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SI PLANT BIOTIC INTERACTIONS

The role of water in plant–microbe interactions

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SUMMARY

Throughout their life plants are associated with various microorganisms, including commensal, symbiotic and pathogenic microorganisms. Pathogens are genetically adapted to aggressively colonize and proliferate in host plants to cause disease. However, disease outbreaks occur only under permissive environmental conditions. The interplay between host, pathogen and environment is famously known as the 'disease triangle'. Among the environmental factors, rainfall events, which often create a period of high atmospheric humidity, have repeatedly been shown to promote disease outbreaks in plants, suggesting that the availability of water is crucial for pathogenesis. During pathogen infection, water-soaking spots are frequently observed on infected leaves as an early symptom of disease. Recent studies have shown that pathogenic bacteria dedicate specialized virulence proteins to create an aqueous habitat inside the leaf apoplast under high humidity. Water availability in the apoplastic environment, and probably other associated changes, can determine the success of potentially pathogenic microbes. These new findings reinforce the notion that the fight over water may be a major battleground between plants and pathogens. In this article, we will discuss the role of water availability in host–microbe interactions, with a focus on plant–bacterial interactions.

Keywords: high humidity, water-soaking, plant disease, plant immunity, stomata.

INTRODUCTION

Water is essential for all living organisms. It functions as a solvent, a temperature buffer and a metabolite in living cells. Unlike animals, plants and microorganisms rely largely on their immediate surroundings for water. Land plants obtain water mainly from soil, whereas microbes that live in or on land plants gain water from the plant and/or water vapor from the atmosphere. Water availability is known to have a great impact on plant diversity and microbial community structure (Lau and Lennon, 2012; Blazewicz *et al.*, 2014; Jonas *et al.*, 2015; Taketani *et al.*, 2017).

Although plants are often surrounded by a multitude of various microbes, most microorganisms cannot colonize plants. This is largely attributed to plants having evolved layers of active defense mechanisms that are effective in protecting them from most microbes. For example, plants have developed strong physical barriers such as a hydrophobic wall on mature roots, bark on stems and waxy cuticles on leaves to prevent microorganisms from entering plant tissues. At the cellular level, microbes can be detected by plasma-membrane-bound receptors (called pattern recognition receptors, PRRs) on the cell surface. Each PRR recognizes a specific pathogen-associated molecular pattern (PAMP). Many PAMPs, such as bacterial flagellin or elongation factor Tu (EF-Tu), are broadly conserved in microbes. Recognition of PAMPs triggers an ancient form of plant defense called pattern-triggered immunity (PTI), which halts the proliferation of most non-pathogenic microbes (reviewed in Segonzac and Zipfel, 2011; Macho and Zipfel, 2014; Li *et al.*, 2016).

Over the course of plant–microbe co-evolution, some microorganisms have adapted to colonize and proliferate pathogenically in plants. As a major pathogenetic

mechanism, many pathogens translocate proteinaceous virulence proteins (called effectors) into host cells, targeting different components of PTI and other forms of plant defense to disarm the plant (reviewed in Jones and Dangl, 2006; Grant *et al.*, 2006; Buttner and He, 2009; Rafiqi *et al.*, 2012). In addition, pathogenic bacteria utilize effectors to create a suitable living environment by redirecting sugar (Chen *et al.*, 2010; Cohn *et al.*, 2014; Cox *et al.*, 2017) and water (Xin *et al.*, 2016; Schwartz *et al.*, 2017) into the extracellular space, where many of them live inside the plant.

In addition to a PRR-based surveillance system at the cell surface, plants have evolved an intracellular surveillance system. Specifically, intracellular immune receptors (also known as disease resistance (R) proteins) can detect microbial effector proteins inside the plant cell and trigger a second layer of plant defense, termed effector-triggered immunity (ETI; reviewed in Chisholm *et al.*, 2006; Jones *et al.*, 2016). Effector-triggered immunity is generally a more robust form of plant defense than PTI as it is often accompanied by plant cell death, known as the hypersensitive response (HR). The HR may help to restrict the proliferation of microbes from infection sites (reviewed in Khan *et al.*, 2016).

It has long been observed that the interaction between a virulent pathogen and a genetically susceptible host plant does not always lead to disease. For a pathogenic microbe to aggressively proliferate in a host plant, favorable environmental conditions are also required. The triangular interaction between pathogen, plant and environment is known as the 'disease triangle' (Stevens, 1960). Among the environmental factors that influence disease development, high atmospheric humidity has been repeatedly found to be associated with disease outbreaks (Miller *et al.*, 1996; Pernezny and Zhang, 2005; Schwartz, 2011). In this article, we discuss the critical role of high humidity and water on host–microbe interactions. We will start with an overview of water transportation and homeostasis in land plants as a preamble to an in-depth discussion on the effect of water on microbes on the plant surface, microbial pathogenesis inside the plant and the effectiveness of plant defense.

