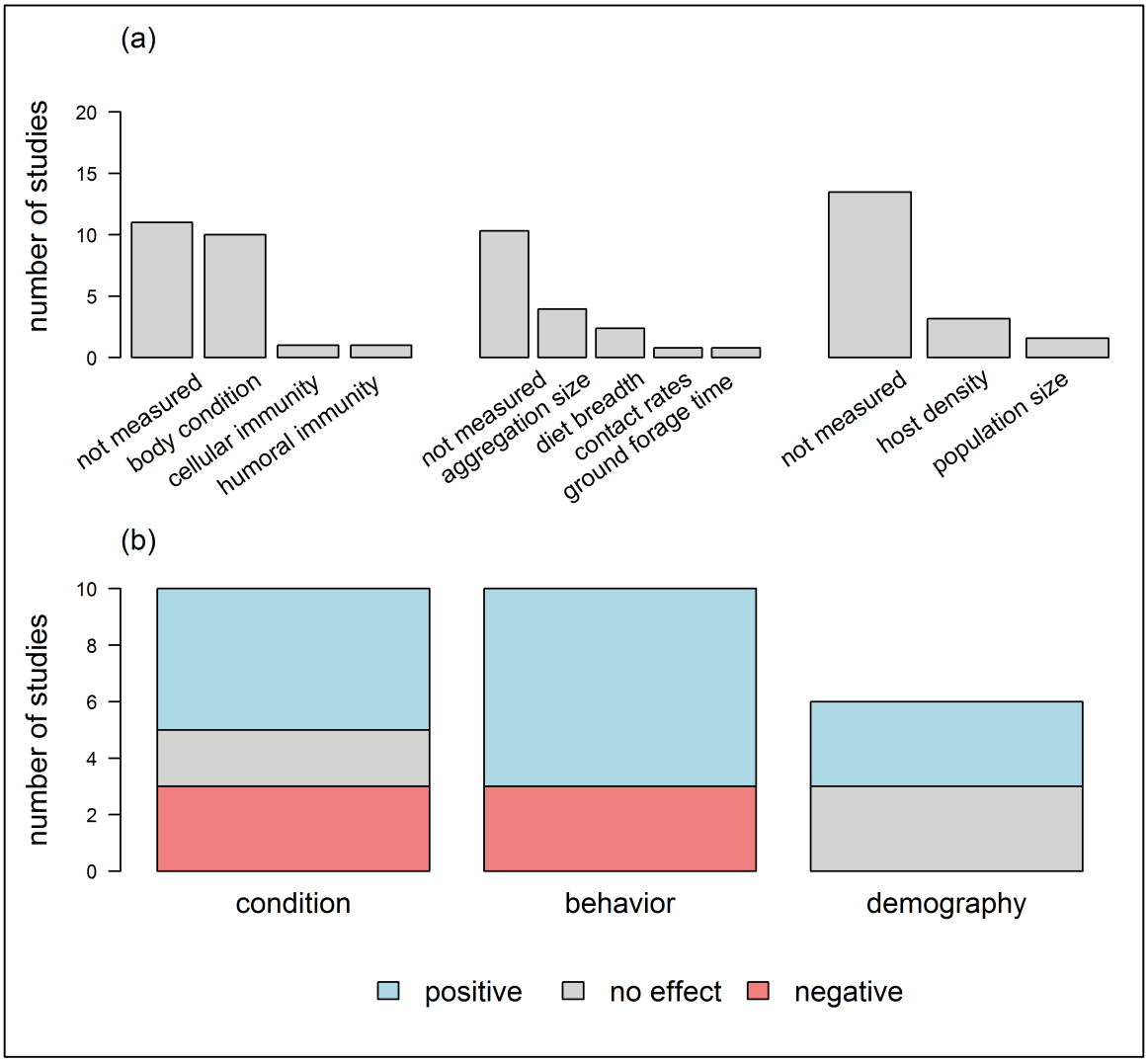


Figure S3.3. Distribution of (a) mechanism variables measured and (b) the directional effect of provisioning on each variable subset.

BS3 Publication bias

In addition to the main aims of our analysis, we assessed publication bias in our dataset using funnel plots displaying standardized effect sizes against their corresponding standard errors. A symmetrical plot indicates no publication bias, as effects from studies with high precision will remain close to the estimated true effect, while effects from studies with large standard errors should be spread equally on both sides of the mean [338,339]. We evaluated funnel plot asymmetry for the REM using the rank correlation test, which tests for a relationship between observed effect sizes and standard errors [339,340]. We further explored publication bias using the trim and fill method [341], a non-parametric rank-based technique that estimates the number of studies missing from analysis due to suppression of extreme and non-significant observations and tests if addition of these results would affect the estimation of the mean true effect.

Using the REM Egger test, we did not detect a relationship between effect size and standard error ($z = 1.83$, $p = 0.07$), suggesting a lack of publication bias in the study of provisioning and wildlife disease (Figure S3.4a). However, trim and fill analyses using the $L0^+$ estimator suggested 45 lines of data demonstrating effects less than the estimated true effect ($\mu = 0.096$) were missing from analysis due to suppression of extreme or non-significant results. Incorporating these missing lines of data shifted the confidence interval of the estimated true effect to cross zero (Figure S3.4b), negating the observed positive relationship between provisioning and disease outcomes observed in our main analysis ($\mu = –0.06$, $z = –1.80$, $p = 0.07$).

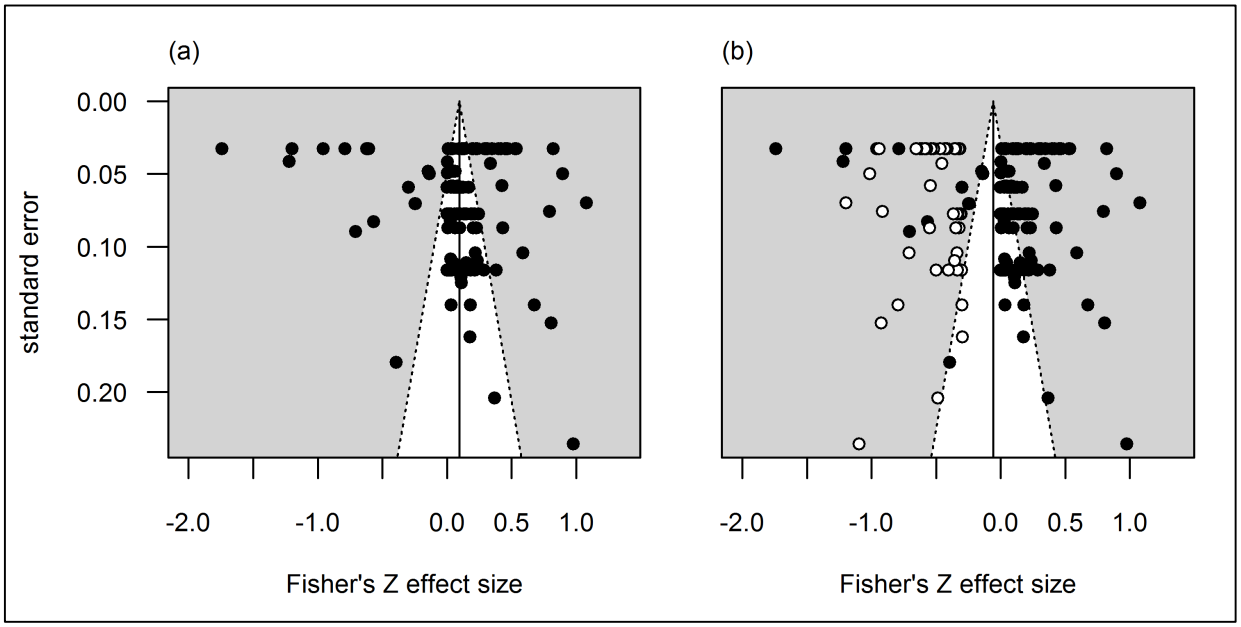


Figure S3.4. Funnel plots illustrating (a) the relationship between effect size and standard error and (b) the effect of correcting funnel plot asymmetry through trim and fill analysis on the estimated true effect of provisioning on disease outcomes.

BS4 Predictors of host–pathogen responses to anthropogenic resources

MEM analyses demonstrated significant effects of all individual covariates on infection outcomes in provisioned populations, although model comparison identified provisioning source and pathogen type as individually explaining the most variation (Table S3.2).

Table S3.2. Univariate MEM analyses ranked by AIC.

predictor variables	τ^2	Q_M	Q_M p-value	ΔAIC	R^2 (%)
~ provisioning source	0.12	19.71	0.001	0	7.58
~ provisioning type	0.13	12.18	0.002	2	2.51
~ pathogen type	0.13	18.44	0.005	5	6.77
~ host taxonomy	0.13	12.44	0.014	6	2.59
~ transmission mode	0.13	9.55	0.049	9	0.61

We here visualize the influence of the other three predictors (Figure S3.5). Host taxonomy and provisioning source explained roughly equal amounts of variation in infection outcomes, with intentional forms of provisioning and all classes of hosts associated with increased disease. Transmission mode explained the least amount of variation.

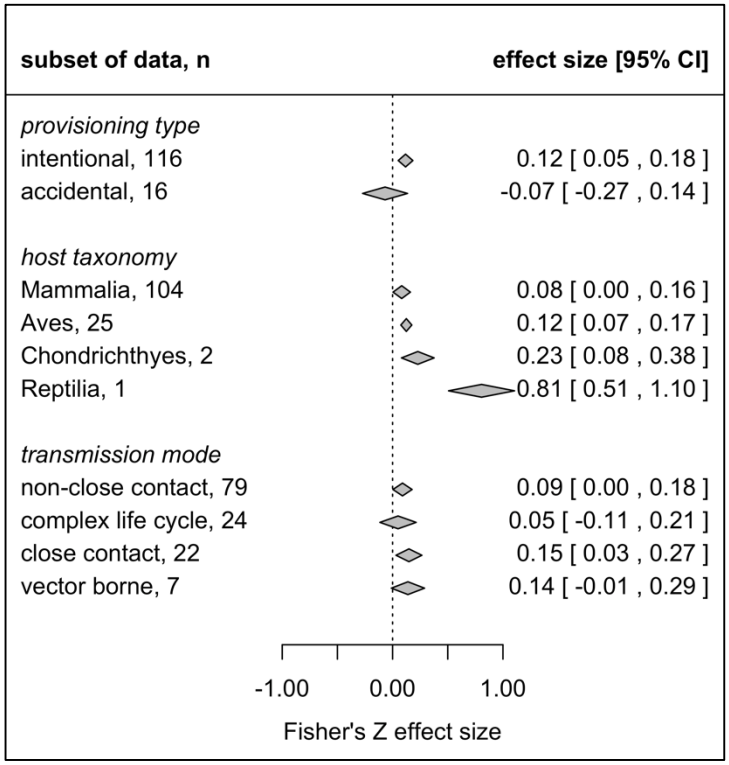


Figure S3.5. Remaining predictors of heterogeneity in infection outcomes of resource provisioning, ranked by AIC. Diamonds with 95% confidence intervals show the estimated mean effect size for each level of each factor.

