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INVITED REVIEW

Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in *Annals of Botany*

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• *Background* Jasmonates are important regulators in plant responses to biotic and abiotic stresses as well as in development. Synthesized from lipid-constituents, the initially formed jasmonic acid is converted to different metabolites including the conjugate with isoleucine. Important new components of jasmonate signalling including its receptor were identified, providing deeper insight into the role of jasmonate signalling pathways in stress responses and development.
• *Scope* The present review is an update of the review on jasmonates published in this journal in 2007. New data of the last five years are described with emphasis on metabolites of jasmonates, on jasmonate perception and signalling, on cross-talk to other plant hormones and on jasmonate signalling in response to herbivores and pathogens, in symbiotic interactions, in flower development, in root growth and in light perception.
• *Conclusions* The last few years have seen breakthroughs in the identification of JASMONATE ZIM DOMAIN (JAZ) proteins and their interactors such as transcription factors and co-repressors, and the crystallization of the jasmonate receptor as well as of the enzyme conjugating jasmonate to amino acids. Now, the complex nature of networks of jasmonate signalling in stress responses and development including hormone cross-talk can be addressed.

Key words: Jasmonic acid, oxylipins, enzymes in biosynthesis and metabolism, perception, JA signalling, JAZ, SCF, COI1, responses to herbivores and pathogens, symbiotic interaction, light regulation, JA in development.

1. INTRODUCTION

In 2007, an 'Update on jasmonates' was published in *Annals of Botany* covering aspects of biosynthesis, signal transduction and action in plant stress responses, growth and development (Wasternack, 2007). In this previous review, genes and enzymes/proteins involved in biosynthesis, metabolism and signalling were described with respect to the wound response and some developmental processes regulated by jasmonic acid (JA). In 2007, however, there was a breakthrough in analysis of JA signalling with the discovery of the so-called JAZ proteins (JAZMONATE ZIM DOMAIN proteins) as negative regulators in JA-induced gene expression. Three groups identified independently JAZ proteins as targets of the SCF^{COI1} complex, where COI1 is the F-box protein as part of the Skp1/Cullin/F-box protein complex which functions as an E3 ubiquitin ligase (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007). COI1 (CORONATINE INSENSITIVE1) was identified in *Arabidopsis thaliana* in 1998, and the corresponding mutant *coi1-1* is the most prominent JA signalling mutant (Xie *et al.*, 1998). With the JAZ proteins, however, the first mechanistic explanations were possible on JA perception, including identification of (+)-7-*iso*-jasmonoyl-L-isoleucine (JA-Ile) as the ligand of a JA receptor (Fonseca *et al.*, 2009). This was complemented by crystallization of the COI1–JAZ co-receptor complex (Sheard *et al.*, 2010), its potentiation by inositol pentakisphosphate (IP_5) and identification of the general co-repressor TOPLESS (TPL) and the adaptor protein Novel Interactor of JAZ (NINJA) (Pauwels *et al.*, 2010). Finally, in 2012, JAR1 (JASMONOYL ISOLEUCINE CONJUGATE SYNTHASE1), the essential enzyme in generation of the most bioactive jasmonate compound active as the ligand of the receptor, was crystallized (Westfall *et al.*, 2012).

The identification of these key components in JA perception and signalling allowed identification of downstream targets, the transcription factors (TFs), acting specifically in numerous JA-dependent processes. This led to the first mechanistic explanations of how cross-talk among the different hormones and signalling pathways may occur. That a similar modular principle occurs in jasmonate, auxin, gibberellin (GA) and ethylene (ET) perception and signalling represents one of the most fascinating discoveries in the last few years of plant hormone research.

Beside these fundamental breakthroughs, there has been remarkable improvement in our knowledge on the metabolic fate of JA/JA-Ile, on short- and long-distance signalling, and on cross-talk to other hormones. The role of JA/JA-Ile in plant immunity, herbivory and mycorrhiza has been intensively studied. Several developmentally regulated processes such as seed germination, seedling development, root growth, flower development, seed development, tuber formation and senescence were shown to be regulated by JA/JA-Ile. Finally, the first

hints were found for the regulation of JA/JA-Ile signalling by light. Several of these numerous aspects on JA/JA-Ile have been repeatedly discussed in excellent reviews (Katsir *et al.*, 2008*a*; Kazan and Manners, 2008, 2011, 2012; Browse, 2009*a*, *c*; Grant and Jones, 2009; Koo and Howe, 2009; Kuppusamy *et al.*, 2009; Wasternack and Kombrink, 2010; Ballaré, 2011; Pauwels and Goossens, 2011; Robert-Seilaniantz *et al.*, 2011; Dave and Graham, 2012; Kombrink, 2012; Pieterse *et al.*, 2012).

In view of these recent developments, there is an emerging need to complement the earlier update on jasmonates (Wasternack, 2007). Taking new information and fundamental breakthroughs into consideration, we will discuss here in parallel the multifarious roles of jasmonates in plant stress responses and development. However, the amount of published data on various aspects of jasmonates is too exhaustive to cite here due to space limitations.

Furthermore, some subjects such as 'JA in response to pathogens', 'JA in herbivory and plant–insect interactions' and 'JA in light signalling' are not covered in detail because some excellent reviews have been published recently (see above).

2. JA BIOSYNTHESIS

The biosynthesis of JA has been repeatedly and extensively reviewed in recent years (Wasternack, 2007; Browse, 2009*a*, *c*; Schaller and Stintzi, 2009; Acosta and Farmer, 2010; Wasternack and Kombrink, 2010; Kombrink, 2012). These reviews present excellent information on reactions, genes, enzymes (including, in several cases, the crystal structures and mechanistic explanations on substrate specificity) and finally regulation of JA biosynthesis. In Fig. 1 we introduce reaction steps, names of enzymes and substrates and refer the reader to the above mentioned reviews for details. Here, we cover only some aspects, where interesting developments have been reported over the last couple of years.

2.1. Release of linolenic acid from galactolipids involved in JA biosynthesis

The fatty acid substrate of JA biosynthesis is α-linolenic acid (18:3) (α-LeA) released from galactolipids of chloroplast membranes. It is generally accepted that a phospholipase1 (PLA_1) releasing α-LeA from the *sn1* position of galactolipids is responsible for generation of the JA substrate, whereas the large family of PLA_2s are not involved in JA biosynthesis (for nomenclature of phopsholipase A enzymes see Scherer *et al.*, 2010). It was, however, a matter of debate as to which of the PLA_1s are involved in JA biosynthesis. Initially, DEFECTIVE IN ANTHER DEHISCENSE 1 (DAD1) was shown to be responsible for JA formation as the mutant *dad1* showed reduced JA levels exclusively in flowers and was therefore male-sterile like the *coi1* mutant (Ishiguro *et al.*, 2001). This DAD1 function was strongly substantiated by identification of *DAD1* as a target of the homeotic protein AGAMOUS (Ito *et al.*, 2007). AGAMOUS binds to the *DAD1* genomic region only during late stamen development. In this way, AGAMOUS orchestrates elongation of filaments, maturation of pollen and dehiscence of anthers, the three critical events in late stamen development (Ito *et al.*, 2007). However, this flower-specific action of DAD1 raised doubts regarding the active roles of PLA_1s in wound-induced JA formation in leaves. DONGLE (DGL), a PLA_1 from *A. thaliana*, was thought to be involved in wound-induced and basal JA biosynthesis (Yang *et al.*, 2007; Hyun *et al.*, 2008, respectively). But there were still doubts due to highly ambiguous leaf-specific data on DAD1 and DGL lines generated in different laboratories. More recently, *DAD1* and *DGL* RNA*i* lines were generated, and these lines were similar to the wild-type in the early wound response. The DGL protein was detected in lipid bodies but not in plastids as required for JA biosynthesis (Ellinger *et al.*, 2010), suggesting that both enzymes are not involved in JA biosynthesis. Of an additional 16 lipase mutants screened, only *PLA1y1* (At1g06800) had a reduced level of JA in wounded leaves. However, there might still be unidentified lipases involved in wound- and pathogen-induced JA formation (Ellinger *et al.*, 2010). These Arabidopsis data were complemented by data from RNA*i* lines suppressing the expression of the *GALACTOLIPASE* A_1 (*GLA1*) of *Nicotiana attenuata*, which indicated its involvement in JA formation in leaves and roots, but not during *Phytophthora parasitica* infection (Bonaventure *et al.*, 2011*a*). It is thus obvious that there are pathway- and stimuli-specific lipases acting in oxylipin formation.

2.2. The LIPOXYGENASE (LOX) gene family members are involved in JA-dependent responses

Oxygenation of α-LeA is the initial step in JA biosynthesis. The oxygen has to be inserted in the C-13 position by a lipoxygenase (LOX) (Fig. 1). Among the six LOXs of Arabidopsis, four of them are 13-LOXs (LOX2, LOX3, LOX4, LOX6) (Bannenberg *et al.*, 2009), although their functions are only partly understood. LOX2 was thought to be involved in the wound response for a long time (Bell *et al.*, 1995) and subsequent studies revealed that LOX2 was responsible for the bulk of JA formation in the first h upon wounding (Glauser *et al.*, 2009; Schommer *et al.*, 2008). Similarly, an involvement of LOX2 in the generation of oxylipins during natural and dark-induced senescence as well as under sorbitol stress was demonstrated by using *LOX2-RNA*i lines. The *LOX2-RNA*i lines carry basal levels of *cis*-12-oxo-phytodienoic acid (OPDA) and JA, but do not show an enhanced accumulation during natural and dark-induced senescence (Seltmann *et al.*, 2010). Therefore, the regulation of *LOX2* may be under a COl1-dependent transcriptional control, but the gain-of-function mutant *fou2* indicated also a Ca^{2+}-dependent control of LOX2 protein leading to constitutively elevated JA levels (Bonaventure *et al.*, 2007*a*). The *fou2* mutant was initially identified in a screen on elevated fatty acid oxidation and thought to be affected in a vacuolar Ca^{2+} channel (Bonaventure *et al.*, 2007*a*, *b*). However, later FOU2/TPC1 was identified as a Ca^{2+}- and voltage-dependent vacuolar cation channel (Beyhl *et al.*, 2009). Moreover, *FOU2/TPC1* itself is a target of the large family of TCP (TEOSINTE BRANCHED/TEOSINTE BRANCHED1/CYCLOIDEA/PROLIFERATING CELL FACTOR1) TFs, which are involved in growth-related processes, such as leaf growth, shoot branching and floral organ morphogenesis (Danisman *et al.*, 2012). Interestingly, several

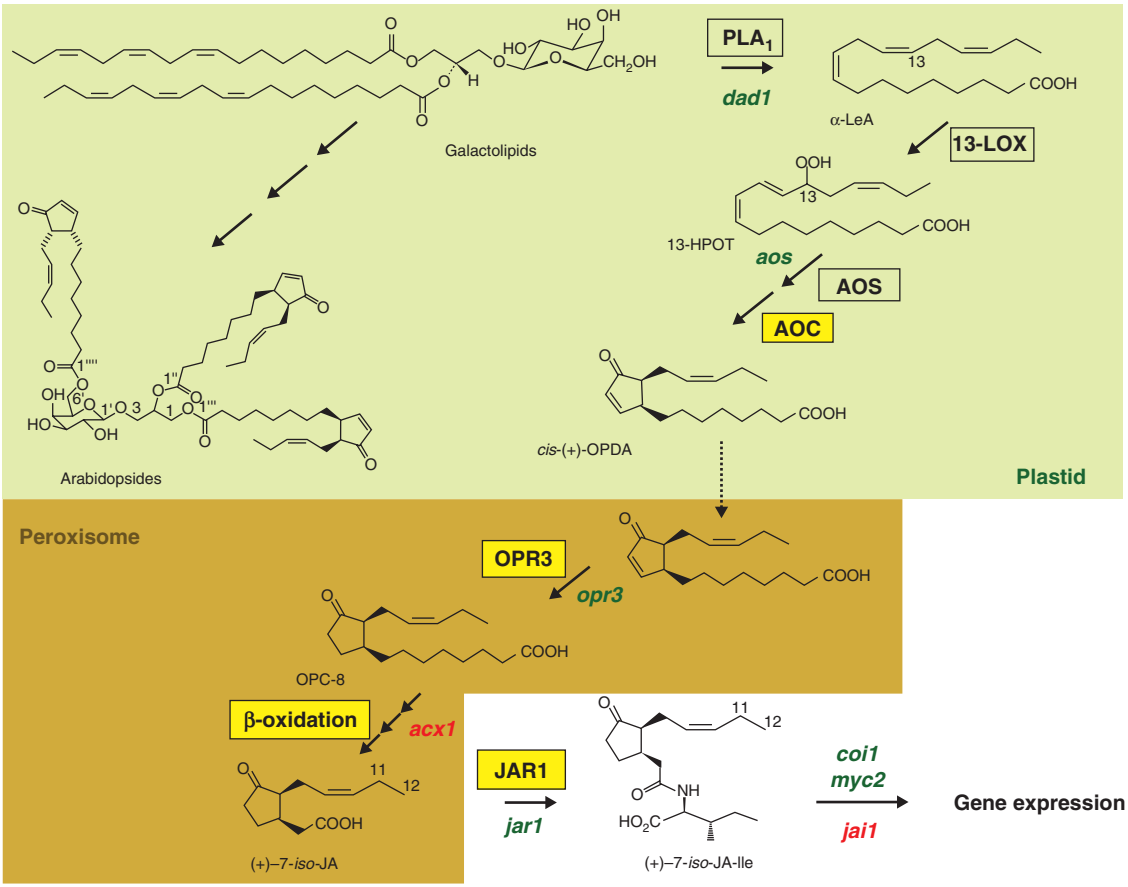

FIG. 1. Synthesis of jasmonic acid (JA)/JA-Ile from α-linolenic acid generated from galactolipids. Enzymes which have been crystallized are given in yellow boxes. Steps impaired in mutants of Arabidopsis (green) or tomato (red) are indicated. *acx1*, *acyl-CoA-oxidase1*; AOC, allene oxide cyclase; AOS, allene oxide synthase; *coi1*, *coronatine insensitive1*; *dad1*, *delayed anther dehiscence1*; 13-HPOT, (13*S*)-hydroperoxyoctadecatrienoic acid; *jai1*, *jasmonic acid insensitive1*; JAR1, JA-amino acid synthetase; α-LeA, α-linolenic acid; 13-LOX, 13-lipoxygenase; *myc2*, *bHLHzip transcription factor MYC2*; OPR3, OPDA reductase3; OPC-8, 3-oxo-2-(2-pentenyl)-cyclopentane-1-octanoic acid; *cis*-(+)-OPDA; *cis*-(+)-12-oxophytodienoic acid; PLA_1, phospholipase A_1.

TPCs are targets of miR319. Among them TPC4 is preferentially involved in the control of JA biosynthesis and leaf senescence (Schommer *et al.*, 2008). This control takes place via LOX2, and clearly indicates a developmental regulation of *LOX2* expression, which is partially uncoupled from its transcriptional regulation during wounding (Schommer *et al.*, 2008). Meanwhile, *LOX2* was identified as a target of additional TCPs such as TCP20, thereby regulating leaf development and senescence (Danisman *et al.*, 2012). Another level of LOX2-mediated control may occur in translation. The availability of eukaryotic initiation factor 4E (elf4E) is modulated by elf4E-binding proteins. AtLOX2 was identified as an elf4E-binding protein, suggesting a translational control via LOX2 activity (Freire *et al.*, 2000). LOX2 is also involved in lipid peroxidation that occurs under abiotic and biotic stresses. Here, a LOX2-mediated double oxygenation of plastid galactolipids leading to arabidopsides was recorded upon pathogen infection, but was not responsible for the pathogen-induced increase in JA (Zoeller *et al.*, 2012). Interestingly, the formation of lipid peroxides was accompanied by the synthesis of azelaic acid, a new signalling compound that has been shown to prime the immune response (Jung *et al.*, 2009; Dempsey and Klessig, 2012; Zoeller *et al.*, 2012) (see section 7.2).

In the *lox2-1* mutant, however, JA and JA-Ile are still synthesized in the first 5 min upon wounding (Glauser *et al.*, 2009) indicating the activity of other 13-LOXs. Moreover, a detailed proteome analysis of JA-induced proteins in *A. thaliana* showed a marked increase in LOX3 protein (Gfeller *et al.*, 2011). These data and recent work revealed that all four 13-LOX forms contribute to JA formation at least in the wound response (Caldelari *et al.*, 2011; Chauvin *et al.*, 2013). Among them LOX-6 showed a preferential role in the wound response in the early stage of leaf cell differentiation. Using single and different combinations of double, triple and quadruple mutants of *lox2-1*, *lox3B*, *lox4A* and *lox6A* as well as *LOX6*-promoter GUS lines, the dominant role of LOX6 in early wound-induced JA formation was confirmed. The *LOX6* promoter was specifically active in and near the xylem cells of young tissues which complement promoter activity of *LOX3* and *LOX4* in vascular tissues (Vellosillo *et al.*, 2007), where other JA biosynthesis genes such as *AOS* and *AOC* are also expressed (Kubigsteltig *et al.*, 1999; Stenzel *et al.*, 2012).

In contrast to the wound response, the role of 13-LOX forms in fertility and flower development is different. Both processes are clearly JA-dependent, but fertility does not require LOX2.

In contrast, the double mutant *lox3lox4* is male sterile, accompanied by abnormal anther maturation, defective dehiscence and non-viable pollen. Additionally, the mutant has a global proliferative arrest as evident by an abnormal carpelloid flower (Caldelari *et al.*, 2011). The remaining LOXs of *A. thaliana*, LOX1 and LOX5, are 9-LOXs and are not involved in JA biosynthesis. Their products are active in local and systemic defence mechanisms against bacterial pathogens (Vicente *et al.*, 2012). LOXs of fungi are different from plant and mammalian LOXs, and generate 9- and 13-hydroperoxides (reviewed by Brodhun and Feussner, 2011).

2.3. ALLENE OXIDE CYCLASE (AOC)

Recently, differential expressions of the four *AOC*s of *A. thaliana* were demonstrated by corresponding promoter::GUS lines (Stenzel *et al.*, 2012). In leaves, *AOC1*, *AOC2* and *AOC3* were expressed in all leaf tissues, whereas the *AOC4* promoter was preferentially active in the main veins of fully developed leaves. In roots, promoters of all AOCs were highly active in the meristematic tissues and the elongation zone, including the lateral root primordia. Results obtained in distinct flower organs indicated redundant and non-redundant expression of *AOC*s. An additional level of regulation of AOCs was indicated by interaction studies using BiFC, where homo- and heterodimerization of all the four AOCs were detected (Stenzel *et al.*, 2012). In soybean, where six genes encode AOCs, initial data showed functional diversity in terms of expression for stress responses (Wu *et al.*, 2011). Recently, the crystal structure of AOC1 and AOC2 of *Physcomitrella patens* revealed new mechanistic insights into AOC catalysis, including tight binding of the substrate, accompanied by conformational changes within the binding pocket (Neumann *et al.*, 2012). Both PpAOCs are similar in structure and oligomeric to the AtAOC2 crystalized previously as a trimer (Hofmann *et al.*, 2006).

AOC and other enzymes in JA biosynthesis such as LOX and ALLENE OXIDE SYNHASE (AOS) are partially associated with chloroplast membranes (Farmaki *et al.*, 2007). For AOS the level of the protein within the envelope is affected by rhomboids, a family of intra-membrane serine proteases of inner envelope membrane (Knopf *et al.*, 2012). The association of LOX, AOS and AOC with chloroplast membranes implies that the esterified OPDAs, called arabidopsides, may be formed from fatty acids esterified in galactolipids by membrane-bound enzymes, as indicated by recent labelling experiments (Nilsson *et al.*, 2012). Arabidopsides that occur exclusively in Arabidopsis are a diverse group of compounds of the galactolipids MGDG and DGDG, where OPDA is esterified in the *sn-1* and/or *sn-2* position. The occurrence of the different types of arabidopsides, their formation and putative function has been reviewed (Göbel and Feussner, 2009; Mosblech *et al.*, 2009).

2.4. OPDA REDUCTASE3 (OPR3)

Among the six OPRs of *A. thaliana* only OPR3 is involved in JA biosynthesis, which was substantiated by mechanistic studies with the crystal structure of OPR3 and OPR1 (Breithaupt *et al.*, 2001, 2006) (reviewed by Schaller and Stintzi, 2009; Wasternack and Kombrink, 2010; Kombrink, 2012). OPR1 might be involved in reduction of phytoprostanes, a group of non-enzymatically formed compounds with structural similarity to OPDA (Mueller *et al.*, 2008).

Initially, *opr3*, a JA-deficient and OPDA-accumulating mutant carrying a 17-kb T-DNA insertion in an *OPR3* intron, showed resistance to *Alternaria brassicicola*, which was discussed as a direct role of OPDA in pathogen defence (Stintzi *et al.*, 2001). In many studies, *opr3* was permanently used to distinguish between JA- and OPDA-dependent signalling. Recently, JA accumulation in *opr3* upon infection with *Botrytis cinerea* has also been reported (Chehab *et al.*, 2011). *opr3* is not a null mutant, and is able to generate mature full-length *OPR3* transcript upon splicing of the T-DNA containing intron under specific conditions, such as *B. cinerea* infection leading to JA formation. Therefore, at least under some conditions, *opr3* is not an ideal platform for dissecting OPDA-specific signalling.

