CURRENT REVIEW

Weights in the Balance: Jasmonic Acid and Salicylic Acid Signaling in Root-Biotroph Interactions

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Work on the interaction of aerial plant parts with pathogens has identified the signaling molecules jasmonic acid (JA) and salicylic acid (SA) as important players in induced defense of the plant against invading organisms. Much less is known about the role of JA and SA signaling in root infection. Recent progress has been made in research on plant interactions with biotrophic mutualists and parasites that exclusively associate with roots, namely arbuscular mycorrhizal and rhizobial symbioses on one hand and nematode and parasitic plant interactions on the other hand. Here, we review these recent advances relating JA and SA signaling to specific stages of root colonization and discuss how both signaling molecules contribute to a balance between compatibility and defense in mutualistic as well as parasitic biotroph-root interactions.

Biotrophic plant invaders encompass a wide variety of organisms including bacteria, fungi, worms, and parasitic plants. Their relationships with host plants result in different outcomes that range from mutually beneficial to parasitic. Biotrophs either insert feeding structures into living plant cells or induce and inhabit special plant-derived organs to exploit living material (Fuller et al. 2008; Glazebrook 2005; Oldroyd and Downie 2008). Their recognition by the plant triggers complex, multi-step signaling cascades leading to accommodation or combat of the colonizer. Phytohormones contribute to modulating such defense and accommodation processes and, among them, jasmonates and salicylic acid (SA) have received considerable attention (Bari and Jones 2009; Beckers and Spoel 2006; Ding and Oldroyd 2009; Glazebrook 2005; Hause et al. 2007). According to genetic evidence retrieved from studies on *Arabidopsis thaliana* defense against biotrophic leaf pathogens generally involves SA-dependent signaling, whereas inducible defense against leaf-chewing insects and necrotrophic microbes is mediated by jasmonic acid (JA)-dependent signaling (Balbi and Devoto 2008; Beckers and Spoel 2006; Browse 2009; Glazebrook 2005; Loake and Grant 2007). However, exceptions and more complex scenarios exist, depending on environmental or experimental conditions or a combination of biotrophic and necrotrophic lifestyles for various pathogens (Glazebrook 2005; López et al. 2008; Robert-Seilaniantz et al. 2007; Spoel and Dong 2008). Furthermore, SA and JA signaling frequently interact either antagonistically or synergistically, depending on the combination of their respective concentrations (Koornneef et al. 2008; Mur et al. 2006). This means that both SA and JA

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can impact biotrophic interactions and that the negative crosstalk between JA and SA signaling can be exploited by pathogens to enhance plant susceptibility. A prominent example is provided by the hemibiotrophic bacterium *Pseudomonas syringae*, which uses the phytotoxin coronatine, a substance similar to jasmonate-isoleucine (JA-Ile) (Katsir et al. 2008) to repress the SA-mediated defense pathway (Brooks et al. 2005; Laurie-Berry et al. 2006).

A number of excellent reviews describe the current understanding of the complex signaling network involved in the plant's defense against biotrophic foliar pathogens (Bari and Jones 2009; Beckers and Spoel 2006; Glazebrook 2005; Lipka et al. 2008; López et al. 2008; Robert-Seilaniantz et al. 2007). In contrast, very little is known about the importance of SA- and JA-mediated signaling in root biotrophic interactions. This field is presently emerging through studies on plant interaction with exclusively root-colonizing biotrophs. Among these are beneficial rhizobial and arbuscular mycorrhizal (AM) symbioses on one hand and detrimental infections by nematodes and parasitic plants on the other hand. Interestingly, plant signaling components leading to root colonization are partly shared between these interactions (Mathesius 2003; Paszkowski 2006), which is inviting for comparisons of compatibility and defense. In this review, we describe current knowledge of the role of SA and JA signaling in root-biotroph interactions. When possible, we relate SA and JA signaling to defined stages of the colonization process. Also, when available reports permit, we put observations on SA and JA signaling in root-biotroph interactions in relation to the general model for leaf parasites (Glazebrook 2005) and conclude with a discussion of the differences and similarities between the interaction of mutualistic and parasitic biotrophs with the "hidden plant organ."

MUTUALISTIC ASSOCIATIONS: AM AND LEGUME-RHIZOBIUM SYMBIOSES

Precontact recognition: JA modulates electrophysiological signals.

Diffusible elicitors released by microbes trigger plant responses before physical cell-to-cell contact occurs (Garcia-Brugger et al. 2006; Garcia-Garrido and Ocampo 2002; Kosuta et al. 2003; Olah et al. 2005). Similarly, the colonization of plant roots by AM fungi and rhizobia is preceded by a molecular dialogue to establish recognition. Rhizobia react to host root-derived flavonoids by the production of Nod factors, which, upon perception, induce the plant's nodulation program (Hirsch et al. 2001; Radutoiu et al. 2007). Nod factors and approaching AM fungi induce calcium spiking in root hairs (Kosuta et al. 2008). In rhizobial symbioses, calcium spiking

acts as a secondary messenger and activates Nod factor–induced gene expression, infection thread (IT) formation, and nodule organogenesis (Oldroyd and Downie 2008). Its role in AM symbioses has not yet been directly addressed.

