J Plant Res (2011) 124:489–499
DOI 10.1007/s10265-011-0409-y

The roles of ABA in plant–pathogen interactions

Feng Yi Cao · Keiko Yoshioka · Darrell Desveaux

Received: 17 November 2010 / Accepted: 25 January 2011 / Published online: 5 March 2011

Abstract Defence against abiotic and biotic stresses is crucial for the fitness and survival of plants under adverse or suboptimal growth conditions. The phytohormone abscisic acid (ABA) is not only important for mediating abiotic stress responses, but also plays a multifaceted and pivotal role in plant immunity. This review presents examples demonstrating the importance of crosstalk between ABA and the key biotic stress phytohormone salicylic acid in determining the outcome of plant–pathogen interactions. We then provide an overview of how ABA influences plant defence responses against various phytopathogens with particular emphasis on the Arabidopsis–*Pseudomonas syringae* model pathosystem. Lastly, we discuss future directions for studies of ABA in plant immunity with emphasis on, its role in the crosstalk between biotic and abiotic stress responses, the importance of distinguishing direct and indirect effects of ABA, as well as the prospect of utilizing the recently elucidated core ABA signaling network to gain further insights into the roles of ABA in plant immunity.

Keywords ABA · Plant immunity · *Pseudomonas syringae*/Arabidopsis

Introduction

As sessile organisms, plants are obligated to respond more effectively than animals to stresses in their environment in order to survive and reproduce. These stresses fall into two broad categories: abiotic and biotic. Abiotic stresses are inanimate factors that contribute to an unfavourable environment, including temperature changes, water and nutrient deficiency and salinity stress. Biotic stresses are those imposed by other organisms and include pathogenic interactions with microbes, fungi, oomycetes, animals, as well as other plants. Responses to abiotic and biotic stresses have historically been studied independently. However, recently it has become apparent that responses to abiotic and biotic stresses heavily influence one another, and that there is important crosstalk between their respective signaling pathways (Fujita et al. 2006). A key player in this crosstalk is the phytohormone abscisic acid (ABA).

We begin this review by presenting recent studies dealing with the crosstalk between ABA-mediated abiotic stress signaling and salicylic acid (SA)-mediated biotic stress signaling. We follow this with an overview of the role of ABA in plant–microbe interactions. Recent review articles by Asselbergh et al. (2008b) and Ton et al. (2009) provide extensive discussions of the multifaceted role of ABA in plant–pathogen interactions. In order to limit overlap, we focus on recent publications that have elaborated our understanding of the role of ABA in plant immunity with particular emphasis on the *Arabidopsis thaliana*/*Pseudomonas syringae* model pathosystem.

Plant immunity and pathogen lifestyles

The lifestyles of phytopathogens can be broadly categorized into biotrophs, necrotrophs and hemibiotrophs base on their nutrient acquisition strategies. Biotrophic pathogens derive nutrients from living plant host tissues and have evolved sophisticated strategies to exploit their hosts

F. Y. Cao · K. Yoshioka · D. Desveaux (✉)
Department of Cell and Systems Biology, University of Toronto, 25 Willcocks St, Toronto, ON M5S 3B2, Canada
e-mail: darrell.desveaux@utoronto.ca

K. Yoshioka · D. Desveaux
Centre for the Analysis of Genome Evolution and Function, University of Toronto, Toronto, Canada

for energy while keeping them alive in order to complete their life cycle (Glazebrook 2005; Laluk and Mengiste 2010). In contrast, necrotrophic pathogens derive nutrients from dead or dying cells, causing extensive necrosis, tissue maceration and ultimately death of the plant host (Laluk and Mengiste 2010). Hemibiotrophs behave both as biotroph and necrotrophs depending on the stage of their life cycles and environmental conditions. Importantly, plants have evolved sophisticated immune responses to contend with the various lifestyles and infection strategies of phytopathogens.

The phytohormones SA, jasmonic acid (JA) and ethylene (ET) play well-established roles in defence responses against phytopathogens (Glazebrook 2005). SA activates signaling cascades that lead to resistance against biotrophs and hemibiotrophs which often culminates in a localized cell death termed the hypersensitive response (HR; Durrant and Dong 2004; Vlot et al. 2009). On the other hand, JA and ET are predominantly associated with defence responses against necrotrophic pathogens (Zimmerli et al. 2004). More recently, the "abiotic" stress hormone ABA as well as the "developmental" hormones auxin, gibberellic acid, cytokinins and brassinosteroids have also been recognized as important players in plant immunity (Mauch-Mani and Mauch 2005; Robert-Seilaniantz et al. 2007; Grant and Jones 2009). Therefore, all major plant hormones appear to contribute to biotic stress responses. These contributions may be direct or indirect since there is extensive crosstalk between plant hormone signaling pathways, such as the well documented antagonism between SA and JA/ET (Gupta et al. 2000; Spoel et al. 2003; Chen et al. 2009). In addition ABA has also been demonstrated to interact antagonistically with SA and JA/ET (Mauch-Mani and Mauch 2005; Robert-Seilaniantz et al. 2007; Yasuda et al. 2008; Anderson et al. 2004). Therefore, targeting hormone pathways and alteration of hormone homeostasis can be an effective strategy by which pathogens alter host immunity (Grant and Jones 2009). The inter-relationships between hormone pathways and their contributions to plant immunity have been extensively discussed in recent reviews (Spoel and Dong 2008; Grant and Jones 2009). Below, we focus on recent literature dealing with the roles of ABA in plant immunity, beginning with the crosstalk between ABA and SA responses.

