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Revisiting the Concept of Host Range of Plant Pathogens

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Abstract

Strategies to manage plant disease—from use of resistant varieties to crop rotation, elimination of reservoirs, landscape planning, surveillance, quarantine, risk modeling, and anticipation of disease emergences—all rely on knowledge of pathogen host range. However, awareness of the multitude of factors that influence the outcome of plant–microorganism interactions, the spatial and temporal dynamics of these factors, and the diversity of any given pathogen makes it increasingly challenging to define simple, all-purpose rules to circumscribe the host range of a pathogen. For bacteria, fungi, oomycetes, and viruses, we illustrate that host range is often an overlapping continuum—more so than the separation of discrete pathotypes—and that host jumps are common. By setting the mechanisms of plant–pathogen interactions into the scales of contemporary land use and Earth history, we propose a framework to assess the frontiers of host range for practical applications and research on pathogen evolution.

INTRODUCTION

Knowing which plants are likely to harbor and/or succumb to a given pathogen has numerous practical applications for the management of crop health, forests, parks, and recreational areas and for preserving biodiversity. The notion of the host range of a plant pathogen is epitomized by the sets of plants used as differentials to characterize strains of rust fungi, resulting in matrices of compatible and incompatible reactions and the identification of races. Likewise, the ranges of plant species on which strains of a given species of bacteria or fungi cause disease are used to define pathovar, forma specialis, and other denominations of pathotype. Host range has also been used for decades as a taxonomic criterion for plant viruses. These categorizations orient the creation of diagnostics, breeding for disease resistance, recommendations for crop rotations, and modeling of disease epidemics. They are also the foundation for concepts about the evolution of pathogenicity.

Behind the seeming straightforwardness of the notion of host range are numerous questions about its scope. Is host range the set of plants in which microorganisms cause disease or in which they can proliferate regardless of apparent symptoms? Does host range comprise those plants in which microorganisms have been observed to proliferate or cause disease under natural conditions—which includes the multitude of vagaries leading to encounters with plants under optimal environmental conditions—or should the results of experimental conditions also be considered? What are the advantages and disadvantages of a flexible scope when considering the host range of plant pathogens? In this review, we revisit the notion of the host range of plant pathogens to provide a framework that can reconcile research on the evolution of host range with practical applications related to managing plant health in the context of intensifying global change. We use the terms host and pathogen for convenience while keeping in mind the debate on whether these are processes rather than attributes of organisms (see sidebar titled Pathogens and Hosts: Attributes or Processes?).

FLEXIBLE DETERMINANTS OF HOST RANGE

The ability of a microorganism to proliferate and cause disease in a plant is, in part, determined by complex molecular interactions between the plant and the microorganism. These interactions

PATHOGENS AND HOSTS: ATTRIBUTES OR PROCESSES?

The terms pathogen and host are historically and almost universally used to describe attributes of organisms and categorize them into behavioral groups. These notions have their origin in the early theories of contagion and were at one time useful in the adoption of hygienic practices to control disease (107). But with increasing awareness of the vast diversity of the microbial world and the varieties of microbial lifestyles and their malleability, the distinction between pathogens and nonpathogens is blurred. Furthermore, accumulating evidence about microbial traits and genomic architecture reveals that there is no structure or function unique to pathogens (107).

Recent debates, particularly in medical microbiology, have highlighted the significance of disease as a process, making the notion of pathogen, in particular, more and more indefensible (107). Likewise, the host is one part of a complex ecological and environmental context that contributes to the outcome of the interaction between a microorganism and another organism—animal, plant, or other. The main message of this debate is that a better understanding of virulence would come from identifying the environmental circumstances in which the outcome of these interactions leads to disease rather than searching for the traits that delimit pathogens from commensals, symbionts, mutualists, etc.

occur at all levels of organization of plant tissues and cells, from those at the cutinaceous and corky covers of the epidermis of leaves and stems and at root hairs and root cap cells to those in the apoplast, at the cell wall, and at the level of organelles in the cell lumen, including the nucleus, as summarized in numerous recent reviews (56, 78, 84, 89, 94, 125, 163). These molecular interactions are commonly referred to as a component of an arms race in the coevolution of microorganisms with plants. Nevertheless, there is growing evidence that host jumps—and not coevolution per se—have contributed to the host range of plant pathogens more than has been generally accepted (33, 44, 105, 113). Host jumps can occur because of genetic variation of pathogens via horizontal gene transfer, mutations, and recombination. But host jumps could also be fostered by inherent flexibility in microbial specificity due to environmental factors that can modulate the molecular interactions. Notably, plant responses to microorganisms are modulated by temperature, water availability, and soil nutrients in particular (47, 156). Physical environmental conditions influence the outcome of plant–microbe interactions via, for example, their modulation of the effectiveness of PAMP (pathogen-associated molecular pattern)-triggered immunity, regulation of defense hormones, expression of proteins involved in effector-triggered immunity of the plant, expression of phytotoxins, and regulation of the type III secretion system (T3SS) by bacteria (156). The specificity of molecular interactions can also be influenced by the amino acids that compose secreted proteins of pathogens. In eukaryotic and prokaryotic animal pathogens, the abundance of primitive amino acids and intrinsically disordered residues in secreted proteins contributes to wider host ranges by enhancing the flexibility of their interactions with host proteins (22). Likewise, viral proteins are particularly rich in intrinsically disordered domains, i.e., domains that fail to fold into a unique 3D conformation but adopt different conformations depending on their ligands (161). Hence, intrinsic disorder may help viruses interact with multiple ligands in a plant, increasing their host-range breadth and favoring host jumps (28). Co-occurrence with other microorganisms can also influence the outcome of plant–microbe interactions (14). Opportunity is also a factor in determining the apparent host range of pathogens. For example, the expansion of eucalyptus plantations outside of the natural range of this tree has revealed the capacity of *Erwinia psidii*, which is pathogenic on guava in South America, to also infect eucalyptus (4). Another example is leaf blast of wheat caused by *Magnaporthe oryzae* that probably arose from strains infecting other Poaceae, as described below. In light of the multitude of factors involved in host range and the complexity of their interactions and modulation by environmental parameters, it is reasonable to wonder whether there are indeed clear patterns of host range that distinguish groups of microorganisms into pathotypes.

Pathogen-associated molecular pattern (PAMP): pathogen molecules that trigger the innate immunity in plants and are relatively conserved among groups of pathogens or microbes. The term is also used in vertebrate–microbe interactions

Type III secretion system (T3SS): protein appendage of several Gram-negative bacteria that acts as a sensor to detect eukaryotic hosts and allows the secretion of proteins within the host cell to promote bacterial infectivity

PATTERNS OF HOST RANGE

Host ranges of plant pathogens and other parasites are characterized both by host-range breadth, i.e., the total number of host taxa whose members can be infected, and by the genetic diversity of taxa containing hosts. Some pathogens are able to infect only a single plant species or a subset of genotypes within a plant species, whereas others are able to infect very large sets of plant species or even members of other kingdoms. *Tomato spotted wilt virus* (order *Bunyavirales*), for example, multiplies in thrips (insects in the order Thysanoptera) as well as in >1,000 plant species (117). Likewise, the fungus *Phytophthora ramorum* (69) and members of the Sclerotiniaceae (113), the bacterium *Xylella fastidiosa* (30, 41), and the *Pseudomonas syringae* complex (108) can multiply and/or cause disease in species in multiple plant families. In contrast, obligate biotrophic fungi usually have much narrower host ranges than facultative necrotrophic pathogens (118).

It is important to distinguish the host range observed in naturally occurring epidemics under field conditions, which depends on both extrinsic (e.g., exposure of plants to microorganisms and

environmental conditions favorable for infection) and intrinsic determinants, from the potential host range that can be evaluated by experimental cross-inoculations. Because environmental conditions and exposure of plants to pathogens are highly variable and fluctuating, comparing pathogens for their potential host range is often more meaningful when searching for underlying mechanisms or anticipating future emergences. Yet even when host-range breadth is compared in controlled experimental conditions, large differences occur between pathogen species and genotypes, leading to the notion of specialists and generalists (11). This suggests that host-range breadth is determined by the pathogens' biological properties and evolutionary history. In an extensive database analysis, Gilbert et al. (62) showed that bacteria, fungi, and oomycetes have a significantly higher tendency for host specialization (known hosts belonging to <10 plant genera) than do viruses or other pests (insects, mites, nematodes, parasitic plants), probably because of the more intimate relationships and higher dependencies of microorganisms on their hosts than those for other pests. Viruses may represent an exception because their host range is in many cases determined by that of their biological vectors (e.g., arthropods and nematodes).

