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Ecology and Genomic Insights into Plant-Pathogenic and Plant-Nonpathogenic Endophytes

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Abstract

Plants are colonized on their surfaces and in the rhizosphere and phyllosphere by a multitude of different microorganisms and are inhabited internally by endophytes. Most endophytes act as commensals without any known effect on their plant host, but multiple bacteria and fungi establish a mutualistic relationship with plants, and some act as pathogens. The outcome of these plant-microbe interactions depends on biotic and abiotic environmental factors and on the genotype of the host and the interacting microorganism. In addition, endophytic microbiota and the manifold interactions between members, including pathogens, have a profound influence on the function of the system plant and the development of pathobiomes. In this review, we elaborate on the differences and similarities between nonpathogenic and pathogenic endophytes in terms of host plant response, colonization strategy, and genome content. We furthermore discuss environmental effects and biotic interactions within plant microbiota that influence pathogenesis and the pathobiome.

INTRODUCTION

Plants are colonized by complex microbial communities that play different roles regarding plant growth and health. Whereas some microbial members are detrimental and cause diseases, others promote plant growth and enhance nutrient acquisition and tolerance to biotic and abiotic stresses via a multitude of mechanisms.

A large fraction of the microbial community can be defined as commensal microorganisms that find their niche in the association with plants but have no known function to their hosts. Depending on different plant environments, microbiota associated with plants can be found on the exterior of plants, such as the rhizo- or the phyllosphere, or in the interior of plants, such as the endosphere. Plants may be colonized by pathogens and nonpathogenic endophytes, which spend at least part of their life cycle inside plants.

In the literature, the term endophyte most commonly refers to microorganisms that can be isolated from surface-disinfected plant tissues without visible disease symptoms (48). However, it has been recently discussed whether endophytes can be defined at a functional level (49). In this review, we adopt the concept of Hardoim et al. (49), who suggested that endophytes be defined solely by their colonization niche but not by their function. Therefore, there are both pathogenic and nonpathogenic endophytes. This is based on the following considerations. First, most isolated endophytes are tested on a single or a few plant species, and even if they show no deleterious effects on these plants, they may exhibit pathogenicity on other plant hosts. Furthermore, pathogenicity depends on a number of environmental parameters and biotic interactions. For instance, fluorescent pseudomonads, frequently showing plant-beneficial effects, can cause disease on leatherleaf ferns under specific conditions (61). In addition, with the advent of molecular tools to investigate microbiomes without the need of cultivation leading to a number of uncharacterized taxa in an environment, functions such as pathogenicity or mutualism can rarely be predicted. The advent of molecular tools enables the investigation of microbiomes without the need of cultivation, which leads to the identification of a number of uncharacterized endophytic microorganisms. Their functions, such as pathogenicity or mutualism, can rarely be predicted.

Pathogenicity is a complex phenomenon. The combination of many factors, such as pathogen and host genotypes and abiotic and other environmental stresses, as well as microbial interactions, determines the outcome of the reaction of a plant to a (pathogenic) microbe. Furthermore, a single microbial species may comprise strains that are capable of exhibiting pathogenicity, mutualism, or no effect on their hosts (74, 117). For instance, *Fusarium oxysporum* is well known for its phytopathogenic properties. Collectively, strains within the *F. oxysporum* species complex can cause diseases on more than 100 plant species and exhibit strong host-specificity (90). However, most isolates are nonpathogenic toward nonhosts, and for some isolates even biocontrol properties have been characterized (2). Belonging to the same species, these pathogenic and nonpathogenic microorganisms share the majority of their genomes. Therefore, it is not surprising if plants use similar immune responses and defense mechanisms to interact with both pathogenic and nonpathogenic strains. However, the distinct interactions dictate the outcome.

In this review, we address several aspects that determine the ecology and functioning of an endophyte that ultimately lead to pathogenicity or mutualism. In particular, we elaborate on how the microbial environment may influence the function of an endophyte and elucidate the differences between pathogenic and nonpathogenic endophytes, frequently mutualists, in regard to plant response, pathogen colonization behavior, and genetic variation. Overall, we postulate that plant-colonizing microorganisms, irrespective of their function as a pathogen or nonpathogen, employ shared mechanisms to interact with their plant hosts, and that pathogenicity is a consequence of fine-tuned interactions between host, environment, and other organisms.

Commensal microorganism: organism living on exudates and nutrients provided by the plant host without having a negative effect on the host

Microbiota: all microorganisms sharing a given environment

Phyllosphere: the leaf surfaces colonized by microorganisms

Endophytes: microorganisms (archaea, bacteria, fungi, oomycetes, protista) inhabiting the interior of plants (endosphere) irrespective of the function in association with the plant

Microbiome: genomes of all microorganisms sharing a given environment

Mutualism: mutual beneficial relationship between host and microorganism

THE DISEASE TRIANGLE CONCEPT VERSUS THE DISEASE TETRAHEDRON

Classical plant pathology textbooks have told us that several prerequisites are necessary for the occurrence of a plant disease, including a virulent microbial pathogen (containing virulence/pathogenicity factors), a susceptible plant host (host factor), and environmental factors in favor of disease development, such as humidity and temperature (34). If any of these factors is missing or less than optimal, disease severity in a population is reduced. Since the 1960s, this paradigm has been often illustrated as a disease triangle (1) (**Figure 1*a***).

The disease triangle can be used to indicate severity or likelihood of disease. Each side of the triangle is hereby proportional to the factors favoring disease, and, consequently, the area of the triangle illustrates the sum of factors in favor of plant disease (1) (**Figure 1*a***). **Figure 1*b*** illustrates that host factors, either (e.g., resistant or tolerant) genotypes and other genetic variance, the density of host populations, or the host age, could minimize the potential for disease development. In addition, certain environmental conditions favor disease development, including optimal humidity/temperature and their timely changes at different stages of infection, topographic exposure, wind, etc. All factors determine the shape of the triangle sides, illustrating increased severity

