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Jasmonates - Signals in Plant-Microbe Interactions

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Abstract

Within their environment, plants interact with a wide range of microorganisms, some of which are pathogenic and cause disease, and others that are beneficial and stimulate plant growth or activate natural defenses. To recognize and respond to this variety of pathogenic and beneficial microorganisms, plants have developed sophisticated strategies to ''perceive'' microorganisms and translate that ''perception'' into an appropriate adaptive response. This plant innate immune response is surprisingly complex and highly flexible in its capacity to recognize and respond to different invaders. Jasmonic acid and derivatives, collectively called jasmonates (JAs), have emerged as important signals in the regulation of plant responses to pathogenic and beneficial microorganisms. The complex interplay of JAs with the alarm signals salicylic acid (SA) and ethylene (ET) provides plants with a regulatory potential that shapes the ultimate outcome of the plant-microbe interaction. In this review, we present an overview of the key role of JAs in basal and induced resistance to pathogens, their possible implication in the establishment and functioning of beneficial plant-microbe associations; and our current knowledge on how the JA signaling pathway cross-communicates with SA- and ET-dependent signaling pathways to fine-tune defense.

Key words: Jasmonates; Signaling; Defense; Basal resistance; Induced resistance; Pathogen; Cross-talk; Ethylene; Salicylic acid; Symbiosis

Introduction

During their lifetime, plants encounter a large and diverse community of microorganisms that compete and interact with each other and the plant. Within this microbial community, a whole range of beneficial and deleterious organisms can be found, leading to the establishment of mutualistic and pathogenic interactions, respectively. The complexity of plant-microbe interactions involves highly coordinated cellular processes that determine the final outcome of the relationship. It is well known that considerable communication between plants and microbes occurs during the early stages of their association, in which signal molecules play an essential role.

Because of their agronomic importance, plant-pathogen interactions have been a major focus in plant biology research (Dangl and Jones 2001; Feys and Parker 2000; Holt and others 2003; Michelmore 2003; Pieterse and Van Loon 1999; Slusarenko and others 2000). Resistance against pathogens relies on the recognition of the pathogen by the plant and the subsequent activation of effective defense mechanisms. Resistance against specific races of a pathogen depends on the recognition of avirulence (AVR)

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gene products from the pathogen by resistance (R) gene products in the plant. In the absence of such a gene-for-gene recognition system, defenses seem to be elicited nonspecifically, similar to those observed in the innate immune response in animals (recently reviewed in Nürnberger and others 2004). This type of defense is known as basal resistance, and restricts the development of the disease after pathogen attack. Common features exist between the signaling processes involved in gene-for-gene-mediated resistance (incompatible interactions) and the restriction of virulent pathogens during compatible interactions by basal resistance (Feys and Parker 2000). For example, there is a significant overlap in transcriptional changes during both compatible and incompatible interactions. The effectiveness of the resistance response, therefore, seems to depend more on the timing and amplitude of the transcriptional activation than on qualitative differences in global expression patterns (Nimchuk and others 2003; Tao and others 2003).

Among the defense-related genes that are activated upon pathogen attack are genes encoding proteins with antimicrobial properties, signaling functions, proteins involved in the reinforcement of the cell wall, and in the ''oxidative burst'' (reviewed by Somssich and Hahlbrock 1998). All these defense responses are primarily activated locally at the site of infection, but under certain circumstances a state of enhanced defensive capacity can also be achieved throughout the plant. This systemic resistance confers long lasting protection against a broad range of pathogens (Van Loon 2000). Together, the battery of plant defense responses that are activated upon pathogen attack are in general sufficiently effective, and despite the high number of potential deleterious organisms, disease is not the common outcome of a plant-microbe interaction.

The plant hormones jasmonic acid (JA), salicylic acid (SA) and ethylene (ET) are major regulators of plant innate immunity. Plants respond with the production of a specific blend of these alarm signals after pathogen attack. The production of these signals varies greatly in quantity, composition and timing, and results in the activation of differential sets of defense-related genes that eventually determine the nature of the defense response against the attacker encountered (Reymond and Farmer 1998; Rojo and others 2003; Van Oosten and others 2004). Other plant hormones, such as abscisic acid (ABA), brassinosteroids and auxins have also been reported to play a role in plant defense against pathogens (Audenaert and others 2002; Jameson 2000; Krishna 2003; Nakashita and others 2003; Thaler and Bostock 2004; Ton and Mauch-Mani 2004).

