Jasmonates in Plant Growth and Stress Responses

Claus Wasternack

Abstract Jasmonates are lipid-derived compounds which are signals in plant stress responses and development. They are synthesized in chloroplasts and peroxisomes. An endogenous rise occurs upon environmental stimuli or in distinct stages of development such as that of anthers and trichomes or in root growth. Hydroxylation, carboxylation, glucosylation, sulfation, methylation, or conjugation of jasmonic acid (JA) leads to numerous metabolites. Many of them are at least partially biologically inactive. The most bioactive JA is the (+)-*7-iso*-JA–isoleucine conjugate. Its perception takes place by the SCF^{COI1}-JAZ-co-receptor complex. At elevated levels of JAs, negative regulators such as JAZ, or JAV are subjected to proteasomal degradation, thereby allowing positively acting transcription factors of the MYC or MYB family to switch on JA-induced gene expression. In case of JAM negative regulation takes place by anatagonism to MYC2. JA and COI1 are dominant signals in gene expression after wounding or in response to necrotrophic pathogens. Cross-talk to salicylic acid, ethylene, auxin, and other hormones occurs. Growth is inhibited by JA, thereby counteracting the growth stimulation by gibberellic acid. Senescence, trichome formation, arbuscular mycorrhiza, and formation of many secondary metabolites are induced by jasmonates. Effects in cold acclimation; in intercropping; during response to herbivores, nematodes, or necrotrophic pathogens; in pre- and post-harvest; in crop quality control; and in biosynthesis of secondary compounds led to biotechnological and agricultural applications.

Keywords Jasmonates • Oxylipins • Jasmonate biosynthesis • Jasmonate metabolites • Jasmonate perception • Jasmonate signaling • Cross-talk • Biotic stress • Abiotic stress • Root development • Flower development • Applied aspects

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L.-S.P. Tran and S. Pal (eds.), *Phytohormones: A Window to Metabolism, Signaling and Biotechnological Applications*, DOI 10.1007/978-1-4939-0491-4_8,

Abbreviations

ABA	Abscisic acid
AM	Arbuscular mycorrhiza
AOC	Allene oxide cyclase
AOS	Allene oxide synthase
BR	Brassinosteroids
COI1	CORONATINE INSENSITIVE1
ET	Ethylene
GA	Gibberellic acid
DAD1	DEFECTIVE IN ANTHER DEHISCECE1
13-HPOT	13-hydroperoxy octadecatrienoic acid
ISR	Induced systemic resistance
JA	Jasmonic acid
JA–Ile	JA–isoleucine conjugate
JAMe	JA methyl ester
JMT	JA methyltransferase
JAR1	JA resistant1
JAZ	JASMONATE ZIM DOMAIN
α-LeA	α-Linolenic acid (18:3)
LOX	Lipoxygenase
MYC	bHLHzip transcription factor
OPDA	12-Oxophytodienoic acid
OPR	OPDA reductase
PLA1	Phospholipase A1
RNS	Root nodule symbiosis
SA	Salicylic acid
ST	Sulfotransferase
TF	Transcription factor
SCF	Skp1/Cullin/F-box

Introduction

Jasmonic acid (JA) and its derivatives, commonly named jasmonates (JAs), are involved in developmental processes such as growth, lateral and adventitious root formation, seed germination, leaf senescence, glandular trichome formation as well as development of embryos and pollen (Fig. 1). Plants with their sessile lifestyle need constant adaptation to altering environmental cues, such as light, water deficit, salt, cold, and nutrient deficiency, in which JA-mediated responses play a crucial role. Furthermore, JAs are involved in biotic interactions such as responses to herbivores, pathogens, nematodes, or mutualistic symbiotic microorganisms, such as mycorrhizal fungi (Fig. 1). In these numerous interactions during plant stress

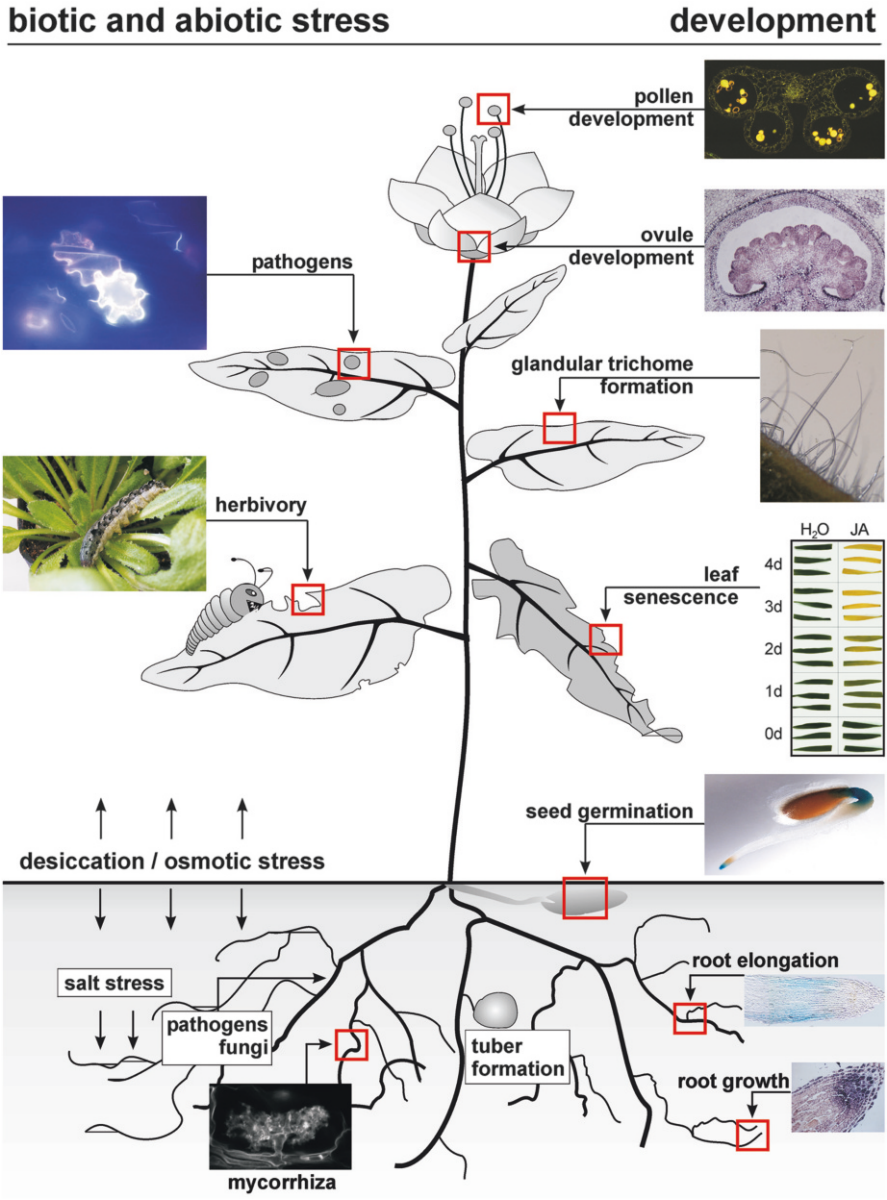

Fig. 1 Jasmonates in plant development (*right*) and plant responses to biotic and abiotic stress (*left*). Pictures for stress responses are given by a hypersensitive response upon pathogen attack, by herbivory on *Arabidopsis*, and by arbuscular mycorrhiza. The role of jasmonates in development is illustrated by a cross section of anthers of *Arabidopsis* showing pollen release, by immunocytochemical detection of allene oxide cyclase in cross section of tomato ovules, by trichomes, by senescing barley leaf segments upon treatment with jasmonate, by seedling growth and root elongation of a tomato seedling showing allene oxide cyclase promoter activity via GUS staining, and by root growth showing immunocytochemical detection of the allene oxide cyclase protein in the root tip. Jasmonates are also involved in growth inhibition, lateral root formation, adventitious root formation, attack by nematodes, light signaling, and freezing tolerance (with permission)

responses and development via JAs, various signal transduction pathways are involved. These pathways exhibit cross-talk to other plant hormones such as ethylene (ET), auxin, gibberellic acid (GA), salicylic acid (SA), brassinosteroids (BR), or abscisic acid (ABA).

The key components of JA biosynthesis, JA perception, and JA signaling have been identified. Several of these proteins were crystallized which allowed first mechanistic explanations. Since JA is perceived as its isoleucine conjugate (JA–Ile, cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones"), I will use here the term JA/JA–Ile. The present chapter will give an overview on JA/JA–Ile biosynthesis, JA/JA–Ile metabolism, JA/JA–Ile perception, JA/JA–Ile signal transduction and cross-talk to other plant hormones, and JA/JA–Ile functions in biotic and abiotic interactions as well as in plant growth and development and will discuss some biotechnological and horticultural applications of JA/JA–Ile. All these aspects have been continuously discussed in excellent reviews (Ballaré 2011; Browse 2009a, b; Kazan and Manners 2008, 2011, 2012; Kombrink 2012; Pauwels and Goossens 2011; Pieterse et al. 2012; Wasternack and Hause 2013; Wasternack and Kombrink 2010). Therefore, emphasis will be given on recently published data. The great amount of published data on JAs can be cited here only partially due to space limitation.

JA Biosynthesis

The JA and its derivatives are members of the class of oxylipins. Whereas JAs are generated by *13-lipoxygenases* (13-LOXs), other oxylipins are products of 9-lipoxygenases (9-LOXs, e.g., LOX1 and LOX5 of *Arabidopsis thaliana*) and α-dioxygenases (α-DOX) which form chemically unstable 2(*R*)-hydroperoxides. α-DOX is involved in defense against aphids (Avila et al. 2013), whereas AtLOX1 together with Atα-DOX1 is involved in the local and systemic response to *Pseudomonas syringae* pv. *tomato* (Vicente et al. 2012). AtLOX1 is also involved in an ABA-independent stomata closure and an immune defense response including SA and the MAP kinases MPK3 and MPK6 (Montillet et al. 2013).

