Pathogens and Mutualists as Joint Drivers of Host Species Coexistence and Turnover: Implications for Plant Competition and Succession

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ABSTRACT: The potential for either pathogens or mutualists to alter the outcome of interactions between host species has been clearly demonstrated experimentally, but our understanding of their joint influence remains limited. Individually, pathogens and mutualists can each stabilize (via negative feedback) or destabilize (via positive feedback) host-host interactions. When pathogens and mutualists are both present, the potential for simultaneous positive and negative feedbacks can generate a wide range of possible effects on host species coexistence and turnover. Extending existing theoretical frameworks, we explore the range of dynamics generated by simultaneous interactions with pathogens and mutualists and identify the conditions for pathogen or mutualist mediation of host coexistence. We then explore the potential role of microbial mutualists and pathogens in plant species turnover during succession. We show how a combination of positive and negative plant-microbe feedbacks can generate a coexistence state that is part of a set of alternative stable states. This result implies that the outcomes of coexistence from classical plant-soil feedback experiments may be susceptible to disturbances and that empirical investigations of microbially mediated coexistence would benefit from consideration of interactive effects of feedbacks generated from different distinct components of the plant microbiome.

Keywords: plant-soil feedback, coexistence, succession, mutualist, pathogen.

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Introduction

Classic ecological theory identifying resource partitioning as a primary reason for coexistence of species within guilds has played a central role in our understanding of the structure of communities (Schoener 1974; Ross 1986; Tilman 2004). While resource partitioning has been shown to be important, strong evidence of pathogen and mutualist effects on interactions among hosts has generated interest in the potential for these symbionts to drive host-host interactions. This is particularly true in plant ecology, where arguments have emerged that pathogens and mutualists are dominant forces structuring plant communities (van der Heijden et al. 2008; Mangan et al. 2010; Bever et al. 2015; Eppinga et al. 2018). The potential conflicts emerging from joint influences of pathogens and mutualists on plant-plant interactions, however, have rarely been considered.

Dynamics of pathogens can drive dynamics among their hosts. For example, cross-species infection (pathogen spillover) can lead to reinforcing dynamics and competitive exclusion (Holt et al. 2003; Power and Mitchell 2004; Rudolf and Antonovics 2005). However, there are several general conditions under which dynamics of pathogens can facilitate plant species coexistence. Perhaps most importantly, a pathogen with density-dependent transmission may enable plant species coexistence when the competitively superior host is more vulnerable to the pathogen (Holt et al. 1994; Mordecai 2013*a*). Dynamics of a shared pathogen can also lead to plant species coexistence when pathogen transmission is more common within than among plant species (Holt and Pickering 1985; Mordecai 2013*b*).

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Inclusion of multiple pathogens allows for specialization on hosts, generating broad conditions for host coexistence (Bever et al. 1997; Chesson 2000; but see Spear et al. 2015; Parker and Gilbert 2018). While tests focusing on individual pathogens have given variable results as to their influence on plant species coexistence (Mordecai 2013*b*; Spear and Mordecai 2018), plant-soil feedback studies, which integrate across multiple groups of pathogens as well as mutualists, identify pathogens as playing an important role in plant species coexistence (Crawford et al. 2019).

Host-host interactions can also be influenced by interactions with mutualists. Accumulation of mutualists is classically thought to be destabilizing of host-host interactions because mutualist-responsive hosts are often assumed to also be better hosts for mutualists, inducing a positive feedback (Hartnett and Wilson 1999; Hart et al. 2003). For example, plant preferential allocation of resources to the most effective mutualists, as has been demonstrated in rhizobia (Kiers et al. 2003; Oono et al. 2011) and arbuscular mycorrhizal (AM) fungi (Bever et al. 2009; Kiers et al. 2011; Ji and Bever 2016), could generate symbiont specialization and positive feedbacks. However, changes in density of microbial mutualists could generate a negative feedback and stabilize host-host interactions if the most responsive plant species is also a poorer host for mutualists (Umbanhowar and McCann 2005). This could happen when the preferential allocation of resources to mutualists results in a cost to the host (Steidinger and Bever 2014; Jiang et al. 2017). In fact, host-specific changes in mutualist composition can feed back positively or negatively on plant dynamics depending on correlations of plant and fungal fitness effects (Bever 1999). While negative feedbacks through the AM fungal community have been observed (Bever 2002), meta-analyses reveal that feedbacks through mutualisms are generally less negative than those through pathogen communities (Crawford et al. 2019).

Hosts commonly interact with both pathogens and mutualists simultaneously. For example, most plant species simultaneously interact with both root pathogens and beneficial mycorrhizal fungi (Bennett et al. 2006; van der Heijden et al. 2008; Bever et al. 2010; Rúa and Umbanhowar 2015). It is therefore important to consider the net effects of joint pathogen and mutualist dynamics on host-host interactions. When pathogens and mutualists generate complementary dynamics (e.g., both generate negative feedback), the net dynamics may not qualitatively differ from the sum of the individual dynamics. Yet it is possible, and not unlikely, that pathogens and mutualists generate contrasting dynamics (one positive feedback and the other negative feedback). In this case, the net dynamics could be qualitatively different than the sum of the effects of the individual interactions and difficult to infer from knowledge of only the individual interactions.

The goal of this article is to explore the implications of hosts interacting simultaneously with pathogens and mutualists. We are particularly interested in exploring these interactions in the context of likely life-history correlations in the types of host-pathogen and host-mutualist interactions occurring at the same time. For example, early-successional plant species have been found to have low responsiveness to mycorrhizal fungi, while late-successional plant species have high responsiveness (Janos 1980; Koziol and Bever 2015). Early-successional plant species have also been found to be more poorly defended against pathogens than late-succession plant species, which together might explain observations of rapid accumulation of negative soil community feedbacks on early-successional plants and weaker negative feedbacks on late-successional plant species (van der Putten et al. 1993; Kardol et al. 2006; Middleton and Bever 2012; Bauer et al. 2015).

Here we use a dynamical modeling framework to explore how pathogens and mutualists simultaneously affect host plant species coexistence. We identify the conditions under which the joint actions of pathogens and mutualists can mediate coexistence. Coexistence of host plant species is possible as long as at least one of the plant-microbe feedbacks is negative and given certain constraints on resource availability and relative competitive abilities of host plants. Interestingly, joint plant-pathogen and plant-mutualist feedbacks could result in a coexistence state as an alternative stable state, alongside exclusion of some of the community members. Surprisingly, coexistence can arise in cases where coexistence would not be possible in either plant-microbe subsystem. Finally, we illustrate the potential role of plant-microbe feedbacks on species turnover during succession.

