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Species interactions affect the spread of vector-borne plant pathogens independent of transmission mode

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Abstract. Within food webs, vectors of plant pathogens interact with individuals of other species across multiple trophic levels, including predators, competitors, and mutualists. These interactions may in turn affect vector-borne pathogens by altering vector fitness and behavior. Predators, for example, consume vectors and reduce their abundance, but often spur movement of vectors as they seek to avoid predation. However, a general framework to predict how species interactions affect vectors of plant pathogens, and the resulting spread of vector-borne pathogens, is lacking. Here we developed a mathematical model to assess whether interactions such as predation, competition, and mutualism affected the spread of vector-borne plant pathogens with nonpersistent or persistent transmission modes. We considered transmission mode because interactions affecting vector–host encounter rates were expected to most strongly affect nonpersistent pathogens that are transmitted with short feeding bouts; interactions that affect vector feeding duration were expected to most strongly affect persistent pathogens that require long feeding bouts for transmission. Our results show that interactions that affected vector behavior (feeding duration, vector–host encounter rates) substantially altered rates of spread for vector-borne plant pathogens, whereas those affecting vector fitness (births, deaths) had relatively small effects. These effects of species interactions were largely independent of transmission mode, except when interactions affected vector–host encounter rates, where effects were strongest for nonpersistent pathogens. Our results suggest that a better understanding of how vectors interact with other species within food webs could enhance our understanding of disease ecology.

Key words: *community ecology; disease ecology; food web; mathematical model; vector-borne pathogen.*

Introduction

Plant pathogens transmitted by arthropod vectors drive global biodiversity loss, reduce agricultural yields, and cause regional economic burden (Strange and Scott 2005, Perilla-Henao and Casteel 2016). Numerous studies have in turn assessed the roles of vector population dynamics and vector–host–pathogen interactions on the spread of plant pathogens (e.g., Hogenhout et al. 2008, Mauck et al. 2012, Conway et al. 2014). Within food webs, vectors also interact with predators, competitors, and mutualists, and such interactions might affect vector-borne pathogens by altering characteristics of vector populations (Blaustein et al. 2010, Seabloom et al. 2015).

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For example, competitors and predators decrease vector fitness (Awmack and Leather 2002, Finke 2012), whereas mutualists increase it (Rice and Eubanks 2013). Predators often induce vector defensive behaviors, increasing plant-to-plant movement (Roitberg et al. 1979, Nelson and Rosenheim 2006, Kersch-Becker and Thaler 2015), while decreasing feeding time on hosts (Long and Finke 2015, Ingerslew and Finke 2017). Vectors can also respond to predators by limiting plant-to-plant movement to avoid detection (Denno et al. 2003). Herbivory by nonvector competitors can also stimulate vector movement because of displacement (De Barro 1992) or reducing nutrients (Weibull 1987, De Mazancourt et al. 1998). Mutualists have been shown to both reduce (Mgochecki and Addison 2010) and increase vector movement (Styrsky and Eubanks 2007).

Models have increasingly been used to predict how such ecological interactions may affect pathogens,

although most focus on directly transmitted pathogens. For example, when hosts are co-infected, competition among pathogens selects for increased virulence (Alizon et al. 2013). Yet, co-infection also increases host susceptibility to certain pathogens (Alizon et al. 2013). Models of dilution effects show that interactions among hosts strongly affect pathogens, although effects depend on whether transmission is frequency- or density-dependent (Roberts and Heesterbeek 2018). The few models that assess how interactions affect vector-borne pathogens focus on predator–prey interactions, and show pathogens spread more slowly when predators reduce vector abundance (Jeger et al. 2011, Finke 2012). Yet, when vectors avoid predators by moving to new hosts, rates of pathogen spread may actually increase due to predators (Jeger et al. 2011, Finke 2012).

Although accumulating evidence shows that species interactions may affect vector-borne pathogens, it remains largely unclear whether these effects depend on pathogen transmission mode. Most vector-borne plant pathogens are classified as either "non-persistent" or "persistent" (Gray and Banerjee 1999). Nonpersistent pathogens are transmitted on contaminated vector mouthparts and require brief probes for transmission, but vectors may lose the pathogen over time (Mauck et al. 2010, 2012). Persistent pathogens are transmitted after circulation through a vector, and require long sustained feeding bouts for transmission (Mauck et al. 2012). We hypothesized that ecological interactions that affect vector–host encounter rates might most strongly affect nonpersistent pathogens because they are transmitted with short feeding bouts, whereas interactions that affect the duration of feeding bouts might most strongly affect persistent pathogens that require sustained feeding for transmission (Long and Finke 2015).

Here, we modeled effects of species interactions on vector fitness and behavior, and the resulting effects on the spread of nonpersistent and persistent plant pathogens. We modeled three interaction types: predation, competition, and mutualism. Although such interactions occur for vectors of animal and plant pathogens (Blaustein et al. 2010, Johnson et al. 2015), we focused on plants, given the substantial empirical data assessing effects of species interactions on movement and vital rates of plant-pathogen vectors. Our results show that integrating community ecology into theoretical pathogen models may greatly enhance our understanding of disease dynamics.

