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Review

The wound hormone jasmonate

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ABSTRACT

Plant tissues are highly vulnerable to injury by herbivores, pathogens, mechanical stress, and other environmental insults. Optimal plant fitness in the face of these threats relies on complex signal transduction networks that link damage-associated signals to appropriate changes in metabolism, growth, and development. Many of these wound-induced adaptive responses are triggered by *de novo* synthesis of the plant hormone jasmonate (JA). Recent studies provide evidence that JA mediates systemic wound responses through distinct cell autonomous and non-autonomous pathways. In both pathways, bioactive JAs are recognized by an F-box protein-based receptor system that couples hormone binding to ubiquitin-dependent degradation of transcriptional repressor proteins. These results provide a framework for understanding how plants recognize and respond to tissue injury.

Contents

1. Introduction

Terrestrial plants encounter numerous environmental conditions that have the potential to injure or destroy above- and below-ground tissues. Arthropod herbivores and pathogens pose one of the most serious threats to plant survival. Tissue injury inflicted by plant-eating organisms does not go unnoticed; strong selection pressure imposed by biotic stress has fueled the evolution of innovative mechanisms by which plants perceive and respond to tissue damage (Howe and Jander, 2008). Plant hormones occupy a central role in regulating these highly dynamic and adaptive responses (Pieterse et al., 2009; Santner et al., 2009).

The idea that plants actively respond to tissue injury dates back to work by Wiesner in the 19th century (Wiesner, 1892). Bioassays were developed and used to purify the so-called wound hormone traumatin, which exhibits mitogenic properties related to wound healing (Wehnelt, 1927; English et al., 1939). Traumatin is a reactive fatty acid derivative (12-oxo-*trans*-10-dodecenoic acid) produced by the hydroperoxy lyase branch of the lipoxygenase pathway (Zimmerman and Coudron, 1979). Although the apparent limited activity of traumatin to specific bean cultivars dampened enthusiasm for the concept of a wound hormone, this early work helped to arouse interest in biochemical research on lipoxygenase-based pathways for oxidative metabolism of polyunsaturated fatty acids (Vick and Zimmerman, 1984; Hamberg and Gardner,

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1992; Farmer, 1994; Matsui, 2006). Today, it is widely accepted that oxylipins (oxygenated fatty acid derivatives) produced by these pathways perform a variety of essential functions in the plant life cycle (Blee, 2002; Feussner and Wasternack, 2002; Howe and Schilmiller, 2002; Browse, 2009).

Wound signaling research in plants was revitalized in the 1970s following the discovery that leaf damage induces systemic accumulation of proteinase inhibitors (PIs) that impair insect digestive enzymes (Green and Ryan, 1972). This landmark paper introduced the idea that mobile signals generated at the site of injury orchestrate systemic protection against insect herbivores. During the past 35 years, systemic wound responses have been demonstrated in species throughout the plant kingdom, and intense research efforts have focused on the identification of systemic wound signals and the mechanisms by which they are generated, transported, and perceived (Karban and Baldwin, 1997; Ryan, 2000; Leon et al., 2001; Howe, 2004). The concept that biotic agents activate local and systemic host defense responses is a cornerstone of current theories of plant immunity (Jones and Dangl, 2006; Browse and Howe, 2008; Howe and Jander, 2008; Boller and He, 2009; Dicke et al., 2009; Wu and Baldwin, 2009). Oxylipins play a central role in many of these defense signaling pathways.

Tissue injury invariably results in perturbation or damage to cell membranes. In both plants and animals, oxylipins derived from membrane lipids are sentinels of wound stress. These signaling compounds are typically synthesized *de novo* in specific cell types upon activation of lipases that release fatty acids from membrane lipids. The biochemical logic underlying oxylipin synthesis in plants and animals is remarkably similar, with species in both kingdoms using lipoxygenases (or other oxygenases) and cytochromes P450 to oxidize fatty acids (Bergey et al., 1996; Howe et al., 2000; Blee, 2002; Lee et al., 2008). Several intrinsic features of oxylipins make them well suited for this purpose: (i) a large supply of precursors in the form of membrane lipids, (ii) rapid synthesis in response to cell damage or other inductive cues, (iii) structural diversity that permits functional specificity, (iv) capacity for intra- and intercellular transport, and (v) rapid turnover or inactivation. Knowledge gained from the study of plant oxylipins is providing insight into the underlying mechanisms and evolutionary origins of fatty acid-based signaling pathways in diverse biological systems (Lee et al., 2008).

Here, we discuss recent advances in our understanding of the role of the jasmonate (JA) family of oxylipins in plant responses to wound stress. First, we consider key features of JA as a wound hormone, including its mechanism of perception by plant cells. Second, we describe damage-associated signals that activate JA synthesis and discuss what these signals tell us about how plants perceive tissue injury. Finally, we review recent studies concerning the role of JA in regulating systemic wound responses. Because of the focus on JA, we do not discuss JA-independent wound responses or other oxylipins (e.g., green leaf volatiles produced by the hydroperoxide lyase pathway) that affect plant interactions with other organisms. Readers are referred to several review articles for additional information on these topics (Leon et al., 2001; Howe, 2004; Matsui, 2006; Felton and Tumlinson, 2008; Heil and Ton, 2008; Dicke et al., 2009).

2. A wound hormone and its receptor

The discovery of JA as a potent elicitor of proteinase inhibitor (PI) expression in tomato (Farmer and Ryan, 1990) and secondary metabolism in plant cell cultures (Gundlach et al., 1992) provided initial insight into the importance of JA as a defense hormone. Subsequent genetic studies established the indispensable role of JA in promoting resistance to herbivores and pathogens that betray their presence by activating JA synthesis in damaged or infected host tissue (Howe et al., 1996; McConn et al., 1997; Vijayan et al., 1998; Kessler et al., 2004; Browse and Howe, 2008; Howe and Jander, 2008). JA exerts its protective effects by regulating a wide range of defense-related processes, including the synthesis of toxic secondary metabolites (Pauwels et al., 2009), production of morphological barriers (Yoshida et al., 2009), and changes in the rate of vegetative growth (Yan et al., 2007; Balbi and Devoto, 2008; Zhang and Turner, 2008). These broad effects of JA imply a general role for the hormone in controlling tradeoffs between growth and defense (Herms and Mattson, 1992; Baldwin, 1998).

JA responses typically depend on large-scale changes in gene expression. In unstressed cells containing low JA levels, transcription factors that promote expression of JA-responsive genes are repressed by members of the JASMONATE ZIM-domain (JAZ) protein family (Chini et al., 2007; Thines et al., 2007; Yan et al., 2007). Increased JA levels stimulate JAZ binding to CORONATINE INSENSITIVE1 (COI1), which is the F-box protein component of the E_3 ubiquitin ligase SCF^{COI1} (Balbi and Devoto, 2008; Thines et al., 2007). Hormone-induced COI1-JAZ interaction triggers JAZ degradation via the ubiquitin/26S proteasome pathway, thereby releasing transcription factors from repression. Recent evidence indicates that the repressive action of JAZ proteins depends on their ability to dimerize via the conserved ZIM-domain (Chini et al., 2009; Chung and Howe, 2009). Additional details about the SCF^{COI1}/JAZ pathway are described by Chung et al. (2009).

A complete understanding of JA as a sentinel of tissue injury requires knowledge of how specific chemical forms of the hormone interact with cognate receptor(s). For this purpose, it is useful to distinguish bioactive JAs, which directly promote the formation of COI1-JAZ complexes, from non-bioactive JAs, which are either precursors or deactivated forms of the bioactive compounds (Katsir et al., 2008a). This distinction is important for defining the identity of specific components of the "signal signature" (Weber et al., 1997; de Vos et al., 2005) that evoke a particular response. Following its synthesis by the octadecanoid pathway in response to wounding or other inductive cues, the (3*R*,7*S*) stereoisomer of JA (also known as (+)-7-*iso*-JA) is metabolized to a wide range of derivatives (Fig. 1). The physiological function of the vast majority of these compounds is not known. It is well established, however, that many JA derivatives exert distinct biological activities when applied to plant tissues (Blechert et al., 1999; Miersch et al., 1999; Stintzi et al., 2001; Ribot et al., 2008; Wang et al., 2008). Because exogenous JAs do not target specific cell types, are often administered at non-physiological concentrations, and are subject to unknown metabolic fates, additional approaches are required to determine the *in vivo* role of specific JA derivatives.

A major breakthrough in identifying bioactive JAs came from the development of techniques to study hormone action with cell-free and yeast-based protein–protein interaction assays (Dharmasiri et al., 2003; Thines et al., 2007). These approaches were used to demonstrate that COI1-JAZ interactions are stimulated by the JA-amino acid conjugate jasmonoyl-L-isoleucine (JA-Ile), but not by JA, methyl-JA (MeJA), or the JA precursor 12-oxo-phytodienoic acid (OPDA) (Thines et al., 2007; Katsir et al., 2008b; Melotto et al., 2008). Identification of JA-Ile as a causal signal for COI1-JAZ binding extends previous work showing that conjugation of JA to Ile by the acyl-adenylate/thioester-forming enzyme JASMONATE RESISTANT1 (JAR1) activates the JA signal (Staswick and Tiryaki, 2004; Staswick, 2008).

