Model analysis for plant disease dynamics co-mediated by herbivory and herbivore-borne phytopathogens

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Plants are subject to diseases caused by pathogens, many of which are transmitted by herbivorous arthropod vectors. To understand plant disease dynamics, we studied a minimum hybrid model combining consumer–resource (herbivore–plant) and susceptible–infected models, in which the disease is transmitted bi-directionally between the consumer and the resource from the infected to susceptible classes. Model analysis showed that: (i) the disease is more likely to persist when the herbivore feeds on the susceptible plants rather than the infected plants, and (ii) alternative stable states can exist in which the system converges to either a disease-free or an endemic state, depending on the initial conditions. The second finding is particularly important because it suggests that the disease may persist once established, even though the initial prevalence is low (i.e. the $R_0$ rule does not always hold). This situation is likely to occur when the infection improves the plant nutritive quality, and the herbivore preferentially feeds on the infected resource (i.e. indirect vector–pathogen mutualism). Our results highlight the importance of the eco-epidemiological perspective that integration of tripartite interactions among host plant, plant pathogen and herbivore vector is crucial for the successful control of plant diseases.

Keywords: basic reproductive number; pathosystem; pest control; plant epidemiology

1. INTRODUCTION

Plant disease control is a continuing and pressing need for agriculture and forestry. The recent dramatic, worldwide prevalence of plant diseases in natural environments, so-called emerging plant diseases, are also gaining attention in the face of climate change, biological invasion and ecosystem destabilization owing to human activity [1]. Virtually all plant species are susceptible to diseases caused by various pathogens (viruses, bacteria and fungi), many of which are transmitted by herbivorous arthropod vectors [2]. The incidence rate and prevalence of plant pathogens depend on herbivore population dynamics and behaviour, plant resistance to infection and herbivory, and pathogen transmission between plant and vector [3–5]. Therefore, integration of tripartite interactions among host plant, plant pathogen and herbivore vector in community contexts is needed to control plant diseases successfully.

During the last two decades, several mathematical models describing plant–pathogen–vector dynamics have been developed, mainly for agricultural purposes [6–12]. These models are simple, mathematically tractable and can be easily extended to other more complicated or system-specific designs. However, the roles of trophic interactions between plant and herbivore vector in plant disease dynamics have not been consistently or explicitly considered, probably owing to the strategies for managed systems or for mathematical convenience, and thus remain a major concern. Some models assume that the plant is not damaged, although the vector is supported by the plant [6,7], while others assume that plant density or vector fecundity is constant, thereby decoupling plant and vector population dynamics [7–12]. To remedy these assumptions and to better understand plant disease dynamics, we explored a mathematical model in which we explicitly assumed that the vector is a herbivore (or functionally similar) that sustains its population through plant exploitation.

2. MODEL

We studied a minimum hybrid model combining consumer–resource (herbivore–plant) and susceptible–infected models:

$$\frac{dR_S}{dt} = r \left(1 - \frac{R_S + R_I}{K_S}\right)R_S - a_SR_S(G_S + G_I), \quad (2.1a)$$

$$\frac{dR_I}{dt} = vR_SC_1 - a_IR(S + I) - mR_I, \quad (2.1b)$$

$$\frac{dC_S}{dt} = (a_Sb_SR_S + a_bIR_I)(G_S + G_I) - dC_S - wR_GC_S \quad (2.1c)$$

and

$$\frac{dC_I}{dt} = wR_GC_S - dC_I, \quad (2.1d)$$

where $R_I$ and $C_I$ represent resource and consumer densities, respectively ($i = S$ or $I$, a susceptible or infected class, respectively). We made the simplest assumptions: the resource exhibits logistic growth ($r$, intrinsic growth rate; $K$, carrying capacity), all interactions are linear ($a$, feeding rate; $b$, conversion efficiency) and the consumer has a density-independent death rate ($d$). We adopted the conventional assumption that the infected resource is non-vigorous and is lost owing to natural and disease-induced additional mortality (i.e. virulence) with the rate $m$, while limiting population growth via density dependence. The pathogen is transmitted bi-directionally between the resource and the consumer, and the parameter $v$ (or $w$) determines transmission efficiency from the infected consumer (or resource) to the susceptible resource (or consumer). We considered neither vertical (mother to offspring) nor horizontal (between conspecifics) infection. Note that the pathogen is harmful only to the resource, and the susceptible and infected classes