The substrate of JA biosynthesis (Fig. 2) is derived from galactolipids of chloroplast membranes. α-Linolenic acid (18:3) (α-LeA) is released from the *sn-1* position of galactolipids by a phospholipase1 (PLA1). Initially, the PLA1 DEFECTIVE IN ANTHER DEHISCENCE1 (DAD1) was shown to be involved in JA formation (Ishiguro et al. 2001). A DAD1-activating factor (DAF) was identified upstream of DAD1 as putative RING-finger E3 ligase which positively regulates *DAD1* expression (Peng et al. 2013). DAD1 occurs preferentially in flowers and is controlled by the homeobox protein AGAMOUS. Involvement of DAD1 and DONGLE, another PLA1, in JA biosynthesis of leaves was excluded by wild-type-like phenotypes of *DAD1*- and *DONGLE*-RNAi lines in respect to leaf wounding and localization of the DONGLE protein in lipid bodies (Ellinger et al. 2010). Among the 16 lipase mutants of *Arabidopsis*, only that of PLA1γ1 (At1g066800) showed reduced JA

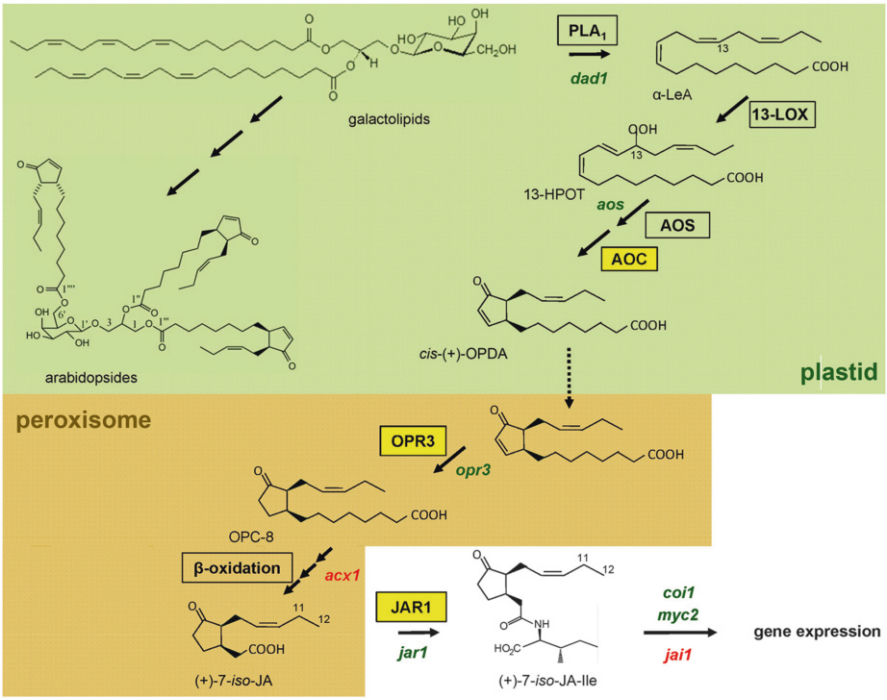

Fig. 2 Biosynthesis of jasmonic acid (JA) and its conjugate JA–isoleucine (JA–Ile) is initiated by the release of α-linolenic acid (α-LeA) from galactolipids of chloroplast membranes. A 13-lipoxygenase (13-LOX), an allene oxide synthase (AOS), and an allene oxide cyclase (AOC) catalyze formation of the cyclopentenone *cis*-(+)-12-oxophytodienoic acid (*cis*-(+)-OPDA). OPDA is released from the chloroplast and transported into peroxisomes, where reduction to the cyclopentanone ring by an OPDA reductase3 (OPR3) and shortening of the carboxylic acid side chain by the fatty acid ß-oxidation machinery take place. (+)-*7-iso*-JA is released into the cytosol, where conversion to JA–Ile and other metabolites takes place. Mutants of *Arabidopsis* are indicated in red, that of tomato in green. *acx1* acyl-CoA oxidase1, *coi1* coronatine insensitive1, *dad1* delayed anther dehiscence1, *13-HPOT* (13*S*)-hydroperoxy octadecatrienoic acid, *jai1* jasmonic acid insensitive1, *JAR1* JA amino acid synthetase1, *myc2* bHLHzip transcription factor MYC2, *OPC-8* 3-oxo-2-(2-pentenyl)-cyclopentane-1-octanoic acid, *PLA$_1$* phospholipase A$_1$ (with permission)

levels upon wounding. The question, however, on activity of other PLA1s in other stress-induced JA formation is still open (Ellinger et al. 2010).

Free α-LeA is oxygenated in the C-13 position by 13-LOXs which occur among the six LOXs of *A. thaliana* as a family with four members (*LOX2, LOX3, LOX4, LOX6*) (Bannenberg et al. 2009). LOX2 is preferentially involved in early wound-induced JA formation (Glauser et al. 2009; Schommer et al. 2008) and JA formation during natural and dark-induced senescence (Seltmann et al. 2010). LOX2 is controlled by Ca^{2+} and a voltage-dependent vacuolar cation channel (Beyhl et al. 2009). This channel is under the control of members of the transcription factor (TF) family TEOSINTE BRANCHED/CYCLOIDEA/PROLIFERATING CELL FACTOR (TCP). Some of them such as TCP4 are targets of miR319 leading to control of JA

biosynthesis via LOX2 (Schommer et al. 2008). This and other examples indicate a developmental control of LOX2 (Danisman et al. 2012). Besides, LOX2 and also LOX3, LOX4, and LOX6 contribute to JA formation (Caldelari et al. 2011; Chauvin et al. 2013). The *LOX6* promoter is preferentially active in developing xylem cells of young tissues, whereas LOX3 and LOX4 are active in mature vascular tissues (Chauvin et al. 2013; Vellosillo et al. 2007), where other genes of JA biosynthesis such as allene oxide synthase (*AOS*) and allene oxide cyclase4 (*AOC4*) are expressed (Kubigsteltig et al. 1999; Stenzel et al. 2012). During fertility and anther development, JA formation including LOX3 and LOX4 activity is required, but LOX2 is not involved (Caldelari et al. 2011). LOX6 location attributes to the rapid increase in JA and JA–Ile after wounding in local and distal leaves (Chauvin et al. 2013). Only LOX6 is required for JA/JA–Ile formation in roots and is involved in responses to abiotic and biotic factors (Grebner et al. 2013). There are increasing examples that distinct isoforms catalyzing identical reactions in JA biosynthesis are involved in different JA/JA–Ile-mediated responses. Examples are the families of LOXs, AOCs, OPDA reductases (OPRs), and acyl-CoA oxidases (ACXs). In contrast to the four 13-LOXs of *A. thaliana*, LOX1 and LOX5 are 9-LOXs and are involved in defense reactions. Interestingly, in *Fusarium oxysporum* known to form many different jasmonates (Miersch et al. 1999), a nonheme iron 13*S*-LOX with multifunctional activity towards dihydroxy, keto, and epoxy alcohol derivatives has been identified (Brodhun et al. 2013). *F. oxysporum* infection activates expression of defense genes such as *THIONINS* (Vignutelli et al. 1998). The 13*S*-LOX detected in *F. oxysporum* suggests that fungal oxylipins including JA might modulate plant defense reactions upon *F. oxysporum* infection.

In JA biosynthesis the 13-LOX product 13-hydroperoxy octadecatrienoic acid (13-HPOT) is converted by the chloroplast-located *AOS,* the first specific step in the JA-specific branch of the LOX pathway. Other branches lead to leaf aldehydes and leaf alcohols as well as divinyl ether-, epoxyhydroxy-, keto-, and hydroxy-polyunsaturated fatty acids (Feussner and Wasternack 2002). AOS is a CYP450 enzyme (CYP74A) which does not require molecular oxygen nor NAD(P)H-dependent cytochrome P450 reductase as cofactor. Gene families of AOS, its substrate specificity and tissue-specific expression as well as the enzyme mechanism have been reviewed (Kombrink 2012; Schaller and Stintzi 2009; Wasternack and Kombrink 2010). Recently, a divinyl ether synthase could be converted into an AOS by a single point mutation indicating the close relationship of CYP74 enzymes (Toporkova et al. 2013). The AOSs of fungi seem to be evolved independently of CYP74, as suggested by the identification of a dioxygenase-cytochrome P450 fusion protein, a novel AOS with catalytic similarities to CYP74 and CYP8A1. This novel AOS has an analogous reaction mechanism to CYP74A enzymes (Hoffmann et al. 2013). A new type of CYP74 enzymes, CYP74C3 could be recently characterized with 9*S*-hydroperoxylinoleic acid as substrate (Brash et al. 2013). This enzyme forms besides the regularly generated *E*-isomer also a *Z*-isomer. Like the LOXs carrying positional specificity for carbon-9 or carbon-13, AOSs show at least preference for C-9 or C-13. An exception is the AOS1 of rice which shows dual specificity (Yoeun et al. 2013). The AOS of *A. thaliana* has been crystallized

(Lee et al. 2008). The highly unstable epoxide formed by AOS is converted by a chloroplast-located *AOC*. In the AOC-catalyzed step, *cis*-(+)-12-oxophytodienoic acid (OPDA) (9*S*,13*S*)-OPDA) is formed which contains the enantiomeric structure of the naturally occurring (+)-*7-iso*-JA. Even not proved experimentally so far, the exclusive occurrence of (9*S*,13*S*)-OPDA suggests that AOS and AOC act in a close vicinity avoiding the formation of a racemic mixture of *cis*-(+)-OPDA and *cis*-(−)-OPDA or spontaneous chemical decomposition leading to α-ketol and γ-ketol. The AOC2 of *A. thaliana* and both AOCs from *Physcomitrella patens* have been crystallized which allowed mechanistic explanation on the binding pocket (Hofmann et al. 2006; Neumann et al. 2012). The AOC of *A. thaliana* is encoded by a family of four members with different but overlapping expression pattern in organs and tissues (Stenzel et al. 2012). As suggested by the redundant expression in leaves and flower organs, interactions of all four AOCs occur by homo- and heteromerization which represents an additional regulatory level (Stenzel et al. 2012). The close association of LOX, AOS, and AOC within chloroplast membranes (Farmaki et al. 2007) may attribute to the formation of OPDA esterified within chloroplast membranes. This diverse group of abundantly accumulating compounds, called arabidopsides due to their exclusive occurrence in *Arabidopsis*, may be a storage form of OPDA (for review cf. Göbel and Feussner 2009; Ibrahim et al. 2011). In rice two photomorphogenic mutants (*hebiba, coleoptile photomorphogenesis 2* (*cpm2*) have been recently found to be defective in AOC genes. These genes encode functional AOCs which are active in defense against *Magnaporthe oryzae* (Riemann et al. 2013).

The second part of JA biosynthesis takes place in peroxisomes. *cis*-(+)-OPDA is assumed to be transported by the peroxisomal ATP-binding cassette (ABC) transporter protein COMATOSE (CTS1) and/or an ion trapping mechanism (cf. reviews of Hu et al. 2012; Wasternack and Kombrink 2010). In peroxisomes OPDA and/or its subsequently generated metabolites are activated by 4CL-like acyl-CoA synthetases (Hu et al. 2012; Kienow et al. 2008; Koo et al. 2006).The cyclopentenone ring of activated OPDA is reduced by an *OPR*. Among the six OPRs of *A. thaliana*, only OPR3 is involved in JA biosynthesis as shown by substrate specificity tests and crystallization of OPR1 and OPR3 (Breithaupt et al. 2001, 2006; Schaller and Stintzi 2009). In contrast, OPR1 seems to be involved in the synthesis of phytoprostanes, a group OPDA-like structures which are preferentially formed by nonenzymatic reactions (Mueller et al. 2008). Moreover, most of the OPRs except OPR3 are involved in detoxification by reduction of α,β-unsaturated aldehydes, ketones, maleimides, or acrolein. The OPRs of *A. thaliana*, rice, maize, and soybean occur in gene families of up to ten members. Their involvement in stress responses and development and even sex determination has been shown (Li et al. 2011).

The following reactions in JA biosynthesis include 4CL-like acyl-CoA synthetases, shortening of the carboxylic acid side chain by the fatty acid ß-oxidation machinery with acyl-CoA oxidase (ACX), the multifunctional protein (MFP), and 3-ketoacyl-CoA thiolase (KAT) (Kombrink 2012; Wasternack and Kombrink 2010). JA generated in peroxisomes is released into the cytosol, where it is metabolized.

The membrane-derived compounds JA and JA–Ile are involved in many responses to biotic and abiotic stress via distinct or overlapping signaling cascades

(cf. sections "Perception of JA-Ile and Cross-Talk to Other Hormones," "JA/JA-Ile in Biotic Interactions of Plants," "JA/JA-Ile in Abiotic Stress Response of Plants," and "JA/JA-Ile in Plant Growth and Development"). Another group of membrane-derived compounds are reactive electrophile species (RES), generated by lipid peroxidation. Whereas JA/JA–Ile- and CORONATINE INSENSITIVE1 (COI1)-mediated processes are involved in wounding, responses to necrotrophic pathogens, and developmentally regulated processes, RES are linked to the SA pathway that involves class II DNA-binding proteins (TGAs) (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones"). There are numerous RES-mediated detoxification processes suggesting a "REScue" by cellular damage including photo-inhibition (reviewed in Farmer and Mueller 2013).

