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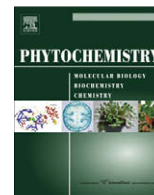

Review

Enzymes in jasmonate biosynthesis – Structure, function, regulation

Andreas Schaller *, Annick Stintzi

Institute of Plant Physiology and Biotechnology, University of Hohenheim, D-70599 Stuttgart, Germany

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Dedicated to the memory of Klaus Dreßler

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ABSTRACT

Jasmonates are a growing class of lipid-derived signaling molecules with diverse functions ranging from the initiation of biotic and abiotic stress responses to the regulation of plant growth and development. Jasmonate biosynthesis originates from polyunsaturated fatty acids in chloroplast membranes. In a first lipoxygenase-catalyzed reaction molecular oxygen is introduced to yield their 13-hydroperoxy derivatives. These fatty acid hydroperoxides are converted by allene oxide synthase and allene oxide cyclase to 12-oxophytodienoic acid (OPDA) and dinor-OPDA, i.e. the first cyclic intermediates of the pathway. In the subsequent step, the characteristic cyclopentanone ring structure of jasmonates is established by OPDA reductase. Until recently, jasmonic acid has been viewed as the end product of the pathway and as the bioactive hormone. It becomes increasingly clear, however, that biological activity extends to and may even differ between the various jasmonic acid metabolites and conjugates as well as its biosynthetic precursors. It has also become clear that oxygenated fatty acids give rise to a vast variety of bioactive compounds including but not limited to jasmonates. Recent insights into the structure, function, and regulation of the enzymes involved in jasmonate biosynthesis help to explain how this variety is generated while specificity is maintained.

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1. Introduction to jasmonate biosynthesis

Polyunsaturated fatty acids (PUFAs) including linoleic acid (18:2), linolenic acid (18:3) and hexadecatrienoic acid (16:3) are abundant in chloroplast membranes and are readily oxidized to yield the corresponding fatty acid hydroperoxides. Under conditions of oxidative stress, fatty acid hydroperoxides are formed by free-radical-catalyzed oxidation of PUFAs and may be further oxidized non-enzymatically to generate phytoprostanes, which are considered to be archetypal mediators of oxidative stress responses (Mueller, 2004). Alternatively, fatty acid hydroperoxides are synthesized enzymatically involving lipoxygenase (LOX) or α-dioxygenase (DOX) activities. While numerous positional isomers are generated as racemic mixtures during chemical lipid peroxidation, the LOX-catalyzed regio- and stereo-specific dioxygenation of PUFAs at C9 or C13 results in the specific formation of 9(*S*) and 13(*S*) hydroperoxy fatty acids, respectively. These hydroperoxides are substrates for at least six different families of enzymes, resulting in the formation of different classes of oxylipins including jasmonates (JAs) (Blee, 2002; Feussner and Wasternack, 2002; Mosblech et al., 2009; Wasternack, 2007).

The committed step of JA biosynthesis (Fig. 1) is catalyzed by allene oxide synthase (AOS), an unusual cytochrome P450 which does not bind molecular oxygen but uses already oxygenated fatty acid hydroperoxide substrates as oxygen donor and as source for reducing equivalents (Howe and Schilmiller, 2002; Werck-Reichhart et al., 2002). The dehydration of 13(*S*)-hydroperoxy-octadecatrienoic acid (13-HPOT) by AOS results in the formation of an unstable allylic epoxide (allene oxide), 12,13(*S*)-epoxy-octadecatrienoic acid

* Corresponding author. Tel.: +49 711 459 21197; fax: +49 711 459 23751.
E-mail address: schaller@uni-hohenheim.de (A. Schaller).