The important and versatile role of OPRs was recently illustrated for maize. The *opr7opr8* double mutant has dramatically reduced levels of JA in all organs tested, accompanied by strong defects in development, including sex determination leading to feminized tassels and the elongation of ear shanks (Yan *et al.*, 2012) (see section 9.12). This double mutant was highly susceptible to root-rotting oomycetes (*Pythium* spp.) and herbivory. In rice, an OPR involved in JA biosynthesis is encoded by *OsOPR7*, whereas the other 12 members of this gene family belong to another subgroup, which is not involved in JA formation (Tani *et al.*, 2008). *OsOPR7* is expressed upon wounding or drought stress, and can complement the *opr3* phenotype in *A. thaliana* (Tani *et al.*, 2008). The OsOPR7 protein can convert both enantiomeric forms of *cis*-OPDA, (+)-*cis*-OPDA and (−)-*cis*-OPDA.

2.5. Regulation of JA biosynthesis

As described previously (Wasternack, 2007; Browse, 2009*a*, *c*), the regulation of JA biosynthesis is determined by a positive feedback loop, substrate availability and tissue specificity. Additional regulation is provided by the concurrent action of the branches in the LOX pathway. Among the seven different branches known for the LOX pathway (Feussner and Wasternack, 2002) the AOS and HYDROPEROXIDE LYASE (HPL) reactions are concurrent on the same substrate, the product of a 13-LOX. The HPL branch leads to volatile and non-volatile oxylipins, e.g. the leaf aldehydes and leaf alcohols (Andreou *et al.*, 2009). Many of them are defence compounds and are formed upon herbivore attack (Matsui *et al.*, 2006; Schuman *et al.*, 2012). The HPL branch involved in the formation of green leafy volatiles (GLVs) is selectively suppressed by chewing herbivores, which might be an evolutionary advantage (Savchenko *et al.*, 2013). One of the three HPLs of rice positively regulates the formation of GLVs but negatively regulates JA biosynthesis by substrate competition (Tong *et al.*, 2012). Consequently, the direct and indirect defence is modulated. Non-volatile oxylipins, such as various traumatic acids and azelaic acid, are formed upon stress in pea seedlings (Mukhtarova *et al.*, 2011), suggesting, for the first time, a central role of azelaic acid as a defence signal (Jung *et al.*, 2009; Dempsey and Klessig, 2012; Zoeller *et al.*, 2012) (see section 7.2.).

Additional components of regulation were obtained from characterization of JAZ proteins, Ca^{2+}-related signalling, JA-related transcription factors and mitogen-activated protein kinases (MAPKs). The positive feedback loop in JA biosynthesis can be explained now by the SCF^{COI1}–JAZ regulatory module that is known to be active in the expression of *LOX*, *AOS*, *AOC*, *OPR3* and *ACX*. The formation of JA/JA-Ile will subject the negative regulator JAZ to proteasomal degradation, which allows MYC2 to activate the JA-responsive promoters of JA biosynthesis genes. JAZ and MYC genes are, however, JA/JA-Ile responsive, allowing a permanent replenishment of the negative (JAZs) and positive (MYC2) regulators that result in an adjustment of the expression of JA biosynthesis genes (Chung *et al.*, 2008). The Arabidopsis microarray datasets from various developmental stages and stress conditions reveal transcriptional regulation of all JA biosynthesis genes (Pauwels *et al.*, 2009; van Verk *et al.*, 2011). There are, however, indications for post-translational regulation of enzyme activities. The OPR3 activity seems to result from a monomer/dimer equilibrium including a self-inhibition by dimerization (Breithaupt *et al.*, 2006; Schaller and Stintzi, 2009). The above interaction studies with all four AOCs of *A. thaliana* revealed interaction among them all. The observed homo- and hetero-dimerization led at least partially to altered enzyme activity (Stenzel *et al.*, 2012).

Ca^{2+} and MAPK cascades are also involved in the regulation of JA biosynthesis. In *A. thaliana*, MKK3 and MPK6 are activated by JA leading to negative regulation of *MYC2* expression and repression of JA biosynthesis genes (Takahashi *et al.*, 2007). In a parallel pathway, however, there is an MKK3/MPK6-independent activation of MYC2 by JA, and the MKK3/MPK6 cascade is epistatic to MYC2 (Takahashi *et al.*, 2007). There exists a link between JA biosynthesis and MAPK pathways, as revealed by co-expression analyses of microarray datasets in *A. thaliana* (van Verk *et al.*, 2011). Here, it became obvious that *OPR3* and to a minor extent *AOS* are co-expressed with *MYC2*, *MEK1*, *MEKK1*, *MKK4* and *MPK3*.

For wound- and herbivore-induced JA accumulation in *Nicotiana attenuata*, the Ca^{2+}-dependent protein kinases CDPK4 and CDPK5 are negative regulators (Yang D-H *et al.*, 2012), whereas a wound-induced protein kinase (WIPK) is rapidly activated near the wound region thereby activating JA biosynthesis (Wu *et al.*, 2007). In tomato, a MPK1, MPK2 and MPK3 are involved in expression of JA biosynthesis genes (Kandoth *et al.*, 2007). Here, the activation of MPKs is systemin-dependent (see section 6). Further control of JA biosynthesis is mediated by the COP9 (CONSTITUTIVE PHOTOMORPHOGENESIS 9) signalosome (CSN), a multiprotein complex involved in the regulation of CULLIN-RING E3 ubiquitin ligases. CSN not only is required for optimum plant development, but is also involved in plant defence against herbivores and pathogens by its modulation of JA levels (Hind *et al.*, 2011).

Ca^{2+} is an early acting second messenger in response to many biotic and abiotic stimuli (Kudla *et al.*, 2010). Although several of these stimuli are associated with increased JA biosynthesis, the involvement of Ca^{2+} upstream of JA biosynthesis is poorly understood. Besides Ca^{2+}-mediated control of LOX2 (Bonaventure *et al.*, 2007*a*) as discussed earlier (see section 2.2), three additional examples will be given here to elucidate, how Ca^{2+} is involved in the regulation of JA biosynthesis and signalling:

(1) In the family of Ca^{2+}/CaM-binding TFs, AtSR1 is required for down-regulation of salicylic acid (SA) levels in plant immune responses (Du *et al.*, 2009). Upon wounding, however, the negative impact of SA in both basal and induced JA biosynthesis is abolished by AtSR1 (Qiu *et al.*, 2012).
(2) The calmodulin-like protein CLM42 negatively regulates the defence response during herbivory by decreasing the COI1-mediated JA sensitivity (Vadassery *et al.*, 2012). The cytosolic and nuclear located protein CLM42 is active downstream of herbivore-induced Ca^{2+} elevation but is upstream of COI1-mediated JA-Ile perception.
(3) In *A. thaliana*, the overexpression of a plasma membrane-located glutamate receptor results in increased glutamate-mediated Ca^{2+} influx and resistance to necrotrophic pathogens (Kang *et al.*, 2006). A putative link to JA biosynthesis is lacking, but is suggested by the up-regulation of *VSP1*, *LOX2* and other JA-responsive genes.

Ca^{2+} is clearly a key player in plant responses to environmental stimuli, leading to context-dependent Ca^{2+} fluctuations upstream and downstream of JA biosynthesis or in parallel to JA generation, and is a part of the regulatory network of evolutionary divergent metabolic pathways (Pauwels *et al.*, 2009).

2.6. *JAR1 catalysing the final step in the generation of ligand*

The cloning of JAR1 as a member of the *GH3* gene family, which belongs to the large group of enzymes forming acyl-adenylate/thioester intermediates, was a breakthrough in the JA field. This enzyme catalyses the final step in the formation of the bioactive JA compound (Staswick and Tiryaki, 2004). The identification of (+)-*7-iso*-JA-Ile as the ligand of the COI1–JAZ co-receptor complex (Fonseca *et al.*, 2009) and the crystallization of the receptor complex (Sheard *et al.*, 2010) provided mechanistic explanations for JA/JA-Ile perception (see section 4). Meanwhile, the JA-specific JAR1/AtGH3.11 and the benzoate-specific PBS3/AtGH3.12 have been crystallized (Westfall *et al.*, 2012). For crystallization of JAR1, a racemic mixture of JA was used, but only (−)-JA-Ile was found in the structure. The authors assumed that (−)-JA is accepted as substrate by JAR1 and is converted to (+)-JA-Ile (Westfall *et al.*, 2012). However, the initial *in vivo* product in JA biosynthesis is (+)-*7-iso*-JA, and its conjugate with L-Ile is the ligand of the receptor (Fonseca *et al.*, 2009; Sheard *et al.*, 2010). Although the JA epimer used by JAR1 still continues to be ambiguous, the water-mediated hydrogen bond to the cyclopentanone ring of JA and the hydrophobic binding pocket for the pentenyl side observed in the crystal structure of JAR1 (Westfall *et al.*, 2012) explain now mechanistically the repeatedly recorded structure/activity relationships for numerous JA compounds (for a review see Wasternack, 2007). The crystal structures of PBS3 and JAR1 define the role of conformational changes in the carboxy-terminal domain for conjugation of amino acids to various acyl acid substrates and illustrates how a promiscuous enzyme might evolve by a highly adaptable

structure (Westfall *et al.*, 2012). For a long time, equilibration between the enantiomers of JA and of JA-Ile was assumed (Wasternack, 2007), and epimerization was suggested as a mechanism to sustain the most bioactive JA compound, (+)-*7-iso*-JA-Ile (Fonseca *et al.*, 2009). Meanwhile, an assay has been developed for quantification of (+)-*7-iso*-JA-Ile from tomato extracts, indicating that the compound is less unstable than assumed earlier (Suza *et al.*, 2010). These data indicate that (+)-*7-iso*-JA-lle is exclusively formed upon wounding and by a recombinant JAR1, with a strong preference for Ile compared with other amino acids. In *SlJAR1-RNA*i lines, wound-induced formation of (+)-*7-iso*-JA-Ile was downregulated by 50–75 % suggesting the existence of other JA-conjugating enzymes than JAR1 (Suza *et al.*, 2010). Note that this must be taken into consideration while evaluating the *jar1* mutant data. The homeostasis of JA-Ile is highly dependent on its hydrolysis *in vitro*. In *JA-Ile-hydrolase 1*-silenced plants of *N. attenuata*, the herbivore-induced burst in JA-Ile and its following reactions in direct and indirect defence responses are strongly attenuated (Woldemariam *et al.*, 2012).

3. THE METABOLIC FATE OF JA

In an earlier update, only four enzymes involved in JA metabolism were described in terms of enzymatic properties and cloning of their cDNAs (Wasternack, 2007). Meanwhile, new JA metabolites have been identified, with additional enzymes having been cloned and characterized.

Due to the central role of JA-Ile in JA signalling and the parallel occurrence of JA and JA-lle as sustained by JAR1, we will combine them in the subsequent sections as 'JA/JA-Ile', this being active as a signalling module. However, there are three important caveats: (1) Are active JA metabolites involved in specific responses that are not directly caused by JA/JA-lle? (2) Is JA/JA-Ile signalling switched off by metabolic conversion? (3) Do JA metabolites function as a storage form of JA?

3.1. Profiles of JA/JA-Ile metabolites

In the early days of JA research, numerous JA compounds were identified as constituents of distinct plant tissues or as volatiles emitted from flowers (reviewed by Wasternack *et al.*, 2013). The profiles of JA-related compounds are presented in Fig. 2. Many of them were already known in 2007. Meanwhile, glucosylated forms of JA, JA-Ile, 12-OH-JA and 12-OH-JA-Ile have been described (Chung *et al.*, 2008; Glauser *et al.*, 2008, 2009). Most of them accumulate very rapidly (within minutes) in wounded Arabidopsis or tomato leaves. Corresponding wound-induced formation of 11-OH-JA, 12-OH-JA, 12-OH-JA-Ile, 12-COOH-JA-Ile and 12-HSO_4-JA were also recorded (Gidda *et al.*, 2003; Guranowski *et al.*, 2007; Glauser *et al.*, 2008, 2009; Miersch *et al.*, 2008). A large-scale screening for different JA/JA-Ile metabolites in different organs of various plant species showed their relative abundance up to three orders of magnitude higher than that of JA or OPDA (Miersch *et al.*, 2008). Immature seeds and leaves of *Glycine max* contain high levels of 12-OH-JA, 12-HSO_4-JA and 12-*O*-Glc-JA. In most cases, however, it is not known whether these abundantly occurring JA/JA-Ile metabolites are biologically active or function as storage forms of JA/JA-Ile (Miersch *et al.*, 2008). It has been suggested that higher levels of 12-OH-JA, 12-HSO_4-JA and 12-*O*-Glc-JA in the tassels of *Zea mays* may be associated with sex determination during development of this male reproductive structure in monoecious species (Acosta *et al.*, 2009; Browse, 2009*b*). Support for the involvement of a JA compound in sex determination also came from the maize double mutant *opr7opr8* (Yan *et al.*, 2012) (see section 9.12).

3.2. cis*-Jasmone (CJ)*

CJ is a volatile compound and represents the main constituent of the floral bouquet of different plants thereby attracting insect pollinators. It is emitted in response to herbivory, application of insect oral secretions or JA treatment. However, the biosynthetic route leading to the formation of CJ is still unclear. Initially, CJ was regarded as a decarboxylated product of JA, being responsible for the disposal of JA due to its high volatility (Koch *et al.*, 1997). Isomerization of *cis*-(+)-OPDA into *iso*-OPDA, however, allows a direct route to CJ via β-oxidation to 3,7-didehydro-JA and decarboxylation (Dabrowska and Boland, 2007; Schulze *et al.*, 2007; Dabrowska *et al.*, 2009). CJ is clearly biologically active, preferentially in plant–insect interactions as summarized by Matthes *et al.* (2010). Most evidence derives from the microarray-based transcriptome analysis of CJ-treated Arabidopsis plants (Matthes *et al.*, 2010). The set of CJ-induced genes was different from those induced by JA, and CJ-induced gene expression was independent of that induced by COI1 and JAR1. Furthermore, key components that are not involved in JA signalling are assumed to have distinct roles in CJ signalling; for example, TFs TGA 2, 5 and 6, and SCARECROW-like 14 have been shown to play a key role for CJ in indirect defence (Matthes *et al.*, 2010).

3.3. CYP94 enzymes generate hydroxylated and carboxylated JA-Ile

Most recently, three groups independently identified the cytochrome P450 enzyme CYP94B3 that hydroxylates JA-Ile at the terminal carbon atom of the pentenyl side chain (Kitaoka *et al.*, 2011; Koo *et al.*, 2011; Heitz *et al.*, 2012). Additionally, Heitz *et al.* (2012) characterized the enzyme CYP94C1, which is active in the subsequent oxidation step to the oxidized 12-OH-JA-Ile (Fig. 2). Heterologous expression in yeast showed substrate preference of CYP94B3 for JA-Ile (Kitaoka *et al.*, 2011; Koo *et al.*, 2011; Heitz *et al.*, 2012). Typical JA-lle-deficient phenotypes as observed in the CYP94B3 overexpressors that show higher susceptibility to insect attack provided further evidence for the involvement of both enzymes (Koo *et al.*, 2011). Accordingly, the wounded *cyp94b3* mutant exhibited increased accumulation of JA-Ile (Koo *et al.*, 2011; Koo and Howe, 2012). These data together with the fact that hydroxylated JA-Ile was less effective in the COI1–JAZ interaction assay (Koo *et al.*, 2011) support the assumption that hydroxylation and carboxylation of JA-Ile may switch off JA/JA-Ile signalling. Such a role of hydroxylation is also known for other hormones, and was initially shown for hydroxylation of JA to 12-OH-JA (Gidda *et al.*, 2003; Miersch *et al.*, 2008). Here, typical JA responses, such as expression of JA-inducible genes, root growth inhibition or seed germination

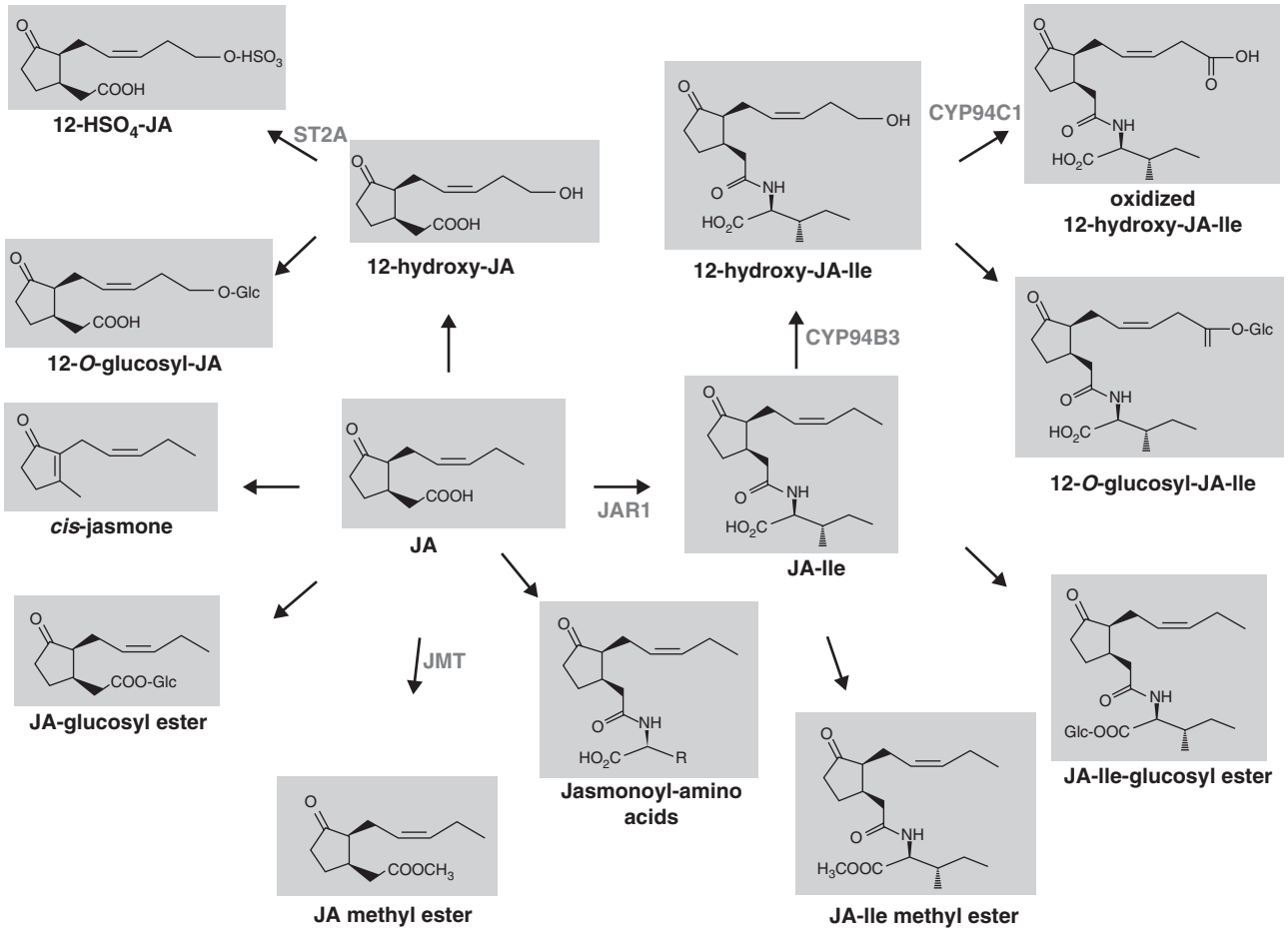

FIG. 2. Metabolic fate of jasmonic acid (JA) and JA-Ile. Enzymes which have been cloned are given in grey. JAR1, JA-amino acid synthetase; JMT, JA methyl transferase; ST2A, sulfotransferase 2A.

inhibition were compromised by treatment with 12-OH-JA. It will be interesting to examine the role of other members of the CYP94 gene family, e.g. a putative JA hydroxylase. Six members are known for the CYP94 gene family, which seems to have evolved rapidly and active in conversion of fatty acid-derived compounds (Nelson and Werck-Reichhart, 2011; Koo and Howe, 2012). The broad specificity of CYP94s for fatty acyl substrates *in vitro* (Kandel *et al.*, 2007; Pinot and Beisson, 2011) and the activity of other hydroxylases, such as CYP709C1 active for long-chain fatty acids, illustrate the diversity in hydroxylated fatty acid-derived compounds.

3.4. Methyl jasmonate (MeJA)

Prior to 2007 when there was not much information about JAZ proteins, any discussion on bioactivity of JA and MeJA was controversial. JA levels were always recorded and correlated to JA responses. Furthermore, transgenic lines of Arabidopsis over-expressing the JA carboxy methyl transferase (JMT) led to the assumption that MeJA is the preferentially active signal in JA responses (Seo *et al.*, 2001). An ectopic expression of JMT in *N. attenuata*, however, negatively affected the formation of JA-Ile, and the biological activity of MeJA was only apparent when MeJA was converted to JA followed by its conjugation to JA-Ile (Stitz *et al.*, 2011). The specificity of JA-Ile in the COI1–JAZ interaction (see section 4.3) was the final proof that there is no direct bioactivity of JA and MeJA.

3.5. Sulfated jasmonates

Among the 18 sulfotransferases of *A. thaliana*, the gene *AtST2a* has been cloned, and its recombinant protein has been shown to be specific for the conversion of 11-OH-JA and 12-OH-JA to the corresponding sulfated derivatives (Gidda *et al.*, 2003). Besides OPDA, JA and JA-lle, 12-OH-JA also mediates the expression of *AtST2a*. Subsequent cloning of the homologous gene from tomato showed similar properties, and transgenic lines over-expressing or repressing *SlST2a* showed a dramatic shift among the three involved compounds 12-OH-JA, 12-HSO_4-JA and 12-*O*-Glc-JA (J. Heise and C. Wasternack, unpubl. res.). Interestingly, in the adenosine 5′-phosphosulfate kinase gene family consisting of four members and being involved in generation of active sulfate for the sulfotransferase reaction, the *apk1apk2* double mutant exhibits a five-fold decrease in 12-OH-JA and 12-HSO_4-JA accompanied with a concomitant increase in 12-*O*-Glc-JA (Mugford *et al.*, 2009). This indicates that conversions of 12-OH-JA into

either 12-*O*-Glc-JA or 12-HSO_4-JA are concurrent reactions (see section 3.6).