BS5 Sensitivity to studies with proximate measures of provisioning

Our main tests used studies that directly included provisioning in their analyses alongside studies that used more proximal measures of anthropogenic resources. Because infection outcomes in these proximal studies could be affected by various other factors of urbanized environments, we here present sensitivity analyses for a reduced dataset only containing studies where inclusion of provisioning was direct. Importantly, our results vary very little when proximal studies were excluded from analysis (Table S3.3). As differences between analyses were minimal, we justify inclusion of proximal studies in the main text to optimize statistical power.

We first ran REM and publication bias analyses only for studies directly including provisioning as a variable of interest (Table S3.3). As in the REM analysis of our full dataset, after accounting for missing lines of data (k = 42) due to suppression of extreme or non-significant results, there was no net effect of provisioning on infection for the reduced dataset (μ = –0.06, p = 0.06). We similarly did not detect a relationship between effect sizes and standard errors when excluding proximal studies (z = 1.79, p = 0.07) using the Egger test.

MEM analysis of this reduced dataset also differed little (Table S3.4). Stepwise model simplification again identified pathogen type and source as together explaining the most variation in infection outcomes (20.81%) in comparison to the base REM (LRT = 28.7, df = 2, p < 0.001). As in the full analysis, the reduced analysis indicated that management- and recreation-based provisioning sources promote increased infection outcomes, whereas foraging on urban waste was associated with reduced disease. Similarly, the reduced dataset also found that the transmission of bacteria, viruses, and helminths are expected to increase in response to provisioning. Lastly, analyses of the reduced dataset likewise supported behavioral and

immunological mechanisms by which provisioning affects infection outcomes, including greater aggregation, dietary simplification, and improved tolerance predicting higher infection rates.

Table S3.3. Results of REM and publication bias tests for full and reduced dataset

	records	**μ**	**p**	**Q_E**	**p**	**Egger's**	**p**	**missing**	**bias-**	**p**
full	132	0.095	0.004	11274	<	1.83	0.07	45	-0.059	0.07
reduced	126	0.090	0.008	11206	<	1.79	0.07	42	-0.063	0.06

Table S3.4. Results of MEM analyses for full and reduced dataset

	best model	**R^2**	**Q_M**	**p**	**Q_M**	**p**	**Q_M**	**p**
full	~ pathogen +	17.8	24.84	<	45.29	<	3.59	0.1
reduce	~ pathogen +	20.8	20.48	<	45.29	<	3.59	0.1

BS6 Studies included in the meta-analysis

1. Aponte, V., Locke, S.A., Gentes, M.-L., Giroux, J.-F., Marcogliese, D.J., McLaughlin, D., *et al.* (2014). Effect of habitat use and diet on the gastrointestinal parasite community of an avian omnivore from an urbanized environment. *Canadian Journal of Zoology*, 92, 1–8.
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3. Bradley, C.A., Gibbs, S.E.J. & Altizer, S. (2008). Urban land use predicts West Nile virus exposure in songbirds. *Ecol Appl*, 18, 1083–1092.
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6. Hill, N.J., Deane, E.M. & Power, M.L. (2008). Prevalence and genetic characterization of Cryptosporidium isolates from common brushtail possums (Trichosurus vulpecula) adapted to urban settings. *Applied and environmental microbiology*, 74, 5549–5555.
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12. Miller, R.A., Kaneene, J.B., Fitzgerald, S.D. & Schmitt, S.M. (2003). Evaluation of the influence of supplemental feeding of white-tailed deer (Odocoileus virginianus) on the prevalence of bovine tuberculosis in the Michigan wild deer population. *Journal of Wildlife Diseases*, 39, 84–95.
13. Monello, R.J. & Gompper, M.E. (2010). Differential effects of experimental increases in sociality on ectoparasites of free-ranging raccoons. *Journal of animal ecology*, 79, 602–609.
14. Monello, R.J. & Gompper, M.E. (2011). Effects of resource availability and social aggregation on the species richness of raccoon endoparasite infracommunities. *Oikos*, 120, 1427–1433.
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BS7 Model equations and derivation of R_0

The modeling framework outlined by [52] used simple compartmental models to describe the effect of provisioning on microparasite dynamics. The basic susceptible–infected–recovered system is described by three differential equations.

$$dS/dt = \big(b_0 - b_1(S + I + R)\big)(S + I + R) - \mu S - \alpha\delta SI$$

$$dI/dt = \alpha\delta SI - (\mu + \nu + \gamma)I$$

$$dR/dt = \gamma I - \mu R$$

Host demography is described by a natural mortality rate μ and a density-dependent birth rate $b_0 - b_1 N$, where N is the population size and b_0 and b_1 are constants. Together these parameters give K, the host carrying capacity $(b_0 - \mu/b_1)$. The framework assumes density-dependent pathogen transmission, but states the transmission parameter as the product of terms describing contact rate (α) and probability of infection upon encounter (δ). Pathogen clearance and disease-induced mortality occur at rates γ and ν respectively.

For the SIR system, R_0 is the product of the disease-free equilibrium host population size (i.e., carrying capacity), the transmission rate, and the infectious period (the inverse of the rate at which infected individuals leave the infectious class):

$$R_0 = \frac{\alpha\delta K}{\mu + \upsilon + \gamma}$$

To derive the provisioning-dependent R_0, parameters are set to depend on the functional forms stated in the main text (Box 1). Under provisioning, α and b_0 are increasing functions of the

provisioning parameter ρ, whereas δ, υ, and μ are decreasing functions. R_0 is then attained by substituting expressions for the resource-dependent parameters into the above equation [52,312].

For our empirically revised modeling framework of microparasite transmission, we follow this same procedure for the analytic derivation of R_0. However, unlike in [52], following our meta-analysis results we here set δ to increase with provisioning. Additionally, in the simulation examining the influence of reduced dietary exposure, we further alter this framework by changing α to decrease with provisioning. Parameter values are the same as those used in [52], although θ_x for resource-dependent parameters is here set equal for behavioral and immunological mechanisms.

APPENDIX C

CHAPTER 4 SUPPLEMENTAL INFORMATION

CS1 Examples of managed species exhibiting metapopulation structure

Table S4.1. Examples of metapopulations experiencing resource supplementation, their conservation status, and the consequences of resource improvement.

Managed species	Status	Patch quality	Consequence	References
Bearded vulture (*Gypaetus barbatus*)	Imperiled	Improved by supplemental feeding	Increased fecundity and carrying capacity, reduced extinction risk	[342]
Iberian lynx (*Lynx pardinus*)	Imperiled	Improved by supplemental feeding	Increased recruitment and survival, higher population viability	[175,343]
Little owl (*Athene noctua*)	Imperiled	Improved by supplemental feeding	Increased recruitment and survival	[344,345]
South Island takahe (*Porphyrio hochstetteri*)	Imperiled	Improved by supplemental feeding	Smaller home range, larger carrying capacity	[346]
Red squirrel (*Sciurus vulgaris*)	Persistent	Improved by recreational feeding	Higher host densities and population viability, improved occupancy	[347,348]
Northern red-legged frogs (*Rana aurora*	Persistent	Improved by supplemental feeding	Increased body size, survival, and emigration	[349]

aurora)				
Song sparrow (*Melospiza melodia*)	Persistent	Improved by urban resource subsidies	Increased recruitment, lower connectivity	[350,351]

CS2 Model equilibria and stability analysis.

The full system of equations describing disease dynamics in a metapopulation with low- and high-quality patches is:

$$\frac{dS_L}{dt} = c(1 - f - S_L - I_L)(S_L + S_H) - x_L S_L - \delta c S_L (I_L + I_H) \quad \text{(B1a)}$$

$$\frac{dI_L}{dt} = c(1 - f - S_L - I_L)(I_L + I_H) - (x_L + \nu_L) I_L + \delta c S_L (I_L + I_H) \quad \text{(B1b)}$$

$$\frac{dS_H}{dt} = c(f - S_H - I_H)(S_L + S_H) - \frac{x_L}{q} S_H - \delta c S_H (I_L + I_H) \quad \text{(B1c)}$$

$$\frac{dI_H}{dt} = c(f - S_H - I_H)(I_L + I_H) - \left(\frac{x_L + \nu_L}{q}\right) I_H + \delta c S_H (I_L + I_H) \quad \text{(B1d)}$$

The parameter definitions are outlined in Table 4.1 of the main text. There are four possible equilibrium states (obtained by setting each of the above derivatives to zero and solving): metapopulation extinction, disease-free persistence, endemic equilibrium, and pandemic equilibrium. Where feasible, we derived analytical expressions for the equilibrium total occupancy (given for each equilibrium below) and threshold conditions for their stability in terms of the two key model parameters f and q (derived below and summarized in Table 4.2 in main text). In order to derive stability criteria, we calculate the Jacobian matrix J, whose (i, j) entries describe the partial derivatives for the rate of change of variable i with respect to variable j ($S_L = 1$, $I_L = 2$, $S_H = 3$, $I_H = 4$, so that $J_{1,2} = \frac{\partial S_L}{\partial I_L}$ and so on).

$$J_{1,1} = -c(S_L + S_H) + c(1 - f - S_L - I_L) - x_L - \delta c(I_L + I_H)$$

$$J_{1,2} = -c(S_L + S_H) - \delta c S_L$$

$$J_{1,3} = c(1 - f - S_L - I_L)$$

$$J_{1,4} = -\delta c S_L$$

$$J_{2,1} = -c(I_L + I_H) + \delta c(I_L + I_H)$$

$$J_{2,2} = -c(I_L + I_H) + c(1 - f - S_L - I_L) - x_L - \nu_L + \delta c S_L$$

$$J_{2,3} = 0$$

$$J_{2,4} = c(1 - f - S_L - I_L) + \delta c S_L \quad \text{(B2)}$$

$$J_{3,1} = c(f - S_H - I_H)$$

$$J_{3,2} = -\delta c S_H$$

$$J_{3,3} = -c(S_L + S_H) + c(f - S_H - I_H) - \frac{x_L}{q} - \delta c(I_L + I_H)$$

$$J_{3,4} = -c(S_L + S_H) - \delta c S_H$$

$$J_{4,1} = 0$$

$$J_{4,2} = c(f - S_H - I_H) + \delta c S_H$$

$$J_{4,3} = -c(I_L + I_H) + \delta c(I_L + I_H)$$

$$J_{4,4} = -c(I_L + I_H) + c(f - S_H - I_H) - \frac{x_L}{q} - \frac{\nu_L}{q} + \delta c S_H$$

An equilibrium is only stable if all eigenvalues of the Jacobian evaluated at the equilibrium are negative. For a four-by-four matrix, this means that an equilibrium is unstable if its determinant is negative; we take advantage of this condition to describe conditions under which equilibria lose their stability. In all cases, the stability transitions between equilibria result from saddle-node bifurcations (i.e., when $\det(J) = 0$). Below we outline the stability analysis in detail for the trivial equilibrium; a similar approach was used to determine the stability of the remaining three equilibria.