JA Metabolism

The most important reaction in metabolism of JA is its conjugation to amino acids catalyzed by JASMONATE RESISTANT1 (JAR1) (Fig. 3). *JAR1* is member of the *GRETCHEN HAGEN3* (*GH3*) gene family mainly involved in auxin conjugation (Staswick and Tiryaki 2004). The important role of JAR1 became obvious upon identification of (+)-*7-iso*-JA–Ile as the most bioactive compound among more than 40 JA compounds (Fonseca et al. 2009). JAR1 is a jasmonoyl amino acid conjugate synthase forming an acyl-adenylate/thioester intermediate by use of (+)-*7-iso*-JA as the substrate. JAR1/AtGH3.11 has been crystallized (Westfall et al. 2012). Most structure–activity relationships, recorded for numerous JA-dependent responses during the last two decades (for review cf. Wasternack 2007), can be explained now. In many plants JA and JA–Ile accumulate in a ratio of about 10:1. For a long time, the initial product of JA biosynthesis, (+)-*7-iso*-JA, was assumed to epimerize to the more stable (−)-JA. (−)-JA was taken as an indicator of endogenous rise of JAs upon any environmental stimuli. Now, an assay for quantification of (+)-*7-iso*-JA–Ile is available (Suza et al. 2010). Usually, however, levels of JA and JA–Ile are recorded without detection of the individual enantiomers. In *JAR1*-RNAi lines of tomato, up to 25–50 % residual JA–Ile was found upon wounding, suggesting the existence of other JA conjugating enzymes than JAR1 (Suza et al. 2010). Auxin homeostasis is sustained by amido-hydrolases such as IAA-LEUCINE RESISTANT (ILR)-LIKE GENE 6 (ILL6) and IAA-ALANINE RESISTANT 3 (IAR3) which cleave auxin amino acid conjugates. Recently, IAR3 and ILL6 were identified as JA–Ile and 12-OH-JA–Ile amido-hydrolases (Widemann et al. 2013). These enzymes attribute to homeostasis of the active signaling compound JA–Ile as well as formation of 12-OH-JA. Their activities represent a new and unexpected route of 12-OH-JA formation. A similar activity with JA–Ile occurs in *N. attenuata.* Here, a homologue of IAR3 has been cloned and shown to act as a JA–Ile amido-hydrolase (Woldemariam et al. 2012).

Besides amino acid conjugates of JA and their metabolites, twelve other JA derivatives have been identified in plant tissues, preferentially upon wounding

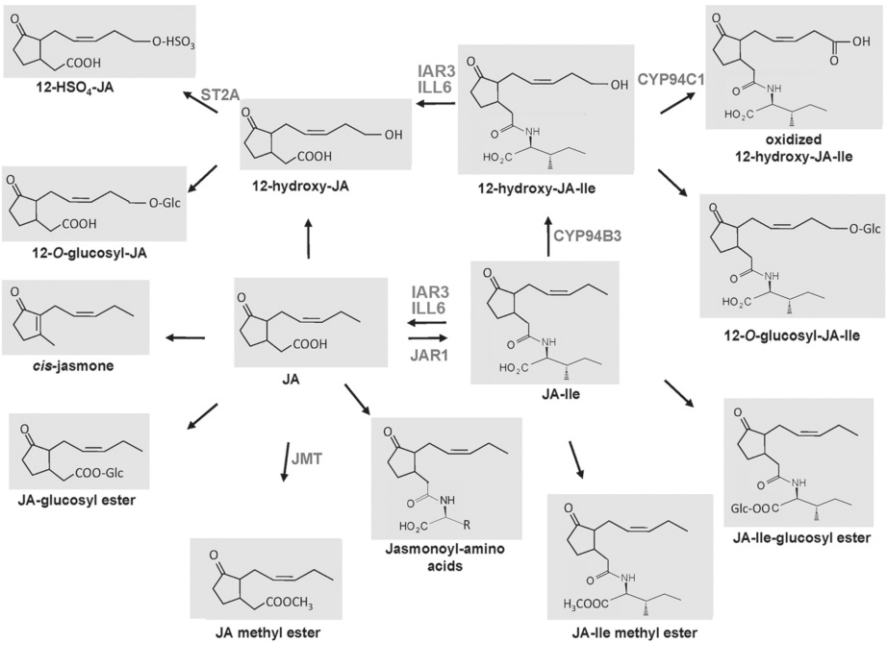

Fig. 3 Metabolism of jasmonic acid (JA) and JA–isoleucine conjugate (JA–Ile). Enzymes which have been cloned are indicated. *JAR1* JA amino acid synthetase, *JMT* JA methyltransferase, *ST2A* 12-OH-JA sulfotransferase 2A, *CYB94B3* JA–Ile hydroxylase, *CYP94C1* 12-OH-JA–Ile oxidase. Degradation of 12-hydroxy-JA–Ile and JA–Ile to 12-hydroxy-JA and JA, respectively, takes place by IAR3 and ILL6, two auxin amido-hydrolases (with permission and modified after Wasternack and Hause 2013)

(Wasternack and Hause 2013). Among them are JA methyl ester (JAMe), JA glucosyl ester, *cis*-jasmone, 12-*O*-glucosyl-JA, 12-HSO_4-JA, 12-hydroxy-JA, 12-hydroxy-JA–Ile, 12-COOH-JA–Ile, 12-*O*-glucosyl-JA–Ile, JA–Ile-glucosyl ester, and JA–Ile methyl ester. Similar derivatives can be assumed for OPDA, but such compounds were not identified so far.

Except JAR1, several enzymes active in JA metabolism have been cloned for *A. thaliana*, tomato, and tobacco. Among them are JA methyltransferases (JMT) (Seo et al. 2001): 12-OH-JA sulfotransferases (AtST2a) (Gidda et al. 2003), a JA–Ile hydroxylase (CYP94B3) (Heitz et al. 2012; Kitaoka et al. 2011; Koo et al. 2011), and a 12-OH-JA–Ile oxidase (CYP94C1) (Heitz et al. 2012). Some JAs accumulate abundantly and constitutively in distinct developmental stages and organs. Among them are 12-OH-JA, 12-HSO_4-JA, and 12-*O*-glucosyl-JA which can reach levels three orders of magnitude higher than that of OPDA, JA, or JA–Ile (Miersch et al. 2008). Many metabolites of JA and JA–Ile such as 12-HSO_4-JA, 12-*O*-glucosyl-JA, 12-hydroxy-JA, 12-hydroxy-JA–Ile, 12COOH-JA–Ile, JAMe, *cis*-jasmone, and 12-*O*-glucosyl-JA–Ile accumulate transiently upon wounding or other environmental stimuli (Glauser et al. 2008, 2009; Heitz et al. 2012; Koo et al. 2011; Miersch et al. 2008). Hydroxylation or other metabolic conversions can be an at least partial

deactivation of bioactivity of JA and JA–Ile (Heitz et al. 2012; Koo et al. 2011; Miersch et al. 2008). In case of the volatile *cis*-jasmone, the decarboxylated JA, bioactivity has been shown by expression data. A subset of genes is expressed by *cis*-jasmone which is different from that induced by JA or JA–Ile (Matthes et al. 2010). Pyrethrins such as cinerolone, jasmonolone, and pyrethrolone are thought to be synthesized from 7-OH-JA (Ramirez et al. 2013). Also 12-*O*-glucosyl-JA has been shown to be active. A distinct enantiomer of the jasmonoyl moiety of this compound was identified as leaf-closing factor of *Albizia* and *Samanea* (Nakamura et al. 2011).

Perception of JA–Ile and Cross-Talk to Other Hormones

One of the most exciting results of the last couple of years in plant biology was the genetic and biochemical proof on hormone perception via the ubiquitin—proteasome system. Similar modules were identified for perception of JA–Ile, auxin, GA, and ET (Chini et al. 2009; Kelley and Estelle 2012). In case of auxin and JA/JA–Ile, similarities are exceptional (Perez and Goossens 2013). A Skp1/Cullin/F-box (SCF) complex functioning as an E3 ubiquitin ligase binds the hormone to the complex. Subsequently, negative regulators of transcription can be recognized by the F-box protein of the complex and are ubiquitinated and thereby subjected to proteasomal degradation (Fig. 4). This allows positively acting TFs to become active. In case of JA–Ile the SCF complex contains the F-box protein *COI1* which was identified via the JA/JA–Ile insensitive mutant of *A. thaliana coi1* (Xie et al. 1998). Coronatine is a bacterial toxin of *Pseudomonas syringae* acting as a molecular mimic of JA–Ile (Zheng et al. 2012), but does not occur in plants. The structural similarity between coronatine and (+)-*7-iso*-JA–Ile led to identification of the latter compound as the most bioactive JA (Fonseca et al. 2009) and finally as the ligand of the JA–Ile receptor (Sheard et al. 2010; Yan et al. 2009). The *SCF^{COI1}-JAZ-co-receptor complex* has been crystallized and mechanism of binding of (+)-*7-iso*-JA–Ile together with inisitol-5-bisphosphate, a co-activator, was shown (Mosblech et al. 2011; Sheard et al. 2010). Targets of the SCF^{COI1} complex are JASMONATE ZIM (ZINC-FINGER PROTEIN EXPRESSED IN INFLORESCENCE MERISTEM) (JAZ) proteins, a new protein family with twelve members in *Arabidopsis* (Chini et al. 2007; Thines et al. 2007; Yan et al. 2007). At low JA–Ile levels, TFs such as MYC2 which binds to the G-box of a promoter of a JA-inducible gene are repressed by JAZ proteins (Fig. 4). At higher JA–Ile levels, however, the SCF^{COI1} complex binds a JAZ protein via JA–Ile binding resulting in ubiquitinylation and degradation of the JAZ protein and derepression of the transcriptional activators. This basic scenario of JA–Ile perception via the SCF^{COI1}-JAZ-co-receptor complex and the subsequent activation of JA/JA–Ile-induced gene expression became more complex upon identification of the corepressor TOPLESS (TPL) and the adaptor protein "Novel Interactor of JAZ" (NINJA) (Pauwels et al. 2010). NINJA interacts with JAZ and TPL. Repression of gene expression takes place by binding of JAZ to TFs such as the *basic*

