Journal of Experimental Botany, Vol. 68, No. 6 pp. 1303–1321, 2017
doi:10.1093/jxb/erw443 Advance Access publication 10 December 2016

REVIEW PAPER

Jasmonates: biosynthesis, metabolism, and signaling by proteins activating and repressing transcription

Claus Wasternack[1,*,†] and Susheng Song[2]

[1] Laboratory of Growth Regulators, Centre of the Region Haná for Biotechnological and Agricultural Research, Palacký University, Institute of Experimental Botany AS CR, Šlechtitelů 11, CZ 78371 Olomouc, Czech Republic
[2] Beijing Key Laboratory of Plant Gene Resources and Biotechnology for Carbon Reduction and Environmental Improvement, College of Life Sciences, Capital Normal University, Beijing 100048, China

* Correspondence: cwastern@ipb-halle.de
† Permanent address: Leibniz Institute of Plant Biochemistry, Department of Molecular Signal Processing, D-06120 Halle (Saale), Weinberg 3, Germany.

Received 2 September 2016; Editorial decision 4 November 2016; Accepted 7 November 2016

Editor: Richard Napier, University of Warwick

Abstract

The lipid-derived phytohormone jasmonate (JA) regulates plant growth, development, secondary metabolism, defense against insect attack and pathogen infection, and tolerance to abiotic stresses such as wounding, UV light, salt, and drought. JA was first identified in 1962, and since the 1980s many studies have analyzed the physiological functions, biosynthesis, distribution, metabolism, perception, signaling, and crosstalk of JA, greatly expanding our knowledge of the hormone's action. In response to fluctuating environmental cues and transient endogenous signals, the occurrence of multilayered organization of biosynthesis and inactivation of JA, and activation and repression of the COI1–JAZ-based perception and signaling contributes to the fine-tuning of JA responses. This review describes the JA biosynthetic enzymes in terms of gene families, enzymatic activity, location and regulation, substrate specificity and products, the metabolic pathways in converting JA to activate or inactivate compounds, JA signaling in perception, and the co-existence of signaling activators and repressors.

Key words: Activators, amino acid conjugates, biosynthesis, jasmonic acid, metabolism, perception, repressors, SCF^{COI1}–JAZ co-receptor complex, signaling.

Introduction

Plants have to adapt permanently to an altered environment due to their sessile lifestyle. Adaptation to abiotic factors such as light, salt, nutrient deficiency, cold, or water deficit, and biotic interactions such as responses to pathogens, herbivores, nematodes, or mutualistic symbiotic microorganisms such as mycorrhizal fungi are mediated by plant hormones. Among the most prominent plant hormones active in stress responses is jasmonic acid (JA). Additionally, JA is a signal in numerous developmental processes such as seed germination, growth of roots and whole plants, stamen development, and senescence. JA and its derivatives, collectively called jasmonates (JAs), are lipid-derived signaling compounds. JAs are formed from α-linolenic acid (α-LeA) of chloroplast membranes by oxidative processes occurring in different branches of the lipoxygenase pathway. Consequently, JAs are members of the family of oxylipins.

One of the first isolated JA compounds was the methyl ester of JA (MeJA) which was detected as an odorant of *Jasminium grandiflorum* flowers (Demole *et al.*, 1962). Only two decades later, however, the first physiological processes caused by JA or MeJA were described, such as senescence promotion and growth inhibition (Ueda and Kato, 1980; Dathe *et al.*, 1981). In the late 1980s and early 1990s, the first indication for altered gene expression by JA was described by four groups: (i) so-called JA-induced proteins (JIPs) which accumulated abundantly upon degradation of housekeeping proteins were detected in JA-treated barley leaves (Weidhase *et al.*, 1987; Müller-Uri *et al.*, 1988); (ii) induction of vegetative strorage proteins (VSPs) was found upon wounding of soybean leaves (Staswick, 1990); (iii) rapid accumulation of alkaloids was detected upon elicitation of plant cell cultures leading to an increase in endogenous JA (Gundlach *et al.*, 1992); and (iv) accumulation of proteinase inhibitors (PIs) in tomato plants upon wounding by herbivores accompanied by interplant communication by MeJA became the most prominent example of JA-mediated alteration of gene expression (Farmer and Ryan, 1990). These historical aspects of JA research and new findings of the last two decades have been reviewed (Wasternack, 2015).

Jasmonates are signals in plant stress responses and development

Since the early 1990s, a tremendous increase in our knowledge of JA biosynthesis, its mode of action in numerous stress responses and developmental processes, its perception, and its local and systemic signaling has occurred. These aspects have been reviewed (Kazan and Manners, 2011, 2013; Kombrink, 2012; Pieterse *et al.*, 2012; Wasternack and Hause, 2013; Campos *et al.*, 2014; Kazan and Lyons, 2014; Song *et al.*, 2014*b*; Wasternack, 2015; Yan and Xie, 2015; Schuman and Baldwin, 2016; Zhou and Memelink, 2016). Here, we will provide an overview on new aspects of the last few years on JA biosynthesis, metabolism, perception, transcription factors (TFs), as well as repressors and activators in JA-induced gene expression. The balance in activity of TFs, repressors, and activators will be a focus. We apologize for references not cited due to space limitation.

Occurrence and biosynthesis

After initial identification of MeJA as an odor of flowering plants (Demole *et al.*, 1962) and of JA in the culture medium of the fungus *Lasiodiplodia theobromae* (Aldrige *et al.*, 1971), several JA compounds were detected ubiquitously in land plants (Meyer *et al.*, 1984). High levels were found in fruits. Subsequent screening revealed occurrence in all organs carrying plastids. Such a basal level was found to increase rapidly upon wounding or other environmental cues. Recently, 12-oxo-phytodienoic acid (OPDA) but not JA has been identified in *Marchantia polymorpha*, the most basal lineage of extant land plants (Yamamoto *et al.*, 2015). OPDA but not JA was also detected in *Physcomitrella patens* (Stumpe *et al.*, 2010). In one of the oldest vascular plants, the spikemoss *Selaginella martensii*, OPDA has been detected abundantly (Ogorodnikova *et al.*, 2015). The green and brown algae contain numerous lipoxygenase (LOX) pathway intermediates including JAs.

The biosynthetic pathway of JA from α-LeA esterified in chloroplast membranes was established in the 1980s. Initially, a sequence of a LOX, a hydroperoxide cyclase, a reductase, and ß-oxidation of the carboxylic acid side chain was proposed (Vick and Zimmerman, 1983). Later on, the single step of hydroperoxide cyclase was shown to be a two-step reaction of membrane-associated allene oxide synthase (AOS), whose highly unstable product is cyclized by an allene oxide cyclase (AOC) to OPDA. Beside the enzymatic reactions, spontaneous hydrolysis takes place of the unstable epoxide to α- and γ-ketols and non-enzymatic cyclization to racemic OPDA (Brash *et al.*, 1988). This has to be taken into account in enzyme assays as well as JA and OPDA quantification.

All enzymes of OPDA formation are located in chloroplasts (Fig. 1**).** The second half of JA biosynthesis occurs in peroxisomes. Here, OPDA is reduced by an OPDA reductase followed by enzymes of ß-oxidation, the acyl-CoA-oxidase (ACX), the multifunctional proteins (MFPs), and the L-3-ketoacyl-CoA-thiolase (KAT) upon activation to co-esters by fatty acid CoA-synthetases and 4-coumaroyl: CoA-ligases (Fig. 1).

An important determinant in JA biosynthesis is the stereochemistry of intermediates and products. In the AOC-catalyzed step, the enantiomeric form (*7R*, *7S*) is established which occurs in the naturally occurring *cis*-(+)-*7-iso*-JA. Initially, epimerization to the more stable (–)-JA was assumed. Since identification of the most bioactive JA compound, however, it is clear that (+)-*7-iso*-JA-Ile, an isoleucine conjugate of JA carrying the (*7R,7S*) configuration, is required for JA perception and signaling (Fonseca *et al.*, 2009).

JA biosynthesis has frequently been reviewed (Browse, 2009*a*, *b*; Schaller and Stintzi, 2009; Acosta and Farmer, 2010; Wasternack and Kombrink, 2010; Kombrink, 2012; Wasternack and Hause, 2013) including mechanistic explanations upon enzyme crystallization (Lee *et al.*, 2005). Here, we will focus on new aspects of the last few years.

Galactolipases involved in JA biosynthesis

JA biosynthesis is initiated by release of α-LeA from galactolipids of chloroplast membranes by phospholipase A1 (PLA_1) which hydrolyzes with *sn-1* specificity. The flower-specific protein DEFECTIVE IN ANTHER DEHISCENCE 1 (DAD1), a PLA_1, is absolutely required for JA formation (Ishiguro *et al.*, 2001). *DAD1* is a target of the homoeotic protein AGAMOUS, thereby affecting late stamen development (Ito *et al.*, 2007). *DAD1* expression is positively controlled by DAF (DAD1-ACTIVATING FACTOR) which is a RING type E3 ubiquitin ligase (Peng *et al.*, 2013). Furthermore, *DAD1* and most of the subsequently acting genes involved in JA biosynthesis [*AOS*, *AOC*, and 12-oxo-phytodienic acid reductase3 (*OPR3*)] are repressed by the NAC-like gene *ANTHER INDEHISCENCE FACTOR (AIF)* (Shih *et al.*, 2014).

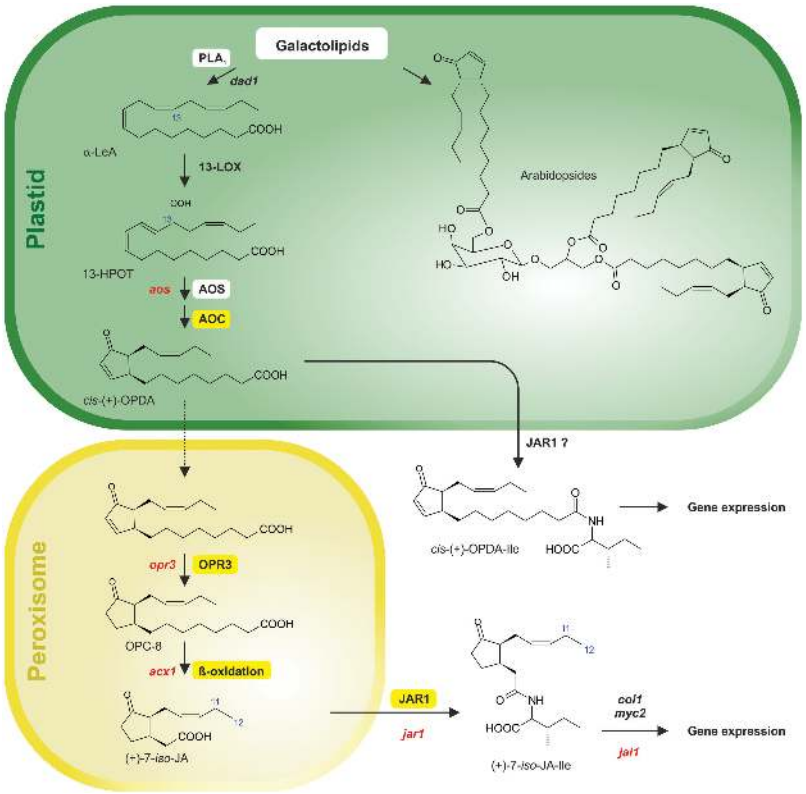

Fig. 1. Synthesis of JA/JA-Ile from α-linolenic acid generated from galactolipids. Enzymes which have been crystalized are given in yellow boxes. Steps which are affected in mutants of Arabidopsis (green) or tomato (red) are indicated. Abbreviations for compounds: α-LeA, α-linolenic acid; 13-HPOT, (13*S*)-hydroperoxyoctadecatrienoic acid; *cis*-(+)-OPDA, *cis*-(+)-12-oxophytodienoic acid; OPC-8, 3-oxo-2-(2-pentenyl)-cyclopentane-1-octanoic acid. Abbreviations for enzymes/proteins: *acx1*, *acyl CoA-oxidase1*; AOC, allene oxide cyclase; AOS, allene oxide synthase; *coi1*, *coronatine insensitive1*; *dad1*, *delayed anther dehiscence1*, *jai1*, *jasmonic acid insensitive1*; JAR1, JA-amino acid synthetase; 13-LOX, 13-lipoxygenase; *myc2*, *bHLHzip transcription factor MYC2*; OPR3, OPDA reductase3; PLA_1, phospholipase A1. [Redrawn based on Wasternack and Hause (2013). Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in Annals of Botany. Annals of Botany 111, 1021–1058, by permission of Oxford University Press.]

Altogether, anther dehiscence is absolutely dependent on intact JA biosynthesis and is regulated positively by AGAMOUS and DAF and negatively by AIF.

Beside these clear data on involvement of DAD1 in JA formation of anthers, there were controversial data on PLA_1s in JA formation of leaves. Here, DAD1 and six homologs such as DONGLE (DGL) and DAD1-like LIPASES (DALL) were found to be expressed upon wounding in a CORONATINE INSENSITIVE1 (COI1)-dependent and COI1-independent manner (Ruduś *et al.*, 2014). DGL was thought to be involved in wound-induced JA formation (Hyun *et al.*, 2008), whereas a PLA_1 is an acylhydrolase rather than a specific phospholipase A and is involved in the basal level of JA (Yang *et al.*, 2007). However, detailed studies on DGL location, numerous lipase mutants, and PLA_1 RNAi lines excluded involvement of DAD1 and DGL in wound-induced JA formation in Arabidopsis leaves (Ellinger *et al.*, 2010). A galactolipase A1 (GLA1) of *Nicotiana attenuata* is involved in JA formation in leaves and roots but not during infection with *Phytophothora parasitica* (Bonaventure *et al.*, 2011). Further studies revealed that upon *Phythophtora* infection, the GLA1 lipase is involved in generation of 9-OH-18:2 and other C_{18} and C_{19} oxylipins (Schuck *et al.*, 2014). The numerous data on involvement of lipases in JA formation suggest their pathway- and stimuli-specific action.

An interesting link between biosynthesis of the lipid digalactosyldiacylglycerol (DGDG) and JA biosynthesis was described recently (Lin *et al.*, 2016). The DGDG synthase1 (DGD1) mutant *dgd1* showed a short inflorescence stem, accompanied by lignified phloem cap cells, following up-regulation of JA-responsive genes including JA biosynthesis genes and a $PLA_1\gamma3$ as well as elevation of levels of JA, JA-Ile, OPDA, and arabidopsides.

13-Lipoxygenase (LOX)

The occurrence and mechanism of lipoxygenase have been repeatedly reviewed (e.g. Andreou and Feussner, 2009; Andreou *et al.*, 2009). Among the seven different branches of the LOX pathway, only the AOS branch leads to JA formation (Feussner and Wasternack, 2002). Oxygen insertion in position C-13 of α-LeA is the ultimate reaction in JA formation (Fig. 1). In Arabidopsis, four of the six-member gene family are 13-LOXs (LOX2, LOX3, LOX4, and LOX6), whereas LOX1 and LOX5 are 9-LOXs, obviously involved in cell death processes by formation of phytoalexins such as death acids (Christensen *et al.*, 2015).

All 13-LOXs are involved in wound-induced JA formation with partially specific activity: (i) a dominant role in wounding (LOX2); (ii) lipid peroxidation (LOX2); (iii) early wound

response of xylem cells (LOX6); (iv) wound response in vascular tissue (LOX3, LOX4); (v) natural and dark-induced senescence (LOX2); and (vi) fertility and flower development (LOX3, LOX4). These aspects including transcriptional and translational control as well as regulation by a vacuolar Ca^{2+} channel have already been reviewed (Wasternack and Hause, 2013).

Meanwhile, some new data on 13-LOXs of Arabidopsis accumulated. AtLOX6 was shown to be exclusively required for JA accumulation in roots upon abiotic and biotic stress and generation of the basal level of OPDA (Grebner *et al.*, 2013). Involvement of AtLOX3 in the salinity stress response has been detected (Ding *et al.*, 2016). Different activities of AtLOX3 and AtLOX4 with a major role of AtLOX4 in defense control against a root-knot nematode and a cyst nematode were identified (Ozalvo *et al.*, 2014). The four 13-LOXs of Arabidopsis seem to act in a paired hierarchical organization, where LOX2 and LOX6 are linked upstream of the LOX3 and LOX4 pair in different responses and in a defined time sequence. These aspects have been discussed and documented recently (Chauvin *et al.*, 2016).

