PHILOSOPHICAL TRANSACTIONS B

royalsocietypublishing.org/journal/rstb

Review

Cite this article: Hawkes CV, Bull JJ, Lau JA. 2020 Symbiosis and stress: how plant microbiomes affect host evolution. *Phil. Trans. R. Soc. B* **375**: 20190590.
http://dx.doi.org/10.1098/rstb.2019.0590

Accepted: 2 April 2020

One contribution of 16 to a theme issue 'The role of the microbiome in host evolution'.

Subject Areas:
evolution, ecology

Keywords:
choice, fidelity, horizontal transmission, incidental benefits, infectious model, quantitative genetics

Author for correspondence:
Christine V. Hawkes
e-mail: chawkes@ncsu.edu

Electronic supplementary material is available online at https://doi.org/10.6084/m9.figshare.c.5044128.

THE ROYAL SOCIETY PUBLISHING

Symbiosis and stress: how plant microbiomes affect host evolution

Christine V. Hawkes[1], James J. Bull[2] and Jennifer A. Lau[3]

[1]Department of Plant and Microbial Biology, North Carolina State University, Raleigh, NC 27607, USA
[2]Department of Biological Sciences, University of Idaho, Moscow, ID 83844, USA
[3]Department of Biology, Indiana University, Bloomington, IN 47405, USA

CVH, 0000-0002-1043-9469; JJB, 0000-0002-9798-8275; JAL, 0000-0002-8344-5421

Existing paradigms for plant microevolution rarely acknowledge the potential impacts of diverse microbiomes on evolutionary processes. Many plant-associated microorganisms benefit the host via access to resources, protection from pathogens, or amelioration of abiotic stress. In doing so, they alter the plant's perception of the environment, potentially reducing the strength of selection acting on plant stress tolerance or defence traits or altering the traits that are the target of selection. We posit that the microbiome can affect plant microevolution via (1) manipulation of plant phenotypes in ways that increase plant fitness under stress and (2) direct microbial responses to the environment that benefit the plant. Both mechanisms might favour plant genotypes that attract or stimulate growth of the most responsive microbial populations or communities. We provide support for these scenarios using infectious disease and quantitative genetics models. Finally, we discuss how beneficial plant–microbiome associations can evolve if traditional mechanisms maintaining cooperation in pairwise symbioses, namely partner fidelity, partner choice and fitness alignment, also apply to the interactions between plants and diverse foliar and soil microbiomes. To understand the role of the plant microbiome in host evolution will require a broad ecological understanding of plant–microbe interactions across both space and time.

This article is part of the theme issue 'The role of the microbiome in host evolution'.

1. Introduction

Diverse microorganisms can influence plant responses to environmental stress in an apparently beneficial manner. For example, endophytes from salt- or heat-stressed environments increase plant fitness under stressed conditions [1] and soil microbial communities that evolved under drought increase plant drought tolerance [2].

While these examples suggest that microbiomes can maintain plant fitness under stress, potentially indicating a novel mechanism of plant adaptation to stress, why plants would rely on diverse symbionts remains a significant problem for the evolution of cooperation. Theory developed over the last several decades has identified basic mechanisms for the evolution and maintenance of cooperation. Cooperation is easy to explain when the interacting partners are co-transmitted across generations (known as partner fidelity). It is more problematic when cooperators re-associate horizontally each generation [3] and include multiple species. Yet, such plant–microbiome evolutionary outcomes may have far-reaching ramifications because they point to the possibility of interspecies symbioses, involving many players, arising in many diverse contexts.

Here, we address some aspects of this beneficial-microbiome problem with simple mathematical models. The goal is to understand how a beneficial microorganism or a community of beneficial microorganisms can come to routinely colonize and benefit host fitness responses to environmental stress. Previous work addressed this problem by treating a complex microbiome as a single

unit subject to evolution, with its own properties of variation and inheritance [4]. We instead simplify the process by asking how interactions between the plant host and a single microorganism can evolve to benefit the host, an approach that seems justified from examples in which a single microorganism or a single microbe-provided function has profound benefit to the plant. The models take two forms and address somewhat different questions. Our first set of models is directed at how a beneficial microorganism can evolve an association with the host and be maintained. The microorganism is treated as an infectious agent, and the host can evolve to allow or block the infection. The second modelling effort is directed at the more basic question of why a plant might evolve to rely on a microorganism for its 'adaptation' instead of merely evolving directly in response to the environmental stress.

(a) Biological motivation

In anticipation of the models that follow, we describe the biology that underlies processes by which plant–microbe interactions that improve plant stress responses may arise. Our focus on stress does not preclude the relevance of these arguments for other contexts for beneficial symbioses.

(i) Intrinsically aligned benefits (or by-product cooperation)

Certain properties may make microorganisms more likely to survive and reproduce under stress. In cases where these same microbial traits either directly benefit plants or modify the environment in ways that benefit plants, microbial and plant fitness are aligned, and the microbial benefit to the plant host is a happy accident of stress-induced selection that favours certain microbial traits. One such trait might be biofilm production. Biofilm-producing bacteria perform well under low soil moisture [5], but also could potentially reduce water loss from plant roots [6]. As another example, root fungal endophytes in drought-stressed plants synthesize anti-stress biochemicals, including osmolytes and antioxidants, that reduce plant wilting and enhance growth [7]. While these cases illustrate how microorganisms might promote host plant fitness under stress, no evolutionary change in the host is required (i.e. this mechanism does not meet the strict definition of adaptation), although one might predict that in such cases selection might favour traits that promote interactions with beneficial microbes.

(ii) Coevolution

Cooperative exchanges can also arise in the absence of aligned benefits [8]. One such process starts with the release of a substance by one species (the donor) that coincidentally benefits another species (the recipient). Under appropriate conditions of spatial structure and limited diffusion, the recipient species may then evolve to reciprocate and benefit the donor—provided that the donor responds by releasing an even greater benefit to the recipient. The exchange may then elaborate through an ongoing coevolution of both species. Many classic examples of mutualistic plant–microbe interactions are strict pairwise coevolved interactions between a plant host and a single microbial taxon, often involving the exchange of nutrients or other services (e.g. defence). In these interactions, the evolution of one partner affects selection on the other partner and interactions are often maintained through complex signalling pathways, many of which alter gene expression in one or both partners [9]. Diverse microorganisms also have been shown to manipulate plant gene expression in numerous ways, often via complex signals that stimulate plant biochemical pathways [10]. These microbial effects on plant traits may be the product of selection for plants to eavesdrop on their microbial partners when microorganisms are better predictors of future environmental conditions than other environmental cues [11]. Selection might also act on plant traits that attract microorganisms with large phenotypic effects if the microbial effects are larger than those allowed solely by plant genetic change. In this case, coevolutionary dynamics may result if improved plant stress responses feed back to benefit microorganisms, resulting in selection favouring microorganisms producing stronger signals or greater phenotypic effects on plants.

