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# Modeling the interactive effects of predation and infection in food-subsidized wildlife populations

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**Abstract**

Feeding wildlife is a globally popular activity, but direct nutritional benefits of feeding may be offset by elevated mortality risk from predators and infectious diseases. Effects of food subsidy and predation on infection dynamics are typically studied in isolation, but their interactions could have synergistic or antagonistic effects on wildlife mortality. We developed a mathematical model of wildlife-pathogen interactions around feeders in the presence of predators, where food subsidy can alter host movement and survival, pathogen transmission, and predation risk. We derive general analytical conditions for the ability of predators to prevent pathogen outbreaks under food subsidy and the net effect of predators on host mortality. We found that predators most effectively prevent outbreaks when subsidies decrease host susceptibility to infection and are least effective when subsidies increase host abundance at feeders. Even when predators cannot prevent outbreaks, they can reduce overall mortality by regulating host numbers near feeding stations, thus slowing pathogen transmission and mortality. We then applied our model to a well-studied bird-feeding system, mycoplasmal conjunctivitis in House Finches, where variation in pathogen virulence influences the relative predation risk of infected birds. When feeding increases host density and contacts, feeding can reduce, but not overcome, additional mortality from predators in the presence of a low-virulence pathogen strain. For high-virulence pathogen strains, predators can slightly reduce overall mortality at feeders by reducing transmission, but these effects are reduced when food subsidy increases host immunity, contacts, and movement. These findings highlight the importance of understanding community interactions around anthropogenic food subsidies.

Keywords: food subsidy, model, disease, predation, bird feeding

## Introduction

Providing food resources for wildlife, either accidentally through agriculture or intentionally through the deliberate feeding of wildlife, can have profound effects on animal ecology, behavior, and health (Robb et al. 2008; Oro et al. 2013; Murray et al. 2016). Nutritionally appropriate food subsidies can have direct fitness benefits, including improved body condition and increased survival probability through harsh conditions (Becker and Hall 2014; Murray et al. 2016). However, increases in wildlife abundance around food subsidies can also intensify interactions with natural enemies, such as infectious diseases and predators. Increased aggregation, population growth rates, and site fidelity in response to food subsidies can all increase the transmission of infectious diseases between individuals (Becker et al. 2016; Teitelbaum et al. 2022); however, immune benefits derived from consuming quality food can reduce infection impacts, and reduced dispersal from regularly provisioned sites can reduce spatial spread of infection (Becker and Hall 2014; Becker et al. 2018). Higher densities, decreased movement rates, and increased spatio-temporal predictability of wildlife around food subsidies can also attract predators (Roth and Vetter 2008), and increased fat reserves due to food provisioning can augment predation rates on fed wildlife (Krama et al. 2023). Since provisioning can simultaneously influence both infectious disease and predation risk, and wildlife are increasingly using human-altered habitats where subsidies are common, understanding food subsidy effects on concurrent species interactions from individual to population scales is crucial.

While past research has explored the connections between food provisioning and species interactions, such as parasitism or predation, few empirical studies have considered the effects of food subsidy on multiple species interactions concurrently. The Healthy Herds Hypothesis suggests that predators can reduce infection prevalence by reducing host population size and, therefore, density-dependent transmission (Richards et al. 2022). Alternatively, predators have the potential to increase infection transmission through predator-induced changes in prey behavior (e.g., reduced foraging and increased flocking) that enhance prey susceptibility to disease (Duffy et al. 2011). Food subsidy has the potential to either intensify or mitigate predator and pathogen effects on wildlife. For instance, predator and pathogen effects on population size can be mitigated through increased immigration or reproduction of prey in response to food subsidy. Conversely, predator and pathogen effects on population size can be intensified through increased infection transmission and predation success. Civitello et al. (2018) investigated the effects of food provisioning, predators, and competitors on wildlife disease dynamics in an aquatic invertebrate–fungal disease system, finding that food provisioning is most likely to aggregate hosts and increase disease transmission, while predators can decrease infection prevalence. However, little is known about how food subsidy influences the concurrent effects of predators and pathogens on host mortality.

A globally popular type of resource provisioning is residential bird feeding, with goals that include supporting declining bird populations and providing benefits to the mental wellbeing of the people that own bird feeders (Cox and Gaston 2016). Bird-feeding is associated with outbreaks of pathogens in feeder-visiting finches, including mycoplasmal conjunctivitis in North America, trichomoniasis in Europe, and *Salmonella* associated with the irruptive movements of Pine Siskins that have resulted in human cases (Fischer et al. 1997; Anderson et al. 2009; Söderlund et al. 2019). In addition to direct disease-induced mortality, these pathogens can

induce sub-lethal effects including lethargy, impaired vision, and increased time at bird feeders, which can lead to elevated predation risk. It remains unclear whether predators increase overall mortality during disease outbreaks or reduce mortality through selective culling of infected hosts that reduces pathogen transmission. Further, the observation of predation and infection can evoke negative emotional responses and lead to management actions taken (e.g., feeder cleaning and chasing off predators) by people who feed birds (Dayer et al. 2019), which may or may not be helpful for bird populations. Therefore, studies are urgently needed to quantify the impacts of these interactions on both birds and the people who watch them (Dayer et al. 2024).

Mathematical models provide a useful approach for understanding the population impacts of changes to host behavior and survival associated with food availability and natural enemies, especially since mortality from infection or predation is imperfectly observed (Hosseini et al. 2004). Here we develop a mathematical model to explore how bird feeding influences population, infection, and predation dynamics in birds visiting feeders. We first conduct an equilibrium analysis to derive general conditions for when predators can prevent outbreaks under different food subsidy effects on host, pathogen, and predation processes and when predators can reduce host mortality via a reduction in infection impacts on hosts. We then apply our model to mycoplasmal conjunctivitis (*Mycoplasma gallisepticum*) in House Finches (*Haemorhous mexicanus*) in North America, a highly visible and commonly observed disease transmitted at bird feeders that can alter behavior and predation risk. We explore how strain variation in the pathogen can influence predator effects on mortality over a 5-month nonbreeding season. We discuss the implications of our results for the management of feeders to reduce negative impacts on birds and other wildlife that receive food subsidies.