of the herbivore have identical demographic performances. We intentionally ignored any parameter correlations (e.g. virulence–transmission trade-off), so that we evaluated the confounding effects of associated ecological and physiological processes independently.

Using this model, we performed a local stability analysis for possible equilibria, and derived an epidemic threshold determining pathogen inviability (i.e. basic reproductive number $R_0 > 1$; [13]; see electronic supplementary material, S1 and S2, respectively).

### 3. RESULTS

Three equilibria are possible: only the resource exists ($E_1$), and the resource and consumer coexist without and with disease ($E_2$ and $E_3$, respectively). The equilibrium values of $(R_S, R_C, C_S, C_I)$ are represented as follows:

$$E_1 = (K, 0, 0, 0),$$

$$E_2 = \left( \frac{d}{a_S b_S}, 0, \frac{-r}{a_S} \left(1 - \frac{R_S}{K}\right), 0 \right),$$

and

$$E_3 = \left( R_S, \frac{vwR_S - ad}{a_S w C_S} - \frac{dm}{C_S}, \frac{b_I d m}{v(a_S b_S + b_I w) R_S} - \frac{d(a_S b_I + w)}{a_S d}, \frac{(vwR_S - ad) C_I - dm}{a_S d} \right).$$

where asterisks denote equilibrium values (see electronic supplementary material, S1 for $R_1$ at $E_1$).

The conditions under which $E_1$ or $E_2$ exists and is locally stable are derived as follows:

$$K < \frac{d}{a_S B_S}$$

for $E_1$, and

$$K > \frac{d}{a_S B_S}$$

and

$$r(K a_S B_S - d) (v w - a_S a_B S) - K a_S^2 b_S^2 m < 0$$

for $E_2$. The conditions for $E_3$ are too complex to present here, but are determined by numerically evaluating the analytically derived conditions. We also confirmed that the pathogen invasion criterion $R_0 > 1$ is identical to the inverse of inequality 4b (electronic supplementary material, S2).

We show representative equilibrium states in a parameter space of resource-carrying capacity $K$ and consumer death rate $d$ in figure 1. A common trend is that, at low $K$ and high $d$, only the resource persists (region I above solid lines), at intermediate levels, the resource and consumer coexist without disease (region II bounded by solid and dashed lines), and they can be infected at high $K$ and low $d$ (grey regions III and iii). The consumer-resource dynamics with the disease oscillate when $K$ is too high and $d$ is too low (dark grey region iii). Intriguingly, the system shows alternative stable states in some parameter space, in which the system converges to either a disease-free (region I or II) or an endemic (region III or iii) state, depending on the initial conditions (grey regions above solid or dashed lines in figure 1f and i). Numerical simulations demonstrated that the disease can invade the disease-free state if the initial density of the infected individuals is sufficiently high (figure 2); however, if they are too high, invasion fails owing to population oscillations caused by trophic interactions (electronic supplementary material, S3).

We explored the community structure for some biologically meaningful scenarios (figure 1). Here, we specifically focused on varying the relative feeding preference for the susceptible and infected resources ($a_S$ and $a_I$, respectively) and infection-induced alternation of resource quality ($b_I$), while keeping other parameters fixed. We note in advance that the results below are qualitatively similar when other parameters are varied (electronic supplementary material, S4). We observed that the endemic regions shrink with decreasing $a_S$ and increasing $a_I$ (compare the grey regions within each row of figure 1). Meanwhile, the endemic regions expand with increasing $b_I$, and this effect is more pronounced when $a_S$ is low and $a_I$ is high (compare the grey regions in the right column of figure 1). Note that the solid and dashed lines do not vary with $b_I$ (see inequalities (3.2) and (3.3b)). As a result, the alternative stable states described above emerge, particularly at low $a_S$, high $a_I$ and high $b_I$ (figure 1f and i).

