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Plant susceptible responses: the underestimated side of plant–pathogen interactions

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ABSTRACT

Plant susceptibility to pathogens is usually considered from the perspective of the loss of resistance. However, susceptibility cannot be equated with plant passivity since active host cooperation may be required for the pathogen to propagate and cause disease. This cooperation consists of the induction of reactions called susceptible responses that transform a plant from an autonomous biological unit into a component of a pathosystem. Induced susceptibility is scarcely discussed in the literature (at least compared to induced resistance) although this phenomenon has a fundamental impact on plant–pathogen interactions and disease progression. This review aims to summarize current knowledge on plant susceptible responses and their regulation. We highlight two main categories of susceptible responses according to their consequences and indicate the relevance of susceptible response-related studies to agricultural practice. We hope that this review will generate interest in this underestimated aspect of plant–pathogen interactions.

Key words: conditionally beneficial pathogens, plant susceptible responses, plant infectious diseases, plant tolerance, S-genes

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I. INTRODUCTION

A variety of phytopathogenic microorganisms can cause plant damage and lead to extensive yield losses in agriculture. It is generally considered that pathogen invasion results in disease development if the plant immune system is not activated or is activated too late to prevent pathogen propagation. Hence, plant immunity has been extensively studied, and immunity-related genes exploited in frameworks of breeding strategies to create resistant plant cultivars.

However, the introduction of immunity-related genes is not the only way of obtaining resistant plant varieties. Plant resistance also can be achieved due to the loss-of-function of so-called susceptibility (S)-genes. Plant S-gene products provide signals attracting pathogens and increasing their aggressiveness and contribute to the accommodation of pathogens *in planta* (Eckardt, 2002; Lapin & Van den Ackerveken, 2013; van Schie & Takken, 2014).

In several cases, S-gene loss-of-function has been shown to be an effective agricultural strategy that provides more durable field resistance than the resistance conferred by introduced immunity-related genes (Engelhardt, Stam & Hückelhoven, 2018). Compared to S-gene loss-of-function, the introduction of resistance genes imposes a stronger selective pressure on a pathogen, potentially leading to the rapid emergence of new strains that are able to avoid the improved host plant immunity (Mundt, 2014). Moreover, since host plant S-gene products provide different 'services' to pathogens, to overcome S-gene loss-of-function, a pathogen must adapt to interact with a host plant without these 'services', which may be more difficult than evading increased immunity.

Although S-gene loss-of-function may lead to plant pathogen resistance, it often simultaneously has a negative effect on plant growth, development and other traits (van Schie & Takken, 2014). Clearly, if S-genes provided only pathogen susceptibility, they would be eliminated from the plant genome. However, S-genes are indispensable for normal plant development and are usually expressed irrespective of the presence of pathogens, unlike many immunity-related genes. Therefore, S-genes have not been excluded by evolution, although they may determine plant vulnerability to pathogens. For this reason, S-gene loss-of-function has significant restrictions in terms of its application in plant breeding, and hence, relatively few examples of S-gene-deficient varieties are currently used in agriculture (Engelhardt *et al.*, 2018). Nevertheless, the control of S-gene-mediated plant susceptibility represents a promising strategy for reducing disease in crops that is poorly realized due to a lack of fundamental knowledge.

Plant S-genes were previously divided into three main categories: (*i*) genes that facilitate host recognition and penetration; (*ii*) genes that encode negative regulators of immune signalling; and (*iii*) genes that fulfill metabolic or structural requirements of a pathogen that allow its proliferation (van Schie & Takken, 2014). S-genes of the first category determine plant susceptibility at the preinvasion stage. S-genes of the second and third categories are often manipulated by the pathogen at the post-invasive stage to contribute to plant susceptibility. By inducing the expression of S-genes of the second category, a pathogen forces the host plant to produce proteins that repress post-invasive activation of the immune system. In turn, the pathogen-mediated upregulation of S-genes of the third category enables the pathogen to modify the host plant so as to ensure disease progression and/or pathogen propagation *in planta*. Host plant reactions mediated by S-genes of the third category that promote pathogen propagation *in planta* and/or disease symptom development are referred to as susceptible responses (SRs). A number of studies have demonstrated that the development of pathogen-induced diseases may require host plant reactions that are typical of normal physiological processes (e.g. cell growth and differentiation, transport of water and photosynthetic assimilates, absorption of inorganic substances from the soil, and reactions related to the abiotic stress response) (see Section II). Therefore, it appears that an intact plant (before pathogen invasion) may not be an optimal environment for the pathogen, and the transformation of this plant into a component of the pathosystem requires certain reactions to be induced. Moreover, disease symptoms caused by many phytopathogens also appear to be a consequence of plant responses (see Section V). Thus, in many cases, it is not S-genes *per se* that promote plant pathogen susceptibility; the susceptibility is a result of the pathogen-mediated upregulation of S-genes, which leads to the induction of SRs. Even in the presence of functional S-genes, the host plant can be non-susceptible if a pathogen is unable to induce a S-gene-mediated SR.

A particular S-gene can be both sensitive and insensitive to pathogen manipulation depending on different factors. The particular plant physiological reaction that represents the SR during a plant–pathogen interaction is usually part of normal plant development and, therefore, is controlled by a variety of regulatory networks depending on ontogenetic stage, physiological status, tissue type, environmental conditions, and many other factors. Hence, plant susceptibility depends on whether the current plant physiological status restricts the pathogen-mediated activation of the specific

SRs. Presumably, this is one of the reasons that individual genetically susceptible plants display different degrees of pathogen susceptibility (from full susceptibility to full resistance) at different ontogenetic stages, nutritional status, environmental conditions, etc. (Block *et al.*, 2005; Walters & Bingham, 2007; Gohlke & Deeken, 2014).

Different sets of SRs and their quantitative levels in each particular case may provide fine-tuning of various aspects of pathosystem development, leading to a variety of forms and outcomes of the interaction between a particular plant and pathogen species. Knowledge on SRs and their regulation is of great importance since the control of SRs seems to represent a more effective agricultural strategy than S-gene knockout, which often has negative effects on plant development. However, in spite of their great theoretical and applied importance, SRs have received much less attention than defence responses. Although S-genes and some plant reactions related to pathogen susceptibility have been reviewed previously (Hok, Attard & Keller, 2010; Lapin & Van den Ackerveken, 2013; van Schie & Takken, 2014; Faris & Friesen, 2020), there is no review to date that provides a comprehensive overview of plant SRs and their regulation mechanisms from the perspectives of both the pathogen and plant. Herein we focus mainly on bacterial plant diseases, but other pathogens are also considered. We first describe SRs related to different physiological processes (stomatal movement, plant cell wall modification, water and assimilate transport, programmed cell death, metal exchange, and neoplastic growth) and discuss how these processes affect plant–microbe interactions. We then discuss plant regulators that mediate different SRs and bacterial virulence factors that can act as inducers of SRs. Finally, we highlight two main categories of SRs according to their consequences and indicate the practical relevance of SR-related studies. Our goal is to attract attention to SRs, which are an underestimated aspect of plant–microbe interactions. Future in-depth investigations of SRs have high potential to provide a basis for designing novel plant protection approaches for agriculture as well as enhancing our fundamental knowledge on plant–pathogen interactions.

II. PHYSIOLOGICAL PROCESSES INVOLVED IN PLANT SUSCEPTIBLE RESPONSES

(1) Motion of stomatal guard cells

Stomatal opening necessary for normal water and gas exchange represents a major route for bacteria to infiltrate a host plant. Plants are able significantly to reduce bacterial penetration through a mechanism related to pathogen-induced molecular pattern (PAMP)-induced stomatal closure (Melotto, Underwood & He, 2008; McLachlan, Kopischke & Robatzek, 2014). Plant pathogens have been shown to counteract this stomatal closure defence response by targeting a variety of regulators and processes: salicylic acid (SA)-signalling (Misas-Villamil *et al.*, 2013; Geng *et al.*, 2014) (*Pseudomonas syringae*), OST1 (open stomata 1) kinase and abscisic acid (ABA) signalling (Melotto *et al.*, 2006), the mitogen-activated protein kinase 3 (MPK3) pathway (Gudesblat, Torres & Vojnov, 2009*b*) (*Xanthomonas campestris*), proton pump (Gudesblat, Torres & Vojnov, 2009*a*) (*X. campestris*), proteasome-mediated protein degradation (Lozano-Durán *et al.*, 2014) (*P. syringae*), K^+ accumulation and the breakdown of starch in guard cells (Guimarães & Stotz, 2004) (*Sclerotinia sclerotiorum*). These actions result in either the blockage of stomatal closure or the induction of their reopening. *Pseudomonas syringae* is able to both stimulate opening of the stomata during the invasion stage of infection and induce stomatal closure after entering the host interior in order to reduce water loss (Goel *et al.*, 2008; Freeman & Beattie, 2009). Thus, some pathogens can induce SRs related to the coordination of stomatal movements in order to allow them to penetrate the plant and to engineer the most beneficial environment inside the host (Fig. 1).

(2) Modification of the plant cell wall

The plant cell wall (PCW) serves as a physical barrier to microorganisms and a sensor surface involved in pathogen perception. Pathogen recognition is well known to induce many distinct host plant reactions that lead to an increase in the antipathogenic properties of the PCW (Underwood, 2012). However, some PCW-related reactions may contribute to plant susceptibility to biotic stressors and thus represent SRs.

To infect the host plant, most (if not all) phytopathogens have to modify the structure and properties of the PCW. This modification is a very complex process due to the high variability of polysaccharides constituting the greatest portion of this compartment. Although the number of different PCW polysaccharide types (as defined by basic elements of the backbone and branching points) is relatively limited (around 10), the fine structure and functional characteristics of a particular polysaccharide can be adjusted for particular physiological roles (e.g. according to cell type and/or cell developmental stage) (Albersheim *et al.*, 2010). Moreover, even within a particular physiological role, individual molecules of a particular polysaccharide type are rather variable due to their non-template synthesis. Therefore, a particular polysaccharide is actually a 'population' of similar molecules distinguished from one another in the degree of polymerization, the pattern of possible modifications (methylation and acetylation), the presence/absence/location of the side chains, etc. (Gorshkova, Kozlova & Mikshina, 2013).