## Methods

### 1. Model

We developed a differential equation model tracking the number of Susceptible ($S$) and Infected ($I$) hosts through time, as well as the cumulative number of hosts dying ($C$) from infection, predation, or natural causes. Hosts arrive at food subsidies at a constant immigration rate ($\lambda$) and emigrate at a constant per capita rate ($m$). Uninfected hosts experience natural mortality at a constant per capita rate ($\mu$) and are predated at a constant per capita rate ($\pi$), which accounts for predator-host contact, the likelihood a predator attempts an attack, and predation success. We assume pathogen transmission is density dependent, and the transmission rate per susceptible-infected encounter is the product of the host contact rate ($\alpha$) and host susceptibility ($\delta$) (i.e., probability of infection given contact). Infected hosts recover and return to the susceptible class at a constant per capita rate ($\gamma$). Infected hosts experience additional disease-induced mortality at per capita rate $\nu$, and their risk of predation relative to uninfected hosts is represented by $\theta$ (so that $\theta > 1$ represents higher predation risk of infected hosts relative to susceptible hosts). We also assume that infected hosts do not disperse away and remain at subsidized sites until they recover from infection. Food subsidy effects are modeled as the fold increase or decrease in parameters influencing host population, infection, and predation processes due to changes in host or predator foraging behavior and aggregation, or host condition and immunity; the associated parameters that can be influenced by food in the model have a subscript $F$. In other words,

$$P_F = \begin{cases} P * F & \text{if parameter increases due to food subsidy} \\ P \div F & \text{if parameter decreases due to food subsidy} \end{cases}$$

where $F$ is the fold change in the parameter due to food subsidy and $P$ is the value of the parameter in the absence of food subsidy.

The model (Fig. 1a) is described by the following system of equations:

$$\frac{dS}{dt} = \lambda_F - m_F S - \mu_F S - \pi_F S - \alpha_F \delta_F SI + \gamma_F I \quad (1)$$

$$\frac{dI}{dt} = \alpha_F \delta_F SI - \mu_F I - \theta \pi_F I - v_F I - \gamma_F I \quad (2)$$

$$\frac{dC}{dt} = \mu_F S + \pi_F S + \mu_F I + \theta \pi_F I + v_F I \quad (3)$$

We allowed food subsidies to affect individual parameters relating to movement, survival, predation, and infection. The direction of the effect of food subsidy on each parameter was based on reported or hypothesized associations from the published literature (Becker and Hall 2014; Aberle et al. 2020; Krama et al. 2023).

## 2. Mathematical Analysis

### i. Pathogen Outbreak Potential

To understand how food subsidy and predation influence outbreak potential, we calculated the pathogen basic reproduction number ($R_0$), which represents the number of new infections caused by one infectious individual introduced to a wholly susceptible population (Anderson 1982). The expression for pathogen outbreak potential under predation (indicated with a subscript "p") can be derived by solving equation (1) for the disease-free equilibrium $S_{df}$ ($\frac{dS}{dt} = 0$, $I = 0$), then substituting $S_{df}$ into equation (2) and evaluating for an increase in the number of infected hosts (i.e., $\frac{dI}{dt} > 0$ when $S = S_{df}$), and rearranging:

$$R_{0p} = \frac{\alpha_F \delta_F \lambda_F}{(\mu_F + v_F + \gamma_F + \theta \pi_F)(m_F + \mu_F + \pi_F)} \quad (4)$$

This expression is the product of the pathogen transmission rate, $\alpha_F \delta_F$, the infectious period, $1/(\mu_F + v_F + \gamma_F + \theta \pi_F)$, and the disease-free host population size, $S_{df} = \lambda_F/(m_F + \mu_F + \pi_F)$ under food subsidy and predation.

In the absence of predation ($\pi_F = 0$), the predator-free outbreak potential, indicated with a subscript "np", $R_{0np}$ is:

$$R_{0np} = \frac{\alpha_F \delta_F \lambda_F}{(\mu_F + v_F + \gamma_F)(m_F + \mu_F)} \quad (5)$$

To understand under what conditions predators could prevent pathogen invasion, we identified the minimum predation rate ($\pi_{min}$) necessary to prevent pathogen invasion (i.e., the predation rate that results in $R_{0p} = 1$ in equation 4) under various food subsidy effects. For each parameter altered by food subsidy, we solved for $\pi_{min}$ when $R_{0p} = 1$ and simplified the resulting

equation to examine the qualitative dependence of $\pi_{\min}$ on the food subsidy effect size ($F$) (S2 in the Supplement).

### ii. Effects of Predators on Host Mortality

Predators can both directly increase feeder bird mortality and, by reducing transmission of virulent pathogens, reduce disease-induced mortality. To determine the net effect of predators on host mortality, we calculated the daily death difference (DDD) (equation 6) as the difference between the number of hosts dying per day (equation 3) when the model reaches equilibrium ($\frac{dS}{dt} = \frac{dI}{dt} = 0$) in the presence and absence of predators. Using subscripts "p" and "np" to denote the equilibrium values of variables in the presence and absence of predators, respectively, this can be written as:

$$DDD = \frac{dCp}{dt} - \frac{dCnp}{dt} = \mu_F(S_p - S_{np}) + \pi_F S_p + \mu_F(I_p - I_{np}) + \theta\pi_F I_p + v_F(I_p - I_{np}) \quad (6)$$

A positive value of *DDD* indicates that predators increase daily host mortality, while a negative value indicates that predators reduce daily host mortality. For each parameter affected by food subsidy, we took the partial derivative of the resulting daily death difference equation with respect to $F$ to better understand how the effect of predators on host mortality changes with increasing food subsidy effect size.

## 3. Application to the House Finch-Mycoplasmal Conjunctivitis System

To determine the net effect of predators on outbreak potential and feeder bird mortality, we applied our model to a well-studied host-pathogen system in North America, conjunctivitis caused by the bacterial pathogen *Mycoplasma gallisepticum* (hereafter *Mg*) in House Finches (*Haemorhous mexicanus*). This system is ideally suited to explore host-pathogen-predation interactions because House Finches easily transmit bacteria at bird feeders and published field and laboratory experiments, along with continent-wide citizen science studies, provide extensive information to parameterize our model (Williams et al. 2014; Flemings-Davies et al. 2018). Notably, there is variation in virulence among *Mg* strains that influence pathogen transmissibility, disease-induced mortality, recovery, and predation risk that can be estimated by visually assessing eye pathology of infected birds (i.e., size of eye swelling and crust covering one or both eyes). Pathogen virulence can influence whether infected birds experience higher predation risk: infection with low virulence *Mg* strains result in low eye pathology that likely do not increase predation risk, while high virulence infections result in lethargy and impaired vision that likely exacerbate predation risk by hawks and other predators (Williams et al. 2014; Adelman et al. 2017).