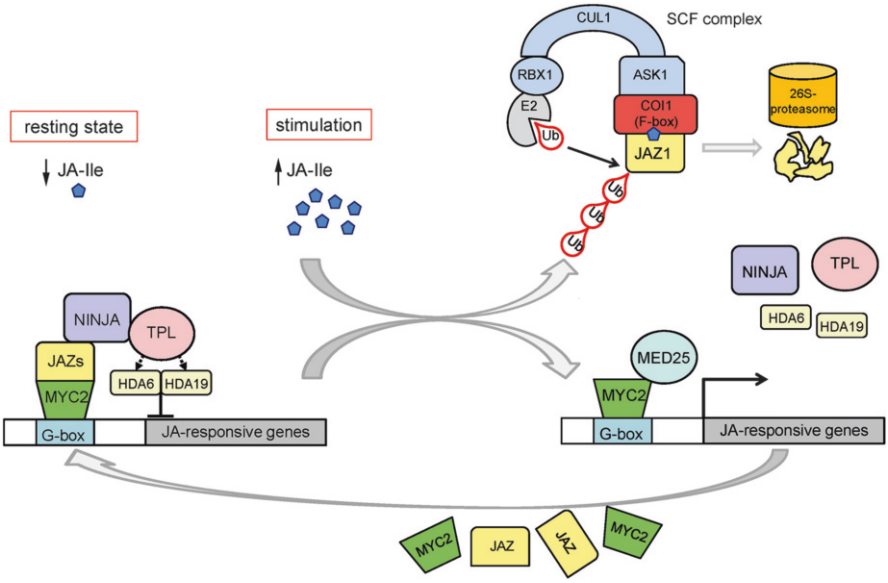

Fig. 4 JA/JA–Ile perception by the SCF^{COI1}-JAZ-co-receptor complex leads to JA/JA–Ile-induced gene expression. There is a low JA/JA–Ile level without environmental stimuli. MYC2 which bounds to a G-box of a JA/JA–Ile-responsive gene is repressed by negative regulators such as JAZs, mediated by corepressors NINJA and TOPLESS (TPL) which act via the HISTONDEACETYLASE6 (HDA6) and HDA19. In addition to JAZ proteins, JAMs (JASMONATE-ASSOCIATED MYC2-LIKE1, JAM2, JAM3) (Nakata et al. 2013) and JAV1 (JASMONATE-ASSOCIATED VQ MOTIF GENE 1) act as repressors. In case JAV1 the interacting ubiquitin E 3 ligase is unknown (Hu et al. 2013a), whereas JAMs compete with MYC2 in binding to the G-box. Dimerization is experimentally shown only for JAZ proteins so far. Upon increase of JA/JA–Ile levels by any stress, JAZs, and JAV1 proteins are subjected to ubiquitinylation and subsequent degradation by the 26S proteasome. Therefore, MYC2 can switch on transcription of JA/JA–Ile-responsive genes including early genes such as *JAZs* and *MYC2*. MED25, the subunit 25 of the Mediator complex, mediates transcription (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones"). Ub, ubiquitin; E2, Rbx, Cullin, ASK1, and the F-box protein COI1 are components of the SCF complex (with permission)

helix-loop-helix (bHLH) TF MYC2 and corepressor activity of TPL mediated by histone deacetylases 6 and 19. In the derepressed state JA/JA–Ile-responsive gene expression is mediated by subunit 25 of the Mediator complex (MED 25) (Çevik et al. 2012; Chen et al. 2012). TFs such as MYC2 and the JAZ proteins are JA/JA–Ile inducible. Therefore, a futile cycle may occur which will attribute to a fine tuning of JA/JA–Ile-induced gene expression at different levels.

The interaction between MYC2 and JAZ takes place via the JAZ INTERACTING DOMAIN (JID) of MYC2 and the Jas domain of JAZ. Jas is absolutely required for repressor function of JAZ (Browse 2009a; Thines et al. 2007). The ZIM domain of JAZ mediates interaction to NINJA but is also responsible via its TIFY domain for homo- and heterodimerization of JAZs (Chung and Howe 2009). The NINJA–TPL interaction takes place via the ET-RESPONSIVE ELEMENT BINDING FACTOR-ASSOCIATED AMPHIPHILIC REPRESSION (EAR) motif of NINJA. Some JAZ

proteins contain such an EAR motif which allows direct binding of TPL without NINJA. These versatile interaction domains occur also in homologous components of ABA and auxin signaling (Pauwels et al. 2010). Consequently, NINJA and TPL are integrators of different signaling pathways. The SCF^{COI1}-JAZ-co-receptor complex and its interactors exhibit several exciting regulatory components:

1. The Jas domain of JAZ interacts with COI1 in the presence of JA–Ile and is strongly increased by IP_5 (Mosblech et al. 2011; Sheard et al. 2010). Stability of COI1 depends on its integration in the SCF complex (Yan et al. 2013).
2. Alternative splice variants of JAZ attribute to multiple JAZ functions and negative feedback control of JA/JA–Ile signaling (Moreno et al. 2013).
3. Enhanced stability of JAZ proteins such as that of JAZ8 being unable to strongly interact with COI1 may attribute to JAZ activity (Shyu et al. 2012).
4. Homo- and heterodimerization of JAZ proteins is another regulatory level (Chung and Howe 2009).
5. *JASMONATE-ASSOCIATED VQ MOTIF GENE 1* (*JAV1*) has been identified recently as another negative regulator of JA/JA–Ile-mediated plant defense with similarities to JAZ (Hu et al. 2013a; Zhu and Zhu 2013). The interacting ubiquitin E 3 ligase, however, is unknown for JAV1. In contrast to JAZ proteins, JAV1 is a repressor against necrotrophic pathogens and herbivorous insects, but not active in plant growth and development.
6. A JASMONATE-ASSOCIATED MYC2-LIKE1 TF, called JAM1, was identified as an ABA-inducible bHLH-type transcriptional repressor of JA responses against herbivores and in JA-dependent growth and development (Nakata et al. 2013). JAM1 competes with MYC2 to target sequences of MYC2 thereby attributing to a fine tuning in JA/JA–Ile-induced gene expression. Together with JAM2 and JAM3, many JA/JA–Ile responses are negatively regulated by JAM1 (Sasaki-Sekimoto et al. 2013). This includes also expression of genes involved in JA biosynthesis and metabolism. The degree of repression by JAZs or/and JAMs is unknown so far.
7. MYC2 activity is sustained by a phosphorylation-coupled proteolysis leading to a distinct amount of "fresh" MYC2 which is able to activate transcription in a positive manner (Zhai et al. 2013). This nuclear located regulatory loop has similarity to SA signaling via the NPR1 protein, the NONEXPRESSOR OF *PR* GENE1 active in SA-induced transcription as co-activator of defense gene expression (cf. Pieterse et al. 2012).
8. Among the bHLH TFs, the subgroup IIId has been identified as novel target of JAZ proteins and as transcriptional repressors in root growth inhibition and anthocyanin formation (Song et al. 2013a). These repressors act redundantly to JAZs indicating a fine tuning in JA/JA–Ile signaling by increased number of signaling components.
9. ILL6, a member of *GH3* gene family coding for amido-hydrolases, has been identified as a new negatively acting regulatory component in JA/JA–Ile responses by comparing expression profiles of individual wild-type plants (Bhosale et al. 2013). ILL6 is involved in cleavage of JA–Ile and 12-OH-JA–Ile,

thereby attributing to JA–Ile homeostasis as well as generation of 12-OH-JA without direct hydroxylation of JA (Widemann et al. 2013).

10. A screen with a JAZ10 reporter system revealed mutants of NINJA which showed constitutive activation of JA responses in roots and hypocotyls indicating organ-specific activation of JA signaling (Acosta et al. 2013).

This plethora of components and regulatory principles in JA signaling is used by downstream components as well as in the cross-talk to other hormones. Targets of JAZs in JA signaling are TFs of the bHLH-type MYC and the *R2R3-type MYB* family. MYC2 was the first TF for which an interaction with a JAZ protein was shown (Chini et al. 2007). MYC2 is a key player in JA/JA–Ile-induced gene expression and is involved in synthesis of auxin, tryptophan, glucosinolates (GS), ET, and JA as well as in responses to herbivores, oxidative stress, pathogens, and ABA-dependent drought stress (Dombrecht et al. 2007; Kazan and Manners 2008). The central role of MYC2 is documented by (1) the regulation of its cross-talk with SA, ABA, GA, and auxin signaling pathways; (2) the link between JA/JA–Ile and other signaling pathways such as light, phytochrome and circadian clock; (3) the regulation of lateral and adventitious root formation, flowering, and shade avoidance syndrome; (iv) the innate immunity in roots; (5) induced systemic resistance (ISR) by beneficial soil microbes; as well as (6) the antagonistic coordination of responses to herbivores and pathogens. Some of the MYC2-dependent JA-regulated processes have been verified by proteome analysis of wild-type and *myc2* mutant plants (Guo et al. 2012). All these aspects reflect the central role of MYC2 and have been reviewed recently (Kazan and Manners 2013). Besides the master regulator MYC2, other targets of JAZs are MYC3, MYC4, MYB21, and MYB24. All MYC TFs have a JID domain and a conserved ACT-like domain at the C-terminus being involved in homo- and heterodimerization of MYCs (Cheng et al. 2011; Fernández-Calvo et al. 2011, Pauwels and Goossens 2011). MYC2, MYC3, and MYC4 are partially redundant (Fernández-Calvo et al. 2011). The *myc2,3,4* triple mutant plants are free of GS and show altered insect performance and feeding behavior (Schweizer et al. 2013). MYC2 binds directly to promoters of GS biosynthesis genes. All three MYCs interact with GS-related MYB TFs indicating the complex scenario in JA/JA–Ile-induced gene expression (Schweizer et al. 2013). The bHLH TFs involved in anthocyanin formation and trichome initiation contain also a JID domain and are targets of JAZ1 and JAZ8 (Qi et al. 2011). JAZ targets active in development were identified in a transcriptome analysis of developing stamen of JA-treated *opr3* plants (Mandaokar et al. 2006). Among them are *MYB21* and *MYB24* which interact with JAZ1 and JAZ8 via the N-terminal R2R3 domain (Song et al. 2011). Both TFs are specifically involved in fertility but less in other JA/JA–Ile-dependent processes such as root growth or anthocyanin formation.

The *cross-talk* between *JA/JA–Ile and auxin* was shown in several processes. Prominent examples are (1) the MYC2-mediated suppression of PLETHORA, a central regulator in auxin-mediated root meristem and root stem cell niche development (Chen et al. 2011); (2) the regulatory activity of JA/JA–Ile in expression of *ANTHRANILATE SYNTHASE1* (*ASA1*), which encodes the initial enzyme in auxin

biosynthesis (Sun et al. 2009); and (3) COI1- and JA/JA–Ile-dependent regulation of *YUCCA8* and *YUCCA9*, two important genes in auxin biosynthesis (Hentrich et al. 2013).

The *cross-talk* between *JA/JA–Ile and ET* is synergistic and takes place by MYC2 activated upon herbivore attack and by ETHYLENE RESPONSE FACTOR1 (ERF1). ERF1 is activated upon infection by necrotrophic pathogens and JA/JA–Ile-dependent degradation of JAZs, the repressors of MYC2 and TFs in ET signaling such as ETHYLENE INSENSITIVE3/EIN-LIKE1 (EIN3/EIL1) and OCTADECANOID-RESPONSIVE *ARABIDOPSIS* AP2/ERF domain protein (ORA59) (Pieterse et al. 2012). The final output of JA/JA–Ile-ET cross-talk is an antagonistic activity between the MYC2 branch and the ERF1 branch and is of benefit for plants due to the naturally occurring simultaneous attack by herbivores and necrotrophic pathogens (Pieterse et al. 2012; Verhage et al. 2011).

Cross-talk between *JA/JA–Ile and GA* signaling takes place synergistically during stamen development and antagonistically in the balance between growth and defense (Kazan and Manners 2012; Wasternack and Hause 2013). During stamen development, the repressors in GA signaling, the DELLA proteins, repress *DAD1* and *LOX* expression in the absence of GA leading to JA/JA–Ile deficiency, to downregulation of *MYB21* and *MYB24* by JAZ, and finally to male sterility (Cheng et al. 2009; Song et al. 2011). The opposite scenario takes place by GA-induced SCF^{GID}-mediated DELLA degradation. JA/JA–Ile and GA act antagonistic in growth and defense which is of benefit for the plant, since plant defense is costly and occurs at the expense of plant growth (Hou et al. 2013; Kazan and Manners 2012). Plant growth can occur at sufficient GA level which represses DELLAs and attenuates DELLA binding to JAZ followed by JAZ binding to MYC2. Consequently, JA-dependent defense response is suppressed during growth (Kazan and Manners 2012; Wager and Browse 2012; Wasternack and Hause 2013). There is a balance of the modules of the SCF complexes for JA and GA. It has to be kept in mind, however, that these complexes are part of the COP9 signalosome (CSN) multiprotein complex which regulates both SCF activities (Stratmann and Gusmaroli 2012). In addition to the GA—JA/JA–Ile cross-talk, the balance between disease resistance and growth is regulated by ABA, SA, and auxin (Denancé et al. 2013). Here, pathogens evade hormone-mediated defense responses with a negative effect on fitness leading to less growth and development.