Nestedness and Modularity in Infection Matrices

To determine host ranges, pathogens have been inoculated on different sets of plants. The distribution of infection data in matrices from such inoculations can be analyzed statistically and can provide invaluable insights into phylogenetic, ecological, and/or genetic bases of parasite infectivity and host resistance and into the evolution of these traits. However, to date, inoculation trials usually deploy plants related to the original host of isolation. This makes it difficult to compare host ranges between pathogen species or genotypes. More recently, the general structure of host–pathogen interactions has been described in network-based analyses (55, 157) of matrices containing infection data between every member of a set of pathogens and a set of hosts. Two characteristics of the matrices are particularly important: nestedness and modularity (**Figure 1**). Nestedness occurs when there is a continuum of host-range breadth among strains of the pathogen (and likewise a continuum of resistance spectrum among hosts) and when these ranges overlap. Modularity occurs when compatible interactions between hosts and pathogens are distributed among distinct groups (modules) with few or no cases of overlap between modules. Infection data can correspond to either counts or frequency of observed interactions of a given pathogen with a given host, mostly from ecological or epidemiological studies, or experimental cross-inoculation studies where the strains of all the pathogens in the study have been inoculated on every host. Few network analyses of plant–pathogen interactions are available (**Table 1**).

The network analyses that have been conducted for viruses, fungi, and bacteria point to a consistent trend for nestedness. Analyses of data from surveys of forest health at the French national scale for interactions between tree and fungal species revealed that the matrix was globally both significantly nested and modular (151). The two main modules also had significant internal nested patterns. There was a strong link between modules and higher tree taxonomic ranks but not higher fungal taxonomic ranks, likely because of very early divergence of the major fungal taxa. In contrast, nestedness was poorly related to the taxonomy of the trees or fungi. Both nestedness and modularity were linked to the fungal life history strategy, with root decay fungi having the largest host ranges. This could be due to the high saprophytic ability of these fungi, which allows them to survive without a host and may have increased their likelihood of host jumps (121). Similarly, analysis of a plant–virus species interaction matrix revealed both a modular and a nested pattern (109). Modules were strongly linked with host taxa (families) but not with virus taxa. The matrix distinguished specialist viruses, infecting mostly plants from a given family, and generalist viruses. These studies focused mostly on interactions at the species level for both plants and pathogens.

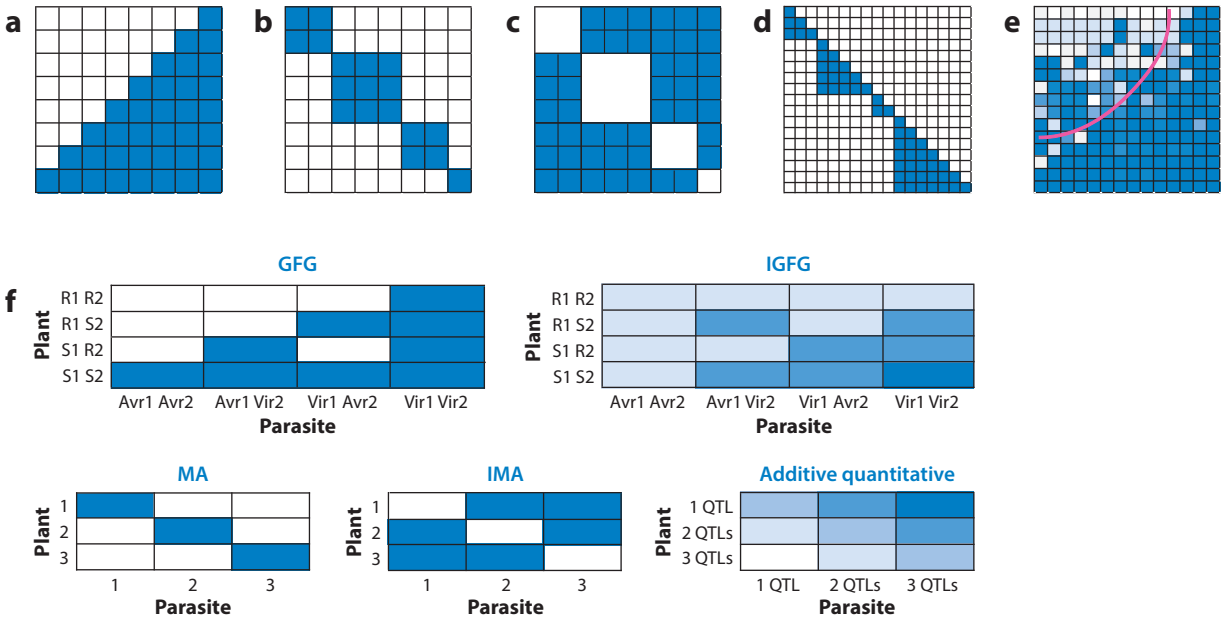


Figure 1

Common structural patterns observed in host–parasite interaction matrices and correspondence with genetic models. (*a*) Nested. (*b*) Modular. (*c*) Antimodular. (*d*) Modular with nested patterns embedded into modules. (*e*) Nestedness observed in a quantitative interaction matrix between barley genotypes (*columns*) and *Puccinia hordei* isolates (*rows*) (data from table 4 in Reference 66). The pink line (isocline) corresponds to a perfectly nested matrix. (*f*) Host–parasite models of interaction in haploids. Gene-for-gene (GFG) and inverse gene-for-gene (IGFG) models, respectively, have two loci (1 and 2) with two alleles each, resistance (R) or susceptibility (S) and avirulence (Avr) or virulence (Vir) (models partly adapted from Reference 52). Matching-allele (MA) and inverse matching-allele (IMA) models have one locus and three alleles (1, 2, and 3). The additive quantitative model has pathogens carrying one to three infectivity quantitative trait loci (QTLs) and plants carrying one to three resistance QTLs. Blue and white cells correspond to infection or lack of infection, respectively. Intermediate shades correspond to intermediate phenotypes (partial infectivity).

Analyses of matrices at the levels of microbial isolates/strains and/or plant genotypes correspond to much more recent evolutionary timescales. Nestedness was prevalent in all matrices from wild populations of *Linum marginale* and the fungal pathogen *Melampsora lini* (9). Modularity was also significant in a few matrices, especially those with a spatial sampling design. Modularity reflected partly local and ecological adaptations: Plants belonging to some of the modules were more prone to be resistant to fungi isolated from other locations and belonging to other modules. In contrast, there were no obvious links between nestedness and either the spatial or temporal structure of sampled populations. Importantly, there was also a strong correlation between the efficiency and spectrum of action of plant resistance: Total resistance had a narrower spectrum of action than partial resistance. The co-occurrence of both kinds of resistance in plant populations may therefore contribute to the maintenance of generalism and specialism in plant–pathogen matrices.

Nested (but not modular) patterns have also been revealed in *P. syringae*–plant species matrices (108), in a *Tobacco etch virus–Arabidopsis thaliana* genotype matrix (75) and in a rust (*Puccinia hordei*)–barley (*Hordeum vulgare*) matrix (66) (**Figure 1*e***). Finally, in an analysis of *Potato virus Y* mutants interacting with genotypes of pepper (*Capsicum annuum*) or tomato (*Solanum* spp.), Moury et al. (110) did not detect any modular patterns, and nestedness was significant only when plant genotypes susceptible to all viruses were included.

Table 1 Structural patterns in matrices of interactions of plants and microbial pathogens

Microbe	Plant host	Data	Nestedness	Modularity	Reference
Fungus species (Dikarya: Ascomycota and Basidiomycota)	Tree (mostly species)	Counts (binary data)	Yes, linked with tree abundance	Yes, strongly linked with tree taxonomy; linked with tree distributional range and fungus life history	151
Virus species	Plant species	Experimental cross-inoculation	Yes	Yes, linked with plant families	109
Melampsora lini populations	*Linum marginale* populations	Experimental cross-inoculation	Yes	Yes/no, linked with ecological and spatial factors	9
Pseudomonas syringae strains	Plant species and genotypes	Experimental cross-inoculation	Yes	No	108
Tobacco etch virus variants from experimental evolution	*Arabidopsis thaliana* ecotypes	Experimental cross-inoculation	Yes	No	75
Potato virus Y strains/mutants	Plant species and genotypes	Experimental cross-inoculation	No (except when including fully susceptible plant)	No	110
Puccinia hordei strains	Barley genotypes	Experimental cross-inoculation–quantitative measure of infection	Yes	No	**Figure 1** based on table 4 in Reference 66

Mechanisms That Underlie Patterns in Infection Matrices

The absence of significant modularity in most of these matrices raises the question of the relevance of within-species classifications of pathogens based on host ranges such as the pathovar or forma specialis concepts for bacteria or fungi (108). Furthermore, the pervasiveness of nestedness that has also been demonstrated for other host–pathogen interactions such as bacteria and phages suggests the existence of widespread mechanisms that are driving and maintaining continua of pathogen host range and the host resistance spectrum. One mechanism to explain the nested or modular structure of interaction matrices lies in the genetic determinism of plant–pathogen interactions. Several genetic models have been proposed based on experimental evidence for these interactions (**Figure 1**). Under the gene-for-gene (GFG) model, host resistance requires recognition of an elicitor, produced by the pathogen, by a host's receptor (54). Consequently, loss or alteration of the elicitor by the pathogen or absence of the host matching resistance allele results in infection. In contrast, under the matching-allele (MA) model, infection success of a pathogen depends on a perfect match with a host's genotype to either circumvent the host's immune system or exploit an essential function of the host (68, 128). Inverse MA (IMA) and inverse GFG (IGFG) models have also been proposed. In the IMA model, infections occur when pathogen genotypes mismatch the host, as in the adaptive immune systems of vertebrates where the host resists infection through recognition of the pathogen alleles (87). In the IGFG model, infection requires recognition of

Elicitor: molecule of plant pathogen, pest, or symbiont that can interact with specific plant receptor proteins and stimulate an immune response in plants

the host by the pathogen and the host becomes resistant by losing the receptor targeted by the pathogen (70, 92).

