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REVIEW

Signaling Pathways for the Biosynthesis and Action of Jasmonates

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The jasmonate family comprises lipid-derived oxidation compounds (oxylipins), which function as plant hormones to regulate diverse developmental processes and defense responses. The pleiotropic effects of jasmonates are ascribed to a variety of biologically active derivatives synthesized along different branches in the octadecanoic pathway. Jasmonate biosynthesis occurs in the first round of the signaling pathway, which is initiated by certain external signal molecules or developmental cues connected to the release of fatty acid precursors from membrane lipids. Newly synthesized jasmonate molecules then mediate the second round of that pathway, inducing the expression of related genes. In particular, certain jasmonates produced in a localized site along their biosynthetic pathway act as a long-distance signal that transmits to distal parts of the plant, eliciting an immune response against a broad spectrum of pathogens and herbivores. The jasmonate-signaling pathway is connected to other signaling pathways associated with various phytohormones, all constituting a complex regulatory network linked to ubiquitin/proteasome-mediated protein degradation of repressors that negatively regulate transcription. In this review article, we highlight the pioneering research conducted on signaling pathways for the biosynthesis and action of jasmonates.

Keywords: jasmonate, octadecanoids, oxylipins, plant hormone, signaling pathway

JASMONATE BIOSYNTHESIS

Jasmonates are a family of cyclopentanone-based compounds that function as plant hormones to regulate many developmental processes and defense responses. Jasmonic acid (JA), the free-acid form, was initially identified from a fungal culture filtrate, and has since been proven to be distributed ubiquitously in higher plants. JA is considered the primary signal transducer for wound responses based on its structural similarity to the animal anti-inflammatory prostaglandin. Methyl jasmonate (MeJA), a component of volatiles emitted from several plant species, was first discovered in the etheric oil of jasmine. It has long been recognized that higher plants release volatiles into their surroundings to make themselves attractive to specific pollinators.

The highest levels of JA and MeJA are found in flowers and reproductive tissues (Creelman and Mullet, 1995), implying roles for jasmonates in developmental processes. This has been demonstrated in studies of flower/fruit formation, pollen viability, and senescence (Creelman and Mullet, 1997). Other lines of research have also found that jasmonates serve as local or systemic signal transducers that activate the expression of defense genes in response to wounds and pathogen infection (Creelman and Rao, 2002; Farmer et al., 2003).

The many effects of jasmonates can be explained by the diversity of biologically active members in that family. Numerous biosynthetic intermediates, isomers, and derivatives are synthesized along different branches in the JA biosynthetic pathway. About 20 naturally occurring jasmonates have already been described (Gfeller and Farmer, 2004). Recent intensive studies based on their molecular biology and functional genomics have identified many important components comprising the jasmonate-mediated gene activation processes.

JA is synthesized by the octadecanoid pathway (Fig. 1). Certain stimuli activate phospholipase(s) that release linolenic acid from the membrane lipid. The linolenic acid is oxygenated by lipoxigenase (LOX) to form 13(*S*)-hydroxy linolenic acid (13-HPOT), which is then converted to 12-oxo-phytodienoic acid (OPDA) by allene oxide synthase (AOS) and allene oxide cyclase (AOC). JA is synthesized from OPDA with one step of reduction and three steps of β-oxidation.

OPDA and JA can be further transformed into various derivatives by methylation, oxidation, hydroxylation, glycosylation, or amino acid conjugation (Beale and Ward, 1998; Hause et al., 2000; Wasternack and Hause, 2002; Schaller et al., 2005). However, the functioning of several JA derivatives remains unclear, because of much contradictory data pertaining to their biological activity. Studies with gene manipulation, such as those that use overexpression or knockouts of the genes responsible for the derivative formation, have been limited by the functional redundancy and overlapping substrate-utility of many homologous methyltransferases, esterases, and glycosylases present in plant tissues.

MeJA is considered a vital regulator that mediates unique cellular responses (Cheong and Choi, 2003). However, no clear distinction has been made between JA and MeJA in many of the exogenous application experiments conducted to mimic jasmonate-mediated gene activation. The two forms of jasmonate are presumably inter-convertible by JA carboxyl methyltransferase(s) (Seo et al., 2001; Song et al., 2005) and MeJA esterase(s) (Stuhlfelder et al., 2004), constituting an equilibrium within plants. Therefore, their relative importance in each jasmonate response is poorly characterized.

