Review

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 geneactivation control and systemic long-distance signaling. Recent approaches using functional genomics and bioinformatics have identified a whole set of MeJAresponsive 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, plantherbivore 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 stressprotective 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



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).

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].

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 $\sim 5\%$ 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 jasmonateinsensitive 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 pollendeveloping 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 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 jasmonatedependent 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 412

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 jasmonatemediated 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 MeJArelated 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.

Acknowledgements

We apologize to those whose work could not be cited owing to space limitations. We thank Dr Nam-Hai Chua (Rockefeller University, New York, NY, USA) and Johan Memelink (Institute of Biology Leiden, Netherlands) for critical reading of the manuscript. This work was supported by a grant from the Crop Functional Genomics Center (Korea) and by the Brain Korea 21 Project of the Ministry of Education.

References

- Pichersky, E. and Gershenzon, J. (2002) The formation and function of plant volatiles: perfumes for pollinator attraction and defense. *Curr. Opin. Plant Biol.* 5, 237–243
- 2 Paré, P.W. and Tumlinson, J.H. (1999) Plant volatiles as a defense against insect herbivores. *Plant Physiol.* 121, 325-331
- 3 Arimura, G. et al. (2000) Herbivory-induced volatiles elicit defence genes in lima bean leaves. Nature 406, 512–515
- 4 Creelman, R.A. and Rao, M.V. (2002) The oxylipin pathway in *Arabidopsis*. In *The Arabidopsis Book* (Somerville, C.R. and Meyerowitz, E.M., eds), doi10.1199/tab.0012, American Society of Plant Biologists
- 5 Wasternack, C. and Hause, B. (2002) Jasmonates and octadecanoids: signals in plant stress responses and development. *Prog. Nucleic Acid Res. Mol. Biol.* 72, 165–221
- 6 Wasternack, C. and Parthier, B. (1997) Jasmonate-signalled plant gene expression. Trends Plant Sci. 2, 302–307
- 7 Schenk, P.M. et al. (2000) Coordinated plant defense responses in Arabidopsis revealed by microarray analysis. Proc. Natl. Acad. Sci. U. S. A. 97, 11655–11660
- 8 Sasaki, Y. et al. (2001) Monitoring of methyl jasmonate-responsive genes in Arabidopsis by cDNA macroarray: self-activation of jasmonic acid biosynthesis and crosstalk with other phytohormone signaling pathways. DNA Res. 8, 153–161
- 9 Reymond, P. et al. (2000) Differential gene expression in response to mechanical wounding and insect feeding in Arabidopsis. Plant Cell 12, 707–719
- 10 Ozturk, Z.N. et al. (2002) Monitoring large-scale changes in transcript abundance in drought- and salt-stressed barley. Plant Mol. Biol. 48, 551–573
- 11 Cheong, Y.H. et al. (2002) Transcriptional profiling reveals novel interactions between wounding, pathogen, abiotic stress, and hormonal responses in Arabidopsis. Plant Physiol. 129, 661–677
- 12 Chen, W. et al. (2002) Expression profile matrix of Arabidopsis transcription factor genes suggests their putative functions in response to environmental stresses. Plant Cell 14, 559-574
- 13 Kreps, J.A. *et al.* (2002) Transcriptome changes for *Arabidopsis* in response to salt, osmotic, and cold stress. *Plant Physiol.* 130, 2129–2141
- 14 Lorenzo, O. *et al.* (2003) Ethylene response factor1 integrates signals from ethylene and jasmonate pathways in plant defense. *Plant Cell* 15, 165–178
- 15 Seo, H.S. et al. (2001) Jasmonic acid carboxyl methyltransferase: a key enzyme for jasmonate-regulated plant responses. Proc. Natl. Acad. Sci. U. S. A. 98, 4788–4793
- 16 Song, J.T. et al. (2000) NTR1 encodes a floral nectary-specific gene in Brassica campestris L. ssp. pekinensis. Plant Mol. Biol. 42, 647–655
- 17 Birkett, M.A. et al. (2000) New roles for cis-jasmone as an insect semiochemical and in plant defense. Proc. Natl. Acad. Sci. U. S. A. 97, 9329–9334
- 18 Feussner, I. and Wasternack, C. (2002) The lipoxygenase pathway. Annu. Rev. Plant Biol. Physiol. Plant Mol. Biol. 53, 275–279
- 19 Weber, H. et al. (1997) Dinor-oxo-phytodienoic acid: a new hexadecanoid signal in the jasmonate family. Proc. Natl. Acad. Sci. U. S. A. 94, 10473–10478
- 20 Harms, K. et al. (1995) Expression of a flax allene oxide synthase cDNA leads to increased endogenous jasmonic acid (JA) levels in transgenic potato plants but not to a corresponding activation of JA-responding genes. Plant Cell 7, 1645–1654
- 21 Ishiguro, S. *et al.* (2001) The *Defective in Anther Dehiscence* 1 gene encodes a novel phospholipase A1 catalyzing the initial step of jasmonic

acid biosynthesis, which synchronizes pollen maturation, anther dehiscence, and flower opening in Arabidopsis. Plant Cell 13, 2191–2209