A BRIEF OVERVIEW OF WATER TRANSPORTATION AND HOMEOSTASIS IN LAND PLANTS

Plants have evolved ways to take up water and maintain water homeostasis to survive and grow. For land plants, soil provides a major water source. Soil pores between and within aggregates function as storage compartments for water. To maximize water uptake, land plants have developed an elaborate network of roots that spread through the soil to gain access to water. Although all parts of the root system might be involved in absorbing water, root tips and root hairs account for bulk water uptake as those cell types are more permeable to water. Mature regions of the root, on the other hand, have developed specialized tissues, named exodermis or hypodermis, which hinder efficient water permeability (Hose *et al.*, 2001).

Upon absorption by the root hairs or epidermal cells, water traffics across the cortex, the endodermis and finally the pericycle before being unloaded into the vasculature for long-distance transport in plants. From the root epidermis to the endodermis water moves through three pathways: the apoplastic pathway, symplastic pathway and transmembrane pathway (Figure 1b). The apoplastic space includes plant cell walls and extracellular spaces between the plasma membranes. In the apoplastic pathway, water is absorbed into root tissues through the cell wall of root hairs or epidermal cells and traffics apoplastically without going into cells. In the symplastic pathway, water is absorbed into root hairs or epidermal cells and traffics to the pericycle through the plasmodesmata, which are membrane-lined channels that connect between cells. On the other hand, if water enters and exits from one cell to the other directly through the plasma membrane the route is known as the transmembrane pathway (Figure 1b; Steudle and Peterson, 1998). Apoplastic and transmembrane movement of water are forced into the symplastic pathway at endodermal cells because they are surrounded by the Casparian strip (Figure 1b), which blocks water diffusion. Given the amount of water that needs to move from the root to the shoot, water-transporting channel proteins, aquaporins, play an active role in facilitating movement of water across living cells (Kjellbom *et al.*, 1999).

After water has trafficked through the endodermis and the pericycle, it flows into the xylem by osmosis for long-distance transport (Figure 1c). In higher plants, the xylem is mostly made of tracheids. During the xylem maturation process, cells undergo cell wall lignification and programmed cell death, creating cylinder-shaped pipes made of the remaining cell walls with pits on the wall. The xylem is composed of vertically overlapped tracheids, whereas pits of the adjacent tracheids often align with each other, creating pit pairs. Pit pairs allow water to move through a low-resistance path between tracheids (Taiz and Zeiger, 2010). After water is transported to leaves, it exits the xylem and is distributed to cells in the leaf (Figure 1d).

Plants have developed a robust system to maintain water homeostasis under various environmental conditions. The upward water flow from root to shoot appears to be governed by negative pressure (Wheeler and Stroock, 2008). It is generally believed that the base of the root possesses positive pressure whereas the leaf retains negative pressure. The negative pressure in the leaf is created by the mesophyll cell walls, which are composed of hydrophilic materials (e.g. cellulose microfibrils and pectins; Taiz and Zeiger, 2010), and transpiration of water through stomata (Landsberg and Waring, 2017), which are microscopic pores in leaves involved in the uptake of