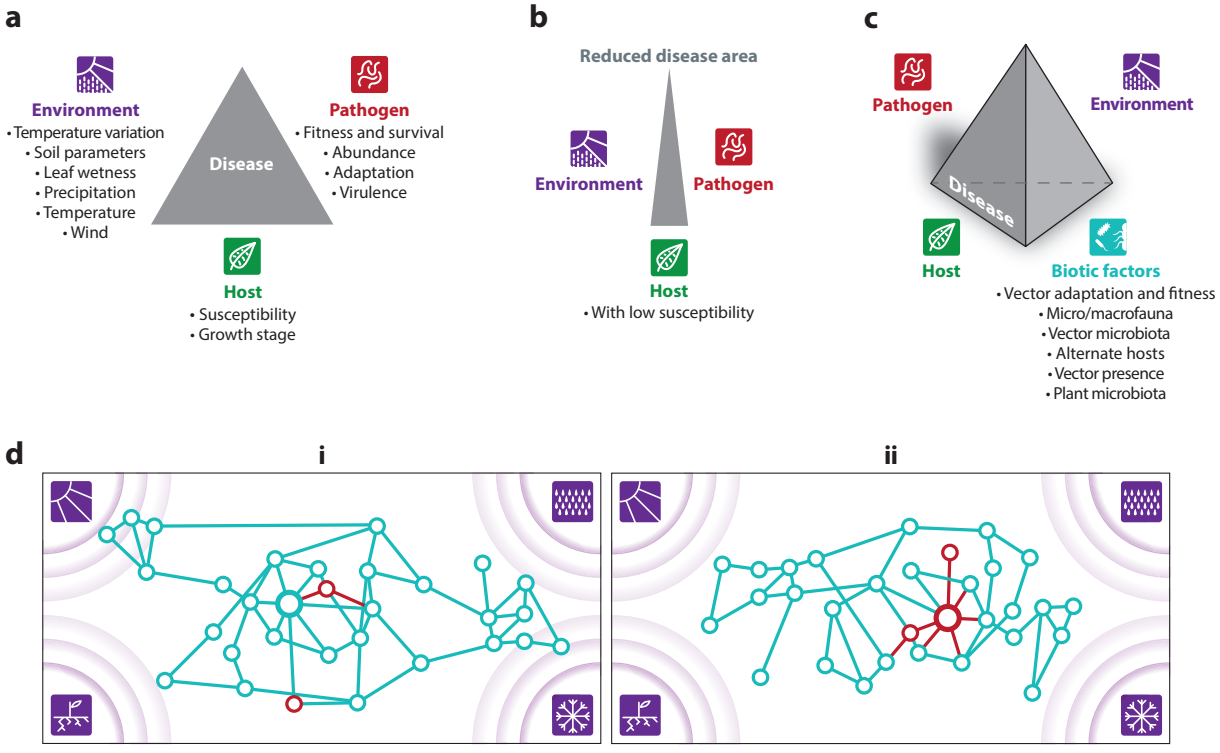

Figure 1

Factors for the manifestation of a plant disease. (*a*) The classical disease triangle. (*b*) A modified disease triangle that illustrates limited amounts of disease in a less susceptible or resistant host. (*c*) A disease tetrahedron that considers the biotic factor separately. According to the disease tetrahedron, the development of a plant disease could be inhibited or modulated by, or may even depend on, a transmitting vector (animal), an alternate host (plant or animal), and other plant-associated microbes. (*d*) Network of microbiome interactions that either (*i*) suppress or (*ii*) favor pathogen establishment and performance. Environmental factors, such as (*clockwise from top left*) radiation, precipitation, temperature, and soil parameters may affect the microbiome directly or indirectly via the plant host.

and likelihood of disease. Additional parameters such as the human factor, disease vectors, and time have been added to the disease triangle (1, 34).

The recognition that plants are colonized by a large number of microorganisms, primarily commensals or mutualists (49, 107), and the observation that certain diseases might be caused or altered by cooperation/coinfection of different pathogens (e.g., 64) lead to the postulation of the pathobiome, in which the pathogen is described as an integrated component of its biotic environment (135). In addition, the microbiome plays a prominent role in plant health and pathogen suppression (10). Taking these considerations into account and considering the natural complexity of biotic interactions, we propose a disease tetrahedron that implements biotic factors as the fourth dimension to illustrate the interplay among various disease-determining factors (**Figure 1*c***). These additional factors include vectors transmitting pathogens and especially microbiota modulating pathogenesis and plant defense.

Pathobiome: a pathogen, its surrounding microbial community, and their interactions leading to plant disease

Disease tetrahedron: analogous to the disease triangle, in which the area symbolizes likelihood of disease, the disease tetrahedron is a visualization of the components' (abiotic) environment, host, pathogen, and additional biotic factors and their interactions responsible for plant disease development

Interaction Between Factors Responsible for Disease Development

Disease development is influenced and modulated by all interconnected faces of the disease tetrahedron. Environmental factors affect not only the host plants but also the pathogen and biotic factors. Similarly, biotic factors directly influence plant performance, pathogens, and other biotic factors, including vectors and microbiota.

Influence of the environment. Environmental factors have been long recognized as key components influencing the development of plant diseases (60). Aerial environmental factors include temperature, precipitation (i.e., dew, rainfall, and snow) intensity and duration, wind, and aerial pollution (including CO_2 and ozone content). Soil environment parameters include soil organic matter, pH, nutrient content, and content of toxic components (e.g., metals, salt, and pesticides). Intuitively, plants growing under suboptimal or stress conditions are prone to disease. Indeed, factors such as light quality and quantity required for plant growth are essential for plant immunity (52). However, optimal growth conditions are not feasible in most environments; therefore, plants are exposed to a number of challenges and have to cope with combinations of abiotic stresses. Responding to a single abiotic stress or combined abiotic stresses, plants could activate sophisticated response cascades leading to the induction of plant defense. Molecular responses to multiple abiotic stresses have been only recently systematically investigated (40).

Signaling pathways responding to abiotic and biotic stresses are highly sophisticated and interconnected. If an abiotic stress signaling pathway overlaps with host defense against a pathogen, priming such a response by applying abiotic stress may provide some measure of protection against pathogen invasion (123). This was documented by a study showing the influence of abiotic factors on attacks by phloem-feeding aphids (33). This phenomenon not only illustrates the tremendous effects of abiotic factors on host plants but also increases the complexity of plant-microbe interactions, as plant-feeding animals may act as vectors of both pathogens and commensal endophytes.

Environmental and soil parameters have been shown to affect pathogen resistance and tolerance. Ozone and UV stress have resulted in enhanced plant tolerance or resistance to subsequent pathogen attack, a phenomenon referred to as cross-tolerance (16). Signal pathways used by plants to cope with temperature stress also overlap with pathways involved in the expression of disease-resistance proteins and RNA silencing and defense mechanisms against fungi, nematodes, and viruses (52). A metastudy suggested that N fertilization leads to an increased plant susceptibility to various fungi and oomycetes (137). Also, potassium deficiency increased plant defense and enhanced the entry and development of pathogens (5).

As the environment influences host responses with consequences in plant defense, it affects the potential of a pathogen to cause disease. If conditions for pathogen development are not

appropriate, disease is reduced. For instance, wheat leaf rusts and powdery mildew infections are more severe in moist Australian coastal areas compared to drier parts of the country (60). Humidity plays an important role in infection processes. Various factors, such as precipitation characteristics, wetness of leaves, soil moisture, and microclimatic conditions, must be considered. The observations that environmental conditions play a key role in the development of pathogen pressure have led to various disease prediction models (41). The predictive power of such models must be continuously improved to avoid the costs of overseen disease outbreaks and to allow acceptance of agricultural management (41).

Environmental conditions also influence biotic environments and the microbiome in general and, therefore, disease development. For instance, certain phytoplasma pathogens depend on the climatic requirements of specific vectors for their disease transmission (140). Local climatic conditions that prevent the survival of these vectors also impact the disease outcome.

The most important biotic environment directly influenced by environmental conditions is probably the microbiota, i.e., the ecological community containing all endophytes and microbes in the rhizosphere, phyllosphere, and other plant habitats. For instance, high nitrate levels suppress nodule formation in legumes and, consequently, inhibit nitrogen fixation by rhizobia (86). Soil parameters such as phosphate and nitrogen also influence arbuscular mycorrhizal development (15), and these effects have, in turn, strong implications on plant performance.

As plant microbiota are clearly shaped by environmental conditions (49, 97), the question is how climate changes, such as altered temperature, humidity, and CO_2 levels (29, 42), affect microbiota and, consequently, plant health. For instance, endophytic fungal communities of the Hawaiian tree *Metrosideros polymorpha* are shaped by environmental factors such as precipitation and temperature (146).