Methods

Model overview

We developed a mathematical model to study effects of species interactions and pathogen transmission mode on vector-borne plant pathogens. Our model involved a single plant host, vector, and pathogen. Species interactions, reflecting predation, competition, and mutualism, were incorporated by adjusting parameters for vector fitness (fecundity, mortality) and behavior (feeding duration, host encounter rates) in specific ways (see Model Formulation and Fig. 1). We assumed predators and competitors decrease vector fitness by reducing vector fecundity and/or increasing mortality (Awmack and Leather 2002, Finke 2012), while mutualists increase it (Rice and Eubanks 2013). We assumed that all interaction types may have positive, negative, or neutral effects on vector movement, given that empirical studies have documented such variable effects (Roitberg et al. 1979, Denno et al. 2003, Nelson and Rosenheim 2006, Styrsky and Eubanks 2007, Mgochecki and Addison 2010, Kersch-Becker and Thaler 2015, Long and Finke 2015, Ingerslew and Finke 2017). Our model examined "nonpersistent" and "persistent" transmission modes. Because altered vector behavior due to species interactions may alter vector–host contact times, our model was designed to test if effects of species interactions varied across transmission modes. The dynamics of each transmission mode were captured by varying parameters for the probability of pathogen acquisition and inoculation (see Model Formulation; Table 1; Appendix S1: Table S1).

Model formulation

Our model tracked numbers of susceptible $P_s(t)$ and infected $P_i(t)$ hosts, and susceptible $V_s(t)$ and infectious $V_i(t)$ vectors, in continuous time t over a typical agricultural season (120 d; Fig. 2; Table 1). Transmission rates from infectious vectors to susceptible plants, or infectious plants to susceptible vectors, were β_P and β_V, respectively. Vectors had two states, moving or feeding, so there were eight compartments: susceptible

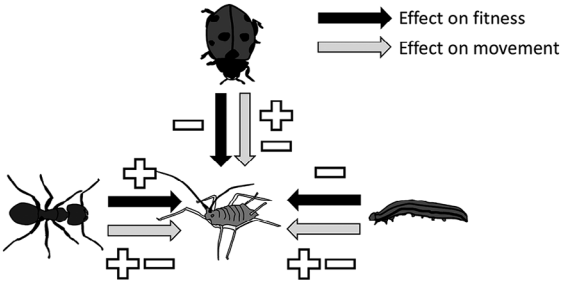

Fig. 1. Predicted effects of interacting competitors (caterpillar), predators (lady beetle), and mutualists (ant) on vector (aphid) fitness and movement. Symbols next to the arrows show expected results of each interaction type, where "+" is positive, "−"is negative, and "+−" is either positive or negative. Positive effects on fitness (by increasing births or decreasing deaths) are expected to increase pathogen spread. Positive effects on movement are expected to increase the spread of pathogens if the vector–host encounter rate increases, but decrease pathogen spread if vector feeding bout duration decreases.

TABLE 1. Model parameters and initial conditions. The range of values are shown only for parameters that were varied due to species interactions.

Parameter	Description	Standard value	Range
ε	Rate of vectors encountering new plants	3 d^{-1}	1–9 d^{-1}
Φ	Fraction of time vectors spend feeding	0.5	0.1–0.9
μ_f	Mortality rate for feeding vectors	0.02 d^{-1}	0.0067–0.06 d^{-1}
μ_m	Mortality rate for moving vectors	0.04 d^{-1}	0.013–0.12 d^{-1}
ρ	Fecundity of feeding vectors	0.08 d^{-1}	0.027–0.24 d^{-1}
K_V	Carrying capacity of vectors per plant	100	–
$V_{fs}(0)$	Initial number of feeding susceptible vectors	0	–
$V_{ms}(0)$	Initial number of moving susceptible vectors	100	–
$V_{fi}(0)$	Initial number of feeding infected vectors	0	–
$V_{mi}(0)$	Initial number of moving infected vectors	1	–
$P_s(0)$	Initial number of susceptible plants	10,000	–
$P_i(0)$	Initial number of infected plants	0	–
Non–persistent transmission			
β_V	Transmission rate from plants to vectors	500 d^{-1}	–
β_P	Transmission rate from vectors to plants	10,000 d^{-1}	–
α	Rate of vectors becoming able to acquire or transmit	86,400 d^{-1}	–
γ_P	Rate of pathogen clearance in feeding vectors	288 d^{-1}	–
γ_m	Rate of pathogen clearance in moving vectors	24 d^{-1}	–
Persistent transmission			
β_V	Transmission rate from plants to vectors	8.3 d^{-1}	–
β_P	Transmission rate from vectors to plants	5.5 d^{-1}	–
α	Rate of vectors becoming able to acquire or transmit	48 d^{-1}	–
γ_f	Rate of pathogen clearance in feeding vectors	0.0 d^{-1}	–
γ_m	Rate of pathogen clearance in moving vectors	0.0 d^{-1}	–

moving vectors (V_{sm}), susceptible vectors feeding on susceptible plants (V_{sfs}), susceptible vectors feeding on infectious plants that are unable to acquire the pathogen (V_{sfip}), susceptible vectors feeding on infectious plants that are able to acquire the pathogen (V_{sfit}), infectious moving vectors (V_{im}), infectious vectors feeding on susceptible plants that are unable to transmit the pathogen (V_{ifsp}), infectious vectors feeding on susceptible plants that are able to transmit the pathogen (V_{ifst}), and infectious vectors feeding on infectious plants (V_{ifi}). A flowchart is shown in Fig. 2, and lists of state variables and conditions are provided in Table 1 and Appendix S1: Table S1. All simulations and analyses were conducted in Python. All files used in the model and original source code used to conduct the simulations and generate the figures are publicly available (see Data Availability).