ABA–SA antagonism

SA is a critical signaling molecule that mediates plant defence responses against numerous biotrophic/hemibiotrophic pathogens, as well as induction of systemic acquired resistance that confers a long-lasting, broad spectrum resistance against pathogen infection (Durrant and Dong 2004). The importance of ABA in abiotic stress responses, in particular drought and salinity stress, is well recognized and has been extensively reviewed in this special issue as well as elsewhere (Tuteja 2007; Xiong et al. 2002; Zhu 2002). Several studies have demonstrated antagonistic interactions between ABA and SA responses. This antagonism can significantly contribute to plant immunity as well as responses to environmental stresses, providing an excellent system to investigate the crosstalk between biotic and abiotic stress responses.

Lesion mimic mutants (LMMs) display constitutive defence responses reminiscent of an HR (Moeder and Yoshioka 2008). In addition to spontaneous cell death, the LMM *cpr22* (*constitutive expresser of PR genes 22*) and *ssi4* (*suppressor of SA insensitivity of npr1-5 4*) also exhibit constitutive expression of defence-related genes such as *PATHOGENESIS-RELATED GENE 1* (*PR1*), enhanced resistance against virulent *P. syringae* pv. *maculicola* (*Pma*) ES4326 (a hemibiotroph) and *Hyaloperonospora arabidopsidis* (a biotroph), as well as constitutively elevated SA accumulation (Yoshioka et al. 2006; Mosher et al. 2010; Shirano et al. 2002). Interestingly, *cpr22* displays ABA insensitive phenotypes including enhanced water loss, as well as reduced sensitivity to ABA-induced stomatal closure, germination arrest and reduced ABA marker *RESPONSIVE TO ABA 18* (*RAB18*) and *RESPONSIVE TO DESICCATION 29B* (*RD29B*) expression (Mosher et al. 2010). Expression of the bacterial salicylate hydroxylase gene *NahG* in *cpr22* to prevent SA accumulation led to attenuation of ABA associated phenotypes suggesting that SA antagonizes ABA signaling in *cpr22* Arabidopsis plants (Mosher et al. 2010; Moeder et al. 2010).

Yasuda et al. (2008) demonstrated that bidirectional antagonism between SA and ABA can occur at multiple points of their signaling pathways. Biologically active SA analogs BIT (1,2-benzisothiazol-3(2H)-one1,1-dioxide) and BTH (benzo(1,2,3)thiadiazole-7 carbothioic acid S-methyl ester) were utilized to activate upstream and downstream of SA biosynthesis, respectively (Yasuda et al. 2008; Durrant and Dong 2004). Both BIT and BTH treatment reduced growth of virulent *P. syringae* pv. *tomato* (*Pst*) DC3000 in Arabidopsis which was abolished by ABA pre-treatment. ABA also effectively suppressed BIT-induced SA accumulation and expression of the SA-biosynthetic gene *ISOCHORISMATE SYNTHASE 1 (ICS1)* [a.k.a. *SA INDUCTION DEFICIENT 2* (*SID2*)] in Arabidopsis wildtype, as well as BTH-induced *PR1* expression in *sid2-1* and SA-related mutant *enhanced disease susceptibility 5-1* (*eds5-1*; Yasuda et al. 2008). These results indicate that ABA can suppress SA signaling both upstream and downstream of SA biosynthesis.

Similar to the effect of ABA, pretreatment of NaCl was able to suppress BIT-induced SA accumulation and BIT/BTH-induced *PR1* induction and disease resistance against *Pst* DC3000 (Yasuda et al. 2008). Thus, NaCl treatment, like ABA, can inhibit SA-mediated signal transduction both upstream and downstream of SA biosynthesis. In Arabidopsis plants constitutively expressing the ABA-degrading cytochrome P450 family 707 subfamily A polypeptide 3 (CYP707A3), NaCl pretreatment failed to suppress BIT- or BTH-induced *PR* gene expression, SA accumulation and *Pst* DC3000 resistance, demonstrating a prominent role for ABA in resistance suppression by NaCl. Inversely, BIT treatment was able to suppress NaCl-induced expression of ABA biosynthesis as well as induction of the ABA-responsive genes *ABA DEFICIENT 1* (*ABA1*), *NINE-CIS-EPOXYCAROTENOID DIOXYGENASE 3* (*NCED3*), *RESPONSIVE TO ABA 18* (*RAB18*), *COLD-REGULATED 15A* (*COR15A*), *JASMONATE INSENSITIVE 1* (*JIN1/MYC2*) and *RESPONSIVE TO DEHYDRATION 22* (*RD22*) (Yasuda et al. 2008). Suppression of *NCED3*, *RAB18* and *JIN1/MYC2* induction was independent of the SA response regulator *NONEXPRESSOR OF PATHOGENESIS-RELATED PROTEINS 1* (*NPR1*) while induction of *ABA1*, *COR15A* and *RD22* was *NPR1*-dependent. Thus, the SA signaling pathway antagonizes ABA-mediated signal transduction in both an NPR1-dependent and -independent manner. These data support the existence of multiple nodes of antagonism between the ABA- and SA-mediated stress signaling pathways.