JA and its derivatives, collectively called jasmonates (JAs), are ubiquitous plant regulators. Their role in different aspects of plant biology has received considerable attention in recent years and is reviewed in this issue. JAs can act as signals in plant cellular responses to different abiotic and biotic stresses, in plant-herbivore interactions (Baldwin and others this issue) and in plant-plant interactions (Baldwin and others 2002; Karban and others 2000). Although the role of JAs in plant defense against insects and during wounding has been well documented, the importance of JAs in defense against pathogenic microorganisms has only been envisaged in the last decade. The involvement of JAs in defense responses against pathogens was evidenced by the fact that JAs often accumulate in response to pathogen attack, the altered susceptibility/resistance of mutant plants affected in JA biosynthesis or signaling, and the effects of exogenous application of JAs on plant resistance. Moreover, JA-dependent responses are associated with enhanced expression of several defense genes that encode antimicrobial proteins, such as plant defensins and thionins (Pieterse and Van Loon 1999). This review gives an overview of the current knowledge on the role of JAs in signaling during plant-microbe interactions, with special emphasis on their role in induced resistance against pathogens.

Role of JA in Disease Resistance

Genetic Evidence

Depending on the host-pathogen interaction, JA, SA, and ET appear to be differentially involved in basal resistance. It has been proposed that the defense signaling pathways that are induced upon pathogen attack are influenced by the pathogen's lifestyle. Pathogens can generally be divided into those that require living plant cells (biotrophs) and those that kill host cells and feed on the dead tissue (necrotrophs) (Parbery 1996). SA-dependent defense responses are usually associated with a form of programmed cell death known as the hypersensitive response. This response can restrict the growth of biotrophic pathogens by killing the infected cells. In fact, this type of defense is effective against a wide range of biotrophs, but usually fails to protect against, or can even be beneficial for necrotrophic pathogens (Govrin and Levine 2000; Thomma and others 2001). JA-dependent defense responses, which are not associated with cell death, are considered to provide an alternative defense against necrotrophs (McDowell and Dangl 2000).

Compelling evidence for the role of JAs in basal resistance came from genetic analyses of plant mutants and transgenics that are affected in the biosynthesis or perception of JAs. The available *Arabidopsis* mutants defective in JA-related processes have been compiled by Berger (2002). For example, both the *jar1* mutant, with reduced sensitivity to methyl jasmonate (MeJA), and the *fad3fad7fad8* triple mutant, which is defective in JA biosynthesis, exhibit susceptibility to normally nonpathogenic soil-borne oomycetes of the genus *Pythium* (Staswick and others 1998; Vijayan and others 1998). Recently, increased susceptibility of *jar1* to *Fusarium oxysporum* (Berrocal-Lobo and Molina 2004) and impairment of induced resistance against *Cucumber mosaic virus* in *fad3fad7fad8* mutants have been reported (Ryu and others 2004). The JA-insensitive mutant *coi1* shows enhanced susceptibility to the bacterial leaf pathogen *Erwinia carotovora* (Norman-Setterblad and others 2000) and the necrotrophic fungi *Alternaria brassicicola* and *Botrytis cinerea* (Thomma and others 1998). Accordingly, overexpression of a JA carboxyl methyl transferase increased endogenous levels of MeJA and resulted in higher resistance to *B. cinerea* (Seo and others 2001). Furthermore, constitutive activation of the JA signaling pathway in *Arabidopsis* resulted in enhanced resistance to the biotrophs *Erysiphe cichoracearum*, *Erysiphe orontii*, and *Oidium lycopersicum* (Ellis and others 2002). All these examples clearly point to a role of JAs in resistance against pathogens with diverse lifestyles, challenging the general notion that JA-dependent defense responses are predominantly effective against necrotrophic pathogens.