Further cross-talk between 12-OH-JA and sulfate metabolism was demonstrated by using the mutant *fou8*, which was identified in a screen for mutants with altered fatty acid oxidation. In *fou8* plants, an increment in the LOX2 level is attributed to increased fatty acid oxidation. Consequently, the JA pathway is permanently activated, as indicated by the appearance of JA-related phenotypes in *fou8* plants (Rodriguez *et al.*, 2010*b*). In *fou8* plants, the conversion of 3′phosphoadenosine-5′-phosphate (PAP) to AMP, the byproduct of the sulfotransferase reaction, is also affected (Lee *et al.*, 2012), and as a result sulfur metabolism including sulfation of glucosinolates and 12-OH-JA is dramatically altered (Lee *et al.*, 2012). However, the most convincing evidence for the cross-talk between sulfur metabolism and JA biosynthesis is the fact that in the triple mutant *fou8apk1apk2*, the *fou8* phenotypes are genetically suppressed, indicating that a component of the sulfur futile cycle affects the LOX activity necessary for JA biosynthesis (Rodriguez *et al.*, 2010*b*).

3.6. Glucosylated jasmonates

The plethora of jasmonate compounds is enormous. Besides the compounds mentioned above, JA also occurs conjugated to 1-aminocyclopropane-1-carboxylic acid (ACC), the ET precursor (Staswick and Tiryaki, 2004). However, there is no information on its biological activity. Another group of jasmonate compounds are the glucosylated derivatives. They may occur as glucosyl esters, which are presumably inactive compounds, as the conjugation with amino acids by JAR1 required for most JA-like activities cannot take place. Initially, 12-OH-JA as tuberonic acid (TA) and its *O*-glucoside (TAG) were identified in potato leaflets and shown to have tuber-inducing properties (Yoshihara and Greulich, 1998) (see section 9.5).

The *O*-glucosylated jasmonates modified at C-11 and C-12 of hydroxylated JA accumulate rapidly upon leaf wounding (see above) (Glauser *et al.*, 2008; Miersch *et al.*, 2008). Jasmonates with other sugar moieties such as gentiobiose were also detected during the cell cycle of tobacco BY2 cells (Swiatek *et al.*, 2004). In unwounded leaves of *Glycine max*, the accumulation of 12-*O*-Glc-JA has been shown to be up to three orders of magnitude higher than that of JA (Miersch *et al.*, 2008). In wounded tomato leaves, 12-*O*-Glc-JA accumulates subsequently to JA and 12-OH-JA (Miersch *et al.*, 2008; O. Miersch, unpubl. res.). In transgenic tomato lines constitutively over-expressing the gene *ST2a*, accumulation of 12-*O*-Glc-JA upon wounding has been shown to be much less due to its shift to the sulfated derivative (J. Heise *et al.*, unpubl. res.). However, the biological role of 12-*O*-Glc-JA in the wound response is not clear. Possibly, 12-*O*-Glc-JA is a transport form of 12-OH-JA, or it represents a sequestration of JA as known for the glucosides of SA and benzoic acid.

12-*O*-Glc-JA was identified as a leaf closing factor (LCF) in motor cells of nyctinastic plants, such as *Albizzia* and *Samanea saman* (Nakamura *et al.*, 2011) (see section 9.8). As with the JA-Ile receptor, only a specific enantiomer, here the (−) form, of LCF is active. In addition to the enantiomer specificity of the jasmonoyl moiety, the D/L-stereochemistry of the glucon moiety is important (Ueda *et al.*, 2012). This accords with the weak activity of 12-OH-JA and inactivity of JA and JA-Ile in leaf closing. The LCF was inactive in all classical JA responses such as *LOX2* expression or leaf volatile emission, and is perceived in a COI1/JAZ-independent manner (Nakamura *et al.*, 2011). The involvement of JA-related compounds in nyctinastic leaf movement was confirmed by the gene expression data from a *Medicago truncatula* mutant with a defective pulvinus that is required for nyctinasty (Zhou *et al.*, 2012). This mutant, called *petiolule-like pulvinus*, showed down-regulation of genes involved in JA biosynthesis and metabolism.

From rice cell cultures, a putative SA glucosyl transferase (OsSGT) has been purified that shows glucosylation not only of SA but also of 12-OH-JA (Seto *et al.*, 2009). The *OsSGT* mRNA accumulating in cell cultures upon treatment with JA, 12-OH-JA and SA as well as in leaves after wounding is indicative of its putative role in the wound response.

4. JA PERCEPTION AND SIGNALLING

4.1. SCF complexes

The ubiquitin-proteasome system is the central regulator in plant hormone sensing and signalling. It consists of an Skp1/Cullin/F-box (SCF) complex that functions as an E3 ubiquitin ligase, where the F-box protein recognizes a target protein which is ubiquitinated and subsequently subjected to proteasomal degradation. For JA perception and signalling, COI1 acts as an F-box protein (Xie *et al.*, 1998). One of the most interesting aspects in plant hormone research is that several of them are perceived by an SCF complex with similar modules, where the F-box protein confers the hormone specificity. Since these facets have been extensively reviewed over the past couple of years (Katsir *et al.*, 2008*a*; Chini *et al.*, 2009*a*; Santner and Estelle, 2010; Kelley and Estelle, 2012; Shan *et al.*, 2012) only JA-related aspects will be discussed here.

4.2. JAZ proteins

In 2007, members of a new protein family of Arabidopsis were discovered by chance and called JASMONATE ZIM DOMAIN (JAZ) proteins (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007). Initially observed to be early up-regulated by wounding or JA treatment, JAZ proteins were recognized as targets of the SCF^{COI1} complex. The degradation of JAZ allows the release of positively acting TFs, such as MYC2 that binds to JA-responsive elements occurring in promoters of JA-responsive genes, thereby initiating transcription. This basic scheme (Fig. 3) has been independently developed in three different laboratories (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007) and was subsequently extended by identification of new up- and downstream components. Among the upstream components, the RING-type ubiquitin ligases, RING DOMAIN LIGASE3 (RGLG3) and RGLG4, were identified as modulators of JA/JA-Ile signalling in response to various stimuli (Zhang X *et al.*, 2012*b*). As downstream components the general co-repressors TOPLESS (TPL) and TPL-related proteins and their interaction with the adaptor protein ‘Novel Interactor of JAZ’ (NINJA) were identified (Pauwels *et al.*, 2010). Furthermore, while searching the

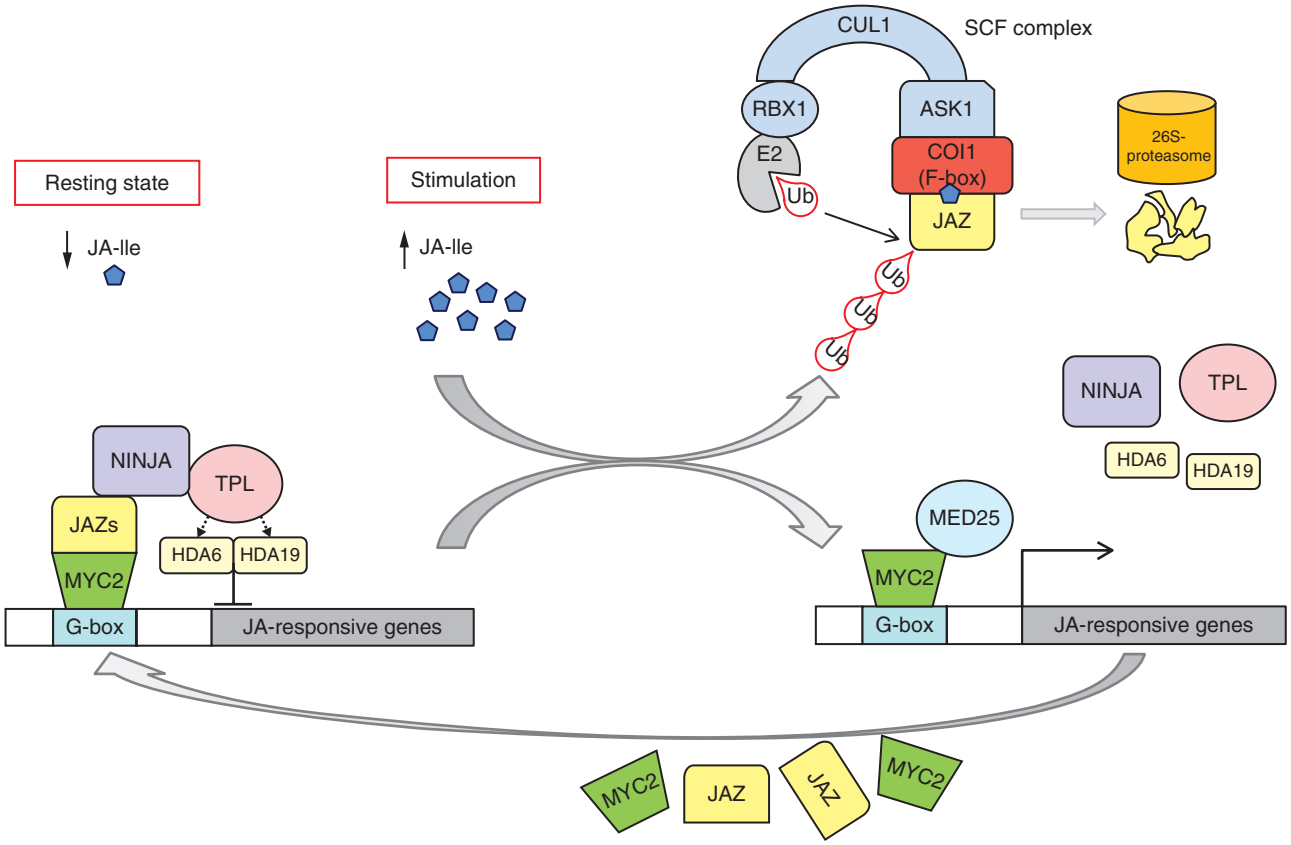

FIG. 3. Jasmonic acid (JA) perception via the COI1–JAZ co-receptor complex – mechanisms in JA-induced gene expression. In the resting state (left, low JA-Ile level), the binding of MYC2 to a G-box within the promoter of a JA-responsive gene does not activate transcription due to binding of the repressors Jasmonate ZIM domain proteins (JAZs) to MYC2. The co-repressors Novel Interactor of JAZ (NINJA) bound to JAZs, and TOPLESS (TPL) repress transcription via HISTONE DEACETYLASE 6 (HDA6) and HDA19. Upon stimulation (right, high JA-Ile level), JAZs are recruited by COI1 and subjected to ubiquitinylation and subsequent degradation by the 26S proteasome. Subsequently, MYC2 can activate transcription of early JA-responsive genes such as those encoding JAZ and MYC2. Transcription is mediated by the subunit 25 of Mediator complex (MED25; see section 4). ASK1, Arabidopsis SKP1 (S-phase kinase-associated protein 1) homologue; CUL, CULLIN; E2, ubiquitin-conjugating enzyme; MYC2, bHLHzip transcription factor; RBX, RING-H2 protein; SCF-complex, complex consisting of Skp1, Cullin-1 and F-box protein; Ub, ubiquitin.

JAZ targets numerous new TFs and JAZ interactors were discovered (Pauwels and Goossens, 2011; Wager and Browse, 2012).

In addition to the F-box protein COI1, JAZ interactors are: (1) bHLH TFs (MYC2, MYC3, MYC4, GL3, EGL3 and TT8), (2) R2R3 MYB TFs (PAP, GL1, MYB 21 and MYB 24), (3) TFs of other hormone signalling pathways (EIN3, EIL, GAI, RGA and RGL1), (4) co-repressor proteins (NINJA, TPL, HDA6 and HDA19) and (5) JAZ proteins due to their homo- and hetero-dimerizations (Chini *et al.*, 2009*b*; Pauwels and Goossens, 2011).

There are 12 JAZ proteins in *A. thaliana* (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007; Chung and Howe, 2009; Chung *et al.*, 2009; Pauwels and Goossens, 2011; Wager and Browse, 2012). They contain a weakly conserved N-terminal domain, a highly conserved C-terminal Jas domain that mediates the interaction with the transcription factors, and the conserved ZIM (TIFY) domain responsible for JAZ dimerization and interaction with NINJA (Vanholme *et al.*, 2007; Chung *et al.*, 2009; Pauwels and Goossens, 2011). The Jas domain is exclusively required for the repressive activity of JAZ proteins (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007). The expression of truncated JAZs lacking the Jas domain was associated with dominant insensitivity to exogenous JA. The initial assumption that individual JAZ proteins act specifically with different targets was subsequently revised by numerous interaction studies and the fact that there is a common occurrence of the ZIM and the Jas domain; for example, all 12 JAZs interact with MYC2, and JAZ1 interacts with nearly all target proteins mentioned above. The JA signalling is, however, mediated by a JAZ-regulatory network that entails interaction with multiple transcription factors, formation of homo- and hetero-dimers, alternative splicing of JAZ-encoding genes and differential stability of JAZs (Pauwels and Goossens, 2011; Kazan and Manners, 2012; Shyu *et al.*, 2012). All these processes may result in a large repertoire and combinatorial diversity in JAZ–JAZ interactions, the *in vivo* function of which is not known (Chung *et al.*, 2009, 2010).

The alternative splicing of JAZ genes can form dominant JAZ variants leading to JA-insensitive plants, if the Jas domain is abolished during splicing. The Jas domain is absolutely required for binding the downstream components, the TFs, and for intact JA signalling. For JAZ10, there are naturally occurring splice

variants lacking parts of the Jas domain (JAZ10.3) or the complete Jas domain (JAZ10.4) (Yan *et al.*, 2007; Chung *et al.*, 2009, 2010). The JAZ proteins are localized in the nucleus (Chini *et al.*, 2007; Thines *et al.*, 2007; Grunewald *et al.*, 2009), but an assumed involvement of the Jas domain is not completely clear because the splice variants JAZ10.3 and JAZ10.4 affected in the Jas domain are still localized in the nucleus (Chung *et al.*, 2009). Recently, the nuclear targeting of JAZ1 and JAZ9 has been shown to be dependent on physical interaction with MYC2 via a highly conserved region of the Jas domain (Withers *et al.*, 2012).

There were several hints at the transcriptional repression by JAZs, but generation of mutants with the expected JA-hypersensitive phenotype was upset by the obvious redundancy among the JAZ proteins. Only the T-DNA insertion mutant *jaz10-1* and RNA*i* lines of *JAZ1* and *JAZ10* exhibited enhanced JA sensitivity (Grunewald *et al.*, 2009), whereas other JAZ mutants and the T-DNA insertion mutant *jaz1-1* did not show such a phenotype (Demianski *et al.*, 2012). Most recently, however, transcriptional repression by full-length JAZ8 has been described (Shyu *et al.*, 2012), which is based on increased stability of JAZ8 due to lack of the conserved LPIARR motif. This hexapeptide within the Jas domain represents the conserved degron motif, and is required for closing off the binding pocket of JA-Ile within the receptor complex (Sheard *et al.*, 2010). Due to its absence in JAZ8, a strong interaction with COI1 in the presence of JA-Ile is excluded, leading to the increased stability of JAZ8. However, the consequences of JAZ8 removal from cells are unknown. The residual interaction between JAZ8 and COI1 occurs only at the higher JA-Ile concentration, whereas JAZ1–COI1 interaction takes place when the JA-Ile concentration is low. Such a scheme, in which the COI1–JAZ interaction is determined by the concentration of the ligand, is quite similar to the auxin TIR1-Aux/indole-3-acetic acid (IAA) receptor system (Katsir *et al.*, 2008*a*; Kelley and Estelle, 2012). Besides this new feature on JAZ function via protein stability, the JAZ8-mediated repressor function was shown to depend on an LxLxL-type EAR (ERF associated amphiphilic repression) motif at the N terminus (Shyu *et al.*, 2012). This motif of JAZ8 can directly bind the co-repressor TPL. In that event, however, the ZIM domain is not required, in contrast to other JAZs, which recruit TPL through the EAR-motif containing adaptor NINJA (see below).

The JAZ gene expression is JA responsive (Chung *et al.*, 2008). Consequently, there is a futile cycle which may contribute to a fine-tuning of JA signalling. MYC2 is involved in the expression of many JA-responsive genes (Dombrecht *et al.*, 2007). For *JAZ* gene expression, however, other components might be involved, as in *myc2* mutants most *JAZ* genes are expressed upon infection with *Pseudomonas syringae*, which is known to be a JA- mediated process (Demianski *et al.*, 2012). One candidate could be the MEDIATOR25 (MED25) subunit of the eukaryotic Mediator complex (Fig. 3). MED25 has been recently identified as an integrative hub in JA-mediated gene expression (Çevik *et al.*, 2012). In the *pft1*/*med25* mutant, pathogen-responsive *JAZ9* expression is diminished (Kidd *et al.*, 2009), while the JA-induced expression of *JAZ6* and *JAZ8* is significantly reduced in the *med25* mutant lines (Chen *et al.*, 2012).

4.3. COI1–JAZ co-receptor complex

Ten years after cloning of the F-box protein COI1 (Xie *et al.*, 1998), its function as a JA receptor was finally established. Initially, COI1 was assumed to function as a receptor due to its analogy to the auxin receptor TIR1 (Woodward and Bartel, 2005). Photoaffinity-based cross-linking of JA-Ile to COI1 substantiated this idea (Yan *et al.*, 2009). However, the requirement of the SCF^{COI1}–JAZ complex for JA perception is now generally accepted. Since the identification of JAZs in 2007 followed by the crystallization of the COI1–JAZ co-receptor complex (Sheard *et al.*, 2010), it is now possible to establish a mechanistic view on JA-Ile perception. In this complex, the Jas domain of JAZ proteins interacts with COI1, if the ligand JA-Ile is present. This interaction takes place via the N-terminal 20 amino acid residues of the Jas degron, and is strongly increased by IP_5 (Sheard *et al.*, 2010; Mosblech *et al.*, 2011). IP_5 is closely located in the binding pocket of JA-Ile and co-ordinates three arginine residues of COI1 and R206 of the Jas peptide (Sheard *et al.*, 2010). The IP_5-free receptor complex is inactive. Previous pull-down experiments revealed that (+)-*7-iso*-JA-Ile is the most bioactive ligand (Fonseca *et al.*, 2009). This is now substantiated by the crystal structure: most of the ligand is surrounded by COI1 residues, but the keto-group of JA in JA-Ile and the COOH-group of Ile can interact with the Jas domain (Sheard *et al.*, 2010). Initial binding assays with labelled JA-Ile and COI1 protein showed a strong (50-fold) increase in binding and in specificity, if JAZ1 or JAZ6 were used as co-receptor complex component (Katsir *et al.*, 2008*b*). Site-directed mutagenesis revealed essential amino acid residues for binding of the ligand in the binding pocket established by the COI1–JAZ interaction (Melotto *et al.*, 2008). Although the basic concept of JA-Ile perception is established, there are still several caveats as to the ubiquitination of the JAZs, the exact interaction maps of all the complex members at both low and high JA-lle concentrations and their half-lives.

Recent results show the possibility of the existence of new properties of COI1. Although there is no doubt about the role of COI1 as an F-box protein in JA-dependent signalling via the SCF^{COI1} complex, JA-independent signalling by COI1 appeared in analysing a new allele of COI1 involved in regulation of innate immune receptor (NB-LRRs) accumulation (He *et al.*, 2012).

4.4. JA signalling versus OPDA signalling

When the basic concept of JA/JA-Ile perception was established in 2007, a striking exception in binding assays with jasmonate compounds appeared – the JA precursor OPDA was not an active ligand in COI1–JAZ pull-down assays (Thines *et al.*, 2007), although OPDA-specific gene expression had already been reported (Taki *et al.*, 2005). Mechanistic proof came from the crystal structure of the COI1–JAZ co-receptor complex, where OPDA does not fit into the binding pocket for JA-Ile (Sheard *et al.*, 2010). Consequently, there is an increasing number of examples describing an JA/COI1-independent role of OPDA (Wasternack *et al.*, 2013):

(1) Tendril coiling is mainly promoted by OPDA but much less by JA, as previously shown (Stelmach *et al.*, 1998; Blechert *et al.*, 1999).
(2) A distinct set of genes is expressed by OPDA, but only a partial overlap appeared with the expression of JA-induced genes (Taki *et al.*, 2005; Mueller *et al.*, 2008).
(3) *Physcomitrella patens* is unable to form JA, but accumulates OPDA. The fertility of *AOC*-knockout lines is decreased, suggesting a requirement for OPDA (Stumpe *et al.*, 2010).
(4) A similar observation was made with developing tomato embryos (Goetz *et al.*, 2012). Here, a preferential and abundant accumulation of OPDA in the seed coat is required for proper embryo development, as shown with tomato mutants defective in OPDA or JA synthesis and JA signalling.
(5) Seed germination is inhibited by JA. However, JA biosynthetic and signalling mutants of Arabidopsis demonstrated that OPDA is the causal compound that inhibits seed germination together with abscisic acid (ABA) in a COI1-independent manner (Dave *et al.*, 2011). According to this scenario, chloroplast-derived OPDA is active in transcriptional activation, but it is not known how the rise in the OPDA is regulated. Here, the above-mentioned esterified OPDA and dinor-OPDA of galactolipids, called arabidopsides, may function as a storage pool of OPDA (Göbel and Feussner, 2009; Mosblech *et al.*, 2009; Dave and Graham, 2012). Moreover, the cytosolic OPDA pool is thought to be regulated via its conjugation with glutathionine (GSH) by GSH transferases and subsequent sequestration in vacuoles (Ohkama-Ohtsu *et al.*, 2011).
(6) OPDA is also thought to have a specific role in the expression of the *PHO1;H10* gene, which occurs in several stress responses (Ribot *et al.*, 2008), PHY A signalling and shade avoidance syndrome (SAS) (Robson *et al.*, 2010), hypocotyl growth inhibition (Brüx *et al.*, 2008) and COI1-independent defence signalling via ARF2 (Stotz *et al.*, 2011).
(7) Insect-induced closure of the Venus flytrap, *Dionea muscipula*, requires OPDA, which affects the secretion of digestive enzymes (Escalante-Pérez *et al.*, 2011).

