Disease resistance or growth: the role of plant hormones in balancing immune responses and fitness costs

Nicolas Denancé1,2,†, Andrea Sánchez-Vallet3,4,5,†, Deborah Goffner1,2 and Antonio Molina4,5

1 UFR 5546, Laboratoire de Recherche en Sciences Végétales, Université de Toulouse, Castanet-Tolosan, France
2 UFR 5546, Laboratoire de Recherche en Sciences Végétales, Centre National de la Recherche Scientifique, Castanet-Tolosan, France
3 Centro de Biotecnología y Genómica de Plantas, Instituto Nacional de Investigación y Tecnología Agraria y Alimentaria, Universidad Politécnica de Madrid, Pozuelo de Alarcón, Spain
4 Centro de Biotecnología y Genómica de Plantas, Instituto Nacional de Investigación y Tecnología Agraria y Alimentaria, Universidad Politécnica de Madrid, Pozuelo de Alarcón, Spain
5 Departamento de Biotecnología, Escuela Técnica Superior de Ingenieros Agrónomos, Universidad Politécnica de Madrid, Madrid, Spain

Edited by: Serge Marin-Ochoa, University of Barcelona, Spain
Reviewed by: Victor Park, Universitat Jaume I, Spain
*Correspondence: Nicolas Denancé, Laboratoire des Interactions Plantes-Microorganismes, UMR INRA/CNRS 441/2594, 24 Chemin de Borde Rouge, B.P.5207, Aucuney, 31220 Castanet-Tolosan, France. e-mail: denancen@toulouse.inra.fr
†Nicolas Denancé and Andrea Sanchez-Vallet have contributed equally to this work.

INTRODUCTION

In their natural environments, plants are under continuous biotic stress caused by different attackers (e.g., bacteria, fungi, viruses, oomycetes, and insects) that compromise survival and offspring. Given that green plants are the ultimate source of energy for most organisms, it is not surprising that plants have evolved a variety of resistance mechanisms that can be constitutively expressed or induced after pathogen or pest attack (Glazebrook, 2005; Panstruga et al., 2009). Plants have developed molecular mechanisms to detect pathogens and pests and to activate defense responses. The plant innate immune system relies on the specific detection by plant protein recognition receptors (PRRs) of relatively conserved molecules of the pathogen called pathogen-associated molecular patterns (PAMPs). This resistance response is known as PAMP-triggered immunity (PTI). Successful pathogens secrete effector proteins that deregulate PTI. To counteract this, plant resistance (R) proteins recognize effectors and activate effector-triggered immunity (ETI) (reviewed in Dodds and Rathjen, 2010).

A fine-tune regulation of these immune responses is necessary because the use of metabolites in plant resistance may be detrimental to other physiological processes impacting negatively in other plant traits, such as biomass and seed production (Walters and Hehl, 2007; Kempel et al., 2011). These physiological constraints, together with other factors such as the co-existence of plants with natural attackers, have contributed to drive the evolution of a dynamic and complex network system. Defense layers from separate cellular components and from diverse physiological processes are interconnected to reduce the inherent fitness cost of being well-defended (Chisholm et al., 2006; Panstruga et al., 2009; Schulze-Lefert and Panstruga, 2011). The resistance response is regulated by phytohormones, that are small molecules which synergistically and/or antagonistically work in a complex network to regulate many aspects of plant growth, development, reproduction, and response to environmental cues (Pieterse et al., 2009; Santer et al., 2009; Jaffé and Chory, 2010). Recent progresses have been made in understanding the complex hormone network associated fitness costs. The molecular mechanisms that govern these hormonal networks are largely unknown. Moreover, hormone signaling pathways are targeted by pathogens to disturb and evade plant defense responses. In this review, we address novel insights on the regulatory roles of the ABA, SA, and auxin in plant resistance to pathogens and we describe the complex interactions among their signal transduction pathways. The strategies developed by pathogens to evade hormone-mediated defensive responses are also described. Based on these data we discuss how hormone signaling could be manipulated to improve the resistance of crops to pathogens.

Keywords: abscisic acid, auxin, hormone crosstalk, pathogens, salicylic acid, trade-off, virulence factor

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Auxins are a group of molecules including IAA (indole-3-acetic acid), ABA, auxin, gibberellins, and cytokinins pathways are considered flagellin protein (Boller and Felix, 2009; Pel and Pieterse, 2012), with other hormone signaling pathways or with PTI (Robert-Seilaniantz et al., 2011a). Additionally, other hormones such as auxins and abscisic acid (ABA), originally described for their function in the regulation of plant growth processes and the response to abiotic stresses, have recently emerged as crucial players in plant-pathogen interactions (Mauch-Mani and Mauch, 2005; Kazan and Manners, 2009; Ton et al., 2009; Fu and Wang, 2011). All the phytohormone pathways are linked to each other in a huge, complex and still obscure network. For example, ET, ABA, auxin, gibberellins, and cytokinins pathways are considered as hormone modulators of the SA–JA signaling backbone (Pieterse et al., 2012).

To develop hormone-based breeding strategies aiming to improve crop resistance to pathogens, we need to understand the intricate regulation of hormone homeostasis during plant–pathogen interactions, and how pathogens interfere with this hormone regulation. Indeed, manipulation of a plant hormone pathway can result in enhanced resistance to a particular pathogen, but it could also have a strong negative effect on plant growth and resistance to a distinct type of pathogen with a different life style (Holeski et al., 2012). In this review, we will discuss novel insights on the complex role of phytohormones in balancing plant innate immunity and development, with a special focus on the regulatory crosstalk of auxins, SA, and ABA. We will also learn about decoy strategies employed by the attackers to disturb hormone-mediated defense responses in plants, and we will describe how misregulation of these hormone pathways leads to strong effects on developmental features and on disease resistance to pathogens. Finally, we will discuss the potential of manipulating hormone homeostasis/signaling to improve crop resistance to pathogens.

HORMONE REGULATORY NETWORKS IN DISEASE RESISTANCE

Auxins
Auxins are a group of molecules including IAA (indole-3-acetic acid) that regulate many aspects of plant development, such as apical dominance, root gravitropism, root hair, lateral root, leaf, and flower formation, and plant vasculature development (Kieffer et al., 2010; Swarup and Péret, 2012). Both direct and indirect effects of auxins on the regulation of pathogen resistance responses in plants have been described (Kazan and Manners, 2009).