In some cases, JA has been implicated in enhanced susceptibility to pathogen infection. For instance, *coi1* and the MAP kinase 4 mutant *mpk4*, which is impaired in JA-responsive gene expression (Petersen and others 2000), show reduced susceptibility to the bacterial pathogen *Pseudomonas syringae* (Feys and others 1994; Kloek and others 2001; Petersen and others 2000), suggesting that in wild-type plants, JA-dependent responses promote susceptibility to this pathogen. Similarly, He and others (2004) provided evidence that in this plant-pathogen interaction the JA-signaling pathway plays an important role during early stages of pathogenesis. In this study, different type III effectors of *P. syringae* and its phytotoxin coronatine were shown to augment the JA-signaling pathway to promote parasitism. The above-mentioned studies with mutant *coi1* and *mpk4* clearly show that JA signaling promotes susceptibility to *P. syringae* in *Arabidopsis*. However, other studies with *jar1* and mutant *cev1*, which constitutively activates JA responses, show that JA signaling promotes basal resistance against this pathogen (Ellis and others 2002; Pieterse and others 1998; Ton and others 2002a). Apparently, the role of JA signaling in promoting either resistance or susceptibility seems to depend on a delicate balance of so far unknown factors.

In tomato, Thaler and others (2004) checked the effectiveness of JA-dependent responses on a wide range of pathogens with different lifestyles. The JA-insensitive mutant *jai1* showed higher mortality due to stem wilting caused by *Fusarium* spp. in field experiments, indicating that JA-dependent defense against these pathogens was compromised in the *jai1* mutant. Considering biotrophy and necrotrophy as a continuum, they selected pathogenic fungi, bacteria and oomycetes ranging from true biotrophs, such as *Oidium* spp., to true necrotrophs, such as *Septoria* spp., including different hemibiotrophs with predominantly biotrophic or necrotrophic lifestyles. The JA-deficient *def1* mutant of tomato was not affected in its resistance to the clear biotrophs, but exhibited increased susceptibility to all intermediate and/or difficult-to-classify species. This work nicely illustrates that JA-mediated basal resistance in tomato is effective against a wide range of pathogens, overlapping partially with the range of effectiveness of the SA-dependent pathway.

Pharmacological Evidence

Besides the genetic studies that clearly demonstrated the important role of JAs in plant defense, another line of evidence came from experiments in which the effect of exogenous application of JAs on the level of resistance was investigated. The most commonly used treatment is the application of naturally occurring methyl jasmonate (MeJA). MeJA is a key compound in the JA signaling pathway and regulates the JA biosynthetic pathway by a positive feedback mechanism (Cheong and Choi 2003; Sasaki and others 2001). Early experiments showed that addition of MeJA to cell suspension cultures of different plant species induced defense-related gene expression and elicited the accumulation of secondary metabolites (Gundlach and others 1992). In *Arabidopsis*, Thomma and others (1998) demonstrated that pretreatment of plants with MeJA provides significant protection against *A. brassicicola* through induction of resistance *in planta*, and not by direct effects on the pathogen. Furthermore, Vijayan and others (1998) demonstrated that exogenous application of MeJA compensated the extreme susceptibility of the JA-deficient *Arabidopsis* mutant *fad3fad7fad8* to *Pythium mastophorum*, thereby reducing the incidence of the disease to similar levels as in wild type plants. Disease caused by necrotrophic fungi such as *B. cinerea* or *Plectosphaerella cucumerina* was also reduced in *Arabidopsis* after treatment with MeJA (Thomma and others 2000). Using different *Arabidopsis* signaling mutants, the authors showed that the SA- and ET-dependent pathways were not required for the induction of resistance by MeJA.

Induction of resistance against other necrotrophic or hemi-biotrophic pathogens by MeJA treatment has also been shown in other plant species. For example, pre-treatment with MeJA resulted in enhanced levels of resistance in potato and tomato against *Phytophthora infestans* (Cohen and others 1993), in cut roses against *B. cinerea* (Meir and others 1998), in *Picea abies* against *Pythium ultimum* (Kozlowski and others 1999) and in grapefruit against *Penicillium digitatum* (Droby and others 1999). MeJA treatment was also effective in inducing resistance in melon against gummy stem blight (*Didymella bryoniae*) and white mould (*Sclerotinia sclerotiorum*), while SA treatment was ineffective (Buzi and others 2004). In addition, MeJA treatment has been shown to be effective against *P. syringae* in *Arabidopsis* and tomato (Pieterse and others 1998; Thaler and others 2002; Van Wees and others 1999).