Cross-talk between *BR and JA/-JA–Ile* is antagonistic in respect to growth as shown by mutants (Huang et al. 2010) and is synergistic in case of anthocyanin biosynthesis, where BR acts upstream of JA/JA–Ile (Peng et al. 2011; Song et al. 2011). Another cross-talk of BR and JA/JA–Ile occurs in defense to herbivores (Yang et al. 2013). Surprisingly, BR receptor impairment downregulates herbivore-induced accumulation of JA–Ile and diterpene glycosides without effects on JA levels and trypsin proteinase inhibitor levels (Yang et al. 2013). An important gene in BR biosynthesis is *DWF4* (*DWARF4)* which encodes a steroid C22 α-hydroxylase (CYP90B1). Its expression is auxin inducible and is repressed by JA/JA–Ile. Consequently, the balance between growth and defense is sustained by JA/JA–Ile via BR (Kim et al. 2013).

The *cross-talk* between *ABA and JA/JA–Ile* was clearly detected for the wound response. Here, the rise of ABA and JA/JA–Ile and JA/JA–Ile-induced formation of PYL4 and PYL5, which are ABA receptors, have been shown (Kazan and Manners 2008; Lackman et al. 2011). Many components of the *cross-talk* between *JA/JA–Ile and SA* have been identified, and synergistic and antagonistic interactions were shown (Boatwright and Pajerowska-Mukhtar 2013; Gimenez-Ibanez and Solano 2013; Pieterse et al. 2012). JA/JA–Ile is the key player in responses to necrotrophic pathogens and herbivores, whereas SA is the central signaling compound in responses to biotrophic pathogens (Pieterse et al. 2012). Key components of both pathways such as glutaredoxins, thioredoxins, TFs such as WRKY70 for the SA pathway, and MYC2 as well as COI1 for the JA pathway are involved in the cross-talk. Final steps in this cross-talk are nuclear modulation of both signaling pathways (Gimenez-Ibanez and Solano 2013; Pieterse et al. 2012). The well-known suppression of JA-responsive gene expression takes place downstream of JA formation (Leon-Reyes et al. 2010) and of the SCF^{COI1}-JAZ-co-receptor function. The suppression includes the TF ORA59 (Van der Does et al. 2013). Another interesting cross-talk was shown by coronatine-mediated increase in *P. syringae* virulence (Zheng et al. 2012). Here, ARABIDOPSIS NAM, ATAF1,2, CUC2 (NAC) TFs (ANACs) are involved. Coronatine activates the three homologous TFs, ANAC019, ANAC055, and ANAC072, in an MYC2-dependent manner, leading to inhibition of initial steps in SA synthesis. A similar scenario for these ANAC TFs was found during senescence (cf. section "JA/JA-Ile in Plant Growth and Development"). In parallel, coronatine allowed bacterial propagation locally and systemically upon induction of stomata reopening (Xin and He 2013) or inhibition of stomatal closure (Lee et al. 2013). These data reflect the multiple virulence activities of coronatine (Zheng et al. 2012). The properties of coronatine as a multifunctional suppressor of defense include also COI1- and SA-independent signaling (Geng et al. 2012). The JA/JA–Ile - SA cross-talk is a conserved mechanism and is transmitted to the next generation (Luna et al. 2012). Obviously, these pathways allow in nature the flexibility of plants to adapt to simultaneously and/or subsequently occurring changes in the environment (Thaler et al. 2012). It is interesting to note that nuclear targeted effectors of pathogenic fungi, nematodes, and beneficial microbes are similar in their action and reprogramming of hormonal pathways such that of SA and JA/JA–Ile (Gimenez-Ibanez and Solano 2013).

JA/JA–Ile signaling versus OPDA signaling is an intriguing question rose by the fact that the SCF^{COI1}-JAZ-co-receptor complex accept exclusively (+)-*7-iso*-JA–Ile (Fonseca et al. 2009) but not OPDA (Thines et al. 2007). The mechanistic proof was given upon crystallization of the complex (Sheard et al. 2010). There are, however, OPDA-specific reactions such as tendril coiling (Blechert et al. 1999), gene expression (Mueller et al. 2008; Taki et al. 2005), embryo development in tomato (Goetz et al. 2012), inhibition of seed germination (Dave et al. 2011), activation of *PHO1* genes which are involved in phosphate accumulation (Ribot et al. 2008), PHYTOCHROME A signaling (Robson et al. 2010), hypocotyl growth inhibition (Brüx et al. 2008), or insect-induced closure of the Venus flytrap (Escalante-Pérez et al. 2011) (reviewed in Wasternack and Hause 2013, Wasternack et al. 2012).

In *P. patens* which does not contain JA (Stumpe et al. 2010), OPDA is involved in responses to *B. cinerea* infection by reinforcement of the cell wall and programmed cell death (Ponce de Leon et al. 2012). Even JA is absent in *P. patens*, the moss can respond to applied JA suggesting perception via the SCF^{COI1}-JAZ-co-receptor complex or a perception mechanism not yet identified.

Some of the OPDA-specific effects might be mediated by RES since OPDA contains an α,β-unsaturated carbonyl group (Farmer and Mueller 2013). An interesting new example of OPDA-specific signaling was given recently by data on OPDA-binding to cyclophilin 20-3 which is involved in stress responses (Park et al. 2013). As a consequence of OPDA-binding to this cyclophilin, a hetero-oligomeric cysteine synthase complex is formed in the chloroplast leading to activation of sulfur assimilation and cellular redox homeostasis (Park et al. 2013).

JA/JA–Ile-Regulated Metabolism of Secondary Compounds

Besides JA-induced proteins of barley (Weidhase et al. 1987) and wound-induced PROTEINASE INHIBITOR (PIN) formation in tomato (Farmer and Ryan 1990), the elicitor-induced alkaloid synthesis of plant cell cultures was among the first JA-induced gene expression programs which were analyzed (Gundlach et al. 1992). Meanwhile, JA/JA–Ile-induced synthesis of secondary compounds has been shown for many plant species and diverse secondary compounds. This led to biotechnological and agricultural applications (reviewed in Wasternack 2013). OCTADECANOID DERIVATIVE RESPONSIVE CATHARANTHUS AP2 DOMAIN2 and 3 (ORCA2 and ORCA3) were the first TFs involved in synthesis of secondary metabolites, here terpenoid indole alkaloids (TIA) in *Catharanthus roseus* (van der Fits and Memelink 2000). Transcriptional control of secondary metabolite biosynthesis has been shown in detail and includes the SCF^{COI1}-complex, JAZ proteins, MYC2, ORCAs and/or ERFs, MYBs, and WRKYs which are active in distinct pathways. For *nicotine* biosynthesis requirement of functional SCF^{COI1}-JAZ-co-receptor complex, MYC2, and AP2/ERFs has been shown (De Boer et al. 2011; Shoji and Hashimoto 2011). AP2/ERFs are encoded by the *NIC* locus in tobacco, comprise 239 members (Rushton et al. 2008), and are close homologues of ORCA3 of *C. roseus*. Obviously, these TFs evolved as a regulatory module in two species and two pathways in parallel due to evolutionary advantage.

The abovementioned "machinery" of SCF^{COI1}, JAZ, MYC2, ORCA2, and ORCA3 is also active in *vinblastine* biosynthesis of *C. roseus* (Zhang et al. 2011), whereas *artemisinin* biosynthesis is controlled by ERF1, ERF2, MYC2, and WRKY1 (Ma et al. 2009). The trichome-specific TF of *Artemisia annua* ORA, a member of the AP2/ERF TF family, is a key player in artemisinin biosynthesis (Lu et al. 2013). Interestingly, artemisinin biosynthesis genes are coordinately activated with genes involved in the formation of trichomes, the storage organ of artemisinin (Maes et al. 2011).

Many genes encoding enzymes of *glucosinolate/camalexin* biosynthesis are JA/JA–Ile regulated via SCF^{COI1}, JAZ, MYC2, MYC3, MYC4, and an MAP

kinase—WRKY cascade (De Geyter et al. 2012; Schweizer et al. 2013). Members of the NAC TF family such as ANAC42 are also involved. In summary, the TFs active in alkaloid biosynthesis belong to the families of bHLH, MYC, ERF, and WRKY TFs, and most of them are JA/JA–Ile inducible. These aspects have been reviewed recently (Yamada and Sato 2013).

Anthocyanin is the most prominent secondary compound formed upon JA/JA–Ile treatment or any environmental stimuli leading to endogenous rise of JA/JA–Ile. Any stress of plant tissues is frequently visible by red cell layers indicating anthocyanin formation. Involvement of JA/JA–Ile biosynthesis and signaling has been repeatedly shown by lack of anthocyanin formation in mutants of *A. thaliana* or tomato affected in JA biosynthesis or signaling. Prominent examples are *coi1* and *opr3* for *A. thaliana* and *jai1*, *spr2,* and *acx1* for tomato (Browse 2009b) (Table 1). Important TFs active in anthocyanin synthesis are PRODUCTION OF ANTHOCYANIN PIGMENT1 (PAP1), ENHANCER OF GLABRA3 (EGL3), GLABRA3 (GL3), MYB75, and TRANSPARENT TESTA8 (TT8). All of them are targets of JAZ proteins (Qi et al. 2011). Like artemisinin, anthocyanin formation and trichome formation are coordinately regulated as shown by identification of the tomato homologue of COI1, JAI1 (Li et al. 2004). In *jai1* mutant plants no anthocyanin formation and trichome formation takes place.

JA/JA–Ile in Biotic Interactions of Plants

Due to their sessile lifestyle, plants have to respond to any attack by herbivores, leaf or root pathogens, nematodes, and sucking insects. Biotic interactions can be, however, also beneficial for plants as in case of mutualistic interactions, such as arbuscular mycorrhiza (AM), growth-promoting rhizobacteria leading to ISR, or root nodule symbiosis (RNS). Even plant–plant interactions occurring by near growth of different plant species can be beneficial for both partners. Leaf volatiles or root exudates can attribute to such interaction. The benefit for the plants is obvious by the so-called intercropping, the mixed growth of two or more plant species (cf. section "Applied Aspects on Jasmonates"). In all these interactions JA is a signal.