It is not yet known how OPDA is perceived during OPDA-specific responses. Some of these responses might be explained by the occurrence of an α,β-unsaturated carbonyl group in OPDA, a characteristic of reactive electrophile species (RES) (Farmer and Davoine, 2007).

4.5. Co-repressors interacting with JAZs

Except for the EAR motif of JAZ8 as described above, the JAZ proteins lack a repression motif that is required for direct repression. Consequently, the JAZ proteins are suggested to recruit co-repressors. Indeed, using tandem affinity purification (TAP) NINJA was identified via TAP-tagged JAZ1 and shown to interact with TPL (Pauwels *et al.*, 2010). The hypothetical model for repression of JA-induced gene expression includes the TFs (e.g. MYC2), any JAZ protein and the adaptor NINJA linked to the co-repressor TPL via the EAR motif (Fig. 4). Whereas the JAZ proteins bind to TFs via the Jas domain, the ZIM domain of JAZs mediates homo- and heterodimerization as well as binding of NINJA. For JAZ5, JAZ6, JAZ7 and JAZ8 that carry an EAR motif, direct binding to TPL without NINJA is possible (Fig. 4). This is supported by the following data: (1) NINJA over-expressers and knockout lines have a decreased JA response, and (2) the EAR motif of NINJA and its homologue in ABA signalling act specifically in both adaptors (Pauwels *et al.*, 2010). NINJA and TPL were identified as integrators of JA/JA-Ile signalling. Both of them act as co-repressors of JA responses, and link JA and auxin signalling (see section 4.7).

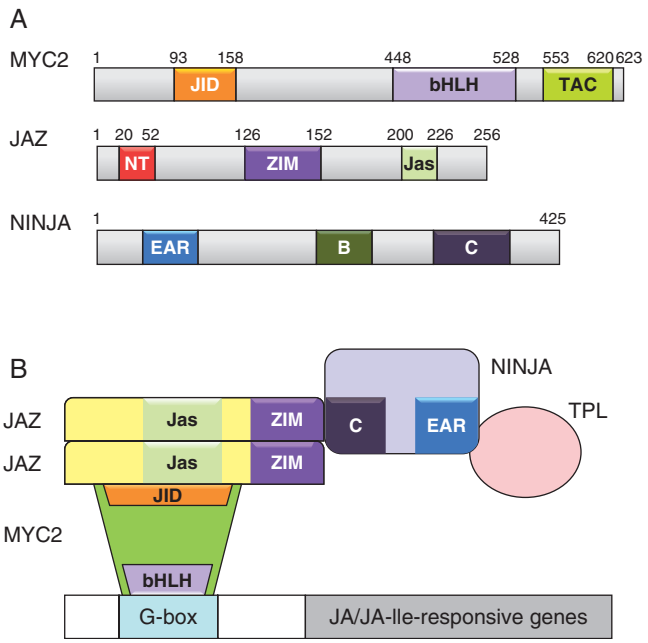

FIG. 4. The domain structure of MYC2, Jasmonate ZIM domain proteins (JAZ) and Novel Interactor of JAZ (NINJA) (A) and a hypothetical scheme of interaction between MYC2, JAZ, NINJA and TOPLESS (TPL) (B). Data adapted from Pauwels and Goossens (2011). B, conserved protein domain of NINJA; bHLH, DNA binding domain of MYC2; C, conserved protein domain of NINJA mediating binding to ZIM of JAZ; EAR, ethylene-responsive element binding factor-associated amphiphilic repression motif of NINJA-mediating binding to TPL; Jas, domain of JAZ for binding to COI1, MYC and other TFs; JID, JAZ-interacting domain of MYC2; NT, binding domain of JAZ to other TFs; TAC, domain of MYC2 for homo- and heteromerization; ZIM, domain of JAZ for binding to NINJA and for homo- and heteromerization.

Chromatin modifications performed by histone deacetylases (HDAs) are a basic mechanism underlying the suppression of gene expression, and are involved in Arabidopsis defence responses upon pathogen attack (Berr *et al.*, 2012). HDA6 and HDA19 are known to interfere with JA signalling, thereby affecting pathogen response, senescence and flowering (Zhou *et al.*, 2005; Wu *et al.*, 2008). They are genetically linked to TPL that cannot directly bind to DNA. Possibly, JAZ-mediated repression might finally result from suppression via HDA19 due to its binding to the co-repressor TPL (Fig. 3).

4.6. JAZ targets – TFs mediating JA-specific gene expression

As mentioned above, the plethora of JA signalling is sustained to a remarkable extent by the multiplicity in negative

regulation by JAZ proteins and co-repressor activities. The TFs preferentially acting as positive regulators bind to specific elements of the promoters of JA-responsive genes leading to separately acting pathways via singular or combinatorial activities of the TFs. Among them, MYC2 is the most prominent TF, a master switch in JA signalling, because it has been shown to regulate the expression of its most prominent marker gene *VEGETATVE STORAGE PROTEIN2* (*VSP2*). MYCs belong to the bHLH domain-containing TFs, and act as both activator and repressor of distinct JA-responsive gene expressions in Arabidopsis (Lorenzo *et al.*, 2004).

MYC2. This is a prominent member of the MYC-TF-family (Kazan and Manners, 2013). In 2007, it was the only DNA-binding TF known to bind also JAZ family members (Chini *et al.*, 2007). Its central role in numerous signalling pathways such as synthesis of glucosinolates, auxin, tryptophan, ET and JA as well as responses to wounding/insects, oxidative stress, pathogens and ABA-dependent drought stress has already been established (Dombrecht *et al.*, 2007; Kazan and Manners, 2008). It is an activator of JA-induced root growth inhibition, anthocyanin biosynthesis and oxidative stress tolerance, but a repressor in mediating resistance to necrotrophic pathogens and biosynthesis of tryptophan and indol glucosinolates (Lorenzo *et al.*, 2004; Dombrecht *et al.*, 2007). MYC2 activity takes place in a competitive interaction with the ET response factor ETR1 (Lorenzo *et al.*, 2004). The weak phenotype of the *myc2/jin1* mutant not only suggested the existence of other MYC-related TFs, but also indicated that JA-responsive gene expression is exclusively controlled by MYC2 (Montiel *et al.*, 2011). MYC2 suppresses the expression of *PLETHORA* (*PLT1* and *PLT2*) TFs, which are central regulators in auxin-mediated root meristem and root stem cell niche development by directly binding to the promoters (Chen *et al.*, 2011). The *PLT1/2* suppression complement the known JA-mediated regulation of auxin biosynthesis in the enzymatic step of anthranilate synthase α1 (ASA1) (Sun *et al.*, 2009), and represents a mechanistic framework for JA-induced root growth inhibition via auxin homeostasis and action. MYC2 is also involved in the circadian clock of JA signalling. Here, TIME FOR COFFEE (TIC) is one of the key components of the circadian clock, which negatively regulates JA signalling (Shin *et al.*, 2012). TIC inhibits MYC2 accumulation, thereby repressing *COI1* expression (Shin *et al.*, 2012). There are other examples where MYC2 also acts as a JAZ target: in nicotine biosynthesis (Shoji *et al.*, 2008; Zhang H-B *et al.*, 2012) and in the synthesis of terpenoid indol alkaloids of *Catharanthus roseus* (Montiel *et al.*, 2011) (see section 5).

Additional TFs are also active downstream of MYC2. The two members of the NAC TF family, ANAC019 and ANAC055, were identified by genetic and biochemical approaches as positive regulators of JA-induced *LOX2* and *VSP1* expression downstream of COI1 and MYC2 (Bu *et al.*, 2008). In summary, MYC2 is a master regulator in most JA-mediated signalling pathways involved in defence and development in Arabidopsis (Kazan and Manners, 2013): MYC2 mediates (1) antagonistic coordination of two branches in defence responses against herbivores and pathogens, (2) the establishment of induced systemic resistance (ISR) by beneficial soil microbes, (3) effector-mediated suppression of innate immunity in roots, (4) the regulation of cross-talk with SA, ABA, GAs and auxin, (5) a link between JA and other signalling pathways, such as light, phytochromes and circadian clock, and (6) the regulation of development, such as lateral and adventitious root formation, flowering time and SAS.

MYC3 and MYC4. Homologous proteins of MYC2 were picked up independently in three groups by yeast-two-hybrid screening using JAZ as bait (Cheng *et al.*, 2011; Fernández-Calvo *et al.*, 2011; Niu *et al.*, 2011). MYC3 and MYC4 are closely related to MYC2. Double and triple mutants of the three MYCs and over-expressers of MYC3 and MYC4 showed weak activity of both new MYCs in root growth inhibition as compared with MYC2, but strong involvement in the expression of wound-responsive genes. However, both responses are typical of that mediated by MYC2, thus indicating redundancy. Mutational analysis, however, revealed that the MYC2-regulatory effect was enhanced by MYC3 and MYC4, illustrating another level of modulation in JA signalling by modular and common activities of several TFs. This is supported by the fact that all the three MYC TFs show identical DNA binding specificities and bind preferentially to the G-box (Fernández-Calvo *et al.*, 2011), the *cis*-element to which most bHLH proteins can potentially bind (Dombrecht *et al.*, 2007).

All the three MYC TFs have two important domains: (1) a JAZ interaction domain (JID) adjacent to the N terminus, which is responsible for JAZ interaction, and (2) a conserved TAC-like domain at the C terminus, which is essential for homo- and hetero-dimerization of MYCs (Cheng *et al.*, 2011; Fernández-Calvo *et al.*, 2011). The JID domain occurring in MYC2, MYC3 and MYC4 is also present in other bHLH TFs, like GL3, EGL3 and TT8, which are known to be involved in anthocyanin formation and trichome initiation, and they have been shown to interact with JAZ1 and JAZ8 (Qi *et al.*, 2011). The WD40/bHLH (GL3, EGL3 and TT8)/MYB (PAP1 and GL1) complex is a regulatory module for anthocyanin and trichome initiation (Qi *et al.*, 2011) (see sections 5.5 and 9.7).

MYB21 and MYB24. Male sterility is the most prominent phenotype of the JA biosynthetic and signalling mutants of Arabidopsis, such as *coi1* and *opr3* (reviewed by Browse, 2009*a*, c). In a transcriptome analysis of developing stamens of *opr3* plants treated with JA, an up-regulation of TFs occurred, and MYB21 and MYB24 were the TFs identified (Mandaokar *et al.*, 2006). Later, both of them were identified as targets of JAZ1 and JAZ8 by yeast two-hybrid screening (Song *et al.*, 2011), showing that the interactions of both JAZs with MYB21 and MYB24 occur via the N-terminal R2R3 domain (Song *et al.*, 2011). The over-expression of *MYB21* in *coi1* or *opr3* partially rescued stamen filament length for both mutants, but insensitivity to JA in root growth and anthocyanin biosynthesis and susceptibility to *Bradysia* were not affected (Song *et al.*, 2011). Therefore, MYB21 and MYB24 are more specifically involved in fertility than in other JA-dependent processes.

4.7. Cross-talk between JAZ proteins and other hormone signalling cascades

ET-JA (EIN3/EIL1 and ERF1/ORA59 versus MYCs). In the JA signalling pathway, there is a parallel branch to the above-

mentioned MYC branch, the ETHYLENE RESPONSE FACTOR1 (ERF1), with the marker gene *PLANT DEFENSIN1.2* (*PDF1.2*) (Lorenzo *et al.*, 2004; Pieterse *et al.*, 2012). The synergistic cross-talk between JA and ET is known to occur preferentially for the response to necrotrophic pathogens (Pieterse *et al.*, 2012). Two central TFs of ET signalling, ETHYLENE-INSENSITIVE3 (EIN3) and EIN3-like (EIL1) bind JAZ1, JAZ3 and JAZ9 via the Jas domain of JAZs, resulting in the suppression of EIN3/EIL1 activity (Zhu *et al.*, 2011). This model is the first mechanistic explanation on synergistic cross-talk between ET and JA. Here, as the repressors of JA signalling, JAZs prevent ET signalling by inhibiting the ET-dependent TFs, but in the presence of JA-Ile, where the JAZs are subjected to proteasomal degradation, EIN3/EIL1 becomes free and requires ET for their stabilization as usual.

A second tier of synergistic signalling of JA and ET is conferred by the TFs ORA59 and ERF1 (Pre *et al.*, 2008) that act downstream of EIN3/EIL1 (Leon-Reyes *et al.*, 2009). Here, the synergistic action of JA and ET is mediated by two GCC-boxes, e.g. ORA59 binds to the *PDF1.2* promoter (Zarei *et al.*, 2011). The ERF/ORA59 branch is activated upon infection by necrotrophic pathogens leading to the expression of *PDF1.2*, thus antagonizing the MYC-mediated branch, which is activated by herbivorous insects leading to the expression of *VSP2* (Pieterse *et al.*, 2012). Consequently, the defence response against insect attack is expected to be compromised in plants with an activated ERF/ORA59 branch (Fig. 5). Accordingly, an activated MYC-branch of the JA pathway will prevent herbivore-induced stimulation of the ERF branch, and the plants will be less attractive to the herbivores (Verhage *et al.*, 2011).

JA-GA (DELLAs versus JAZs). There are synergistic as well as antagonistic cross-talks between GA and JA depending of the process in which these hormones are involved. For stamen development, both hormones act synergistically (Fig. 6). The DELLA proteins, accumulating upon GA deficiency, prevent JA biosynthesis via the suppression of *DAD1* and *LOX* expression (Cheng *et al.*, 2009; Song *et al.*, 2011). This leads to JA deficiency that causes male sterility by repression of JA-dependent gene expression of the essential TFs MYB21 and MYB24. In the absence of JA/JA-lle, this down-regulation is even attenuated by an inhibition of MYB21 and MYB24 actions through binding of JAZs (Cheng *et al.*, 2009). In contrast, an antagonistic cross-talk between JA and GA occurs in plant growth and defence responses, which are themselves antagonistic because plant defence occurs at the expense of plant growth (Hou *et al.*, 2010; Kazan and Manners, 2012; Yang D-L *et al.*, 2012) (Fig. 6 and see section 9.6).

The JAZ proteins have their counterparts in five DELLA proteins of Arabidopsis which are active in GA signalling; GAI/SLY is the homologue of COI1, GID1 is the GA receptor and the DELLAs RGL and RGL1-like (RGL1, RGL2 and RGL3) are the repressors (Schwechheimer, 2012).

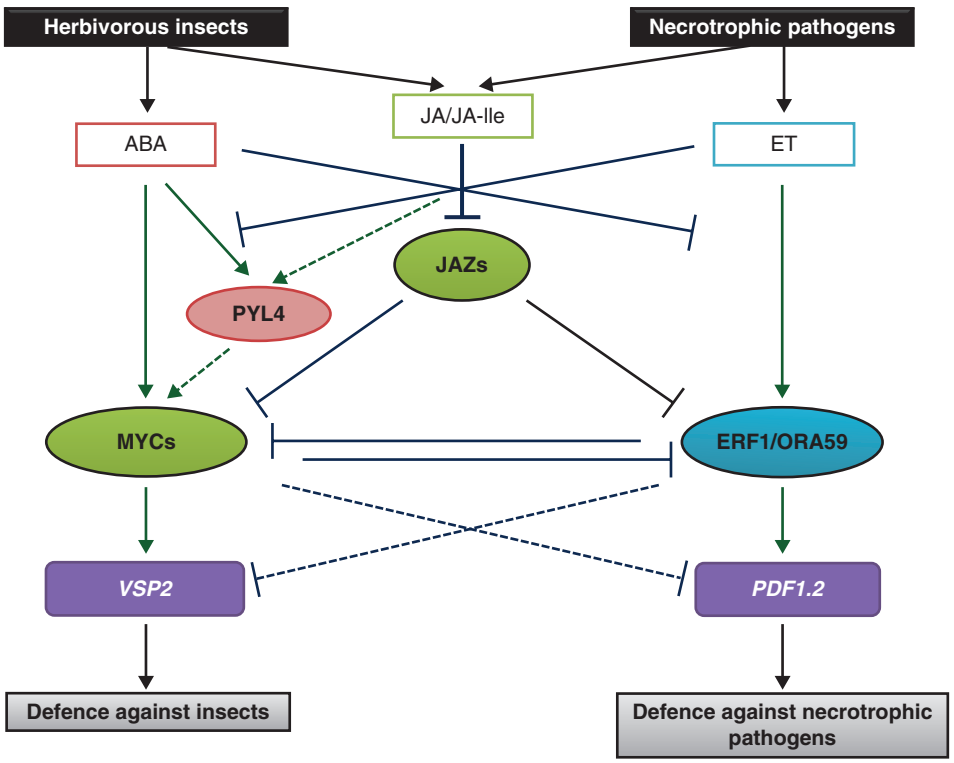

FIG. 5. The cross-talk between jasmonate (JA), ethylene (ET) and abscisic acid (ABA) triggered in response to herbivorous insects and necrotrophic pathogens (adapted from Pieterse *et al.*, 2012). Attack by herbivorous insects induces JA- and ABA-dependent signalling pathways, whereas infections by necrotrophic pathogens induce JA- and ET-dependent signalling pathways. Both branches are antagonistically regulated. Solid lines, known interactions; dashed lines, hypothetical interactions; green arrows, positive effects; blue inhibition lines, negative effects. Compounds are given in rectangles, transcriptional regulators in circles, regulated genes in purple. ERF1, ethylene response factor 1; ORA59, octadecanoid-responsive Arabidopsis AP2/ERF-domain protein 59; PYL4, PYR1-like protein 4 (ABA receptor); other acronyms are given in Fig. 3.

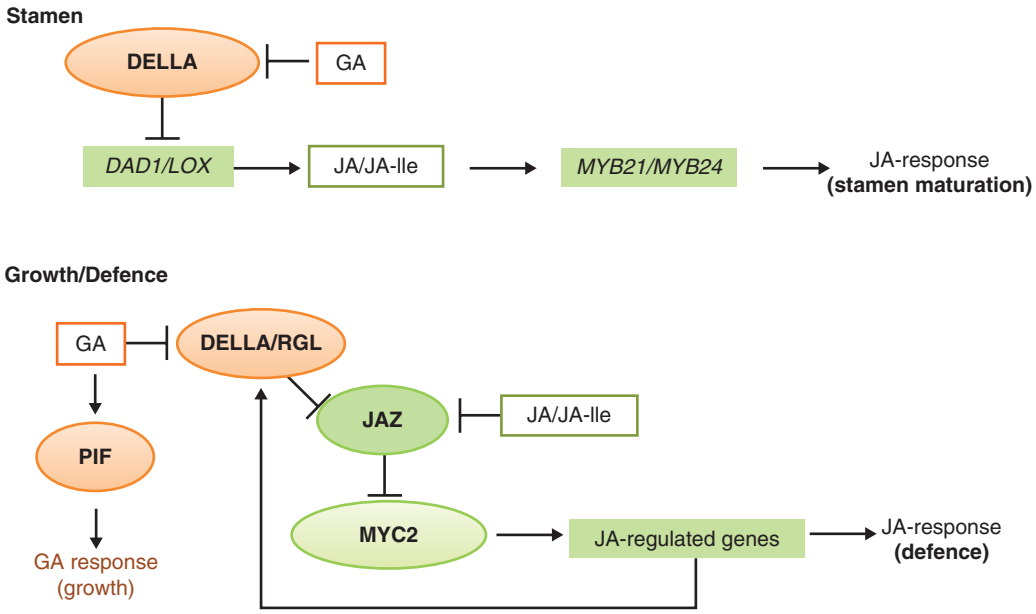

FIG. 6. Cross-talk between jasmonic acid (JA)- and gibberellic acid (GA)-signalling pathways in stamen maturation and during growth and defence processes. In stamen, DELLA negatively affects the expression of genes encoding JA biosynthetic enzymes. An increase in GA will result in the removal of DELLA leading to enhanced synthesis of JA/JA-Ile. In turn, this induces the expression of *MYB21/24*, which is crucial for stamen maturation. In vegetative tissues, the DELLA-TF DELLA RGA-like (RGL) competes with MYC2 for binding to JAZ. With an increasing level of JA/JA-Ile, MYC2 is released and mediates the transcription of not only JA-regulated genes involved in defence but also encoding RGL. An increase in RGL will amplify the defence response by recruiting Jasmonate ZIM domain protein (JAZ) followed by release of MYC2. In contrast, accumulation of GA will lead to degradation of RGL, thereby releasing JAZ to inhibit MYC2. In parallel, GA activates the growth response via phytochrome interacting factor (PIF); other acronyms are given in Fig. 1.