Model conditions

Vector population dynamics were governed by fecundity and mortality; the number of plants (P) was constant. Vector fecundity was proportional to the number of feeding vectors, as we assumed moving vectors do not reproduce, with a logistic term dependent on the total number of vectors, the intrinsic growth rate (ρ), and carrying capacity (κ). All offspring are susceptible (no maternal transmission) and join the moving state, as newly born vectors need to move before establishing a feeding site. Moving and feeding vectors die at rates of μ_m and μ_f, respectively.

Vector movement was incorporated with parameters for feeding duration and vector–host encounter rates. Vectors encounter new plants at rate ε and spend a proportion ϕ of time feeding and the remainder, $1 - \phi$, moving. Feeding duration and encounter rates were independently modeled to identify different mechanisms by which interactions could affect vectors, although these factors are often linked (i.e., vectors that feed more also likely encounter more hosts).

Susceptible vectors transition from moving to feeding at a rate of $\frac{\epsilon}{1-\phi}$, with $\frac{P_s}{P}$ landing on susceptible plants (V_{sfs}) and $\frac{P_i}{P}$ landing on infectious plants (V_{sfip}). For the vectors that land on susceptible plants, the plants can be infected by other infectious vectors at a rate of $\beta_P \frac{V_{ifst}}{P_s}$, moving those susceptible vectors to compartment V_{sfip}. Susceptible vectors establishing on infectious plants (V_{sfip}) are unable to acquire the pathogen until after they have fed for an average time of $\frac{1}{\alpha}$; after this period they become able to acquire the pathogen and join compartment V_{sfit}. Susceptible vectors able to acquire the pathogen are infected (V_{ifi}) at rate β_V.

Infectious vectors also transition from moving to feeding at a rate of $\frac{\epsilon}{1-\phi}$, with $\frac{P_i}{P}$ landing on infectious plants (V_{ifi}) and $\frac{P_s}{P}$ landing on susceptible plants (V_{ifsp}).

Infectious vectors establishing on susceptible plants (V_{ifsp}) are unable to transmit the pathogen until after they have fed for an average time of $\frac{1}{\alpha}$, after which they become able to transmit (V_{ifst}). The susceptible plant being fed on becomes infected at a rate of $\beta_P \frac{V_{ifst}}{P_s}$, moving the feeding infectious vectors to V_{ifi}.

Rates of pathogen loss for feeding and moving vectors are γ_f and γ_m. Infectious vectors that clear the pathogen move back to the susceptible state. We assume that only pathogens with the nonpersistent transmission mode can be lost from vectors (Gray and Banerjee 1999).

Model equations

The full model is given by the vector dynamics equations (Fig. 2):

$$\frac{dV_{sm}}{dt} = \underbrace{-\frac{\varepsilon}{1-\phi}V_{sm}}_{\text{to feeding}} + \underbrace{\frac{\varepsilon}{\phi}V_{sf}}_{\text{from feeding}} + \underbrace{\gamma_m V_{im}}_{\text{loses pathogen}} - \underbrace{\mu_m V_{sm}}_{\text{death}} + \underbrace{\rho V_f\left(1-\frac{V_f}{\kappa P}\right)}_{\text{birth}}$$

