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REVIEW

Perception, signaling and cross-talk of jasmonates and the seminal contributions of the Daoxin Xie's lab and the Chuanyou Li's lab

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Abstract Jasmonates (JAs) are lipid-derived signals in plant responses to biotic and abiotic stresses and in development. The most active JA compound is (+)-*7-iso*-JA-Ile, a JA conjugate with isoleucine. Biosynthesis, metabolism and key components of perception and signal transduction have been identified and numerous JA-induced gene expression data collected. For JA-Ile perception, the SCF^{COI1}–JAZ co-receptor complex has been identified and crystalized. Activators such as MYC2 and repressors such as JAZs including their targets were found. Involvement of JA-Ile in response to herbivores and pathogens and in root growth inhibition is among the most studied aspects of JA-Ile signaling. There are an increasing number of examples, where JA-Ile shows cross-talk with other plant hormones. Seminal contributions in JA/JA-Ile research were given by Daoxin Xie's lab and Chuanyou Li's lab, both in Beijing. Here, characterization was done regarding components of the JA-Ile receptor, such as COI1 (JAI1) and SCF, regarding activators (MYCs, MYBs) and repressors (JAV1, bHLH IIId's) of JA-regulated gene expression, as well as regarding components of auxin biosynthesis and action, such as the transcription factor PLETHORA active in the root stem cell niche. This overview reflects the work of both labs in the light of our present knowledge on biosynthesis, perception and signal transduction of JA/JA-Ile and its cross-talk to other hormones.

Keywords Jasmonate biosynthesis · Jasmonate perception · Jasmonate signal transduction · Cross-talk · Daoxin Xie's lab · Chuanyou Li's lab

Abbreviations

ABA	Abscisic acid
AOS	Allene oxide synthase
BR	Brassinosteroid
COI1	CORONATINE INSENSITIVE1
ET	Ethylene
GA	Gibberellic acid
HPL	Hydroperoxide lyase
JA	Jasmonic acid
JA-Ile	JA isoleucine conjugate
JAMe	JA methyl ester
JAR1	JA resistant1
JAZ	JASMONATE ZIM DOMAIN
α-LeA	α-Linolenic acid (18:3)
LOX	Lipoxygenase
MYB	R2R3-type TFs
MYC	bHLHzip-type TFs
OPDA	12-Oxophytodienoic acid
PIN2	PIN-FORMED2
PLT	PLETHORA
SA	Salicylic acid
TF	Transcription factor
SCF	Skp1/Cullin/F-box

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Introduction

The methyl ester of jasmonic acid (JA) was isolated and identified in the odor of flowers of *Jasminum grandiflorum* in 1962 (Demole et al. 1962). Two decades later, senescence promotion (Ueda and Kato 1980) and growth inhibition (Dathe et al. 1981) were the first physiological effects described for JA. The JA biosynthesis pathway was elucidated by Vick and Zimmerman (1983) in the 80s of the last century followed in the early 90s by identification of JA as the key player in herbivore-induced synthesis of defense proteins such as proteinase inhibitors (PIs) (Farmer and Ryan 1990) and of many other processes such as alkaloid synthesis (Gundlach et al. 1992). Meanwhile, JA and its conjugate with isoleucine (JA-Ile) have been described as important signals in plant responses to biotic and abiotic stress as well as in development (cf. review of Wasternack and Hause 2013). Numerous gene expression programs induced by JA have been elucidated. Key components of JA perception and JA signal transduction were identified and characterized. Several proteins involved in synthesis of JA-Ile and in JA-Ile perception have been crystallized. First insights into the cross-talk of JA to other hormones building a regulatory network of action have been obtained.

Two groups in Beijing were heavily involved in several breakthroughs in JA research during the last 15 years. Daoxin Xie's lab (School of Life Sciences, Tsinghua University, Beijing) identified and characterized the first JA-specific F-box protein, COI1 of Arabidopsis, and contributed by identification of essential components of JA-induced gene expression and proteasomal degradation of repressors. Chuanyou Li's lab (Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, Beijing) identified essential components of JA biosynthesis and signaling of tomato, aspects of systemic signaling including defense responses and the role of auxin and its cross-talk to JA in root growth of Arabidopsis. The following overview is a summary on JA perception, JA signal transduction and cross-talk to other hormones highlighting the contributions of Daoxin Xie's lab and Chuanyou Li's lab.

JA biosynthesis

The biosynthesis of JA has been repeatedly reviewed in recent years (Wasternack and Kombrink 2010; Kombrink 2012; Wasternack and Hause 2013). To avoid repetitions, only some recent aspects required for the following paragraphs are discussed here.

The first half of JA biosynthesis takes place in plastids (Fig. 1). Substrate is α-linolenic acid (18:3) (α-LeA). An ω-3-fatty acid desaturase is required for its formation from