Both aligned benefits and coevolutionary mechanisms have the potential to change plant phenotypes rapidly and, in some cases, to a greater extent than plant genetics alone. Inoculation with different soil microbial communities altered *Boechera stricta* flowering time by up to approximately 5 days and did so consistently across 48 plant genotypes, despite significant genetic variation for flowering time [12]. Similarly, in three generations of selection, *Brassica rapa* failed to evolve accelerated flowering times in response to drought, but microorganisms from dry environments caused plants to flower approximately 5 days earlier, whereas plastic plant responses to current drought stress only accelerated flowering by approximately 3 days [2]. Although these microbe-induced shifts in flowering time phenotypes might sound small in magnitude, the *B. rapa* population studied is rapid cycling and goes from seed to seed in approximately 40 days. A delay of even a few days can have large consequences for productivity and fitness.

More generally, microbiome effects on plant stress responses and plant adaptation have received increasing attention; however, limited theory has been developed to predict when and how such effects are likely to occur. It is also unclear whether the extensive theory developed for tightly coevolved symbioses applies to plants interacting with diverse, horizontally transmitted microbial communities that occupy roots and leaves. Plant microbiomes have the potential to open up new mechanisms of plant adaptation given the wide array of gene functions available to microorganisms, high functional redundancy in microbial populations that could promote resilience of both the host and microbiome in the face of stress, and the ubiquitous nature of plant–microbe interactions.

Here, we consider how plants can adapt to a novel environment chiefly by symbiont acquisition, rather than by a direct genetic response to the environment. *A priori*, there are two general processes by which beneficial plant–microbe interactions may become prevalent: infectious and genetic. Both may be involved in any system, but we model them separately.

2. Infectious models for the spread of beneficial microorganisms

We use an epidemiological or 'infectious' model to investigate how and when an infectious process—a symbiont colonizing a host —will lead to a population-wide association between a beneficial microorganism and its host. This modelling has three stages. The first stage merely assumes that an infectious microorganism exists, and the model describes how extensively it may spread in a population of sensitive plants—a process that is purely demographic without

evolution. This process works similarly for a beneficial symbiont or for a (moderately) harmful one, leading to an abundance of symbiont-associated plants. The second stage considers that only some plants are susceptible to infection by the microorganism and addresses the evolution of plant susceptibility; this stage has different consequences for a beneficial versus a harmful microbe. Stage three considers the possibility that some microbial variants are not beneficial and evaluates which strains evolve, and why.

(a) Model 1: an infectious symbiont altering the fitness of its host

An infectious process, with the symbiont invading the host population, can be illustrated with a differential equation model similar to those used in epidemiology [13] and phage growth [14]. Our assumptions are

(1) Uninfected (naive) plants lack symbionts. Uninfected plants are lost through death and through colonization by symbionts, which converts them into infected plants. Births occur at a fixed rate and are always of naive plants.
(2) If acquired by the plant, symbionts affect plant death rate. A beneficial symbiont lowers the death rate below that of uninfected plants; a harmful one increases it. We are interested in how the dynamics differ when the symbiont is beneficial versus harmful. Biologically, beneficial symbionts might pre-exist in stressful environments when the traits allowing the symbionts to persist under those stressors also benefit associated host plants (e.g. biofilms)—a case of intrinsically aligned benefits.
(3) Infected plants come into existence only through symbiont colonization (infection) of naive plants; no plants are born infected. Infected plants are lost only through death—there is no recovery back to an uninfected state.
(4) Free-living symbionts are born from infected plants. They are lost through infection of naive plants and through death.

These processes are represented by the following set of equations (2.1). For tractability, we assume that the infection process follows 'mass action,' reasonable for developing intuition; spatially structured spread would be more biologically accurate but not nearly so tractable for intuition, and the general features of evolution and dynamics are not likely to be qualitatively misled by mass action. The notation is given in table 1; all variables are functions of time and non-negative; a superior dot ($\dot{}$) indicates a derivative with respect to time.

$$\left.\begin{aligned} \dot{P}_u &= b_p - \beta S P_u - d_u P_u \\ \dot{P}_i &= \beta S P_u - d_i P_i \\ \text{and} \quad \dot{S} &= b_s P_i - \beta S P_u - d_s S. \end{aligned}\right\} \tag{2.1}$$

The equilibrium for this system (with all variables and parameters positive) is

$$\left.\begin{aligned} \hat{S} &= \frac{\beta b_p b_s - \beta b_p d_i - d_i d_u d_s}{\beta d_i d_s} \\ \hat{P}_u &= \frac{d_i d_s}{\beta(b_s - d_i)} \\ \text{and} \quad \hat{P}_i &= \frac{\beta b_p b_s - \beta b_p d_i - d_i d_u d_s}{\beta d_i (b_s - d_i)}, \end{aligned}\right\} \tag{2.2}$$

Table 1. Model variables and parameters for equations (2.1)–(2.4).

notation	description
variables	
P_u	density of plants lacking symbionts
P_i	density of plants infected with symbionts
S	density of free-living symbionts
P_r	density of plants resistant to symbiont infection
parameters[a]	
b_p	birth rate of plants (born uninfected)
b_s	birth rate of symbionts per infected plant
β	infection rate parameter
d_u	death rate of uninfected plants
d_i	death rate of symbiont-infected plants
d_s	death rate of free symbiont
d_r	death rate of symbiont-resistant plants

[a]Parameter values are fixed throughout a run. Values used in numerical trials are given in the figure legends.

the ^ indicating a steady-state value. This equilibrium exists and is biologically meaningful for reasonable parameter values (e.g. birth rates substantially greater than death rates). Of particular interest is the expected ratio of infected to naive plants,

$$\frac{\hat{P}_i}{\hat{P}_u} = \frac{\beta b_p (b_s - d_i) - d_u d_i d_s}{d_i^2 d_s}, \tag{2.3}$$

which will be positive and quite large, with a sufficient magnitude of β and $b_s > d_i$.

In trials (figure 1), the symbiont quickly infects virtually the entire plant population, regardless of whether the symbiont is beneficial or harmful; uninfected plants persist only as newborns. This behaviour is straightforward and highly intuitive, although noteworthy in that it contrasts with some infectious disease models where uninfected hosts often remain a large fraction of all hosts [13]. Here and in standard disease models, persistence of high levels of uninfected hosts depends on recovery (absent here) and the effect of symbiont/parasite on host death rate, with a greater host benefit leading to a higher ratio of symbiont-infected to uninfected hosts (equation (2.3) and figure 1). Thus, although it seems that a beneficial symbiont might experience much more favourable dynamics than a (moderately) harmful one, a harmful one can also sweep the population, provided host survival is not profoundly compromised.

