Published in "Current Opinion in Plant Biology 8: 409-414, 2005" which should be cited to reference this work

The role of abscisic acid in plant–pathogen interactions

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The effect of the abiotic stress hormone abscisic acid on plant disease resistance is a neglected field of research. With few exceptions, abscisic acid has been considered a negative regulator of disease resistance. This negative effect appears to be due to the interference of abscisic acid with biotic stress signaling that is regulated by salicylic acid, jasmonic acid and ethylene, and to an additional effect of ABA on shared components of stress signaling. However, recent research shows that abscisic acid can also be implicated in increasing the resistance of plants towards pathogens via its positive effect on callose deposition.

Introduction

The plant hormone abscisic acid (ABA) plays important roles in many aspects of plant development, in the regulation of stomatal aperture, and in the initiation of adaptive responses to various environmental conditions. Adaptation to drought, low temperature and salinity is regulated by the combinatorial activity of interconnected ABA-dependent and ABA-independent signaling pathways [1]. By contrast, the plant hormones salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) play major roles in disease resistance. These biotic stress hormones do not control isolated linear signaling pathways but are part of a complex network of synergistic and antagonistic interactions [2,3]. Although ABA-controlled and biotic-stress signaling appear to share many common elements, the role of ABA in plant disease resistance is not well defined. Abiotic and biotic stress signaling have remained mostly separate fields of research despite the awareness that plants have to cope with and adapt to situations in which they are simultaneously exposed to several stresses in their natural environment. Recent evidence suggests the existence of a significant overlap between signaling networks that control abiotic stress tolerance and disease resistance.

The role of ABA in disease resistance

On the basis of experiments with exogenous application of ABA, inhibition of ABA biosynthesis and/or the use of ABA-deficient mutants it has been shown that enhanced ABA levels correlated with increased susceptibility and that a reduction below wildtype (WT) levels increased resistance to many pathogens [4–12,13•,14,15]. Changes in ABA concentration following the inoculation of plants with pathogens were rarely measured in these experiments. Reduced ABA levels were observed in beans upon inoculation with rust [16]. In soybeans that were inoculated with *Phytophthora*, a decrease in ABA concentration occurred only in the incompatible reaction [17]. By contrast, viral infection of tobacco led to an increase in ABA concentration [18]. The observed changes in ABA concentration were, however, modest compared to the dramatic changes in SA, JA and/or ET production during pathogenesis.

Abiotic stress has a strong effect on ABA accumulation and is known to influence the outcome of plant–pathogen interactions [19]. The susceptibility of rice plants to *Magnaporthe grisea* was increased by application of ABA and following cold stress [15]. Inhibition of ABA synthesis prevented the cold-induced susceptibility; hence, ABA is a key factor in the suppression of disease resistance to *M. grisea*. With regard to ABA-induced susceptibility, it is worth noting that several fungal pathogens can produce ABA [20,21].

There are also reports of a positive correlation between ABA levels and disease resistance. Viral infection increased ABA concentrations in tobacco, and treatment with ABA increased virus resistance [18,22]. Interestingly, ABA inhibited the transcription of a basic β-1,3-glucanase [23] that can degrade the β-1,3-glucan callose, forming a physical barrier to viral spread through plasmodesmata. Plants that were deficient in basic β-1,3-glucanase were more resistant than WT plants to viral infection [24]. The downregulation of β-1,3-glucanase by ABA can function therefore as a resistance factor in plant–virus interactions but also has the potential to compromise basic resistance towards fungal and oomycete pathogens.

In *Arabidopsis*, ABA treatment or simulated drought stress that resulted in a large increase in ABA concentrations increased susceptibility to avirulent bacteria [13•]. These treatments did not affect the interaction with an avirulent

isolate of the oomycete *Hyaloperonospora parasitica*, but inoculation of the ABA-deficient mutant *aba1-1* with virulent isolates of *H. parasitica* resulted in partial resistance. In contrast to the WT, *aba1-1* mutants developed necrotic spots at the site of inoculation. Sexual reproduction was suppressed and asexual reproduction was markedly reduced in *aba1-1* mutants when infected with virulent *H. parasitica*. The ABA-insensitive mutant *abi1-1* remained susceptible to virulent isolates of *H. parasitica*. Thus, the concentration of ABA rather than the presence of a functional ABA-signaling pathway is important for the development of disease susceptibility in *Arabidopsis*. This suggests that ABA interferes indirectly with disease resistance by interacting with biotic stress signaling.