JA exogenously applied to the growth medium decreases the number of nodules induced by *Sinorhizobium meliloti* on *Medicago truncatula* roots in a dose-dependent manner (Sun et al. 2006). This result could be attributed to JA-mediated cell-cycle inhibition (Swiatek et al. 2002) during nodule formation. However, JA seems to even suppress nodule initiation, as nodule primordia were absent and induction of two Nod factor–responsive marker genes (*RIP1* and *ENOD11*) was blocked. Assessment of the impact of JA application on calcium spiking revealed that JA decreases the responsiveness of calcium spiking to Nod factor in a dose-dependent manner. Whereas high concentrations (100 μM) of JA strongly inhibited calcium spiking, lower concentrations (10 to 50 μM) modified the calcium signal by lengthening the spike period, thereby decreasing the frequency of spiking (Sun et al. 2006). Modulation of the calcium signature might have an important role in fine tuning the initiation of colonization in an interplay of Nod factor and JA concentrations. Indeed, the number of calcium spikes is critical for triggering the Nod factor signaling pathway (Miwa et al. 2007). Similar calcium oscillations are also important for other strictly regulated systems, such as stomatal opening, for which calcium oscillation in guard cells needs to follow defined parameters for a change in the stomatal state to occur (Allen et al. 2001). It is tempting to speculate that JA participates in adjusting the amount of colonization to a level that the plant can support under a given environmental condition. However, it should be noted that the effect of JA on calcium spiking has been shown exclusively by pharmacological application. Therefore, the relevance of endogenous JA for calcium spiking at the onset of rhizobial colonization remains to be determined by genetic studies. If JA acts on calcium spiking, it could also be expected to influence AM symbioses at this early stage. Identification of one or more targets of JA-mediated inhibition of calcium spiking will immensely increase our understanding of symbiosis regulation.

Roots of soybean and wheat have been reported to secrete JA into the rhizosphere (Creelman and Mullet 1995; Dathe et al. 1994) showing that rhizosphere organisms might be exposed to JA molecules. The direct effect of JA on AM fungi is not known. However, JA and methyl jasmonate (MeJA) exogenously applied to rhizobia induced the expression of Nod genes (Rosas et al. 1998) and the production of Nod factors (Mabood et al. 2006). The effect of JA on Nod-factor production was enhanced when JA was coapplied with flavonoids (Mabood et al. 2006; Rosas et al. 1998), which are known to induce nod-factor production (Hirsch et al. 2001). In the natural ecosystem, rhizobia can thus potentially balance the JA-mediated decrease in nod-factor responsiveness of calcium spiking by a JA-promoted augmentation of nod-factor production. In summary, JA signaling appears to be involved in balancing the interplay between roots and microbes at the initial events of colonization.

Cell-to-cell contact: Inhibition vs. mediation of cell-autonomous compatibility.

Recognition between plants and their colonizers is a multistep process allowing decisions to be made at each level of colonization. Precontact signaling molecules, like rhizobial Nod factors or AM fungal Myc factors, prime the plant for the symbiotic program. Upon physical contact however, the microbe has to overcome further checkpoints. AM fungi form a hyphopodium from which a penetration hyphae passes through the rhizodermal cell cytoplasm guided by a prepenetration apparatus (PPA) (Genre et al. 2005). The fungus then apoplastically passes the outer cell layers and develops arbuscules in cell files of the inner cortex. Most rhizobia invade through root hairs and migrate within a structure similar to the PPA, the so-called IT (Fournier et al. 2008). The IT grows inward through the root hair, the rhizodermis, and cortex and releases rhizobia into the inner cells of the simultaneously initiated nodule, in which nitrogen fixation occurs (Oldroyd and Downie 2008). PPA and IT formation might provide a first checkpoint at which the plant can manipulate the success of the colonizer. The intercellular colonization of cells by arbuscules or of nodules by rhizobia is another process that must be under tight plant control (Parniske 2000, 2008).

SA signaling in cell-to-cell contact. Indeed, legumes strictly control the number of successful rhizobial infection events and can reject the bacteria at any stage of the interaction through a rapid defense reaction (Baron and Zambryski 1995). For example, once a certain number of nodules is reached in wild-type *Medicago sativa* colonized by *Sinorhizobium meliloti*, hypersensitive response (HR)-like abortions of IT accompanied by cell necrosis and accumulation of phenolic compounds occur at the sites of IT formation (Vasse et al. 1993). Moreover, rhizobia with defects in surface polysaccharides that serve for recognition upon attachment elicited defense reactions similar to HR in alfalfa and pea (Niehaus et al. 1993; Perotto et al. 1994). Additionally, it has been reported that plant mutants that do not allow colonization by AM fungi show various responses similar to HR that likely contribute to the rejection of the colonizer (Genre and Bonfante 2002; Gianinazzi-Pearson et al. 1996; Gollotte et al. 1993).

It could be hypothesized that these HR-like symptoms might be linked to SA-mediated defense signaling if SA function in leaves was conserved in roots. In support of such a hypothesis, it was shown by exogenous application to rice roots that SA interferes with susceptibility to AM fungi by slowing down the onset of intraradical colonization without influencing the number of extraradical hyphopodia (Blilou et al. 2000). More importantly, at early stages of the AM interaction, SA levels transiently increase in roots of wild-type rice and pea, probably in response to hyphopodia formation on the root surface (Blilou et al. 1999, 2000). This SA increase is mirrored by a transient elevation in defense-gene expression in *Medicago truncatula* roots at early stages of AM colonization (Liu et al. 2003). In the pea mutant *sym30,* which is resistant to colonization by AM fungi and rhizobia (Duc et al. 1989), the initial SA increase persists (Blilou et al. 1999). The same was found when wild-type pea roots or alfalfa roots were inoculated with an incompatible *Rhizobium* strain or a mutant strain defective in Nod-factor production, which abolishes symbiont recognition by the plant (Blilou et al. 1999; Martinez-Abarca et al. 1998). This suggests that, after physical contact, the symbiont is confronted with a defense response that involves SA and subsequently needs to be switched off to allow colonization. In the above examples, defense suppression does not occur if the symbiosis is defective at an early step of the interaction—be it on the side of the plant or the microbe. To investigate whether the correlation between increased SA levels and rejection of symbiotic colonization is a general phenomenon, it would be interesting to determine if elevated SA levels are also persistent in other nonmycorrhizal or nonnodulating mutants.