Indirect effects may be caused by auxins regulation of development-associated processes, such as cell wall architecture, root morphology, and stomata pattern. For example, treatment of rice with IAA impaired the resistance to Xanthomonas oryzae pv. oryzae probably as a consequence of the activation of the biosynthesis of cell wall-associated expansins that lead to cell wall loosening, which facilitates pathogen growth (Ding et al., 2008).

Auxins can negatively impact plant defense by interfering with other hormone signaling pathways or with PTI (Robert-Seilaniantz et al., 2011a). The bacterial PAMP flg22, a peptide from flagellin protein (Boller and Felix, 2009; Pel and Pieterse, 2012), induces an Arabidopsis microRNA (miR393), which negatively regulates the mRNA levels of auxins receptors TIR1 (transport inhibitor response 1), AFB2 (auxin signaling F-box 2), and AFR3. Thus, the flg22-triggered suppression of auxin signaling leads to increased resistance to the bacterium Pseudomonas syringae pv. tomato DC3000 (Park et al. 2006) and also to the oomycete Hyaloperonospora arabidopsidis (Navarro et al., 2006; Robert-Seilaniantz et al., 2011b). The flg22-induced resistance to these biotrophic pathogens was explained by the observed induction of the SA signaling pathway. Supporting this hypothesis, it was found independently that treatment of Arabidopsis leaves with flg22 induces SA accumulation (Tsuda et al., 2008).

In Arabidopsis, SA treatment stabilizes the Aux/IAA proteins, leading to down-regulation of the expression of auxin-related genes. Moreover, the enhanced susceptibility to P. syringae pv. maculicola 4326 (Park et al., 2006) of plants expressing the NahG gene (encoding a bacterial salicylate hydroxylase that degrades SA) is partially reverted by the aux2-1 mutation, that disrupts auxin signaling, further indicating that auxin signaling is part of the SA-induced resistance signaling pathway (Wang et al., 2007). Interaction between SA and auxins was further clarified by the characterization of the regulatory pattern of GH3.3 gene, which is involved in auxin homeostasis in Arabidopsis plants. Lines overexpressing GH3.3 have lower levels of Aux/IAA proteins, overexpression of SA signaling pathway and enhanced resistance to P. syringae (Park et al., 2007). Moreover, these transgenic lines also displayed enhanced resistance to abiotic stress and induction of the ABA regulatory pathway (Park et al., 2007).

The conjugated auxin–aspartic acid (IAA–Asp) has been recently reported to play a key role in regulating resistance to the necrotrophic fungus Botrytis cinerea and PstDC3000. In Arabidopsis, tomato, and Nicotiana benthamiana infected with these pathogens there is an enhanced expression of GH3.2 and GH3.4 genes, which encode two enzymes required for conjugation of auxins with Asp. Thus, upon pathogen infection, accumulation of IAA–Asp takes place, promoting the development of disease symptoms in infected plants (Conejero-Lamothe et al., 2013). The negative effects of auxins on the activation of plant resistance is further supported by the observed enhanced susceptibility of auxin-treated rice to X. oryzae (Ding et al., 2008) and of auxin-treated Arabidopsis to PstDC3000 (Navarro et al., 2006) and Fusarium culmorum, leading to enhanced resistance to F. oxysporum (Kidd et al., 2011). Nevertheless, auxins have also been shown to positively regulate Arabidopsis immunity as aux2-1 and aux1-1 mutants were more susceptible than wild-type plants to the necrotrophic fungi B. cinerea and Plectosphaerella cucumerina (Llorente et al., 2008).

One of the biosynthetic pathways of auxins is partially shared with those required for the biosynthesis of tryptophan-derived antimicrobials, such as indole glucosinolates and camalexin. This might lead to competition for the biosynthetic precursor of auxin and antimicrobials (Barlier et al., 2008; Grubb and Abel, 2006). The recently characterized Arabidopsis wat1 (watts are thin1) mutant exhibits specific enhanced resistance to vascular pathogens such as Ralstonia solanacearum. This response was associated to a misregulation of tryptophan derivatives (i.e., lower...
Abscisic acid is an isoprenoid compound that regulates developmental processes, such as seed development, desiccation, and dormancy (Wasilewska et al., 2008). In addition, the function of ABA as a regulator of abiotic stress has been thoroughly described (Shinozaki and Yamaguchi-Shinozaki, 2007). ABA has also emerged as a complex modulator of plant defense responses (Asselebergh et al., 2008; Feng et al., 2012; Sánchez-Vallet et al., 2012). ABA can function as a positive or a negative regulator of plant defense depending on the plant–pathogen interaction analyzed (Mash-Chani and Mash, 2005; Asselbergh et al., 2008; Ton et al., 2009). ABA-impaired (biosynthesis or signaling) mutants in tomato (situs) and Arabidopsis (abi1-1, abi2-1, abi1-6, abi2-12, aaos-2, and pyr1pyl2pyl4) were shown to overexpress defensive-signaling pathways, leading to enhanced resistance to different pathogens such as B. cinerea, P. syringae, F. oxysporum, Plectosphaerella cucumerina, and Hyaloperonospora parasitica (Andersen et al., 2002; Meir and Cahill, 2003; de Torres-Zabala et al., 2007; de Torres-Zabala et al., 2009; García-Andrade et al., 2011; Sánchez-Vallet et al., 2012). Negative interactions of ABA with the major hormones involved in plant defense response (SA, JA, and ET) have been described by means of exogenous hormone treatments (Yasuda et al., 2008; de Torres-Zabala et al., 2009; Sánchez-Vallet et al., 2012). For instance, almost 65% of the up-regulated genes and 30% of the down-regulated genes in abi1-6 mutant were found to be up- or down-regulated by either ET, JA, or SA treatment (Sánchez-Vallet et al., 2012). Remarkably, these genes constitutively up-down-regulated in abi1-6 mutant were differentially expressed in Arabidopsis wild-type plants inoculated with Plectosphaerella cucumerina, indicating that they form part of the defensive responses activated upon pathogen infection (Sánchez-Vallet et al., 2012). In addition, ABA plays a direct role in regulating R (resistance) protein activity. ABA and exposition of plants to high temperature both reduce the nuclear accumulation of SNC1 (suppressor of npi-1, constitutive1) and RPS4 (resistant to Pseudomonas syringae 4) compromising disease resistance to P. syringae (Mang et al., 2012).

