JASMONATE-TRIGGERED PLANT IMMUNITY

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Abstract

The plant hormone jasmonate (JA) exerts direct control over the production of chemical defense compounds that confer resistance to a remarkable spectrum of plant-associated organisms, ranging from microbial pathogens to vertebrate herbivores. The underlying mechanism of JA-triggered immunity (JATI) can be conceptualized as a multi-stage signal transduction cascade involving: i) pattern recognition receptors (PRRs) that couple the perception of danger signals to rapid synthesis of bioactive JA; ii) an evolutionarily conserved JA signaling module that links fluctuating JA levels to changes in the abundance of transcriptional repressor proteins; and iii) activation (de-repression) of transcription factors that orchestrate the expression of myriad chemical and morphological defense traits. Multiple negative feedback loops act in concert to restrain the duration and amplitude of defense responses, presumably to mitigate potential fitness costs of JATI. The convergence of diverse plant- and non-plant-derived signals on the core JA module indicates that JATI is a general response to perceived danger. However, the modular structure of JATI may accommodate attacker-specific defense responses through evolutionary innovation of PRRs (inputs) and defense traits (outputs). The efficacy of JATI as a defense strategy is highlighted by its capacity to shape natural populations of plant attackers, as well as the propensity of plant-associated organisms to subvert or otherwise manipulate JA signaling. As both a cellular hub for integrating informational cues from the environment and a common target of pathogen effectors, the core JA module provides a focal point for understanding immune system networks and the evolution of chemical diversity in the plant kingdom.

Keywords

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Introduction

Plants are a source of nutrition for a vast biota in terrestrial environments. Selective pressure imposed by pathogens and herbivores has shaped the evolution of an astonishing array of specialized plant defense compounds that exert direct toxic, anti-nutritional, or repellant effects on plant consumers. Other defensive compounds work indirectly by attracting natural enemies of plant-associated organisms. Strategies to deploy these protective chemical shields and associated morphological structures may be constitutive or inducible. It is thought that natural selection, at least in some plant species, favored the evolution of induced defenses because they have lower resource allocation costs than constitutive resistance traits (Baldwin 1998; Herms and Mattson 1992; Thaler et al. 2012). A key feature of many induced defense traits is their expression in tissues distal to the site of infection or attack. The combined effect of local and systemic defense responses provides broad-spectrum resistance against biotic attack, and constitutes a form of induced immunity (Fu and Dong 2013; Howe and Jander 2008; Jones and Dangl 2006).

Intensive research efforts to understand the molecular mechanisms and evolutionary ecology of induced immunity have focused on the question of how plants recognize foreign threats. Significant insight has come from molecular genetic analyses of plant-pathogen interactions. Pattern-triggered immunity (PTI) confers basal resistance and is mediated by cell surface-localized pattern recognition receptors (PRRs) that bind conserved foreign molecules, known collectively as microbial/pathogen-associated molecular patterns (MAMPs) (Chisholm et al. 2006; Dodds and Rathjen 2010; Jones and Dangl 2006). A second layer of induced resistance, referred to as effector-triggered immunity (ETI), relies on polymorphic intracellular resistance (R) proteins to detect effector molecules that plant attackers deliver into host cells to counteract defense. ETI responses often include localized host cell death and are qualitatively similar, though typically more robust, than PTI responses (Dodds and Rathjen 2010). Major conceptual contributions of the PTI/ETI paradigm include the distinction between plant defense responses triggered by conserved patterns *versus* effectors, and a theory to explain how these forms of immunity drive the evolution of plant-pathogen associations (Jones and Dangl 2006). The PTI/ETI model also has influenced current views on how plants recognize attack by arthropod herbivores, which constitute the majority of plant-consuming species on Earth (Erb et al. 2012; Howe and Jander 2008). Accordingly, eliciting compounds produced by plant-eating animals have been dubbed herbivore-associated molecular patterns (HAMPs) (Felton and Tumlison 2008; Mithofer and Boland 2008).

In addition to cell surveillance systems that recognize foreign threats in the form of MAMPs/HAMPs and effectors, it has long been known that plant-derived (i.e., self) signals also are potent elicitors of local and systemic defense responses (Bergey et al. 1996; Green and Ryan 1972; Heil et al. 2012; Huffaker et al. 2006, 2011; Krol et al. 2010; Mousavi et al. 2013). These endogenous elicitors are produced in response to general cellular injury and may be classified as damage-associated molecular patterns (DAMPs). Because DAMPs are generated in response to diverse types of tissue injury, their role in cellular recognition of pathogen attack traditionally has been ignored. However, the recent identification of DAMP receptors and associated signal transduction components (Brutus et al. 2010; Choi et al

2014; Mousavi et al. 2013; Yamaguchi et al. 2006, 2010) is shaping a broader view of how plant cells perceive and respond to injurious threats (Boller and Felix 2009; ; De Lorenzo et al. 2011; Heil 2009; Koo and Howe 2009). The diversity of conserved patterns that trigger local and systemic defense reactions supports the concept that cellular perception of "danger", regardless of its source, is a unifying principle of induced immunity in plants and animals (Boller and Felix 2009; Koo and Howe 2009; Lotze et al. 2007; Matzinger 2002).

A second major question surrounding induced immunity concerns the extent to which cellular recognition of a given threat is translated into a host response that specifically neutralizes the attacking pathogen or herbivore. Indeed, genome-wide transcriptome studies indicate a significant degree of overlap in molecular responses triggered by different MAMPs/HAMPs/DAMPs and effectors (Bidart-Bouzat and Kliebenstein 2011; Caillaud et al. 2013; Gouhier-Darimont et al. 2013; Kim et al. 2014; Navarro et al. 2004; Reymond et al. 2004; Tao et al. 2003; Thilmony et al. 2006; Tsuda et al. 2008, 2009; Wise et al. 2007; Zhurov et al. 2014). There also is evidence to indicate that PTI and ETI converge on similar downstream signaling components, including MAP kinase pathways, ROS production, and calcium-dependent signaling events (Romeis and Herde 2014; Sato et al. 2010). Although quantitative differences in the timing and strength of induction are likely to shape the outcome of specific plant-attacker associations (De Vos et al. 2005; Katagiri and Tsuda 2010; Tao et al. 2003; Wise et al. 2007), most evidence indicates that specific danger signals trigger general host defense responses that are effective against broad classes of pathogens and herbivores (Erb et al. 2012).