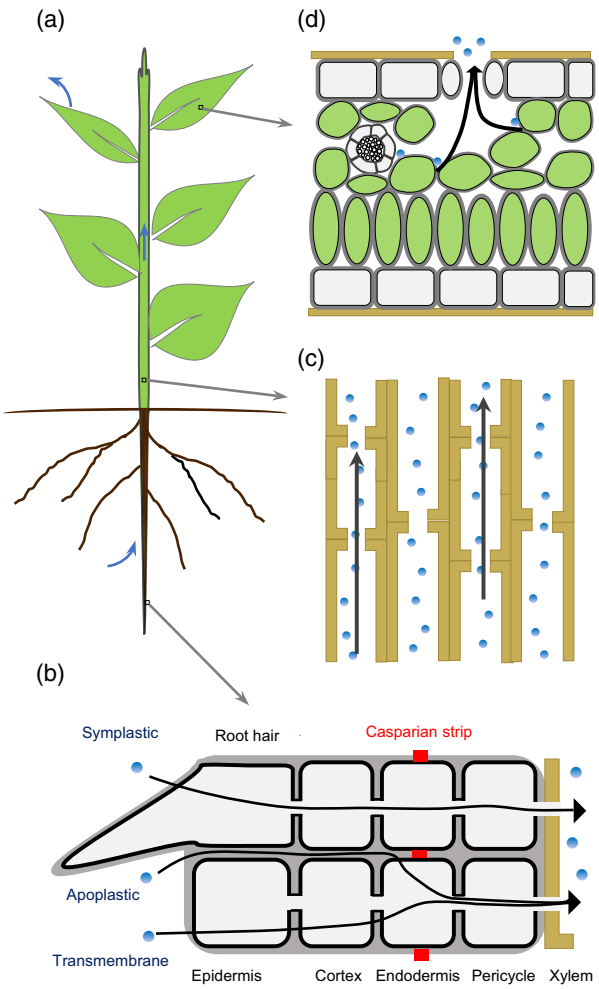

Figure 1. Movement of water from soil to the atmosphere through a plant.
(a) A land plant takes up water from the soil by roots, distributes water through the xylem to other parts of the plant, and transpires water vapor into the atmosphere from the leaves. Root hairs and epidermal cells are mainly responsible for water uptake. Blue arrows indicate the water flow from soil to atmosphere via a plant.
(b) Water enters root cells through three distinct pathways: apoplastic, symplastic and transmembrane pathways. All three pathways converge into a symplastic movement at the endodermis.
(c) Water is unloaded into the xylem and subjected to long-distant transport.
(d) In the leaf, water leaves the vascular bundle and is distributed to mesophyll cells and epidermal cells. Water is then drawn into plant cell walls. The water vapor from cell walls moves to the atmosphere through stomata during transpiration. Black arrows indicate the direction of water flow in a plant.

carbon dioxide (CO_2) necessary for photosynthesis. It is estimated that plants retain only around 5% of the water absorbed by the roots, and stomata are responsible for almost 97% of plant water loss through the transpiration process (reviewed in Ruggiero *et al.*, 2017). In addition, specialized water pores on the leaf edge, called hydathodes, have a minor role in plant water loss and water homeostasis in plants (Taiz and Zeiger, 2010).

Plant water content is subjected to changes in environmental conditions. The transpiration rate can be affected by environmental factors including atmospheric humidity, temperature, light and wind velocity. When atmospheric humidity is low, plants reduce their stomatal aperture to prevent excess water loss through transpiration. High atmospheric temperature, high light intensity and high wind velocity increase the transpiration rate (Moreshet, 1970). Similarly, low atmospheric humidity, high temperature and strong winds increase the evaporation of water from plant surfaces. Thus, the availability of water to microorganisms that live in or on plants varies under different environmental conditions.

EFFECT OF WATER ON MICROBES PRIOR TO ENTERING PLANTS

Water is a prerequisite for microorganisms to grow and proliferate; however, most microorganisms do not possess mechanisms to actively take up water. Instead, they rely on osmotically active substances in the cytoplasm to maintain a positive turgor. The osmotic gradient triggers a water flux into the microorganism in a hypotonic environment (Kempf and Bremer, 1998), where the exterior has higher water potential than the interior of the microorganism.

Effect of water on rhizosphere microbes

Soil microorganisms are ubiquitous, but they thrive only where water is accessible. A low water content in soil has a profound effect on its microbial inhabitants by affecting not only water availability but also nutrient availability. Thus, water content in soil poses a major selective pressure in shaping soil microbial communities. Recent studies have documented that soil relative humidity is positively correlated with the richness of the soil microbiota (Lau and Lennon, 2012; Blazewicz *et al.*, 2014; Jonas *et al.*, 2015; Taketani *et al.*, 2017). Rhizosphere bacterial communities from semi-arid ecosystems show a drastic increase in bacterial abundance during the wet season compared with the dry season. In addition, the dry season promotes a much higher population of desiccation-resistant bacteria (e.g. Actinobacteria), some of which can form spores to withstand desiccation stress. On the contrary, the wet season favors the growth of desiccation-sensitive bacteria (e.g. Proteobacteria; Taketani *et al.*, 2017). Similar to rhizosphere bacterial populations, rhizosphere fungal populations are also affected by soil humidity (Lau and Lennon, 2012; Blazewicz *et al.*, 2014).

Water flooding also affects rhizosphere microbes. In a controlled greenhouse experiment, a significant decrease in the overall microbial population was observed after flooding (Unger *et al.*, 2009; Ferrando and Fernandez Scavino, 2015). Flooding might also change the movement and distribution of microbes in soils and damage plant

tissues, potentially creating wounds through which microbes can enter plants.

Effect of water on phyllosphere microbes

Compared with rhizosphere microorganisms, phyllosphere microbes (microbes dwelling on and within above-ground tissues) are generally more vulnerable to water stress due to their closer proximity to the atmosphere. Phyllosphere microbes that live on the leaf surface (called epiphytic microbes) rely on trace amounts of water (also nutrients) available on that habitat. Since the vast area of the leaf surface is covered with waxy cuticles, which effectively block water transpiration, nutrient release and gas exchange, epiphytic microbes tend to live where trace amounts of water and nutrients are available. As discussed in the previous section 'A brief overview of water transportation and homeostasis in land plants', the majority of plant-associated water moves to the atmosphere through stomata; however, other aqueous pathways in the leaf cuticle have been shown to allow water transpiration (Schonherr, 2006). These minor aqueous pathways are found near the base of trichome and anticlinal cell walls, which are mostly located near the vascular tissues. Interestingly, these aqueous pathways are where epiphytic microbes tend to colonize (Monier and Lindow, 2003, 2004), supporting the notion that microbes aggregate near water (probably also nutrient) sources on the leaf surface. Phyllosphere bacteria also require water for motility on the leaf surface. A positive correlation between leaf surface water abundance and bacterial motility (both swimming and twitching) has been reported (Beattie, 2011). Flagellar motility of *Pseudomonas putida*, a Gram-negative bacterium, requires liquid films thicker than 1.5 μm (Dechesne *et al.*, 2010), suggesting that flagellum-dependent bacteria are able to move when the thickness of water films is greater than the size of a bacterium. Movement across the leaf surface increases the chance for bacteria to gain access to the leaf interior, where more water and nutrients are available.