Further support for multiple nodes of ABA–SA antagonism was observed in rice where ABA suppressed SA- and BTH-induced expression of both *WRKY45* and *OsNPR1*, which regulate two separate branches of SA signaling in rice (Jiang et al. 2010). Overexpression of *WRKY45* or *OsNPR1* alleviated ABA-induced susceptibility to the hemibiotrophic rice blast fungus *Magnaporthe grisea*, indicating that suppression of SA signaling by ABA occurs upstream of *WRKY45* and *OsNPR1*. Since ABA-induced susceptibility was not completely abolished by *WRKY45* or *OsNPR1* overexpression, ABA also seems to act downstream of *WRKY45* and *OsNPR1* to enhance disease susceptibility.

The ABA-deficient tomato mutant *sitiens* also displays elevated expression of the SA biosynthesis gene *PHENYLALANINE AMMONIA LYASE* (*PAL*), hypersensitivity to BTH-induced *PR1* expression and enhanced resistance to *Botrytis cinerea* compared to wildtype plants (Audenaert et al. 2002). Together, these results indicate that ABA levels in wildtype tomato negatively influences SA-mediated defence pathway(s) potentially via the suppression of *PAL* expression and SA biosynthesis.

In Arabidopsis, expression analyses of the SA-biosynthetic gene *ICS1* and the ABA-biosynthetic gene *NCED3* following *Pst* DC3000 infection revealed that upregulation of *ICS1* and *NCED3* preceded SA and ABA increase, suggestive of de novo biosynthesis in response to pathogen (de Torres-Zabala et al. 2009). In the ABA deficient *abscisic aldehyde oxidase 3* (*aao3*) mutant, increased resistance was accompanied by a lower basal ABA level but a higher basal SA level. In addition, basal *ICS1* transcript level was approximately five times higher in *aao3* than wildtype, and showed an earlier and stronger increase in response to *Pst* DC3000, suggesting that ABA is able to suppress SA biosynthesis via downregulation of *ICS1*. In contrast, a reduction of endogenous ABA was observed in *Pst* DC3000-infected *sid2-1* compared to wildtype, indicating positive regulation of ABA by SA (de Torres-Zabala et al. 2009). This is also supported by recent results showing that the transcription factor MYB96 positively regulates both ABA-mediated abiotic stress signaling and SA-mediated pathogen resistance (Seo and Park 2010). As such, the interactions between SA and ABA signaling may not be strictly antagonistic. Nevertheless, the crosstalk between ABA and SA signaling pathways appears to be predominantly antagonistic and many studies have demonstrated that ABA can compromise plant immunity.

The role of ABA in plant–microbe interactions: compromising immunity

Various approaches have been used to investigate the impact of ABA on plant–pathogen interactions. These include the use of mutants with altered ABA biosynthesis or signaling, as well as exogenous application of ABA (Asselbergh et al. 2008a, b). The results of these studies largely support a negative role of ABA in plant immunity.

Bacterial growth of the avirulent *Pst* 1065 in Arabidopsis increased with ABA pretreatment via root uptake (Mohr and Cahill 2003). Similarly, floating virulent *Pst*-infected Arabidopsis leaves on 10 μM ABA also enhanced bacterial growth (Fan et al. 2009). Spraying of 4 and 20 μM ABA on susceptible rice leaf sheaths also led to more severe disease responses to the rice blast fungus *M. grisea* (Koga et al. 2004). Jiang et al. (2010) analyzed the results of compatible and incompatible interactions between rice and *M. grisea* after pretreatment of 0.1 mM ABA by spraying. Disease symptoms, including lesion development and quantification of fungal DNA relative to rice DNA, were more severe for both virulent and avirulent pathogens following ABA pretreatment. Exogenous application of ABA was also able to suppress the resistance of the ABA deficient tomato mutant *sitiens* to the biotroph *Oidium neolycopersici* and the necrotroph *B. cinerea*

(Achuo et al. 2006). Using genetic approaches, Audenaert et al. (2002) demonstrated that ABA negatively influences tomato immunity against *B. cinerea*. The ABA-deficient tomato mutant *sitiens* contains only 8% of wildtype level of ABA and exhibited enhance resistance against *B. cinerea*. This was also supported in Arabidopsis where the ABA biosynthetic mutants *aba2-12* and *aao3-2* also displayed enhanced resistance to *B. cinerea* (Adie et al. 2007). Similarly, ABA insensitive Arabidopsis mutants, *ABA insensitive 2-1* (*abi2-1*) and *ABA insensitive 1-1* (*abi1-1*) were more resistant to *P. syringae* whereas the ABA hypersensitive mutant *enhanced response to abscisic acid 1* (*era1*) was more susceptible (de Torres-Zabala et al. 2007; Goritschnig et al. 2008). Therefore, increases in ABA levels appear to correlate with *P. syringae* virulence supporting a negative role in plant immunity. These examples demonstrate that ABA levels or ABA sensitivity negatively correlate with resistance to both biotrophic and necrotrophic pathogens. In other words, increased ABA levels or ABA sensitivity result in decreased resistance and vice versa.