Another volatile jasmonate, *cis*-jasmone, aka (Z)-jas-

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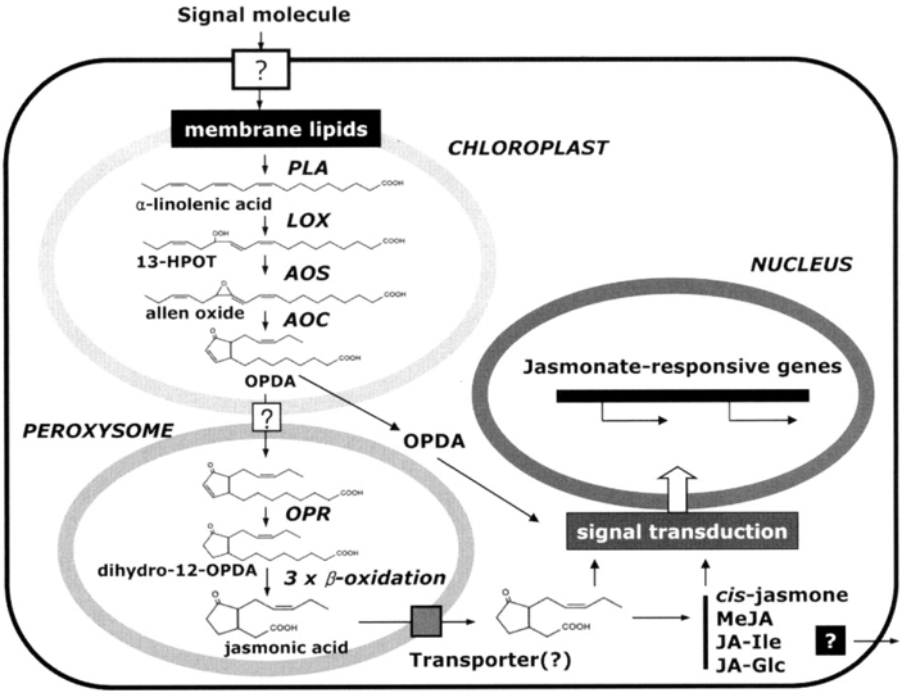

Figure 1. Main pathway of jasmonate biosynthesis. A phospholipase releases linolenic acid from membrane lipids. Linolenic acid is then oxygenated by lipoxigenase (LOX) to form 13(*S*)-hydroxy linolenic acid (13-HPOT), which is converted to 12-oxo-phytodienoic acid (OPDA) by allene oxide synthase (AOS) and allene oxide cyclase (AOC). Jasmonic acid (JA) is synthesized from OPDA through reduction and three steps of β-oxidation, and further converted to various derivatives including an unknown transmissible long distance signal molecule(s). Chloroplasts are thought to be primary site of JA biosynthesis for developmental processes. A similar pathway in the cytoplasm has been reported, but omitted in this figure.

mone, is produced by an additional round of β-oxidation of JA. This particular flower volatile is induced and accumulated in vegetative tissues when damaged, exerting a role as an insect semiochemical and in plant defense (Birkett et al., 2000). In addition, JA is metabolized to its glucose and gentiobiose esters upon uptake in tobacco culture cells, followed by hydroxylation at C-11 or C-12 (Świątek et al., 2004). Glucose ester is the most abundant metabolite of JA in tomato cell culture (Meyer et al., 1989). The 12-OH-JA (supertonic acid) plays a signaling role in tuberization and flowering (Koda, 1997; Gidda et al., 2003). As well, a considerable number of amino acid conjugates, including *N*-jasmonylisoleucine (JA-Ile), are detected in developing flowers (Hause et al., 2000). The JA adenylation activity of JAR1 is responsible for forming JA conjugates with the amino acids (Staswick et al., 2002). JA-Ile is necessary for optimal signaling in developmental processes (Staswick and Tiryaki, 2004) and for activating defense responses (Kang et al., 2006). Moreover, high levels of JA-Ile methyl ester (Hause et al., 2000) and JA-tyramine conjugate (Miersch et al., 1998) accumulate in different proportions depending on the suborgans of the flower.