As an interconnected system, the impact of environmental factors on plants results in plant physiological changes, which could affect their associated microorganisms. For instance, the combination of environmental factors and the age and genotype of the host plant *Boechera stricta* shapes the endophytic community that colonizes plant leaves (139). Similarly, plant microbiota such as endophytes also influence the environmental stress tolerance of plants (49). Taking into account the complexity of all possible interactions and the fact that each environmental factor may contribute differently to plant host performance, pathogen pressure, and biotic environment, more complexity is to be expected with changing environmental parameters due to climate change and long-distance travel.

Multitrophic interactions. Pathogens, as with any microorganism occurring in natural ecosystems, are embedded in complex microbial communities that interact with each other as well as with their host or other higher organisms. Although we understand some of these interactions in great detail, such as those of specific phytopathogens or plant symbionts, we are at the very beginning of understanding multilateral interactions, particularly microbial community interactions. Increasing research on signaling and communication between microorganisms has shed light on this area.

One well-known example for intercell communication is quorum sensing (QS), a cell density–dependent microbial communication system that modulates gene expression (36, 141) and is involved in environmental adaptation via modulation of virulence determinants in pathogens and beneficial interactions with plants as well as biofilm formation (6, 141). Generally, freely diffusible chemicals, such as *N*-acylhomoserine lactones (AHLs), produced by many Gram-negative bacteria, act as signaling molecules (36). Other QS molecules include long-chain fatty acids and methyl esters [diffusible signal factors (DSF)], furanones, peptides, and 2-alkyl-4-quinolones (66, 141). QS molecules not only represent a language spoken by the producing microorganisms but also influence communication with other strains or taxa. Consequently, the virulence of a pathogen is

Rhizosphere: the zone close to the surface of the root system, which is inhabited by a large number of microorganisms and influenced by the roots (and root exudates)

Quorum sensing (QS): population density–dependent regulatory mechanism based on diffusible compounds regulating gene expression according to cell density

not only regulated in a cell density–dependent manner by its own population but could well be modulated by other community members speaking a similar chemical language. Some microbes may also degrade QS molecules, a phenomenon called quorum quenching, or can produce compounds influencing the synthesis or the perception of QS compounds (66). A range of different bacteria (129) and fungi (130) produce AHL-modifying or -degrading enzymes, including lactonases and different types of hydrolases, which all have the potential to modulate pathogenic or beneficial functions of nearby microbes. Modulating and quenching properties have also been shown to influence other QS signaling compounds such as DSF of *Xylella fastidiosa* but are less well investigated (66) and maybe more common than we currently know.

Type III/VI secretion system (T3SS/T6SS): consists of distinct protein composition and is responsible for injection of effectors and toxins into host cells

There is increasing evidence that volatiles can act as infochemicals mediating microbe-microbe and microbe-host interactions. To date, approximately 1,000 volatiles produced by a wide range of bacteria and fungi have been described (112). As bacteria often live in close association with fungi or are jointly associated with plants, volatiles produced by community fellows may affect the survival as well as pathogenicity of co-occurring pathogens. Indeed, some bacterial volatiles have antimicrobial activities by altering fungal gene expression, changing pathogen activities and resulting in modifications in morphology (39, 82). For instance, Minerdi et al. (82) reported that bacteria associated with hyphae of *F. oxysporum* produced the volatile sesquiterpene caryophyllene, which repressed the expression of virulence genes. Also, the oomycete pathogen *Phytophthora infestans* was reported to be sensitive against the bacterial volatiles hydrogen cyanide and 1-undecen (53). We have just started to understand the functional role of microbial volatiles and their influence on the outcome of the disease triangle or tetrahedron. In fact, microbial communities produce a complex mix of volatile compounds (39). It has to be taken into consideration that volatiles travel faster and over a longer distance than diffusible compounds (32).

Oxylipins are other important signaling molecules used by mammals, plants, and fungi and are able to facilitate cross-kingdom communication. Plant-derived oxylipins (e.g., jasmonates) regulate plant defense against fungal attack. The importance of endogenous oxylipins in fungal QS modulating sporulation as well as mycotoxin production was also reported (18). Fungi may also exploit host oxylipins to colonize a host, reproduce, and synthesize toxins to facilitate their own virulence and pathogenic development (23). As oxylipins represent a rather universal eukaryotic chemical language, it may be that such signals also derive from neighboring fungi modulating mycotoxin production or pathogenicity. For instance, it was reported that the fungal endophyte *Paraconiothyrium variabile* antagonized the phytopathogen *F. oxysporum* but did so only when both microorganisms acted together (24). The pathogen *F. oxysporum* induced the production of an oxylipin, which led to the negative modulation of beauvericin, a highly potent toxin of *F. oxysporum*. In addition to modulating the synthesis of toxins, plant-associated fungi and bacteria are also known to degrade toxins (e.g., 80). The alteration of the oxylipin signaling of plants is exploited by *Pseudomonas syringae* pv. *tomato* to produce the jasmonate analog coronatine, which acts as a toxin and virulence factor by suppressing plant defense (13). Modification and manipulation of oxylipin signaling of eukaryotes by bacteria might be much more common phenomena than currently believed.

Community interactions may also play an important role in the development of emerging pathogens through the acquisition of new DNA fragments from other organisms (9). Such a horizontal gene transfer (HGT) is driven by a donor and recipient colonizing the same niche, facilitating niche colonization by new microbial species (120). HGT processes may involve the acquisition of plasmids, bacteriophages, and pathogenicity islands, leading to the development of microorganisms with altered pathogenicity as well as to the emergence of new pathogens (9, 56). There is evidence that in phytopathogenic bacteria *hrp/hrc* genes encoding the central core of the type III secretion system (T3SS) have been subject to HGT (7, 9). Also, genome comparison of

Xanthomonas genomes revealed that pathogenicity genes and genes related to the suppression of host defense have been acquired by HGT (69). In *Pectobacterium atrosepticum*, it was further shown that in planta colonization favored transfer of one of the integrative and conjugative elements, which play a central role in HGT and may lead to the acquisition or loss of virulence genes (134). Genome expansion through HGT was suggested to have contributed to the recent evolution of *Erwinia tracheiphila*, a cucurbit bacterial wilt pathogen (115). The *Fusarium* comparative genomics revealed the phenomenon of the horizontal transfer of supernumerary chromosomes (SP) that convey host-specific pathogenicity in the *F. oxysporum* genome (74). Other studies also suggested that HGT between fungi is widespread and has played an important role in the evolution of fungal pathogens (3, 72, 105). Additionally, endofungal bacteria that live as fungal intracellular symbionts not only influence the function of their fungal hosts, they also facilitate transkingdom gene transfer (127).

Fungi, as well as bacteria, are well known to produce a range of antimicrobial substances that may directly antagonize or weaken pathogens as well as other microorganisms. In this respect, the interplay between microorganisms is an important element in the disease tetrahedron. Bacteria with the capacity to modulate the synthesis of fungal secondary metabolites (113), to contribute directly to fungal pathogenicity (110), or to alter fungal metabolic potential (17) have all been observed. These findings and the fact that endofungal bacteria, such as the endobacterium *Burkholderia* sp. of *Rhizopus microsporus*, are required for host sporulation and toxin production (93) reinforce the concept that bacterial-fungal interactions are important components in plant disease development. Such interactions may also influence beneficial functions of plant-associated microorganisms (14, 44). Similarly, fungal-fungal interactions may have an impact on plant disease. For instance, sesquiterpenoids and polyketides produced by *Trichoderma arundinaceum* regulate the expression of virulence factors of the pathogen *Botrytis cinerea* (75). Likewise, viruses may influence the functionality of plant microbiota, e.g., by determining the thermal tolerance of a tripartite system comprising plants and fungal endophytes infected with a virus (78).

