REVIEW

Jasmonate signaling in plant development and defense response to multiple (a)biotic stresses

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Abstract Plants frequently live in environments characterized by the presence of simultaneous and different stresses. The intricate and finely tuned molecular mechanisms activated by plants in response to abiotic and biotic environmental factors are not well understood, and less is known about the integrative signals and convergence points activated by plants in response to multiple (a)biotic stresses. Phytohormones play a key role in plant development and response to (a)biotic stresses. Among these, one of the most important signaling molecules is an oxylipin, the plant hormone jasmonic acid. Oxylipins are derived from oxygenation of polyunsaturated fatty acids. Jasmonic acid and its volatile derivative methyl jasmonate have been considered for a long time to be the bioactive forms due to their physiological effects and abundance in the plant. However, more recent studies showed unambiguously that they are only precursors of the active forms represented by some amino acid conjugates. Upon developmental or environmental stimuli, jasmonates are synthesized and accumulate transiently. Upon perception, jasmonate signal transduction process is finely tuned by a complex mechanism comprising specific repressor proteins which in turn control a number of transcription factors regulating the expression of jasmonate responsive genes. We discuss the latest discoveries about the role of jasmonates in plants resistance mechanism against biotic and abiotic stresses. Finally, the deep interplay of different phytohormones in stresses signaling will be also discussed.

Keywords Abiotic stresses · Biotic stresses · Hormones signaling · Jasmonates · Transcription factors

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The jasmonate biosynthesis pathway

The first step of JA synthesis occurs in the membranes of chloroplasts, where a phospholipase releases α-linolenic acid (C18:3) and hexadecatrienoic acid (C16:3) from membrane phospholipids (Ishiguro et al. 2001; Hyun et al. 2008). In plants, generally, JAs synthesis occurs mainly from the C18:3 precursor through the octadecanoid pathway (Farmer and Ryan 1992; Mueller et al. 1993; Fig. 1). In the second step, αLA is oxidized by the action of a chloroplastic 13-lipoxygenase (13-LOX) generating the 13-hydroperoxy derivative of linolenic acid (13-HPOT) (Vick and Zimmerman 1983; Bell and Mullet 1993; Melan et al. 1993; Bannenberg et al. 2009). The *Arabidopsis* genome harbors six genes encoding lipoxygenases and three of them (LOX2, LOX3 and LOX4) have been associated with the production of JA (Bell and Mullet 1993; Caldelari et al. 2011). The subsequent enzyme along the pathway is the allene oxide synthase (AOS) which is responsible for dehydration of the 13-HPOT into the

epoxy-octadecatrienoic-acid, also called allene oxide. AOS protein represents a key enzyme in this pathway and belongs to the CYP74A subfamily inside the large family of cytochrome P450 (Laudert et al. 1996; Laudert and Weiler 1998). *AOS* is induced by JA and wounding (Laudert and Weiler 1998). T-DNA insertion in the unique *AOS* gene present in the *Arabidopsis* genome resulted in male sterile mutants unable to synthesize JA (Park et al. 2002; von Malek et al. 2002).

Allene oxide cyclase (AOC) is the enzyme responsible for the conversion of the unstable allene oxide into (9S,

13S)-*cis*-(+)-oxophytodienoic acid (OPDA) by a cyclization reaction (Ziegler et al. 2000). AOS and AOC are both chloroplastic enzymes (Ziegler et al. 2000; Hause et al. 2003; Fig. 1). A small amount of hexadecatrienoic acid (16:3) can be converted by the same enzymes to dinor-12-oxo-fitodienoic acid (dnOPDA), an OPDA structural homolog (Weber et al. 1997). The next steps of JA synthesis take place in the peroxisome, where OPDA is oxidized to 3-oxo-2-(2′(Z)-pentenyl)-cyclopentane-1-octanoic acid (OPC:8) by the enzyme OPDA reductase (OPR) (Schaller and Weiler 1997; Fig. 1).

