Methyl jasmonate as a vital substance in plants

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The plant floral scent methyl jasmonate (MeJA) has been identified as a vital cellular regulator that mediates diverse developmental processes and defense responses against biotic and abiotic stresses. The pleiotropic effects of MeJA have raised numerous questions about its regulation for biogenesis and mode of action. Characterization of the gene encoding jasmonic acid carboxyl methyltransferase has provided basic information on the role(s) of this phytohormone in gene-activation control and systemic long-distance signaling. Recent approaches using functional genomics and bioinformatics have identified a whole set of MeJA-responsive genes, and provide insights into how plants use volatile signals to withstand diverse and variable environments.

Plants emit a variety of volatile substances from flowers, fruit, and vegetative tissues. This enables them to communicate with their surroundings, attract specific pollinators and be less susceptible to harmful insects [1]. In addition, the synthesis of plant volatiles is induced by certain external challenges caused by pathogens, herbivores or adverse weather conditions [2]. Moreover, such gaseous compounds act as airborne signals that mediate inter-plant communication thus affecting not only the challenged plant but also its neighbors [3]. Methyl jasmonate (MeJA) is a fragrant volatile compound initially identified from flowers of *Jasminum grandiflorum*, and has proven to be distributed ubiquitously in the plant kingdom. The volatile nature of MeJA led to the discovery of its role as a signal in plant cellular responses, plant–herbivore interactions and plant–plant interactions. This review highlights recent advances in molecular genetic studies on its hormone-like properties and mode-of-action as a signal transducer.

Methyl jasmonate-mediated responses

Methyl jasmonate and its free-acid jasmonic acid (JA) – collectively referred to as jasmonates – are important cellular regulators involved in diverse developmental processes, such as seed germination, root growth, fertility, fruit ripening, and senescence [4,5] (Fig. 1). In addition, jasmonates activate plant defense mechanisms in response to insect-driven wounding, various pathogens, and environmental stresses, such as drought, low temperature, and salinity [6].

The way in which jasmonates regulate these processes has been studied by observing gene expression patterns in a wide range of jasmonate-responsive metabolism. Microarray analysis has enabled massive screening of numerous genes induced by jasmonate application [7,8]. Jasmonate-regulated responses to wounding, microbial attack and abiotic stresses can be studied in parallel using this approach, enabling global and simultaneous analyses of gene expression profiles [9–14]. Numerous jasmonate-responsive genes whose biological functions are not yet known have been identified in these experiments.

Genes upregulated by MeJA treatment include those involved in jasmonate biosynthesis, secondary metabolism, cell-wall formation, and those encoding stress-protective and defense proteins. By contrast, genes involved in photosynthesis, such as ribulose bisphosphate carboxylase/oxygenase, chlorophyll a/b-binding protein, and light-harvesting complex II are downregulated. The genes discovered in these experiments are under intensive analysis using functional genomics and bioinformatics approaches to better define MeJA-mediated signaling pathways and cellular responses.

Regulation of MeJA biosynthesis

Jasmonates are synthesized in plants via the octadecanoid pathway (Fig. 2), and are similar to animal anti-inflammatory prostaglandins in structure and biogenesis [4,5]. Certain stimuli activate phospholipases to release α-linolenic acid (18:3) from membrane lipid. The linolenic acid is oxygenated by lipoxygenase (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). Jasmonic acid is synthesized from OPDA through reduction and three steps of β-oxidation after which it is catabolized further by JA carboxyl methyltransferase (JMT) to form its volatile counterpart MeJA [15,16]. A component of flower volatile *cis*-jasmone is formed by a β-oxidation of JA [17]. In addition, various derivatives can be formed from JA by hydroxylation, *O*-glycosylation and conjugation with amino acids.