Abscisic acid can also positively regulate the resistance to some pathogens, such as Alternaria brassicicola, R. solanacarum, and Pythium irregulare, as ABA-deficient and insensitive mutants (abi1-1, abi2-1, abi1-6, abi2-12, aaos-2, and npq2-1) were found to be more susceptible than wild-type plants to these pathogens (Adie et al., 2007; Hernandez-Bloise et al., 2007; Flores et al., 2008; Garcia-Andrade et al., 2011). In Arabidopsis, ABA has been shown to be required for JA biosynthesis that is essential for resistance to Pythium irregulare (Adie et al., 2007). This contrasts with the negative interaction of ABA- and JA-signaling in the modulation of Arabidopsis resistance to the necrotrophic fungus Plectosphaerella cucumerina (Sánchez-Vallet et al., 2012). Similarly, although ABA and SA have been shown to function antagonistically in the control of the resistance to some pathogens, they trigger stomata closure to avoid penetration of the bacteria P. syringae in Arabidopsis (Melotto et al., 2006). Plant treatment with fgl22 is known to interfere with ABA signaling to induce stomata closure. The ABA- or fgl22-induced stomata closure are impaired in lines overexpressing HSC70-1 (heat shock cognate70-1) and mutants in HSP90 (heat shock protein90; Clément et al., 2011), resulting in an increased susceptibility to both virulent and avirulent strains of P. syringae (Hubbert et al., 2005; Takashishi et al., 2003; Noel et al., 2007). ABA is a key hormone in Arabidopsis response to R. solanacarum infection, as 40% of the genes up-regulated during the development of wilting symptoms were related to ABA, including those encoding proteins for ABA biosynthesis [i.e., 9-cis-epoxy carrotenoid dioxygenase3 (NCED3)] or signaling [i.e., ABA-insensitive1 (ABI1) and ABI5; Hu et al., 2008]. More recently, it has been shown that pre-inoculation of Arabidopsis with an avirulent strain of R. solanacarum activates plant resistance to virulent isolates of this bacterium, and this resistance was correlated with the enhanced expression of ABA-related genes that resulted in a hostile environment for the infection development. These results suggest that ABA may be used in biological control of bacterial wilt caused by R. solanacarum (Feng et al., 2012).

Polyamines

Polyamines, such as putrescine, spermidine, and spermine, are important nutrients that are also involved in plant defense responses (Cao et al., 1997), and NPR3 and NPR4 proteins have been recently described as SA receptors (Fu et al., 2012; Wu et al., 2012). NPR1 localizes at the cytosol as an oligomer, and in the presence of SA, redox changes occur in NPR1 that lead to the dissociation of NPR1 complex and to the translacation of the corresponding monomers to the nucleus. There, NPR1 protein activates the transcription of defensive genes, such as polyamines-related protein, by interacting with YGA (TGACG sequence-specific binding protein) transcription factors (Dong, 2004; Tada et al., 2008; Robert-Seilaniantz et al., 2011a). In Arabidopsis, EDS1 (enhanced disease susceptibility 1) is a major node required both for SA-dependent basal resistance against...
viral pathogens and for the activation of the ETI mediated by the TIR-NB-LRR (Toll-interleukin receptor domain–nucleotide binding domain–leucine rich repeat) resistance proteins (Parker et al., 1996; Falk et al., 1999). ED1 protein is present in distinct pools at nuclei and cytoplasm, and these two ED1 locations are required for a complete immune response (Garcia et al., 2010). Several ED1 interactors have been identified, including PAD4 (pyrroloquinoline quinone-dependent), RPS4, RPS6, SAG101 (senescence-associated gene101), SFR1 (suppressor of RPS4-RLD1), and SNC1 (Frey et al., 2001, 2005; Bhattacharjee et al., 2011; Heidrich et al., 2011; Rietz et al., 2011). The ED1–PAD4 complex is necessary for basal resistance and activation of SA-defense response (Rietz et al., 2011). Indeed, mutations in ED1 and PAD4 lead to resistance to pathogens such as Hyaloperonospora parasitica and deficiency of the SA signaling pathway (Park et al., 1996; Falk et al., 1999; Wang et al., 2005). Transcriptional regulation of SA-defensive genes is also mediated by HDA19 (histone deacetylase19) that repressed SA-mediated basal defense to Pseudomonas syringae (Choi et al., 2012). Up-regulation of SA marker genes (PR1, PR2, ICS1, ED1, PAD4) and over-accumulation of SA take place in hda19 mutant, which correlates with its enhanced resistance phenotype to Pseudomonas syringae pathogenic bacteria. Indeed, HDA19 targets PR1 and PR2 promoters to regulate gene expression. The mutation hda19 causes hyper-acetylation of histones in the promoters of PR genes and priming of SA-associated plant defense (Choi et al., 2012).

Cross-talk between SA and JA signaling pathways has been thoroughly described (Gimenez-Ibanez and Solano, 2013). For example, WRKY33, a positive regulator of JA-related genes, is a repressor of the SA pathway. In the wrky33 mutant there is an enhanced expression of several SA-regulated genes (SIDD/SID1, ICS1, ED5, ED1, NIMIN1, PR1, PR2, PR5) and increased accumulation of SA levels. In turn, SA induction contributes to down-regulate JA-signaling, and to increase the susceptibility of wrky33 mutant to necrotrophic fungi (Birkenbihl et al., 2012; Sánchez-Vallet et al., 2012). NPRI1 is a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003). The Arabidopsis mediator subunit 16 (MED16) was recently described to be a positive regulator of SA-induced defense response and a negative regulator of JA/ET signaling pathway (Zhang et al., 2012). The role of ABA as a regulator of SA-mediated suppression of the JA/ET signaling pathway, as revealed using npri1 mutant (Spool et al., 2003).

**Decoy Strategies of Pathogens: Manipulation of the Host Hormone Machinery**

**Pathogens Produce and Degrade Hormones**

**Auxins**

Many pathogenic microbes and plant growth promoting rhizobacteria have evolved complete pathways for auxin biosynthesis with tryptophan as the main precursor (Spaepen et al., 2007). Auxin-producing phytopathogenic bacteria are mostly, but not exclusively, gall-inducing microbes. They include, for instance, Agrobacterium tumefaciens (Liu and Nester, 2006), Agrobacterium rhizogenes (Gaudin and Jouannin, 1995), Erwinia chrysanthemi (Yang et al., 2007), Erwinia herbicola (Brandl and Lindow, 1998), Pseudomonas fluorescens (Suzuki et al., 2003), P. putida (Leveau and Lindow, 2005), Pseudomonas savastanoi (Glickmann et al., 1998), P. syringae (Glickmann et al., 1998), R. solanacearum (Seguin and Williams, 1964; Valls et al., 2006), and Rhodococcus fascians (Vandeputte et al., 2005). In R. solanacearum, auxin biosynthesis is governed by HrpG, a major regulator of bacterial virulence and response to metabolic signals (Valls et al., 2006). In Agrobacterium tumefaciens, two genes required for conversion of tryptophan to auxin are localized on the T-DNA region of the Ti plasmid injected into plant cells. Auxin biosynthesis is necessary for tumor gall formation and for pathogenicity of Agrobacterium (Lee et al., 2009); auxins negatively regulate the expression of genes necessary for the transfer of Agrobacterium T-DNA in plants and also inhibit the growth of several bacterial species in vitro (Liu and Nester, 2006).