The high variability of PCW polysaccharides requires a large number of enzymes with particular substrate specificities for the modification or breakdown of complex carbohydrates. Plants are well equipped with polysaccharide-modifying enzymes that facilitate PCW loosening and stretching during cell growth and fruit ripening as well as other PCW modifications related to different physiological processes (Fry, 1995). Plants maintain large numbers of genes of multigene family proteins with particular substrate and environmental (e.g. pH, osmolarity, and cofactors) specificities. For example, the *Arabidopsis thaliana* genome

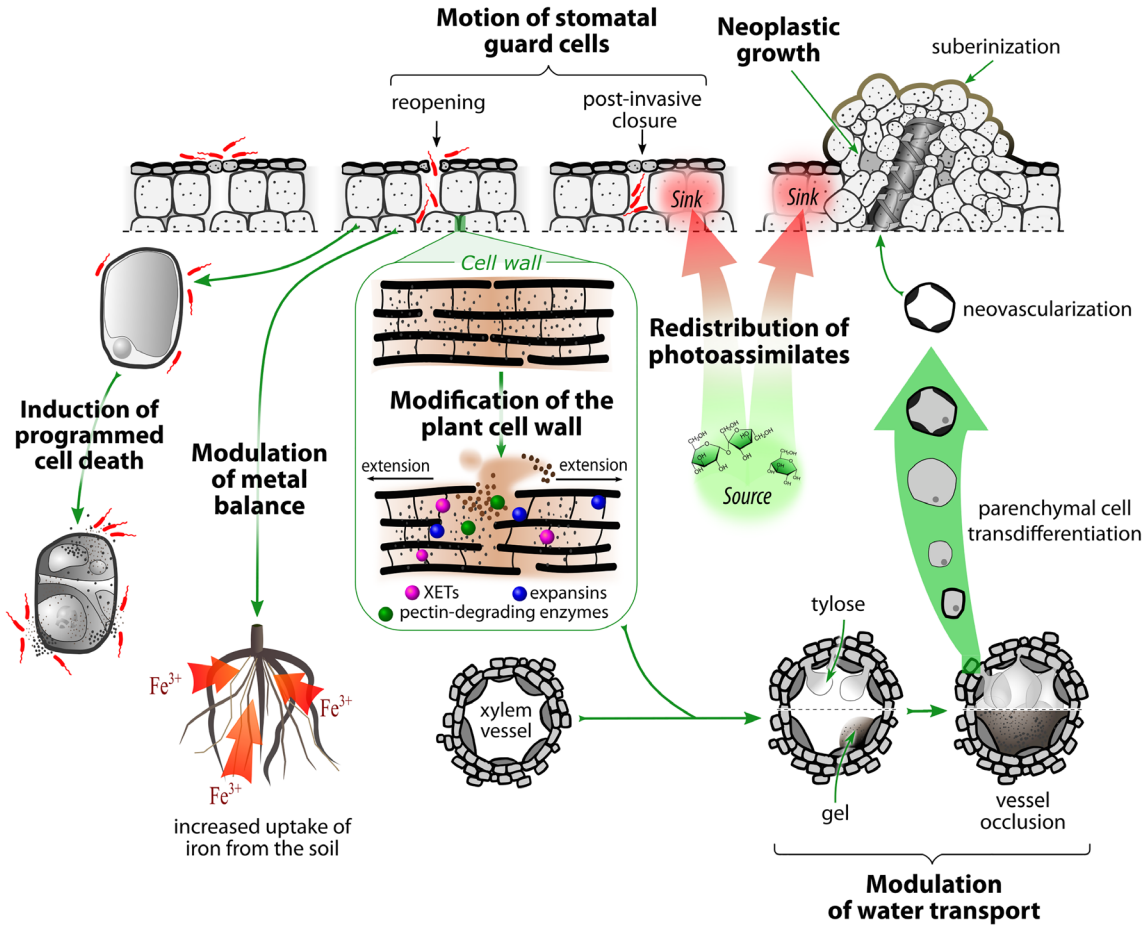

Fig 1. Schematic representation of pathogen-induced plant susceptible responses. XET, xyloglucan endotransglucosylases/hydrolase.

contains 67 genes encoding pectin methylesterases and 52 encoding polygalacturonases (The Arabidopsis Genome Initiative, 2000). In other plant species, multigene families can be even larger: the genomes of *Solanum lycopersicum*, *Populus trichocarpa*, and *Linum usitatissimum* contain 79, 89, and 105 genes, respectively, encoding pectin methylesterases (Pelloux, Rustérucci & Mellerowicz, 2007; Vandevenne *et al.*, 2009; Pinzón-Latorre & Deyholos, 2013). Pathogens tend to have reduced genome sizes which prohibit the possession of dozens or even hundreds of similar proteins. Therefore, many pathogens have acquired the ability to exploit host plant PCW enzymes/proteins, and the prevention of such exploitation results in reduced host susceptibility.

The action of several plant enzymes degrading glycosidic bonds in PCW polysaccharides (endo-β-glucanases, polygalacturonase, and pectate lyase-like protein) was shown to enhance plant susceptibility to pathogens (Vogel *et al.*, 2002; Flors *et al.*, 2007; Cantu *et al.*, 2008). Non-enzymatic PCW proteins – expansins that breakdown cellulose–hemicellulose hydrogen bonds and promote 'polymer creep' associated with PCW loosening (Cosgrove, 2015) – were also demonstrated to have a role in plant susceptibility (Kay *et al.*, 2007; Cantu *et al.*, 2008; Abuqamar *et al.*, 2013). PCW loosening is obviously beneficial for pathogens since it enables their spread within the host plant, facilitates depolymerization of PCW constituents yielding low-molecular-weight nutrients, and enhances the mobility of polymers *in muro*, leading to their release from the PCW for utilization by the pathogen. Prior to its invasion of xylem vessels, *Pectobacterium atrosepticum* induces the plant-mediated release of rhamnogalacturonan I (RG-I, a pectic polysaccharide) from the PCW into the vessel lumen (Gorshkov *et al.*, 2014, 2016, 2021). The released RG-I is utilized by these bacteria as an extracellular matrix necessary for the assemblage of bacterial emboli (biofilm-like multicellular structures). Expansins, rhamnogalacturonan lyases, β-galactosidases, and some other PCW proteins encoded by infection-upregulated genes were proposed to be involved in the SR related to the release of RG-I (Tsers *et al.*, 2020).

Plant methyl- and acetylesterases of PCW polysaccharides were also shown to be exploited by pathogens. The enzymatic digestion of the pectic polysaccharide homogalacturonan – the main 'target' of many phytopathogens – is impeded by its esterification with methyl and/or acetyl groups (Bellincampi, Cervone & Lionetti, 2014). Although many phytopathogens

possess their own methyl- and acetylesterases, microorganisms often rely on host plant esterases during plant colonization. Plant genes encoding esterases were demonstrated to be induced during infection by *Botrytis cinerea* and *Pectobacterium carotovorum*, while the inhibition of plant pectin esterases was shown to increase plant resistance to pathogens (Lionetti *et al.*, 2007; An *et al.*, 2008; Raiola *et al.*, 2011).

PCW-related SRs are associated not only with polysaccharide degradation/modification but also with their biosynthesis. Inactivation of cellulose- and callose-related biosynthetic genes appeared to increase plant resistance to pathogens (Nishimura *et al.*, 2003; Hernández-Blanco *et al.*, 2007). This involvement of callose in plant susceptibility was confusing since this polymer has been widely shown to determine plant resistance (Piršelová & Matušíková, 2013): callose can be used to plug holes in the PCW during cell division or pathogen invasion. Callose was also presumed to be beneficial to pathogens by protecting them from plant defence metabolites, forming a structural basis for the haustorial neck, and acting as a barrier that limits the diffusion of pathogen-derived elicitors of plant defence responses (Jacobs *et al.*, 2003; Bellincampi *et al.*, 2014). In addition, reduced cellulose and callose biosynthesis appears to activate plant defence systems (Jacobs *et al.*, 2003; Nishimura *et al.*, 2003; Hernández-Blanco *et al.*, 2007) indicating that a plant can detect an impaired PCW and then deploy alternative mechanisms to protect itself from invaders.

During plant–pathogen interactions, the deposition of callose is often associated with the formation of specific PCW appositions (plugs, or papillae) which are structures that contain many different compounds together with callose. Papillae may be formed irrespective of whether a plant is resistant or susceptible to a pathogen. The papillae are referred to as either effective (if a plant is resistant) or non-effective (if a plant is susceptible). The effectiveness of papillae presumably depends on their composition, size, and timing of formation, which are determined by the parameters of endomembrane vesicle-associated transport (Hückelhoven, 2014; Pessina *et al.*, 2016). One of the presumed regulators of such transport – the Mlo (Mildew locus o) protein – was shown to have a role in plant SRs. Mlo-deficient mutants are highly resistant to powdery mildew and *Xanthomonas campestris* in contrast to wild-type plants (Piffanelli *et al.*, 2004; Consonni *et al.*, 2006). Therefore, it was proposed that pathogens may exploit Mlo to control metabolite transport during papillae formation and thus 'force' the plant to form only non-effective papillae (Bhat *et al.*, 2005; Miklis *et al.*, 2007; Pessina *et al.*, 2016).

Thus, alterations in PCW composition and properties during disease development are the result of action not only of pathogen enzymes/proteins but also those of the host plant. Pathogens intensively exploit the PCW-biogenesis machinery of the host plant to induce PCW modification (Fig. 1). PCW-related SRs contribute to the release of growth substrates from polysaccharides, facilitate systemic pathogen spread due to PCW loosening, and repress the defence systems of the host plant.

(3) Modulation of water transport

Wilt, a frequent symptom of plant infectious diseases, was long considered to be a result of the occlusion of the plant vascular system by microbial cells and their metabolites, such as exopolysaccharides. However, the bacterial biomass in diseased plants is often too low to cause significant occlusion of the conductive elements, and most xylem vessels remain bacteria-free (Sun *et al.*, 2013). It was found instead that blockage of the vascular system and wilting of the infected plants is a result of plant responses, namely the formation of tyloses and gels in the vessels (Klosterman *et al.*, 2009; Beattie, 2011; Sun *et al.*, 2013; Yadeta & Thomma, 2013). Tyloses are outgrowths of parenchyma cells protruding through pit-membranes into the vessels, leading to their occlusion. Tyloses and gels are often considered to prevent the systemic spread of pathogens *via* the vascular system, thus acting as defence structures. However, tyloses (as well as papillae, see Section II.2) can be both effective and non-effective, i.e. these structures are formed in both resistant and susceptible plant varieties (Sun *et al.*, 2013; Yadeta & Thomma, 2013; Wang *et al.*, 2015). Moreover, tyloses can be more abundant in susceptible plants than in resistant ones (Sun *et al.*, 2013).

The extensive formation of gels and tyloses significantly reduces water conductivity in infected plants, exacerbating disease symptoms (Sun *et al.*, 2013; Wang *et al.*, 2015; Bispo *et al.*, 2016). Mutant plants unable to form tyloses were shown to have increased resistance to *Xylella fastidiosa* and *Clavibacter michiganensis* compared to wild-type tylose-forming plants (Pérez-Donoso *et al.*, 2007; Balaji *et al.*, 2008). Some pathogens induce SRs related to the occlusion of xylem vessels by gels and tyloses in order to reduce water transport and create water deficiency in the host plant. In turn, water deficiency is coupled with enhanced plant susceptibility to some bacterial and fungal phytopathogens (Daugherty, Lopes & Almeida, 2010; Oliva, Stenlid & Martínez-Vilalta, 2014).