The central role of small-molecule hormones in controlling the expression of chemical and morphological defense traits provides an impetus for describing induced immunity from the perspective of phytohormone networks (Erb et al. 2012; Pieterse et al. 2009; Reymond and Farmer 1998). It now is evident that diverse danger signals converge on the immune-promoting effects of two major defense hormones, jasmonic acid (JA) and salicylic acid (SA). Here, we focus on recent advances in understanding JA-triggered immunity (JATI). We describe JATI as a multistage process in which a highly conserved core JA module links a variety of PRR-based recognition systems (inputs) to the expression of specific defense traits (outputs). Based on these considerations, we propose that the regulatory structure of JATI has potential to create new specificities of host resistance through evolutionary innovation of input and output modules. We also highlight the various ways in which plant-associated organisms manipulate JATI to their own advantage. These new mechanistic insights will help to explain how JATI shapes patterns of chemical diversity and species interaction in the plant kingdom, and how these relationships affect genome evolution to modulate phenotypic plasticity. Our focus on JA is not intended to minimize the role of other signals in coordinating plant defense responses, or to distract from the important challenge of understanding the complexities of phytohormone networks and their relationship to induced immunity (Ballaré 2014; Kazan and Manners 2012; Kim et al. 2014; Mukhtar et al. 2011; Pieterse et al. 2009). Nevertheless, we subscribe to the view that an accurate, and ultimately predictive, understanding of interconnected signaling networks depends on knowledge of how individual signals are produced and perceived at the molecular level. Readers are referred to several excellent review articles for a comprehensive discussion of JA-mediated signal transduction, its interaction with other

signaling pathways, and the function of JA in developmental processes (Ballaré 2011; Browse 2009; Huot et al. 2014 Kazan and Manners 2008, 2012; Kombrink 2012; Meldau et al. 2012; Pauwels and Goossens 2011; Robert-Seilaniantz et al. 2011; Wasternack and Hause 2013).

JATI Confers Broad-spectrum Resistance in Dicots and Monocots

The central role of JA as an activating signal for induced immunity is grounded in three general observations. First, biotic attack and other forms of tissue injury result in the rapid synthesis of JA and its receptor-active derivative, jasmonoyl-L-isoleucine (JA-Ile). Stress-induced accumulation of JA-Ile occurs in both above- and below-ground tissues and, depending on the eliciting signal and tissue type, is a systemic response (Chauvin et al. 2013; Fragoso et al. 2014; Grebner et al. 2013; Koo et al. 2009; Mousavi et al. 2013; Schilmiller and Howe 2005). Second, JA promotes the expression of virtually all major classes of secondary metabolites and proteins that have established roles in defense, including alkaloids, terpenoids, phenylpropanoids, amino acid derivatives, anti-nutritional proteins, and some pathogenesis-related (PR) proteins (Browse and Howe 2008; De Geyter et al. 2012; De Vleesschauwer et al. 2013; Farmer and Ryan 1990; Gonzales-Vigil et al. 2011; Mohan et al. 2006; Van Loon et al. 2006). The JA pathway also promotes the development of morphological structures, including glandular trichomes, resin ducts, and nectaries that produce a rich variety of compounds serving direct and indirect roles in defense (Dicke and Baldwin 2010; Hudgins et al. 2004; Li et al. 2004; Peiffer et al. 2009; Qi et al. 2011; Radhika et al. 2010; Traw and Bergelson 2003; Van Poecke and Dicke 2002; Yoshida et al. 2009). Finally, studies employing JA mutants have demonstrated the crucial role of this hormone in plant protection against diverse biota (Browse and Howe 1998). Among the plant-associated organisms whose fitness is curtailed by JATI are necrotrophic and (hemi)biotrophic pathogens, mutualistic fungi, nematodes, leafhoppers, beetles, caterpillars, aphids, thrips, spider mites, fungus gnats, slugs, crustaceans, and some vertebrate herbivores (Table 1). Indeed, it is reasonable to think that the number of plant-eating species affected by JATI may exceed the total number of plant species on Earth.

Much of our knowledge about the protective effects of JATI comes from studies on a limited number of dicot plants, including Arabidopsis, tomato, and tobacco. These studies have led to the generalization that tissue-consuming insect herbivores and necrotrophic pathogens are particularly sensitive to JATI, whereas biotrophic organisms that obtain nutrients from living host tissues succumb to the effects of SA-triggered immunity (SATI) (Cailldaud et al. 2013; Glazebrook 2005; Pieterse et al. 2009). There is every indication that JA promotes resistance of monocot and gymnosperm species to a wide range of pathogens and herbivores (Hudgins et al. 2004; Schmelz et al. 2014; Yan et al. 2012; Ye et al. 2012; Zulac and Bohlmann 2010). In contrast to the biotroph/necrotroph dichotomy that has emerged from studies with Arabidopsis, it is noteworthy that JA is required for induced immunity of rice to (hemi)biotrophic parasites, including the root knot nematode (*Meloidogyne graminicola*) and *Xanthomonas oryzae* (De Vleesschauwer et al. 2013; Nahar et al. 2011). This conclusion is consistent with the ability of JA to elicit expression of many *PR* genes and defensive secondary metabolites in rice and maize (Mitsuhara et al. 2008; Schmelz et al. 2011, 2014; Yamane 2013;). Given these findings, together with the high endogenous levels

of free SA in rice leaves (Silverman et al. 1995), the precise role of SA as a signal for induced immunity in monocots awaits further clarification (De Vleesschauwer et al. 2013; Schmelz et al. 2014).

The serendipitous discovery that JA mutants maintained in artificial growth environments often succumb to attack by unsuspected pathogens and herbivores vividly demonstrates the robust protection afforded by JATI (Browse and Howe 2008). Elegant field studies have employed natural and synthetic genetic variation to demonstrate this phenomenon in natural habitats, and have established the ecological importance of JATI in shaping herbivore community composition (Kallenbach et al. 2012; Kessler et al. 2004; Thaler et al. 2001; Züst et al. 2012). We employed this unbiased "ask the plant" genetic approach to query the biological role of JA in mediating interaction of cultivated tomato (*S. lycopersicum*) with potential biotic attackers in the field. Previous studies at this field site showed that glandular trichomes, whose development on tomato leaves is controlled in part by the JA pathway (Boughton et al 2005; Li et al 2004; Peiffer et al 2009), provide an important layer of anti-insect defense (Kang et al. 2010a, 2010b, 2014). Replicated field trials showed that a tomato mutant (*jai1*) lacking the JA-Ile receptor suffered 100% mortality from root rot disease caused by the oomycete pathogen *Pythium* (Fig. 1). Similar results have been reported for JA mutants of Arabidopsis and maize (Staswick et al 1998; Vijayan et al 1998; Yan et al. 2012). These collective studies demonstrate the efficacy of JATI in protecting highly diverse plants against the same soil-borne pathogen.

Core JA Signaling Module

The signal transduction events that couple perception of danger signals at the cell surface to the expression of JA-responsive defense genes relies on an evolutionarily conserved core apparatus to synthesize and perceive JA-Ile (Fig. 2) (Chico et al. 2008; Katsir et al. 2008a). A crucial feature of JA-Ile as an endogenous trigger of immune responses is its rapid and reversible accumulation in vegetative tissues that are frequently targeted for attack. Unstressed leaves of Arabidopsis, for example, contain extremely low or undetectable amounts of bioactive JA (Glauser et al. 2008; Koo and Howe 2009). JA synthesis is initiated in plastids from the pre-existing C18 precursor linolenic acid (LA). LA is converted to a cyclic 12-oxo-phytodienoic acid (OPDA) intermediate, which is then transported to the peroxisome for subsequent reduction and β-oxidation steps that give rise to JA (Howe and Schilmiller 2002; Schaller and Stintzi 2009; Wasternack and Hause 2013). JA is conjugated to Ile in the cytosol to produce JA-Ile (Kang et al. 2006; Staswick and Tiryaki 2004). As the receptor-active form of the hormone (Fonseca et al. 2009; Katsir et al. 2008b; Sheard et al. 2010; Staswick 2008; Thines et al. 2007), JA-Ile presumably diffuses into the nucleus where it is perceived by its receptor. Many genes required for JA-Ile biosynthesis are coordinately upregulated by the JA signaling pathway (Browse 2009; Koo et al. 2006; Wasternack and Hause 2013). Although this observation suggests a positive feedback loop to amplify JA responses, the existence of JA-inducible negative feedback loops (see below) highlights the complexity of processes involved in JA-Ile homeostasis.