Auxin biosynthesis in fungal pathogens seems to be limited to a few species. In Ustilago maydis, U. euchenta, and U. scirpinaeuxin is produced (Chung and Tzeng, 2004; Reineke et al., 2008). In this case, auxin does not seem to be required for U. maydis-induced tumor formation or for pathogenicity, as a mutant defective in four genes encoding key auxin biosynthetic enzymes was compromised in auxin levels but not in tumor formation (Reineke et al., 2008). Additionally, other fungi have enzymatic tools to produce auxins, such as Colletotrichum gloeosporioides f. sp. aschynomenei, Colletotrichum acutatum, and F. proliferatum (Robinson et al., 2000; Chung et al., 2003, 2005; Tavakelova et al., 2012). Nevertheless, the production of auxins by fungal pathogens has not been clearly demonstrated to be a virulent factor that favors plant colonization.

**Abscisic Acid**

Several fungal species produce ABA, including B. cinerea, Rhizoctonia solani, Cercospora fimbriata, and Rhizopus nigricans (Dorfling et al., 1984; Inomata et al., 2004a). ABA biosynthesis by B. cinerea requires a cluster of four genes, BcAR1 to BcAR4 (Hirai et al., 2000; Inomata et al., 2004b; Shevers et al., 2004, 2006). Unlike plants, fungi, such as B. cinerea and Corynebacterium, use the mevalonate pathway to produce ABA (Hirai et al., 2000; Inomata et al., 2004a). The role of ABA in B. cinerea virulence factors has not been fully demonstrated, but several published data support this hypothesis: i) ABA biosynthesis in the fungus is stimulated by the host plant (Kettner and Darfling, 1995); ii) exogenous treatment with ABA increased disease symptoms caused by the fungus on roses (Shaul et al., 1996); and iii) ABA contributes to susceptibility to B. cinerea and other pathogens by suppressing...
defense responses in plants (Audenaert et al., 2002; Sánchez-Vallet et al., 2012).

Salicylic acid
Although SA biosynthesis has not been described in plant pathogens, it is known that some plant-associated bacteria can degrade salicylate. Indeed, the enzyme salicylate hydroxylase (NahG), that catalyzes the formation of catechol from salicylate, has been identified in various bacteria, such as P. putida and P. fluorescens (You et al., 1991; Chung et al., 2001).

PATHOGEN EFFECTORS INTERFERE WITH HORMONE SIGNALING IN PLANTS
Effectors are proteins secreted by pathogens during infection to deregulate host immune responses. One common strategy implemented by effectors is the manipulation of the homeostasis of plant phytohormones, resulting in deactivation of the appropriate defense response (Robert-Seilaniantz et al., 2007; Bari and Jones, 2009; Figures 1 and 2).

Bacteria and phytoplasma
In addition to the common example of the phytotoxin COR produced by P. syringae strains to manipulate the plant hormonal balance (Zhang et al., 2012), many phytopathogenic bacteria have developed large repertoires of type III effectors (T3E) which are necessarily injected through the syringe-like type III secretion system inside plant cells to deregulate plant immunity (Figure 1; Jones and Dangl, 2006; Boller and He, 2009; Büttner and He, 2009). The roles of bacterial effectors in plant immunity have been extensively reviewed elsewhere (Cai et al., 2009; Rivas and Genin, 2011; Deslandes and Rivas, 2012; Dou and Zhou, 2012; Howden and Huitema, 2012). Xanthomonas sp. bacteria synthesized TAL (transcription activator-like) effectors, such as AvrR3 from X. axonopodis pv. vesicatoria (formerly X. campestris pv. vesicatoria), that are imported to the plant nuclei where they activate the expression of host target genes (Buch et al., 2009; Moscou and Bogdanove, 2009; Bogdanove et al., 2011). Five targets, designed as sp-regulated by AvrR3 1 to 5 (UPA1–5), are auxin-induced genes members of the SAUR (small auxin up RNA) family (Marois et al., 2012).
FIGURE 2 | Balancing plant immune responses and fitness costs. Plant disease resistance responses are induced upon recognition of PAMPs/ Effectors from pathogens interfere with hormonal balance and the activation of PTI and ETI. Pathogens can also negatively impact plant growth and developmental-associated processes (transcriptional expression of genes, negative regulation of signaling pathways, etc.; see text for details). Positive and negative interactions are indicated by arrows and squares, respectively. GA, gibberellic acid; BR, brassinosteroids.

Additionally, induction of the TAL target UPA20 provokes cell hypertrophy, a feature which is characteristic of auxin accumulation (Kay et al., 2007). Auxin is a susceptibility factor in Arabidopsis plants infected with PstDC3000, and consequently, auxin was hypothesized to be a potential target for bacterial effectors. Thus, the cysteine protease bacterial effector AvrRpt2 triggers auxin signaling pathway to enhance bacterial virulence in Arabidopsis lines lacking the resistance gene that normally recognizes this T3E. Transgenic plants expressing AvrRpt2 accumulated higher auxin levels and showed a constitutive activation of the auxin signaling pathway. Additionally, auxin levels in Arabidopsis leaves inoculated with PstDC3000avrRpt2 were higher than those in plants infected with PstDC3000 (Chen et al., 2007), indicating that AvrRpt2 modulates auxin pathway to enhance bacterial virulence, but this effect was found to be independent of SA (Chen et al., 2004). Auxin signaling seems to be a preferential target of phytoplasmas, some bacteria-like, obligate plant pathogens belonging to the class of Mollicutes that require sap-feeding insect herbivores as vectors for transmission to plants (Sugio et al., 2011).