Metapopulation extinction

The trivial equilibrium $(S_L^*, I_L^*, S_H^*, I_H^*) = (0,0,0,0)$ always exists. The Jacobian evaluated at this equilibrium is:

$$J = \begin{bmatrix} c(1-f) - x_L & 0 & c(1-f) & 0 \\ 0 & c(1-f) - x_L - \nu_L & 0 & c(1-f) \\ cf & 0 & cf - \frac{x_L}{q} & 0 \\ 0 & cf & 0 & cf - \frac{x_L}{q} - \frac{\nu_L}{q} \end{bmatrix} \quad \text{(B3)}$$

Exploiting the zero structure of the Jacobian, we find that the determinant factorizes as follows:

$$\det(J) = \left(J_{1,1}J_{3,3} - J_{1,3}J_{3,1}\right)\left(J_{2,2}J_{4,4} - J_{4,2}J_{4,4}\right) = \det\left(J_{1,3}\right)\det\left(J_{2,4}\right) \quad \text{(B4)}$$

where

$$J_{1,3} = \begin{bmatrix} J_{1,1} & J_{1,3} \\ J_{3,1} & J_{3,3} \end{bmatrix} = \begin{bmatrix} c(1-f) - x_L & c(1-f) \\ cf & cf - \frac{x_L}{q} \end{bmatrix} \quad \text{(B5)}$$

and

$$J_{2,4} = \begin{bmatrix} J_{2,2} & J_{2,4} \\ J_{4,2} & J_{4,4} \end{bmatrix} = \begin{bmatrix} c(1-f) - x_L - \nu_L & c(1-f) \\ cf & cf - \frac{x_L}{q} - \frac{\nu_L}{q} \end{bmatrix} \quad \text{(B6)}$$

Noting that $Tr(J) = Tr(J_{1,3}) + Tr(J_{2,4})$, the equilibrium is only stable if $Tr(J_{1,3}), Tr(J_{2,4}) < 0$ and $\det(J_{1,3}), \det(J_{2,4}) > 0$. The condition $\det(J_{1,3}) > 0$ is therefore

$$\frac{x_L^2}{q} - c(1-f)\frac{x_L}{q} - cfx_L > 0 \quad \text{(B7)}$$

which rearranges to

$$\frac{c(1-f)}{x_L} + \frac{cfq}{x_L} < 1 \quad \text{(B8)}$$

The terms on the left-hand side of this expression represent respectively the fraction of occupied low- and high-quality patches of resulting from individual low- and high-quality patches over their "lifetimes" (i.e., the duration of occupancy) when introduced to an empty metapopulation.

The condition $Tr(J_{1,3}) < 0$ yields the following inequality:

$$c(1-f) + cf - x_L - \frac{x_L}{q} < 0 \quad \text{(B9)}$$

Dividing through by x_L, noting that $q \geq 1$, and finally using the condition for $\det(J_{1,3}) > 0$ (i.e., $\frac{x_L^2}{q}\left(\frac{c(1-f)}{x_L} + \frac{cfq}{x_L} - 1\right) < 0$, we find

$$\frac{c(1-f)}{x_L} + \frac{cf}{x_L} - 1 - \frac{1}{q} < \frac{c(1-f)}{x_L} + \frac{cfq}{x_L} - 1 - \frac{1}{q} < 0 - \frac{1}{q} < 0 \quad \text{(B10)}$$

i.e., the trace is negative whenever the determinant is positive.

Finally, since $x_L + \nu_L > x_L$, we have

$$\frac{c(1-f)}{x_L+\nu_L} + \frac{cf}{x_L+\nu_L} < \frac{c(1-f)}{x_L} + \frac{cfq}{x_L} \tag{B11}$$

and thus $\det(J_{2,4}) > 0$. Therefore, the trivial equilibrium is stable if and only if the following condition is met:

$$\frac{c(1-f)}{x_L} + \frac{cfq}{x_L} < 1 \tag{B12}$$

Disease-free persistence

The disease-free equilibrium $(S_L(df), 0, S_H(df), 0)$ is found by setting the ordinary differential equations for S_L and S_H to zero when $I_L = I_H = 0$, and solving the simultaneous equations, yielding the following expressions:

$$S_L(df) = 1 - f - \frac{1}{2}\left[\theta + \frac{q}{q-1}\left(1 - \sqrt{\left(1-\theta\left(1-\frac{1}{q}\right)\right)^2 + 4f\theta\left(1-\frac{1}{q}\right)}\right)\right] \tag{B13a}$$

$$S_H(df) = f - \frac{1}{2}\left[\frac{\theta}{q} - \frac{1}{q-1}\left(1 - \sqrt{\left(1-\theta\left(1-\frac{1}{q}\right)\right)^2 + 4f\theta\left(1-\frac{1}{q}\right)}\right)\right] \tag{B13b}$$

$$N(df) = \frac{1}{2}\left[1 - \theta\left(1+\frac{1}{q}\right) + \sqrt{\left(1-\theta\left(1-\frac{1}{q}\right)\right)^2 + 4f\theta\left(1-\frac{1}{q}\right)}\right] \tag{B13c}$$

where $\theta = x_L/c$. The condition for the existence of this equilibrium is the condition for the extinction equilibrium to lose its stability; i.e., when

$$\frac{c(1-f)}{x_L} + \frac{cfq}{x_L} > 1 \tag{B14}$$

We can derive a threshold condition for the fraction of high-quality patches necessary for persistence by rearranging the above inequality to yield

$$f > f_{df} = \frac{1}{q-1}\left(\frac{x_L - c}{c}\right) \tag{B15}$$

The disease-free equilibrium is stable provided the pathogen cannot invade (see below).

Endemic equilibrium

The endemic equilibrium $(S_L^*(e), I_L^*(e), S_H^*(e), I_H^*(e))$ is difficult to solve analytically; however, when the metapopulation consists only of low-quality ($f = 0$) or high-quality ($f = 1$) patches, the total equilibrium occupancies are, respectively,

$$N_0(e) = \frac{\nu_L}{\delta c}, N_1(e) = \frac{\nu_L}{q\delta c} \tag{B16}$$

The condition for its existence is that the disease-free equilibrium loses its stability. This occurs when the per-generation growth rates of low- and high-quality infected patches, introduced into a

metapopulation at its disease-free equilibrium, (i.e., the pathogen basic reproductive number) is greater than one:

$$R_0 = \frac{c(1-f-S_L(df))+\delta c S_L(df)}{x_L+\nu_L} + \frac{qc(f-S_H(df))+q\delta c S_H(df)}{x_L+\nu_L} > 1 \quad \text{(B17)}$$

Substituting the derived expressions for the disease-free equilibrium densities and rearranging yields the following expression for R_0:

$$R_0 = \frac{x_L(1-\delta)+\delta c(1+(q-1)f)}{x_L+\nu_L} \quad \text{(B18)}$$

We can rearrange the pathogen invasion threshold $R_0 > 1$ to yield the critical fraction of high-quality patches necessary for the pathogen to invade and become endemic:

$$f > f_e = \frac{1}{q-1}\left(\frac{x_L-c}{c} + \frac{\nu_L}{\delta c}\right) \quad \text{(B19)}$$

Pandemic equilibrium

The pandemic equilibrium $(0, I_L(p), 0, I_H(p))$ occupancies can be written by noting that the ordinary differential equations for I_L and I_H with no uninfected patches resemble those for S_L and S_H, with the per-patch extinction rate x_L replaced with $x_L + \nu_L$:

$$I_L(p) = 1 - f - \frac{1}{2}\left[\theta + \phi + \frac{q}{q-1}\left(1 - \sqrt{\left(1-(\theta+\phi)\left(1-\frac{1}{q}\right)\right)^2 + 4f(\theta+\phi)\left(1-\frac{1}{q}\right)}\right)\right] \quad \text{(B20)}$$

$$I_H(p) = f - \frac{1}{2}\left[\frac{\theta+\phi}{q} - \frac{1}{q-1}\left(1 - \sqrt{\left(1-(\theta+\phi)\left(1-\frac{1}{q}\right)\right)^2 + 4f(\theta+\phi)\left(1-\frac{1}{q}\right)}\right)\right] \quad \text{(B21)}$$

$$N(p) = \frac{1}{2}\left[1-(\theta+\phi)\left(1+\frac{1}{q}\right) + \sqrt{\left(1-(\theta+\phi)\left(1-\frac{1}{q}\right)\right)^2 + 4f(\theta+\phi)\left(1-\frac{1}{q}\right)}\right] \quad \text{(B22)}$$

where $\phi = \nu_L/c$. This equilibrium always exists when the pathogen can invade, but is only stable when the per-generation growth rate of uninfected patches when introduced to a wholly infected population is less than one:

$$\frac{c(1-f-I_L(p))}{x_L+\delta cN(p)} + \frac{c(f-I_H(p))}{\frac{x_L}{q}+\delta cN(p)} < 1 \quad \text{(B23)}$$

In order to find the threshold condition for the endemic–pandemic stability switch, we set the above inequality to 1 and rearrange to derive a quadratic in $N(p)$. The algebra is simplified by using the expressions for $I_L(p)$ and $I_H(p)$ to yield the following identity:

$$\frac{1}{q}\left(1-f-I_L(p)\right) + f - I_H(p) = \frac{\theta+\phi}{q} \quad \text{(B24)}$$