Some phytopathogenic organisms are known to produce ABA, including fungal pathogens such as *Cercospora* spp., *Ceratocystis* spp., *Fusarium* spp., *Rhizoctonia* spp., *B. cinerea* and *M. grisea* (Assante et al. 1977; Dörffling et al. 1984; Jiang et al. 2010). Kettner and Dörffling (1995) showed that at least four processes can control ABA levels in tomato leaves infected with *B. cinerea*: (1) stimulation of fungal ABA biosynthesis by the host; (2) release of ABA or its precursor by the fungus; (3) stimulation of plant ABA biosynthesis by the fungus and (4) inhibition of ABA catabolism by the fungus. In addition, ABA was shown to accumulate in the hyphae and conidia of *M. grisea* and was also detected in culture filtrate indicative of ABA secretion (Jiang et al. 2010). The ABA biosynthetic pathway of pathogens was likely not acquired by horizontal gene transfer from their respective host plants since characterized ABA biosynthesis pathways in pathogens are different from their plant counterparts (Siewers et al. 2006; Robert-Seilaniantz et al. 2007). Since there is no evidence supporting the role of ABA in the physiology of pathogens, it is therefore likely that pathogens have evolved ABA biosynthetic machinery to dampen plant immunity and promote the infection process.

Focus on the *Pseudomonas syringae*–Arabidopsis pathosystem

Much of our current understanding of the molecular aspects of plant immunity has come from studies in Arabidopsis (Nishimura and Dangl 2010). The interaction between the Gram negative bacterial phytopathogen *Pseudomonas syringae* and Arabidopsis has emerged as a model pathosystem to study the molecular details of plant–microbe interactions including the role of ABA in plant immunity (Gimenez-Ibanez and Rathjen 2010).

A current model of the plant immunity divides the plant immune system into two major branches (Jones and Dangl 2006; Chisholm et al. 2006). We describe this model below and discuss how ABA influences both branches of plant immunity with particular emphasis on the Arabidopsis/*P. syringae* pathosystem.

The zig-zag model of plant immunity

The plant immune system can be divided into two major branches based on their mode of phytopathogen recognition (Jones and Dangl 2006; Chisholm et al. 2006). Pathogen-associated molecular pattern (PAMP)-triggered immunity or PTI is triggered by recognition of conserved structural components of microbes (Jones and Dangl 2006). PTI-induced responses include stomatal closure, callose deposition and upregulation of PAMP-induced genes such as *FLG22-INDUCED RECEPTOR-LIKE KINASE 1* (*FRK1*) and the glycerol kinase encoding *NONHOST RESISTANCE TO P. S. PHASEOLICOLA 1* (*NHO1*) (Hauck et al. 2003; Melotto et al. 2006; Asai et al. 2002; Li et al. 2005). Phytopathogens have evolved the ability to secrete phytotoxins, extracellular polysaccharides and proteinaceous effectors into plant cells to enhance pathogen virulence and suppress plant immune responses (Boller and He 2009). Many Gram negative bacterial pathogens such as the hemibiotroph *P. syringae* employ a type III secretion system (TTSS) for delivery of type III effector proteins into host cells. Plant resistance or R proteins can recognize the presence or activities of pathogen effectors to induce effector-triggered immunity or ETI, which is also known as R-gene-mediated resistance, the second major branch of the plant immune response (Jones and Dangl 2006). Recognition of effectors results in a robust immune response that is often associated with a localized programmed cell death or HR, rendering the pathogen avirulent. Induction of both PTI and ETI involves SA (DebRoy et al. 2004; Dempsey et al. 1999; Loake and Grant 2007).

ABA and pre-invasive PAMP-triggered immunity

Studies have demonstrated the influence of endogenous ABA on pre- and post-invasive PAMP-triggered immune responses in Arabidopsis. Stomata represent a major route of entry into plant tissues for many phytopathogens (Zeng et al. 2010). PAMP-induced stomatal closure (a.k.a. stomatal immunity) can actively prevent *P. syringae* invasion of leaf tissue via stomatal openings (Melotto et al. 2006). The role of ABA in regulating stomatal closure is well established, mainly in relation to abiotic stress such as

drought (Schroeder et al. 2001). However, ABA also contributes to stomatal immunity in response to biotic stress in conjunction with the SA pathway. *Pst* DC3000 induced stomatal closure in Arabidopsis Col-0 wildtype but not the SA deficient mutants *eds16-1* or *NahG* transgenic plants (Melotto et al. 2006). Also, the ABA deficient mutant *aba3-1* was not responsive to stomatal closure induced by two PAMPs, flg22 (a conserved peptide from flagellin) and lipopolysaccharide (constituent of outer membrane of Gram negative bacteria). When dip inoculated, these ABA and SA mutants supported more growth of the coronatine deficient *Pst DC3118* (the bacterial toxin coronatine suppresses stomatal immunity), demonstrating the importance of both SA and ABA in preventing bacterial invasion (Melotto et al. 2006). More recently, it was demonstrated that the SA signaling regulator NPR1 acts downstream of SA but upstream of ABA in stomatal immunity (Zeng and He 2010). Interestingly, elements that were shown to be required for PAMP-induced stomatal closure, namely ABA biosynthesis, nitric oxide production and the kinase *OPEN STOMATA 1*(*OST1*), are also important for sensing abiotic stresses in guard cells (Schroeder et al. 2001; Fan et al. 2004; Yoshida et al. 2002). Therefore, at the early preinvasion stage of the Arabidopsis–Pseudomonas interaction, there is important crosstalk between ABA- and SA-signaling pathways with both ABA and SA playing a positive role in regulating stomatal immunity (Fig. 1).