Indeed, TENGU (tengu-su inducer) is an effector of Candidatus phytoplasma asteris that, when expressed in Arabidopsis transgenic lines, causes dwarfism and abnormal reproductive organogenesis and flower sterility. These phenotypes, which are similar to the disease symptoms provoked by the phytoplasma, have been associated to alterations in hormone balance. Microarray analysis of transgenic Arabidopsis plants expressing TENGU demonstrated that many auxin-related genes were down-regulated, including genes of the Aux/IAA, SAUR, GH3, and PIN families (Hoshi et al., 2009). Thus, TENGU effector could interfere with auxin signaling in plants.

Several P. syringae effectors target SA. HopPtoM and AvrE are repressors of SA-dependent callose deposition but do not affect SA-responsive genes in Arabidopsis infected leaves (DebRoy et al., 2004). The effector HopI1 (previously named HopPmaI), that is essential for the virulence of P. syringae pv. maculicola (Pma) in Arabidopsis, N. benthamiana, and N. tabacum, has been found to be a modulator of SA-mediated defense responses. Indeed, the expression of HopI1 in Arabidopsis acd6-1 (accelerated cell
ABA-mediated responses in plants: it enhances stomata closure, Ws was corroborated by the fact that Arabidopsis showed enhanced colonization by the avirulent conditions (Goel et al., 2008). Thus, HopZ1a contributes to Ppv virulence by suppressing SA-mediated defenses that takes place during ETI induced by other effectors such as AvrRpt2. EDS1, a key regulatory node of basal and induced resistance, is also targeted by bacterial pathogen effectors. AvrRps4 and HopA1, two PvdDC3000 effectors, bind to EDS1 interfering with the interaction between EDS1 and TIR–NB–LRR resistance proteins, and consequently preventing the activation of the immune response (Bhattacharjee et al., 2011; Hehrich et al., 2011). In contrast to other effectors, HopW1-1, that forms part of the T3E repertoire of Pma, but not of that of PvdDC3000 (Guatman et al., 2002), induces resistance in the Ws accession of Arabidopsis to Pma (Lee et al., 2008). This effect of HopW1-1 on Ws was corroborated by the fact that PvdDC3000 strain expressing HopW1-1 has reduced growth and caused weak disease symptoms in the Ws plants. In a yeast two-hybrid screen, three Arabidopsis HopW1-1-interacting proteins (WIN2, WIN3) were found to bind to the effectors (Lee et al., 2008). The enhanced resistance triggered by HopW1-1 was not caused by activation of a hypersensitive response, but it was dependent on an enhanced accumulation of SA. Indeed, pad4 mutants were almost completely compromised in their resistance response to HopW1-1. HopAM1 contributes to P syringae virulence by manipulating ABA-mediated responses in plants: it enhances stomata closure, suppresses infection-triggered callose deposition, and inhibits seed germination. Remarkably, HopAM1 increased P syringae virulence on Arabidopsis plants grown under water-stressed conditions (Goel et al., 2008). Arabidopsis lines expressing HopAM1 showed enhanced colonization by the avirulent PvdDC3000 rectC mutant, impaired in TSS, and did not develop callose-rich papillae that are normally induced by RecC strain in wild-type plants (Goel et al., 2008). An effector of P syringae pv. phaseolicola, HopB2, promotes virulence on Arabidopsis and bean plants, and suppresses basal resistance to PvdDC3000 hrpA-, a mutant compromised in TSS (de Torres et al., 2006). Expression of HopB2 in Arabidopsis plants induces the expression of NCE33, resulting in enhanced biosynthesis of ABA, which interferes with the accumulation of SA levels and the activation of SA-mediated resistance (de Torres-Zabala et al., 2007). Thus, HopAM1 and HopB2 are suppressors of defense mechanisms by enhancing ABA responses and promoting disease susceptibility in plants.

FILAMENTOUS PATHOGENS: OOMYCETES AND FUNGI

Oomycete genomes contain a class of cytoplasmic proteins known as XLRs that contain a conserved BXL amino acid motif (arginine, any amino acid, leucine, arginine; Rehmany et al., 2005; Morgan and Kamoun, 2007). Two effectors from this class, HalR1.96 from Hypothenorum arabidopsidis, the causal agent of downy mildew on Arabidopsis, and its ortholog PsvAvh163 from Phytophthora sojae, which causes soybean rot disease, interfere with plant immunity (Anderson et al., 2012). Remarkably, Arabidopsis plants expressing HalR1.96 or PsvAvh163 became more susceptible to virulent and avirulent pathogens, indicating that these effectors repress basal resistance and ETI. In fact, the induction of SA-defensive genes, but not SA biosynthesis, that take places upon infection with avirulent strains of Hypothenorum arabidopsidis, was suppressed in the transgenic lines expressing HalR1.96 or PsvAvh163, indicating that these effectors interfere with SA signaling to trigger plant susceptibility to oomycetes (Anderson et al., 2012).

Filamentous extracellular or obligate fungal pathogens secrete effectors via hyphae or haustoria (Stergiopoulos and de Wit, 2009; de Jonge et al., 2011). U. maydis is a basidiomycete fungus that causes smut disease on maize and its relative teosinte (Brefort et al., 2009; Djamie and Kahmann, 2012). Maize infection by U. maydis results in the repression of SA-associated PRI defense gene expression during the early biotrophic phase of the interaction, while auxin production in the host is induced later during tumor formation (Doelemann et al., 2008). One of the most highly expressed genes of U. maydis during plant colonization is the Cmu1 effector, a chorismate mutase protein (Skåbbe et al., 2010). Cmu1 is required for full virulence since the induction of tumors is significantly reduced in a U. maydis cmu1 mutant (Djamie et al., 2011). Once inside plant cells, Cmu1 is localized in the cytoplasm, the nucleus and guard cells and it is spread to neighbor cells through plasmodesmata. A yeast two-hybrid analysis showed that Cmu1 interacts with two maize chorismate mutases, ZmCm1 and ZmCm2, which are found in plastids and cytoplasm in plants, respectively. Interestingly, SA levels were higher in maize inoculated with a cmu1 mutant than with a wild-type strain, resulting in an increased resistance of the mutant to U. maydis. It was hypothesized that Cmu1 could act together with ZmCm2 in the plant cytoplasm to enhance the flow of the SA-precursor chorismate from the plastid (where SA biosynthesis takes place) to the cytosol. Consequently, in plastids, less chorismate would be available for SA biosynthesis (Djamie et al., 2011). These results indicate that SA biosynthesis pathway of maize is hijacked by U. maydis as a mechanism of virulence. Interestingly, such a mechanism was also described for the soybean cyst nematode Heterodera glycines and the root-knot nematode Meloidogyne javanica (Rokal et al., 2003; Doyle and Lambert, 2003). The virulence factor of Chalasosporium falbura Avr2 targets the tomato papain-like cysteine protease (PLCP) RCR3 and Phytophthora-inhibited protease 1 (PIP1) in order to deregulate basal immunity. RCR3 and PIP1 are specifically induced by treatment of tomato plants with the SA analog benzo[1,2-b:4,3-b′]dithiophene (BTH). Therefore, Avr2 seems to interfere with tomato SA signaling pathway (Shabab et al., 2008).