The quadratic for $N(p)$ simplifies to

$$(1+\delta)N(p)^2 + \left(\theta\left(1+\frac{1}{q}\right)-1\right)N(p) - \frac{\theta\phi}{q\delta} = 0 \quad \text{(B25)}$$

and using the quadratic formula, the value of $N(p)$ at the threshold is:

$$N(p) = \frac{1}{2(1+\delta)}\left[1 - \theta\left(1+\frac{1}{q}\right) + \sqrt{\left(1-\theta\left(1+\frac{1}{q}\right)\right)^2 + \frac{4\theta\phi(1+\delta)}{q\delta}}\right] \quad \text{(B26)}$$

Equating this expression to the previously derived expression for $N(p)$ yields

$$\frac{1}{2}\left[1 - (\theta+\phi)\left(1+\frac{1}{q}\right) + \sqrt{\left(1-(\theta+\phi)\left(1-\frac{1}{q}\right)\right)^2 + 4f(\theta+\phi)\left(1-\frac{1}{q}\right)}\right] \quad \text{(B27)}$$

$$= \frac{1}{2(1+\delta)}\left[1 - \theta\left(1+\frac{1}{q}\right) + \sqrt{\left(1-\theta\left(1+\frac{1}{q}\right)\right)^2 + \frac{4\theta\phi(1+\delta)}{q\delta}}\right] \quad \text{(B28)}$$

Rearranging this expression, we can derive an expression for the critical fraction of high-quality patches at which the pandemic equilibrium becomes stable:

$$f > f_p = \frac{Z^2 - Y^2}{4(1-Y)} \quad \text{(B29)}$$

where

$$Y = 1 - (\theta+\phi)\left(1-\frac{1}{q}\right) \quad \text{(B30)}$$

$$Z = \left(1+\frac{1}{q}\right)\left(\frac{\delta\theta + (1+\delta)\phi - \delta - \sqrt{W}}{1+\delta}\right) \quad \text{(B31)}$$

and

$$W = \left(1 - \theta\left(1 + \frac{1}{q}\right)\right)^2 + \frac{4\theta\phi(1+\delta)}{q\delta} \quad \text{(B32)}$$

CS3 Sensitivity of pathogen dynamics to how patch quality affects disease-induced extinction.

In the model presented in the main text, we assumed patch improvement reduces disease-induced extinction in the same way as it reduces disease-free extinction ($\nu_H = \nu_L/q$). Here we present results from an alternative scenario where disease-induced extinction is unaffected by the degree of patch improvement ($\nu_H = \nu_L$). Given the case of a declining metapopulation rescued through supplemental feeding (scenario A), we find that restricting patch quality improvement to affect only the disease-free extinction rate makes pathogen invasion less likely; for our parameterization only occurring at very high degrees of resource improvement and deployment of management across the landscape (Figure S4.1). This is because disease-induced extinction of high-quality patches remains high even when provisioning reduces the "natural" extinction rate of patches. The region of parameter space across f and q where disease-free persistence occurs is much larger within this scenario than for that described in the text. Therefore when resource-based management does not influence disease-induced extinction, resource improvement is predicted to have primarily positive effects on patch occupancy.

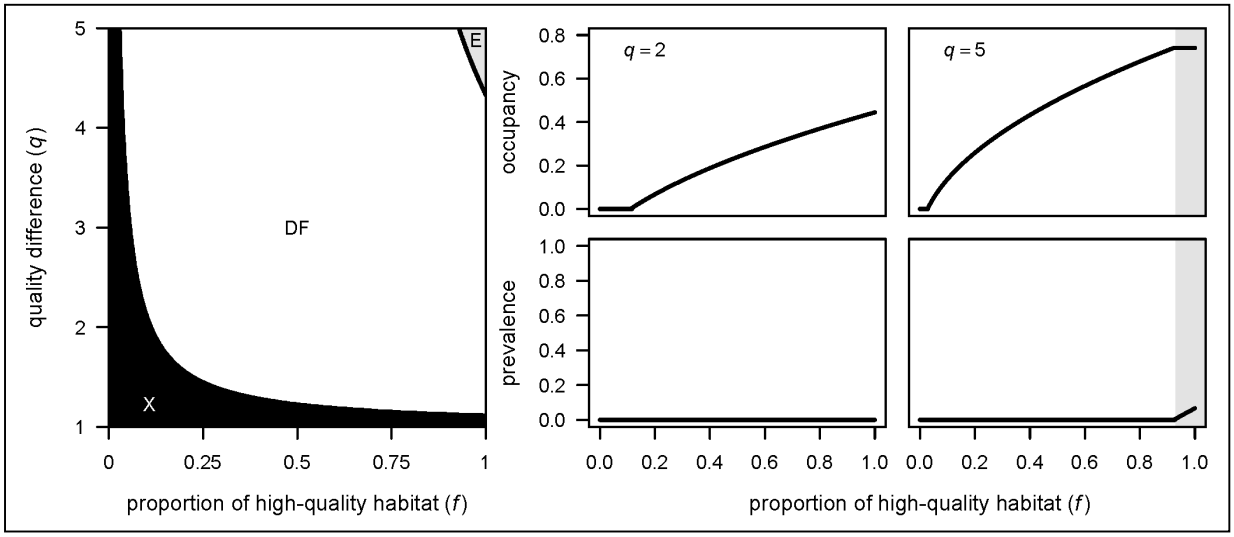

Figure S4.1. Thresholds for equilibria as a function of resource improvement under scenario A ($c < x_L$), where the disease-induced extinction rate is unaffected by the degree of patch improvement ($\nu_H = \nu_L$). Shaded regions depict where the metapopulation is extinct (X, black), persists in a disease-free state (DF, white), and where the pathogen invades and becomes endemic (E, light grey).

Given the case of a persistent metapopulation provisioned by resource subsidies (scenario B), we likewise find that pandemic equilibrium only occurs at very high degrees of resource improvement and deployment of management across the landscape (Figure S4.2). The region of parameter space across f and q where pathogen invasion and endemic equilibrium occurs is much larger within this scenario than for that described in the text. Therefore when provisioning does not influence disease-induced extinction, resource improvement does facilitate pathogen invasion but allows infection-free patches to persist. In both cases we used the same disease-induced extinction rate ($\nu_L = 0.5$, so that low-quality infected patches go extinct 1.5 times faster than uninfected patches) for direct comparison with the parameterization in the main text. If the disease-induced extinction rate ν_L is lower, pathogen invasion, and the outcomes described in the text, will occur in a larger region of provisioning parameter space.

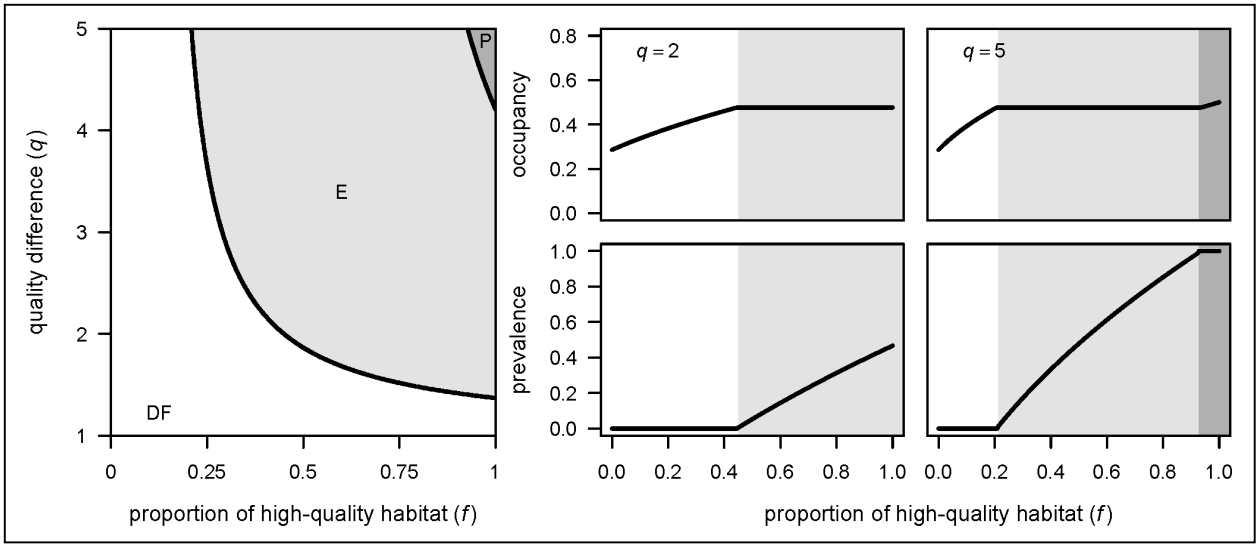

Figure S4.2. Thresholds for equilibria as a function of resource improvement under scenario B ($c > x_L$), where the disease-induced extinction rate is unaffected by the degree of patch improvement ($\nu_H = \nu_L$). Shaded regions depict where the metapopulation persists in a disease-free state (DF, white) and where the pathogen invades and becomes endemic (E, light grey) or pandemic (P, dark grey).

CS4 Sensitivity of the global maximum and minimum in occupancy to pathogen traits.

In Scenario A, where the metapopulation is declining prior to management and is rescued through supplemental feeding ($c < x_L$), we can use the invasion threshold derived in Appendix B to calculate f_e as functions of pathogen virulence (ν_L) and transmissibility (δ). We then solve the models numerically to obtain the equilibrium patch occupancy $N^*(f_e)$ and compare this to occupancy in a fully provisioned metapopulation $N^*(f = 1)$. Whenever their difference is positive, maintaining some low-quality patches results in higher occupancy than a fully provisioned population (Figure S4.3). In scenario B, where a persistent metapopulation is subject to provisioning ($c > x_L$), we can calculate the fraction of provisioned patches at which infection becomes pandemic (f_p) as functions of pathogen virulenence and transmissibility, and use simulations to calculate the occupancy ($N^*(f_p)$). Whenever this quantity is less than the unprovisioned occupancy ($N^*(0)$), provisioning is dertimental to the metapopulation (Figure S4.4).

