Jasmonate Biosynthesis in *Arabidopsis thaliana* – Enzymes, Products, Regulation

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Abstract: **Among the plant hormones jasmonic acid and related derivatives are known to mediate stress responses and several developmental processes. Biosynthesis, regulation, and metabolism of jasmonic acid in *Arabidopsis thaliana* are reviewed, including properties of mutants of jasmonate biosynthesis. The individual signalling properties of several jasmonates are described.**

Key words: **Jasmonates, jasmonate metabolites, regulation, *Arabidopsis thaliana,* biosynthesis.**

Introduction

Jasmonates (JAs) and their precursor, 12-oxo-phytodienoic acid (OPDA), are signals in plant stress responses and development. Upon elucidation of the biosynthetic pathway in the 1980s, reverse genetic approaches and mutant analyses, mostly with *A. thaliana,* revealed the regulation and individual signalling properties of OPDA, JA, and its metabolites in development and plant defence. External stimuli, such as wounding or pathogen attack, lead to an endogenous rise in OPDA and JA, followed by expression of numerous defence genes. During development, levels of JA and OPDA differ remarkably between various organs and developmental stages. How these levels of both compounds are sustained and transduced into specific responses is an important question to understand plant stress responses and JA-mediated developmental processes. Here, we review aspects of biosynthesis and metabolism of jasmonates in *A. thaliana,* including regulation and mutant properties. For functions of jasmonates and aspects of the jasmonate signalling network, recent reviews should be consulted (Berger, 2002; Farmer et al., 2003; Schaller et al., 2004; Howe, 2004; Pozo et al., 2004; Pauw and Memelink, 2004; Lorenzo and Solano, 2005; Wasternack, 2006).

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Jasmonate Biosynthesis – Enzymes

Jasmonic acid (JA) and its derivatives, collectively referred to as jasmonates, as well as their precursors, the octadecanoids, are lipid-derived signals originating from α-linolenic acid (18 : 3) (α-LeA) of chloroplast membranes. Therefore, JA biosynthesis is initiated by liberation of α-LeA from these membranes. Among the various gene families of *A. thaliana* coding for enzymes with lipid hydrolyzing activities (Dörmann, 2005), only the following may have a role in JA biosynthesis:

1. phospholipase A_1 cleaving the acyl moiety in *sn*-1 position and encoded by one gene;
2. phospholipases A_2 cleaving the acyl moiety in *sn*-2 position, being active in many stress responses and encoded by two genes;
3. patatin-like acyl hydrolases with phospholipase and glycolipase activities, encoded by 10 genes;
4. DAD-like lipases which are involved in phospholipid and galactolipid acyl hydrolysis and encoded by 12 genes;
5. SAG (senescence-associated gene) 101-like acyl hydrolases encoded by a gene family of at least 21 members.

Although many attempts were made to identify JA-related lipases, only for a wound-induced phospholipase A_2 (Narváez-Vásquez et al., 1999) and a DAD1-like lipase were hints found on their role in JA biosynthesis. Clear proof was given by the phenotype of the mutant delayed anther dehiscence 1 *(dad1)*. The *dad1* mutant is affected in gene coding for a plastid-located phospholipase A_1, which led to the gene family name. The *dad1* plants exhibit only 25% JA content compared to that of the wild type in flowers before anthesis (Ishiguro et al., 2001). Due to this JA deficiency and shorter filament length, the role of JA in filament elongation for proper pollination was proposed. The proteins fulfilling DAD1 function in leaves are unknown so far.

The α-LeA is the substrate of a chloroplast-located 13-lipoxygenase (13-LOX), which catalyzes oxygen insertion at carbon 13 of the carbon backbone, leading to (13*S*)-hydroperoxyoctadecatrienoic acid (13-HPOT) (Fig. **1**). Among the six LOX genes of *A. thaliana* (Feussner and Wasternack, 2002), the 13-LOX encoded by *LOX2* seems to be involved in JA biosynthesis. Transgenic approaches revealed that LOX2 is responsible for at least the wound-induced JA biosynthesis (Bell et al., 1995). Recent proteome analysis of chloroplast stromal proteins showed that monomeric LOX2 protein is 1.5% of the total amount of stromal

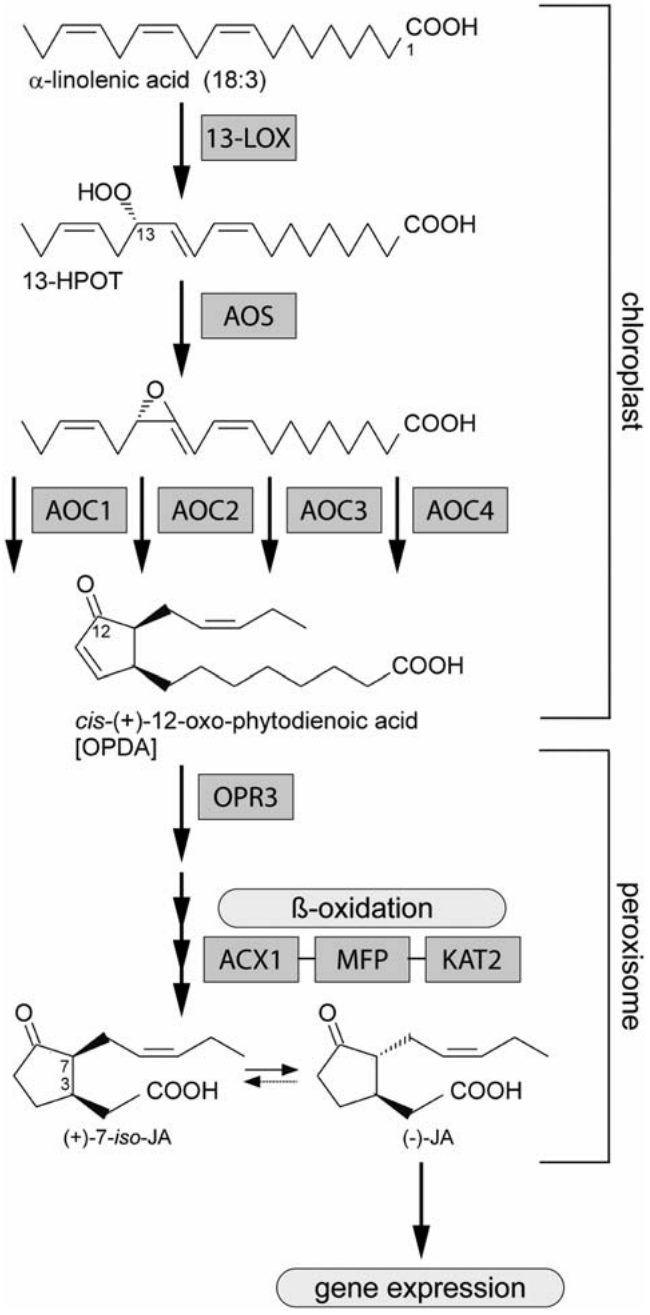

Fig. 1 Scheme of JA biosynthesis in *A. thaliana*.

proteins, thus making it among the group of abundant enzymes of the Calvin cycle (Peltier et al., 2006). Beside the 13-LOX products which finally lead to octadecanoids and jasmonates, 9-LOX products occur and are of increasing interest in the analysis of plant pathogen interactions. In addition to the jasmonate branch of the LOX pathway, six other branches generate oxylipins (Feussner and Wasternack, 2002). These oxylipins occur, like the fatty acids, in free and esterified form. Surprisingly, the level of esterified fatty acids and oxylipins substantially exceeds that of the corresponding free compounds in *A. thaliana* leaves (Stenzel et al., 2003 b; Müller et al., 2002). This is also true for OPDA, which occurs abundantly in esterified form in chloroplast membranes (Stelmach et al., 2001).