Apart from octadecanoids in the main pathway of jasmonate biosynthesis, a variety of compounds are synthesized from α-linolenic acid and 13-HPOT through different branches of the pathway [18]. In addition, dinor-OPDA is derived directly from plastid 16:3 fatty acid rather than by β-oxidation of the 18-carbon OPDA [19]. All the metabolites from polyunsaturated

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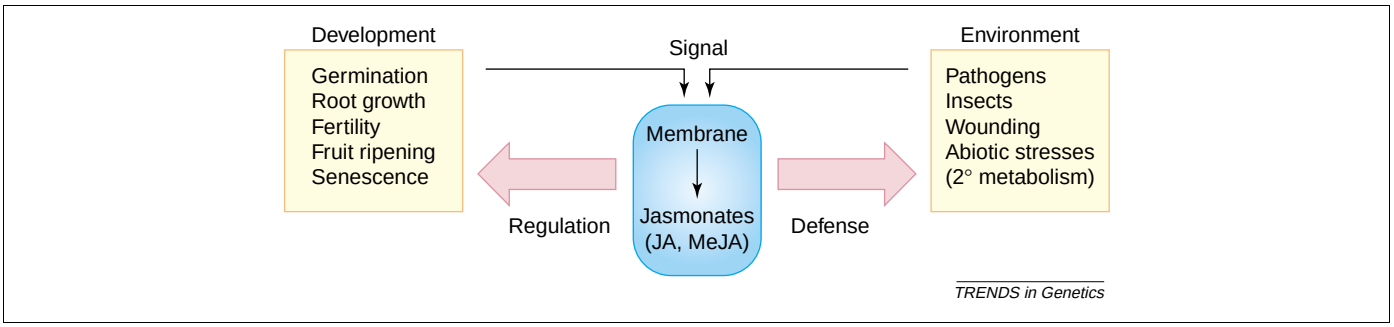

Fig. 1. Synthesis of jasmonates in response to developmental and environmental signals. Jasmonates act as cellular regulators in diverse developmental processes, such as seed germination, root growth, fertility, fruit ripening, and senescence. In addition, jasmonates activate plant defense mechanisms in response to insect-driven wounding, various pathogens, and environmental stresses such as drought, low temperature and salinity. Jasmonates are also involved in the regulation of some stages of secondary metabolism.

fatty acids – including jasmonates and octadecanoids – are collectively named oxylipins.

Several biosynthetic intermediates, isomers and derivatives of the octadecanoid pathway are also powerful cellular regulators, depending on the biological system [4,5]. Dinor-OPDA, α-linolenic acid, 13-HPOT, OPDA and isoleucine conjugate of JA are known to have their own biological activities in jasmonate-regulated responses. For example, *cis*-jasmone is induced upon damage to vegetative tissues, is an insect semiochemical (repellent or attractant) and is involved in plant defense [17].