There are several reasons for the increased susceptibility of water-deficient plants to pathogens. First, water deficiency leads to disturbance of the primary metabolism that supplies plant defence systems with energy and intermediates (Bolton, 2009; Rojas *et al.*, 2014; Bispo *et al.*, 2016). Moreover, adaptation to water stress is a higher priority for plants than activation of defences against pathogens (Beattie, 2011); therefore, by forcing the plant to reduce xylem sap flow, a pathogen induces drought-adaptive responses rather than phytoimmune reactions.

Second, blockage of xylem flow may provoke sugar transport from phloem or parenchyma into the vessels to increase the osmolarity of xylem sap, with a resulting water inflow and hence the restoration of water transport (Nardini, Lo Gullo & Salleo, 2011; Brodersen & McElrone, 2013). Sugar transport to nutrient-poor vessels may be beneficial for phytopathogens residing in xylem. Thus, by stimulating blockage of the transpiration stream, a pathogen may maximise the presence of nutrients in xylem vessels.

Third, the decrease in water flow may promote microbial 'social behaviour', consisting of cell-to-cell exchanges of diffusible autoinducers (quorum sensing) and the formation of biofilms and biofilm-like structures (Liu *et al.*, 2008; Gorshkov *et al.*, 2014). Intensive xylem sap flow can cause the leakage of autoinducers from the bacterial microcosm and reduce their ability to communicate and perform coordinated actions. Thus, blockage or a reduction in water flow may facilitate both the accumulation of autoinducers and biofilm formation, both of which may enhance virulence.

Fourth, cessation of upward water flow in a vessel may be necessary for bacteria to implement downward migration. The downward vascular translocation widely shown for phytopathogenic bacteria (Fuente, Burr & Hoch, 2007; Czajkowski *et al.*, 2010; Misas-Villamil *et al.*, 2013; Sun *et al.*, 2013; Gorshkov *et al.*, 2014) promotes bacterial colonization of the underground parts and lateral shoots of the host. Since the upward flow rate of the xylem sap stream significantly exceeds the speed of bacterial movement, downward vascular bacterial translocation is likely to be impeded unless the water transport is reduced or blocked. *Xylella fastidiosa* intensively colonized lateral shoots (located below the inoculation site) of susceptible grapevine varieties with a high percentage of tylose-occluded vessels, while in resistant varieties with a low percentage of occluded vessels, the pathogen was localized only around the inoculation site and did not colonize lateral shoots (Sun *et al.*, 2013).

Although reduction/blockage of the transpiration stream in the host plant clearly provides many advantages to pathogens, too much occlusion may cause negative effects on pathogen fitness. A host plant suffering significantly from water deficiency may die, with the pathogen then losing its ecological niche. To prevent host plant death due to infection-associated water deficiency, new vessels may arise due to the transdifferentiation of sheath and xylem parenchyma cells or enhanced cambial activity (Baayen, 1986; Reusche *et al.*, 2012). Such pathogen-induced host plant responses neutralize water deficiency, prolong the life of a pathosystem, and increase plant drought tolerance (Reusche *et al.*, 2012).

Pathogens may induce water transport-related SRs not only in the vascular system but also in the outer host plant tissues. Some phytopathogens (e.g. *Pseudomonas* and *Xanthomonas* species) stimulate host plants to absorb water condensed on the leaf surface, causing the presence of water-soaking lesions (hydroses or wet apoplast) (Xin *et al.*, 2016; Aung, Jiang & He, 2018). Enhanced water absorption by leaves is thought to be driven by a pathogen-induced increase in hygroscopicity of PCWs and water permeability of the plasma membrane. The function of this pathogen-mediated plant reaction is unclear. However, it was shown that the virulence of some pathogens depends on their ability to induce the rapid absorption of water by leaves (Xin *et al.*, 2016). It is likely that such intensive water absorption promotes bacterial invasion into the host plant interior.

Thus, the success of some phytopathogens depends on their ability to induce water transport-related SRs in the host plant. These SRs are related to (*i*) blockage of the xylem sap flow due to the formation of gels and tyloses, (*ii*) recovery of the transpiration stream due to the *de novo* formation of vessels, and (*iii*) intensive water absorption by the leaf surface (Fig. 1). Water transport-related SRs may reduce the plant's immune status, promote microbial social behaviour and systemic plant colonization, enhance the nutrient content of xylem vessels, facilitate pathogen invasion, and prolong the life of a pathosystem.

(4) Redistribution of photoassimilates

The ability to acquire nutrients from host plants is essential for pathogens to establish successful infections. Some pathogens are known to attract photoassimilates by manipulating the source–sink status of the infection site: source tissues (which produce assimilates) after an infection may acquire a sink status (which consume assimilates) (Berger, Sinha & Roitsch, 2007; Seo *et al.*, 2007; Proels & Hückelhoven, 2014). The transition from source to sink status largely depends on increased activity of apoplast invertases. These enzymes convert the main transport sugar, sucrose, into glucose and fructose and thus participate in phloem unloading and forming a sucrose gradient. Invertases are known to be induced in infection sites resulting in an increase in glucose/sucrose ratio (Biemelt & Sonnewald, 2006; Bonfig *et al.*, 2006; Kocal, Sonnewald & Sonnewald, 2008; Liu *et al.*, 2015).

Increased invertase activity is known to promote so-called 'high sugar resistance' when an increased hexose level leads to a reduction in photosynthesis, activation of defence genes, and accumulation of reactive oxygen species (ROS) (Seo *et al.*, 2007; Essmann *et al.*, 2008). In addition, high invertase activity at the infected site may divert a large quantity of assimilates, leading to starvation of other sink tissues. The resulting systemic carbon disbalance in the plant was presumed to induce plant immune responses (Seo *et al.*, 2007). Indeed, invertases have been demonstrated to participate in plant immune responses (Bonfig *et al.*, 2006; Essmann *et al.*, 2008; Sonnewald *et al.*, 2012; Sun *et al.*, 2013). However, simultaneously, invertases were shown to be manipulated by pathogens in order to attract nutrients and promote host plant susceptibility (Kocal *et al.*, 2008; Siemens *et al.*, 2011; Liu *et al.*, 2015). Invertases play a specific role in the development of disease symptoms related to hypertrophy and hyperplasia that are not possible without intensive photoassimilate inflow (Deeken *et al.*, 2006; Siemens *et al.*, 2011).

In addition to plant invertases, pathogens also exploit plant SWEETs (sugar will eventually be exported transporters). SWEETs transfer sugars from the cytoplasm to the apoplast where they can be accessed more easily by a pathogen. Diseases caused by *Xanthomonas axonopodis* and *B. cinerea* are associated with the upregulation of SWEET genes, while their knockout or silencing leads to enhanced plant resistance to pathogens (Chen *et al.*, 2010; Chong *et al.*, 2014; Cohn *et al.*, 2014).

Pathogens may manipulate host plants to supply them not only with sugars but also with organic acids. *Xanthomonas oryzae* integrates a specific porin into its host plant's plasma

membrane causing leakage of α-ketoglutarate from the cytoplasm to the apoplast where it is consumed by the pathogen (Guo *et al.*, 2012). The exhaustion of α-ketoglutarate in plant cells leads to its increased biosynthesis and thereby to further enhanced leakage, ensuring a continuous supply to the bacteria.

Thus, some pathogens *via* SR induction can control access to host plant photoassimilates and primary metabolites that support the active proliferation of these microorganisms, leading to disease progression (Fig. 1).

(5) Induction of programmed cell death

The best-known example of programmed cell death (PCD) during plant–pathogen interactions is a hypersensitive response (HR). HR, a PCD induced only during biotic stress, is associated with a local oxidative burst that rapidly kills the infected plant cells together with the invading pathogen (Coll, Epple & Dangl, 2011; Dickman & Fluhr, 2013). However, PCD types other than HR, autophagy-related PCD and apoptosis-like PCD, in addition to taking part in normal plant development and adaptation to abiotic stressors, are also involved in plant–microbe interactions (Kwon, Cho & Park, 2010; Liu & Bassham, 2012). It is thought that pathogens with a necrotrophic lifestyle induce host PCD in order to obtain nutrients effectively from the digested host cells. PCD is often thought to be harmful to biotrophs that obtain nutrients from living cells. However, PCD can both promote and prevent the development of diseases caused by both biotrophs and necrotrophs depending on the particular host and pathogen species and type of activated PCD (Lenz *et al.*, 2011; Dickman & Fluhr, 2013; Kabbage, Williams & Dickman, 2013).

Autophagy allows the degradation of organelles and macromolecules in the vacuole. Autophagy may both save cells from death due to the recycling/removal of damaged cell components or toxic compounds (prosurvival function) and promote the digestion of cell constituents before death (prodeath function) (van Doorn *et al.*, 2011; Yoshimoto, 2012). A number of investigations indicate that the induction of autophagy can aid disease progression of some pathogens, predominantly biotrophs (Lai *et al.*, 2011; Lenz *et al.*, 2011; Dagdas *et al.*, 2016). The contribution of autophagy to disease progression is likely determined by its negative effect on HR as well as salicylic acid and ROS accumulation (Patel & Dinesh-Kumar, 2008; Lenz *et al.*, 2011; Wang *et al.*, 2011). In addition, selective autophagy was proposed to cause the specific elimination of defence compounds in host plant cells (Dagdas *et al.*, 2016).

The induction of the apoptosis-like PCD, which displays some, but not all, of the features of apoptosis in animal cells (Reape & McCabe, 2010) may also reflect a SR. The repression of runaway apoptotic-like cell death by the expression of heterologous anti-apoptotic gene *CED-9* from *Caenorhabditis elegans* in transgenic *A. thaliana* resulted in increased plant resistance to *Sclerotinia sclerotiorum* (Kabbage *et al.*, 2013). The action of plant caspase-like proteases (metacaspases or phytaspases) was shown to be necessary for disease progression of some pathogens (Richael *et al.*, 2001; Lincoln *et al.*, 2002; Baarlen *et al.*, 2007; Zhang *et al.*, 2014).

Even the HR, usually considered a very strong defence response that generally leads to full plant resistance (Torres, Jones & Dangl, 2006; Zurbriggen, Carrillo & Hajirezaei, 2010; Coll *et al.*, 2011; Dickman & Fluhr, 2013), may facilitate infections caused by some pathogens. The growth of *B. cinerea* was repressed in HR-deficient mutant plants, while preinfection induction of HR led to increased plant susceptibility to this pathogen (Govrin & Levine, 2000). Interestingly, the R receptor involved in the activation of HR appeared to determine both plant resistance to one pathogen and susceptibility to another (Lorang, Sweat & Wolpert, 2007).