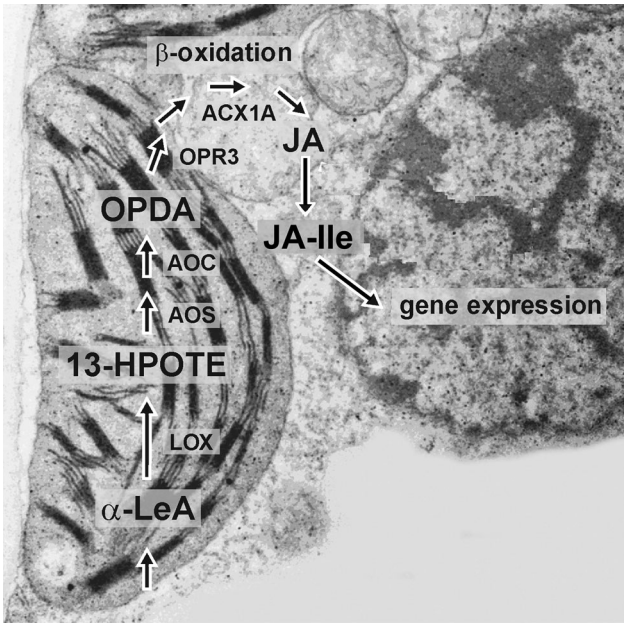

Fig. 1 Scheme on synthesis of jasmonic acid isoleucine conjugate (*JA-Ile*) in three different compartments illustrated on an electron microscopic picture of a barley mesophyll cell. α-Linolenic acid (*α-LeA*) is released from chloroplast membranes and is converted by a lipoxygenase (*LOX*) to 13-hydroperoxy octadecatrieneoic acid (*13-HPOTE*). Allene oxide synthase (*AOS*) and allene oxide cyclase (*AOC*) catalyze formation of 12-oxophytodienoic acid (*OPDA*) which is transported into the peroxisome, where an OPDA reductase3 (*OPR3*), an acyl-CoA oxidase1A (*ACX1A*) and further enzymes of the fatty acid β-*oxidation* machinery catalyze formation of jasmonic acid (*JA*). In the cytosol, a synthetase (*JAR1*) catalyzes formation of JA-Ile which leads to gene expression in the nucleus. A part of the vacuole can be seen in the lower part of the picture (photograph B. Hause) [modified after Wasternack (2007) with permission]

α-linoleic acid (18:2). The tomato enzyme has been cloned by Chuanyou Li in G. Howe's lab (East Lansing, USA) using the *spr2* mutant (suppressor of prosystemin-mediated response2) (Table 1); (Li et al. 2003). This mutant is JA deficient. Therefore, the mutant became a strong tool in analysis of wound-induced JA formation and systemic signaling (Li et al. 2002). The released α-LeA is oxygenated by plastid-located lipoxygenases (LOXs) at C-13 (13-LOXs). LOXs occur in gene families with six members in Arabidopsis and four members in tomato. Four of the six Arabidopsis LOXs are 13-LOXs and are involved in wound-induced JA biosynthesis in an organ- and tissue-specific manner. LOX3 and LOX4 are involved in anther-specific JA formation (cf. review of Wasternack and Hause 2013). A new 13-LOX of tomato (TomLOXD) has been recently identified in Chuanyou Li's lab by characterizing the *spr8* mutant (Table 1); (Yan et al. 2013b). The lack of JA formation in *spr8* mutant plants leads to deficiencies in immune responses, such as defense protein synthesis and glandular trichome formation, and to compromised resistance to herbivores or necrotrophic pathogens. In contrast,

Table 1 Key components in jasmonate (*JA*) biosynthesis, perception and signaling of Arabidopsis and tomato (contributions of the Daoxin Xie's lab and the Chuanyou Li's lab are indicated by small letters)

Gene	Protein	Function	Reference
Biosynthesis			
SPR2 (*tomato*)	SUPRESSOR OF PROSYSTEMIN RESPONSE2/fatty acid ω-3-desaturase	JA biosynthesis	Li et al. (2003)[a,b]
SPR8 (*tomato*)	SUPRESSOR OF PROSYSTEMIN RESPONSE8/LIPOXYGENASED	JA biosynthesis	Yan et al. (2013b)[a]
AOC	ALLENE OXIDE CYCLASE	JA biosynthesis	Stenzel et al. (2003)
OPR3	OPDA REDUKTASE3	JA biosynthesis	Stintzi and Browse (2000); Sanders et al. (2000)
JAR1	JASMONATE RESISTANT1/JA amino acid synthase	JA-Ile biosynthesis	Staswick and Tiryaki (2004)
Perception			
SCF^{COI1}	SKP/CUL1/F-box protein (JA)	E3 ubiquitin ligase	Xu et al. (2002)[c]
ASK1,2	Arabidopsis SKP-LIKE PROTEIN1,2	SCF components	Liu et al. (2004)[c]
CUL1	CULLIN1	adaptor protein in SCF^{COI1} complex	Ren et al. (2005)[c]
COI1 (*Arabidopsis*)	CORONATINE INSENSITIVE1	F-box protein	Xie et al. (1998)[c,d]; Yan et al. (2013a, b)[c]
JAI1 (tomato)	JASMONATE INSENSITIVE1	F-box protein	Li et al. (2004)[a,b]
Signaling			
COS1	COI1 SUPPRESSOR	Riboflavin synthesis	Xiao et al. (2004)[c]
MYC2/JIN1	JASMONATE INSENSITIVE1	TF (activating and repressing)	Lorenzo et al. (2004)
MYB21, *24*	R2R3 TYPE TF	TF (activating)	Song et al. (2011)[c]
PLT1,2	PLETHORA1,2	TF (activating)	Chen et al. (2011a)
JAM1, *2*, *3*	JASMONATE ASSOCIATED MYC2-LIKE1, 2, 3	TF (repressing)	Sasaki-Sakimoto et al. (2013); Nakata et al. (2013)
JAZ	JASMONATE ZIM DOMAIN	Repressor of TF	Thines et al. (2007); Chini et al. (2007); Yan et al. (2007)
JAV1	JASMONATE ASSOCIATED VQ MOTIF	Repressor of TF	Hu et al. (2013)[c]
NINJA TPL	NOVEL INTERACTOR OF	Co-repressor	Pauwels et al. (2010)
	JAZ TOPLESS	Co-repressor	Pauwels et al. (2010)
MED25	MEDIATOR SUBUNIT25	Mediator complex (RNA polymerase interactor)	Chen et al. (2012)[a]

TF Transcription factor

[a] Chuanyou Li's lab

[b] Chuanyou Li's lab in collaboration with Gregg Howe's lab

[c] Daoxin Xie's lab

[d] Daoxin Xie's lab in collaboration with John Turner's lab

the wound-inducible TomLOXC is involved in HYDROPEROXIDE LYASE (HPL)-independent C-5 and C-6 volatile formation without impact on pathogen resistance (Shen et al. 2014).