Methods

Model Description

Our model tracks the density of biomass per ground unit area of two plant species, P_1 and P_2; density of mutualistic microbes, M; and pathogenic ("enemy") microbes, E. In the absence of interactions with microbes, the plant populations grow and compete according to the Lotka-Volterra competition model with density-independent mortality. Because resource supply and acquisition are not explicitly represented in the Lotka-Volterra model, we define plant competitive ability as its intrinsic growth rate in the absence of microbes. Each plant's maximum population growth rate increases as a result of interactions with mutualistic microbes that increase resource uptake. We assume that the pathogenic microbes increase each plant's mortality rate, although our conclusions still hold if we instead model pathogenic effects in a similar way as mutualist effects (figs. S1,

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S2; figs. S1–S8 are available online). Both microbe populations grow in response to the plant abundances and experience density-independent mortality. The model equations are

$$\frac{dP_j}{dt} = (a_j + \mu_j M)P_j\left(n - \sum_{i=1}^{2} P_i\right) - (d_j + \beta_j E)P_j, \quad (1a)$$

$$\frac{dM}{dt} = \ell M \sum_{i=1}^{2} \frac{b_i P_i}{1 + b_i M} - d_M M, \quad (1b)$$

$$\frac{dE}{dt} = E \sum_{i=1}^{2} c_i P_i - d_E E, \quad (1c)$$

where $j = 1, 2$. This is a two-plant extension of the model described by Rúa and Umbanhowar (2015).

The intrinsic population growth rate for plant i is a_i in the absence of the mutualist, and it is increased by μ_i per unit of mutualistic microbe density. The parameter n quantifies the total resource supply available to plants, expressed in units of plant biomass density. Therefore, the current resource level available to support plant population growth is $n - \sum P_i$. As dominant plant mutualists (mycorrhizal fungi and nitrogen-fixing bacteria) promote plant fitness through improved growth rather than decreased mortality, we assume that mutualists increase plant growth rates (Umbanhowar and McCann 2005; Bennet et al. 2006; Rúa and Umbanhowar 2015). Pathogens are assumed to increase mortality as in the common modeling approach (Holt et al. 1994; Eppinga et al. 2006; Mordecai 2013*a*). Plant i's density-independent mortality rate is d_i in the absence of the pathogen and is increased by β_i per unit pathogen biomass density.

The mutualist population grows in a density-dependent manner in response to photosynthate allocated to it by each plant species. The baseline growth rate of the mutualist is given by ℓ, augmented by a factor of up to b_i per unit of plant i biomass. The mutualist population then grows at a maximum per capita rate of $\ell \sum b_i P_i$, and with increasing mutualist density this growth rate saturates according to the Beverton-Holt type of density dependence (Beverton and Holt 2012). The mutualistic microbe's density-independent mortality rate is d_M.

Finally, the pathogenic microbe grows in proportion to plant density, depending on the parameter c_i, which measures how well the pathogen grows on plant species i. We note that our main conclusions do not depend on the different assumptions of density dependence for mutualists and pathogens. More specifically, we arrive at the same conclusions when both mutualists and pathogens exhibit the Beverton-Holt type of density dependence (figs. S1, S2). The pathogen's density-independent mortality rate is d_E. The definitions of all parameters, along with the values used in most of our analyses, can be found in table 1.

Biological Interpretation of Key Parameters

The parameters that directly describe the interaction between plant species i and the mutualistic microbe are μ_i and b_i. A plant that is a good host to the mutualist (i.e., more strongly promotes mutualist population growth) will create a high b_i. A highly responsive plant—one that derives a strong benefit from associating with the mutualist—will have a high μ_i. A positive pairwise plant-mutualist feedback occurs when μ_i and b_i are positively correlated, so that plants that are more affected by the mutualist (high μ_i) also allocate more energy to supporting it (high b_i). If μ_i and b_i are negatively correlated, a negative pairwise plant-mutualist feedback occurs (Bever 1999; Umbanhowar and McCann 2005).

The interaction between plant species i and the pathogen is described by the parameters β_i and c_i. A plant that

Table 1: Parameters used in the model

		Default value (used in figures unless noted otherwise)		
Symbol	Description	$i = 1$	$i = 2$	Dimensions
a_i	Maximum population growth rate of plant i	.5	1.0	B^{-1} A T^{-1}
b_i	Ability of mutualist to grow on plant i	.17	.68	B^{-1} A
c_i	Ability of enemy to grow on plant i	.83	1.5	B^{-1} A T^{-1}
d_i	Mortality rate of plant i	.13	.10	T^{-1}
d_M	Mortality rate of mutualist	.5	.5	T^{-1}
d_E	Mortality rate of enemy	1.0	1.0	T^{-1}
ℓ	Baseline growth rate of mutualist	1.0	1.0	T^{-1}
n	Availability of plants' resource	1.5	1.5	B A^{-2}
β_i	Plant i's responsiveness to enemy	.25	1.0	B^{-1} A T^{-1}
μ_i	Plant i's responsiveness to mutualist	1.5	.1	B^{-2} A^2 T^{-1}

Note: A = area; B = biomass; T = time.

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strongly promotes pathogen growth will have a high c_i, and a highly responsive plant to which the pathogen is particularly detrimental will have a large β_i. Therefore, a negative plant-pathogen feedback occurs when β_i and c_i are positively correlated, so that plants that are a better host to the pathogen (high c_i) also receive more damage from it (high β_i). If β_i and c_i are negatively correlated, a positive pairwise plant-pathogen feedback occurs (Holt et al. 2003).