Synthetic preparations of JA-Ile used for initial COI1-JAZ interaction studies (Thines et al., 2007; Katsir et al., 2008b; Melotto et al., 2008) contained a mixture of the two naturally occurring stereoisomers, namely (3*R*,7*S*)-JA-Ile (also known as (+)-7-*iso*-JA-Ile) and (3*R*,7*R*)-JA-Ile (also referred to as (−)-JA-Ile) (Fig. 1). In plant tissues and in solution, these two isomers are in equilibrium,

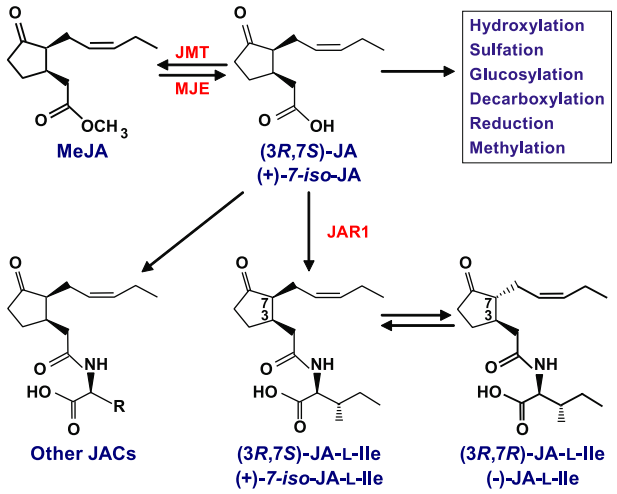

Fig. 1. The biologically synthesized form of jasmonic acid, (3*R*,7*S*)-JA (also known as (+)-7-*iso*-JA), is metabolized to various bioactive and non-bioactive derivatives. JA carboxyl methyltransferase (JMT) converts (3*R*,7*S*)-JA to the volatile compound, methyl-JA (MeJA). The reverse reaction is catalyzed by MeJA esterase (MJE). (3*R*,7*S*)-JA is also converted to the active form of the hormone, (3*R*,7*S*)-JA-L-Ile (also known as (+)-7-*iso*-JA-L-Ile), via JAR1-mediated conjugation to L-isoleucine. (3*R*,7*S*)-JA-L-Ile may epimerize to the more stable (3*R*,7*R*)-JA-L-Ile stereoisomer (also known as (−)-JA-L-Ile) in which the side chains at position 7 and 3 are in the *trans* orientation with respect to one another. (3*R*,7*S*)-JA can be conjugated to other amino acids to produce various JA-amino acid conjugates (JACs). Both free and amino acid-conjugated forms of (3*R*,7*S*)-JA are subject to several other modifications (boxed).

with the thermodynamically more stable (3*R*,7*R*)-JA-Ile in excess (~94:6) over the less stable (3*R*,7*S*)-JA-Ile isomer (Miersch et al., 1986; Creelman and Mullet, 1997; Wasternack, 2007; Fonseca et al., 2009). Preparations of JA-Ile synthesized in this manner have been shown to activate JA-responsive gene expression and JA-dependent physiological processes (Kramell et al., 1997, 2000; Wasternack et al., 1998; Staswick and Tiryaki, 2004; Wang et al., 2008). Initial COI1-JAZ interaction studies (Thines et al., 2007; Katsir et al., 2008b; Melotto et al., 2008) used these preparations to demonstrate that JA-Ile is the relevant signal for COI1-JAZ partnering but, because of the mixed composition of isomers, did not draw conclusions about the relative activity of (3*R*,7*S*)-JA-Ile and (3*R*,7*R*)-JA-Ile. Recent experiments performed with purified isomers showed that (3*R*,7*S*)-JA-Ile is much more active than stereochemically pure (3*R*,7*R*)-JA-Ile in promoting JAZ9 interaction with COI1, as well as JA-dependent phenotypic responses in *Arabidopsis* (Fonseca et al., 2009). These findings demonstrate that (3*R*,7*S*)-JA-Ile is an endogenous bioactive JA, whereas the (3*R*,7*R*)-JA-Ile isomer is largely inactive.

The relative potency of (3*R*,7*S*)-JA-Ile in promoting COI1-JAZ interaction is consistent with the fact that this compound is stereochemically similar to the bacterial toxin coronatine that acts as a potent agonist of the JA receptor (Katsir et al., 2008b; Melotto et al., 2008; Staswick, 2008). The ability of radiolabeled coronatine to bind COI1-JAZ complexes with high affinity ($K_d \sim 20$ nM) in a manner that depends on COI1 indicates that COI1 or a COI1-JAZ complex is a component of the coronatine receptor, and most likely the receptor for active stereoisomers of JA-Ile as well (Katsir et al., 2008b; Melotto et al., 2008). The conclusion that COI1 is part of a receptor for JA-Ile is supported by extensive genetic evidence showing that loss of COI1 function abolishes JA responses *in planta* (Feys et al., 1994; Li et al., 2004; Paschold et al., 2007). Formal biochemical proof that COI1 or a COI1-JAZ complex is a JA receptor will require demonstration that specific isomers of JA-Ile bind specifically, saturably, and reversibly to the purified receptor.

3. Synthesis in distress

Current models indicate that JA signaling is initiated upon accumulation of JA-Ile to levels that are sufficient to promote COI1-JAZ interactions. A detailed understanding of JA/JA-Ile homeostasis in healthy and distressed tissues is therefore paramount to understanding how the pathway is controlled. It is well established that JA levels vary widely depending on developmental stage and environmental conditions (Creelman and Mullet, 1997; Wasternack, 2007). Leaf damage inflicted by mechanical wounding and herbivory are highly effective triggers for *de novo* JA synthesis (Blechert et al., 1995; McCloud and Baldwin, 1997; Weber et al., 1997; Strassner et al., 2002; Koo et al., 2009). Mechanical damage results in large increases (~25-fold) in JA and JA-Ile accumulation within 5 min of tissue injury (Chung et al., 2008; Glauser et al., 2008; Koo et al., 2009). The remarkable speed of this response indicates that all biosynthetic enzymes involved in production of JA/JA-Ile are present in resting cells. Wounded leaves also accumulate other JA-amino acid conjugates, including JA-Val and JA-Leu (Wang et al., 2007; Suza and Staswick, 2008; Koo et al., 2009). The ability of JA-Val and JA-Leu to promote interaction of tomato COI1 and JAZ1/3 *in vitro* (Katsir et al., 2008b) suggests that these compounds may be active signals in the wound response. However, because the wound-induced level of JA-Ile is at least 10-fold higher than that of JA-Val and JA-Leu, it seems likely that JA-Ile is the major signal for COI1-dependent wound responses in *Arabidopsis* (Koo et al., 2009).

The available evidence is consistent with a model in which wound-induced production of (3*R*,7*S*)-JA-Ile promotes SCF^{COI1}-dependent degradation of JAZs, thereby allowing downstream transcription factors to engage early response genes. The transient nature of JA-Ile accumulation and JA responses in wounded leaves (Wang et al., 2007; Chung et al., 2008; Suza and Staswick, 2008; Koo et al., 2009) implies the existence of efficient mechanisms to metabolize or inactivate the signal. Signal attenuation may be accomplished by hydroxylation of JA/JA-Ile (Glauser et al., 2008; Miersch et al., 2008) or by epimerization of (3*R*,7*S*)-JA-Ile to (3*R*,7*R*)-JA-Ile (Fig. 1; Farmer, 1994; Fonseca et al., 2009). Rigorous testing of these ideas will require identification and transgenic manipulation of enzymes involved in JA-Ile metabolism. Plant cells likely use additional mechanisms to attenuate JA-mediated wound responses. In *Arabidopsis* leaves, for example, wound-induced expression of JA response genes declines sharply during a period when JA-Ile levels remain high (Koo et al., 2009). This observation suggests that damaged leaves become desensitized to the JA-Ile signal. Wound-induced production of JAZ repressors that are stabilized against hormone-induced degradation may be involved in this form of negative feedback control (Chung and Howe, 2009).

4. Damage-associated signals activate JA synthesis

JA synthesis is controlled at the level of substrate availability (Stenzel et al., 2003b; Wasternack, 2007). Lipases that release JA precursors (e.g., linolenic acid) from plastid lipids generate substrates for JA synthesis (Ishiguro et al., 2001; Hyun et al., 2008). Expression of these lipases in reproductive tissues appears to be regulated by developmental cues (Ito et al., 2007). However, the manner in which lipase or other rate-limiting enzymes in the JA biosynthetic pathway are controlled in response to wounding and herbivory is not known. Elucidation of these mechanisms will significantly advance our understanding of how JA synthesis is regulated and, more broadly, how plants recognize tissue injury.

Several observations are pertinent to the question of how wounding controls *de novo* synthesis of JA. First, mechanical tissue damage is sufficient to trigger a rapid and transient JA/JA-Ile burst.

The timing and amplitude of JA accumulation is affected by the temporal and spatial patterns of leaf damage (McCloud and Baldwin, 1997; Mithöfer et al., 2005; Stork et al., 2009). Second, many wound-induced defense responses are stimulated by compounds in insect oral secretions and by host-derived elicitors. Numerous studies have shown, for example, that insect feeding or application of oral secretions to wound sites elicits a different volatile response than mechanical damage alone (Turlings et al., 1990; De Moraes et al., 2001; Arimura et al., 2004a,b; Schmelz et al., 2006). This and related phenomena suggest that plants have evolved recognition systems to perceive compounds of insect origin (Truitt et al., 2004; Schmelz et al., 2006; Felton and Tumlinson, 2008; Mithöfer and Boland, 2008; Wu and Baldwin, 2009). Finally, *de novo* JA synthesis can be activated in the absence of wound stress. Signals produced during reproductive development, for example, promote JA synthesis in specific cell types (Hause et al., 2000; Ito et al., 2007). Several chemical elicitors, including the peptide systemin and cell wall-derived oligosaccharides, also induce synthesis of the hormone in undamaged leaves (Mueller et al., 1993; Doares et al., 1995; Lee and Howe, 2003). As discussed in greater detail below, wounding can also activate *de novo* JA synthesis in undamaged parts of the plant.

These collective observations imply the existence of cell surface receptors or other sensory systems that, upon recognition of nonself or damage-associated (self) signals, activate the octadecanoid pathway for JA synthesis (Fig. 2). This concept is consistent with models of plant innate immunity in which pathogen- or microbe-associated molecular patterns (PAMPs/MAMPs) trigger host defense responses by activating pattern recognition receptors (PRRs) at the plant cell surface (Boller and He, 2009). The term herbivore-associated molecular pattern (HAMP) has been used to describe herbivore-derived compounds that elicit host defense responses (Mithöfer and Boland, 2008). Several compounds that modulate the host response have been identified in insect oral secretions, saliva, and oviposition fluids (Felton and Tumlinson, 2008; Schmelz et al., 2009; Wu and Baldwin, 2009). The mechanisms by which these compounds are perceived by plant cells, however, remain elusive.