Among the seven different branches of the LOX pathway (Feussner and Wasternack 2002), one branch via the ALLENE OXIDE SYNTHASE (AOS) leads to JA, whereas a concurrent branch via the HPL leads to volatile leaf alcohols and leaf aldehydes. Chuanyou Li's lab identified a HPL of rice affected in the so-called *cea62* mutant (Liu et al. 2012). *cea62* plants exhibit constitutive expression of *OsAOS* and JA overproduction including growth defects. In these plants, there is a competing activity between the AOS branch and the HPL branch as observed also in Arabidopsis with the consequences for indirect and direct defense mechanisms (Chehab et al. 2008). Most attention has been paid to the AOS branch due to the central role of JA-Ile in stress responses and development. The AOS and the subsequently active ALLENE OXIDE CYCLASE (AOC), both of them partially bound to plastid membranes (Farmaki et al. 2007), catalyze formation of the cyclopentenone *cis*-(+)-12-oxophytodienoic acid (OPDA). In the AOC-catalyzed step, the enantiomeric form of

the naturally occurring JA is established (Ziegler et al. 2000). OPDA is transported into peroxisomes, where an OPDA reductase3 (OPR3) catalyzes reduction of the cyclopentenone ring (Fig. 1). *opr3* plants are JA deficient but able to form OPDA (Stintzi and Browse 2000; Stintzi et al. 2001). Subsequent shortening of the carboxylic acid side chain takes place by the fatty acid β-oxidation machinery and is initiated by an ACYL-CoA-OXIDASE1 (ACX1). Characterization of ACX1 of tomato done by Chuanyou Li et al. (2005) in G. Howe's lab was among the first proofs for an involvement of fatty acid β-oxidation in JA biosynthesis. Like the *opr3* mutant of Arabidopsis, *acx1* plants of tomato became a tool to distinguish between OPDA- and JA-Ile-dependent signaling (Li et al. 2005; Schilmiller et al. 2007; Koo et al. 2009; Goetz et al. 2012). Several enzymes of JA biosynthesis have been crystallized, such as AOS, AOC, OPR3 and ACX1 (cf. review of Wasternack and Kombrink 2010). JA biosynthesis is regulated by substrate availability, a positive feedback loop and tissue specificity, and Ca^{2+} signaling and MAPK cascades are involved (cf. reviews of Wasternack 2007; Balbi and Devoto 2008; Wasternack and Hause 2013). Expression of JA biosynthesis genes is regulated in a coordinate manner by transcription factors (TFs) such as MYC2 and repressed by ANTHER INDEHISCENCE FACTOR, a NAC-like TF (Shih et al. 2014) as well as by JAZ proteins (cf. below).

There are 12 different metabolic routes of JA. Among them, conjugation of JA with amino acids such as isoleucine to JA-Ile by a member of the GH3 protein family, the JA amino acid synthetase (JAR1), is the most important reaction (Staswick and Tiryaki 2004). A specific enantiomeric form, (+)-7-*iso*-JA-Ile, is the most bioactive JA compound (Fonseca et al. 2009) and is bound by the JA receptor (Sheard et al. 2010) (cf. below). Therefore, JA/JA-Ile is used here as a module. JAR1 has been crystallized (Westfall et al. 2012). Other metabolic routes of JA include hydroxylation to 12-hydroxy-JA or 12-hydroxy-JA-Ile, sulfonation of 12-hydroxy-JA, carboxylation and glucosylation of 12-hydroxy-JA-Ile. Many of these metabolites are biologically inactive. Consequently, their formation represents a switch-off in JA signaling (Miersch et al. 2008; Koo et al. 2011; Heitz et al. 2012).

COI1, the SCF complex and repressors such as JAZs, JAMs and JAV1

In the 90s of the last century, elucidation of biosynthesis of most plant hormones was completed, but was improved in case of auxin, strigolactones and brassinosteroids (BR) in the following decades. The perception of plant hormones, however, was largely unknown except initial work on ethylene. Fundamental breakthroughs were published in 1998 for auxin by identification of TIR1 as an F-box protein, active as auxin receptor, in M. Estelle's lab (Ruegger et al. 1998) and for JA by identification of COI1 as an F-box protein by Xie et al. (1998), at that time in J. Turner's lab in Norwich, UK. In a mutant screen with the bacterial toxin coronatine, which is a molecular mimic of JA-Ile, the Arabidopsis mutant *coi1* (*coronatine insensitive1*) was initially identified in J. Turner's lab (Feys et al. 1994). The subsequent sequencing of *COI1* and identification of the encoded protein as an F-box protein via its F-box domain strongly suggested a role of COI1, similar to TIR1 in case of auxin, in an SKP/CULLIN/F-BOX (SCF)-mediated proteasomal degradation (Xie et al. 1998). This role of COI1 was shown in Daoxin Xie's lab by a functional proof on physical interaction between COI1 and other components of the SCF^{COI1} complex such as AtCUL1, AtRbx1 and one of the Skp-like proteins ASK1 and ASK2 leading to an active ubiquitin ligase complex designated as SCF^{COI1} (Xu et al. 2002). CULLINs, encoded by a gene family of 11 members in *Arabidopsis thaliana*, function as scaffold proteins within the SCF complexes and occur as evolutionary conserved proteins in fungi, plants and mammals (cf. review of Stratmann and Gusmaroli 2012). AtRbx1 is a RING-box domain protein, which binds to CULLIN and SKP1, thereby attributing to interaction of CULLIN-RING 3 ubiquitin ligase and the E2 ubiquitin-conjugating enzyme (Lechner et al. 2002). A single amino acid substitution in $COI1_{E22A}$ in the F-box motif of COI1 indicated the absolute requirement of intact COI1 for formation of the SCF^{COI1} complex (Xu et al. 2002). The SCF^{COI1} complex is only active in JA signaling, if an intact AXR1 required for CULLIN1 modification is present. Mutations in *COI1* and *AXR1* showed a synergistic genetic interaction in the double mutant (Xu et al. 2002). This requirement for a functional SCF^{COI1} complex in JA signaling was supported in a collaborative work with J. Turner's group (Norwich) (Devoto et al. 2002). In this stage of work on the SCF^{COI1} complex, its role in flower development suggested by the male sterile phenotype of *coi1* mutants was demonstrated (Ni et al. 2004). In further studies by Daoxin Xie's lab, the essential role of ASK1 and ASK2 in embryogenesis and seedling growth of Arabidopsis (Liu et al. 2004) and the essential role of CUL1 in complex assembling were shown by analysis of the mutants *axr6-1*, *axr6-2*, and *cul1*, all affected in JA responses (Ren et al. 2005).