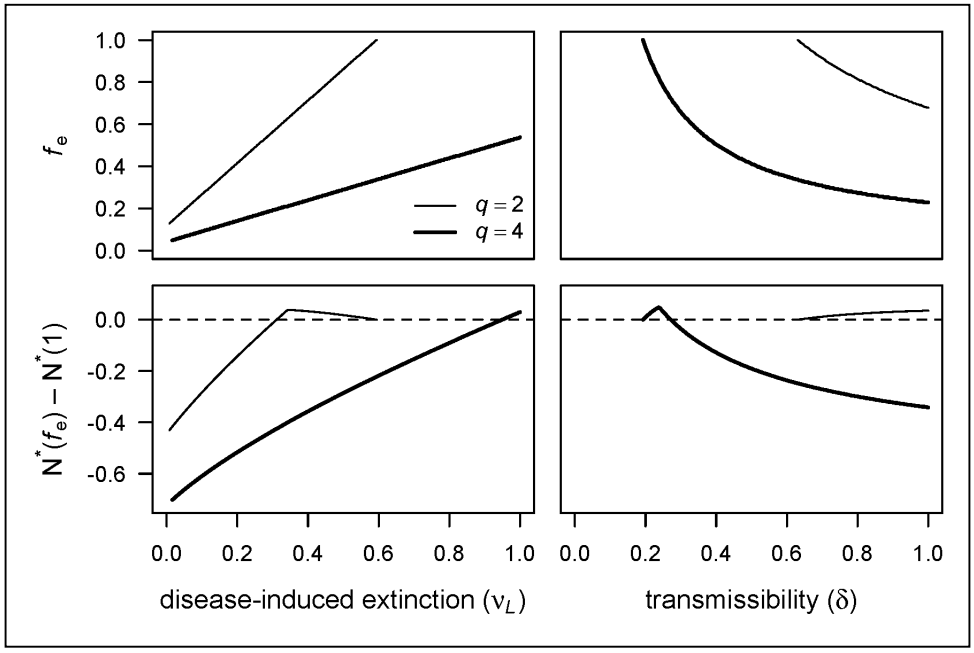

Figure S4.3. Sensitivity of key model outputs to variation in pathogen virulence (ν_L; left column) and transmissibility (δ; right column) for scenario A. Top row: fraction of provisioned patches at the pathogen invasion threshold (f_e). Bottom row: difference between occupancy at the invasion threshold and in a fully provisioned metapopulation ($N^*(f_e) - N^*(1)$). Occupancy is maximized for patch mixtures whenever these curves lie above the dashed line. Outcomes are illustrated for "moderate" ($q = 2$, thin line) and "high" ($q = 4$, thick line) provisioning scenarios.

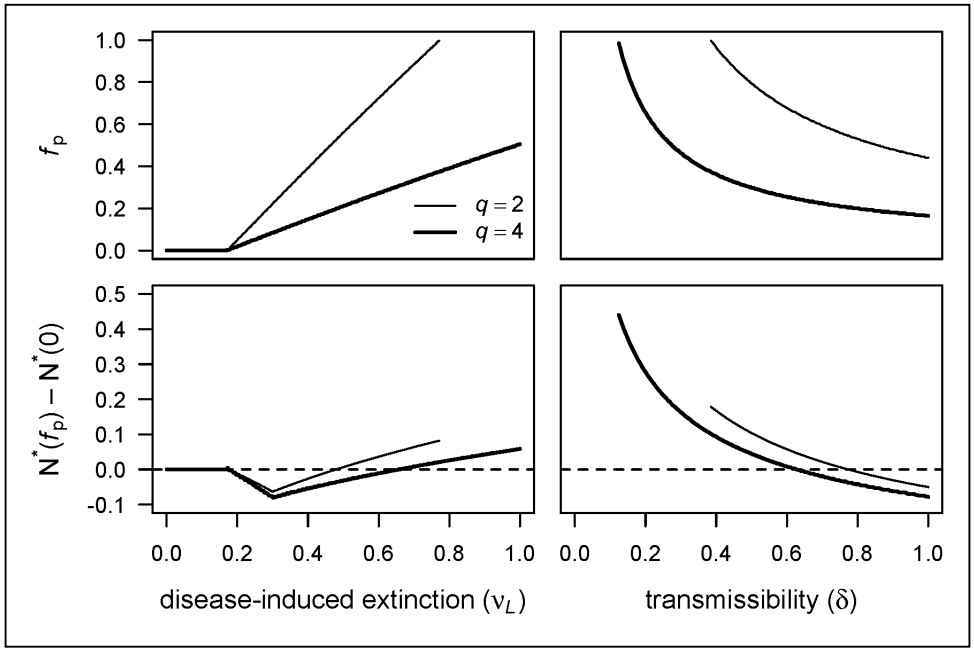

Figure S4.4. Sensitivity of key model outputs to variation in pathogen virulence (ν_L; left column) and transmissibility (δ; right column) for scenario B. Top row: fraction of provisioned patches at the pandemic threshold (f_p). Bottom row: difference between occupancy at the pandemic threshold and in an unprovisioned metapopulation ($N^*(f_p) - N^*(0)$). Occupancy is minimized for provisioned metapopulations relative to unprovisioned metapopulations whenever these curves below above the dashed line. Outcomes are illustrated for "moderate" ($q = 2$, thin line) and "high" ($q = 4$, thick line) provisioning scenarios.

APPENDIX D

CHAPTER 5 SUPPLEMENTAL INFORMATION

DS1 Model derivation and analysis

Provisioning effects in the absence of disease

In the absence of provisioning, patch occupancy dynamics are described by a classic Levins metapopulation with colonization rate c and extinction rate x. A fraction f of patches is provisioned across the landscape, and the fractions of occupied unprovisioned and provisioned patches are described by N_U and N_P respectively. Provisioning changes the extinction rates of provisioned patches relative to unprovisioned patches by a factor α, and colonization rates by altering the relative number of dispersers produced (θ) and the relative attractiveness of provisioned patches to dispersers (ϕ). We assume that the proportion of dispersers that arrive in a patch of type j $(= U, P)$ depends on both its relative attractiveness (1 or ϕ) and the frequency of that patch type across the landscape ($1 - f$ or f respectively). Thus, the colonization rate for each patch type is the product of the total number of dispersers produced per unit time, the probability that they arrive at a patch of type j (based on its relative attractiveness), and the probability that the patch is unoccupied:

$$C_U = (cN_U + \theta cN_P) \times \frac{1.(1-f)}{\phi f + 1.(1-f)} \times \frac{1-f-N_U}{1-f}$$

$$= \frac{c}{1+(\phi-1)f}(1-f-N_U)(N_U+\theta N_P) \quad (1a)$$

$$C_P = (cN_U + \theta cN_P) \times \frac{\phi f}{\phi f + 1.(1-f)} \times \frac{f-N_P}{f} = \frac{\phi c}{1+(\phi-1)f}(f-N_P)(N_U+\theta N_P) \quad (1b)$$

The equations describing metapopulation dynamics are as follows:

$$\frac{dN_U}{dt} = \frac{c}{1+(\phi-1)f}(1-f-N_U)(N_U+\theta N_P) - xN_U \quad (2a)$$

$$\frac{dN_P}{dt} = \frac{\phi c}{1+(\phi-1)f}(f-N_P)(N_U+\theta N_P) - \alpha x N_P \quad (2b)$$

Conditions for coexistence

A provisioned metapopulation persists if, on average, every occupied patch replaces itself during the time it is occupied (1/extinction rate). We define the provisioned metapopulation's *lifetime colonization success*, ρ, as the proportion of patches in an empty metapopulation that would be colonized by each patch type over its occupancy time, weighted by their relative frequency on the landscape:

$$\rho = \frac{c(1-f)}{(1+(\phi-1)f)x} + \frac{\theta\phi c f}{(1+(\phi-1)f)\alpha x}. \quad (3)$$

Note that $\rho > 1$ is exactly the condition for the extinction equilibrium to destabilize, and occurs when the determinant of the Jacobian of system of equations (2) evaluated at the extinction equilibrium is negative. Expression (3) can be re-written as follows:

$$\rho = \frac{c}{x}\left(1 + \frac{\phi(\frac{\theta}{\alpha}-1)}{\frac{1}{f}+\phi-1}\right) \quad (4)$$

From this we can see that ρ is a monotonic function of f. Provisioning increases the likelihood of metapopulation persistence (i.e. ρ increases with f) only when provisioned patches produce more dispersers over their lifetime than unprovisioned patches (i.e. $\theta/\alpha > 1$); when provisioned patches act as a sink (e.g. by promoting site fidelity or by having higher extinction rates, so that $\theta/\alpha < 1$), increasing the fraction of provisioned patches reduces ρ and the likelihood of persistence. By rearranging expression (4) when $\rho = 1$, we can derive an expression for the critical fraction of patches necessary for the disease-free metapopulation to persist (f_{df}):

$$f_{df} = \left(1 + \frac{\phi(1 - \frac{\theta c}{\alpha x})}{\frac{c}{x} - 1}\right)^{-1} . \quad (5)$$

If the unprovisioned population cannot persist ($c < x$), then a necessary condition is that a wholly provisioned metapopulation ($f = 1$) can, i.e. $\theta c/\alpha x > 1$, and provisioning allows persistence whenever $f > f_{df}$. If the unprovisioned population persists, and provisioned patches produce more dispersers over their lifetime than unprovisioned ($\theta/\alpha > 1$), then the metapopulation will persist for all frequencies of provisioning. However, if provisioned patches on average don't replace themselves ($\theta c/\alpha x < 1$, meaning that a fully provisioned metapopulation would not persist), then persistence is only possible in provisioned metapopulations when $f < f_{df}$.

Full model

We adapt Hess's approach to modeling disease dynamics, where we assume infected colonizers can infect an already occupied patch with probability δ, and that infection increases the

extinction rate of unprovisioned patches by an additive factor v; additionally, we assume that infection reduces the number of dispersers produced by a factor ψ relative to uninfected patches. For simplicity, we consider the effects of provisioning and disease to be independent (so that their effects on colonization and extinction rates are multiplicative). Denoting occupied patches by their infection status (S=Susceptible, I= Infected) and using subscripts to denote whether the patch is Provisioned or Unprovisioned, yields the following system of equations:

$$\frac{dS_U}{dt} = \frac{c}{1+(\phi-1)f}((1-f-S_U-I_U)(S_U+\theta S_P) - \delta\psi S_U(I_U+\theta I_P)) - xS_U \quad (6a)$$

$$\frac{dI_U}{dt} = \frac{\psi c}{1+(\phi-1)f}(1-f-S_U-I_U+\delta S_U)(I_U+\theta I_P) - (x+v)I_U \quad (6b)$$

$$\frac{dS_P}{dt} = \frac{\phi c}{1+(\phi-1)f}((f-S_P-I_P)(S_U+\theta S_P) - \delta\psi S_P(I_U+\theta I_P)) - \alpha x S_P \quad (6c)$$

$$\frac{dI_P}{dt} = \frac{\psi\phi c}{1+(\phi-1)f}(f-S_P-I_P+\delta S_P)(I_U+\theta I_P) - \alpha(x+v)I_P \quad (6d)$$

There are four possible equilibrium states (obtained by setting combinations of the above derivatives to zero and solving): metapopulation extinction, disease-free persistence, pathogen invasion (endemic equilibrium), and all occupied patches become infected (pandemic equilibrium).