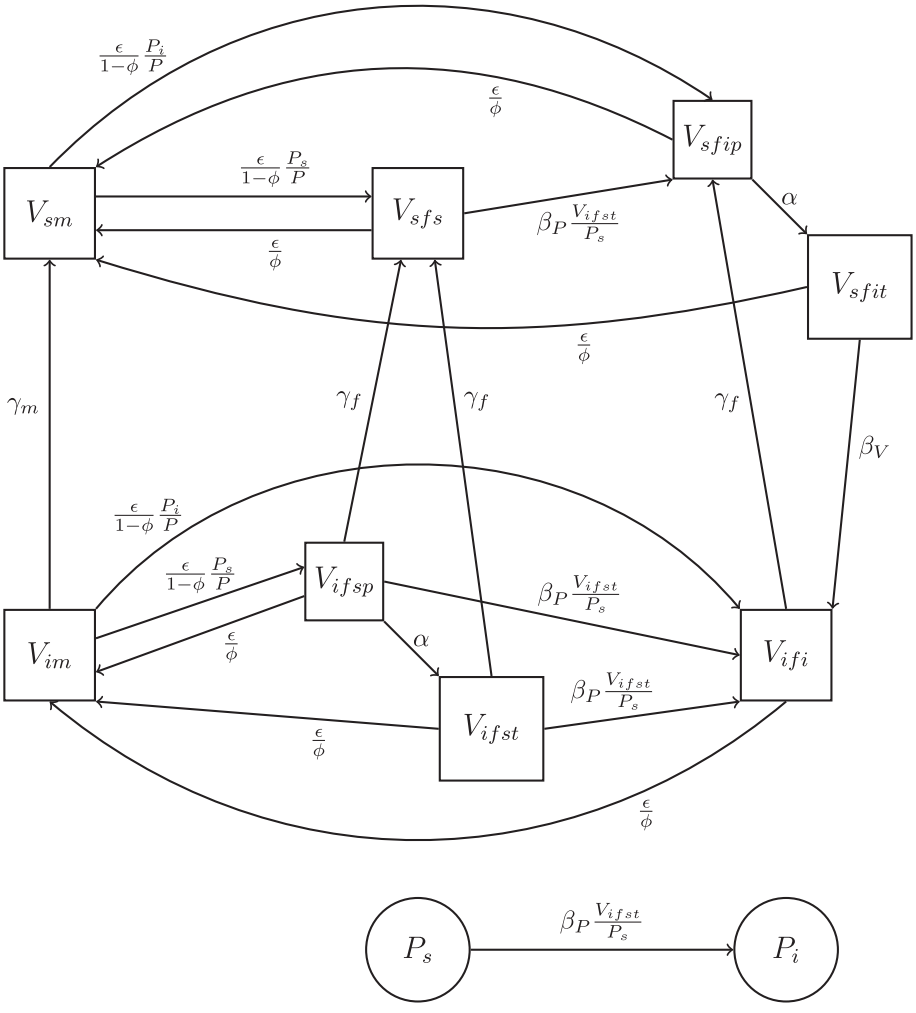

FIG. 2. Flowchart of the model. The model includes eight vector compartments: susceptible moving vectors (V_{sm}), susceptible vectors feeding on susceptible plants (V_{sfs}), susceptible vectors feeding on infectious plants that are unable to acquire the pathogen (V_{sfip}), susceptible vectors feeding on infectious plants that are able to acquire the pathogen (V_{sfit}), infectious moving vectors (V_{im}), infectious vectors feeding on susceptible plants that are unable to transmit the pathogen (V_{ifsp}, infectious vectors feeding on susceptible plants that are able to transmit the pathogen (V_{ifst}), and infectious vectors feeding on infectious plants (V_{ifi}); the model includes two plant compartments: susceptible plants (P_s) and infected plants (P_i). Pathogen transmission is governed by the rate of vectors encountering new plants (ε), the fraction of time vectors spend feeding (Φ), the rate of vectors becoming able to acquire or transmit (α), the transmission rate from plants to vectors (β_V), the transmission rate from vectors to plants (β_P), the rate of pathogen clearance in feeding vectors (γ_f), and the rate of pathogen clearance in moving vectors (γ_m). Lines with arrows show the transitions in the model, with the formula adjacent to each line showing the rate at which the transition occurs.

$$\frac{dV_{sfs}}{dt} = \underbrace{\frac{\varepsilon}{1-\phi}\frac{P_s}{P}V_{sm}}_{\text{from moving}} - \underbrace{\frac{\varepsilon}{\phi}V_{sfs}}_{\text{to moving}} + \underbrace{\gamma_f V_{ifs}}_{\text{loses pathogen}} - \underbrace{\mu_f V_{sfs}}_{\text{death}} - \underbrace{\beta_P \frac{V_{ifst}}{P_s}V_{sfs}}_{\text{plant infected}}$$

$$\frac{dV_{sfip}}{dt} = \underbrace{\frac{\varepsilon}{1-\phi}\frac{P_i}{P}V_{sm}}_{\text{from moving}} - \underbrace{\frac{\varepsilon}{\phi}V_{sfip}}_{\text{to moving}} + \underbrace{\gamma_f V_{ifi}}_{\text{loses pathogen}} - \underbrace{\mu_f V_{sfip}}_{\text{death}} + \underbrace{\beta_P \frac{V_{ifst}}{P_s}V_{sfs}}_{\text{plant infected}} - \underbrace{\alpha V_{sfip}}_{\text{acquiring pathogen}}$$

$$\frac{dV_{sfit}}{dt} = \underbrace{-\frac{\varepsilon}{\phi}V_{sfit}}_{\text{to moving}} - \underbrace{\mu_f V_{sfit}}_{\text{death}} + \underbrace{\alpha V_{sfip}}_{\text{acquiring pathogen}} - \underbrace{\beta_V V_{sfit}}_{\text{infection}}$$

$$\frac{dV_{im}}{dt} = \underbrace{-\frac{\varepsilon}{1-\phi}\frac{P_i}{P}V_{im}}_{\text{to feeding}} + \underbrace{\frac{\varepsilon}{\phi}V_{if}}_{\text{from feeding}} - \underbrace{\gamma_m V_{im}}_{\text{loses pathogen}} - \underbrace{\mu_m V_{im}}_{\text{death}}$$

$$\frac{dV_{ifsp}}{dt} = \underbrace{\frac{\varepsilon}{1-\phi}\frac{P_s}{P}V_{im}}_{\text{from moving}} - \underbrace{\frac{\varepsilon}{\phi}V_{ifsp}}_{\text{to moving}} - \underbrace{\gamma_f V_{ifsp}}_{\text{loses pathogen}} - \underbrace{\mu_f V_{ifsp}}_{\text{death}} - \underbrace{\beta_P \frac{V_{ifst}}{P_s}V_{ifsp}}_{\text{plant infected}} - \underbrace{\alpha V_{ifsp}}_{\text{transmitting pathogen}}$$

$$\frac{dV_{ifst}}{dt} = \underbrace{-\frac{\varepsilon}{\phi}V_{ifst}}_{\text{to moving}} - \underbrace{\gamma_f V_{ifst}}_{\text{loses pathogen}} - \underbrace{\mu_f V_{ifst}}_{\text{death}} - \underbrace{\beta_P \frac{V_{ifst}}{P_s}V_{ifst}}_{\text{infection}} + \underbrace{\alpha V_{ifsp}}_{\text{transmitting pathogen}}$$

$$\frac{dV_{ifi}}{dt} = \underbrace{\frac{\varepsilon}{1-\phi}\frac{P_i}{P}V_{im}}_{\text{from moving}} - \underbrace{\frac{\varepsilon}{\phi}V_{ifi}}_{\text{to moving}} - \underbrace{\gamma_f V_{ifi}}_{\text{loses pathogen}} - \underbrace{\mu_f V_{ifi}}_{\text{death}} + \underbrace{\beta_P \frac{V_{ifst}}{P_s}V_{ifs}}_{\text{plant infected}} + \underbrace{\beta_V V_{sfit}}_{\text{infection}},$$