JA-Ile controls defense gene expression by promoting the destruction of JAZ (JAsmonate ZIM domain) transcriptional repressors via the ubiquitin-26S proteasome system (Chini et

al. 2007; Thines et al. 2007; Yan et al. 2007). JAZ proteins are defined by two highly conserved sequence motifs referred to as ZIM (or TIFY) and Jas (Browse 2009; Chung et al. 2009; Yan et al. 2007). In the absence of stress, low levels of JA-Ile permit JAZ proteins to bind and repress transcription factors (TFs) in the nucleus. The basic helix-loop-helix TF MYC2 and its closely related paralogs (MYC3 and MYC4) are the most extensively studied JAZ-interacting TFs having a direct role in JATI (Fernández-Calvo et al. 2011; Kazan and Manners 2013; Schweizer et al. 2013). Nuclear localization of some JAZ repressors is dependent on physical association with MYC2 (Withers et al. 2012). Within the nucleus, the repressive activity of some JAZ requires the NINJA (Novel Interactor of JAZ) protein to mediate interaction with corepressors such as TOPLESS (TPL) (Acosta et al 2013; Pauwels et al 2010). NINJA contains an EAR (ERF-associated amphiphilic repression) motif that binds TPL and TPL-related corepressors (Pauwels et al. 2010; Szemenyei et al. 2008). Other JAZ proteins (e.g., JAZ8) contain an EAR motif, thereby allowing direct recruitment of TPL independently of NINJA (Shyu et al. 2012). A rapidly expanding list of JAZ- and MYC2-interacting regulatory proteins that participate in other hormone-response pathways indicates that the JAZ-TF interactome is a cellular hub for integrating diverse environmental and developmental signals (Ballaré 2014; Hou et al. 2010; Hu et al. 2013a; Kazan and Manners 2013; Nakata et al. 2013; Pauwels et al. 2010; Qi et al. 2011, 2014; Song et al. 2011, 2014; Toda et al. 2013; Yang et al. 2012b; Zhu et al. 2011).

In response to perception of signals that trigger JA-Ile synthesis, JA-Ile promotes direct binding of JAZ repressors to the F-box protein COI1 (CORONATINE INSENSITIVE1), which is the specificity determinant of the E3 ubiquitin ligase SCF^{COI1} (Katsir et al. 2008b; Melotto et al. 2008; Sheard et al. 2010; Thines et al. 2007; Xie et al. 1998). Ubiquitin-dependent degradation of JAZ proteins relieves repression of TFs, thereby allowing expression of JA-response genes (Fig. 2). The amplitude and duration of JA responses appear to be controlled primarily by the intracellular level of JA-Ile (Koo et al. 2009; Staswick 2008). Moreover, the speed with which danger signals are transmitted to gene activation via the core pathway may be remarkably fast. Crush-type wounds inflicted to Arabidopsis leaves, for example, result in increased JA-Ile levels within minutes of tissue damage, with increased accumulation of primary JA-response transcripts observed within 5 min of wounding (Chauvin et al. 2013; Chung et al. 2008). Mechanical tissue damage also triggers rapid systemic responses, including JA-Ile accumulation, degradation of JAZ proteins, activation of JA-response genes, and induced resistance (Acosta et al 2013; Green and Ryan 1972; Koo et al. 2009; Mousavi et al. 2013; Zhang and Turner 2008).

Activation of the Core JA Module

MAMPs, HAMPs, and DAMPs

Available evidence indicates that JA/JA-Ile synthesis is controlled at the post-transcriptional level via activation of pre-existing JA biosynthetic enzymes (Wasternack and Hause 2013). Although the precise mechanism of activation remains to be determined, a wide range of endogenous (DAMP) and foreign (MAMP/HAMP) signals have been implicated in the process (Fig. 2; Table 2). Analysis of phytohormone production and defense gene expression in response to elicitors such as flagellin, elongation factor-Tu (EF-Tu), and

chitin, for which the cognate PRRs have been identified, indicate that these conserved bacterial and fungal patterns activate multiple branches of induced immunity, including JATI (Kim et al. 2014). Several HAMPs, including fatty acid-amino acid conjugates (FACs), also amplify JA responses via unknown mechanisms (McCloud and Baldwin 1997; Schmelz et al. 2003, 2007). Elicitation of JA-mediated defense responses by DAMPs, including the 18-amino-acid peptide systemin and cell wall-derived oligogalacturonides (OGs), is consistent with the ability of these compounds to stimulate endogenous JA synthesis (Doares et al. 1995; Lee and Howe 2003). Likewise, endogenous peptide elicitors from Arabidopsis (AtPep1) and maize (ZmPep3) exert potent stimulatory effects on JATI (Table 2) (Huffaker et al. 2006, 2013). Identification of plant receptors for OGs and AtPep1 marks a major advance in efforts to understand the contribution of DAMPs to plant immunity (De Lorenzo et al., 2011; Krol et al., 2010; Yamaguchi et al., 2006). Equally exciting is the recent discovery of the receptor for extracellular ATP, which exhibits properties of a danger signal released by damaged cells (Choi et al. 2014; Song et al. 2006).

Ca^{2+} signaling, ROS and MAPKs

It generally is accepted that JA synthesis is initiated in the plastid by stress-induced activation of lipases that release fatty acid precursors of JA (Bergey et al. 1996; Hyun et al. 2008; Wasternack and Hause 2013). Alternatively, there is evidence to suggest that tissue damage may stimulate JA synthesis from an existing pool of OPDA (Koo et al. 2009). Regardless of the precise mechanism involved, a major gap in our understanding of JATI concerns the molecular events that link perception of MAMP/HAMP/DAMPs by PRRs to accumulation of JA-Ile (Fig. 2). Among the intracellular signals implicated in this process are calcium ions, reactive oxygen species (ROS), and mitogen-activated protein (MAP) kinase cascades.

Calcium ions have long been recognized as ubiquitous second messengers in signal transduction pathways. The involvement of Ca^{2+} in JATI is supported by studies showing that cytosolic Ca^{2+} levels increase in response to herbivore feeding and treatment with exogenous MAMP/HAMP/DAMPs (Arimura and Maffei 2010; Jeter et al. 2004; Maffei et al. 2004, 2006). Changes in membrane polarization caused by wounding and insect attack also increase the level of cytosolic Ca^{2+} (Maffei et al. 2006). Ca^{2+} fluxes and associated Ca^{2+}-binding proteins, including calmodulin and Ca^{2+}-dependent protein kinases (CDPKs), exert control during the activation of JA-response genes (Bonaventure et al. 2007; Boudsocq et al. 2010; Levy et al. 2005; Romeis and Herde 2014; Yang et al. 2012a). Dynamic changes in cytosolic Ca^{2+} levels during plant-attacker interactions are linked to the production of ROS, including hydrogen peroxide (Arimura and Maffei 2010). Alterations in cellular redox status are associated with local and systemic JATI, and are associated with the activity of the respiratory burst oxidase homolog D (RBOHD) (Miller et al. 2009; Orozco-Cárdenas et al. 2001). Direct phosphorylation of RBOHD by the PRR-associated kinase BIK1 provides a mechanism to integrate MAMP perception with calcium-based regulation of immune function (Kadota et al. 2014; Li et al. 2014).