Through variation of parameter values, we first explore the range of dynamical outcomes that occurs in different scenarios of resource availability, host competitive ability, and host-microbe feedbacks. Specifically, we keep the parameter values for plant 2 fixed while varying plant 1's responsiveness to the pathogen (β_1), pathogen-hosting ability (c_1), responsiveness to the mutualist (μ_1), and mutualist-hosting ability (b_1) to create a full combination of positive/negative plant-pathogen/mutualist feedbacks crossed with which plant is better at hosting the pathogen and mutualist. We then explore the consequences of correlations in parameter values expected for plants of different successional stages, such that plant 2 is an early-successional species and plant 1 is a late-successional species. Here plant 2 wins in the absence of microbes ($d_2/a_2 < d_1/a_1$) but is more susceptible to pathogens ($\beta_2 > \beta_1$, $c_2 > c_1$) and less responsive to mutualists ($\mu_2 < \mu_1$, $b_2 < b_1$) than plant 1.

Model Analyses

Nullcline Analysis of the Component Plant-Pathogen and Plant-Mutualist Feedbacks. Because our goal is to understand the effect of multiple, simultaneous plant-microbe feedbacks, we begin by analyzing the three-species submodels that include two plants with one of the microbes, in which these feedbacks originate. For completeness, we also analyzed the other three-species submodel (one plant with two microbes) and both two-species submodels (one plant with one microbe) and present those results in appendix S1 (apps. S1, S2 are available online).

To understand how mutualists and pathogens each generate feedbacks to mediate the coexistence of host plants, we conduct invasion analyses for systems of two plants and one microbial species using nullcline plots, where the nullclines for each plant-microbe pair are overlaid following Rúa and Umbanhowar (2015). This overlay is useful for this analysis due to our assumption that available resources can be represented as $n - \sum P_i$. This means that we can determine at any single pair of plant-microbe densities, represented as a point, whether the other plant species can increase when rare. More formally, invasion analysis proceeds by investigating whether a given plant species can invade when the resident plant species has reached an equilibrium with its one microbe. Resident plant equilibrium density is denoted $\hat{P}_i$, and the microbial density at this equilibrium is denoted $\hat{M}$ or $\hat{E}$. According to the invasion criteria, plant j can invade when rare if $dP_j/dt > 0$—that is, if the resident plant species equilibrium point is below the invader nullcline—and will be excluded if the reverse holds.

Linear Stability Analysis of the Full Four-Species Model. We calculated the equilibrium values for the full four-species model (eqq. [1]) and performed linear stability analyses for each of the equilibrium points by evaluating the Routh-Hurwitz stability criteria across different parameter combinations. We defined four scenarios, which we call cases A–D, on the basis of each plant's ability to host the mutualist (b_i) or the pathogen (c_i) across a range of resource levels (n) and relative plant population growth rates (a_1/a_2 ratios). In case A, both plants are better at hosting the pathogen than the mutualist (i.e., $d_E/c_i < d_M/\ell b_i$). In cases B and C, one plant is better at hosting the pathogen and the other plant is better at hosting the mutualist. In case D, both plants are better at hosting the mutualist (i.e., $d_E/c_i > d_M/\ell b_i$).

Within each of the four scenarios, the plant-mutualist feedback and the plant-pathogen feedback can be positive or negative, creating four subscenarios. The only exception occurs in case A, in which the mutualist cannot persist, and therefore only two subscenarios are possible (i.e., positive and negative plant-pathogen feedback). Hence, the four scenarios comprise 14 subscenarios with unique combinations of plant characteristics and directions of plant-microbe feedbacks. The linear stability analyses were carried out for equilibrium points in each of the 14 subscenarios. All calculations were performed in MATLAB R2016b (MathWorks).

Numerical Simulations of Succession. To investigate how mutualists and pathogens drive plant successional trajectories, we simulated the dynamics of two-plant communities with differences in life-history traits, subjected to the introduction of microbes. As noted above, plant 2 is an early-successional species with parameter values as defined in table 1, while plant 1 is a late-successional species whose life-history traits vary in different scenarios. All simulations start with only the two plant species present, in which case plant 1 would eventually be excluded according to Lotka-Volterra competition. We introduced the microbes before complete exclusion of plant 1, and we varied the order in which pathogens and mutualists were introduced to examine which pathways enabled succession (i.e., turnover to a system state in which the late-successional plant 1 persists with the mutualist). The first microbe was introduced after 50 time steps, the second microbe was introduced 100 time steps after initialization, and then the model was run until equilibrium was reached. All simulations were performed using MATLAB R2016b.

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Results

Positive and Negative Feedbacks within Two-Plant, One-Microbe Subcommunities

Nullcline analysis identified that pathogens and mutualists can each initiate negative feedback facilitating coexistence or positive feedback leading to competitive exclusion (fig. 1). Our results for the two-plant, one-microbe subsystems agree well with previously developed theory on competitors with a shared predator or shared mutualist (Holt et al. 1994, 2003; Umbanhowar and McCann 2005), which we summarize here to provide necessary context for interpreting the four-species model.

In the plant-pathogen subsystem, pathogens drive negative feedback when the plant species that is the best host for the pathogen (higher c_i) is also most sensitive to the pathogen (higher β_i), and both plants can invade when rare (fig. 1*a*). In contrast, under positive feedback both single-plant equilibrium points are locally stable, and neither plant can invade when rare (fig. 1*b*). These results are not sensitive to whether pathogens influence plant growth or mortality or whether pathogen growth is density dependent (fig. S1). The plant-mutualist subsystem has similar dynamics, in which both negative and positive feedbacks are possible (fig. 1*c*, 1*d*). Negative feedback occurs when the plant that is the best host for the mutualist (higher b_i) is the least responsive to the mutualist (lower μ_i). Positive feedback and alternative stable states emerge when the plant that is the best host for the mutualist (higher b_i) is also the most responsive to the mutualist (higher μ_i), as is often assumed of mutualisms (fig. 1*d*).

Given differences in microbial response, how different must the plants' hosting abilities be for them to coexist through negative plant-soil feedback? This depends in part on how the plants compete for resources. The ratio b_1/b_2 (where b_i is plant i's ability to host the mutualist) provides a continuous measure of the plants' difference in hosting ability toward the mutualist. The bar to the left of the *Y*-axis in figure 2 shows the outcomes of competition in the two-plant, one-mutualist subcommunity, when plant 1 is a competitively inferior species that receives higher benefits from the mutualist. We clearly see cases of negative plant-mutualist feedback enabling coexistence (low b_1/b_2 ratios, marked with a red box labeled $P_1 + P_2 + M$) and positive plant-mutualist feedback generating alternate stable states (high b_1/b_2 ratios, marked with a green box labeled $P_1 + M$ or $P_2 + M$) as previously described. However, we also see that when the plants' abilities to host the mutualist are relatively similar (b_1/b_2 near 1, marked with the blue box), the plant-mutualist feedback is not strong enough to drive the dynamics, meaning that the competitively superior plant 2 excludes plant 1.