In addition to the octadecanoids in the main pathway of jasmonate biosynthesis, a variety of compounds can be synthesized from α-linolenic acid, 13-HPOT, and OPDA through different branches of the pathway (Feussner and Wasternack, 2002; Howe and Schilmiller, 2002; Wasternack and Hause, 2002). Damage sustained by *Arabidopsis* leaves triggers a significant transient increase in the level of OPDA-monogalactosyl diglyceride, which occurs by esterification of plastid-specific galactolipids (Stelmach et al., 2001). Studies with dinor-OPDA also have shown that hexadecanoid jasmonates are derived directly from plastid 16:3 fatty acid, not from octadecanoid precursors (Weber et al., 1997).

Chloroplasts are considered the primary sites of JA biosynthesis in various developmental processes because the JA biosynthetic enzymes LOX, AOS, and AOC are localized within that sub-cellular organelle (Creelman and Mullet, 1997; Wasternack and Hause, 2002). However, accumulating evidence suggests that wound- or pathogen-induced JA biosynthesis occurs in the cytoplasm (Wang, 1999; Laudert et al., 2000).

The common perception is that the OPDA synthesized in the chloroplasts or cytoplasm is transported to the peroxisomes, where OPDA reduction and β-oxidation occur to produce JA. The reductase OPR3, which catalyzes the reduction of OPDA to 3-oxo-2(2′[*Z*]-pentenyl)-cyclopentane-1-octanoic acid (OPC8), contains an SRL peptide sequence at the carboxyl terminus that localizes it to the peroxisome (Stintzi and Browse, 2000). Enzymes for β-oxidation, which remove six carbons from the octanoate side chain of OPC8, are also located in that sub-cellular organelle (Li et al., 2005; Schneider et al., 2005; Koo et al., 2006; Schilmiller et al., 2007).

A portion of the OPDA remains in the cytoplasm and triggers the expression of a distinct set of genes, playing a role in wound-induced expression in *Arabidopsis* (Taki et al.,

2005). Because of their greater complexity, enzymes that modify JA into JA-derivatives are presumably cytoplasmic (Turner et al., 2002), as demonstrated with *Brassica* JMT in an immuno-localization experiment (Song et al., 2000). It is unclear how these compounds are transported between the intracellular organelles and the cytoplasm.

SIGNALING FOR JASMONATE BIOSYNTHESIS

The first step in jasmonate biosynthesis is to release fatty acid precursors from membrane lipids. DAD1, a chloroplast-located phospholipase A1 (PLA1), catalyzes the initial step of JA biosynthesis for anther and pollen development in *Arabidopsis* (Ishiguro et al., 2001). This observation is in accordance with the theory that initiation occurs in the chloroplast.

However, substantial evidence suggests that initiation of JA biosynthesis as a wound- or pathogen-induced response differs from that for developmental processes (Turner et al., 2002). Upon sustaining damage, the rate of JA induction increases by more than 100-fold in the *dad1* mutant (Ishiguro et al., 2001). This implies that other phospholipase activities, apart from DAD1, are involved in the wound-related induction of JA. In connection with this, a rapid and systemic rise in phospholipase A (PLA) activity has been observed in tomato leaves in response to wounding (Narváez-Vásquez et al., 1999). Soluble PLA_2 activity is induced before oxylipin accumulates in TMV-infected tobacco leaves (Dhondt et al., 2000). In addition, the role of phospholipase D (PLD) in wound-induced accumulation of JA has been elucidated in *Arabidopsis* (Wang et al., 2000; Bargmann and Munnik, 2006). This enzyme activation appears to result from the intracellular translocation of PLD from the cytosol to membranes, mediated by an increase in cytoplasmic Ca^{2+} concentrations.

In wound- or pathogen-induced JA biosynthesis, oligosaccharide elicitors derived from plant or fungal cell walls and peptide inducers are believed to stimulate JA biosynthesis through receptor-mediated processes (Turner et al., 2002; Schilmiller and Howe, 2005). Among the stimulatory signals implicated in the systemic response within tomato plants is systemin, an 18-amino acid peptide (Farmer and Ryan, 1992). Systemin is produced at the wound site by proteolytic cleavage of prosystemin, which induces the expression of protease inhibitor genes by activating the JA biosynthesis pathway. Systemin binds to the cell surface receptor SR160, a leucine-rich repeat receptor kinase (Scheer and Ryan, 2002). However, the signal-transduction event that couples SR160 activation at the plasma membrane to the initiation of JA biosynthesis remains to be elucidated.