MAP kinase signaling cascades serve a prominent role in the early steps of induced immunity (Asai et al. 2002; Schweighofer et al. 2007; Seo et al. 2007; Wu and Baldwin

2010; Zhang and Klessig 2001). Plants silenced in the expression of specific MAPKs showed reduced JA biosynthesis and decreased expression of JA-related defense genes, suggesting that these kinases control an early step in the activation of JA synthesis (Kandoth et al. 2007; Wu et al. 2007). The manner in which MAPK cascades are linked to a specific step in the JA biosynthesis pathway, however, remains unknown.

Long-distance electrical signals and glutamate-like receptors

One of the most fascinating and least understood aspects of JATI concerns the mechanism by which mechanical tissue injury, including that elicited by chewing herbivores, results in rapid systemic induction of JA biosynthesis and defense gene expression (Koo and Howe 2009). Studies in Arabidopsis, for example, show that a signal generated at the site of leaf injury travels rapidly (2–3 cm/min) to trigger JA-Ile synthesis and associated JA responses in undamaged leaves (Glauser et al. 2008; Koo et al. 2009). Despite the importance and wide-spread occurrence of this phenomenon, the molecular and genetic basis of rapid systemic JATI signaling has been uncertain. A recent study by Mousavi et al. (2013) built on previous work showing that leaf damage causes changes in electrical activity and membrane depolarization in undamaged systemic leaves (Mousavi et al. 2013; Wildon et al. 1992; Zimmermann et al. 2009). A screen for mutants that exhibit reduced wound-triggered changes in electric potential showed that members of the GLUTAMATE RECEPTOR-LIKE (GLR) family of ion channel proteins are required for the response; *glr3.3* and *glr3.6* mutants are deficient in electrical activity in wounded leaves and show reduced expression of JA-responsive genes in distal undamaged leaves (Mousavi et al. 2013). Systemic depolarization events were triggered in a GLR3.3/GLR3.6-dependent manner by caterpillar feeding but not by caterpillar walking on the leaf (Mousavi et al. 2013; Salvador-Recatalà et al. 2014). The ability of mechanical leaf wounding and insect chewing to elicit comparable changes in electrical activity indicates that insect-derived factors are not required for this response. Current evidence thus indicates that insect feeding generates long-distance electrical signals through the action of GLRs, and that decoding of this signal in systemic responding leaves results in JA-Ile synthesis, JA-Ile perception via the COI1-JAZ co-receptor system, and activation of defense gene expression. The JATI-eliciting electrical signal thus has all the hallmarks of a DAMP (Fig. 2). The mechanism by which the propagating signal is perceived and subsequently linked to JA biosynthesis remains to be determined. However, there is evidence to suggest that calcium ions may be involved in propagating and/or interpreting the signal in responding target cells (Felle and Zimmermann 2007; Maffei et al. 2006; Qi et al. 2006) and a role for RBOHD-dependent ROS production was excluded (Mousavi et al. 2013).

Negative Regulation of JATI

Although JATI confers effective resistance to a broad spectrum of pathogens and herbivores, hyperactivation of the pathway can negatively affect plant growth and fitness. Many specialized defense compounds, for example, are toxic to the plant that produces them (Baldwin and Callahan 1993; Gog et al. 2005). In addition, increased allocation of limited metabolic resources to defense compounds may reduce the extent to which these resources can be used to fuel plant growth and reproduction (Agrawal 1999; Baldwin 1998; Herms

and Mattson 1992; Yan et al. 2007; Zhang and Turner 2008). JATI may, therefore, provide a cost-saving strategy to coordinate the timing of chemical defense production with perceived threats from the environment. Until recently, relatively little attention has been paid to understanding mechanisms that restrain defense signaling pathways or desensitize plant cells to the presence of eliciting signals. As described below, molecular studies have elucidated several JA-induced negative feedback loops within the core JA signaling module.

Catabolism of JA-Ile

The dependence of JATI on intracellular accumulation of JA-Ile suggests that turnover of the hormone could provide a mechanism to attenuate JA responses. Initial support for this hypothesis came from studies showing that various oxidized and conjugated derivatives of JA-Ile accumulate in wounded leaves (Glauser et al. 2008; Miersch et al. 2008; Paschold et al. 2008). Recent studies have employed genetic approaches to elucidate two metabolic routes for JA-Ile catabolism, referred to as the JA-Ile ω-oxidation and hydrolysis pathways (Fig. 2) (Koo and Howe 2012). The latter pathway is catalyzed by aminohydrolases that cleave JA-Ile to JA and Ile (Bhosale et al. 2013; Widemann et al. 2013; Woldemariam et al. 2012). This reaction is readily reversible by the JA-conjugating enzyme JAR1 (Staswick and Tiryaki 2004), suggesting that the relative level of conjugating and aminohydrolase activity is an important factor in the control of JA-Ile homeostasis. In contrast to JA-Ile hydrolysis, the ω-oxidation pathway provides a mechanism for permanent inactivation of JA-Ile. This pathway involves at least two members (CYP94B3 and CYP94C1) of the CYP94 family of cytochromes P450 that oxidize the ω-carbon of JA-Ile to produce 12OH-JA-Ile, which is further oxidized to 12COOH-JA-Ile (Heitz et al. 2012; Kitaoka et al. 2011; Koo et al. 2011). 12OH-JA-Ile is less active than JA-Ile in promoting COI1 binding to JAZ proteins (Koo et al. 2011). The fact that 12OH-JA-Ile retains some activity in COI1-JAZ interaction assays, however, suggests that CYP94-mediated oxidation of 12OH-JA-Ile to 12COOH-JA-Ile, or conjugation of 12OH-JA-Ile to other small molecules (Gidda et al. 2003; Kitaoka et al. 2014), is required for complete inactivation of JA-Ile.

Consistent with a role in negative feedback regulation of JATI, genes encoding enzymes in both the ω-oxidation and hydrolysis pathways are rapidly induced in response to wounding, herbivory, and JA treatment (Bhosale et al. 2013; Heitz et al. 2012; Kitaoka et al. 2011; Koo et al. 2011; Widemann et al. 2013; Woldemariam et al. 2012). Remarkably, Bhosale et al. (2013) found that the *IIL6* gene encoding a JA-Ile aminohydrolase in Arabidopsis is co-expressed with other JA-response genes in plants grown under tightly controlled growth conditions in which stress treatments were not intentionally imposed. This finding highlights the exquisite sensitivity of JA-associated surveillance and response systems, and suggests a broader role for JA signaling in modulating phenotypic plasticity in response to subtle changes in the environment. It can be anticipated that future research will uncover mechanisms by which JA responses are integrated with various environmental perturbations, including changes in light, water status, nutrient availability, soil microbe communities, and wind/touch (e.g., Chehab et al. 2012).