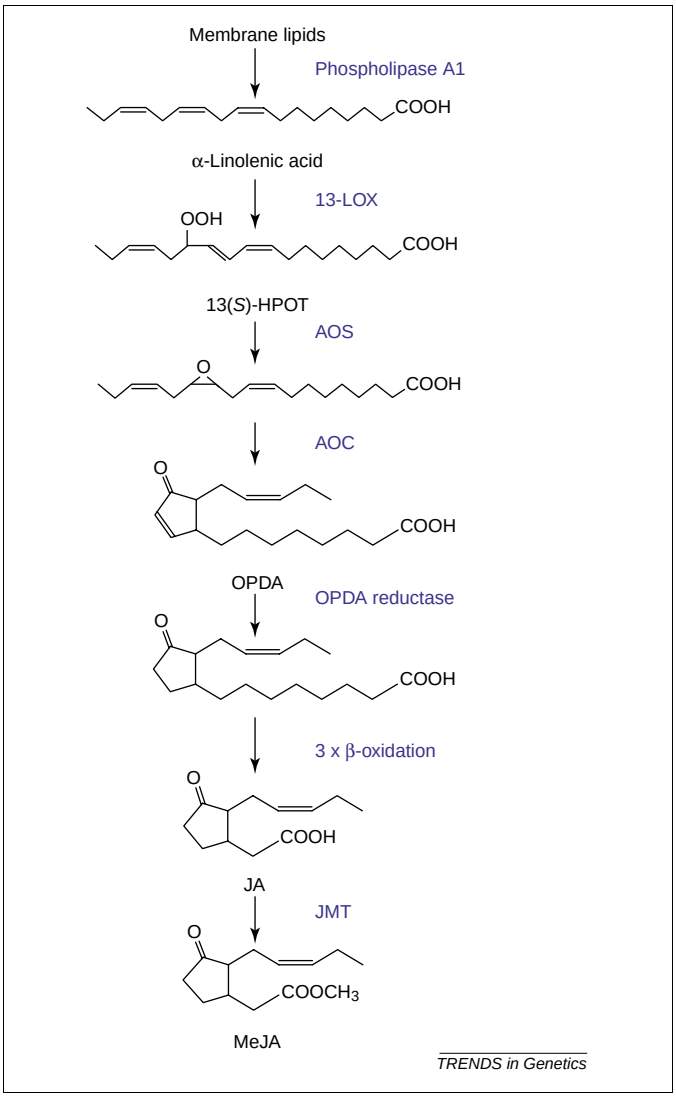

Fig. 2. Main pathway of jasmonate biosynthesis. A phospholipase A1 releases α-linolenic acid from membrane lipids. The α-linolenic acid is oxygenated by lipoxygenase (LOX) to form 13(*S*)-hydroxy linolenic acid (13-HPOT), which is then converted to 12-oxo-phytodienoic acid (OPDA) by allene oxide synthase (AOS) and allene oxide cyclase (AOC). Jasmonic acid (JA) is synthesized from OPDA through reduction and three steps of β-oxidation, and is further converted to methyl jasmonate (MeJA) by JA carboxyl methyltransferase (JMT).

Methyl jasmonate formation could be one of several important control points for jasmonate-regulated plant responses. This hypothesis was tested with transgenic *Arabidopsis* (*Arabidopsis thaliana*) overproducing the gene encoding JMT, where various jasmonate-responsive genes were constitutively expressed in the absence of wounding or jasmonate treatment [15]. Indeed, the transgenic plants showed enhanced resistance to a virulent fungal pathogen compared with non-transgenic plants. However, although transgenic potatoes overexpressing AOS have an increased concentration of JA, transcription of jasmonate-responsive genes is not enhanced in these plants [20].

Expression of *JMT* was not detected in young seedlings but its expression initiates at the floral nectary in developing flower [15,16]. In addition, expression of the gene was induced both locally and systemically either by wounding or by treatment with methyl jasmonate. This result suggests that *JMT* can perceive and respond to local and systemic signals generated by external stimuli, and that the signals might include methyl jasmonate itself. The cues of JMT activation in diverse jasmonate-regulated responses are being extensively investigated.

Cellular organelles such as chloroplasts and peroxisomes are considered to be the primary sites of JA biosynthesis. Enzymes necessary for the initiation of jasmonate biosynthesis, including phospholipase A1 [21], have been identified in the chloroplast. It is generally accepted that the OPDA synthesized in chloroplasts is transported to peroxisomes where OPDA is reduced and β-oxidized to produce JA [22]. Moreover, JMT is located in the cytoplasm, as observed by immuno-localization experiments [16]. It would be of particular interest to understand how these compounds are transported between the intracellular organelles and cytoplasm.

Mode of MeJA signaling

Analysis of mutants impaired in jasmonate biosynthesis as well as in signaling has shed light on the complexity of a

role for jasmonates as signaling compounds [23,24]. For example, the *coi1* mutation defines an *Arabidopsis* gene that functions in the jasmonate signaling pathway required for pollen development and defense against pathogens or insects. It was reported that the *COI1* gene encodes a protein containing leucine-rich repeats and an F-box motif [25]. F-box proteins are known to function as receptors that selectively recruit regulatory proteins as substrates for ubiquitination. Thus, it appears that COI1 is required to degrade a repressor of the jasmonate signaling pathway. Experiments using co-immunoprecipitation suggest that COI1 forms part of a functional E3-type ubiquitin ligase complex and mediates ubiquitination of a histone deacetylase, leading to the activation of jasmonate-responsive genes [26]. The target(s) of COI1 might be a key regulator of gene activation; however, these await identification.