Fig. 1 JAs biosynthesis is an enzymatic process occurring in different subcellular compartments. JA biosynthesis involves three different cellular compartments: the plastid (*green square*) where OPDA (*cis*-(+)-12-oxophytodienoic acid) and dn-OPDA (dinor-OPDA) are synthesized, the peroxisome (*red square*) where (dn)OPDA is converted to JA (jasmonic acid) and where β-oxidation cycle takes place by a set of four enzymatic reactions: oxidation, hydration, oxidation and thiolysis, and the cytosol where further modifications of JA take place. Ja-Ile, jasmonoyl-isoleucine; MeJa, methyl jasmonate; 13-HPOT, (13S)-hydro-peroxyoctadecatrienoic acid; *AOS* allene oxide synthase, *AOC* allene oxide cyclase, *OPR* 12-oxophytodienoate reductase 3, *OPC-8* 12-oxophytoenoic acid, *KAT* 3-keto-acyl-CoA-thiolase, *ACX* fatty acyl-CoA oxidase, *JMT* jasmonic acid methyl transferase, *JAR1* jasmonate resistant 1
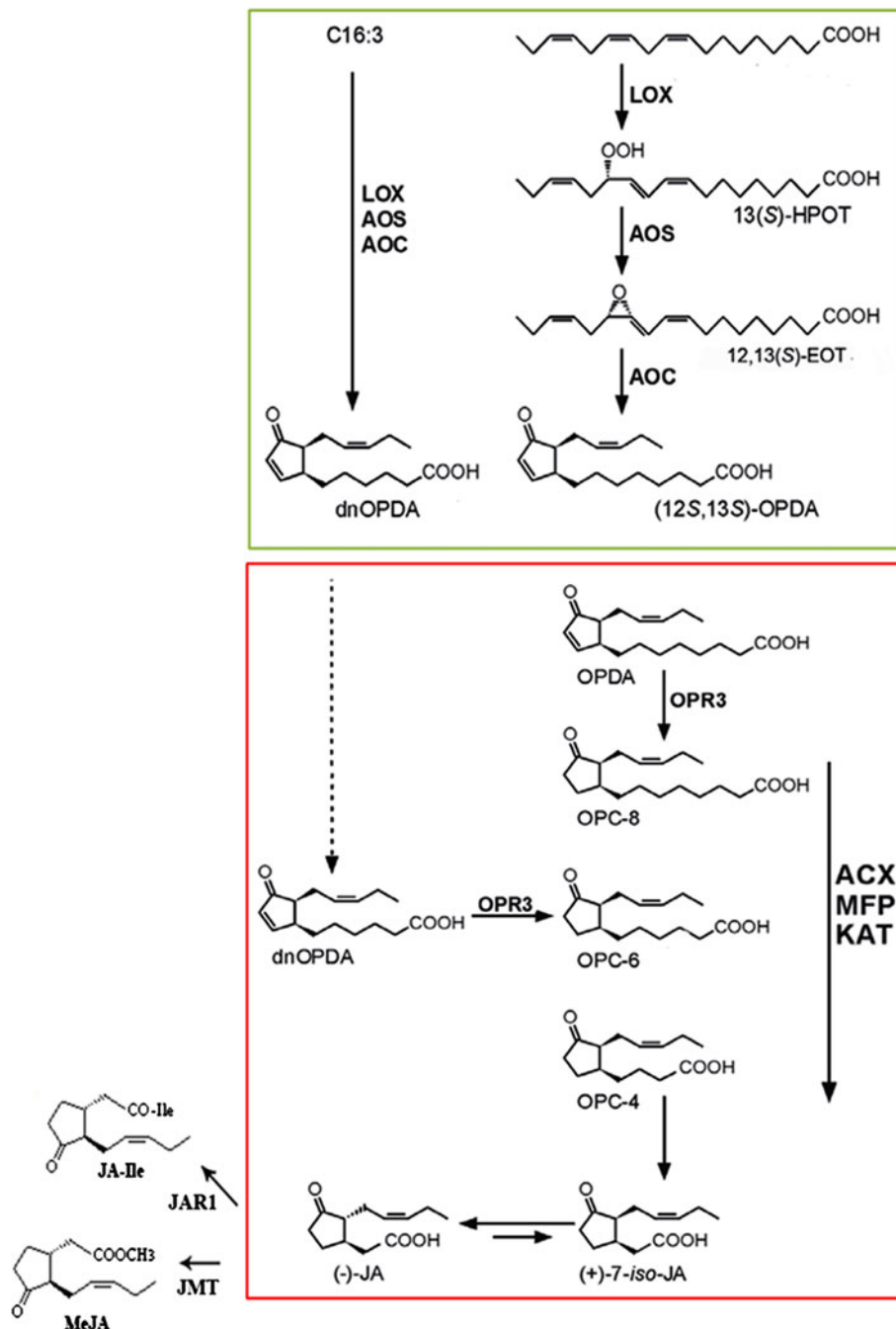
The mechanism of OPDA export from the chloroplast to peroxisome has not been completely understood even though it depends on the carrier COMATOSE1/PEROXI-MAL 1/PEROXISOME ABC TRANSPORTER (ABC CTS1/PXA1/PED3) (Zolman et al. 2001; Theodoulou et al. 2005; Footitt et al. 2007).

In the genomes of *Arabidopsis*, tomato and rice several *OPR* encoding genes have been identified (Schaller et al. 1998; Strassner et al. 2002; Tani et al. 2008). However, in *Arabidopsis* and tomato, only the OPR3 isoform is localized in the peroxisome and shows a strict specificity for the enantiomer (9S, 13S)-*cis*-(+) of OPDA (Schaller et al. 1999; Strassner et al. 2002). *OPR*3 gene expression is inducible by the exogenous application of JA or wounding, and the *Arabidopsis* mutant *opr3* is incapable of producing JA (Costa et al. 2000; Strassner et al. 2002).

JA synthesis proceeds with three rounds of β-oxidation that shorten the carbon side chain from the precursor molecule (Miersch and Wasternack 2000). These β-oxidation steps start with the activation of OPC:8 by esterification to OPC:8-CoA (Baker et al. 2006; Koo and Howe 2007). In the central steps of β-oxidation three different enzymes are involved sequentially: (1) an acyl CoA oxidase (ACX) as ACX1 and ACX5 involved in the synthesis of OPC:6 (Schilmiller et al. 2007), (2) a multifunctional protein (MFP) encoded by the gene *ABNORMAL INFLORESCENCE MERISTEM 1* (*AIM1*) involved in the synthesis of OPC:4CoA and (3) a 3-ketoacyl-CoA thiolase (KAT2) which catalyzes the formation of JA-CoA (Castillo and León 2008). Once synthesized, the (3R, 7S)-JA is released into the cytoplasm from peroxisome by an unknown mechanism (Fig. 1).

Jasmonate signal transduction

JA and MeJA have been considered for a long time to be the bioactive forms because of their physiological effects and abundance in the plant (Creelman and Mullet 1995). However, the characterization of *JAR1* gene encoding the enzyme responsible for conjugation of JA to some amino acids, i.e. isoleucine, valine, etc., and the identification of the corresponding *jar1* mutant demonstrated that JA and MeJA are only precursors of the bioactive molecule represented by the isoleucine conjugated jasmonoyl derivative (JA-Ile; Staswick and Tiryaki 2004; Fonseca et al. 2009a; Suza et al. 2010). Indeed, many data indicate that JA-Ile is the bioactive compound in JA signaling (Thines et al. 2007; Katsir et al. 2008; Fonseca et al. 2009b).