The 13-LOX product 13-HPOT is converted by an allene oxide synthase (AOS). The highly unstable AOS product rapidly decays into ketols and racemic OPDA or is enzymatically converted by an allene oxide cyclase (AOC) to *cis*-(+)-OPDA. The AOS contains a chloroplast target sequence and is encoded by a single copy gene in *A. thaliana*. Therefore, AOS is regarded as a step in the regulation of JA biosynthesis (Laudert et al., 1996; Laudert and Weiler, 1998). As expected for a single copy gene, the *aos*-knockout mutant is JA-deficient, lacks wound-induced accumulation of JA and JA-responsive gene expression (Park et al., 2002). The AOS promoter showed developmental regulation, e.g., high activity in anther filaments, early stages of carpel development and in the flower abscission zone (Kubigsteltig et al., 1999). The *AOS* of *A. thaliana* is expressed locally and systemically upon wounding, but treatment with OPDA or JA only locally induced the AOS promoter activity (Kubigsteltig et al., 1999).

The AOC catalyzed step is of special regulatory importance due to the establishment of the ultimate enantiomeric structure of the naturally occurring JA. Since the first cloning of an AOC from tomato (Ziegler et al., 2000), more than 58 *AOC* sequences have been deposited in the database and partially characterized by functional assays. The phylogenetic tree analysis for *AOC*s revealed small, but clearly distinct gene families for monocotyledonous and dicotyledonous plants (Stenzel et al., 2003 b).The number of gene family members per species might be of regulatory impact, as multiple genes indicate a possible junction of differential regulation in JA biosynthesis.

Four genes code for AOCs in *A. thaliana*. All of them carry a chloroplast target sequence which is functional, as revealed by immunocytological analysis (Stenzel et al., 2003 b) (Fig. **2 D**) and import studies (J. E. Froehlich, pers. communication). In fully developed leaves, AOCs occur in chloroplasts of all leaf tissues, together with AOS and LOX (Figs. **2 A – C**). The four recombinant AOCs use 18 : 3 and 16 : 3 generated substrates leading to the cyclopentenones OPDA and dinor-OPDA, respectively (C. Dorer, pers. communication). *AOC1 – AOC4* are expressed locally and systemically in response to wounding, with preferential expression of *AOC2* (Stenzel et al., 2003 b). *AOC* gene expression is also up-regulated upon treatment with jasmonates, octadecanoids, glucose, and sorbitol, but not in response to NaCl and salicylate. Recent analyses of transgenic lines carrying the GUS reporter gene under the control of the individual *AOC* promoters revealed non-redundant promoter activities during distinct stages of development (Stenzel et al., in prep.). In roots, only promoter activities for *AOC3* and *AOC4* can be observed (Fig. **3**). The *AOC4* promoter shows activity in the root tip throughout root development, whereas the promoter of *AOC3* first shows activity in the meristematic and elongation zone 7 days after germination, and the activity shifts into the stele of the elongation zone about 14 days after germination. Interestingly, this corresponds to diminished root growth of *AOC3* knockout plants (C. Delker, unpubl.). During flower development, non-redundant, spatially and temporally different activities were found for all four *AOC* promoters. The sum of these promoter activities is reflected in the AOC protein pattern detected immunocytologically with an anti-AOC2 antibody which recognizes all four AOCs (Hause et al., 2003) (Fig. **4**). Assuming a correlative link between *AOC* promoter activity and JA levels, JA-dependent gene expression may occur in flower organs carrying high *AOC* promoter activ-

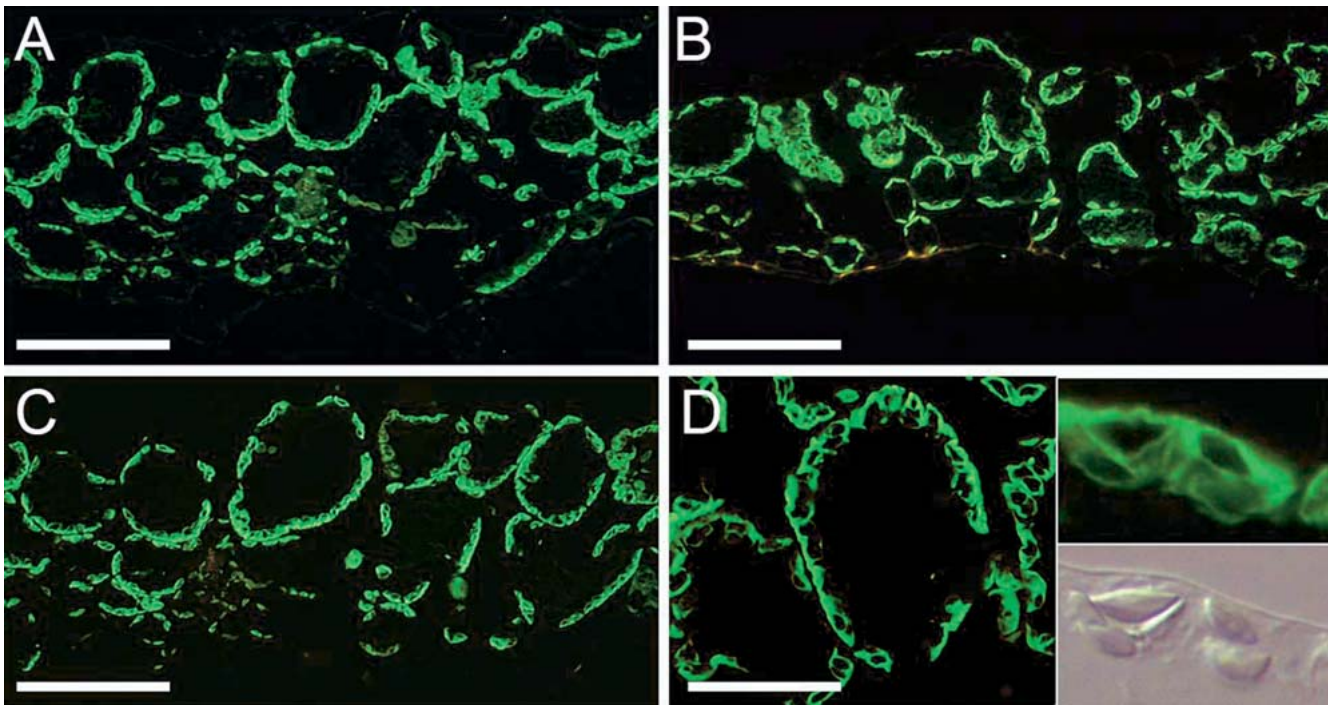

Fig. 2 Occurrence of LOX (**A**), AOS (**B**), and AOC (**C**) in all tissues of a fully developed leaf and location of AOC in chloroplasts (**D**). Cross-sections of WT (COL-0) leaves of the ecotype Columbia were probed with an anti-LOX antibody (dilution 1 : 200), an anti-AOS antibody (dilution 1 : 2000), or an anti-AOC antibody (dilution 1 : 2000). The anti-LOX antibody recognized all six recombinant LOX proteins of *A. thaliana* (Feussner, unpublished). The anti-AOC antibody was raised against recombinant AOC2 and exhibited weak cross-reactivity to AOC1, AOC3, and AOC4 (Stenzel et al., 2003b). The green fluorescence indicates occurrence of LOX, AOS, and AOC protein. Yellow-brown fluorescence appeared only upon treatment with pre-immune serum (data not shown). Intracellular distribution of AOC is shown indicating occurrence in chloroplasts, which contain starch granules (inset in **D**). Bars represent 100 μm for **A – C** and 50 μm for **D**.

ity. The promoter activity of the JA-responsive *THIONIN2.1* gene fits in this scenario. Its site of activity correlates with that of the *AOC* promoters (Vignutelli et al., 1998). In tomato, the link between AOC and JA levels could be measured directly. In the various flower organs, abundant occurrence of AOC protein correlated with elevated levels of JA and OPDA (Hause et al., 2000).