Although MeJA treatment seems to be generally effective against necrotrophic pathogens, the effects on biotrophic pathogens are less clear. MeJA application failed to induce resistance to *Peronospora parasitica* in *Arabidopsis* (Thomma and others 1998) or to *Blumeria graminis* in barley (Schweizer and others 1993). However, other reports showed a significant systemic protection of barley to powdery mildew (*E. cichoracearum* and *B. graminis*) after MeJA treatment (Ellis and others 2002; Walters and others 2002). Overall, independent studies highlight the key role of JAs in basal and induced resistance to necrotrophic pathogens in different plant species. In addition, evidence accumulates that JA-dependent defense responses can also contribute to resistance against pathogens with a (hemi)biotrophic lifestyle, possibly by acting in concert with other defense signaling pathways.

Role of JA in Beneficial Plant-Microbe Interactions

Symbiotic Associations

Besides pathogenic interactions, mutually beneficial relationships are frequent in nature, improving plant nutrition and/or helping the plant to overcome abiotic and biotic stresses. These associations can involve fungi, such as the ubiquitous mycorrhizal symbiosis, or bacteria, such as the nitrogen-fixating associations between legumes and *Rhizobium* spp. The establishment of mutualistic associations involves mutual recognition and a high degree of coordination at the morphological and physiological level that should be based on a continuous cellular and molecular dialogue between both symbionts (Gianinazzi-Pearson 1996; Kistner and Parniske 2002; Parniske 2000). There is evidence indicating that plant defense-related mechanisms are involved in the establishment and control of these intimate symbioses, and that plant symbionts could have evolved mechanisms to use host defense-recognition systems for symbiotic signal perception (Liu and others 2003; Pozo and others 1998; Pozo and others 2002a). A recent study showed that colonization of barley roots by an arbuscular mycorrhizal fungus induced elevated levels of endogenous JA, and expression of JA-responsive genes and genes involved in JA biosynthesis in arbuscule-containing cells (Hause and others 2002). Moreover, several studies showed that treatment with JA stimulated mycorrhizal development in endo- and ectomycorrhiza associations (Regvar and others 1996; Regvar and others 1997) and induced expression of the symbiotic nod genes in *Rhizobium* (Rosas and others 1998).

Rhizobacteria-induced Systemic Resistance

Another important group of beneficial microorganisms is formed by nonpathogenic, plant growth-promoting rhizobacteria (PGPR). Fluorescent *Pseudomonas* spp. are among the most effective PGPR and have been shown to be responsible for the reduction of soil-borne diseases in natural disease-suppressive soils (Raaijmakers and Waller 1998). Part of their effect on growth promotion is caused by their ability to antagonize deleterious microorganisms in the soil (Schippers and others 1987). In addition to direct antimicrobial effects, selected strains of rhizobacteria are able to induce a plant-mediated systemic resistance that is effective against a broad spectrum of pathogens. This phenomenon is called rhizobacteria-mediated induced systemic resistance (ISR) and has been demonstrated in many different plant species (Van Loon and others 1998). Specific recognition between the plant and the ISR-inducing rhizobacterium is required for the induction of ISR, and the ability to express this type of resistance is determined genetically in the plant (reviewed in Pieterse and others 2003; Pieterse and others 2002). Genetic studies using *Arabidopsis* mutants demonstrated that *Pseudomonas fluorescens*

WCS417r-mediated ISR requires responsiveness to both JA and ET, but functions independently of SA (Pieterse and others 1996; Pieterse and others 1998). Apart from *P. fluorescens* WCS417r, other fluorescent *Pseudomonas* spp. strains have been shown to induce the SA-independent ISR pathway in *Arabidopsis* (Iavicoli and others 2003; Ryu and others 2003; Van Wees and others 1997), tobacco (Press and others 1997; Zhang and others 2002) and tomato (Yan and others 2002), indicating that the ability to trigger a SA-independent pathway controlling systemic resistance is not uncommon among resistance-inducing rhizobacteria.