### *Model Scenarios*

We explored the following combinations of pathogen and food subsidy effects in a factorial design (Fig. 1b).

*Pathogen scenarios*: We considered low virulence and high virulence pathogen strains, where strain virulence was determined from data on eye lesion scores caused by *Mg* in House Finches in the eastern United States (described in S1 of the Online Resource). The low virulence strain was parameterized so uninfected and infected birds experienced no difference in predation

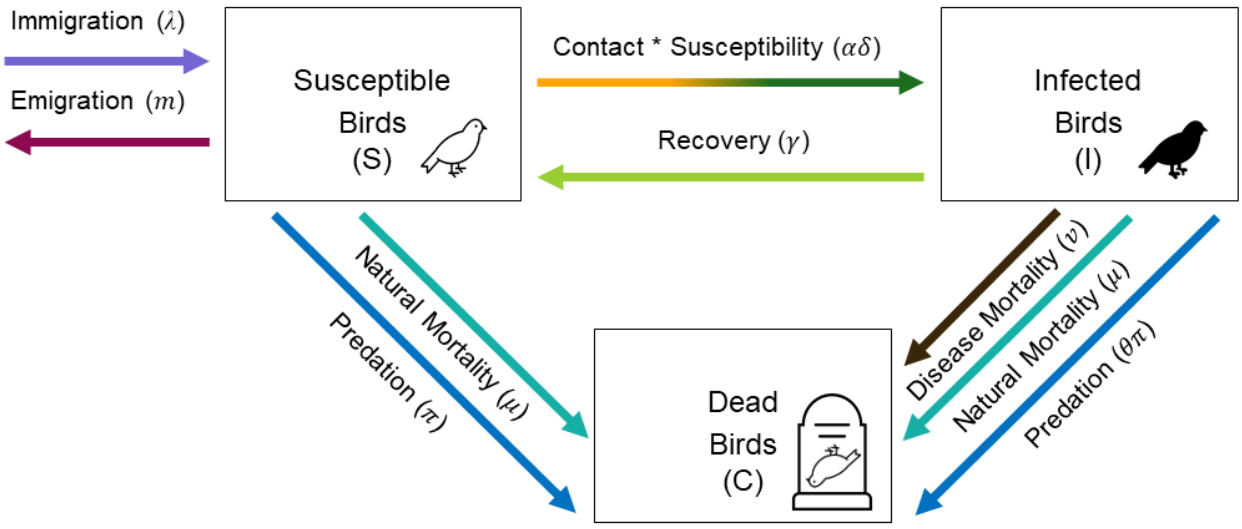
risk. Additionally, infected birds experienced a low disease-induced mortality rate, fast recovery from infection, and overall transmission rates were relatively low. The high virulence strain was parameterized to reflect increased vulnerability to predation of infected birds, high disease-induced mortality, slow recovery from infection, and higher transmissibility.

*Food subsidy scenarios*: We allowed food subsidy to affect one host demographic, infection, or predation parameter at a time. For each affected parameter, we considered food subsidy effects over a range of $F = 1$ (no effect) to $F = 5$ (i.e., a 5-fold increase or decrease in the affected parameter). We considered that food subsidies could increase host attraction to, or decrease departure from, subsidized sites, increase predation and pathogen exposure due to reliable host aggregations at feeders, potentially reduce natural or disease-induced mortality, and increase recovery from infection due to improvements to condition and immunity arising from reliable access to food. The direction of the food subsidy effect on each parameter is summarized in Table 1.

**Table 1.** Definition of the model parameters, the direction of the effect food subsidy has on the parameter (+ = increase, - = decrease, 0 = not affected by subsidy), and their default (subsidy free) value.

| Parameter | Definition | Food Effect | Value |
|---|---|---|---|
| $\lambda$ | Host immigration. | + | 0.48 |
| $m$ | Uninfected host emigration rate. | - | 0.03 |
| $\mu$ | Host natural mortality rate. | - | 0.004 |
| $\pi$ | Predation rate of uninfected hosts. | + | 0.002 |
| $\theta$ | Predation risk of infected hosts relative to uninfected hosts ($\theta \geq 1$). | 0 | 1 (Low virulence strain)<br>3 (High virulence strain) |
| $v$ | Disease induced host mortality rate. | - | 0.01 (Low virulence strain)<br>0.25 (High virulence strain) |
| $\alpha$ | Contact rate between uninfected and infected hosts. | + | 1 |
| $\delta$ | Probability of infection given contact. | - | 0.004 (Low virulence strain)<br>0.024 (High virulence strain) |
| $\gamma$ | Infection recovery rate. | + | 0.086 (Low virulence strain)<br>0.003 (High virulence strain) |

(a)




(b)

| | Low Virulence Strain | High Virulence Strain |
|---|---|---|
| **No Predation** | High recovery ($\gamma$ = 0.086)<br>Low transmission ($\beta$ = 0.004)<br>Low disease induced mortality ($\upsilon$ = 0.01)<br>No predation ($\pi$ = 0) | Low recovery ($\gamma$ = 0.003)<br>High transmission ($\beta$ = 0.024)<br>High disease induced mortality ($\upsilon$ = 0.25)<br>No predation ($\pi$ = 0) |
| **Predation** | High recovery ($\gamma$ = 0.086)<br>Low transmission ($\beta$ = 0.004)<br>Low disease induced mortality ($\upsilon$ = 0.01)<br>Low predation risk ($\theta$ = 1) | Low recovery ($\gamma$ = 0.003)<br>High transmission ($\beta$ = 0.024)<br>High disease induced mortality ($\upsilon$ = 0.25)<br>High predation risk ($\theta$ = 3) |

**Fig. 1** Model Schematic and Crossed Experimental Design. (**a**) Differential equations track the number of Susceptible, Infected, and Dead birds (boxes) through movement, host mortality, and pathogen infection (arrows). (**b**) Cells represent different combinations of predation and pathogen virulence

For each of the pathogen strains and food subsidy effect sizes described above, we quantified the impacts of predators and food subsidy on House Finches by calculating the percent change in cumulative host mortality over a nonbreeding season between feeding stations with and without predators. To do this, we solved the differential equation model in R Version 4.2.2 (using the *lsoda* function in the *desolve* package) and recorded the cumulative mortality ($C$) through time (in days) over a 150-day period (the approximate length of a nonbreeding season in temperate North America). All models were initiated with the host at its disease-free equilibrium population size, $S_{df}$, and we introduced one infected individual ($S_0 = S_{df} - 1$, $I_0 = 1$).