The CULLIN1-based SCF^{COI1} complex is regulated by the COP9 signalosome (CSN), a multiprotein complex (cf. review of Stratmann and Gusmaroli 2012). Daoxin Xie's lab along with X. W. Deng's lab (Beijing) showed that physical interaction of the COP9 signalosome with the SCF^{COI1} complex and its role in modulation of the JA response (Feng et al. 2003). JA signaling was further substantiated by identification of downstream components. In a mutant screen of Daoxin Xie's lab for mutants carrying suppression of JA-dependent defects of *coi1* mutant plants, the *COS1* gene was cloned (Xiao et al. 2004). *COS1*

encodes a lumazine synthase, a key component of the riboflavin pathway of bacteria, fungi and plants. The riboflavin pathway was shown to be active downstream of COI1 and is required for suppression of COI1-mediated inhibition of root growth, senescence and plant defense (Xiao et al. 2004). These data further supported the role of COS1 in SCF^{COI1}-mediated JA signaling. The *cos1* mutation restored the defect of *coi1-2* suggesting that the riboflavin pathway is required for suppression of a putative negative regulator. Here, Daoxin Xie's lab showed one of the first hints that in JA signaling, proteasomal degradation via the SCF^{COI1} complex is required for removal of a repressor of gene expression similar to animal systems. 5 years later, these negative regulators were occasionally identified in three different labs by characterization of the JASMONATE ZIM DOMAIN (JAZ) proteins (Chini et al. 2007; Thines et al. 2007; Yan et al. 2007).

In summary, between 2002 and 2004 Daoxin Xie's lab identified and characterized, partially by collaborative work, the key components of a functional SCF^{COI1} complex such as COI1, ASK1, ASK2, COP9, CULLIN1 and AXR1 of *A. thaliana*. Details on JA perception, however, were still unclear. The above-mentioned similarities between COI1 and TIR1, the auxin receptor, led to the suggestion that COI1 might be a JA receptor (Woodward and Bartel 2005). A functional proof by pulldown-experiments, however, was not successful in several labs. A breakthrough was the identification of JAZ genes, a family of 12 members in Arabidopsis (Chini et al. 2007; Thines et al. 2007; Yan et al. 2007). JAZ proteins are negative regulators of positively acting TFs in JA-dependent gene expression. Based on these new data, a basic scenario on JA-Ile perception could be proposed in 2007 (Fig. 2): In a resting state, the positively acting TF MYC2 binds to a G-box of a JA-responsive gene and is repressed by a JAZ protein. Upon stress, an endogenous rise of JA-Ile occurs and is perceived by the SCF^{COI1} complex. The perception of JA-Ile causes release of the repressing JAZ protein from MYC2 and interaction of JAZ with the SCF^{COI1} complex. JAZ, the target of COI1, is subsequently subjected to proteasomal degradation upon ubiquitination (cf. review of Wasternack and Hause 2013). Based on this scenario and in order to identify a JA receptor, Daoxin Xie's lab developed a photoaffinity probe containing a coronatine moiety and a photoreactive group which allowed cross-linking to COI1 (Gu et al. 2010). This probe, called PACOR, was designed upon molecular modeling of COI1 with coronatine (Yan et al.