As extensively reviewed (Balbi and Devoto 2008; Chico et al. 2008; Staswick 2008; Browse 2009; Chini et al. 2009a; Gfeller et al. 2010; Pauwels and Goossens 2011; Kazan and Manners 2008, 2012; Wager and Browse 2012), the primary signal transduction process following JA perception converges on basic-helix-loop-helix (bHLH) related transcription factors (TFs). Of these, the best characterized is the multifunctional MYC2 (Fig. 2). Under stress conditions, JA response is controlled by a group of nuclear proteins, called JASMO-NATE-ZIM-DOMAIN (JAZ) repressors. JAZ repressors interact with the F-box protein COI1 (CORONATINE INSENSITIVE1), an integral part of the SCF (Skp-Cullin-F-box) complex involved in the co-reception of biologically active JA (JA-Ile) (Chini et al. 2007; Thines et al. 2007; Yan et al. 2007). To repress the transcriptional activity of downstream TFs involved in specific aspects of JA signaling, Pauwels et al. (2010) suggested a model in which the JAZ proteins block MYC2 activity in the absence of bioactive JAs by recruiting the general co-repressors TOPLESS (TPL) and TPL-related proteins through an interaction with the adaptor protein Novel Interactor of JAZ (NINJA). MYC2 blockage can be released by JA-Ile–induced and SCF^{COI1}-mediated degradation of the JAZ proteins (Balbi and Devoto 2008; Chico et al. 2008; Katsir et al. 2008; Kazan and Manners 2008; Staswick 2008; Browse 2009; Chini et al. 2009b; Chung et al. 2009; Fonseca et al. 2009a; Memelink 2009; Gfeller et al. 2010). Noteworthy, TPL was initially described as a co-repressor of auxin response that interacts with AUX/IAA repressors through the ETHYLENE RESPONSE FACTOR (ERF)-associated amphiphilic repressor (EAR) motif present in these proteins (Long et al. 2002, 2006; Szemenyei et al. 2008).

JAZ repressors have been shown to possess a particularly strong affinity for a number of related bHLH TFs such as MYC2, MYC3 and MYC4 as well as EGL1 (ENHAN-CER OF GLABRA3 1), GL3 (GLABRA3) and TT8 (TRANSPARENT TESTA 8) involved in both specific and overlapping aspects of JA signaling (Cheng et al. 2011; Fernández-Calvo et al. 2011; Niu et al. 2011; Qi et al. 2011).

A new regulating element in JA signaling has recently been described. The *JASMONOYL-L-ISOLEUCINE HYDROLASE 1* (*JIH1*) encodes a new homeostatic step in JA metabolism that, together with other inactivation steps of JA-Ile such as carboxylation or hydroxylation, attenuates the Ja-Ile burst contributing to regulate plant defense responses (Woldemariam et al. 2012). *jih1* silenced lines of *Nicotiana attenuata* showed reduced growth levels of the specialist *Manduca sexta* and the generalist *Spodoptera littoralis* and accordingly accumulate higher levels of JA-Ile derived metabolites. Interestingly, silenced plants also showed an increase in the release of herbivore-elicited volatiles resulting in an enhanced attraction of natural predators of *M. sexta* eggs.
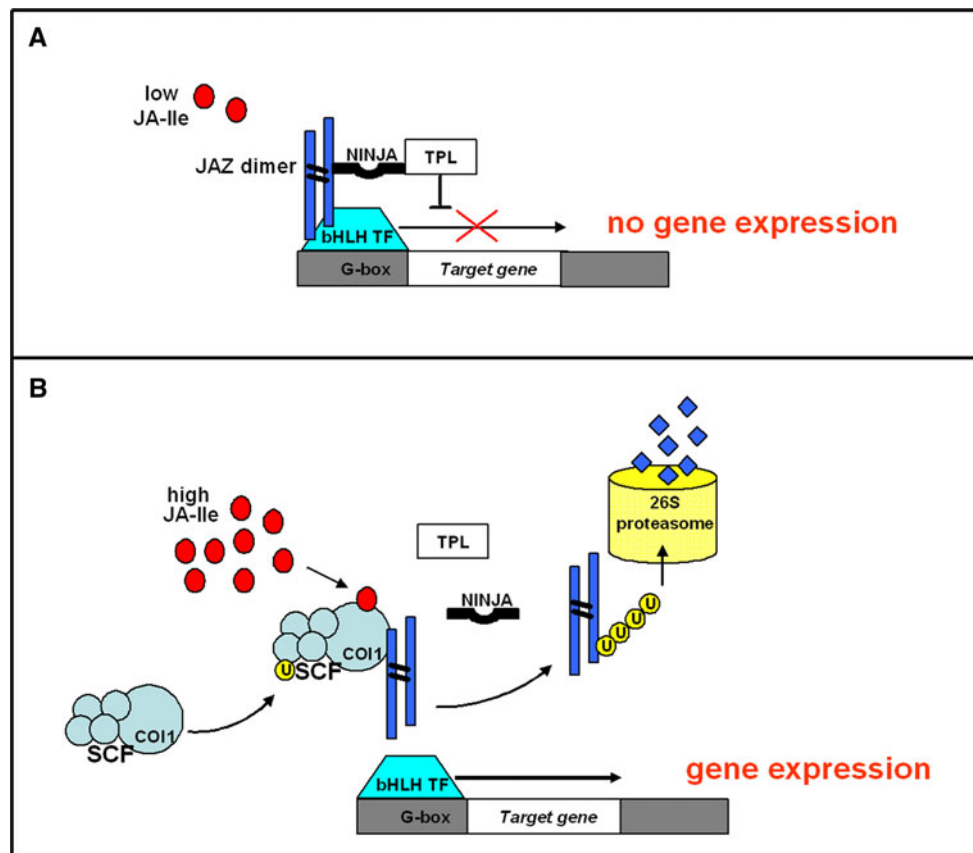
Fig. 2 Model of jasmonate perception and regulation of expression of jasmonate target genes. **a** bHLH transcription factors interact with the target sequence inside the jasmonate-responsive gene (G-box) promoters but their activity is repressed by homo- or heterodimers of JAZ proteins when JA-Ile levels are low. **b** JA-Ile level synthesis increases upon stress or developmental cues and it binds to the jasmonate receptor COI1, which is part of the SCF^{COI1} complex and the E2 ubiquitin-conjugating enzyme. Subsequently, JAZ proteins are recruited from their initial binding site to the COI1-JA-Ile unit of the SCF^{COI1} complex, which acts as an ubiquitin ligase transferring ubiquitin (U) from the ubiquitin-conjugating enzyme to the COI1-interacting JAZ proteins. Upon polyubiquitination, JAZ proteins are directed to the 26S proteasome for degradation, thus releasing bHLH TFs and activating expression of JA responsive genes, including those encoding JAZ proteins. Synthesis of new JAZ repressors results in repression of genes encoding for enzymes in JA biosynthesis pathway