The second half of JA biosynthesis takes place in peroxisomes (Fig. **1**). So far, it is not known whether the AOC product OPDA or its CoA ester is transported from the chloroplast to the peroxisomes. The ABC transporter COMATOSE (CTS), also known as PXA1, may transport OPDA or its CoA ester, since the *cts* mutant is JA-deficient (Theodoulou et al., 2005). However, due to residual formation of JA in the *cts* mutant, the CTS function might be bypassed, possibly by ion trapping of $OPDA^H$ (Theodoulou et al., 2005). The conversion of OPDA, a cyclopentenone, to cyclopentanones is catalyzed by the OPDA reductase (OPR) encoded by a small gene family in *A. thaliana*. However, only the OPR3 carries a peroxisomal target sequence and acts specifically with *cis*-(+)-OPDA (Schaller et al., 2000; Strassner et al., 2002). The mutants *opr3* and *dde1* (delayed dehiscence1), both affected in the *OPR3* gene (Sanders et al., 2000; Stintzi and Browse, 2000), are JA-deficient and thus support this specificity. OPR1 and OPR2 cannot substitute the OPR3 function. Consequently, mutants impaired in OPR3 function are JA-deficient (Stintzi and Browse, 2000; Stintzi et al., 2001; Stenzel et al., 2003b).

In the final steps of JA biosynthesis, the carboxylic acid side chain is shortened in three rounds of β-oxidation. Feeding experiments in tomato with compounds carrying different lengths of the carboxylic acid side chains revealed that only even numbered OPC derivatives were converted to JA, suggesting β-oxidative steps (Miersch and Wasternack, 2000). In accordance, derivatives with an *O* or *S* altered carboxylic side chain were not converted to JA (Blechert et al., 1995). Several lines of genetic evidence have revealed recently that fatty acid β-oxidation enzymes are active in JA biosynthesis. These steps are catalyzed by an acyl-CoA synthase, an acyl-CoA oxidase (ACX), a multifunctional protein MFP, and a L-3-ketoacyl-CoA thiolase (KAT) (Fig. **1**). In *Arabidopsis, ACX1* and *KAT2* expression is up-regulated upon wounding and antisense expression of *ACX1* and *KAT2* led to JA deficiency upon wounding, indicating involvement of β-oxidation in JA biosynthesis (Castillo et al., 2004). Several mutants affected in *ACX* genes exhibit reduced fatty acid acyl-CoA oxidase activity and enhanced resistance to the endogenous auxin analogue indole-3-butyric acid (IBA) due to the role of fatty acid acyl-CoA oxidase in IBA formation (Adham et al., 2005). Consequently, mutants affected in the *ACX* gene may have JA deficiency upon wounding due to the involvement of ACX and a β-oxidative step in JA biosynthesis. A comparable effect is seen in the *aim1* mutant affected in one of the two MFP genes of *A. thaliana* (Richmond and Bleecker, 1999). Indeed, *aim1* mutant plants lack wound-induced accumulation of JA and show reduced expression of JA-responsive genes encoding vegetative storage proteins *(VSPs)* (C. Delker, unpubl.).

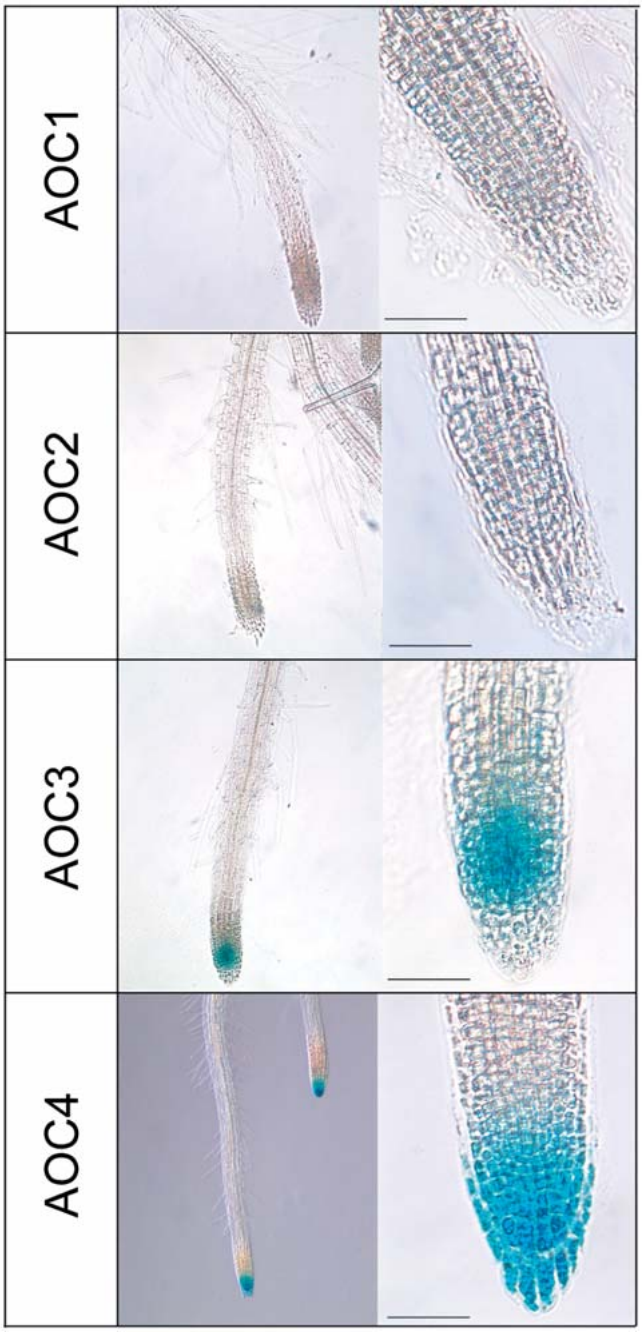

Fig. 3 Promoter activities of *AOC* gene family members in roots of 7-day-old light-grown seedlings transformed with a construct carrying the GUS reporter gene *µidA* under the control of the full-length promoter of *AOC1, AOC2, AOC3,* or *AOC4*.

Another class of enzymes may also function in JA biosynthesis. One of the 4-coumarate: CoA ligase-like enzymes of *A. thaliana* can activate *in vitro* OPDA to the CoA ester and is located in peroxisomes (Schneider et al., 2005). Thus, two different scenarios may function in the final steps of JA biosynthesis:

1. OPDA transported into peroxisomes by ion trapping (Theodoulou et al., 2005), may be directly reduced by OPR3 or activated by a 4-Cl-like enzyme (Schneider et al., 2005) and subsequently reduced and β-oxidized by OPR3, ACX1, MFP, and KAT.
2. The ABC transporter CTS/PXA1 located in the peroxisomal membrane may catalyze import of OPDA or its CoA ester, which are further converted by OPR3, ACX1, MFP, and KAT2 (Fig. **1**). In the case of CoA ester transport, the 4-CL-like enzyme activity would be redundant to an acyl CoA-ligase.