- 22 Stintzi, A. and Browse, J. (2000) The Arabidopsis male-sterile mutant, opr3, lacks the 12-oxophytodienoic acid reductase required for jasmonate synthesis. Proc. Natl. Acad. Sci. U. S. A. 97, 10625–10630
- 23 Berger, S. (2002) Jasmonate-related mutants of Arabidopsis as tools for studying stress signaling. *Planta* 214, 497–504
- 24 Turner, J.G. $et\ al.$ (2002) The jasmonate signal pathway. Plant Cell 14, S153–S164
- 25 Xie, D-X. et al. (1998) COI1: an Arabidopsis gene required for jasmonate-regulated defense and fertility. Science 280, 1091-1094
- 26 Devoto, A. et al. (2002) COI1 links jasmonate signalling and fertility to the SCF ubiquitin-ligase complex in Arabidopsis. Plant J. 32, 457–466
- 27 Rojo, E. et al. (1998) Reversible protein phosphorylation regulates jasmonic acid-dependent and -independent wound signal transduction pathways in Arabidopsis thaliana. Plant J. 13, 153–165
- 28 Jensen, A.B. et al. (2002) Fusion genetic analysis of jasmonatesignalling mutants in Arabidopsis. Plant J. 29, 595-606
- 29 Petersen, M. et al. (2000) Arabidopsis MAP kinase 4 negatively regulates systemic acquired resistance. Cell 103, 1111–1120
- 30 León, J. et al. (1998) Jasmonic acid-dependent and -independent wound signal transduction pathways are differentially regulated by Ca²⁺/ calmodulin in Arabidopsis thaliana. Mol. Gen. Genet. 258, 412–419
- 31 Menke, F.L.H. *et al.* (1999) A novel jasmonate- and elicitor-responsive element in the periwinkle secondary metabolite biosynthetic gene *Str* interacts with a jasmonate and elicitor-inducible AP2-domain transcription factor, ORCA2. *EMBO J.* 18, 4455–4463
- 32 van der Fits, L. and Memelink, J. (2000) ORCA3, a jasmonateresponsive transcriptional regulator of plant primary and secondary metabolism. *Science* 289, 295–297
- 33 Memelink, J. et al. (2001) ORCAnization of jasmonate-responsive gene expression in alkaloid metabolism. Trends Plant Sci. 6, 212–219
- 34 Ellis, C. and Turner, J.G. (2001) The Arabidopsis mutant cev1 has constitutively active jasmonate and ethylene signal pathways and enhanced resistance to pathogens. *Plant Cell* 13, 1025–1033
- 35 Ellis, C. *et al.* (2002) The *Arabidopsis* mutant *cev1* links cell wall signaling to jasmonate and ethylene responses. *Plant Cell* 14, 1557–1566
- 36 Tiryaki, I. and Staswick, P.E. (2002) An Arabidopsis mutant defective in jasmonate response is allelic to the auxin-signaling mutant axr1. *Plant Physiol.* 130, 887–894

- 37 Hellmann, H. and Estelle, M. (2002) Plant development: regulation by protein degradation. Science 297, 793-797
- 38 Ellis, C. et al. (2002) Protein complexes mediate signalling in plant responses to hormones, light, sucrose and pathogens. Plant Mol. Biol. 50, 971–980
- 39 Lopez-Molina, L. et al. (2003) AFP is a novel negative regulator of ABA signaling that promotes ABI5 protein degradation. Genes Dev. 17, 410–418
- 40 Schwechheimer, C. et al. (2001) Interactions of the COP9 signalosome with the E3 ubiquitin ligase SCF^{TIR1} in mediating auxin response. *Science* 292, 1379–1382
- 41 Capron, A. et al. (2003) First glance at the plant APC/C, a highly conserved ubiquitin-protein ligase. Trends Plant Sci. 8, 83-89
- 42 Mladek, C. *et al.* (2003) Identification and characterization of the *ARIADNE* gene family in *Arabidopsis*. A group of putative E3 ligases. *Plant Physiol.* 131, 27–40
- 43 Kurepa, J. et al. (2003) The small ubiquitin-like modifier (SUMO) protein modification system in Arabidopsis: accmulation of SUMO1 and -2 conjugates is increased by stress. J. Biol. Chem. 278, 6862–6872
- 44 León, J. et al. (2001) Wound signalling in plants. J. Exp. Bot. 52, 1-9 45 Pieterse, C.M.J. et al. (1998) A novel signaling pathway controlling
- induced systemic resistance in Arabidopsis. Plant Cell 10, 1571–1580 46 Li, L. et al. (2002) Distinct roles for jasmonate synthesis and action in
- 40 Li, L. et al. (2002) Distinct roles for jasmonate synthesis and action in the systemic wound response of tomato. Proc. Natl. Acad. Sci. U. S. A. 99, 6416–6421
- 47 Staswick, P.E. *et al.* (2002) Jasmonate response locus JAR1 and several related *Arabidopsis* genes encode enzymes of the firefly luciferase superfamily that show activity on jasmonic, salicylic, and indole-3-acetic acids in an assay for adenylation. *Plant Cell* 14, 1405–1415
- 48 Karban, R. *et al.* (2000) Communication between plants: induced resistance in wild tobacco plants following clipping of neighboring sagebrush. *Oecologia* 125, 66–71
- 49 Ruiz-Medrano, R. et al. (2001) The phloem as a conduit for inter-organ communication. Curr. Opin. Plant Biol. 4, 202–209
- 50 Koo, Y.J. et al. (2002) Systemic activation of jasmonic acid carboxyl methyltransferase gene during jasmonate-regulated plant responses. In Plant, Animal and Microbe Genomes X Conference, Abstract P-777, p. 272 (http://www.intl-pag.org/pag/10/)

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