Kinase activity also appears to be involved in the signal-transduction pathway for JA biosynthesis. Minutes after damage is incurred, transcription of the tobacco mitogen-activated protein (MAP) kinase gene *WIPK* is induced (Seo et al., 1995). Suppression of *WIPK* inhibits wound-induced accumulation of JA in transgenic plants, while its overexpression results in greater accumulations of JA and protease inhibitor gene (*PIN2*) transcripts (Seo et al., 1999). In *Arabidopsis*, activation of MPK4 occurs rapidly, within 2 to 5 min after wounding, without a corresponding increase in the amount of mRNA or protein (Ichimura et al., 2000), suggesting that this enzyme is activated by the primary wound signal. However, the *mpk4* mutant fails to induce jasmonate-responsive gene expression upon treatment with JA (Petersen et al., 2000). Thus, it is not clear whether wound-triggered MPK4 activation is caused by the primary wound-signal perception or as a consequence of JA biosynthesis.

LONG-DISTANCE SIGNALING BY JASMONATES

The jasmonate-insensitive *Arabidopsis* mutant *coi1* is unable to produce viable pollen, thereby rendering the male sterile (Feys et al., 1994). This indicates that jasmonate sensitivity plays a crucial role in that process. Because JA-biosynthetic genes are specifically expressed in female flower organs, such as the ovary, sepals, petals, and filaments, it is likely that a jasmonate molecule produced in one of these is transported to the pollen-developing tissue (Wasternack and Hause, 2002).

The role of jasmonates in plant-defense responses as a long-distance signaling molecule has been investigated based on the common observation that local damage induces a systemic defense reaction against a broad spectrum of pathogens and herbivores (Schilmiller and Howe, 2005; Wasternack et al., 2006). A well-characterized example is the accumulation of protease inhibitors in distal parts of wounded plants (Farmer and Ryan, 1992) and the induced systemic resistance (ISR) observed after exposure to specific biotic stimuli (Pieterse et al., 1998), which require JA and ethylene.

Reciprocal grafting experiments with wild-type tomato plants have shown that the jasmonate biosynthesis mutant *spr2* is defective in its production, but not recognition, of a graft-transmissible wound signal. In contrast, jasmonate-insensitive mutant *jai1* plants do not recognize this signal, but can produce it (Li et al., 2002). The manufacture of a transmissible wound signal is abolished in the tomato *acx1* mutant, which is defective in the peroxisomal β-oxidation stage of JA biosynthesis, whereas signal recognition in undamaged, distal leaves is unaffected (Li et al., 2005). These data indicate that JA or one of its derivatives is the transmissible wound signal, and that JA biosynthesis is not necessary for the response to the mobile signal. Indeed, JA accumulates locally and not systemically in the leaves of wounded tomato plants, suggesting that local and systemic tissues undergo distinct signaling events (Strassner et al., 2002). The identity of that transmissible wound signal has yet to be defined.

Free-acid JA, a strong candidate for the transmissible signal molecule, may be unable to cross the cellular membrane without a carrier due to its acidic nature. In this regard, lipid transfer proteins (LPTs) are believed to be capable of loading and transferring lipid-derived hydrophobic molecules, thereby promoting long-distance signaling. The apoplastic LPT-defective *Arabidopsis dir1-1* mutant exhibits normal local resistance, yet fails to acquire systemic resistance to virulent pathogens (Maldonado et al., 2002). Inter-

estingly, tobacco LPT1 has been shown to load JA (Buhot et al., 2004). That study has revealed that the LPT1-JA complex forms a strong interaction with the plasma membrane receptors and provides enhanced long-distance protection against fungal pathogens, which is not observed when plants are treated only with LPT1 or JA.

MeJA is also a potential candidate for the role of systemic signal molecule because this gaseous compound can diffuse to distal regions of the plant through the vapor phase (Farmer and Ryan, 1990; Franceschi and Grimes, 1991; Karban et al., 2000). Such migration appears less probable, however, because the wound-induced JA burst in *Nicotiana attenuata*, a wild tobacco species, has no association with the release of significant quantities of MeJA into the headspace (von Dahl and Baldwin, 2004). Instead, it has been speculated that intercellular migration of MeJA occurs through the phloem (Cheong and Choi, 2003). Certain signal(s) generated during an early event in the developmental processes or defense responses initiate the activation of *JMT*, which subsequently produces MeJA (Seo et al., 2001). Although MeJA may diffuse through the phloem to activate jasmonate biosynthesis genes throughout the entire plant, little direct evidence is available to support this theory.