Both *P. syringae* and *Xanthomonas campestris* pv. *campestrisis* (*Xcc*) have evolved virulence factor(s) to suppress stomatal immunity in order to allow stomatal reopening and pathogen entry. The *P. syringae* phytotoxin coronatine can overcome stomatal immunity induced by PAMP-triggered stomatal closure (Melotto et al. 2006; Zeng and He 2010). Similarly, *Xcc* induces rapid stomatal closure in response to infection (1 hpi) followed by reopening soon after (3hpi) (Gudesblat et al. 2009). It was recently demonstrated that the diffusion signal factor (DSF) system important for cell to cell communication was also required to revert stomatal closure. The *Xcc* mutants *rpfF* and *rpfC* result in defective DSF synthesis and perception, respectively. These mutations abolished the ability of *Xcc* to revert stomatal closure demonstrating that an intact DSF system is required to suppress stomatal immunity (Gudesblat et al. 2009). *Mitogen-Activated Protein Kinase 3* (*MPK3*) was found to be necessary for PAMP-induced stomatal closure but not ABA-induced stomatal closure. Furthermore, *MPK3* was required for *Xcc* to reopen stomata (Gudesblat et al. 2009). These results support a model where PAMP-triggered stomatal closure requires *MPK3* whereas ABA-induced stomatal closure is downstream of *MPK3*.

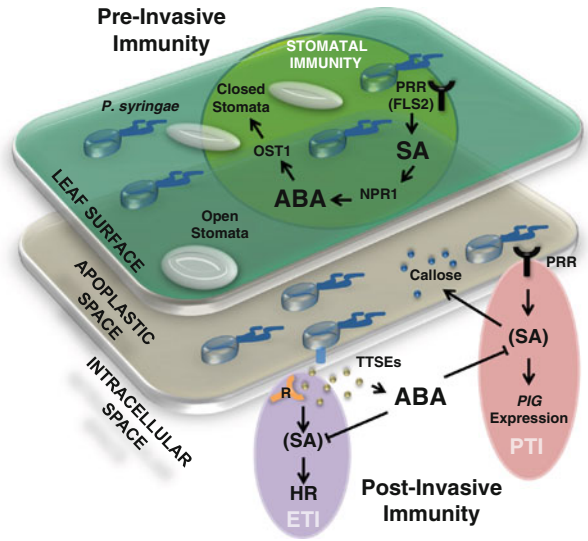

Fig. 1 The involvement of ABA in pre- and post-invasive immune responses in the *Arabidopsis thaliana–Pseudomonas syringae* pathosystem. ABA plays a positive role in pre-invasive stomatal immunity by induction of stomatal closure to prevent pathogen entry. On the other hand, ABA plays a negative role in post-invasive PAMP-triggered immunity (PTI) and effector-triggered immunity (ETI). Both stomatal immunity and post-invasive PTI are triggered by the recognition of PAMPs (pathogen associated molecular patterns) by pattern recognition receptors (PRRs). Post-invasive ETI against *P. syringae* is triggered by the recognition of type III secreted effector (TTSEs) by resistance (R) proteins. The hypersensitive response (HR) is often associated with ETI whereas markers of PTI include activation of *PIG* (*PAMP-induced gene*) expression as well as callose deposition. Crosstalk between ABA and SA signaling pathways plays a role in both stomatal and post-invasive immunity. In stomatal immunity SA appears to act upstream of ABA to close stomata, whereas antagonism of the SA pathway by ABA likely contributes to suppression of PTI and ETI. Note that there are SA-independent components to both PTI and ETI (DebRoy et al. 2004; Lewis et al. 2010). *P. syringae* TTSEs can upregulate ABA biosynthesis and/or signaling, potentially to suppress post-invasive immunity

ABA and post-invasive PAMP-triggered immunity

In addition to stomatal immunity, recognition of PAMPs also leads to post-invasive resistance responses such as callose deposition that acts as physical barriers against pathogen penetration as well as the upregulation of *PAMP-induced genes* (*PIGs*) (Truman et al. 2006). Using pressure infiltration of *P. syringae* into Arabidopsis leaves to determine post-invasive growth, it has been demonstrated that plants defective in ABA biosynthesis or perception display enhanced resistance against *P. syringae*, whereas ABA hypersensitive mutants display enhanced susceptibility (de Torres-Zabala et al. 2007; Goritschnig et al. 2008). Furthermore, ABA treatment before *Pst* DC3000 inoculation resulted in reduced formation of callose deposits compared to the mock treatment (de Torres-Zabala et al. 2007). These results support a negative role

for ABA in the post-invasive PTI response to *P. syringae* (Fig. 1).