Stable JAZ proteins

Among the 12 *JAZ* genes in Arabidopsis, most are rapidly and strongly expressed in response to exogenous JA or stress-induced accumulation of endogenous JA (Chini et al. 2007; Chung et al. 2008; Thines et al. 2007; Yan et al. 2007;). This pattern of expression suggests that *de novo* synthesis of JAZ proteins is part of a negative feedback system to desensitize cells to the presence of the hormone. Such a mechanism of feedback control, however, presumably depends on JAZ proteins that are relatively resistant to JA-Ile-induced destruction. Whereas initial studies demonstrated that some JAZ members (e.g., JAZ1) are rapidly degraded ($t_{1/2} < 2$ min) in the presence of JA-Ile (Chini et al. 2007; Grundwald et al. 2009; Pauwels et al. 2010; Thines et al. 2007), recent studies have advanced the concept that other JAZs exhibit a wide range of stability, which could allow fine-tuning of TF activity in response to fluctuating JA-Ile levels (Chung and Howe 2009; Chung et al. 2009, 2010; Shyu et al. 2012).

The conserved Jas motif of JAZ proteins contains a degradation signal (degron) that binds COI1 in a JA-Ile-dependent manner (Katsir et al. 2008b; Melotto et al. 2008; Sheard et al. 2010; Yan et al. 2007). Point mutations within the degron disrupt JAZ-COI1 interaction without affecting JAZ binding to TFs, thereby stabilizing and enhancing the activity of the repressor (Melotto et al. 2008; Withers et al. 2012). Natural sequence variation within degron also affects JAZ stability and associated physiological outputs of JATI (Shyu et al. 2012). JAZ8, for example, contains a non-canonical degron that evidently does not interact with COI1 in the presence of JA-Ile. As a consequence, JAZ8 maintains the ability to interact with target TFs and repress transcription through recruitment of a co-repressor complex. Stress-induced expression of JAZ8 may thus provide a mechanism to desensitize cells to the presence of JA-Ile (Shyu et al. 2012).

JAZ repressors also are stabilized by alternative splicing (AS) events that remove or modify the Jas motif and its associated degron. AS of *JAZ10* pre-mRNA produces several splice variants that differentially interact with COI1 in the presence of JA-Ile. These isoforms of JAZ10 exhibit a range of stability in JA-stimulated cells and, when overexpressed *in planta*, attenuate JA signal outputs to varying degrees (Chung and Howe 2009; Chung et al. 2010; Moreno et al. 2013). A direct role for *JAZ10* AS in negative feedback control of JA signaling is supported by the JA-hypersensitive phenotype of *jaz10* null mutants, as well as the ability of specific JAZ10 splice variants to complement the hypersensitive phenotype of *jaz10* mutants (Cerrudo et al. 2012; Demianski et al. 2012; Moreno et al. 2013; Yan et al. 2007). The AS event responsible for generating the stable JAZ10.3 isoform involves retention of an intron whose location within the Jas motif results in truncation of the C-terminal end of the motif. Interestingly, this intron is present in most *JAZ* genes from diverse land plants, suggesting that this conserved AS event provides a general mechanism to desensitize cells to high JA-Ile levels (Chung et al. 2010). It remains to be determined how stable JAZ repressors are removed from cells in order to reset full sensitivity of the JA response.

Transcriptional JAMming

A third mechanism to negatively regulate JA responses involves a phylogenetic clade of bHLH-type proteins (JAM1/bHLH017, JAM2/bHLH013, JAM3/bHLH003) that is closely related to the positively acting MYC2 TF and its functional paralogs, MYC3, and MYC4 (Fonseca et al. 2014; Nakata et al. 2013; Sasaki-Sekimoto et al. 2013; Song et al. 2013). JAM proteins compete with MYC2 for binding to *cis*-acting G-box elements within the promoters of JA-responsive genes. However, because they lack the conserved activation domain found in MYC2/3/4, JAMs function as transcriptional repressors rather than activators. JAM TFs also interact directly with JAZ proteins, which may serve to increase the strength of transcriptional repression through recruitment of the co-repressors NINJA and TOPLESS (Fonseca et al. 2014; Song et al. 2013). Similar to other negative feedback loops, the expression of *JAM1* is strongly upregulated by JA treatment and associated stress responses (Fonseca et al. 2014; Nakata et al. 2013; Sasaki-Sekimoto et al. 2013; Song et al. 2013).

Other modes of negative regulation

The multiple negative feedback loops described above likely act in concert to restrain the amplitude and duration of JATI after the response is initiated. It should be noted, however, that the onset of JATI can be actively suppressed by other signals when the benefit of growth outweighs the cost of defense. A compelling example is repression of JATI during the shade avoidance response in which changes in light quality, as perceived by the photoreceptor phytochrome B, modulates the stability of MYC TFs and JAZs to prioritize elongation growth over defense (Ballaré 2014; Cerrudo et al. 2012; Chico et al. 2014; Izzaguirre et al. 2013; Moreno et al. 2009). Recent studies have also provided insight into the mechanisms by which JATI is suppressed by the growth-related hormones gibberellic acid (Hou et al. 2010; Yang et al. 2012b) and ethylene (Kim et al. 2014; Song et al. 2014), as well as other negative regulators whose mode of action is just beginning to be understood (Hu et al. 2013b).

Manipulation of JATI by Plant-associated Organisms

The efficacy of any given immune response is often reflected by the extent to which host-associated pathogens and parasites evolved to evade that response. Consistent with its role in re-directing primary and secondary metabolism, perhaps it is not surprising that plant pathogens and herbivores evolved strategies to manipulate (activate or suppress) JATI. Current views on this topic are influenced by the notion that JATI and SATI are often mutually antagonistic (Caillaud et al. 2013; Kunkel and Brooks, 2002; Robert-Seilaniantz et al. 2011; Thaler et al. 2012;). Studies performed with Arabidopsis, for example, have led to the generalization that increased activity of the JA sector of immunity enhances the virulence of biotrophic pathogens that are sensitive to SATI, whereas expression of SATI favors the performance of insect herbivores and necrotropic pathogens that are more sensitive to JATI. Some of the strongest evidence for JATI-SATI antagonism comes from studies showing that many plant-associated organisms use effector-based strategies to create JA-SA imbalances that suppress one or the other branches of immunity (Table 2).

An important emerging paradigm in plant-herbivore interactions is the ability of herbivores to activate the SA pathway and thereby reduce the effectiveness of JATI as a basal defense (Hogenhout and Bos 2011; Walling 2008). For example, phloem feeding by silverleaf whitefly (*Bemisia tabaci*) results in increased expression of SA-related defense genes and concomitant repression of JATI (Zarate et al. 2007; Zhang et al. 2013). Similarly, insect egg-associated effectors trigger SA accumulation and JATI suppression in host tissues surrounding the egg, thus favoring the survival of newly hatched larvae (Bruessow et al. 2010; Reymond 2013). Secretion of SA into the locomotion mucus (slime trail) by some molluskan herbivores (Kästner et al. 2014), or excretion of SA into honeydew by some aphid species (Schwartzberg and Tumlinson 2013), may reflect additional mechanisms to suppress JATI. The Coleopteran herbivore *Leptinotarsa decemlineata* (Colorado potato beetle) employs an alternative but no less effective strategy to hijack JATI; symbiotic bacteria in the oral secretion of the beetle activate SA-dependent responses and repress local and systemic JATI (Chung et al. 2013). That this phenomenon also occurs in a root-feeding insect herbivore (*Diabrotica* virgifera, western corn rootworm) of maize suggests that host defense suppression by symbiotic bacteria may be a general feeding strategy adopted by insect herbivores (Barr et al. 2010).