Importantly, different types of PCD may have different or even opposite effects on a particular plant–pathogen interaction. Disease development caused by *S. sclerotiorum* is associated with apoptosis-like PCD, which is induced by oxalic acid produced by the pathogen (Kabbage *et al.*, 2013). The avirulent oxalic acid-deficient strain does not induce apoptosis-like PCD; instead, it activates the defence response associated with autophagic PCD. Moreover, the oxalic acid-deficient strain displayed virulence towards the autophagy-deficient plant mutant. Therefore, it was concluded that it is not PCD *per se* that dictates the outcome of certain plant–microbe interactions, but that the mechanism of PCD involved is crucial (Kabbage *et al.*, 2013).

Thus, some phytopathogens are able to manipulate PCD to enhance the detoxification of defence compounds and digestion of host cell contents (Fig. 1).

(6) Modulation of metal balance

The concentration of different metals in plants can be non-optimal for the extensive propagation of some phytopathogens. Therefore, pathogens have evolved ways to change the metal content in the host plant (or particular host plant compartments) to be able to propagate *in planta*. *Dickeya dadantii* requires significant quantities of iron for full virulence. To increase the iron level in the host plant, *D. dadantii* induces a leaf-to-root iron deficiency signal that activates host plant iron assimilation machinery, including the major root iron-chelate reductase and iron-regulated transporter (IRT) transporter (Dellagi *et al.*, 2009; Segond *et al.*, 2009) (Fig. 1). An inability of *D. dadantii* to induce iron acquisition (e.g. due to the absence of iron in the plant growth media) results in increased plant resistance, while preinfection induction of iron assimilation increases plant susceptibility to this pathogen (Franza, Mahé & Expert, 2005; Dellagi *et al.*, 2009; Kieu *et al.*, 2012).

Pathogens can also use host plant metal transporters to reduce the levels of toxic metals. Xylem vessel-colonizing *X. oryzae*, which is very sensitive to copper, induces host copper transport proteins to withdraw this metal from the xylem (Yuan *et al.*, 2010). Thus, some pathogens can exploit the metal-transporting machinery of the host plant to increase or reduce the levels of different metals in the infected area.

(7) Neoplastic growth: hypertrophy and hyperplasia

Some phytopathogens induce symptoms related to neoplastic growth, leading to galls, witches' brooms, hairy roots, etc. By inducing host plant SRs related to hypertrophy (pathological cell enlargement) and/or hyperplasia (a pathological increase in cell number) pathogens construct specific niches where they are likely to have a selective advantage compared to other potential plant parasites (Kado, 2014; Harris & Pitzschke, 2020).

The best-known example of neoplastic growth is crown galls caused by *Agrobacterium tumefaciens* (Pitzschke & Hirt, 2010). This bacterium introduces a fragment of tumour-inducing (Ti)-plasmid (T-DNA, transfer DNA) into a plant genome. This T-DNA contains genes for the biosyntheses of phytohormones (auxin and cytokinins) and opines – modified amino acids that are utilized by agrobacteria as a major growth substrate (Gordon & Christie, 2015). Expression of these T-DNA genes leads to the synthesis of auxin and cytokinins, which stimulate the proliferation and extension of opine-producing plant cells that provide food for the pathogen (Pitzschke & Hirt, 2010). The genetic transformation of host cells is not the only route by which a pathogen can cause hypertrophy and/or hyperplasia, and several phytopathogenic bacteria, fungi, protists, and nematodes induce such symptoms in other ways (e.g. by producing various effector proteins and phytohormones) (Jameson, 2000; Kay *et al.*, 2007; Robert-Seilaniantz, Grant & Jones, 2011).

The simultaneous induction of a set of SRs is required to produce neoplastic growth. Plant cell extension and differentiation are not possible without PCW modification (see Section II.2; Marois, Van den Ackerveken & Bonas, 2002; Kay *et al.*, 2007). Enhanced photoassimilate flow (Section II.4) is necessary to support intensive opine synthesis and active metabolism of the infected tissue, promoting cell proliferation and growth (Deeken *et al.*, 2006; Siemens *et al.*, 2011; Gohlke & Deeken, 2014). Neovascularization (Section II.3) is needed to supply water to the gall and prevent water deficiency (Aloni, Pradel & Ullrich, 1995; Ullrich & Aloni, 2000; Efetova *et al.*, 2007). Water loss is a potential problem for the functioning of a gall since its growth (associated with the excrescence of parenchymatous tissue) results in exfoliation of the epidermis which could lead to increased water evaporation and vulnerability of the gall to multiple stressors (Aloni *et al.*, 1995; Ullrich & Aloni, 2000; Efetova *et al.*, 2007). However, in addition to neovascularization, water balance in a gall is supported by the activation of suberin production in the outer periderm-like cell layer, enhanced osmoprotectant synthesis, and upregulation of aquaporins (Efetova *et al.*, 2007). In addition, plant SRs during neoplastic growth lead to the formation of fibre-like cells conferring mechanical strength to a neoplasm (Aloni *et al.*, 1995; Ullrich & Aloni, 2000).

Thus, during neoplastic growth, a set of different plant SRs cause cell hypertrophy and tissue hyperplasia and prolong the life of a neoplasm, supporting the pathogens within it (Fig. 1).

III. PLANT MEDIATORS COORDINATING SUSCEPTIBLE RESPONSES

To induce SRs, many phytopathogens often manipulate the global regulatory systems of the host plant. Such pathogen-induced physiological modification of a plant during pathosystem formation is known to be mediated *via* phytohormones and ROS.

(1) Phytohormones

Salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) are often referred to as phytohormones of biotic stress (Robert-Seilaniantz *et al.*, 2011; Kazan & Lyons, 2014). Although the induction of SA-, JA-, and ET-mediated hormonal systems is usually attributed to plant defence responses, upregulation of these systems often reflects SRs.

JA and ET are agonists that act in antagonism with SA. Consequently, after pathogen invasion, a plant prioritizes only one type of defence response: mediated by either JA/ET or SA (Beckers & Spoel, 2006; Halim *et al.*, 2006). Pathogens usually display differential sensitivity to these two types of responses, being highly susceptible to one of them and tolerant (or much less susceptible) to the other (Zhao *et al.*, 2003; Rahman *et al.*, 2012). Therefore, many phytopathogens induce the one defence system that is harmless for the pathogen, leading to repression of the system that is harmful to the pathogen (Robert-Seilaniantz *et al.*, 2011; Pieterse *et al.*, 2012; Zheng *et al.*, 2012). Although the repression of SA- or JA/ET-mediated hormonal systems was shown to reduce plant resistance to some pathogens (Gaffney *et al.*, 1993; Shoresh, Yedidia & Chet, 2005; van Loon, Geraats & Linthorst, 2006), in some cases, it can cause the opposite effect. For example, JA-insensitive plants are resistant to *P. syringae* due to post-infection accumulation of SA, while in susceptible wild-type plants, only a low level of SA is present due to the pathogen-induced activation of JA-mediated responses (Laurie-Berry *et al.*, 2006; Zheng *et al.*, 2012). Induction of JA/ET-mediated SRs also occurs during the development of soft rot caused by *Pectobacterium atrosepticum* (Gorshkov *et al.*, 2018; Tsers *et al.*, 2020). By contrast, *Xanthomonas campestris* pv. *vesicatoria* induces an increase in the SA level in the susceptible host but cannot cause disease development in SA-insensitive plants (O'Donnell *et al.*, 2001).

Other phytohormone systems mediated by auxin, cytokinins (CKs), gibberellins (GAs), and abscisic acid (ABA) were also demonstrated to participate in SRs (Audenaert, Meyer & Höfte, 2002; Navarro *et al.*, 2006; Depuydt *et al.*, 2008; Yang *et al.*, 2008). Often, the positive effect of these phytohormones on disease development is attributed to their influence on the SA- and/or JA-mediated hormonal systems: auxin and GAs

repress SA- and JA-induced reactions, respectively, while ABA inhibits both SA- and JA-regulated responses (Fig. 2). For example, *Pseudomonas syringae*-induced pathogenesis is associated with an increase in auxin level, which leads to the downregulation of SA-regulated processes; auxin-insensitive plants are less susceptible to this pathogen than the wild type (Djami-Tchatchou *et al.*, 2020). By contrast, *X. oryzae* induces a GA-regulated pathway that represses JA-mediated defence responses (Lu *et al.*, 2015).

ABA, a hormone of abiotic stress, acts to divert energetic resources from growth and defence systems in order to promote adaptation to abiotic stressors (Sah, Reddy & Li, 2016). Plants are likely to encounter abiotic stressors more often than pathogenic organisms and, therefore, ABA-regulated responses take priority over SA- or JA/ET-mediated ones (Mauch-Mani & Mauch, 2005; Asselbergh, De Vleesschauwer & Höfte, 2008*b*; Saijo & Loo, 2020). Due to this, ABA treatment or ABA-hypersensitivity/overproduction represses SA- and JA-regulated gene expression and reduces resistance to biotic stressors, while ABA-deficient or ABA-insensitive mutants demonstrate increased expression levels of SA- and JA-regulated genes and increased resistance to pathogens (Audenaert *et al.*, 2002; Anderson *et al.*, 2004; Thaler & Bostock, 2004; Kariola *et al.*, 2006; de Torres-Zabala *et al.*, 2007; Yasuda *et al.*, 2008; Asselbergh *et al.*, 2008*a*; Plessis *et al.*, 2011; Aalto *et al.*, 2012; Survila *et al.*, 2016; Van Gijsegem *et al.*, 2017). Therefore, pathogen-induced upregulation of the ABA-mediated hormonal system expressed in a 'reorientation' of a plant from the activation of defence reactions towards adaptation to 'imaginary' abiotic stressors represents a typical SR.

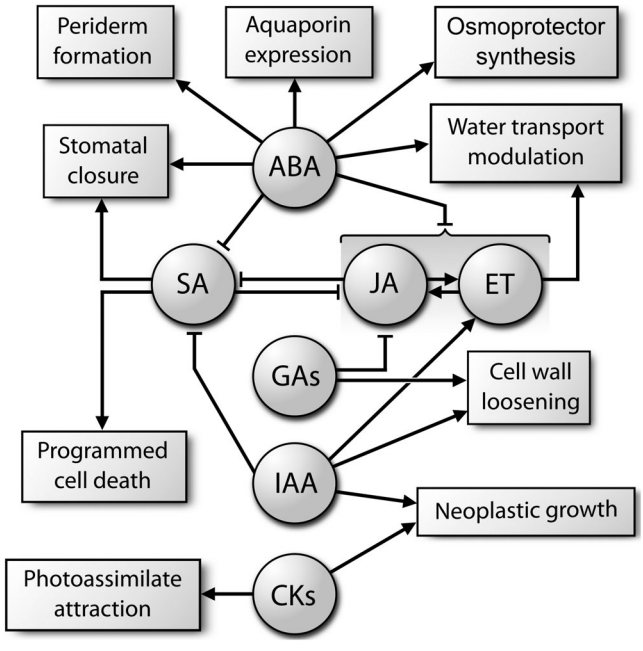

Fig 2. Phytohormone crosstalk and phytohormone-mediated susceptible responses. ABA, abscisic acid; CKs, cytokinins; ET, ethylene; GAs, gibberellins; IAA, indole-3-acetic acid (auxin); JA, jasmonic acid; SA—salicylic acid.