Exogenous addition of SA inhibited formation of indeterminate nodules in vetch but not of determinate nodules in *Lotus japonicus* (van Spronsen et al. 2003) and also inhibited rhizobial growth (Stacey et al. 2006). To test the effect of endogenous SA levels on nodulation, Stacey and associates (2006) expressed NahG, a bacterial salicylate hydroxylase that degrades SA to catechol (Gaffney et al. 1993) and therefore leads to de-

creased SA levels, in *Lotus japonicus* plants. Roots of these transgenic plants displayed an increase in formation of determinate nodules, which was inversely correlated with SA levels in individual transgenic lines. The increase in nodule number was caused by enhanced IT formation, suggesting that endogenous SA inhibits the formation of this early infection structure.

Also, the establishment of an AM symbiosis was affected by endogenous SA levels at early stages of colonization. In *Nicotiana tabacum* plants expressing NahG, the level of SA accumulation was reduced, which led to an elevation of AM colonization at early timepoints postinoculation, including higher numbers of infection units and arbuscules (Herrera Medina et al. 2003). On the contrary, in tobacco plants that constitutively produce elevated levels of SA, lower colonization levels were observed at early timepoints. The effects of modified SA levels on AM colonization were no longer detectable at later timepoints postinoculation (Herrera Medina et al. 2003), indicating that SA-mediated inhibition of AM colonization mainly leads to a colonization delay. This might be interpreted as a repression of early infection events that is, at later stages, compensated by extensive spread of fungal intraradical hyphae. In summary, SA acts at the first cell-to-cell contact between the symbionts to reduce colonization. During rhizobial colonization, SA suppresses IT formation (Stacey et al. 2006). For AM colonization the exact stage of inhibition has not been described, but PPA formation is a good candidate target (Fig. 1A). In this context, it is intriguing that, in *Medicago truncatula* hairy roots, a class 10 pathogenesis-related protein (PR-10) was upregulated in root cells close to the hyphopodium and subsequently repressed during formation and fungal passage of the PPA (Siciliano et al. 2007). It can be experimentally addressed whether the spatio-temporal expression pattern of this PR-10–encoding gene marks SA activity. It would further be interesting to combine pharmacological SA application with live imaging (Genre et al. 2005; Fournier et al. 2008) to document the effect of SA on early infection structures.

JA signaling in cell-to-cell contact. How might initial SA-mediated defense signaling be switched off to avoid abortion of the symbiotic colonization event at the rhizodermis? Mutually inhibitory crosstalk between JA and SA signaling is a common and generally accepted phenomenon in plant shoots (Beckers and Spoel 2006; Glazebrook 2005; Glazebrook et al. 2003; Lorenzo and Solano 2005). Although, in the absence of evidence, several hypotheses can be brought forward, it is a particularly appealing possibility that JA signaling might counterbalance SA signaling at early stages of root symbioses—possibly in response to perception of microbial effector molecules by the plant. Genes encoding JA-biosynthetic enzymes are upregulated in *Lotus japonicus* at early colonization steps by *Mesorhizobium loti* and then repressed upon nodule formation (Kouchi et al. 2004), while SA-responsive PR-encoding genes are repressed in *Medicago truncatula* (Godiard et al. 2007; Mitra and Long 2004). This suggests that JA might be acting early in rhizobial colonization, during a step downstream of calcium spiking and upstream of nodule formation. At present however, it still remains an open question if JA signaling is actually occurring and counteracting or, rather, supporting SA signaling (Koornneef et al. 2008; Mur et al. 2006) in the abortion of early infection structures. JA-deficient legume mutants are necessary to address these issues.

Colonization by AM fungi leads to increased JA levels in roots of barley and barrel medic (Hause et al. 2002; Isayenkov et al. 2005; Stumpe et al. 2005). The increase is most pronounced at later stages of the symbiosis and has been related to arbuscule formation, which will be discussed below (Hause et al. 2002). However, a weak JA elevation measured in complete root systems at early timepoints postinoculation (Hause

et al. 2002) suggests that JA accumulation might also occur in response to early infection structures. It could be interpreted as dilution of considerably high JA concentrations in a small number of cells that are in contact with early structures such as hyphopodia or penetrating hyphae. Since JA levels were correlated to overall AM colonization by Hause and associates (2002), no conclusions can be drawn with respect to the presence and quantity of individual fungal structures. Plants transformed with reporter genes fused to JA-responsive promoters (Stenzel et al. 2008) are useful tools to visualize JA distribution in roots at the cellular level during all stages of colonization by AM fungi and rhizobia. They could be used to get a first glimpse on a possible interaction of SA and JA signaling at early stages of colonization.

A cell-specific role for JA signaling has been shown in cortex cells containing arbuscules. It has been reported that JA-induced genes and enzymes are expressed specifically in these
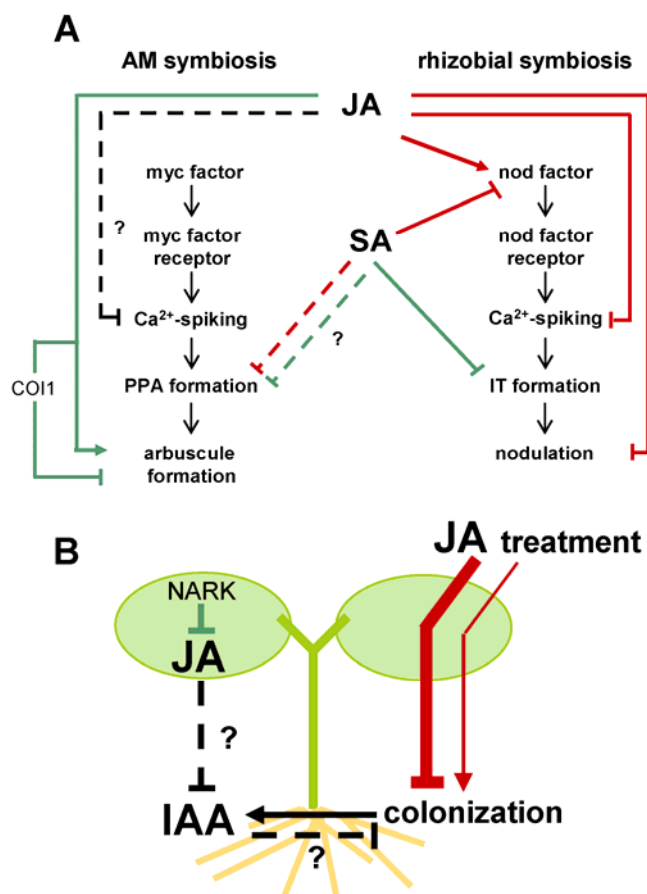