Rapid accumulation of JA in response to mechanical stress or wounding indicates that *de novo* JA synthesis can be efficiently triggered by plant-derived signals. The danger model of vertebrate immunity has used the term damage-associated molecular patterns (DAMPs) to describe host-derived signals that trigger immune responses (Matzinger, 2002; Seong and Matzinger, 2004). In plants, these signals may include peptide or cell wall-derived oligosaccharide fragments (Narvaez-Vasquez et al., 2007), as well as wound-induced hydraulic or electrical signals (Malone, 1993; Zimmermann et al., 2009). Recognition of the latter physical signals may involve sensors of mechanical stress or cell wall integrity. Genetic and pharmacological disruption of cell wall integrity has been shown to constitutively activate the JA pathway (Ellis et al., 2002; Hamann et al., 2009). There is also evidence to indicate that mitogen-activated protein (MAP) kinase cascades are involved in the activation of JA synthesis in response to wound damage and wound-associated elicitors, including fatty-acid amino acid conjugates and systemin (Seo et al., 1999; Kandoth et al., 2007; Wu et al.,

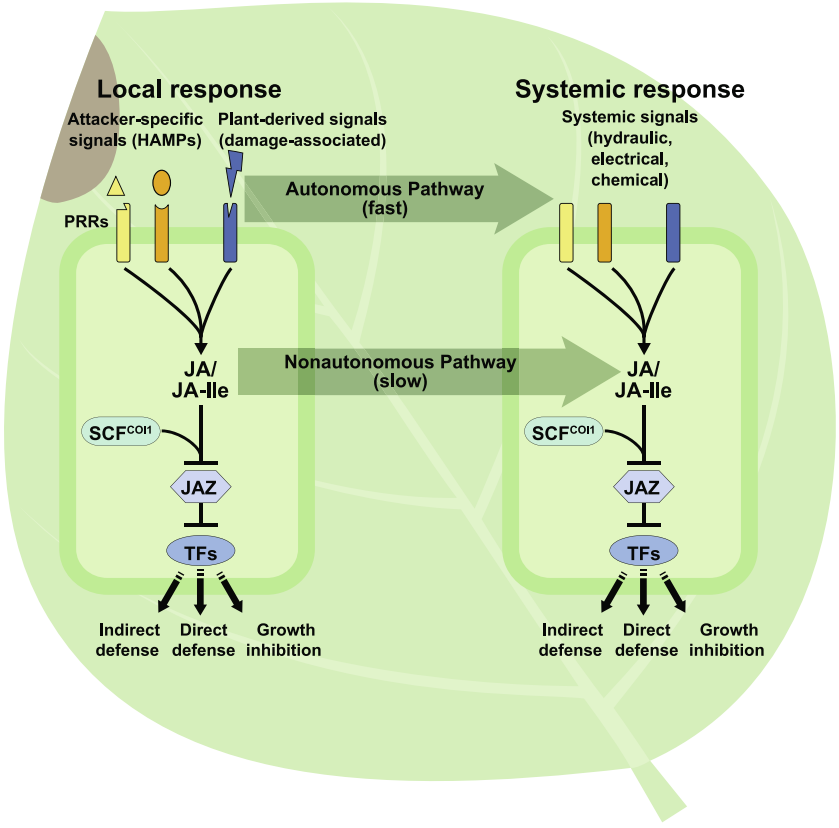

Fig. 2. Role of JA in plant responses to tissue injury. Local response: tissue damage results in the production of plant- and attacker-derived signals (either chemical or physical in nature) that are recognized by pattern recognition receptors (PRRs) at the cell surface. By mechanisms that are largely unknown, this recognition event activates *de novo* synthesis of JA and JA-Ile. JA-Ile is the active signal for SCF^{COI1}/26 proteasome-mediated degradation of JAZ proteins that repress transcription factors (TFs) involved in the expression of defense-related traits. Systemic response: wound-induced systemic defense responses are mediated by two distinct pathways involving JA. In the cell-nonautonomous pathway, JA (or a derivative) produced in the damaged leaf is translocated to distal sites (e.g., an undamaged leaf) where it triggers JA responses in target cells. In the cell autonomous pathway, wound-induced production of a mobile signal (other than JA) activates JA/JA-Ile synthesis and subsequent responses in distal tissues. The two pathways may work synergistically to optimize the spatial and temporal expression of responses elicited by various forms of tissue injury. For simplicity, the figure does not include pathogen- and microbe-associated molecular patterns (PAMPs/MAMPs) that may also activate JA-based defenses.

2007). Wound-induced ion fluxes have been implicated in the early steps of wound recognition, but the underlying mechanisms are still vague (Schaller and Oecking, 1999; Bonaventure et al., 2007).

5. JA action at a distance

Biotic stress often results in systemic expression or priming of host defense responses that serve to protect healthy tissues from secondary attack (Conrath et al., 2006; Frost et al., 2007; Ton et al., 2007; Erb et al., 2008; Heil and Ton, 2008; Vlot et al., 2008). A definitive role for JA in long-distance responses to wounding and herbivory was established through the use of mutants that are defective in JA synthesis or perception (Leon et al., 2001; Schilmiller and Howe, 2005; Wasternack et al., 2006). JA-dependent systemic responses are highly diverse and are expressed over various temporal and spatial scales. Rapid systemic changes in gene expression and hormone levels occur within minutes of tissue injury. Other wound responses, including the production of toxins, feeding deterrents (e.g., PIs), and volatiles that mediate tritrophic interactions, are slower (e.g., measured in hours) (Green and Ryan, 1972; Engelberth et al., 2004; Mithöfer et al., 2005; Baldwin et al., 2006; Matsui, 2006; Heil and Silva Bueno, 2007; Pauwels et al., 2009). Wound-induced developmental responses such as trichome initiation are manifested over the course of days (Yoshida et al., 2009).

There are two general models to explain how the JA pathway components are spatially organized between damaged and undamaged leaves: (i) JA produced at the wound site serves as a mobile signal to activate responses in systemic tissues, and (ii) wound-induced production of a mobile signal other than JA activates synthesis of the hormone in systemic tissues. We refer to these two scenarios as the cell non-autonomous and autonomous pathways, respectively, for what they imply about sites of JA synthesis and action. Both models predict wound-induced systemic increases in JA/JA-Ile levels and subsequent perception of JA-Ile by the SCF^{COI1}/JAZ receptor complex (Fig. 2). The models differ in several key respects, including the source of systemic JA/JA-Ile (i.e., transported or *de novo* synthesized), the requirement for JA synthesis in the production of the mobile signal and, of course, the identity of the mobile signal.

5.1. JA as a cell-non-autonomous wound signal

There is considerable evidence to support the hypothesis that JA (or a JA derivative) acts cell non-autonomously in the systemic wound response. This topic has been extensively reviewed (Stratmann, 2003; Howe, 2004; Schilmiller and Howe, 2005; Wasternack et al., 2006; Heil and Ton, 2008; Howe and Jander, 2008) and thus we summarize only the main points here. Exogenous JA exerts systemic responses in solanaceous plants (Farmer et al., 1992; Halitschke and Baldwin, 2003; Zhao et al., 2003; Pluskota et al., 2007). Moreover, isotope labeling experiments indicate that JA/MeJA can be transported in the phloem and xylem (Zhang and Baldwin, 1997; Thorpe et al., 2007). In *Arabidopsis*, exogenous JA did not elicit systemic expression of a reporter gene that is expressed in distal leaves of wounded plants (Kubigsteltig et al., 1999). Results from these and other JA application experiments are thus inconsistent and, as mentioned above, are inadequate to infer a role for endogenous JA in the wound response.

A function for JA as a mobile signal in the wound response is supported by immunocytochemical studies showing that tomato JA biosynthetic enzymes are located in the companion cell-sieve element complex of vascular tissue (Hause et al., 2000, 2003). The preferential accumulation of JA and OPDA in vascular bundles further indicates that the JA synthetic pathway is operational in these tissues (Stenzel et al., 2003a). In *Arabidopsis*, various JA derivatives are enriched in the midveins of wounded leaves (Glauser et al., 2008), and it was also shown that petiole exudates contain JA (Truman et al., 2007; Chaturvedi et al., 2008). Additional support for a phloem-mobile wound signal comes from studies showing that spatial patterns of wound-induced systemic gene expression are influenced by the strength of vascular connections between damaged and responding leaves (Davis et al., 1991; Orians et al., 2000; Schittko and Baldwin, 2003). Estimates of the speed (1–5 cm/h) of the systemic signal for wound-induced PI expression in tomato are consistent with the involvement of the phloem in signal transport (Nelson et al., 1983; Ryan, 2000). Taken together, these observations support the hypothesis that JA or one of its derivatives is a mobile signal in the systemic wound response (Fig. 2; cell non-autonomous pathway).

Grafting experiments have provided functional evidence that JA is a component of the long-distance signal for PI expression in tomato. Grafts between wild-type and COI1-deficient plants showed that JA responsiveness is not required for production of the systemic signal in damaged leaves, but rather is necessary for recognition of the signal in undamaged systemic leaves (Li et al., 2002). Experiments performed with JA synthesis mutants showed that wound-induced systemic expression of PIs depends on JA production in rootstock tissues but not in distal leaves of the scion (Li et al., 2002, 2005; Lee and Howe, 2003). The most straightforward interpretation of these results is that JA or another product of the octadecanoid pathway is a component of the transmissible wound signal for systemic PI expression (Schilmiller and Howe, 2005; Wasternack et al., 2006).