These different models, corresponding to different genetic interactions between hosts and pathogens, may correspond to different structural patterns in interaction matrices provided that a representative set of host and pathogen genotypes has been studied. The GFG and IGFG models show an inherent nested structure, whereas the MA model shows a modular structure (**Figure 1**). The IMA shows an antimodular structure, i.e., it is less modular than randomly defined matrices. In the case of quantitative resistance and infectivity, often characterized by a continuum of phenotypes, a purely polygenic and additive model, where several resistance quantitative trait loci (QTLs) confer the same increase of resistance whatever the pathogen genotype (and vice versa for pathogen infectivity QTLs), also leads to a nested structure in the interaction matrix.

A second factor contributing to matrix structure beyond the genetic bases of the plant–pathogen interaction is the occurrence of costs linked to the increase of the pathogen's host range and the plant's resistance spectrum (10, 19, 20, 25, 86, 150). These costs could impede the predominance of pathogens with universal pathogenicity and of plants with universal resistance, as in the GFG model, and could explain the absence of generalist pathogens and plant resistance in the MA model. Fitness costs linked to host jumps or host-range expansion have been widely studied in the context of the breakdown of plant resistance genes by pathogen mutants, and costs are apparently frequent and quite high on average (5, 6, 59, 133), although some counter-examples show that resistance breakdown may also confer fitness gains in plants devoid of the resistance gene (5, 57).

Host–pathogen coevolutionary simulations have also been used to explain matrix patterns. Valverde et al. (152) showed that the spatial limitation of host–pathogen interactions favored the occurrence of nested interaction matrices. In their simulation, no nested pattern emerged in the absence of such spatial limitation. Importantly, their analysis considered matrices with quantitative data such as numbers of infections observed in epidemiological surveys. Analyses of matrices of binary data from experimental cross-inoculations might not necessarily lead to the same results.

CHANGES IN HOST RANGE AND THE UNDERLYING MECHANISMS

Variability, Shifts, and Evolution of Host Range

Many reports illustrate that pathogens tend to infect plants that are closely related, rendering the phylogenetic distance between plant taxa an important predictor of the risk of host jump of a given pathogen. Gilbert et al. (62) showed that this tendency was widespread among all categories of plant pathogens and pests, except mollusks that have little plant specificity. They proposed a formula to estimate the probability that a target plant genus is a host for a given pathogen as a function of its phylogenetic distance (in millions of years) to the source plant genus. Their estimates suggest that viruses are less prone to host jumps than other plant pathogens and pests when host distance increases. Similar trends were observed for models predicting the risk of host jumps for animal viruses based on the phylogenetic distance between hosts, such as the steep decline of risk of host jump estimated for RNA viruses infecting primates, carnivores, or terrestrial ungulates (38). When the host range of plant viruses was evaluated experimentally, plant families were revealed to be the taxonomic threshold beyond which barriers to infection increase substantially, whereas barriers to infection were less stringent or less frequent within plant families (109). The relationship between the phylogenetic distance between plant species and their host status for fungi, oomycetes, and bacteria has not been tested experimentally with the same exhaustiveness as for viruses (44). However, the predictions of Gilbert et al. (62) suggest that such experiments

would not reveal barriers to infection at the plant family level that are as stringent as they are for viruses.

Nonhost resistance: plant defense that protects all members of a plant species from infection by a given pathogen

Pattern recognition receptors (PRRs): plant receptors that trigger an immune response upon perception of PAMP/MAMP

One explanation for this phylogenetic signal in pathogen host ranges is that closely related plant taxa offer similar environments to pathogens, both in terms of resources required and immune defenses that need to be circumvented. Indeed, nonhost resistance in plants (i.e., plant resistance that protects all members of a plant species against a given pathogen) usually depends on either constitutive defense traits or the affinity between plant receptors [pattern recognition receptors (PRRs)] and pathogen factors (elicitors) (63, 136). Because these plant factors are relatively conserved, the capacity of a given pathogen to circumvent these barriers to infection in several plants decreases with the degree of divergence between the plant factors and consequently with the divergence time between these plants.

There are, however, examples of plant pathogens that are able to infect distant hosts, sometimes in different kingdoms. **Table 2** lists examples of host jumps via either natural infections or experimental inoculation and/or inferred from phylogenetic analyses. Various microorganisms are known to associate with hosts across a range of kingdoms. For example, members of the *Narnaviridae*, a family of RNA viruses, are widespread in filamentous fungi but also in invertebrates. In many plants, the mitochondrial genome contains sequences of mitoviruses, a genus within the *Narnaviridae*. Mitovirus sequences were also found in plant nuclear genomes and were apparently transferred from the mitochondria. Transcribed sequences suggest that they correspond to true

Table 2 Examples of cross-kingdom host jumps of plant microbial pathogens or endophytes[a]

Microorganism	Primary host[b]	Secondary host[c]	Evidence[d]	Reference
Bacteria				
Propionibacterium acnes type Zappae	Human	*Vitis vinifera* (endophyte)	N	120
Erwinia aphidicola	Gut of pea aphid	Pea, bean, pepper	N	99
Fungi				
Trichoderma spp.	Plant/fungi	Fungi/plant	P	31
Phytomyxea	Angiosperms	Oomycetes	P	114
Phytomyxea	Brown algae/diatoms	Angiosperm	P	114
Cryptococcus gattii	Mammals, birds	Various angiosperms and gymnosperms	N, E	141
Viruses				
Cucumber mosaic virus	Plants	*Rhizoctonia solani*, *Valsa mali*	N, E	1
Bunyavirales (*Orthotospovirus*, *Emaravirus*, *Tenuivirus*)	Insects/vertebrates	Plants	P	159
Rhabdoviridae (*Cytorhabdovirus*, *Nucleorhabdovirus*, *Dichorhavirus*, *Varicosavirus*)	Insects/vertebrates	Plants	P	159
Reoviridae (*Oryzavirus*, *Fijivirus*, *Phytoreovirus*)	Insects/vertebrates/fungi	Plants	P	159
Ourmiavirus	Invertebrate/fungi	Plants	P	130
Apple mosaic virus, *Cytorhabdovirus*	Lichens or plants	Plants or lichens	P, N, E	123
Tobacco ringspot virus	Plants	*Apis mellifera* (honeybee)	N	36, 93

[a]Numerous other examples, not shown in the table, of bacteria and fungi that are mostly opportunistic pathogens or associated with immunocompromised patients and have been observed to also associate with plants as endophytes or pathogens have been reviewed elsewhere (153).
[b]Host on which the microorganism was first observed or the original host inferred from phylogenetic analyses.
[c]Host on which the microorganism was observed at a later date than the primary host or secondary host inferred from phylogenetic analyses.
[d]Type of evidence suggesting host jump: phylogenetic (P), natural infection (N), experimental infection (E).

plant mitoviruses rather than sequences integrated into the genome of mitochondria. The origin of these viruses and their mode of transmission between plants and fungi are still unknown (129).

Ourmiavirus is a genus with an RNA-dependent RNA polymerase (RdRp), the essential protein involved in the genome replication of RNA viruses, similar in sequence to that of the *Narnaviridae*. However, ourmiaviruses acquired additional protein-coding sequences that seem to have increased their capacity to infect plants—in particular, an open reading frame that codes a movement protein (MP) most closely related to plant tombusviruses. Hence, ourmiaviruses seem to be derived partly from the fungus- and invertebrate-infecting *Narnaviridae*. In addition, the *Amalgaviridae*, *Chrysoviridae*, *Endornaviridae*, *Partitiviridae*, and *Totiviridae*, which contain plant-persistent viruses (i.e., viruses that are exclusively vertically transmitted), also have members that infect plant-interacting fungi. These families comprise double-stranded RNA viruses, except the *Endornaviridae*, which is a group of single-stranded RNA viruses. The origin of these viruses and the direction of putative host shifts are not known with certainty. However, in most cases, virus and host phylogenies are not congruent, suggesting extensive virus shifts between plants and fungi.