Interestingly, all these proteins can interact with JAZs via the Jas domain, thereby competing with MYC2 in JAZ binding (Hou *et al.*, 2010; Wild *et al.*, 2012; Yang D-L *et al.*, 2012). In this model GA triggers the degradation of DELLA, thereby allowing JAZ1 to bind to MYC2, which leads to the repression of JA signalling, whereas in the absence of GA, DELLAs exist and bind to JAZs resulting in de-repression of MYC2 (Hou *et al.*, 2010; Wager and Browse, 2012). This was shown in particular for RGL3 (Wild *et al.*, 2012); its expression is induced in a COI1- and MYC2-dependent manner due to direct binding of MYC2 to the *RGL3* promoter, and it interacts with JAZ1, again representing a competitive binding for MYC2. Consequently, the rise in JA-Ile will result in an accumulation of RGL3 leading to trapping of JAZ1 and enhancement of the MYC2 activity. In the presence of GA, however, RGL3, a negative regulator in GA signalling, will be subjected to degradation, thereby allowing JAZ1 to inhibit the MYC2 activity and resulting in depression of JA-induced gene expression (Wild *et al.*, 2012). Obviously, there is a flexible balance of both negative regulators DELLAs and JAZs, which sustain the antagonistic behavior of the growth of above-ground plant parts vis-à-vis their defence. Note that cross-talks between plant hormones can differ dramatically between different plant organs. In contrast to this antagonism in above-ground plant parts, there occurs no cross-talk between GA and JA in roots.

Auxin-JA (ARFs versus MYBs). In roots, the well-known growth inhibition by JA occurs via a cross-talk with auxin. This root growth inhibition does not take place in *coi1* and *myc2* mutants, but is increased in the *jaz10* mutant, indicating the involvement of COI1, MYC2 and JAZ10 (Chen *et al.*, 2011). As MYC2 represses the expression of *PLETHORA* (see section 4.6), the central regulator of root meristem activity, cell elongation and cell number, this might counteract the auxin–TIR1-AUX/IAA–ARFs signalling cascade, leading to diminished expression of both *PLETHORA* genes (Chen *et al.*, 2011). Additionally, JA may increase auxin levels by inducing the expression of *ASA1* that encodes the first enzyme in auxin biosynthesis (Sun *et al.*, 2009). On the other hand, there is an auxin-induced expression of *JAZ1*, which might have an integrator function in auxin–JA interaction leading to a regulatory loop in sustaining auxin and JA signalling (Grunewald *et al.*, 2009). Interestingly, the tryptophan-conjugates of JA and IAA are endogenous auxin inhibitors that affect auxin sensitivity in a COI1-independent and/or TIR1-dependent manner (Staswick, 2009), thereby illustrating another mechanism of JA-auxin cross-talk.

In flowers, auxin signalling requires AUXIN RESPONSE FACTOR6 (ARF6) and ARF8, both of which induce the expression of JA biosynthesis genes in filaments (Nagpal *et al.*, 2005). Consequently, *arf6-2arf8*-3 filaments are characterized by low JA levels. Obviously, petal and stamen growth are determined by a common regulatory network that entails JA-dependent transcription factors MYB21 and MYB24 (see section 4.6) as well as auxin-dependent transcription factors ARF6 and ARF8 (Reeves *et al.*, 2012). In addition, the regulatory effects of ARF6 and ARF8 on JA biosynthesis result in a negative regulation of class1 KNOX genes, which are important negative regulators of optimal flower development (Tabata *et al.*, 2010). It is thus clear that ARF6 and ARF8 function via JA in the progression of floral development.

Brassinosteroid (BR)-JA (DWARF4 versus MYBs). In contrast to the well-known growth-inhibitory effect of JA, BRs promote the growth of above-ground plant parts. The BR signalling cascade is well described, including the BR receptor BRI1, the BR-associated kinase1 (BAK1) and the transcription factors that are involved in BR-induced expression BES1 and BZR1 (Clouse, 2002). The main phenotype of mutants that are defective in the BR receptor is dwarfism. Interestingly, in a genetic screen on suppressors of *coi1*, a *psc1* mutant was found with partially suppressed *coi1*-phenotype (Ren *et al.*, 2009). This mutant carries a mutation in *DWF4* that encodes a key enzyme in BR biosynthesis, suggesting that BRs might counteract JA signalling. Indeed, *psc1* in a background of wild-type *COI1* displays JA hypersensitivity, especially in respect to JA-induced inhibition of root growth (Huang *et al.*, 2010). The BR application leads to anthocyanin accumulation, which is a hallmark of JA-induced responses (see section 5.5), whereas JA-induced anthocyanin accumulation is reduced in BR-biosynthetic mutants (Peng *et al.*, 2011). Here, JA-mediated induction of 'late' anthocyanin biosynthesis genes was suppressed by reduced BR synthesis (*dwf4-102*) or disturbed BR perception (*bri1-4*) via the reduced expression of two MYB genes *PAP1* and *PAP2* (Peng *et al.*, 2011; Song *et al.*, 2011). In contrast, JA inhibits COI1-dependent *DWF4* expression, indicating that *DWF4* itself is down-regulated by JA and is located downstream of *COI1* in the JA signalling pathway.

JA-ABA (PYL4 versus JA-dependent TFs). Cross-talk between ABA and JA is not surprising given their common central role in several stress responses (Cutler *et al.*, 2010). ABA was identified as an essential signal in *Pythium irregulare*-induced defence responses of *A. thaliana* (Adie *et al.*, 2007). Although contentious, the positive and negative roles of ABA in JA/ET-mediated defence have been described and at least partially linked to callose formation (Ton *et al.*, 2009). Besides its role in plant resistance, there is a role of ABA in JA-mediated wound response (Kazan and Manners, 2008). The recent identification of the direct ABA receptors, the PYR/PYL/RCAR proteins, has allowed the synthesis of a mechanistic view on the cross-talk between ABA and JA. For instance, the tobacco *NtPYL4* gene has been shown to encode a functional ABA receptor and is induced by JA (Lackman *et al.*, 2011). A similar link between ABA and JA exists in Arabidopsis, where homologues of *NtPYL4*, *PYL4* and *PYL5* are also induced by JA. The *pyl4* and *pyl5* mutants exhibiting hypersensitivity in JA-mediated biomass reduction recorded a decline in JA-induced anthocyanin accumulation (Lackman *et al.*, 2011). This unequivocally suggests that the ABA–JA cross-talk contributes to maintaining the balance between growth and defence (Fig. 5).

JA–SA (COI1/MYC2 versus NPR1/TGAs). JA–SA cross-talk has been known for a long time and is the most studied cross-talk among plant hormones. It has been recently reviewed in detail, with reference to its role in plant immunity (Pieterse *et al.*, 2012). Therefore, we will discuss here only key aspects to complement the cross-talks mentioned above. In principle, JA signalling is involved in responses to necrotrophic pathogens and herbivorous insects with key components such as JA biosynthetic enzymes, COI1, JAZs, NINJA, TPL and MYC2 as described above. In response to biotrophic pathogens, however, SA is the central regulator (Vlot *et al.*, 2009; Pieterse *et al.*, 2012). Here, the SA biosynthesis occurs via two parallel pathways – the well-known PAL reaction and the ISOCHORISMATE SYNTHASE (ICS/SID2) reaction (Garcion and Metraux, 2006). As an experimental tool, overexpressing lines of the bacterial *NahG* gene encoding an SA dehydrogenase have been generated (Mur *et al.*, 1997) significantly compromising the accumulation of SA (Delaney *et al.*, 1994). The central regulator in SA signalling is NONEXPRESSOR OF PR GENES1 (NPR1) that, in the presence of SA, is a transcriptional co-activator for many defence genes. There are NPR1 multimers which monomerize by SA-induced changes of the redox state via thioredoxin followed by the transport of the monomeric forms into the nucleus. Here, they bind as activators to TGA TFs specific for SA-inducible genes, are phosphorylated during transcription initiation and are subsequently ubiquitinylated following binding to a CULLIN3-based ligase (Fu *et al.*, 2012), and are subjected to proteasomal degradation. Later, new nuclear-imported NPR1 monomers can again allow SA-induced gene expression (Spoel *et al.*, 2009). In this model, turnover of the co-activator NPR1 has dual roles in both preventing and stimulating gene expression. In addition to this basic component, several other factors are also known to be involved in the SA signal transduction pathways (Pieterse *et al.*, 2012).

The SA–JA cross-talk was initially observed in the wound response of tomato (Harms *et al.*, 1998). In nature, however, plants are attacked simultaneously and sequentially by a single or several attackers that induce the SA- and/or JA-signalling pathways. The preferential induction of one pathway and its antagonistic interaction with another pathway has been repeatedly demonstrated (Koornneef and Pieterse, 2008; Pieterse *et al.*, 2012) and can be shifted from an antagonistic to synergistic interaction depending on SA and JA concentrations (Wees *et al.*, 2000; Mur *et al.*, 2006). Environmental cues, such abiotic stresses as thermotolerance (Clarke *et al.*, 2009) or shade avoidance (Ballaré, 2011), seem to be involved in maintaining a balance between the SA and JA pathways. The cross-talk between SA and JA has been observed in many Arabidopsis accessions (Koornneef and Pieterse, 2008), and is even transmitted to the next generation (Luna *et al.*, 2012). The adaptability of plants in nature may be attributed to the flexibility of both pathways as conditioned by their individual components as well as interactions.

The putative roles of several new players in the above-mentioned model are as follows (Pieterse *et al.*, 2012):

(1) Mitogen-activated protein (MAP) kinases are clearly involved (Rodriguez *et al.*, 2010*a*). MPK4 acts as a negative and positive regulator of SA and JA signalling pathways, respectively.
(2) The redox regulators glutaredoxins (GRXs) and thioredoxins (TRXs) that sustain the redox state of proteins in terms of disulfide bridges in a glutathionin-dependent manner represent other regulatory modules, where JA decreases and SA increases the glutathionin pool (Spoel and Loake, 2011).
(3) The above-mentioned NPR1, active as a monomer in the nucleus by binding to TGAs, has a distinct role in the

cytoplasm as a multimer (Ramirez *et al.*, 2010) and is regulated by ET (Leon-Reyes *et al.*, 2009).

(4) Some of the clade II TGAs, such as TGA2, TGA5 and TGA6, have been shown to have a positive effect on JA/ET-dependent defence gene expression in the absence of SA (Zander *et al.*, 2010). Obviously, these TGAs counteract the negative effect of MYC2 in the JA/ERF branch (see above). GRXs (called ROXYs) have been postulated to modulate the activities of these TGAs in the JA/ERF branch (Zander *et al.*, 2012; Gatz, 2013). Consequently, the outcome of the JA/ET-induced and SA-suppressed expression of defence genes would be sustained by the simultaneous and sequential action of several key factors, such as the levels of JA, ET and SA, and the expression of MYC2, ROXY19, TGA2, TGA5, ERF/EIN and PDF1.2 (Gatz, 2013).

(5) WRKY TFs, such as WRKY50 and WRKY51, are essential for SA-mediated suppression of JA signalling (Gao *et al.*, 2011), while WRKY62 is a negative regulator of JA signalling acting downstream of cytosolic NPR1 (Mao *et al.*, 2007). Another WRKY TF involved in the cross-talk between SA and JA is WKRY70, which is directly controlled by AtMYB44 that activates the SA-induced defence response and represses the JA branch (Shim *et al.*, 2013).

(6) Subunit 16 of the Mediator complex (MED16) is required for positive regulation of systemic acquired resistance (SAR) via NPR1, but is a negative regulator of resistance to necrotrophic fungal pathogens via the JA/ET pathway. It is thus clear that the SA and JA/ET pathways converge to MED16, which links specific transcriptional regulators with the RNA polymerase II transcription machinery (Zhang X *et al.*, 2012*a*).

(7) The Ca^{2+}/CaM-binding TF AtSR1 causes down-regulation of SA levels, thereby abolishing its negative impact on basal and induced JA levels upon wounding (Qiu *et al.*, 2012).

The targets of these enumerated effectors of SA–JA signalling are not well understood. In earlier dissection of SA–JA cross-talk, a direct inhibitory effect of SA on JA biosynthesis was assumed due to the inhibitory effect of aspirin (Harms *et al.*, 1998). However, recent mutant analyses have clearly shown that SA antagonizes the JA pathway downstream of JA biosynthesis (Leon-Reyes *et al.*, 2010). Another target for SA-mediated suppression of JA signalling occurs at the transcriptional level. Here, components of the SA signalling, such as TGAs and WRKYs, inhibit the expression of JA-dependent TFs (Pieterse *et al.*, 2012). The numerous components of SA–JA cross-talk are superimposed by other hormones that modulate this cross-talk (Pieterse *et al.*, 2012). This may provide a simpler explanation for the mechanistic basis of the above-mentioned cross-talks such as JA–ET, JA–GA, JA–ABA and JA–BR. Furthermore, evolution of JA–SA cross-talk is a matter of interest because this obviously ancient phylogenetic cross-talk is thought to be of adaptive significance (Thaler *et al.*, 2012).

5. REGULATION OF PLANT SECONDARY METABPOLISM BY JASMONATES

Twenty years ago, it was demonstrated that an endogenous rise in JA levels upon elicitation with a rough yeast elicitor was associated with the induction of alkaloid synthesis in plant cell cultures (Gundlach *et al.*, 1992). Later, the proof of concept for JA-mediated induction of biosynthesis of secondary metabolites came from studies on constitutive activation of the JA signalling pathway in tomato, leading to the constitutive accumulation of caffeoylputrescine (Chen *et al.*, 2006). In 2000, the first TFs involved in JA-dependent terpenoid indole alkaloid (TIA) synthesis in *C. roseus* were identified (van der Fits and Memelink, 2000). These TFs were called OCTADECANOID-DERIVATIVE RESPONSIVE CATARANTHUS AP2-DOMAIN2 and 3 (ORCA2 and ORCA3) (reviewed by Memelink *et al.*, 2001). Meanwhile, several TF families involved in the synthesis of TIA, nicotine, artemisinin, anthocyanins, camalexin, indol glucosinolates and volatile terpenes have been identified. A common facet underlying JA-mediated transcriptional control of secondary metabolite biosynthesis is involvement of the SCF^{COI1} complex, JAZ proteins and MYC2 together with additional components, such as WRKYs, ORCAs, ERFs, MYBs, PAP1 and ZCTs, all of them being active in distinct pathways.

As the involvement of TFs in JA-mediated regulation of secondary metabolite biosynthesis has been recently reviewed (Memelink, 2009; De Geyter *et al.*, 2012), we address here only a few aspects. The examples shown below illustrate that similar and homologous TFs are involved in the JA-dependent biosynthesis of different secondary compounds in different plant species, suggesting an early conserved evolution of the JA signalling network regulating the biosynthesis of secondary metabolites (De Geyter *et al.*, 2012).

5.1. Nicotine

Most of the structural genes encoding enzymes for nicotine biosynthesis are transcriptionally regulated by JA, and depend on a functional COI1–JAZ co-receptor (Shoji *et al.*, 2008). In a genomic screen, bHLH TFs, such as MYC2, were identified to be active in nicotine biosynthesis (Todd *et al.*, 2010; De Boer *et al.*, 2011; Shoji and Hashimoto, 2011). NtMYC2 acts together with AP2/ERFs that occur in the NIC2 locus (Shoji *et al.*, 2010; De Boer *et al.*, 2011). The family of tobacco ERFs has 239 members (Rushton *et al.*, 2008). Seven of them resulting from gene duplication are involved in nicotine biosynthesis, and are clustered in the NIC2 locus. Together, they represent a positive regulatory unit (Shoji *et al.*, 2010). NIC2/ERFs are close homologues of ORCA3 of *C. roseus* and activate TIA biosynthesis genes via a GCC-box. Here, ORCA/ERF221 and MYC2 act synergistically in binding, and both of them are post-translationally up-regulated by a JA-modulated phosphorylation cascade, where MAPKK1 (JAM1) is active (De Boer *et al.*, 2011). It is interesting that, in two different pathways and two different plant species (nicotine biosynthesis in tobacco and TIA biosynthesis in *C. roseus*), homologous TFs evolved obviously independently. The widely distributed induction of secondary

metabolite biosynthesis by JA may indicate an evolutionary advantage of an established regulatory module.

5.2. Vinblastine

Vinblastine is a TIA and is synthesized in *C. roseus* cells, where expression of the enzyme-encoding genes is regulated by a cascade of transcription factors including CrMYC2 that regulate the expression of the AP2/ERF domain TFs, such as *ORCA2* and *ORCA3* (Zhang *et al.*, 2011). CrMYC2 is encoded by an immediate-early JA-responsive gene. CrMYC2 binds *in vitro* to jasmonate regulatory elements (JREs) in the promoter of *ORCA3* leading to the expression of *ORCA3*. A down-regulation of *CrMYC2*, however, does not down-regulate the TIA biosynthesis, indicating sufficient basal expression of *ORCAs* (Zhang *et al.*, 2011). Moreover, at least ORCA3 regulates most but not all steps in TIA synthesis (Suttipanta *et al.*, 2011). The negative regulation of *CrMYC2* by JAZ as observed in many JA-dependent pathways remains to be elucidated.

5.3. Artemisinin

Biosynthesis of the antimalarial sesquiterpene lactone artemisinin is positively controlled by two JA-responsive ERFs, namely ERF1 and ERF2 (Yu *et al.*, 2012), which act in a concerted manner with MYC2. WRKY1, a TF in artemisinin biosynthesis, is also assumed to act in such a concerted manner with MYC2 (Ma *et al.*, 2009). Artemisinin is synthesized and stored in glandular secretory trichomes. Global transcript profiling revealed the expression of trichome-specific genes correlating with the expression of genes active in artemisinin biosynthesis (Maes *et al.*, 2011). All these genes are simultaneously activated in a JA-dependent manner.

The artemisinin biosynthetic machinery is confined to specialized cells of glandular trichomes (Olsson *et al.*, 2009). In this context, it is interesting to note another JA-related process in *Artemisia annua*. The volatile MeJA released from this species has been correlated with the expression of defence genes in a neighbouring tomato plant (Farmer and Ryan, 1990).

5.4. Glucosinolates/camalexin

Glucosinolates are a large group of secondary metabolites involved in plant resistance to insects and pathogens. They are amino acid-derived compounds and can be classified into aliphatic, benzenic and indolic glucosinolates synthesized in numerous steps, most of which are JA-inducible (Sønderby *et al.*, 2010). The main components of the JA/JA-Ile signalling pathway leading to glucosinolates and camalexin have been identified. For camalexin biosynthesis, an SCF^{COI1}–JAZ-MYC2 branch and an MEK1-MKK3-MPK6-WRKY33 branch have been identified (De Geyter *et al.*, 2012). Camalexin biosynthesis seems to be controlled by ANAC042, a member of the NAC TF family. Expression of *ANAC042* is induced by flagellin and depends on ET signalling, but is repressed by the application of MeJA (Saga *et al.*, 2012) indicating a modulation of camalexin formation by JA via ANAC042 (De Geyter *et al.*, 2012). An additional control is given by a member of the DNA-binding one finger (DOF) TF family. Here, the JA/JA-Ile-inducible DOF1.1 is a positive regulator of indole glucosinolates via *CYP83B1* expression (Skirycz *et al.*, 2006).

5.5. Anthocyanin

Anthocyanin accumulation represents the most prominent JA/JA-Ile phenotype. TFs, such as PAP1, EGL3, GL3, MYB75 and TT8 being essential components of the WD-repeat/bHLH/MYB transcriptional complexes, are involved in anthocyanin biosynthesis (and trichome development, see section 9.7). Recent biochemical and genetic evidence indicated that these TFs are targets of JAZs, thereby providing a mechanistic framework for JA-induced anthocyanin formation (Qi *et al.*, 2011).

5.6. Benzylisoquinoline alkaloids

This group of compounds comprising about 2500 structures including the most prominent compound morphine is of pharmacological interest. Several of the numerous enzymes in their biosynthesis consisting of different branches (Ziegler and Facchini, 2008) are encoded by JA-inducible genes. These pathways were among the first to be identified, where an endogenous rise in JA upon elicitation was shown to be the reason for alkaloid synthesis (Gundlach *et al.*, 1992; Blechert *et al.*, 1995).

6. JA IN HERBIVORY AND PLANT–INSECT INTERACTIONS

The molecular recognition of pathogens and herbivores by plants exhibits remarkable similarities in the modules which are used for recognition and responses (Erb *et al.*, 2012). For herbivores, at least three different responses can be conceptually distinguished: (1) herbivore-induced immunity (HTI) can appear upon recognition of oviposition-associated compounds, (2) HTI can occur upon perception of herbivore-associated molecular patterns (HAMPs) or damage-associated molecular patterns (DAMPs), and (3) wound-induced resistance (WIR) is generated by mechanical wounding during herbivory. These responses are linked in several tiers of activity. Oral secretions of insects are produced in a species-specific manner with quantitative and qualitative differences among the elicitor compounds, whereas plants respond to these elicitor combinations differentially (Erb *et al.*, 2012).

The elicitation of a wound response in plants appearing upon mechanical wounding or herbivore attack is one of the most prominent examples and extensively studied areas, where JA/JA-Ile is involved as a signal. The data generated in this area of research over the last five years has been thoroughly reviewed (Howe and Jander, 2008; Koo and Howe, 2009; Felton and Tumlinson, 2008; Walling, 2009; Dicke and Baldwin, 2010; Heil and Karban, 2010; Bonaventure *et al.*, 2011*b*; Sun *et al.*, 2011*a*; Erb *et al.*, 2012; Meldau *et al.*, 2012). Therefore, only some aspects will be discussed here. Moreover, *N. attenuata* has been intensively studied in the last decade regarding different aspects of herbivory, JA/JA-Ile biosynthesis and JA/JA-Ile signalling, including field experiments in the desert of Utah (USA). The complexity of JA/JA-Ile biosynthesis and signalling in relation to herbivory as dissected in this species compared with that in other

model plants, such as Arabidopsis and tomato, is unique and has been repeatedly reviewed (Kant and Baldwin, 2007; Schwachtje and Baldwin, 2008; Gális *et al.*, 2009; Wu and Baldwin, 2009; Baldwin, 2010; Dicke and Baldwin, 2010; Kessler *et al.*, 2010; Wu and Baldwin, 2010; Bonaventure *et al.*, 2011*b*; Kessler and Baldwin, 2011; Meldau *et al.*, 2012).