Response to *herbivory* and *mechanical wounding* is one of the most prominent and early observed JA responses. There was the observation by C. A. Ryan (Pullman, USA) that a sagebrush plant led to less attack by herbivores of a neighboring tomato plant (Farmer and Ryan 1990). Volatile JAMe was identified as the compound emitted by sagebrush leaves which induced in the neighboring tomato leaves formation of PIN2, a deterrent protein for the gut of herbivores. Worldwide is a dramatic loss in agriculture by herbivores, mechanical wounding, or sucking/piercing insects. This led to intensive research. Plant responses to herbivores are induced by oral secretion of the herbivore which contain inducers of wound-induced gene expression such as volicitin (cf. rev. of Wasternack and Hause 2002). There are two defense mechanisms: (1) *direct defense* by formation of toxic compounds such as nicotine in tobacco or other deterrent secondary metabolites, by synthesis of many defense proteins such as PINs or polyphenol oxidase (PPO) which have deterrent role in the

Table 1 Mutants of JA biosynthesis and JA signaling in *Arabidopsis* and tomato (modified after Wasternack 2006)

Mutants	Phenotype	Gene product	References
Deficient JA biosynthesis			
dad1	Reduced filament elongation, *d*elayed *a*nther *d*ehiscence, JA deficient in flowers	Phospholipase A1	Ishiguro et al. (2001)
fad3-2fad7-2fad8[a]	Male sterile, delayed anther development, altered α-LeA level	ω-7-*f*atty *a*cid *d*esaturase	McConn and Browse (1996)
spr2[a,b]	Deficient in α-LeA and JA levels, no wound response, *s*uppressed *pr*osystemin expression	ω-3-fatty acid desaturase	Li et al. (2003)
aos	JA deficient, decreased resistance to pathogens	*AOS*	Park et al. (2002)
dde2-2	Male sterile, delayed anther development	*AOS*	von Malek et al. (2002)
opr3	JA deficient, decreased resistance to pathogens, reduced filament elongation	*OPR3*	Stintzi and Browse (2000)
acx1[a]	JA-deficient, reduced wound response	Acyl-CoA oxidase1	Li et al. (2005)
aim1[c]	Abnormal inflorescence meristem	Multifunctional protein1	Richmond and Bleecker (1999)
comatose	Reduced JA content	*COMATOSE/PXA1*	Theodoulou et al. (2005)
Constitutive JA response			
cev1	*C*onstitutive *e*xpression of *v*egetative storage proteins	Cellulose synthase CeS3A	Ellis et al. (2002)
cet1-9	*C*onstitutive *e*xpression of *t*hionins, increased JA levels	?	Hilpert et al. (2001)
cas1	Constitutive expression of *AOS*	?	Kubigsteltig and Weiler (2003)
ore9/max2[d]	Delayed leaf senescence, strigolactone-dependent shoot branching	F-box protein	Woo et al. (2001) Domagalska and Leyser (2011)
Reduced sensitivity to JA			
coi1	Reduced root growth inhibition, male sterile, reduced filament elongation, enhanced sensitivity to necrotrophic pathogens	F-box leucine reach repeat (LRR) COI1	Xie et al. (1998)
jai1[a]	Female sterile, altered trichome development, increased sensitivity to pathogens, decreased wound response	Tomato homologue of COI1	Li et al. (2004)
jar1/jin4/jai2	Reduced root growth inhibition by JA, increased sensitivity to necrotrophic pathogens	JA amino acid conjugate synthase	Lorenzo et al. (2004), Staswick et al. (1992), Staswick and Tiryaki (2004)
cyp94b3	Reduced JA responses	JA–Ile-hydroxylase	Koo et al. (2011), Heitz et al. (2012)

cyp94c1	Reduced JA responses	JA–Ile-carboxylase	Heitz et al. (2012)
jin1/jai1/myc2	Reduced root growth inhibition	AtMYC2 (bHLHzip TF)	Lorenzo et al. (2004)
mpk4	Dwarf phenotype, altered expression of JA- and SA-response genes	*AtMPK4*	Petersen et al. (2000)
axr1	Reduced root growth inhibition by JA	RUB-activating enzyme	Xu et al. (2002)
jai4/sgt1b[e]	Reduced root growth inhibition in the *ein3* background	*AtSGT1b*	Lorenzo et al. (2004)
Increased JA response			
jam1[f]	Increased anthocyanin formation and resistance to herbivores	JAM1 TF	Nakata et al. (2013)
jam2[f], *jam3*[f]	Increased defense responses, anthocyanin formation, and root growth inhibition	JAM2 TF JAM3 TF	Sasaki-Sekimoto et al. (2013)
jav1[g]	Increased resistance to pathogens and herbivores, but unaltered development	JAV1 TF	Hu et al. (2013a)

[a]Tomato mutants
[b]*SUPPRESSOR OF PROSYSTEMIN EXPRESSION*
[c]*ABNORMAL INFLORESCENCE MERISTEM1*
[d]*ORESA9/MORE AXILLARY GROWTH2*
[e]*JASMONATE INSENSITIVE4/SUPPRESSOR OF THE G2 ALLELE OF SKP 1*
[f]*JASMONATE-ASSOCIATED MYC2-LIKE1,2,3*
[g]*JASMONATE-ASSOCIATED VQ MOTIF GENE 1*

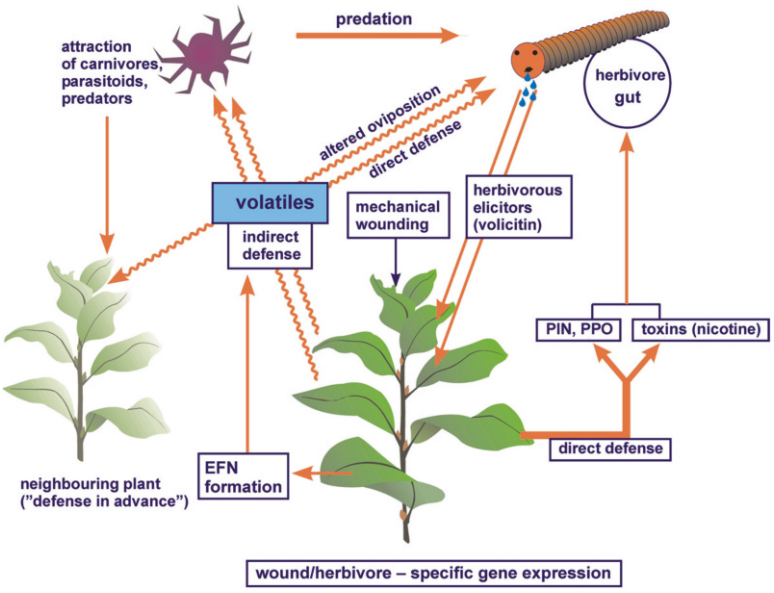

Fig. 5 Mechanical wounding and herbivory leads to direct and indirect defense. Upon elicitation by oral secretions of herbivores or mechanical damage of leaves, defense proteins such as proteinase inhibitors (PINs) or polyphenol oxidase (PPO) as well as toxic compounds such as nicotine in case of tobacco are formed. All of them affect digestion of the leaf tissues in the herbivorous gut due to deterrent properties of these proteins or compounds. Indirect defense upon herbivory is initiated by emission of leaf volatiles which attract parasitoids and carnivores or alter oviposition of herbivores. Additionally, volatiles can induce defense reactions in neighboring plants. Extra floral nectar (EFN) formation can also attribute to defense (with permission)

digestion of the herbivorous gut, and (2) *indirect defense* by emission of volatiles such as leaf alcohols or aldehydes or terpenoids (Fig. 5). These volatiles attract carnivores, parasitoids, or predators and alter the oviposition of herbivores. There is a specific volatile blend which differs among various insect communities. Under field conditions, the volatile emission can reduces the number of herbivores up to 90 % (Kessler et al. 2004). The scenario, however, is more complex than previously recognized, e.g., oral secretions of herbivores contain bacteria which downregulate plant defense reactions (Chung et al. 2013). Another issue is the reallocation of resources within a plant by herbivore attack. JA/JA–Ile-mediated defense is costly, e.g., herbivore attack on leaves reduces sugar and starch levels in roots and reduces regrowth from the rootstock (Machado et al. 2013). Besides wounding by mechanical damage or herbivores, touch of aboveground plant parts increases endogenous JA/JA–Ile levels and leads to growth inhibition (Tretner et al. 2008). This is even different to soft mechanical stress which generates ROS (*reactive oxygen species*) in a JA-independent manner leading to resistance to *B. cinerea* (Benikhlef et al. 2013).

Due to the overwhelming literature on wound responses and herbivory available already in reviews, we refer here to some of them to avoid overlap (Ballaré 2011; Bonaventure et al. 2011; Dicke and Baldwin 2010; Erb et al. 2012; Fürstenberg-Hägg et al. 2013; Meldau et al. 2012; Reymond 2013; Santino et al. 2013).

Arbuscular mycorrhiza (*AM*) is a mutualistic interaction of about 80 % of land plants with fungi of the phylum *Glomeromycota* (Schüssler et al. 2001). AM leads to supply of mineral nutrients and water as well as improved tolerance to some abiotic and biotic stressors (Cameron et al. 2013; Hause and Schaarschmidt 2009). Some of participating proteins have been identified mainly by RNAi approaches. Among them are components of membrane biosynthesis, transport, sucrose cleavage, and carotenoid biosynthesis (Recorbet et al. 2013). Several data accord with a role of JA/JA–Ile in the establishment and maintenance of AM: (1) AM roots of *M. truncatula* have increased JA levels and increased expression of JA biosynthesis genes (Hause et al. 2002; Isayenkov et al. 2005), (2) transgenic tomato lines with enhanced JA levels exhibit increased mycorrhization (Tejeda-Sartorius et al. 2008), (3) AOC-RNAi lines of *M. truncatula* carrying reduced JA biosynthesis have significantly less mycorrhization (Isayenkov et al. 2005), and (4) repeated wounding of *M. truncatula* leaves elevates JA levels and increases AM (Landgraf et al. 2012) (cf. also review of Wasternack and Hause 2013). The establishment of AM leads to systemic protection against many attackers similar to systemic acquired resistance (SAR) following pathogen attack and ISR after colonization by nonpathogenic rhizobacteria (Cameron et al. 2013). Therefore, the term "mycorrhiza-induced resistance" (MIR) was proposed. Four phases have been proposed, where in the last phase a systemic priming of JA- and ET-dependent defense reactions occur (Cameron et al. 2013).

ISR is induced by nonpathogenic microbes and, as mentioned above, by mycorrhizal fungi. JA/JA–Ile is the central regulator in generation of ISR (Van der Ent et al. 2009). There is a close interconnection between ISR and MIR due to putative priming of JA-dependent defenses caused by ISR-related rhizobacteria in the mycorrhizosphere (Cameron et al. 2013).

RNS has been controversially discussed in respect to putative role of JA/JA–Ile (cf. rev. of Wasternack and Hause 2013). Whereas in limited light supply JA/JA–Ile seems to be a positive regulator (Shigeyama et al. 2012; Suzuki et al. 2011), no increased JA level during nodulation under normal growth conditions was found (Landgraf et al. 2012). Autoregulation, a systemic effect in RNS, is a complex scenario, for which involvement of shoot-derived JA/JA–Ile has been proposed (Hause and Schaarschmidt 2009; Kinkema and Gresshoff 2008). RNS and AM have some common signaling components. Ca^{2+} and calmodulin-dependent protein kinases are the central signaling hubs, whereas specificity for AM and RNS is given by transcriptional regulators (Singh and Parniske 2012). These common sequences in AM and RNS seems to be inhibited by shoot-derived JA/JA–Ile during autoregulation (Hause and Schaarschmidt 2009).

JA/JA–Ile in Abiotic Stress Response of Plants

Involvement of JA/JA–Ile has been shown for plant responses to salt, drought, and osmotic and chilling stresses and has been reviewed recently (Santino et al. 2013). For several of these signaling pathways, JA/JA–Ile-specific signaling modules such as

SCF^{COI1}, JAZ, and MYC2 or expression of JA/JA–Ile biosynthesis genes has been identified. An example is the response to cold stress being positively regulated by JA/JA–Ile (Hu et al. 2013b). Key players in cold stress response are JA/JA–Ile inducible, and the INDUCER OF CBF EXPRESSION1 (ICE) is a target of JAZ1 and JAZ4.