Interactions between microbial community members involve not only substances and compounds with signaling activities but also microbial metabolites utilized by other community members. This cross feeding is especially important for a number of cofactors, but highly specialized interactions might lead to the development of a syntrophic metabolism, in which two partners are needed to establish an energetic positive metabolism (114). Nitrogen availability in plant microbiota is severely influenced by nitrogen fixation and nodulation, and, consequently, quantitative changes of biologically fixed nitrogen also influence community composition (147). Within plant microbiota, limitation of resources (i.e., competition for certain nutrients) also plays a role in microbe-microbe interactions (114). A good example demonstrating the potential involvement of microbiota in temporal pathogenicity is grapevine trunk disease (GTD). Pathogenic fungi are responsible for the necrosis associated with GTD such as esca disease, involving, primarily, *Phaeomoniella chlamydospora*, *Phaeoacremonium minimum*, and *Fomitiporia mediterranea*, but also *Stereum hirsutum* and *Botryosphariaceae* spp., which are often isolated together from necrotic tissues. However, these fungi also frequently occur in healthy, asymptomatic plants (51) and often do not induce disease symptoms after inoculation (122). Despite the similarity in fungal communities (19), microbiome analyses revealed that healthy versus diseased grapevine plants are associated with distinct bacterial assemblages (20), which might alter plant physiology and may contribute to disease development. Some specific microorganisms such as bacterial taxa are present within the same niches as pathogens and might influence pathogen infection in a positive or negative way (45). **Figure 2** summarizes the interactions between beneficial, commensal, and pathogenic microorganisms and their host plant.

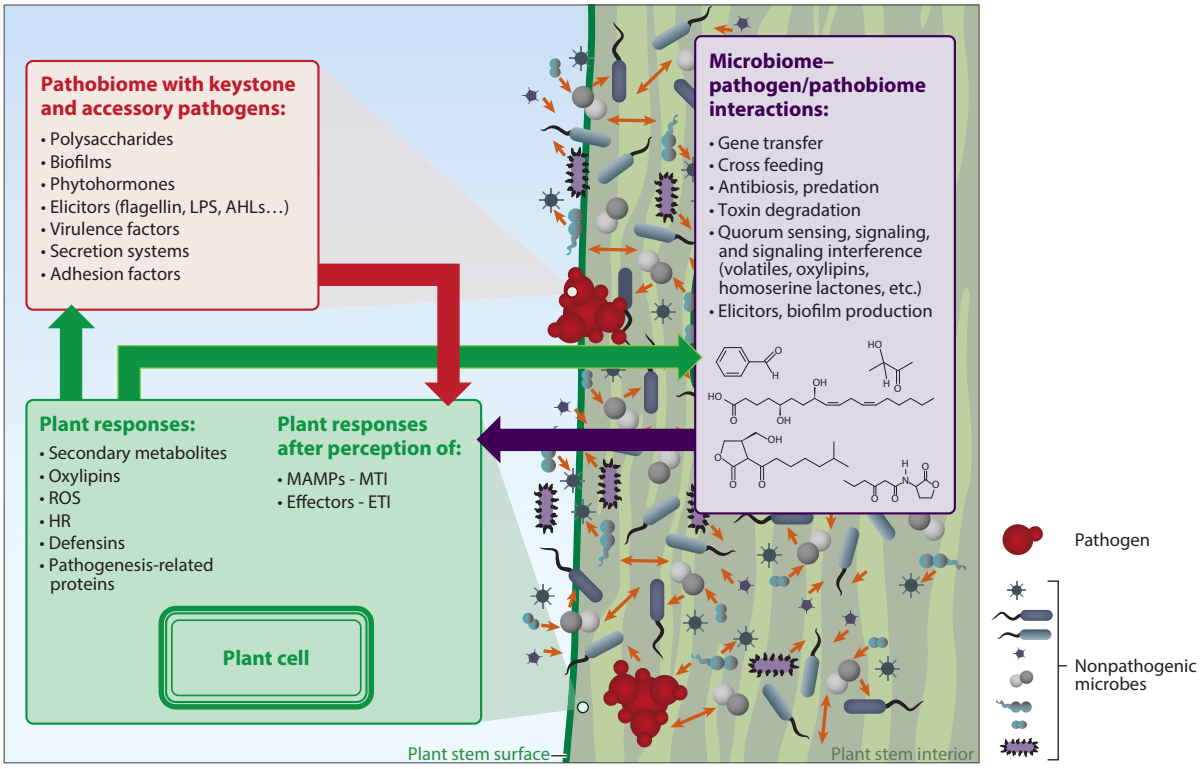

Figure 2

Interactions between pathogen(s), the plant microbiome and the plant. Pathogens produce virulence factors mediated via secretion systems and adhesions to the plant to suppress defense (*red arrows and blocks*). Endophytes may produce toxins and antibiotics, regulate their behavior with quorum sensing molecules, and produce a number of phytohormones, biofilms, polysaccharides, elicitors of plant defense, and metabolic products. All endophytic organisms interact with each other (*arrows*) and can use, degrade, and interfere with the products and the signaling of the others or even directly act on other organisms. An ever-changing system of exchange of genetic information and horizontal gene transfer shapes further the microbial environment. Abbreviations: AHLs, acylhomoserine lactones; ETI, effector-triggered immunity; HR, hypersensitive response; LPS, lipopolysaccharide; MAMPs, microbe-associated molecular patterns; MTI, MAMP-triggered immunity; ROS, reactive oxygen species.

RESPONSE OF PLANTS TO PATHOGENS AND NONPATHOGENS

Our understanding of plant immunity has advanced tremendously in the past few decades (30, 59). However, information on plant responses to nonpathogenic microorganisms is still sparse. Generally, plants have a two-layer innate immune system essential for defense against invading microorganisms. The first layer includes plant surface–localized pattern recognition receptors (PRRs), typically plasma membrane–localized receptor-like kinases (RLKs) or receptor-like proteins (128), which recognize microbe- (or pathogen-) associated molecular patterns (MAMPs/PAMPs) of invading microorganisms and initiate MAMP-triggered immunity (MTI). Typically, MAMPs perform essential functions and are conserved in microbes regardless of their pathogenicity (88). Known MAMPs include flagellin, elongation factor Tu (EF-Tu), peptidoglycan (PGN), lipopolysaccharides (LPSs), fungal chitin, and β-glucans from oomycetes.