In other pathosystems, ABA appears to influence callose deposition differently. Callose is crucial for BABA-induced resistance against *Plectosphaerella cucumerina* (a necrotroph) since the callose deficient mutant *powdery mildew resistant 4-1* (*pmr4-)1* failed to establish β-amino butyric acid (BABA)-induced resistance to this fungus (Ton and Mauch-Mani 2004). Mutations in the ABA biosynthetic gene *ABA1* (*ABA deficient 1*; a.k.a. *impaired BABA-induced sterility 3*), resulted in significant reductions of BABA-induced callose deposition as well as decreased resistance against the biotrophic oomycete pathogen *H. arabidopsidis* (Ton et al. 2005). In contrast, Adie et al. (2007) showed that ABA deficiency (using mutant *aao3-2*) did not influence callose production in response to the necrotrophic oomycete *Pythium irregulare*, but did enhance resistance to this pathogen, indicating that callose deposition may not always correlate with resistance.

Upregulation of *PIGs* represents an additional important marker of PTI (de Torres et al. 2003; Truman et al. 2006). It was shown that both before and after *Pst* DC3000 infection, expression of a number of *PIGs*, including *FLAGELLIN-SENSITIVE 2* (*FLS2*) encoding the flagellin sensing kinase, the flagellin-induced transcription factor *WRKY30*, as well as *FRK1* and *NHO1*, was elevated in the ABA deficient mutant *aao3* but attenuated in SA deficient mutant *sid2-1* (de Torres-Zabala et al. 2009). Thus, SA and ABA play opposite roles in the regulation of *PIG* expression. Through epistatic analysis, *sid2-1* enhanced susceptibility was determined to be dominant over *aao3* acquired resistance as single mutant *sid2-1* and the double mutant *aao3 sid2-1* showed similar low levels of *FRK1* expression and enhanced susceptibility to *Pst* DC3000 (de Torres-Zabala et al. 2009). Overall, these results support the hypothesis that ABA suppresses SA-mediated PAMP-induced accumulation of defence genes (Fig. 1).

ABA and effector-triggered immunity

In addition to PTI, studies of ETI indicate that this important branch of plant immunity is also influenced by ABA signaling. Arabidopsis plants treated with 100 μM ABA prior to inoculation with avirulent *Pst* 1065 (ETI due to recognition of the TTSS AvrRpt2 by the R protein RPS2; Whalen et al. 1991; Bent et al. 1994; Mindrinos et al. 1994) led to more extensive spread of chlorosis and increased bacterial numbers that were comparable to that observed with virulent *P. syringae* (Mohr and Cahill 2003). Deposition of lignin was associated with HR in response to *Pst* 1065 infection and was abolished by exogenous ABA treatment before infection (Mohr and Cahill 2007). In addition, ABA pretreatment also suppressed conjugated SA accumulation induced by *Pst* 1065. However, free SA was not significantly affected (Mohr and Cahill 2007). Together, these data support the role of ABA as a regulator of ETI responses in Arabidopsis (Fig. 1). Intriguingly, it was recently demonstrated that virulent *Pst* DC3000 was more effective than avirulent *Pst* carrying *avrRpt2* at reopening the stomata of Arabidopsis leaves indicating that ETI can influence pre-invasive immunity (Melotto et al. 2006).

Type III effectors manipulate ABA signaling

Recent studies have demonstrated the direct manipulation of ABA signaling by type III effectors as a virulence strategy for *P. syringae*. Comparison of *Pst* DC3000 wildtype and the TTSS-defective *hrpA-* demonstrated the ability of type III effectors to induce ABA biosynthesis and expression of the ABA-biosynthesis gene *NCED3* (de Torres-Zabala et al. 2007). Microarray analysis showed a significant overlap of genes that are differentially induced by *P. syringae* type III effectors and genes that are either associated with ABA biosynthesis/signaling or responsive to abiotic stresses (de Torres-Zabala et al. 2007). Remarkably, conditional overexpression of a single effector, AvrPtoB, was sufficient for inducing endogenous ABA accumulation. In another example, transgenic Arabidopsis plants expressing HopAM1 were more susceptible to the weakly virulent *P. syringae* strain *Pma* M6CΔE, with greater enhanced susceptibility under slight drought conditions (Goel et al. 2008). Unlike AvrPtoB, HopAM1 did not alter ABA biosynthesis. Instead, ABA sensitivity was altered. HopAM1 was found to enhance stomatal closure and germination arrest induced by ABA, as well as salt tolerance, indicative of ABA hypersensitivity. Since *P. syringae* does not require HopAM1 for virulence in Arabidopsis, the authors hypothesized that HopAM1 may work in concert with other type III effectors and may provide advantages for infection under specific environmental conditions, such as during drought stress. Furthermore, once secreted into host plant cells, HopAM1 may alter ABA signaling in order to optimize osmotic conditions for bacterial growth. Thus, it appears that *P. syringae* has evolved multiple type III effector proteins to target different points of ABA biosynthesis or perception/signaling in order to promote virulence. Upregulation of the ABA pathway would represent an effective strategy to dampen both PTI and ETI branches of post-invasive immunity (Fig. 1). However, Fan et al. 2009 reported that infection of Arabidopsis with virulent *Psm* but not avirulent *Psm* resulted in induction of *NCED2* and *NCED5* (two *NCED* genes involved in ABA biosynthesis) suggesting that ETI can interfere with *P. syringae* induced ABA biosynthesis and compromise this virulence strategy.