Generation of various 13-LOX mutants carrying a JA reporter system improved our knowledge on intercellular JA transport in the axial (root to shoot) and radial (cell to cell) direction (Gasperini *et al.*, 2015*a*). This biosensor for JA was designed with a specific Jas motif of JA ZIM-domain9 (JAZ9) (see below) and the VENUS variant of the yellow fluorescent protein carrying a nuclear localization signal (NLS) and the *Cauliflower mosaic virus* (CaMV) 35S promoter (Larrieu *et al.*, 2015). It is necessary to mention here that a breakthrough in wounding being behind 13-LOX-generated JA was found by surface potential measurements and transcriptomic analysis. This led to identification of glutamate receptor-like genes (GLRs) known from synaptic activity of animal cells (Mousavi *et al.*, 2013). GLRs mediate organ to organ wound signaling via alteration of surface potentials.

13-LOXs of many other species have been cloned and characterized, but only a few examples can be given here. Among the six tomato LOXs (TomloxA–F), TomloxC and TomloxD are chloroplast-localized 13-LOX enzymes. TomloxC is essential for C5 flavor volatiles without any important defensive function (Shen *et al.*, 2014). Genetic and biochemical evidence obtained by characterizing wound response *spr8* mutants showed that TomloxD is involved in JA generation and consequently linked to all defense reactions against herbivores (Yan *et al.*, 2013). This mutant carries a point mutation in the catalytic domain of TomloxD. The poplar LOX gene family consisting of 20 members was characterized comprehensively (Chen *et al.*, 2015).

In rice, crosstalk between a 13-LOX and 9-LOX1 was observed. Antisense lines of *r9-LOX1* exhibited increased JA levels and JA-mediated responses against chewing and phloem-feeding herbivores (Zhou *et al.*, 2014). Another interesting 13-LOX property was found in the fungus *Fusarium oxysporum*. This species can form and release numerous JA compounds (Miersch *et al.*, 1999). Now, one of the two predicted LOXs of *F. oxysporum* has been cloned (Brodhun *et al.*, 2013). The recombinant protein exhibited a multifunctional activity towards four different branches of the LOX pathway.

13-LOXs are located within plastids, as shown for the first time for barley using an immunological approach (Feussner *et al.*, 1995), as well as by immunogold labeling (Bachmann *et al.*, 2002). The recent localization of a 13-LOX of barley lacks novelty and sufficient quality in the activity data (Springer *et al.*, 2016).

Several crystal structures of LOXs allowed an improved mechanistic explanation of its catalysis (Newcomer and Brash, 2015; Newie *et al.*, 2016). A highly conserved Fe-coordination is in a helical core, and the fatty acid substrate lies near the active site in a hydrophobic U-shaped channel. Alternative orientation for substrates is allowed by the conserved core leading to different products (Newcomer and Brash, 2015). In contrast to plant LOXs, the LOX1 of the cyanobacterium *Cyanothece* sp. PCC 8801 differs in the N-terminal domain and has an α-helical extension which is active in substrate acquisition directly from the membrane (Newie *et al.*, 2016).

Allene oxide synthase (AOS)

AOS is a member of the CYP74 gene family, together with hydroperoxide lyase (HPL) and divinylether synthase (DES) (Feussner and Wasternack, 2002; Hughes *et al.*, 2009). The cloning, characterization, mechanism, and function of AOS in stress responses and development have been reviewed (Brash, 2009; Schaller and Stintzi, 2009; Kombrink, 2012). In many plants, AOSs occur in gene families, except Arabidopsis, where a single-copy gene allowed generation of JA- and OPDA-deficient mutant plants (Park *et al.*, 2002). AOS was crystalized from guayule (Li *et al.*, 2008). Crystallization of AOS from Arabidopsis led to a discussion on its evolutionary origin (Lee *et al.*, 2008).

Upon characterization of two AOSs from *M. polymorpha* combined with phylogenetic analysis, two evolutionarily distinct clusters were discussed, suggesting that CYP74 enzymes have arisen multiple times prior to divergence of the flowering plants (Koeduka *et al.*, 2015). This is supported by data for AOS1 and AOS2 of *P. patens*, where the PpAOS1 could be converted by a single amino acid change to a PpHPL (Scholz *et al.*, 2012). Correspondingly, mutation of the tobacco DES led to AOS activity (Toporkova *et al.*, 2013). Like LOX enzymes with 9- and 13-positional specificity, AOSs prefer 9- or 13-hydroperoxide derivatives. However, dual positional substrate specificity was also found, for example, for rice AOS1 (Yoeun *et al.*, 2013) and barley AOS1 (Maucher *et al.*, 2000). The rice AOS1 has a chloroplast targeting sequence (Haga and Ino, 2004), and the enzyme has been successfully used without a chloroplast target signal in a combined AOS/AOC enzymatic assay as a functional AOS (Riemann *et al.*, 2013). In rice, mutant analysis revealed an interesting crosstalk between the AOS and the HPL branch. Screening of a library on constitutive AOS expression led to the isolation of the mutant *cea62* affected in OsHPL3 but exhibiting a dramatic overproduction of JA (Liu *et al.*, 2012). Another interesting rice mutant was identified recently with the recessive *precious* (*pre*) mutant which has a low level of JA and a long leaf phenotype (Hibara *et al.*, 2016). The mutated gene codes

for an OsAOS1, indicating that JA is a negative regulator of vegetative phase transition.

Allene oxide cyclase (AOC)

AOCs which occur in small gene families have been cloned and characterized, including mechanistic studies from numerous plant species (see reviews by Hofmann and Pollmann, 2008; Schaller and Stintzi, 2009; Kombrink, 2012), and crystal structures are available for AtAOC2 (Hofmann *et al.*, 2006) and PpAOC1 and PpAOC2 (Neumann *et al.*, 2012).

Recently, an AOC of the liverwort *M. polymorpha* was cloned (Yamamoto *et al.*, 2015). The recombinant enzyme has similar properties to that of flowering plants. *Marchantia polymorpha* accumulates OPDA, but no JA, suggesting signaling properties of OPDA (Yamamoto *et al.*, 2015). Generally, overexpression of a JA biosynthesis gene such as *AOS* or *AOC* did not lead to elevated JA levels due to lack of the substrate α-LeA which is generated only upon external stimuli such as wounding (Laudert *et al.*, 2000; Stenzel *et al.*, 2003). Constitutive overexpression of the *AOC* of *Salvia miltiorrhiza* and of wheat, however, led to elevated JA levels and JA responses in the corresponding plants (Gu *et al.*, 2012; Zhao *et al.*, 2014). The wheat studies also showed involvement of JA in the salinity response. AOC mutants of rice (*cpm2*, *hebiba*) showed roles of JA in defense against the blast fungus *Magnaporthe oryzae* (Riemann *et al.*, 2013). Both mutants are impaired in the AOC function and exhibited deficiency in OPDA accompanied by increased salt tolerance and reactive species (ROS)-scavenging activity (Hazman *et al.*, 2015). In Arabidopsis, the four members of the *AOC* gene family have tissue- and organ-specific promoter activities including *in vivo* heteromerization (Stenzel *et al.*, 2012). Further inspection of heteromeric pairs of AOCs are in line with enzyme activity control via heteromerization (Otto *et al.*, 2016). Functional proof by site-directed mutagenesis of AtAOC2 showed its trimeric structure, salt bridges, and a hydrophobic core (Otto *et al.*, 2016), which corresponds to the previously discussed crystal structure (Hofmann *et al.*, 2006).

OPDA reductase (OPR3)

The following steps in JA biosynthesis take place in peroxisomes (Fig. 1). OPDA, formed within the chloroplast, has to be transported across two membranes. While no transporter of the inner and outer envelope of the chloroplast has been identified so far, the import into peroxisomes seems to occur by COMATOSE exhibiting PXA1 activity or by anion trapping (Theodoulou *et al.*, 2005). The OPRs occur in small gene families where only the OPR3 of Arabidopsis and its homologs are involved in JA biosynthesis. In rice, the homolog of AtOPR3 is OsOPR7 (Tani *et al.*, 2008). OPRs have been cloned, localized, biochemically characterized, and functionally analyzed from different plant species including mechanistic studies upon crystallization. These aspects have been reviewed (Schaller and Stintzi, 2009; Kombrink, 2012; Wasternack and Hause, 2013). In rice, the 10 member gene family was comparatively characterized in terms of expression, structure, and function. They were divided into five subgroups, and OPR7 was identified as a JA-forming enzyme (Li *et al.*, 2011). In Arabidopsis there is a gene family of six members, with OPR3 exclusively involved in JA formation. *opr3* mutants are male sterile, the most obvious phenotypes for JA deficiency, due to a defect in stamen development. All of them are affected in JA-induced root growth inhibition. A JA-independent direct role for OPR3 was identified recently in primary root growth under P deficiency (Zheng *et al.*, 2016). Even though primary root growth inhibition by P deficiency is a well-known phenomenon, the underlying mechanism was unknown. Now, a novel function of *AtOPR3* has been shown via suppression of root tip growth at the transcriptional level under P deficiency, including a crosstalk with the ethylene and gibberellin (GA) signaling pathways (Zheng *et al.*, 2016). The initial characterization of the *opr3* mutant of *Arabidopsis thaliana* showed that the mutant is OPDA and JA deficient (Stintzi and Browse, 2000) and was therefore used to distinguish between OPDA- and JA-specific responses (see below). Later on, however, conditional JA formation was shown due to intronic T-DNA insertion in the *opr3* mutant (Chehab *et al.*, 2011). Therefore, for tomato, *OPR3-RNAi* lines were generated for inspection of OPDA-specific responses (Bosch *et al.*, 2014; Scalschi *et al.*, 2015) (see below).

ß-Oxidation of the pentenyl side chain (ACX, MFP, and KAT)

Similar to the final steps in auxin biosynthesis, the pentenyl side chain of JA is shortened by the fatty acid ß-oxidation machinery (Hu *et al.*, 2012). This was shown by labeling experiments (Miersch and Wasternack, 2000), as well as biochemical and genetic evidence for the enzymes involved such as acyl-CoA oxidase (Li *et al.*, 2005; Schilmiller *et al.*, 2007), L-3-ketoacyl CoA thiolase (Cruz Castillo *et al.*, 2004), and 4-coumarate:CoA ligase-like enzymes (Schneider *et al.*, 2005; Koo *et al.*, 2006). Additional evidence was collected by diminished JA formation in *pex* mutants affected in components of the peroxisomal import complex (Hu *et al.*, 2012). These aspects have been reviewed (Hu *et al.*, 2012; Kombrink, 2012; Wasternack, 2014). Among the *pex* mutants, PEX13 interacts with DAYU/ABERRANT PEROXISOME MORPHOLOGY9 (DAU) which is critical in pollen maturation as well as JA formation (X-R Li *et al.*, 2014). An interesting link between the evolutionarily conserved KAT1 and KAT5 and Arabidopsis inflorescence development was shown, suggesting the requirement for JA for normal inflorescence development (Wiszniewski *et al.*, 2014). This is reminiscent of the flower inflorescence phenotype initially observed in the mutant *aim1*, which is affected in the MFP protein (Richmond and Bleecker, 1999).

Intracellular localization of 13-LOXs, AOS, and AOCs

Enzymes of OPDA formation such as 13-LOXs, AOS, and AOCs carry a target sequence for chloroplast import, with some exceptions, but all of them were localized in chloroplasts by import studies and immunocytochemistry including electron microscopy Farmaki *et al.*, 2007; Wasternack, 2007;

Schaller *et al.*, 2008). Corresponding data were found by a proteomic approach with wounded and unwounded Arabidopsis leaves (Gfeller *et al.*, 2011). Even though AOS1 of barley and AOS1 of rice lack the putative chloroplast targeting sequence, both of them localize to the chloroplasts (Maucher *et al.*, 2000; Yoeun *et al.*, 2013). AOS was found in the lipid-rich plastoglobuli, where the high quinone and α-tocopherol content may stabilize the highly unstable epoxide formed in the AOS reaction (Ytterberg *et al.*, 2006). For tomato, in vitro import studies revealed import of AOS to the inner envelope membrane (Froehlich *et al.* 2001). A common localization of AOS in the Arabidopsis inner envelope with the rhomboid-like protease AtRBL8/9 leads to regulation of AOS (Knopf *et al.*, 2012).

Regulation of JA biosynthesis

Regulation of JA biosynthesis has been repeatedly reviewed in terms of three main principles by (i) requirement for release α-LeA as the substrate upon external stimuli; (ii) positive feedback loop in expression of JA biosynthesis genes by JA; and (iii) tissue specificity (Wasternack, 2007; Schaller and Stintzi, 2009; Wasternack and Hause, 2013). Additional regulatory factors may occur by concurrent activity between the AOS and HPL, and other branches of the LOX pathway, by the negative regulators JAZs, post-translational control (e.g. OPR3), heteromerization (e.g. AOCs of Arabidopsis) (Otto *et al.*, 2016), Ca^{2+} signaling, and the mitogen-activated protein kinase cascade (see the review by Wasternack and Hause, 2013). More recent data argue against a positive feedback loop in JA formation, but still show such a regulation in expression of JA biosynthesis genes, thereby suggesting involvement of post-translational modifications (Scholz *et al.*, 2015).

Arabidopsides

Beside the free forms, 18:2 and 18:3 fatty acids and their 9- and 13-hydroperoxides formed by the corresponding LOXs occur in esterified form in many plant species (Ibrahim *et al.*, 2011; Yu *et al.*, 2014). Specifically in Arabidopsis, OPDA and dinor-OPDA have been detected esterified in the *sn-1* or *sn-2* position of galactolipids such as mono- or digalactosyldiacylglycerol (MGDG or DGDG) and are called arabidopside A–F (reviewed by Andreou *et al.*, 2009; Göbel and Feussner, 2009). The large amount of OPDA esterified in arabidopsides and their involvement in the hypersensitive response as well as abiotic stress led to an ongoing discussion on the putative role of arabidopsides. The formation of arabidopsides is dependent on LOX2, which oxidizes bound fatty acids (Glauser *et al.*, 2009), followed by AOS, active with the bound substrate (Park *et al.*, 2002), and is completed with bound oxidized fatty acid products (Nilsson *et al.*, 2012). An interesting new aspect was found by inspection of natural variation of accumulation of arabidopsides (Nilsson *et al.*, 2016) which highlights the repeatedly observed competition between AOS and HPL also for arabidopside formation. High accumulation of arabidopsides was negatively correlated with expression of the HPL which competes with AOS on bound LOX2-generated hydroperoxides (Nilsson *et al.*, 2016). Whereas arabidopsides occur specifically in Arabidopsis, acylated MGDG, the parallel constituent of galactolipids, appears in most plant species (Nilsson *et al.*, 2015). In Arabidopsis, these acylated MGDGs frequently incorporate OPDA. Mutants of the enzyme involved in acylation have complete loss of OPDA-containing acyl-MGDG, but no clear biological role has been identified so far for these acyl-MGDGs (Nilsson *et al.*, 2015).

OPDA as a signal

A distinct set of genes is induced specifically by OPDA (Taki *et al.*, 2005). OPDA perception does not take place via the SCF^{COI1}–JAZ co-receptor complex (Thines *et al.*, 2007). These observations led to numerous studies, where expression by OPDA was shown to occur independently of COI1 in different processes of different higher plants, as well as in lower plants which are JA deficient. These aspects have been repeatedly reviewed (e.g.Wasternack and Hause, 2013; Wasternack and Strnad, 2016). Due to the requirement for JA-Ile as the ligand of the receptor complex, the identification of a conjugate of OPDA, OPDA-Ile (Floková *et al.*, 2016), led to the suggestion that this compound might mediate OPDA-specific responses. Indeed, OPDA-Ile has biological activity in terms of expression of OPDA-specific genes (Arnold *et al.*, 2016). Its perception, however, has not been studied so far.

Metabolism

Twelve metabolic pathways are known today that convert JA into active and inactive compounds, or compounds which are partially active (Fig. 2) (Koo and Howe, 2012; Wasternack and Hause, 2013; Heitz *et al.*, 2016). Whereas the majority of JA signaling is provided by homeostasis among different JA-Ile derivatives (see below), some reactions lead to compounds with activity in specific reactions of stress responses and development such as leaf movement (see below).