Studies of the Arabidopsis-*Pseudomonas syringae* strain DC3000 (*Pst* DC3000) pathosystem have provided considerable insight into how bacterial pathogens manipulate JATI-SATI antagonism to their own advantage. In this system, immunity to *Pst* DC3000 is mediated in large part by SATI. Interestingly, *Pst* DC3000 uses multiple effectors to activate JA responses through targeted destruction of JAZ proteins, which in turn suppresses SATI (Fig. 2). One well-studied effector is the polyketide coronatine (COR) that acts as a potent agonist of the COI1-JAZ co-receptor system (Bender et al. 1993; Katsir et al. 2008b; Sheard et al. 2010). COR-induced degradation of JAZ repressors strongly upregulates the expression of JA-responsive defense genes and downregulates growth-related genes, and impairs multiple aspects of SATI (Attaran et al. 2014; Brooks et al. 2005; Melotto et al. 2006; Uppalapatti et al. 2007; Zhao et al. 2003; Zheng et al. 2012). Suppression of SATI by COR is mediated in part by NAC-type TFs that concomitantly repress the expression of the key SA biosynthetic enzyme ICS1 and activate expression of a methyltransferase (BSMT1) that converts SA to volatile MeSA (Attaran et al. 2009, 2014; Zheng et al. 2012). That release of MeSA is observed in several other plant-enemy interactions (Dempsey et al. 2011) suggests that disposal of SA through volatilization may be a general mechanism to antagonize SATI by stresses that trigger JA signaling.

Pseudomonas syringae strains produce at least two type III secreted protein effectors that also promote degradation of JAZ proteins to increase pathogenicity. HopZ1a is a putative acetyltransferase that modifies JAZ proteins to stimulate their degradation in a COI1-dependent manner (Jiang et al. 2013). HopX1 is a cysteine protease that destroys JAZs independently of COI1 (Gimenez-Ibanez et al. 2014). Interestingly, HopX1 is produced by a strain of *P. syringae* that does not synthesize COR, indicating that distinct mechanisms to activate JA signaling through proteolytic destruction of JAZs have arisen independently in the evolution of this pathogen (Gimenez-Ibanez et al. 2014). These findings are consistent with results of large-scale protein-protein interaction screens showing that JAZs are targets

of effectors from both *P. syringae* and the obligate biotrophic oomycete *Hyaloperonospora arabidopsidis* (Mukhtar et al. 2011). In contrast to other biotrophic organisms, colonization of host tissues by some mutualistic ectomycorrhizal fungi is inhibited by JATI (Plett et al. 2014a). A recent study showed that the ectomycorrhizal fungus *Laccaria bicolor* produces an effector (MiSSP7) that binds to and stabilizes a host JAZ protein to repress JA responses that presumably inhibit establishment of the symbiosis (Plett et al. 2014b). These collective studies highlight the COI1-JAZ co-receptor system as a central hub of plant immunity and portend the discovery of additional effectors that target the core JA module.

Synthesis and Future Perspectives

Recent research on many fronts has tremendously advanced our understanding of the mechanism of JA signaling and its relationship to induced plant immunity. These efforts have coalesced around a simple model (Fig. 2) to explain how fluctuating levels of a small-molecule hormone (JA-Ile) exert transcriptional control over complex morphological and chemical defense traits. We suggest that the modular structure of JATI allows the conserved core JA module to link different combinations of PRR-based recognition systems (inputs) and defense traits (outputs) to create new specificities of host resistance. Indeed, there is good evidence that JATI is a significant driving force in shaping plant-animal associations in natural environments (Kallenback et al. 2012; Züst et al. 2012). This conceptual framework provides a foundation for studies aimed at understanding the underlying mechanisms by which recognition-response systems give rise to phenotypic plasticity, and for revealing how interactions between genomes and environments have spawned highly diverse, idiosyncratic defense traits in the plant kingdom. Meeting this challenge will require integrative approaches spanning the ecosystem-to-gene continuum, as applied to experimental systems that offer both genomic and ecological resources.

With the exception of a few model plants, remarkably little is known about the identity of JA-regulated compounds that provide resistance against specific attackers. It is currently unclear, for example, whether JA/COI1-mediated resistance of Arabidopsis, tomato, and maize to soil-borne *Pythium* (Table 1) involves similar or divergent defense chemistries. Major differences in specialized defense compounds between these species, however, suggests that each plant may use the core JA module to deploy different suites of chemical defense against the same pathogen. Similarly, there is evidence that tomato and Arabidopsis use highly distinct JA-regulated defense chemistries for protection against the generalist herbivores *Tetranychus urticae* (two-spotted spider mite) and *Trichoplusia ni* (cabbage looper) (Herde and Howe 2014; Li et al. 2002,2004; Zhurov et al. 2014). The modular architecture of JATI thus appears to support the evolution, in different host plants, of independent chemical solutions to the same pathogen or herbivore, which may contribute to the diversity and sporadic distribution of secondary metabolites in higher plants (Fraenkel 1959). On the other hand, there are several examples of similar defense compounds that evolved independently in diverse plant families (Berenbaum and Zangerl 2008). Modern omics-based technologies offer tremendous potential to better understand the evolution of constitutive and induced defense compounds by elucidating gene-pathway-metabolite relationships in diverse groups of plants (Berenbaum and Zangerl 2008; Kliebenstein 2012). Insight into the evolutionary forces that drive the diversity of chemical defenses also will

benefit from a better understanding of how these defense systems are matched by equally complex counter-defenses in plant attackers (Herde and Howe 2014).

It is becoming increasingly evident that the JA/COI1/JAZ/TF module is a convergence point for direct crosstalk with other signaling pathways that control growth and development (Ballaré 2014; Erb et al. 2012; Huot et al. 2014). It appears that this crosstalk occurs primarily through direct interaction between nuclear factors that regulate transcription, including the Mediator complex (Caillaud et al. 2013; Çevik et al. 2012; Kidd et al. 2011). Future research aimed at understanding changes in chromatin structure, epigenetic modification, and *cis*-regulatory codes (Zou et al. 2011) that direct TF-DNA interactions is expected to provide new insight into how transcriptional networks control complex JATI outputs, including transgenerational immunity (Rasmann et al. 2012). Various negative feedback loops act in concert to restrain JATI outputs, but whether these control mechanisms constitute an adaptive response to balance tradeoffs between JATI and growth, or perhaps other forms of immunity, remains to be determined. Knowledge of how proteins in different signaling pathways functionally interact to regulate growth-defense antagonism has potential practical application in the development of crop varieties that are both high yielding and stress tolerant. These efforts may be aided by mathematical models to predict how environmental inputs are integrated within phytohormone networks to generate specific physiological outcomes (Middleton et al. 2012).