along with the host dynamics equations

$$\frac{dP_s}{dt} = \underbrace{-\beta_P V_{ifst}}_{\text{infection}}$$

$$\frac{dP_i}{dt} = \underbrace{\beta_P V_{ifst}}_{\text{infection}}$$

The number of susceptible vectors feeding on infectious plants is $V_{sfi} = V_{sfip} + V_{sfit}$, and the number of infectious vectors feeding on susceptible plants is $V_{ifs} = V_{ifsp} + V_{ifst}$. The number of susceptible feeding vectors is $V_{sf} = V_{sfs} + V_{sfi}$, the number of infectious feeding vectors is $V_{if} = V_{ifs} + V_{ifi}$, and the number of feeding vectors is $V_f = V_{sf} + V_{if}$. The number of moving vectors is $V_m = V_{sm} + V_{im}$, and the number of vectors total is $V = V_m + V_f$.

Parameters

Parameters for each transmission mode were derived from well-studied vector-borne plant pathosystems (Appendix S1: Table S2). Key parameters were birth rate, death rate, carrying capacity, vector–host encounter rates, feeding-bout duration, and transmission rate (Appendix S1: Table S2). We derived initial conditions from the average and range of these studies, and ensured parameters produced realistic rates of pathogen spread that reflected key differences in rates of pathogen acquisition and inoculation observed for each transmission mode (Table 1). Species interactions were modeled by altering vector fitness or behavior based on typical effects of each interaction type on vector fitness and behavior (see Model Overview; Fig. 1, Table 1; Appendix S1: Tables S3–S6). We did not model population dynamics of nonvectors, such that the interaction strength was assumed to be constant with any specific initial conditions; this allowed us to test hypotheses related to how species interactions may affect the spread of plant pathogens without confounding feedback loops between vector and nonvector species.

Numerical analysis

We ran simulations to assess how growth rates of nonpersistent and persistent pathogens were altered by species interactions. In a first simulation set, we varied single parameters, which assumes interactions affect a single vector trait. Interactions can alter vector birth or death rates, fraction of time spent feeding, or vector–host encounter rates (Fig. 1, Table 1; Appendix S1: Tables S3–S6). In a second set of simulations, we varied two parameters to reflect interactions affecting two traits. Our approach was analogous to a factorial experiment, with assessment of main effects and interactions. In both simulation sets, focal parameters were varied over a range from 1/3 to 3 times their baseline value, except for fraction of time spent feeding, which was varied from 0.1 to 0.9 (see Appendix S1 for more details). In both simulation sets, the parameters not being varied were fixed at baseline values. For each set of parameter values, the intrinsic growth rate of the pathogens was calculated as the dominant eigenvalue of the Jacobian matrix restricted to the infected classes only, evaluated at the disease-free state on day 100 after starting with 100 vectors for 10,000 plants on day 0. To summarize the pairwise effects from the second simulation set that varied two parameters concurrently, we

considered changes where the two parameters were either perfectly positively correlated or perfectly negatively correlated.

Results

Effects of species interactions that alter fitness

For both transmission modes, interactions that increased vector fitness (increased fecundity or decreased mortality), typical of mutualisms, increased rates of plant-pathogen spread (Fig. 3). Interactions that decreased fecundity, typical of competition, or increased mortality, typical of predation, slowed rates of plant-pathogen spread (Fig. 3). When interactors enhanced fitness by both increasing fecundity and decreasing mortality, or reduced fitness by both decreasing fecundity and increasing mortality, the concurrent fitness changes additively affected rates of plant-pathogen spread (Fig. 4; Appendix S1: Fig. S1).

Effects of species interactions that alter movement

The effects of vector movement on pathogen spread differed depending on whether greater movement reduced time spent feeding, or increased vector–host encounter rates. Interactions that decreased vector feeding time, relative to moving, decreased rates of spread for nonpersistent and persistent pathogens as long as there was no concurrent reduction in vector–host encounter rates (Figs. 3 and 4). However, interactions that increased vector–host encounter rates increased rates of nonpersistent pathogen spread, but did not alter the rate of spread of persistent pathogens (Figs. 3 and 4). As a result, when an interactor simultaneously increased vector–host encounter rates but decreased vector feeding duration, which commonly happens with greater plant-to-plant movement, the counteracting effects of these two aspects of movement dampened the overall effect of the interaction on rates of plant-pathogen spread (Fig. 4; Appendix S1: Fig. S2).

Effects of species interactions that alter both fitness and movement

Interactions influencing multiple vector conditions had effects on plant pathogens mediated by vector movement. When interactions altered vector–host encounter rates, the largest impacts occurred if vector fitness and movement changed in the same direction. Rates of plant-pathogen spread were increased by interactions that increased vector fitness and vector–host encounter rates, as may occur with mutualism; rates were reduced by interactions that decreased vector fitness and vector–host encounter rates, as may occur with competition (Figs. 4 and 5). However, when interactions altered vector feeding duration, the largest impacts occurred when interactions affected vector fitness and movement in opposite directions. An interactor that increased vector fitness and decreased feeding duration increased rates of plant-pathogen spread (Figs. 4 and 5), whereas an interactor that decreased vector fitness and increased feeding duration reduced spread (Figs. 4 and 5). The largest reduction in rates of pathogen spread occurred in the presence of an interactor that increased vector mortality and decreased vector feeding duration, as might occur with a predator (Figs. 4 and 5). All of these results were similar for