Given that high atmospheric humidity might relieve water stress on epiphytic microbes on the leaf surface, it has been shown to shape the phyllosphere microbiome. In field research, high atmospheric humidity shows a strong positive correlation with the abundance and richness of culturable fungi on the leaf surface (Talley *et al.*, 2002). Under controlled laboratory conditions, high atmospheric humidity is required for the survival of newly infected *Pseudomonas syringae* bacteria on bean leaves (Monier and Lindow, 2005). Similarly, the population, spore germination and disease outbreak of filamentous pathogens are also influenced by atmospheric moisture and water availability on the leaf surface (Huber and Gillespie, 1992; Talley *et al.*, 2002). In particular, dormant fungal spores require water and/or elicitor cues (e.g. components of the cuticle; Serrano *et al.*, 2014) to break dormancy. Upon germination, filamentous pathogens penetrate through the cuticle layer to gain access to stable sources of water and nutrients in the leaf apoplast (Van Der Does and Rep, 2017).

It has been well documented that heavy precipitation events increase water availability to phyllosphere microbes, which allows them to grow and multiply (Hirano and Upper, 2000). In addition, rainfall may liberate and disperse pathogens from infected tissues to surrounding tissues and plants. Wounds created on the leaf due to rainfall also increase the opportunity for microbes to enter the plant. High atmospheric humidity increases the permeability of cuticle, which provides more water and nutrients to microbes, and it promotes stomatal opening, which allows microbes to enter the apoplast (Melotto *et al.*, 2017).

Effect of humidity on stomatal defense

Although primarily serving as a portal for gas exchange and transpiration, stomata put plants at risk as foliar pathogens exploit stomata as entry sites to gain access to the apoplast (Melotto *et al.*, 2006). To prevent the invasion of pathogens through stomata, the cells that make up stomata, guard cells, have evolved to recognize a variety of PAMPs, including flagellin, chitin, chitosan and oligogalacturonic acid (Arnaud and Hwang, 2015). Such recognition triggers downstream signaling events, ultimately resulting in narrowing of the stomatal aperture as a defense mechanism (Melotto *et al.*, 2017).

Some pathogenic microbes have evolved specific virulence factors to actively manipulate the stomatal aperture. Many of these virulence factors, including the fungal toxin fusicoccin, the bacterial toxins coronatine and syringolin A, as well as an increasing list of proteinaceous effectors (e.g. AvrB, HopF2, HopM1, HopX1 and HopZ1), have been shown to promote stomatal opening to facilitate bacterial entry into the plant (reviewed in Melotto *et al.*, 2017). Intriguingly, pathogenic microbes might also directly or indirectly induce stomatal closure at a later stage of infection, presumably to maintain leaf apoplastic water potential for sustained multiplication. Consistent with this notion, pathogen-induced stomatal opening appears to be a transient phenomenon (Freeman and Beattie, 2009). It was reported that an effector of *P. syringae*, HopAM1, plays an important role in promoting its virulence activity in water-stressed plants. HopAM1 was found to promote stomatal closure in an abscisic acid (ABA)-dependent manner (Goel *et al.*, 2008). Abscisic acid is a plant hormone known to induce stomatal closure (Melotto *et al.*, 2017). Together, the findings suggest that pathogenic bacteria deploy virulence factors to manipulate stomatal movements, allowing bacteria to enter the apoplast at an early infection stage. Once bacteria have entered the plant they induce stomatal closure, presumably to increase the availability of water for bacterial multiplication inside the leaf.

Although pathogens actively manipulate stomatal movements using virulence factors, high atmospheric humidity also promotes stomatal opening. Therefore, some phyllosphere microbes that do not have active mechanisms to open stomata could potentially take advantage of high atmospheric humidity to gain entry into the plant. Recent studies have begun to shed light on the molecular mechanisms that regulate humidity-dependent stomatal movement at the molecular level. High atmospheric humidity triggers the degradation of ABA by upregulating key enzymes involved in ABA catabolism, resulting in stomatal opening (Okamoto *et al.*, 2009). Additionally, a recent study has shown that bacteria-triggered stomatal closure is suppressed by high humidity due to early activation of jasmonate (JA) hormone signaling and suppression of the salicylic acid (SA) defense signaling pathway within stomatal guard cells (Panchal *et al.*, 2016). The effect of high humidity on JA and SA signaling pathways in the guard cells is observed as early as 15 min after the high-humidity treatment (Panchal *et al.*, 2016). Jasmonate and SA are plant hormones that are involved in plant defense (Campos *et al.*, 2014; Yang and Dong, 2014). Together, these findings show that high humidity modulates positive and negative regulators of the stomatal aperture that could contribute to the abundance and movements of phyllosphere microbes.