Local wounding by herbivores leads to a burst in newly synthesized JA. The constitutive occurrence of enzyme proteins involved in JA biosynthesis in all leaf tissues (as in Arabidopsis; Stenzel *et al.*, 2003*b*) or in vascular bundles (as in tomato; Hause *et al.*, 2000, 2003) may be ascribed to such an immediate rise in JA levels within several minutes (Glauser *et al.*, 2008; Mielke *et al.*, 2011), whereas later (from 15 min onwards) the transcriptional machineries for the expression of *LOX*, *AOS*, *AOC*, *OPR3* and *JAZs* are activated (Stenzel *et al.*, 2003*a*, *b*; Chung *et al.*, 2008; Koo and Howe, 2009). In this signalling network compounds of insects' oral secretions, such as volicitin (Bonaventure *et al.*, 2011*b*), the peptide systemin, H_2O_2, NO and ET, act as additional positive signals (see Wasternack, 2006; Koo and Howe, 2009).

The involvement of jasmonates in systemic response upon local wounding has been a matter of debate. Primarily, grafting experiments with tomato mutants defective in JA biosynthesis and signalling revealed strong evidence that signalling but no JA biosynthesis is required in systemic leaves (Li *et al.*, 2002). Support for the involvement of JA compounds in systemic response came from observations that showed the occurrence of JA biosynthesis enzymes in mid veins of wounded leaves (Hause *et al.*, 2000, 2003), enrichment of jasmonate compounds in mid veins (Stenzel *et al.*, 2003*a*; Glauser *et al.*, 2008) as well as their occurrence in phloem exudates of systemic leaves (Truman *et al.*, 2007; Gaupels *et al.*, 2012). However, in Arabidopsis, it has been shown that the occurrence of JA/JA-Ile in systemic leaves requires the presence of intact OPR3 and JAR1, accompanied by a rapid decline in OPDA levels, thus arguing against the transport of any JA compound (Koo *et al.*, 2009). In feeding experiments, labelled JA-Ile could not be recovered in systemic leaves, suggesting that it is not a long-distance signal in *N. attenuata* (Wang *et al.*, 2008). Less stronger support for this view also came from studies on systemic transport of labelled JA-Ile in tomato (Matsuura *et al.*, 2012). Besides chemical signalling by JA compounds, the involvement of hydraulic and electrical signalling, due to action and variation potentials, in systemic response has been discussed (see Koo and Howe, 2009). More recently, a 'system potential' has been proposed for systemic wound signalling that involves stimulation of an H^+-ATPase in the plasma membrane concomitant with ion fluxes (Zimmermann *et al.*, 2009). Although volatile MeJA that is released from locally wounded leaves has been proposed to act as a long-distance signal (Heil and Ton, 2008) to evoke the systemic response, this has not yet been experimentally substantiated (Koo and Howe, 2009).

Systemin was the first peptide identified as a signalling compound in wounded tomato leaves (Pearce *et al.*, 1991). Initially, systemin was thought to be a systemic signal involved in long-distance signalling. After two decades of research on wound-induced systemic response, systemin is thought to have a minor role by amplification of systemic wound signalling in a tissue-specific manner (Hind *et al.*, 2010; Sun *et al.*, 2011*a*). Due to the central role of JA/JA-Ile in SCF^{COI1}-mediated signalling, one amplification activity of systemin seems to be its positive regulation of the expression of *AOC* and JA formation upon wounding (Stenzel *et al.*, 2003*a*). This activation of JA biosynthesis by systemin involves activity of MPK1, MPK2 and MPK3 (Kandoth *et al.*, 2007) (for details see Koo and Howe, 2009; Sun *et al.*, 2011*a*; Yamaguchi and Huffaker, 2011).

Wounding by herbivores leads to (1) direct defence via synthesis of toxic compounds as well as toxic or antinutritional proteins such as proteinase inhibitors (PIs) and (2) indirect defence by release of volatiles to affect the attraction of carnivores, parasitoids and predators or by altered oviposition of herbivores (Wasternack and Hause, 2002; Howe and Jander, 2008). In this event plants become immunized by the *PI* expression that affects protein digestion in the herbivore gut. Of the herbivore- and JA-induced proteins, threonine deaminase 1 (TD1) is required for the formation of isoleucine. An interesting adaptive evolution by gene duplication of TD has been demonstrated for tomato (Chen *et al.*, 2007; Gonzales-Vigil *et al.*, 2011). The proteolytic cleavage of the regulatory domain of *TD2* may be attributed to plant resistance in response to herbivory.

However, different defence strategies adopted by plants against phloem-feeding insects, such as aphids and whiteflies, must be distinguished (Walling, 2008). These strategies include hormonal signalling by ET, SA and JA affecting pre-entry, entry and colonization by the insects. Recently, the importance of root-derived oxylipins in colonization of the above-ground organs by insects has been elucidated. A phloem sap-consuming green peach aphid of Arabidopsis needs LOX5-derived oxylipins produced within the roots for infestation of the foliage (Nalam *et al.*, 2012). The strategy used by aphids and whiteflies for delivering salivary compounds and proteins is similar to that adopted by phytopathogenic microbes. Here, toxic compounds or effector proteins are released to suppress the host's PAMP-triggered immunity (PTI), but may be perceived by the host plant via different strategies leading to effector-triggered immunity (ETI) that results in various defence responses (Jones and Dangl, 2006; Pieterse *et al.*, 2012).

Finally, plants such as Arabidopsis synchronize defence against herbivores by circadian JA accumulation with circadian insect behavior (Goodspeed *et al.*, 2012). Accumulation of JA and SA is circadian-regulated in different phases, and cabbage loopers (*Trichoplusia ni*) feed rhythmically on plants grown in light/dark cycles with only moderate tissue damage.

7. JA IN PLANT–PATHOGEN INTERACTIONS

As mentioned above, recognition and response modules can be defined for microbe-, pathogen- and damage-associated molecular patterns (MAMPs, PAMPs and DAMPs) which are similar to that active in herbivory. Pattern recognition receptors (PRRs) that recognize PAMPs develop PTI, which is suppressed by pathogen effectors, whereas the resistance gene products that recognize the effectors lead to ETI (Jones and Dangl, 2006; Erb *et al.*, 2012).

Current results suggest that JA induces resistance against necrotrophic pathogens, some phloem-feeding insects and chewing herbivores, whereas SA induces resistance against biotrophic pathogens and some phloem-feeding insects. This

antagonistic SA–JA cross-talk and its evolutionary significance have been discussed recently (Thaler *et al.*, 2012). We have already discussed some molecular aspects of SA–JA cross-talk in section 4.7, while the role of JA in plant interactions with plant–necrotrophic pathogen interactions has been thoroughly reviewed over the last couple of years. However, we refer to a recent and elegant review on hormonal modulation of plant immunity in relation to JA and its cross-talk with SA (Pieterse *et al.*, 2012). Thus, our discussion here will be limited to only some aspects.

7.1. JA, ET and SA in plant–pathogen interactions

The JA signalling cascade via $SCF^{COI1-JAZ}$ is the backbone representing a link between responses to necrotrophic pathogens and resistance to herbivorous insects (Fig. 5). In both cases, JA is generated. JA acts synergistically with ET upon attack by necrotrophic pathogens, but with ABA during herbivory. ET confers the defence response via the expression of *ERF1/ORA59* and *PDF1.2*, whereas ABA operates via PYL4 and MYCs to elicit the defence response through the expression of *VSP2*. There are two antagonistic interactions between both pathways: (1) the level of ET and ABA; and (2) at the level of TFs, e.g. MYCs versus ERF/ORA59 (Fig. 5). However, additional antagonistic interactions also originate from the backbone of the JA-mediated signalling cascade that intersects the two pathways. Furthermore, a bioinformatic approach with more than 300 publicly available microarray datasets on the co-expression of ET, JA and SA biosynthesis and signalling components illustrated the signal transduction network intersecting these hormones in plant defence (van Verk *et al.*, 2011). In nature, plants are attacked simultaneously or subsequently by biotrophic or necrotrophic pathogens or by sucking or chewing insects. Consequently, the cross-talk between various signalling components becomes increasingly important. Plants survive in response to interactions between multiple attackers by prioritizing a specific signalling pathway and rewiring the hormone signalling network. Necrotrophic pathogens, such as *B. cinerea*, have evolved strategies to overcome the host defence system by negotiating the SA–JA cross-talk. Upon infection of tomato, they release β-(1,3)-(1,6) glucan, an exopolysaccharide, that activates the SA–NPR1 pathway, but simultaneously suppresses the JA pathway required for acquiring the host resistance (El Oirdi *et al.*, 2011). *Pseudomonas syringae* represents an exceptional example of how pathogens hijack hormone-regulated host signalling cascade. This biotrophic pathogen is able to form the plasmid-encoded bacterial toxin coronatine, which is highly active as a molecular mimic of JA-Ile (see sections 2.6 and 4). Biotrophic pathogens such as *P. syringae* are defended via the SA-NPR1-TGA signalling cascade, but its simultaneous injection of toxins, such as coronatine or bacterial effector proteins into the host, alters the homoeostasis of JA and other hormones such as ABA and auxin, leading to the suppression of host immunity (Pieterse *et al.*, 2012). Recent results show that MYC2-mediated activation of the TFs, such as ANAC019, ANAC055 and ANC972, leads to stomata reopening allowing an entry of *P. syringae* (Zheng *et al.*, 2012). This provides a mechanistic explanation for coronatine-induced increase in the virulence of *P. syringae*. Coronatine acting preferentially by suppression of SA signalling (Pieterse *et al.*, 2012) is a multifunctional defence suppressor that also suppresses SA-independent signalling leading to callose deposition and even promotes bacterial growth in a COI1-independent manner (Geng *et al.*, 2012).

Additional oxylipins, such as 9-LOX and α-DOX products, are also involved in conferring resistance against biotrophic pathogens via JA signalling (Vicente *et al.*, 2012). It has been shown that plants produce N-acylamides that confer resistance to necrotrophic pathogens by activating JA biosynthesis and signalling (Méndez-Bravo *et al.*, 2011). Interestingly, arachidonic acid (AA), the counterpart of the JA precursor α-LeA occurring in metazoan species but not in plants, is perceived by plants and acts through an increase in JA levels concomitantly with resistance to necrotrophic pathogens (Savchenko *et al.*, 2010). Obviously, AA is an evolutionarily conserved signalling molecule that acts in plants in response to stress similar to that in animal systems. However, the transport of AA from the pathogen into the plant is unknown.

7.2. Systemic signalling in pathogen defence

SAR has long been known to be induced upon primary infection of a plant. It is an inducible defence mechanism against pathogens established distal to the primarily infected organ. Another type of resistance is the ISR appearing in leaves upon colonization of roots by plant growth-promoting rhizobacteria and during arbuscular mycorrhization (see section 8). Systemic responses occur mainly as priming effects. Similar to systemic signalling upon herbivory (see section 6), mobile signals are involved in linking the local infection with the distal response. Initially, SA was assumed to act as a phloem-mobile systemic signal for inducing SAR. Grafting experiments using transgenic plants in which SA was degraded by a bacterial salicylate hydroxylase (*nahG* plants), however, argue against the role of SA as a mobile signal in SAR (Mur *et al.*, 1997).

The putative role of JA in SAR is controversial. There are several studies that reported increased JA levels in phloem exudates of systemic leaves, an SAR-induced systemic increase in the expression of JA biosynthesis genes and an attenuation of pathogen-induced SAR in JA biosynthetic and signalling mutants when challenged with an avirulent strain of *P. syringae* (Truman *et al.*, 2007; Chaturvedi *et al.*, 2008). These all point to a definitive role of JA in SAR. On the other hand, several studies could not detect a role of JA in SAR (Mishina and Zeier, 2007; Attaran *et al.*, 2009). However, note that the dose of SAR-inducing pathogens was remarkably different in the two sets of experiments, without taking into account the impact of the hypersensitive response (HR; Shah, 2009). Possibly, JA is conditionally required to induce SAR depending on whether HR is involved or not (Shah, 2009).

The establishment of SAR is much more complex than previously suggested. At present, methyl salicylate (MeSA) is regarded as a critical mobile signal (Park *et al.*, 2007). Its activity seems to depend on the balance between light-dependent formation of SA and MeSA and interactions with additional compounds, such as azelaic acid (Jung *et al.*, 2009), the abietane diterpenoid dehydroabietanal (DA), the lipid transfer protein DIR1 and pipecolic acid (reviewed by Dempsey and Klessig, 2012). Once these signals are transported to systemic

leaves, they may synergistically interact to induce SAR via NPR1.

8. JASMONATES IN SYMBIOTIC INTERACTIONS

Mutualistic symbioses are important in nature and sustainable agriculture, the most important of which are the almost ubiquitously occurring arbuscular mycorrhiza (AM) and root nodule symbiosis (RNS). Whereas the former entails an association with obligate biotrophic fungi of the phylum Glomeromycota (Schüssler *et al.*, 2001), the latter is the interaction of the leguminous roots with nitrogen-fixing bacteria. In these two forms of intracellular (endo)symbioses, the heterotrophically growing microbial partners are accommodated within living root cells (for a review see Oldroyd *et al.*, 2009). The establishment and maintenance of both symbioses require plant resources, such as photosynthetically assimilated carbon. In turn, the respective microbial partners deliver mineral nutrients, mainly phosphate and nitrate in the case of AM and RNS, respectively. Both mutualistic interactions are based on a complex molecular cross-talk between the plant and the microsymbiont (Bonfante and Genre, 2010; Gough and Cullimore, 2011; Geurts *et al.*, 2012). This cross-talk involves recognition of the partners, establishment of mutualistic interactions, regulation of nutrient exchange and maintenance of mutualism. The role of jasmonates in these processes has been intensively studied and reviewed over the last two decades (Ludwig-Müller, 2000; Pozo *et al.*, 2005; Hause *et al.*, 2007; Pozo and Azcon-Aguilar, 2007; van Wees *et al.*, 2008; Hause and Schaarschmidt, 2009; Gutjahr and Paszkowski, 2009; Mortier *et al.*, 2012). Here, we focus on new data showing the involvement of jasmonates in AM and RNS, with a reference to plant interactions with the mutualistic endophyte *Piriformospora indica*.

8.1. Arbuscular mycorrhiza (AM)

AM are the most common type of mycorrhiza (Smith and Read, 2008). They originated more than 400 million years ago and enabled plants to colonize the land (Brundrett, 2002). Today, this mutualistic interaction is still very common among land plants. About 80 % of plants can interact with the AM fungi (Smith and Read, 2008). Besides supplying mineral nutrients and water, AM can improve the tolerance of plants to certain abiotic and biotic stressors, including drought, salt, heavy metals, and different pathogens and herbivorous insects (García-Garrido and Ocampo, 2002; Pozo and Azcon-Aguilar, 2007; Hartley and Gange, 2009). Therefore, AM are also regarded as inducers of ISR, as evident from plant interactions with plant growth-promoting rhizobacteria (PGPR) (Van der Ent *et al.*, 2009). ISR is induced by non-pathogenic microbes upon interaction with plant roots and confers a broad spectrum of effectiveness in many plant species (Van Wees *et al.*, 2008; Pineda *et al.*, 2010). JA is a central player in mediating ISR, which is depressed in JA biosynthesis and signalling mutants (Van der Ent *et al.*, 2009). Moreover, it has been demonstrated that ISR is based on priming of JA-regulated responses by increasing the sensitivity to JA rather than the production of JA (Pieterse *et al.*, 2002). Similarly to the ISR induced by rhizobacteria, JA might trigger increased local or systemic resistance of the AM plants against pathogens (Pozo and Azcon-Aguilar, 2007; Gutjahr and Paszkowski, 2009; Hause and Schaarschmidt, 2009).

JA seems to be involved in the establishment and maintenance of AM, but the results are partially contradictory. The AM roots exhibit enhanced JA levels accompanied by the expression of JA-induced genes and genes encoding enzymes of JA biosynthesis (Hause *et al.*, 2002; Isayenkov *et al.*, 2005; Lopéz-Ráez *et al.*, 2010). In tomato, however, mycorrhization led also to an accumulation of oxylipins derived from the 9-LOX pathway in colonized parts of the root (Lopéz-Ráez *et al.*, 2010; León-Morcillo *et al.*, 2012). Here, a local induction of expression of *LOXA* and *AOS3* occurred. Both genes are induced by JA (García-Garrido *et al.*, 2010; León-Morcillo *et al.*, 2012), implying that it might control the spread of the fungus via 9-LOX-derived oxylipins. This is supported by the fact that application of JA to mycorrhizal plants results in a diminished mycorrhization rate (Vierheilig, 2004; Herrera-Medina *et al.*, 2008). However, other data show that JA application is associated with an enhanced plant–fungus interaction (Tejeda-Sartorius *et al.*, 2008; Kiers *et al.*, 2010; Landgraf *et al.*, 2012; León-Morcillo *et al.*, 2012). Most probably, these contrasting data accrue from differences in the experimental designs, such as JA concentrations, timing and frequency of JA application, plant organs treated and plant nutritional status. Even the analyses of mycorrhization in mutants and transgenic plants defective in JA biosynthesis or perception did not yield unequivocal results. In comparison with tomato wild-type plants, an increase of mycorrhization occurred in the JA-insensitive mutant *jai1*, suggesting that JA may control the fungal spread (Herrera-Medina *et al.*, 2008; León-Morcillo *et al.*, 2012). In contrast, JA-deficient mutants *spr2* and *def1* were characterized by a decrease (Tejeda-Sartorius *et al.*, 2008; León-Morcillo *et al.*, 2012), while the *overexpressors of prosystemin* (enhanced JA levels) mutants by an increase of mycorrhization (Tejeda-Sartorius *et al.*, 2008), supporting the assumption that JA may act as a positive regulator of AM (Fig. 7). Further support for this assumption came from *AOC-RNA*i *Medicago truncatula* roots with reduced JA biosynthesis showing a significant decrease in mycorrhization (Isayenkov *et al.*, 2005). Moreover, an endogenous rise in JA levels induced by repeatedly wounding the leaves of *M. truncatula* led to enhanced mycorrhization (Landgraf *et al.*, 2012).

Comparing all data obtained from analyses of mutants and transgenic plants, the positive role of JA in AM can be explained by a systemic signalling to and from the shoot. Jasmonates produced in the roots by AM might result in systemic alterations in the shoot, which in turn might enforce the AM in the root. When JA levels are artificially increased in roots and shoots (JA application, *Prosystemin* overexpression, wounding), this reinforcement of AM might be increased. In the absence of JA biosynthesis in roots or shoots, the systemic root-to-shoot signals in either direction are missing, resulting in a decline in AM. However, how the shoot supports AM remains to be elucidated, but the role of JA may be attributed to an enhanced allocation of assimilates into the root system, as shown for other wounded or herbivore-affected plants (Babst *et al.*, 2005; Schwachtje *et al.*, 2006; Kaplan *et al.*, 2008). Accordingly, mycorrhization was found to be positively

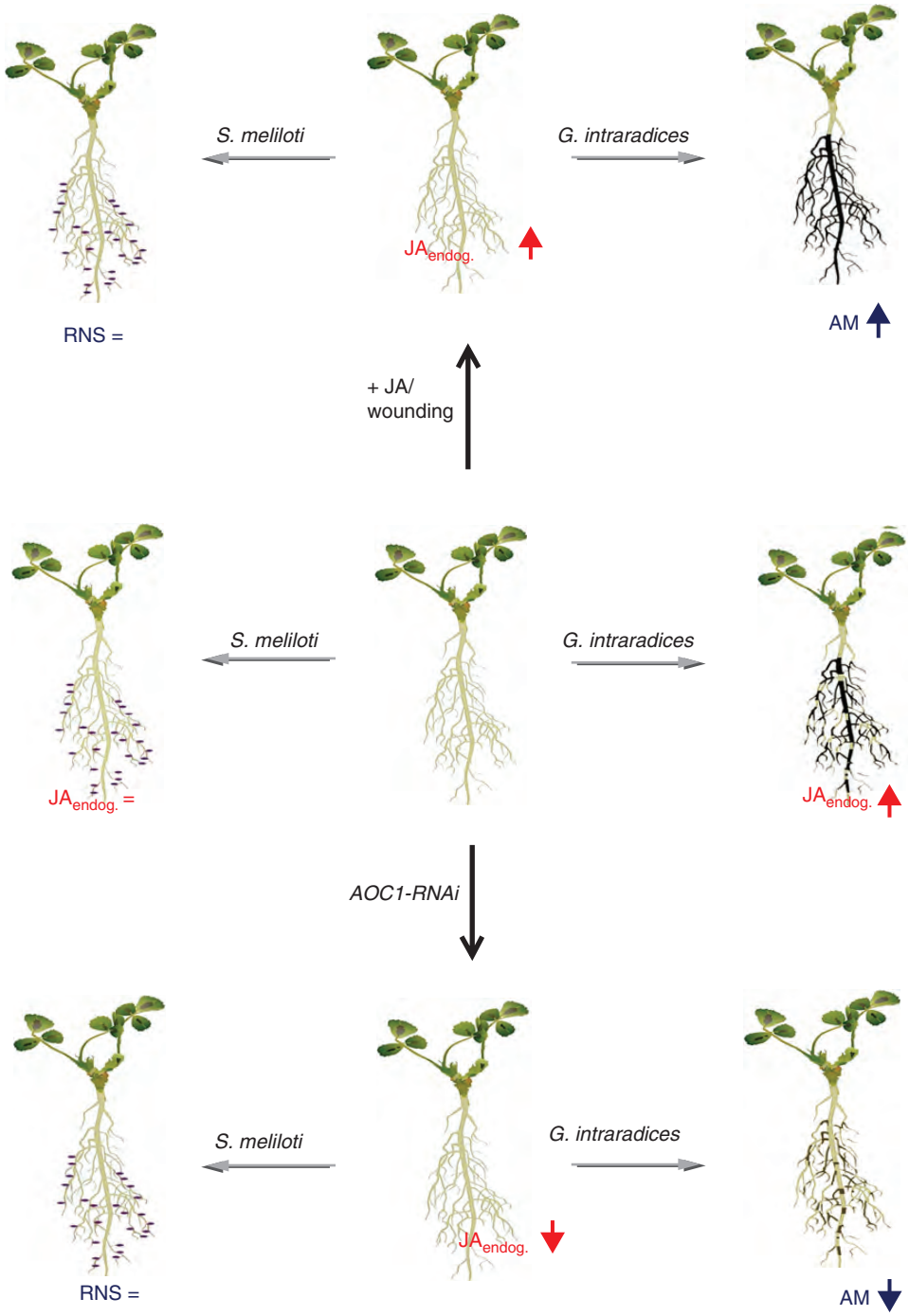

FIG. 7. Effects of arbuscular mycorrhization (AM) and root nodule symbiosis (RNS) on endogenous levels of jasmonates as well as the effects of modulated JA levels on AM and RNS in *Medicago truncatula*. Endogenous jasmonate levels are increased and remain unaffected in roots of AM and RNS plants, respectively. The application of jasmonic acid (JA) or wound-induced rise in JA results in enhanced AM, but no effects on RNS. Similarly, reduced JA levels in transformed *ALLENE OXIDE CYCLASE* (*AOC*)-*RNA*i roots suppress AM, but do not affect RNS.

correlated with deregulated carbon (Tejeda-Sartorius *et al.*, 2008). Additionally, JA may influence the action of other hormones (see section 4.7), because ET, ABA, gibberellin and auxin are involved in the regulation of AM (Martín Rodriguez *et al.*, 2010; García-Garrido *et al.*, 2010; de Los Santos *et al.*, 2011; Hanlon and Coenen, 2011; Ortu *et al.*, 2012). A deregulation of the cross-talk between JA and other hormones might occur in JA-insensitive plants, thus resulting in an enhanced mycorrhization.