In theory, wound-induced production of volatile MeJA could provide a cell non-autonomous, airborne route for chemical communication between damaged and undamaged leaves of the same plant (Heil and Ton, 2008). However, experiments conducted with sentinel plants located in close proximity to the wounded plant showed that systemic expression of PIs in tomato is mediated by a signal traveling within the plant rather than a factor diffusing through the atmosphere (Farmer et al., 1992). In addition, wounding of *Nicotiana attenuata* leaves did not result in the release of significant quantities of MeJA (Von Dahl and Baldwin, 2004).

5.2. JA as a cell-autonomous wound signal

Recent studies in *Arabidopsis* implicate JA as a cell autonomous wound signal for rapid systemic changes in gene expression. Mechanical tissue damage inflicted on rosette leaves resulted in a large (~10-fold) and rapid (within 5 min) increase in JA and JA-Ile levels in undamaged leaves of the rosette (Glauser et al., 2008; Koo et al., 2009). The systemic increase in JA-Ile preceded the onset of expression of early JA-response genes, which was detected in systemic leaves within 15 min of wounding. As predicted by current models of JA signaling, these rapid changes in gene expression were spatially and temporally correlated with degradation of JAZ proteins (Koo et al., 2009). Wound-induced systemic increases in JA-Ile levels and turnover of JAZ proteins have also been reported in other recent studies (Suza and Staswick, 2008; Wang et al., 2008; Zhang and Turner, 2008).

Systemic accumulation of JA-Ile could result either from its transport between wounded to unwounded leaves or from *de novo* synthesis in undamaged leaves in response to a long-distance signal. A transgenic system in which conversion of OPDA to JA can be spatially manipulated by dexamethasone-inducible expression of the JA synthetic enzyme OPR3 was used to distinguish these possibilities (Koo et al., 2009). Wounding did not elicit systemic JA/JA-Ile accumulation in plants that had the ability to produce the hormone in damaged (local) leaves only. However, wounding did induce systemic production of JA-Ile in plants that were complemented for

OPR3 function in the systemic undamaged leaves. Significantly, production of this systemic pool of JA-Ile occurred even in the absence of JA synthesis in the local damaged leaves. It was concluded that wound-induced systemic JA/JA-Ile accumulation in *Arabidopsis* results, at least in part, from *de novo* synthesis of these compounds in systemic leaves rather than transport from the site of wounding (Koo et al., 2009). Metabolic labeling experiments in *N. attenuata* have also provided evidence that JA-Ile is synthesized *de novo* in leaves distal to the wound site (Wang et al., 2008). These new findings are consistent with a model in which a rapidly transmitted wound signal activates systemic synthesis of JA which, upon conversion to JA-Ile, mediates expression of early response genes by the SCF^{COI1}/JAZ pathway (Fig. 2).

The speed of the wound signal responsible for triggering *de novo* JA/JA-Ile synthesis in systemic leaves was estimated to be less than 2 cm/min (Glauser et al., 2008; Koo et al., 2009). This rate of movement is at least 20-fold faster than the estimated speed of the phloem-borne signal for PI expression in tomato. A significant body of literature implicates electrical potentials and/or hydraulic forces as signals for a variety of wound-induced rapid systemic responses (Wildon et al., 1992; Malone, 1993, 1996; Seo et al., 1995; Herde et al., 1996; Stankovic and Davies, 1996, 1998; Davies et al., 1997; Coker et al., 2005; Beaubois et al., 2007; Zimmermann et al., 2009). In maize, hydrostatic pressure changes induced by wounding affect stomatal aperture in systemic leaves within seconds (Raschke, 1970). An abscisic acid-independent, rapid hydraulic signal plays a role in communicating root-to-shoot water status in tomato and *Arabidopsis* (Holbrook et al., 2002; Christmann et al., 2007). Based on studies performed in the tomato system, Malone proposed a hydraulic dispersal mechanism in which rapid wound-induced changes in hydraulic pressure generate a basipetal mass flow that delivers chemical elicitors to distal leaves via the xylem, in a time frame that can account for rapid (e.g., within 20 min of wounding) activation of gene expression (Malone, 1993; Malone and Alarcon, 1995). In accordance with the involvement of a rapid physical signal in systemic wound responses in *Arabidopsis*, simple excision of petioles of undamaged leaves was sufficient to cause rapid systemic changes in JA/JA-Ile levels and gene expression (Koo et al., 2009).

These recent studies indicate that all components of the JA pathway, ranging from the production of JA precursors to activation of JA-response genes, are actively engaged in systemic undamaged leaves of the *Arabidopsis* rosette. In referring to this mode of JA action as the cell autonomous pathway (Fig. 2), we assume for simplicity that systemic synthesis and subsequent action of JA-Ile occurs in the same responding cell. A potential clue to the mechanism by which *de novo* JA synthesis is activated in distal responding cells came from experiments showing that the steep rise in systemic JA-Ile levels correlated with a rapid decline in levels of free OPDA (Koo et al., 2009). Although a precursor-product relationship was not established, the magnitude of the decline in OPDA content during the first 5 min after wounding was sufficient to account for the systemic increase in JA-Ile. These findings suggest that, in *Arabidopsis*, OPDA metabolism may be a control point for JA/JA-Ile production in distal leaves.

The speed with which wounding triggers systemic changes in OPDA and JA levels suggests rapid mobilization of a JA precursor or a post-translational event that activates a pre-existing biosynthetic enzyme. Previous studies in tomato and tobacco have shown that leaf wounding produces a rapid xylem-borne signal that activates MAP kinases in systemic leaves (Seo et al., 1995; Stratmann and Ryan, 1997). Reversible phosphorylation is also implicated in the control of JA-dependent wound responses (Rojo et al., 1998; Seo et al., 1999; Kandoth et al., 2007; Wu et al., 2007). Potential points of post-translational regulation of OPDA metabolism include transport of OPDA into peroxisomes via the ATP-binding cassette transporter COMATOSE (Theodoulou et al., 2005) or peroxisomal matrix enzymes involved in the conversion of OPDA to JA. Structural studies showing that OPR3 forms a self-inhibited dimer provide evidence that the *in vivo* activity of OPR3 may be regulated by phosphorylation (Breithaupt et al., 2006).

6. A new perception of the wound response

The occurrence of distinct JA-dependent wound response pathways (Fig. 2), together with the well established but poorly understood phenomenon of JA-independent wound signaling (Leon et al., 2001; LeBrasseur et al., 2002; Howe, 2004), indicates that the mechanisms by which plants perceive and respond to tissue injury are highly complex. It seems clear that the final outcome of the plant wound response varies depending on the developmental stage of the plant, the specific nature of the threat, and the environmental conditions under which the threat is encountered. Multiple systemic signaling pathways may interact functionally with one another to optimize the plant's response to different types of wound stress or during different stages of plant development. It is possible, for example, that rapid elicitation of systemic responses via the cell autonomous pathway primes undamaged tissues for enhanced resistance to secondary attacks. A similar model was recently proposed for priming of rhizobacteria-induced systemic resistance in *Arabidopsis* (Pozo et al., 2008). Rapidly induced systemic responses may also prime undamaged tissues for increased responsiveness to a slower moving, transmissible JA signal. Such a two-step mechanism may allow plants to better discriminate between real (e.g., insect attack) threats and false alarms, and to fine tune the systemic response to a particular type of aggressor. This view is consistent with recent studies (Wu et al., 2007) indicating that wound-induced systemic responses in *N. attenuata* involve multiple long-distance signaling systems that operate at different temporal and spatial scales. The involvement of multiple long-distance signals in controlling systemic PI expression in tomato may help to integrate opposing theories about the chemical *versus* physical nature of the systemic signal in this classic model system (Wildon et al., 1992; Malone and Alarcon, 1995; Ryan, 2000).

7. Moving forward

A wealth of information establishes JA as a wound hormone in land plants. Nevertheless, several major gaps in our understanding of how JA exerts its local and systemic effects in injured plants remain to be filled. Further characterization of rapid wound signals and elucidation of the mechanisms by which they are perceived in responding cells is centrally important. A related challenge is to determine how early signaling events at the cell surface are linked to activation of the JA biosynthetic pathway. Among the earliest biochemical events implicated in response to tissue damage are changes in plasma transmembrane potential and Ca^{+2} concentration (Maffei et al., 2007; Zimmermann et al., 2009; Knight et al., 1991; Lecourieux et al., 2006; Bonaventure et al., 2007), generation of reactive oxygen species (Orozco-Cardenas et al., 2001; Foyer and Noctor, 2005; Almagro et al., 2009), activation of mechanosensitive ion channels and cytoskeletal-linked stretch sensors (Kung, 2005; Nakagawa et al., 2007; Haswell et al., 2008; Chehab et al., 2009; Na et al., 2008), and post-translational protein modification (Seo et al., 1995; Stratmann and Ryan, 1997). It remains to be determined which of these processes are directly involved in activating JA synthesis in response to wounding. It will be equally important to determine the biochemical mechanisms involved in signal inactivation. Additional work is needed, for example, to determine whether enzymatic modification of JA-Ile (Miersch et al., 2008; Glauser et al., 2008), epimerization of (3*R*,7*S*)-JA-Ile (Fonseca

et al., 2009), production of stabilized JAZ proteins (Chung and Howe, 2009), or other mechanisms play a role in attenuating wound responses.

Continuing research to discern the inner workings of the SCF^{COI1}/JAZ complex (Chung et al., 2009) will enhance our understanding of F-box proteins as intracellular sensors of wound stress. The identification of bioactive forms of the hormone (Thines et al., 2007; Fonseca et al., 2009) places increasing importance on efforts to quantify the endogenous level of these compounds, including specific stereoisomers, in healthy and stressed tissues. Because wound-induced JA signal output is determined largely by the intracellular concentration of JA-Ile (Koo et al., 2009), there is a clear need to increase our understanding of the cellular mechanisms of JA/JA-Ile homeostasis.