Some pathogens, mostly viruses, are shared between plants and insects. Several virus groups (*Bunyavirales*, *Rhabdoviridae*, and *Reoviridae*) comprise both plant-infecting and insect-infecting genera or clades. Phylogenetic analyses suggest that plant-infecting *Bunyavirales* and *Rhabdoviridae* became specialized on plants secondarily and were perhaps derived from insect viruses after the insects themselves adapted to plants. Acquisition of an additional protein or protein domain (the MP) involved in cell-to-cell movement fostered the adaptation of these viruses to plants (88). Indeed, for some viruses, the MP is the essential factor limiting host shifts from insects to plants, as demonstrated by complementation experiments (39). Other examples are indicated in **Table 2**.

As for the phylogenetic signal of host range in plants, there are a few reports of a phylogenetic signal for host range in pathogens (44). Closely related pathogen species or genotypes tend to share similar host ranges at the plant species level but usually not at the plant intraspecific level. This could be due to the conservation of elicitors (MAMP-like bacteria flagellins, components of the fungal cell wall, membrane polysaccharides) in pathogens that are the basis of fundamental functions and that trigger nonhost resistance based on PRR (148).

Host ranges of pathogenic microorganisms and other plant pests are not immutable. However, we know relatively little about the frequency and timescale of host jumps in pathogens. The most widespread method of inferring host jumps is cophylogeny analysis of hosts and pathogens. When the phylogeny of pathogens mirrors perfectly that of hosts (i.e., cases of phylogenic congruence), the most parsimonious hypothesis is the codivergence (cospeciation in case of species) between the two groups of taxa and absence of host jumps—although this conclusion might be inaccurate (43). In contrast, complete lack of correlation between both phylogenies (incongruence) suggests frequent host jumps but does not necessarily allow precise inference of these events. For partial incongruence, different algorithms allow the reconstruction of host jumps and their differentiation from events of codivergence, extinction, and duplication of pathogen clades (44, 81). Cophylogeny analyses are also useful to date host jump events provided that absolute dates can be attributed to nodes in the phylogenetic trees of hosts and pathogens.

Cophylogeny analyses show that host jumps are the rule rather than the exception and can be detected in almost all studies (**Table 3**). Furthermore, they confirm that jumps to closer host relatives are more probable. Usually, fewer host jumps are detected than codivergence events (**Table 3**), but there is certainly a bias because frequent host jumps blur the cophylogenetic signal and preclude host jump inference. Host jumps can also be associated with the emergence of generalist pathogens as shown for the Sclerotiniaceae (113). However, many genetic and ecological factors, as well as pathogen traits, are involved in pathogen evolution toward generalism or specialism (11, 27, 72).

Table 3 Results of cophylogenic analyses between plants and microbial pathogens

Microbial taxon	Plant taxon	Events	Reference
Fungi			
Exobasidiales (Basidiomycota)	Asteraceae/Lauraceae	0–8 HJ, 16–24 CD, 8–16 DU	81
Entyloma spp. (Basidiomycota)	Asteraceae	0–7 HJ, 6–14 CD, 6–14 DU	81
Cintractia spp. (Basidiomycota)	Poales	0–3 HJ, 16–18 CD, 4–6 DU	81
Ustilago spp. (Basidiomycota)	Poaceae	0–7 HJ, 8–16 CD, 8–16 DU	81
Tilletia spp. (Basidiomycota)	Pooideae/Panicoideae	0–7 HJ, 10–16 CD, 8–14 DU	81
Microbotryum spp. (Basidiomycota)	Caryophyllales	0–6 HJ, 12–18 CD, 8–14 DU	81
Cronartium spp. (Basidiomycota)	*Pinus* spp.	0–6 HJ, 16–18 CD, 12–14 DU	81
Phragmidium spp. (Basidiomycota)	Rosaceae	0–8 HJ, 10–18 CD, 8–16 DU	81
Uromyces spp. (Basidiomycota)	Rosidae	0–7 HJ, 10–16 CD, 8–14 DU	81
Homobasidiomycetes (Basidiomycota)	Monotropoideae	0–1 HJ, 12–16 CD, 2–6 DU	81
Puccinia spp. (Basidiomycota)	Brassicaceae	HJ > CD	132
Microbotryum spp. (Basidiomycota)	Caryophyllaceae	Cospeciation not the rule; host jumps pervasive but not to too distant host species	127
Epichloë spp. (Ascomycota)	Pooideae	0–7 HJ, 8–18 CD, 6–16 DU	81
Claviceps spp. (Ascomycota)	Poaceae	0–6 HJ, 10–16 CD, 8–14 DU	81
Erysiphe spp. (Ascomycota)	Asteridae/Rosidae	0–5 HJ, 14–18 CD, 8–12 DU	81
Golovinomyces spp. (Ascomycota)	Asteraceae	0–6 HJ, 18–24 CD, 6–12 DU	81
Monilinia spp. (Ascomycota)	Asteridae/Rosidae	0–6 HJ, 12–16 CD, 8–12 DU	81
Epichloë spp. (Ascomycota)	Pooideae	5 HJ, CD	135
Cyttaria (Ascomycota)	Nothofagus	1–2 HJ, 7–8 CD, 1–2 DU	122
Anthracoidea spp.	*Carex* spp.	19–22 HJ + DU, 7–10 CD	50
Family Sclerotiniaceae (Ascomycota)	Plants	30–37% HJ, 13–18% CD, 31–37% DU[a]	113
Blumeria graminis (Ascomycota)	Poaceae	2 HJ, CD	106
Bremia spp. (Oomycetes)	Asteraceae	25–43 HJ, 18–29 CD, 0–5 DU[a]	33
Bacteria			
Frankia spp.	Angiosperm	8 CD, 9 DU	82
Virus			
Wheat dwarf virus	Poaceae	2 HJ, 6 CD	162
Tobamovirus	Monocots and dicots	No congruence	119
Partitiviridae	Viridiplantae and fungi (Ascomycota and Basidiomycota)	Two virus families with codivergence and two without. Many duplications and jumps even in cases of codivergence	65

[a]Only results from the CoRe-PA software are presented.
Abbreviations: CD, codivergence; DU, duplication; HJ, host jump.

Direct examples of host jumps to new plant genotypes (often perceived as a breakdown of resistance) are widespread (60, 104) and are usually discerned through direct epidemiological evidence rather than through cophylogeny analyses because phylogenies are poorly resolved at the within-species level for both hosts and pathogens. Breakdown can be rapid and frequent, sometimes occurring a few months or years after deployment of plant genotypes with a new resistance gene. As underlined above, they are frequently associated with fitness costs on alternative hosts. They can also affect other fitness traits linked to, for example, transmission (for viruses) or survival (16, 58).

Mechanisms Underlying Host Jumps

A large diversity of mechanisms are responsible for host jumps among plant pathogens, including hybridization, horizontal gene transfer, partial or total gene deletion, and amino acid substitutions. Host jumps can occur through intraspecific hybridization, as in the cases of *Blumeria graminis* f. sp. *triticale*, which resulted from the hybridization of two *B. graminis* subspecies specialized on wheat and rye, leading to an added adaptation to triticale crops, a wheat–rye hybrid (106), and of *X. fastidiosa* subsp. *morus*, where recombination led to the adaptation to mulberry (115). Interspecific hybridization has also contributed to host jumps and host-range expansion, as in the case of *Phytophthora* species hybrids (42). In the case of plant viruses, a major trait determining the plant host-range breadth is the number of nucleic acid segments in the virus genome (109). Larger host ranges were observed for viruses with three RNA or DNA genome segments. One hypothesis is that this segment number is the optimal compromise between (*a*) the capacity to exchange entire genome segments between viral strains (i.e., reassortment), which could promote host-range expansion and virus adaptation more generally, and (*b*) the necessity that all segments penetrate into plant cells for efficient infection. Gene loss can also contribute to host jumps. Extensive loss of genes in the genome of the smut fungus *Melanopsichium pennsylvanicum* was correlated with a jump from monocots to dicots (139). For *Magnaporthe oryzae*, its emergence on wheat was due to the loss of function of a single avirulence gene targeted by a particular resistance gene widespread in wheat cultivars (80), a mechanism similar to host jumps at the within-plant species level when resistance breaks down. In contrast, it was demonstrated that *Citrus tristeza virus*, in spite of having a small and constrained genome like all plant viruses, acquired several genes independently that enabled it to extend its host range on *Citrus* spp. (146). Most changes in viruses that alter the range of host plant species are conferred by one or several amino acid substitutions in the viral genome, as in the case of acquisition of infectivity on *Raphanus* spp. by *Brassica* species–infecting *Turnip mosaic virus* populations (142), jumps of *Papaya ringspot virus* from papaya to cucurbit (32), the jump of *Rice yellow mottle virus* (RYMV) from *Oryza sativa* to *Oryza glaberrima* (124), and *Potato virus Y* adaptation to pepper from other solanaceous species (155). Similarly, the jump of *Phytophthora* spp. between *Solanum* spp. and *Mirabilis jalapa* and subsequent specialization involved amino acid substitutions in protease inhibitors (46).