A significant gap in our understanding of JATI is how recognition of danger signals at the cell surface activates JA biosynthesis. By analogy to stress-responsive regulation of ethylene biosynthesis (Liu and Zhang 2004), identification of direct targets of the relevant MAPK cascades may provide important clues. Attention also should be given to the hypothesis that JA biosynthesis is controlled by calcium-dependent signaling events that are coupled to PRR activation (Romeis and Herde 2014). Further analyses of how GLRs generate and/or propagate long-distance electrical signals that trigger systemic JA-Ile synthesis will undoubtedly yield important new insights as well. A systems-level view of JA-Ile homeostasis, including pathways by which JA derivatives are transported within and between cells, is ultimately needed to understand how specific TFs are controlled by thresholds and time-dependent signatures of the hormone. Finally, it should be noted that although there is molecular evidence that plant resistance to insect herbivores is mediated by PRRs (Abuqamar et al. 2008; Prince et al. 2014; Truitt et al. 2004; Yang et al. 2011), HAMP receptors remain to be identified in any plant.

One of the most exciting recent advances in the field of induced plant immunity is evidence that the core JA module is a common target of effectors from multiple plant-associated microbes (Table 2). This finding is consistent with the idea that different pathogens independently evolved virulence factors that converge on common host targets within the PTI network (Jones and Dangl 2006; Mukhtar et al. 2011). Only time will tell whether the current list of COI1/JAZ-targeting effectors is complete or, more likely, will continue to expand as effector repertoires from diverse plant-associated microbes, arthropods, and nematodes are systematically scrutinized (Boller and He 2009; Elzinga and Jander 2013; Hogenhout and Bos 2011; Kandoth and Mitchum 2013). The strong selection pressure imposed by JATI on arthropod herbivores and pathogens, together with evidence that many

of these organisms actively suppress JA-based defenses, suggests the existence of novel mechanisms by which plant-associated organisms disrupt JATI. Interdisciplinary approaches aimed at understanding how the JA module promotes broad-spectrum immunity through the control of specialized metabolism, and how this branch of immunity is subverted by plant attackers, offers tremendous potential to help solve pressing problems facing the world (Plant Science Research Summit 2013). From a biotechnological perspective, for example, these efforts may inform synthetic approaches to harness specialized biochemical pathways for metabolic engineering of new chemistries for a variety of plant-based products, including pigments, fragrances, flavors, pesticides, and pharmaceuticals. Given the current pace of discovery and technological tools available, exciting new discoveries may be just around the corner.

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Fig. 1.
Jasmonate perception by the COI1 receptor system is essential for resistance of cultivated tomato to the oomycete pathogen *Pythium*. Wild-type (cv Castlemart) and *jai1* mutant plants grown for three weeks in a growth chamber without visible signs of disease were transplanted to a field plot at Michigan State University, East Lansing, MI. Two weeks after transplanting, all *jai1* plants (*N* = 30) died from a disease that was diagnosed as *Pythium* stem/root rot by the MSU Diagnostics Lab. Sequencing of PCR products derived from 5.8S ribosomal gene and internal transcribed spacer region in infected tomato tissue confirmed the presence of *Pythium ultimum*. Of several hundred wild-type (*Jai1/Jai1*) plants grown side-by-side at the same field site, none showed symptoms of the disease. The figure shows photographs of representative wild-type (A and B) and *jai1* mutant (C and D) plants two weeks after transplantation. Identical results were obtained in three independent trails performed at the same site.
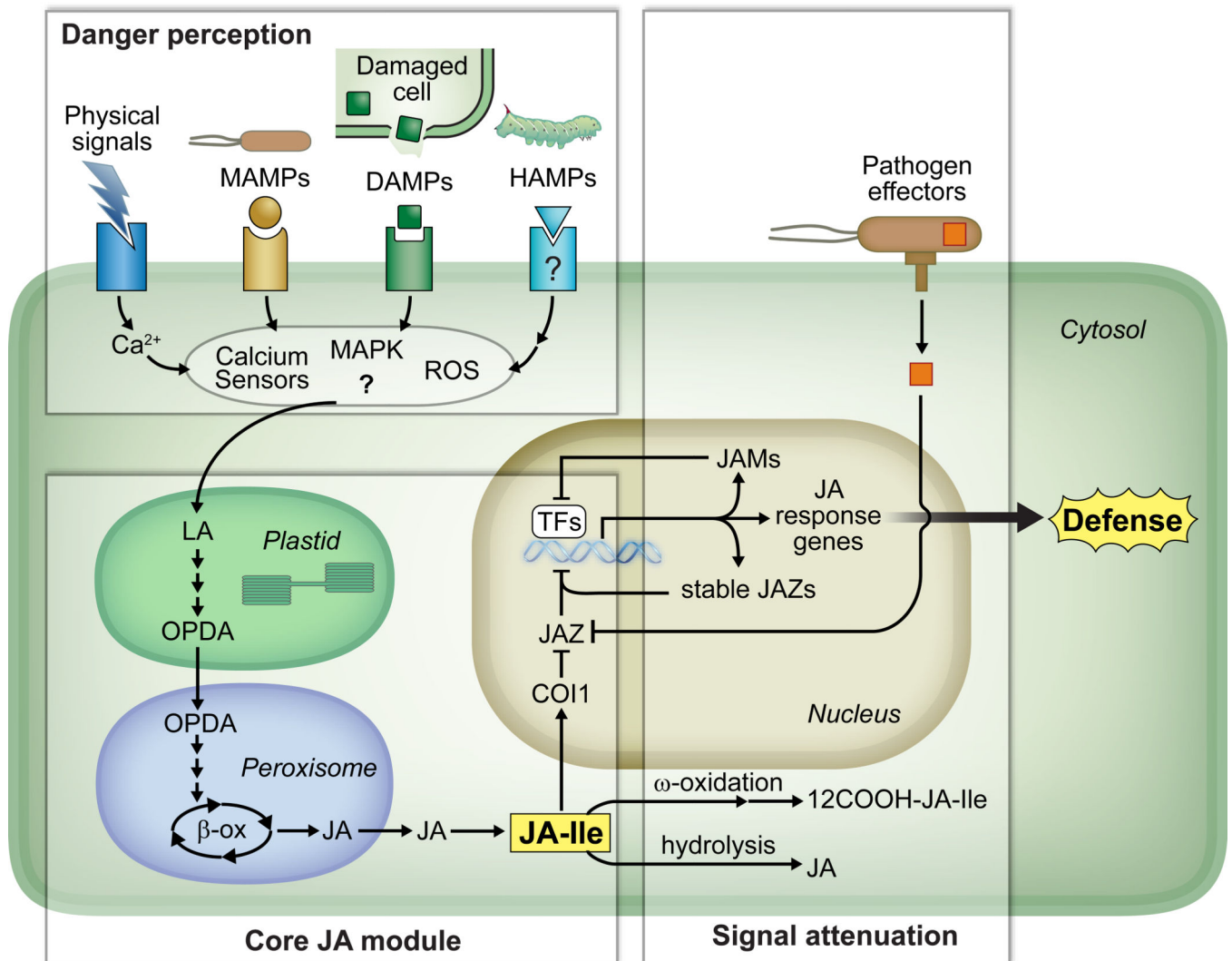
Fig. 2.
Model of jasmonate-triggered plant immunity (JATI). Danger signals (MAMPs/HAMPs) derived from attacking organisms and damaged plant cells (DAMPs) are recognized by pattern recognition receptors (PRRs) at the cell surface. PRR activation is coupled to intracellular signaling systems involving MAP kinase pathways, calcium ion-sensing proteins, and reactive oxygen species (ROS), among others. How these signaling events are connected to activation of the core JA signal module, which includes JA biosynthesis from its precursor linolenic acid (LA), is largely unknown (?). Plastidic and peroxisomal enzymes convert LA to jasmonic acid (JA), which is the substrate for synthesis of JA-Ile in the cytosol. Within the nucleus, JA-Ile promotes JAZ-COI1 interaction and targets JAZs for proteolytic degradation by the ubiquitin-proteasome system. Removal of JAZ alleviates TFs from repression, thereby activating the expression of JA-responsive genes and the expression of chemical and morphological defense traits (Defense). Several mechanisms to attenuate signaling through the core module have been elucidated, including catabolism of JA-Ile via ω-oxidation and hydrolysis, *de novo* synthesis of JAZ repressors that are stable in the presence of JA-Ile, and accumulation of JAM TFs that negatively regulate transcription.