FITNESS COSTS OF DEFENSE RESPONSES REGULATED BY PHYTOHORMONES

The involvement of many plant growth regulatory phytohormones in the control of plant resistance responses to both biotic and abiotic stresses indicates the existence of a tight interconnection between two physiological processes: development and adaptation to environmental cues. The regulatory potential of the hormone network allows plants to quickly respond to environmental
changes and, thus, to use the limited nutrient resources in a cost-efficient manner. This hypothesis is based on the idea that being well-defended (i.e., having strong, pre-existing defensive mechanisms) may not always be the best defensive strategy, most likely because allocation of metabolites and proteins to resistance may constrain other plant physiological processes (Walters and Heil, 2007; Manzanedo et al., 2010; Kempel et al., 2011). In line with this hypothesis, it is generally believed that hormone-induced resistance evolved to save energy under enemy-free conditions, as they will only incur energy costs when these defensive mechanisms are activated upon pathogen infection or insect attack (Walters and Heil, 2007). However, pathogens and pests evolve to get adapted to the continuous exposure to defensive genetic traits (i.e., antibiotic or antodeterrent proteins and/or metabolites). Therefore, it is also possible that hormone-induced resistance evolved to slow down the potential adaptation of putative attackers to these biochemical barriers (Walters and Heil, 2007). All these physiological constraints, together with the co-existence of plants with natural attackers, have evolutionary driven the selection of plant innate immune system.

In different plant species there have been characterized mutants or transgenic lines showing constitutive activation of defensive mechanisms and enhanced resistance to particular pathogens. These resistance phenotypes are generally associated with the misregulation of particular hormone signaling pathways (Robert-Seilaniantz et al., 2011a). The characterization of these mutants and transgenic plants has contributed to the identification of the molecular components involved in hormone biosynthesis and signaling pathways, and to the discovery of cross-regulatory nodes among these signaling pathways. Thus, Arabidopsis mutants constitutively overexpressing a specific hormone-dependent pathway (SA, ET, JA, ET + JA, etc.) show enhanced resistance to particular type of pathogens (reviewed by Robert-Seilaniantz et al., 2011a; Holoski et al., 2012). However, this enhanced, constitutive resistance negatively impact plant fitness as these mutants have phenotypic alterations such as dwarfism, spontaneous lesions in different organs, accelerated senescence, delayed flowering, sterility, or reduced seed production (for a review, see Robert-Seilaniantz et al., 2011a; Holoski et al., 2012; Thaler et al., 2012).

These data indicate that plants have genetic determinants to fine-tune fitness/resistance balance. An example of this fine-tune regulation is represented by the SA receptor NPR3, that is a negative regulator of defensive response during Arabidopsis early flower development through its interaction with NFR1 and TGA2. Remarkably, the npr3 plants exhibit increased resistance to P. syringae infection of immature flowers, but showed reduced resistance in comparison to that of wild-type plants (Shi et al., 2012).

Alteration of a particular hormone signaling pathway generally results in the mis-regulation of other signaling pathways due to the described complex regulatory network that exist among hormones. Thus, the negative cross-regulations among hormone pathways, such as auxin, ABA, and SA described in this review, lead to alterations in the pattern of resistance to natural attackers. That is, enhanced resistance to a particular pathogen (i.e., necrotroph, Spoe1 et al., 2007; Robert-Seilaniantz et al., 2011a) or...
with model and crop plants under field conditions should be done to determine the potential use of hormone-mediated resistance in crop protection, as these experiments will provide information on the hormone-mediated effectiveness of disease control, but also on plant trade-offs and changes in the population structure of pathogens and pests. Also, a better understanding of the functional role of 

Hormone homeostasis/signaling and improve crop resistance to pathogens.

REFERENCES

Aike, B. A., Perez-Perez, J., Perez-Perez, M. M., Guzman, M., Sanchez-Serrano, J. J., Schmelz, E. A., et al. (2007). ABA is an essential signal for plant resistance against fungal pathogens and the activation of defence in Arabidopsis. Plant Cell 19, 1665–1681.

Anderson, R. G., Cundy, M. S., Fox, R. A., Vaughan, M. D., Dib, D., Fukushige, K., et al. (2012). Homologous EKR1 effectors from Hyaloperonospora arabidopsidis and Phytophthora species suppress immunity in distantly related plants. Plant J. 72, 882–893.

Andersson, L., Del Mastro, J., Narodzinska, T., Levin, A., Ljung, K., Bhalerao, R., Bennett, K. N. (2003). A chorismate mutase encodes the plant cytochrome P450 CYP83B1, a modifier of basal susceptibility of Arabidopsis to Heterodera glycines. Proc. Natl. Acad. Sci. U.S.A. 100, 14819–14824.

Bekal, S., Niblack, T. L., and Lambert, M. T. (1998). Contribution of indole-3-acetic acid biosynthesis of indole-3-acetic acid to the host defense. J. Exp. Bot. 49, 473–488.

Barbel, R., Nauen, R., Lopes, J. D. (2009). Role of plant hormones in plant defense responses. Plant Mol. Biol. 69, 473–488.

Barber, J. K., Kowalczyk, M., Marchant, A., Izen, S., Bhukar, R., Bennett, M., et al. (2008). The AUR2 gene of Arabidopsis thaliana encodes the cysteine-rich P403 CYRPS81, a modulator of auxin homeostasis. Proc. Natl. Acad. Sci. U.S.A. 97, 14819–14824.

Bekal, S., Niblack, T. L., and Lambert, K. N. (2003): A chorionic mutase from the so-called cyst nontoxic Hevea brasiliensis shows polymorphism that correlates with virulence. Mol. Plant Pathol. 16, 439–446.

Belkhadir, Y., Gallie, B. R., and Chung, K. R. (2004). Transcriptome analysis of the tomato to Botrytis cinerea interaction reveals type III effector AvrPtoB suppresses the RXLR response. Mol. Plant Microbe Interact. 17, 644–655.