The previous examples concern jumps between plant species, a phenomenon that is less frequently described than jumps within plant species. Furthermore, the molecular determinants responsible for pathogen jumps between genotypes of a given plant species have been more frequently unraveled than jumps at larger taxonomic scales (133). Such jumps usually involve horizontal gene transfer, partial or total gene deletion, and nucleotide and/or amino acid substitutions. Several of these mechanisms can operate independently, leading to the same host jump (40). Given the relatively small number of between-plant-species jumps elucidated at the molecular level, it is difficult to compare them with jumps at the within-plant-species level. However, it seems that the same kinds of molecules and the same mechanisms can be at play in both cases. One of the most striking examples comes from RYMV, for which jumps between *O. sativa* and *O. glaberrima* and breakdown of resistances within these plant species involve the same viral protein and even mutations at contiguous amino acid positions (124). Similarly, contiguous amino acid positions in a given viral protein can be responsible for both a host jump and a resistance breakdown (76, 144, 155). The example of the shift of *M. oryzae* to wheat described above also supports the similarity of mechanisms involved in jumps between or within plant species. However, in spite of the similarities between nonhost and cultivar-specific plant resistance (49, 112) and adaptation of pathogens to these resistances, nonhost resistance can involve widely different mechanisms, including large arrays of constitutive defenses, and hence a greater diversity of ways for a pathogen to adapt to these defenses can be expected (63).

THE EVOLUTIONARY HISTORY OF PLANTS AS HOSTS TO MICROORGANISMS

The diversity of host shift processes described above begs for a more comprehensive perspective on the context in which pathogenicity and host range evolve. A growing number of reports are pushing back, by hundreds of millions of years, the time frame in which the evolutionary history of pathogens is being considered (15, 33, 71, 80, 113, 116). These reports are built on robust methods for dating evolutionary timelines of prokaryotes and eukaryotes that are now consolidated and available at **http://www.timetree.org** as described in two milestone publications (73, 100). By pushing the evolutionary context beyond the advent of agriculture, these works provide perspectives that could be useful to outsmarting the pathways of host-range evolution via novel plant breeding and other disease-management strategies. For human pathogens such as multidrug-resistant enterococci, the drivers of the evolution of traits that foster their survival and dissemination in hospital environments—resistance to desiccation and disinfectants and their capacity to adapt to the changing carbohydrate availability that typifies intestines—date back to the Paleozoic Era (90) and are unlikely to vary much within this group of bacteria. To foster an analogous perspective for plant pathogens, here we situate their evolutionary history in the context of the colonization of land by vascular plants.

Ancient Interactions That Helped Plants Take a Foothold on Land

The early interactions of microorganisms with ancestors of land plants, beginning around 400 million years ago (Mya), were first and foremost a means for microorganisms to obtain food and protected habitats. From the point of view of long-term evolution across the archaeological periods of Earth's history, plants in turn have gained remarkable benefits from these intimate interactions, including the establishment of root architecture and access to recalcitrant resources such as atmospheric nitrogen and soil minerals and motors of genome expansion (i.e., genetic material that leads to increases in genome size). At the onset of these interactions, there were already the foundations of signaling, defense, metabolic regulation, and degradative enzymatic functions in plants and microorganisms that facilitated contemporary plant–microbe interactions. Today, these mechanisms are interpreted as parts of an arms race. But on a long-term evolutionary scale, they are the foundation of processes that have favored coexistence and diversification since the existence of the first organisms on Earth.

Modern terrestrial plants are the descendants of the early colonizers of the land masses that emerged approximately 450 Mya. These land masses were devoid of soil per se, extremely poor in nutrients, and inhabited by bryophyte-like streptophytes that interacted with fungi (17). The tissues of these primitive plants were highly permeable to water and nutrients and also to penetration by fungi (26). Their cell walls contained pectin, a component common to all streptophytes for the past 750 million years (17). They did not have roots but stems that were on, or under, the ground. Being essentially aerial, the plant body was covered with cutin. They produced strigolactones, which are molecules involved in developmental processes throughout the plant kingdom from Charophycean algae to liverworts, mosses, and flowering plants (23). These plants also already had defense systems that recognized fungi. Plants ranging from streptophyte algae to angiosperms have lysin motif receptor–like kinases and calcium- and calmodulin-dependent protein kinases that are sensors for oligomers of *N*-acetylglucosamine, the building block of chitin, suggesting that their most common ancestor also had these sensors (17). Oligomers of *N*-acetylglucosamine, and, in particular, long-chain oligomers, induce innate immunity in plants (23, 95). Furthermore, shorter and acylated oligomers are factors that favor symbioses of plants with nitrogen-fixing

bacteria and with vascular arbuscular mycorrhizae (23, 95). Therefore, the first plants were prepared to be hosts of microorganisms (138).

Fungi were rather well diversified when they started to colonize land. Well before 450 Mya, the main families of fungi and oomycetes had already separated into the major genetic lineages that constitute the extant fungi (17) and most likely had the capacity to degrade pectin. Fungi in the Chytridiomycota and those that evolved afterward—comprising most of today's plant-associated fungi—diverged after the appearance of pectin in streptophytes 750 Mya and all have orthologs of pectin-degrading enzymes (17). Before the divergence of the Chytridiomycota, there was also an expansion of genes in fungi for carbohydrate-active enzymes such as cellulases (17), and the wide diversity of such enzymes is still evident in extant fungi (164). In addition, by 700 Mya fungi in the Mucoromycota and the Dikarya had developed the capacity to grow as hyphae. This provided them with expanding tips that facilitated absorption of nutrients because of the weaker cross-linking of cell walls at the tips (17). Therefore, the mechanistic foundations for fungal–plant interactions were already in place at the onset of the colonization of land.

Starting with the early land plant *Aglaophyton major* from approximately 400 Mya, there are fossil traces of arbuscular mycorrhizal (AM) associations with plants representing lycopods, sphenophytes, ferns, cycads, gingkoes, conifers, gnetales, and angiosperms throughout all archaeological periods. AM associations in plants with true roots appeared in the fossil record at 385 Mya. The first ancestors of arbuscular mycorrhizae probably acquired protection from competition with other soilborne microorganisms by penetrating into plant tissues (26). The main triggers for the establishment of AM associations—strigolactones, cutin monomers, and chitin-related molecules (23)—are very likely to have played key roles in the earliest establishments of plant–fungal associations. Strigolactones could have stimulated fungal metabolism, hyphal branching, and the production of short-chain chitin oligomers. Cutin monomers could have facilitated hyphopodium differentiation, and the short-chain chitin oligomers that AM ancestors produced could have been perceived by plants to foster their passage into a symbiosis, as they do for extant AM associations (23).

The morphology of AM associations with plants has not changed across the evolutionary history of plants. Furthermore, there is no specificity in the capacity of AM fungal strains to form associations with plants, and there is no evidence for evolution of host specificity (137). Therefore, the effector repertoires of extant AM fungi can give an indication of the ancient profile at the time of the first interactions with plants. *Rhizophagus irregularis*, for example, has the capacity to produce 220 putative effectors representing 23% of its secreted proteins. Compared to the whole body of secreted proteins for this fungus, the effector pool is enriched for proteins that are involved in hormone-related functions and contains markedly fewer proteins involved in immune responses (137). This profile is compatible with processes whereby these fungi modified root architecture and did not elicit defense reactions of the plant.

At the same time that fungi were helping early plants in adapting to Earth's newly forming land masses, bacteria in the genus *Streptomyces* were also contributing to this success. But whereas fungi developed intimate interactions involving entry into the lumen of plant cells, *Streptomyces* modulated the external environment of plants and contributed to the evolution of soil as a habitat. The most recent common ancestor of *Streptomyces* was present on land at least 440 Mya (29) but probably originated another 500 million years before that (12, 100) (**http://www.timetree.org**). These bacteria produce spores that are highly resistant to desiccation, from which branching hyphal filaments emerge. The filaments become masses of mycelia that entrap debris that is subsequently degraded by the wide range of metabolites secreted by these bacteria. They have complex enzymatic systems for utilizing chitin and cellulose, the two most abundant polysaccharides on Earth. They also produce proteases and protease inhibitors as well as approximately 7,000 different secondary

metabolites, including antibiotics, siderophores, and pigments. *Streptomyces* spp. deploy three secretion pathways to export enzymes and metabolites (the Sec pathway, the Esx secretion system, and the Tat pathway), but like other Gram-positive bacteria, they do not have a T3SS. In addition to providing mycelial nets on which early, rootless plants could repose, the significant influence of *Streptomyces* spp. on their extracellular environment is believed to have been conducive to the establishment of land plants, and other soilborne organisms as well, via degradation of recalcitrant biopolymers that accumulated as biomass developed on Earth's newly emerged continents (29).