A detailed analysis of the effectiveness of *P. fluorescens* WCS417r-mediated ISR in *Arabidopsis* demonstrated that it is predominantly effective against pathogens that in non-induced plants are resisted through JA/ET-dependent basal resistance, including *A. brassicicola*, *X. campestris*, and *P. syringae* (Ton and others 2002a). Therefore, ISR seems to be based on an enhancement of extant JA- and/or ET-dependent defense responses. Interestingly, the level of protection achieved by MeJA treatment in *Arabidopsis* against *A. brassicicola* and *P. syringae* was similar to that observed in ISR-expressing plants (Ton and others 2002a; Van Wees and others 1999). When several *Arabidopsis* mutants with reduced basal resistance (*eds* mutants, for enhanced disease susceptibility) were screened for their responsiveness to ISR-inducing rhizobacteria, three mutants unable to mount ISR were identified (*eds4-1*, *eds8-1*, and *eds10-1*). Further analysis of these mutants showed that the inability of *eds8-l* to mount ISR was associated with reduced sensitivity to MeJA (Ton and others 2002b). Together, these lines of evidence confirm that JA-dependent defense responses are essential for ISR. However, analysis of local and systemic levels of JA and ET in plants expressing ISR revealed that this type of induced resistance is not associated with detectable changes in their production (Pieterse and others 2000). Thus, rhizobacteria-mediated ISR is associated with an enhanced sensitivity of the induced tissues to these hormones, rather than an increase in their production. This phenomenon is known as ''sensitization'', ''conditioning'', or ''priming''.

Priming of JA-Dependent Defense Responses

Priming is the enhanced capacity of induced tissues for rapid -and effective activation of cellular defense responses after infection with a challenging pathogen (Conrath and others 2002). Priming has been implicated in several types of induced resistance. In most cases JA, SA, or ET has been suggested to act as potentiation signals of defense-related gene expression. The ability of JAs to prime plant tissues for a faster and more efficient response has been observed in several studies. Pretreatment of parsley cell cultures with MeJA potentiates elicitor-induced accumulation of active oxygen species and elicitation of phenylpropanoid defense responses in these cells (Kauss and others 1994; Kauss and others 1992). In rice, JA potentiates the expression of the *PR-1* gene that is activated in response to fungal elicitors, and the level of resistance induced by low doses of the SA analog INA against the fungus *Magnaporthe grisea* (Schweizer and others 1997). Tobacco cells also showed faster and stronger lipid peroxidation and protein phosphorylation in response to fungal elicitors after preconditioning by MeJA treatment (Dubery and others 2000).

Priming seems to be an important mechanism involved in rhizobacteria-mediated ISR in *Arabidosis*. Van Wees and others (1999) and Hase and others (2003) showed that ISR-expressing *Arabidopsis* plants are primed to express the JA- and/or ET-responsive genes *VSP2*, *PDF1.2* and *HEL* to a higher level after subsequent elicitation. Recently, microarray analyses showed that *P. fluorescens* WCS417r-induced ISR in *Arabidopsis* is not associated with detectable changes in gene expression in systemic tissues (Verhagen and others 2004). However, after challenge inoculation of WCS417r-induced plants with the bacterial leaf pathogen *P. syringae* pv. *tomato* DC3000, a large set of genes showed an augmented expression pattern in ISR-expressing leaves, suggesting that these genes were primed to respond faster and/or more strongly upon pathogen attack. The majority of the primed genes were predicted to be regulated by JA and/or ET signaling. To assess the importance of JA in priming during ISR, the transcript profile of MeJA- inducible genes was recently analyzed in ISR-expressing plants using whole-genome ATH1 Affymetrix GeneChips. More than one-third of all the MeJA-responsive *Arabidopsis* genes showed a quicker and/or stronger response in ISR expressing plants after MeJA treatment in comparison to MeJA-treated control plants (M.J. Pozo and C.M.J. Pieterse, unpublished results). These results support a central role for JA in the priming phenomenon associated with rhizobacteria-mediated ISR. Priming of pathogen-induced genes allows the plant to react more effectively to the invader encountered, which might explain the broad-spectrum effectiveness of ISR.