ABA-induced resistance

Although most studies support a negative role of ABA in defence against biotic stresses, a number of notable exceptions exist. For example, exogenous ABA application was able to reduce fungal spreading of the virulent necrotrophs *Cochliobolus miyabeanus* in mesophyll tissue of rice leaf sheaths (Vleesschauwer et al. 2010). ABA induced resistance against *C. myabeanus* appears to be achieved by suppression of *C. miyabeanus*-triggered activation of ethylene signaling (Vleesschauwer et al. 2010). In another example of a positive role for ABA in plant immunity, soil drenching Arabidopsis with 80 μM ABA before inoculation of necrotrophic fungal pathogens *Alternaria brassicicola* and *P. cucumerina* reduced lesion size demonstrating that exogenously applied ABA can also promote resistance (Ton and Mauch-Mani 2004). ABA also plays a positive role in resistance against the necrotrophic pathogens *P. irregulare* (oomycete) and *A. brassicicola* (fungus) since the ABA defective mutants *aba2-12*, *aao3-2* and *abi4-1* were more susceptible to these pathogens (Adie et al. 2007). Jasmonic acid (JA) insensitive *coi1-1* plants were highly susceptible to *P. irregulare* suggesting that JA plays a prominent role in immunity against *P. irregulare* (Adie et al. 2007). This is in contrast to *B. cinerea* resistance which appeared to be JA-independent (Audenaert et al. 2002). Additionally, *aba2-12* exhibited lower increases in JA in response to *P. irregulare* infection. These results support a model where ABA production contributes to JA accumulation and activation for resistance against *P. irregulare*. In addition, resistance against some viral pathogens also appears to be positively influenced by ABA (Mauch-Mani and Mauch 2005).

Another facet of the plant immune response is priming, a phenomenon in which plants that are preexposed to certain pathogens or chemical compounds develop faster and/or stronger activation of defence responses against various types of abiotic and biotic stress (Conrath et al. 2002). Application of the nonprotein amino acid BABA enhances tolerance to abiotic stress as well as resistance against biotic stress (Ton et al. 2005). This example of priming appears to be positively influenced by ABA signaling since ABA insensitive mutant plants, *abi1-5* and *abi4-1* failed to develop BABA-induced resistance against the necrotrophic fungal pathogen *P. cucumerina* (Ton and Mauch-Mani 2004).

It is apparent that ABA can also promote disease resistance in certain plant pathosystems, particularly resistance against some viral and necrotrophic pathogens. However, the molecular mechanisms of ABA-mediated resistance remain to be elucidated.

Crosstalk between biotic and abiotic stress

The observations that ABA can antagonistically interact with the prominent defence phytohormones SA, JA/ET suggest that plant abiotic stress responses can take precedence over biotic stress responses and that abiotic stress may be detrimental to plant immunity (Mauch-Mani and Mauch 2005; Robert-Seilaniantz et al. 2007; Yasuda et al. 2008; Anderson et al. 2004). In support of this, a number of abiotic stresses such as increases in temperature and humidity as well as drought and salinity stress, have been demonstrated to have a negative effect on resistance to biotic stress (Mohr and Cahill 2003; Koga et al. 2004; Moeder and Yoshioka 2009; Yoshioka and Shinozaki 2009). It has been reported that drought stress resulted in susceptible phenotypes including necrosis and chlorosis, as well as enhanced bacteria growth in Arabidopsis infected with avirulent *Pst* 1065 (Mohr and Cahill 2003). As described above, drought conditions also enhanced growth of *Pma* M6CΔE in transgenic Arabidopsis plants expressing the effector HopAM1 (Goel et al. 2008). Similarly, drought stress has also been shown to increase the susceptibility of bean plants (*Phaseolus vulgaris*) to the charcoal rot causal fungus *Macrophomina phaseolina*, as well as vine (*Parthenocissus quinquefolia*) to the xylem-limited bacteria *Xylella fastidiosa* (Mayek-Perez et al. 2002; McElrone et al. 2001). Although in most cases abiotic stress responses increase disease susceptibility, exceptions are resistance of tomato against *Botrytis cinerea* (necrotroph) and *Oidium neolycopersici* (biotroph) where pre-exposure to drought stress resulted in elevated resistance against these pathogens (Achuo et al. 2006). These exceptions as well as those pathosystems where ABA promotes resistance cannot be generalized to one particular pathogen lifestyle (biotroph, necrotroph, hemibiotroph) and likely represent consequences of the specific virulence strategies adopted by individual pathogen species. In addition, since SA–ABA antagonism appears to be bidirectional, it will be interesting to further investigate the influence of biotic stress on abiotic stress tolerance (Yasuda et al. 2008; Mosher et al. 2010; Moeder et al. 2010; Yoshioka and Shinozaki 2009).