Viruses have also contributed to the evolutionary success of land plants by their effects on the diversification of plants. Among the various plant viruses, genomes of retroviruses integrate into eukaryotic genomes. Reverse-transcribing viruses are believed to be at the origin of some of the long terminal repeat–retrotransposons (LT-RTs) that are found in plants (see Reference 48). LT-RTs have contributed greatly to the expansion of the size of plant genomes and constitute, for example, 25% and 75% of the genomes of modern rice and maize, respectively. LT-RTs can also regulate the expression of genes near their sites of insertion. Numerous examples of the *Copia* and *Gypsy* lineages of LT-RTs that evolved from reverse-transcribing viruses are believed to have integrated into plant genomes before the divergence of monocots from eudicots (48) that occurred more than 120 Mya (8) (**Figure 2**).

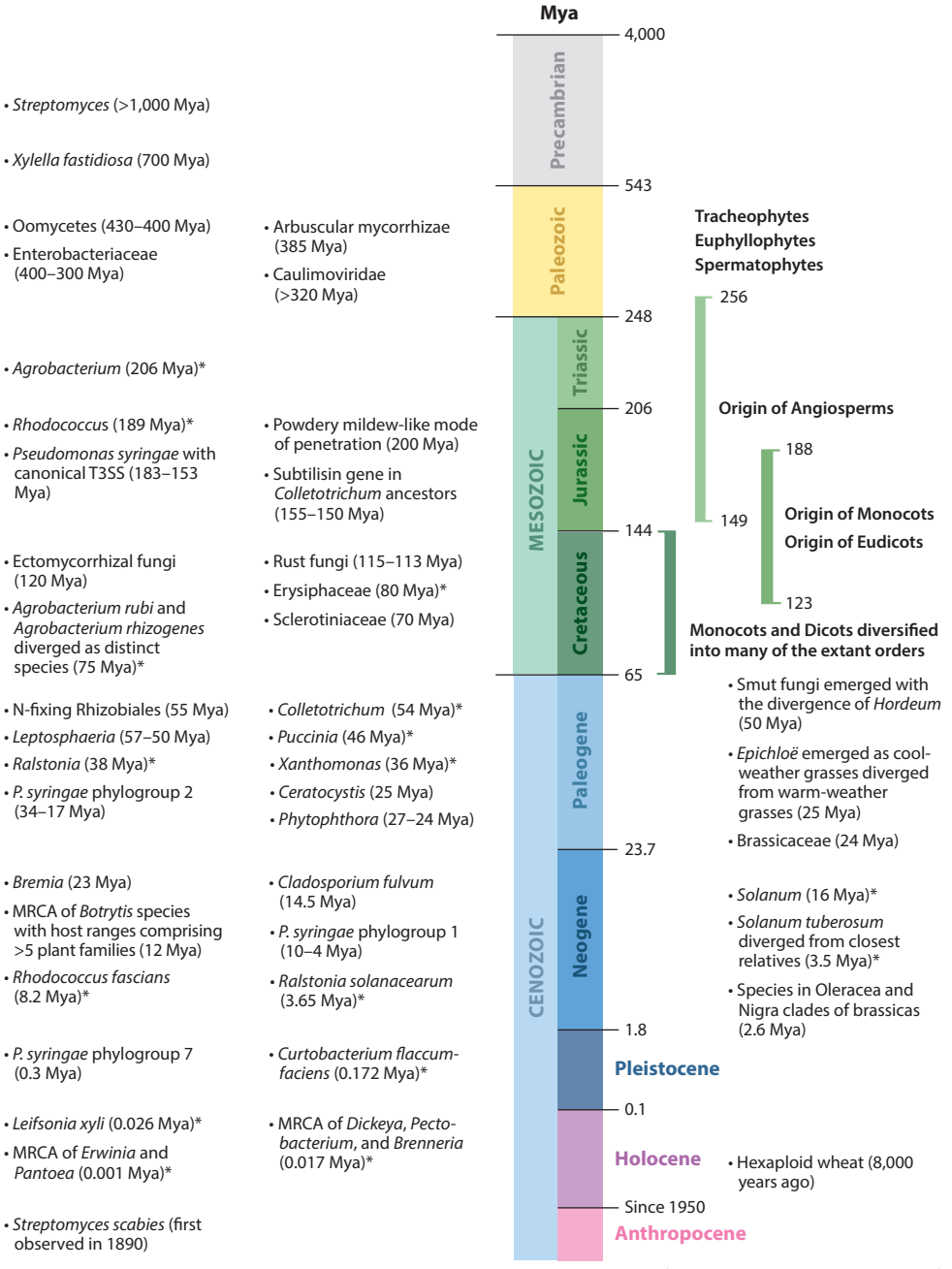
From the Lumen to the Apoplast: The Retreat of Microbial Intimacy with Plants over Evolutionary History

The diversity of plant–microbe interactions that have evolved since these early interactions are, overall, variations on the themes of early interactions. For example, AM fungi penetrate into the lumen of cells, forming specialized structures delimited by the plasma membrane of the plant. This same overall process is deployed by numerous fungi, such as rusts (emerging 115–113 Mya) (105), *Colletotrichum* spp. (54 Mya) (100; **http://www.timetree.org**), smuts (50 Mya) (111), *Phytophthora* spp. (27–24 Mya) (103), and *Bremia* spp. (23 Mya) (33) and by the nitrogen-fixing bacteria in the Rhizobiales (55 Mya) (140).

The main differences among all of these variations of plasma membrane–bound penetration are whether or not the plasma membrane of the plant is breached by the microorganism and to what extent microbial proliferation is restricted in the plant tissues. Breaching of the plasma membrane and unconstrained proliferation of a microorganism could depend on the effectiveness of that microorganism in hindering plant defenses and the way in which plant cells eventually die, if they indeed die (83). Apoptotic death of plant cells makes nutrients more readily available to microorganisms, whereas autophagic death leads to the formation of vacuoles that compartmentalize cytoplasmic contents and renders them less available, and these outcomes can be influenced by the molecular communication between plants and microorganisms (83). The capacity of microorganisms to deploy effectors in waves or pulses could also influence the duration of the balance between restricted and unrestricted growth of the microorganism in plant tissues (83). Species of *Streptomyces* causing hypertrophy and other alterations to belowground plant parts (*Streptomyces scabies* and related species) also penetrate directly into the lumen of plant cells, but the behavior of the plant's plasma membrane has not been described (97). The acquisition of this capacity in this ancient genus of bacteria, conferred by genes for toxins such as thaxtomin, borrelidin, and concanamycins (21), is likely to be very recent. The only species that contain genes for these toxins diverged at a time too recent to be estimated with the tools at **http://www.timetree.org**, but the disease has been known for approximately 150 years (**Figure 2**).

Colonization of the apoplast and the intercellular spaces outside of the plant cell wall was an innovation that probably began with fungi that formed ectomycorrhizal associations and

with various bacteria. The most recent common ancestor of the lines of the species in the *P. syringae* complex with the canonical T3SS, well-known for its dynamic behavior in the apoplast, diverged approximately 183–153 Mya (116). At this date, this ancestral line of *P. syringae* probably had a T3SS that was relatively functional in terms of secretion of proteins given that the core



(*Caption appears on following page*)

Figure 2 (*Figure appears on preceding page*)

Timeline of divergence of plant-associated microorganisms and major plant lineages. The timeline is set in the context of Earth's major geological periods. Estimates of single dates represent the mean age, expressed as million years ago (Mya), of divergence from the most recent common ancestor (MRCA) with the most closely related taxonomic groups. Asterisks indicate estimates of age made with the tools available at TimeTree (**http://www.timetree.org**). *Streptomyces scabies* diverged too recently to be estimated with TimeTree but was first observed in 1890 (147). The most recent estimates of the dates of diversification of plants presented on the right side of the figure (Tracheophytes, Euphyllophytes, Spermatophytes, Angiosperms, Monocots, Dicots, and Eudicots) are provided by Barba-Montoya et al. (8). Additional references: *Streptomyces* and *Xylella*, 12; oomycetes and *Phytophthora*, 103; arbuscular mycorrhizae, 26; Enterobacteriaceae, 13; *Caulimoviridae*, 45; powdery mildew, 35; *Pseudomonas syringae* (canonical T3SS, phylogroup 2, and phylogroup 1), 116; subtilisin gene in *Colletotrichum* ancestors, 3; ectomycorrhizal fungi, 26; rust fungi, 105; Sclerotiniaceae and *Botrytis*, 113; N-fixing Rhizobiales, 140; *Leptosphaeria*, 131; smut fungi and *Hordeum*, 111; *Ceratocystis*, 51; *Epichloë*, 158; *Bremia*, 33; *Cladosporium fulvum*, 37; *Pseudomonas syringae* phylogroup 7, 85; Brassicaceae and species in the Oleracea/Nigra clades of brassicas, 2; hexaploid wheat, 160.