(b) Model 2: adding plant evolution to the infectious model

The epidemic model shows that a susceptible plant population can quickly be overwhelmed by an infectious symbiont, whether the symbiont is good or bad for the plant. All plants may not be susceptible initially, however. How might a symbiont-sensitive plant evolve if most of the plant population is resistant to colonization by the symbiont? A simple addition to model (1) reveals one possible evolutionary process: competition between symbiont-sensitive and symbiont-resistant

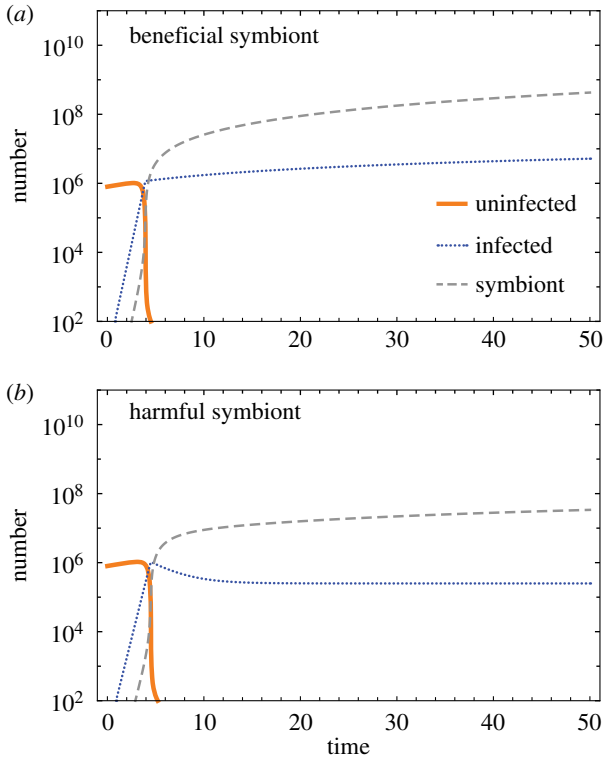

Figure 1. Two numerical trials of equation (2.1). The dynamics quickly have symbiont-infected plants displace nearly all uninfected plants, regardless of whether the symbiont is beneficial (*a*) or is harmful to the plant (*b*). The process is fundamentally the same as infectious disease dynamics (for the case of no recovery from infection). Uninfected plants are maintained because all newborn plants are uninfected, but the levels at which uninfected plants are maintained is low and not evident on the scale shown. Symbiont density increases from the output of infected plants, which in turn boosts infected plant density. Time could be treated as weeks, but parameter values were chosen for illustration, not to fit any system; dynamics out to time 5000 merely show continued approach to equilibrium. (*a*) The symbiont is beneficial to the plant: death rate is lower (half that) for infected than for uninfected plants. The equilibrium ratio of symbiont-infected to uninfected plants is nearly 5×10^9 (from equation (2.3)). Parameter values: $b_p = 10^5$, $b_s = 3$, $\beta = 0.0005$, $d_u = 0.008$, $d_i = 0.004$, $d_s = 0.002$. Initial densities: $P_u = 800\,000$, $P_i = 0$, $S = 10$. (*b*) The same parameter values as in (*a*), except the symbiont is now harmful: the death rate of infected plants (d_i) is increased 100-fold to 0.4. Densities of free symbionts and symbiont-infected plants are lower, and the equilibrium level of symbiont-infected to uninfected plants is approximately 4×10^5, much lower than in (*a*), but still high. Thus, a harmful symbiont can invade with a qualitatively similar dynamical effect to a beneficial symbiont. (Online version in colour.)

plants, affected only by the impact of symbiont acquisition. The revised model in equations (2.4) merely adds one new equation for the symbiont-resistant plant (P_r) (table 1) and now partitions plant births between sensitive and resistant plants in proportion to the current abundance of each type

$$\left.\begin{aligned} \dot{P}_u &= b_p\left(\frac{P_i + P_u}{P_i + P_r + P_u}\right) - \beta S P_u - d_u P_u, \\ \dot{P}_i &= \beta S P_u - d_i P_i, \\ \dot{P}_r &= b_p\left(\frac{P_r}{P_i + P_r + P_u}\right) - d_r P_r \\ \text{and} \quad \dot{S} &= b_s P_i - \beta S P_u - d_s S. \end{aligned}\right\} \tag{2.4}$$

Equilibria are no longer straightforward, but some special cases are easily analysed. If the population of hosts is stable ($N = P_u + P_i + P_r$), with $X = P_u + P_i$, equation (2.4) yields

$$\left.\begin{aligned} \dot{X} &= b_p\frac{X}{N} - d_r X + \Delta(P_r, P_i) = X\left(\frac{b_p}{N} - d_r\right) + \Delta(P_r, P_i) \\ \dot{P}_r &= b_p\frac{P_r}{N} - d_r P_r = P_r\left(\frac{b_p}{N} - d_r\right), \end{aligned}\right\} \tag{2.5}$$

where the function $\delta(P_r, P_i) = P_u(d_r - d_u) + P_i(d_r - d_i)$. If $\delta(P_r, P_i) = 0$, it is easily seen that resistant and sensitive plant types grow at the same rate and thus are equally fit. For example, $\delta(P_r, P_i) = 0$ if $d_r = d_u = d_i$. If the only death rate that differs is d_i, then resistant plants will evolve if the symbiont is beneficial ($d_i < d_r, d_u$) and not if it is harmful ($d_i > d_r, d_u$). Thus, as we might expect for simple cases, plants are selected to accept beneficial symbionts and resist harmful ones. Although a harmful symbiont can sweep the population, the plant is selected to block it. Limited simulations support these outcomes.

(c) Model 3: adding symbiont evolution to the infectious model

The crux to many problems in the evolution of cooperation is the maintenance of cooperation against invasion by 'cheaters'—individuals that usurp benefits from the partner but provide nothing in return. In the context of our biological problem, this would be evolution of a microorganism that offers no benefit while taking resources from the plant. This question is one of the evolution of parasite virulence, for which there is a moderately expansive theoretical literature [15].

Expanding our model to this process requires five equations: one for a beneficial microorganism, one for a non-beneficial microorganism, one for uninfected plants, and one each for plants infected by each microorganism (electronic supplementary material, appendix S1, disallowing co-infection of plants by both microorganisms). Again, all fitness effects of the microorganism on the plant are through survival (the model could be reformulated to have the symbiont affect fecundity). Numerical trials suggest that, if the symbionts differ only in their effect on plant survival, the more beneficial microorganism evolves, thus maintaining the mutual benefit. Combining these results with those of the previous models, the evolutionary interests of plants and microbes are aligned: plants are selected to accept only beneficial symbionts, and symbionts are selected to benefit the plant (but see below).

(d) Biological appraisal of the infectious models

In sequential fashion, the models show that it is easy for a horizontally transmitted symbiont to invade and spread throughout a sensitive host population. Furthermore, acceptance of the symbiont is selected when the microorganism is beneficial to the plant, not when it is harmful. The challenge is the usual one in the maintenance of cooperation in horizontally transmitted associations: suppression of cheaters. Our third model shows that beneficial symbionts displace those that harm the host—under some conditions, at least when there are no pleiotropic effects of differences in symbiont benefit.

At face value, this last result contradicts the consensus in the evolution of virulence literature, which shows that

virulent infections are often favoured—the symbiont/parasite evolves to harm the host [15]. A key difference between our model and evolution of virulence models is that we allowed the symbiont to vary in only one trait, its effect on host survival. Evolution of virulence models specify covariation in two traits, transmission and effect on the host, with a trade-off in which higher transmission causes increased harm. Virulence to the host is always bad for the parasite, but it can be offset by gains in parasite transmission. Allowing our symbiont to increase transmission while reducing its benefit could also lead to evolution of harm, depending on parameter values. Thus our model merely applies to a different kind of symbiont from those in virulence models, one that can benefit the host at the same time it transmits; those types of symbionts appear to exist. Indeed, the intrinsically aligned benefits model satisfies this property.