EFFECT OF WATER ON MICROBES AFTER THEY HAVE ENTERED PLANTS

Pathogenic bacteria create an aqueous habitat in the leaf apoplast under high humidity

In addition to its effect on promoting survival and movement of microbes on the leaf surface and invasion of bacteria into the plant interior, a recent study has revealed a crucial role for high atmospheric humidity in modulating bacterial population even after bacteria have entered the leaf apoplast (Xin *et al.*, 2016). It has been known for a long time that many bacterial pathogens induce water-soaked spots as an early disease symptom under conditions of high atmospheric humidity (Johnson, 1937). Water-soaking symptoms are also induced by other types of pathogens, including fungi and oomycetes. For example, the fungal pathogen *Magnaporthe oryzae*, the causal agent of rice blast disease, induces water-soaked lesions during an early infection phase (Ahn *et al.*, 2005). In 1937, it was reported that an artificially water-soaked apoplast allowed aggressive proliferation of *Bacterium angulatum* and *Bacterium tabacum* in tomato, bean and apple, as well as other plants which are not normally susceptible to colonization by these microbes (Johnson, 1937). A similar observation was made by Young in 1974. The pathogenic bacterium *Pseudomonas phaseolicola* as well as non-pathogenic bacteria *Pseudomonas lachrymans* and *P. syringae* could multiply to similar levels in bean leaves when water is supplied to the apoplast during the infection (Young, 1974). These findings suggest that water in the apoplast can fundamentally change host–microbe interactions (Ramos, 2010).

A detailed study of the development of water-soaking symptoms in the Arabidopsis–*P. syringae* pv. *tomato* (*Pst*) DC3000 pathosystem showed that water-soaking is a transient process that appears during early infection (about 24 h; Figure 2a) and disappears before the appearance of late disease symptoms, including tissue chlorosis and necrotic lesions. *In vivo* imaging showed that water-soaked regions are where bacteria aggressively proliferate (Xin *et al.*, 2016). Remarkably, unlike the virulent strain *Pst* DC3000, an avirulent strain, *Pst* DC3000 (avrRpt2), which activates ETI, fails to induce water-soaking symptoms (Xin *et al.*, 2016). This finding suggests that activation of the plant immune response can block the water-soaking process, possibly as an integral part of the plant defense mechanism against bacterial pathogenesis. In line with this intriguing finding, a previous study showed that virulent and avirulent bacteria experience different water stress levels in the leaf apoplast. Specifically, virulent *Pst* DC3000 bacteria experience suitable water potentials for pathogen multiplication in the leaf apoplast, whereas avirulent *Pst* DC3000 (avrRpm1) bacteria experience a very high level of water stress in the resistant plant that would inhibit bacterial growth *in vitro* (Wright and Beattie, 2004). Together, these results suggest that activation of ETI may restrict water supply at the infection sites of avirulent bacteria.

How pathogens are able to create an aqueous apoplast environment under high humidity is not clear, but specific pathogen virulence factors are required. In the case of bacteria, several effector proteins have been shown to be involved in the development of water-soaking spots in host plants. Activities of the effector proteins AvrE and HopM1 from *Pst* DC3000 as well as WtsE, an AvrE-family effector protein from *Pantoea stewartii* subsp. *stewartii*, cause an aqueous apoplast in Arabidopsis (Xin *et al.*, 2016) and maize (Ham *et al.*, 2006; Asselin *et al.*, 2015), respectively. In addition, the *Pst* DC3000 *avrE*$^{-}$*hopM1*$^{-}$ mutant, which lacks water-soaking-inducing effectors, fails to cause an aqueous apoplast during infection (Xin *et al.*, 2016). Interestingly, although AvrE and HopM1 share no amino acid sequence similarity they are functionally redundant in *Pst* DC3000 pathogenesis (Debroy *et al.*, 2004). HopM1 targets and degrades a plant ARF-family guanine nucleotide exchange factor protein, AtMIN7, involved in vesicle trafficking (Nomura *et al.*, 2006). Correspondingly, the Arabidopsis *atmin7* mutation, which partially mimics the virulence action of HopM1, promotes spontaneous, albeit limited, water-soaked spots in certain Arabidopsis genotypes under high atmospheric humidity (Xin *et al.*, 2016). These results suggest that AtMIN7 is normally involved in maintaining water homeostasis in the apoplast and that