Jasmonate Biosynthesis – Regulation

Several lines of evidence indicate that three different mechanisms are involved in the regulation of JA biosynthesis of *A. thaliana*. (i) Substrate availability: Transgenic plants over-expressing *AOS* constitutively did not show elevated levels of JA, but generate more JA than the wild type upon wounding (Laudert et al., 2000; Park et al., 2002). Furthermore, in the fully developed *Arabidopsis* leaf LOX, AOS, and AOC proteins occur abundantly, but JA and OPDA are formed only upon external stimuli such as wounding (Laudert et al., 1998; Stenzel et al., 2003 b; Stintzi et al., 2001). This transient rise in JA takes place before transcript accumulation of JA biosynthetic genes. (ii) Positive feedback: Treatment of *A. thaliana* leaves with JA activates expression of all JA biosynthetic genes (Reymond et al., 2000; Stenzel et al., 2003 b; Castillo et al., 2004). Furthermore, mutants having elevated levels of JA exhibit increased AOC protein levels, whereas JA-deficient mutants such as *opr3* contain less AOC protein than the wild type (Stenzel et al., 2003 b, and unpublished data). (iii) Tissue specificity: The distinct AOS promoter activities (Kubigsteltig et al., 1999) and the non-redundant promoter activities of *AOC1 – AOC4* (Stenzel et al., in prep.) strongly suggest regulation of JA biosynthesis by tissue specificity. Possibly, the combined activity of the single copy gene *AOS* with the differential activity of the four *AOC* genes allows a spatially and temporally distinct generation of JA during development in *Arabidopsis*.

Metabolism of Jasmonic Acid

JA is not the only cyclopentanone compound occurring in plants. Although its accumulation is taken as a first indicator for its role in any JA-dependent process analyzed, at least six metabolic conversions of JA can take place (Fig. **5**). This raises the question whether JA or its metabolites exhibit separate biological activity.

1. Methylation by a JA-specific methyl transferase (Seo et al., 2001): This reaction seems to sustain, together with unspecific esterases (Stuhlfelder et al., 2004), the ratio of JA and its methyl ester. Usually, plant tissues including those from *A. thaliana,* contain much more JA than its methyl ester (Kramell et al., 2000; Miersch, unpublished data). Constitutive over-expression of the JA-specific methyl transferase led to a higher amount of the methyl ester, an unchanged JA level, and increased pathogen resistance, indicating that the methyl ester can be an active form of JA under specific conditions (Seo et al., 2001).
2. Decarboxylation to *cis*-jasmone (Koch et al., 1997): The accumulation of *cis*-jasmone increased resistance to insects (Birkett et al., 2000). *Cis*-jasmone may induce emission of volatiles which affect aphid repulsion and attraction of aphid antagonists.
3. Reduction of the keto group of the cyclopentanone ring: Although not studied in detail, this reaction is indicated by the occurrence of cucurbic acid and its derivatives in various plant species (Sembdner and Parthier, 1993).

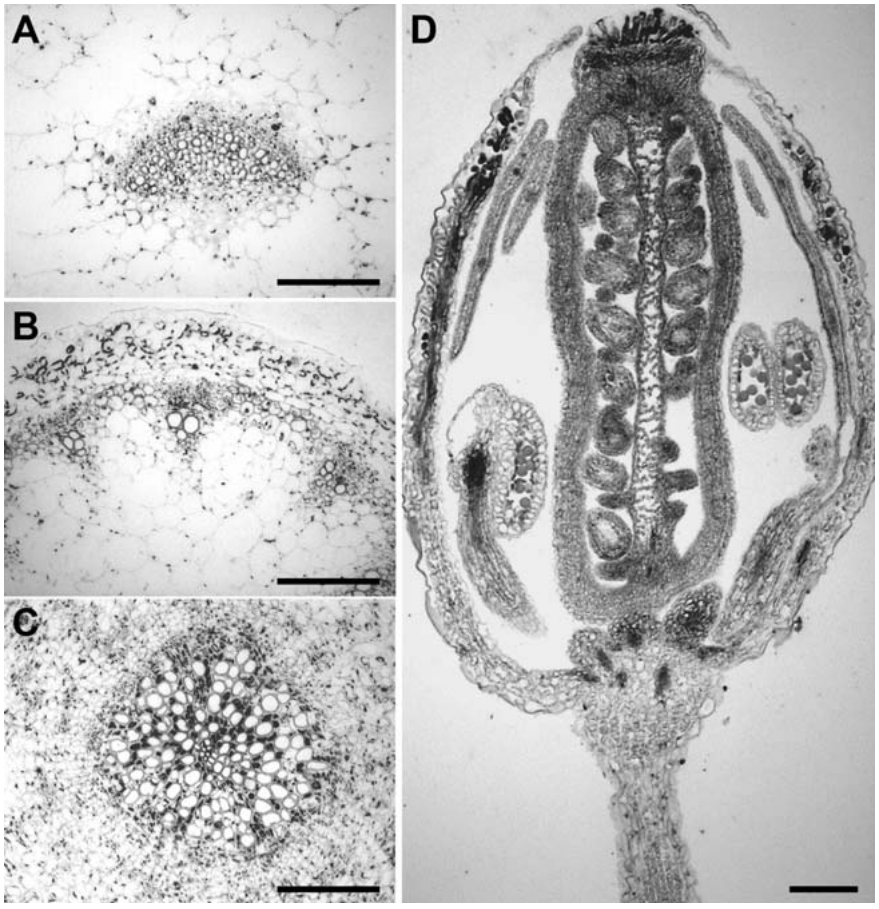

Fig. 4 Immunocytochemical detection of AOC protein in untreated WT (COL-0) tissues. Cross-sections of the leaf stalk of a rosette leaf (**A**), the stem (**B**), and the central cylinder of a root (**C**), as well as a longitudinal section of a flower bud (**D**) were probed with an anti-AOC antibody (dilution 1 : 2000). For properties of the anti-AOC antibody see legend of Fig. **2**. AOC protein is indicated by dark-stained tissues, which occurred in vascular tissues, filaments, the stigma, and ovules. Bars: 100 μm in all figures.

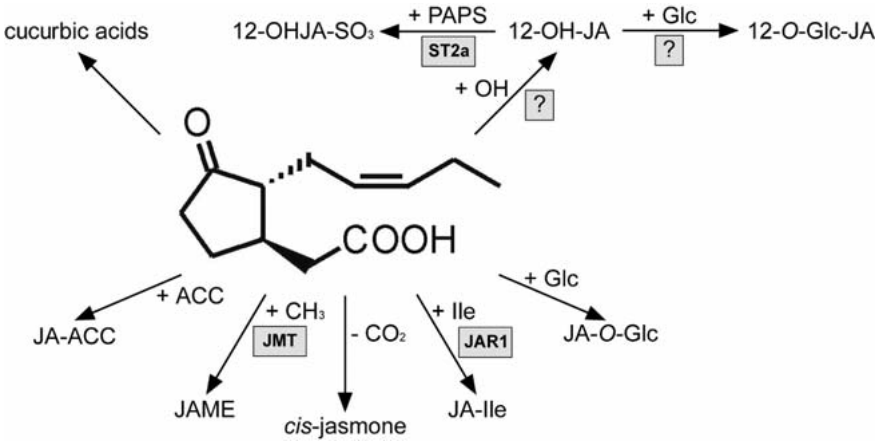

Fig. 5 Metabolism of jasmonic acid. The carboxylic acid side chain can be glucosylated, methylated, conjugated with amino acids, decarboxylated, or conjugated with the ethylene precursor 1-aminocylopropane-1-carboxylic acid (ACC). The pentenone ring can be reduced to cucurbic acids. The pentenyl side chain can be hydroxylated and subsequently glucosylated or sulfated. The enzymes JA methyl transferase (JMT), JA conjugate synthetase (JAR1), and 12-OH-JA-sulfotransferase (ST2a) have been cloned from *Arabidopsis*.