Auxin, CKs, GAs, and ABA participate in SRs not only due to their crosstalk with SA, JA, and ET but also *via* the direct modulation of complex metabolic processes in favour of a pathogen. Many SRs (see Section II) require the coordinated action of multiple gene products. Therefore, exploitation of host hormonal systems is a more effective way to systemically influence complex metabolic processes than targeting many individual gene products separately (Fig. 2).

PCW loosening is a normal physiological process that takes place during cell growth and fruit ripening and involves the coordinated action of many plant enzymes/proteins, such as expansins, endo-β-1,4-glucanases, xyloglucan endotransglucosylases/hydrolases (XETs), and pectinases (Sánchez-Rodríguez *et al.*, 2010; Cosgrove, 2015; Section II.2). The expression of many of the corresponding genes is controlled by auxin, CKs, and GAs. Therefore, a pathogen can induce host-mediated PCW loosening by exploiting these hormonal systems. *X. axonopodis* and *X. oryzae* increase the levels of auxin and GAs in infected plants, leading to the induction of auxin- and GA-mediated responses including those related to PCW loosening (Ding *et al.*, 2008; Cernadas & Benedetti, 2009). Neoplastic growth-causing pathogens induce auxin- and CK-mediated hormonal systems to enable PCW stretching required for cell division and extension (Devos *et al.*, 2005; Kay *et al.*, 2007; Section II.7). ABA is also likely to mediate PCW loosening since ABA deficiency increases the degree of pectin methylation that hampers PCW decomposition, leading to enhanced plant resistance to *B. cinerea* (Curvers *et al.*, 2010).

Pathogen-stimulated regulation of stomatal movements (Section II.1) is also thought to be implemented *via* the manipulation of different plant hormone systems (Melotto *et al.*, 2006, 2008; McLachlan *et al.*, 2014). The attraction of photoassimilates towards infected sites and the formation of green islands (Section II.4) are achieved by pathogen-induced activation of CK-mediated responses (Walters & McRoberts, 2006; Robert-Seilaniantz *et al.*, 2007; Depuydt *et al.*, 2009). The formation of tyloses and gels in the xylem vessels causing a reduction in the transpiration stream (Section II.3) is mediated by increased ET levels in infected plants. ET-deficient plants (in contrast to wild-type plants) do not form tyloses after *X. fastidiosa* infection and display resistance to this pathogen, while ET treatment induces tylose formation even in the absence of the pathogen (Pérez-Donoso *et al.*, 2007; Balaji *et al.*, 2008).

SR-related hormonal rearrangements are especially vivid during the development of galls. The activation of cell division and growth is well known to be due to increased levels of auxin and CKs in the gall (Sakakibara *et al.*, 2005; Lee *et al.*, 2009; Mashiguchi *et al.*, 2019). CKs also enhance photoassimilate flow into the gall to maintain the intensive metabolism of hypertrophic cells (Robert-Seilaniantz *et al.*, 2007; Walters, McRoberts & Fitt, 2008; Depuydt *et al.*, 2009). In addition, due to the positive effect of auxin on ET production (Abts *et al.*, 2017; Zemlyanskaya

et al., 2018), the synthesis of ET is activated in the gall (Riov & Yang, 1989; Vandenbussche *et al.*, 2003, 2010). In turn, ET induces the synthesis of ABA in the leaves of the infected plants, and ABA is transported into the gall to induce the synthesis of osmolytes and suberin (Efetova *et al.*, 2007). These reactions prevent water deficiency in the gall, neutralizing the negative effect of the breakdown of integumentary tissues during gall growth (Section II.7). Thus, auxin not only induces its conventional responses related to cell division and growth but also initiates systemic hormonal rearrangements that have fundamental importance for gall functioning. Moreover, the neovascularization of the gall and the formation of fibre-like cells (Section II.7) is also likely a result of hormonal rearrangements, the exact parameters of which remain to be determined.

(2) Reactive oxygen species

ROS are known participants of plant defence responses, fulfilling a range of resistance-related functions (O'Brien *et al.*, 2012; Huang *et al.*, 2019). However, in some cases, ROS have been shown to be regulators of SRs. ROS mediate different forms of PCD that may take part in both defence responses and SRs depending on the particular pathogen species and type of PCD (Section II.5). ROS accumulation particularly contributes to the progression of diseases caused by necrotrophic pathogens that consume nutrients released from lysed host cells (Williams *et al.*, 2011). Pro-oxidant treatment of plants increased their susceptibility to *B. cinerea* and *Sclerotinia sclerotiorum* (Govrin & Levine, 2000), while the inactivation of a gene for NADPH oxidase *Rboh* (respiratory burst oxidase homolog) led to a decrease in ROS levels coupled with increased resistance to *B. cinerea* (Asai & Yoshioka, 2009). In addition to ROS, reactive nitrogen species (RNS) were proposed to serve as a susceptibility factor since RNS content increases during the development of some diseases (Baarlen, Staats & Van Kan, 2004; Wang *et al.*, 2010; Sarkar *et al.*, 2014).

Although ROS are well known to provide PCW fortification by promoting the oxidative coupling of polysaccharides and lignin polymerization (Kärkönen & Kuchitsu, 2015), they may also cause non-enzymatic decomposition of some polysaccharides, leading to PCW loosening. ROS-mediated scission of polysaccharides resulting in PCW stretching was shown to occur during extension growth (Fry, 1998; Schopfer, 2001; Müller *et al.*, 2009). Therefore, it is reasonable to propose that ROS could also serve this function during pathogenesis. Indeed, plant-mediated reorganization of the PCW of xylem vessels during *Pectobacterium atrosepticum*-induced disease was shown to be coupled with ROS accumulation (Gorshkov *et al.*, 2016). This reorganization leads to the release of PCW polysaccharide (RG-I) into the vessel lumen, where it serves as an extracellular matrix for bacterial cells. Despite the high ROS level, no visible damage to bacterial cells was noticed.

Thus, to induce SRs, many phytopathogens target hormonal systems and ROS-biosynthetic enzymes of the host plants. By regulating the levels of phytohormones and ROS, microorganisms influence a wide range of host metabolic processes and thus systemically optimize the plant interior for their own benefit.

IV. BACTERIAL VIRULENCE FACTORS INDUCING PLANT SUSCEPTIBLE RESPONSES

To induce SRs in the host plant, phytopathogens possess specific factors that enable host manipulation: effectors, phytohormones, phytotoxins, and siderophores.

(1) Susceptible response-inducing effectors

Effectors are transported by bacteria directly into the cytoplasm (or, sometimes, to the plasma membrane) of host cells mostly *via* the type III, IV and VI secretion systems (Dou & Zhou, 2012). Most of the described effectors are repressors of phytoimmunity that act as blockers of defence-related signalling cascades or receptors of different elicitors (Khan *et al.*, 2018*a*); these effectors are outside the scope of this review. Some effectors act as inducers of plant SRs (see Section II). These effectors induce SRs *via* different regulatory mechanisms, including the direct activation of S-genes and manipulation of the ubiquitin–proteasome system (UPS) (Fig. 3).

(*a*) *Transcription activator-like effectors*

Some SR-inducing effectors belong to transcription activator-like effectors (TALEs) described for *Xanthomonas* species. They act as inducers of the transcription of plant S-genes and thus are referred to as trans-kingdom transcription factors (Mak *et al.*, 2013). TALEs interact with the plant basal transcriptional factor IIA (TFIIA), a component of the transcription preinitiation complex (PIC) that consists of three subunits (α, β, and γ). TALEs hijack TFIIA by binding to the α + γ subcomplex (i.e. substituting the β subunit) to enhance the transcription of specific S-genes (Ma *et al.*, 2018).

The targets of TALEs are different. PthXo1 (pathogenicity Xo1) and AvrXa7 (avirulence Xa7) effectors of *X. oryzae* pv. *oryzae* directly interact with *OsSWEET11* and *OsSWEET14* genes, respectively, inducing their expression (Chen *et al.*, 2010). Upregulation of *SWEET* genes leads to photoassimilate flow towards the pathogen (Section II.4). The *X. translucens* pv. *undulosa* transcription activator-like effector protein 8 (Tal8) enhances the expression of wheat *TaNCED* encoding 9-*cis*-epoxycarotenoid dioxygenase, a crucial enzyme for ABA biosynthesis. This leads to elevated ABA levels, resulting in the repression of SA-signalling and water-soaked lesion development (Peng *et al.*, 2019). The *Xanthomonas gardneri* AvrHah1 effector has multiple indirect targets in the host plant. This effector directly induces the expression of tomato transcription factors (TFs) (bHLH3 and bHLH6, basic helix–loop–helix 3/6). In turn, these