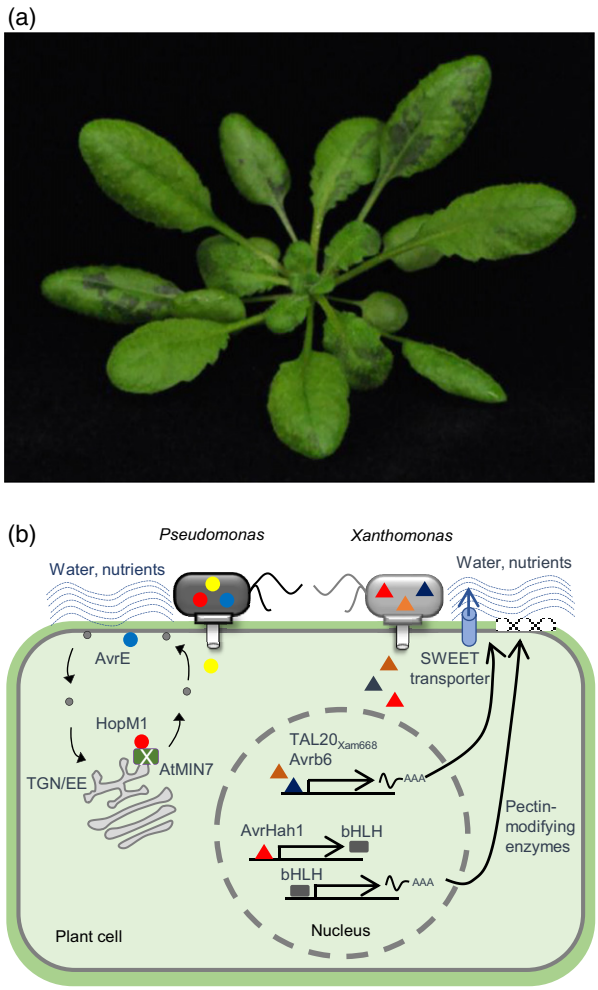

Figure 2. Pathogenic bacteria create water-soaking spots on host plants during pathogenesis.
(a) An Arabidopsis plant is infected with a bacterial pathogen, *Pst* DC3000. The image was taken 1 day after infection. Dark areas on the leaves indicate water-soaking spots.
(b) A model illustrates how pathogenic bacteria create an aqueous environment in the leaf apoplast to support their aggressive growth. *Pst* DC3000 utilizes two protein effectors, HopM1 and AvrE, to create an aqueous habitat in the apoplast. Once inside the plant cell, HopM1 is targeted to the trans-Golgi network/early endosome (TGN/EE) and degrades a plant ARF-family guanine nucleotide exchange factor protein, AtMIN7, involved in vesicle trafficking. AvrE is localized to the plasma membrane. These two effectors likely affect the plant plasma membrane integrity, creating osmotic sinks to draw water (possibly nutrients) into the apoplast. *Xanthomonas gardneri*, on the other hand, employs AvrHah1, which is a transcription activator-like (TAL) effector (TALE), to induce water-soaking symptoms in plants. AvrHah1 upregulates expression of two basic helix-loop-helix (bHLH) transcription factors, which subsequently induce the expression of two genes that encode pectin-modifying enzymes. The actions of pectin-modifying enzymes might change the composition of plant cell walls, affecting the hygroscopicity of the cell walls. In addition, *Xanthomonas axonopodis* pv. *manihoti* and *Xanthomonas citri* subsp. *malvacearum* deliver TALEs $TAL20_{Xam668}$ and Avrb6, respectively, to upregulate expression of the sugar transporter genes *SWEET* in plants. By redirecting the distribution of sugar in their host plants, the pathogenic bacteria might facilitate their nutrition as well as increase osmotic potential in the apoplast, leading to an aqueous apoplast environment in the infected leaves.

Pst DC3000 uses HopM1 to destroy the AtMIN7 protein as part of its mechanism to change water availability in the apoplast. How HopM1-mediated degradation of AtMIN7 and the molecular actions of AvrE and WtsE lead to an aqueous environment is not yet known. Given the functional role of AtMIN7 in regulating vesicle trafficking and maintaining the integrity of the plasma membrane (PM) and the PM localization of AvrE and WtsE (and its host target protein phosphatase 2A) in Arabidopsis (Jin *et al.*, 2016; Xin *et al.*, 2016), these bacterial effectors might manipulate the PM integrity and/or phosphorylation of host cells to possibly create osmotic sinks that draw water into the apoplast to benefit the bacteria (Figure 2b).

Xanthomonas gardneri, a bacterial pathogen, can also induce water-soaked disease symptoms in tomato. AvrHah1, a transcription activator-like (TAL) effector (TALE) of *X. gardneri* was recently found to target two basic helix-loop-helix (bHLH) transcription factors in plants. Activation of these transcription factors causes expression of two pectin-modifying genes, a pectate lyase and a pectinesterase. Using designer TALEs (dTALEs), ectopic expression of the two bHLH transcriptions and the pectin lyase, but not the pectinesterase, ultimately leads to a water-soaked leaf apoplast upon bacterial infection. Strikingly, tobacco leaf infected with *X. gardneri* containing AvrHah1 can draw externally added water from the leaf surface into the apoplast (Schwartz *et al.*, 2017). How the pectin lyase causes water-soaking remains to be elucidated. One possibility is that changes in properties of the plant cell wall might affect the hygroscopicity of the cell wall, causing water to accumulate in the leaf apoplast (Figure 2b).