JA in plant development and fertility

JA has a central role in plant development and reproduction (Creelman and Mullet 1997; Wasternack 2007; Browse 2009). It has been reported that jasmonates are involved in a large number of different physiological processes of plants, i.e. floral development, senescence induction, growth inhibition, fruit ripening, tendril coiling, mechanotransduction, potato tuberization, trichome formation and fungi arbuscular mycorrhizal association (Parthier 1991; Koda et al. 1992; Sembdner and Parthier 1993; Koda 1997; Creelman and Mullet 1995, 1997; Wasternack and Hause 2002; Browse 2005; Wasternack 2007; Balbi and Devoto 2008; Pauwels et al. 2008, Zhang and Turner 2008; Reinbothe et al. 2009; Yoshida et al. 2009). JA was also described as an important regulator of leaf and root morphogenesis in soybean (Xue and Zhang 2007).

Furthermore, JA also controls plant fertility. *Arabidopsis* mutants deficient in JA synthesis, (McConn and Browse 1996; Sanders et al. 2000; Stintzi and Browse 2000; Ishiguro et al. 2001; Park et al. 2002; von Malek et al. 2002) or JA-perception (Feys et al. 1994; Browse 2009) are male sterile. These data demonstrate a regulatory role of JA in fertility control. In *Arabidopsis*, JA acts at three levels, controlling filament elongation, anther dehiscence and pollen viability (Browse 2009). Furthermore, JA induces the expression of numerous genes in stamen tissue, including several TFs of the MYB family, such as MYB21, MYB108 and MYB24, that play an essential role in fertility (Mandaokar et al. 2003, 2006; Cheng et al. 2009, Mandaokar and Browse 2009). Therefore, it has been hypothesized that JA controls male fertility, regulating stamen elongation and anther development, through MYB24 and MYB21 that are the targets of JAZ repressors (Song et al. 2011).

Jasmonate signaling in response to biotic stresses

Plants have to deal with a complex environment in a continuous exposure to biotic stresses caused by different attackers such viruses, bacteria, fungi and insects. However, along the battle against potential invaders, plant can also establish beneficial relationships with others microorganisms that can promote defense and growth. Responses to biotic stresses are mainly mediated by plant hormones such as salicylic acid (SA), JAs and ethylene (ET), which act as primary signals in the regulation of plant defense (Fig. 3). In general, local and systemic defense response, including systemic acquired resistance (SAR), against biotrophic pathogens is mediated by SA (Durrant and Dong 2004), whereas JA and ET mediate responses against necrotrophs (Glazebrook 2005). However, this classification appears to be simplistic since many potential pathogens change their pathogenic style along their life cycle. Even the crosstalk between SA and ET/JA pathways can be either mutually antagonistic or synergistic, and the result is

a more complex crosstalk among pathways (Beckers and Spoel 2006; Adie et al. 2007; Spoel et al. 2007).

Jasmonates in plant–insect interaction

The role of jasmontes in plant defense signaling in response to insects attack was shown to be very specific. It has been demonstrated that *Arabidopsis* attacked by the specialist insect herbivore *Pieris rapae* activates the expression of only one branch of the downstream JA signaling that is regulated by *MYC2*. This specific response induces the expression of the *VEGETATIVE STORAGE PROTEIN 2* (*VSP2*) gene. Interestingly, the ERF-branch, which is activated upon necrotrophyc pathogenic attack, was repressed together with the down-regulation of the *OCTADECANOID-RESPONSIVE ARABIDOPSIS 59* (*ORA59*) transcription factor and the marker gene *PLANT DEFENSIN 1.2* (*PDF1.2*) (Verhage et al. 2010). When the MYC2 branch is suppressed in *myc2* mutant or in the JA-Ile defective mutant *jar1-1*, JA-dependent response upon
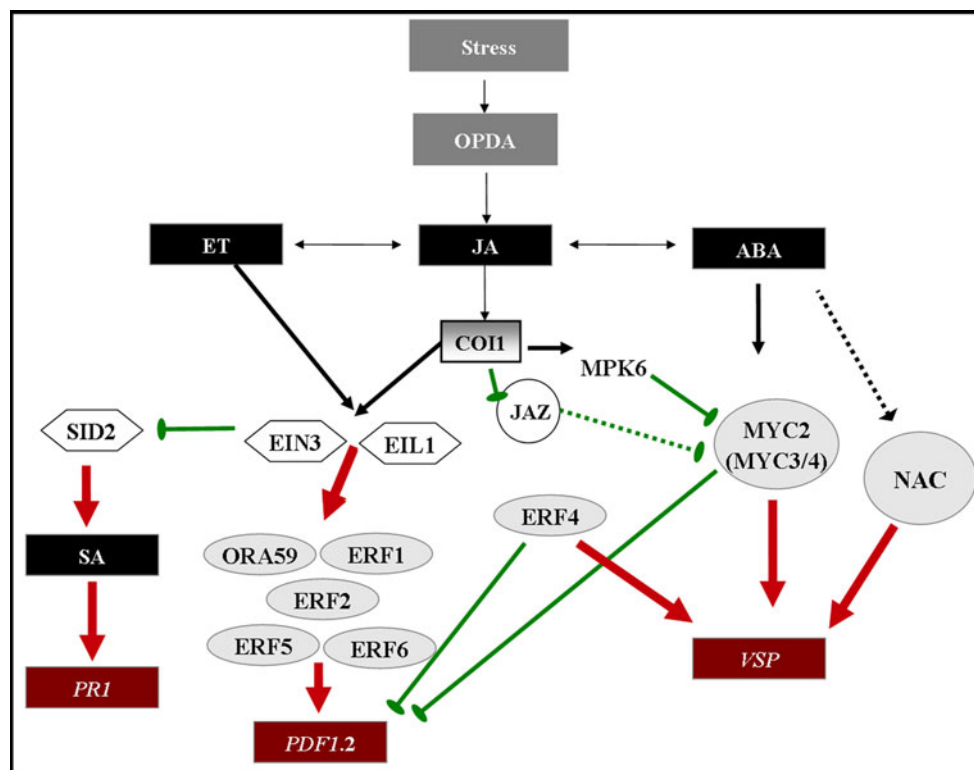