Similarly, the ratio c_1/c_2 (where c_i is plant i's ability to host the pathogen) provides a continuous measure of the plants' difference in pathogen-hosting ability. When plant 1 is more resistant to the pathogen, we see the expected negative feedback and coexistence for low c_1/c_2 ratios (red $P_1 + P_2 + E$ box under the *X*-axis) and positive feedback and alternative stable states for high c_1/c_2 ratios (green box). Again, when the plants have similar hosting abilities (c_1/c_2 near 1, blue box), competition is the primary determinant of the outcome and plant 2 excludes plant 1. Changing our assumptions about how plants and pathogens interact does not qualitatively alter these results (fig. S2).

Feedbacks through Pathogens and Mutualists Jointly Mediate Plant Coexistence and Alternative Stable States in Two-Plant, Two-Microbe Communities

Now we can consider interactions between microbes, where every parameterization of the four-species system corresponds to an (x, y)-coordinate in the parameter space of figure 2. There are regimes where mutualists are excluded in the presence of pathogens and hence the dynamics of the system reduce to the two-plant, pathogen system (from the bottom left corner to the top right corner of fig. 2). However, when both microbes persist, the four-species system also shows fundamentally different behavior than either subsystem containing only one microbial species (fig. 2). For example, in the upper left region of parameter space, the positive plant-mutualist feedback would enable either plant to exclude the other if the system contained only the mutualist. However, the presence of pathogens creates a negative feedback that prevents competitive exclusion of plant 1 but is not strong enough to cancel out the existence of alternative stable states driven by the positive mutualist feedback. The result is alternative stable states, still caused by the positive plant-mutualist feedback but now involving a different pair of states: the equilibrium from the pathogen-only subsystem plus an equilibrium with one plant species and the two microbes (fig. 2). A similar outcome can be observed in the lower right quadrant, where the interaction between a positive plant-pathogen feedback and a negative plant-mutualist feedback creates a coexistence equilibrium with both plants and both microbial species present, even though plants could not coexist in either subsystem (fig. 2). At the coexistence equilibrium, the negative plant-mutualist feedback dominates, but a disturbance that would weaken this feedback, such as a reduction in mutualist density or a reduction in the better host for the mutualist, could be amplified by the positive plant-pathogen feedback, initiating the development to an alternative, pathogen-dominated state in which only the most pathogen-resistant plant persists (fig. 2).

Although we present only this example in the main text, when we consider all 14 possible combinations of feedbacks and plant characteristics (table 2) across a range of

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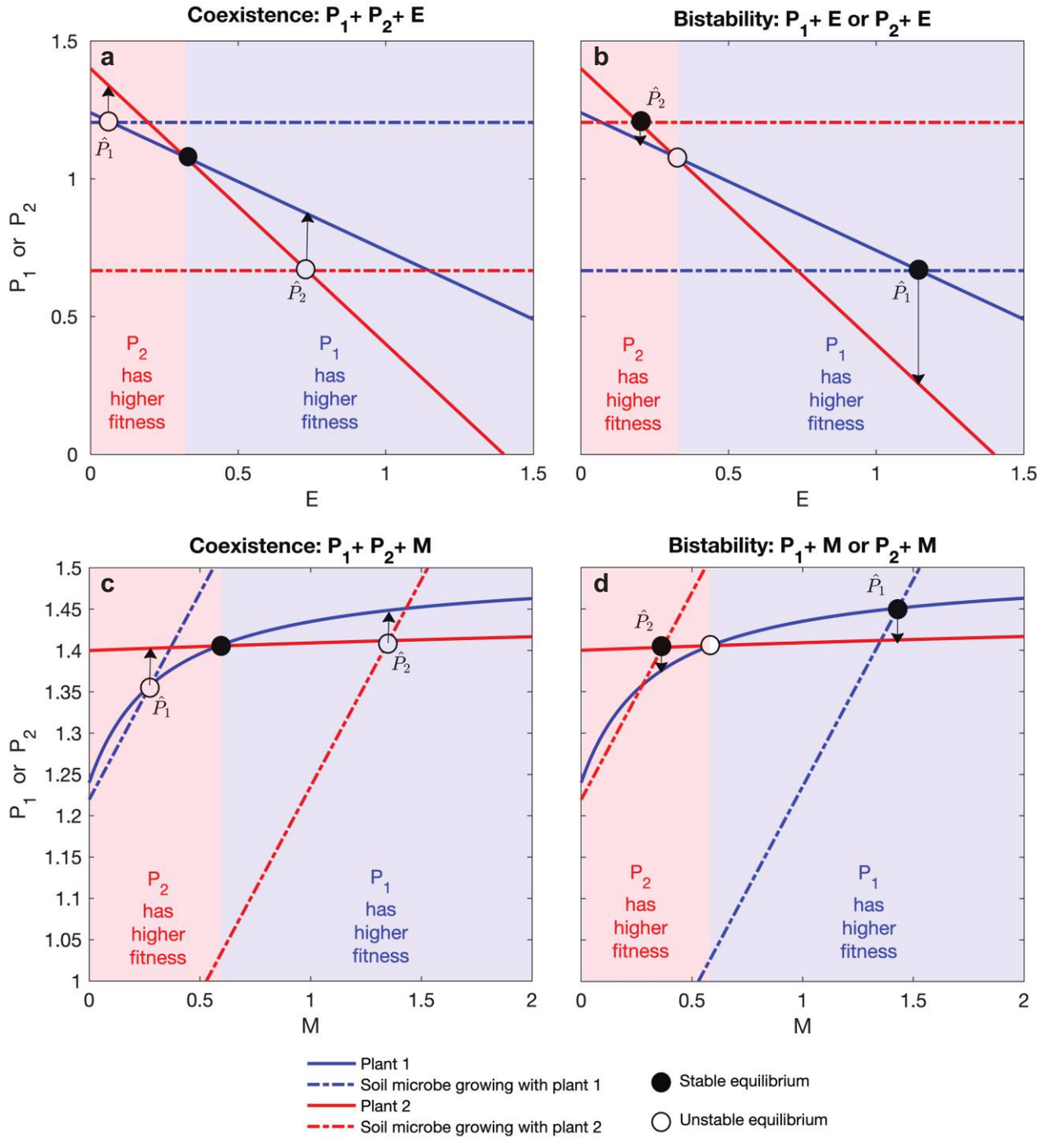

