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Jasmonates and Related Compounds in Plant-Insect Interactions

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ABSTRACT

Herbivore attack elicits defense responses in host plants by a complex chain of events that starts with the introduction of herbivore-specific elicitors into the wounds at the feeding or oviposition site, their recognition by the plant, and activation of several signaling cascades that trigger defense responses that finally increase resistance. Oxylipin signaling plays a central role in the activation of these herbivore-induced responses. Wounding activates some but not all of these defense responses, but herbivore attack frequently amplifies the oxylipin responses well beyond that elicited by wounding alone, suggesting recognition of herbivore attack. In addition to their signaling role within the plant, oxylipins can also directly influence the performance of herbivores or attract natural enemies to feeding herbivores. Here we review the literature on the regulation and function of herbivore-specific oxylipin signaling and the direct effects of oxylipins on herbivore performance.

Key words: jasmonates; oxylipin signaling; herbivore-induced response; wounding

OXYLIPIN SIGNALS INVOLVED IN PLANT-HERBIVORE INTERACTIONS

The activation of herbivore- and wound-induced defense responses involves a complex network of plant signaling cascades including peptide signals, such as systemin (Ryan and Pearce 2003; Ryan and others 2002) and phytohormones, such as salicylic acid, ethylene, H_2O_2, and lipid-derived oxylipins. Responses specific to the attack by particular herbivore species likely result from extensive crosstalk among the individual cascades, which includes antagonistic as well as synergistic interactions (Pieterse and others 2001; Pozo and others this issue; Rojo and others 2003; Walling 2000). Jasmonic acid (JA), a linolenic acid-derived oxylipin produced by the octadecanoid pathway, its biosynthetic precursor, 12-oxo-phytodienoic acid (OPDA), and derivatives such as its methyl ester (MeJA) or amino acid conjugates, collectively called jasmonates (JAs), represent the best characterized class of signals mediating the elicitation of defense responses to wounding and herbivory (Beale and Ward 1998; Blee 1998; Creelman and Mullet 1997; Devoto and Turner 2003; Farmer and others 2003). JAs produced by the octadecanoid pathway are not the only oxylipin signals involved in defense activation (Blee 2002), and the development of oxylipin profiling techniques in combination with an increasing number of mutants and transgenic plants impaired in oxylipin biosynthesis or perception are facilitating the identification and characterization of additional signaling compounds. Here we review the recent progress in understanding the herbivore-

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specific regulation of oxylipin biosynthesis and the function of individual oxylipin compounds in plant-insect interaction. The literature reporting effects of treatments with synthetic oxylipins is increasing (Mithöfer and others this issue) but herbivore-induced responses are rarely faithfully mimicked by exogenous treatments of signal compounds (Baldwin 1990). Therefore, in this review, we highlight recent studies that use genetically modified plants to manipulate the production of components of the signaling cascades *in planta* and examine their consequences for plant-herbivore interactions. In addition, we review the role of oxylipins in higher-order interactions including signaling among plants and their direct effects on insects that feed on plants.

HERBIVORE-SPECIFIC ELICITATION OF OXYLIPIN SIGNALING

In all species where it has been examined, wounding results in a transient accumulation of JAs (Howe this issue) and a release of green leaf volatiles (GLVs) (Sembdner and Parthier 1993). In the native tobacco species *Nicotiana sylvestris* (Baldwin and others 1997) and *N. attenuata* (Schittko and others 2000; Ziegler and others 2001) and in corn (*Zea maize*; Schmelz and others 2003), attack by adapted herbivores amplifies the wound-induced JA accumulation (JA burst), which is elicited when oral secretions of the feeding larvae are introduced into wounds during feeding. Oral secretions contain a complex cocktail of potential elicitors, but only a suite of fatty acid-amino acid conjugates (FACs) has been demonstrated to be necessary and sufficient to elicit the JA burst and several JA-dependent defense responses (Halitschke and others 2001, 2003; Roda and others 2004; Schmelz and others 2003). In addition to eliciting the JA-burst, the herbivore's oral secretions are also known to amplify the wound-induced release of GLVs from *N. attenuata* (Halitschke and others 2004). Although the elicitors are known, the mechanisms responsible for these amplifications are not yet understood.