How does ABA influence disease resistance?

Little is known about the primary causes of ABA-induced disease susceptibility. ABA does not directly stimulate or inhibit fungal growth [4,12]. The possibility that ABA could influence disease resistance through its control of stomatal aperture and water relations is not discussed in this review.

ABA treatment has been shown to suppress phytoalexin synthesis and to inhibit the activity and transcript accumulation of phenylalanine ammonium lyase [4,8,11]. The ABA-deficient *sitiens* mutant of tomato has increased resistance to infection by *Botrytis cinerea* [12]. Application of ABA restored the susceptibility of *sitiens* and increased the susceptibility of WT plants to *B. cinerea*. In contrast to *Arabidopsis*, the resistance of tomato against *B. cinerea* depended on SA and not on JA/ET signaling. Increased activity of phenylalanine ammonium lyase was measured in *sitiens* plants upon inoculation. Treatment with the SA functional analog benzothiadiazole induced higher levels of PR1 protein and restored resistance to *B. cinerea* in WT plants. The *sitiens* mutant had greater SA-mediated responses and was more resistant to *P. syringae* pv. *tomato* than WT plants [14]. These results suggest an antagonistic effect of ABA on SA-mediated defense signaling. Thus, high ABA concentrations interfere with resistance against pathogens controlled by the SA signaling pathway.

There is overwhelming evidence that ABA interacts with ET- and sugar-mediated signaling [25]. High ABA concentrations inhibit ET production [26], and the ABA and ET signaling pathways interact mostly antagonistically in plant development [27,28] and in vegetative tissues [29••]. Mutant screens for ET insensitivity or for enhanced response to ABA led to the identification of the same gene (*ETHYLENE INSENSITIVE2* [*EIN2*]/*ENHANCED RESPONSE TO ABA3* [*ERA3*]) thus identifying the encoded protein as a point of convergence in ABA and ET signaling [28]. The *ein2* mutant overproduces ABA and it is therefore not clear whether ethylene insensitivity and/or ABA overproduction causes its susceptibility to necrotrophic pathogens.

Synergistic and antagonistic effects were reported for the interaction of the ABA and JA signaling pathways [30,31]. The complex interplay was recently analyzed in *Arabidopsis* by Anderson *et al.* [29••]. High ABA concentrations strongly reduced the transcript levels of JA- or ET-responsive defense genes, whereas ABA-deficient mutants showed a corresponding increase. Interestingly, the inhibitory effect of ABA could not be overcome by the application of methyl-JA or ET. This suggests that abiotic stress signaling has the potential to override biotic stress signaling in situations of simultaneous stress.

Disruption of the transcription factor AtMYC2, which is a positive regulator of ABA signaling [32••], resulted in elevated levels of basal and induced expression from JA- and ET-responsive defense genes [29••]. Analysis of the jasmonate-insensitive *jin1* mutant revealed that *JIN1* is allelic to *AtMYC2* [33••]. AtMYC2 activates genes that are involved in JA-mediated systemic responses to wounding but represses JA-mediated genes that are involved in defense against pathogens. AtMYC2 is a late point of convergence of ABA and JA signaling: it activates ABA-regulated gene expression and inhibits a subset of JA-regulated defense genes. Consequently, *jin1*, knockout mutants of *AtMYC2* and the ABA-biosynthetic mutant *aba2-1* were more resistant to various fungal pathogens [29••,33••]. Resistance to these pathogens was previously shown to be JA- and ET-dependent [34].

ABA and biotic-stress signaling do not always have opposing effects. The *Arabidopsis* MYB-related protein BOTRYTIS SUSCEPTIBLE1 (BOS1) shows high sequence similarity to AtMYB2, which functions as a transcriptional activator in ABA signaling [35•]. The expression of BOS1 was induced by *B. cinerea* via the jasmonate pathway, and the promoter of BOS1 contained ABA-responsive elements. BOS1 appears to control the expression of a subset of jasmonate- and ABA-inducible target genes whose expression is important for the establishment of abiotic and biotic stress tolerance. Loss of BOS1 function caused enhanced susceptibility to necrotrophic pathogens and impaired tolerance towards water deficit, salinity, and oxidative stress.