MAMP/PAMP: microbe/pathogen -associated molecular pattern

MTI: MAMP-triggered immunity

One of the best-studied MTI mechanisms is based on the interaction between the flagellin epitope (flg22) and the plant receptor FLS2. FLS2 can recognize bacterial flagellin and

initiate plant defense. For instance, the recognition of flagellin from *P. syringae* in *Arabidopsis* and in *Nicotiana benthamiana* triggered stomata closure (54) and MAP kinase activation. This leads to transcriptional induction of pathogen-responsive genes, production of reactive oxygen species, and deposition of callose to reinforce the cell wall at infection sites and to prevent microbial growth. Variable responses are observed between beneficial microbes and their plant hosts. In the interaction between *Arabidopsis* and a plant-beneficial *Bacillus subtilis* strain, a similar type of response was observed (63). Flagellin of the mutualist endophyte *Paraburkholderia phytofirmans* PsJN triggered a weak and transient defense reaction with an oxidative burst but to a lower extent compared to pathogenic interactions (128). Several plant growth–promoting *Pseudomonas* strains, such as the WCS strains deposited in the Willie Commelin Scholten Phytopathological Laboratory, have been investigated in detail regarding their beneficial functions and plant response (reviewed by 11). *Pseudomonas fluorescens* strain WCS417 actually suppressed the local root immune response in *Arabidopsis* typically triggered by flg22 (81). The alkaline phosphatase AprA of *Pseudomonas aeruginosa* and *P. syringae* was suggested to repress flg22-triggered immunity (11) and is able to degrade flagellin monomers, therefore preventing the recognition by the receptor FLS2 (96). In the symbiotic interaction between *Lotus japonicus* and *Sinorhizobium meliloti*, a Gram-negative N_2-fixing bacterium, the defense reaction after the recognition of the flg22 resulted in a nodulation delay. However, once the symbiosis was established, the formation of new nodules was not inhibited, and the expression of the FLS2 receptor was down-regulated in nodular tissue (73). In general, plant immunity responses seem to be crucial for the rhizobia-legume symbiosis, and plants use their defense system to build up and maintain the balance (43).

In addition to flagellin, other MAMPs and effector proteins are known to be involved in plant-microbe interactions. LPSs, which are basic components of the outer membrane of Gram-negative bacteria, are also able to activate the host immune response (145). But LPSs from pathogens and nonpathogens may induce different host responses. For instance, LPSs from *Sinorhizobium meliloti* can suppress host defense via an oxidative burst in its symbiotic host, *Medicago truncatula.* However, the same molecules elicit an oxidative burst on nonhost plants (125). This suggests that a sophisticated LPS perception system has evolved in legumes in order to establish symbiotic interactions (143). Infiltrating potato leaves with cells of the plant-beneficial strain *P. phytofirmans* PsJN and its LPSs downregulated defense genes, such as defense-like *PR1*, superoxide dismutase, and the COP9 signalosome complex, indicates that plants recognize LPSs derived from nonpathogenic endophytes (F. Trognitz & A. Sessitsch, unpublished data). Using a nonpathogenic fungus to probe host immunity has also been reported among fungal species. For instance, the nonpathogenic *F. oxysporum* strain Fo47 was reported to induce overexpression of plant defense genes prior to and after pathogen challenge in both pepper and tomato hosts (2, 136). Similar findings were obtained with the nonpathogenic *F. oxysporum* strain CS-20 (35). However, primed defense is sometimes associated with fitness cost. For instance, Lara-Chavez et al. (65) compared gene expression in switchgrass genotypes, which respond differently to strain PsJN in terms of plant growth promotion and found higher defense gene activation in the nonresponsive genotype. The strong induction of defense-related genes may be a trade-off for growth promotion. Similarly, in a generally beneficial interaction between *Gluconacetobacter diazotrophicus* and *Arabidopsis* roots, a growth inhibition associated with salicylic acid (SA)-mediated plant defense was observed, least, at the initial growth stages (106). In the interaction between *Arabidopsis* leaves and a plant growth–promoting bacterium, *Bacillus cereus* strain AR156, genes involved in several defense pathways, such as salicylate- and jasmonate/ethylene-dependent signaling pathways, were upregulated (89). This indicates that beneficial microorganisms can manipulate host immunity to establish a successful relationship with the host. Some sophisticated strategies may be used by both pathogenic and beneficial microbes to overcome the host immunity (95).

ETI: effector-triggered immunity

It was documented that both pathogenic and nonpathogenic microorganisms produce effectors to modify the structures and functions of host defense elements to overcome plant MTI (102). Microbial effectors include small secreted proteins and some secondary metabolites. Collectively, they protect the microbe, suppress the host immunity, and alter host cell physiology. However, the functions of most secreted proteins remain to be elucidated (102).

In the second layer of defense, plants recognize microbial effectors, leading to so-called effector-triggered immunity (ETI) (59). Plant ETI is either directly mediated by resistance proteins or indirectly mediated involving accessory proteins (59). Effectors also play important roles in symbioses such as between plants and arbuscular mycorrhizal fungi (AMF). For instance, the SP7 effector of the AMF *Glomus intraradices* interacts with the *M. truncatula* ERF19 transcription factor to regulate the expression of several defense-related genes (62). Constitutive expression of SP7 leads to a higher degree of mycorrhization and reduced defense responses. In contrast, the overexpression of the target of SP7, MtERF19, significantly impairs root colonization by the fungus (62). Similarly, the MiSSP7 effector of the ectomycorrhizal fungus *Laccaria bicolor*, the MiSSP7 effector is essential for the establishment of mutualism in its host poplar plants (101).

Bacterial pathogens transfer multiple effectors into eukaryotic cells. Frequently it is the combination of several effector proteins that exhibit a specific function (37). To effectively transfer effectors into the plant cytosol, bacteria have evolved complex delivery machineries, primarily the T3SS and type VI secretion system (T6SS) (37). Rarely but surely, these secretion systems are also found in a number of bacterial mutualists. For instance, rhizobia contain core T3SS components, and diversification in some T3SS proteins suggests adaptations to serve specific interactions between rhizobia and legumes (124).

Plant-microbe interactions lead to physiological changes in the host plant. These changes are likely to result in altered root exudation, which impacts the composition and functioning of associated microbiota as well as microbial interactions. Overall, we believe the underlying mechanisms that facilitate mutualistic and pathogenic interactions are related. Careful investigation is needed to further dissect modulating elements that lead to different outcomes.

COLONIZATION STRATEGIES OF PATHOGENS AND NONPATHOGENIC ENDOPHYTES

Microorganisms usually colonize plant surfaces before entering the plant. From their entry points, microorganisms may systemically colonize plants from roots to shoots, shoots to flowers or fruits and/or from flowers to fruits and seeds, and they may also cause localized colonization inside/outside plant organs (25, 49) (**Figure 3**). For both pathogenic and nonpathogenic endophytes, commonalities and differences exist in their colonization routes. Some bacteria, such as *P. syringae*, use multiple colonization strategies. For instance, *P. syringae* pv. *syringae* strain B728a colonizes and maintains large populations on healthy plants before invading plant tissues, whereas *P. syringae* pv. *tomato* strain DC3000 reaches stomata and invades via wounds after colonizing the phyllosphere and overcoming plant defense reactions (50). On pear, *P. syringae* pv. *syringae* multiplied on the leaf surface, particularly on trichomes and in depressions of the cuticular layer, before entering leaf tissues through the trichome base and fissures in the cuticular cell layer (76). Furthermore, this pathogen may also colonize distant sites such as xylem vessels and the plant apoplast, if aided by the virulence factor syringolin A, which suppresses acquired resistance in adjacent tissues by blocking SA signaling (83). Other colonization strategies used by *P. syringae* include colonization of xylem and phloem of fruits (38) or seed to seedling colonization (144), whereas *P. syringae* pv. *coriandricola*, which causes bacterial blight of coriander, is systemically transmitted (126).