Bekal, S., Niblack, T., and Lambert, M. T. (1998). Contribution of indole-3-acetic acid biosynthesis of indole-3-acetic acid to the host defense. J. Exp. Bot. 49, 473–488.

Belkhadir, Y., Gallie, B. R., and Chung, K. R. (2004). Transcriptome analysis of the tomato to Botrytis cinerea interaction reveals type III effector AvrPtoB suppresses the RXLR response. Mol. Plant Microbe Interact. 17, 644–655.

Bekal, S., Niblack, T. L., and Lambert, M. T. (1998). Contribution of indole-3-acetic acid biosynthesis of indole-3-acetic acid to the host defense. J. Exp. Bot. 49, 473–488.

Belkhadir, Y., Gallie, B. R., and Chung, K. R. (2004). Transcriptome analysis of the tomato to Botrytis cinerea interaction reveals type III effector AvrPtoB suppresses the RXLR response. Mol. Plant Microbe Interact. 17, 644–655.

Bekal, S., Niblack, T. L., and Lambert, M. T. (1998). Contribution of indole-3-acetic acid biosynthesis of indole-3-acetic acid to the host defense. J. Exp. Bot. 49, 473–488.

Belkhadir, Y., Gallie, B. R., and Chung, K. R. (2004). Transcriptome analysis of the tomato to Botrytis cinerea interaction reveals type III effector AvrPtoB suppresses the RXLR response. Mol. Plant Microbe Interact. 17, 644–655.

Bekal, S., Niblack, T. L., and Lambert, M. T. (1998). Contribution of indole-3-acetic acid biosynthesis of indole-3-acetic acid to the host defense. J. Exp. Bot. 49, 473–488.
de Torres-Zabala, M., Bennett, M. H., Truman, W. M., and Grant, M. R. (2009). Antagonism between salicylic acid and abscisic acid reflects early host-pathogen conflict and modulates plant defense responses. Plant J. 59, 375–386.

de Torres-Zabala, M., Truman, W. N., Bennett, M. H., Lalflor, G., and Manfield, J. W. (2007). Rosette Pseudomonas syringae pv. tomato hijacks the Arabidopsis abscisic acid signaling pathway to cause disease. EMBO J. 26, 1434–1445.

Ding, X., Cao, Y., Huang, L., Zhao, J., Xu, C., Li, X., et al. (2008). Activation of the indica 3-3-acetic acid synthetase GH3-8 suppresses expansin expression and promotes salicylate- and jasmonate-independent basal immunity in rice. Plant Cell 20, 226–240.

Dijkmans, A. and Kalmijn, R. (2012). Unifying models: dissecting the molecular interface between pathogen and plant. PLoS Pathog. 8:e1002855. doi: 10.1371/journal.ppat.1002855

Dijkmans, A., Schepers, K., Roos, F., Ghosh, A., Vincx, V., Kuhnt, J., et al. (2011). Metalloprotease by a secreted fungal effector. Nature 495, 395–398.

Dodda, R. N. and Rathjen, J. F. (2010). Plant immunity: towards an integrative view of plant-pathogen interactions. Trends Plant Sci. 15, 123–130.

Doehlemann, G., Wahl, R., Horst, D., Djamei, A., Schipper, K., Rabe, F., de Torres Zabala, M., Bennett, M., Denancé et al. Hormone balance in plant resistance.

Doyle, E. A., and Lambert, K. N. (2008). Activation of the indole-3-acetic acid synthetase GH3-8 (2008). Activation of the indole-3-acetic acid synthetase GH3-8 is important for salicylic acid signaling in plants. Nature 456, 228–232.

Dörffling, K., Petersen, W., Sprecher, E., Urbasch, I., and Hanssen, H. (2008). Transcriptional responses of Arabidopsis thaliana during mild disease caused by the soil-borne phytopathogenic bacterium, Ralstonia solanacearum. Planta 228, 2587–2598.

Dörffling, K., Petersen, W., Sprecher, E., Urbasch, I., and Hanssen, H. (2008). Transcriptional responses of Arabidopsis thaliana during mild disease caused by the soil-borne phytopathogenic bacterium, Ralstonia solanacearum. Planta 228, 2587–2598.

Dörffling, K., Petersen, W., Sprecher, E., Urbasch, I., and Hanssen, H. (2008). Transcriptional responses of Arabidopsis thaliana during mild disease caused by the soil-borne phytopathogenic bacterium, Ralstonia solanacearum. Planta 228, 2587–2598.