JA/JA–Ile in Plant Growth and Development

The involvement of jasmonates in plant growth and development has been unequivocally shown by *mutants* affected in JA/JA–Ile biosynthesis and JA/JA–Ile signaling. These mutants preferentially identified for *A. thaliana* and tomato showed an altered phenotype in root growth inhibition and flower development. These aspects have been reviewed (Browse 2009a, b). For comparison, a brief summary of several mutants is shown in Table 1. These mutants can be subdivided into mutants of JA biosynthesis, mutants with reduced sensitivity to JA/JA–Ile, mutants with constitutive JA response, and mutants with increased JA response. Among JA biosynthesis mutants, *fad3-2fad7-2fad8*, *spr2*, *aos*, and *dde2-2* are prominent examples for JA/JA–Ile and OPDA deficiency. In contrast, *opr3* and *acx1* plants are JA deficient but still able to accumulate OPDA upon wounding. Constitutive JA/JA–Ile responses occur in *cev1* plants, where the subunit 3 of the cellulose synthase complex of *A. thaliana* is altered (Ellis et al. 2002). Recently, a set of mutants with increased JA responses was identified. Here, *JAM1, JAM2, and JAM3* were identified as bHLH TF/JA-associated MYC2-like negative regulators of MYC2 signaling (Nakata et al. 2013) (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones") . Another negative regulator is encoded by the *JAV1* gene. In *jav1* mutant plants defense responses to necrotrophic pathogens and herbivores are increased without influencing growth and development (Hu et al. 2013a). This indicates repressor function of JAV1 at least partially like the JAZ proteins (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones"). Male sterility is among the most prominent phenotypes described for JA-insensitive (*coi1, jai1*) or JA-deficient plants (*opr3, dde2-2, fad3-2 fad7-2 fad8*).

Flower Development: The altered phenotype of mutants affected in JA/JA–Ile biosynthesis and signaling led to detailed analyses of flower development (Browse 2009a; Song et al. 2013b; Wilson et al. 2011). Among the male sterile *A. thaliana* plants, insufficient filament elongation (*opr3*), nonviable pollen, and delayed anther dehiscence (*dad1*) have been described. Stamen transcriptome analysis in JA-treated *opr3* plants led to the identification of several MYB-type TFs (Mandaokar et al. 2006) (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones"). Among them, MYB21, MYB24, and MYB57 were identified as JAZ targets being essential for stamen development (Song et al. 2011). Cross-talk to auxin in anther development was clearly shown by control of JA biosynthesis genes such as *DAD1*, *LOX2, AOS,* or *OPR3* by AUXIN RESPONSE FACTOR6 (ARF6) and ARF8 (Nagpal et al. 2005; Reeves et al. 2012) and accumulation of JA in auxin receptor quadruple mutant (*tir1, afb1-3*) (Cecchetti et al. 2013) (cf. review of Song et al. 2013b). There is also a cross-talk between JA/JA–Ile and GA as briefly described in section "Perception of JA-Ile

and Cross-Talk to Other Hormones". Here, DELLAs suppress expression of JA biosynthesis genes, thereby reducing JA/JA–Ile levels which are required for *MYB21/MYB24/MYB57* expression, the essential TFs in stamen development (Song et al. 2011, 2013b). Another indication for the role of JA/JA–Ile in flower development is given by binding of the TF AGAMOUS to the promoter of *DAD1*, encoding the PLA1 involved in JA formation in flowers (Ishiguro et al. 2001) (cf. section "JA Biosynthesis"), and by controlling of the bHLH TF BIGPETALp by JA/JA–Ile. This TF is involved in petal growth (Brioudes et al. 2009).

Seed Germination: Although GA, ABA, and ET are key players in seed germination, also JA/JA–Ile is active in an inhibitory manner (cf. review of Linkies and Leubner-Metzger 2012). Seed germination data for many mutants affected in JA biosynthesis and JA signaling revealed involvement of COI1. The mechanism of the suggested involvement of the SCF^{COI1}-JAZ-co-receptor complex is, however, not clear. The compound which inhibits seed germination is OPDA and not JA/JA–Ile, as checked with mutants of enzymatic steps downstream of OPDA formation (Dave et al. 2011; Dave and Graham 2012; Goetz et al. 2012). OPDA cannot be perceived via the SCF^{COI1}-JAZ co-receptor complex (Thines et al. 2007) (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones").

Growth and Light: Plant growth is influenced by light in developmental programs such as photomorphogenesis, skotomorphogenesis, and shade avoidance syndrome (SAS) which have been studied intensively (Chory 2010; Lau and Deng 2010). Involvement of JA/JA–Ile, however, was analyzed only recently. Requirement for MYC2 activity, decreased defense against herbivores or necrotrophic pathogens upon silencing of JA/JA–Ile signaling components, and involvement of the JA/JA–Ile-linked MED25 (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones") in phytochrome B-mediated SAS are few examples. The different aspects of JA/JA–Ile in light signaling have been reviewed (Lau and Deng 2010; Ballaré 2011; Ballaré et al. 2012; Kazan and Manners 2011; Wasternack and Hause 2013) and are not repeated here to avoid overlap.

Growth inhibition is an early observed physiological effect of JAs (Dathe et al. 1981). An explanation could be given by wound-induced inhibition of mitosis (Zhang and Turner 2008). The endogenous rise in JA after wounding of leaves occurs in all dicotyledonous plants tested so far. Even repeated touching of leaves leads to increase in JA which is sufficient to inhibit growth (Chehab et al. 2012; Tretner et al. 2008). Recently performed analysis of effects of JA showed COI1-dependent arrest in endo-reduplication cycle, in mitotic cycle during the G1 phase, and in downregulation of key determinants of DNA replication (Noir et al. 2013). The final output of these JA/JA–Ile effects is reduced expansion, growth, size, and number of cells which leads to reduced leaf size.

Root growth inhibition is a regularly performed assay for action of jasmonates and was used for screening of mutants in JA biosynthesis and JA/JA–Ile signaling, e.g., *jar1*, a JA-insensitive mutant (cf. Table 1), has been identified via root growth inhibition (Staswick et al. 1992). Root growth inhibition is COI1 dependent. Involvement of JA/JA–Ile is also indicated by the stunted root growth phenotype of *cev1* plants which have constitutively elevated JA/OPDA levels (Ellis et al. 2002).

NINJA, the corepressor of JA/JA–Ile signaling acting together with JAZ proteins (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones"), is indispensible in repressing JA/JA–Ile signaling in roots and keeps normal root growth (Acosta et al. 2013). The complex nature of root growth is now studied by system biology approaches (Band et al. 2012a) which showed hierarchic interaction of GA, auxin, CK, and JA. Due to the abovementioned cross-talk among these hormones during JA/JA–Ile perception and signaling (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones"), the outcome of root growth inhibition is given by altered cell division, membrane traffic, cell wall loosening and synthesis, as well as altered turgor and growth rate. All of them affect hormonal and mechanic signaling (Band et al. 2012b). Auxin, the key player in root growth, is influenced by (1) JA/JA–Ile-induced *ASA1* expression, required for auxin biosynthesis (Sun et al. 2009); (2) JA-induced redistribution of PIN-FORMED2, an auxin transporter (Sun et al. 2011); and (3) JA/JA–Ile-induced MYC2-dependent repression of PLETHORA, required for stem cell niche activity (Chen et al. 2011). Furthermore, in rice the outcome of root growth inhibition is determined by root cell elongation which is regulated by a ternary complex of JAZ proteins, bHLH TFs, and a nuclear factor active in rice salt stress (Toda et al. 2013).

Lateral root formation is influenced by JA/JA–Ile via the abovementioned cross-talk with auxin. Genes involved in JA/JA–Ile formation such as *AtAOC3* and *AtAOC4* have high promoter activity in emerging lateral roots (Stenzel et al. 2012), and the JA/JA–Ile-insensitive *coi1-16* plants have less lateral roots (Zhang and Turner 2008). But also a JA/JA–Ile-independent signaling seems to be involved, since 9-LOX products derived from LOX1 and LOX5 negatively regulate lateral root formation (Vellosillo et al. 2007).

Adventitious root formation is a multifactorial process with involvement of auxin, cytokinin, and JA/JA–Ile (Da Costa et al. 2013). Key player is auxin that acts as an inducer by regulating JA/JA–Ile homeostasis (Gutierrez et al. 2012). Auxin regulates ARF6 and ARF8 in a positive manner. Downstream of auxin, adventitious root formation is negatively regulated by JA/JA–Ile in a COI1- and MYC2-dependent manner. Consequently, *coi1-16, myc2, myc3, myc4*, and *jar1* mutant plants have more adventitious roots than the wild type (Gutierrez et al. 2012).

Gravitropism is a morphogenic response caused by auxin redistribution and intra- and intercellular communication. Besides the mechanistic framework of cross-talk in auxin and JA/JA–Ile signaling, gradients of auxin, JA/JA–Ile, and auxin responsiveness have been detected during gravitropic response. This supports the traditionally used Cholodny–Went hypothesis for explanation of asymmetric growth (Gutjahr et al. 2005).

Trichomes, preferentially glandular trichomes, are "factories" for production of secondary metabolites such as terpenoids, flavonoids, alkaloids, and defense proteins (Tian et al. 2012; Tissier 2012). Therefore, glandular trichomes are involved in resistance to insects as shown by the *odorless-2* tomato mutant (Kang et al. 2010). Identification of *jai1*, the tomato homologue of *AtCOI1,* clearly showed requirement for intact JA/JA–Ile-signaling in trichome formation (Li et al. 2004). Trichome density and JA/JA–Ile-inducible defense compounds such as monoterpenes,

sesquiterpenes, and PINs are involved in resistance to herbivores (Tian et al. 2012). Trichome initiation is dependent on TFs such as MYB75, GL3, and EGL3 which are targets of JAZ proteins (Qi et al. 2011) (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones").<> Among trichome-specific enzymes involved in synthesis of secondary metabolites such as pyrethrins of *Pyrethrum* are two LOXs which convert α-LeA to 13-HPOT (Ramirez et al. 2013). The pyrethrins cinerolone, jasmolone, and pyrethrolone are assumed to be synthesized from the JA derivative 7-OH-JA (cf. section "Metabolism").

Tuber formation was assumed to be dependent on 12-OH-JA. In the late 1980s, 12-OH-JA was named tuberonic acid (TA) due to its tuber-inducing activity (reviewed by Wasternack and Hause 2002). Later on, involvement of StLOX1 in tuber formation (Kolomiets et al. 2001) and accumulation of JA and TA in stolons under low tuber-inducing temperature were shown (Nam et al. 2008). These data on TA, however, are only correlative. The effect could be indirect. Meanwhile, a conclusive scenario of tuber formation has been established. In this scenario, the potato orthologues of CONSTANS and FLOWERING LOCUS T are involved (Rodríguez-Falcón et al. 2006).The gene encoding the homeobox TF BEL5 is expressed in a phytochrome B-dependent manner, and its mRNA is transported under short-day conditions and at low temperature from leaves to the stolon tip via the phloem (Hannapel 2010; Lin et al. 2013). Finally, the *GA-20 oxidase1* promoter binds StBEL5 and another TF, POTH1, leading to increased GA levels (Banerjee et al. 2006; Lin et al. 2013). Interestingly, the phloem transport of *StBEL1* mRNA is accompanied with a phloem transport of mRNAs of Aux/IAA-encoding genes which leads to suppression of root growth (Hannapel 2013). Possibly, the role of TA is indirect by altering cell expansion.

Senescence: Senescence is a complex developmentally and environmentally regulated process. Nutrient availability, biotic and abiotic stress, and light/dark conditions influence senescence. Among senescence-related hormones, JA is known for a long time as a senescence-promoting factor (Ueda and Kato 1980). Aspects on senescence were reviewed recently (Guo and Gan 2012; Zhang and Zhou 2013). Transcript profiling in different stages of senescence led to a leaf senescence database (Buchanan-Wollaston et al. 2005; Liu et al. 2011) and identification of JA-linked TFs such as WRKY53 (Miao and Zentgraf 2007), WRKY54, and WRKY70 (Besseau et al. 2012) and TFs of the NAC family (Balazadeh et al. 2010). For the latter, e.g., ANAC019, ANAC055, and ANAC072, a regulatory network was shown recently indicating similarities and divergence among activities of TFs in stress responses (cf. section "Perception of JA-Ile and Cross-Talk to Other Hormones") and senescence downstream of MYC and MYB TFs (Hickman et al. 2013). The NAC TF ORE1 (ANAC092) is a positive and central regulator of senescence (Matallana-Ramirez et al. 2013). Other components of JA/JA–Ile-mediated senescence are (1) the COI1-dependent downregulation of RUBISCO activase (Shan et al. 2011), (2) the JA/JA–Ile-induced chlorophyll degradation (Tsuchiya et al. 1999), (3) the cross-talk to ET (Wang et al. 2013) or CK (van Doorn et al. 2013), and (4) the recruitment of JA/JA–Ile signaling in the absence of functional plastoglobule kinases accompanied with conditional de-greening (Lundquist et al. 2013).