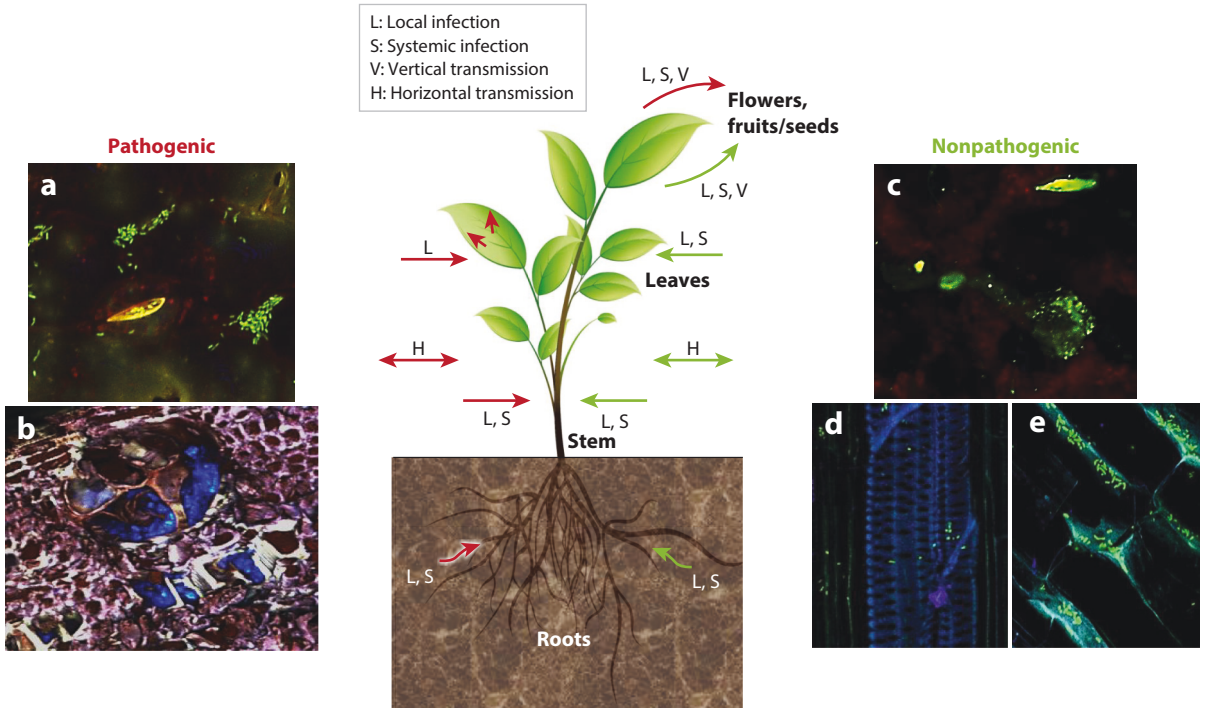

Figure 3

Diagram showing the colonization routes of pathogenic and nonpathogenic microorganisms. Red arrows depict colonization of pathogenic microorganisms, and green arrows depict colonization of nonpathogenic microorganisms. Local infection (L): colonization via stomata and hydathodes; root colonization; xylem invasion; phloem infection. Systemic infection (S): colonization of whole plant; vertical transmission. Vertical transmission (V): spread of microorganism to the next plant generation via reproductive organs. Horizontal transmission (H): spread of microorganisms by vectors or air from one plant to another. Different infection examples are shown in the surrounding micrographs. (*a*) *Pseudomonas syringae* DC3000 colonizing stomata of tomato (Syto9 staining; *green fluorescent*). (*b*) Grapevine trunk pathogen in trunk xylem vessels (stained with WGA-Alexa 350; *blue fluorescent*). (*c*) *Pseudomonas viridiflava* colonizing stomata of tomato (Syto9 staining; *green fluorescent*). (*d*,*e*) *Paraburkholderia phytofirmans* strain PsJN (*gfp* marked strain) colonizing root xylem vessels and intercellular spaces of root cortical cells of a maize seedling. Center plant illustration adapted with permission of Springer (from Reference 28; © Springer International Publishing, Switzerland 2016).

Xanthomonas campestris pv. *malvacearum* was found within the buds of field-grown cotton and further transmitted to seeds (132). Similarly, *Xanthomonas fuscans* subsp. *fuscans* is transmitted via seeds (31), and *X. campestris* pv. *campestris* colonizes multiple sites, including leaves, flowers, and seeds (133). However, *Xanthomonas citri* pv. *citri*, which usually causes local infection of leaves, stems, and fruits, enters mainly by stomata and wounds and does not systemically spread (109). Another bacterial pathogen, *Ralstonia solanacearum*, invades root elongation zones and axils of emerging or developed lateral roots or intact root tips (4). Furthermore, it can enter roots through physical wounds, colonize the cortical cell layer, and then reach the central cylinder, where it uses xylem vessels to spread to the upper parts and clogged vessels (4).

Pathogenic microorganisms that do not colonize plants directly may enter the plants with the help of vectors. One example is illustrated in the case of *X. fastidiosa*, which is transmitted via xylem-feeding insects such as sharpshooters and spittlebugs. In this case, infected plants exhibit foliar symptoms, and disease may lead to occlusion of xylem vessels by tyloses or colonization by

bacterial cells. Bacteria-diffusible signaling factors such as *Xf* DSF are used to further stimulate pathogen adhesion and to increase cell aggregation, surface attachment, and biofilm formation (21). Most bacterial pathogens colonize substomatal chambers and the xylem, whereas only specific pathogens are able to colonize the phloem. The phloem-colonizing bacteria include rickettsias, spiroplasmas, and phytoplasmas introduced by phloem-feeding insects, or via cultural practices like grafting (142).

Nonpathogenic endophytes have been reported to colonize additional niches, but are mostly found in lower cell numbers than their pathogenic counterparts (25). For example, the plant-beneficial *P. phytofirmans* strain PsJN is known to colonize root surfaces, entering from the exodermis and traveling to the cortical cell layer before passing the endodermis barrier. A subpopulation reaches the central cylinder and uses xylem vessels to spread throughout the whole plant (26, 27). When inoculated onto maize flowers, this strain could be systemically transmitted to the next generation of seeds (85). *Candidatus* Burkholderia kirkii, an endosymbiont of the plant *Psychotria kirkii*, is known to be seed transmitted but may be also horizontally transferred from the environment (67). Other nonpathogenic bacteria are known to reach upper plant parts using intercellular spaces (26). Another plant growth–promoting strain, *Herbaspirillum seropedicae* Z67, showed similar colonization routes in rice as the strain PsJN (57). Some nonpathogenic strains only colonize locally. For instance, a *P. fluorescens* strain was observed only in roots of olive plants, colonizing from the root surface and root hairs to the root cortical cell layer (104). Rhizobial nodule symbionts represent nonpathogenic microbes, which are primarily encountered in nodule tissues (87).