Endemic Infection

The disease-free equilibrium becomes unstable when the pathogen can invade, i.e. when the pathogen basic reproductive number, R_0, exceeds 1. We use the next generation matrix method

to derive a matrix whose dominant eigenvalue is the expression for R_0. First, we identify the terms in the above system of equations resulting in gains and losses to infected classes I_U and I_P.

$$I_U \text{ gains: } \frac{\psi c}{1+(\phi-1)f}(1-f-S_U-I_U+\delta S_U)(I_U+\theta I_P) \quad (7a)$$

$$I_P \text{ gains: } \frac{\psi \phi c}{1+(\phi-1)f}(f-S_P-I_P+\delta S_P)(I_U+\theta I_P) \quad (7b)$$

$$I_U \text{ losses: } (x+v)I_U \quad (7c)$$

$$I_P \text{ losses: } \alpha(x+v)I_P \quad (7d)$$

Then, we calculate the matrix, F, whose entries are the partial derivatives of the gain terms with respect to each infected patch type, and evalauted at the disease-free equilibrium ($I_U = I_P = 0$):

$$F_{1,1} = \frac{\psi c}{1+(\phi-1)f}(1-f-S_U+\delta S_U) \quad (8a)$$

$$F_{1,2} = \frac{\theta\psi c}{1+(\phi-1)f}(1-f-S_U+\delta S_U) = \theta F_{1,1} \quad (8b)$$

$$F_{2,1} = \frac{\psi \phi c}{1+(\phi-1)f}(f-S_P+\delta S_P) \quad (8c)$$

$$F_{2,2} = \frac{\theta\psi \phi c}{1+(\phi-1)f}(f-S_P+\delta S_P) = \theta F_{2,1} \quad (8d)$$

The matrix loss terms differentiated with respect to each infectious patch type, $V = \text{diag}(x+v, \alpha(x+v))$. Next we calculate the matrix

$$G = FV^{-1} = \begin{bmatrix} \frac{F_{1,1}}{x+v} & \frac{\theta F_{1,1}}{\alpha(x+v)} \\ \frac{F_{2,1}}{x+v} & \frac{\theta F_{2,1}}{\alpha(x+v)} \end{bmatrix} \quad (9)$$

whose dominant eigenvalue is R_0, found by solving the characteristic equation:

$$\left(\frac{F_{1,1}}{x+v} - \lambda\right)\left(\frac{\theta F_{2,1}}{\alpha(x+v)} - \lambda\right) - \frac{\theta F_{1,1}F_{2,1}}{\alpha(x+v)^2} = 0,$$

yielding

$$R_0 = \frac{F_{1,1}}{x+v} + \frac{\theta F_{2,1}}{\alpha(x+v)}. \quad (10)$$

Substituting (8a) and (8c) into (10) yields R_0 as a function of the disease-free equilibrium occupancies:

$$R_0 = \frac{\psi c}{(1+(\phi-1)f)(x+v)}\left[1 - f - S_U + \frac{\theta\phi}{\alpha}(f - S_P) + \delta\left(S_U + \frac{\theta\phi}{\alpha}S_P\right)\right] \quad (11)$$

Next we need to eliminate the S_U and S_P terms so that R_0 is a function only of model parameters. Evaluating (6a) and (6c) at the disease-free equilibrium yields

$$\frac{c}{1+(\phi-1)f}(1-f-S_U)(S_U+\theta S_P)=xS_U \quad (12a)$$

$$\frac{\phi c}{1+(\phi-1)f}(f-S_P)(S_U+\theta S_P)=\alpha x S_P \quad (12b)$$

Taking $(12a)+\theta/\alpha\times(12b)$, and dividing by $S_U+\theta S_P$ yields

$$\frac{c}{1+(\phi-1)f}\left(1-f-S_U+\frac{\theta\phi}{\alpha}(f-S_P)\right)=x \quad (13a)$$

which can be rearranged to

$$\frac{c}{1+(\phi-1)f}\left(S_U+\frac{\theta\phi}{\alpha}S_P\right)=\frac{c}{1+(\phi-1)f}\left(1+\left(\frac{\theta\phi}{\alpha}-1\right)f\right)-x. \quad (13b)$$

Substituting (13a) and (13b) into (11) yields

$$R_0=\frac{\psi}{x+v}\left[x+\delta\left(\frac{c}{1+(\phi-1)f}\left(1+\left(\frac{\theta\phi}{\alpha}-1\right)f\right)-x\right)\right] \quad (14)$$

We can use expression (3) to directly relate the basic reproductive number to the metapopulation's lifetime colonization success:

$$R_0=\frac{\psi x}{x+v}(1+(\rho-1)\delta) \quad (15)$$

Finally, by setting $R_0 = 1$ in (15) and substituting for ρ using (4), we can rearrange to find an expression for the threshold fraction of patches at which the pathogen can invade and attain an endemic equilibrium (f_e):

$$f_e = \left(1 + \frac{\phi\left(1 - \frac{\theta c}{\alpha x} + \frac{1}{\delta}\left(\frac{x+v}{\psi x} - 1\right)\right)}{\frac{c}{x} - 1 - \frac{1}{\delta}\left(\frac{x+v}{\psi x} - 1\right)} \right)^{-1} . \quad (16)$$

Below we describe each scenario for metapopulation persistence and pathogen invasion for unprovisioned ($f = 0$) and fully provisioned ($f = 1$) metapopulations, and what this means for pathogen invasion for partially provisioned metapopulations ($0 < f < 1$).

(i) When the unprovisioned metapopulation can't persist ($c < x$), necessary conditions for pathogen invasion are that the fully provisioned metapopulation persists ($\theta c/\alpha x > 1$), and pathogen invasion is possible (i.e $R_0(f = 1) > 1$); in this case pathogen invasion occurs when $f > f_e > f_{df}$.

(ii) When the unprovisioned metapopulation persists ($c > x$), and the pathogen is unable to establish ($R_0(f = 0) < 1$); then we have the same necessary conditions as above and invasion occurs when $f > f_e > 0$.

(iii) When the unprovisioned metapopulation persists, and the pathogen can establish ($R_0(f = 0) > 1$), the pathogen can always invade provided ($R_0(f = 1) > 1$). Note that this is always true when provisioning increases the lifetime number of dispersers ($\theta/\alpha > 1$).

(iv) If $R_0(f = 1) < 1$ and the fully provisioned metapopulation persists ($\theta c/\alpha x > 1$), the pathogen can only invade when $f < f_e < 1$.

(v) If $R_0(f = 1) < 1$ and the fully provisioned metapopulation cannot persist ($\theta c/\alpha x < 1$), the pathogen can only invade when $f < f_e < f_{df}$.

APPENDIX E

CHAPTER 6 SUPPLEMENTAL INFORMATION

ES1 Site coordinates and livestock density

Table S6.1. Site geography, coordinates, livestock biomass, elevation, and sampling history.

Site	Department	Country	Longitude	Latitude	Livestock biomass (kg)[1]	Elevation (m)	Captures	Nights
LR3	Loreto	Peru	-73.218	-4.307	3.530	100	21	7
LR2	Loreto	Peru	-73.199	-4.290	4.201	100	23	8
LR4	Loreto	Peru	-73.204	-4.211	4.271	100	17	7
AM1	Amazonas	Peru	-78.292	-5.212	6.704	660	57	13
AM2	Amazonas	Peru	-78.290	-5.212	6.704	660	3	2
AM3	Amazonas	Peru	-78.288	-5.201	6.704	660	26	4
LR1	Loreto	Peru	-73.303	-4.241	7.109	100	17	7
OW1[2]	Orange Walk	Belize	-88.731	17.816	7.692	30	50	9
CA1	Cajamarca	Peru	-78.791	-5.077	8.661	1702	103	12
OW2[2]	Orange Walk	Belize	-88.654	17.753	8.793	11	72	11

[1]Log transformed

[2]Based out of the Lamanai Field Research Center

ES2 Stable isotopes of bats and prey

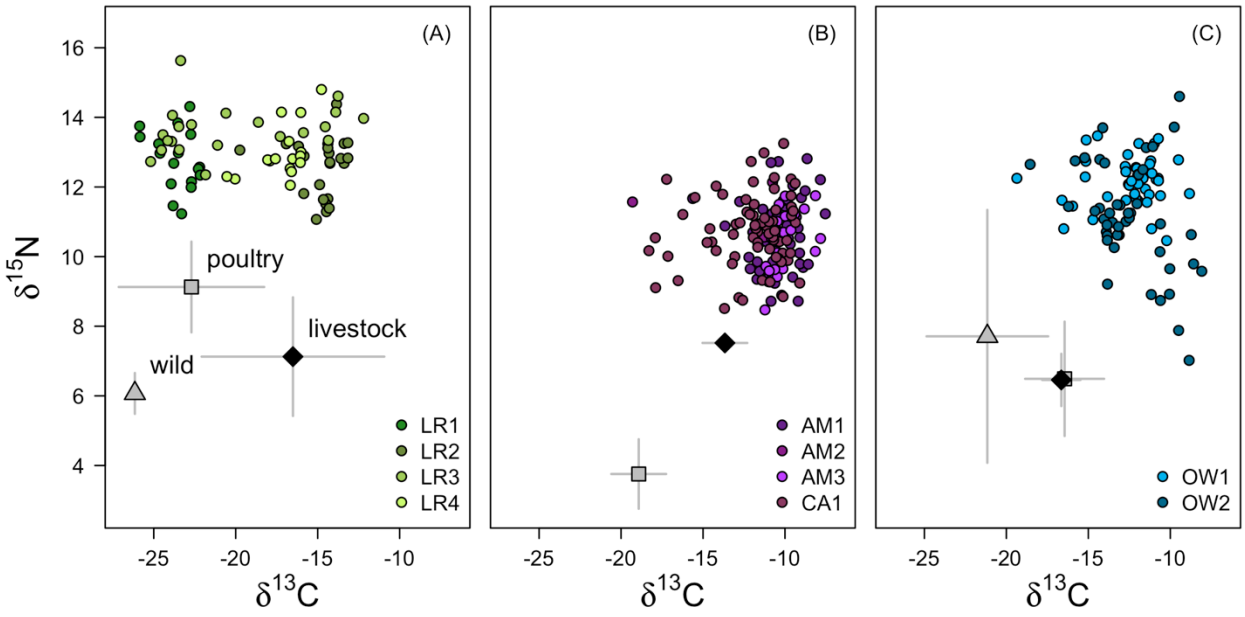

Figure S6.1. Bat hair isotopes of $\delta^{13}C$ and $\delta^{15}N$ per region. Points are colored by sampling site and displayed by study region (A=Loreto, B=Amazonas/Cajamarca, C=Belize). The means and standard deviations of $\delta^{13}C$ and $\delta^{15}N$ from potential prey groupings per region are displayed in grey (triangle=wildlife, square=poultry), with livestock shown in the black diamonds.