## Results

### Question 1: When can predators prevent outbreaks?

#### *Theory*

Food subsidies that increase host immigration or contact rates, decrease host emigration, or decrease natural or disease-induced mortality rates, can make it harder for predators to prevent outbreaks (i.e., a higher predation rate, $\pi_{min,}$ is needed to bring $R_{0p}$ below one; Fig. 2a). While $\pi_{min}$ increases and saturates with increasing food subsidy effects on host emigration and mortality, $\pi_{min}$ continues to increase monotonically with food subsidy-increased immigration or contact rates, meaning that the predation rate necessary to bring $R_{0p}$ below one eventually becomes impossible for predators to achieve if the strength of the food subsidy effect on host immigration or contact is sufficiently large.

Food subsidies can make it easier for predators to prevent outbreaks (i.e., a lower $\pi_{min}$ is required to bring $R_{0p}$ below one) when food subsidies decrease host susceptibility to infection, increase recovery from infection, or increase predation success (Fig. 2b). While strong food subsidy effects on susceptibility and recovery can prevent outbreaks in the absence of predators, the relationship between $\pi_{min}$ and food subsidy effect on predation success asymptotically decreases towards zero; this means that when food subsidy increases predation success, predators only can only reduce, but not prevent, pathogen outbreaks.

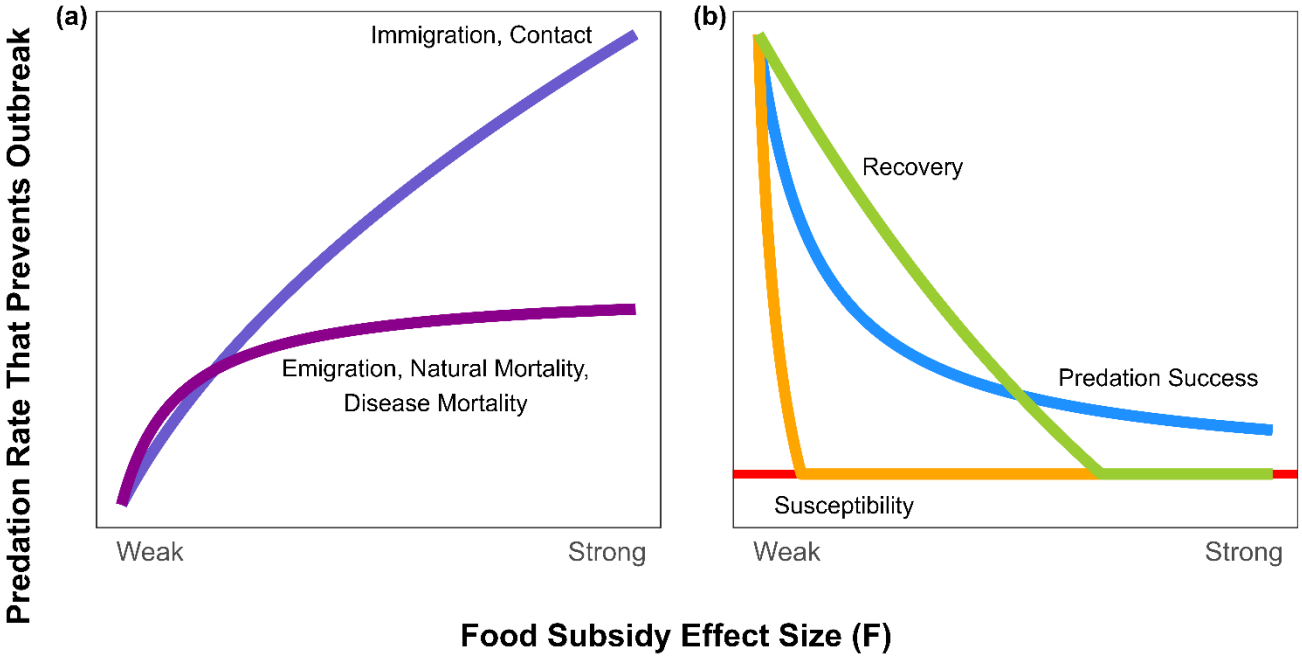



**Fig. 2** The qualitative behavior of the predation rate ($\pi_{min}$) necessary to prevent an outbreak (i.e., to reduce the basic reproduction number, $R_{0p}$, to 1) as the strength of food subsidy effects ($F$) on host, pathogen and predation parameters (colored lines) increase. Equations underlying the relationships between $\pi_{min}$ and $F$ are shown in S2 in the Supplement. (**a**) Increases in host immigration or contact rates, and reductions in host emigration or mortality, make it harder for

predators to prevent outbreaks under food subsidy. (**b**) Reductions in host susceptibility, or increases in host recovery or predation success under food subsidy, make it easier for predators to prevent outbreaks. The horizontal red line indicates that $\pi_{min} = 0$ and $R_{0p} < 1$ even in the absence of predators

***Application to the House Finch – Mg system***

The qualitative relationships between the predation rate necessary to prevent pathogen invasion ($\pi_{min}$) and the strength of food subsidy effects matched the analytically-derived generalized results (Fig. 2) for both low virulence (L) and high virulence (H) strains of *Mg* (Fig. 3). However, in the empirically parameterized system, the pathogen strain greatly impacts the ability of predators to prevent an outbreak, with a much higher predation rate required to prevent the high virulence strain from invading than that required to prevent invasion of the low virulence strain.