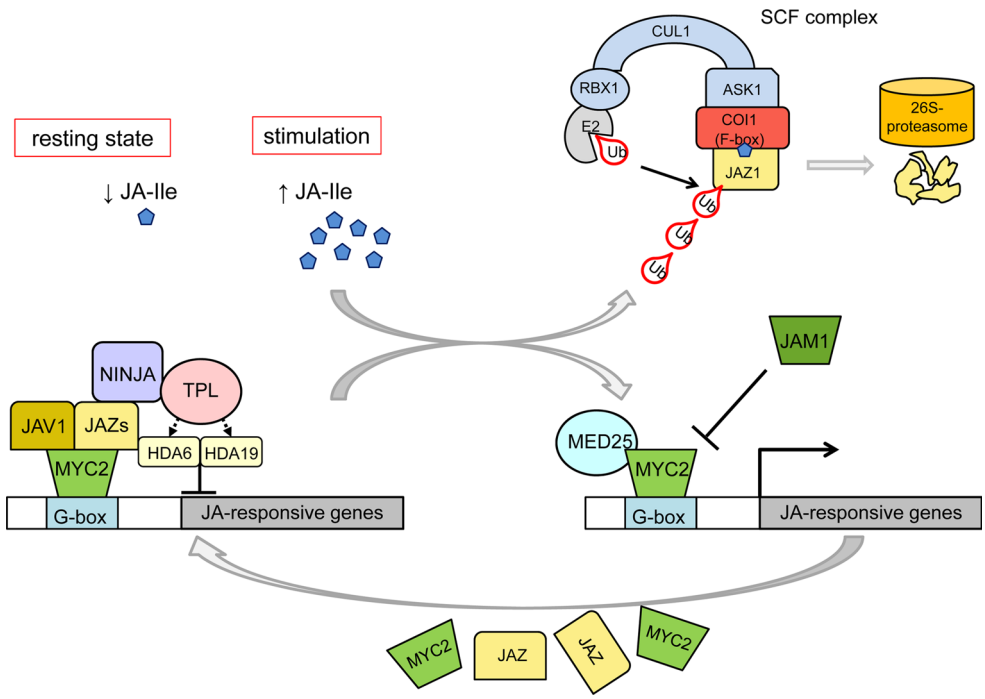

Fig. 2 JA/JA-Ile perception by the SCF^{COI1}–JAZ co-receptor complex leads to JA/JA-Ile-induced gene expression. MYC2 which bounds to a G-box of a JA/JA-Ile-responsive gene is repressed by negative regulators such as JAZs, mediated by co-repressors NINJA and TOPLESS (*TPL*) which acts via the HISTONDEACETYLASE6 (*HDA6*) and HDA19. In addition to JAZ proteins, JASMONATE ASSOCIATED VQ MOTIF GENE 1 (*JAV1*) acts as a repressor (*left side*), whereas JAMs (JASMONATE ASSOCIATED MYC2-LIKE1, JAM2, JAM3) (*right side*) are antagonists of MYC2 in its binding to the G-box. JAZs and JAV1 are ubiquitinylated and subjected to proteasomal degradation. 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. *Ub* ubiquitin; E2, Rbx, Cullin, ASK1, and the F-box protein COI1 are components of the SCF complex [modified after Wasternack and Hause (2013) with permission]

2009). Free PACOR which specifically interacts with COI1 was biologically active in vivo in a competitive manner to JA-Ile and could be cross-linked by UV light to COI1 (Yan et al. 2009). Additionally, Surface Plasmon Resonance measurements revealed the interaction of JA-Ile, COI1 and JAZ1. Finally, molecular modeling supported the idea that (+)-*7-iso*-JA-Ile binds similar as coronatine to the surface binding pocket of COI1 (Yan et al. 2009). Nearly simultaneously, (+)-*7-iso*-JA-Ile was identified as the most bioactive JA compound in 2009 (Fonseca et al. 2009). The final proof for a JA-Ile-receptor was given by crystallization of the SCF^{COI1}–JAZ1 co-receptor complex in 2010 (Sheard et al. 2010).

A further breakthrough in understanding JA perception was the identification of the co-receptor TOPLESS (TPL) and the adaptor protein "Novel interactor of JA" (NINJA) by tandem affinity purification using advantage of specific domains of JAZ proteins (Pauwels et al. 2010). Whereas the conserved *jas* domain of JAZ proteins is required for repression via interaction with the TF, the ZIM (TIFY) domain mediates homo- and hetero-dimerization as well as binding of NINJA which interacts with the second co-repressor TPL. Like JAZ proteins, TPL cannot bind directly to DNA. Obviously, histone deacetylases (HDAs) are involved, which attribute to suppression of gene expression by chromatin modification. HDA6 and HDA19 are genetically linked to TPL and can bind to TPL. In summary, the repression of JA-responsive gene expression by JAZ is based on *jas* domain-mediated binding to TFs and TPL binding to the chromatin-modifying HDA6 and HDA19 (Fig. 2). This scenario of repression in JA signaling was recently complemented by identification of an additional repressor, JAV1, in Daoxin Xie's lab (Hu et al. 2013). JAV1 was identified in a screen with about 20,000 transgenic plants. These plants contained a rolling circle amplification-mediated hairpin RNA (RMHR)-based JA-Ile-inducible hairpin RNA library and were screened on enhanced resistance against *Botrytis cinerea*, a strictly COI1- and JA-Ile-dependent defense response. The identified gene (At3g22160) with unknown function contains a VQ motif (Hu et al. 2013). Therefore, the gene was designated as *JA-associated VQ motif gene1* (*JAV1*). JAV1 integrates as a negative regulator defense reaction against insects and pathogens without a role in development (Hu et al. 2013). So far, all identified repressors were active in JA-dependent defense and development. Expression of *JAV1* and *JAZs* is inducible by JA-Ile. JAZ proteins are degraded in a COI1-dependent manner via proteasomal degradation. JAV1, however, does not interact directly with COI1 as it takes place with JAZ proteins and COI1 (Hu et al. 2013). Possibly, an unidentified COI1-dependent E3 ligase may direct JAV1 to proteasomal degradation. The concerted action of JAV1 in different defense responses suggests that downstream of JAV1 unidentified regulators of herbivory and infection might be active. Involvement of WRKY28 and WRKY51 could be suggested (Hu et al. 2013). The dual activity of JAV1 in responses to pathogens and herbivores allows agricultural application already at this stage of research.