Systemin has been implicated as the long-distance transmissible signal in the systemic wound response (Farmer and Ryan, 1992). However, it is now generally accepted that systemin acts at the site of damage as a local mediator of jasmonate synthesis, amplifying the production of a JA-based mobile signal (Ryan and Moura, 2002; Schilmiller and Howe, 2005). Grafting experiments have demonstrated that prosystemin-overexpressing (*35S-Prosys*) transgenic tomato plants constitutively generate a graft-transmissible signal in the absence of wounding (McGurl et al., 1994). Recognition of the *35S-Prosys*-derived signal in responsive leaves requires the capacity to sense, but not synthesize, an octadecanoid pathway signal (Li et al., 2002). The tomato *spr1* mutant, which is defective in its systemin perception, impairs the wound-induced accumulation of JA and the systemic expression of protease inhibitor genes (Lee and Howe, 2003). Grafting with wild-type plants has shown that *spr1* impedes the systemic expression of protease inhibitor genes by blocking the production of those long-distance wound signals in damaged leaves, rather than by inhibiting the systemic recognition of that signal in undamaged leaves.

JASMONATE-MEDIATED SIGNALING

Exogenous application of JA or MeJA to plants alters a wide range of cellular metabolic activities, including the up- or down-regulation of expression in various genes that mediate jasmonate-responsive cellular metabolism (Wasternack and Hause, 2002). Recently developed DNA microarray technology has facilitated the massive screening of jasmonate-inducible genes (Sasaki et al., 2001; Jung et al., 2007a). Genes up-regulated by MeJA-treatment include those for jasmonate biosynthesis, defense proteins, stress-protective proteins, and cell wall formation. In contrast, genes involved in photosynthesis, such as ribulose bisphosphate carboxylase/oxygenase (Rubisco), chlorophyll a/b-binding protein, and light-harvesting complex II, are down-regulated. Transgenic *Arabidopsis* producing an excess of endogenous MeJA constitutively expresses numerous jasmonate-responsive genes in the absence of wounding or jasmonate treatment (Seo et al., 2001; Jung et al., 2007b).

Characterization of mutants impaired in their jasmonate-mediated responses has provided considerable insight into the complexity of that signaling cascade (Berger, 2002; Turner et al., 2002). In particular, the mechanism for jasmonate perception has been investigated with several mutants that are insensitive to JA treatment, including *Arabidopsis coi1* (Feys et al., 1994). The *COI1* gene encodes a protein containing an F-box motif (Xie et al., 1998), which functions as a receptor that selectively recruits regulatory proteins as substrates for ubiquitination. Its gene product forms a functional E3-type ubiquitin ligase complex known as SCF (Xu et al., 2002).

Ligand(s) that bind to COI1 are believed to regulate jasmonate-responsive gene activation, although that mechanism is undefined. As hypothesized (Creelman and Rao, 2002), a jasmonate may activate a protein kinase to phosphorylate a signal messenger protein that binds to COI1. Alternatively, COI1 could be the JA receptor itself (Lorenzo and Solano, 2005), when comparisons are drawn to the auxin-receptor F-box protein TIR1 (Dharmasiri et al., 2005; Kepinski and Leyser, 2005).

Constitutive repression, therefore, negatively controls the jasmonate-signaling pathway, which is activated by repressor(s) degradation (Fig. 2). Histone deacetylases have been proposed as possible substrates for the COI1-associated SCF complex (Devoto et al., 2002; Zhou et al., 2005). In addition, the *cos1* mutant has been identified as a suppressor of *coi1* that restores some JA-regulated responses, suggesting that COS1 also acts downstream of COI1 in the JA-signaling pathway (Xiao et al., 2004). The *COS1* gene encodes lumazine synthase, a key component in riboflavin biosynthesis that is essential for several cellular processes. The COP9 signalosome, a nucleus-enriched multiprotein complex, physically interacts with SCF^{COI1} to modulate JA-responsive gene expression (Feng et al., 2003). This interaction may act as a negative control mechanism for tight regulation, as observed in the COP9-SCF^{TIR} interaction for the auxin-signaling pathway (Schwechheimer et al., 2001).