Figure 1: Nullclines for single-plant systems on the plant-pathogen nullcline space (*a*, *b*) or plant-mutualist nullcline space (*c*, *d*). Solid lines represent plant nullclines, and dashed lines represent pathogen or mutualist nullclines. In the cases of coexistence (*a*, *c*), equilibrium plant densities $\hat{P}_i$ can be invaded by the competing species that has higher fitness (up arrows). In the cases of bistability (*b*, *d*), equilibrium plant densities $\hat{P}_i$ cannot be invaded by the competing species (down arrows). The four panels differ in the plants' abilities at hosting the mutualist (b_1, b_2) or pathogen (c_1, c_2). Parameter values are $c_1 = 0.83$, $c_2 = 1.5$ in *a* and $c_1 = 1.5$, $c_2 = 0.83$ in *b*. The other parameters are set to the default values in table 1. In *c* and *d*, the parameters are the same as in *a* except that $b_1 = 0.41$, $b_2 = 0.68$ in *c* and $b_1 = 0.68$, $b_2 = 0.41$ in *d*.

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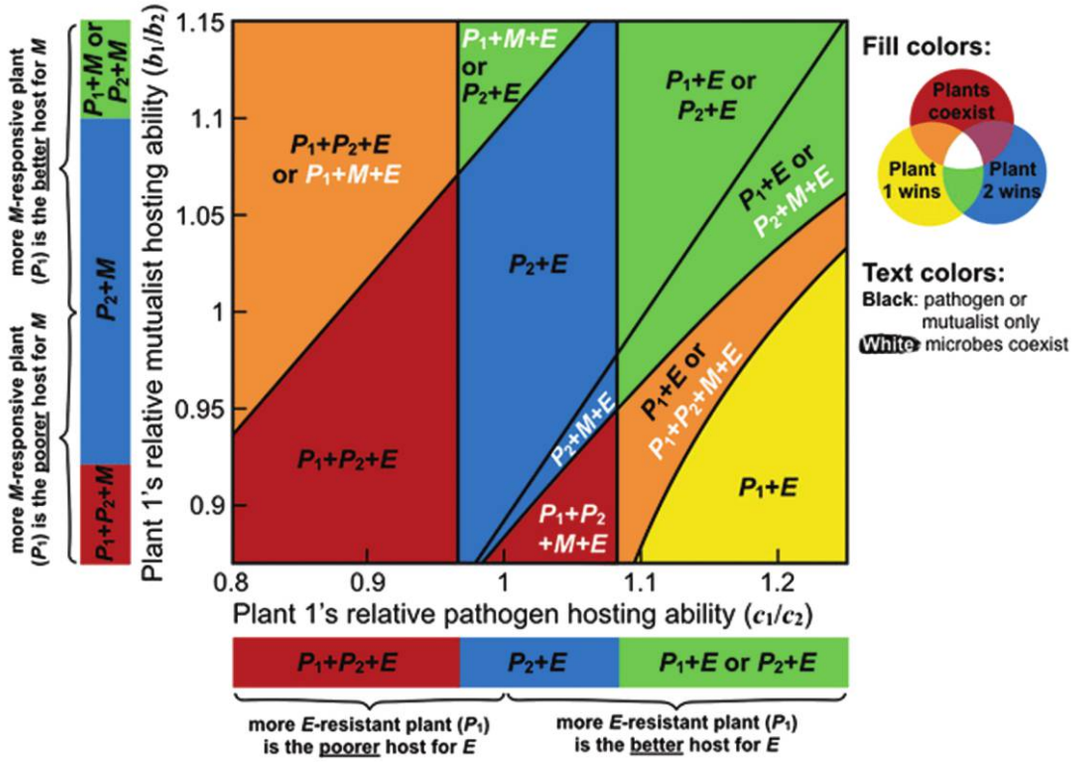

Figure 2: Stable states in the four-species system across b_1/b_2 – c_1/c_2 parameter space. When the b_1/b_2 (or c_1/c_2) ratio is >1, plant 1 is better at hosting the mutualist (or pathogen) than plant 2. These ratios were adjusted by varying b_2 between 0.4 and 0.46 and c_2 between 0.8 and 1.0 while holding $c_1 = 1.8 - c_2$ and $b_1 = 0.86 - b_2$. The other parameters are set to the default values in table 1. The colored bar below the *X*-axis shows the behavior of the two-plant–pathogen subsystem for this range of b_1/b_2 ratios, and the colored bar to the left of the *Y*-axis does the same for the two-plant–mutualist subsystem. Primary colors (red, yellow, blue) mark parameter combinations with one stable plant community at equilibrium (plant coexistence, plant 1 alone, or plant 2 alone, respectively). Secondary colors (orange, purple, green) mark regions where two of these plant communities exist as alternative stable states, as illustrated in the key. Text colors correspond to the number of microbial taxa present in the stable equilibrium state(s).

resource availabilities, we find that these alternative stable states can emerge readily when multiple microbe species are explicitly considered (app. S2). In the following section, we explore the implications of this notion within the context of succession.

Implications for Succession

We explored the potential role of life-history correlations in plants' ability to resist pathogens (β) and their ability to benefit from the mutualist (μ), because the plant community is thought to increase pathogen resistance and mutualist responsiveness in a correlated manner through succession (see the introduction). We further assumed a negative plant-pathogen feedback, in which the higher ability of a plant to host the pathogen (c) is correlated with higher vulnerability to the pathogen (β), and a positive plant-mutualist feedback, in which higher ability to host the mutualist (b) is correlated with stronger responsiveness to the mutualist (μ). We varied the strengths of these patterns for both plant species as shown in figure 3, with plant 1 becoming more typical of a late-successional species (with higher pathogen resistance, lower pathogen-hosting ability, higher mutualist responsiveness, and better mutualist-hosting ability) relative to plant 2 when moving upward or rightward (fig. 3). When plant 1 is at a similar successional stage as plant 2 (i.e., has similar hosting and response traits; bottom left corner of fig. 3), the competitively superior plant 2 will always exclude plant 1. In contrast, when the successional stage difference is greater, the late-successional species plant 1 will always exclude plant 2 (fig. 3, top right corner). Coexistence of both plants, pathogens, and mutualists is also possible if plant 1 has a weak interaction with the mutualist but plant 2 also has a strong interaction with the pathogen.