Other studies using chemical inhibitors of protein kinases and protein phosphatases suggest that a reversible protein phosphorylation step is involved in the signal transduction pathway, leading to jasmonate-induced gene transcription [27,28]. A transposon-inactivation study revealed that mitogen-activated protein kinase 4 (MPK4) is required for jasmonate-responsive gene expression in *Arabidopsis* [29]. It also appears that Ca^{2+} and calmodulin act downstream of the reversible MAP kinase phosphorylation events [30].

Several *cis*-elements responsible for gene activation, including a G-box sequence (CACGTG), have been identified in the promoter regions of jasmonate-responsive genes [5]. In particular, a jasmonate- and elicitor-responsive element (JERE) was identified in the promoter of the strictosidine synthase gene *Str*, which is a secondary metabolite biosynthetic gene of *Catharanthus roseus* [31]. Subsequently, two octadecanoid-responsive *Catharanthus* AP2-domain proteins (ORCAs), ORCA2 and ORCA3, were found to mediate jasmonate-responsive gene expression via interaction with the JERE [31,32]. The ORCAs belong to the AP2/ERF-domain family of transcription factors. It should be noted that this family of transcription factors respond to many plant responses induced by ethylene (EREBF/ERF), cold stress (CBF/DREB1), and drought stress (DREB2) [33].

Jasmonates act synergistically or antagonistically with other phytohormones, such as ethylene, salicylic acid, auxin, and abscisic acid. In particular, combinations of MeJA and ethylene synergistically induced a group of defense genes [4,5]. The *Arabidopsis* mutant *cev1* constitutively produces jasmonates and ethylene, and exhibits enhanced resistance to pathogens [34]. As the *CEV1* gene encodes a cellulose synthase it is interesting that inhibition of cell-wall synthesis activates jasmonate- and ethylene-dependent stress responses [35]. Very recently, microarray analysis revealed that the transcription factor ethylene response factor 1 (ERF1) regulates the expression of a large number of genes responsive to both ethylene and jasmonate, integrating both signals for the activation of defense-response genes [14]. A link between jasmonate and the auxin signaling pathway was demonstrated by the observation that the auxin response mutant *axr1* was also defective in MeJA responses [36].

It is obvious that the jasmonate signaling pathway is connected to other signaling pathways, constituting a complex regulatory network. Recent studies have shown that ubiquitin–proteasome-mediated protein degradation is common to many plant signaling pathways, including those responsive to light, sucrose, pathogens, and hormones such as jasmonate, auxin, and abscisic acid [37–39]. Such a negative-control mechanism might provide tight regulation as observed in the auxin signaling pathway [40]. Currently, many of the cellular components involved in the ubiquitination process – estimated to be ~5% of the total proteins predicted from *Arabidopsis* genome sequence – have been extensively investigated in plants [41–43]. Such genomic studies will provide a framework for the complex signaling networks that control plant ubiquitination processes, thereby contributing to an understanding of the jasmonate signaling pathway, and crosstalk with other signaling pathways.

MeJA as a long distance signal

A group of jasmonate biosynthesis mutants are defective in pollen development [5]. In addition, the jasmonate-insensitive mutant *coi1* is also unable to produce viable pollen and becomes male-sterile, indicating that certain member(s) of the jasmonate family play a crucial role in this process. As jasmonate biosynthesis genes are specifically expressed in female flower organs, such as ovary, sepal, petal, and filaments, signal(s) produced in these organs must somehow be transported to the pollen-developing tissue (stomium). Such jasmonate-related systemic inter-cellular or -organ signal propagations have also been described in several defense responses. The best-studied examples are the accumulation of proteinase inhibitors in distal parts of wounded plants [44], and the ISR (induced systemic resistance) observed after exposure to specific biotic stimuli [45].