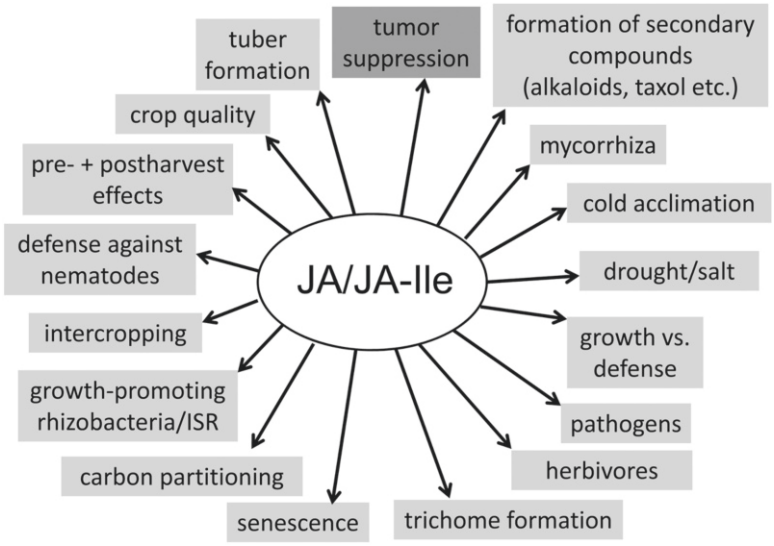

Fig. 6 Scheme on applied aspects of jasmonates in horticulture, pharmacy, and biotechnology. The accumulated knowledge on role of jasmonates in formation of secondary compounds; in defense reactions against pathogens, nematodes, or herbivores; in senescence, pre- and post-harvest, crop quality; or in arbuscular mycorrhiza led to their increased application (with permission)

Applied Aspects on Jasmonates

Upon two decades of JA research on JA-biosynthesis and JA-mediated signal transduction pathways in plant stress responses and development, an increasing interest is obvious to use this knowledge for horticultural applications. There are several examples summarized in Fig. 6, showing how JA/JA–Ile-mediated processes can be used in agriculture for improved plant growth, harvest, biotechnological production of secondary metabolites, or improvement of plant immunity. Applied aspects on jasmonates have been reviewed recently (Wasternack 2014). Therefore, only few examples will be briefly discussed here.

Freezing Tolerance: JA/JA–Ile is clearly a positive regulator of freezing tolerance (Hu et al. 2013b). Inhibition of JA/JA–Ile biosynthesis and signaling leads to hypersensitivity to freezing. The key players in cold stress, CBF1/DREB1, are JA/JA–Ile inducible, and ICE (INDUCER OF CBF EXPRESSION1) is a target of JAZ1 and JAZ4.

Defense Against Root Nematodes: Roots are attacked by root-knot and cyst nematodes which are endoparasites. These parasites use plant nutrients for their own lifestyle (Gheysen and Mitchum 2011). Worldwide there is about 5 % crop loss by root-knot nematodes of the genus Melogyne which attack about 200 mono- and

dicotyledonous species. Nematodes inject after invasion effector proteins into the host leading to a dramatic reprogramming of gene expression. Besides auxin, ET, and BR, JA is involved in systemically induced defense reactions against root nematodes (Nahar et al. 2013). Knowledge on participating signaling components will improve putative application. Here, simultaneously active shoot-feeding insects have to be taken into account. There is a compensatory plant growth response by herbivores which affects nematode invasion (Wondafrash et al. 2013).

Intercropping: Mixed growth of two or more crops, called intercropping, is of increasing interest due to obvious disadvantages of plant growth in monocultures. More than 28 million hectare in China is used already by intercropping. An interesting example is the maize/peanut intercropping which improves iron content of plants on calcareous soil (Xiong et al. 2013). In both plants, stress-related proteins are downregulated in a JA-dependent manner, initiated by interactions via the rhizosphere. A JA/JA–Ile-mediated advantage in intercropping systems is also given by volatile organic compounds (VOCs) which strongly interfere with insect interactions (Poveda and Kessler 2012).

A pesticide-free management of agroecosystems is envisaged by growing the right plants together. Maize plants growing together with legumes are much less attacked by the adult stem borer moth due to VOC emission, whereas grasses growing at the boarder of a maize field can attract gravid females away from maize plants (Hassanali et al. 2008). There are increasing examples, how plant–plant communications can be used for agricultural improvement. In the rhizosphere, root exudates attribute to communication, whereas in the atmosphere volatile compounds such as VOCs including JAMe are active.

Pre- and Post-harvest Effects and Crop Quality: Infection by *Botrytis* and green mold is the reason for the most frequently appearing loss in post-harvest (Rohwer and Erwin 2008). The role of JA/JA–Ile in infection by necrotrophic pathogens like *B. cinerea* is well understood. Consequently, application of JA and JA/JA–Ile-mediated volatile production are frequently used to establish resistance by pre- and post-harvest treatments. Crop quality can be improved by JAMe treatment. Here, (1) accumulation of "healthy" compounds such as resveratrol in case of *Vitis vinifera* leaves (Ahuja et al. 2012), (2) JA-induced accumulation of anthocyanins and antioxidant compounds in fruits and vegetables (Wang and Zheng 2005), or (3) JA/JA–Ile-induced GS formation in cruciferous vegetables (Grubb and Abel 2006) can be of interest. The latter aspect can be reached by JA treatment under field conditions without loss in post-harvest quality (Ku et al. 2013). Compounds of pharmaceutical interest such as alkaloids, taxol, or saponins are "produced" in plant cell cultures or via transgenic approaches due to their induction by JA/JA–Ile. During post-harvest of crops, herbivore resistance can be enhanced by using plant-circadian clock function for fitness (Goodspeed et al. 2013).

Jasmonates in Cancer Therapy: Jasmonates are unique for plants and do not occur in human tissues. There is, however, an anticancer activity of several JA compounds at least in several human cell lines (cf. review of Cohen and Flescher 2009).

JAs exert cytotoxic effects on cancer cells by direct cell death induction via interference with energy production, mitochondrial perturbation, and ROS production and/or via cell cycle arrest, redifferentiation, and anti-inflammatory properties (Raviv et al. 2013). Most strategies for use of JAs in anticancer therapy are based on improved chemical synthesis, increase in pharmacokinetic stability, and development of new JA compounds. There are, however, already natural sources of plants which are used for a long time for preparation of pharmaceutical drugs with anticancer activity. Among them are extracts of mistletoe (*Viscum album*). A putative explanation was found recently. Mistletoe plants have a JA content of about four orders of magnitude higher levels than most other plants, such as *A. thaliana*, tomato, or tobacco, even if these plants were wounded (Miersch and Wasternack, unpublished). Natural sources such as algae extracts or treatment with JAMe have been repeatedly described to exert anticancer activity in prostate cancer (Farooqi et al. 2012).

Soil Microbe Communities: There is a remarkable growth promotion of *Arabidopsis* by soil microbes which includes a facilitation of iron uptake, downregulation of genes involved in nitrogen uptake, redox signaling, and SA-mediated signaling, whereas genes involved in JA signaling, photosynthesis, and cell wall synthesis were upregulated (Carvalhais et al. 2013). There are about 10^{11} microbes with up to 30,000 prokaryotic species per gram roots in the rhizosphere near the roots (Berendsen et al. 2012). Among them are pathogenic, beneficial, and commensal microbes. Pathogen infection leads to damage by root growth inhibition caused by toxic compounds of bacterial origin. Colonization by beneficial microbes, however, can result in growth promotion or ISR. Soil-borne beneficial microbes such as *Pseudomonas* spp. *rhizobacteria* can establish protection against abiotic stress, may prime the plant immune system, and can change the root architecture (Zamioudis et al. 2013).

Simultaneously Applied Stresses: Most analyses of stress responses include single stress scenarios. In nature, however, several biotic and abiotic stresses occur simultaneously and/or subsequently. Consequently, for any application in agriculture, data collection has to be envisaged by simultaneously performed, multiple stresses. In an initial transcriptome-based comparison of single and double stresses, about 60 % of transcripts upon double stress could not be predicted by single stress data (Rasmussen et al. 2013). Another transcriptome data set on simultaneously performed biotic and abiotic stress showed regulation of specific genes, which are involved in several stress responses, but also an overriding property of abiotic stress on the response to biotic stress (Atkinson et al. 2013). Transcriptome and metabolome analyses of a multifactorial stress experiment including heat, drought, and virus infection revealed specific genes for single, double, and triple stress conditions including altered biotic stress responses by abiotic stress application (Prasch and Sonnewald 2013). This balance between abiotic and biotic stress responses was inversed in case of photoprotection versus defense. *Arabidopsis* mutants affected in key components of the chloroplast photoprotection system showed elevated oxylipin levels (JA/JA–Ile, OPDA) and increased defense against herbivores and pathogens (Demmig-Adams et al. 2013). Obviously, any balance between abiotic and biotic stresses is not optimal in plants and is of great impact on any agricultural application.

Conclusions

After two decades of JA research based on analytical, genetic, molecular, and cell biological approaches, principles in biosynthesis, perception, signaling, and action of JA/JA–Ile have been elucidated. Signaling modules and similarities to other hormones as well as the network of cross-talk among all of them are milestones in this new knowledge. Transcriptomic, proteomic, lipidomic, and metabolomic analyses led to a vast amount of data which will be extended on new conditions and will lead to system biology approaches. Complex analyses will be performed on:

1. JA/JA–Ile action in stress responses and development under natural conditions
2. Simultaneous and/or subsequent action of two or more stresses in relation to JA/JA–Ile signaling
3. JA/JA–Ile-dependent balance of growth and development
4. JA/JA–Ile-based communication of plants via the rhizosphere and the atmosphere
5. JA/JA–Ile-mediated plant productivity in terms of secondary and macromolecular compounds

These global questions will be underpinned by mechanistic studies in JA/JA–Ile-signaling leading to identification of:

1. New regulatory components around the well-established SCF^{COI1}-JAZ-co-receptor complex
2. Translational and posttranslational control mechanisms including protein phosphorylation and protein stability
3. Epigenetic regulation of biosynthesis and signaling of JA/JA–Ile
4. Stress-specific and developmentally specific regulators active in JA/JA–Ile signaling

It will be fascinating to see the concerted progress in plant hormone research including JA/JA–Ile.

Acknowledgements The author's work was supported by the Deutsche Forschungsgemeinschaft (SFB 363 project C5 and SFB 648 project C2) and the Region HANA for Biotechnological and Agricultural Research, Czech Republic (grant no. ED0007/01/01). The author thanks Prof. Dr. B. Hause (IPB, Halle/Saale, Germany) for the helpful discussions and critical reading of the manuscript. I thank Wiley for copyright transfer of Fig. 1 (initially published in Wasternack 2006; Plant Hormone Signaling, Eds. Hedden, P. and Thomas, S. G.); Oxford University Press for Figs. 2, 3, and 4 (initially published in Wasternack and Hause 2013); Elsevier for Fig. 5 (initially published in Wasternack and Hause 2002); and Elsevier for Fig. 6 (initially published in Wasternack 2014).

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