Fig. 3 Scheme of JA signaling pathway and phytohormone crosstalk. Plants challenged by different (a)biotic stresses respond by inducing the synthesis and activation of JA-dependent responses. The active form of JA, JA-Ile is perceived by COI1 and modulates downstream gene response through *MYC2/MYC3/MYC4*. These TFs, also regulated by ABA signaling, drive the expression of JA-dependent defenses, such as *VSP*. ABA signaling can also regulate NAC TFs. The other branch of JA signaling involves the crosstalk with ET,

which regulates the expression of *PDF1.2* through some TFs such as *ORA59* and some *ERFs* (*ERF1*, *ERF2*, *ERF5* and *ERF6*), which can induce or repress *PDF1.2* or *VSP*. The JA-activated MPK6 kinase exerts a positive and negative regulation on *PDF1.2* and *VSP*, respectively. JA/ET also induce the expression of *EIN3* and *EIL1*, which suppress the synthesis of SA through the repression of *SID2*. This blocks the induction of *PR1* and could explain the antagonistic action between JA and SA

P. rapae infestation was redirected through the ERF-branch (Verhage et al. 2011). Despite that the *jar1* and *jin1* mutants showed an enhanced expression of the ERF-branch, in a series of choice experiments, both are preferred by *P. rapae* compared with Col-0 plants. This clearly points to the MYC2 as the main branch involved in the *Arabidopsis* insect-wounding resistance mechanism (Verhage et al. 2010; Fig. 3). In a latter experiment, Verhagen and co-workers demonstrated that *P. rapae* salivae elicited the ERF branch that may be responsible for secretion of insect attractants. However, a full insect attack switched plant defenses into a more active MYC2 branch and the concomitant repression of the ERF-controlled branch. Other authors also demonstrated that *MYC2* induction mediated by ABA may have a role in the redirection of the JA pathway upon insect attack. This was concluded by the finding that the *aba2* mutant did not show *MYC2* activation and from the strong repression of *ERF* observed upon exogenous ABA treatments.

In addition to the specialist *P. rapae*, a very similar pattern of response has also been observed after the attack of a generalist insect such as *Spodoptera litoralis* (Bodenhausen and Reymond 2007).

Although our knowledge of the role of JA in the root inter-phase is limited, it is known that belowground responses of plants are rather different (Erb et al. 2012). Roots can perceive belowground attackers; however, it is not clear whether this response is specific or due to the perception of tissue disruption (Van Dam 2009; Erb et al. 2012). The mediation of JA in such insect–root interaction seems clear with some differences since the local amounts of JA accumulated in the roots are modest compared with the systemic JA levels found in leaves upon belowground root infestation. These responses were reported in *Medicago truncatula*, *Arabidopsis* and *Nicotiana attenuata* (Erb et al. 2012). Thus, the JA-burst in the roots is attenuated compared with the leaves. Furthermore, it is also found that the JA increase in the root was not connected to volatile compounds production and release as it happens in leaves; therefore, it was speculated that the root may perceive other jasmonates different from JA or JA-Ile that can effectively regulate belowground plant responses to insects and wounding (Erb et al. 2012).

Apart from the already commented roles of JA in plant–insect interactions, several new aspects of JA mediation in plant defense have been described. Plants previously exposed to abiotic or biotic stresses can keep a memory of the stress to respond more efficiently after a second stress experience. This so called priming has been mainly studied in plant–pathogen interactions (Conrath et al. 2006; Pastor et al. 2012). Most of the priming reactions are mediated by SA-dependent signals (Zimmerli et al. 2000; Ahmad et al. 2011). But the fact that SA can be a hormone that mediates

priming does not exclude a role for ABA and JA as other mediators of such responses against different challenges. Indeed, JA and its conjugates have been proposed as the mobile signals to provide long-distance priming in arthropod-infested plants (Frost et al. 2008). The initial reports of priming against insects were proposed to be mediated by green leaf volatiles (GLVs; Engelberth et al. 2004). Maize plants exposed to these volatiles induced higher amounts of JA and produced more herbivore-induced volatiles upon caterpillar infestation or wounding treatments. It is noteworthy that treatments with (Z)-3-hexenal primes the expression of the octadecanoid pathway, the production of α-linoleic acid and JA in poplar leaves (Frost et al. 2008). Unfortunately, the molecular mechanisms behind JA-mediated priming against insects are still elusive. What is clear is that priming is a horizontal multicomponent phenomenon that involves many different signaling pathways that differ depending on the challenge to which a plant is exposed (Frost et al. 2008; Pastor et al. 2012). Similar to the mechanisms described for SA-mediated priming, the participation of ROS and mitogen-activated protein kinases (MAPKs) are very likely mechanisms since they are already involved in those plant responses against insects that end-up with the accumulation of oxylipins and the activation of the JA-dependent signals. A recent breakthrough report pointed to the occurrence of long-lasting priming against insects that is mediated by JA (Rasmann et al. 2012). The authors demonstrated that *Arabidopsis* or tomato plants that have been pre-treated with MeJA or wounded were able to transfer to their offspring an enhanced resistance against caterpillars which grew smaller in primed plants. Priming was blocked in *coi1* and also in mutants with impaired biogenesis of small interfering RNA, suggesting that an epigenetic inheritance mechanism is behind the JA-mediated stress memory against insects.