For the low virulence strain of *Mg*, food subsidy effects on natural and disease-induced mortality, and host susceptibility to and recovery from infection, are sufficient to reduce $R_0$ below one without the addition of predators (Fig. 3). While food subsidy effects on host contact or movement make it harder for predators to prevent outbreaks, the predation rates required to prevent a low virulence strain outbreak are less than or equal to empirically observed predation rates at low food subsidy effect sizes (Fig. 3a gray line). This means that the typical activity of predators near a food source should be sufficient to prevent an outbreak. Furthermore, it remains possible for predators to prevent a low virulence strain outbreak even when food subsidy strongly reduces host emigration. However, when host immigration or contact is more than doubled by food subsidy effects, the predation rate required to prevent invasion of the low virulence strain is over five times the predation rate observed in literature and, thus, predators are unlikely to be able to prevent pathogen outbreak at subsidized sites.

For the high virulence strain of *Mg*, food subsidy effects on mortality, predation success, and disease parameters are not sufficient to reduce $R_0$ below one in the absence of predators (Fig. 3). Moreover, food subsidy effects of any size on host movement, contact, and disease mortality make it highly improbable that predators can prevent an outbreak (Fig. 3a). In contrast, food subsidy effects on host recovery, susceptibility, or predation success make it possible for predators to prevent an outbreak (Fig. 3b). The predation rates required to prevent a high virulence strain outbreak drop below observed predation rates at low food subsidy effects on host susceptibility and moderate-to-high food subsidy effects on predation success.

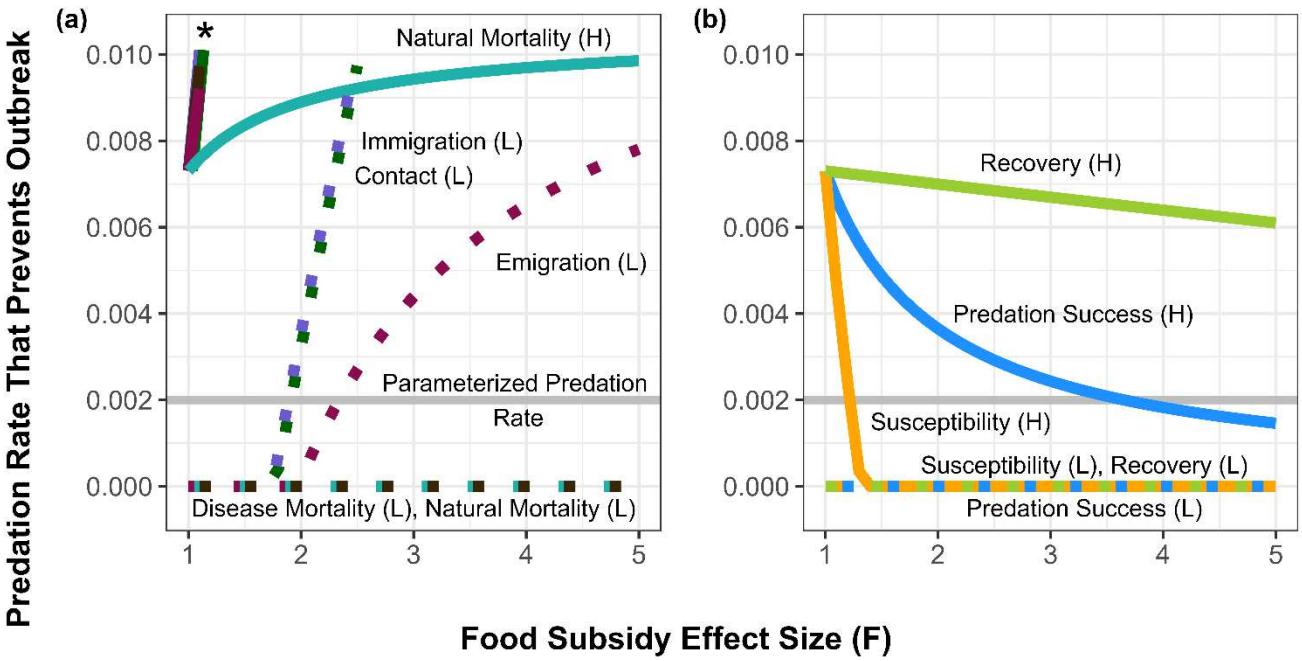



**Fig. 3** The predation rate ($\pi_{min}$) necessary to prevent an outbreak ($R_{0p} = 1$) as a function of the strength of food subsidy effects ($F$) on host, pathogen and predation parameters for the House Finch-*Mg* system, applied to a low virulence (L) pathogen strain (dashed lines) and a high virulence (H) pathogen strain (solid lines). The horizontal grey line represents the empirically observed predation rate (Lindström, 1989). (**a**) colored lines (solid and dashed) represent parameters that typically make it harder for predators to prevent outbreaks under food subsidy. (* represents Immigration (H), Contact (H), Disease Mortality (H), and Emigration (H)), (**b**) colored lines (solid and dashed) represent parameters that typically make it easier for predators to prevent outbreaks under food subsidy

**Question 2: Can predators reduce host mortality?**

***Theory***

We quantified whether predators are harmful or beneficial to food-subsidized host populations using the equilibrium difference in daily host deaths in the presence and absence of predators (daily death difference, DDD; equation 6) under food subsidy effects on each model parameter (Fig. 4). For a given food subsidy effect, $F$, the sign of DDD indicates whether predators increase overall mortality (DDD > 0) or decrease overall mortality (DDD < 0) of hosts. The sign of the slope of the relationship between DDD and $F$ determines whether additional effects of food subsidy make predators more harmful (positive slope) or less harmful (negative slope).

For all host and pathogen parameters, the qualitative relationship between DDD and $F$ (monotonic increasing, monotonic decreasing, or flat) was dependent on whether pathogen outbreaks could occur in the presence or absence of predators ($R_{0p}$ and $R_{0np}$, respectively) under food subsidy effects. The sign or slope of the relationship typically differed in the three regions of parameter space where (i) pathogens were unable to invade irrespective of the presence or absence of predators ($R_{0p} < R_{0np} < 1$), (ii) pathogens could only invade in the absence of

predators but not when predators were present ($R_{0p} < 1 < R_{0np}$), or (iii) pathogens could always invade in the presence and absence of predators ($1 < R_{0p} < R_{0np}$) (Fig. 4). The analytically derived functional relationships between DDD and *F* for each parameter are given in section S3.4 of the Supplement.