components of this secretion system appeared in bacteria approximately 700 Mya (77). Strains in phylogroup 2 of this complex diverged approximately 34–17 Mya (116), those in phylogroup 1 10–4 Mya (116), and those in phylogroup 7 with noncanonical T3SS only 0.3 Mya (85) (see Reference 18 for definition of the phylogroups). The other genera of γ-Proteobacteria that can thrive in plant apoplasts emerged much more recently, with *Xanthomonas* being the most ancient among them at approximately 36 Mya followed by others until as recently as approximately 1,000 years ago (**Figure 2**). *Agrobacterium* spp. (a genus of α-Proteobacteria) and *Rhodococcus* spp. (a Gram-positive genus) can also invade the apoplast. These genera diverged approximately 200 Mya (**Figure 2**), and the diversification of both led to species that acquired plasmids with genes that alter plant architecture and enhance proliferation of the bacteria (64, 134). The greater diversity of virulence plasmids in *Agrobacterium* spp. than in *Rhodococcus* spp. suggests that this behavior evolved earlier in *Agrobacterium* than in *Rhodococcus* (134). This is consistent with the age of the most recent common ancestor of the oldest extant tumorigenic *Agrobacterium* species—*Agrobacterium rhizogenes* and *Agrobacterium rubi* (75 Mya)—compared to that of the pathogenic species of *Rhodococcus*, i.e., those closely related to *Rhodococcus fascians* (8.2 Mya) (**Figure 2**).

The first ectomycorrhizal fungi appeared approximately 120 Mya in conifers in the Pinaceae and in various angiosperms (26). After the emergence of the first ectomycorrhizal associations, this behavior evolved independently over a dozen times in Ascomycetes and Basidiomycetes (reviewed in Reference 74). A signature trait of ectomycorrhizal fungi is the loss of many of the carbohydrate active enzymes involved in plant cell wall degradation (74, 101) that are otherwise widespread across the fungal kingdom (164) and that were probably present in fungi at the time of the transition to ectomycorrhizal associations (17). However, some endophytic fungal colonizers, such as *Colletotrichum tofieldiae* that diverged only 8.8 Mya did not lose their patrimony of carbohydrate active enzymes. The expression of these enzymes is nevertheless downregulated during intercellular and intracellular growth (71).

Many of the species in the approximately 8,000 genera of fungi that are considered to be pathogens to plants (**http://www.plantpathogen.org/**) move through the intercellular spaces of plant tissues after penetrating through wounds or natural openings. Among this vast diversity, there are examples of fungi that change their behavior in the apoplast with regard to the damage they cause to plant cells and illustrate the plasticity of their interaction with plants. The Sclerotiniaceae, comprising numerous broad-host-range species, emerged approximately 70 Mya (113). Fungi in this family, such as *Sclerotinia* and *Botrytis* species, enter plant tissues and can rapidly rupture plant cells with a range of exoenzymes and then proliferate on the liberated nutrients. But *Sclerotinia sclerotiorum*, for example, can also maintain relatively constrained growth of hyphae in

the apoplast in distal plant tissues, and these outcomes depend on how it deploys oxalic acid and effectors (83). Likewise, *Botrytis deweyae* grows in a restricted manner in the apoplast of daylily (*Hemerocallis* sp.), but in certain hybrids of this plant it degrades plant cells and grows in an unrestricted manner (154). *Fusarium* spp. can also vary in their behavior. The age of this genus has been difficult to assess. Estimates range from 110 to 420 million years depending on the taxonomic group used for calibration of time trees (143). The *Fusarium graminearum* species complex represents strains that are indigenous to North America and were initially endophytes in wild grasses in the plains growing in a relatively constrained manner in grass apoplasts. Growth of the fungus became more unrestricted as the practice of monoculturing a few breeds of wheat and barley was initiated in North America approximately 400 years ago (96).

The capacity for microorganisms to colonize xylem cells of plants is also likely to be ancient, with *X. fastidiosa* as a probable precursor. *X. fastidiosa* emerged as a species approximately 700 Mya (12). Initial entry of extant *X. fastidiosa* into xylem cells depends on xylem-feeding insects. The first such insects, in the Hemiptera (126), emerged on land 160–100 Mya (**http://www.timetree.org**) and could have initiated this interaction as the angiosperms were emerging during this same time period (**Figure 2**). Other extant bacteria apt at multiplying prolifically in the xylem—species of *Leifsonia*, *Clavibacter*, and *Curtobacterium* (all members of the Microbacteriaceae) and members of the *Ralstonia solanacearum* species complex—do not require insects to access the xylem. Although the Microbacteriaceae family is nearly as ancient as *X. fastidiosa*, the species that formed associations with plants are much younger. For example, *Leifsonia xyli* diversified from its most closely related species approximately 26,000 years ago and *Curtobacterium flaccumfaciens* diverged as a species 172,000 years ago (**Figure 2**). For *R. solanacearum*, the biogeography of its phylotypes led to the hypothesis that this species complex emerged approximately 200–150 Mya. But recent molecular clock analyses have revealed that the *Ralstonia* genus diverged only around 38 Mya (98) (**Figure 2**).

Apart from the reverse-transcribing viruses, the origin and diversification of many plant viruses, in contrast to most of the pathogens listed above, appear to be rather recent. Extant viral species seem to be only decades to centuries old and the genera (and sometimes families) to which they belong have diverged since the advent of agriculture in the Holocene (61, 119). One notable exception is the split of the genus *Begomovirus* into two subclades dating between 20 and 30 Mya (91).

A FRAMEWORK TO DEFINE THE PROBABLE FRONTIERS OF HOST RANGE

As described above, the capacity of microorganisms to exploit plants for their resources has been under constant evolution since long before humans roamed the Earth. Likewise, host range—whether it is defined in terms of the plants in which microorganisms multiply or those in which they cause disease symptoms—is also constantly evolving. Although much of this evolution is at scales of time that surpass human lifetimes and therefore is difficult to perceive, the processes driving this evolution are in progress and are continuously influencing microbial diversity. In this light, it is difficult to define host range as a fixed trait of a given genetic line of a microorganism. Nevertheless, the pace of evolution of host range over the scale of decades might be sufficiently restricted to allow us to identify the probable frontiers of the contemporary host range of a pathogen and transform this information into useful tools for disease management.

Xylella fastidiosa: Modern Stronghold on an Ancient Pathogen

The management of diseases due to *X. fastidiosa*, for example, could benefit from a consideration of long-term evolutionary history. *X. fastidiosa* is currently the target of intense surveillance on

national and international levels to protect crops such as olive and grapevine. This species as a whole infects over 560 plant species in more than 260 genera and 80 families of plants, including monocots, dicots, and gymnosperms (41), and in hundreds of these species it does not cause symptoms and seems to live as an endophyte (30). Because of the age of *X. fastidiosa* (**Figure 2**), we propose that its ubiquity is analogous to the ubiquity of AM associations across the plant kingdom that were established early in the history of plant evolution. From its likely early interactions with plants, *X. fastidiosa* probably evolved mostly in genetic and ecological isolation in its hosts, with some cases of recombination, leading to genetic lines with restricted host range (34). As a consequence, the genetic diversity of *X. fastidiosa* has been underestimated, and this can undermine the utility of molecular detection (7). In this light, it is likely that *X. fastidiosa* is present in species of plants well beyond the lists reported at present. Therefore, an efficient approach to assessing its contemporary host-range potential would be to predict which perennial plant species are most likely to suffer from water stress in the foreseeable future. Because of the difficulty in detecting *X. fastidiosa*, this strategy would allow efforts to be concentrated on the most probable candidates for eventual disease symptoms. Symptoms of obstruction of xylem vessels by *X. fastidiosa* are generally observed mainly when the water requirements of the plant are higher than the amount that can flow through the vessels (30). Interestingly, models for the occurrence of *X. fastidiosa* based on observations from Corsica revealed that the best predictor was water stress (severe droughts and high seasonality of precipitation) (102). Hence, research on *X. fastidiosa* host range could focus on the plant species at the most risk—based on previsions of future water deficits in relation to plant biogeography—before signs of new disease appearance. Focusing research in this way could lead to discoveries of a new diversity of the bacterium that will be directly applicable to disease surveillance and also to the development of approaches for avoiding disease emergence where it is possible to mitigate water stress to the plant.