In addition, two *Xanthomonas* species cause water-soaked disease symptoms in their host plants via TALE-mediated induction of plant sugar transporters. *Xanthomonas axonopodis* pv. *manihotis,* a pathogen that causes bacterial blight of cassava, delivers $TAL20_{Xam668}$ to regulate expression of the sugar transporter gene *MeSWEET10a* in cassava (Cohn *et al.*, 2014). On the other hand, *Xanthomonas citri* subsp. *malvacearum* (*Xcm*), the causal agent of bacterial blight of cotton, secretes Avrb6 to induce the expression of the sugar transporter gene *GhSWEET10* in cotton (Cox *et al.*, 2017). These two studies indicate that pathogenic bacteria can redirect the distribution of sugar in their host plants not only to facilitate their nutrition but also to alter osmotic potential in the apoplast, resulting in an aqueous apoplast environment in the infected leaves. Together, the above-mentioned findings suggest that different pathogens have convergently evolved distinct mechanisms to establish an aqueous living space in the leaf apoplast, impacting different aspects of plant–bacterial interactions.

Pathogen-induced water soaking symptoms may affect plant–microbe interactions beyond pathogenesis. Avrb6-

induced water-soaking is associated with greatly increased release of *Xcm* bacteria from infected plant tissues (Yang *et al.*, 1994). In addition, it was recently reported that water-soaking and necrosis in leaves infected by *Xanthomonas euvesicatoria* and *X. gardneri*, can promote colonization of the human pathogen *Salmonella enterica* inside plant tissues (Potnis *et al.*, 2015). This is interesting in light of the finding by Xin *et al.* (2016) that the levels and abundance of leaf endophytic bacterial communities are altered in plant genotypes that are prone to water-soaking under high humidity. Thus, apoplast water-soaking affects not only pathogenesis, but probably also affects the endogenous microbiome in the leaf apoplast. The observed promotion of human pathogen colonization by apoplast water-soaking illustrates the importance of elucidating the water-soaking mechanisms in understanding the dynamics of foodborne pathogens in plants.

High atmospheric humidity affects the *R* gene-mediated HR

High humidity has been shown to suppress the *R* gene-mediated HR, which involves rapid plant cell death at the site of pathogen infection. It was reported that high atmospheric humidity delays the HR in tomato plants expressing an *R* gene (Cf-4 or Cf-9) and a matching avirulence gene (Avr4 or Avr9) from the fungal pathogen *Cladosporium fulvum* (Wang *et al.*, 2005). In addition, several Arabidopsis 'autoimmune' mutants, in which immune responses are spontaneously activated without pathogen infections, show a humidity-dependent phenotype. Arabidopsis mutants *ssi1* and *shl1* (Zhou *et al.*, 2004; Noutoshi *et al.*, 2005), caused by a gain-of-function mutation in the *R* genes *cpn1*/*bon1* and *cpr22* (Yoshioka *et al.*, 2006; Mosher *et al.*, 2010), exhibit retarded growth and chlorotic and enhanced disease resistance phenotypes under moderate humidity; however, the phenotypes are suppressed by high atmospheric humidity (>95%; Zhou *et al.*, 2004; Noutoshi *et al.*, 2005; Yoshioka *et al.*, 2006; Mosher *et al.*, 2010). In addition, the autoimmune mutants exhibit elevated defense hormone SA production when the plants are grown under moderate humidity; whereas SA production is suppressed by high humidity (Zhou *et al.*, 2004; Noutoshi *et al.*, 2005; Mosher *et al.*, 2010). However, as mentioned above, the avirulent bacterium *Pst* DC3000 (avrRpt2) still can mount ETI and block water-soaking symptoms under conditions of high humidity, even though the macroscopic tissue collapse (i.e. HR) was effectively prevented (Xin *et al.*, 2016). Together, the findings suggest that high humidity may have a negative impact on some, but not all, ETI-associated immune responses. Understanding how high atmospheric humidity negatively affects the function and activity of *R* genes and downstream signaling steps has significant practical implications for the deployment of *R* genes for disease control in the context of changing climate.

Effect of drought on plant resistance

The ongoing changes in climate conditions have significant implications for crop production around the globe. It is predicted that a warmer climate will increase the occurrence of prolonged drought and flooding (Wetherald and Manabe, 2002). Two recent studies showed that drought-stressed Arabidopsis and chickpea plants are more resistant to bacterial pathogens, *Pst* DC3000 and *P. syringae* pv. *phaseolicola*, respectively (Gupta *et al.*, 2016; Sinha *et al.*, 2016).