Of course, in the myriad of microbes that a plant encounters as potential symbionts, most will not be so benevolent: many microbes will be outright harmful, and others that could be or are initially beneficial will be selected towards harm once they develop an association with the plant. The plant faces a special challenge in maintaining the benefit from this latter category of symbiont, because it may be difficult for the plant to interact with the beneficial mutants while avoiding harmful mutants of the same species. As noted above, our model does not address this category of symbiont, and it may be an important one.

If harmful symbionts can indeed evolve, what factors could promote the evolutionary maintenance of beneficial microbiomes against harmful ones? We suggest two possibilities to this challenge, both standard ones. One solution is 'partner choice'—the plant discriminates against less beneficial or detrimental symbionts [3]. It is not clear how feasible such discrimination is, and it would certainly involve an arms race between the plant and symbiont as cheaters acquire signals characterizing high-quality symbionts, reducing a plant's ability to differentiate between them. However, sanctioning or reducing resources allocated to low-quality symbionts after infection may be more likely (sometimes considered a negative form of partner choice). A second possibility is that, despite the horizontal acquisition of microbiomes, partner fidelity operates on a long enough time scale (the plant's lifetime) to maintain the mutual benefit. This model is especially plausible when the association enables the plant to survive in an otherwise lethal environment. It may also be relevant that the symbiont may colonize the host soon after seed germination, potentially facilitating a many-fold increase in host survival, size and reproduction, also promoting massive increases in symbiont fecundity. The model does not explain why harmful endophytes do not invade the mutualism once established; likely other processes are at work, perhaps microbe–microbe interactions.

3. Forces promoting microbiome-mediated adaptation: partner fidelity, partner choice and intrinsically shared benefits

We alluded to three potential mechanisms that can limit cheaters and result in microbiome-mediated host adaptation: partner fidelity, partner choice and intrinsically aligned benefits. Here, we discuss evidence for and against these mechanisms in diverse plant microbiomes in contrast to what is known from coevolved mutualisms.

(a) Partner fidelity

We have little understanding of the degree of repeated interactions in diverse microbiomes. Nevertheless, when repeated interactions do occur, plants engaged in interactions with particularly beneficial microbial communities should experience increased growth, resulting in more resources available to those microbial communities. Over time, such repeated interactions can create a mutually beneficial cycle through partner-fidelity fitness feedbacks that serve to increase the frequency of beneficial microbial communities and the plants best able to interact with them [16].

Vertical transmission is considered the primary mechanism supporting partner fidelity. While the majority of the plant microbiome is horizontally transmitted, a small number of endophytes are inherited through plant seed or via a mixture of the two transmission pathways [17]. Yet vertical transmission is often imperfect even in classic cases such as *Epichloë* fungi in cool-season grasses, with the rate of transmission to progeny controlled by host genotype and environment [18]. New evidence that some common soil microbial taxa also occupy seeds suggests the potential for broader vertical transmission [19]; however, their transmission via seed appears to be low [20]. Thus, partner fidelity in the form of vertical transmission is unlikely to broadly support host adaptation via its microbiome.

Nevertheless, partner fidelity can occur through other mechanisms that mimic vertical transmission by resulting in repeated interactions across generations, including forces that promote spatial structure in both plant and microbial populations (e.g. limited dispersal and strong location-specific effects on microbial communities or plant genotypes), and host genetic control. In cases where the spatial scales of both plant and microbial dispersal are small, offspring plants may be more likely to form associations with the same microbial communities as their parental genotypes. Although microbial dispersal and colonization dynamics remain poorly understood, there is a growing body of evidence suggesting that microbial dispersal can be geographically limited [21]. For example, ectomycorrhizal fungi form spatially structured hyphal networks in soil that can persist beyond the life of individual trees, thus creating the potential for high heritability provided that plant dispersal is also limited [22]. These networks often consist of a limited number of fungal species and genotypes [23] that benefit survival, growth, and stress tolerance of new seedlings [24]. In *Pinus pisaster*, location affected root ectomycorrhizal community composition more so than host genotype, suggesting that the fungal networks in different environments are an important controller of associations in each host generation [25]. However, in some cases, the ectomycorrhizal networks vary spatially through time [26], which points to a combination of fidelity and choice mechanisms (see §3b).

Strong host genetic control of the microbiome may also act as a form of partner fidelity. For example, *Pinus edulis* genotypes that differ in drought tolerance maintain different root ectomycorrhizal communities even when reciprocally inoculated [27]. Genotype–microbiome associations were also found in *Arabidopis thaliana* and associated with plant genes related to cell wall integrity [28,29]. However, it is not clear that host genotype

is an effective means of maintaining fidelity in diverse microbiomes. When multiple locations and years are considered, fewer than approximately 12% of microbial taxa were genotype-associated in *Arabidopsis* [30]. Similarly, in maize, only a small portion of the leaf and root microbiomes appear to be heritable, with large genotype by environment interactions [31,32]. These genotype by environment interactions may indicate fidelity of only a small number of taxa, with many transient or opportunistic colonizers. However, they also suggest that plants could potentially associate preferentially with microorganisms that are particularly advantageous in one environment or another, likely via partner choice.

(b) Partner choice

In a diverse microbiome community, rewards and sanctioning can occur if plants respond to spatial variation in microbial community benefit with increased resource allocation, as observed for rhizobia nodules with different levels of nitrogen fixation [33] and for arbuscular mycorrhizal fungi that provide different amounts of phosphorus [34]. The mechanisms of how plants detect and preferentially allocate resources to beneficial symbionts in a diverse, mixed community is less clear than for specific taxa with coevolved signalling mechanisms, particularly when microbial taxa with different benefits coexist in close proximity. However, partner choice could occur at two levels: (1) preferential allocation to plant organs associated with more cooperative microbial communities compared with other plant organs associated with less cooperative microbial communities, or (2) preferential allocation to more cooperative taxa within the microbial community even within the same plant organ. Models of host discrimination have identified conditions under which coexisting mixtures of mutualists and non-mutualists can be maintained, such as when host preferential allocation to plant organs is a saturating function of mutualist density [35]. Empirical tests are still needed to document plant preferential allocation or sanctioning of diverse microbial communities both at the level of the whole community and at that of specific microbial taxa within the community.

Some host plants can select microbial partners via active recruitment from available species pools. Most known active recruitment of microorganisms by plants is via chemical attractants or inhibitors. Differential chemotaxis by rhizobial strains to bulk root exudates of different plants has been recognized for some time [36]. More recently, specific rhizosphere metabolites have been identified as drivers of root colonization. In *Arabidopsis*, for example, microbial community assembly is dependent on salicylic acid [37]. Similarly, salicylic and other aromatic organic acids appear to be preferred by rhizobacteria associated with the annual grass *Avena barbata*, and their exudation patterns drive microbial succession in the rhizosphere [38]. The ability to chemically attract beneficial taxa or repel non-beneficial taxa should support preferential, beneficial associations via specific functions that may be independent of microbial identity *per se*. In maize leaves, for example, only five bacterial clades but 200 metabolic functions were heritable [32]. Given that different host exudates stimulate the growth of different microbial taxa, shifts in exudate profiles may be one potential mechanism for partner choice [38], although such chemical signals also may be subject to cheating if non-beneficial or even antagonistic symbionts cue in on these same signals or prefer the same substrates.