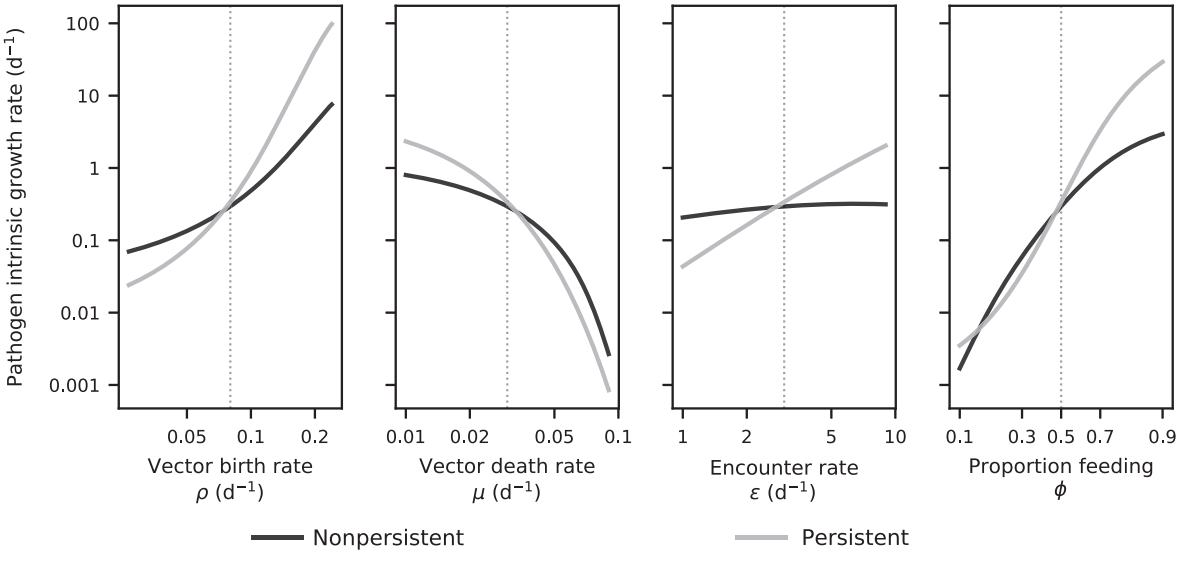

Fig. 3. Intrinsic growth rates of pathogens with various parameter values for vectors that transmit nonpersistent (black) and persistent (gray) pathogens. In each panel, one parameter was varied from its standard value (Appendix S1: Table S3), and all other parameter values were as in Table 1.

nonpersistent and persistent transmission modes (Figs. 4 and 5), although increased vector–host encounter rates, and feeding duration, had stronger impacts on rates of spread for nonpersistent compared to persistent plant pathogens (Figs. 4 and 5).

Discussion

Our model shows interactions in food webs can strongly mediate rates of plant-pathogen spread, with alterations of vector movement having stronger effects than fitness alterations of similar magnitude (Figs. 3 and 4 and Appendix S1: Figs. S1, S2). This is consistent with the few models that show predators can increase rates of pathogen spread by promoting vector movement, even though predators reduce vector abundance (Jeger et al. 2011, Finke 2012). Empirical studies also show that predator–vector interactions that affect vector movement and feeding behavior can override consumptive effects on vector abundance (Long and Finke 2015). Although most vector-borne pathogen models track vector abundance, vector movement is often implicit or represented by simple diffusion (McCallum et al. 2001; but see Shaw et al. 2017, 2019). Our results show that explicit inclusion of movement in models may fundamentally alter predictions. Moreover, strategies that limit vector movement might be more effective in mitigating pathogens than those targeting vector abundance, such as the use of insecticides (Perring et al. 1999).

Empirical studies show interactions with predators, competitors, and mutualists can affect vector behavior, and our model shows this can mediate the spread of pathogens through several mechanisms. For example, when increased movement decreased feeding duration, pathogen spread slowed because of a tradeoff between time feeding and moving. When time spent moving is