Auxin-mediated gene transcription occurs by promoting the degradation of a family of transcription repressors (Badescu and Napier, 2006; Parry and Estelle, 2006). This concept is also applicable to the JA-mediated signaling pathway, in which a number of transcription factor(s) operate on the promoter regions of jasmonate-responsive genes. Lorenzo et al. (2003) have reported that the AP-2 domain transcription factor ERF1 in *Arabidopsis* integrates signals from the ethylene and jasmonate pathways to aid in plant defense. Transcription factors WRKY70, WRKY11, and WRKY17 negatively regulate the expression of JA-induced genes (Li et al., 2004; Journot-Catalino et al., 2006). The *Arabidopsis BOS1* (*BOTRYTIS-SUSCEPTIBLE1*) gene encodes an R2R3MYB transcription factor that mediates the response to both biotic and abiotic stress signals

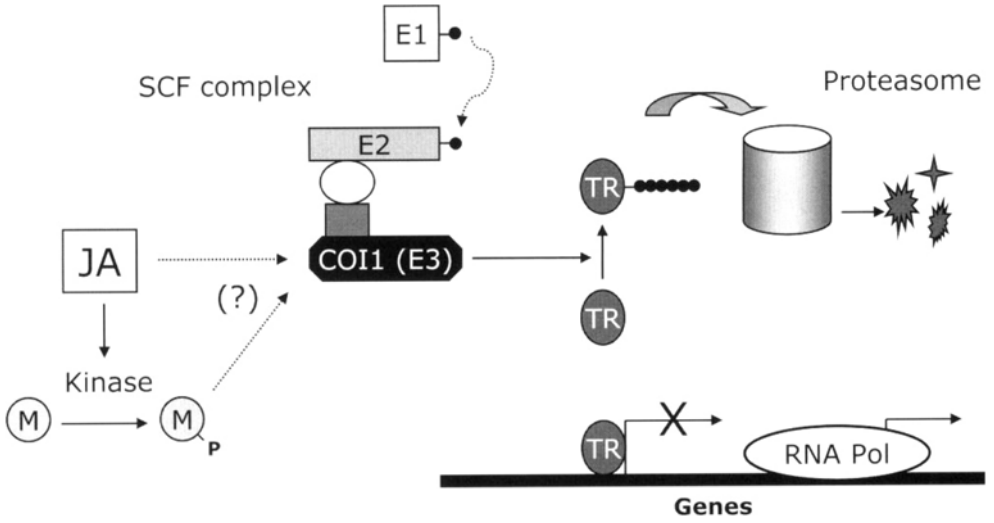

Figure 2. Hypothetical model for jasmonate-mediated signaling pathway, showing function of COI1 in ubiquitination/proteasome-mediated control of gene expression. Jasmonate may activate protein kinase to phosphorylate signal messenger protein (M) that binds to COI1. Alternatively, jasmonate itself may directly bind to COI1. An SCF complex, which includes COI1, acts as ubiquitin ligase, transferring ubiquitin(s) to negative-acting transcription repressor(s) (TR). After polyubiquitination, transcription repressor is degraded by proteasomes and, thus, unable to act on promoter region. Instead, RNA polymerase binds to promoter, activating transcription of jasmonate-responsive gene.

(Mengiste et al., 2003). The JA-related MYC class of transcription factors, a group of basic helix-loop-helix (bHLH) proteins, has also been found in *Arabidopsis* (Lorenzo et al., 2004) and tomato (Boter et al., 2004). *Arabidopsis* MYC2, aka JIN1 (JASMONATE-INSENSITIVE1), activates wound-response genes (Lorenzo et al., 2004), while also repressing blue-light-mediated photomorphogenic growth (Yadav et al., 2005). Further investigation is needed to determine if the transcription factors described above are the primary target transcriptional repressors of SCF^{COI1} and, if so, what the mechanisms are for the interaction between proteins.

INTERACTIONS WITH OTHER SIGNALING PATHWAYS

Jasmonates act either synergistically or antagonistically with other plant regulators, such as ethylene, light, auxin, salicylic acid, and abscisic acid. Several molecules, including kinases and transcription factors, play an integral role in the crosstalk between hormonal signaling pathways (Fujita et al., 2006).

The prototypical examples for such interactions are the signaling pathways induced by ethylene and JA, which cooperate to concurrently activate JA/ethylene-dependent genes (Wang et al., 2002). Treating either jasmonate- (*coi1*) or ethylene- (*ein2*) signaling mutants with MeJA or ethylene fails to induce the plant defensin gene *PDF1.2* (Penninckx et al., 1998). This suggests that concomitant activation of both pathways is required for this gene expression. As previously mentioned, the transcription factor ERF1 integrates signals from the two signaling pathways in plant defense responses (Berrocal-Lobo et al., 2002; Lorenzo et al., 2003).