Fig. 1. A, Schematic representation of the influence of jasmonic acid (JA) and salicylic acid (SA) signaling on various steps of root colonization by mutualistic arbuscular mycorrhizal (AM) fungi and rhizobia. **B,** Modulation of mutualist colonization by systemic JA signaling. Data from rhizobial and AM symbioses are united in one model. Application of JA to shoots at high concentrations or high frequency (bold red arrow) inhibits and at low concentrations and low frequency (fine red arrow) increases root colonization. AM colonization induces indole-acetic acid (IAA) levels in roots, which possibly inhibits further colonization. *Gm*NARK suppresses JA biosynthesis in shoots, thereby suppressing colonization. In the *Gmnark* mutant, JA levels in shoots are increased, which enhances root colonization. This might occur through suppression of IAA levels in the root. Green indicates evidence obtained via genetic approaches, and red displays evidence obtained by pharmacological application of the hormone. Black indicates hypotheses or data that were obtained in the wild type without pharmacological hormone application. Dotted lines decorated with question marks represent hypothetical scenarios for which no experimental evidence is yet available.

cells, indicating cell-specific JA accumulation. Arbusculated cells of *Medicago truncatula* express the enzyme allene oxide cyclase (AOC) (Isayenkov et al. 2005), which catalyzes a key step in JA biosynthesis. In barley transcripts and proteins of the JA-biosynthetic enzyme, allene oxide synthase (AOS) and the jasmonate-inducible protein JIP23 accumulate in arbusculated cells (Hause et al. 2002). The importance of JA in arbuscule formation was demonstrated by RNAi-mediated knockdown of *AOC* in roots of *Medicago truncatula* (Isayenkov et al. 2005). A strong decrease in root JA levels due to silencing of *AOC* was correlated with a significant delay of mycorrhizal colonization and especially a reduction in arbuscule numbers. *AOC* was downregulated in roots of composite RNAi lines but not in shoots, in which JA levels still increased in response to AM (Isayenkov et al. 2005). Although the authors were therefore not able to exclude effects of a systemic hormone imbalance, this finding represents an important step forward in assigning a role to JA in arbuscule formation. Recently, these results were confirmed in the tomato JA-deficient mutant *spr2* (suppressor of prosystemin-mediated responses 2), which is defective in a fatty acid desaturase required for JA biosynthesis (Li et al. 2003). In this mutant, arbuscule numbers were reduced as well and could be restored to wild-type levels by exogenous application of JA. In turn, an *overexpressor of prosystemin* displaying elevated JA levels showed enhanced colonization (Tejeda-Sartorius et al. 2008). AM colonization thus appears to be related to endogenous JA levels. In contrast to these results, AM colonization was accelerated and arbuscule abundance and functionality was increased in the tomato mutant *jai1* (Herrera Medina et al. 2008), which is defective in COI1, a component of the JA-Ile-receptor complex (Katsir et al. 2008; Li et al. 2001). *Nicotiana attenuata* plants post-transcriptionally silenced for COI1 also displayed elevated AM colonization (Riedel et al. 2008). These contrasting results between mutants perturbed in JA accumulation and JA-Ile perception can be explained if COI1-mediated JA signaling limits mycorrhizal colonization while JA-mediated support of colonization and arbuscule formation is independent of COI1 function. However, this hypothesis needs further confirmation through the simultaneous examination of mutants defective in JA biosynthetic enzymes and COI1 in the same genetic background and the same experimental setting.

How could JA support AM colonization and arbuscule formation? Simultaneous localization of the JA-responsive JIP23 protein in barley phloem companion cells and in arbusculated cells led Hause and associates (2002) to speculate that JA could be involved in increasing the sink strength of arbusculated cells for carbohydrates that the plant supplies to the fungus. Differential expression of genes encoding enzymes involved in carbon partitioning in tomato mutants with modified endogenous JA levels point in this direction (Tejeda-Sartorius et al. 2008). JA signaling is further known to be involved in defenses against parasites that breach the cell wall and has been designated, therefore, the "cell-ingress pro-hormone" (Farmer 2007). Thus, in an alternative scenario, JA could be necessary for accommodation of the arbuscule in the cell (Hause et al. 2007) independent of the nourishment of the fungus. Arbuscule formation causes invagination and a several-fold extension of the plant cell plasma membrane relative to the original surface area (Hause and Fester 2005). The apoplastic space between the plant and the fungal membrane remains continuous with the cell wall of neighboring cells and contains loosely assembled cell-wall material (Balestrini and Bonfante 2005). Extension of cell wall and membrane in response to the dramatic force applied to the cortex cell by a growing arbuscule must require a fine-tuned dialogue between the symbionts to avoid an abortive defense reaction. Thus, in response to developing arbuscules, JA signaling might create a supportive environment for cell-wall extension by promoting modifications in cell-wall composition. An example for a possible link between cell-wall integrity and JA signaling is given by the *Arabidopsis* mutants *cev1*, deficient in the cellulose synthase *CeSA3* and therefore root cellulose content, and *cobra5*, deficient in a glycosylphosphatidylinositol (GPI)-anchored protein that modulates cellulose deposition; both mutants lack crucial cell-wall components and show elevated JA levels and increased JA-related gene expression (Ellis et al. 2002; Ko et al. 2006). Alternatively or additionally, JA signaling might directly activate cell expansion, as in developing potato tubers (Koda 1997; Takahashi et al. 1994), for example through activation of cell wall–cleaving enzymes. Also cytoskeletal rearrangements, which have been observed in arbusculated cells (Genre and Bonfante 1998), might be induced by JA (Koda 1997; Matsuki et al. 1992).