These mechanistic questions concerning JA-dependent wound responses should be complemented by parallel studies at the whole plant and ecological levels. Although it is firmly established that JA-mediated responses to tissue injury are critical for plant fitness in natural environments, the relevance of the cell autonomous and non-autonomous pathways of JA signaling in plant interactions with insects and pathogens is largely unknown. Identification of receptors for herbivore- and pathogen-derived signals that modulate the JA response will be a key part of this effort, as will elucidation of the molecular basis of cross-talk between JA and other hormone signaling pathways. Undoubtedly, progress in all of these research areas will help to address the fundamental question of how plants recognize and respond to tissue injury.

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References

Almagro, L., Gómez Ros, L.V., Belchi-Navarro, S., Bru, R., Ros Barceló, A., Pedreño, M.A., 2009. Class III peroxidases in plant defence reactions. J. Exp. Bot. 60, 377–390.

Arimura, G., Huber, D.P., Bohlmann, J., 2004a. Forest tent caterpillars (*Malacosoma disstria*) induce local and systemic diurnal emissions of terpenoid volatiles in hybrid poplar (*Populus trichocarpa* × *deltoides*): cDNA cloning, functional characterization, and patterns of gene expression of (−)-germacrene D synthase, PtdTPS1. Plant J. 37, 603–616.

Arimura, G., Ozawa, R., Kugimiya, S., Takabayashi, J., Bohlmann, J., 2004b. Herbivore-induced defense response in a model legume. Two-spotted spider mites induce emission of (*E*)-β-ocimene and transcript accumulation of (*E*)-β-ocimene synthase in *Lotus japonicus*. Plant Physiol. 135, 1976–1983.

Balbi, V., Devoto, A., 2008. Jasmonate signalling network in *Arabidopsis thaliana*: crucial regulatory nodes and new physiological scenarios. New Phytol. 177, 301–318.

Baldwin, I.T., 1998. Jasmonate-induced responses are costly but benefit plants under attack in native populations. Proc. Natl. Acad. Sci. USA 95, 8113–8118.

Baldwin, I.T., Halitschke, R., Paschold, A., von Dahl, C.C., Preston, C.A., 2006. Volatile signaling in plant–plant interactions: 'talking trees' in the genomics era. Science 311, 812–815.

Beaubois, E., Girard, S., Lallechere, S., Davies, E., Paladian, F., Bonnet, P., Ledoigt, G., Vian, A., 2007. Intercellular communication in plants: evidence for two rapidly transmitted systemic signals generated in response to electromagnetic field stimulation in tomato. Plant Cell Environ. 30, 834–844.

Bergey, D.R., Howe, G.A., Ryan, C.A., 1996. Polypeptide signaling for plant defensive genes exhibits analogies to defense signaling in animals. Proc. Natl. Acad. Sci. USA 93, 12053–12058.

Blechert, S., Brodschelm, W., Holder, S., Kammerer, L., Kutchan, T.M., Mueller, M.J., Xia, Z.Q., Zenk, M.H., 1995. The octadecanoic pathway: signal molecules for the regulation of secondary pathways. Proc. Natl. Acad. Sci. USA 92, 4099–4105.

Blechert, S., Bockelmann, C., Fusslein, M., Von Schrader, T., Stelmach, B., Niesel, U., Weiler, E.W., 1999. Structure–activity analyses reveal the existence of two separate groups of active octadecanoids in elicitation of the tendril-coiling response of *Bryonia dioica* Jacq. Planta 207, 470–479.

Blee, E., 2002. Impact of phyto-oxylipins in plant defense. Trends Plant Sci. 7, 315–322.

Boller, T., He, S.Y., 2009. Innate immunity in plants: an arms race between pattern recognition receptors in plants and effectors in microbial pathogens. Science 324, 742–744.

Bonaventure, G., Gfeller, A., Proebsting, W.M., Hortensteiner, S., Chetelat, A., Martinoia, E., Farmer, E.E., 2007. A gain-of-function allele of TPC1 activates oxylipin biogenesis after leaf wounding in *Arabidopsis*. Plant J. 49, 889–898.

Breithaupt, C., Kurzbauer, R., Lilie, H., Schaller, A., Strassner, J., Huber, R., Macheroux, P., Clausen, T., 2006. Crystal structure of 12-oxophytodienoate reductase 3 from tomato: self-inhibition by dimerization. Proc. Natl. Acad. Sci. USA 103, 14337–14342.

Browse, J., 2009. Jasmonate passes muster: a receptor and targets for the defense hormone. Annu. Rev. Plant Biol. 60, 183–205.

Browse, J., Howe, G.A., 2008. Update on jasmonate signaling: new weapons and a rapid response against insect attack. Plant Physiol. 146, 832–838.

Chaturvedi, R., Krothapalli, K., Makandar, R., Nandi, A., Sparks, A.A., Roth, M.R., Welti, R., Shah, J., 2008. Plastid omega3-fatty acid desaturase-dependent accumulation of a systemic acquired resistance inducing activity in petiole exudates of *Arabidopsis thaliana* is independent of jasmonic acid. Plant J. 54, 106–117.

Chehab, E.W., Eich, E., Braam, J., 2009. Thigmomorphogenesis: a complex plant response to mechano-stimulation. J. Exp. Bot. 60, 43–56.

Chini, A., Fonseca, S., Fernández, G., Adie, B., Chico, J.M., Lorenzo, O., García-Casado, G., López-Vidriero, I., Lozano, F.M., Ponce, M.R., Micol, J.L., Solano, R., 2007. The JAZ family of repressors is the missing link in jasmonate signalling. Nature 448, 666–671.

Chini, A., Fonseca, S., Chico, J., Fernández-Calvo, P., Solano, R., 2009. The ZIM domain mediates homo- and heteromeric interactions between *Arabidopsis* JAZ proteins. Plant J. 59, 77–87.

Christmann, A., Weiler, E.W., Steudle, E., Grill, E., 2007. A hydraulic signal in root-to-shoot signalling of water shortage. Plant J. 52, 167–174.

Chung, H.S., Howe, G.A., 2009. A critical role for the TIFY motif in repression of jasmonate signaling by a stabilized splice variant of the JASMONATE ZIM-domain protein JAZ10 in *Arabidopsis*. Plant Cell 21, 131–145.

Chung, H.S., Koo, A.J.K., Gao, X., Jayanty, S., Thines, B., Jones, A.D., Howe, G.A., 2008. Regulation and function of *Arabidopsis JASMONATE ZIM*-domain genes in response to wounding and herbivory. Plant Physiol. 146, 952–964.

Chung, H.S., Niu, Y., Browse, J., Howe, G.A., 2009. Top hits in contemporary JAZ: new information on jasmonate signaling. Phytochemistry. 70, 1547–1559.

Coker, J.S., Vian, A., Davies, E., 2005. Identification, accumulation, and functional prediction of novel tomato transcripts systemically upregulated after fire damage. Physiol. Plant. 124, 311–322.

Conrath, U., Beckers, G.J., Flors, V., Garcia-Agustin, P., Jakab, G., Mauch, F., Newman, M.A., Pieterse, C.M., Poinssot, B., Pozo, M.J., Pugin, A., Schaffrath, U., Ton, J., Wendehenne, D., Zimmerli, L., Mauch-Mani, B., 2006. Priming: getting ready for battle. Mol. Plant Microbe Interact. 19, 1062–1071.

Creelman, R.A., Mullet, J.E., 1997. Biosynthesis and action of jasmonates in plants. Annu. Rev. Plant Physiol. Plant. Mol. Biol. 48, 355–381.

Davies, E., Vian, A., Vian, C., Stankovic, B., 1997. Rapid systemic up-regulation of genes after heat-wounding and electrical stimulation. Acta Physiol. Plant. 19, 571–576.

Davis, J.M., Gordon, M.P., Smit, B.A., 1991. Assimilate movement dictates remote sites of wound-induced gene-expression in poplar leaves. Proc. Natl. Acad. Sci. USA 88, 2393–2396.

De Moraes, C.M., Mescher, M.C., Tumlinson, J.H., 2001. Caterpillar-induced nocturnal plant volatiles repel conspecific females. Nature 410, 577–580.

de Vos, M., van Oosten, V.R., van Poecke, R.M., van Pelt, J.A., Pozo, M.J., Mueller, M.J., Buchala, A.J., Metraux, J.P., van Loon, L.C., Dicke, M., Pieterse, C.M., 2005. Signal signature and transcriptome changes of *Arabidopsis* during pathogen and insect attack. Mol. Plant Microbe Interact. 18, 923–937.

Dharmasiri, N., Dharmasiri, S., Jones, A.M., Estelle, M., 2003. Auxin action in a cell-free system. Curr. Biol. 13, 1418–1422.

Dicke, M., van Loon, J.J., Soler, R., 2009. Chemical complexity of volatiles from plants induced by multiple attack. Nat. Chem. Biol. 5, 317–324.

Doares, S.H., Syrovets, T., Weiler, E.W., Ryan, C.A., 1995. Oligogalacturonides and chitosan activate plant defensive genes through the octadecanoid pathway. Proc. Natl. Acad. Sci. USA 92, 4095–4098.

Ellis, C., Karafyllidis, I., Wasternack, C., Turner, J.G., 2002. The *Arabidopsis* mutant *cev1* links cell wall signaling to jasmonate and ethylene responses. Plant Cell 14, 1557–1566.

Engelberth, J., Alborn, H.T., Schmelz, E.A., Tumlinson, J.H., 2004. Airborne signals prime plants against insect herbivore attack. Proc. Natl. Acad. Sci. USA 101, 1781–1785.

English, J., Bonner, J., Haagen-Smit, A., 1939. Structure and synthesis of a plant wound hormone. Science 90, 329.

Erb, M., Ton, J., Degenhardt, J., Turlings, T.C., 2008. Interactions between arthropod-induced aboveground and belowground defenses in plants. Plant Physiol. 146, 867–874.

Farmer, E.E., 1994. Fatty acid signalling in plants and their associated microorganisms. Plant Mol. Biol. 26, 1423–1437.