(c) Intrinsically aligned benefits

Plant and microbiome fitness also might be expected to become aligned when microbial traits promote both host and microorganism fitness. Cases where host and microbial fitness are not aligned, as for microbial taxa where the host is only one of many habitats, may disrupt adaptive trajectories. However, we argue that the high functional redundancy in diverse microbial communities leads to decoupling of taxonomy from function [39] and may, therefore, mitigate these constraints. Functional redundancy is well known in microbial communities such as soils, where high spatial heterogeneity in the habitat combined with phylogenetic niche conservatism are likely to support functional overlap among microbial lineages [40]. High levels of functional redundancy were also found in tropical tree phyllospheres, with approximately 50% of microbial protein functional domains shared across four different host species despite high taxonomic divergence [41]. We are only beginning to understand function in host-associated microbiomes with horizontally transmitted taxa [42], but we expect functional redundancy will play an important role. In particular, functional redundancy in the microbial community will allow host selection on microbiome function rather than taxa *per se*. Redundancy may, therefore, rescue microbiome effects on host adaptation that might not occur if relying on taxon-specific interactions.

4. Context-dependence and host adaptation via microbiomes

Both partner choice and partner fidelity are well-studied mechanisms explaining the maintenance of coevolved pairwise mutualisms that can also apply to diverse microbiomes. Nevertheless, the efficacy of both choice and fidelity is likely weakened when microbiome effects depend on environmental conditions and when microbiome composition is spatially and temporally variable. Similarly, while intrinsically aligned benefits could also explain how microbiomes can be mutualistic under stress, fitness alignment is likely susceptible to this same variation in microbial community function and composition.

(a) Plasticity and environmental context

One of the biggest challenges in diverse plant microbiomes is that broad functional plasticity in microorganisms results in the outcome of particular plant–microbiome associations depending on environmental conditions. Many plant–microbe interactions are context-dependent and range from mutualistic to pathogenic, such that the same microbiomes are not consistently beneficial. For example, specific resource mutualists like rhizobia or mycorrhizal fungi can shift towards parasitism when resource availability increases [43], and leaf endophytes that are beneficial under drought can be parasitic under well-watered conditions [44]. Context-dependence, combined with temporal variation in abiotic environmental conditions, may reduce the effectiveness of partner choice through signalling with specific taxa (unless choice itself is context-dependent). Instead, other mechanisms that effectively alter the intensity or likelihood of host–symbiont interaction, like sanctioning and reducing allocation to symbionts when they are less beneficial, may be necessary to stabilize the interaction.

(b) Local microbial heterogeneity

Variation in plant–microbiome composition across sites and time [45] may be a double-edged sword for plant hosts. On

the one hand, temporal and spatial variation in microbial communities poses challenges because it likely limits associations that can promote partner fidelity. On the other hand, site- and environment-specific microbiome assembly may be advantageous, allowing plants the flexibility to associate with the most advantageous microbial community in a particular set of conditions. This flexibility may promote plant–microorganism fitness alignments. If plants can choose the most beneficial microbiomes in a particular environment, then the ability to adjust associations to maximize beneficial microbiomes in space and time may parallel plant adaptive plasticity (i.e. genotype by environment interactions in which some plant genotypes effectively shift their phenotype in a manner that increases fitness) [4]. Flexibility in partner choice may allow finer control than traditional genetic mechanisms of adaptation, if plants can increase fitness across a wide range of environmental conditions and spatial scales. Limited evidence, however, suggests that partner choice varies across environmental conditions in even tightly coevolved mutualisms [46], although few conditions have been tested. Alternatively, fitness alignments may be coincidental, with spatial and temporal variation in microbiome availability proportional to the probability of colonization.

5. When are plants likely to capitalize on microorganisms for local adaptation?

Our initial infectious model shows that a beneficial microbial symbiont can rapidly infect a plant population, that plants that associate with the symbiont are favoured by selection, and that the beneficial symbiont can displace a non-beneficial symbiont. The infectious model essentially shows that plants can capitalize on a symbiont to maintain and increase fitness; however, it does not identify when this microbe-mediated mechanism of adaptation is likely to dominate over non-symbiont evolution of plant stress tolerance traits. Essentially, the infectious model treats plant genotype as a simple trait: plants are either able or unable to form associations with the symbiont, and the symbiont provides the benefit. The only plant evolutionary response allowed is acquisition of the symbiont. A more realistic evolutionary process would include two plant responses: acquisition of a beneficial symbiont and a direct plant response to the environmental selection—the standard quantitative genetic response. What happens when both evolutionary processes are possible—which one will dominate? This question is somewhat more fundamental than those addressed above in that, here, the question is whether to expect the plant to evolve an association with the symbiont at all or just evolve the response on its own. Of course, should the plant be unable to respond to the selection, then a symbiont is the only possible solution. But there may be many cases when both responses are possible.

Initial insight to alternative pathways of evolution may be sought by considering the trivial model of two bi-allelic loci, *A*/*a* and *B*/*b*: the *A* allele directly increases the plant phenotype in a beneficial way, the *B* allele does so through acquisition of a symbiont. It is straightforward that whichever allele has the larger fitness effect will increase more rapidly at the outset (before the two alleles start interfering with each other). Even if the symbiont is capable of a large beneficial response, however, the average effect of *B* may be reduced either if the symbiont is not everywhere available or if the symbiont invades the plant somatic tissues to only a limited degree; the magnitude of effect is what matters.

A quantitative genetics framework provides additional insight. We borrow and slightly modify the quantitative genetics framework of Gomulkiewicz *et al*. [47], which assumes two traits evolving toward a common fitness optimum with a quadratic fitness function. For our purposes, we let z_1 be a trait that directly affects the plant phenotype and z_2 be a trait that indirectly affects the plant phenotype through acquisition of the symbiont. Each trait has its own heritability and variance (h_i^2,σ_i^2). The symbiont is everywhere equally abundant; its dynamics and evolution are omitted.

It is not immediately obvious how to represent or interpret the symbiont-based phenotype as a quantitative trait affecting fitness. Larger values of z_2 need to be associated with a larger effect or presence of the symbiont, but we need a biological translation by which z_2 allows the effect of the symbiont to be manifest. One option is to imagine that increasing values of z_2 have a higher probability of the plant acquiring the symbiont, and that plants with the symbiont have the full effect. The fitness effect of a specific value of z_2 would be the population average of all plants at that value of z_2 (only some of which have the symbiont). Alternatively, z_2 might influence how extensively the symbiont is able to invade the plant's somatic tissues; a low z_2 would mean the symbiont cells remain a tiny portion of the total plant biomass, thereby limiting the benefit.

The issue is then how to compare the relative fitness gains by increasing z_1 versus z_2. We write αz_2 as the plant phenotype associated with trait value z_2. When $\alpha > 1$, increasing z_2 by an amount μ causes a greater phenotype (and fitness) increase than is achieved when increasing z_1 by the same amount μ. This corresponds to allele *B* having a larger effect than allele *A* in the two-locus model.