### 4. DISCUSSION

To our knowledge, this is the first attempt to formally integrate the traditional consumer–resource and susceptible–infected models of ecology and plant epidemiology, respectively (see Hatcher & Dunn [14] for eco-epidemiology in animal systems). Our model analysis provides new insights into plant disease dynamics mediated by herbivore-borne phytopathogens.

First, we observed that disease is more likely to persist when the herbivore preferentially feeds on the susceptible plant. This is because the infected plant is maintained in the system owing to reduced herbivory. This may seem counterintuitive from the viewpoint of disease transmission, because the herbivore would have fewer opportunities to acquire the pathogen. However, we suggest that, as well as efficiency effects (i.e. pathogen transmission), mass effects (i.e. infected plant abundance) are crucial for disease persistence. Many herbivore vectors prefer feeding on healthy plants [4], and such preferences may allow infected plants to survive (until killed by disease) and continue infecting vectors, although inefficiently, thereby contributing to disease invasion and prevalence.

Another important finding is that disease-free and endemic states can be alternative stable states. This suggests that once established, the disease might persist, even though the initial prevalence is low. This finding has significant implications for plant disease management because plant epidemiologists have conventionally used the $R_0$ criterion (i.e. $R_0 > 1$) to predict disease invasion potential [13]. However, we have shown that this criterion might not coincide with the stability domain of the epidemic state (i.e. neither disease-free nor epidemic equilibria are globally
stable); this indicates that the $R_0$ rule does not necessarily determine pathogen invasibility. The possibility that the $R_0$ rule may underestimate the pandemic risk of herbivore-borne plant diseases should be considered. We showed that such a situation is likely to occur when infection improves the plant nutritive quality, and the herbivore preferentially feeds on infected plants. Therefore, our prediction can be explained by an indirect, mutual herbivore–pathogen relationship that contributes to invasion success and disease pandemics through a positive feedback. In the case where infected-plant density increases, population growth of the infected plant-feeding herbivore also increases, which in turn facilitates pathogen transmission to the plant; the opposite direction of the positive feedback applies to disease extinction. An increasing amount of empirical evidence supports this idea [15–20].

In conclusion, trophic interactions between plant and vector critically affect the plant disease dynamics mediated by herbivore-borne phytopathogens. We emphasize that our results are not presented in previous models that did not explicitly include such trophic relationships [6–12]. Future research is required to extend the present model to incorporate other factors that affect plant disease dynamics (e.g.

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**Figure 1.** Community structure in the $K–d$ parameter space. Only the resource exists in region I (above solid lines). The resource and consumer coexist free from disease in region II (bounded by solid and dashed lines). The consumer and resource can be infected, and their population dynamics are stable and unstable in regions III (light grey) and iii (dark grey), respectively. Slash marks indicate alternative stable states (grey regions above solid or dashed lines). Parameter values are $(a_S, a_I) = (0.75, 0.25), (0.5, 0.5)$ and $(0.25, 0.75)$, from left to right, and $b_I = 0.1, 0.2$ and 0.5, from top to bottom. Other parameters are $r = 1$, $b_S = 0.2$, $m = 0.5$, $v = 0.5$ and $w = 0.5$.

**Figure 2.** Disease invasibility according to the initial densities of infected resource and consumer. We estimated that invasion succeeds if the infected consumer density is greater than $10^{-4}$ at $t = 10^4$ (grey pixels) and fails otherwise (white pixels). All simulations began at equilibrium $E_1$. Parameter values are $(K, d) = (17.5, 1)$ and others are identical to those in figure 1.
parameter correlations, spatial structure, plant and vector immunity or other interspecific interactions).

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