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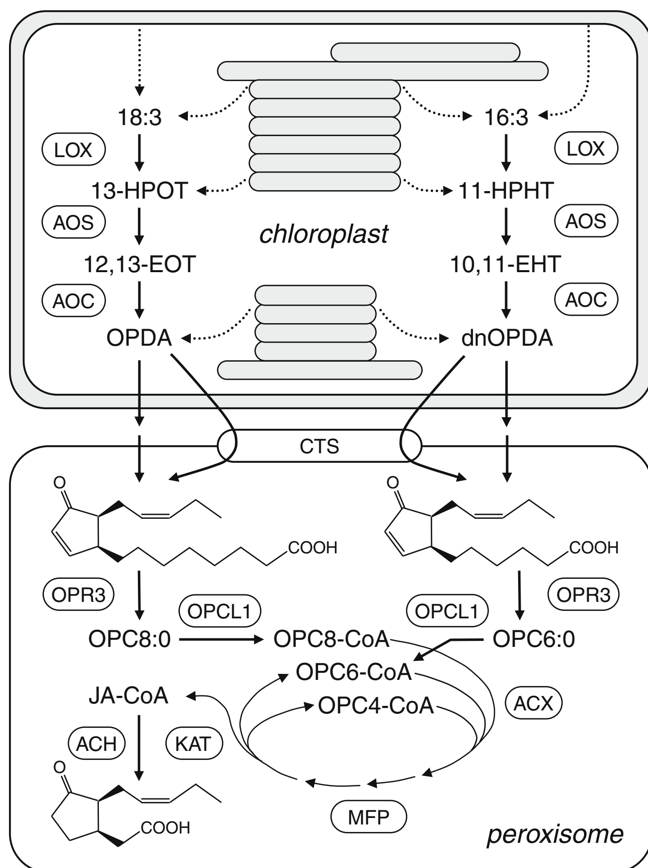

Fig. 1. Jasmonate biosynthesis. Polyunsaturated fatty acids (18:3 and 16:3) are converted to OPDA and dnOPDA by the consecutive action of plastid-localized lipoxygenase (LOX), allene oxide synthase (AOS), and allene oxide cyclase (AOC). Within peroxisomes, jasmonic acid is formed by oxophytodienoate reductase 3 (OPR3) followed by three cycles of β-oxidation (see text for further details and abbreviations). Broken arrows represent the lipase-mediated release of pathway substrates and intermediates from chloroplast membranes, which is still partly hypothetical (modified from Schaller and Stintzi (2008)).

(12,13-EOT). In aqueous media, 12,13-EOT is hydrolyzed spontaneously into α- and γ-ketols, or undergoes cyclization to form 12-oxo-phytodienoic acid (OPDA) (Brash et al., 1988). As opposed to spontaneous cyclization which yields a racemate of OPDA enantiomers, optically pure (9*S*,13*S*)-OPDA is formed as the predominant product in the presence of allene oxide cyclase (AOC) (Hamberg and Fahlstadius, 1990). In a parallel pathway, dinor-OPDA (dnOPDA) is generated by AOS and AOC from 11(*S*)-hydroperoxy-hexadecatrienoic acid (11-HPHT; Fig. 1) (Weber et al., 1997). The short half-life of allene oxides in water (20 s at 0 °C and pH 7.4) (Brash et al., 1988) and the optical purity of endogenous OPDA (Laudert et al., 1997) suggest tight coupling of the AOS and AOC reactions *in vivo*. While coupling is in fact required to establish the absolute configuration of the substituted cyclopentanone ring of JAs, physical contact of AOS and AOC in an enzyme complex does not seem to be required for stereochemical control of the cyclization reaction (Zerbe et al., 2007).

The formation of (9*S*,13*S*)-OPDA and (7*S*, 11*S*)-dnOPDA as the first cyclic intermediates concludes the plastid-localized part of the JA biosynthetic pathway. The remaining steps of JA biosynthesis are located in peroxisomes raising the question as to how OPDA (and/or dnOPDA) is released from chloroplasts and taken up by peroxisomes. While specific transporters for OPDA have not been identified, there is evidence that the peroxisomal ATP-binding cassette (ABC) transporter COMATOSE (CTS (Footitt et al., 2002), also known as PXA1 (Zolman et al., 2001) or PED3 (Hayashi et al., 2002)) mediates import of OPDA, and thus contributes to the biosynthesis of JAs (Theodoulou et al., 2005). CTS catalyzes the ATP-dependent uptake of multiple β-oxidation substrates into peroxisomes. Reduced levels of JAs, impaired wound-induced JA accumulation, and reduced expression of the JA-dependent *VSP1* gene in the *cts* mutant suggest that (dn)OPDA or the corresponding CoA esters are among the CTS substrates (Theodoulou et al., 2005). However, as indicated by residual JA levels in the *cts* mutant, additional pathways for (dn)OPDA import must exist. CTS-independent uptake of (dn)OPDA into peroxisomes may in part be explained by anion trapping as a result of the pH difference between peroxisomes and the cytoplasm (Theodoulou et al., 2005).