With this, and from Gomulkiewicz *et al*. [47], we have

$$\Delta\bar{z}_1 = h_1^2\sigma_1^2\frac{\partial \ln \bar{W}}{\partial \bar{z}_1},$$

$$\Delta\bar{z}_2 = h_2^2\sigma_2^2\frac{\partial \ln \bar{W}}{\partial \bar{z}_2}$$

$$\text{and} \quad \bar{W} = W_{\max}\{1 - \gamma[(\bar{z}_1 + \alpha\bar{z}_2 - \theta)^2 + \sigma_1^2 + \alpha^2\sigma_2^2]\}. \tag{5.1}$$

For equal heritabilities and variances of Z_1 and Z_2, evolution of Z_2 proceeds at a rate α times that of Z_1. The quantitative genetics model thus shows that a symbiont with a large beneficial effect on plant phenotype can allow the plant to evolve faster than it evolves via a typical, direct genetic response to the selection. Perhaps surprisingly, the rate of evolution does not depend on the mere fact that the symbiont can ultimately provide a (much) higher final fitness than genetics alone. Instead, the relative rate of evolution depends on how the genetic variation in symbiont 'acceptance' by the plant translates into variation in plant fitness; if increases in z_2 lead to smaller improvements in phenotype/fitness than do corresponding increases in z_1, then the intrinsic genetic response will evolve faster—up to the point that genetic variation is exhausted. For symbiont-based adaptation to dominate, the plant must translate symbiont acquisition into a larger genetic effect than operates without the symbiont.

The two types of quantitative variation are in principle measurable. z_1 would be standard for an environmentally influenced fitness trait (e.g. variation in plant heat tolerance, salt tolerance, etc.). z_2 would be measured in the same way, except in the presence of the symbiont. Complications in measuring z_2 would include (i) controlling for covariation in z_1 and (ii) ensuring that the plant response was due specifically to the symbiont. The most direct approach might be to analyse variation in z_2 as just variation in the plant's acquisition of a known beneficial symbiont, then address the phenotypic effect on fitness separately.

Although empirical results to support these predictions are limited, some evidence points to strong microbial effects on particular plant traits in stressful environments. For example, as discussed earlier, microbial communities that evolved under drought stress also caused plants to flower significantly earlier, a common drought avoidance trait. Notably, these microbe-mediated effects on plant phenotypes were substantially stronger than the effects of plant evolution (three generations of selection) on plant flowering time [2]. Similarly, microbial endophytes can mediate plant drought physiology, growth and survival [44,48] or protect plants from pathogens [49], suggesting microbiome fitness effects that could promote host adaptation to these stressors may be widespread.

6. Future directions

Mechanisms maintaining tightly coevolved mutualisms are well understood, but their application to diverse plant microbiomes including a range of beneficial and non-beneficial taxa is not straightforward. Studies of repeated interactions and host-association mechanisms are much needed, as well as a focus on the relationship between host fitness and microbial function rather than taxa *per se*. For example, many researchers have sought to define the 'core' microbiome associated with host plant species based on a variety of metrics to indicate microbial taxa found in a majority of replicate plants [50]. However, to address the potential for microbiome effects on host adaptation, it is worth considering studies of core microbiomes that prioritize functional characterizations and repeated interactions of host plants with specific functional groups across generations and under different environmental conditions.

When considering microbiome effects on host plant ecology and evolution, microbial interactions among themselves must also be considered. In the process of microbiome assembly on the host, colonizing microorganisms will potentially compete with or facilitate each other and any resident taxa. These microbial interactions can alter the resulting microbiome and host benefits [51] even if individual taxa were actively recruited or rewarded. Colonization sequence, for instance, can have a large impact on the microbiome by creating priority effects: the first colonists to arrive can gain a numerical advantage, allowing plant microsites to be occupied by competitively inferior taxa [52]. Because priority effects alter microbial interactions, they can also shift microbiome function; for example, arrival order can determine whether endophyte–pathogen interactions result in disease for the host plant [53]. Thus, future studies of how plant microbiomes affect host adaptation should consider not just pairwise interactions, but multi-species interactions occurring simultaneously.

The effects of temporal and spatial heterogeneity on microbiome composition and function can also shed light on mechanisms supporting cooperation. For example, studies are needed in which microbiomes are experimentally transmitted from plant generation to generation in the context of realistic microbial and environmental variation. Most microbial communities do not move directly between the same hosts over time, and some may inhabit soils or water in between time spent in plant hosts. A simple example comes from agriculture, where the majority of crops are planted in rotation systems throughout the year, such as corn–soy–wheat. One benefit of agricultural rotations is a reduction in the incidence of negative components of the microbiome [54], supporting the need to track microbiome occupancy through different habitats over time. Passaging directly between the same host plants [16] therefore does not necessarily shed light on mechanisms occurring in the field, although such studies may provide insight into the potential for engineering the microbiome.

From a modelling perspective, the models here either explicitly or implicitly assume interactions between a plant and a single microbial species. This level of simplicity is justified for some observed systems, especially those in which a single component of the microbiome has major effects on plant survival. The single-symbiont assumption stands apart from modelling efforts that treat a complex microbiome as a unit [4]. An obvious next step is to formally model dynamics of a multi-species microbiome interacting and evolving with a host. Such an approach would simultaneously reveal whether results from a single-symbiont model can be generalized and also reveal whether a multi-species microbiome can indeed be treated as a unit of selection. Models of diffuse evolution from plant–herbivore communities that account for multi-species interactions may provide some guidance on diverse microbiomes [55].

7. Conclusion

There is increasing interest, from both applied and basic researchers, in how the microbiome can facilitate host adaptation to stress. However, while a long and extensive history of both ecological and evolutionary theories developed for tightly coevolved mutualisms has led to key predictions for the maintenance and evolution of bipartite cooperation, limited theory exists for the diverse microbiomes that dominate nature. The explicitly quantitative and heuristic models that have emerged suggest potential effects of microbial cues, repeated interactions and partner fidelity, and the magnitude of microbial versus host-genetic effects on plant phenotypes. Empirical work has advanced at a more rapid pace, but is still limited to simple systems (e.g. single plant species mesocosms) and few host traits. Future theoretical and empirical work will need to fully consider the diversity of the microbiome (a both a functional and a taxonomic perspective), how classic mechanisms maintaining mutualism apply to microbiomes, and the context-dependence of host–microbiome interactions. In addition, it remains to be seen whether strong genotype by environment associations of plants and microbiomes are an evolutionary challenge or an evolutionary advantage and a novel arsenal for plants to adapt to temporally and spatially varying stressors.

Ethics. This work did not involve human or animal subjects.

Data accessibility. Model equations supporting this article are provided in the manuscript and in electronic supplementary material, appendix S1.

Authors' contributions. C.V.H., J.J.B. and J.A.L. conceived of the ideas and wrote the paper. J.J.B. carried out the modelling.

Competing interests. We declare we have no competing interests.

Funding. We received no funding for this study.

Acknowledgements. C.V.H. was supported by USDA NIFA Physiology of Agricultural Plants (grant no. 2017-67013-29207), USDA Hatch (accession no. 1018688) and a DOE Genomic Sciences Program SFA (SCW1039) subaward from Lawrence Livermore National Lab. J.A.L. was supported by the Environmental Resilience Institute, funded by Indiana University's Prepared for Environmental Change Grand Challenge Initiative. J.J.B. received support from NIH R01GM122079.