Conjugation

Formation of JA-Ile by jasmonoyl-isoleucine synthetase (JAR1), a member of the *GH3* gene family (AtGH3.11) (Staswick and Tiryaki, 2004), is the ultimate step in JA perception. The GH3 protein family comprises acyl acid-amido synthetases with auxin, JA, or benzoate as substrates (Westfall *et al.*, 2013). The role of JA-Ile became obvious upon its identification as the most biologically active JA compound (Fonseca *et al.*, 2009), by initial reports on JA-Ile-mediated promotion of SCF^{COI1}–JAZ co-receptor complex formation (Thines *et al.*, 2007; Katsir *et al.*, 2008; Melotto *et al.*, 2008), and by properties of JA-Ile as the ligand of the complex (Yan *et al.*, 2009; Sheard *et al.*, 2010). This scenario has been used to design a ligand-based antagonist of JA-Ile by modifying coronatine (Monte *et al.*, 2014) or for generation of inhibitors of JAR1 such as jarin1 by chemical screening (Meesters *et al.*, 2014). Crystallization of JAR1 led to a mechanistic explanation of why the epimer (+)-7-*iso*-JA-Ile is formed (Westfall *et al.*, 2012). Even though (+)-7-*iso*-JA-Ile is exclusively

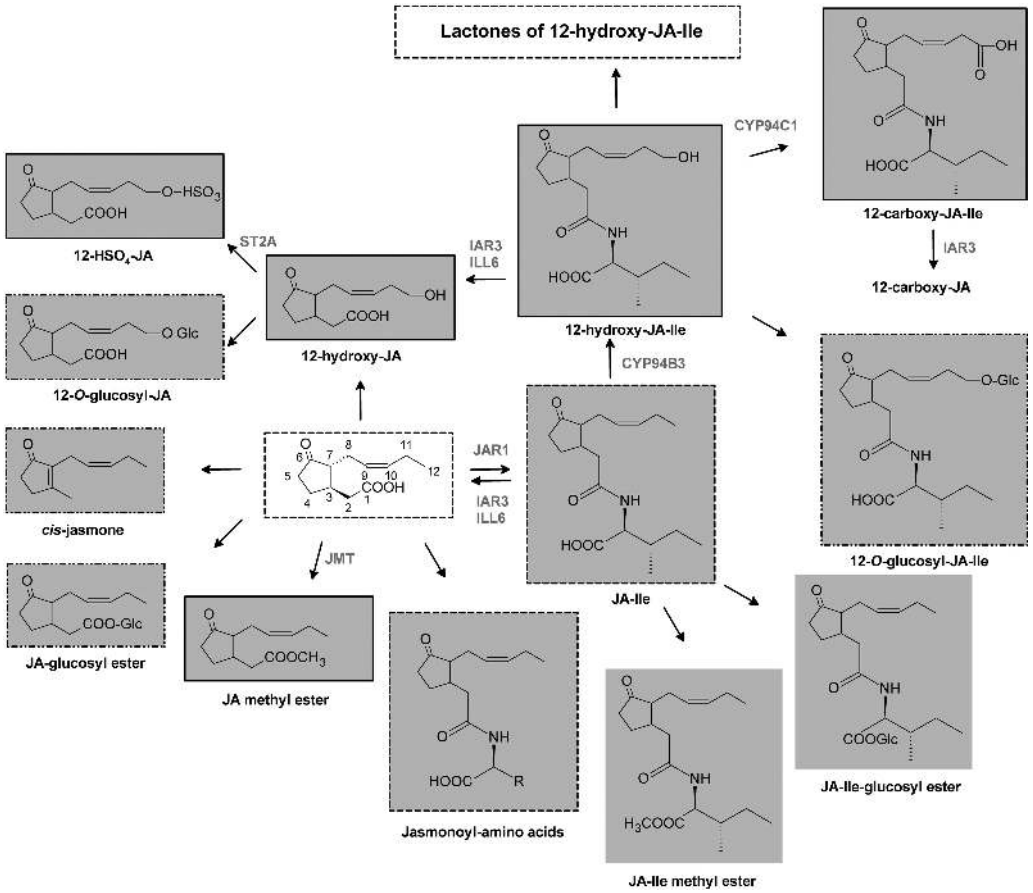

Fig. 2. Metabolic conversion of JA. Methylation to JA-Me, JA glucosyl ester formation, decarboxylation to *cis*-jasmone, hydroxylation to 12-OH-JA, sulfation of 12-OH-JA, *O*-glucosylation of 12-OH-JA, conjugation with amino acids, preferentially isoleucine to give JA-Ile, methylation to JA-Me-Ile, 12-hydroxylation of JA-Ile, carboxylation of 12-OH-JA-Ile, *O*-glucosylation of JA-Ile, and JA-Ile glucosyl ester formation are indicated, together with the known enzymes involved: JAR1, jasmonoyl isoleucine synthetase; JA-Ile-12-hydroxylase; CYP94B3; 12-OH-JA-Ile carboxylase, CYP94C1; amidohydrolases IAR3 and ILL6; JMT, JA methyl transferase; ST2a, 12-OH-JA sulfotransferase. The lactone of 12-OH-JA-Ile was added even though its detection in plants is missing to date. Biologically inactive compounds are outlined with solid lines, partially active compounds are outlined with dashed and dotted lines, and active compounds are outlined with dotted lines; the biological activity of 12-carboxy-JA, as well as of JA-Ile-glucosyl ester and JA-Ile methyl ester are unknown [modified after Wasternack and Strnad (2016). Jasmonate signaling in plant stress responses and development—active and inactive compounds. New Biotechnology 33, 604–613, Copyright (2016), with permission from Elsevier].

formed upon wounding, tomato JAR1-RNAi lines showed only 50–75% down-regulation, suggesting the occurrence of another JA-conjugating enzyme other than JAR1 (Suza *et al.*, 2010). This has to be taken into account in the use of *jar1* mutants. In rice, OsJAR1, exhibiting similar substrate specificity to AtJAR1, is the most important JA-conjugating enzyme in the wounding response (Fukumoto *et al.*, 2013; Svyatyna *et al.*, 2014), seed development under field conditions (Fukumoto *et al.*, 2013), as well as floret opening and anther dehiscence (Xiao *et al.*, 2014). In photomorphogenesis, OsJAR1 activity is also required, but acts partially redundantly to OsJAR2 (Svyatyna *et al.*, 2014). The two major functions of OsJAR1 cannot be complemented by OsJAR2 (Fukumoto *et al.*, 2013). The level of JA-Ile plays an important regulatory role for all JA-dependent processes and is sustained by JAR1 activity and subsequent metabolic reactions such as hydroxylation, carboxylation, and hydrolysis of conjugates. Recently, (+)-7-*iso*-JA-Ala, (+)-7-*iso*-JA-VAL, (+)-7-*iso*-JA-Leu, and (+)-7-*iso*-JA-Met have also been identified as endogenous bioactive JA derivatives via corresponding coronafacic acid conjugates as stable molecular mimics (Yan *et al.*, 2016). These conjugates showed distinct binding in the COI1–JAZ interaction assay (see below).

Hydroxylation and carboxylation

Direct hydroxylation of JA to 12-OH-JA has not been detected so far, except a putative activity of a fungal monooxygenase (Patkar *et al.*, 2015). Several members of the CYP94 gene family have been identified to hydroxylate JA-Ile (CYP94B3, CYP94C1) and to carboxylate 12-OH-JA-Ile (CYP94C1) (Koo *et al.*, 2011; Heitz *et al.*, 2012). This work was recently extended to additional family members such as CYP94B1 (Koo *et al.*, 2014; Bruckhoff *et al.*, 2016) and CYP94B2 (Bruckhoff *et al.*, 2016). Hydroxylation and carboxylation conribute to JA catabolism via an ω-oxidation pathway. The family members act in a semi-redundant manner, as shown with quadruple mutants (Bruckhoff *et al.*, 2016). Data on overexpression lines and mutants of CYP94 gene family members revealed involvement in JA-Ile signaling during herbivory and pathogen attack by *Botrytis cinerea* (Koo *et al.*, 2011; Heitz *et al.*, 2012). There is, however, a complex sustainment of JA-Ile

homeostasis, in which hydroxylation and carboxylation remove the active JA-Ile, whereas the amido-hydrolases ILL6 and IAR3 cleave JA-Ile and 12-hydroxy-JA-Ile (see review by Heitz *et al.*, 2016). The dynamics in JA-Ile homeostasis differ between leaf injury, *B. cinerea*-infected leaves, and developing flowers (Widemann *et al.*, 2016). Even though the basic scenario of activation and inactivation via CYP94s seems to be clear, a complex regulation may occur: triple mutants of *CYP94B1*, *CYP94B3*, and *CYP94C1* revealed accumulation of JA-Ile without a stronger wound response, but symptoms known for JA-Ile deficiencies and normal responses to exogenous JA (Poudel *et al.*, 2016).

ILL6 and IAR3 are active in simultaneous regulation of JA and auxin homeostasis, indicating an important crosstalk between JA and auxin signaling (Zhang *et al.*, 2016). The amido-hydrolases are active in the endoplasmic reticulum (Sanchez Carranza *et al.*, 2016) which corresponds to the intracellular location of the CYP94s (Koo *et al.*, 2014). Interestingly, a novel JA derivative, 12-carboxy-JA, was identified recently by a non-targeted metabolite analysis in floral tissues which is formed presumably from 12-carboxy-JA-Ile via IAR3 (Bruckhoff *et al.*, 2016). In *N. attenuata*, JIH1, a homolog of AtIAR3, also regulates the JA-Ile level (Woldemariam *et al.*, 2012), and *N. attenuata* CYP94B3 contributes to attenuation of resistance to herbivores (Luo *et al.*, 2016).

Decarboxylation

The volatile *cis*-jasmone has been known for a long time as a constituent of the floral bouquet of many plants attracting insects for pollination. Wounding leads to *cis*-jasmone formation and its activity in multitrophic interactions of plants with aphids and their parasitoids (Bruce *et al.*, 2008). A distinct set of genes, different from those of JA, is expressed by *cis*-jasmone treatment (Matthes *et al.*, 2010). In maize, *cis*-jasmone can prime formation of volatile organic compounds (Oluwafemi *et al.*, 2013). Beside decarboxylation of JA, isomerization of *cis*-(+)-OPDA into *iso*-OPDA followed by ß-oxidation to 3,7-didehydro-JA and decarboxylation was proposed for synthesis of *cis*-jasmone (Schulze *et al.*, 2007).

Methylation and esterification

Even though MeJA has been known for a long time as a constituent of floral scent active in pollination and as a signaling compound in all reactions where JA or JA-Ile are active, Me JA is not active *per se*. Transgenic approaches showed clear evidence that MeJA has to be cleaved by esterases and subsequently converted to JA-Ile by JAR1 (Stitz *et al.*, 2011). These data correspond to the inactivity of the JA-Ile–receptor complex in binding MeJA (Sheard *et al.*, 2010). MeJA transferases have been cloned from Arabidopsis, tomato, rice, grapevine, strawberry, and black cottonwood, and are involved, among others, in stress responses and fruit development via sustaining JA levels (see reviews by Wasternack and Hause, 2013; Preuß *et al.*, 2014; Qi *et al.*, 2016; Zhao *et al.*, 2016). *Lasiodiplodia theobromae* was the fungal species from which (+)-7-*iso*-JA was first isolated (Miersch *et al.*, 1987). Meanwhile, several JA esters, so-called lasiojasmonates, were isolated from *Lasiodiplodia* sp., a grapevine pathogen (Andolfi *et al.*, 2014). In different phytotoxic and zootoxic assays, however, these new compounds were inactive.

Sulfation

The composition, occurrence, substrate specificity, and functions of 21 sulfotransferases (STs) of Arabidopsis have been comprehensively described (Hirschmann *et al.*, 2014; Koprivova and Kopriva, 2016). Among them, AtST2a (AtSOT15) is active specifically with 12-OH-JA as substrate (Gidda *et al.*, 2003). The sulfuryl group donor is 3'-phosphoadenosine 5'-phosphosulfate (PAPS). Therefore, in kinase mutants involved in PAPS formation, 12-HSO_4-JA levels are altered (Mugford *et al.*, 2009). A new addition to the plant ST gene family is the tyrosylprotein ST active on peptides and tyrosyl residues of proteins (Koprivova and Kopriva, 2016), thereby active in signaling such as the auxin/plethora pathway in root stem cell niche maintenance (Zhou *et al.*, 2010).

O-Glycosylation

Glycosylation, preferentially glucosylation, is a basic modification of secondary compounds as well as hormones. In the case of jasmonates, 12-OH-JA (tuberonic acid) and its glucosyl derivative were described as tuber-inducing factors of potato in the 1990s (see Wasternack and Hause, 2013). This is obviously an indirect role, since strong evidence supports a light-dependent tuber induction via leaf-generated mRNAs and two TFs activating temperature-dependent GA formation within the stolon (Hannapel, 2010; Wasternack and Hause, 2013). 12-*O*-Glucosyl-JA is a late but abundantly accumulating JA compound in wounded leaves of tomato or Arabidopsis (Glauser *et al.*, 2008; Miersch *et al.*, 2008). Constitutive accumulation in tissues such as filaments of *Zea mays* or leaves of *Glycine max* can occur at up to two to three orders of magnitude higher levels than JA (Miersch *et al.*, 2008). Interestingly, in Albizzia and Samanea species, 12-*O*-glucosyl-JA acts as a leaf closing factor in motor cells with high structural specificity of the 12-OH-JA (Nakamura *et al.*, 2011) as well as the glucone moiety (Ueda *et al.*, 2015). Only a few reports are available so far on characterization of glucosyltransferase of 12-OH-JA and a 12-OH-JA-glucoside hydrolase (Wakuta *et al.*, 2010; Seto *et al.*, 2011).

A new derivative of 12-OH-JA and 12-OH-JA-Ile was identified as jasmine ketolactone (Jimenez-Aleman *et al.*, 2015). Based on these naturally occurring compounds, macrolactones of 12-OH-JA-Ile were synthesized which exhibited biological activity as (*3R,7R*) and as (*3S,7S*) forms (Jimenez-Aleman *et al.*, 2015), indicating conversion of an inactive JA derivative into an active one.

Jasmonate signaling

SCF^{COI1}–JAZ co-receptor complex

Since the 1990s, research on JA signaling has become a hotspot. The isolation of the F-box protein COI1, which forms

the SCF^{COI1} E3 ligase with SKP1 and CULLIN1 to mediate JA responses, is the first milestone in JA signaling (Feys *et al.*, 1994; Xie *et al.*, 1998). In 2007, the family of Arabidopsis JAZ proteins were discovered to be the substrates of SCF^{COI1}, and interact with and inhibit the TF MYC2 to repress JA responses (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007). In the resting state, JAZs interact with and repress downstream TFs (e.g. MYC2) to suppress of JA responses. In response to JA signal, COI1 interacts with and ubiquitinates JAZs tagging them for degradation through the 26S proteasome, thereby releasing downstream TFs to regulate gene expression and activate JA responses (Fig. 3) (Wasternack and Hause, 2013).

(+)-7-*iso*-JA-Ile is the most bioactive endogenous JA in promoting COI1 interaction with JAZ1, and the JA analog coronatine synthesized by some strains of *Pseudomonas syringae* is more active than (+)-7-*iso*-JA-Ile (Fonseca *et al.*, 2009), and can be a potent agonist of the JA receptor complex (Katsir *et al.*, 2008). The recently identified conjugates (+)-7-*iso*-JA-Ala, (+)-7-*iso*-JA-Val, (+)-7-*iso*-JA-Leu, and (+)-7-*iso*-JA-Met showed distinct and highly different binding activity in the COI1–JAZ interaction assay, if COI1 paralogs of various species were used (Yan *et al.*, 2016). This suggests evolution of the COI1 function by only a few amino acid substitutions.

Studies on the structure of the COI1–ASK1–JA-Ile/coronatine–JAZ1 degron showed that COI1, the JAZ1 degron, and IP_5 constitute a co-receptor with high affinity for JA-Ile and coronatine (Sheard *et al.*, 2010). A modified COI1 that is able to maintain high fertility and defense against insect attack, but is resistant to *P. syringae* pv. *tomato*, was isolated (L. Zhang *et al.*, 2015), and coronatine-*O*-methyloxime was identified as a JA antagonist that prevents the COI1 interaction with JAZs, and enhances plant resistance to *P. syringae* (Monte *et al.*, 2014). This latter aspect corresponds to the initial observation that coronatine is a potent agonist of the JA receptor complex (Katsir *et al.*, 2008).

The family of Arabidopsis JAZs with 13 members normally have a conserved C-terminal Jas domain for binding to COI1, MYC2, or other TFs, a highly conserved ZIM domain in the middle for interacting with the Novel Interactor of JAZ (NINJA)/TOPLESS (TPL) co-repressors and dimerizations of JAZs, and a variable N-terminal NT domain for interaction with several signal transduction factors (e.g. DELLA proteins) (Chung and Howe, 2009; Chung *et al.*, 2009; Pauwels and Goossens, 2011).