When the pathogen cannot invade a predator-free system, and food subsidy increases host immigration, predators are always harmful (i.e., DDD > 0), and increasing effects of food subsidy on host immigration make predators more harmful (i.e., DDD increases with *F*) (Fig. 4a). However, once food subsidy increases immigration enough for pathogens to successfully invade a predator-free system, increasing food effects on immigration reduce the contribution of predators to host mortality, and further increases in immigration can actually make predators beneficial (i.e., DDD < 0) as predators continue to prevent pathogen invasion. When the food subsidy effect on host immigration is large enough that predators cannot prevent an outbreak, the benefit of predators in reducing daily bird deaths are maximized and does not change further with increasing food subsidy effects on immigration.

When food subsidy weakly influences host contact rates, the presence of predators is harmful (Fig. 4b). Once food subsidy effects on host contacts are large enough to allow pathogens to invade a predator-free system, daily deaths decrease with subsidy effect size and predators can become beneficial (i.e., additional deaths from predation are fewer than the deaths that would occur from large outbreaks in the absence of predators). When increases in contact rates are large enough that predators cannot prevent an outbreak, this benefit of predators in reducing disease-related deaths decreases until both locations experience the same number of daily deaths.

Since food subsidy effects improve host immunity (by reducing host susceptibility or increasing recovery), food effects on immunity always decrease outbreak potential. Therefore, predators are always harmful when food subsidy effects are strong and can only be beneficial when food subsidy effects on immunity are weak enough to allow pathogen invasion in the absence of predators (Fig. 4c, d).

When food subsidy decreases host emigration rate, increases predation success, or decreases natural or disease-induced mortality, the qualitative dependence of DDD on food subsidy effects was context dependent; in other words, the parameter values associated with a focal system determine whether predation exacerbates or reduces predation (see S3.4 in the Online Resource for derivations).

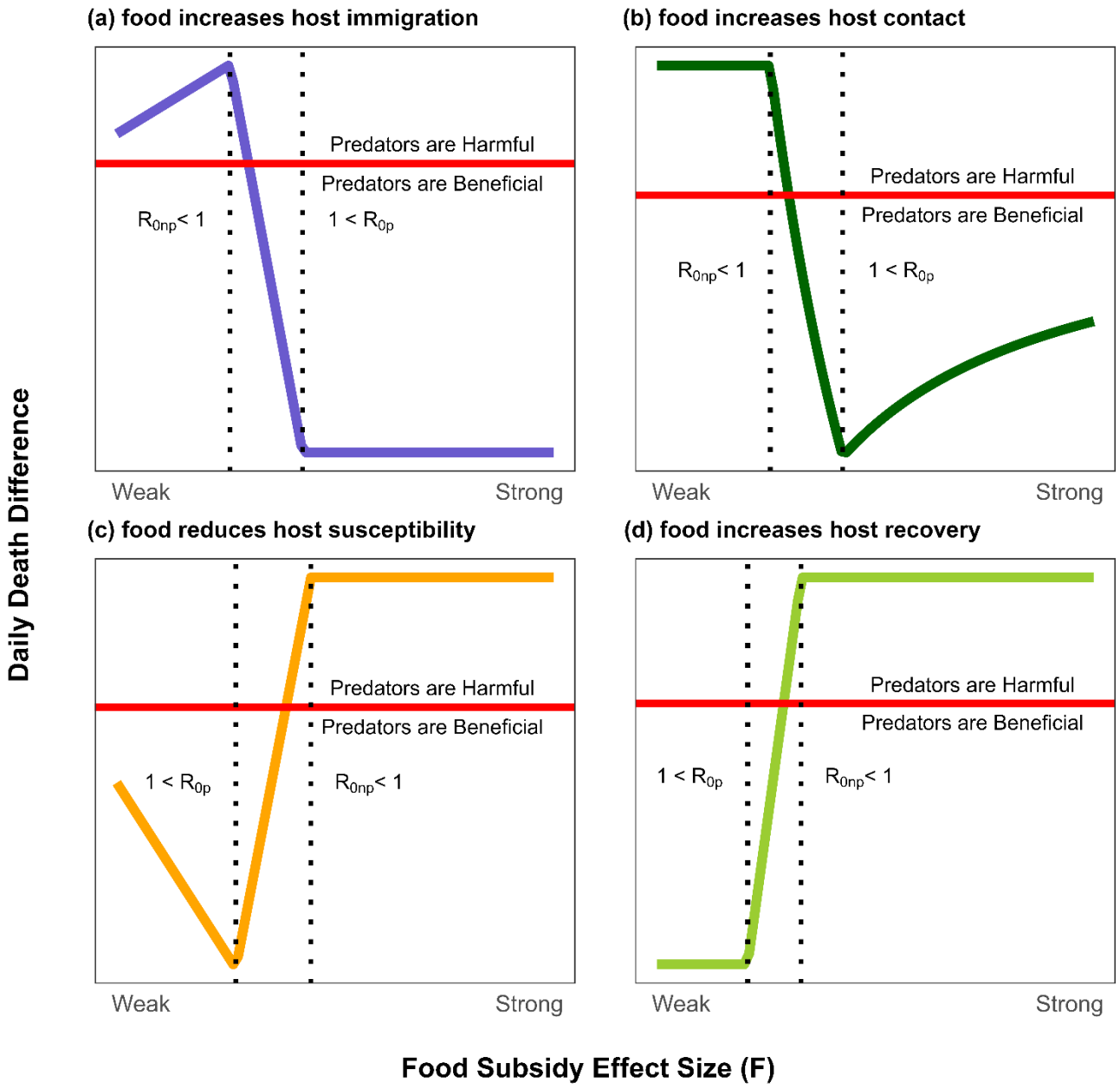



**Fig. 4** Qualitative behavior of the daily death difference (DDD = $\frac{dC}{dt}$ with predators - $\frac{dC}{dt}$ without predators) as food subsidies change (**a**) the rate of host immigration, (**b**) host contact rates, (**c**) host susceptibility to infection, and (**d**) host recovery rates. The horizontal red line indicates when there is no difference in host deaths per day between locations with and without predators (DDD = 0). Values above the red line indicate that the presence of predators increase the number of hosts that die per day, while values below the red line indicate that the presence of predators decrease the number of daily host deaths. Dashed vertical lines show when $R_0$ values change relative to 1