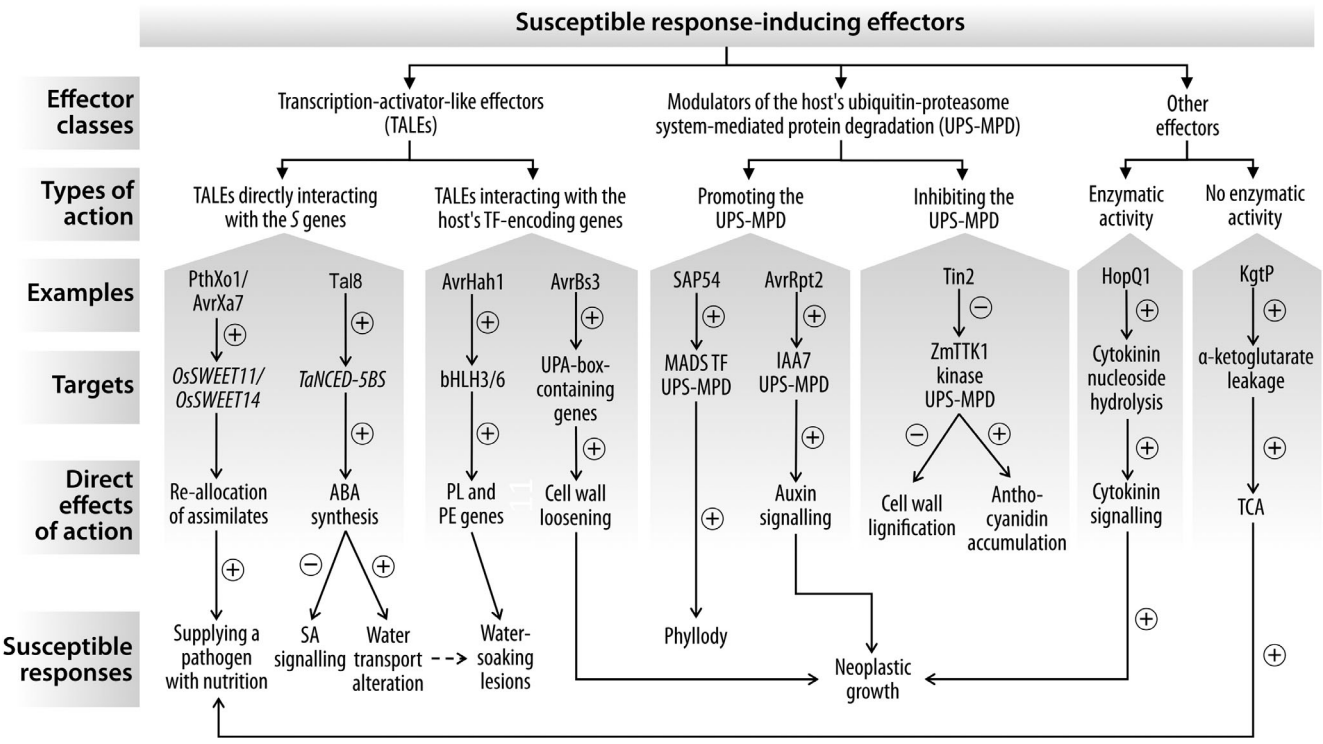

Fig 3. The action of bacterial effectors inducing host plant susceptible responses. See text for further details. +/−, positive or negative regulation. ABA, abscisic acid; AvrBs3, avirulence Bs3; AvrHah1, avirulence Hah1; AvrRpt2, avirulence Rpt2; AvrXa7, avirulence Xa7; bHLH3/6, basic helix–loop–helix 3/6; HopQ1, hypersensitive response and pathogenicity-dependent outer protein Q1; IAA7, indoleacetic acid-induced protein 7; KgtP, alpha-ketoglutarate permease; OsSWEET 11/14, *Oryza sativa* sugar will eventually be exported transporters 11/14; PE, pectinesterase; PL, pectate lyase; PthXo1, pathogenicity Xo1; *S* genes, susceptibility genes; SA, salicylic acid; SAP54, secreted aster yellows-witches' broom protein 54; Tal8, transcription activator-like effector protein 8; TaNCED, *Triticum aestivum* 9-cis-epoxycarotenoid dioxygenase; TALE, transcription-activator-like effector; TCA, tricarboxylic acid cycle; TF, transcription factor; UPA-box, upregulated by AvrBs3 box; UPS-MPD, ubiquitin-proteasome system-mediated protein degradation.

TFs enhance the expression of their target genes including those for pectate lyase and pectinesterase that presumably cause increased hygroscopicity of the PCW and water soaking of the infected leaf (Schwartz *et al.*, 2017; Section II.3).

Some TALEs induce the expression of tens of plant genes targeting both TF-encoding and non-TF-encoding genes. AvrBs3, one of the most studied TALEs produced by *X. campestris* pv. *vesicatoria*, enhances the expression of more than 20 genes that have the so-called UPA (upregulated by AvrBs3) box in their promoters (Kay *et al.*, 2009). One of the UPA genes, *upa20*, encodes the bHLH TF, acting as a master regulator of plant cell size (Kay *et al.*, 2007). Upa20 enhances the expression of genes encoding SAUR (small auxin up RNA), α-expansin, and pectate lyase, contributing to neoplastic growth (Marois *et al.*, 2002; Kay *et al.*, 2009; Section II.7).

(*b*) *Effectors that mediate the ubiquitin–proteasome system*

Many effectors have been shown to act *via* the UPS, causing (or preventing) the degradation of specific regulatory proteins (Fig. 3). Most of these effectors lead to the inactivation of immunity-related regulatory proteins (Göhre & Robatzek, 2008; Gimenez-Ibanez *et al.*, 2009; Kim, Stork & Mudgett, 2013; Chen *et al.*, 2017). However, some UPS-mediating effectors induce SRs. SAP54 (secreted aster yellows-witches' broom protein 54) produced by *Phytoplasma* species contributes to the malformation of flowers by deflecting shoot apical meristems from their genetically preprogrammed reproductive destiny. SAP54 causes the UPS-dependent degradation of MTFs (MADS-domain transcription factors) *via* interaction with the RAD23 protein. Since MTFs are required for flower development, their degradation enhances the transition of shoot apical meristems from generative to vegetative state. This leads to the formation of sterile leaf-like green flowers attractive to leafhoppers, which are the vectors for phytoplasma distribution (MacLean *et al.*, 2014; Wei *et al.*, 2019). By contrast, the Tin2 effector of fungus *Ustilago maydis* prevents the UPS-mediated degradation of its target, ZmTTK1 kinase, a positive regulator of anthocyanidin biosynthesis. This leads to enhanced anthocyanidin accumulation at the expense of lignification since the same precursor (4-coumaroyl CoA) is utilized for anthocyanidin and lignin biosynthesis (Tanaka *et al.*, 2014). In

other words, using Tin2, the pathogen induces the anthocyanidin pathway to divert metabolites away from lignin biosynthesis.

Some UPS-exploiting effectors can induce phytohormone signalling independently of phytohormone biosynthesis. The *P. syringae* AvrRpt2 effector causes the UPS-dependent degradation of AXR2/IAA7 (auxin-resistant protein 2/indoleacetic acid-induced protein 7) proteins (which are negative regulators of auxin responses), leading to the de-repression of auxin-regulated genes (Cui *et al.*, 2013). *P. syringae* also produces the HopZ1 (hypersensitive response and pathogenicity (Hrp)-dependent outer protein Z1) effector, which causes the acetylation and further UPS-dependent degradation of JAZ (jasmonate ZIM domain) proteins (which are negative regulators of jasmonate signalling). This enables the pathogen to induce jasmonate-mediated responses in order to antagonize SA-mediated ones (Jiang *et al.*, 2013; Section III.1).

The HopM1 effector of *P. syringae* causes the UPS-mediated degradation of MIN7, a guanine nucleotide exchange factor involved in vesicle trafficking necessary for the endocytic recycling of the plasma membrane (Nomura *et al.*, 2006). HopM1-mediated degradation of MIN7 seems to compromise the integrity of the host cell membrane and contribute to the establishment of wet apoplast symptoms (Xin *et al.*, 2016; Section II.3). In addition to HopM1, another effector, AvrE, induces the formation of wet apoplast, although the action of AvrE is mediated by a different mechanism than the UPS-dependent one (Xin *et al.*, 2016).

(*c*) *Other effectors*

For some SR-inducing effectors, the mechanisms of action are either not clear to date or are unrelated to transcription activation and UPS-dependent protein degradation. Some SR effectors possess catalytic activities. The HopQ1 effector found in *Pseudomonas*, *Xanthomonas*, and *Ralstonia* species was shown to mimic plant cytokinin-activating nucleoside hydrolases [LONELY GUY (LOG)] and to increase the free cytokinin level, causing the downregulation of defence systems (Hann *et al.*, 2014). The effector ripTPS (*Ralstonia* protein injected into plant cells trehalose-6-phosphate synthase) from *Ralstonia solanacearum* phosphorylates trehalose, yielding trehalose-6-phosphate, which is involved in a large number of cellular processes and serves as a substrate for the pathogen (Poueymiro *et al.*, 2014). *P. syringae* HopX1, with protease activity, cleaves JAZ proteins (Gimenez-Ibanez *et al.*, 2014).

KgtP (alpha-ketoglutarate permease) of *X. oryzae* pv. *oryzae* is a unique effector due to its specific localization in the host cell plasma membrane. KgtP acts as a permease for α-ketoglutarate (an intermediate of the tricarboxylic acid cycle) causing its leakage from the host cell into the apoplast where it is captured by the pathogen (Guo *et al.*, 2016). The loss of α-ketoglutarate forces the plant to increase its biosynthesis and thus permanently to provide the pathogen with a growth substrate.

Several effectors of *P. syringe* are able to manipulate stomatal movement. HopM1, HopF2, and AvrB prevent PAMP-induced stomatal closure (Hurley *et al.*, 2014; Lozano-Durán *et al.*, 2014; Zhou *et al.*, 2015), while HopAM1, conversely, stimulates stomatal closure at the post-invasive infection stage (Goel *et al.*, 2008; Section II.1). HopAM1 increases plant sensitivity to ABA (without altering ABA levels) and thus induces abiotic stress-related reactions (including ABA-dependent stomatal closure) at the expense of defence responses. However, the mechanism by which HopAM1 increases ABA sensitivity remains unknown. AvrPtoB of *P. syringe* also induces ABA-regulated outcomes at the expense of the defence responses by upregulating the ABA-biosynthetic gene *via* an unknown mechanism (de Torres-Zabala *et al.*, 2007).

Some effectors induce SR *via* unknown or only partially elucidated mechanisms. Among these are HopG1 and HopE1 of *P. syringe* and DspE (disease-specific protein E) synthesised by *Erwinia amylovora* and *Pectobacterium* species. HopG1 and HopE1 affect the organization of the cytoskeleton that coordinates most (if not all) metabolic processes in the cells (Guo *et al.*, 2016; Shimono *et al.*, 2016). DspE induces programmed cell death (Section II.5) *via* the indirect inhibition of serine palmitoyltransferase, resulting in the depletion of sphingolipid precursors (Siamer *et al.*, 2014). Thus, effectors act not only as repressors of immunity but in addition they can induce different host plant SRs *via* the activation of plant S-gene expression, serving as trans-kingdom transcription factors, by the modulation of ubiquitin-mediated degradation of regulatory proteins, in the formation of pores for nutrient leakage, the enzymatic conversion of primary metabolites and phytohormone precursors, and presumably *via* other mechanisms that remain to be determined.

(2) Phytopathogen-derived phytohormones

Many (if not all) plant SRs are mediated by phytohormones (Section III.1). Within a pathosystem, phytohormones can be synthesized not only by the plant but also by some pathogens. The role of bacteria-produced phytohormones is best understood in *Pseudomonas savastanoi*, *Pantoea agglomerans*, *Rhodococcus fascians*, and *Agrobacterium tumefaciens* which cause neoplastic growth using self-produced auxin and CKs. These two phytohormones cause enhanced cell proliferation and growth, cell wall loosening and stretching, photoassimilate flow towards the gall, and a general shift in hormonal homeostasis, which is necessary for the coordination of many physiological processes in the gall (Sections II.7 and III.1).