Other beneficial microorganisms may also boost plant defenses in a JA-dependent manner. For example, Wasternack and Hause (2002) suggested a causal relationship between the enhanced defense status demonstrated in mycorrhizal plants (Cordier and others 1998; Pozo and others 1999; Pozo and others 2002b), and the elevated levels of JA observed upon mycorrhization. Furthermore, a strong accumulation of JA has also been associated with induction of resistance by the biological control fungus *Trichoderma longibrachiatum* (Martinez and others 2001).

Cross-Talk Between JA and Other Defense Signaling Pathways

The plasticity of the plant response to deleterious and beneficial microorganisms seems to rely on complex interplay between the different signaling pathways. Cross-talk between defense signaling pathways might help the plant to either prioritize the action of a particular pathway over another, or activate multiple pathways to achieve the most efficient response to the microorganism encountered. Global gene expression profiling studies strongly support the existence of substantial cross-talk between the SA, JA and ET signaling pathways (Glazebrook and others 2003; Schenk and others 2000). Despite the extraordinary complexity of the defense-signaling network, considerable advances have been made in this field of research. The main obstacles in cross-talk research are the possible pleiotropic effects of signaling mutants (Heck and others 2003; Van Wees and Glazebrook 2003), and inconsistencies in the correlation between gene expression patterns and disease resistance (Clarke and others 2000). Recently, several reviews have discussed the data available on the interactions between JA, SA and ET signaling pathways, illustrating the existence of cooperative, synergistic and antagonistic effects on disease resistance (Feys and Parker 2000; Kunkel and Brooks 2002; Pieterse and others 2001; Pieterse and Van Loon 2004; Reymond and Farmer 1998; Rojo and others 2003). Here, we will focus on cross-communication between pathways that can affect the role of JA during plant-microbe interactions.

JA and ET Signaling

ET often acts in concert with JA in activating the expression of defense-related genes (O'Donnell and others 1996; Penninckx and others 1998; Rojo and others 2003; Xu and others 1994). There are, however, some reports of negative interactions between the ET and JA pathways. For instance, ET and JA have antagonistic effects in the biosynthesis of the anti-herbivore compound nicotine in tobacco (Shoji and others 2000), in ozon-induced oxidative cell death in *Arabidopsis* (Overmyer and others 2000; Tuominen and others 2004) and in wounding responses (Lorenzo and others 2004; Rojo and others 1999). A classic example of synergism between JA and ET is the pathogen-induced expression of the plant defensin gene *PDF1.2* in *Arabidopsis*, which requires a concomitant activation of the JA and ET signaling pathway for full expression (Penninckx and others 1998). Recently, Lorenzo and others (2003) demonstrated that upstream of *PDF1.2* activation, the JA and ET pathways converge in the transcriptional activation of *ERF1*, encoding ethylene-response factor 1. Transcript profiling of *ERF1*-overexpressing *Arabidopsis* plants revealed that ERF1 regulates a large number of genes that are responsive to both JA and ET, suggesting that ERF1 plays a key role in the integration of both signals (Lorenzo and others 2003). The concerted action of JA and ET in defense-related gene expression suggests that both alarm signals act concomitantly in the activation of defense responses. Indeed, pharmacological and genetic studies showed overlapping roles of JA and ET in both induced and basal disease resistance (Berrocal-Lobo and others 2002; Ellis and Turner 2001; Pieterse and others 1998; Thomma and others 2001; Van Wees and others 1999).