ES3 Multivariate analysis of immune function

Table S6.2. Pearson correlation coefficients between measures of immune function (*n*=166).

	N	L	M	E	B	TWBC	BKA	IgG
N[1]	1							
L[2]	–0.98	1						
M[3]	–0.38	0.27	1					
E[4]	–0.23	0.14	0.30	1				
B[5]	–0.02	–0.01	0.05	–0.06	1			
TWBC[6]	0.15	–0.18	0.14	0.13	–0.07	1		
BKA[7]	0.23	–0.22	–0.18	–0.15	0.12	0.16	1	
IgG[8]	–0.20	0.18	0.10	0.10	0.06	–0.08	0.03	1

[1]Percent neutrophils from blood smears

[2]Percent lymphocytes from blood smears

[3]Percent monocytes from blood smears

[4]Percent eosinophils from blood smears

[5]Percent basophils from blood smears

[6]Quarter-root transformed total WBC estimates

[7]Percent *E. coli* killed in plasma relative to positive control

[8]Optical density of immunoglobulin G antibody in plasma

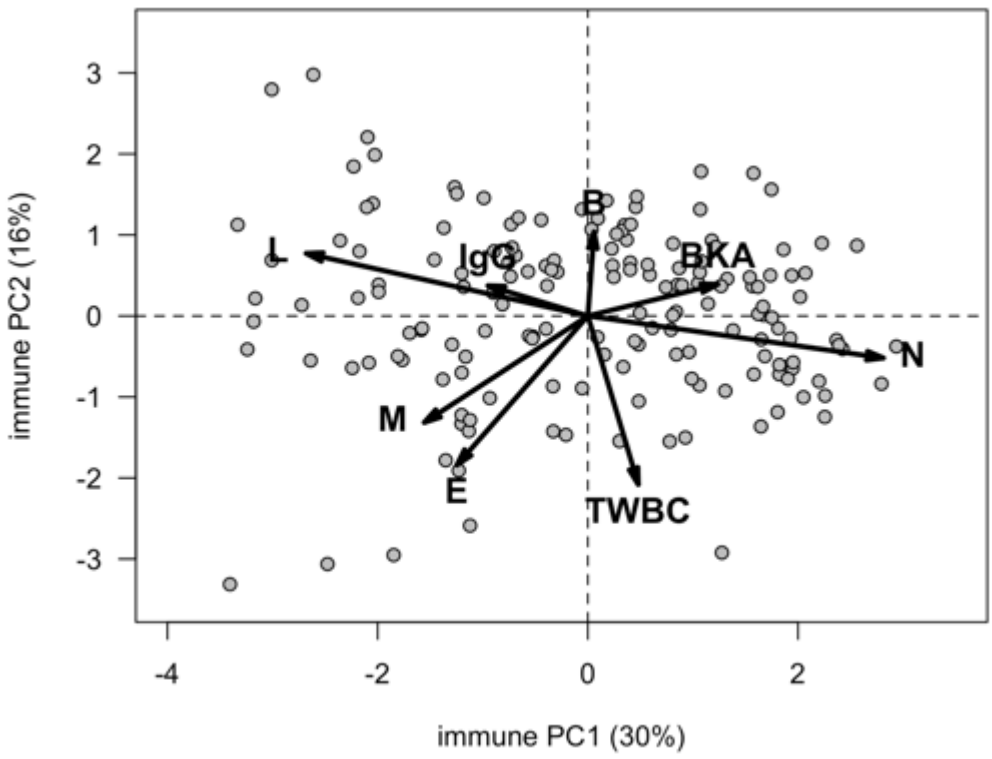

Figure S6.2. Biplot of the first two PCs of eight immune measures. Arrows indicate PC loadings.

ES4 Livestock biomass and isotopic distance

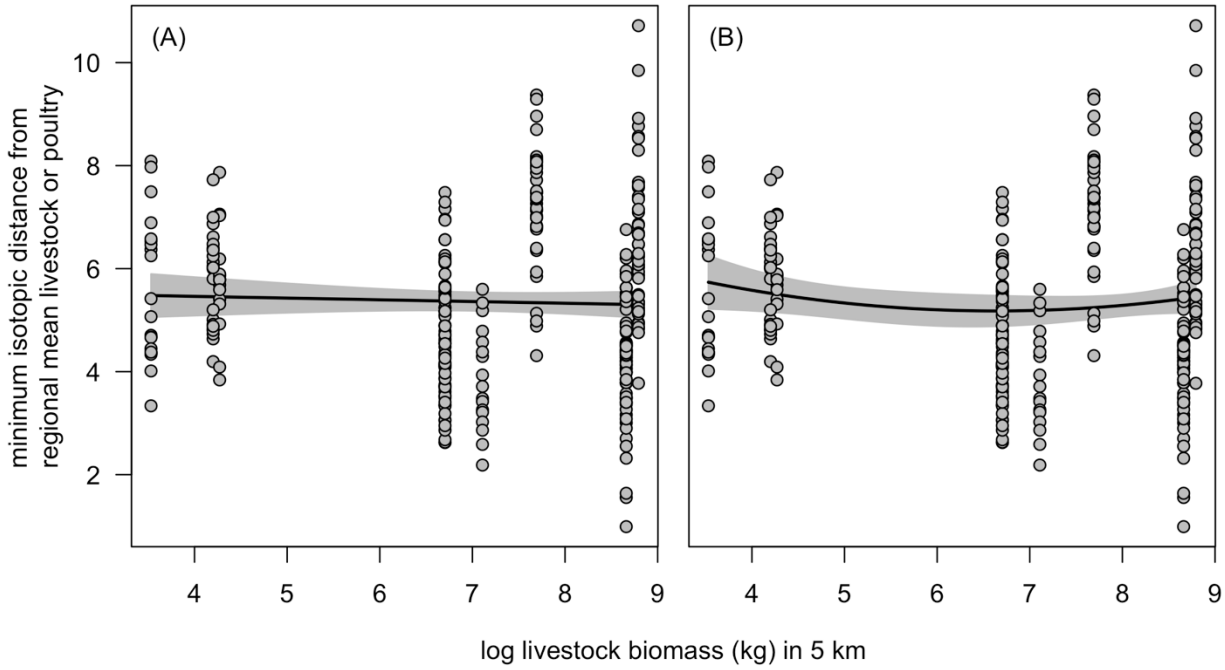

Figure S6.3. Relationships between livestock biomass and the minimum bat isotopic distance from mean livestock or poultry prey per study region. The curves show the REML fit from the LMMs.

ES5 PC1 model selection

Table S6.3. 95% confidence set of LMMs predicting the immune component PC1. LMMs are ranked by ΔAICc with the renormalized Akaike weights (w_i), number of estimated coefficients (k), and marginal and conditional r^2 statistics. A random effect of bat ID is denoted by (1|ID).

Immune function LMM	***k***	**ΔAICc**	w_i	r^2_m	r^2_c
PC1 ~ reproduction + livestock biomass + (1|ID)	3	0.00	0.203	0.36	0.36
PC1 ~ isotopic distance + reproduction + livestock biomass + (1|ID)	4	1.76	0.084	0.36	0.36
PC1 ~ age + reproduction + livestock biomass + (1|ID)	4	2	0.075	0.36	0.36
PC1 ~ reproduction + sex + livestock biomass + (1|ID)	4	2.14	0.07	0.35	0.35
PC1 ~ reproduction + livestock biomass + (1|ID) + reproduction:livestock biomass	4	2.16	0.069	0.35	0.35
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:sex	6	3.68	0.032	0.36	0.42
PC1 ~ isotopic distance + reproduction + livestock biomass + (1|ID) + isotopic distance:reproduction	5	3.82	0.03	0.36	0.36
PC1 ~ age + isotopic distance + reproduction + livestock biomass + (1|ID)	5	3.82	0.03	0.36	0.36
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID)	5	3.9	0.029	0.36	0.36
PC1 ~ isotopic distance + reproduction + livestock biomass + (1|ID) + reproduction:livestock biomass	5	3.95	0.028	0.36	0.36
PC1 ~ reproduction + sex + livestock biomass + (1|ID) +	5	4.06	0.027	0.35	0.35

sex:livestock biomass					
PC1 ~ age + reproduction + sex + livestock biomass + (1|ID)	5	4.18	0.025	0.35	0.35
PC1 ~ age + reproduction + livestock biomass + (1|ID) + reproduction:livestock biomass	5	4.19	0.025	0.35	0.35
PC1 ~ reproduction + sex + livestock biomass + (1|ID) + reproduction:livestock biomass	5	4.33	0.023	0.35	0.35
PC1 ~ age + reproduction + sex + livestock biomass + (1|ID) + age:sex	6	5.54	0.013	0.36	0.36
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:reproduction + isotopic distance:sex	7	5.61	0.012	0.36	0.41
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + sex:livestock biomass	6	5.86	0.011	0.35	0.35
PC1 ~ age + isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:sex	7	5.89	0.011	0.36	0.42
PC1 ~ age + isotopic distance + reproduction + livestock biomass + (1|ID) + isotopic distance:reproduction	6	5.9	0.011	0.35	0.35
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:sex + reproduction:livestock biomass	7	5.92	0.011	0.36	0.43
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:sex + sex:livestock	7	5.93	0.01	0.36	0.42

biomass					
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:reproduction	6	6	0.01	0.35	0.36
PC1 ~ age + isotopic distance + reproduction + sex + livestock biomass + (1|ID)	6	6.02	0.01	0.35	0.35
PC1 ~ age + isotopic distance + reproduction + livestock biomass + (1|ID) + reproduction:livestock biomass	6	6.03	0.01	0.35	0.35
PC1 ~ isotopic distance + reproduction + livestock biomass + (1|ID) + isotopic distance:reproduction + reproduction:livestock biomass	6	6.04	0.01	0.35	0.35
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + reproduction:livestock biomass	6	6.12	0.01	0.35	0.35
PC1 ~ age + reproduction + sex + livestock biomass + (1|ID) + sex:livestock biomass	6	6.16	0.009	0.35	0.35
PC1 ~ reproduction + sex + livestock biomass + (1|ID) + reproduction:livestock biomass + sex:livestock biomass	6	6.19	0.009	0.35	0.35
PC1 ~ age + reproduction + sex + livestock biomass + (1|ID) + reproduction:livestock biomass	6	6.4	0.008	0.35	0.35
PC1 ~ age + isotopic distance + reproduction + sex + livestock biomass + (1|ID) + age:sex + isotopic distance:sex	8	7.06	0.006	0.36	0.44
PC1 ~ age + isotopic distance + reproduction + sex + livestock biomass + (1|ID) + age:sex	7	7.31	0.005	0.36	0.36