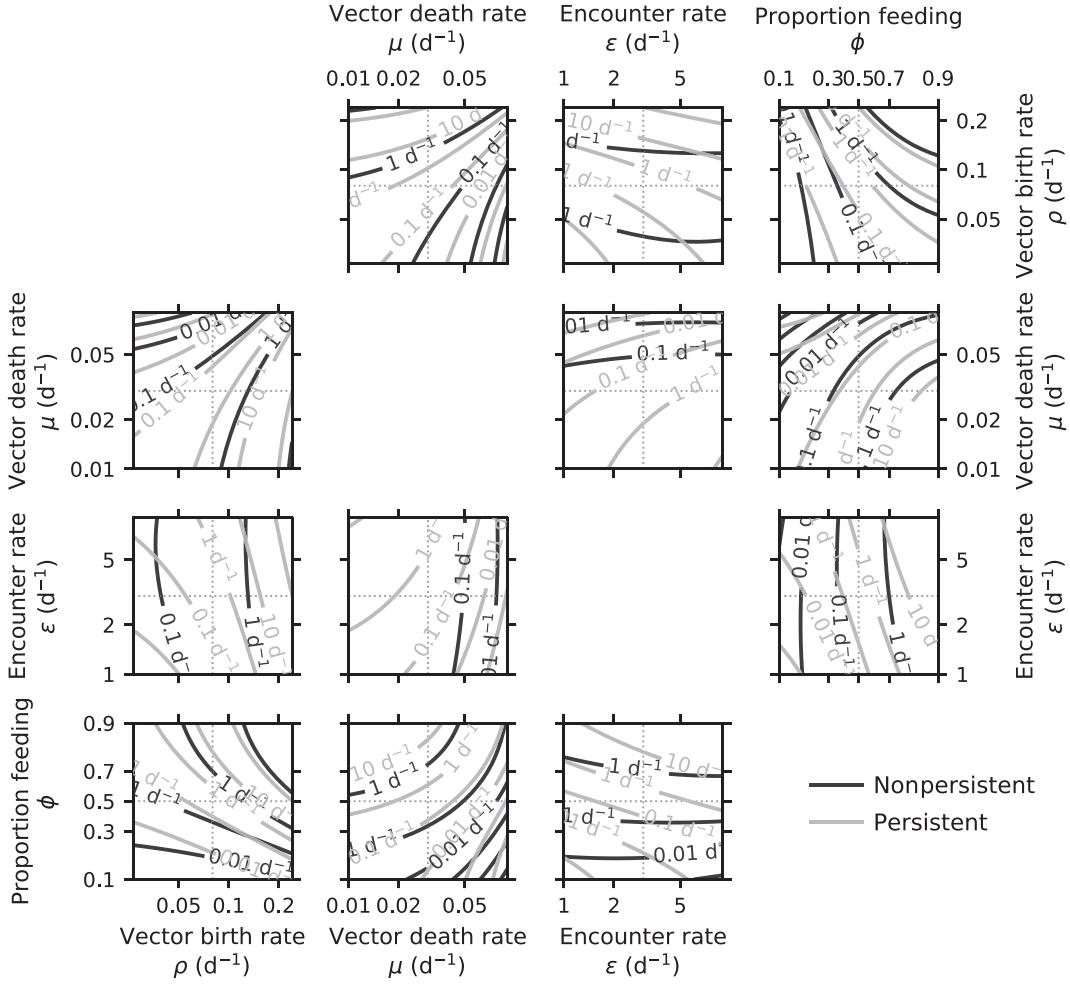

Fig. 4. Intrinsic growth rates of pathogens with various parameters for vector birth rate, vector death rate, vector–host encounter rates, and proportion of time spent feeding (nonpersistent pathogen shown in black; persistent pathogen shown in gray). Each panel shows isoclines for pathogen growth rates (number of new hosts infected per day) with varying parameter values (the pathogen growth rate along each line is the same). Each panel shows how pathogen growth rates would be expected to change because of a species interaction that affects two parameters for vector fitness and/or movement.

long relative to pathogen retention time, a vector may acquire a pathogen with a brief probe on an infected host but clear it in transit before colonizing a new susceptible host (Ng and Falk 2006, Killiny and Almeida 2014). However, if increased movement took the form of greater vector–host encounter rates, rates of pathogen spread increased, as long as feeding duration was unchanged, a result consistent with empirical observations. For example, Hodge et al. (2011) found parasitoid wasps enhanced aphid movement, and this facilitated the spread of bean yellow mosaic virus as colonization of susceptible hosts after acquisition occurred before vectors cleared the pathogen.

Overall, our results show that the impacts of species interactions on vector behavior, and the resulting spread of vector-borne pathogens, depend on how movement affects transmission efficiency. This is in line with models of directly-transmitted (McCallum et al. 1991; Alizon et al. 2013) and vector-borne pathogens (Shaw et al. 2017, 2019; Roberts and Heesterbeek 2018), which all show strong effects of transmission efficiency on pathogens. For example, co-infection of hosts with many pathogens can increase rates of pathogen spread because pathogen–pathogen competition selects for increased transmission efficiency (Alizon et al. 2013). In our model, increasing plant-to-plant movement by vectors decreased the duration of feeding bouts and thus indirectly reduced the probability of transmission. This likely explains why we found strong effects of movement on pathogen transmission, whereas other models that did not explicitly link movement with transmission efficiency show relatively small effects of movement (Shaw et al. 2017, 2019).

Unlike movement, species interactions that solely affected vector fitness had predictable effects on the spread of pathogens that were similar for nonpersistent and persistent pathogens. Interactions that reduced vector fitness, such as predation and competition, slowed the spread of pathogens, as seen in other models (Moore et al. 2010, Finke 2012, Okamoto and Amarasekare 2012) and empirical studies (Landis and van der Werf 1997, Smyrnioudis et al. 2001). In contrast, mutualisms, such as ants protecting aphids from predation (Styrsky and Eubanks 2007, Rice and Eubanks 2013) should increase rates of pathogen spread by increasing vector fitness.