8.2. Root nodule symbiosis (RNS)

RNS is characterized by the intracellular uptake of nitrogen-fixing bacteria (rhizobia) concomitant with the formation of

specialized organs, the root nodules. Specialized Gram-negative bacteria involved in RNS show a narrow host range and exclusively affect legumes. Among them are important agricultural crops such as soybean (*Glycine max*), common bean (*Phaseolus vulgaris*) and pea (*Pisum sativum*) (Kistner and Parniske, 2002). Root nodules provide a suitable microenvironment for nitrogenase activity of nitrogen-fixating bacteria and for protected and controlled development of a high-density bacterial population to maintain the symbiosis.

The application of JA to roots affects RNS in several ways (for reviews see Ding and Oldroyd, 2009; Gutjahr and Paszkowski, 2009; Hause *et al.*, 2009). On the one hand, JA application to rhizobia causes induction of *nod* genes (Rosas *et al.*, 1998; Mabood *et al.*, 2006), thereby enhancing the effectiveness of subsequent root nodulation. On the other hand, JA application has been shown to affect the response of *M. truncatula* to rhizobial Nod factors by inhibiting the Nod factor-induced transcription of *ENOD11* and *RIP1* and calcium spiking, leading to decreased numbers of nodules (Miwa *et al.*, 2006; Sun *et al.*, 2006). In soybean, JA or OPDA application affects the morphology of nodules by changing the number and size of central and peripheral nodule cells (Costanzo *et al.*, 2012). However, all these pharmacological experiments do not demonstrate an endogenous role of JA in RNS.

Endogenous levels of JA in nodulated roots do not differ from that in non-infected roots (Zdyb *et al.*, 2011). Moreover, no differences in nodule morphology and number were oberserved in transgenic *M. truncatula* roots with altered JA biosynthesis (Zdyb *et al.*, 2011) (Fig. 7). However, transient transformation of roots resulted in chimeric plants, an observation that could not entirely preclude a possible role of shoot-derived JA in RNS.

Results documenting the role of shoot-derived JA on nodulation are again controversial: (1) JA could act as a negative regulator because MeJA application to *Lotus japonicus* shoots is known to reduce nodulation (Nakagawa and Kawaguchi, 2006; Seo *et al.*, 2007); (2) JA could act as a positive regulator, as shoot-specific suppression of JA biosynthesis in soybean plants by foliar application of the inhibitor propyl gallate severely reduces nodule number without affecting root growth (Kinkema and Gresshoff, 2008); and (3) JA does not regulate RNS as enhanced JA levels in *M. truncatula* after wounding and JA application have been shown not to alter nodulation (Landgraf *et al.*, 2012) (Fig. 7). The use of different plant species and growth conditions might have produced such conflicting results. However, under light-limiting conditions, JA seems to have a regulatory function (see section 10). In *L. japonicas*, nodulation is photomorphogenetically controlled via JA, as demonstrated by the reduced nodule number in *phyB* mutants (Suzuki *et al.*, 2011). In wild-type plants, PHYB is part of a monitoring system to detect suboptimal light conditions and mediates the initiation of SAS as well as the suppression of nodule development under low R/FR light. Consequently, *phyB* mutants exhibit a constitutive SAS phenotype under white light. Interestingly, in low R/FR light-grown wild-type and white light-grown *phyB* plants, transcript levels of JA-induced genes, such as *JAR1*, are down-regulated, resulting in a decrease of JA-Ile content. Here, two effects – roots do not synthesize JA-Ile due to down-regulation of *JAR1* and the translocation of JA-Ile from shoot to root is probably impeded – seem to be involved (Shigeyama *et al.*, 2012). It is thus clear that shoot-derived JA-Ile controls nodule formation in the SAS as a positive regulator.

Another systemic effect in RNS is the 'autoregulation of nodulation' (AON). To restrict microbial infections and thereby nodule number, a feedback inhibition occurs, which is controlled by the shoot through CLAVATA1(CLV1)-like receptor kinase (NARK in soybean, HAR1 in *L. japonicus* and SUNN in *M. truncatula*) (for reviews see Hause *et al.*, 2009; Reid *et al.*, 2011; Mortier *et al.*, 2012). Mutant plants with a defective receptor kinase have a supernodulating phenotype. Although the nature of the root-derived AON signal is still elusive, CLV3/ESR-related (CLE) peptides binding to the receptor kinase might be involved (Mortier *et al.*, 2012). This signal is transmitted into the formation of an unknown 'shoot-derived inhibitor' (SDI) that is transferred back to the roots. Transcript profiling in soybean identified the components of AON acting downstream of NARK in the leaf (Seo *et al.*, 2007; Kinkema and Gresshoff, 2008). Of these, *AOC* and *MYC2* were found to be systemically down-regulated when roots of wild-type, not the *nark* mutant, were inoculated. Moreover, AON mutants exhibit enhanced levels of JA in leaves (Seo *et al.*, 2007; Kinkema and Gresshoff, 2008). Together with the fact that application of JA biosynthesis inhibitors also reduces nodulation in *nark*, these data suggest that JA signalling is a positive regulator of RNS and might suppress activity of the SDI (Kinkema and Gresshoff, 2008).

8.3. JA in plant interactions with Piriformospora indica

P. indica has been characterized as a mutualistic, biotrophically living endophyte which colonizes plant roots without causing any disease symptoms (for a review see Qiang *et al.*, 2012). It is a Basidiomycete, belongs to the order Sebacinales (Weiss *et al.*, 2004) and can be cultivated in axenic culture (Lahrmann and Zuccaro, 2012). *P. indica* is highly effective in root colonization accompanied by immune suppression (Schäfer *et al.*, 2009; Jacobs *et al.*, 2011). It colonizes a broad range of hosts, where it confers significant growth promotion including enhanced seed yield (Franken, 2012). The interaction between plants and *P. indica* is mutualistic – *P. indica* enhances the phosphate supply of plants depending on its phosphate transporters (Yadav *et al.*, 2010) and receives carbohydrates from the plant (Schäfer *et al.*, 2009). Additionally, colonization of roots by *P. indica* induces enhanced resistance against a wide variety of root and leaf pathogens (Molitor and Kogel, 2009). This is similar to ISR and depends on an operative JA pathway as the mutants *jar1* and *jin1*/*myc2* are compromised in *P. indica*-mediated resistance (Stein *et al.*, 2008). In barley, a *P. indica*-mediated priming leads to enhanced PR and heat-shock gene expression after infection of leaves with powdery mildew (Molitor *et al.*, 2011).

The colonization of roots by *P. indica* itself is regulated by JA. Mutants, which are impaired in JA biosynthesis or perception, show elevated root immune responses leading to reduced root colonization (Jacobs *et al.*, 2011). This led to the assumption that JA regulates early immune responses by suppression of SA- and glucosinolate-related defence pathways (Jacobs *et al.*, 2011). Indeed, Arabidopsis mutants impaired in SA-associated defence are more susceptible to *P. indica*. Moreover, there might be a cross-talk with ET, GA and cytokinin (CK). ET

biosynthesis, signalling and ET-targeted TFs are required for colonization and the beneficial effects of *P. indica* in barley and *A. thaliana* (Camehl *et al.*, 2010; Khatabi *et al.*, 2012). The colonization of barley roots depends on GA as its biosynthesis and perception mutants are significantly less colonized (Schäfer *et al.*, 2009). Moreover, *P. indica* is able to produce auxin and CK, but not JA or ABA (Sirrenberg *et al.*, 2007; Vadassery *et al.*, 2008), and therefore this mutualistic endophyte might recruit additional plant hormones to manipulate plant defence and development (Lahrmann and Zuccaro, 2012).

9. JASMONATE IN PLANT GROWTH AND DEVELOPMENT

9.1. Seed germination

Inhibition of seed germination was described for JA. However, recent genetic and biochemical evidence has shown that OPDA is the inhibitory compound which acts together with ABA in a COI1-independent manner (Dave *et al.*, 2011) (see section 4.4).

9.2. Root growth inhibition by JA

Growth inhibition and senescence promotion were the first two physiological responses described for JA (Ueda and Kato, 1980; Dathe *et al.*, 1981). Root growth inhibition by JA application has been used in mutant screens since the 1990s. The first mutant insensitive to JA was *jar1* (Staswick *et al.*, 1992). Subsequently, *JAR1* was cloned as JA-Ile synthase (Staswick and Tiryaki, 2004). Root growth inhibition by JA was also strongly supported by short-root phenotype of mutants with constitutive elevation of JA levels, such as *cev1* (Ellis *et al.*, 2002), and reduced sensitivity to JA in *coi1* and *myc2* mutants (Xie *et al.*, 1998; Lorenzo *et al.*, 2004). For inhibition of root growth, JA requires COI1, as indicated by the JA-unresponsiveness of the *coi1* mutant. However, ET and its precursor ACC, which occurs only in the light but not in the dark, are also known to inhibit root growth (Adams and Turner, 2010). The ACC/ET-induced root growth inhibition is light- and COI1-dependent, but JA-independent.

However, JA-induced root growth inhibition needs to be analysed in relation to other factors controlling the complex process of root development (Petricka *et al.*, 2012*b*; Ubeda-Tomás *et al.*, 2012). Initially, cell- and tissue-specific gene expression maps revealed non-overlapping areas of auxin-, GA- and JA-dependent gene expression (Birnbaum *et al.*, 2003). JA-induced gene expression appeared in outer layers of roots. But such expression data have to be taken into account cautiously, as the cellular proteome map of *A. thaliana* roots indicated a positive but weak correlation between protein and RNA profiles (Petricka *et al.*, 2012*a*). Meanwhile, system biology approaches are being used to analyse the complex and hierarchical link between hormonal and mechanic signalling in root growth (Band *et al.*, 2012*a*). Key players are CK, GA and auxin. However, the cross-talk with other hormones, such as auxin, GA and BR, underlying JA perception and signalling indicates the involvement of JA in root growth. The outcome of root growth is an integration of hormonal and mechanic signalling that affect cell division, membrane traffic, cell-wall loosening and synthesis, turgor, and growth rate (Band *et al.*, 2012*b*). Many of these processes could be a direct effect of JA or an indirect effect of JA via auxin. The biosynthesis of auxin, the key player in root growth, is affected by JA-induced expression of *ASA1* (Sun *et al.*, 2009) (Fig. 8). JA induces auxin redistribution via modulation of endocytosis and an accumulation of PIN2 in the plasma membrane (Sun *et al.*, 2011*b*). Auxin is also affected by JA-induced MYC2-dependent repression of *PLETHORA*, the key player in root stem cell niche activity (Chen *et al.*, 2011). Another example of auxin/JA cross-talk is given by the *axr1* mutant defective in an SCF-complex component required for auxin signalling (Mockaitis and Estelle, 2008). This mutant shows reduced root growth inhibition by MeJA, indicating that the AXR1-dependent modification of the CULLIN1 subunit of the SCF^{COI1} complex is required for JA/JA-Ile signalling (Xu *et al.*, 2002).

Taken together, the JA-induced root growth inhibition seems to occur preferentially via modulation of the effects of auxin in root growth and development (Fig. 8).

9.3. Lateral root formation

Most of the Arabidopsis lateral root mutants are affected in auxin homeostasis, signalling and transport and in PINs (Péret *et al.*, 2009), thereby indicating the dominant role of auxin in lateral root formation (Petricka *et al.*, 2012*b*). The various possibilities for cross-talk between JA and auxin as described above strongly suggest a role of JA in lateral root formation. The JA-insensitive mutant *coi1-16* that produces fewer lateral roots lends further credence to this idea. Furthermore, the high promoter activities of *AtAOC3* and *AtAOC4* in emerging lateral roots suggest that JA is involved in lateral root formation (Stenzel *et al.*, 2012). It has been shown that lateral root formation is induced by auxin, but is inhibited by the conjugate of JA with tryptophan (Staswick, 2009).

9.4. Adventitious root formation

Adventitious roots are formed naturally or induced by environmental stimuli in aerial organs. Like root growth, adventitious root formation is a complex process regulated by hormones and environmental factors. Auxin is a positive regulator mediated by ARF6 and ARF8, which are targets of miR167 (Gutierrez *et al.*, 2012). Interestingly, downstream of this auxin-induced adventitious root formation, there is a negative COI1- and MYC2-dependent regulation via altered JA/JA-Ile homeostasis. Whereas JAR1, which is the GH3.11 of the *GH3* gene family of conjugating enzymes, generates JA-Ile, the other members (*GH3.3, GH3.5, GH3.6*) conjugate Asp, Met and Trp with JA. The triple-mutant of these genes has fewer adventitious roots and increased expression of JA biosynthesis genes, whereas mutants impaired in JA perception and signalling, such as *coi1-16*, *myc2*, *myc3*, *myc4* and *jar1* form far more adventitious roots than the wild-type (Gutierrez *et al.*, 2012). These data are in agreement with auxin–JA cross-talk that occurs during adventitious root formation. Here, the positive regulatory effects of ARF6 and ARF8 are increased by the *GH3.3, GH3.5, GH3.6* module that attenuates the negative regulatory effect of JA/JA-lle via

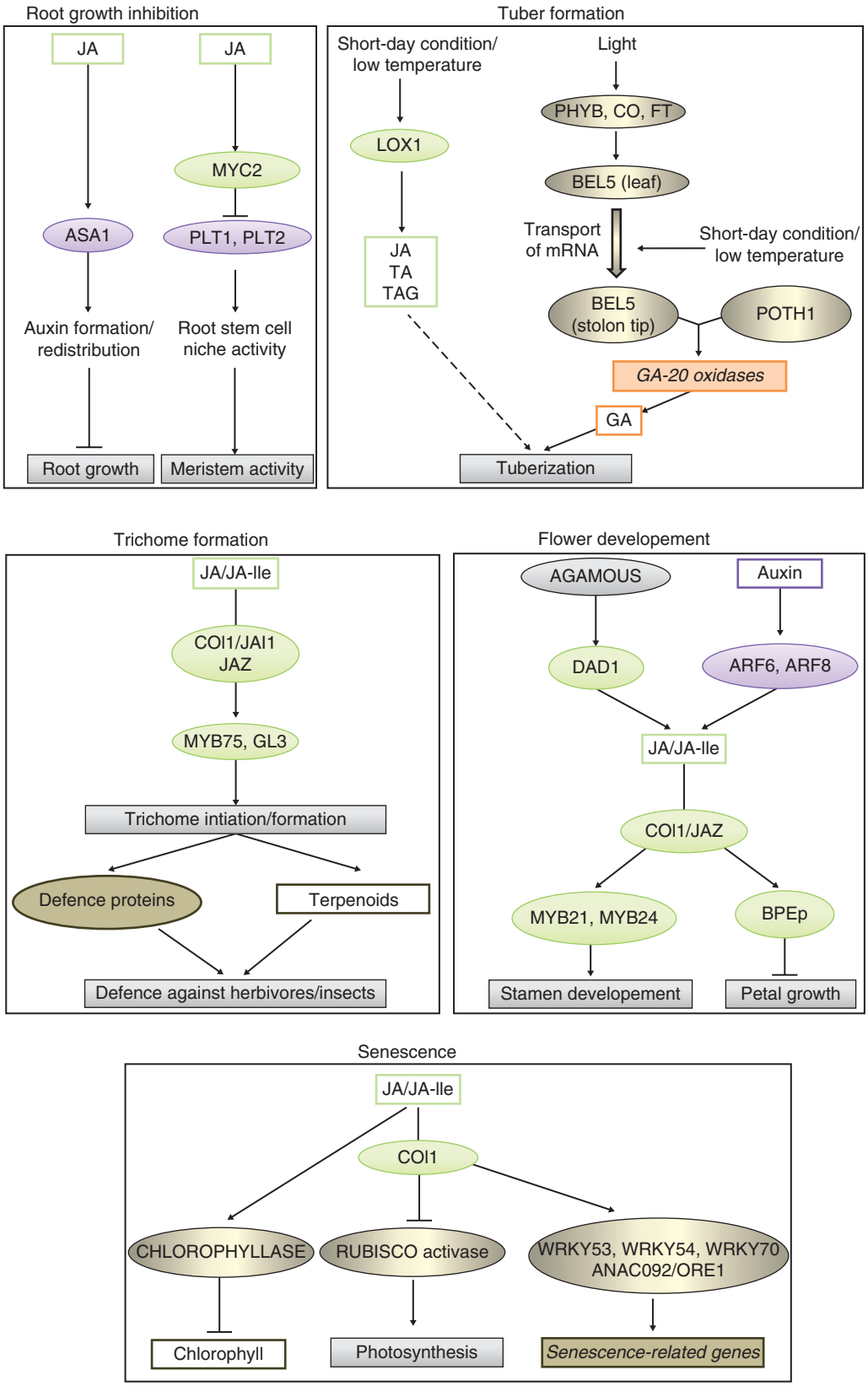

FIG. 8. The role of jasmonic acid (JA)/JA-Ile in plant development. JA induces root growth inhibition by stimulating auxin biosynthesis via anthranilate synthase α1 (ASA1) and inhibiting the expression of genes encoding the TFs PLETHORA 1 (PLT1) and PLT2, which ensure the maintenance and activity of stem cells in the root. In tuber formation, jasmonates [JA, tuberonic acid (TA) and TA glucoside (TAG)] might act directly after their rise following activity of LIPOXYGENASE 1 (LOX1).

conjugation of JA to JA-Asp, JA-Met and JA-Trp (Gutierrez *et al.*, 2012). However, this is in opposition to the positive regulatory effects of ARF6 and ARF8 on JA biosynthesis, as recorded in filament elongation during flower development (see section 4.7) (Nagpal *et al.*, 2005; Reeves *et al.*, 2012). Moreover, in leafy cuttings of *Petunia hybrida*, an increase in JA levels precedes a corresponding increase in auxin levels (Ahkami *et al.*, 2009). However, whether this increase in JA levels is essential for subsequent adventitious rooting remains to be elucidated.

9.5. Tuber formation

For a long time, tuber-inducing activities of jasmonates, particularly 12-OH-JA (TA) and its glucoside (TAG), have been suggested (reviewed by Wasternack and Hause, 2002). StLOX-1 was shown to be involved in tuber yield and tuber formation (Kolomiets *et al.*, 2001), and LOX-derived metabolites such as JA, TA and TAG accumulate at low, tuber-inducing temperature (Nam *et al.*, 2008). There were, however, only correlative data on the endogenous content of jasmonates in stolons and tuber formation. The cloning of a 12-OH-JA sulfotransferase from *A. thaliana* and tomato (Gidda *et al.*, 2003; J. Heise and C. Wasternack, unpubl. res.) and the occurrence of 12-OH-JA, 12-HSO_4-JA and 12-*O*-Glc-JA in different non-tuber-bearing plant species (Miersch *et al.*, 2008) argue against a specific role of jasmonates in tuber formation. Possibly, tuber-inducing effects might be caused indirectly via cell expansion in stolons accompanied by changes in microtubule orientation (Abe *et al.*, 1990), because JA biosynthesis may occur in developing stolons (Cenzano *et al.*, 2007).

Multiple pathways are involved in tuber formation (Sarkar, 2008) (Fig. 8). Besides hormonal control, tuberization in potato is strictly photoperiod-dependent. Low night temperature and a short-day photoperiod produce a systemic signal in leaves which induces tubers in roots. Tuberization depends on conserved function of the potato orthologue of CONSTANS (CO) and FLOWERING LOCUS T (FT) (Rodríguez-Falcón *et al.*, 2006). Both of them are key players in flower induction. Phytochrome B-mediated photoperiodic control of tuberization is well described (Rodríguez-Falcón *et al.*, 2006). Identification of the BEL5 TF of potato shed new light on the regulation of tuber formation. This TF belongs to the homeobox *BEL1* gene family that is involved in different developmental processes. Its mRNA accumulates under short-day conditions in leaves and is transported via the phloem to stolon tips correlating with tuber formation (Banerjee *et al.*, 2006). Additionally, high *StBEL5* promoter activity appears in stolons of short-day plants (Chatterjee *et al.*, 2007). *StBEL5* mRNA accumulation seems to result from the photoperiod-dependent control via CO and FT and is a long-distance signal with increased mobility mediated by its 3′ untranslated region (Hannapel, 2010). The final step in regulation of tuber formation is an altered GA level (Jackson *et al.*, 1996). StBEL5 binds together with the potato KNOX gene product POTH1 to promoter sequences of the gene encoding the GA-20 oxidase1, which leads to its repression and altered GA level that affect tuber formation and other aspects of vegetative development (Banerjee *et al.*, 2006; Lin *et al.*, 2013) (Fig. 8). Initiation of cell division in stolons by cytokinins is another hormonal control active in tuber formation (Xu *et al.*, 1998).