Dörffling, K., Petersen, W., Sprecher, E., Urbasch, I., and Hanssen, H. (2008). Transcriptional responses of Arabidopsis thaliana during mild disease caused by the soil-borne phytopathogenic bacterium, Ralstonia solanacearum. Planta 228, 2587–2598.
Kieffer, M., Neve, J., and Kepinski, L., Zhang, Y., Karlsson Moritz, Lee, M. W., Jelenska, J., and Green-Leveau, J. H., and Lindow, S. E. (2013). Hormone balance in plant resistance. Mol. Plant Pathol. 15, 2365–4079. Panterone, R., Parker, J. E., and Schubert-Lefert, P. (2009). Signpost: plant immune response pathways. Cell 136, 1050–1056. Rawat, S., and Genni, S. (2011). A plethora of virulence strategies hidden behind nuclear targeting of microbial effectors. Front. Plant Sci. 2:104. doi: 10.3389/fpls.2011.00104. Robert-Seilaniantz, A., Grant, M., and Jones, J. D. (2010). Hormone crosstalk in plant disease: more than just jasmonate-salicylate antagonism. Annu. Rev.Phytopathol. 49, 517–543. Robert-Seilaniantz, A., MacLean, D., Ikemura, Y., Hill, L., Yamaguchi, S., Kaneto, Y., et al. (2013). The microRNA miR355 re-directs secondary metabolite biosynthesis away from camalexin and towards glucosinolates. Plant J. 67, 218–231. Robert-Seilaniantz, A., Navarro, L., Bar, R., and Jones, J. D. (2007). Pathological hormone imbalance. Curr. Opin. Plant Biol. 10, 372–379. Rohm, M., Riese, L., and Sharon, A. (1998). Indole-3-acetic acid biosynthesis in Colletotrichum gloeosporioides f. sp. anamorphens. Appl. Environ. Microbiol. 64, 3305–3312. Sequeira, L., and Williams, P. H. (1994). Synthesis of indoleacetic acid by Pseudomonas solanacearum. Phytochemistry 37, 1240–1246. Staswick, P. E., Jeon, J., Yun, J., et al. (2009). The HSP70 chaperones regulates Arabidopsis immunity. Nat. Biotechnol. 27, 1049–1055. Stitt, S., Stamm, M., Maloney, S., Wagner, S., Becker, D., Molina-Encinas, N., et al. (2011). Different roles of enhanced disease susceptibil- ity (EDS) bound- and unbound from diseased tomato plants. Fungal Genet. Biol. 48, 1109–1123. Thalaba, O., Elah, Y., and Zadors, N. (1996). Suppression of Botrytis blight in ear row flowers with gibberellic acid.
acid. Effects of exogenous application of abscisic acid and paclobutrazol. Plant Growth Regul. 7, 145–150.
Shi, Z., Maiti, S., Liu, Y., Ver- ica, J., and Faulkman, M. J. (2012). The salicylic acid receptor NPR1 is a negative regulator of the transcriptional defense response during early flower development in Arabidopsis. Mol. Plant. 10:1035/mp/0091 (Epub ahead of print).
Shimizu, K., and Yamaguchi-Shinozaki, K. (2007). Gene networks involved in drought stress response and tolerance. J. Exp. Bot. 58, 223–235.
Siewers, V., Kolkholka, L., Smedsgaard, J., and Tjulipniki, P. (2006). Identification of an abscisic acid biosynthesis in the grey mold Botrytis cinerea. Appl. Environ. Microbiol. 72, 4619–4626.
Siewers, V., Smedsgaard, J., and Tjulipniki, P. (2004). The P450 monoxygenase ReBC1 is essential for abscisic acid biosynthesis in Botrytis cinerea. FEMS Microbiol. Rev. 31, 425–448.
Spald, S. H., Johnson, J. S., and Dong, A. (2007). Regulation of trade-offs in plant defense against pathogens with different life history. Proc. Natl. Acad. Sci. U.S.A. 104, 18842–18847.
Spald, S. H., Koornneef, A., Claessens, S. M., Konijn, J. P., Van Vliet, J. A., Mueller, M. J., et al. (2003). NPR1 avoid closing the talk between salicylate- and jasmonate-dependent defense pathways through a novel function in the cytosol. Plant Cell 15, 760–770.
Streeppevan, L., and de Wit, P. J. (2009). Fungal effector pro- teins. Annu. Rev. Phytopathol. 47, 235–261.
Sugio, A., Maclean, A. M., Kingdom, H. N., Greve, V. M., Maannmetsa, K., and Hoglund, S. A. (2011). Diverse targets of phytoplasma effectors from plant development to defense against insects. Annu. Rev. Phytopathol. 49, 175–199.
Suzuki, S., H. Y., and Osanai, H. (2015). Indole-3-Acetic acid production and its defense function in the cytosol. Proc. Natl. Acad. Sci. U.S.A. 112, 18842–18847.
Vallé, M., Genin, S., and Bonen, C. (2008). Integrated regulation of the type III secretion system and other virulence determinants in Balteopsis solanacearum. PLAnt Pathol. 57:292. doi: 10.1111/j.1365-3059.2008.02082.x.
Vanderzand, P., Odem, S., Mol, A., Versée, D., Gohrhardt, H., El Jaziri, M., et al. (2005). Biosynthesis of auxin by the gram-positive phytopathogen Rhodococcus fascians is controlled by compounds specific to infected plant tissue. Appl. Environ. Microbiol. 71, 1169–1177.
Veli, A. C., Dempsey, D. M. A., and Khong, D. F. (2007). Salicylic acid, a multifaceted hormone to combat disease. Annu. Rev. Phytopathol. 47, 177–206.
Walten, D., and Hol, M. (2007). Costs and trade-off associated with induced resistance. Physiol. Mol. Plant Pathol. 71, 3–17.
Wang, D., Puzesovska-Mukhtar, K., Calfee, A. H., and Dong, A. (2007). Salicylic acid inhibits defense growth in plants through repression of the auxin signaling pathway. Curr. Biol. 17, 1784–1790.
Waskowska, A., Vlah, S., Starzecka, C., Radko, J., Jamies, E., Volon, C., et al. (2009). An update on abscisic acid signaling in plants and me. Mol. Plant 1, 198–217.
Wu, Y., Zhang, D., Chu, J. Y., Bera, P., Wang, Y., Brindle, I. D., et al. (2012). The Arabidopsis NPR1 pro- tein is a receptor for the plant defence hormone salicylic acid. Cell Rep. 1, 639–647.
Yang, S., Zhang, Q., Jie, C., Chakraborty, A. O., Gask, B. B., Buch, A. M., et al. (2007). Global effect of indole-3-Acetic acid biosynthesis on multiple virulence factors of Erwinia chrysan-theniae 305. Appl. Environ. Microbiol. 73, 1079–1084.
Yudan, M., Ishikawa, A., Ikumori, Y., Seki, M., Unzueme, T., Atami, T., et al. (2008). Antagonistic interaction between systemic acquired resistance and the abscisic acid-mediated abiotic stress responses in Arabidopsis. Plant Cell 20, 1679–1682.
You, I. S., Ghosh, D., and Gamou-Ando, O. C. (1991). Nucleotide sequence analysis of the Pseudomonas putida Pgy7 salicylic acid biosynthesizing gene (nalB) and its 5′ flanking region. Biochemistry 30, 1653–1664.
Zeng, W., Brutus, A., Keil, J. M., Wilburn, J. C., Gao, X., Jones, A. D., et al. (2011). A genetic screen reveals Arabidopsis stomatal and/or apoplastic defenses against Pseudomonas syringae pv. tomato DC3000. PLoS Pathog. 7:e1002291. doi: 10.1371/journal.ppat.1002291.
Zhao, X., Wang, C., Zhang, X., Sun, Y., and Liu, Z. (2012). The Arabidop- sius-mediated complex subunit 1 pos- itively regulates salicylate-mediated systemic acquired resistance and partitioning of abiotic-induced defense pathways. Plant Cell 24, 4926–4939.
Zhong, X. Y., Stettler, W. M., Zeng, W., Liu, P. P., Fu, Z. Q., Kikg, D. F., et al. (2012). Coronatine promotes Pseudo- monas syringae virulence in plants by activating a signaling cascade that inhibits salicylic acid accumulation. Cell Host Microbe 11, 507–516.
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