Jasmonates in plant–pathogen interactions

JA signaling may interact synergistically or antagonistically with SA during plant–pathogen interaction (Durrant and Dong 2004). In the rice-*Xanthomonas oryzae* pv. *oryzae* interaction, the activation of *MITOGEN-ACTIVATED PROTEIN KINASE 6* (*MPK6*) resulted in an accumulation of JA and SA and induced expression of JA and SA responsive genes. Local resistance against the fungal necrotrophic pathogen *Botrytis cinerea* induces SA and JA-mediating signaling in *Arabidopsis* (Ferrari et al. 2003). In systemic resistance it has been found that a fast and transient accumulation of JA in phloem exudates is needed for the initiation of SAR. After challenge with the avirulent *Pseudomonas syringae* avr Rpm1, systemic induction of JA biosynthesis genes was detected prior to SA induction (Truman et al. 2007). Thus, JAs participate in

long-distance information transmission. Interestingly, *MYC2* was also induced systemically, repressing JA responsive markers associated with local defense response in *A. thaliana* (Lorenzo et al. 2004). JA and SA seem to act in tandem, with a first and transient induction of JA followed by SA-mediated defense response. SA and JA can also combine their action to optimize the response to a single attacker. For instance, inoculation with the hemibiotrophic bacteria *Pseudomonas syringae*, which induces SA-mediated defenses, turned plants more susceptible to *Alternaria brassicicola* by suppressing JA-signaling pathway, and it is partially dependent on *NONEXPRESSOR OF PR GENES 1* (*NPR1*). This trade-off is only effective in local tissues, whereas in distal ones *A. brassicicola* infection was not affected. Furthermore, this suppression of JA-dependent defenses is not present in plants infected with an avirulent strain of *Pseudomonas* (Spoel et al. 2007).

COI1 has been described to have a central role in JA signaling for disease resistance against oomycetes and fungi (Vijayan et al. 1998; Glazebrook 2005; Adie et al. 2007; Chini et al. 2007). However, as with many events in biotic interactions, it may depend on specific plant–pathogen interaction. The hemibiotrophic root fungus *Fusarium oxysporum* required COI1 for promoting susceptibility (Thatcher et al. 2009), as well as the soil-borne bacteria *Ralstonia solanacearum* (Hernández-Blanco et al. 2007). *coi1* mutant is more resistant to these pathogens and fails to develop the symptomatic chlorotic lesions caused by the bacterial effector coronatine (COR).

The interplay between JA and SA can be also supported by ethylene (ET; Fig. 3). In the tomato–*Alternaria alternata* interaction, ET and JA pathways are necessary for susceptibility, with JA and ET acting synergistically. Conversely, SA signaling promoted resistance of tomato plants to infection against *Alternaria alternata* f. sp. *lycopersici* and antagonized ET signaling during infection (Jia et al. 2012).

A key step in the activation of plant immune response is mediated by transcription factors (TFs) that control the transcriptional regulation of related defense genes (Fig. 3). Several members of the ERF family have shown to play a role in the JA-responsive gene expression against pathogens. Among them, ORA59, ERF1, ERF2, ERF5 and ERF6 have a clear impact over *PDF1.2* gene expression, conferring resistance to the necrotrophic fungi *B. cinerea* and *Alternariabrassicicola* (Berrocal-Lobo et al. 2002; Brown et al. 2003; Lorenzo et al. 2003; MacGrath et al. 2005; Moffat et al. 2012) by integrating JA/ET signals and the ERF4 TF. ERF4 repressed *PDF1.2* but induced *VSP2* (Memelink 2009). Interestingly, the basic leucine zipper (bZIP) TFs of the TGACC motif family (TGA2, TGA5 and TGA6), required for establishment of SAR and mediated by the hormone SA, were also essential for activation of

JA/ET responses that counteract necrotrophic pathogens. The triple mutant *tga2-tga5-tga6* was blocked in *PDF1.2* expression upon *B. cinerea* and *Pseudomonas syringae maculicola* ES4326 infection (Zander et al. 2010). In this case ET may be the key signal required for TGA TFs since *PDF1.2* expression was unaffected by external treatments of JA (Ndamukong et al. 2007).

Another important TF in JA signaling is MYC2 (Lorenzo et al. 2004). This TF is a key point of the ABA/JA signaling and regulates different responsive genes to both hormones (Fig. 3). TIME FOR COFFEE (TIC) interacts physically with MYC2 in the nucleus and is able to repress the inhibition of root growth mediated by JA in a MYC2-dependent manner and promotes resistance against *Pseudomonas syringae* through a negative regulation of JA signaling that requires the interaction with MYC2. On the other hand, JA promotes MYC2 protein accumulation in a circadian clock-dependent manner. Plants are more susceptible to *P. syringae* DC3000 at the beginning than at the end of light cycle, and the expression of JA responsive genes *JAZ5* and *MYC2* after JA treatment was higher when the light cycle starts (Shin et al. 2012).

The phytotoxin COR is a structural mimic of JA-Ile (Fonseca et al. 2009a) that is secreted by the virulent bacteria *P. syringae* pv tomato DC3000, and it is injected into host cells by a type III effector system to suppress basal defenses. COR is over 1,000 times more active than JA-Ile in vitro and both compounds are recognized by the same receptor (Katsir et al. 2008). It has been described that COR promotes virulence by suppression of SA accumulation via COI1 activation (Uppalapati et al. 2008). COR also suppresses SA-independent callose accumulation mediated by *PEN2*-dependent pathway during *P. syringae* infection. Therefore, COR suppresses both SA-dependent and SA-independent *Arabidopsis* defense responses (Geng et al. 2012). These TFs participate in the COR-mediated reopening of stomata and bacterial propagation by inhibiting the accumulation of SA (Zheng et al. 2012).

Another significant aspect (Kazan and Manners 2012) relies on the balance between plant growth and response to a specific pathogen. DELLAs (GROWTH REPRESSOR of GA1-3, RGA1 and RGA2) compete with MYC2 to bind JAZs in the GA-JA crosstalk, and in the same way JAZ proteins could compete for DELLA targets such as PIF3 and PIF4 (PHYTOCHROME INTERACTING FACTOR). Therefore, the equilibrium between DELLA-JAZ proteins appears to contribute to both, growth suppression when the plant is challenged by a necrotrophic pathogen or defense suppression when the pathogen threat has been overcome (Navarro et al. 2008). Furthermore, JAZ repressors in JA signaling appear to have broad roles in diverse phytohormone signaling pathways. For example, JAZs are known to have synergistic or antagonistic interactions with ABA and

therefore it is possible that JAZ proteins play roles in mediating the JA-ABA crosstalk in *Arabidopsis* and tobacco plants (Lackman et al. 2011).