JAR1 has been identified as a JA-amino synthetase essential for optimal signaling in some jasmonate responses in *Arabidopsis*. In addition, JAR1 mediates the conjugation of JA with 1-aminocyclopropane-1-carboxylic acid (ACC), the ethylene precursor found in *Arabidopsis* leaves (Staswick and Tiryaki, 2004), providing a mechanism that regulates the availability of both JA and ethylene. The *Arabidopsis* mutant *cev1*, which is defective in a cellulose synthase, constitutively produces jasmonates and ethylene while exhibiting enhanced resistance to pathogens (Ellis and Turner, 2001; Ellis et al., 2002a). It is interesting that inhibition of cell wall synthesis activates jasmonate- and ethylene-dependent stress responses.

However, the combined effects of jasmonate and ethylene are antagonistic in the activation of wound-response genes. In *Arabidopsis*, MYC2 (JIN1) represses the expression of defense genes while activating wound-response genes (Rojo et al., 1999), thus distinguishing between the two different jasmonate-regulated responses, which are opposite to ERF1 (Lorenzo et al., 2004). MYC2 also negatively regulates the blue-light signaling pathway (Yadav et al., 2005), constituting a point of crosstalk between the JA and light pathways. This interaction has also been attributed to the COP9 signalosome, because transgenic plants with reduced levels of this protein complex have reduced JA responses (Feng et al., 2003).

The relationship between the jasmonate and auxin signaling pathways has been defined in the auxin-response mutant *axr1*, which is defective in its jasmonate response (Tiryaki and Staswick, 2002). Furthermore, the *Arabidopsis* gene responsible for jasmonate-response mutant *jar1* is similar to the auxin-induced soybean *GH3* gene (Staswick et al., 2002). As first described, JAR1 is an acyl adenylate-forming enzyme that specifically targets JA to form conjugates with several amino acids (Staswick et al., 2002; Staswick and Tiryaki, 2004).

Many studies have shown that jasmonates and abscisic acid (ABA) antagonistically regulate the expression of stress-

inducible proteins, as observed for proteins associated with water deficiency or the defense response in rice roots (Moons et al., 1997). The mutual, antagonistic interaction between the ABA and JA/ethylene signaling pathways has also been found with defense-gene expression and disease resistance (Anderson et al., 2004). Recent microarray analyses have shown that external treatment (Jung et al., 2007a) or endogenous overproduction of MeJA (Jung et al., 2007b) significantly reduces the transcription of abscisic acid-responsive cold/drought-stress genes in *Arabidopsis*.

The interaction between JA and salicylic acid (SA) also is considered oppositional (Kunkel and Brooks, 2002; Lorenzo and Solano, 2005). External application or genetic manipulation of the signaling pathways for either phytohormone reveals mutual, antagonistic effects on gene expression and plant phenotypes. MPK4 appears to regulate such behavior between JA and SA. This MAP kinase is required for the induction of jasmonate-responsive genes, promoting the activation and response to JA, in addition to repressing SA biosynthesis (Petersen et al., 2000; Brodersen et al., 2006). NPR1 also modulates this negative interaction, as observed in the induction of SA biosynthesis and the reduction in JA accumulation during the plant defense response (Dong, 2004; Pieterse and van Loon, 2004). NPR1 interacts with the TGA family of transcription factors in the nucleus to activate SA-responsive genes (Després et al., 2000; Fan and Dong, 2002). A novel function of this protein in the cytosol appears to have a negative impact on JA-signaling (Spoel et al., 2003). An *Arabidopsis NPR1*-like gene, *NPR4*, is integral to the expression of the JA-induced defensin gene *PDF1.2*, and may be implicated in the crosstalk between the SA- and JA-dependent signaling pathways (Liu et al., 2005). The WRKY70 transcription factor is speculated to function as a switch between those two pathways, enhancing the expression of SA-responsive genes and suppressing that of JA-induced genes (Li et al., 2004, 2006). Furthermore, physical damage or MeJA treatment results in the activation of the salicylate carboxyl methyltransferase (SAMT) gene, which converts SA to MeSA that is inactive for SA-dependent gene expression (Chen et al., 2003; Koo et al., 2007). This event may provide an additional mechanism for the antagonism between JA- and SA-signaling.