It is clear, however, that the amplification of metabolite accumulation is not under transcriptional control, because wound- and herbivore-induced increases in the release or accumulation of both JA and GLVs precede increases in gene expression of oxylipin biosynthetic genes. This disconnect between gene expression and product formation has been demonstrated in *N. attenuata* for both the wound- and herbivore-induced accumulation of allene oxide synthase (AOS) and hydroperoxide lyase (HPL) transcripts which are slower than the more rapid and highly transient increase in JA and HPL-derived GLVs, respectively, (Halitschke and others 2004; Ziegler and others 2001). Both products are likely regulated by the flux of fatty acid or fatty acid hydroperoxide substrates which are subsequently metabolized by the constitutive HPL or AOS activity found in unwounded leaf tissues (Vancanneyt and others 2001; Ziegler and others 2001).

The first biosynthetic step in oxylipin formation, for example, the production of JA and GLV, is catalyzed by lipoxygenases (LOX) which comprise a multi-gene family with distinct positional (9- or 13-LOX) and pathway specificity, tissue-specific gene expression and organelle-specific localization (Porta and Rocha-Sosa 2002; Feussner and Wasternack 2002). A specific 13 -LOX (LOX-H1) in potato supplies the substrate for the HPL reaction required for the release of GLVs, but does not supply the hydroperoxide substrate for the octadecanoid pathway (Leon and others 2002), and in *N. attenuata* the wound- and herbivore-elicited increase in JA accumulation is dependent on the expression of a different homolog (NaLOX3). Silencing of NaLOX3 expression did not affect the wound-induced release of GLVs (Halitschke and Baldwin 2003) which demonstrates that distinct LOX isoforms supply fatty acid hydroperoxide substrates to the JA and GLV pathways. Both isoforms possess typical chloroplast transit peptides and 13-LOX products could be detected in the stroma and envelope fraction (Bachmann and others 2002). The localization of specific LOXs would further clarify their role in supplying substrates for the AOS and HPL enzymes which are targeted to different membranes of the chloroplast envelope (Froehlich and others 2001).

Interestingly, the pathway-specific supply of fatty acid hydroperoxides by distinct LOXs (Halitschke and others 2004; Leon and others 2002) does not prevent "substrate crosstalk" between the AOS and HPL cascades in *N. attenuata*. Antisense expression-mediated silencing of HPL activity reduced the release of wound-induced GLVs and increased the wound- and herbivore-induced JA burst. Conversely, silencing of AOS activity resulted in reduced JA accumulation and was accompanied by an increase in the release of HPL-derived GLVs (Halitschke and others 2004). This substrate crosstalk could serve as a regulatory mechanism to amplify (or suppress) individual components of the complex oxylipin signature during attack from specific herbivores.

OXYLIPIN-MEDIATED HERBIVORE RESISTANCE

Most of the evidence for the involvement of oxylipin signals in plant-herbivore interactions comes from two types of studies: i) those establishing correlations between oxylipin accumulation (or release) and the expression of defensive traits during an episode of herbivory or wounding and ii) those that use synthetic compounds applied at more or less realistic concentrations to elicit changes in the plant-herbivore interaction. Interestingly, despite the availability of a large number of mutants and transgenic plants, their potential for elucidating the mechanisms of herbivore resistance elicitation is only recently being realized (Berger 2002; Kessler and Baldwin 2002).

The majority of these mutants are of *Arabidopsis* and recent studies have examined the role of JA and GLVs in herbivore resistance. The *Arabidopsis thaliana fad3 fad7 fad8* triple mutant, impaired in wound-induced JA accumulation, has reduced resistance against the fungal gnat *Bradysia impatiens* (McConn and others 1997; Stintzi and others 2001), which can be a pest of *Arabidopsis* in glasshouses. The resistance could be recovered by JA applications, but not by the C_6 products of the HPL cascade, demonstrating that resistance was dependent on the octadecanoid pathway. In contrast, resistance against *Spodoptera exigua* larvae was not reduced in the JA-deficient *fad* mutant, but surprisingly, treatment with JAs increased resistance (Cipollini and others 2004). Analysis of another mutant further downstream in JA biosynthesis, OPDA reductase (*opr3*), which is deficient in the conversion of OPDA to JA, demonstrated that these last steps in the biosynthesis of JA are not necessary for the elicitation of resistance (Stintzi and others 2001) and suggests a signal function for OPDA or other earlier intermediates of the octadecanoid pathway. The availability of additional JA-biosynthesis mutants (Turner and others 2002) lacking, for example, AOS activity (Park and others 2002; von Malek and others 2002) will facilitate the identification of those biosynthetic steps of the cascade essential in eliciting resistance against particular herbivores. For example, the JA insensitive mutant, *jar1* was found to be more resistant than wild type plants to attack from *S. exigua* (Cipollini and others 2004). The recent identification of JAR1 as a JA-amino acid conjugate synthetase, which activates JA for conjugation to isoleucine (Staswick and Tiryaki 2004), demonstrates that JA-Ile and the activation of JA is not necessary for the elicitation of herbivore resistance in *Arabidopsis*.