Overexpression of *JAZ9*, *JAZ8*, *JAZ7*, *JAZ4*, and *JAZ13*, and alternative splice variants or truncated forms of JAZs (*JAZ10.3*, *JAZ10.4*, *JAZ1Δ3A*, and *JAZ3ΔC*) inhibits JA-inhibitory root growth and hypocotyl elongation, flowering, leaf senescence, and defense against the herbivore *Spodoptera exigua* or the pathogen *Fusarium oxysporum*, and single or multiple mutants of JAZ4, JAZ7, JAZ8, JAZ9, JAZ10, and JAZ13 exhibit JA hypersensitivity in JA-inhibitory root growth and hypocotyl elongation, leaf senescence, flowering, or susceptibility to *P. syringae* (Chini *et al.*, 2007; Thines *et al.*, 2007; Chung and Howe, 2009; Chung *et al.*, 2010; Demianski *et al.*, 2012; Shyu *et al.*, 2012; Yang *et al.*, 2012; Thireault *et al.*, 2015; Thatcher *et al.*, 2016; Yu *et al.*, 2016).

JAZs can be depleted in a COI1-independent manner by bacterial effectors. HopX1, a cysteine protease from

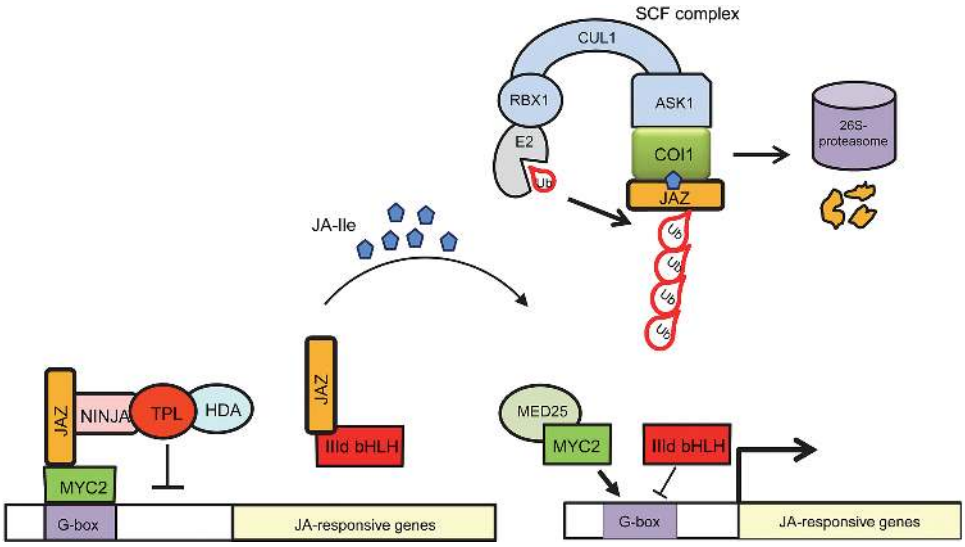

Fig. 3. A simplified model of JA-Ile perception and signaling via the SCF^{COI1}–JAZ co-receptor complex. The JAZ repressors recruit the co-repressor complex consisting of NINJA, TOPLESS (TPL), and histone deacetylase (HDA), interact with and repress positive regulators of JA signaling such as the transcription activator MYC2, and also inhibit negative regulators of JA signaling such as the transcriptional repressors IIId bHLH factors. The perception of JA-Ile by the COI1–JAZ co-receptor leads to degradation of JAZs via the 26S proteasome; the downstream transcription factors are activated to regulate expression of JA-responsive genes and JA responses synergistically or antagonistically. MYC2 associates with the MED25 subunit of the mediator complex, binds to the G-box motif of the target promoters, and activates JA-responsive genes. The IIId bHLH factors antagonize MYC2 via competitive binding to the G-box motif and inhibiting JA-responsive genes. COI1, ASK2, CULLIN1, Rbx, and E2 are components of the SCF^{COI1} complex. Ub, ubiquitin. [Modified with permission from Wasternack and Hause (2013). Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in Annals of Botany. Annals of Botany 111, 1021–1058, by permission of Oxford University Press.]

P. syringae pv. *tabaci*, degrades JAZs, and complements the function of coronatine in susceptibility to *P. syringae* (Gimenez-Ibanez *et al.*, 2014). HopZ1a, a member of the YopJ effector family, interacts with and acetylates JAZs for degradation, and rescues the virulence of the *P. syringae* mutant without coronatine (Jiang *et al.*, 2013).

NINJA/TPL co-repressors

NINJA acts as an adaptor, binds with its C-terminus to the ZIM domain of JAZs, and utilizes its ERF-associated amphiphilic repression (EAR) domain to recruit the co-repressors TPL and TPL-related proteins (TPRs), which affect epigenetic modification with histone deacetylases (e.g. HDA19), to repress JA responses (Fig. 3) (Pauwels *et al.*, 2010). The non-canonical JAZ8 and JAZ13, which harbor a divergent Jas domain exhibiting little interaction with COI1 and are resistant to JA-induced degradation, have an EAR motif that directly recruits the TPL repressors, and interact with MYC2 to inhibit JA responses (Shyu *et al.*, 2012; Thireault *et al.*, 2015).

Activators and repressors in JA signaling

After the discovery of JAZs, a battery of TFs and signal transduction factors acting as activators or repressors were isolated and helped in understanding the JA signaling and crosstalk for different biological processes. We will briefly describe these TFs and their roles in crosstalk.

JA signaling activators

bHLH subgroup IIIe TFs: MYC2. MYC2 was first identified as a master regulator through cloning of the Arabidopsis JA-insensitive mutant *myc2*/*jai1*/*jin1* (Lorenzo *et al.*, 2004). MYC2 contains an N-terminal JAZ interaction domain (JID) that interacts with JAZs (Fernández-Calvo *et al.*, 2011), a transcription activation domain (TAD) required for transactivation and interaction with MEDIATOR25 (MED25) (Chen *et al.*, 2012; Zhai *et al.*, 2013), a C-terminal NLS for recruiting JAZs into the nucleus (Withers *et al.*, 2012), and a C-terminal bHLH (basic helix–loop–helix) domain for dimerization and binding to the G-box (CACGTG) or G-box like motifs (Toledo-Ortiz *et al.*, 2003).

Arabidopsis MYC2 positively regulates most JA-dependent responses, such as expression of wound-responsive genes, wounding response, inhibition of root growth and the apical hook, sesquiterpene synthase, defense against insects (e.g. *Spodoptera littoralis* and *Helicoverpa armigera*), and susceptibility to the pathogen *P. syringae*; however, MYC2 negatively regulates a subset of JA responses, including biosynthesis of indole glucosinolates and tryptophan, resistance to necrotrophic pathogens (e.g. *B. cinerea*), and expression of pathogen-response genes *ETHYLENE RESPONSE FACTOR1* (*ERF1*), *OCTADECANOID-RESPONSIVE ARABIDOPSIS AP2/ERF 59* (*ORA59*), and *PLANT DEFENSIN1.2* (*PDF1.2*) (Lorenzo *et al.*, 2004; Dombrecht *et al.*, 2007; Fernandez-Calvo *et al.*, 2011; Montiel *et al.*, 2011; Hong *et al.*, 2012; Zhai *et al.*, 2013; Song *et al.*, 2014*b*).

MYC2 functions as a transcriptional activator, interacts with the mediator subunit MED25 (Kidd *et al.*, 2009; Çevik *et al.*, 2012; Chen *et al.*, 2012), and binds to and activates the G-box or G-box-like motifs of promoters of JA-responsive genes, such as *TAT1* (Fig. 3) (Hou *et al.*, 2010). However, MYC2 also acts as a transcriptional repressor, binding to and repressing the promoters of *PLETHORA1* (*PLT1*)/*PLT2* or *ORA59*, to attenuate root growth or resistance to *B. cinerea* (Chen *et al.*, 2011). These studies suggest that MYC2 differentially regulates JA responses by acting as a transcriptional activator or repressor, and as a positive or negative regulator.

MYC2 is a node of crosstalks. Both DELLA repressors in the GA pathway and JAZs interact with MYC2 to repress sesquiterpene biosynthesis in flowers (Hong *et al.*, 2012). JA and GA activate MYC2 and induce sesquiterpene biosynthesis. Abscisic acid (ABA) promotes the interaction of the ABA receptor PYL6 with MYC2, and impedes the binding ability of MYC2 to target promoters (Aleman *et al.*, 2016). βC1 of *Tomato yellow leaf curl China virus* interacts with MYC2, suppressing MYC2-regulated terpene synthase, and reducing defense to whitefly, thereby promoting the performance of whitefly and accelerating the spread of the virus (R Li *et al.*, 2014).

bHLH subgroup IIIe TFs: MYC3 and MYC4. These two TFs interact with JAZs (Fernández-Calvo *et al.*, 2011). The α-helix of the Jas domain in JAZ9 nestles between the JID and TAD domains of MYC3, becomes an integral part, and competitively inhibits MYC3's interaction with MED25 (F. Zhang *et al.*, 2015). Specific mutations in the JID of MYC2 and MYC3 could abolish the interactions with most JAZs, and enhance JA sensitivity (Goossens *et al.*, 2015). MYC3 and MYC4 function redundantly with MYC2 in JA-inhibitory root growth, apical hook formation, JA-induced leaf senescence, glucosinolate biosynthesis, susceptibility to *P. syringae*, resistance to *B. cinerea*, defense against *S. littoralis*, and insect growth (Fernández-Calvo *et al.*, 2011; Schweizer *et al.*, 2013; Chico *et al.*, 2014; Song *et al.*, 2014*a*; Qi *et al.*, 2015*b*).

The E3 ligase PLANT U-BOX PROTEIN10 (PUB10) ubiquitinates MYC2, MYC3, and MYC4 for degradation, and inhibits JA responses (Jung *et al.*, 2015). MYC2, MYC3, and MYC4 participate in the daily cycle of plant growth and defense: during the day, blue and red light stabilize MYC2/3/4, inhibit growth, and promote defense; in the night, dark destabilizes MYC2/3/4, promotes growth and suppresses defense (Chico *et al.*, 2014). TIME FOR COFFEE, a key determinant of the circadian clock, binds to and destabilizes MYC2 to regulate plant growth and defense rhythmically (Shin *et al.*, 2012).

bHLH subgroup IIIe TFs: MYC5. MYC5, MYC2, MYC3, and MYC4 function redundantly in regulation of the expression of *MYB21*, *MYB24*, *MYB57*, and *MYB108*, and form the JAZ-inhibited MYC–MYB transcriptional complexes with MYB21 and MYB24 to mediate stamen development (Song *et al.*, 2011, 2013; Figueroa and Browse, 2015; Qi *et al.*, 2015*a*). The *myc2 myc3 myc4 myc5* quadruple mutant exhibits short filament, delayed anther dehiscence, and unviable pollen grains at floral stage 13 (Qi *et al.*, 2015*a*). Moreover, the MYC2^{E165K} mutation increases transactivation activity, and restores the stamen development of *aos ninja* (Gasperini *et al.*, 2015*b*).

TTG1/bHLH/MYB complex. The WD-repeat protein Transparent Testa Glabra1 (TTG1), bHLH transcription factors GLABRA3 (GL3) or ENHANCER of GL3 (EGL3), TRANSPARENT TESTA8 (TT8), and R2R3-MYB transcription factors MYB75 or GLABRA1 (GL1) form the WD-repeat/bHLH/MYB complexes, which activate downstream genes to promote trichome formation and anthocyanin accumulation (Ramsay and Glover, 2005). JAZs interact with bHLH and MYB components of the TTG1/bHLH/MYB complexes to inhibit their transcriptional activity. Environmental stress leads to a rise in endogenous JA; JAZs are degraded, and the TTG1/bHLH/MYB complexes are activated to enhance trichome formation and anthocyanin accumulation, respectively, thereby resulting in increased defense or stress tolerance (Qi *et al.*, 2011). DELLAs also bind to and repress the TTG1/bHLH/MYB complexes, and attenuate GA/JA-induced trichome formation (Qi *et al.*, 2014). JA and GA, respectively, induce degradation of JAZs and DELLAs, activate the TTG1/bHLH/MYB complexes, and synergistically enhance trichome formation.

Other bHLH TFs. The bHLH factors Arabidopsis INDUCER OF CBF EXPRESSION1 (ICE1)/ICE2, OsbHLH148, and RICE SALT SENSITIVE3 (RSS3)/OsbHLH094 interact with JAZs, and respectively regulate tolerance to freezing (Hu *et al.*, 2013) and salt (Toda *et al.*, 2013), suggesting that JA plays important roles in plant tolerance to abiotic stresses. In secondary metabolism, bHLH Iridoid Synthesis 1 (BIS1) and BIS2 regulate monoterpenoid indole alkaloid biosynthesis in *Catharanthus roseus* (Van Moerkercke *et al.*, 2015, 2016), and TRITERPENE SAPONIN BIOSYNTHESIS ACTIVATING REGULATOR1 (TSAR1) and TSAR2 regulate triterpene saponin biosynthesis in *Medicago truncatula* (Mertens *et al.*, 2016).

R2R3-MYB TFs. The JA-responsive flower-specific R2R3-MYB TFs MYB21, MYB24, and MYB57 function as direct targets of JAZs, specifically and redundantly regulate filament elongation, anther dehiscence and pollen maturation (Mandaokar *et al.*, 2006; Cheng *et al.*, 2009). Overpression of *MYB21* can restore the stamen development of *coi1* and *opr3* (Song *et al.*, 2011). MYB108 acts downstream of MYB21, and controls anther dehiscence and pollen maturation (Mandaokar and Browse, 2009). GA induces the degradation of DELLAs, up-regulates *DAD1* expression to enhance JA biosynthesis, and promotes filament elongation via the MYB pathway (Cheng *et al.*, 2009). The key components of the auxin pathway, including Transport Inhibitor Response 1 (TIR1)/Auxin-signaling F-box proteins (AFBs), Indole-3-Acetic Acid protein 8 (IAA8), and Auxin Response Factor 6 (ARF6)/ARF8, modulate late stamen development via JA biosynthesis (Nagpal *et al.*, 2005; Cecchetti *et al.*, 2008; Wang *et al.*, 2013).

The plant metabolites glucosinolates (GSs) can be converted to toxins during insect feeding, and fend off herbivore attack (Bones and Rossiter, 2006). The R2R3-MYB TFs MYB28, MYB29, and MYB76 control the expression of biosynthetic genes, and biosynthesis of aliphatic GS, whereas MYB34, MYB51, and MYB122 regulate indole GS biosynthesis. These MYBs form MYB–MYC transcriptional complexes with MYC2, MYC3, and MYC4, bind to the promoters of GS biosynthetic genes, and activate JA-induced GS biosynthesis and defense against insect attack (Schweizer *et al.*, 2013).

AP2/ERF-domain TFs. Among the APETALA2/ETHYLENE RESPONSE FACTOR (AP2/ERF) domain TFs, JAZs only interact with TARGET OF EAT1 (TOE1) and TOE2, which bind to and repress the promoter of *FLOWERING LOCUS T* for suppression of flowering (Zhai *et al.*, 2015). ERF1, ORA59, ERF5, and ERF6 interact with MED25 (Çevik *et al.*, 2012) and mediate JA/ethylene (ET)-regulated immunity to necrotrophic fungi (Pre *et al.*, 2008; Moffat *et al.*, 2012). Salicylic acid (SA) antagonizes JA-regulated plant immunity via repressing the *ORA59* promoter (Van der Does *et al.*, 2013). JA-responsive ERF109 transactivates the auxin biosynthetic genes ANTHRANYLATE SYNTHASE1 (*ASA1*) and *YUCCA2*, activates auxin biosynthesis, and promotes JA-induced lateral root formation (Cai *et al.*, 2014). OCTADECANOID-DERIVATIVE RESPONSIVE CATARANTHUS AP2-DOMAIN2 (CrORCA2) and CrORCA3 function in a complementary manner with BIS1 and BIS2 to promote monoterpenoid indole alkaloid biosynthesis in *C. roseus* (Van Moerkercke *et al.*, 2015, 2016).

EIN3/EIL1. ETHYLENE-INSENSITIVE3 (EIN3) and EIN3-LIKE 1 (EIL1) are the core TFs in the ET pathway. JAZ1 interacts with and represses EIN3/EIL1 through recruiting HDA6, and JA activates EIN3 and EIL1 via suppression of JAZ1, and promotes the expression of *ERF1/ORA59* to enhance resistance and root hair formation (Zhu *et al.*, 2011). On the other hand, the interactions between EIN3/EIL1 and MYC2/3/4 mediate the antagonism between JA and ET in various plant developmental processes and defense responses against herbivore attack: ET-stabilized EIN3 and EIL1 interact with and repress the transcriptional activity of MYC2/3/4 to suppress expression of wound-responsive genes and defense against the generalist *S. exigua*; meanwhile, JA-activated MYC2/3/4 interact with and inhibit EIN3 and EIL1 to reduce apical hook formation of seedlings under dark (Song *et al.*, 2014*a*, *b*). JA also activates the expression of EIN3 BINDING F-BOX PROTEIN1 (EBF1) to suppress EIN3 and EIL1 (Zhang *et al.*, 2014).