JAZ and JAV1 proteins achieve their repressor properties by binding to the TF and are subjected to proteasomal degradation, if JA-Ile levels are elevated by environmental and developmental stimuli. Simultaneously, however, *JAZ* and *JAV1* gene expression takes place, since they are JA-Ile inducible. This JA-Ile-dependent balance between repression and induction is adjusted by additional regulatory components. Among them is the subunit 25 of the eukaryotic Mediator complex (MED25). This component was first identified in Chuanyou Li's lab in a screen on bestatin-resistant (*ber*) mutants (Zheng et al. 2006). Bestatin was known not only as an inhibitor of aminopeptidases but also as a powerful inducer of expression of wound response genes in tomato, a well-studied JA-Ile-dependent process (Schaller et al. 1995). These *ber* mutants could be classified into (1) bestatin-insensitive mutants with normal JA responses, (2) JA-insensitive mutants and (3) JA-hypersensitive mutants. The *ber* mutants became an excellent source for identification of novel loci involved in JA signaling. The strength and success of such a chemical genetics approach became obvious 6 years later: identification and characterization of the *ber6* mutant, initially detected by JA insensitivity in the JA-induced root growth inhibition assay, showed that a central regulator of JA and ABA signaling was affected. The affected gene encodes MED25 (Chen et al. 2012). Physical interaction of MED25 with MYC2 (JA signaling) and with ABI5 (ABA-signaling) highlighted how the MED25 subunit links different signaling pathways. The MED25 is a further piece of evidence for the complex regulation of hormone activity via common modules such as SCF^{COI1}–JAZ co-receptor complex, TFs such as MYC2 and adaptors and additional repressors (Fig. 2) (cf. review of Wasternack and Hause 2013).

Another type of repressors than JAZ and JAV1 was identified with the JASMONATE ASSOCIATED MYC2-like TFs, called JAM1, JAM2, and JAM3 (Nakata et al. 2013; Sasaki-Sekimoto et al. 2013). These proteins are ABA-inducible bHLH-type TFs which compete with MYC2 on target sequences of MYC2. Consequently, many JA-Ile-dependent processes can be repressed even JAZ and JAV1 are already degraded, thereby allowing a fine tuning of JA-Ile-induced gene expression. Another regulation in JA-Ile signaling is given by the stability of COI1 which was addressed recently in Daoxin Xie's lab (Yan et al. 2013a). In previous studies on the SCF^{COI1}–JAZ co-receptor complex, the interaction of the partners was preferentially analyzed. Now, the stability of COI1 was

inspected. A strict requirement of ASK1 and stabilization of COI1 by integrity of the SCF^{COI1} complex were found and suggest a dynamic balance of stabilization and degradation via the 26S proteasome. The Lys residue 297 was identified as the active ubiquitination site in COI1. Deletion of the F-box motif of COI1 accelerated its degradation. These and further data suggest that COI1 is not degraded auto-catalytically as known for other F-box proteins, but is recruited by another, unidentified E3 ligase for ubiquitination and proteasomal degradation (Yan et al. 2013a).

Surprisingly, the Arabidopsis F-box protein COI1 and its orthologue JAI1 of tomato are involved in different processes. COI1 is required for male fertility (Xie et al. 1998), whereas JAI1 is required for female fertility (Li et al. 2004). Furthermore, *jai1* mutant plants showed the absolute requirement of JA for trichome development and herbivore resistance (Li et al. 2004). In the last decade, this dual role of JA in defense and development has been described for many plant species.

Identification of JAZ targets and of new TFs involved in JA responses

An important issue in JA signaling is the type and the specificity of TFs which are under control of repressors such as JAZs, JAV1 or JAMs. So far, several interaction screens were performed to find targets of JAZ proteins. Solano's lab (Madrid) already identified MYC2 as a TF of JA-Ile-induced gene expression (Lorenzo et al. 2004) and showed the interaction of MYC2 with JAZ1 (Chini et al. 2007). MYC2 was described as a "master regulator" in JA-Ile signaling (cf. review of Kazan and Manners 2013). Nevertheless, Daoxin Xie's group was looking for other TFs in JA-Ile-regulated gene expression and their action as targets of JAZ proteins. First, JA-Ile-induced steps in anthocyanin biosynthesis and TFs involved in their regulation were characterized (Shan et al. 2009). Subsequently, TFs of the bHLH-MYC type (TT8, GLABRA3, EGL3) and of the R2R3 MYB type (MYB75 and GLABRA1) were shown in vitro and in planta to interact with JAZ proteins leading to suppression of anthocyanin formation and trichome initiation. Correspondingly, over-expression of *MYB75*, *GLABRA3* and *EGL3* in the *coi1*-mutant background led to restoration of both processes (Qi et al. 2011). Similar approaches revealed interaction of MYB21 and MYB24 with JAZ1, JAZ8 and JAZ11 and its specific involvement in pollen maturation, anther dehiscence and filament elongation (Song et al. 2011), which all are affected in the *coi1* mutant (Xie et al. 1998). Over-expression of *MYB21* in *coi1* mutant rescued fertility partially, but did not recover JA/JA-Ile-induced root growth inhibition and plant defense (Song et al. 2011). This was an important proof that specificity among the various JA-Ile-dependent processes, such as root growth inhibition, plant defense, anthocyanin formation, trichome initiation and anther development, is generated by interaction of JAZ proteins with different types of TFs. These aspects of MYB-type TFs in development, particularly in stamen development, and a more general view on phytohormone signaling via SCF^{COI1} and other SCF modules and JAZ proteins have been reviewed by Daoxin Xie's group (Shan et al. 2012; Song et al. 2013b). Additionally to the above-described TFs, MYC3 was identified as a TF in JA/JA-Ile-induced root growth inhibition with partial redundancy to MYC2 (Cheng et al. 2011). These data correspond to the results of R. Solano's lab (Madrid), where MYC3 and MYC4 were characterized (Fernández-Calvo et al. 2011). Recently, TFs of the bHLH subgroup IIId (bHLH3, bHLH13, bHLH14 and bHLH17) were identified as JAZ targets by Daoxin Xie's group (Song et al. 2013a). These TFs act redundantly as transcriptional repressors. They antagonize to positively acting TFs such as MYC2 and WD-repeat/bHLH/MYB complexes by binding to corresponding DNA target sequence (Song et al. 2013a). These data highlight a new level of regulation: downstream of the SCF^{COI1} complex and JAZ proteins, a balance of a subset of positively activating TFs (e.g. MYC2) and repressing TFs (e.g. bHLH3) may sustain fine tuning of gene expression and may regulate the ratios between the various JA/JA-Ile-dependent processes such as anthocyanin formation, plant defense and trichome development (Song et al. 2013a).