Although the JA-biosynthesis mutants have clearly demonstrated the importance of oxylipins in eliciting resistance in feeding assays, the direct defense traits that are responsible for the observed resistance are not well understood in *Arabidopsis*. For example, although the activity of defensive proteins [for example, polyphenol oxidase (PPO) and proteinase inhibitor (PI)] and the accumulation of glucosinolates were increased by JA treatment, no reduction of these putative direct resistance traits was observed in the JA biosynthesis (*fad*) or response (*jar1*) mutants, and induced expression of these putative defense traits did not correlate with the performance of feeding *S. exigua* larvae. Similarly, the elicitation of an indirect defense, the attraction of the parasitoid wasp *Cotesia rubecula* by volatile organic compounds (VOCs) released from *Pieris rapae* larvae-attacked *Arabidopsis* plants, was found to be JAR1-independent (van Poecke and Dicke 2003). However, silencing JA biosynthesis at an earlier step, namely, at the lipoxygenase reaction required for the hydroperoxide supply (Bell and others 1995), effectively reduced the attractiveness of attacked plants for *Cotesia* wasps, demonstrating that the elicitation of this indirect defense is also oxylipin-dependent (van Poecke and Dicke 2002).

In addition to mutants impaired in JA biosynthesis, mutants impaired in downstream signaling components involved in JA perception and signal transduction have been examined for effects on plant-herbivore interactions. The best characterized signal perception mutant is the *coi1* mutant (Feys and others 1994), which is deficient in a F-box protein of an E3-type ubiquitin ligase complex that targets proteins for degradation by the ubiquitin proteasome (Devoto and others 2002; Xie and others 1998; Xu and others 2002). This JA- and OPDA-insensitive mutant is impaired in its resistance to *B. impatiens* and *Spodoptera littoralis* larvae in a degree similar to that of the JA-deficient *fad* triple mutant (Stintzi and others 2001; Stotz and others 2002). The identification of protein targets of this SCF^{COI1} complex, which are likely transcription factors that negatively regulate COI1-dependent JA responses, would facilitate the separation of the resistance-mediating functions of JA signaling from JA's effects on pollen development and male function (Feys and others 1994), which currently limits the value of this mutant for ecological research.

With the notable exception of the identification of VOC emissions as an indirect defense, the analysis of JA-induced defense responses in *Arabidopsis* has been largely limited to transcript profiling of defense-related genes; the metabolites or proteins that determine herbivore performance remain to be discovered. These resistance traits have been more thoroughly studied in other plant species, and it is likely that advances in our understanding of the role of JA signaling in herbivore resistance will come from taxa in which the traits responsible for herbivore resistance are better understood.

In addition to indirect defense mechanisms, several direct defense responses, including antidigestive proteins and toxic alkaloids, are well characterized in several solanecaous plants including tomato, potato, and tobacco. Genetic approaches that directly manipulate the expression of individual traits powerfully demonstrate their resistance-mediating function and an increasing number of transgenic plants and mutants impaired in oxylipin biosynthesis or perception shed new light on the importance of individual signaling components and their regulation in response to herbivory.

JA deficiency of the tomato *def1* mutant abolishes the elicitation of indirect (for example, VOC release) and direct (for example, PI and PPO activity) defense traits (Howe and others 1996; Thaler and others 2002) and increases the susceptibility of this mutant to a range of herbivores: the chewing *Manduca sexta* (Howe and others 1996) and *S. exigua* (Thaler and others 2002) larvae and the spider mite *Tetranychus urticae* (Li and others 2002a). When *def1* plants are treated with synthetic JAs, resistance is recovered suggesting that the mutant harbors a defect between the LOX and OPR reactions (Howe and others 1996). However, because the *def1* mutation is not in the genes coding for either AOS and AOC (Li and others 2002a), its function in JA biosynthesis remains to be understood. The mechanism responsible for JA deficiency in a second JA biosynthesis mutant of tomato has been recently identified. The *spr2* mutant lacks the fatty acid desaturase activity required for the biosynthesis of the JA precursor, linolenic acid, and is more susceptible to attack by *M. sexta* larvae (Li and others 2003). Increased larval performance is associated with a suppression of elicited PIs during larval attack in *spr2* plants. Recently, the tomato homolog of COI1 has been identified and this mutant (JA-insensitive *jai1*; Li and others 2001) lacks JA-elicited PI and PPO activity and glandular trichrome development and is susceptible to spider mite infestation (Li and others 2004). The silencing of a specific lipoxygenase (LOX-H3) in potato by antisense expression abolished the elicitation of PIs and enhanced the performance of Colorado potato beetle *Leptinotarsa decemlineata* and *S. exigua* larvae (Royo and others 1999). However, resistance of these antisense lines could not be recovered by JA treatment, and no reduction in wound-induced JA accumulation was observed, so the role of oxylipins in herbivore resistance in these lines remains unclear.