YABs. The YABBY (YAB) family TFs YAB1 and YAB3 interact with JAZ3 to modulate anthocyanin accumulation, chlorophyll degradation, and susceptibility to *P. syringae* (Boter *et al.*, 2015).

NAC019, NAC055, and NAC072. Plants recognize *P. syringae* entry in stomata, and trigger stomatal closure via SA and ABA. Coronatine activates MYC2, and MYC2 directly transactivates the NAM/ATAF/CUC (NAC) TFs NAC019, NAC055, and NAC072, which repress the SA biosynthetic gene *ISOCHORISMATE SYNTHASE1* (*ICS1*) and activate the SA metabolic gene *BSMT1* to inhibit SA accumulation, and promote stomatal reopening and bacterial entry (Zheng *et al.*, 2012). In addition, NAC019, NAC055, and NAC072 mediate JA/MYC2/3/4-regulated leaf senescence via up-regulation of

the chlorophyll catabolic genes (e.g. *NYE1* and *NYE2*) and promotion of chlorophyll degradation (Zhu *et al.*, 2015).

DELLAs. The DELLA repressors in the GA pathway interact with JAZs and function as positive regulators of the JA pathway, and mediate the antagonism between JA and GA in regulating plant growth and defense. A herbivore attack-induced increase in JA leads to proteasomal degradation of JAZs and activation of MYC2 plant defense, whereas DELLAs are relieved of JAZs inhibition, and interact with PHYTOCHROME INTERACTING FACTORs (PIFs) to suppress growth. Conversely, GA triggers DELLA degradation, allowing the PIFs to activate growth and meanwhile the JAZ repressors are released to inhibit MYC2-mediated plant defense responses (Hou *et al.*, 2010; Yang *et al.*, 2012). Interestingly, the combined mutations of five JAZs and the photoreceptor phyB, which directly inhibits PIFs, can de-repress both MYCs and PIFs, uncouple the trade-off between growth and defense, and promote both defense and growth, providing a potential way to assemble plant traits (Campos *et al.*, 2016).

JA signaling repressors

bHLH subgroup IIId TFs. The bHLH subgroup IIId TFs, including bHLH17/JASMONATE ASSOCIATED MYC2-like TF (JAM1), bHLH13/JAM2, bHLH3/JAM3, and bHLH14, interact with JAZs and function redundantly as transcriptional repressors, which antagonize the transcription activators (e.g. MYC2, WD-repeat/bHLH/MYB complexes) via competitively binding to the G-box of the mutual target genes (e.g. *TAT1*, *DFR*), and inhibit various JA responses, including anthocyanin biosynthesis, root growth inhibition, trichome formation, stamen development, flowering, chlorophyll degradation, resistance to necrotrophic pathogens, and defense against insect attack (Fig. 3) (Nakata *et al.*, 2013; Sasaki-Sekimoto *et al.*, 2013; Fonseca *et al.*, 2014; Song *et al.*, 2014*b*). The existence of the JA-activated IIId bHLH factors is a strategy for plants to avoid overstunted growth due to excess defense response.

Other JA signaling repressors. Arabidopsis WRKY57 binds to and represses the promoters of senescence-associated genes (e.g. *SAG12* and *SEN4*) to inhibit leaf senescence, and mediates auxin/JA crosstalk in leaf senescence via interactions with JAZ4/JAZ8 and IAA29 (Jiang *et al.*, 2014). The VQ protein JASMONATE-ASSOCIATED VQ MOTIF GENE (JAV1) acts as a repressor that only inhibits plant defense against herbivores and necrotrophic pathogens, but not JA-regulated growth (Hu *et al.*, 2013), indicating that plants can distinguish the signaling branch of defense responses from that of growth.

Conclusions and future perspectives

The homeostasis among active and inactive JA compounds contributes to fine tuning in regulation of JA responses. Even though the number of these compounds has already been increased, new and more sensitive methods for analysis of different jasmonate compounds will lead to identification of new active and inactive JA derivatives. The increased knowledge on gene families in biosynthesis and metabolism of JA led to improved understanding of intracellular and whole plant JA responses. Future work will show how different JA compounds are involved in systemic responses. The hierarchical positive regulators, negative regulators, transcription activators, and repressors collaborate to regulate different JA responses synergistically and antagonistically and mediate the synergy/antagonism of crosstalks and plant–environment interaction. Studies on such aspects will increase our knowledge on the specificity and redundancy in action of the different JA compounds. The co-existence of members of various positive/negative regulators and transcription activators/repressors in JA signaling network improves the complexity and stability of JA action, and adaptability of plants. The research on JA signaling in Arabidopsis and tomato has helped to reveal the scenario of JA signaling. Further studies in crops and medicinal herbs will broaden our knowledge on JA function and signaling events, and help agricultural production and even human health. New future perspectives in JA research will be drawn from studies on plant–plant, plant–herbivore, and plant–microbe interactions, and will lead to increased applications.

Acknowledgements

We are grateful for financial support from the Palacky University Olomouc, Czech Republic, from the program Interhana: Operational Program Education for Competitiveness–European Social Fund (project CZ.1.07/2.3.00/20.0165). We thank O. Blahousek (Palacky University, Olomouc, Czech Republic) and B. Hause (IPB, Halle, Germany) for modification of Fig. 1 and Fig. 2, respectively

References

Acosta IF, Farmer EE. 2010. Jasmonates. Arabidopsis Book **8,** 1–13.

Aldrige D, Galt S, Giles D, Turner W. 1971. Metabolites of Lasiodiplodia theobromae. Journal of the Chemical Society C: Organic 1623–1627.

Aleman F, Yazaki J, Lee M, Takahashi Y, Kim AY, Li Z, Kinoshita T, Ecker JR, Schroeder JI. 2016. An ABA-increased interaction of the PYL6 ABA receptor with MYC2 transcription factor: a putative link of ABA and JA signaling. Scientific Reports **6,** 28941.

Andolfi A, Maddau L, Cimmino A, Linaldeddu BT, Basso S, Deidda A, Serra S, Evidente A. 2014. Lasiojasmonates A–C, three jasmonic acid esters produced by Lasiodiplodia sp., a grapevine pathogen. Phytochemistry **103,** 145–153.

Andreou A, Brodhun F, Feussner I. 2009. Biosynthesis of oxylipins in non-mammals. Progress in Lipid Research **48,** 148–170.

Andreou A, Feussner I. 2009. Lipoxygenases—structure and reaction mechanism. Phytochemistry **70,** 1504–1510.

Arnold A, Gruber C, Flokova K, Miersch O, Strnad M, Novak O, Wasternack C, Hause B. 2016 The recently identified isoleucine conjugate of *cis*-12-oxo-phytodienoic acid is partially active in cis-12-oxo-phytodienoic acid-specific gene expression of *Arabidopsis thaliana*. PLoS One **11,** e0162829.

Bachmann A, Hause B, Maucher H, Garbe E, Vörös K, Weichert H, Wasternack C, Feussner I. 2002. Jasmonate-induced lipid peroxidation in barley leaves initiated by distinct 13-LOX forms of chloroplasts. Journal of Biological Chemistry **383,** 1645–1657.

Bonaventure G, Schuck S, Baldwin IT. 2011. Revealing complexity and specificity in the activation of lipase-mediated oxylipin biosynthesis: a specific role of the *Nicotiana attenuata* GLA1 lipase in the activation

of jasmonic acid biosynthesis in leaves and roots. Plant, Cell and Environment **34,** 1507–1520.

Bones AM, Rossiter JT. 2006. The enzymic and chemically induced decomposition of glucosinolates. Phytochemistry **67,** 1053–1067.

Bosch M, Wright LP, Gershenzon J, Wasternack C, Hause B, Schaller A, Stintzi A. 2014. Jasmonic acid and its precursor 12-oxophytodienoic acid control different aspects of constitutive and induced herbivore defenses in tomato. Plant Physiology **166,** 396–410.

Boter M, Golz JF, Giménez-Ibañez S, Fernandez-Barbero G, Franco-Zorrilla JM, Solano R. 2015. FILAMENTOUS FLOWER is a direct target of JAZ3 and modulates responses to jasmonate. The Plant Cell **27,** 3160–3174.

Brash A, Baertschi S, Ingram C, Harris T. 1988. Isolation and characterization of natural allene oxides: unstable intermediates in the metabolism of lipid hydroperoxides. Proceedings of the National Academy of Sciences, USA **85,** 3382–3386.

Brash AR. 2009. Mechanistic aspects of CYP74 allene oxide synthases and related cytochrome P450 enzymes. Phytochemistry **70,** 1522–1531.

Brodhun F, Cristobal-Sarramian A, Zabel S, Newie J, Hamberg M, Feussner I. 2013. An iron 13S-lipoxygenase with an α-linolenic acid specific hydroperoxidase activity from Fusarium oxysporum. PLoS One **8,** e64919.

Browse J. 2009*a*. Jasmonate passes muster: a receptor and targets for the defense hormone. Annual Review of Plant Biology **60,** 183–205.

Browse J. 2009*b*. The power of mutants for investigating jasmonate biosynthesis and signaling. Phytochemistry **70,** 1539–1546.

Bruce TJA, Matthes MC, Chamberlain K, *et al*. 2008. *cis*-Jasmone induces *Arabidopsis* genes that affect the chemical ecology of multitrophic interactions with aphids and their parasitoids. Proceedings of the National Academy of Sciences, USA **105,** 4553–4558.

Bruckhoff V, Haroth S, Feussner K, König S, Brodhun F, Feussner I. 2016. Functional characterization of CYP94-genes and identification of a novel jasmonate catabolite in flowers. PLoS One **11,** e0159875.

Cai XT, Xu P, Zhao PX, Liu R, Yu LH, Xiang CB. 2014. *Arabidopsis* ERF109 mediates cross-talk between jasmonic acid and auxin biosynthesis during lateral root formation. Nature Communications **5,** 5833.

Campos ML, Kang JH, Howe GA. 2014. Jasmonate-triggered plant immunity. Journal of Chemical Ecology **40,** 657–675.

Campos ML, Yoshida Y, Major IT, *et al*. 2016. Rewiring of jasmonate and phytochrome B signalling uncouples plant growth–defense tradeoffs. Nature Communications **7,** 12570.

Cecchetti V, Altamura MM, Falasca G, Costantino P, Cardarelli M. 2008. Auxin regulates Arabidopsis anther dehiscence, pollen maturation, and filament elongation. The Plant Cell **20,** 1760–1774.

Çevik V, Kidd BN, Zhang P, *et al*. 2012. MEDIATOR25 acts as an integrative hub for the regulation of jasmonate-responsive gene expression in Arabidopsis. Plant Physiology **160,** 541–555.

Chauvin A, Lenglet A, Wolfender J-L, Farmer EE. 2016. Paired hierarchical organization of 13-lipoxygenases in Arabidopsis. Plants (Basel) **5,** 16.

Chehab EW, Kim S, Savchenko T, Kliebenstein D, Dehesh K, Braam J. 2011. Intronic T-DNA insertion renders Arabidopsis opr3 a conditional jasmonic acid-producing mutant. Plant Physiology **156,** 770–778.

Chen Q, Sun J, Zhai Q, *et al*. 2011. The basic helix–loop–helix transcription factor MYC2 directly represses *PLETHORA* expression during jasmonate-mediated modulation of the root stem cell niche in Arabidopsis. The Plant Cell **23,** 3335–3352.

Chen R, Jiang H, Li L, *et al*. 2012. The Arabidopsis mediator subunit MED25 differentially regulates jasmonate and abscisic acid signaling through interacting with the MYC2 and ABI5 transcription factors. The Plant Cell **24,** 2898–2916.

Chen Z, Chen X, Yan H, Li W, Li Y, Cai R, Xiang Y. 2015. The lipoxygenase gene family in poplar: identification, classification, and expression in response to MeJA treatment. PLoS One **10,** e0125526.

Cheng H, Song S, Xiao L, Soo HM, Cheng Z, Xie D, Peng J. 2009. Gibberellin acts through jasmonate to control the expression of MYB21, MYB24, and MYB57 to promote stamen filament growth in Arabidopsis. PLoS Genetics **5,** e1000440.

Chico JM, Fernández-Barbero G, Chini A, Fernández-Calvo P, Díez-Díaz M, Solano R. 2014. Repression of jasmonate-dependent defenses by shade involves differential regulation of protein stability of MYC transcription factors and their JAZ repressors in Arabidopsis. The Plant Cell **26,** 1967–1980.

Chini A, Fonseca S, Fernandez G, *et al*. 2007. The JAZ family of repressors is the missing link in jasmonate signalling. Nature **448,** 666–671.

Christensen SA, Huffaker A, Kaplan F, *et al*. 2015. Maize death acids, 9-lipoxygenase-derived cyclopente(a)nones, display activity as cytotoxic phytoalexins and transcriptional mediators. Proceedings of the National Academy of Sciences, USA **112,** 11407–11412.

Chung HS, Cooke TF, Depew CL, Patel LC, Ogawa N, Kobayashi Y, Howe GA. 2010. Alternative splicing expands the repertoire of dominant JAZ repressors of jasmonate signaling. The Plant Journal. **63,** 613–622.

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

Chung HS, Niu Y, Browse J, Howe GA. 2009. Top hits in contemporary JAZ: an update on jasmonate signaling. Phytochemistry **70,** 1547–1559.

Cruz Castillo M, Martínez C, Buchala A, Métraux JP, León J. 2004. Gene-specific involvement of beta-oxidation in wound-activated responses in Arabidopsis. Plant Physiology **135,** 85–94.

Dathe W, Rönsch H, Preiss A, Schade W, Sembdner G, Schreiber K. 1981. Endogenous plant hormones of the broad bean, Vicia faba L. (–)-jasmonic acid, a plant growth inhibitor in pericarp. Planta **153,** 530–535.

Demianski AJ, Chung KM, Kunkel BN. 2012. Analysis of Arabidopsis JAZ gene expression during Pseudomonas syringae pathogenesis. Molecular Plant Pathology **13,** 46–57.

Demole E, Lederer E, Mercier D. 1962. Isolement et détermination de la structure du jasmonate de méthyle, constituant odorant charactéristique de lèssence de jasmin. Helvetica Chimica Acta **45,** 675–685.

Ding H, Lai J, Wu Q, Zhang S, Chen L, Dai Y-S, Wang C, Du J, Xiao S, Yang C. 2016. Jasmonate complements the function of Arabidopsis lipoxygenase3 in salinity stress response. Plant Science **244,** 1–7.

Dombrecht B, Xue GP, Sprague SJ, *et al*. 2007. MYC2 differentially modulates diverse jasmonate-dependent functions in Arabidopsis. The Plant Cell **19,** 2225–2245.

Ellinger D, Stingl N, Kubigsteltig II, Bals T, Juenger M, Pollmann S, Berger S, Schuenemann D, Mueller MJ. 2010. DONGLE and DEFECTIVE IN ANTHER DEHISCENCE1 lipases are not essential for wound- and pathogen-induced jasmonate biosynthesis: redundant lipases contribute to jasmonate formation. Plant Physiology **153,** 114–127.

Farmaki T, Sanmartín M, Jiménez P, Paneque M, Sanz C, Vancanneyt G, León J, Sánchez-Serrano JJ. 2007. Differential distribution of the lipoxygenase pathway enzymes within potato chloroplasts. Journal of Experimental Botany **58,** 555–568.

Farmer EE, Ryan CA. 1990. Interplant communication: airborne methyl jasmonate induces synthesis of proteinase inhibitors in plant leaves. Proceedings of the National Academy of Sciences, USA **87,** 7713–7716.

Fernández-Calvo P, Chini A, Fernández-Barbero G, *et al*. 2011. The Arabidopsis bHLH transcription factors MYC3 and MYC4 are targets of JAZ repressors and act additively with MYC2 in the activation of jasmonate responses. The Plant Cell **23,** 701–715.

Feussner I, Hause B, Vörös K, Parthier B, Wasternack C. 1995. Jasmonate-induced lipoxygenase forms are localized in chloroplasts of barley leaves (*Hordeum vulgare* cv. Salome). The Plant Journal **7,** 949–957.

Feussner I, Wasternack C. 2002. The lipoxygenase pathway. Annual Review of Plant Biology **53,** 275–297.

Feys B, Benedetti CE, Penfold CN, Turner JG. 1994. Arabidopsis mutants selected for resistance to the phytotoxin coronatine are male sterile, insensitive to methyl jasmonate, and resistant to a bacterial pathogen. The Plant Cell **6,** 751–759.

Figueroa P, Browse J. 2015. Male sterility in Arabidopsis induced by overexpression of a MYC5–SRDX chimeric repressor. The Plant Journal **81,** 849–860.