In summary, SA signaling controls mutualist colonization by inducing cell-autonomous defense reactions during early encounters between the symbionts. JA seems to play a role at multiple steps of the colonization. It appears to promote accommodation of AM fungi at later stages of colonization and to suppress AM colonization via the COI1-mediated signaling pathway (Fig. 1A).

Systemic influence on the quantity of colonization: JA-balanced susceptibility.

Biotrophic symbionts consume considerable amounts of carbohydrates. For example up to 20% of the plant's photoassimilate are invested in the AM symbiosis (Kucey and Paul 1982; Wang et al. 1989; Wright et al. 1998). To avoid excessive carbon losses, plants need to control the amount of colonization. This autoregulation is most evident in split-root systems in which rhizobial or AM colonization of one half of the split root suppresses subsequent colonization of the other half. The two symbioses also mutually suppress each other (Catford et al. 2003), indicating that related mechanisms control the amount of colonization by both microbes. It is, therefore, not surprising that some hypernodulating mutants also display elevated AM colonization (Meixner et al. 2005; Shrihari et al. 2000; Solaiman et al. 2000).

Systemic effects of endogenous JA. LjHAR1/MtSUNN/GmNARK is a CLAVATA1-like leucine-rich repeat (LRR) receptor kinase required for autoregulation of nodulation. Through grafting experiments, it was shown to act systemically from the shoot (Krusell et al. 2002; Nishimura et al. 2002; Schnabel et al. 2005; Searle et al. 2003). A search for downstream signals of *GmNARK* (*Glycine max* nodule autoregulation receptor kinase) via transcriptional profiling revealed that a group of jasmonate-responsive genes, including those encoding JA biosynthetic enzymes and an orthologue of the transcription factor *MYC2*, which is involved in JA signaling (Lorenzo et al. 2004), was constitutively upregulated in shoots of *Gmnark* mutants (Kinkema and Gresshoff 2008; Seo et al. 2007). The SA-inducible gene encoding PR protein PR1a, in turn, was suppressed (Seo et al. 2007). Furthermore, while, in wild-type shoots, JA-responsive genes were downregulated upon nodulation, expression levels remained high in the *GmNARK* mutant shoots irrespective of rhizobial colonization (Kinkema and Gresshoff 2008). The differences in gene expression were correlated with JA levels that were twice as high in *Gmnark* mutant leaves than in wild-type leaves (Seo et al. 2007). Functional evidence for the relevance of systemic JA signaling in enhancing nodule formation was obtained by application of the JA-biosynthesis inhibitor n-propyl gallate to shoots (Kinkema and Gresshoff 2008). The treatment significantly reduced nodule numbers in *Gmnark* mutant roots without affecting wild-type nodulation. *GmNARK* thus dampens JA signaling constitu-

tively and controls nodulation via further suppression of JA biosynthesis and signaling. Conversely, enhanced levels of JA in the shoot systemically promote root nodulation.

How could elevated JA levels in the *Gmnark* mutant contribute to increasing nodulation? Meixner and associates (2005) showed, in split-root systems using soybean wild type, that mycorrhizal colonization of one part of the split root suppressed colonization of the second part. In the *Gmnark* mutant, no such suppression was observed. Measurement of phytohormones in mycorrhizal and nonmycorrhizal parts of split roots revealed an increase of indole-acetic acid (IAA) levels in the colonized half. The increase was twofold higher in wild type than in *nark* mutant roots, suggesting that high auxin might participate in the systemic suppression of mycorrhizal colonization. The fact that application of auxin transport inhibitors increases nodulation and mycorrhizal colonization (Wasson et al. 2006; Xie et al. 1998) supports this idea. On the basis that equal mechanisms of NARK-dependent autoregulation control rhizobial and mycorrhizal colonization, it could be suggested that enhanced JA levels and signaling in the shoots of *nark* mutants might either inhibit auxin transport to the colonized parts of the root or suppress auxin biosynthesis, thereby limiting the amount of auxin that can be transported. It fits the picture that, in *Arabidopsis*, the JA-responsive transcription factor MYC2, the orthologue of a gene upregulated in *Gmnark* shoots (Kinkema and Gresshoff 2008), inhibits the synthesis of the IAA precursor tryptophan and induces the production of flavonoids that act as IAA-transport inhibitors (Dombrecht et al. 2007). Systemic JA signaling might also affect localized SA signaling responsible for abortion of infection events (discussed above). Elevated JA levels in the shoot might further lead to enhanced carbohydrate partitioning to the root (Babst et al. 2005; Henkes et al. 2008), such that a higher number of rhizobia can be nourished. Suppression of endogenous JA biosynthesis in the shoot thus could contribute to keeping colonization levels within boundaries. On the other hand, enhanced JA levels have been implicated in signaling nutrient deficiency (Armengaud et al. 2004; Schmelz et al. 2003; Tang et al. 2001) and systemic JA signaling might increase root colonization (or release suppression) in conditions in which the plant needs enhanced nutrient supplies from the symbiont.