Irrespective of the type of interaction, both pathogenic and nonpathogenic bacteria use plant-derived nutrients, albeit resulting in commensal, mutualistic, or parasitic relationships. Fungi and oomycetes use similar niches and common colonization strategies. The ascomycete *Colletotrichum graminicola* causes anthracnose disease of maize and stalk rot diseases and devastates foliar leaves after phyllosphere colonization. The same fungus may colonize maize roots asymptomatically, even after systemic colonization (121). Members of the Clavicipitaceae family can establish symbiosis with different hosts and colonize the entire host plant systemically. They proliferate within the apical shoot meristem, colonize intercellular spaces of the new shoots and leaves, and are vertically transmitted via seeds (22). Some *Neotyphodium* and *Epichloë* species, known for their plant growth–promoting activities, may also be transmitted horizontally. Another plant-beneficial fungus, *Piriformospora indica*, colonizes the root zone and stops in the central cylinder. In the case of vascular pathogens of grapevine trunk diseases, some are soil-derived, whereas others are transmitted by pruning, and infect piths, fibers, and xylem vessels before being blocked by tyloses, provoking one of the mechanisms leading to disease (12). All these studies show that colonization behavior does not necessarily depend on the pathogenicity or function of a particular strain. However, the number of bacterial or fungal cells colonizing usually differs between nonpathogenic and pathogenic strains, the latter frequently colonizing in far higher cell densities (47) (**Figure 4**). On one hand, high concentrations of nonpathogenic microorganisms may have harmful effects on their host, as seen under artificial laboratory conditions even when these effects have not been observed in the field. On the other hand, low cell density of certain pathogens could also be harmful because of the production of toxic compounds, the production of specific effectors, or the lack of plant resistance (25, 47).

F. oxysporum is well known for its phytopathogenic properties and causes vascular wilts and root/crown rots in more than 100 plant species; however, it also contains strains with biocontrol properties that show distinct colonization behavior in comparison to the pathogenic formae speciales. Pathogenic *F. oxysporum* hyphae penetrate the vascular stele of susceptible root tissue to proliferate within the xylem elements and result in disease (1, 131). This type of interaction was described as a compatible fungal-host interaction (58). In contrast, the incompatible interactions

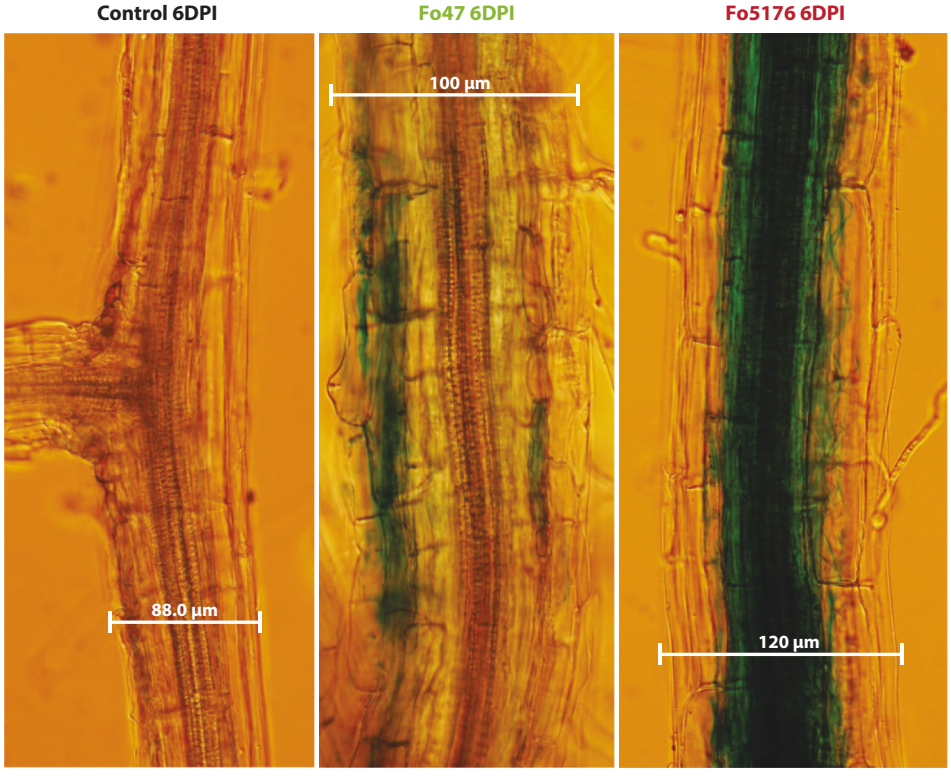

Figure 4

Colonization of *Arabidopsis thaliana* Col-0 roots by the nonpathogenic and pathogenic strains of *Fusarium oxysporum* Fo47 and Fo5176, respectively, six days postinoculation (DPI). Blue stain evident in fungal infected roots is from X-ARA (5-bromo-4-chloro-3-indoxyl-α-L-arabinofuranoside), which reacts with arabinofuranosidase enzymes secreted by *F. oxysporum* to generate a blue-colored product. All pictures were taken at 20× with a 1/15 s exposure time and are differential interference contrast (DIC) images with the resolution optimized by Kohler Illumination. Images were optically stained by altering the objective Nomarski prism to produce yellow-hued Newtonian interference colors.

are usually asymptomatic and are often observed among resistant plant varieties or nonhost plants (68). With a strong host specificity among the species complex *F. oxysporum*, strains from nonhost *F. oxysporum* formae speciales are rarely observed or at least may be restricted within the root vascular cortex (91). It is noteworthy that there are no or minimal initial differences in terms of root attachment and colonization of pathogenic versus nonpathogenic *F. oxysporum*, as well as several mutants that are critical for fungal virulence, in either compatible or incompatible interactions (70, 99). Depending on the host species, noticeable differences in root colonization by fungal hyphae develop several days post inoculation. Nonpathogenic *F. oxysporum* predominantly form hyphal networks within the root epidermal and outer cortical cells, whereas pathogenic *F. oxysporum* sink hyphae into the xylem and proliferate within this specific vascular compartment (**Figure 4**).

GENOMIC INSIGHTS INTO PATHOGENICITY AND NONPATHOGENICITY

Regardless of whether they are nonpathogenic endophytes or plant pathogens, microbes possess very similar physiological capacities, such as rhizosphere competence, motility to reach the host

plant, mechanisms for entrance and spreading inside the plant, and the ability to overcome plant immunity. These are characteristics that enable them to successfully colonize their plant hosts (84, 107). However, the outcome of such interaction differs drastically. This leads inevitably to the question about the features that differentiate nonpathogenic endophytes from pathogens.

Genomics of Bacterial Taxa Comprising Pathogenic and Nonpathogenic Members

Genomes of closely related mutualistic and pathogenic bacteria have very similar gene contents (70, 117), which poses a challenge for predicting bacterial pathogenicity based on global genomic analysis. However, several reports point to the importance of protein secretion in determining plant-microbe interactions. The transport of effector proteins into host cells is an important strategy of bacteria to suppress host plant defense (59) and may have had a key role in the evolution of the parasitic lifestyle (8). Effector proteins are often recognized by the plant immune system and activate effector-triggered immune responses in plants (59). Particularly important in this context are T3SSs and T6SSs.