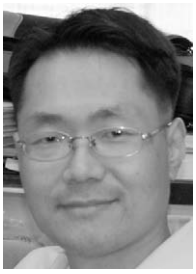
Farmer, E.E., Ryan, C.A., 1990. Interplant communication: airborne methyl jasmonate induces synthesis of proteinase inhibitors in plant leaves. Proc. Natl. Acad. Sci. USA 87, 7713–7716.

Farmer, E.E., Johnson, R.R., Ryan, C.A., 1992. Regulation of expression of proteinase-inhibitor genes by methyl jasmonate and jasmonic acid. Plant Physiol. 98, 995–1002.

Felton, G.W., Tumlinson, J.H., 2008. Plant-insect dialogs: complex interactions at the plant-insect interface. Curr. Opin. Plant Biol. 11, 457–463.
Feussner, I., Wasternack, C., 2002. The lipoxygenase pathway. Annu. Rev. Plant Biol. 53, 275–297.
Feys, B., Benedetti, C.E., Penfold, C.N., Turner, J.G., 1994. *Arabidopsis* mutants selected for resistance to the phytotoxin coronatine are male sterile, insensitive to methyl jasmonate, and resistant to a bacterial pathogen. Plant Cell 6, 751–759.
Fonseca, S., Chini, A., Hamberg, M., Adie, B., Porzel, A., Kramell, R., Miersch, O., Wasternack, C., Solano, R., 2009. (+)-7-*iso*-Jasmonoyl-L-isoleucine is the endogenous bioactive jasmonate. Nat. Chem. Biol. 5, 344–350.
Foyer, C.H., Noctor, G., 2005. Redox homeostasis and antioxidant signaling: a metabolic interface between stress perception and physiological responses. Plant Cell 17, 1866–1875.
Frost, C.J., Appel, H.M., Carlson, J.E., De Moraes, C.M., Mescher, M.C., Schultz, J.C., 2007. Within-plant signalling via volatiles overcomes vascular constraints on systemic signalling and primes responses against herbivores. Ecol. Lett. 10, 490–498.
Glauser, G., Grata, E., Dubugnon, L., Rudaz, S., Farmer, E.E., Wolfender, J.L., 2008. Spatial and temporal dynamics of jasmonate synthesis and accumulation in *Arabidopsis* in response to wounding. J. Biol. Chem. 283, 16400–16407.
Green, T.R., Ryan, C.A., 1972. Wound-induced proteinase inhibitor in plant leaves – possible defense mechanism against insects. Science 175, 776–777.
Gundlach, H., Müller, M.J., Kutchan, T.M., Zenk, M.H., 1992. Jasmonic acid is a signal transducer in elicitor-induced plant cell cultures. Proc. Natl. Acad. Sci. USA 89, 2389–2393.
Halitschke, R., Baldwin, I.T., 2003. Antisense LOX expression increases herbivore performance by decreasing defense responses and inhibiting growth-related transcriptional reorganization in *Nicotiana attenuata*. Plant J. 36, 794–807.
Hamann, T., Bennett, M., Mansfield, J., Somerville, C., 2009. Identification of cell-wall stress as a hexose-dependent and osmosensitive regulator of plant responses. Plant J. 57, 1015–1026.
Hamberg, M., Gardner, H.W., 1992. Oxylipin pathway to jasmonates: biochemistry and biological significance. Biochim. Biophys. Acta 1165, 1–18.
Haswell, E.S., Peyronnet, R., Barbier-Brygoo, H., Meyerowitz, E.M., Frachisse, J.M., 2008. Two MscS homologs provide mechanosensitive channel activities in the *Arabidopsis* root. Curr. Biol. 18, 730–734.
Hause, B., Stenzel, I., Miersch, O., Maucher, H., Kramell, R., Ziegler, J., Wasternack, C., 2000. Tissue-specific oxylipin signature of tomato flowers: allene oxide cyclase is highly expressed in distinct flower organs and vascular bundles. Plant J. 24, 113–126.
Hause, B., Hause, G., Kutter, C., Miersch, O., Wasternack, C., 2003. Enzymes of jasmonate biosynthesis occur in tomato sieve elements. Plant Cell Physiol. 44, 643–648.
Heil, M., Silva Bueno, J.C., 2007. Within-plant signaling by volatiles leads to induction and priming of an indirect plant defense in nature. Proc. Natl. Acad. Sci. USA 104, 5467–5472.
Heil, M., Ton, J., 2008. Long-distance signalling in plant defence. Trends Plant Sci. 13, 264–272.
Herde, O., Atzorn, R., Fisahn, J., Wasternack, C., Willmitzer, L., Pena-Cortes, H., 1996. Localized wounding by heat initiates the accumulation of proteinase inhibitor II in abscisic acid-deficient plants by triggering jasmonic acid biosynthesis. Plant Physiol. 112, 853–860.
Herms, D.A., Mattson, W.J., 1992. The dilemma of plants – to grow or defend. Q. Rev. Biol. 67, 283–335.
Holbrook, N.M., Shashidhar, V.R., James, R.A., Munns, R., 2002. Stomatal control in tomato with ABA-deficient roots: response of grafted plants to soil drying. J. Exp. Bot. 53, 1503–1514.
Howe, G.A., 2004. Jasmonates as signals in the wound response. J. Plant Growth Regul. 23, 223–237.
Howe, G., Jander, G., 2008. Plant immunity to insect herbivores. Annu. Rev. Plant Biol. 59, 41–66.
Howe, G.A., Schilmiller, A.L., 2002. Oxylipin metabolism in response to stress. Curr. Opin. Plant Biol. 5, 230–236.
Howe, G.A., Lightner, J., Browse, J., Ryan, C.A., 1996. An octadecanoid pathway mutant (JL5) of tomato is compromised in signaling for defense against insect attack. Plant Cell 8, 2067–2077.
Howe, G.A., Lee, G.I., Itoh, A., Li, L., DeRocher, A.E., 2000. Cytochrome P450-dependent metabolism of oxylipins in tomato. Cloning and expression of allene oxide synthase and fatty acid hydroperoxide lyase. Plant Physiol. 123, 711–724.
Hyun, Y., Choi, S., Hwang, H.J., Yu, J., Nam, S.J., Ko, J., Park, J.Y., Seo, Y.S., Kim, E.Y., Ryu, S.B., Kim, W.T., Lee, Y.H., Kang, H., Lee, I., 2008. Cooperation and functional diversification of two closely related galactolipase genes for jasmonate biosynthesis. Dev. Cell 14, 183–192.
Ishiguro, S., Kawai-Oda, A., Ueda, J., Nishida, I., Okada, K., 2001. The DEFECTIVE IN ANTHER DEHISCIENCE 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.
Ito, T., Ng, K.H., Lim, T.S., Yu, H., Meyerowitz, E.M., 2007. The homeotic protein AGAMOUS controls late stamen development by regulating a jasmonate biosynthetic gene in *Arabidopsis*. Plant Cell 19, 3516–3529.
Jones, J.D., Dangl, J.L., 2006. The plant immune system. Nature 444, 323–329.
Kandoth, P.K., Ranf, S., Pancholi, S.S., Jayanty, S., Walla, M.D., Miller, W., Howe, G.A., Lincoln, D.E., Stratmann, J.W., 2007. Tomato MAPKs LeMPK1, LeMPK2, and LeMPK3 function in the systemin-mediated defense response against herbivorous insects. Proc. Natl. Acad. Sci. USA 104, 12205–12210.
Karban, R., Baldwin, I.T., 1997. Induced Responses to Herbivory. University of Chicago Press, Chicago.
Katsir, L., Chung, H.S., Koo, A.J.K., Howe, G.A., 2008a. Jasmonate signaling: a conserved mechanism of hormone sensing. Curr. Opin. Plant Biol. 11, 428–435.
Katsir, L., Schilmiller, A.L., Staswick, P.E., He, S.Y., Howe, G.A., 2008b. COI1 is a critical component of a receptor for jasmonate and the bacterial virulence factor coronatine. Proc. Natl. Acad. Sci. USA 105, 7100–7105.
Kessler, A., Halitschke, R., Baldwin, I.T., 2004. Silencing the jasmonate cascade: induced plant defenses and insect populations. Science 305, 665–668.
Knight, M.R., Campbell, A.K., Smith, S.M., Trewavas, A.J., 1991. Transgenic plant aequorin reports the effects of touch and cold-shock and elicitors on cytoplasmic calcium. Nature 352, 524–526.
Koo, A.J.K., Gao, X., Jones, A.D., Howe, G.A., in press. A rapid wound signal activates systemic synthesis of bioactive jasmonates in *Arabidopsis*. Plant J.
Kramell, R., Miersch, O., Hause, B., Ortel, B., Parthier, B., Wasternack, C., 1997. Amino acid conjugates of jasmonic acid induce jasmonate-responsive gene expression in barley (*Hordeum vulgare* L.) leaves. FEBS Lett. 414, 197–202.
Kramell, R., Miersch, O., Atzorn, R., Parthier, B., Wasternack, C., 2000. Octadecanoid-derived alteration of gene expression and the "oxylipin signature" in stressed barley leaves. Implications for different signaling pathways. Plant Physiol. 123, 177–188.
Kubigsteltig, I., Laudert, D., Weiler, E.W., 1999. Structure and regulation of the *Arabidopsis thaliana* allene oxide synthase gene. Planta 208, 463–471.
Kung, C., 2005. A possible unifying principle for mechanosensation. Nature 436, 647–654.
LeBrasseur, N.D., MacIntosh, G.C., Perez-Amador, M.A., Saitoh, M., Green, P.J., 2002. Local and systemic wound-induction of RNase and nuclease activities in *Arabidopsis*: RNS1 as a marker for a JA-independent systemic signaling pathway. Plant J. 29, 393–403.
Lecourieux, D., Ranjeva, R., Pugin, A., 2006. Calcium in plant defence-signalling pathways. New Phytol. 171, 249–269.
Lee, G.I., Howe, G.A., 2003. The tomato mutant *spr1* is defective in systemin perception and the production of a systemic wound signal for defense gene expression. Plant J. 33, 567–576.
Lee, D.S., Nioche, P., Hamberg, M., Raman, C.S., 2008. Structural insights into the evolutionary paths of oxylipin biosynthetic enzymes. Nature 455, 363–368.
Leon, J., Rojo, E., Sanchez-Serrano, J.J., 2001. Wound signalling in plants. J. Exp. Bot. 52, 1–9.
Li, L., Li, C., Lee, G.I., Howe, G.A., 2002. Distinct roles for jasmonate synthesis and action in the systemic wound response of tomato. Proc. Natl. Acad. Sci. USA 99, 6416–6421.
Li, L., Zhao, Y., McCaig, B.C., Wingerd, B.A., Wang, J., Whalon, M.E., Pichersky, E., Howe, G.A., 2004. The tomato homolog of CORONATINE-INSENSITIVE1 is required for the maternal control of seed maturation, jasmonate-signaled defense responses, and glandular trichome development. Plant Cell 16, 126–143.
Li, C., Schilmiller, A.L., Liu, G., Lee, G.I., Jayanty, S., Sageman, C., Vrebalov, J., Giovannoni, J.J., Yagi, K., Kobayashi, Y., Howe, G.A., 2005. Role of β-oxidation in jasmonate biosynthesis and systemic wound signaling in tomato. Plant Cell 17, 971–986.
Maffei, M.E., Mithöfer, A., Boland, W., 2007. Before gene expression: early events in plant–insect interaction. Trends Plant Sci. 12, 310–316.
Malone, M., 1993. Hydraulic signals. Phil. Trans. Royal Soc. Lond. Ser. B-Biol. Sci. 341, 33–39.
Malone, M., 1996. Rapid, long-distance signal transmission in higher plants. Adv. Bot. Res. 22, 163–228.
Malone, M., Alarcon, J.J., 1995. Only xylem-borne factors can account for systemic wound signaling in the tomato plant. Planta 196, 740–746.
Matsui, K., 2006. Green leaf volatiles: hydroperoxide lyase pathway of oxylipin metabolism. Curr. Opin. Plant Biol. 9, 274–280.
Matzinger, P., 2002. The danger model: a renewed sense of self. Science 296, 301–305.
McCloud, E.S., Baldwin, I.T., 1997. Herbivory and caterpillar regurgitants amplify the wound-induced increases in jasmonic acid but not nicotine in *Nicotiana sylvestris*. Planta 203, 430–435.
McConn, M., Creelman, R.A., Bell, E., Mullet, J.E., Browse, J., 1997. Jasmonate is essential for insect defense in *Arabidopsis*. Proc. Natl. Acad. Sci. USA 94, 5473–5477.
Melotto, M., Mecey, C., Niu, Y., Chung, H.S., Katsir, L., Yao, J., Zeng, W., Thines, B., Staswick, P.E., Browse, J., Howe, G.A., He, S.Y., 2008. The bacterial toxin coronatine and the plant hormone jasmonoyl isoleucine target the physical interaction between the *Arabidopsis* COI1 F-box protein and the Jas domain of JAZ repressor proteins. Plant J. 55, 979–988.
Miersch, O., Meyer, A., Vorkefeld, S., Sembdner, G., 1986. Occurrence of (+)-7-*iso*-jasmonic acid in *Vivia faba* L. Abd its biological activity. J. Plant Growth Regul. 5, 91–100.
Miersch, O., Kramell, R., Parthier, B., Wasternack, C., 1999. Structure–activity relations of substituted, deleted or stereospecifically altered jasmonic acid in gene expression of barley leaves. Phytochemistry 50, 353–361.
Miersch, O., Neumerkel, J., Dippe, M., Stenzel, I., Wasternack, C., 2008. Hydroxylated jasmonates are commonly occurring metabolites of jasmonic acid and contribute to a partial switch-off in jasmonate signaling. New Phytol. 177, 114–127.