The rapidly accumulating data from large-scale expression profiling strongly supports the existence of regulatory networks. Biotic and abiotic stress, as well as ABA, SA, JA and ET, control the expression of different but overlapping sets of genes. A detailed comparison of the downstream targets of ABA and biotic stress signaling is beyond the scope of this review.

ABA and biotic stress signaling share additional elements

The signaling responses of plants to ABA and biotic stress share many similarities that might act as additional nodes of competitive or synergistic interaction. The rapid

generation of reactive oxygen species (ROS) is a central component of disease resistance responses and of ABA signaling [36,37]. The same NADPH-dependent respiratory burst oxidase homologs seem to be implicated in ROS generation in both systems [38,39]. Similarly, nitric oxide has emerged as an important mediator of plant defense responses and as a component of ABA-signaling in the control of stomatal aperture and in adaptive plant responses to drought stress [40,41].

Ca^{2+} signaling is important in the expression of disease resistance and in ABA-controlled stomatal movements and responses to dehydration [42,43]. Klüsner *et al.* [44] presented evidence that fungal elicitors activate a branch of the signaling network that is shared with ABA signaling in the regulation of plasma-membrane-localized Ca^{2+} channels. The expression of various calcium-dependent protein kinases (CDPKs) of tobacco was upregulated by ABA, JA, pathogens, fungal elicitors and abiotic stress (reviewed in [45]). Similarly, *CaCDPK3* was induced by abiotic stress factors, ABA, SA, ET, JA and an avirulent bacterial pathogen [46].

The expression and activity of the rice mitogen-activated protein kinase OsMAPK5 was activated by ABA, various abiotic stresses and pathogen attack [47••]. OsMAPK5 inversely modulates broad-spectrum disease resistance and abiotic stress tolerance. Suppression of *OsMAPK5* expression resulted in constitutive expression of PATHOGENESIS-RELATED (PR)-proteins and in increased disease resistance. However, the plants in which *OsMAPK5* expression was suppressed showed reductions in drought, salt and cold tolerance. By contrast, the overexpression of OsMAPK5 kinase activity had the opposite effect.

Overexpression of *ACTIVATED DISEASE RESISTANCE1* (*ADR1*), which encodes a coiled-coil (CC)–nucleotide binding site (NBS)–leucine-rich repeat (LRR) gene, caused enhanced resistance to virulent pathogens and to drought stress but decreased tolerance towards thermal and salinity stress. The drought tolerance established by ADR1 was dependent on ABI1 function and components of SA signaling [48•].

ABA-dependent priming of biotic and abiotic stress tolerance

Plants that have been treated with the non-protein amino acid β-aminobutyric acid (BABA) develop an enhanced capacity to resist biotic and abiotic stresses. This BABA-induced resistance (BABA-IR) is associated with an increased capacity to express defense responses in stress situations, a phenomenon called priming [49,50]. Interestingly, the treatment of plants with BABA has the potential to prime the expression of both SA- and ABA-regulated genes, thus suggesting that BABA affects a shared signaling component. Mutants that are impaired in the production of or in sensitivity to ABA were found to be blocked in BABA-induced priming of biotic and abiotic stress tolerance ([51•]; G Jakab B Mauch-Mani, unpublished). The *Arabidopsis* mutant *impaired BABA-induced sterility3* (*ibs3*) is defective in the ABA-biosynthetic zeaxanthin epoxidase gene and showed reduced levels of BABA-IR against *H. parasitica* and decreased callose deposition [52••]. *Arabidopsis* mutants that had impaired ABA sensitivity and the callose-deficient mutant *powdery mildew resistant4-1* (*pmr4-1*) did not express BABA-IR against necrotrophic fungi [51•]. The link between ABA and callose is further strengthened by the fact that the application of ABA mimicked the effect of BABA treatment on both callose deposition and resistance against necrotrophic fungi [51•,53]. However, the observed link might also be an indirect one due to the interference of callose synthase with other proteins implicated in cell wall integrity.