Unlike in plant pathogens, genes for T3SSs are largely missing or incomplete in genomes of mutualistic endophytes (49, 84, 107). The genome of the mutualistic strain *P. phytofirmans* PsJN, for example, encodes all T3SSs components except the needle-forming protein (84). With regard to the absence of T3SSs in many endophytes, Reinhold-Hurek & Hurek (107) proposed that nonpathogenic endophytes should be regarded as disarmed pathogens. This is supported by the finding that a loss in functional T3SSs enables evolution of an endophytic lifestyle. For example, T3SS mutants of *Salmonella enterica* ser. Typhimurium showed increased endophytic colonization in *M. truncatula* (55). Furthermore, inactivation of T3SS structural and virulence regulator genes has been demonstrated to facilitate experimental evolution of the pathogen *R. solanacearum* carrying the symbiotic, rhizobial plasmid to a nodulating symbiont (77). However, T3SSs are also a key determinant of colonization of nonleguminous plants by rhizobia (e.g., 98), and several nonpathogenic *Pseudomonas* strains contain a T3SS but do not elicit a hypersensitive response (HR) in plants and thereby support their establishment in roots (103). In other studies, it was shown that the T3SS of *Pseudomonas* strains is involved in biocontrol activity against *Pythium ultimum* (108) or in the synergistic interaction between *M. truncatula* and mycorrhiza (138).

Genes for T6SSs are commonly found in genomes of mutualistic proteobacterial endophytes, indicating a general role for T6SSs in plant-microbe interactions (84, 107). Mutations in T6SSs of the potato pathogen *P. atrosepticum* resulted in increased disease symptoms and colonization (79). In contrast, mutations in the T6SSs significantly reduced virulence in other bacterial pathogens (111). This indicates that protein secretion systems are not per se determinants of a specific phenotype. Instead, the outcome of plant-microbe interaction seems to be determined by the nature of the effector protein secreted. Deletions and rearrangements in effector genes may be mechanisms to broaden host range and maintain virulence in bacteria (8). It has been observed that *P. syringae* pv. *phaseolicola*, which repeatedly traveled through plant tissues undergoing HR, lost the gene for an avirulence effector protein and developed pathogenicity (100). Similar findings were made when comparing pathogenic and mutualistic *Pantoea ananatis* seed endophytes. The *hcp* effector protein gene cluster was present in the beneficial and neutral strains but absent in the pathogen (117).

All in all, finding a clear distinction between mutualistic and pathogenic endophytes based on differences in the genome is difficult. López-Fernández et al. (70) compared virulence genes in endophytes and other symbiotic bacteria and concluded that there are only minor differences between endophytes and pathogens and that the similarities between these two groups are set above

the species level. However, we should not forget that prediction of gene functions is still limited, and many bacterial genes that were found to be expressed during plant-microbe interaction are of yet unknown function (118, 119). A better understanding of functions encoded by bacterial genomes could reveal new insight into the mechanisms of plant-microbe interactions and identify genomic determinants of bacterial lifestyle that are yet unknown.

The *Fusarium oxysporum* Complex as an Interesting Model to Understand Pathogenic and Nonpathogenic Relationships

Similar to bacteria, fungi are prominent plant colonizers comprising pathogens, commensals, and mutualists (49). Among many fungal pathosystems, the *F. oxysporum* species complex represents an interesting comparative model for understanding pathogenic versus symbiotic relationships (71–73). Members within this species complex cause diseases in more than 100 plant species, but this species complex also comprises strains with biocontrol properties. One particular nonpathogenic *F. oxysporum* strain, Fo47, has been studied as a potential biocontrol agent of *Fusarium* wilt and other root diseases directly through several mechanisms, including nutrient competition, antibiosis, and mycoparasitism, or indirectly through inducing plant defense gene expression and alterations to root cell architecture (92).

A distinct feature of genomes within the species complex also made *F. oxysporum* an effective model to investigate the genomic dynamics and plant-fungal interactions. A comparative genomics study revealed the structural and functional compartmentalization of the *F. oxysporum* genome, which essentially comprises two components: a basal genome encoding many of the basic fungal functions and an adaptive genome encoding accessory as well as more sophisticated features such as host preferences (74). The adaptive genome is presented in the form of accessory or lineage-specific (LS) chromosomes. They are horizontally transferred and are required for host-specific pathogenicity. Distinguished from the core, these accessory chromosomes contain distinct features, such as the ability to encode virulence factors, that are enriched for repeats and transposons (72, 74). Interestingly, the nonpathogenic strain Fo47 also contains an LS chromosome, suggesting that the accessory chromosomes also contribute to the endophytic and symbiotic plant-fungal interactions. However, this chromosome lacks transposons and the signature plant pathogenic SIX genes (named after the effectors that are secreted in xylem). Even though the Fo47 genome lacks the pathogenicity related SIX genes, the genome encodes for many small secreted proteins that are actively induced during the course of interactions with the plant host *Arabidopsis thaliana* (L.-J. Ma, unpublished data). Effectors also play significant roles in mediating endophytic and symbiotic plant-fungal interactions; however, research in this field is scarce. A recent study demonstrated that a small, secreted protein isolated from the nonpathogenic *F. oxysporum* strain CS-20 primed tomato defense genes and attenuated symptoms from *F. oxysporum* f. sp. *lycopersici* (116). Comprehensive understanding of the LS chromosomes in Fo47 will further shed light on this important research field.

CONCLUSIONS AND PERSPECTIVES

Plants are associated with a multitude of microorganisms, most of them without known function or acting as commensals. Few microorganisms show a mutualistic relationship with their host or exhibit pathogenicity if a set of conditions prevails, i.e., if the pathogen has the capacity to act as a pathogen on a specific host, if the host is susceptible, if the pathogen population size is sufficient to cause disease, or if the specific abiotic and biotic environments allow pathogen spread and disease expression. Furthermore, the microbial environment has to be appropriate to facilitate expression

of disease factors or colonization of the pathogen. The recognition that plant microbiota may contribute substantially to disease severity and development has led to the introduction of the term pathobiome (135). A number of pathogens, particularly viruses, were reported to act synergistically in disease development (64). However, insights from intestinal inflammation in human guts point to more complex relationships between pathogens, commensals, and mutualists (94). Hajishengallis & Lamont (46) identified keystone pathogens (or bacterial drivers) as directly manipulating host defense and disturbing microbiota composition. They are aided by accessory pathogens and pathobionts (or bacterial passengers), which exploit compromised defense, in their establishment in the community (by, e.g., nutritional or colonization support). We furthermore propose that disease is driven by a highly complex disease network in which different abiotic and biotic factors interact and influence each other (**Figure 1*d***). For instance, exposure of the plant-microbe system to a specific temperature or soil environment results in a specific plant condition, which, together with temperature/soil environment, shapes the associated microbiota and its interactions. Therefore, disease occurs only at specific points of this network or at specific combinations of all factors. However, the establishment of certain microbial communities can support disease suppression. We need to better understand all these interactions to better predict disease incidence and severity as well as to identify novel approaches for combating diseases. Furthermore, plant-associated microorganisms are applied in agriculture as biofertilizers or biopesticides, and before entering the market they are usually subject to a rigorous risk assessment. A better understanding of mechanisms involved in pathogenicity and mutualism will facilitate and promote the development and application of sustainable microbial solutions in crop production.

Keystone pathogens: highly adapted pathogens directly manipulating plant defense with a strong influence on microbiome composition

Accessory pathogens: support keystone pathogens in their establishment in the community by nutritional or colonization support and find their niche in the pathobiome

Pathobionts: members of the microbiome normally living in commensal or mutualistic relationship but acting as pathogens after breakdown of the plant-microorganism balance; includes necrophytic and saprophytic microorganisms

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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Errata

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