References

1. Rodriguez RJ, Henson J, Van Volkenburgh E, Hoy M, Wright L, Beckwith F, Kim Y-O, Redman RS. 2008 Stress tolerance in plants via habitat-adapted symbiosis. *ISME J.* **2**, 404–416. (doi:10.1038/ismej.2007.106)
2. Lau JA, Lennon JT. 2012 Rapid responses of soil microorganisms improve plant fitness in novel environments. *Proc. Natl Acad. Sci. USA* **109**, 14 058–14 062. (doi:10.1073/pnas.1202319109)
3. Sachs JL, Mueller UG, Wilcox TP, Bull JJ. 2004 The evolution of cooperation. *Q. Rev. Biol.* **79**, 135–160. (doi:10.1086/383541)
4. Henry LP, Bruijning M, Forsberg SKG, Ayroles JF. 2019 Can the microbiome influence host evolutionary trajectories? *bioRxiv*, 700237. (doi:10.1101/700237)
5. Lennon JT, Aanderud ZT, Lehmkuhl BK, Schoolmaster Jr DR. 2012 Mapping the niche space of soil microorganisms using taxonomy and traits. *Ecology* **93**, 1867–1879. (doi:10.1890/11-1745.1)
6. Naseem H, Ahsan M, Shahid MA, Khan N. 2018 Exopolysaccharides producing rhizobacteria and their role in plant growth and drought tolerance. *J. Basic Microbiol.* **58**, 1009–1022. (doi:10.1002/jobm.201800309)
7. Dastogeer KMG, Li H, Sivasithamparam K, Jones MGK, Wylie SJ. 2018 Fungal endophytes and a virus confer drought tolerance to *Nicotiana benthamiana* plants through modulating osmolytes, antioxidant enzymes and expression of host drought responsive genes. *Environ. Exp. Bot.* **149**, 95–108. (doi:10.1016/j.envexpbot.2018.02.009)
8. Bull JJ, Harcombe WR. 2009 Population dynamics constrain the cooperative evolution of cross-feeding. *PLoS ONE* **4**, e4115. (doi:10.1371/journal.pone.0004115)
9. Heath KD, Burke PV, Stinchcombe JR. 2012 Coevolutionary genetic variation in the legume-rhizobium transcriptome. *Mol. Ecol.* **21**, 4735–4747. (doi:10.1111/j.1365-294X.2012.05629.x)
10. Khare E, Mishra J, Arora NK. 2018 Multifaceted interactions between endophytes and plant: developments and prospects. *Front. Microbiol.* **9**, 2732. (doi:10.3389/fmicb.2018.02732)
11. Metcalf CJE, Henry LP, Rebolleda-Gómez M, Koskella B. 2019 Why evolve reliance on the microbiome for timing of ontogeny? *mBio* **10**, e01496-19. (doi:10.1128/mBio.01496-19)
12. Wagner MR, Lundberg DS, Coleman-Derr D, Tringe SG, Dangl JL, Mitchell-Olds T. 2014 Natural soil microbes alter flowering phenology and the intensity of selection on flowering time in a wild *Arabidopsis* relative. *Ecol. Lett.* **17**, 717–726. (doi:10.1111/ele.12276)
13. Keeling M, Rohani P. 2008 *Modeling infectious diseases in humans and animals*. Princeton, NJ: Princeton University Press.
14. Levin BR, Stewart FM, Chao L. 1977 Resource-limited growth, competition, and predation: a model and experimental studies with bacteria and bacteriophage. *Am. Nat.* **111**, 3–24. (doi:10.1086/283134)
15. Cressler CE, McLeod DV, Rozins C, Van Den Hoogen J, Day T. 2016 The adaptive evolution of virulence: a review of theoretical predictions and empirical tests. *Parasitology* **143**, 915–930. (doi:10.1017/S003118201500092X)
16. Morella NM, Weng FC-H, Joubert PM, Metcalf CJE, Lindow S, Koskella B. 2019 Successive passaging of a plant-associated microbiome reveals robust habitat and host genotype-dependent selection. *Proc. Natl Acad. Sci. USA* **117**, 1148–1159. (doi:10.1073/pnas.1908600116)
17. Clay K, Schardl C. 2002 Evolutionary origins and ecological consequences of endophyte symbiosis with grasses. *Am. Nat.* **160**, S99–S127. (doi:10.1086/342161)
18. Gagic M *et al.* 2018 Seed transmission of *Epichloë* endophytes in *Lolium perenne* is heavily influenced by host genetics. *Front. Plant Sci.* **9**, 1580. (doi:10.3389/fpls.2018.01580)
19. Shade A, Jacques M-A, Barret M. 2017 Ecological patterns of seed microbiome diversity, transmission, and assembly. *Curr. Opin. Microbiol.* **37**, 15–22. (doi:10.1016/j.mib.2017.03.010)
20. Rezki S, Campion C, Simoneau P, Jacques M-A, Shade A, Barret M. 2018 Assembly of seed-associated microbial communities within and across successive plant generations. *Plant Soil* **422**, 67–79. (doi:10.1007/s11104-017-3451-2)
21. Peay KG, Schubert MG, Nguyen NH, Bruns TD. 2012 Measuring ectomycorrhizal fungal dispersal: macroecological patterns driven by microscopic propagules. *Mol. Ecol.* **21**, 4122–4136. (doi:10.1111/j.1365-294X.2012.05666.x)
22. Mueller RC, Scudder CM, Whitham TG, Gehring CA. 2019 Legacy effects of tree mortality mediated by ectomycorrhizal fungal communities. *New Phytol.* **224**, 155–165. (doi:10.1111/nph.15993)
23. Beiler KJ, Durall DM, Simard SW, Maxwell SA, Kretzer AM. 2010 Architecture of the wood-wide web: *Rhizopogon* spp. genets link multiple Douglas-fir cohorts. *New Phytol.* **185**, 543–553. (doi:10.1111/j.1469-8137.2009.03069.x)
24. van der Heijden MGA, Horton TR. 2009 Socialism in soil? The importance of mycorrhizal fungal networks for facilitation in natural ecosystems. *J. Ecol.* **97**, 1139–1150. (doi:10.1111/j.1365-2745.2009.01570.x)
25. Pérez-Izquierdo L, Zabal-Aguirre M, González-Martínez SC, Buée M, Verdú M, Rincón A, Goberna M. 2019 Plant intraspecific variation modulates nutrient cycling through its below ground rhizospheric microbiome. *J. Ecol.* **107**, 1594–1605. (doi:10.1111/1365-2745.13202)
26. Pickles BJ, Genney DR, Potts JM, Lennon JJ, Anderson IC, Alexander IJ. 2010 Spatial and temporal ecology of Scots pine ectomycorrhizas. *New Phytol.* **186**, 755–768. (doi:10.1111/j.1469-8137.2010.03204.x)
27. Gehring CA, Sthultz CM, Flores-Rentería L, Whipple AV, Whitham TG. 2017 Tree genetics defines fungal partner communities that may confer drought tolerance. *Proc. Natl Acad. Sci. USA* **114**, 11 159–11 174. (doi:10.1073/pnas.1704022114)
28. Horton MW *et al.* 2014 Genome-wide association study of *Arabidopsis thaliana* leaf microbial community. *Nat. Commun.* **5**, 5320. (doi:10.1038/ncomms6320)
29. Bergelson J, Mittelstrass J, Horton MW. 2019 Characterizing both bacteria and fungi improves understanding of the *Arabidopsis* root microbiome. *Scient. Rep.* **9**, 24. (doi:10.1038/s41598-018-37208-z)
30. Brachi B *et al.* 2017 Plant genes influence microbial hubs that shape beneficial leaf communities. *bioRxiv*, 181198. (doi:10.1101/181198)
31. Peiffer JA, Spor A, Koren O, Jin Z, Tringe SG, Dangl JL, Buckler ES, Ley RE. 2013 Diversity and heritability of the maize rhizosphere microbiome under field conditions. *Proc. Natl Acad. Sci. USA* **110**, 6548–6553. (doi:10.1073/pnas.1302837110)
32. Wallace JG, Kremling KA, Kovar LL, Buckler ES. 2018 Quantitative genetics of the maize leaf microbiome. *Phytobiomes J.* **2**, 208–224. (doi:10.1094/pbiomes-02-18-0008-r)
33. Kiers ET, Rousseau RA, Denison RF. 2006 Measured sanctions: legume hosts detect quantitative variation in rhizobium cooperation and punish accordingly. *Evol. Ecol. Res.* **8**, 1077–1086.
34. Ji B, Bever JD. 2016 Plant preferential allocation and fungal reward decline with soil phosphorus: implications for mycorrhizal mutualism. *Ecosphere* **7**, e01256. (doi:10.1002/ecs2.1256)
35. Steidinger BS, Bever JD. 2016 Host discrimination in modular mutualisms: a theoretical framework for