Systemic effects of exogenously applied JA. Systemic effects on symbiotic root associations of exogenous SA application to shoots have not been observed (Ludwig-Müller et al. 2002; Nakagawa and Kawaguchi 2006). In turn, contradictory results were obtained when JA was exogenously applied to plants. In contrast to the findings of Kinkema and Gresshoff (2008) based on elevated endogenous JA levels, exogenous application of JA to shoots inhibited nodulation in a dose-dependent manner even in the *Ljhar1* and *Gmnark* mutant (Nakagawa and Kawaguchi 2006; Seo et al. 2007). This contradiction might be due to the specific distribution within the tissue that distinctively determines the outcome of JA signaling. In hypernodulating *Gmnark* mutants, an increase in JA might occur in restricted tissue, e.g., in vascular bundles (Hause et al. 2003; Nontachaiyapoom et al. 2007; Stenzel et al. 2003), whereas it is in contact with the entire leaf surface upon exogenous spraying. To understand how JA contributes to increasing nodulation in *nark* mutants, it is essential to determine the location of its activity through the use of JA-responsive reporter genes. Secondly, high concentrations of JA, mimicking wounding and pathogen attack and causing pleiotropic effects such as inhibition of shoot growth, lateral root formation, and loss in chlorophyll content (Nakagawa and Kawaguchi 2006), might further be responsible for the observed inhibition of colonization. The importance of JA concentration is illustrated by lateral root formation in rice, which is stimulated by exogenous application of JA up to a concentration of 2 μM but is inhibited by higher concentrations (Wang et al. 2002). This is also evident from studies of the AM symbiosis in which JA application to shoots led to a diversity of results depending on JA concentration and frequency of application. Low concentrations of JA (5 μM) applied once per week stimulated AM development in *Allium sativum* (Regvar et al. 1996). In contrast, spraying tomato leaves with 5 and 50 μM MeJA twice a week reduced AM colonization in tomato (Herrera Medina et al. 2008) and so did treatment of leaves of *Tropaeolum majus*, *Carica papaya*, and *Cucumis sativus* with high concentrations of JA (0.05 to 5 mM) every second day (Ludwig-Müller et al. 2002). Also, the timing of JA application to mycorrhizal plants influenced colonization (Tejeda-Sartorius et al. 2008), possibly because JA responsiveness varies with the developmental stage of plants (Matthes et al. 2008). Tejeda-Sartorius and associates (2008) showed that a single treatment of two different tomato leaves with one drop of 5 μM JA was sufficient to enhance AM colonization and arbuscule formation in the wild type and the JA-deficient mutant *spr2*. The extent to which colonization levels were raised depended on which day after AM inoculation JA was applied. When plants were treated with the same amount of JA for four times instead of only once, colonization was suppressed. These examples illustrate that conclusions drawn on the influence of systemic JA signaling on root symbioses might be biased if they are based on the application of a single JA concentration, a range of high concentrations, or on frequent application only, and it is necessary to carefully establish dose-response relationships. Furthermore, as stated before, pharmacological hormone and inhibitor application need to be combined with the use of plant mutants perturbed in JA biosynthesis and signaling in the same experimental setting to deepen our understanding of systemic JA signaling in root symbioses. A model summarizing the current knowledge on systemic JA action in mutualistic symbioses is given in Figure 1B.

EXPLOITIVE ASSOCIATIONS WITH PLANT PARASITIC NEMATODES AND PARASITIC PLANTS

Plant-parasitic nematodes and parasitic plants are obligate biotrophic pathogens of many plant species. In contrast to the symbiotic microorganisms discussed earlier, their infection process does not appear to be actively supported by the plant via plant-derived infection structures such as the PPA or the IT. Instead, they pierce the root in a cell-destructive manner and subsequently become biotrophic. Both parasites connect to the vascular cylinder to directly access plant nutrients. Nematodes induce dramatic developmental changes in plant roots by fundamentally manipulating root-cell behavior. In the root differentiation zone, root-knot nematodes (RKN) (members of the genus *Meloidogyne*) induce nuclear divisions without cell division leading to the formation of multinucleate giant cells that are connected to the vascular tissue and serve as feeding sites. The root cells that surround giant cells divide and swell, leading to the formation of root knots (Caillaud et al. 2008). Cyst nematodes (members of the genera *Heterodera* and *Globodera*) pierce and disrupt cortical cells while entering the root. Induction of feeding sites at the vascular cylinder leads to the breakdown of cell walls between neighboring cells, creating a multinucleate syncytium (Williamson and Gleason 2003). Parasitic plants of the genera *Orobanche* and *Striga* rely on carbohydrate nutrition from their dicotyledoneous or monocotyledoneous hosts, respectively, to complete their life cycle. After germination, parasitic plants form a haustorium that breaks into the host root, crosses the root cortex, and fuses

with the vascular cylinder of the host plant to exploit nutrients (Rispail et al. 2007).