9.6. Growth versus defense

Besides the above discussed root growth inhibition by JA, growth of above-ground plant parts is also inhibited by JA. Any growth response depends on cell division reflecting the cell cycle activity and on cell expansion mediated by macromolecule formation, ploidy-dependent cell growth, cell-wall elasticity, microtubule organization and turgor pressure (Rymen and Sugimoto, 2012). All these processes are under hormonal control and depend on environmental cues such as biotic and abiotic stresses that are known to suppress the growth of above-ground plant parts. Plant hormones such as auxin, ET and GA have been shown to be involved in such stress-induced growth inhibition (Band *et al.*, 2012*a*; Murray *et al.*, 2012; Petricka *et al.*, 2012*b*). JA is the key player in responses to herbivores and mechanical wounding, both of which are known to repress plant growth in *A. thaliana* (Zhang and Turner, 2008). Endogenously generated JA, but not OPDA, has been shown to repress plant growth by suppressing mitosis in a COI1/JAZ/MYC2-dependent manner. This accords well with a JA-induced reprogramming of the expression of cell cycle-regulated genes in a COI1/JAZ/MYC2-dependent manner (Pauwels *et al.*, 2008). A similar effect was observed when *M. truncatula* plants were mechano-stimulated by repeatedly touching their leaves, which resulted in an increase of endogenous JA levels concomitant with stunted growth (Tretner *et al.*, 2008). The same experimental setup has been used recently in *A. thaliana* for analysing touch-induced morphogenesis, which enhances resistance to *B. cinerea* in a JA-dependent manner (Chehab *et al.*, 2012). Even some aspects of thigmomorphogenesis are ET-dependent; the ET-response mutants show touch-induced thigmomorphogenesis. Mutant analysis revealed a JA-mediated signalling pathway underlying thigmomorphogenesis (Chehab *et al.*, 2012). In contrast to the negative effect of JA and mechano-stimulation on longitudinal growth, a positive effect of mechano-inducible COI1/MYC2/JAZ during secondary growth in cambium formation has been reported (Sehr *et al.*, 2010).

Growth is promoted by GAs that, however, repress defence gene activation (Fig. 6). These antagonistic responses are caused by an imbalanced ratio of GA and JA (see Kazan and Manners, 2012). In the absence of JA, the formation of GA is

However, most importantly levels of gibberellic acid (GA) are regulated by the combined action of the TFs BEL1-like 5 (BEL5) and POTATO HOMEOBOX 1 (POTH1) at the promoters of GA-20 oxidase-encoding genes. In trichome initialization, JA/JA-Ile act via the COI1 co-receptor complex to activate the trichome-specific TFs MYB75 and GLABRA 3 (GL3), leading to formation of defence proteins as well as terpenoids. Role of jasmonates in flower development is depicted for *Arabidopsis thaliana*. The TF AGAMOUS activates the phospholipase A_1 DAD1, but also auxin induces rise in JA/JA-Ile via the function of the TFs AUXIN RESPONSE FACTOR 6 (ARF6) and ARF8. Jasmonates induce COI1-dependently expression of MYB21 and MYB24, leading to proper stamen development, whereas expression of the TF BIGPETALp (BPEp) restricts petal growth. In senescence, jasmonates act on different levels. On the one hand, chlorophyllase is activated, which leads to chlorophyll breakdown, and RUBISCO activase is inhibited, which switches off photosynthesis. On the other hand, specific TFs, such as WRKY53, WRKY54, WRKY70 and ANAC092/ORE1, are induced, leading to expression of senescence-related genes.

associated with growth promotion and defence containment, while in the absence of GA, the formation of JA is accompanied by opposing responses. The already described proteins DELLA and PIF (for GA signalling) as well as JAZ and MYC (for JA signalling) are components of this GA–JA cross-talk (see section 4.7), where JA prioritizes defence over growth (Yang D-L *et al.*, 2012). Incidentally, cell elongation and meristem activity required for plant growth are regulated by auxins. Here, JA affects auxin formation and distribution by inducing the expression of *ASA1* and by regulating the PINs and *PLETHORA*, respectively (see sections 4.6 and 4.7).

9.7. Trichome formation

Glandular trichomes are multicellular and often involved in resistance to insects due to formation of terpenoids, flavonoids, alkaloids and defence proteins (Tian *et al.*, 2012). They represent a useful tool for production of secondary metabolites (Tissier, 2012). Genetic evidence for the involvement of JA in glandular trichome formation were obtained by characterizing the tomato homologue of COI1, the central component of JA perception (Li *et al.*, 2004) (Fig. 8). The corresponding tomato mutant *jai1* is female sterile, but is impaired in grandular trichome formation, trichome monoterpene content and spider mite resistance. Further support for the link between trichome formation, JA and defence came from the recessive tomato mutant *odorless-2* (*od-2*), which exhibits altered morphology, density and chemical composition of glandular trichomes (Kang *et al.*, 2010). Under natural field conditions, *od-2* plants were highly susceptible to Colorado potato beetle larvae and the solanaceous specialist *Manduca sexta*, indicating that trichome-borne compounds determine host plant selection under natural conditions (Kang *et al.*, 2010; Meldau *et al.*, 2012). Recently, an antagonism between herbivore-induced plant volatiles and trichome formation has been observed in tomato. Using the JA-deficient *spr2* mutant and the trichome-free JA-insensitive *jai1* mutant, preferential oviposition that was observed on trichome-free JA-insensitive plants indicated a greater impact of trichomes over volatile emission in this tritrophic interaction (Wei *et al.*, 2013). Furthermore, glandular and non-glandular trichomes are involved in defence against herbivores via trichome density and JA-inducible defence compounds, such as PI2, monoterpenes and sesquiterpenes (Tian *et al.*, 2012). It is to be noted here that the cotton fibre represents a special type of single-cell seed trichome. It is well known that its initiation and elongation are under hormonal control including JA. Recently, it has been shown that a member of the class I bHLH TF family of *Gossypium barbadense* positively regulates JA biosynthesis (Hao *et al.*, 2012). Consequently, the elevated JA level in cotton fibre activates downstream genes involved in Ca^{2+} signalling and ET biosynthesis. In Arabidopsis, targets of JAZ proteins are TFs such as MYB75, GL3 and EGL3, which are involved in anthocyanin biosynthesis and trichome initiation (Qi *et al.*, 2011) (see section 4.6). JA regulates trichome initiation in a dose-dependent manner via the key TF in trichome formation GL3 and its interaction with JAZ proteins (Yoshida *et al.*, 2009) (Fig. 8).

9.8. Leaf movement

There are several types of leaf movements. Among them are the upward leaf movement (hyponastic growth) and the leaf movement of nyctinastic plants such as *Albizzia*. Both of them are altered by JA compounds. Hyponastic growth is induced by ET, heat and low light intensity and is stimulated by JA but inhibited by SA (van Zanten *et al.*, 2012).

Nyctinastic leaf movement depends on activity of motor cells (see section 3.6). Here, TAG has a role. Among the enantiomeric forms of TAG, only one mediates activity of motor cells of nyctinastic plants, such as *Albizzia* and *Samanea saman* in a COI1-independent manner (Ueda and Nakamura, 2007; Nakamura *et al.*, 2011).

9.9. JA in leaf senescence

Leaf senescence is a complex developmental programme that depends on light/dark conditions, nutrients, biotic and abiotic stresses, and several hormones including JA. Over the last few years, several reviews on leaf senescence in relation to JA have been published (Reinbothe *et al.*, 2009; Guo and Gan, 2012; Zhang and Zhou, 2013). Therefore, only a few aspects will be discussed here. In *A. thaliana*, comparative large-scale transcript profiling between environmentally and developmentally regulated leaf senescence revealed only limited similarities in early stages, but showed convergence and divergence of gene expression profiles (Guo and Gan, 2012). High-resolution transcript profiling of senescing leaves identified a distinct group of TFs that link metabolic pathways, leaf development and senescence (Breeze *et al.*, 2011). The JA-linked TFs identified to be active in leaf senescence are WRKY53 (Miao and Zentgraf, 2007), WRKY54 and WRKY70 (Besseau *et al.*, 2012), and ANAC092/ORE1 (Balazadeh *et al.*, 2010) (Fig. 8). The F-box protein ORE9 was initially identified from a screen of ABA-, JA- and ET-induced senescence mutants (Woo *et al.*, 2001), but it was found to have different regulatory properties in photomorphogenesis, shoot branching (Stirnberg *et al.*, 2007) and cell death. Leaf senescence is characterized by JA-inducible chlorophyll breakdown. In *A. thaliana*, between the two key enzymes involved in chlorophyll degradation, the gene encoding the CHLOROPHYLLASE1 is strongly induced by JA (Tsuchiya *et al.*, 1999). Moreover, a mechanistic explanation for the senescence-promoting effects of JA in leaves was provided only recently. It has been shown that Rubisco-activase is down-regulated by JA in a COI1-dependent manner (Shan *et al.*, 2011).

9.10. Gravitropism

Gravitropism is a well-studied, morphogenic response, in which intra- and intercellular communication by auxin takes place. Traditionally, the Cholodny–Went hypothesis is used to explain the asymmetric growth as a consequence of auxin redistribution. Regarding the repeatedly discussed cross-talk between auxin and JA (see sections 4.7 and 9.2), it is not surprising that JA has a role in gravitropism (Gutjahr *et al.*, 2005). Using rice coleoptiles, the Cholodny–Went hypothesis was found to be true. In addition to an auxin gradient, gradients of JA and auxin responsiveness were found to be involved in gravitropism (Gutjahr *et al.*, 2005). A mechanistic framework

might be given by an interaction between auxin and JA signalling pathways. This became evident by identification of tryptophan conjugates of indolyl-3-acetic acid and JA as endogenous inhibitors of the gravitropic response, one of the most prominent auxin responses (Staswick, 2009). In rice a gravitropism-related gene, *LAZY1*, was identified that is required for gravity responses in leaf lamina, but not in roots (Yoshihara and Ino, 2007). The function of its gene product remains unknown.

9.11. JA in development of reproductive organs of dicotyledonous plants

The most diagnostic phenotype of Arabidopsis mutants impaired in JA biosynthesis and perception, such as *coi1*, *opr3*, *dde1*, *dde2*, *dad1*, *aos* and *fad3-2fad7-2fad8* (Browse, 2009*c*; Wilson *et al.*, 2011), is male sterility (Browse, 2009*a*, c). Three characteristic phenotypes were identified: (1) insufficient filament elongation, (2) non-viable pollen and (3) delayed anther dehiscence. In mutants impaired in JA biosynthesis, fertility can be restored by JA treatment when applied in stages 11 and 12 of floral development, but not by OPDA (Mandaokar *et al.*, 2006). Transcript profiling of JA-treated stamens of *opr3* plants allowed detection of stamen- and JA-specific mRNAs preferentially regulating genes involved in metabolic pathways required for the synthesis of terpenoid volatiles, wax and pollen constituents (Mandaokar *et al.*, 2006) (Fig. 8). Most interestingly, new TFs required for stamen development were identified in the stamen transcriptome of *opr3* plants. Among them were MYB21 and MYB24 (Mandaokar *et al.*, 2006). Subsequent genetic analysis identified MYB108, which, together with MYB24, is involved in JA-regulated stamen and pollen developments (Mandaokar and Browse, 2009). MYB21 and MYB24 were further identified as targets of JAZ repressors (Song *et al.*, 2011) (see section 4.6). In the *coi1* mutant, the overexpression of MYB21 could partially restore the delayed anther dehiscence, but not JA insensitivity in terms of root growth inhibition, anthocyanin formation and susceptibility to necrotrophic pathogens (Song *et al.*, 2011). These data suggest a dominant role of MYB21 in stamen and pollen development.

The essential role of JA in stamen development is also obvious by DAD1, an Arabidopsis PLA_1 involved in JA formation of flowers. *DAD1* is expressed in flowers, and *dad1* shows a phenotype similar to *coi1* (Ishiguro *et al.*, 2001). This gene is a target of the central TF AGAMOUS (Ito *et al.*, 2007) (see section 2.1). Of all the JA cross-talks involving other hormones, the JA–auxin cross-talk is the most important in flower development. It has been unequivocally demonstrated that ARF6 and ARF8 regulate JA biosynthesis in anther filaments (Nagpal *et al.*, 2005; Reeves *et al.*, 2012) (see section 4.7).

In contrast to the male sterile phenotype of *coi1*, the homologous tomato mutant *jai1* impaired in the tomato homologue of COI1 is female sterile, suggesting that JA signalling plays distinct roles in flower development in Arabidopsis and tomato (Li *et al.*, 2004). Recently, embryo development in tomato has been shown to be OPDA-specific, but JA-independent (Goetz *et al.*, 2012), implying the difference in flower development vis-à-vis fertility between Arabidopsis and tomato.

Beside its role in stamen development in Arabidopsis, JA has a role in petal growth (Brioudes *et al.*, 2009). The final stages of petal growth are largely dependent on cell proliferation and/or cell expansion. The bHLH TF BIGPETALp (BPEp), expression of which is controlled by JA, limits petal size by controlling cell expansion. Consequently, the *opr3 bpe-1* mutants are characterized by a larger petal size that can be restored by JA treatment (Brioudes *et al.*, 2009).

9.12. JA in development of reproductive organs of monocotyledonous plants

JA has a central role in sex determination of maize (Acosta *et al.*, 2009; Browse, 2009*b*). In maize, sex organs are located on the same plant in the male tassel at the top and the female ear(s). Originating from a bisexual floral meristem, the pistil primordia are aborted undergoing a *tasselseed*-mediated cell death (Acosta *et al.*, 2009). There are two *tasselseed* genes in maize, namely *ts1* and *ts2*. Whereas the *ts2* gene encodes a short-chain dehydrogenase/reductase with broad substrate specificity, the *ts1* gene has been identified recently by positional cloning and encodes a plastid-targeted 13-LOX (Acosta *et al.*, 2009). The homozygous *ts1* mutant is characterized by a loss of 13-LOX activity and lower JA levels in inflorescences, but the mutant phenotype could be rescued by JA application. TS1 and TS2 are both required for sex determination. Possibly, TS2 plays a role similar to TS1 in JA biosynthesis by regulating β-oxidation steps of the carboxylic acid side chain of OPDA (Acosta *et al.*, 2009; Browse, 2009*b*).

Cytoplasmic male sterility (CMS), a maternally inherited phenomenon leading to pollen abortion, is associated with JA biosynthesis. In rice, proteins of mitochondrial complexes together with a sex-determining TASSELSEED2-like protein were found to be affected in a CMS line YuetaiB, leading to aberrant changes in JA biosynthesis during microspore development (Liu *et al.*, 2012).

10. JA IN LIGHT SIGNALLING

The amount and quality of light sensed by plants is important in different developmental programmes such as skotomorphogenesis, photomorphogenesis and SAS. The molecular mechanism of light signalling and essential components of light perception and responses have been intensively studied over the last two decades (reviewed by Chory, 2010; Kami *et al.*, 2010; Lau and Deng, 2010). The involvement of plant hormones, such as auxins, cytokinins, GA, ET and BRs, in light-dependent regulation of developmental processes has been intensively studied (Wolters and Jürgens, 2009; Chory, 2010). However, the involvement of JA in light signalling has been described only over the last few years. The newly discovered key players in JA perception and signalling (JA receptor, JAZ proteins and MYC/MYB TFs) have provided new mechanistic insights into how JA and light are integrated in growth and development and how competition between growth and defence occurs. This interplay has been most authoritatively reviewed (Lau and Deng, 2010; Kazan and Manners, 2011). To avoid an overlap, we will discuss here only to some important aspects.

(1) The mutant *myc2* impaired in the key TFs of JA signalling pathways shows high sensitivity to SAS or FR light, whereas *phy A* mutant exhibits reduced JA-regulated growth inhibition (Robson *et al.*, 2010). The wound and shade avoidance responses are integrated via JAZ1.
(2) The mutant *hy1-101*, which is affected in a heme-oxygenase required for phytochrome chromophore biosynthesis, has an overproduction of JA concomitant with increased expression of JA-responsive genes (Zhai *et al.*, 2007).
(3) HY5, a bZIP TF, is a positive regulator of photomorphogenesis and a key regulator of light signalling (Lau and Deng, 2010). It binds to the *LOX3* promoter (Lee *et al.*, 2007) involved in JA biosynthesis (Caldelari *et al.*, 2011) (see section 2.2).
(4) Light responses are organ- and tissue-specific. Roots of phytochrome chromophore-deficient mutants, such as *hy1-1* and *hy2-1*, have reduced sensitivity to JA similar to the JA-insensitive mutants *jar1* and *myc2* (Costigan *et al.*, 2011), suggesting a photoregulation of root elongation via an acquisition of JA sensitivity.
(5) The CSN is clearly linked to JA biosynthesis and JA-dependent defence responses. Tomato plants with a silenced CSN subunit are less resistant to herbivorous insects and necrotrophic pathogens (Hind *et al.*, 2011).
(6) The positive regulator of PHY B-mediated SAS is PHYTOCHROME AND FLOWERING TIME1 (PFT1), which is an important regulator of JA signalling. PFT1 encodes the conserved MED25 subunit of the mediator complex active in JA signalling (see section 4.2). Possibly, the light signalling component PFT1/MED25 negotiates the activation of TFs, such as MYC2 and ERF1 and their binding to the RNA polymerase II apparatus (Kidd *et al.*, 2009; Çevik *et al.*, 2012). This integrates a variety of interdependent environmental stimuli, such as flowering time control via CONSTANS, light quality control and JA-dependent defence responses (Iñigo *et al.*, 2012).
(7) Light stress affects JA biosynthesis through plastid proteins called fibrillins (Youssef *et al.*, 2010; Kazan and Manners, 2011). Additionally, anthocyanin accumulation, the most prominent JA/JA-Ile-induced phenotype, is elevated by light stress.
(8) When plants are treated with JA and UV-B light, there occurs an overlap in gene expression resulting in stronger defence even under field conditions (Ballare *et al.*, 2012).
(9) R/FR light ratio causes weaker resistance to necrotrophic pathogens and decreased JA responses in a COI1–JAZ10-dependent but SA-independent manner (Cerrudo *et al.*, 2012).
(10) In rice, light regulates JA/JA-Ile biosynthesis via PHY-dependent up-regulation of *OsAOS1* and *OsJAR1* (reviewed by Svyatyna and Riemann, 2012).
(11) The secretion of extrafloral nectar in lima bean, a JA-dependent indirect defence mechanism, is controlled by light via the formation of JA-Ile irrespective of R/FR light ratio (Radhika *et al.*, 2010).
(12) Hyponastic growth (upward leaf movement), a component of SAS in *A. thaliana*, is an ET- and heat-controlled process, and is modulated by both JA and SA (van Zanten *et al.*, 2012).

11. CONCLUDING REMARKS

In the last decade, a vast amount of data has been accumulated by transcriptomic, proteomic, lipidomic and metabolomic studies. Many of them were performed to elucidate hormone action including that of jasmonates in a developmental or stress-related context. Such analyses will continue to expand by analytical and bioinformatic improvements, which will allow analysis of spatial and temporal hormone-induced changes at the level of organs, tissues and even specific cell types. Many new components or key players in hormone signalling are expected to be identified by these 'omics' studies.

To date, at the protein level, signalling components are analysed by interaction studies, such as yeast two-hybrid and three-hybrid screening and BiFC analyses *in vivo*. Technical improvements in combination with specialized genetic tools will allow us to address the complexity inherent in the plant hormone cross-talk. Another emerging issue will be to optimize sensitive methods for recording hormone homeostasis and detecting active and inactive hormones sustained by their metabolic conversion.

In the jasmonate field the following aspects will become of interest in the near future.

(1) The assembly of the SCF^{COI1}–JAZ co-receptor complex and stability of its components.
(2) A mechanistic insight into gene activation and repression by studying protein interactions between JA/JA-Ile signalling components involving JAZs and TFs.
(3) Epigenetic regulation of JA/JA-Ile signalling components.
(4) Mechanistic insights into the cross-talks between JA/JA-Ile and other hormones such as auxin, ABA or GA in a process-related context.
(5) Fine-mapping of JA, JA-Ile and OPDA levels as well as related metabolites at cells, tissue and organ levels in relation to external stimuli and developmental phases.
(6) Chemical and genetic screens to pick up new components involved in JA/JA-Ile formation and signalling.
(7) Translational and post-translational control mechanisms including protein phosphorylation that affect JA/JA-Ile-dependent processes.

However, a system biology approach will help understand many of these data and elucidate how hormone-dependent processes evolved during adaptation of plants to their environments.

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NOTE ADDED IN PROOF

Recently it was shown that COI1 is stabilized by SCF^{COI1} and degraded via the 26S proteasome (Yan *et al*., 2013, *The Plant Cell* **25**: 486–498). The SA suppression on JA biosynthesis and signalling was localized downstream of SCF^{COI1}–JAZ and includes the TF ORA59 (Van der Does *et al*., 2013, *The Plant Cell* **25**: 744–761).