Erwinia Species: A Very Young Pathogen Facing New Opportunities

The contemporary host-range potential of species of *Erwinia* could also be better apprehended from an evolutionary perspective. *Erwinia* is one of the youngest genera of microorganisms that can cause plant disease, having diverged from *Pantoea* less than 1,000 years ago (**Figure 2**). Based on recent time-tree estimates (100), three groups of species within this genus diverged very recently: a group pathogenic on apples, pears, and other Rosaceae (including *Erwinia amylovora* and closely related species); a group of species that are very persistent in the environment, pathogenic on a wide range of plants, and also found in association with aphids [*Erwinia aphidicola* (99), *Erwinia persicina* (99), and *Erwinia rhapontici* (79)]; and a group with members found in association with guava and eucalyptus. In this latter group, *E. psidii* is emerging as an important pathogen of eucalyptus in Brazil (4), a region where this tree is being intensively cultivated outside of its natural range. *E. psidii* was first reported as a pathogen of guava in Brazil and then moved to eucalyptus. This was not due to any apparent specialization of the strains implicated in the epidemics but has involved cross contamination (4). Increasing occurrence and severity of the disease on eucalyptus are reportedly due to the introduction of plant hybrids that are more and more fragile and to the intensification of eucalyptus production (4). Interestingly, the most ancient of the *Pantoea* species (*Pantoea rodasii* and *Pantoea rwandensis*) (100) cause eucalyptus dieback in Columbia and Rwanda (24) where eucalyptus is also being cultivated outside of its natural range.

On the basis of the epidemiological observations for eucalyptus and guava diebacks, we can postulate that numerous species within the myrtle family are likely to be susceptible to bacteria related to *E. psidii* and certain *Pantoea* spp. Disease on eucalyptus is likely to have emerged from reservoirs of the bacteria on indigenous plants in the myrtle family. Guava (*Psidium* spp.) is native

to Mexico and Central and South America (145, 149), and therefore wild and cultivated *Psidium* plants could have positively selected bacteria for fitness on this genus in Brazil and in Columbia. In Africa, *Syzygium* species are the main representatives of the myrtle family with numerous species occurring in wetlands in Rwanda (53) in addition to the intense production of cloves (*Syzygium aromaticum*) in Zanzibar off the mainland of Tanzania. Hence, these plants could have also driven fitness on eucalyptus. *Eucalyptus*, *Psidium*, and *Syzygium* diverged from their most recent common ancestor more than 70 Mya (**http://www.timetree.org**); therefore, it is reasonable to hypothesize that plants in other genera with younger ancestors in common with eucalyptus (149), or those that are closely related phylogenetically within the myrtle family (67), are also potential hosts of *E. psidii* and *Pantoea* spp. Expansion of the natural range of eucalyptus to overlap with plants in two other very closely related families—Vochysiaceae in South and Central America and Heteropyxidaceae in southeastern Africa (145)—might also have exposed eucalyptus to adapted *E. psidii* and *Pantoea* spp. The divergence of species within the myrtle family and its divergence from other families occurred long before the emergence of *Erwinia* and *Pantoea* as genera. Therefore, the notion of coevolution of a pathogen with its plant hosts is not very useful here for defining a set of candidate plants to assess the breadth of host range. It could be more fruitful to examine the ensemble of families in the rosids clade (8) to determine whether the breadth of host range surpasses the myrtle family.

This evolutionary perspective can also contribute to choosing the sets of strains for which host range is explored. In the case of eucalyptus dieback, we can hypothesize that *E. psidii*, *P. rodasii*, *P. rwandensis*, and perhaps some related strains share common traits for fitness on various plants in the myrtle family—and possibly beyond this family. In addition, we note that the other groups of *Erwinia* species that diverged apart from *E. psidii* are found in association with distinctly different plant families such as the Rosaceae for *E. amylovora* or are very versatile in their associations with both plants and insects as indicated above. Therefore, confronting all of this behavioral diversity of bacteria with a wide range of plants in the rosid clade could clearly reveal the frontiers of host range and also define the spectrum of bacterial genetic diversity for which it is pertinent. This confrontation could involve laboratory inoculations but it could also involve prospection for natural infections. This approach is very different from testing strains of a single microbial species on the hosts of isolation and on a few other plants chosen for reasons such as their physical proximity to diseased cultivated fields or their phylogenetic proximity to the species on which the pathogen was isolated. The approach we propose here is likely to open very novel questions about the underlying molecular mechanisms that confer pathogenicity on whole families or orders of plants and will thereby lead to robust, generic markers for detection and surveillance and to targets for breeding resistant or tolerant plant varieties.

A Comprehensive Concept of Host Range for Disease Management and Research on Evolution

After reviewing the eclectic body of literature relevant to the host range of plant pathogens, we cannot advocate for an all-purpose definition or set of criteria to circumscribe the host range of plant pathogens. Instead, we propose that the concept of host range needs a robust framework that is applicable to any particular model. We propose that this framework should specify the time frame considered and include observations from both the field and experimental inoculations and data on the fundamental microbial trait on which natural selection operates, i.e., fitness. Thus, the notion of contemporary host-range potential would comprise not only current pathogenic capacities of a given phylogenetic range of microorganisms but also estimations of a potential host range throughout a certain number of upcoming decades in light of foreseeable land use,

agronomic practices, and climate for that period. This would provide a context for developing tools for surveillance and diagnostics, breeding for resistance, and organizing the spatial and temporal arrangements of crops in mixed cultures and rotations that could be much more prophylactic than what can be achieved by reacting to urgent crises. It would also highlight the importance of considering symptomless hosts and latent or saprophytic phases as drivers of pathogen evolution and provide a framework for evaluating the potential for biological control agents to cause disease in a given context of time and place. Likewise, the notion of host-range evolutionary history would consider the spectrum of behaviors of a microorganism from its divergence as a genus or species to present. This notion of host range could help reveal underestimated motors of change in the interaction of a given phylogenetic group of microorganisms with plants at various taxonomic levels. For example, we might ask what drove the divergence of *Cladosporium fulvum*, considered to be a specialized pathogen of tomato (37), at the time of divergence of the *Solanum* genus but millions of years before the divergence of tomato as a species (**Figure 2**). The informative value of comparative evolutionary history of pathogens and plants could be bolstered by knowledge on the evolutionary history of the molecular mechanisms involved in the plant–pathogen interactions. In this way, the framework we propose for deriving host range would foster identification of host-range barriers at higher categories of plant taxa such as families or orders, and the search for commonalities that could reveal novel mechanisms for resistance to pathogens with broad host ranges.

SUMMARY POINTS

1. Plants are essential partners for a large variety of microorganisms. Colonization of land plants as hosts for microorganisms has occurred since the emergence of the first land plants more than 400 million years ago.
2. There is a large variation in the contemporary host-range breadth of pathogens, ranging from specialists to generalists, and in the frequency of past host jumps.
3. To define host ranges with practical value for disease management, several parameters should be considered, including both the natural and potential host ranges, various phenotypes (e.g., pathogen fitness, virulence, aggressiveness, and transmissibility under natural and experimental conditions), and the appropriate environmental conditions and timescale in which host range is likely to be stable.
4. Various genomic alterations can be responsible for pathogen host jumps at both the plant inter- and intraspecific levels (e.g., hybridization, horizontal gene transfer, deletions, and point mutations). However, there is much less knowledge about jumps between plant species (or higher taxonomic ranks) than those between plant genotypes within a given plant species.
5. Studies of the evolution of pathogenicity and host range of plant pathogens would benefit from setting the time frame relative to the date of the divergence of the pathogen's taxonomic group, the evolutionary history of plants, and Earth's changing climate over archaeological time.
6. At present, there are only a few predictors of the breadth of the host range of a pathogen and the taxonomic groups of the hosts. The phylogenetic distance between plant hosts is a powerful predictor of the probability of host jumps. Shared distribution range between different host taxa is also an important factor.

FUTURE ISSUES

1. Is there always a benefit for pathogens to expand their host ranges? The links between host range and fitness are complex and deserve more attention.
2. Genetic factors involved in host jumps are still largely unknown, from the perspective of the plant and the pathogen as well as at the plant interspecific level and higher ranks. In this regard, experimental acquisition of data on plant–pathogen interaction matrices and subsequent network analyses could help to establish links with possible underlying genetic models and, in turn, mechanisms of host-range evolution.
3. There are still few predictors of a pathogen's potential host range. Establishment of databases of host range from cross-inoculations could help identify intrinsic pathogen traits that determine host range. This would contribute to the understanding of the role of environmental factors, versus contemporary diversification, in apparent host jumps. This could also contribute to understanding how interactions between ecological, evolutionary, and genetic factors determine pathogen generalism or specialism.
4. For human pathogens, there is evidence that generalist pathogens have a higher rate of emergence than specialists. The possibility that this trend exists for plant pathogens should be addressed.
5. There are growing efforts to date the divergence of various taxonomic groups of plant pathogens and to compare this to the evolutionary history of plants. More studies on this theme will contribute to elucidating major drivers of pathogen diversification that could be the basis for novel disease-management tools and approaches.

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