In contrast, in the native tobacco plant, *N. attenuata*, silencing of NaLOX3, the *N. attenuata* homolog of potato LOX-H3, reduced the wound- and herbivore-induced JA accumulation. This JA-deficiency inhibited direct (for example, PI and nicotine accumulation) and indirect (volatile release) defense elicitation and resulted in reduced resistance against attack from *M. sexta* larvae (Halitschke and Baldwin 2003) and made the plants susceptible to two new herbivores, the leaf-chewing beetle *Diabrotica undecimpunctata* and the piercing-sucking leafhopper *Empoasca* spp. (Kessler and others 2004). The resistance of wild-type *N. attenuata* plants against the leaf beetle is most likely mediated by the accumulation of the toxic alkaloid nicotine, because transgenic plants silenced in putrescine *N*-methyl transferase activity, a key step in nicotine biosynthesis, are similarly susceptible (Steppuhn and others 2004). Interestingly, treatment of NaLOX3 silenced plants with synthetic JA fully recovered the nicotine response but only partially restored the PI response (Halitschke and Baldwin 2003), suggesting that an additional NaLOX3-mediated signal in addition to JA is required for PI elicitation. This requirement for an additional LOX-mediated signal for PI elicitation is in agreement with the proposal from Sivasankar and others for an HPL-derived GLV signal upstream of systemin and JA signaling required for the elicitation of PIs in tomato (Sivasankar and others 2000). Such an HPL-derived signal may also explain the phenotype of the LOX-H3 silenced potato plants (Royo and others 1999).

Although the biosynthesis of GLVs has been thoroughly analyzed in a variety of plants due to their importance for food quality and flavor (Grechkin 2002; Hatanaka 1996; Noordermeer and others 2001), the genetic manipulation of GLV release has only been performed in potato (Leon and others 2002; Vancanneyt and others 2001) and native tobacco (Halitschke and others 2004). Silencing of HPL activity suppressed the wound-induced release of GLVs in both species. In potato this was associated with increased susceptibility to attack from the aphid *Myzus nicotianae*, but this reduced resistance was not associated with alterations in defense gene (including PIs) expression (Vancanneyt and others 2001). In the native tobacco, *M. sexta* larval development was slower on GLV-deficient than on wild-type plants but this

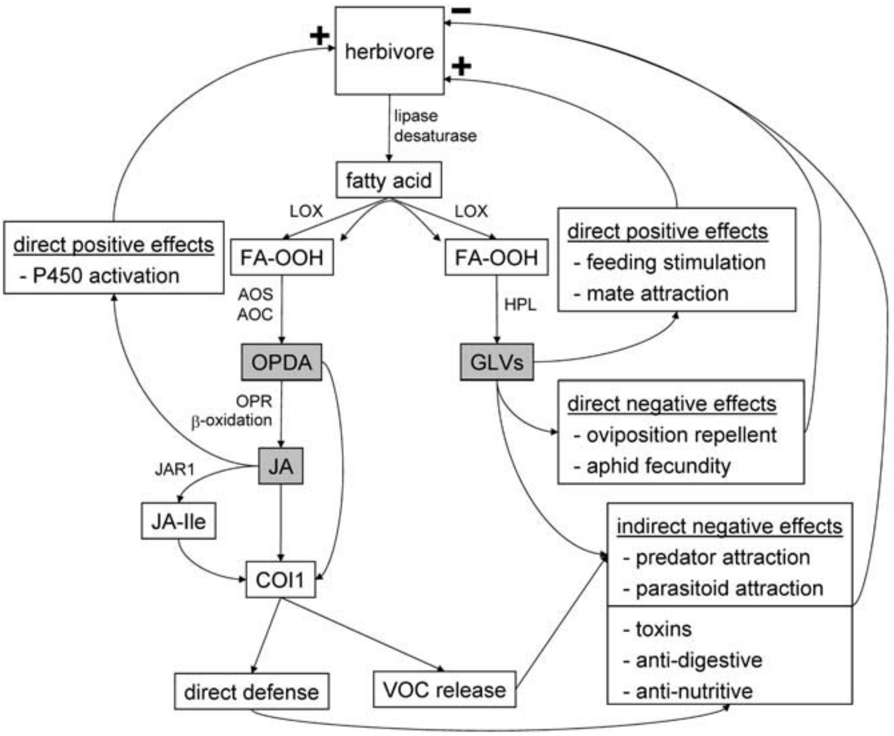

Figure 1.