4. Adenylation at the carboxylic acid side chain by an AMP transferase (Staswick et al., 2002) and subsequent conjugation to an amino acid by JAR1(Staswick and Tiryaki, 2004): *JAR1* is a member of a large gene family coding for enzymes which activate any substrate carrying a carboxylic acid group by adenylation. Among them are also 4-Cl-like ligases (Schneider et al., 2005) and the auxin conjugate synthase (Staswick et al., 2005). *JAR1* was identified *via* the JA-insensitive *jar1* mutant (Staswick et al., 1992). This indicates that conjugation of JA is essential, at least partially, for JA responses. JA adenylated by JAR1 is conjugated by this enzyme preferentially to isoleucine (Staswick and Tiryaki, 2004), which corresponds to the preferential occurrence of JA-Ile in different plant species (Kramell et al., 1997). JA-Ile hydrolyzing enzyme activity has been found (Hertel et al., 1997). Although this enzyme has not been cloned so far, hydrolysis and conjugation may sustain JA/JA-Ile homeostasis. This scenario has been shown for auxins, where auxin conjugate synthase (Staswick et al., 2005) counteracts various auxin conjugate hydrolases (Rampey et al., 2004).

5. Hydroxylation at C11 or C12 of the pentenyl side chain and subsequent *O*-glucosylation (Swiatek et al., 2004) or sulfation (Gidda et al., 2003): Hydroxylated JA and its *O*-glucoside were initially found only in solanaceous species and were called tuberonic acid due to the tuber inducing properties (Yoshihara et al., 1989; Helder et al., 1993). Interestingly, *A. thaliana* and many other species contain 12-OH-JA and its sulfated derivative (Gidda et al., 2003; Miersch, in prep.). Sulfation takes place by one of the 18 sulfotransferases occurring in *A. thaliana* and may represent an inactivation mechanism (Gidda et al., 2003). 12-OH-JA does not induce all JA-responsive genes (Gidda et al., 2003; Stenzel et al., 2003 a), suggesting at least partial inactivation of JA signalling by formation of 12-OH-JA.
6. Formation of jasmonoyl-1-β-glucose, jasmonoyl-1-β-gentiobiose, and hydroxyjasmonoyl-1-β-glucose (Swiatek et al., 2004): These compounds have been identified in tobacco BY2 suspension cultures, and in contrast to JA, they did not inhibit the G2 phase of the cell cycle (Swiatek et al., 2002; Swiatek et al., 2004). It will be interesting to see whether *A. thaliana* plants contain glucosylated JA compounds.

Initially, the term "oxylipin signature" was proposed for the occurrence of distinct metabolite profiles of oxylipins. Since OPDA and JA were found to have individual signalling properties (Stintzi et al., 2001), other jasmonates such as 12-OH-JA and JA-Ile were also found to have separate signalling properties. It will be interesting to see whether the jasmonate/oxylipin profiles sustained by the various metabolic routes are functionally active in distinct signalling pathways.

Mutants in Biosynthesis and Signalling of Jasmonates

An essential tool in studying JA biosynthesis, its regulation and its signalling is the isolation and characterization of mutants. First screens were based on root growth inhibition, a well-known effect of JA, and the diminished sensitivity to the molecular mimic of JA, coronatine. In this way, the JA-insensitive mutants *jar1, coi1,* and *jin1,* which is allelic to *jin4,* were identified (Staswick et al., 1992; Feys et al., 1994; Berger et al., 1996) (Table **1**). Another group of signalling mutants was isolated by using plants transgenic for a reporter gene under the control of JA-responsive promoters. In this way, the *joe1* and *joe2* mutants were found using the *LOX* promoter (Jensen et al., 2002), the *cev1* mutant, using the promoter of genes coding for *VSPs* (Ellis et al., 2001), the *cet* mutants, using the *THIONIN2.1* promoter (Hilpert et al., 2001), and the *cas1* mutant, with the *AOS* promoter (Kubigsteltig et al., 1999). These mutants exhibit constitutive or increased expression of the JA-responsive gene used for screening. Finally, transposon insertion mutants and EMS mutants affected in JA biosynthesis were isolated using a characteristic phenotype, male sterility, which could be normalized by JA treatment. Male sterility is a common phenotype of mutants affected in a gene encoding an enzyme of JA biosynthesis. Among them were *dad1* (Ishiguro et al., 2001), *fad3-2fad7-2fad8* (McConn and Browse, 1996), *dde2-2* (von Malek et al., 2002), *dde1* (Sanders et al., 2000), and *opr3* (Stintzi and Browse, 2000).

In the case of the triple mutant *(fad3-2fad7-2fad8),* the final step in the formation of α-LeA, the substrate of JA biosynthesis, is affected. Due to the occurrence of only α-LeA in the tapetum of anthers, α-LeA deficiency leads to JA deficiency, which may affect correct pollen development and dehiscence (McConn and Browse, 1996). The triple mutant and the *opr3* mutant exhibit three common phenotypes: (i) insufficient filament elongation which does not allow pollination at anthesis and is reminiscent of the *dad1* phenotype (see above), (ii) unviable pollen grains, and (iii) lack of dehiscence of the anther locules during flower opening (McConn and Browse, 1996; Stintzi and Browse, 2000). The triple mutant can be restored by OPDA and JA, whereas the *opr3* is restored by JA but not by OPDA. This indicates that anther development and pollen maturation is absolutely JA-dependent and cannot be substituted by its precursor OPDA.

Different signalling properties for JA and OPDA were also identified by different responses of wild type and *opr3* mutant plants to various pathogens. The *opr3* mutant has been shown to exhibit survival resistance to necrotrophic pathogens and saprophagous insects (Stintzi et al., 2001). A recent large-scale array analysis with JA-forming wild type and OPDA-forming *opr3* mutant plants revealed distinct sets of genes for both plants expressed in response to wounding or pathogen attack (Stintzi et al., 2001; Taki et al., 2005).

In contrast to *dad1, fad3-2fad7-2fad8, dde1, dde2-2,* and *opr3,* which are male sterile, other mutants affected in JA biosynthesis such as *cts*/*pxa1* are JA-deficient but not male sterile. Obviously, the residual JA formation in these mutants (see above) is sufficient for anther development and pollen maturation. Individual signalling properties for JA and OPDA were identified with the *opr3* mutant, and the *jar1* mutant indicated separate signalling properties for JA and JA-Ile. *JAR1* plants are fertile, although these plants are JA-insensitive (Staswick et al., 1992). Furthermore, several JA-dependent responses, such as resistance to the soil fungus *Pythium irregulare* (Staswick et al., 1998) or limited damage from ozone exposure (Overmyer et al., 2003), are affected in *jar1* plants, indicating at least partial differences in JA and JA-Ile signal transduction (Staswick and Tiryaki, 2004).

Beside the mutants affected in genes encoding enzymes of JA biosynthesis or JA metabolism, there are mutants altered in JA responses. The most prominent member is the JA-insensitive mutant *coi1* (Feys et al., 1994). Others are *cet1-9, cev,* and *cas1,* which exhibit constitutive JA responses. Although identified by screens on JA sensitivity, these mutants are altered in the capacity to form JA. The *cas1* mutant, affected in a so far unknown upstream element, which regulates the AOS promoter activity, exhibits constitutive elevated levels of OPDA and JA (Kubigsteltig and Weiler, 2003). The *cet1-cet9* mutants show constitutive expression of *THIONINS* and elevated levels of JA and OPDA (Hilpert et al., 2001). The *cev1* mutant, defective in *CELLULOSE SYNTHASE3,* one of the 10 genes encoding subunits of the cellulose synthase of *A. thaliana,* also has elevated levels of JA and OPDA and constitutive JA responses, such as expression of VSPs (Ellis and Turner, 2001; Ellis et al., 2002). Altered capacity to form JA in all these mutants seems to be caused, at least partially, by a defect in the positive feedback regulation of JA biosynthesis. In the case of *coi1,* JA insensitivity may lead to lower expression of JA biosynthetic genes, since all of them are JA-responsive (Reymond et al., 2000; Stenzel et al., 2003 b). Indeed, *coi1* plants are JA-deficient and exhibit less AOC protein (C. Delker, unpubl. data). In *cev* and *cet* mutant plants, constitutively elevated levels of JA correlate with abun-