JA and SA Signaling

In general, interactions between SA and JA signaling are antagonistic. For instance, SA and its functional analogues INA and BTH have been shown to act as strong suppressors of JA-dependent defense responses (Bowling and others 1997; Doherty and others 1988; Fidantsef and others 1999; Peña-Cortés and others 1993; Van Wees and others 1999), possibly through the inhibition of JA biosynthesis and action (Doares and others 1995; Harms and others 1998; Peña-Cortés and others 1993). As a result, plants are able to prioritize SA-dependent resistance, which is effective against certain types of pathogens, over JA-dependent defense, which is effective against other groups of pathogens. In agreement with this, Preston and others (1999) demonstrated that TMV-infected tobacco plants expressing SA-dependent systemic acquired resistance (SAR) are unable to develop JA-mediated defense responses, probably because of inhibition of JA signaling by increases in SA levels resulting from TMV infection. Recently, Spoel and others (2003) demonstrated that the antagonistic effect of SA on JA-triggered gene expression is mediated by the regulatory protein NPR1 (Dong 2001; Pieterse and Van Loon 2004). Nuclear localization of NPR1, which is essential for SA-mediated PR gene expression (Kinkema and others 2000), appeared not to be required for the suppression of JA signaling. Thus, cross-talk between SA and JA is modulated through a novel function of NPR1 in the cytosol. The mode of action of NPR1 in the cytosol is unknown, but it is tempting to speculate that it interferes with the previously identified SCF^{COI1} ubiquitin-ligase complex (Devoto and others 2002; Xu and others 2002) that regulates JA-responsive gene expression through targeted ubiquitination and subsequent proteasome-mediated degradation of a negative regulator of JA signaling.

Additional key elements in cross-talk between JA and SA signaling have recently been identified. For instance, the *Arabidopsis* transcription factor WRKY70 was shown to act as both an activator of SA-responsive genes and a represser of JA-inducible genes, thereby integrating signals from these antagonistic pathways (Li and others 2004).

Despite the clear antagonism between SA- and JA-dependent pathways, transcript profiling analysis revealed a high number of genes co-induced or co-repressed by the two hormones, pointing to a certain degree of overlap between the two pathways (Glazebrook and others 2003; Schenk and others 2000). In addition, absence of antagonism in the protection against necrotrophs (Thomma and others 2000) and additive effects of SA- and JA- dependent induced resistance against *P. syringae* DC3000 have been shown in *Arabidopsis* (Van Wees and others 2000).

The emerging picture is that multiple genes are involved in balancing the activation of either SA- or JA-mediated resistance. Recent reports show that the biochemical and biological consequences of cross-talk between the different pathways depend on the timing and concentration of hormones (Devadas and others 2002; Thaler and others 2002). These results illustrate the complexity of the interactions between pathways, and support the flexibility of the plant defense response to fine-tune the appropriate mechanisms by tightly regulating the concentrations of the different signals.

Ecological Implications of Pathway Cross-talk

During co-evolution of plants and microbes, pathogens may have evolved mechanisms to suppress defense responses by interfering with key pathway regulators, thereby forcing plants to evolve bypass mechanisms (McDowell and Dangl 2000). This would explain the degree of overlap between the ranges of effectiveness among the different defense signaling pathways. In some cases, pathogens are able to exploit the mechanisms of cross-talk between pathways and benefit from their trade-offs. For example, certain *Pseudomonas* strains produce the phytotoxin coronatine that acts as a structural and functional analog of JA. In tomato, coronatine increases the severity of the disease by targeting the host JA signaling pathway, thereby activating JA response genes and possibly attenuating the SA-dependent defenses that are effective against this pathogen (Zhao and others 2003). Coronatine also augments the JA-dependent pathway to promote parasitism in *Arabidopsis* (He and others 2004). Similarly, harpin, a proteinaceous elicitor from *P. syringae*, may activate JA signaling via MPK4, thereby suppressing SA-mediated resistance mechanisms (Desikan and others 2001). Although the implications of pathway cross-talk on plant resistance to herbivores are the focus of many studies, the impact of cross-talk in plant-microbe interactions is less well-studied. Therefore, assessing the ecological significance of plant trade-offs constitutes an attractive field in future plant research.

Concluding Remarks

Defense responses are vital, but costly for the plant. Thus, instead of maintaining them continuously, the plant activates different inducible mechanisms, depending on the attacker it is encountering. These inducible defenses are subjected to tight regulation, because their rapid activation is the key to successful defense. The spatial and temporal concentrations of the different alarm signals that are generated in response to recognition of a microorganism are instrumental in the regulation of the outcome of the interactions between the different pathways. These interactions can be mutually antagonistic, cooperative, or synergistic, and will finally determine the response to the particular attacker.