Floková K, Feussner K, Herrfurth C, Miersch O, Tarkowská D, Strnad M, Feussner I, Wasternack C, Novák O. 2016. A novel jasmonate compound is hidden in flowering Arabidopsis thaliana—a story of *cis*-(+)-OPDA-Ile identification. Phytochemistry **122,** 230–237.

Fonseca S, Chini A, Hamberg M, Adie B, Porzel A, Kramell R, Miersch O, Wasternack C, Solano R. 2009. (+)-7-iso-Jasmonoyl-L-isoleucine is the endogenous bioactive jasmonate. Nature Chemical Biology **5,** 344–350.

Fonseca S, Fernández-Calvo P, Fernández G, *et al*. 2014. bHLH003, bHLH013 and bHLH017 are new targets of JAZ repressors negatively regulating JA responses. PLoS One **9,** e86182.

Froehlich JE, Itoh A, Howe GA. 2001. Tomato allene oxide synthase and fatty acid hydroperoxide lyase, two cytochrome P450s involved in oxylipin metabolism, are targeted to different membranes of chloroplast envelope. Plant Physiology **125,** 306–317.

Fukumoto K, Alamgir K, Yamashita Y, Mori IC, Matsuura H, Galis I. 2013. Response of rice to insect elicitors and the role of OsJAR1 in wound and herbivory-induced JA-Ile accumulation. Journal of Integrative Plant Biology **55,** 775–784.

Gasperini D, Chauvin A, Acosta IF, Kurenda A, Stolz S, Chételat A, Wolfender JL, Farmer EE. 2015*a*. Axial and radial oxylipin transport. Plant Physiology **169,** 2244–2254.

Gasperini D, Chételat A, Acosta IF, Goossens J, Pauwels L, Goossens A, Dreos R, Alfonso E, Farmer EE. 2015*b*. Multilayered organization of jasmonate signalling in the regulation of root growth. PLoS Genetics **11,** e1005300.

Gfeller A, Baerenfaller K, Loscos J, Chételat A, Baginsky S, Farmer EE. 2011. Jasmonate controls polypeptide patterning in undamaged tissue in wounded Arabidopsis leaves. Plant Physiology **156,** 1797–1807.

Gidda SK, Miersch O, Levitin A, Schmidt J, Wasternack C, Varin L. 2003. Biochemical and molecular characterization of a hydroxyjasmonate sulfotransferase from Arabidopsis thaliana. Journal of Biological Chemistry **278,** 17895–17900.

Gimenez-Ibanez S, Boter M, Fernández-Barbero G, Chini A, Rathjen JP, Solano R. 2014. The bacterial effector HopX1 targets JAZ transcriptional repressors to activate jasmonate signaling and promote infection in Arabidopsis. PLoS Biology **12,** e1001792.

Glauser G, Dubugnon L, Mousavi SA, Rudaz S, Wolfender JL, Farmer EE. 2009. Velocity estimates for signal propagation leading to systemic jasmonic acid accumulation in wounded Arabidopsis. Journal of Biological Chemistry **284,** 34506–34513.

Glauser G, Grata E, Dubugnon L, Rudaz S, Farmer EE, Wolfender JL. 2008. Spatial and temporal dynamics of jasmonate synthesis and accumulation in Arabidopsis in response to wounding. Journal of Biological Chemistry **283,** 16400–16407.

Göbel C, Feussner I. 2009. Methods for the analysis of oxylipins in plants. Phytochemistry **70,** 1485–1503.

Goossens J, Swinnen G, Vanden Bossche R, Pauwels L, Goossens A. 2015. Change of a conserved amino acid in the MYC2 and MYC3 transcription factors leads to release of JAZ repression and increased activity. New Phytologist **206,** 1229–1237.

Grebner W, Stingl NE, Oenel A, Mueller MJ, Berger S. 2013. Lipoxygenase6-dependent oxylipin synthesis in roots is required for abiotic and biotic stress resistance of Arabidopsis. Plant Physiology **161,** 2159–2170.

Gu XC, Chen JF, Xiao Y, Di P, Xuan HJ, Zhou X, Zhang L, Chen WS. 2012. Overexpression of allene oxide cyclase promoted tanshinone/phenolic acid production in Salvia miltiorrhiza. Plant Cell Reports **31,** 2247–2259.

Gundlach H, Müller M, Kutchan T, Zenk M. 1992. Jasmonic acid is a signal transducer in elicitor-induced plant cell cultures. Proceedings of the National Academy of Sciences, USA **89,** 2389–2393.

Haga K, Iino M. 2004. Phytochrome-mediated transcriptional up-regulation of ALLENE OXIDE SYNTHASE in rice seedlings. Plant and Cell Physiology **45,** 119–128.

Hannapel DJ. 2010. A model system of development regulated by the long-distance transport of mRNA. Journal of Integrative Plant Biology **52,** 40–52.

Hazman M, Hause B, Eiche E, Nick P, Riemann M. 2015. Increased tolerance to salt stress in OPDA-deficient rice ALLENE OXIDE CYCLASE mutants is linked to an increased ROS-scavenging activity. Journal of Experimental Botany **66,** 3339–3352.

Heitz T, Smirnova E, Widemann E, Aubert Y, Pinot F, Ménard R. 2016. The rise and fall of jasmonate biological activities. In: Nakamura Y, Li-Beisson Y, eds. Lipids in plant and algae development. Cham: Springer International Publishing, 405–426.

Heitz T, Widemann E, Lugan R, *et al*. 2012. Cytochromes P450 CYP94C1 and CYP94B3 catalyze two successive oxidation steps of plant hormone jasmonoyl-isoleucine for catabolic turnover. Journal of Biological Chemistry **287,** 6296–6306.

Hibara K, Isono M, Mimura M, *et al*. 2016. Jasmonate regulates juvenile-to-adult phase transition in rice. Development **143,** 3407–3416.

Hirschmann F, Krause F, Papenbrock J. 2014. The multi-protein family of sulfotransferases in plants: composition, occurrence, substrate specificity, and functions. Frontiers in Plant Science **5,** 556.

Hofmann E, Pollmann S. 2008. Molecular mechanism of enzymatic allene oxide cyclization in plants. Plant Physiology and Biochemistry **46,** 302–308.

Hofmann E, Zerbe P, Schaller F. 2006. The crystal structure of Arabidopsis thaliana allene oxide cyclase: insights into the oxylipin cyclization reaction. The Plant Cell **18,** 3201–3217.

Hong GJ, Xue XY, Mao YB, Wang LJ, Chen XY. 2012. Arabidopsis MYC2 interacts with DELLA proteins in regulating sesquiterpene synthase gene expression. The Plant Cell **24,** 2635–2648.

Hou X, Lee LY, Xia K, Yan Y, Yu H. 2010. DELLAs modulate jasmonate signaling via competitive binding to JAZs. Developmental Cell **19,** 884–894.

Hu J, Baker A, Bartel B, Linka N, Mullen RT, Reumann S, Zolman BK. 2012. Plant peroxisomes: biogenesis and function. The Plant Cell **24,** 2279–2303.

Hu Y, Jiang L, Wang F, Yu D. 2013. Jasmonate regulates the inducer of cbf expression-C-repeat binding factor/DRE binding factor1 cascade and freezing tolerance in Arabidopsis. The Plant Cell **25,** 2907–2924.

Hughes RK, De Domenico S, Santino A. 2009. Plant cytochrome CYP74 family: biochemical features, endocellular localisation, activation mechanism in plant defence and improvements for industrial applications. ChemBioChem **10,** 1122–1133.

Hyun Y, Choi S, Hwang H-J, *et al*. 2008. Cooperation and functional diversification of two closely related galactolipase genes for jasmonate biosynthesis. Developmental Cell **14,** 183–192.

Ibrahim A, Schütz AL, Galano JM, Herrfurth C, Feussner K, Durand T, Brodhun F, Feussner I. 2011. The alphabet of galactolipids in Arabidopsis thaliana. Frontiers in Plant Science **2,** 95.

Ishiguro S, Kwai-Oda A, Ueda J, Nishida I, Okada K. 2001. The *DEFECTIVE IN ANTHER DEHISCENCE1* gene encodes a novel phospholipase A1 catalyzing the initial step of jasmonic acid biosynthesis, which synchronizes pollen maturation. The Plant Cell **13,** 2191–2209.

Ito T, Ng KH, Lim TS, Yu H, Meyerowitz EM. 2007. The homeotic protein AGAMOUS controls late stamen development by regulating a jasmonate biosynthetic gene in Arabidopsis. The Plant Cell **19,** 3516–3529.

Jiang S, Yao J, Ma KW, Zhou H, Song J, He SY, Ma W. 2013. Bacterial effector activates jasmonate signaling by directly targeting JAZ transcriptional repressors. PLoS Pathogens **9,** e1003715.

Jiang Y, Liang G, Yang S, Yu D. 2014. Arabidopsis WRKY57 functions as a node of convergence for jasmonic acid- and auxin-mediated signaling in jasmonic acid-induced leaf senescence. The Plant Cell **26,** 230–245.

Jimenez-Aleman GH, Machado RA, Görls H, Baldwin IT, Boland W. 2015. Synthesis, structural characterization and biological activity of two diastereomeric JA-Ile macrolactones. Organic and Biomolecular Chemistry **13,** 5885–5893.

Jung C, Zhao P, Seo JS, Mitsuda N, Deng S, Chua NH. 2015. PLANT U-BOX PROTEIN10 regulates MYC2 stability in Arabidopsis. The Plant Cell **27,** 2016–2031.

Katsir L, Schilmiller AL, Staswick PE, He SY, Howe GA. 2008. COI1 is a critical component of a receptor for jasmonate and the bacterial virulence factor coronatione. Proceedings of the National Academy of Sciences, USA **105,** 7100–7105.

Kazan K, Lyons R. 2014. Intervention of phytohormone pathways by pathogen effectors. The Plant Cell **26,** 2285–2309.

Kazan K, Manners JM. 2011. The interplay between light and jasmonate signalling during defence and development. Journal of Experimental Botany **62,** 4087–4100.

Kazan K, Manners JM. 2013. MYC2: the master in action. Molecular Plant **6,** 686–703.

Kidd BN, Edgar CI, Kumar KK, Aitken EA, Schenk PM, Manners JM, Kazan K. 2009. The mediator complex subunit PFT1 is a key regulator of jasmonate-dependent defense in Arabidopsis. The Plant Cell **21,** 2237–2252.

Knopf RR, Feder A, Mayer K, Lin A, Rozenberg M, Schaller A, Adam Z. 2012. Rhomboid proteins in the chloroplast envelope affect the level of allene oxide synthase in *Arabidopsis thaliana*. The Plant Journal **72,** 559–571.

Koeduka T, Ishizaki K, Mwenda CM, Hori K, Sasaki-Sekimoto Y, Ohta H, Kohchi T, Matsui K. 2015. Biochemical characterization of allene oxide synthases from the liverwort Marchantia polymorpha and green microalgae Klebsormidium flaccidum provides insight into the evolutionary divergence of the plant CYP74 family. Planta **242,** 1175–1186.

Kombrink E. 2012. Chemical and genetic exploration of jasmonate biosynthesis and signaling paths. Planta **236,** 1351–1366.

Koo AJ, Chung HS, Kobayashi Y, Howe GA. 2006. Identification of a peroxisomal acyl-activating enzyme involved in the biosynthesis of jasmonic acid in Arabidopsis. Journal of Biological Chemistry **281,** 33511–33520.

Koo AJ, Howe GA. 2012. Catabolism and deactivation of the lipid-derived hormone jasmonoyl-isoleucine. Frontiers in Plant Science **3,** 19.

Koo AJ, Thireault C, Zemelis S, Poudel AN, Zhang T, Kitaoka N, Brandizzi F, Matsuura H, Howe GA. 2014. Endoplasmic reticulum-associated inactivation of the hormone jasmonoyl-L-isoleucine by multiple members of the cytochrome P450 94 family in Arabidopsis. Journal of Biological Chemistry **289,** 29728–29738.

Koo AJK, Cooke TF, Howe GA. 2011. Cytochrome P450 CYP94B3 mediates catabolism and inactivation of the plant hormone jasmonoyl-L-isoleucine. Proceedings of the National Academy of Sciences, USA **108,** 9298–9303.

Koprivova A, Kopriva S. 2016. Sulfation pathways in plants. Chemico-Biological Interactions. May 17. pii: S0009-2797(16)30191-0. doi:10.1016/j.cbi.2016.05.021.

Larrieu A, Champion A, Legrand J, *et al*. 2015. A fluorescent hormone biosensor reveals the dynamics of jasmonate signalling in plants. Nature Communications **6,** 6043.

Laudert D, Schaller F, Weiler EW. 2000. Transgenic Nicotiana tabacum and Arabidopsis thaliana plants overexpressing allene oxide synthase. Planta **211,** 163–165.

Lee D, Polisensky DH, Braam J. 2005. Genome-wide identification of touch- and darkness-regulated Arabidopsis genes: a focus on calmodulin-like and XTH genes. New Phytologist **165,** 429–444.

Lee DS, Nioche P, Hamberg M, Raman CS. 2008. Structural insights into the evolutionary paths of oxylipin biosynthetic enzymes. Nature **455,** 363–368.

Li C, Schilmiller AL, Liu G, *et al*. 2005. Role of β-oxidation in jasmonate biosynthesis and systemic wound signaling in tomato. The Plant Cell **17,** 971–986.

Li L, Chang Z, Pan Z, Fu Z-Q, Wang X. 2008. Modes of heme binding and substrate access for cytochrome P450 CYP74A revealed by crystal structures of allene oxide synthase. Proceedings of the National Academy of Sciences, USA **105,** 13883–13888.

Li R, Weldegergis BT, Li J, *et al*. 2014. Virulence factors of geminivirus interact with MYC2 to subvert plant resistance and promote vector performance. The Plant Cell **26,** 4991–5008.

Li W, Zhou F, Liu B, Feng D, He Y, Qi K, Wang H, Wang J. 2011. Comparative characterization, expression pattern and function analysis of the 12-oxo-phytodienoic acid reductase gene family in rice. Plant Cell Reports **30,** 981–995.

Li X-R, Li H-J, Yuan L, Liu M, Shi D-Q, Liu J, Yang W-C. 2014. Arabidopsis DAYU/ABERRANT PEROXISOME MORPHOLOGY9 is a key regulator of peroxisome biogenesis and plays critical roles during pollen maturation and germination in planta. The Plant Cell **26,** 619–635.

Lin Y-T, Chen L-J, Herrfurth C, Feussner I, Li HM. 2016. Reduced biosynthesis of digalactosyldiacylglycerol, a major chloroplast membrane lipid, leads to oxylipin overproduction and phloem cap lignification in Arabidopsis. The Plant Cell **28,** 219–232.

Liu X, Li F, Tang J, *et al*. 2012. Activation of the jasmonic acid pathway by depletion of the hydroperoxide lyase OsHPL3 reveals crosstalk between the HPL and AOS branches of the oxylipin pathway in rice. PLoS One **7,** e50089.

Lorenzo O, Chico JM, Sánchez-Serrano JJ, Solano R. 2004. JASMONATE-INSENSITIVE1 encodes a MYC transcription factor essential to discriminate between different jasmonate-regulated defense responses in Arabidopsis. The Plant Cell **16,** 1938–1950.

Luo J, Wei K, Wang S, *et al*. 2016. COI1-regulated hydroxylation of jasmonoyl-L-isoleucine impairs Nicotiana attenuata's resistance to the generalist herbivore Spodoptera litura. Journal of Agricultural and Food Chemistry **64,** 2822–2831.

Mandaokar A, Browse J. 2009. MYB108 acts together with MYB24 to regulate jasmonate-mediated stamen maturation in Arabidopsis. Plant Physiology **149,** 851–862.

Mandaokar A, Thines B, Shin B, *et al*. 2006. Transcriptional regulators of stamen development in Arabidopsis identified by transcriptional profiling. The Plant Journal **46,** 984–1008.

Matthes MC, Bruce TJ, Ton J, Verrier PJ, Pickett JA, Napier JA. 2010. The transcriptome of cis-jasmone-induced resistance in Arabidopsis thaliana and its role in indirect defence. Planta **232,** 1163–1180.

Maucher H, Hause B, Feussner I, Ziegler J, Wasternack C. 2000. Allene oxide synthases of barley (*Hordeum vulgare* cv. Salome): tissue specific regulation in seedling development. The Plant Journal **21,** 199–213.

Meesters C, Mönig T, Oeljeklaus J, Krahn D, Westfall CS, Hause B, Jez JM, Kaiser M, Kombrink E. 2014. A chemical inhibitor of jasmonate signaling targets JAR1 in Arabidopsis thaliana. Nature Chemical Biology **10,** 830–836.