Table 1 Mutants affected in biosynthesis and signalling of jasmonates

Mutant	Altered phenotype	Altered process	Affected gene(s)	Locus	Reference
Mutants affected in JA biosynthesis and metabolism					
dad1	male sterile	α-LeA liberation	phospholipase A_1	*At2g44810*	Ishiguro et al., 2001
fad3-2fad7-2fad8	male sterile	α-LeA formation	ER + plastidic fatty	*At2g29980*	
			acid desaturases	*At3g11170*	
				At5g05580	McConn and Browse, 1996
dde2-2	male sterile	OPDA conversion	*AOS*	*At5g42650*	von Malek et al., 2002
dde1	male sterile	OPDA conversion	*OPR3*	*At2g06050*	Sanders et al., 2000
opr3	male sterile	OPDA conversion	*OPR3*	*At2g06050*	Stintzi and Browse, 2000
cts/pxa	JA deficient	β-oxidation	*COMATOSE/PXA1*	*At4g39850*	Theodoulou et al., 2005
acx1	IBA resistant	β-oxidation	*ACX1*	*At4g16760*	Adham et al., 2005
aim1	JA deficient	β-oxidation	*MFP*	*At4g29010*	Richmond and Bleecker, 1999
jar1	JA insensitive	JA conjugation	*JAR1,* JA amino acid synthetase	*At2g46370*	Staswick et al., 2002; Staswick and Tiryaki, 2004
Mutants overproducing JA and/or carrying constitutive JA response					
cet1-9	constitutive JA response	unknown	unknown		Hilpert et al., 2001
cev1	constitutive JA response	cell wall synthesis	*CeS3*	*At5g05170*	Ellis and Turner, 2001; Ellis et al., 2002
cas1	constitutive JA response	unknown	unknown		Kubigsteltig and Weiler, 2003
joe1	increased accumulation of anthocyans	increased expression of *LOX2*	unknown		Jensen et al., 2002
joe2	reduced inhibition of root growth	increased expression of *LOX2*	unknown		Jensen et al., 2002
Mutants insensitive or with reduced sensitivity to JA					
coi1	male sterile	proteolysis *via* SCF	*COI1,* F-box protein	*At2g46370*	Feys et al., 1994; Xie et al., 1998
jin1	reduced root growth inhibition	BHLH zip transcription factor	*AtMYC2*	*At1g32640*	Lorenzo et al., 2004
mpk4	dwarf phenotype	altered expression of JA- and SA-response genes	*AtMPK4*	*At4g01370*	Petersen et al., 2000
rcd1	reduced sensitivity to JA, ethylene, and ABA	impaired in ozone signalling	*RADICAL-INDUCED CELL DEATH 1*	*At1g32230*	Ahlfors et al., 2004
axr1	reduced root growth inhibition by JA	proteolysis via SCF	*RUB*	*At1g05180*	Xu et al., 2002
jai4/sgt1b	reduced root growth inhibition in the *ein3* background	proteolysis via SCF	*AtSGT1b*	*At4g11260*	Lorenzo et al., 2004

dant occurrence of AOC protein (I. Stenzel, unpubl. data). Altered JA biosynthetic capacity was also found for the JA signalling mutant ***jin1*** (Nickstadt et al., 2004). ***JIN1*** codes for the transcription factor MYC2, which differentially regulates two branches in the JA signalling pathway (Lorenzo et al., 2004).

The increasing number of mutants affected in JA biosynthesis, regulation, and signalling will facilitate analyses of JA-regulated gene expression and the various JA-dependent processes in plant development and in response to biotic and abiotic stress. A central element is already known with *COI1,* an F-box protein (Xie et al., 1998). Its putative role in proteolytic degradation *via* the proteasome of any negative or positive regulatory protein is being intensively studied (Turner et al., 2002; Lorenzo and Solano, 2005). More recently, other JA signalling mutants, such as *jai4* and *axr1,* were also found to be affected in proteasome activity (Table **1**) (cf. Wasternack, 2006). Another strategy is the analysis of transcription factors of JA-responsive gene expression. Candidates are the AP2 domain transcription factors such as ORCA3, identified first in *Catharantus roseus* cell suspension cultures (van der Fits and Memelink, 2001), and AtMYC2, a nuclear-located basic helix-loop-helix-leucine zipper transcription factor which is rapidly up-regulated by JA (Lorenzo et al., 2004). The antagonistic action of AtMYC2 and ERF1 (Ethylene-Response-Factor1) highlights how the plant network of different hormones is arranged to respond to different sets of stresses (Lorenzo and Solano, 2005). Future work will focus on this cross-talk between JA signalling and other signalling pathways and on JA perception.

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References

Adham, A. R., Zolman, B. K., Millius, A., and Bartel, B. (2005) Mutations in *Arabidopsis* acyl-CoA oxidase genes reveal distinct and overlapping roles in beta-oxidation. The Plant Journal 41, 859 – 874.

Ahlfors, R., Lang, S., Overmyer, K., Jaspers, P., Brosché, M., Tauriainen, A., Kollist, H., Tuominen, H., Belles-Boix, E., Piippo, M., Inzé, D., Palva, E. T., and Kangasjärvi, J. (2004) *Arabidopsis* RADICAL-INDUCED CELL DEATH1 belongs to the WWE protein-protein interaction domain protein family and modulates abscisic acid, ethylene, and methyl jasmonate responses. Plant Cell 16, 1925 – 1937.

Bell, E., Creelman, R. A., and Mullet, J. E. (1995) A chloroplast lipoxygenase is required for wound-induced jasmonic acid accumulation in *Arabidopsis*. Proceedings of the National Academy of Sciences of the USA 92, 8675 – 8679.

Berger, S., Bell, E., and Mullet, J. E. (1996) Two methyl jasmonate-insensitive mutants show altered expression of *AtVsp* in response to methyl jasmonate and wounding. Plant Physiology 111, 525 – 531.

Berger, S. (2002) Jasmonate-related mutants of *Arabidopsis* as tools for studying stress signaling. Planta 214, 497 – 504.

Blechert, S., Brodschelm, W., Hölder, S., Kammerer, L., Kutchan, T. M., Mueller, M. J., Xia, Z.-Q., and Zenk, M. H. (1995) The octadecanoic pathway: signal molecules for the regulation of secondary pathways. Proceedings of the National Academy of Sciences of the USA 92, 4099 – 4105.

Birkett, M. A., Campbell, C. A. M., Chamberlain, K., Guerrieri, E., Hick, A. J., Martin, J. L., Matthes, M., Napier, J. A., Pettersson, J., Pickett, J. A., Poppy, G. M., Pow, E. M., Pye, B. J., Smart, L. E., Wadhams, G. H., Wadhams, L. J., and Woodcock, C. M. (2000) New roles for *cis*-jasmone as an insect semiochemical and in plant defense. Proceedings of the National Academy of Sciences of the USA 97, 9329 – 9334.

Castillo, M. C., Martínez, C., Buchala, A., Métraux, J. P., and León, J. (2004) Gene-specific involvement of β-oxidation in wound-activated responses in *Arabidopsis*. Plant Physiology 135, 85 – 94.

Dörmann, P. (2005) Membrane lipids. In Plant Lipids – Biology, Utilization and Manipulation (Murphy, D. J., ed.), Oxford: Blackwell Publishing Ltd., pp. 123 – 161.

Ellis, C. and Turner, J. G. (2001) The *Arabidopsis* mutant *cev1* has constitutively active jasmonate and ethylene signal pathways and enhanced resistance to pathogens. Plant Cell 13, 1025 – 1033.