Figure 3 also implies that turnover from the early-successional plant with the enemy to the late-successional plant with the mutualist could, for weaker correlations

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Table 2: Summary of the 14 subscenarios, distinguished by how positive and negative feedbacks of various strengths are combined

Case	*P-M* feedback	*P-E* feedback	Coexistence	Alternative stable states	Example
	Case A: both plants are better hosts to *E* than to *M* $\left(\frac{d_E}{c_i} < \frac{d_M}{lb_i}, \frac{d_E}{c_j} < \frac{d_M}{lb_j}\right)$				
A1	na	−	Yes	No	fig. S3*A*
A2	na	+	No	Yes	fig. S3*B*
	Case B: P_i is a better host to *M* than to *E* $\left(\frac{d_E}{c_i} > \frac{d_M}{lb_i}\right)$; P_j is a better host to *E* than to *M* $\left(\frac{d_E}{c_j} < \frac{d_M}{lb_j}\right)$				
B1	−	−	Yes	No	fig. S4*A*
B2	+	−	Yes	Yes	fig. S4*B*
B3	−	+	Yes	Yes	fig. S4*C*
B4	+	+	No	Yes	fig. S4*D*
	Case C: P_i is a better host to *E* than to *M* $\left(\frac{d_E}{c_i} < \frac{d_M}{lb_i}\right)$; P_j is a better host to *M* than to *E* $\left(\frac{d_E}{c_j} > \frac{d_M}{lb_j}\right)$				
C1	+	−	Yes	Yes	fig. S5*A*
C2	−	−	Yes	No	fig. S5*B*
C3	+	+	No	Yes	fig. S5*C*
C4	−	+	No	Yes	fig. S5*D*
	Case D: both plants are better hosts to *M* than to *E* $\left(\frac{d_E}{c_i} > \frac{d_M}{lb_i}, \frac{d_E}{c_j} > \frac{d_M}{lb_j}\right)$				
D1	−	−	Yes	No	fig. S6*A*
D2	+	−	Yes	Yes	fig. S6*B*
D3	−	+	Yes	Yes	fig. S6*C*
D4	+	+	Yes	Yes	fig. S6*D*

Note: The *P-M* feedback column gives the sign of the plant (*P*)–mutualist (*M*) feedback (if present), and the *P-E* feedback column gives the sign of the plant-pathogen (*E*) feedback. All cases are written so that, of the two plants, plant *i* is the lower-quality host to the enemy ($c_i < c_j$). Negative *P-E* feedbacks thus occur when *i* is also the less responsive plant to *E* (relative to its own density-independent mortality rate: $\beta_i/\beta_j < d_i/d_j$). Negative *P-M* feedbacks occur when the same plant is both a lower-quality host to and most responsive to *M* compared to the other plant species ($b_i < b_j$ and $\mu_i > \mu_j$ or $b_i > b_j$ and $\mu_i < \mu_j$). na = not applicable.

between pathogen resistance and mutualist responsiveness (i.e., for trait values not on the diagonal from bottom left to top right), be driven by positive or negative feedbacks. For example, if the late-successional plant 1 is vulnerable to the pathogen and a good host for the mutualist, it may invade an early-successional system via an alternative stable state that contains both microbes (fig. 3, top left corner). Only when the late-successional plant 1 is much less vulnerable to the pathogen (fig. 3, top right corner) is the late-successional system state the only stable state, comprising the late-successional plant species and the mutualist. Given the prevalence of alternative stable states with weak-to-intermediate life-history correlations, numerical simulations are needed to provide insight into alternative successional pathways.

Specifically, to investigate how pathogens and mutualists might drive successional dynamics, we simulated several examples of two-plant communities under different sequences of microbial invasions. If the late-successional plant 1 is more vulnerable to the pathogen despite its strong mycorrhizal responsiveness, invasion of the mutualist alone (either before or after introduction of the pathogen) cannot drive succession (fig. 4*a*, 4*b*). To reach the late-successional state, which is alternatively stable to the early-successional state in this parameter range (fig. 3), the density of plant 1 has to be large enough at the time of the mutualist introduction (fig. S7). In the absence of pathogens, the early-successional plant 2 coexists with the mutualist temporarily until the introduction of the pathogen reduces plant 2's density to a level that is not suitable for the mutualist (fig. 4*a*). When plant 1 is more resistant to the pathogen, either sequence of microbial invasions can drive the succession to the late-successional state (fig. 4*c*, 4*d*), with different mid-successional stages. If the mutualist is introduced before the pathogen (fig. 4*c*), plant 2 coexists with the mutualist before introduction of the pathogen. However, introduction of the pathogen then reduces the density of plant 2, which favors establishment of plant 1 and the subsequent exclusion of pathogens. For the opposite invasion sequence, where the pathogen is introduced first, negative plant-pathogen feedback permits plant coexistence before introduction of the mutualist (fig. 4*d*). Subsequently, once the mutualist has been introduced, strong mycorrhizal responsiveness of plant 1 allows it to exclude plant 2 in the late-successional stage.

Discussion

Empirical studies have identified an important role of pathogens and mutualists in the dynamics of plant communities