ABA is required for JA production and subsequent defense induction (Adie et al. 2007). The *A. thaliana* mutant *ocp3* (OVEREXPRESSOR OF CATIONIC PEROXIDASE 3; a member of the homeobox transcription factors family) is resistant to *Plectosphaerella cucumerina*, needs ABA for callose induction and shows higher levels of ABA and *ABI4* (*ABSCISSIC ACID INSENSITIVE 4*) transcript levels compared with the control Col-0 (García-Andrade et al. 2011). Interestingly, COI1 is not fundamental for basal callose accumulation since *coi1* and Col-0 showed similar levels of basal callose, but it is necessary for callose deposition after infection. The double mutant *ocp3coi1* is hypersusceptible to necrotrophs (Coego et al. 2005) showing that a correct JA signaling is necessary for resistance. Therefore, JA as well as ABA participate in the regulation of callose deposition in response to pathogen infection (Ton and Mauch-Mani 2004).

Jasmonate signaling in response to abiotic stresses

As sessile organisms, plants must develop various biochemical and physiological mechanisms to respond, adapt and acquire tolerance to a determinate stress. Adaptation to stress has been suggested to be mediated by both preexisting and induced defenses (Bray et al. 2000; Hasegawa et al. 2000; Pastori and Foyer 2002). Abiotic stresses such as drought, salinity, cold and heavy metal cause average yield losses of more than 50 % in major crops. Abiotic stresses are characterized by both ionic and osmotic disequilibrium, components that elicit general as well as specific mechanisms of stress protection mediated by hormones like JAs and ABA (Fig. 3).

Salt, drought and osmotic stresses

The physiological mechanisms governing plant responses to salinity and drought show high similarity, suggesting that both stresses must be perceived by plant cell as deprivation of water (Jakab et al. 2005). High salt (most commonly NaCl) concentrations in the soil lead to water potential decrease, which affects water availability (Hasegawa et al. 2000). In addition to the hyperosmotic shock and the subsequent oxidative stress (Borsani et al. 2001), deleterious consequences of high NaCl concentration in the apoplast also include ion toxicity and nutrient imbalance (Serrano et al. 1999; Hasegawa et al. 2000; Rodriguez-Navarro 2000).

Leaves are the main sites of JA biosynthesis (Creelman and Mullet 1997; Mueller 1997) although there is evidence of the presence of the complete biosynthetic pathway machinery in the roots (Pedranzani et al. 2003). One such evidence is the high expression of the main structural genes of the JA pathway in root tissues of various plant species under different physiological conditions. The observed increase in gene transcripts correlates with higher levels of JA/JA-Ile and their precursor OPDA.

An increase in the endogenous levels of leaf OPDA, JA and methyl jasmonate (Me-JA) was reported in sorbitol-treated tomato plants (Abdala et al. 2003). However, the most remarkable increase was recorded for OPDA (Kramell et al. 2000). An increase in the levels of OPDA, JA and JA-Ile was also reported from salt-treated hairy roots, showing that these oxylipins are involved in the response to salt. The hypothesis that JA and OPDA are powerful and independent signal molecules has been more recently confirmed (Böttcher and Pollmann 2009). Abdala et al. (2003) examined the response to saline stress of hairy roots from two tomato cultivars with different sensitivity to NaCl. The results suggested that changes in endogenous JAs were different in genotypes of contrasting salt tolerance.

In the model legume *M. truncatula*, a transcriptome analysis based on the 16K+ microarrays (Mt16KOLI1) using salt-treated root apices (Gruber et al. 2009) showed that among hormone and secondary metabolism category, four key-genes involved in the oxylipins metabolism, namely lipoxygenase, hydroperoxide-lyase, allene oxide synthase, and allene oxide cyclase were all up-regulated in the salt-tolerant genotype Jemalong A17, under salt stress condition. More recently, we showed that either the AOS or HPL branch is rapidly (already at 2 h after onset of stress) and significantly (up to about 19-fold induction) induced by drought stress in the tolerant chickpea variety ICC 4958. The early activation of the JA pathway was also confirmed by the concomitant increased levels of OPDA, JA and JA-Ile at 2 h after stress onset (De Domenico et al. 2012).

In citrus plants, a rapid increase in JA concentration was observed in roots after salt stress imposition, reaching maximum levels 6 h after the onset of stress. Gene expression analysis of key enzymes in JA biosynthesis pathways indicated a transient up-regulation of the Ct*lox3*, Ct*aos* and Ct *aoc* orthologs at 4 h after the stress onset (Mahouachi et al. 2007; Arbona and Gómez-Cadenas 2008; Arbona et al. 2010).

In *Vitis vinifera* cell cultures three representatives of the JAZ/TIFY gene family have been studied in relation to NaCl stress. Ismail et al. (2012) showed that two JAZ/TIFY members (JAZ1/TIFY10a and JAZ3/TIFY6b) were highly up-regulated in the salt-tolerant *V. rupestris*. In parallel, MYC2 transcription factor as well as the JA receptor gene COI1 was both slightly up-regulated. Since the activity of

JA signaling is primarily regulated at post-translational level (e.g. by proteolytic degradation of the inhibitory JAZ/TIFY proteins), these transcript responses of JAZ1/TIFY10a and JAZ3/TIFY6b as well as of MYC2 and COI1 are probably not directly involved in stress signaling.

Chilling stress

Treatment of whole plants with MeJA before chilling rather than during or after stress imposition greatly improves the survival ratio of chilled rice seedlings (Lee et al. 1997). Chilling injuries such as electrolyte leakage and water loss were also ameliorated by MeJA pre-treatment. Root application of MeJA significantly promotes tolerance of rice seedlings to chilling stress, while shoot application appears to be less effective. It has been hypothesized that MeJA itself or MeJA-induced tolerance signals could be transported from roots to shoots and thus prevent chilling damage. MeJA might prevent water loss in chilled rice seedlings by reducing stomatal opening and enhancing hydraulic conductivity. Thus, rice seedlings pretreated with MeJA maintain their well-watered status after chilling exposure.