Compared with symbiotic interactions, less is known about JA and SA effects on particular steps of nematode or parasitic plant colonization. Therefore, we combine the description of those with effects on overall colonization. For both nematode and parasitic plant interactions, resistant and susceptible plant cultivars are known from a number of plant species. The use of such cultivars in comparative studies is useful to provide clues on the mechanisms involved in defense or compatibility and has yielded some information on the role of SA and JA signaling in these processes. The gene *Mi-1,* encoding a nucleotide binding-LRR protein, confers resistance to RKN in tomato. In the tomato cultivar Motelle, which carries *Mi-1,* RKN can still move to the central cylinder, but localized cell death occurs instead of giant-cell development (Ho et al. 1992). To test whether defense hormone pathways play a role in *Mi-1*–mediated resistance, Motelle hairy roots were transformed with the bacterial salicylate-hydroxylase gene NahG to reduce SA content. In these roots, resistance to the RKN *Meloidogyne javanica* was strongly decreased and could be restored by application of the functional SA analogue benzothiadiazole (BTH). Therefore, resistance conferred by *Mi-1* is, at least in part, mediated via SA signaling (Branch et al. 2004). A second experiment suggested that relatively low levels of SA are sufficient to confer *Mi-1*–dependent resistance, because NahG plants degrading SA less efficiently than the hairy roots used by Branch and associates (2004) were not compromised in resistance (Bhattarai et al. 2008). In a further study, treatment of okra and cowpea with SA foliar spray caused a decrease in RKN infection (Nandi et al. 2003). SA application also reduced parasitism by cyst nematodes. Treatment of white clover and *Arabidopsis* with SA led to increased resistance without directly affecting nematode viability (Kempster et al. 2001; Wubben et al. 2008). Furthermore, a number of SA-deficient *Arabidopsis* mutants, namely *sid2-1*, deficient in isochorismate synthase required for SA synthesis (Wildermuth et al. 2001), *pad4-1*, affected in a lipase gene required for SA synthesis (Jirage et al. 1999), and NahG, were more susceptible to the cyst nematode *Heterodera schachtii* than was the wild type. The same was true for SA-insensitive mutants impaired in the SA-signaling component NPR1 (Cao et al. 1997). In turn, the *npr1*-supressor mutant *sni1* (Li et al. 1999) displayed an elevated resistance to cyst nematodes (Wubben et al. 2008). Interestingly, in wild-type *Arabidopsis*, cyst-nematode infection caused an increase in SA levels in shoots but not in roots, which was paralleled by induction of *PR-1* (encoding PATHOGENESIS-RELATED PROTEIN 1) a marker gene for SA signaling and systemic acquired resistance (SAR) (Wubben et al. 2008). The function of this SA elevation in shoots for cyst-nematode parasitism remains unclear. Nevertheless, it can be concluded that cyst-nematode infection induces SAR in *Arabidopsis*. The absence of SA induction in roots further illustrates that the role of a defense-signaling compound cannot necessarily be inferred directly from its levels in the tissue of interest.

Whereas SA signaling partly explains *Mi-1*-mediated resistance, JA signaling is not involved (Bhattarai et al. 2008). Nevertheless, exogenous application of high concentrations of MeJA (up to 1.5 mM) to roots of oat and spinach and to shoots of tomato enhanced resistance to parasitic nematodes, possibly by elevating the level of compounds that are toxic to nematodes like phytoectosteroids, flavonoids, and proteinase inhibitors (Cooper et al. 2005; Soriano et al. 2004a and b). However, as discussed above, other pleiotropic effects like plant growth depression caused by application of high concentrations of MeJA cannot be excluded. On the contrary, Bhattarai and associates (2008) found that, on roots of the tomato mutant *jai1,* which is defective in the JA-Ile receptor component COI1, a lower number of RKN egg masses was produced as compared with wild-type roots. This indicates that COI1-mediated JA signaling promotes susceptibility to RKN. The endogenous JA level did not seem to be relevant, as the JA-deficient tomato mutant *def1*, defective in a yet-unknown component, had no quantitative phenotype (Bhattarai et al. 2008). On the other hand, the hypernodulating *Lotus japonicus* mutant *har1* was hyperinfected with RKN (Lohar and Bird 2003), which might be partly due to higher JA levels in the shoot, as shown for the mutant *Gmnark*, defective in the soybean orthologue of HAR1 (Kinkema and Gresshoff 2008). It is possible that, analogous to the interaction of *Pseudomonas syringae* with plant leaves, COI1-mediated RKN susceptibility is not actually caused by plant-produced JA-Ile but by an analogous molecule exuded by RKN to suppress SA signaling (Bhattarai et al. 2008).

Transcriptomics studies on whole soybean roots and laser-captured syncytia suggest that JA signaling is suppressed during fully established compatible cyst-nematode interactions (Ithal et al. 2007a and b). Genes encoding JA-biosynthesis enzymes, such as *AOS* and *AOC*, were downregulated in feeding structures induced by *Heterodera glycines*. However, *OPDA Reductase 3* (*OPR3*), encoding the orthologue of *Arabidopsis* OPR3, which is critical for JA biosynthesis downstream of *AOS* and *AOC* (Stintzi and Browse 2000), was induced—a contradictory result that awaits further investigation. The biological role of the downregulation of JA genes encoding JA-biosynthesis enzymes is also unclear. It might be necessary to allow for the dramatic restructuring of root cells during syncytia formation or for the suppression of host defense responses.

Defense reactions during incompatible interactions between parasitic plants and resistant or partially resistant hosts range from i) HR-like necrosis and deposition of callose, lignin, and phenolic compounds in the cortex, at the endodermis or in vascular cylinder to ii) blockage of the vascular tissue by mucilage, probably to impair nutrient flux to the parasite (Lozano-Baena et al. 2007). Whereas application of JA to red clover had no effect on *Orobanche* infection (Kusumoto et al. 2007), several studies have shown that application of SA or its analogue, BTH, to roots or leaves of dicotyledoneous and monocotyledoneous host plants reduced parasitism by *Orobanche* and *Striga* spp., respectively (Hiraoka and Sugimoto 2008; Kusumoto et al. 2007; Müller-Stöver et al. 2005; Perez-de-Luque et al. 2004; Sauerborn et al. 2002). SA-induced defenses were visible at different steps of the interaction of *Orobanche minor* with *Trifolium pratense* (Kusumoto et al. 2007). Directed growth of parasite radicles towards SA-treated clover roots was blocked and radicle tips displayed bifurcate growth and papillate cells, suggesting that SA treatment leads to release of an inhibitory chemical by the host root. The second barrier occurred after cortical invasion by the parasite. The haustorium was stopped at the endodermis of the SA-treated host and was unable to fuse with the host vascular tissue, due to lignin-deposition in endodermal and fiber cells close to the intrusive cells of the parasite. Further evidence for the participation of SA signaling in defense against parasitic plants was provided by transcriptomics studies in which several SA-responsive genes were upregulated during attempted colonization in the rice cultivar Nipponbare, resistant to *Striga hermonthica* (Swarbrick et al. 2008), and the partially resistant sorghum cultivar Wad Ahmed (Hiraoka and Sugimoto 2008). In turn, JA-responsive genes were induced in compatible interactions of *Orobanche* spp. and their dicotyledoneous hosts (Die et al. 2007; Vieira Dos