decrease in larval performance was not associated with increases in direct (nicotine, PI) and indirect (VOC emissions) defense traits (Halitschke and others 2004), also suggesting that GLVs were not functioning as intra-plant signals eliciting induced resistance. Additional experiments with these plants revealed that HPL-derived GLVs play an additional signaling role in interactions outside the plant.

FUNCTIONS OF OXYLIPINS OUTSIDE THE PLANT

Once a biologically active compound is released from a plant, it can play a number of complicated roles in plant-herbivore interactions. Some GLVs, (*E*)-2-hexenal in particular, have been shown to directly inhibit aphid population growth by reducing aphid fecundity (Hildebrand and others 1993; Lyr and Banasiak 1983) and may account for the HPL-dependent resistance of potato plants to aphids discussed above (Vancanneyt and others 2001). Additional extra-plant signaling functions of GLVs include their role in mediating indirect defenses. In tobacco, GLVs attract predators of *M. sexta* larvae (Kessler and Baldwin 2001) and repel ovipositing *Heliothis virescens* moths (DeMoraes and others 2001), thereby effectively reducing herbivory rates.

Oxylipins play numerous other signaling roles in interactions outside the plant and the availability of mutants is again facilitating their discovery. The reduced performance of *M. sexta* larvae feeding on HPL-depleted plants was simply due to the fact that they ate less leaf material. This reduced leaf consumption could be restored by treating HPL-depleted plants with synthetic GLVs (Halitschke and others 2004). Choice tests with additional lepidopteran hebivores, including the closely related specialist *M. quinquemaculata* and the generalist *S. exigua*, demonstrated that these herbivores have a strong preference for leaves with intact GLV biosynthesis and that GLVs function as a feeding stimulant that apparently facilitated the recognition of a potential host plant.

Such plant-derived compounds not only facilitate host recognition, but can serve as a rapid indicator of the ''defensive status'' of the attacked plant. *Helicoverpa zea* larvae use host plant-derived JA and salicylate to activate their own cytochrome P450 genes that are involved in detoxification processes (Li and others 2002b). By responding to the plant signals that elicit plant defense traits, the insect is able to activate its detoxification systems simultaneously or before the biosynthesis of plant toxins.

FUTURE DIRECTIONS

Plant-derived oxylipin signals are clearly involved in a broad spectrum of interactions between plants and their environment including the elicitation of plant defenses as endogenous signals, direct effects on herbivore performance as direct and indirect chemical defenses, and host selection and feeding stimulation (Figure 1). Their ability to elicit responses within a plant may have facilitated a role in interactions between plants. It is clear that organisms that use plants as habitats or food sources have integrated these signals into the signaling systems that they use in their life cycle, as the well-documented synergism of host plant GLVs with insect sex pheromones clearly illustrates (Reddy and Guerrero 2004). Establishing a signaling function for a metabolite within a plant is a complicated task that becomes even more complicated when the receivers of the emitted signals involve additional organisms. Experiments in which these signals have been released or applied at unrealistic concentrations or in unrealistic enclosures suggest that they play a role in many different types of higher-order interactions, but establishing whether these interactions actually occur in nature requires the careful manipulation of the release and perception of these signals. This manipulation is most tractably accomplished through the use of mutants defective in the regulation, production or perception of the signals. The development of well-characterized signaling mutants used in combination with application of synthetic compounds will likely be the most effective way forward.

Biosynthetic mutants, defective in individual steps of signal production, are necessary to identify the compounds essential to the elicitation of wound and herbivore responses inside a plant and to analyze the role of these signals in higher-order interactions. The manipulation of specific signals in the background of an otherwise identical plant will allow researchers to evaluate whether a response occurs under natural conditions. In addition, mutants defective in the perception of specific signals would allow researchers to dissect the function of a signal in developmental processes or stress-induced responses, which would be necessary for a complete characterization of the fitness consequences of oxylipin-mediated plant responses.

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