A peroxisomal OPDA reductase (OPR) catalyzes the subsequent step in JA biosynthesis, i.e. the reduction of the cyclopentenone ring of (9*S*,13*S*)-OPDA and dnOPDA to 3-oxo-2-(2′(*Z*)-pentenyl)-cyclopentane-1-octanoic (OPC-8:0) and hexanoic (OPC-6:0) acids, respectively (Fig. 1). The JA precursors OPDA and dnOPDA (i.e. cyclopentenones) and JAs (i.e. cyclopentanones) differ in bioactivity (Blechert et al., 1999; Stintzi et al., 2001; Taki et al., 2005). The reduction of the cyclopentenone ring may therefore be particularly important, as it controls the relative levels of these two classes of signaling molecules. The reaction is catalyzed by OPR3, which is the only member of a small family of related enone reductases accepting the (9*S*,13*S*)-enantiomer of OPDA as a substrate (Schaller et al., 2000; Stintzi and Browse, 2000; Strassner et al., 2002).

The shortening of the hexanoic and octanoic acid side chains of OPC-6:0 and OPC-8:0 yields jasmonic acid and involves two or three rounds of β-oxidation, respectively. Prior to entry into the β-oxidation cycle, the carboxylic moiety needs to be activated as CoA ester. In *Arabidopsis thaliana*, there is a large family of 63 ATP-dependent acyl-activating enzymes potentially involved in this process (Shockey et al., 2003). Within this family, a subgroup of fatty acyl-CoA synthetases was shown to activate JA precursors *in vitro*, including OPDA, dnOPDA, OPC-8:0, and OPC-6:0 (Kienow et al., 2008; Koo et al., 2006; Schneider et al., 2005). However, a physiological role in JA biosynthesis was confirmed for only one of them, OPCL1 (OPC-8:CoA ligase 1, locus At1g20510) (Koo et al., 2006). Loss-of-function mutants for OPCL1 hyper-accumulate OPC-8:0, OPC-6:0, and OPC-4:0 suggesting a partial metabolic block in OPC-CoA ester formation (Kienow et al., 2008). The mutants are also compromised in wound-induced JA accumulation which is consistent with a role of OPCL1 in JA biosynthesis. However, about 50% of wild-type levels remain in the mutants indicating that OPCL1 is responsible for only part of the wound-induced JA production, and that additional acyl-CoA synthetases may be involved (Kienow et al., 2008; Koo et al., 2006). The available data are consistent with the view that OPCs are activated as CoA esters within peroxisomes and subsequently channeled into β-oxidation. The formation of OPDA-CoA and dnOPDA-CoA either in the cytosol or within peroxisomes prior to the reduction by OPR3 remains an alternative possibility.

Beta-oxidation itself involves three core enzymes, acyl-CoA oxidase (ACX), multifunctional protein (MFP; comprising enoyl-CoA hydratase and β-hydroxy-acyl-CoA dehydrogenase activities), and 3-ketoacyl-CoA thiolase (Fig. 1). Despite early findings implicating β-oxidation in JA biosynthesis (Vick and Zimmerman, 1984), direct evidence for the contribution of these enzymes is very recent. ACX1A was shown to catalyze the first step in the β-oxidation of OPC-8:0-CoA, and was found to be responsible for the bulk of wound-induced JA production in tomato (Li et al., 2005). Consistent with its essential role in JA biosynthesis, the *acx1* tomato mutant is impaired in wound-induced defense gene activation and insect resistance (Li et al., 2005). In *Arabidopsis*, ACX1 is responsible for about 80% of JA production after wounding (Cruz Castillo et al., 2004; Schilmiller et al., 2007), and only the *acx1/5* double mutant showed severe JA deficiency symptoms (Schilmiller et al., 2007).

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