Crown galls caused by *A. tumefaciens* produce large amounts of auxin and CKs that stimulate extensive cell proliferation and growth (Sakakibara *et al.*, 2005; Lee *et al.*, 2009; Mashiguchi *et al.*, 2019). The hyperproduction of these two phytohormones in the gall may be a consequence of the expression of either plant genes or pathogen genes integrated into the plant genome or both. Although the relationship between plant- and *A. tumefaciens*-derived auxin and CKs in the gall is not known, *A. tumefaciens* phytohormone-related genes were shown to contribute to virulence (Liu

et al., 1982; Hwang *et al.*, 2013). Three other neoplastic growth-inducing bacteria, *P. savastanoi*, *R. fascians*, and *P. agglomerans*, do not integrate auxin/CK-related or other genes into the plant genome, but produce these hormones themselves. For *P. savastanoi*, self-produced auxin and CKs were shown to be required for gall formation (Iacobellis *et al.*, 1994), while *P. agglomerans* requires self-produced auxin and CKs for the enlargement of galls but not for their formation (Manulis & Barash, 2003; Chalupowicz *et al.*, 2006). Both virulent (leafy-gall-inducing) and avirulent strains of *R. fascians* synthesize CKs; only virulent strains can produce methylated CKs (1- and 2-methyl isopentenyladenine) that seem to be required for gall formation (Jameson *et al.*, 2019). Thus, the roles of pathogen-produced auxin and CKs in the formation of a pathosystem depend on the particular pathogen species involved.

The production of CKs is not restricted to neoplastic growth-inducing pathogens. Some fungi synthesize this phytohormone to stimulate the formation of 'green islands' – photosynthetically active green areas surrounded by yellow senescing tissues (Walters & McRoberts, 2006). Green islands attract photoassimilates (creating nutrient sinks) to the infected sites, prolonging the life of host cells. Induction of green island formation *via* the production of CKs was also shown for the bacteria *Wolbachia* sp. These bacteria are endosymbionts of leaf-miner larvae (*Phyllonorycter blancardella*) (Kaiser *et al.*, 2010), whose larvae benefit from endosymbiont-induced formation of green islands for feeding.

GAs are also synthesized not only by plants but also by bacteria and fungi. These phytohormones were first described not in plants, but in the fungus *Fusarium fujikuroi* (formerly *Gibberella fujikuroi*) (Kurosawa, 1926), as the causative agent of 'foolish seedling' – a pathological elongation of stems (Tudzynski, 1999). GAs were also identified in diazotrophic bacteria (Bastián *et al.*, 1998; Piccoli *et al.*, 2011; Méndez *et al.*, 2014). Some plant pathogenic bacteria (*X. oryzae*, *X. translucens*, *X. bromi*, and *Erwinia tracheiphila*) are also likely to synthesize GAs since all genes necessary for GA production were found in some strains of these bacteria (Nagel & Peters, 2017*a*). GA-related genes of *X. oryzae* were cloned, and the corresponding recombinant enzymes were shown to catalyse the synthesis of active GAs (Nagel & Peters, 2017*b*). Knockout of these genes reduced the virulence of *X. oryzae* and caused the upregulation of JA-mediated defence responses (Lu *et al.*, 2015), indicating that at least one role of pathogen-produced GAs is related to their antagonistic interaction with JA.

The phytohormone ET is also known to be produced by many bacteria, including both phytopathogenic bacteria [*Agrobacterium tumefaciens*, *Pseudomonas* (*Ralstonia*) *solanacearum*, *Pseudomonas pisi*, some *Xanthomonas* species, and some strains of *P. syringae*] and those that are not associated with plants (Swanson, Wilkins & Kennedy, 1979; Fukuda, Ogawa & Tanase, 1993; Weingart & Volksch, 1997). The physiological role of ET for bacteria (especially those that do not colonize plants) is largely unknown. However, it is reasonable to speculate that the production of ET by plant pathogenic bacteria contributes to host plant manipulation. An increase in ET level in diseased plants after infection by *Pseudomonas syringae* pv. *glycinea* and *P. syringae* pv. *phaseolicola* was shown to be mainly due to its synthesis by the pathogens but not the host plants (Weingart & Volksch, 1997). Moreover, the virulence of the strains was dependent on their ability to synthesize ET. By contrast, ET synthesized during *P. solanacearum*-, *Xanthomonas citri*-, and *B. cinerea*-induced disease was shown to be mostly of plant rather than bacterial/fungal origin (Pegg & Cronshaw, 1976; Goto, Yaguchi & Hyodo, 1980; Lu, Tzeng & Hsu, 1989; Cristescu *et al.*, 2002). Thus, the induction of ET-mediated SRs may be due to ET of different origins depending on the particular host and pathogen species involved.

ABA-producing phytopathogenic bacteria have not been described to date. However, several phytopathogenic fungi (e.g. *B. cinerea*, *Cercospora rosicola*, and *Magnaporthe oryzae*) (Assante, Merlini & Nasini, 1977; Hirai *et al.*, 2000; Jiang *et al.*, 2010) and rhizosphere bacteria (Forchetti *et al.*, 2007; Piccoli *et al.*, 2011; Salomon *et al.*, 2014; Lievens *et al.*, 2017) are able to synthesize ABA. ABA-deficient *M. oryzae* mutants were less virulent compared to the wild-type fungi (Spence *et al.*, 2015). It is reasonable to speculate that the production of ABA by phytopathogens enables them to induce abiotic stress-related plant responses at the expense of defence reactions; however, the particular SRs induced by pathogen-produced ABA remain to be elucidated.

Taken together, almost all phytohormones (except for ABA) can be synthesized by various phytopathogenic bacteria. These synthetic abilities enable pathogens to induce complex SRs implemented by multiple hormone-regulated gene products.

(3) Bacterial phytotoxins

Most known phytotoxins cause the death of host plant cells by inhibiting vital enzymes or disturbing membrane integrity (Duke & Dayan, 2011; Bignell, Fyans & Cheng, 2014). However, some phytotoxins are inducers of SRs. The best known among them are syringolin A and coronatine produced by different strains of *P. syringae*. These phytotoxins enable bacteria to maintain the stomata in the open state (Section II.1). Syringolin A prevents DAMP (damage associated molecular pattern)-induced stomatal closure *via* modulation of the SA-mediated signalling pathway by repressing the UPS-dependent turnover of NPR-1 (nonexpressor of pathogenesis-related genes 1), a crucial component of SA-signalling (Schellenberg, Ramel & Dudler, 2010). Coronatine causes the reopening of stomata (after their PAMP-induced closure) *via* the inhibition of the Arabidopsis H^+-ATPase 1 (AHA1) and AHA2 and activation of K^+ influx into the guard cells (Melotto *et al.*, 2017). In addition, coronatine, as a molecular mimic of JA, induces JA-regulated responses *via* the promotion of UPS-mediated degradation of JAZ transcriptional repressors. Due to the antagonistic interaction of JA- and SA-regulated responses, the action of

coronatine is advantageous for *P. syringae* due to the high susceptibility of these bacteria to SA. Genes for coronatine-related phytotoxins were also found in *Pectobacterium* and *Streptomyces* species (Bell *et al.*, 2004; Bignell *et al.*, 2014; Panda *et al.*, 2016). Knockout of these genes in *Pectobacterium atrosepticum* results in an inability to cause plant rots and induce JA-regulated genes (Bell *et al.*, 2004; Gorshkov *et al.*, 2018). Another non-lethal phytotoxin is thaxtomin A produced by phytopathogenic streptomycetes (Goyer, Vachon & Beaulieu, 1998). Thaxtomin A inhibits cellulose synthase complex and thus causes changes in the composition of the PCW, including a decrease in cellulose and increase in pectins and cross-linking glycans (Bischoff *et al.*, 2009).

(4) Siderophores

Siderophores are low-molecular-weight iron carriers that are produced by many living organisms to obtain iron from the environment (Khan, Singh & Srivastava, 2018*b*). Plant pathogenic bacteria also produce siderophores to outcompete host plant ferritins in iron acquisition and thus access host plant iron. In addition, the siderophores chrysobactin and deferrioxamine (produced by *Dickeya dadantii* and *Erwinia amylovora*, respectively) appear to act as signals towards host plant cells. These siderophores induce a leaf-to-root iron deficiency signal and thereby activate iron uptake by the roots (Dellagi *et al.*, 2009; Segond *et al.*, 2009). The treatment of plants with chrysobactin increased their susceptibility to *D. dadantii*, while plants grown under iron deficiency displayed increased resistance to this pathogen (Kieu *et al.*, 2012). This indicates that chrysobactin (and probably deferrioxamine) induce SRs related to increased iron uptake for the benefit of the pathogen.

Thus, phytopathogenic bacteria can produce specific metabolites that provide global modification of the host plant metabolism. The action of these metabolites results in the transformation of a plant from a relatively autonomous biological unit into a component of an integrated pathosystem.

V. CONSEQUENCES OF PLANT SRs AND CURRENT PERSPECTIVES

Plant SRs can be divided into two main categories depending on their consequences. The first category includes SRs that are necessary for the multiplication of a pathogen inside the host; without these SRs, the proliferation of a microorganism *in planta* is impossible, and disease symptoms do not occur. For example, *Pseudomonas syringae* is unable to propagate (and, therefore, to cause disease) in the host plant if host PCD-related SRs are repressed (Richael *et al.*, 2001). Thus the induction of PCD is necessary to ensure the normal development of *P. syringae* in the host. Similarly, the SR related to the increased synthesis of jasmonates is required for *Xanthomonas campestris* reproduction *in planta*; knockout or silencing of plant genes for jasmonate-biosynthetic enzymes represses the growth of these bacteria in the host (O'Donnell *et al.*, 2003).

The second category of SRs includes that host plant responses that are not required for pathogen propagation *in planta* but are necessary for the manifestation of disease symptoms. Absence of SRs of this category does not hamper pathogen reproduction in the host plant but prevents the pathological process (Norman & Alvarez, 1994; Kover & Schaal, 2002; Block *et al.*, 2005; Swanson *et al.*, 2005). Thus, repression of SRs of the second category leads not to plant resistance but to plant tolerance – a state in which a plant does not show a significant reduction in fitness despite high pathogen numbers *in planta* (Kover & Schaal, 2002). Plant tolerance to pathogens is associated with the development of latent infections (Fig. 4).