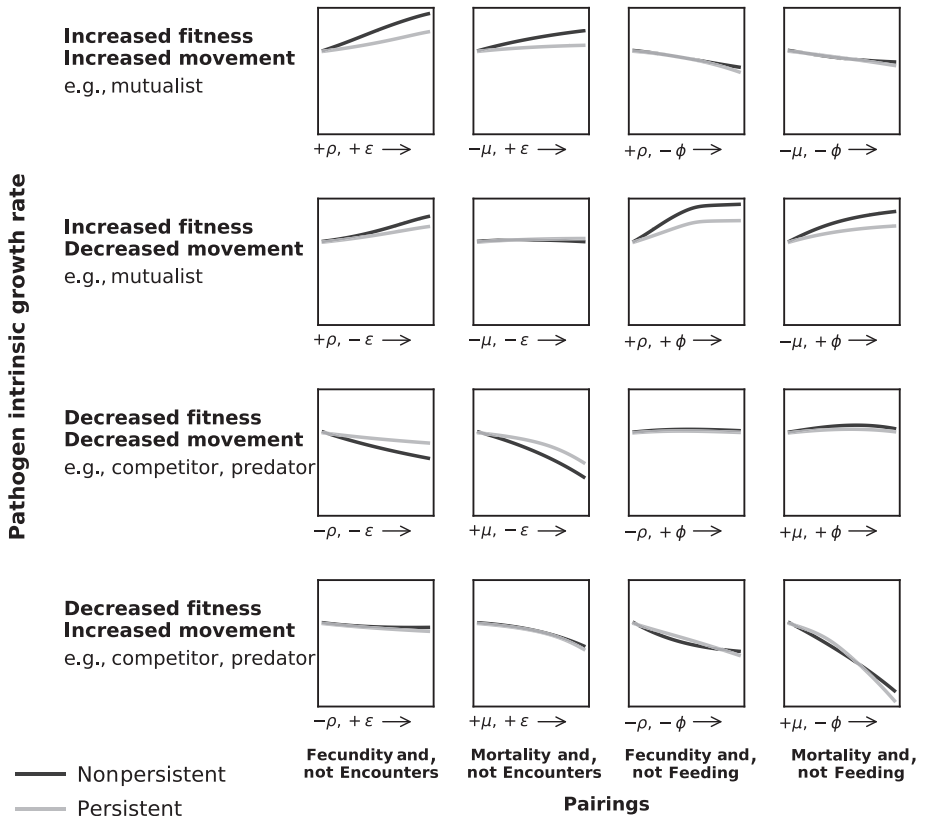

FIG. 5. Effects of interactions that affect both vector fitness (fecundity or mortality) and vector movement (time spent feeding or vector–host encounter rates) on the intrinsic growth rate of pathogens (d^{-1}). The *x*-axis of each panel shows the conditions affected (ρ = fecundity; μ = mortality; ε = encounter rates; Φ = time feeding) and values either increase (+) or decrease (−) from left to right. For each panel, the strength of the interaction increases compared to the standard model (parameters in Table 1 and Appendix S1: Table S4) as you move left to right along the *x*-axis. Thus, where the lines intersect with the left *y*-axis is the prediction of the standard model, and the lines show how the predicted rate of disease spread changes as the interaction strengthens for both nonpersistent (black) and persistent (gray) pathogens.

Species interactions may often impact more than a single vector characteristic. Our model shows that effects of such interactions on rates of pathogen spread range from strongly negative to strongly positive depending on the magnitude and direction of effects, and provide context for empirical observations. For example, vectors may respond to predators by decreasing plant-to-plant movement to avoid detection, as seen in the planthopper *Prokelisia marginata* (Denno et al. 2003). Our model suggests that such an interaction should considerably reduce rates of pathogen spread because of reduced vector fitness and plant-to-plant movement. In contrast, aphids respond to predators by simultaneously increasing nymph production and plant-to-plant movement (Kersch-Becker and Thaler 2015), which should greatly promote pathogen spread. Our model results show that interactions affecting multiple vector characteristics produced similar results for both persistent and nonpersistent pathogens, providing evidence that species interactions might exert effects on pathogens that equal or exceed effects of transmission mode (see Mauck et al. 2012).

Interactions might also produce vector responses that affect rates of pathogen spread in opposite directions. For example, competing herbivores often reduce the nutritional content of plants (Weibull 1987, De Mazancourt et al. 1998), lowering vector fitness (Awmack and Leather 2002), but stimulating plant-to-plant movement (Kareiva 1982). Similarly, predators increase vector plant-to-plant movement but reduce postdispersal fecundity in several aphid species (e.g., Roitberg et al. 1979, Nelson and Rosenheim 2006). Our model predicts that in such cases, effects of species interactions on pathogens should be marginal, unless the effects on one vector response (like movement) are considerably stronger than effects on the other (like fitness).

One key assumption of our model was that species interactions affected infectious and noninfectious vectors equally. However, this may not always hold. For example, parasitoids may prefer infectious compared to noninfectious vectors (De Oliveria et al. 2014), possibly because they complete development more rapidly in infectious vectors (Mauck et al. 2015), which would be expected to increase the effects of parasitoids on suppressing pathogens. Similarly, parasitoids have been shown to be more attracted to volatiles from infected compared to uninfected plants (Martini et al. 2014). Future models that explore such complexities may allow for more precise predictions of effects of species interactions on the rates of pathogen spread.

Our model shows that even relatively small impacts of species interactions on vectors can dramatically affect plant pathogens. Plant pathosystems provide excellent models to test these predictions, as host, vector, and pathogen populations can be manipulated on relevant spatial scales (Antonovics et al. 2002, Mitchell et al. 2002, Power and Mitchell 2004, Chisholm et al. 2018). Although our model may also provide insights into vector-borne pathogens infecting animal hosts, key differences in host movement and defense may cause results to differ from those reported here in animal pathosystems. Importantly, our model demonstrates that in both animal and plant pathosystems, examination of how species interactions affect vector movement and fitness may provide key new insights into the conditions under which interactions among these factors alter infection rates in hosts. In turn, the impacts of species interactions should be incorporated more broadly into the planning of disease management strategies to suppress infectious disease spread across ecological communities with a variety of species interactions.

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Supporting Information

Additional supporting information may be found in the online version of this article at http://onlinelibrary.wiley.com/doi/10.1002/ecy.2782/suppinfo

Data Availability

Data are available from GitHub/Zenodo (https://doi.org/10.5281/zenodo.2656763).