Chilling-induced expression of three Ω-3 fatty acid desaturases (FAD) was greatly reduced in *A. thaliana aos* mutant characterized by impaired JA levels. Also, chilling-induced increase in C18:3 polyunsaturated fatty acids was reduced in the *aos* mutant (Shi et al. 2011). Taken together, these results pointed a role for JAs in chilling-induced response, which affects membrane lipid composition. MeJA has also been shown to improve chilling tolerance and reduce stress injuries in several fruits (Cao et al. 2009; Jin et al. 2009a, b; Sayyari et al. 2011; Zhao et al. 2012). Two MYC2-like genes (Ma*MYC2a* and *MYC2b*) and an ICE (a MYC-type bHLH) transcription factor were both rapidly induced by MeJA and might be involved in MeJA-induced tolerance to chilling stress by interacting with some components of the cold signal pathway (Zhao et al. 2012). It is noteworthy that MaMYC2s interact with Ma-ICE1 to form a heterodimer that can bind to G-box elements within the promoters of their target genes to activate JA-induced transcription activation (Zhao et al. 2012).

JA signaling in multiple biotic/abiotic stresses

Plant needs to counteract pathogen and insect attacks, but at the same time they have to perceive simultaneously abiotic stress signals to decide the best strategy to optimize responses to overcome successfully those challenges. The integration between JA- and ABA-mediated signals along with multiple simultaneous biotic and abiotic stresses remains mainly unknown. Recent studies have pointed to nutrient transporters as transceptors that may activate downstream signals that balance plants response to favor biotic or abiotic signals. However, these investigations were focused in the interaction between bacterial pathogens, SA signaling and nitrogen depletion (Camañes et al. 2012; Dechorgnat et al. 2012).

Multiple stresses are not limited to simultaneous abiotic and biotic challenges. For example, transcription factors also have a relevant role regulating the transcriptome of tobacco following multiple infestations of the sap-feeding mirid (*Tupocoris nnatus*) and the chewing hornworm (*Manduca sexta*) (Voelckel and Baldwin 2004). Interestingly the plant, through TFs action, reorganizes its transcriptome differently when it is attacked by only one insect species or both the insect species at the same time.

Several families of transcription factors involved in the crosstalk among SA, JA and ABA have been characterized. *MYC2, MYB2, ATAF1* and *2, RD26, WRKY82* and *ZAT7* have been reported to regulate the interplay between JA and other hormones after wounding and abiotic stresses (Wu et al. 2009; Atkinson and Urwin 2012). *MYC2*, as reported above, plays multiple roles in ABA, JA, ET, SA signaling and during abiotic stress response (Fujita et al. 2009; Fig. 3). This transcription factor binds to a CACNTG core that is present in genes that respond to drought and ABA (Abe et al. 2003), light (Yadav et al. 2005) and JA-regulated genes (Dombrecht et al. 2007).

An interesting crossroad between the ABA and JA signaling is the *NAC* family of transcription factors. This family is formed by the subfamilies ATAF, NAM and CUC transcription factors (Mauch-Mani and Flors 2009). *ATAF2* is involved in response to wounding, salinity stress and hormones treatments, such as SA and JA. It down-regulates responses to soil pathogens, whereas its homolog *ATAF1* responds to dehydration, wounding and ABA treatments (Collinge and Boller 2001; Wu et al. 2009). Another interesting NAC transcription factor is *RD26* that is stimulated by JA treatments, H_2O_2, pathogen infections and ABA among other signals (Zimmermann et al. 2004; Fujita et al. 2009).

The last group of transcription factors that participate in JA signaling are ERFs. In tobacco the *Tobacco Stress-Induced-1(TSI1)* is activated by MeJA, wounding and also by SA and ethephon (Park et al. 2001). These TFs bind to GCCGCC boxes that are present in many ET-inducible genes encoding PR proteins. *TSI1* over-expression enhanced tolerance to osmotic stress and also to biotrophic pathogen infection by accumulating constitutively higher levels of PR proteins (Park et al. 2001). Similarly, a pepper ERF transcription factor (*ERFLP1*) is also induced by pathogen infection and mechanical wounding contributing to the resistance to both stresses (Lee et al. 2004).

Concluding remarks

The biological significance of the molecular crosstalk between different signaling pathways that operate under various stress conditions and the mechanisms that underlie this crosstalk remain largely obscure. We are just at the beginning to dissect key factors governing this crossroad. As research on the biosynthesis and activity of plant hormones progresses, it is becoming clear that a large diversity of metabolites related to different phytohormones play a role in plant defense and plant–environment interactions. Amino acid conjugation, hydroxylation, methylation and glucosylation, in particular, are prominent biochemical processes that diversify the metabolic arsenal of phytohormone families. The temporal and spatial patterns of these compounds may represent an additional step of regulation. New analytical approaches and a close collaboration between biologists, chemists and agronomists will improve our knowledge on phytohormones and are likely to lead to the discovery of new regulatory elements of plant stress responses.

The involvement of JAs in plant defense signaling has claimed a lot of attention in recent years. Oxylipins and JA-derivatives have active roles in resistance against a large diversity of (a)biotic stresses. However, plant immune system is multicomponent and involves complex signals that interact and crosstalk with each other.

Whereas there is an extensive literature on the response of plants to single stresses under laboratory conditions, the study of how plants respond to multiple (a)biotic stresses is still at its infancy (Mittler and Blumwald 2010). From this overview it clear that there is a deep interplay of phytohormone signaling at local and distant tissues, which regulate transcription factors belonging to different families. The crossroad between stress adaptation and cell death and the determinant events leading to one or the other direction represent a challenging research area in the context of global climate changes. Increasing our knowledge about the molecular, physiological and metabolic aspects of plant response to multiple stresses will be vital to develop new varieties that are able to better cope with future global climate changes.

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