In MYC2-mediated signaling, the phosphorylation-coupled proteolysis of MYC2 was recently identified as a completely new regulatory level (Zhai et al. 2013). Whereas transcriptional regulation of JA/JA-Ile-induced gene expression by MYC2 is relatively well understood, nothing was known on its turnover. Now, a turnover of MYC2 could be correlated with expression of wound-induced genes (positive action by MYC2 accumulation) and de-repression of PR gene expression (negative action of MYC2 accumulation). This turnover is activated by phosphorylation of amino acid Thr 328, which is important for transcriptional activity of MYC2 (Zhai et al. 2013).

Whereas MYC2 is relatively well studied to be a master regulator of JA signaling, acting downstream of the SCF^{COI1}–JAZ co-receptor complex, downstream components of MYC2 were unknown until recently. Such components were found among the NAC proteins known as TFs involved in stress responses and some aspects of development. Chuanyou Li's group could identify ANAC009 and ANAC055, two NACs of Arabidopsis. Both of them function downstream of COI1 and MYC2 in JA-dependent signaling of responses to necrotrophic pathogens (Bu et al. 2008), and also in ABA-signaling (Bu et al.

2009). ANAC019 and ANAC055 interact with the RING-H2 protein RHA2a which is involved in ABA signaling. RHA2a is a functional E3 ligase which positively regulates responses to salt and osmotic stress during seed germination and early seedling development (Bu et al. 2009). Its homolog RHA2b is also an E3 ligase, and both of them act downstream of the protein phosphatase 2 (ABI2), presumably in a redundant manner (Li et al. 2011). The positive regulation by RHA2a and RHA2b is in line with the action of a bHLH-type TF, AtAIB, which positively regulates ABA responses (Li et al. 2007).

Cross-talk of JA with SA, GA, BR, and ET

Cross-talk among plant hormones is a common phenomenon which attributes to plasticity in plant stress responses and fine tuning of regulation of gene expression. A well-studied example is the cross-talk between JA and salicylic acid (SA) preferentially shown for Arabidopsis. Here, this cross-talk links the signal transduction pathways of wounding or attack by necrotrophic pathogens with that of biotrophic pathogens (Pieterse et al. 2012). A similar cross-talk, however, occurs in tomato. In a collaborative work, Chuanyou Li's lab attributed to the elucidation of the cross-talk among SA, JA and ET in tomato plants infected by *Alternaria alternaria* f. sp. *lycopersici* (Jia et al. 2013; Zhang et al. 2011). JA and ET promote cell death induced by an Alternaria toxin in a way that JA signaling is upstream of ET formation. Another example is the preconditioning by whiteflies and its effect on leaf nutrients and SA-mediated defense responses. Data with different JA biosynthesis mutants of tomato showed after preconditioning an increased SA-based defense (Cui et al. 2012).

Daoxin Xie's lab has published several works on the cross-talk between gibberellic acid (GA) and JA and on the cross-talk between brassinosteroid (BR) and JA. The GA–JA cross-talk was preferentially analyzed in stamen development. These aspects have been recently reviewed by Xie's group together with data on the JA–auxin cross-talk during stamen development (Song et al. 2013b). The requirement of JA, GA and auxin for stamen development was known since the identification and characterization of mutants being affected in biosynthesis and signaling of these hormones and showing alterations specifically in stamen development (Song et al. 2013b; Browse 2009a, b). The cross-talk, however, became evident only upon identification of the negative regulators JAZs and their targets such as MYB21, MYB24, and MYB54, which all are specifically active in stamen development (Song et al. 2011, 2013b). Plants, deficient in JA or GA, are male sterile. GA acts through JA leading to up-regulation of *DEFECTIVE IN ANTHER DEHISCENCE* (*DAD1*) and *LOX1*, JA formation and JA-induced expression of *MYB21*, *MYB24*, and *MYB 57* (Cheng et al. 2009). The repressors of GA signaling, the DELLA proteins, are degraded upon GA formation similar to the JAZ proteins upon JA/JA-Ile formation (Cheng et al. 2009; Song et al. 2011). A modified cross-talk between GA and JA exists in the balance of growth and defense. In the presence of GA, growth is supported by GA-mediated degradation of DELLAs. DELLAs, however, compete with JAZ proteins for the binding sites of MYC2. Consequently, growth and defense are permanently sustained by a balance in JA and GA signaling (Kazan and Manners 2013; Wasternack and Hause 2013).

The cross-talk between BR and JA was initially observed in anthocyanin formation. Here, mutants of BR biosynthesis exhibit reduced JA-induced anthocyanin formation, whereas BR treatment leads to anthocyanin formation (Peng et al. 2011). In this respect, Daoxin Xie's lab identified the mutant *psc1*, which exhibits a partial suppression of the male sterile phenotype of *coi1* plants. The mutation of *psc1* was localized in the *DWF4* gene which encodes a key enzyme in BR biosynthesis indicating that BR may affect JA signaling (Ren et al. 2009). Further analysis showed a negative role of BR in JA-induced root growth inhibition and down-regulation of *DWF4* expression by JA downstream of COI1.