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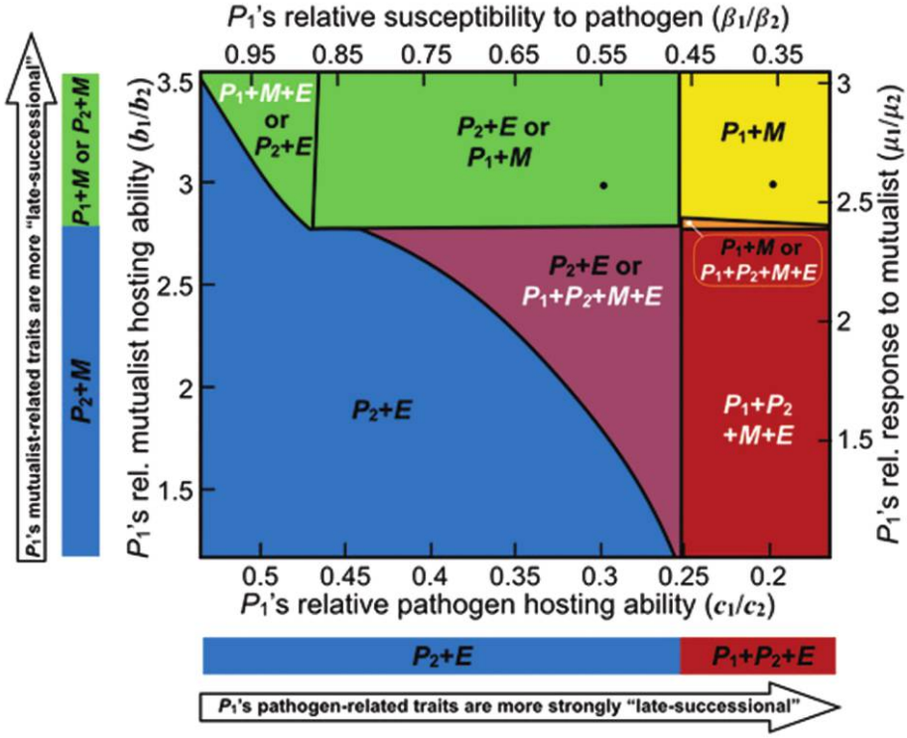

Figure 3: Four-species system across b_1/b_2 – c_1/c_2 parameter space, analogous to figure 2 except that we impose a concurrent decrease in β_1/β_2, plant 1's susceptibility to the pathogen relative to plant 2's, with decreasing of c_1/c_2 due to assumed life-history relationships ($\beta_1/\beta_2 = 1.85c_1/c_2$, $c_2 = 1.5$, $\beta_2 = 1.0$), and a concurrent increase in μ_1/μ_2, plant 1's relative responsiveness to the mutualist, with increasing of b_1/b_2 ($\mu_1/\mu_2 = 0.86b_1/b_2$, $b_2 = 0.68$, $\mu_2 = 0.1$). During succession, we expect parameters to change from those in the bottom left to the top right of this diagram. While these expected relationships generate positive feedback in mutualists, potentially inhibiting establishment of the late-successional plant (P_1), pathogen dynamics can facilitate late-successional plant establishment, even in the presence of positive feedbacks through mutualists. The two dots indicate parameter combinations selected for further simulations in figure 4. Fill and text colors follow the key in figure 2.

(Mangan et al. 2010; Bever et al. 2015). While previous theory has identified that both pathogens (Holt et al. 1994; Mordecai 2013*a*) and mutualists (Bever 1999; Umbanhowar and McCann 2005) can generate negative feedbacks and thereby contribute to plant species coexistence, our work extends this theory by evaluating the joint operation of pathogens and microbial mutualists. The most profound implications of our analyses emerged from systems in which one microbial species generated positive feedback while the other species generated negative feedback. In these systems, the negative feedback may stabilize a coexistence equilibrium, but a sufficiently large disturbance of the system may result in the positive feedback becoming the main driver and force the exclusion of one of the plant hosts (figs. 2, 3). An important implication of this result is that empirical evidence of negative feedback may provide an incomplete understanding of the stabilizing and destabilizing roles of soil microbes. Moreover, as the identified type of coexistence does not require both plant species to be able to recover when rare, it may not be detectable through the typical design of experimental plant-soil feedback studies (e.g., van der Putten et al. 1993; Bever et al. 1997; Revilla et al. 2013). Instead, the presented model predictions could be tested with a new type of pot experiment that independently and factorially manipulates components of the plant microbiome. Such an experiment might evaluate plant fitness and competitive effects across a range of initial densities of two-plant species (i.e., an additive design) factorially manipulated with the presence and timing of introduction of a pathogen and a mutualist.

Utilizing plant-soil microbe interactions to accelerate succession on ex-arable fields is an important theme in restoration ecology (Harris 2009; Kardol et al. 2009; Middleton and Bever 2012; Koziol et al. 2018). Our case study highlighted how the effectiveness of such restoration strategies may depend on the characteristics of the late-successional target species (fig. 4). For example, if late-successional target species are relatively vulnerable to soil pathogens, introducing mutualists may be a necessary but not sufficient restoration measure due to the presence of a

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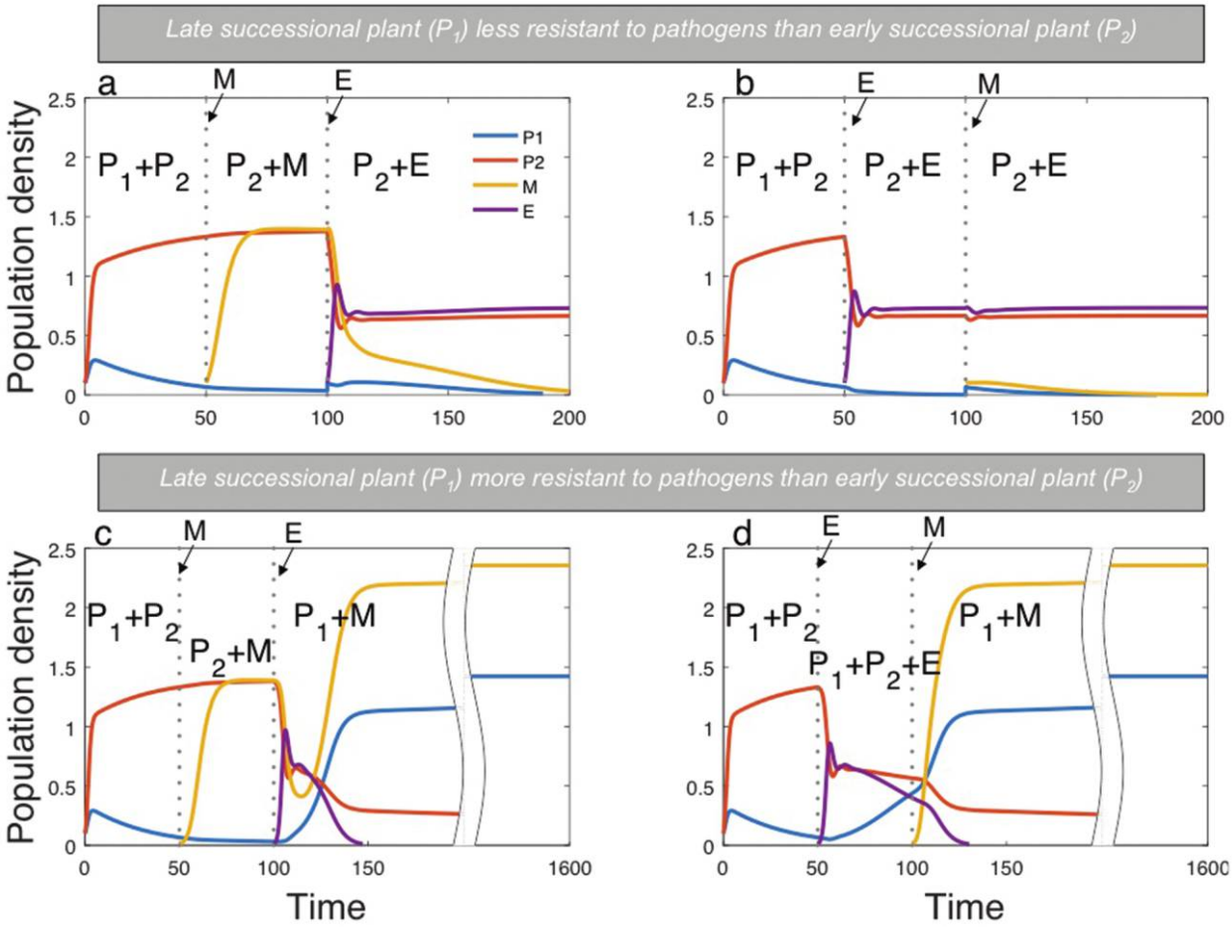