The existence of these two types of SR can be seen clearly in the example of *Pseudomonas syringae–A. thaliana* interactions (Kloek *et al.*, 2001). The induction of JA-regulated responses by coronatine enables *P. syringae* to repress SA synthesis and to cause disease. This pathogen cannot propagate in JA/coronatine-insensitive plants due to the accumulation of SA. However, *P. syringae* develops the ability to reproduce in JA/coronatine-insensitive plants if the accumulation of SA is repressed by heterologous expression of the SA-hydrolase gene *nahG*. Nevertheless, the disease symptoms are not manifested in JA/coronatine-insensitive plants with a heterologous *nahG* gene despite active pathogen propagation (at similar levels of propagation to that in wild-type plants) (Kloek *et al.*, 2001). Thus, *P. syringae* induces two types of JA-mediated SRs. The first provides the conditions for pathogen proliferation by repressing SA synthesis. The second has no effect on pathogen propagation but induces disease manifestation.

Many examples demonstrate that the repression of SRs prevents the formation of disease symptoms (or significantly delays their development) without inhibiting pathogen growth. *Xanthomonas campestris*, *Pseudomonas syringae*, and *Clavibacter michiganensis* induce ET-mediated SRs. These pathogens reproduce in the ET-insensitive mutants as effectively as in the wild-type plants but do not induce symptom development (Bent *et al.*, 1992; Lund, Stall & Klee, 1998; Balaji *et al.*, 2008). The suppression of SA synthesis has no effect on *X. campestris* growth *in planta* but prevents disease development (O'Donnell *et al.*, 2003). Similarly, the silencing of the apoplastic invertase-encoding gene does not inhibit the proliferation of *Xanthomonas campestris* pv. *vesicatoria* but delays the manifestation of disease symptoms (Kocal *et al.*, 2008). These examples clearly indicate that the development of pathologies is a direct consequence of active host plant responses rather than the action of enzymes/toxins of pathogen origin.

Plant tolerance to pathogens can be achieved not only by genetic manipulations (mutations in S-genes) but also by the modulation of plant physiological processes. For example, the inoculation of one tomato (*Solanum lycopersicum*) leaf with *X. campestris* or *P. syringae* results in the intensive development of disease symptoms on this leaf and

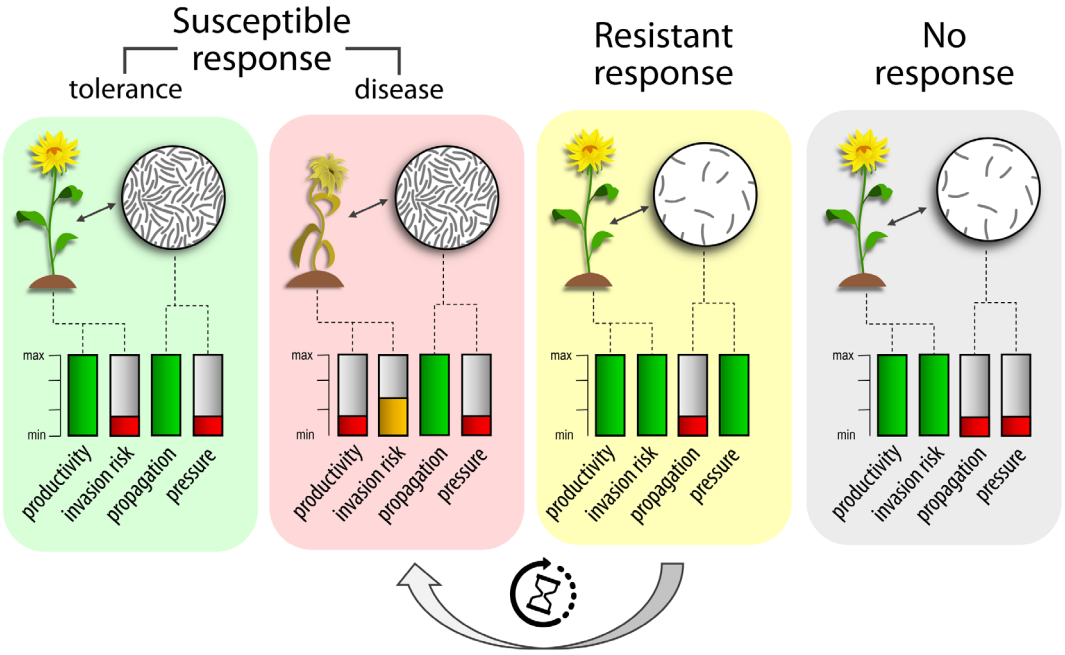

Fig 4. Summary scheme of the consequences of different types of susceptible responses compared to the defence response and pathogen insensitivity. The columns show the relative levels of plant productivity, risk of invasion of the alternative pathogens, pathogen propagation *in planta*, and selective pressure exerted on a pathogen.

simultaneously induces a specific systemic response that leads to a decrease in host plant susceptibility; when a plant is reinoculated on another leaf, the disease symptoms on the second leaf are manifested to a reduced extent compared to the first inoculation (Block *et al.*, 2005). At first glance, this effect resembles systemic acquired resistance (SAR). However, SAR leads to the inhibition of pathogen growth, while the above-described phenomenon, termed systemic acquired tolerance (SAT), is not associated with the repression of pathogen proliferation (Block *et al.*, 2005). The phenomenon of SAT shows that under a particular physiological state, different types of SRs may be induced differentially: SRs that promote pathogen proliferation *in planta* are induced, while those that are responsible for disease symptom development are not. This is consistent with the fact that plants genetically susceptible to a particular pathogen may not show disease symptoms in spite of heavy colonization by this pathogen (Grimault & Prior, 1993; McGarvey, Denny & Schell, 1999; Baumgartner & Warren, 2005; Wistrom & Purcell, 2005; Gambetta *et al.*, 2007).

Numerous examples of asymptomatic plant infections suggest that the peaceful coexistence (equilibrium) of a plant and a pathogen is a natural scenario, while the development of pathologies is a consequence of a disturbance of this equilibrium. Moreover, the survival of the host plant is important to the pathogen since a reduction in host fitness will endanger pathogen survival. A significant task for contemporary phytopathology is to elucidate why and how the apparently mutualistic plant–pathogen interaction is transformed into an antagonistic one. The interaction of a particular plant and pathogen is likely to be determined by a set/timing/strength of SRs induced or repressed as a result of the cross-talk between two organisms. The possibility of external control of the manifestation of SRs may be a promising agricultural approach for plant disease management.

An approach related to the control of plant SRs *via* S-gene loss-of-function has already been introduced into agriculture. The best-known example of this is the breeding of barley (*Hordeum vulgare*) Mlo (Mildew locus o)-deficient varieties. Mlo-deficient plants are not affected by the causative agent of powdery mildew *Blumeria graminis* (formerly *Erysiphe graminis*) since this pathogen is then unable to induce Mlo-mediated SRs (Pavan *et al.*, 2010). However, Mlo loss-of-function is known to have negative effects on plant fitness. First, Mlo deficiency results in a plant growth/developmental defect that is a typical consequence of S-gene loss-of-function (van Schie & Takken, 2014). However, this negative effect can largely be eliminated by introgression into the right genetic background (Bjørnstad & Aastveit, 1990). Second, although Mlo deficiency leads to plant resistance to *B. graminis*, it simultaneously results in increased susceptibility to some other phytopathogens (*Cochliobolus sativus*, *Magnaporthe grisea*, and *Fusarium graminearum*) compared to Mlo-positive plants (Jarosch, Kogel & Schaffrath, 1999; Kumar *et al.*, 2001; Jansen *et al.*, 2005). The underlying reason is likely to be that Mlo-deficient plants do not support *B. graminis* growth, with the 'econiche' occupied by *B. graminis* thus becoming vacant in Mlo-negative plants for occupation by other pathogens. It thus follows that Mlo-positive plants are protected by *B. graminis* from other invaders.

Thus, although pathogens are almost always considered as enemies, it may be the case that they also provide benefits to the host plant, e.g. enhanced resistance to other pathogens or herbivores (Crute & Pink, 1996; Lund *et al.*, 1998; Kover & Schaal, 2002) or increased drought tolerance (Reusche *et al.*, 2012; Section II.3). To emphasize these potentially beneficial traits of pathogens, the term "conditionally beneficial pathogens" was proposed (Partida-Martínez & Heil, 2011). Immune plants that remove a pathogen eliminate not only its negative but also its beneficial effect. The virulence and aggressiveness of pathogens seem to be restored much faster due to rapid pathogen evolution under a selective pressure compared to the restoration of beneficial traits that have arisen during prolonged co-evolution. Therefore, plant breeding should focus on obtaining not immune pathogen-free plant varieties but stable 'equilibrated' pathosystems with tolerant plants and mutualistic pathogens causing asymptomatic harmless infections that provide benefits to the host plant (Fig. 4).

Currently, latent infections are considered objectively as harmful as we cannot control the transition from the asymptomatic stage of infection to the acute one, or even forecast such transitions. However, if such transitions can be controlled (prevented), asymptomatic pathogen colonization would not be a concern. Importantly, tolerant plants were shown to produce ample yields even when they are infected by pathogens (Agrios, 2004).

The comprehensive investigation of plant SRs (especially those that do not promote pathogen reproduction but exacerbate disease development) is key to a deeper understanding of the fundamental basis of the peaceful and antagonistic coexistence of plants and pathogens. Deciphering the regulatory mechanisms of SRs will allow us to understand how the plant–pathogen equilibrium is maintained or disturbed. These regulatory mechanisms can be targeted using contemporary genome editing technologies in order to obtain agricultural plants tolerant to a variety of pathogens. This approach would be beneficial for crop production since it could form a basis for pesticide-free control of pathological symptoms, which are facultative features of plant–pathogen interactions largely determined by host plant SRs.

VI. CONCLUSIONS

(1) Individual plants, even those possessing genetically determined predispositions, are not susceptible to a particular pathogen *a priori*. This susceptibility arises when specific plant SRs are induced.
(2) SRs reflect normal physiological reactions that can be activated by pathogens in those cases when they are normally repressed. Pathogens recruit such host reactions in order to transform the plant organism into a beneficial ecological niche.
(3) The induction of SRs is often carried out *via* the modulation of the hormonal system and ROS levels.
(4) Phytopathogens can make plants more susceptible *via* the production of virulence factors (some effector proteins, pathogen-produced phytohormones, some toxins, and some siderophores).
(5) SR-related reactions are modulated not only by pathogens but also by a variety of factors (e.g. environmental conditions, growth/ontogenetic stage, and tissue type). The set of induced SRs in each particular case largely determines the outcome of the interaction of a pathogen with its host.
(6) Different SRs may lead either to true susceptibility associated with extensive symptom manifestation or to plant tolerance, which reflects an equilibrium between the host and pathogen.
(7) Understanding SRs in greater depth has high potential for the development of novel plant-protection approaches. The hierarchical regulation of SRs provides a window of opportunity for making S-genes insensitive to pathogen manipulation without their knockout, which often causes unwanted negative effects. In turn, the SRs that allow equilibrium should be used to maintain the apparently mutualistic host–pathogen interaction since many pathogens are conditionally beneficial and may contribute to host plant survival.

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