### *Application to the House Finch – Mg system*

When our model was applied to the low virulence (L) strain of *Mg* (Fig. 5a, b), pathogen transmission rates were sufficiently low that the pathogen was unable to invade in the absence of food subsidy, and, therefore, the presence of predators always increased the total number of host deaths over a 150-day period. The harmful effect of predators was exacerbated when increasing food subsidy reduced host natural mortality rates or increased the predation success of predators, resulting in an almost 200% increase in total bird deaths at strong food subsidy effect sizes (Fig. 5a). The harmful effect of predators also increased when food subsidy improved immunity (reduced susceptibility to disease, reduced disease mortality, and increased recovery), but the increase in daily death differences was much weaker (Fig. 5b). When food subsidy increased host density by increasing immigration and host contact rates, and reducing emigration, the harmful effect of predators decreased and began to saturate (Fig. 5a, b).

When our model was applied to the high virulence (H) strain of *Mg* (Fig. 5c, d), pathogen transmission rates were sufficiently high to allow the pathogen to invade in the absence of food subsidy. Moreover, the presence of predators typically decreased cumulative host deaths (Fig. 5c, d) because selective predation of infected birds decreased transmission and prevented disease-induced mortality. However, these reductions in cumulative mortality were relatively small (< 6%) for all food subsidy effects. The striking exception was when food subsidy decreased host susceptibility to infection; in this case, food subsidy alone was sufficient to prevent pathogen invasion. As a result, selective predation of infected birds exacerbated the negative effects of predators and resulted in over a 37% increase in bird mortality (Fig. 5d). Predators can also increase bird mortality when food subsidy increases predation success. While most parameters resulted in a monotonic relationship between the daily death difference and food effect size, this relationship was U-shaped when food subsidy increased predation success (Fig. 5c). This reflects that at low subsidy effect sizes, increasing predation is beneficial by continuing to prevent invasion of a virulent pathogen strain, but, eventually, strong increases in predation success result in more predator-induced deaths than the pathogen would cause in a predator-free population.

Overall, our results in the House Finch-*Mg* system suggest that when feeding primarily increases host density and contact, feeding can reduce, but not overcome, additional mortality from predators in the presence of a low-virulence pathogen strain. For high virulence pathogen strains, predators can reduce overall mortality at feeders by reducing transmission and, thus, disease deaths, but these effects are relatively small and the benefits are generally reduced with increasing food subsidy effects on host immunity, contacts, and movement.

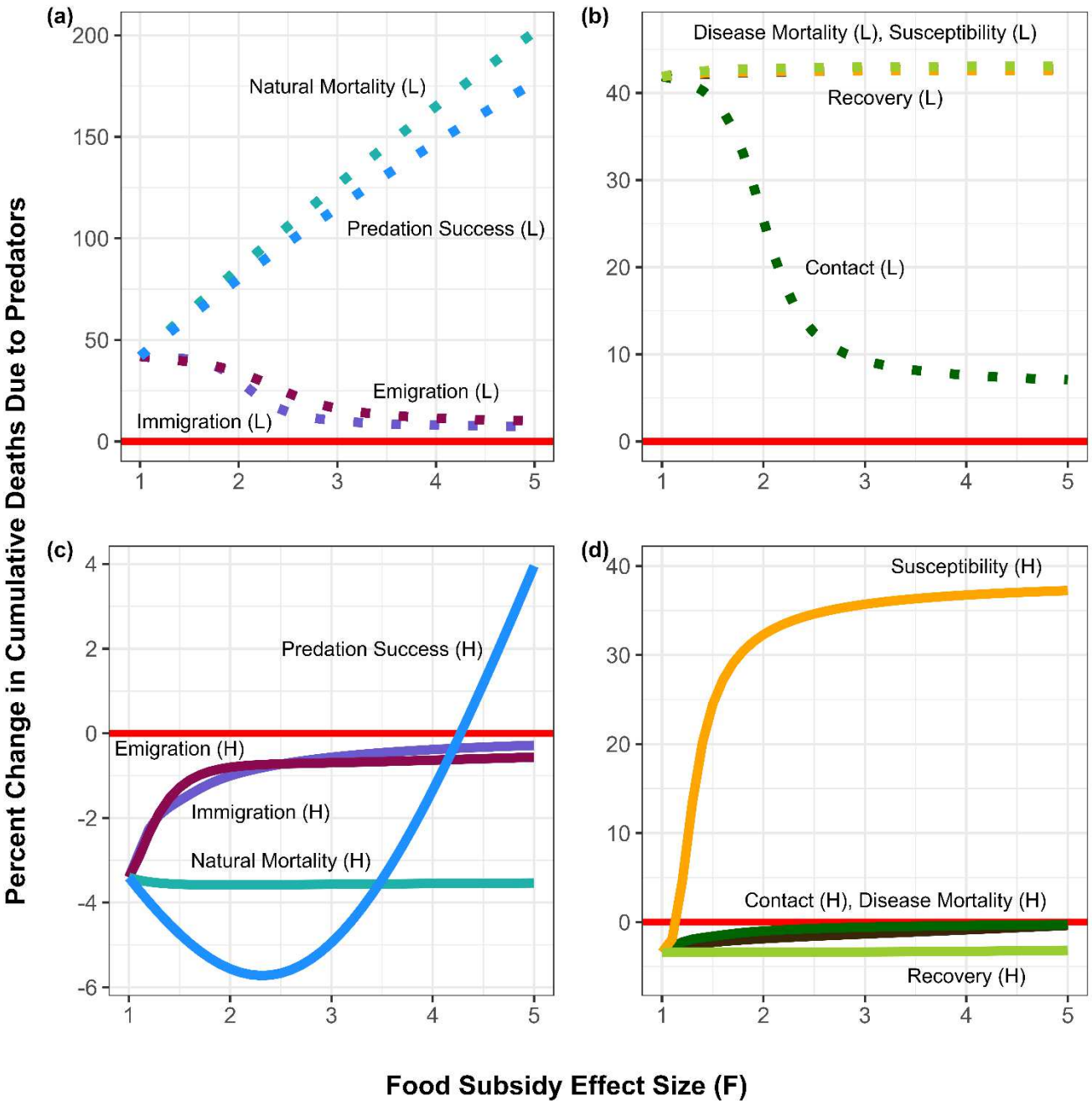



**Fig. 5** Percent change in cumulative bird deaths due to predators over 150 days as the strength of food subsidies (*F*) increase and alter a parameter value, applied to a low virulence (L) pathogen (**a**, **b**; represented with dashed lines) and a high virulence (H) pathogen (**c**, **d**; represented with solid lines), parameterized after the house finch-*Mg* system. The horizontal red line indicates when there is no difference in total bird deaths between a bird feeding site with a predator and one without. Values above the red line indicate that the presence of predators increases the total number of bird deaths, while values below the red line indicate that the presence of predators decreases the total number of bird deaths. For a line representing a given parameter, a positive slope indicates that increasing food subsidy effects increase host mortality due to predators while