Melotto M, Mecey C, Niu Y, *et al*. 2008. A critical role of two positively charged amino acids in the Jas motif of Arabodopsis JAZ proteins in mediating coronatine- and jasmonoyl isoleucine-dependent intreractions with the COI1 F-box protein. The Plant Journal **55,** 979–988.

Mertens J, Pollier J, Vanden Bossche R, Lopez-Vidriero I, Franco-Zorrilla JM, Goossens A. 2016. The bHLH transcription factors TSAR1 and TSAR2 regulate triterpene saponin biosynthesis in Medicago truncatula. Plant Physiology **170,** 194–210.

Meyer A, Miersch O, Büttner C, Dathe W, Sembdner G. 1984. Occurrence of the plant growth regulator jasmonic acid in plants. Journal of Plant Growth Regulation **3,** 1–8.

Miersch O, Bohlmann H, Wasternack C. 1999. Jasmonates and related compounds from *Fusarium oxysporum*. Phytochemistry **50,** 517–523.

Miersch O, Neumerkel J, Dippe M, Stenzel I, Wasternack C. 2008. Hydroxylated jasmonates are commonly occurring metabolites of jasmonic acid and contribute to a partial switch-off in jasmonate signaling. New Phytologist **177,** 114–127.

Miersch O, Preiss A, Sembdner G, Schreiber K. 1987. (+)-*7-iso*-Jasmonic acid and related compounds from Botryodiplodia theobromae. Phytochemistry **26,** 1037–1039.

Miersch O, Wasternack C. 2000. Octadecanoid and jasmonate signaling in tomato (Lycopersicon esculentum Mill.) leaves: endogenous jasmonates do not induce jasmonate biosynthesis. Biological Chemistry **381,** 715–722.

Moffat CS, Ingle RA, Wathugala DL, Saunders NJ, Knight H, Knight MR. 2012. ERF5 and ERF6 play redundant roles as positive regulators of JA/Et-mediated defense against Botrytis cinerea in Arabidopsis. PLoS One **7,** e35995.

Monte I, Hamberg M, Chini A, Gimenez-Ibanez S, García-Casado G, Porzel A, Pazos F, Boter M, Solano R. 2014. Rational design of a ligand-based antagonist of jasmonate perception. Nature Chemical Biology **10,** 671–676.

Montiel G, Zarei A, Körbes AP, Memelink J. 2011. The jasmonate-responsive element from the ORCA3 promoter from Catharanthus roseus is active in Arabidopsis and is controlled by the transcription factor AtMYC2. Plant and Cell Physiology **52,** 578–587.

Mousavi SA, Chauvin A, Pascaud F, Kellenberger S, Farmer EE. 2013. GLUTAMATE RECEPTOR-LIKE genes mediate leaf-to-leaf wound signalling. Nature **500,** 422–426.

Mugford SG, Yoshimoto N, Reichelt M, *et al*. 2009. Disruption of adenosine-5'-phosphosulfate kinase in Arabidopsis reduces levels of sulfated secondary metabolites. The Plant Cell **21,** 910–927.

Mueller-Uri F, Parthier B, Nover L. 1988. Jasmonate-induced alteration of gene expression in barley leaf segments analyzed by in-vivo and in-vitro protein synthesis. Planta **176,** 241–247.

Nagpal P, Ellis CM, Weber H, *et al*. 2005. Auxin response factors ARF6 and ARF8 promote jasmonic acid production and flower maturation. Development **132,** 4107–4118.

Nakamura Y, Mithöfer A, Kombrink E, Boland W, Hamamoto S, Uozumi N, Tohma K, Ueda M. 2011. 12-Hydroxyjasmonic acid glucoside is a COI1–JAZ-independent activator of leaf-closing movement in Samanea saman. Plant Physiology **155,** 1226–1236.

Nakata M, Mitsuda N, Herde M, Koo AJ, Moreno JE, Suzuki K, Howe GA, Ohme-Takagi M. 2013. A bHLH-type transcription factor, ABA-INDUCIBLE BHLH-TYPE TRANSCRIPTION FACTOR/JA-ASSOCIATED MYC2-LIKE1, acts as a repressor to negatively regulate jasmonate signaling in Arabidopsis. The Plant Cell **25,** 1641–1656.

Neumann P, Brodhun F, Sauer K, *et al*. 2012. Crystal structures of Physcomitrella patens AOC1 and AOC2: insights into the enzyme mechanism and differences in substrate specificity. Plant Physiology **160,** 1251–1266.

Newcomer ME, Brash AR. 2015. The structural basis for specificity in lipoxygenase catalysis. Protein Science **24,** 298–309.

Newie J, Andreou A, Neumann P, Einsle O, Feussner I, Ficner R. 2016. Crystal structure of a lipoxygenase from Cyanothece sp. may reveal novel features for substrate acquisition. Journal of Lipid Research **57,** 276–287.

Nilsson AK, Fahlberg P, Ellerström M, Andersson MX. 2012. Oxo-phytodienoic acid (OPDA) is formed on fatty acids esterified to galactolipids after tissue disruption in Arabidopsis thaliana. FEBS Letters **586,** 2483–2487.

Nilsson AK, Fahlberg P, Johansson ON, Hamberg M, Andersson MX, Ellerström M. 2016. The activity of HYDROPEROXIDE LYASE 1 regulates accumulation of galactolipids containing 12-oxo-phytodienoic acid in Arabidopsis. Journal of Experimental Botany **67,** 5133–5144.

Nilsson AK, Johansson ON, Fahlberg P, *et al*. 2015. Acylated monogalactosyl diacylglycerol: prevalence in the plant kingdom and identification of an enzyme catalyzing galactolipid head group acylation in Arabidopsis thaliana. The Plant Journal **84,** 1152–1166.

Ogorodnikova AV, Mukhitova FK, Grechkin AN. 2015. Oxylipins in the spikemoss Selaginella martensii: detection of divinyl ethers, 12-oxophytodienoic acid and related cyclopentenones. Phytochemistry **118,** 42–50.

Oluwafemi S, Dewhirst SY, Veyrat N, Powers S, Bruce TJ, Caulfield JC, Pickett JA, Birkett MA. 2013. Priming of production in maize of volatile organic defence compounds by the natural plant activator cis-jasmone. PLoS One **8,** e62299.

Otto M, Naumann C, Brandt W, Wasternack C, Hause B. 2016. Activity regulation by heteromerization of arabidopsis allene oxide cyclase family members. Plants **5,** 3.

Ozalvo R, Cabrera J, Escobar C, *et al*. 2014. Two closely related members of Arabidopsis 13-lipoxygenases (13-LOXs), LOX3 and LOX4, reveal distinct functions in response to plant–parasitic nematode infection. Molecular Plant Pathology **15,** 319–332.

Park J-H, Halitschke R, Kim H, Baldwin I, Feldmann K, Feyereisen R. 2002. A knock-out mutation in allene oxide synthase results in male sterility and defective wound signal transduction in Arabidopsis due to a block in jasmonic acid biosynthesis. The Plant Journal **31,** 1–12.

Patkar RN, Benke PI, Qu Z, Constance Chen YY, Yang F, Swarup S, Naqvi NI. 2015. A fungal monooxygenase-derived jasmonate attenuates host innate immunity. Nature Chemical Biology **11,** 733–740.

Pauwels L, Barbero GF, Geerinck J, *et al*. 2010. NINJA connects the co-repressor TOPLESS to jasmonate signalling. Nature **464,** 788–791.

Pauwels L, Goossens A. 2011. The JAZ proteins: a crucial interface in the jasmonate signaling cascade. The Plant Cell **23,** 3089–3100.

Peng Y-J, Shih C-F, Yang J-Y, Tan C-M, Hsu W-H, Huang Y-P, Liao P-C, Yang C-H. 2013. A RING-type E3 ligase controls anther dehiscence by activating the jasmonate biosynthetic pathway gene *DEFECTIVE IN ANTHER DEHISCENCE1* in Arabidopsis. The Plant Journal **74,** 310–327.

Pieterse CM, Van der Does D, Zamioudis C, Leon-Reyes A, Van Wees SC. 2012. Hormonal modulation of plant immunity. Annual Review of Cell and Developmental Biology **28,** 489–521.

Poudel AN, Zhang T, Kwasniewski M, Nakabayashi R, Saito K, Koo AJ. 2016. Mutations in jasmonoyl-L-isoleucine-12-hydroxylases suppress multiple JA-dependent wound responses in Arabidopsis thaliana. Biochimica et Biophysica Acta **1861,** 1396–1408.

Pré M, Atallah M, Champion A, De Vos M, Pieterse CM, Memelink J. 2008. The AP2/ERF domain transcription factor ORA59 integrates jasmonic acid and ethylene signals in plant defense. Plant Physiology **147,** 1347–1357.

Preuß A, Augustin C, Figueroa CR, Hoffmann T, Valpuesta V, Sevilla JF, Schwab W. 2014. Expression of a functional jasmonic acid carboxyl methyltransferase is negatively correlated with strawberry fruit development. Journal of Plant Physiology **171,** 1315–1324.

Qi J, Li J, Han X, Li R, Wu J, Yu H, Hu L, Xiao Y, Lu J, Lou Y. 2016. Jasmonic acid carboxyl methyltransferase regulates development and herbivory-induced defense response in rice. Journal of Integrative Plant Biology **58,** 564–576.

Qi T, Huang H, Song S, Xie D. 2015*a*. Regulation of jasmonate-mediated stamen development and seed production by a bHLH–MYB complex in Arabidopsis. The Plant Cell **27,** 1620–1633.

Qi T, Huang H, Wu D, Yan J, Qi Y, Song S, Xie D. 2014. Arabidopsis DELLA and JAZ proteins bind the WD-repeat/bHLH/MYB complex to modulate gibberellin and jasmonate signaling synergy. The Plant Cell **26,** 1118–1133.

Qi T, Song S, Ren Q, Wu D, Huang H, Chen Y, Fan M, Peng W, Ren C, Xie D. 2011. The Jasmonate-ZIM-domain proteins interact with the WD-repeat/bHLH/MYB complexes to regulate Jasmonate-mediated anthocyanin accumulation and trichome initiation in Arabidopsis thaliana. The Plant Cell **23,** 1795–1814.

Qi T, Wang J, Huang H, Liu B, Gao H, Liu Y, Song S, Xie D. 2015b. Regulation of jasmonate-induced leaf senescence by antagonism between bHLH Subgroup IIIe and IIId factors in Arabidopsis. The Plant Cell **27,** 1634–1649.

Ramsay NA, Glover BJ. 2005. MYB–bHLH–WD40 protein complex and the evolution of cellular diversity. Trends in Plant Science **10,** 63–70.

Richmond T, Bleecker A. 1999. A defect in β-oxidation causes abnormal inflorescence development in Arabidopsis. The Plant Cell **11,** 1911–1923.

Riemann M, Haga K, Shimizu T, *et al*. 2013. Identification of rice allene oxide cyclase mutants and the function of jasmonate for defence against Magnaporthe oryzae. The Plant Journal **74,** 226–238.

Ruduś I, Terai H, Shimizu T, *et al*. 2014. Wound-induced expression of DEFECTIVE IN ANTHER DEHISCENCE1 and DAD1-like lipase genes is mediated by both CORONATINE INSENSITIVE1-dependent and independent pathways in Arabidopsis thaliana. Plant Cell Reports **33,** 849–860.

Sanchez Carranza AP, Singh A, Steinberger K, Panigrahi K, Palme K, Dovzhenko A, Dal Bosco C. 2016. Hydrolases of the ILR1-like family of Arabidopsis thaliana modulate auxin response by regulating auxin homeostasis in the endoplasmic reticulum. Scientific Reports **6,** 24212.

Sasaki-Sekimoto Y, Jikumaru Y, Obayashi T, Saito H, Masuda S, Kamiya Y, Ohta H, Shirasu K. 2013. Basic helix–loop–helix transcription factors JASMONATE-ASSOCIATED MYC2-LIKE1 (JAM1), JAM2, and JAM3 are negative regulators of jasmonate responses in Arabidopsis. Plant Physiology **163,** 291–304.

Scalschi L, Sanmartin M, Camanes G, Troncho P, Sanchez-Serrano J. 2015. Silencing of OPR3 in tomato reveals the role of OPDA in callose deposition during activation of defense responses against Botrytis cinerea. The Plant Journal **81,** 304–315.

Schaller A, Stintzi A. 2009. Enzymes in jasmonate biosynthesis—structure, function, regulation. Phytochemistry **70,** 1532–1538.

Schaller F, Zerbe P, Reinbothe S, Reinbothe C, Hofmann E, Pollmann S. 2008. The allene oxide cyclase family of Arabidopsis thaliana: localization and cyclization. FEBS Journal **275,** 2428–2441.

Schilmiller AL, Koo AJK, Howe GA. 2007. Functional diversification of acyl-coenzyme A oxidases in jasmonic acid biosynthesis and action. Plant Physiology **143,** 812–824.

Schneider K, Kienow L, Schmelzer E, Colby T, Bartsch M, Miersch O, Wasternack C, Kombrink E, Stuible HP. 2005. A new type of peroxisomal acyl-coenzyme A synthetase from Arabidopsis thaliana has the catalytic capacity to activate biosynthetic precursors of jasmonic acid. Journal of Biological Chemistry **280,** 13962–13972.

Scholz J, Brodhun F, Hornung E, *et al*. 2012. Biosynthesis of allene oxides in Physcomitrella patens. BMC Plant Biology **12,** 228.

Scholz SS, Reichelt M, Boland W, Mithöfer A. 2015. Additional evidence against jasmonate-induced jasmonate induction hypothesis. Plant Science **239,** 9–14.

Schuck S, Kallenbach M, Baldwin IT, Bonaventure G. 2014. The Nicotiana attenuata GLA1 lipase controls the accumulation of Phytophthora parasitica-induced oxylipins and defensive secondary metabolites. Plant, Cell and Environment **37,** 1703–1715.

Schulze B, Dabrowska P, Boland W. 2007. Rapid enzymatic isomerization of 12-oxophytodienoic acid in the gut of Lepidopteran larvae. ChemBioChem **8,** 208–216.

Schuman MC, Baldwin IT. 2016. The layers of plant responses to insect herbivores. Annual Review of Entomology **61,** 373–394.

Schweizer F, Fernández-Calvo P, Zander M, *et al*. 2013. Arabidopsis basic helix–loop–helix transcription factors MYC2, MYC3, and MYC4 regulate glucosinolate biosynthesis, insect performance, and feeding behavior. The Plant Cell **25,** 3117–3132.

Seto Y, Hamada S, Ito H, Masuta C, Matsui H, Nabeta K, Matsuura H. 2011. Tobacco salicylic acid glucosyltransferase is active toward tuberonic acid (12-hydroxyjasmonic acid) and is induced by mechanical wounding stress. Bioscience, Biotechnology, and Biochemistry **75,** 2316–2320.

Sheard LB, Tan X, Mao H, *et al*. 2010. Jasmonate perception by inositol-phosphate-potentiated COI1–JAZ co-receptor. Nature **468,** 400–405.

Shen J, Tieman D, Jones JB, Taylor MG, Schmelz E, Huffaker A, Bies D, Chen K, Klee HJ. 2014. A 13-lipoxygenase, TomloxC, is essential for synthesis of C5 flavour volatiles in tomato. Journal of Experimental Botany **65,** 419–428.

Shih CF, Hsu WH, Peng YJ, Yang CH. 2014. The NAC-like gene ANTHER INDEHISCENCE FACTOR acts as a repressor that controls anther dehiscence by regulating genes in the jasmonate biosynthesis pathway in Arabidopsis. Journal of Experimental Botany **65,** 621–639.

Shin J, Heidrich K, Sanchez-Villarreal A, Parker JE, Davis SJ. 2012. TIME FOR COFFEE represses accumulation of the MYC2 transcription factor to provide time-of-day regulation of jasmonate signaling in Arabidopsis. The Plant Cell **24,** 2470–2482.

Shyu C, Figueroa P, Depew CL, *et al*. 2012. JAZ8 lacks a canonical degron and has an EAR motif that mediates transcriptional repression of jasmonate responses in Arabidopsis. The Plant Cell **24,** 536–550.

Song S, Huang H, Gao H, *et al*. 2014*a*. Interaction between MYC2 and ETHYLENE INSENSITIVE3 modulates antagonism between jasmonate and ethylene signaling in *Arabidopsis*. Plant Cell **26,** 263–279.

Song S, Qi T, Huang H, *et al*. 2011. The jasmonate-ZIM domain proteins interact with the R2R3-MYB transcription factors MYB21 and MYB24 to affect jasmonate-regulated stamen development in Arabidopsis. The Plant Cell **23,** 1000–1013.

Song S, Qi T, Huang H, Xie D. 2013. Regulation of stamen development by coordinated actions of jasmonate, auxin, and gibberellin in Arabidopsis. Molecular Plant **6,** 1065–1073.