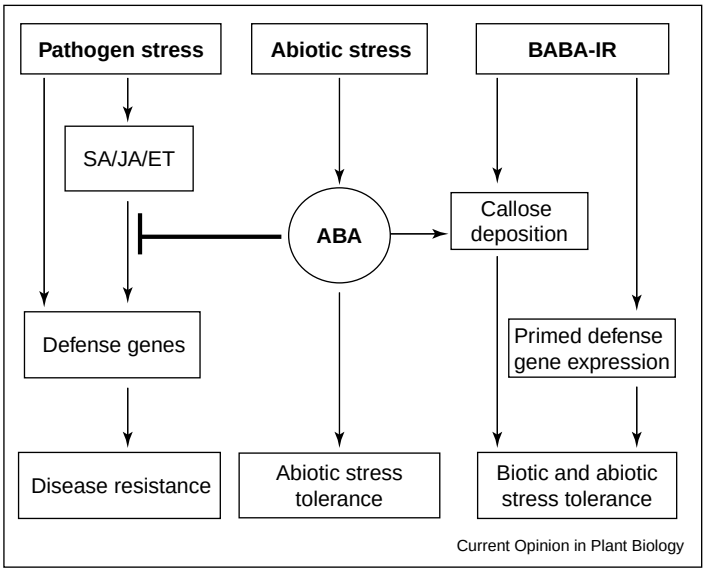
Mutants such as *ibs3*, *aba1-5* and *abi4-1* are not impaired in basal but in primed callose deposition upon pathogen attack. The molecular mechanism of ABA-mediated priming is not known. Callose production is a secretory process and the fusion of the involved secretory vesicles with the plasma membrane is mediated by SNARE (soluble *N*-ethyl-maleimide-sensitive fusion protein attachment protein receptor) proteins. During cell-plate formation, callose synthase is transported in vesicles to the location where its function, the synthesis of callose, is required [54]. Interestingly, SNAREs have repeatedly been implicated in ABA-dependent responses to abiotic stress and to pathogen resistance [55,56]. An additional mutant that is impaired in BABA-IR has a defect in a SNARE gene, suggesting that ABA is involved in callose deposition through the regulation of specific SNAREs (V Flors, B Mauch-Mani, unpublished).

Conclusions

Current evidence suggests that ABA affects disease resistance mainly negatively by interfering at different levels with biotic stress signaling. The involvement of ABA in primed callose production is one of the few examples of a positive role of ABA in disease resistance. It has become increasingly clear that the previously isolated abiotic signaling network that is controlled by ABA and the biotic network that is controlled by SA, JA and ET are interconnected at various levels (Figure 1). Whether all of the potential connections and shared nodes are actually used for cross-talk remains to be determined.

The analysis of this combined network is a difficult task. The concept of marker genes whose expression is believed to be regulated by individual hormones does not do justice to the nature of the network. The apparent cross-talk in stress-hormone signaling makes it difficult to assign a marker gene or a mutant phenotype to a specific hormone-controlled pathway. The signaling network into

Figure 1



Simplified model depicting the role of ABA in disease resistance. For better clarity, positive modulators of biotic and abiotic stress signaling such as BOS1 [35•] are not included. Similarly, the influence of components of biotic stress signaling (e.g. EIN2 [27,28]) on ABA-mediated signaling is not shown.

which the four stress hormones and other signals feed is apparently designed to allow plants to adapt optimally to specific situations by integrating possibly conflicting information from environmental conditions, biotic stress, and developmental and nutritional status. The responses of this complex network to naturally occurring changes are not expected to be digital at any level but rather graded, thus allowing a fine-tuning of adaptive gene expression. The nature of the network cannot be completely understood by overstimulating signal input or by mutational knockout of individual components. These situations tend to produce an extreme out-of-balance state that might occur only rarely in nature. To further progress our understanding of the complex interactions between ABA-induced signaling and biotic-stress signaling it will be necessary to produce more quantitative data at all levels under natural conditions.

Acknowledgements

We gratefully acknowledge funding from the Swiss National Science Foundation (grants 3100-105884/1 and 3100-067038).

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- •• of outstanding interest

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The *OsMAPK5* gene is functionally characterized as a positive regulator of abiotic stress tolerance and as a negative regulator of disease resistance. Expression of *OsMAPK5* was induced by ABA and by various biotic and abiotic stresses. RNA-interference (RNAi)-mediated suppression of *OsMAPK5* caused the constitutive expression of defense-related genes and led to enhanced resistance against *M. grisea* and the bacterial pathogen *Burkholderia glumae*. By contrast, it caused decreased drought, salt and low-temperature tolerance. Plants that overexpressed *OsMAPK5* had the opposite phenotype.

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