meta-populations of mutualists and exploiters. *Proc. R. Soc. B* **283**, 20152428. (doi:10.1098/rspb.2015.2428)

36. Currier WW, Strobel GA. 1976 Chemotaxis of *Rhizobium* spp. to plant root exudates. *Plant Physiol.* **57**, 820–823. (doi:10.1104/pp.57.5.820)
37. Lebeis SL *et al.* 2015 Salicylic acid modulates colonization of the root microbiome by specific bacterial taxa. *Science* **349**, 860–864. (doi:10.1126/science.aaa8764)
38. Zhalnina K *et al.* 2018 Dynamic root exudate chemistry and microbial substrate preferences drive patterns in rhizosphere microbial community assembly. *Nat. Microbiol.* **3**, 470–480. (doi:10.1038/s41564-018-0129-3)
39. Louca S, Parfrey LW, Doebeli M. 2016 Decoupling function and taxonomy in the global ocean microbiome. *Science* **353**, 1272–1277. (doi:10.1126/science.aaf4507)
40. Jia Y, Whalen JK. 2020 A new perspective on functional redundancy and phylogenetic niche conservatism in soil microbial communities. *Pedosphere* **30**, 18–24. (doi:10.1016/S1002-0160(19)60826-X)
41. Lambais MR, Barrera SE, Santos EC, Crowley DE, Jumpponen A. 2017 Phyllosphere metaproteomes of trees from the Brazilian Atlantic Forest show high levels of functional redundancy. *Microb. Ecol.* **73**, 123–134. (doi:10.1007/s00248-016-0878-6)
42. Knief C, Delmotte N, Chaffron S, Stark M, Innerebner G, Wassmann R, von Mering C, Vorholt JA. 2012 Metaproteogenomic analysis of microbial communities in the phyllosphere and rhizosphere of rice. *ISME J.* **6**, 1378–1390. (doi:10.1038/ismej.2011.192)
43. Johnson NC, Wilson GWT, Wilson JA, Miller RM, Bowker MA. 2015 Mycorrhizal phenotypes and the law of the minimum. *New Phytol.* **205**, 1473–1484. (doi:10.1111/nph.13172)
44. Giauque H, Connor EW, Hawkes CV. 2019 Endophyte traits relevant to stress tolerance, resource use and habitat of origin predict effects on host plants. *New Phytol.* **221**, 2239–2249. (doi:10.1111/nph.15504)
45. Maignien L, DeForce EA, Chafee ME, Eren AM, Simmons SL. 2014 Ecological succession and stochastic variation in the assembly of *Arabidopsis thaliana* phyllosphere communities. *mBio* **5**, e00682-13. (doi:10.1128/mBio.00682-13)
46. Heath KD, Stock AJ, Stinchcombe JR. 2010 Mutualism variation in the nodulation response to nitrate. *J. Evol. Biol.* **23**, 2494–2500. (doi:10.1111/j.1420-9101.2010.02092.x)
47. Gomulkiewicz R, Holt RD, Barfield M, Nuismer SL. 2010 Genetics, adaptation, and invasion in harsh environments. *Evol. Appl.* **3**, 97–108. (doi:10.1111/j.1752-4571.2009.00117.x)
48. Vigani G *et al.* 2019 Root bacterial endophytes confer drought resistance and enhance expression and activity of a vacuolar H^+-pumping pyrophosphatase in pepper plants. *Environ. Microbiol.* **21**, 3212–3228. (doi:10.1111/1462-2920.14272)
49. Carrión VJ *et al.* 2019 Pathogen-induced activation of disease-suppressive functions in the endophytic root microbiome. *Science* **366**, 606–612. (doi:10.1126/science.aaw9285)
50. Shade A, Stopnisek N. 2019 Abundance-occupancy distributions to prioritize plant core microbiome membership. *Curr. Opin. Microbiol.* **49**, 50–58. (doi:10.1016/j.mib.2019.09.008)
51. Connor EW, Sandy M, Hawkes CV. 2017 Microbial tools in agriculture require an ecological context: stress-dependent non-additive symbiont interactions. *Agron. J.* **109**, 917–926. (doi:10.2134/agronj2016.10.0568)
52. Fukami T. 2015 Historical contingency in community assembly: integrating niches, species pools, and priority effects. *Annu. Rev. Ecol. Evol. Syst.* **46**, 1–23. (doi:10.1146/annurev-ecolsys-110411-160340)
53. Adame-Álvarez R-M, Mendiola-Soto J, Heil M. 2014 Order of arrival shifts endophyte–pathogen interactions in bean from resistance induction to disease facilitation. *FEMS Microbiol. Lett.* **355**, 100–107. (doi:10.1111/1574-6968.12454)
54. Leandro LFS, Eggenberger S, Chen C, Williams J, Beattie GA, Liebman M. 2018 Cropping system diversification reduces severity and incidence of soybean sudden death syndrome caused by *Fusarium virguliforme*. *Plant Dis.* **102**, 1748–1758. (doi:10.1094/pdis-11-16-1660-re)
55. Strauss SY, Irwin RE. 2004 Ecological and evolutionary consequences of multispecies plant-animal interactions. *Annu. Rev. Ecol. Evol. Syst.* **35**, 435–466. (doi:10.1146/annurev.ecolsys.35.112202.130215)