PC1 ~ age + reproduction + sex + livestock biomass + (1|ID) + age:sex + sex:livestock biomass	7	7.52	0.005	0.36	0.36
PC1 ~ age + reproduction + sex + livestock biomass + (1|ID) + age:sex + reproduction:livestock biomass	7	7.79	0.004	0.35	0.35
PC1 ~ age + isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:reproduction + isotopic distance:sex	8	7.87	0.004	0.36	0.41
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:reproduction + isotopic distance:sex + sex:livestock biomass	8	7.9	0.004	0.36	0.41
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:reproduction + isotopic distance:sex + reproduction:livestock biomass	8	7.9	0.004	0.36	0.41
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + isotopic distance:reproduction + sex:livestock biomass	7	8.02	0.004	0.35	0.35
PC1 ~ age + isotopic distance + reproduction + sex + livestock biomass + (1|ID) + sex:livestock biomass	7	8.02	0.004	0.35	0.35
PC1 ~ isotopic distance + reproduction + sex + livestock biomass + (1|ID) + reproduction:livestock biomass + sex:livestock biomass	7	8.04	0.004	0.35	0.35

ES6 Sensitivity to holding time

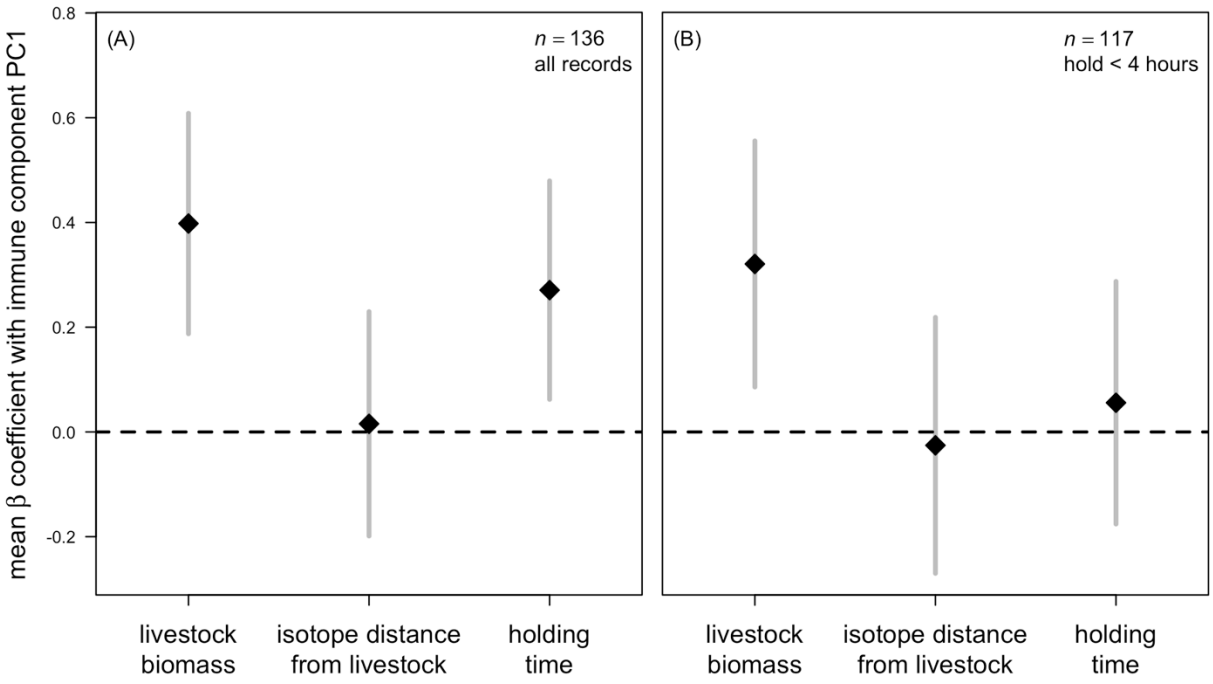

Figure S6.4. Results from model averaging where holding time was included as a covariate in all LMMs (A). Results where we restricted data to only bats held for under four hours (B). The 95% confidence intervals are shown in grey and mean coefficients are shown in black diamonds. The dashed line represents no correlation between covariates and bat immunity PC1 (β=0).

ES7 Correlation between demography and immunity

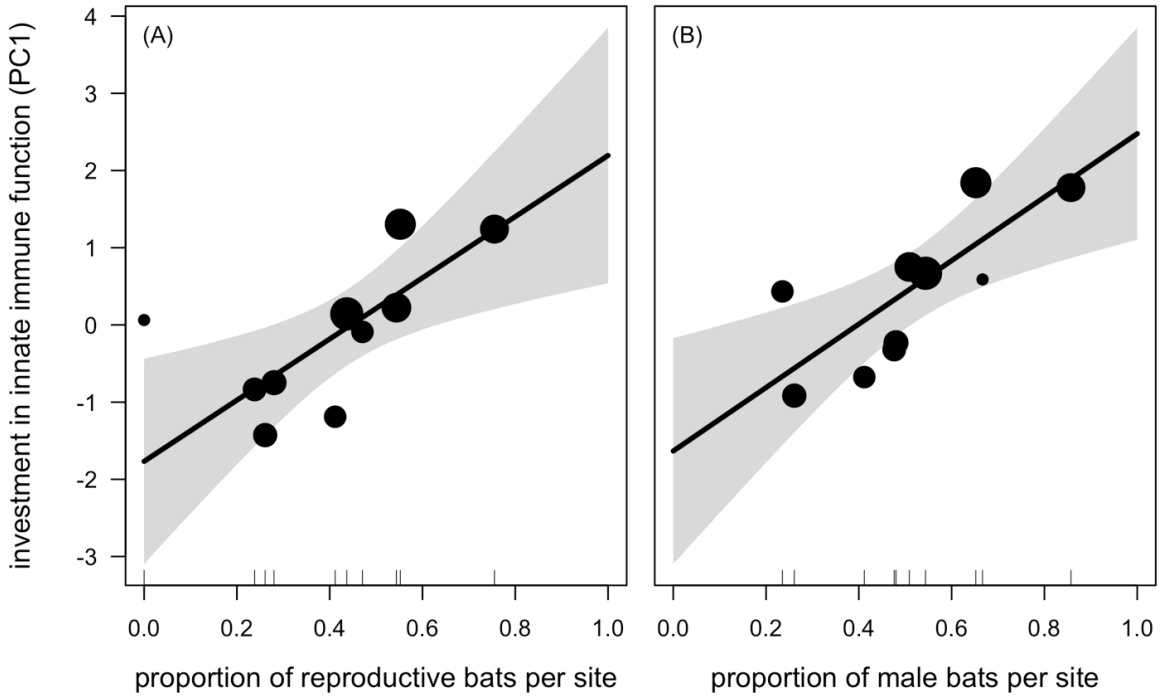

Figure S6.5. Relationships between vampire bat demography and immune profiles (immune component PC1). Associations between mean site PC1 and (A) the proportion of reproductive bats and (B) male bats captured per site. Points are scaled by the log sample size per site. Curves and grey shading display the fit and 95% confidence interval from weighted linear regressions.

ES8 Univariate infection GLMMs

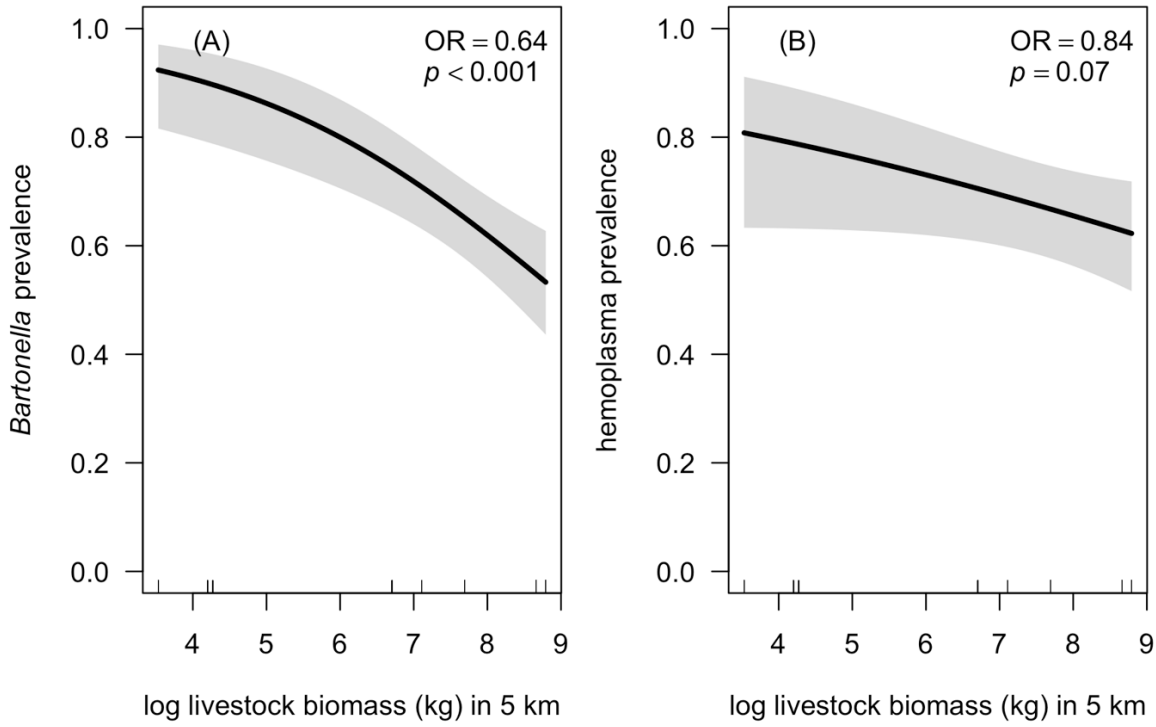

Figure S6.6. Predictions and 95% confidence intervals for univariate GLMMs modeling the relationship between livestock biomass and infection with *Bartonella* (A) and hemoplasmas (B).