Figure 4: Time series for two-plant communities following different sequences of microbial invasion at different successional stages. Plant 2 is more early successional, and plant 1 is more late successional with strong mycorrhizal responsiveness. Plant 1 is more resistant to the pathogen in *c* and *d* than in *a* and *b*. All the simulations were started with only two plants present, and then the mutualist and pathogen were introduced in different orders, one at time step 50, the other at time step 100. Mutualists were introduced before pathogens in *a* and *c* and after pathogens in *b* and *d*. Parameters are set to the defaults in table 1, except that $b_1 = 2.04$ ($b_1/b_2 = 3.0$), $\mu_1 = 0.26$ ($\mu_1/\mu_2 = 2.58$) in all panels; $c_1 = 0.45$ ($c_1/c_2 = 0.3$), $\beta_1 = 0.56$ ($\beta_1/\beta_2 = 0.56$) in *a* and *b*; and $c_1 = 0.3$ ($c_1/c_2 = 0.2$), $\beta_1 = 0.37$ ($\beta_1/\beta_2 = 0.37$) in *c* and *d*, corresponding to the two dots in figure 3.

positive pathogen feedback. To overcome this feedback, introduction of mutualists would need to be accompanied by introducing the target plant species in sufficient densities (figs. 4, S7). In our case study, we also showed how a particular microbial species can play a crucial role in the succession process, despite being absent in the initial and final equilibrium states of the system (fig. 4*c*, 4*d*). Specifically, we showed an example where the late-successional species was more resistant to soil pathogens, and the presence of these pathogens was crucial in reducing the density of early-successional species to a level where late-successional species could establish and the system could develop to a late-successional stage without pathogens (fig. 4*c*, 4*d*). This crucial role of pathogens within the successional trajectory would be missed when analyzing only plant and soil community composition of the early- and late-successional equilibrium states. These model simulations also provide a mechanistic explanation for the limited success of restoration efforts that transplanted plant and soil communities of late-successional states into early-successional communities, emphasizing the potentially important role of soil microbes present only in intermediate-successional stages (Kardol et al. 2009).

Our goal in this study was to provide an overview of the potential feedbacks that could emerge between microbes and competing plant species that differ in their ability to host and respond to these microbes (table 2). We used a mean field modeling approach, considering the minimum number of functional groups needed to study mediation of plant coexistence by pathogens and mutualists. It is important

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to note the limitations of this particular approach. First, spatial interactions through microbial dispersal and infection processes may influence the dynamics but are not included in our model, which could be explicitly represented in individual-based models (Mack and Bever 2014; Vincenot et al. 2017). Second, in more diverse communities, interactions between plant species and soil microbes become more diffuse, as the impact of each plant species to drive changes in soil community composition reduces. Recent theory allows for quantifying the contribution of such diffuse interactions to community stability and coexistence (Eppinga et al. 2018). Interestingly, this theory shows how the combined effect of relatively weak interactions can exert strong feedback effects driving community structure (Neutel et al. 2002; Eppinga et al. 2018). However, this upscaling of interactions to the community level comes at the expense of greatly simplifying soil community dynamics. Hence, there is an important complementarity between the two types of approaches. For example, community-level analyses can provide specific hypotheses regarding final community states and possible (restoration) trajectories toward these states. In cases where these predictions fail to accurately describe observed patterns, the plant-soil feedback formalism can be expanded to explicitly describe pathogen and mutualist dynamics as in the current study. Starting with the community-level model does allow for strongly constraining the parameter space to be studied with more detailed models, which is necessary due to the inherent complexity of the latter type of models. We believe that such a combined approach provides a promising way forward to increase our understanding of the ways in which interactions between plants and soil microbes drive community structure.

While plant succession is often thought to result from changes in abiotic resources such as light (Bazzaz 1979), recent work suggests that soil microbes may mediate successional turnover (van der Putten et al. 1993; Kardol et al. 2006; Middleton and Bever 2012; Bauer et al. 2015). Our model indicates that accumulation of either pathogens or mutualists can drive successional turnover in plant species, depending on interspecific differences in plant life-history traits. Accumulation of mutualists can generate positive feedback during succession (Koziol et al. 2018), and we find that this can be a necessary but not sufficient condition for generating successional dynamics. In contrast, pathogens can drive succession only in cases where the late-successional species is the most pathogen resistant. If late-successional species are more vulnerable to pathogens, indirect suppression of pathogens by mycorrhizal fungi (by promoting the growth of more resistant hosts) provides a potential mechanism for succession. These results suggest that future experiments may benefit from dissecting the independent roles of pathogens and mutualists over time, as these microbes may fundamentally change between successional stages.

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