Ellis, C., Karafyllidis, I., Wasternack, C., and Turner, J. G. (2002) The *Arabidopsis* mutant *cev1* links cell wall signaling to jasmonate and ethylene responses. Plant Cell 14, 1557 – 1566.

Farmer, E. E., Alméras, E., and Krishnamurthy, V. (2003) Jasmonates and related oxylipins in plant responses to pathogenesis and herbivory. Current Opinion in Plant Biology 6, 372 – 378.

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

Feys, J. F., Benedetti, C. E., Penfold, C. N., and Turner, J. G. (1994) *Arabidopsis* mutants selected for resistance to the phytotoxin coronatine are male sterile, insensitive to methyl jasmonate, and resistant to a bacterial pathogen. Plant Cell 6, 751 – 759.

Gidda, K. S., Miersch, O., Schmidt, J., Wasternack, C., and Varin, L. (2003) Biochemical and molecular characterization of a hydroxyjasmonate sulfotransferase from *Arabidopsis thaliana*. Journal of Biology and Chemistry 278, 17895 – 17900.

Hause, B., Stenzel, I., Miersch, O., Maucher, H., Kramell, R., Ziegler, J., and Wasternack, C. (2000) Tissue-specific oxylipin signature of tomato flowers – allene oxide cyclase is highly expressed in distinct flower organs and vascular bundles. The Plant Journal 24, 113 – 126.

Hause, B., Stenzel, I., Miersch, O., and Wasternack, C. (2003) Occurrence of the allene oxide cyclase in different organs and tissues of *Arabidopsis thaliana*. Phytochemistry 64, 971 – 980.

Helder, H., Miersch, O., Vreugdenhil, D., and Sembdner, G. (1993) Occurrence of hydroxylated jasmonic acids in leaflets of *Solanum demissum* plants grown under long- and short-day conditions. Physiologia Plantarum 88, 647 – 653.

Hertel, S., Knöfel, H.-D., Kramell, R., and Miersch, O. (1997) Partial purification and characterization of a jasmonic acid conjugate cleaving amidohydrolase from the fungus *Botryodiplodia theobromae*. FEBS Letters 407, 105 – 110.

Hilpert, B., Bohlmann, H., op den Camp, R., Przybyla, D., Miersch, O., Buchala, A., and Apel, K. (2001) Isolation and characterization of signal transduction mutants of *Arabidopsis thaliana* that constitutively activate the octadecanoid pathway and form necrotic micro lesions. The Plant Journal 26, 435 – 446.

Howe, G. A. (2004) Jasmonates as signals in the wound response. Journal of Plant Growth Regulation 23, 223 – 237.

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

Jensen, A. B., Raventos, D., and Mundy, J. (2002) Fusion genetic analysis of jasmonate-signalling mutants in *Arabidopsis*. The Plant Journal 29, 595 – 606.

Koch, T., Bandemer, K., and Boland, W. (1997) Biosynthesis of *cis*-jasmone: a pathway for the inactivation and the disposal of the plant stress hormone jasmonic acid to the gas phase? Helvetica et Chimica Acta 80, 838 – 850.

Kramell, R., Miersch, O., Hause, B., Ortel, B., Parthier, B., and Wasternack, C. (1997) Amino acid conjugates of jasmonic acid induce jasmonate-responsive gene expression in barley (*Hordeum vulgare* L.). FEBS Letters 414, 197 – 202.

Kramell, R., Miersch, O., Atzorn, R., Parthier, B., and Wasternack, C. (2000) Octadecanoid-derived alteration of gene expression and the "Oxylipin Signature" in stressed barley leaves. Implications for different signaling pathways. Plant Physiology 123, 177 – 187.

Kubigsteltig, I., Laudert, D., and Weiler, E. W. (1999) Structure and regulation of the *Arabidopsis thaliana* allene oxide synthase gene. Planta 208, 463 – 471.

Kubigsteltig, I. and Weiler, E. W. (2003) *Arabidopsis* mutants affected in the transcriptional control of allene oxide synthase, the enzyme catalyzing the entrance step in octadecanoid biosynthesis. Planta 217, 748 – 757.

Laudert, D., Pfannschmidt, U., Lottspeich, F., Holländer-Czytko, H., and Weiler, E. W. (1996) Cloning, molecular and functional characterization of *Arabidopsis thaliana* allene oxide synthase (CYP 74), the first enzyme of the octadecanoid pathway to jasmonates. Plant Molecular Biology 31, 323 – 335.

Laudert, D. and Weiler, E. W. (1998) Allene oxide synthase: a major control point in *Arabidopsis thaliana* octadecanoid signalling. The Plant Journal 15, 675 – 684.

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

Lorenzo, O., Chico, J. M., Sanchez-Serrano, J. J., and Solano, R. (2004) *JASMONATE-INSENSITIVE1* encodes a MYC transcription factor essential to discriminate between different jasmonate-regulated defense responses in *Arabidopsis*. Plant Cell 16, 1938 – 1950.

Lorenzo, O. and Solano, R. (2005) Molecular players regulating the jasmonate signalling network. Current Opinion in Plant Biology 8, 532 – 540.

McConn, M. and Browse, J. (1996) The critical requirement for linolenic acid is pollen development, not photosynthesis, in an *Arabidopsis* mutant. Plant Cell 8, 403 – 416.

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

Müller, A., Duchting, P., and Weiler, E. W. (2002) A multiplex GC-MS/MS technique for sensitive and quantitative single-run analysis of acidic phytohormones and related compounds, and its application to *Arabidopsis thaliana*. Planta 216, 44 – 56.

Narváez-Vásquez, J., Florin-Christensen, J., and Ryan, C. A. (1999) Positional specificity of a phospholipase A2 activity induced by wounding, systemin, and oligosaccharide elicitors in tomato leaves. Plant Cell 11, 2249 – 2260.

Nickstadt, A., Thomma, B. P. H., Feussner, I., Kangasjärvi, J., Zeier, J., Loeffler, C., Scheel, D., and Berger, S. (2004) The jasmonate-insensitive mutant *jin1* shows increased resistance to biotrophic as well as necrotrophic pathogens. Molecular Plant Pathology 5, 425 – 434.

Overmyer, K., Brosché, M., and Kangasjärvi, J. (2003) Reactive oxygen species and hormonal control of cell death. Trends in Plant Sciences 8, 335 – 342.

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

Pauw, B. and Memelink, J. (2004) Jasmonate-responsive gene expression. Journal of Plant Growth Regulation 23, 200 – 210.

Peltier, J.-B., Cai, Y., Sun, Q., Zabrouskov, V., Gicaomelli, L., Rudella, A., Ytterberg, A. J., Rutschow, H., and van Wijk, K. J. (2006) The oligomeric stromal proteome of *A. thaliana* chloroplasts. Molecular Cell Proteomics 5, 114 – 133.

Petersen, M., Brodersen, P., Naested, H., Andreasson, E., Lindhardt, U., Johansen, B., Nielsen, H. B., Lacy, M., Austin, M. J., Parker, J. E., Sharma, S. B., Klessig, D. F., Martienssen, R., Mattsson, O., Jensen, A. B., and Mundy, J. (2000) *Arabidopsis* map kinase 4 negatively regulates systemic acquired resistance. Cell 103, 1111 – 1120.

Pozo, M. J., Van Loon, L. C., and Pieterse, C. M. J. (2004) Jasmonates – signals in plant microbe interactions. Journal of Plant Growth Regulation 23, 211 – 222.

Rampey, R. A., LeClere, S., Kowalczyk, M., Ljung, K., Sandberg, G., and Bartel, B. (2004) A family of auxin-conjugate hydrolases that contributes to free indole-3-acetic acid levels during *Arabidopsis* germination. Plant Physiology 135, 978 – 988.