ABA mutants and plant immunity: proceed with caution

It must be noted that due to the importance of ABA for plant physiology, ABA deficiency often results in growth or developmental defects. Plants with impaired ABA biosynthesis and sensitivity commonly exhibit a dwarf and wilting phenotype [e.g. *aba2-2* (Nambara et al. 1998) and *abi1-1* (Armstrong et al. 1995)]. The ABA hypersensitive

enhanced response to ABA 1-2 (*era1-2*) plants have enlarged meristems and organs (Yalovsky et al. 2000). Evaluation of disease phenotypes of ABA mutants should screen for differences in pathogen susceptibility due to developmental differences between mutant and wild type. For example, the *ABA hypersensitive germination 2-1* (*ahg2-1*) mutant is ABA hypersensitive and has high endogenous ABA accumulation (Nishimura et al. 2005). It was tested for pathogen resistance and exhibited reduced severity of disease symptoms; namely the absence of chlorosis in response to *P. syringae* (*Pst* DC3000 and *Pma*), as well as reduction of necrosis in response to *B. cinerea* (Nishimura et al. 2009). *ahg2-1* also supported a lower *Pst* DC3000 growth compared to Col-0 in bacterial growth assays. In addition, Nishimura et al. (2009) further showed *ahg2-1* contains higher total and free SA accumulation before infection but lower SA accumulation in response to *Pst* DC3000 when compared to wildtype. The ability of *ahg2-1* to accumulate high ABA and SA contradicts other studies that demonstrated the antagonistic relationship between ABA and SA. Additional analysis showed that ABA and SA were not responsible for many of the phenotypes in *ahg2-1*, and that the ABA-related, SA-related and dwarf phenotypes were independent of each other (Nishimura et al. 2009). *ahg2-1* likely affects mitochondrial function via pathways that are independent of ABA or SA, which may contribute to enhanced immunity. In another example, a suppressor screen of the defence related mutant *suppressor of npr1-1* (*snc1*) identified a novel loss-of-function allele of *ERA1* (Goritschnig et al. 2008). *ERA1* encodes the β-subunit of protein farnesyltransferase (Cutler et al. 1996). *era1* plants displayed hypersusceptibility to infection with virulent and avirulent *P. syringae* as well as *H. arabidopsidis* (Cutler et al. 1996; Goritschnig et al. 2008). However, ABA-biosynthetic mutants were only partially able to reduce susceptibility in the *era1* background indicating that ERA1 plays an ABA-independent role in plant immunity. These studies demonstrate the complexity of ABA-related phenotypes and emphasize the importance of dissecting the direct effects of ABA on plant immunity from other pleiotropic phenotypes. One approach for this may involve supplementing ABA auxotrophs with ABA during growth to minimize developmental effects, followed by ABA deprivation during pathogenicity assays. Furthermore, since ABA signaling forms part of a network with other hormone signaling pathways, it will be important to identify the nodes of crosstalk between these pathways in order to understand the individual contributions of each phytohormone to plant immunity.

An important contribution to our understanding of ABA signaling was the recent identification of the RCAR/PYR/PYL ABA receptors (Park et al. 2009; Ma et al. 2009). These pyrabactin resistance 1 (PYR) [also known as pyrabactin resistance 1-like or regulatory component of ABA receptor (PYL/RCAR)], belong to the START-domain or Bet v I-fold superfamily of proteins that are characterized by a conserved hydrophobic ligand-binding pocket (Cutler et al. 2010). The emerging model of ABA signaling network involves the following components: PYR/PYL/RCAR ABA receptors, the negative regulators type 2C protein phosphatases (PP2Cs), the positive regulators SNF1-related kinase 2 (SnRK2 kinases) as well as ABA-responsive element binding factors (ABFs) (Cutler et al. 2010; Fujii et al. 2009). Under non-ABA inducing conditions, active PP2C phosphatases inactivate SnRK2 kinases thereby suppressing ABA signaling. Under ABA inducing conditions, PYR/PYL/RCAR receptor proteins bind to and inactivate the PP2Cs; an interaction mediated by ABA. Active SnRK2 kinases phosphorylate ABF transcription factors which then induce ABA responsive genes. In vivo, PYR/PYLs, PP2Cs, SnRK2s and ABF transcription factors are necessary and sufficient for ABA perception, signaling and activation of ABA responsive gene expression (Fujii et al. 2009). It will be important to determine the involvement of the core ABA signaling network components in disease resistance using loss-of-function and gain-of-function mutations. Since the PYR/PYL/RCAR family includes 14 members, functional redundancy may be an issue. This may be addressed by mutating multiple related and/or co-expressed family members as was done to reveal ABA-related phenotypes (Park et al. 2009). Components of the core ABA signaling pathway represent promising candidates involved in the crosstalk with other hormone signaling pathways, particularly SA, JA and ET. Since mutations of the core ABA genes are likely to affect other hormone signaling pathways, it will be important to conduct global analyses of their effects on other hormone pathways and address pleiotropic effects using combinatorial hormone signaling mutants.

Concluding remarks

With increasing incidence of extreme environmental fluctuations associated with climate change, it is fundamental to understand the crosstalk between abiotic and biotic stress responses if we are to assess their impact on the fitness and yield of the world's vegetation. The "abiotic" stress hormone ABA mediates responses and tolerance against unfavourable environmental conditions, and as demonstrated throughout this review also affects the outcome of biotic stress. It therefore represents a critical player in the interrelationship between abiotic and biotic stress signaling that will be crucial for engineering and

breeding crop species with improved abiotic stress tolerance and pathogen resistance.

Acknowledgments We thank Dr. Peter McCourt and Dr. Shelley Lumba for helpful discussions about ABA signaling. We thank anonymous reviewers for insightful and thorough comments. Work in the Desveaux and Yoshioka labs is supported by the Natural Sciences and Engineering Research Council (NSERC) of Canada. D.D. is a Canada Research Chair in Plant–Microbe Systems Biology.

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