Song S, Qi T, Wasternack C, Xie D. 2014*b*. Jasmonate signaling. Current Opinion in Plant Biology. **21,** 112–119.

Springer A, Kang C, Rustgi S, von Wettstein D, Reinbothe C, Pollmann S, Reinbothe S. 2016. Programmed chloroplast destruction during leaf senescence involves 13-lipoxygenase (13-LOX). Proceedings of the National Academy of Sciences, USA **113,** 3383–3388.

Staswick PE. 1990. Novel regulation of vegetative storage protein genes. The Plant Cell **2,** 1–6.

Staswick PE, Tiryaki I. 2004. The oxylipin signal jasmonic acid is activated by an enzyme that conjugates it to isoleucine in Arabidopsis. The Plant Cell **16,** 2117–2127.

Stenzel I, Hause B, Maucher H, Pitzschke A, Miersch O, Ziegler J, Ryan C, Wasternack C. 2003. Allene oxide cyclase dependence of the wound response and vascular bundle-specific generation of jasmonates in tomato—amplification in wound signaling. The Plant Journal **33,** 577–589.

Stenzel I, Otto M, Delker C, Kirmse N, Schmidt D, Miersch O, Hause B, Wasternack C. 2012. ALLENE OXIDE CYCLASE (AOC) gene family members of Arabidopsis thaliana: tissue- and organ-specific promoter activities and in vivo heteromerization. Journal of Experimental Botany **63,** 6125–6138.

Stintzi A, Browse J. 2000. The Arabidopsis male-sterile mutant, *opr3*, lacks the 12-oxophytodienoic acid reductase required for jasmonate synthesis. Proceedings of the National Academy of Sciences, USA **97,** 10625–10630.

Stitz M, Gase K, Baldwin IT, Gaquerel E. 2011. Ectopic expression of AtJMT in Nicotiana attenuata: creating a metabolic sink has tissue-specific consequences for the jasmonate metabolic network and silences downstream gene expression. Plant Physiology **157,** 341–354.

Stumpe M, Göbel C, Faltin B, *et al*. 2010. The moss Physcomitrella patens contains cyclopentenones but no jasmonates: mutations in allene oxide cyclase lead to reduced fertility and altered sporophyte morphology. New Phytologist **188,** 740–749.

Suza WP, Rowe ML, Hamberg M, Staswick PE. 2010. A tomato enzyme synthesizes (+)-7-iso-jasmonoyl-L-isoleucine in wounded leaves. Planta **231,** 717–728.

Svyatyna K, Jikumaru Y, Brendel R, Reichelt M, Mithöfer A, Takano M, Kamiya Y, Nick P, Riemann M. 2014. Light induces jasmonate–isoleucine conjugation via OsJAR1-dependent and -independent pathways in rice. Plant, Cell and Environment **37,** 827–839.

Taki N, Sasaki-Sekimoto Y, Obayashi T, *et al*. 2005. 12-Oxo-phytodienoic acid triggers expression of a distinct set of genes and plays a role in wound-induced gene expression in Arabidopsis. Plant Physiology **139,** 1268–1283.

Tani T, Sobajima H, Okada K, *et al*. 2008. Identification of the OsOPR7 gene encoding 12-oxophytodienoate reductase involved in the biosynthesis of jasmonic acid in rice. Planta **227,** 517–526.

Thatcher LF, Cevik V, Grant M, Zhai B, Jones JD, Manners JM, Kazan K. 2016. Characterization of a JAZ7 activation-tagged Arabidopsis mutant with increased susceptibility to the fungal pathogen Fusarium oxysporum. Journal of Experimental Botany **67,** 2367–2386.

Theodoulou FL, Job K, Slocombe SP, Footitt S, Holdsworth M, Baker A, Larson TR, Graham IA. 2005. Jasmonic acid levels are reduced in COMATOSE ATP-binding cassette transporter mutants. Implications for transport of jasmonate precursors into peroxisomes. Plant Physiology **137,** 835–840.

Thines B, Katsir L, Melotto M, *et al*. 2007. JAZ repressor proteins are targets of the SCF(COI1) complex during jasmonate signalling. Nature **448,** 661–665.

Thireault C, Shyu C, Yoshida Y, St. Aubin B, Campos ML, Howe GA. 2015. Repression of jasmonate signaling by a non-TIFY JAZ protein in Arabidopsis. The Plant Journal **82,** 669–679.

Toda Y, Tanaka M, Ogawa D, *et al*. 2013. RICE SALT SENSITIVE3 forms a ternary complex with JAZ and class-C bHLH factors and regulates jasmonate-induced gene expression and root cell elongation. The Plant Cell **25,** 1709–1725.

Toledo-Ortiz G, Huq E, Quail PH. 2003. The Arabidopsis basic/helix–loop–helix transcription factor family. The Plant Cell **15,** 1749–1770.

Toporkova YY, Ermilova VS, Gorina SS, Mukhtarova LS, Osipova EV, Gogolev YV, Grechkin AN. 2013. Structure–function relationship in the CYP74 family: conversion of divinyl ether synthases into allene oxide synthases by site-directed mutagenesis. FEBS Letters **587,** 2552–2558.

Ueda J, Kato J. 1980. Isolation and identification of a senescence-promoting substance from wormwood (Artemisia absinthium L.). Plant Physiology **66,** 246–249.

Ueda M, Yang G, Nukadzuka Y, Ishimaru Y, Tamura S, Manabe Y. 2015. Functional importance of the sugar moiety of jasmonic acid glucoside for bioactivity and target affinity. Organic and Biomolecular Chemistry **13,** 55–58.

Van der Does D, Leon-Reyes A, Koornneef A, *et al*. 2013. Salicylic acid suppresses jasmonic acid signaling downstream of SCF^{COI1}–JAZ by targeting GCC promoter motifs via transcription factor ORA59. The Plant Cell **25,** 744–761.

Van Moerkercke A, Steensma P, Gariboldi I, *et al*. 2016. The basic helix–loop–helix transcription factor BIS2 is essential for monoterpenoid indole alkaloid production in the medicinal plant Catharanthus roseus. The Plant Journal **88,** 3–12.

Van Moerkercke A, Steensma P, Schweizer F, *et al*. 2015. The bHLH transcription factor BIS1 controls the iridoid branch of the monoterpenoid indole alkaloid pathway in Catharanthus roseus. Proceedings of the National Academy of Sciences, USA **112,** 8130–8135.

Vick BA, Zimmerman DC. 1983. The biosynthesis of jasmonic acid: a physiological role for plant lipoxygenase. Biochemical and Biophysical Research Communications **111,** 470–477.

Wakuta S, Hamada S, Ito H, Matsuura H, Nabeta K, Matsui H. 2010. Identification of a beta-glucosidase hydrolyzing tuberonic acid glucoside in rice (Oryza sativa L.). Phytochemistry **71,** 1280–1288.

Wang J, Yan DW, Yuan TT, Gao X, Lu YT. 2013. A gain-of-function mutation in IAA8 alters Arabidopsis floral organ development by change of jasmonic acid level. Plant Molecular Biology **82,** 71–83.

Wasternack C. 2007. Jasmonates: an update on biosynthesis, signal transduction and action in plant stress response, growth and development. Annals of Botany **100,** 681–697.

Wasternack C. 2014. Jasmonates in plant growth and stress responses. In: **Tran L-S, Pal S**, eds. Phytohormones: a window to metabolism, signaling and biotechnological applications. New York: Springer, 221–263,

Wasternack C. 2015. How jasmonates earned their laurels: past and present. Journal of Plant Growth Regulation **34,** 761–794.

Wasternack C, Hause B. 2013. Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in Annals of Botany. Annals of Botany **111,** 1021–1058.

Wasternack C, Kombrink E. 2010. Jasmonates: structural requirements for lipid-derived signals active in plant stress responses and development. ACS Chemical Biology **5,** 63–77.

Wasternack C, Strnad M. 2016. Jasmonate signaling in plant stress responses and development—active and inactive compounds. New Biotechnology **33,** 604–613.

Weidhase R, Kramell H-M, Lehmann J, Liebisch H-W, Lerbs W, Parthier B. 1987. Methyljasmonate-induced changes in the polypeptide pattern of senescing barley leaf segments. Plant Science **51,** 177–186.

Westfall CS, Muehler AM, Jez JM. 2013. Enzyme action in the regulation of plant hormone responses. Journal of Biological Chemistry **288,** 19304–19311.

Westfall CS, Zubieta C, Herrmann J, Kapp U, Nanao MH, Jez JM. 2012. Structural basis for prereceptor modulation of plant hormones by GH3 proteins. Science **336,** 1708–1711.

Widemann E, Smirnova E, Aubert Y, Miesch L, Heitz T. 2016. Dynamics of jasmonate metabolism upon flowering and across leaf stress responses in Arabidopsis thaliana. Plants **5,** 4.

Wiszniewski AA, Bussell JD, Long RL, Smith SM. 2014. Knockout of the two evolutionarily conserved peroxisomal 3-ketoacyl-CoA thiolases in Arabidopsis recapitulates the abnormal inflorescence meristem 1 phenotype. Journal of Experimental Botany **65,** 6723–6733.

Withers J, Yao J, Mecey C, Howe GA, Melotto M, He SY. 2012. Transcription factor-dependent nuclear localization of a transcriptional repressor in jasmonate hormone signaling. Proceedings of the National Academy of Sciences, USA **109,** 20148–20153.

Woldemariam M, Ongokesung N, Baldwin I, Galis I. 2012. Jasmonoyl-L-isoleucine hydrolase 1 (JIH1) regulates jasmonoyl-L-isoleucine levels and attenuates plant defenses against herbivores. The Plant Journal **72,** 758–767.

Xiao Y, Chen Y, Charnikhova T, *et al*. 2014. OsJAR1 is required for JA-regulated floret opening and anther dehiscence in rice. Plant Molecular Biology **86,** 19–33.

Xie D-X, Feys B, James S, Nieto-Rostro M, Turner J. 1998. COI1: an Arabidopsis gene required for jasmonate-regulated defense and fertility. Science **280,** 1091–1094.

Yamamoto Y, Ohshika J, Takahashi T, Ishizaki K, Kohchi T, Matsuura H, Takahashi K. 2015. Functional analysis of allene oxide cyclase, MpAOC, in the liverwort Marchantia polymorpha. Phytochemistry **116,** 48–56.

Yan J, Li S, Gu M, Yao R, Li Y, Chen J, Yang M, Nan F, Xie D. 2016. Endogenous bioactive jasmonate is composed of a set of (+)-*7-iso*-JA-amino acid conjugates. Plant Physiology. pp.00906.2016; doi:10.1104/pp.16.00906.

Yan C, Xie D. 2015. Jasmonate in plant defence: sentinel or double agent? Plant Biotechnology Journal **13,** 1233–1240.

Yan J, Zhang C, Gu M, *et al*. 2009. The *Arabidopsis* CORONATINE INSENSITIVE1 protein is a jasmonate receptor. The Plant Cell **21,** 2220–2236.

Yan L, Zhai Q, Wei J, *et al*. 2013. Role of tomato lipoxygenase D in wound-induced jasmonate biosynthesis and plant immunity to insect herbivores. PLoS Genetics **9,** e1003964.

Yan Y, Stolz S, Chételat A, Reymond P, Pagni M, Dubugnon L, Farmer EE. 2007. A downstream mediator in the growth repression limb of the jasmonate pathway. The Plant Cell **19,** 2470–2483.

Yang DL, Yao J, Mei CS, *et al*. 2012. Plant hormone jasmonate prioritizes defense over growth by interfering with gibberellin signaling cascade. Proceedings of the National Academy of Sciences, USA **109,** E1192–1200.

Yang W, Devaiah SP, Pan X, Isaac G, Welti R, Wang X. 2007. AtPLAI is an acyl hydrolase involved in basal jasmonic acid production and Arabidopsis resistance to Botrytis cinerea. Journal of Bological Chemistry **282,** 18116–18128.

Yoeun S, Rakwal R, Han O. 2013. Dual positional substrate specificity of rice allene oxide synthase-1: insight into mechanism of inhibition by type II ligand imidazole. BMB Reports **46,** 151–156.

Ytterberg AJ, Peltier JB, van Wijk KJ. 2006. Protein profiling of plastoglobules in chloroplasts and chromoplasts. A surprising site for differential accumulation of metabolic enzymes. Plant Physiology **140,** 984–997.

Yu H, Shiva S, Roth M, *et al*. 2014. Lipid changes after leaf wounding in Arabidopsis thaliana: expanded lipidomic data form the basis for lipid co-occurrence analysis. The Plant Journal **80,** 728–743.

Yu J, Zhang Y, Di C, *et al*. 2016. JAZ7 negatively regulates dark-induced leaf senescence in Arabidopsis. Journal of Experimental Botany **67,** 751–762.

Zhai Q, Yan L, Tan D, Chen R, Sun J, Gao L, Dong MQ, Wang Y, Li C. 2013. Phosphorylation-coupled proteolysis of the transcription factor MYC2 is important for jasmonate-signaled plant immunity. PLoS Genetics **9,** e1003422.

Zhai Q, Zhang X, Wu F, Feng H, Deng L, Xu L, Zhang M, Wang Q, Li C. 2015. Transcriptional mechanism of jasmonate receptor COI1-mediated delay of flowering time in Arabidopsis. The Plant Cell **27,** 2814–2828.

Zhang F, Yao J, Ke J, *et al*. 2015. Structural basis of JAZ repression of MYC transcription factors in jasmonate signalling. Nature **525,** 269–273.

Zhang L, Yao J, Withers J, *et al*. 2015. Host target modification as a strategy to counter pathogen hijacking of the jasmonate hormone receptor. Proceedings of the National Academy of Sciences, USA **112,** 14354–14359.

Zhang T, Poudel AN, Jewell JB, Kitaoka N, Staswick P, Matsuura H, Koo AJ. 2016. Hormone crosstalk in wound stress response: wound-inducible amidohydrolases can simultaneously regulate jasmonate and auxin homeostasis in Arabidopsis thaliana. Journal of Experimental Botany **67,** 2107–2120.

Zhang X, Zhu Z, An F, Hao D, Li P, Song J, Yi C, Guo H. 2014. Jasmonate-activated MYC2 represses ETHYLENE INSENSITIVE3 activity to antagonize ethylene-promoted apical hook formation in Arabidopsis. The Plant Cell **26,** 1105–1117.

Zhao N, Lin H, Lan S, Jia Q, Chen X, Guo H, Chen F. 2016. VvMJE1 of the grapevine (Vitis vinifera) VvMES methylesterase family encodes for methyl jasmonate esterase and has a role in stress response. Plant Physiology and Biochemistry: PPB **102,** 125–132.

Zhao Y, Dong W, Zhang N, Ai X, Wang M, Huang Z, Xiao L, Xia G. 2014. A wheat allene oxide cyclase gene enhances salinity tolerance via jasmonate signaling. Plant Physiology **164,** 1068–1076.

Zheng H, Pan X, Deng Y, Wu H, Liu P, Li X. 2016. AtOPR3 specifically inhibits primary root growth in Arabidopsis under phosphate deficiency. Scientific Reports **6,** 24778.

Zheng XY, Spivey NW, Zeng W, Liu PP, Fu ZQ, Klessig DF, He SY, Dong X. 2012. Coronatine promotes Pseudomonas syringae virulence in plants by activating a signaling cascade that inhibits salicylic acid accumulation. Cell Host and Microbe **11,** 587–596.

Zhou G, Ren N, Qi J, Lu J, Xiang C, Ju H, Cheng J, Lou Y. 2014. The 9-lipoxygenase Osr9-LOX1 interacts with the 13-lipoxygenase-mediated pathway to regulate resistance to chewing and piercing–sucking herbivores in rice. Physiologia Plantarum **152,** 59–69.

Zhou M, Memelink J. 2016. Jasmonate-responsive transcription factors regulating plant secondary metabolism. Biotechnology Advances **34,** 441–449.

Zhou W, Wei L, Xu J, *et al*. 2010. Arabidopsis tyrosylprotein sulfotransferase acts in the Auxin/PLETHORA pathway in regulating postembryonic maintenance of the root stem cell niche. The Plant Cell **22,** 3692–3709.

Zhu X, Chen J, Xie Z, Gao J, Ren G, Gao S, Zhou X, Kuai B. 2015. Jasmonic acid promotes degreening via MYC2/3/4- and ANAC019/055/072-mediated regulation of major chlorophyll catabolic genes. The Plant Journal **84,** 597–610.

Zhu Z, An F, Feng Y, *et al*. 2011. Derepression of ethylene-stabilized transcription factors (EIN3/EIL1) mediates jasmonate and ethylene signaling synergy in *Arabidopsis*. Proceedings of the National Academy of Sciences, USA **108,** 12539–12544.