Reymond, P., Weber, H., Diamond, M., and Farmer, E. E. (2000) Differential gene expression in response to mechanical wounding and insect feeding in *Arabidopsis*. Plant Cell 12, 707 – 719.

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

Sanders, P. M., Lee, P. Y., Biesgen, C., Boone, J. D., Beals, T. P., Weiler, E. W., and Goldberg, R. B. (2000) The *Arabidopsis DELAYED DEHISCENCE1* gene encodes an enzyme in the jasmonic acid synthesis pathway. Plant Cell 12, 1041 – 1061.

Sembdner, G. and Parthier, B. (1993) The biochemistry and the physiological and molecular actions of jasmonates. Annual Review of Plant Physiology and Plant Molecular Biology 44, 569 – 589.

Schaller, F., Biesgen, C., Müssig, C., Altmann, T., and Weiler, E. W. (2000) 12-Oxophytodienoate reductase 3 (OPR3) is the isoenzyme involved in jasmonate biosynthesis. Planta 210, 979 – 984.

Schaller, F., Schaller, A., and Stintzi, A. (2004) Biosynthesis and metabolism of jasmonates. Journal of Plant Growth Regulation 23, 179 – 199.

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

Seo, H. S., Song, J. T., Cheong, J.-J., Lee, Y.-H., Lee, Y.-W., Hwang, I., Lee, J. S., and Choi, Y. D. (2001) Jasmonic acid carboxyl methyl transferase: a key enzyme for jasmonate-regulated plant responses. Proceedings of the National Academy of Sciences of the USA 98, 4788 – 4793.

Strassner, J., Schaller, F., Frick, U. B., Howe, G. A., Weiler, E. W., Amrhein, N., Macheroux, P., and Schaller, A. (2002) Characterization and cDNA-microarray expression analysis of 12-oxophytodienoate reductases reveals differential roles for octadecanoid biosynthesis in the local versus the systemic wound response. The Plant Journal 32, 585 – 601.

Staswick, P. E., Su, W., and Howell, S. H. (1992) Methyl jasmonate inhibition of root growth and induction of a leaf protein are decreased in an *Arabidopsis thaliana* mutant. Proceedings of the National Academy of Sciences of the USA 89, 6837 – 6840.

Staswick P. E., Yuen G. Y., and Lehmann C. C. (1998) Jasmonate signaling mutants of *Arabidopsis* are susceptible to the soil fungus *Pythium irregulare*. The Plant Journal 15, 747 – 754.

Staswick, P. E., Tiryaki, I., and Rowe, M. (2002) Jasmonate response locus *JAR1* and several related *Arabidopsis* genes encode enzymes of the firefly luciferase superfamily that show activity on jasmonic, salicylic, and indole-3-acetic acids in an assay for adenylation. Plant Cell 14, 1405 – 1415.

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

Staswick, P. E., Serban, B., Rowe, M., Tiryaki, I., Maldonado, M. T., Maldonado, M. C., and Suza, W. (2005) Characterization of an *Arabidopsis* enzyme family that conjugates amino acids to indole-3-acetic acid. Plant Cell 17, 616 – 627.

Stelmach, B. A., Müller, A., Hennig, P., Gebhardt, S., Schubert-Zsilavecz, M., and Weiler, E. W. (2001) A novel class of oxylipins, *sn*1-O-(12-Oxophytodienoyl)-sn2 (hexadecatrienoyl)-monogalactosyl diglyceride, from *Arabidopsis thaliana*. Journal of Biological Chemistry 276, 12832 – 12838.

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

Stenzel, I., Hause, B., Miersch, O., Kurz, T., Maucher, H., Weichert, H., Ziegler, J., Feussner, I., and Wasternack, C. (2003 b) Jasmonate biosynthesis and the allene oxide cyclase family of *Arabidopsis thaliana*. Plant Molecular Biology 51, 895 – 911.

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

Stintzi, A., Weber, H., Reymond, P., Browse, J., and Farmer, E. E. (2001) Plant defense in the absence of jasmonic acid: the role of cyclopentenones. Proceedings of the National Academy of Sciences of the USA 98, 12837 – 12842.

Stuhlfelder, C., Müller, M. J., and Warzecha, H. (2004) Cloning and expression of a tomato cDNA encoding a methyl jasmonate cleaving esterase. European Journal of Biochemistry 271, 2976 – 2983.

Swiatek, A., Lenjou, M., Van Bockstaele, D., Inzé, D., and Van Onckelen, H. (2002) Differential effect of jasmonic acid and abscisic acid on cell cycle progression in tobacco BY-2 cells. Plant Physiology 128, 201 – 211.

Swiatek, A., Van Dongen, W., Esmans, E. L., and Van Onckelen, H. (2004) Metabolic fate of jasmonates in tobacco bright yellow-2 cells. Plant Physiology 135, 161 – 172.

Taki, N., Sasaki-Sekimoto, Y., Obayashi, T., Kikuta, A., Kobayashi, K., Ainai, K., Yagi, K., Sakurai, N., Suzuki, H., Masuda, T., Takamiya, K., Shibata, D., Kobayashi, Y., and Ohta, H. (2005) 12-oxo-phytodienoic acid triggers expression of a distinct set of genes and plays a role in wound-induced gene expression in *Arabidopsis*. Plant Physiology 139, 1268 – 1283.

Theodoulou, F. L., Job, K., Slocombe, S. P., Footitt, S., Holdsworth, M., Baker, A., Larson, T. R., and Graham, I. A. (2005) Jasmonic acid levels are reduced in COMATOSE ATP-binding cassette transporter mutants. Implications for transport of jasmonate precursors into peroxisomes. Plant Physiology 137, 835 – 840.

Turner, J. G., Ellis, C., and Devoto, A. (2002) The jasmonate signal pathway. Plant Cell 14 (Suppl.), S153 – S164.

Van der Fits, L. and Memelink, J. (2001) The jasmonate-inducible AP2/ERF-domain transcription factor ORCA3 activates gene expression *via* interaction with a jasmonate-responsive promoter element. The Plant Journal 25, 43 – 53.

Vignutelli, A., Wasternack, C., Apel, K., and Bohlmann, H. (1998) Systemic and local induction of an *Arabidopsis* thionin gene by wounding and pathogens. The Plant Journal 14, 285 – 295.

von Malek, B., van der Graaff, E., Schneitz, K., and Keller, B. (2002) The *Arabidopsis* male-sterile mutant dde1-2 is defective in the ALLENE OXIDE SYNTHASE gene encoding one of the key enzymes of the jasmonic acid biosynthesis pathway. Planta 216, 187 – 192.

Wasternack, C. (2006) Oxylipins – biosynthesis, signal transduction and action. In Plant Hormone Signaling (Hedden, P. and Thomas, S., eds.), Annual Plant Reviews. Oxford: Blackwell Publishing Ltd., in press.

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

Xu, L., Liu, F., Lechner, E., Genschik, P., Crosby, W. L., Ma, H., Peng, W., Huang, D., and Xie, D. (2002) The SCF^{COI1} ubiquitin-ligase complexes are required for jasmonate response in *Arabidopsis*. Plant Cell 14, 1919 – 1935.

Yoshihara, T., Omer, E.-L. A., Koshino, H., Sakamura, S., Kikuta, Y., and Koda, Y. (1989) Structure of a tuber-inducing stimulus from potato leaves (*Solanum tuberosum* L.). Agricultural Biology and Chemistry 53, 2835 – 2837.

Ziegler, J., Stenzel, I., Hause, B., Maucher, H., Miersch, O., Hamberg, M., Grimm, R., Martin, G., and Wasternack, C. (2000) Molecular cloning of allene oxide cyclase: the enzyme establishing the stereochemistry of octadecanoids and jasmonates. Journal of Biology and Chemistry 275, 19132 – 19138.

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