JAs play a central role in the complex signaling network leading to disease resistance. A model illustrating the role of the JA signaling pathway in basal and induced resistance against pathogens is shown in Figure 1. In addition to their key role in plant-herbivore interactions, it is evident that JAs play a major role in basal and induced resistance against necrotrophic pathogens. However, JAs can also influence resistance to hemibiotrophs and certain biotrophs, which were generally thought to be

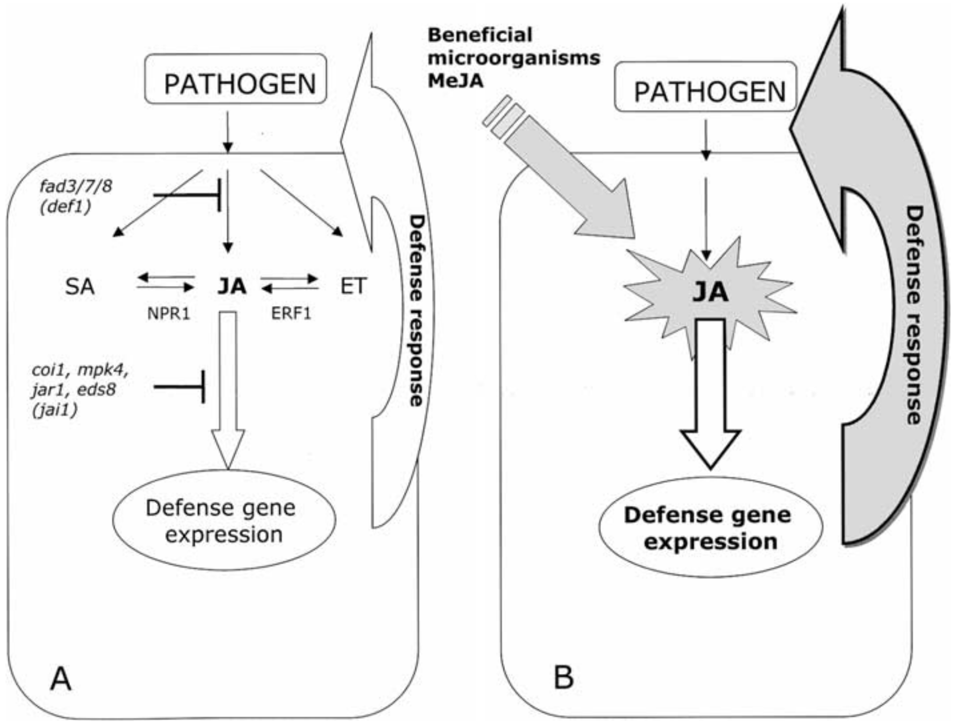

Figure. 1. Model illustrating the role of the JA signaling pathway in basal and induced resistance against pathogens. **A.** Pathogen recognition leads to the production of the alarm signals JA, ET and SA. Cross-talk among the different pathways shapes the outcoming defense response. Key elements in cross-talk and *Arabidopsis* mutants impaired in JA biosynthesis or signalling pathway are indicated (tomato mutants between brackets). **B.** Beneficial microorganisms (such as ISR-inducing rhizobacteria) or pretreatment with MeJA prime the tissues for a quicker and more effective activation of the JA-dependent defense responses after pathogen attack, resulting in enhanced resistance.

resisted exclusively through SA-dependent defenses. Thus, the concept that JA-dependent defense responses are predominantly effective against pathogens with a necrotrophic lifestyle, whereas SA-dependent defense responses are mostly effective against pathogens with a (hemi)biotrophic lifestyle, is not universal and should be used with caution. Besides its role in pathogenic interactions, recent advances in defense signaling research revealed that JAs can also play an important role in the response of plants to beneficial micro-organisms, either in the induction of systemic resistance or in the establishment of a beneficial association with the plant. Understanding the mechanisms regulating JA signaling in plants will provide novel insights into how plant health can be improved in the context of environmentally friendly practices for disease control and sustainable agriculture.

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