The possible role of jasmonates as a long-distance signal was recently suggested in a reciprocal grafting experiment using different combinations of wild-type and mutant tomato plants [46]. The jasmonate biosynthesis mutant *spr-2* was defective in the production – but not recognition – of a graft-transmissible wound signal. By contrast, *jai-1* mutant plants defective in jasmonate responses were compromised in the recognition of this signal but not in its production. These results suggest that activation of jasmonate biosynthesis is required for the production of a long-distance signal. The identity of the graft-transmissible wound signal has yet to be clearly defined.

Mutation at *Arabidopsis JAR1* reduces sensitivity to jasmonate, as observed in a variety of jasmonate-dependent defense responses. However, *jar1* mutants are fertile despite jasmonate being required for male fertility, indicating that this gene is not required for all jasmonate responses. Recently, it was reported that the JAR1 protein belongs to the acyl-adenylate-forming firefly luciferase superfamily, and is specifically active on JA in an assay for adenylation [47]. This result suggests that the role of the enzyme is to covalently modify JA itself rather than to act as a signaling intermediate. Methyl jasmonate is not a substrate for JAR1 [47] despite *jar1* mutants conferring

the same level of resistance to both JA and MeJA. This apparent discrepancy awaits clarification.

Until now, MeJA had become a strong candidate for a role in systemic signaling. This compound could diffuse to distal parts of the plant via the vapor phase [48] or by intercellular migration, possibly through the phloem [49]. Free-acid JA might not be able to move across the cellular membrane without a carrier because of its acidic nature. In relation to the long-distance signaling required for pollen development, it is worth noting that *JMT* expression initiates at the floral nectary during flower development [15,16]. Activation of *JMT* was also induced in distal leaves of *Arabidopsis* by wounding or exogenous MeJA application, indicating that MeJA production can be systemically regulated [15]. Such systemic *JMT* activation can occur during post-floral developmental processes, as it was observed that the gene expression initiates and limits in the flowers, but eventually propagates to the whole plant body, through the phloem of the floral and main stems, to reach to the primary root [50]. Certain signal(s) generated during an early event in developmental processes or defense responses might initiate *JMT* activation to produce MeJA. This diffusible intercellular signal transducer might then activate the same gene in neighboring cells thereby propagating MeJA-mediated cellular responses throughout the whole plant. This hypothesis awaits more conclusive experimental evidence.

Concluding remarks

As described here, an understanding of how MeJA biogenesis is regulated at the molecular level, and how it relates to jasmonate-responsive gene activation, is still incomplete. Until recently, jasmonate-mediated plant responses have been studied mainly through the application of synthesized chemicals comprising various isomers of which actual equilibrium concentration *in planta* is unknown. Thus, in most experiments, clear distinctions between members of the jasmonate family have not been made. More importantly, JA and MeJA have been used in many experiments without consideration of their relative importance in each jasmonate response. In this regard, characterization and transgenic research of the cellular component (JMT) that catalyzes the formation of MeJA have helped to clarify the complexity of jasmonate-mediated plant responses.

Detailed investigation of the *JMT* activation mechanism would provide more information on the role(s) of MeJA in gene-activation control and systemic long-distance signaling. Most importantly, loss-of-function mutants or inducible overexpression of *JMT* in transgenic plants would provide an opportunity to address issues relating to these processes. In addition, massive screening and characterization by functional genomics of more MeJA-related genes would also reveal clues for the role(s) of MeJA as a vital cellular regulator.

As shown with the function of MeJA in various responses, the production and use of various volatiles in development and defense responses is a remarkable feature of plant responses. Further investigation of the gas-phase signaling mechanism will enable an understanding of how plants use volatiles to confront and manage diverse and variable environments. Such advances will also provide a means of manipulating cellular signaling pathways to improve the stress-resistance or metabolite productivity of plants.

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