a negative slope indicates that increasing food subsidy effects decrease host mortality due to predators. Note that the y-axes scales are different in each panel figure

## Discussion

Previous research has shown that food subsidies can increase host abundance and contact, leading to increased outbreak risk and pathogen transmission (Becker and Hall 2014), and that predators can decrease transmission by culling infected individuals (Lopez et al. 2023). Our model unifies these past theories to investigate how food subsidy and predators influence pathogen outbreak potential and the net effect of food, predators, and pathogens on cumulative host mortality. Overall, we found that a predator's ability to prevent pathogen invasion at feeding stations, and whether predators exacerbated or reduced host deaths in the presence of a pathogen, depended strongly on how food subsidy influenced host movement, aggregation, condition, and immunity. Notably, while food subsidy that increased host immigration or intraspecific contact tended to reduce a predator's ability to prevent outbreaks, they also tended to increase the beneficial effect of predators in reducing disease-related deaths. The opposite pattern occurred when food subsidy improved immune defense, in which case predators more readily prevented outbreaks, but, consequently, tended to exacerbate host mortality. In a well-studied empirical system, transmission of House Finch eye disease at bird feeders, pathogen virulence typically dictated the net effects of predators: predators tended to exacerbate mortality with low virulence pathogens but slightly reduced overall mortality in the presence of highly virulent strains; bird feeding could amplify or reduce these predator effects depending on whether food subsidy acted primarily to increase host density, improve immunity, or increase predation success. These findings highlight the importance of considering multiple species interactions, and quantifying effects of feeding on both individual condition and aggregation of conspecifics, to understand the net outcomes of providing food for wildlife.

We found that food subsidies can either enhance or reduce the ability of predators to regulate pathogen transmission and outbreaks. Our work suggested that when food subsidy increased host abundance (via altered host movement), increased host contact, or reduced disease-induced host mortality caused by high virulence pathogen infections, empirically estimated predation rates (Roth and Lima 2003; Lepczyk et al. 2004) would typically be too low to prevent outbreaks. Predators were more likely to reduce outbreak potential rather than prevent outbreaks in the conditions we examined. However, our model only considered direct effects of predators on host mortality; if predators also alter prey behavior in ways that reduce transmission (for example reducing bird attraction to feeders or time birds spend at feeders), predation could have a stronger effect on outbreak potential than we found in our study. Predators were most likely to prevent outbreaks when food subsidies reduced host susceptibility to infection, highlighting the importance of providing nutritionally appropriate food as a tool for managing disease risk (Murray et al. 2016).

While predators always lower pathogen transmission, their impacts on overall host mortality can be positive or negative. When our model was applied to a focal bird-pathogen system (House Finch and mycoplasmal conjunctivitis), we found that predation amplified bird mortality when a low virulence pathogen strain was circulating, but, consistent with past studies (Packer et al. 2003), typically reduced cumulative bird mortality in the presence of a high virulence strain. When mycoplasmal conjunctivitis initially emerged in susceptible House Finch

populations, it caused high disease-induced mortality (Nolan et al. 1998). Since lethargy and vision impairment make infected birds highly vulnerable to predation (Adelman et al. 2017), our results suggest that predators could have been beneficial in reducing initial impacts of the pathogen on House Finch populations. However, pathogen virulence and transmissibility can evolve as host populations develop resistance to infection, suggesting that the role of predators in maintaining 'healthy herds' or exacerbating disease-induced declines could change through time (Cressler et al. 2016). Indeed, the virulence of mycoplasmal conjunctivitis has increased over time (Hawley et al. 2013; Bonneaud et al. 2018), potentially suggesting that predators could increasingly act to mitigate population and infection outcomes in house finches.

Our simple model gives some initial insights into the impacts of food subsidies and predators on disease outcomes in feeder birds against which additional complexities in real systems can be explored in future work. Our model only considered effects of food subsidy on one host or pathogen parameter at a time, but interactive effects of food on multiple host and parasite processes can alter the net effect of feeding on disease (Becker and Hall 2014). Our model also assumes that infected birds do not emigrate out of a feeding site due to lethargy, but it is possible that host movement is not altered when infected with a low virulence pathogen (Kollias et al. 2004). Additionally, some pathogens transmitted at bird feeders, including salmonella or trichomoniasis, can be transmitted from prey to predator, which might reduce the impacts of predation if predators die from consuming infected prey (Tizard 2004; Anderson et al. 2009). Finally, we focused on the direct, lethal effects of predation, but did not account for additional behavioral or physiological linkages between predation and infection. For example, predators might avoid eating sick-looking individuals, or predator presence could induce hormonal stress responses and reduced foraging that result in decreased immune function and increased pathogen susceptibility (Richards et al. 2023). A major knowledge gap remains about the wider implications of bird-feeding over larger, regional species scales; therefore, future modeling work would extend this site-level model to consider predator-prey-pathogen interactions on networks of supplemental feeding sites linked by bird and predator movement. Large-scale community science projects that gather information on bird abundance, predation, and infection, such as Project FeederWatch in North America and Garden Wildlife Health in the UK, offer excellent opportunities to parameterize and validate these models, and to communicate best practices for integrated predator and pathogen management at feeding stations (Bonter and Greig 2021; Lawson et al. 2018). Such future work is key to understanding wider community-level impacts of feeding and developing public guidelines for the increasing number of people that engage in feeding birds and other wildlife.

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**Statements & Declarations**

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