Mithöfer, A., Boland, W., 2008. Recognition of herbivory-associated molecular patterns. Plant Physiol. 146, 825–831.
Mithöfer, A., Wanner, G., Boland, W., 2005. Effects of feeding *Spodoptera littoralis* on lima bean leaves. II. Continuous mechanical wounding resembling insect feeding is sufficient to elicit herbivory-related volatile emission. Plant Physiol. 137, 1160–1168.
Mueller, M.J., Brodschelm, W., Spannagl, E., Zenk, M.H., 1993. Signaling in the elicitation process is mediated through the octadecanoid pathway leading to jasmonic acid. Proc. Natl. Acad. Sci. USA 90, 7490–7494.
Na, S., Collin, O., Chowdhury, F., Tay, B., Ouyang, M., Wang, Y., Wang, N., 2008. Rapid signal transduction in living cells is a unique feature of mechanotransduction. Proc. Natl. Acad. Sci. USA 105, 6626–6631.
Nakagawa, Y., Katagiri, T., Shinozaki, K., Qi, Z., Tatsumi, H., Furuichi, T., Kishigami, A., Sokabe, M., Kojima, I., Sato, S., Kato, T., Tabata, S., Iida, K., Terashima, A., Nakano, M., Ikeda, M., Yamanaka, T., Iida, H., 2007. *Arabidopsis* plasma membrane protein crucial for Ca^{2+} influx and touch sensing in roots. Proc. Natl. Acad. Sci. USA 104, 3639–3644.
Narvaez-Vasquez, J., Orozco-Cardenas, M.L., Ryan, C.A., 2007. Systemic wound signaling in tomato leaves is cooperatively regulated by systemin and hydroxyproline-rich glycopeptide signals. Plant Mol. Biol. 65, 711–718.
Nelson, C.E., Walkersimmons, M., Makus, D., Zuroske, G., Graham, J., Ryan, C.A., 1983. Regulation of synthesis and accumulation of proteinase-inhibitors in leaves of wounded tomato plants. Acs Symp. Ser. 208, 103–122.
Orians, C.M., Pomerleau, J., Ricco, R., 2000. Vascular architecture generates fine scale variation in systemic induction of proteinase inhibitors in tomato. J. Chem. Ecol. 26, 471–485.
Orozco-Cardenas, M.L., Narvaez-Vasquez, J., Ryan, C.A., 2001. Hydrogen peroxide acts as a second messenger for the induction of defense genes in tomato plants in response to wounding, systemin, and methyl jasmonate. Plant Cell 13, 179–191.
Paschold, A., Halitschke, R., Baldwin, I.T., 2007. Co(i)-ordinating defenses: NaCOI1 mediates herbivore-induced resistance in *Nicotiana attenuata* and reveals the role of herbivore movement in avoiding defenses. Plant J. 51, 79–91.
Pauwels, L., Inzé, D., Goossens, A., 2009. Jasmonate-inducible gene: what does it mean? Plant J. 14, 87–91.
Pieterse, C.M., Leon-Reyes, A., Van der Ent, S., Van Wees, S.C., 2009. Networking by small-molecule hormones in plant immunity. Nat. Chem. Biol. 5, 308–316.
Pluskota, W.E., Qu, N., Maitrejean, M., Boland, W., Baldwin, I.T., 2007. Jasmonates and its mimics differentially elicit systemic defence responses in *Nicotiana attenuata*. J. Exp. Bot. 58, 4071–4082.
Pozo, M.J., Van Der Ent, S., Van Loon, L.C., Pieterse, C.M., 2008. Transcription factor MYC2 is involved in priming for enhanced defense during rhizobacteria-induced systemic resistance in *Arabidopsis thaliana*. New Phytol. 180, 511–523.
Raschke, K., 1970. Leaf hydraulic system: rapid epidermal and stomatal responses to changes in water supply. Science 167, 189–191.
Ribot, C., Zimmerli, C., Farmer, E.E., Reymond, P., Poirier, Y., 2008. Induction of the *Arabidopsis* PHO1;H10 gene by 12-oxo-phytodienoic acid but not jasmonic acid via a CORONATINE INSENSITIVE1-dependent pathway. Plant Physiol. 147, 696–706.
Rojo, E., Titarenko, E., Leon, J., Berger, S., Vancanneyt, G., Sanchez-Serrano, J.J., 1998. Reversible protein phosphorylation regulates jasmonic acid-dependent and -independent wound signal transduction pathways in *Arabidopsis thaliana*. Plant J. 13, 153–165.
Ryan, C.A., 2000. The systemin signaling pathway: differential activation of plant defensive genes. Biochim. Biophys. Acta 1477, 112–121.
Santner, A., Calderon-Villalobos, L.I., Estelle, M., 2009. Plant hormones are versatile chemical regulators of plant growth. Nat. Chem. Biol. 5, 301–307.
Schaller, A., Oecking, C., 1999. Modulation of plasma membrane H+-ATPase activity differentially activates wound and pathogen defense responses in tomato plants. Plant Cell 11, 263–272.
Schilmiller, A.L., Howe, G.A., 2005. Systemic signaling in the wound response. Curr. Opin. Plant Biol. 8, 369–377.
Schittko, U., Baldwin, I.T., 2003. Constraints to herbivore-induced systemic responses: bidirectional signaling along orthostichies in *Nicotiana attenuata*. J. Chem. Ecol. 29, 763–770.
Schmelz, E.A., Carroll, M.J., LeClere, S., Phipps, S.M., Meredith, J., Chourey, P.S., Alborn, H.T., Teal, P.E., 2006. Fragments of ATP synthase mediate plant perception of insect attack. Proc. Natl. Acad. Sci. USA 103, 8894–8899.
Schmelz, E.A., Engelberth, J., Alborn, H.T., Tumlinson 3rd, J.H., Teal, P.E., 2009. Phytohormone-based activity mapping of insect herbivore-produced elicitors. Proc. Natl. Acad. Sci. USA 106, 653–657.
Seo, S., Okamoto, M., Seto, H., Ishizuka, K., Sano, H., Ohashi, Y., 1995. Tobacco MAP kinase: a possible mediator in wound signal transduction pathways. Science 270, 1988–1992.
Seo, S., Sano, H., Ohashi, Y., 1999. Jasmonate-based wound signal transduction requires activation of WIPK, a tobacco mitogen-activated protein kinase. Plant Cell 11, 289–298.
Seong, S.Y., Matzinger, P., 2004. Hydrophobicity: an ancient damage-associated molecular pattern that initiates innate immune responses. Nat. Rev. Immunol. 4, 469–478.
Stankovic, B., Davies, E., 1996. Both action potentials and variation potentials induce proteinase inhibitor gene expression in tomato. FEBS. Lett. 390, 275–279.
Stankovic, B., Davies, E., 1998. The wound response in tomato involves rapid growth and electrical responses, systemically up-regulated transcription of proteinase inhibitor and calmodulin and down-regulated translation. Plant Cell Physiol. 39, 268–274.
Staswick, P.E., 2008. JAZing up jasmonate signaling. Trends Plant Sci. 13, 66–71.
Staswick, P.E., Tiryaki, I., 2004. The oxylipin signal jasmonic acid is activated by an enzyme that conjugates it to isoleucine in *Arabidopsis*. Plant Cell 16, 2117–2127.
Stenzel, I., Hause, B., Maucher, H., Pitzschke, A., Miersch, O., Ziegler, J., Ryan, C.A., Wasternack, C., 2003a. Allene oxide cyclase dependence of the wound response and vascular bundle-specific generation of jasmonates in tomato – amplification in wound signalling. Plant J. 33, 577–589.
Stenzel, I., Hause, B., Miersch, O., Kurz, T., Maucher, H., Weichert, H., Ziegler, J., Feussner, I., Wasternack, C., 2003b. Jasmonate biosynthesis and the allene oxide cyclase family of *Arabidopsis thaliana*. Plant Mol. Biol. 51, 895–911.
Stintzi, A., Weber, H., Reymond, P., Browse, J., Farmer, E.E., 2001. Plant defense in the absence of jasmonic acid: the role of cyclopentenones. Proc. Natl. Acad. Sci. USA 98, 12837–12842.
Stork, W., Diezel, C., Halitschke, R., Galis, I., Baldwin, I.T., 2009. An ecological analysis of the herbivory-elicited JA burst and its metabolism: plant memory processes and predictions of the moving target model. PLoS ONE 4, e4697.
Strassner, J., Schaller, F., Frick, U.B., Howe, G.A., Weiler, E.W., Amrhein, N., Macheroux, P., Schaller, A., 2002. Characterization and cDNA-microarray expression analysis of 12-oxophytodienoate reductases reveals differential roles for octadecanoid biosynthesis in the local versus the systemic wound response. Plant J. 32, 585–601.
Stratmann, J.W., 2003. Long distance run in the wound response-jasmonic acid is pulling ahead. Trends Plant Sci. 8, 247–250.
Stratmann, J.W., Ryan, C.A., 1997. Myelin basic protein kinase activity in tomato leaves is induced systemically by wounding and increases in response to systemin and oligosaccharide elicitors. Proc. Natl. Acad. Sci. USA 94, 11085–11089.
Suza, W.P., Staswick, P.E., 2008. The role of JAR1 in jasmonoyl-L-isoleucine production in *Arabidopsis* wound response. Planta 227, 1221–1232.
Theodoulou, F.L., Job, K., Slocombe, S.P., Footitt, S., Holdsworth, M., Baker, A., Larson, T.R., Graham, I.A., 2005. Jasmonoic acid levels are reduced in COMATOSE ATP-binding cassette transporter mutants. Implications for transport of jasmonate precursors into peroxisomes. Plant Physiol. 137, 835–840.
Thines, B., Katsir, L., Melotto, M., Niu, Y., Mandaokar, A., Liu, G., Nomura, K., He, S.Y., Howe, G.A., Browse, J., 2007. JAZ repressor proteins are targets of the SCF(COI1) complex during jasmonate signalling. Nature 448, 661–665.
Thorpe, M.R., Ferrieri, A.P., Herth, M.M., Ferrieri, R.A., 2007. C-11-imaging: methyl jasmonate moves in both phloem and xylem, promotes transport of jasmonate, and of photoassimilate even after proton transport is decoupled. Planta 226, 541–551.
Ton, J., D'Alessandro, M., Jourdie, V., Jakab, G., Karlen, D., Held, M., Mauch-Mani, B., Turlings, T.C., 2007. Priming by airborne signals boosts direct and indirect resistance in maize. Plant J. 49, 16–26.
Truitt, C.L., Wei, H.X., Pare, P.W., 2004. A plasma membrane protein from *Zea mays* binds with the herbivore elicitor volicitin. Plant Cell 16, 523–532.
Truman, W., Bennett, M.H., Kubigsteltig, I., Turnbull, C., Grant, M., 2007. *Arabidopsis* systemic immunity uses conserved defense signaling pathways and is mediated by jasmonates. Proc. Natl. Acad. Sci. USA 104, 1075–1080.
Turlings, T.C.J., Tumlinson, J.H., Lewis, W.J., 1990. Exploitation of herbivore-induced plant odors by host-seeking parasitic wasps. Science 250, 1251–1253.
Vick, B., Zimmerman, D., 1984. Biosynthesis of jasmonic acid by several plant species. Plant Physiol. 75, 458–461.
Vijayan, P., Shockey, J., Levesque, C.A., Cook, R.J., Browse, J., 1998. A role for jasmonate in pathogen defense of *Arabidopsis*. Proc. Natl. Acad. Sci. USA 95, 7209–7214.
Vlot, A.C., Klessig, D.F., Park, S.W., 2008. Systemic acquired resistance the elusive signal(s). Curr. Opin. Plant Biol. 11, 436–442.
Von Dahl, C.C., Baldwin, I.T., 2004. Methyl jasmonate and cis-jasmone do not dispose of the herbivore-induced jasmonate burst in *Nicotiana attenuata*. Physiol. Plant. 120, 474–481.
Wang, L., Halitschke, R., Kang, J.H., Berg, A., Harnisch, F., Baldwin, I.T., 2007. Independently silencing two JAR family members impairs levels of trypsin proteinase inhibitors but not nicotine. Planta 226, 159–167.
Wang, L., Allmann, S., Wu, J., Baldwin, I.T., 2008. Comparisons of LOX3- and JAR4/6-silenced plants reveal that JA and JA-AA conjugates play different roles in herbivore resistance of *Nicotiana attenuata*. Plant Physiol. 146, 904–915.
Wasternack, C., 2007. Jasmonates: an update on biosynthesis, signal transduction and action in plant stress response, growth and development. Ann. Bot. (Lond.) 100, 681–697.
Wasternack, C., Ortel, B., Miersch, O., Kramell, R., Beale, M., Greulich, F., Feussner, I., Hause, B., Krumm, T., Boland, W., Partheir, B., 1998. Diversity of octadecanoid-induced gene expression of tomato. J. Plant Physiol. 152, 345–352.
Wasternack, C., Stenzel, I., Hause, B., Hause, G., Kutter, C., Maucher, H., Neumerkel, J., Feussner, I., Miersch, O., 2006. The wound response in tomato – role of jasmonic acid. J. Plant Physiol. 163, 297–306.
Weber, H., Vick, B.A., Farmer, E.E., 1997. Dinor-oxo-phytodienoic acid: a new hexadecanoid signal in the jasmonate family. Proc. Natl. Acad. Sci. USA 94, 10473–10478.
Wehnelt, B., 1927. Untersuchungen über das Wundhormon der Pflanzen. Jahrb. wiss. Bot. 66, 773–813.
Wiesner, J., 1892. Die Elementarstructur und das Wachstum der lebenden Substanz. Vienna 102.
Wildon, D.C., Thain, J.F., Minchin, P.E.H., Gubb, I.R., Reilly, A.J., Skipper, Y.D., Doherty, H.M., Odonnell, P.J., Bowles, D.J., 1992. Electrical signaling and systemic proteinase-inhibitor induction in the wounded plant. Nature 360, 62–65.