In parallel, however, there is substantial evidence supporting a positive relationship between JA and SA (Grant and Lamb, 2006). As reported, the combination of MeJA and SA synergistically induces pathogenesis-related (PR) proteins (Xu et al., 1994). Microarray analysis has also revealed that more than 50 defense-related genes are coordinately induced by JA and SA in *Arabidopsis* (Schenk et al., 2000). More specifically, simultaneous activation of the salicylate- (SAR) and jasmonate-dependent (ISR) defense pathways by microbial infection leads to the additive enhancement of induced disease resistance in *Arabidopsis* (van Wees et al., 2000). Likewise, co-treatment with low concentrations (10 to 100 μM) of JA and SA in tobacco and *Arabidopsis* enhances the expression of genes associated with either JA- or SA-signaling (Mur et al., 2006). The synergistic influences on gene expression and plant stress are NPR1- and COI1-dependent, respectively. In that study, various antagonistic effects were observed after prolonged treatment times or with higher concentrations, eliciting the generation of reactive oxygen species (ROS) and inducing cell death. Therefore, the synergistic/antagonistic mechanisms employed there appeared to depend on the relative concentrations of the hormones JA and SA, as well as the involvement of other phytohormones, e.g., ethylene and ROS. To this end, the antagonism observed in experiments using external treatment or genetic manipulation might have been due to the determination of cell fate (i.e., which signaling pathway is activated), by controlling MPK4 or NPR1 activity, rather than via true crosstalk between the two signaling pathways.

The interactions of the JA signaling pathway are not limited to those with phytohormone signaling pathways. In practice, several signaling events mediated by systemin, oligogalacturonides, or electric pulse might be simultaneously evoked during wound responses (Leün et al., 2001). Similarly, a variety of elicitors, including oligosaccharides, peptides, and (glyco)proteins, are generated during microbial attacks, which activate their own signaling pathways (Hammond-Kosack and Jones, 1996). Most importantly, JA accumulation does not sufficiently explain the activation of defense and wound responses. As one line of evidence, transgenic potato plants constitutively expressing *AOS* cDNA may contain an increased level of JA, but transcription of the jasmonate-responsive gene is not enhanced in those plants (Harms et al., 1995).

Therefore, depending on the stimulus, a specific elicitor may trigger a signaling pathway that interacts with the jasmonate-mediated signaling pathway, ultimately activating unique cellular metabolism. Reinforcing this theory, differential gene expression (Reymond et al., 2000) and volatile emissions (Paré and Tumlinson, 1999) are observed in response to mechanical wounding and insect feeding. For instance, an elicitor of plant volatiles, volicitin, is formed in response to certain herbivore attacks (Alborn et al., 1997). Furthermore, crosstalk among JA-, ethylene-, and Nod factor- (a lipid-bound oligosaccharide) signaling rapidly integrates diverse inputs for the regulation of nodulation (Sun et al., 2006), thereby supporting the proposed existence of a multiplex interaction.

CONCLUDING REMARKS

The jasmonate family includes numerous biologically active members, which helps explain their pleiotropic effects. Ongoing research is focused on the signal-transduction pathway and regulatory mechanism for the biosynthesis of each jasmonate. Recent approaches using functional genomics and bioinformatics have identified a whole set of MeJA-responsive genes, thereby providing insight into how plants use jasmonate signals to adapt to diverse environments. Connection maps that describe jasmonate signal-transduction elements are maintained at the Signal Transduction Knowledge Environment (STKE; Gfeller and Farmer, 2004). These contribute to our understanding of the jasmonate signaling pathway.

Another aspect of their functional diversity is demonstrated by the complex regulatory networks in which jasmonates and other signaling pathways work together

(Bostock, 2005; Fujita et al., 2006). In this vein, it is noteworthy that ubiquitin/proteasome-mediated protein degradation is common to many cellular metabolic systems (Devoto et al., 2003; Zeng et al., 2006; Chow and McCourt, 2007). Plant cellular metabolism requires a negative control system that includes responsiveness to light, sucrose, pathogens, and hormones such as jasmonate, auxin, and abscisic acid (Hellmann and Estelle, 2002; Ellis et al., 2002b; Lopez-Molina et al., 2003). Cellular components involved in the ubiquitination process have been estimated to comprise about 5% of all total proteins, as predicted from the genome sequence of *Arabidopsis* (Capron et al., 2003; Mladek et al., 2003). Therefore, to gain a complete understanding of jasmonate-signaling, one must consider it as part of a complex plant-signaling network. Currently, comprehensive functional genomics are providing a framework for such a network that controls the cellular metabolism of the entire plant.

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