Drought treatment or *Pst* DC3000 infection are both known to increase the accumulation of ABA in plants (reviewed in Ton *et al.*, 2009; Helander *et al.*, 2016). ABA promotes stomatal closure, which prevents bacteria from entering through stomata (Eisenach and de Angeli, 2017; Inoue and Kinoshita, 2017; Vialet-Chabrand *et al.*, 2017). However, increased ABA levels are associated with enhanced susceptibility to *Pst* DC3000 in Arabidopsis at the post-invasive stage (de Torres-Zabala *et al.*, 2007). Interestingly, when Arabidopsis plants are challenged with drought and pathogen simultaneously, ABA level remains unchanged but the levels of defense hormones SA and JA are raised, providing an explanation for why the combined stress leads to enhanced disease resistance in Arabidopsis (Gupta *et al.*, 2017). In addition, drought-stressed chickpea exhibits higher resistance to a xylem-inhabiting bacterial pathogen, *Ralstonia solanacearum* (Sinha *et al.*, 2016). In contrast, drought-treated rice becomes more susceptible to *M. oryzae* infection (Bidzinski *et al.*, 2016). In this pathosystem, drought stress was found to suppress PTI and ETI (Bidzinski *et al.*, 2016). These contrasting findings suggest that severe water limitation (i.e., drought) might differentially affect plant defense and microbial pathogenesis in different pathosystems. Further study is needed to clarify the underlying causes using different pathosystems, which will shed light on the effect of drought stress on host-pathogen interactions.

Microbial-mediated drought tolerance in plants

Not only does drought alter plant responses to pathogens but drought-adapted microbial communities have been shown to be beneficial for overall plant fitness under drought stress regardless of their historical growth conditions (i.e. dry or wet; Lau and Lennon, 2012). In addition to the effect of the rhizosphere microbiome on plant responses to drought, inoculation of individual bacterial strains has also been shown to improve plant resistance to drought (reviewed in Ngumbi and Kloepper, 2016). The plant-growth-promoting rhizobacterium (PGPR) *Paenibacillus polymyxa* can protect Arabidopsis against drought stress and upregulate expression of drought-stress response genes (Timmusk and Wagner, 1999). Moreover, several bacterial strains (*Bacillus* sp., *Pseudomonas* sp., *Acinetobacter* sp., *Sphingobacterium* sp., *Enterobacter* sp.

and *Delftia* sp.) isolated from drought-treated grapevine rootstocks can improve grapevine resistance to drought by increasing overall plant fitness (Salomon *et al.*, 2014; Rolli *et al.*, 2015). Among these bacteria, *Bacillus licheniformis* Rt4M10 and *Pseudomonas fluorescens* Rt6M10 can produce several plant hormones, including ABA, indole-3-acetic acid (IAA) and gibberellin (Salomon *et al.*, 2014). ABA induces stomatal closure to reduce water consumption in plants and improve their drought tolerance (reviewed in Helander *et al.*, 2016). Inoculation with the two ABA-producing bacterial strains significantly increases the ABA level in the inoculated plants (Salomon *et al.*, 2014), protecting the plants from drought stress. In addition to bacteria, an endophytic fungus, *Piriformospora indica*, can also improve drought tolerance and induces expression of a suite of drought stress-related genes in Arabidopsis (Sherameti *et al.*, 2008). Together, the findings suggest that prolonged water deficit could drastically alter the composition of soil microbial communities and some of the drought-enriched microbial strains might be beneficial for their colonized plants in improving drought tolerance and water homeostasis.

CONCLUSION AND OUTSTANDING QUESTIONS

Clearly, water plays a fundamental role in modulating the biology of plants, microbes and their interactions. With changing climatic conditions, it is increasingly relevant to understand how water availability and homeostasis in plants and their surrounding habitats impact diverse plant–microbe interactions. Current understanding of this topic is very limited in both scope and depth. In the case of plant diseases, it is clear that changing weather patterns will continue to impact the frequencies of disease outbreaks, as well as the emergence of new diseases and the disappearance of some old ones. There is renewed urgency to deepen our understanding of how changing environmental conditions, including temperature, humidity and microbiota, affect plant–pathogen interactions in diverse ecosystems, as such fundamental knowledge is needed to develop climate-resilient crop plants for future generations. Reductionist experiments in the laboratory have proven to be powerful (and will continue to be) in revealing some of the underlying mechanisms of plant–microbe interactions. However, the bulk of current studies have been conducted under artificial environmental conditions that are quite far removed from what plants and microbes experience in nature. In crop fields, simultaneous exposure to biotic and abiotic stresses is likely the rule in a given plant–microbe interaction. As advances in new technologies have enabled the study of plant–microbe interactions at molecular and cellular level it is important to devote substantially more effort to investigating how plants interact with pathogenic, commensal and symbiotic microbes under conditions that closely simulate their native growth environment.

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KA, YJJ and SYH wrote the paper.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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