Cross-talk between JA and auxin

Auxin distribution by PIN-FORMED (PIN) proteins and stem cell niche activity are important players in root development of *A. thaliana*. Work by Chuanyou Li's lab indicated a cross-talk between auxin and JA in auxin biosynthesis and auxin distribution during root development: (1) analysis of mutants defective in JA-induced lateral root formation led to identification of ANTHRANILATE SYNTHASE α1 (ASA1) which catalyzes the first step in auxin biosynthesis, and *ASA1* is JA/JA-Ile inducible (Sun et al. 2009). (2) JA modulates endocytosis and plasma membrane accumulation of AtPIN2 (Sun et al. 2011). PIN proteins are required for polar auxin transport. PIN2 endocytosis was inhibited at lower JA concentration and PIN2 accumulation in the plasma membrane was reduced. This cross-talk between JA and auxin in auxin biosynthesis and transport takes place as a type of positive interaction in defense against necrotrophic pathogens such as *Alternaria brassicicola* (Qi et al. 2012).

Another cross-talk between auxin and JA was studied on Chuanyou Li's lab, partially in collaboration with K. Palme's group (Freiburg, Germany), on role of the TFs PLETHORA1 (PLT1) and PLT2 in root stem cell niche of *A. thaliana*. The four mitotically inactive cells of the quiescent center (QC) and the surrounding mitotically active cells of the stem cell niche are regulated by JA (Chen et al. 2011b). This regulation takes place by binding of MYC2 to

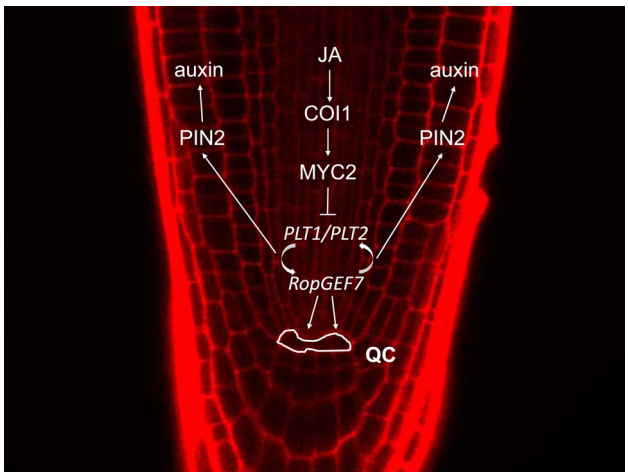

Fig. 3 Maintenance of the root stem cell niche pattering by a regulatory network of JA-mediated repression, auxin distribution, PIN-FORMED2 (*PIN2*) accumulation and action of TFs such as MYC2 and PLETHORA (PLT) 1 and 2 (designed after Chen et al. 2011a, b). JA leads to COI1-dependent *MYC2* expression. MYC2 protein binds to the promoters of *PLT1* and *PLT2* which leads to their repression in a guanine nucleotide exchange factor (*RopGEF7*)-dependent manner. Consequently, the maintenance of the quiescent center (*QC*) and the stem cell niche activity is affected followed by redistribution of PIN2 and auxin

the promoters of *PLT1* and *PLT2* which leads to repression of their expression. PLT1 and PLT2, however, are required for root stem cell niche pattering and expression of *PIN* genes (Fig. 3). These data show how JA signaling is integrated in auxin signaling and represent a mechanistic explanation for root growth inhibition by JA, a process which is known for more than three decades.

Another aspect of the auxin/PLT pathway was found by Tyr-sulfation of some peptides known as meristem growth factors. A tyrosylprotein sulfotransferase is required for maintenance of root stem cell niche via regulation of basal- and auxin-induced expression of *PLT1* and *PLT2* (Zhou et al. 2010). In QC cells, a ROC/ROP GTPase activator (RopGEF7) is expressed and is required for QC cell maintenance (Chen et al. 2011a). RopGEF7 regulates expression of *PLT* genes and its gene is auxin inducible. RopGEF7 is required for expression of the gene encoding the auxin efflux protein PIN1 (Fig. 3); (Chen et al. 2011a). These data complement the complex regulatory network in stem cell niche maintenance including auxin gradients, PIN1 accumulation, and expression of *PLT1*, *PLT2* and *RopGEF7*. Recently, redox-signaling by a plastid-localized glutathione reductase2 (GR2) was identified as another player in root apical meristem maintenance by characterization of the mutant *miao* (Yu et al. 2013). This mutant showed defects in maintenance of root apical meristem which was caused by glutathione oxidation. The intact auxin/PLT pathway, however, requires reduced glutathione (Yu et al. 2013).

Conclusions

In the last decade, fundamental breakthroughs in biosynthesis, perception, signaling and cross-talk of jasmonates were achieved. Among them were functional characterization of the SCF^{COI1}–JAZ co-receptor complex, of repressors such as JAZs, JAV1 and JAMs, of numerous TFs active in JA-Ile signaling and of cross-talk of JA/JA-Ile with other hormones such as auxin, SA, ET, BR, GA and ABA. Many of these were discovered in Daoxin Xie's lab and in Chuanyou Li's lab. In both labs ongoing activities occur in elucidation of regulatory principles and further components in JA-Ile signaling and mode of action. Open questions being addressed in the near future worldwide are (1) assembly and half-life of components of the SCF^{COI1}–JAZ co-receptor complex, (2) translational and post-translational control mechanisms in JA-Ile signaling, (3) new components of JA-Ile signaling identified by chemical and genetic screens, and (4) epigenetic control mechanisms in JA-Ile signaling.

The outcome of such studies will improve our knowledge on action of jasmonates in plant stress responses and development and will include applied aspects (Wasternack 2014).

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Conflict of interest The author declares that there is no conflict of interest.

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