Pathogen-derived effectors target the core JA signal module to disrupt hormonal balance and induced immune responses. Abbreviations: Microbe-associated molecular patterns (MAMPs); Herbivore-associated molecular patterns (HAMPs); Damage-associated molecular patterns (DAMPs), Mitogen-activated protein kinase (MAPK); 12-oxo-phytodienoic acid (OPDA), β-oxidation (β-ox), jasmonoyl-L-isoleucine (JA-Ile), JASMONATE-ZIM domain (JAZ), JA-related transcription factor (TF), JASMONATE-ASSOCIATED MYC2-LIKE (JAM), 12-carboxy-JA-Ile (12COOH-JA-Ile).

Table 1

Examples in which there is genetic evidence for JA-mediated plant resistance to pathogens and herbivores.

ORGANISM	HOST PLANT	REFERENCE
Pathogenic bacteria		
Erwinia carotovora	*Arabidopsis thaliana* (Brassicaceae)	Norman-Setterblad et al. 2000
Xanthomonas oryzae	*Oryza sativa* (Poaceae)	Yamada et al. 2012
Necrotrophic fungi / oomycetes		
Alternaria brassicicola	*A. thaliana* (Brassicaceae)	Thomma et al. 1998
Botrytis cinerea	*A. thaliana* (Brassicaceae)	Thomma et al. 1998
Pythium spp	*A. thaliana* (Brassicaceae)	Vijayan et al. 1998; Staswick et al. 1998;
	Zea mays (Poaceae)	Yan et al. 2012
	Solanum lycopersicum (Solanaceae)	This study
Nematodes		
Meloidogyne graminicola	*O. sativa* (Poaceae)	Nahar et al. 2011
Mollusks		
Arion lusitanicus	*A. thaliana* (Brassicaceae)	Falk et al. 2013
Crustaceans		
Porcellio scaber	*A. thaliana* (Brassicaceae)	Farmer and Dubugnon 2009
Armadillidium vulgare	*O. sativa* (Poaceae)	
Cell content feeders		
Tetranychus urticae (Acari)	*S. lycopersicum* (Solanaceae)	Li et al. 2004
	A. thaliana (Brassicaceae)	Zhurov et al. 2014
Frankliniella occidentalis (Thysanoptera)	*Solanum lycopersicum* (Solanaceae)	Li et al. 2002
	A. thaliana (Brassicaceae)	Abe et al. 2009
Piercing-sucking insects		
Myzus persicae (Hemiptera)	*A. thaliana* (Brassicaceae)	Ellis et al. 2002
Empoasca sp. (Hemiptera)	*Nicotiana attenuata* (Solanaceae)	Kessler et al. 2004
Leafminer insects		
Scaptomyza flava (Diptera)	*A. thaliana* (Brassicaceae)	Whiteman et al. 2011
Leaf / root chewing insects		
Manduca sexta (Lepidoptera)	*N. attenuata* (Solanaceae)	Howe et al. 1996; Kessler et al. 2004;
Spodoptera frugiperda (Lepidoptera)	*S. lycopersicum* (Solanaceae)	Campos et al. 2009
Bradysia impatiens (Diptera)	*A. thaliana* (Brassicaceae)	McConn et al. 1997
Spodoptera exigua (Lepidoptera)	*Zea mays* (Poaceae)	Yan et al. 2012
Vertebrate herbivores		
Eurotestudo boettgeri	*A. thaliana* (Brassicaceae)	Mafli et al. 2012

Table 2

Select examples of danger signals and effectors that modulate JA-mediated plant defense responses.

SIGNAL	MECHANISM OF PERCEPTION / ACTION	REFERENCES
DAMPs		
AtPep1	LRR-RK receptors PEPR1 and PEPR2. Activates JA- and SA-dependent innate immune responses.	Huffaker et al. 2006; Yamaguchi et al. 2006
Systemin	Receptor unknown (presumed LRR-RK). Elicits JA synthesis and production of defense compounds.	Pearce et al. 1991
ZmPep3	Unknown receptor. Activates JA synthesis and production of defense compounds.	Huffaker et al. 2013
Oligogalacturonides	WAK1 receptor. Activates JA synthesis and production of defense compounds.	Doares et al. 1995 Brutus et al. 2010
Extracellular ATP	DORN1 receptor. Activates transcriptional responses that are similar to wound responses.	Choi et al. 2014
MAMPs/HAMPs		
Flagellin (bacterial pathogens)	LRR-RK receptor FLS2. Activates the JA and other branches of induced immunity.	Chinchilla et al. 2006; Kim et al. 2014
Elongation factor-Tu (bacterial pathogens)	LRR-RK receptor EFR. Activates the JA and other sectors of induced immunity.	Zipfel et al. 2006; Kim et al. 2014
Chitin (fungal pathogens)	LysM-RK receptor CERK1. Predominately activates the JA sector of induced immunity.	Wan et al. 2008; Kim et al. 2014
Volictin and other fatty acid-amino acid conjugates (Lepidopteran herbivores)	Unknown receptor. Released from insect oral secretions to stimulate JATI.	Alborn et al. 1997; Halitschke et al. 2001
Inceptin (Lepidopteran herbivores)	Unknown receptor. Activates JA accumulation and associated defense responses.	Schmelz et al. 2007
Physical signals		
Electrical potentials (Herbivory / wounding)	Wound-induced electrical signals are propagated by glutamate-like receptors to activate JA biosynthesis.	Mousavi et al. 2013
Microbial effectors		
Coronatine (*Pseudomonas syringae*)	JA-Ile analog that promotes formation of COI-JAZ co-receptor complexes and JAZ degradation.	Katsir et al. 2008b; Sheard et al. 2010
HopZ1a (*Pseudomonas syringae*)	Putative acetytransferase that promotes COI1-dependent JAZ degradation.	Jiang et al. 2013
HopX1 (*Pseudomonas syringae*)	A cysteine protease that promotes COI1-independent JAZ degradation.	Gimenez-Ibanez et al. 2014
MiSSP7 (*Laccaria bicolor*)	An effector from a mutualistic fungus that binds to and protects JAZ6 from JA/COI1-induced degradation.	Plett et al. 2014b
HaRxL44 (Downy mildew)	Promotes degradation of Mediator subunit 19a to activate JA responses and suppress SATI.	Caillaud et al. 2013