Wu, J., Baldwin, I.T., in press. Herbivory-induced signalling in plants: perception and action. Plant Cell Environ.
Wu, J., Hettenhausen, C., Meldau, S., Baldwin, I.T., 2007. Herbivory rapidly activates MAPK signaling in attacked and unattacked leaf regions but not between leaves of *Nicotiana attenuata*. Plant Cell 19, 1096–1122.
Yan, Y., Stolz, S., Chetelat, A., Reymond, P., Pagni, M., Dubugnon, L., Farmer, E.E., 2007. A downstream mediator in the growth repression limb of the jasmonate pathway. Plant Cell 19, 2470–2483.
Yoshida, Y., Sano, R., Wada, T., Takabayashi, J., Okada, K., 2009. Jasmonic acid control of GLABRA3 links inducible defense and trichome patterning in *Arabidopsis*. Development 136, 1039–1048.
Zhang, Z.P., Baldwin, I.T., 1997. Transport of [2-C-14]jasmonic acid from leaves to roots mimics wound-induced changes in endogenous jasmonic acid pools in *Nicotiana sylvestris*. Planta 203, 436–441.
Zhang, Y., Turner, J., 2008. Wound-induced endogenous jasmonates stunt plant growth by inhibiting mitosis. PLoS ONE e3699.
Zhao, Y., Thilmony, R., Bender, C.L., Schaller, A., He, S.Y., Howe, G.A., 2003. Virulence systems of *Pseudomonas syringae* pv. tomato promote bacterial speck disease in tomato by targeting the jasmonate signaling pathway. Plant J. 36, 485–499.
Zimmerman, D.C., Coudron, C.A., 1979. Identification of traumatin, a wound hormone, as 12-oxo-*trans*-10-dodecenoic acid. Plant Physiol. 63, 536–541.
Zimmermann, M.R., Maischak, H., Mithöfer, A., Boland, W., Felle, H.H., 2009. System potentials, a novel electrical long-distance apoplastic signal in plants, induced by wounding. Plant Physiol. 149, 1593–1600.



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