Santos et al. 2003a and b). The same occurred in the susceptible sorghum cultivars Abu70 and Tabat during colonization by *Striga hermonthica* while, simultaneously, SA-responsive genes were repressed. JA-responsive genes were only weakly upregulated in the more resistant sorghum cultivar Wad Ahmed (Hiraoka and Sugimoto 2008). It remains an open question whether JA signaling favors susceptibility to parasitic plants (e.g., by suppression of SA signaling) or if the increased JA response is a lateral effect induced in the host root due to cell-wall disruption and wounding caused by parasite penetration. Careful genetic analyses should help to shed light on this issue.

In summary, the emerging picture indicates that SA signaling generally promotes defense against root colonizing nematodes and parasitic plants similar to the general model derived from the well-studied leaf-biotroph interactions. Our current understanding of JA signaling in root-exploitive associations is less clear, because our knowledge is, so far, limited to circumstantial evidence derived from gene expression studies (Fig. 2).

CONCLUSIONS

This review summarizes the knowledge recently gained on the role of JA and SA signaling in root interactions with mutualistic and parasitic biotrophs. It has become evident that the signaling molecules SA and JA play a role in both mutualistic and parasitic interactions, showing that mechanisms used by the plant to find a balance between defense and accommodation may share some general features. The idea that SA signaling is preferentially involved in the activation of defense against biotrophic pathogens, which was obtained from studies on leaf pathogenesis, generally holds true for root-pathogen interactions and can be extended to mutualistic symbioses. For AM and rhizobial symbioses, SA-mediated defense responses seem to fine-tune the compatible interaction by providing a checkpoint at each colonization event like a passport control of the symbiotic microorganism. While evidence exists for a role of SA in incompatible interactions, it remains to be determined whether a similar preingress defense response is in place in compatible root interactions with parasitic plants and nematodes.

The role of JA signaling in root-biotroph interaction is less uniform. JA acts at multiple steps of colonization by AM fungi and rhizobia. In some cases, such as during presymbiotic calcium signaling, JA has an inhibitory effect, at least when applied exogenously. In other cases, such as arbuscule formation, it appears to promote accommodation. The colonization process of all four biotrophic colonizers discussed in this review involves dramatic cell-wall modification and degradation. A functional link between cell-wall stress and JA signaling is emerging, and it is tempting to speculate that JA has a role in supporting these cell-wall modifications. JA signaling systemically promotes or suppresses root colonization depending on plant species, concentration, and probably temporal and spatial distribution within the tissue. Divergent results have been obtained for AM and nematode colonization with mutants defective in *coi1* and JA-biosynthesis. This suggests that COI1-independent JA signaling might exist in roots.

Our knowledge on SA and JA signaling in root interactions is still incomplete and fragmentary. In the recent past, it became evident that much can be learned from comparisons of different plant-microbe interactions. It is now time to rigorously dissect the role of SA and JA signaling in root interactions with a combination of genetics, pharmacological treatments, and live imaging. The availability of a set of biosynthetic and signaling mutants in the same plant species would be desirable to allow investigations of different root interactions in parallel.
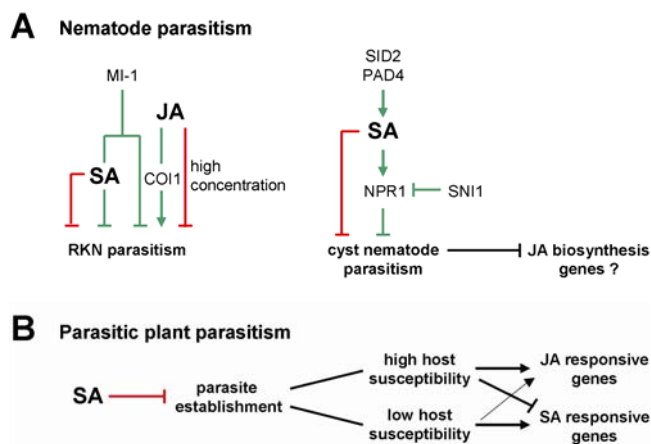

Fig. 2. A, Known impact of jasmonic acid (JA) and salicylic acid (SA) signaling on nematode parasitism. The repression of JA biosynthetic genes in cyst-nematode syncytia needs further investigation. **B,** Pharmacologically applied SA suppresses root colonization by parasitic plants. Highly susceptible host plants induce JA-responsive genes and suppress SA-responsive genes. Lowly susceptible to resistant host plants express SA-responsive genes and some express JA-responsive genes at low levels (thin black arrow). Green indicates evidence obtained via genetic approaches, and red displays evidence obtained by pharmacological application of the hormone. Black indicates that data were obtained in the wild type without pharmacological hormone application.

The model legumes *Medicago truncatula* and *Lotus japonicus*, which form symbiosis with AM fungi and rhizobia and are infected by nematodes and parasitic plants, are excellent candidates for such an approach.

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