Biochimie 95 (2013) 79–85

Contents lists available at SciVerse ScienceDirect

Biochimie

journal homepage: www.elsevier.com/locate/biochi

Review

Jasmonates in flower and seed development

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ARTICLE INFO

Article history:
Received 27 March 2012
Accepted 4 June 2012
Available online 13 June 2012

Keywords:
Jasmonates
Flowers
Seeds
Jasmonate-induced genes
Defense
Development

ABSTRACT

Jasmonates are ubiquitously occurring lipid-derived signaling compounds active in plant development and plant responses to biotic and abiotic stresses. Upon environmental stimuli jasmonates are formed and accumulate transiently. During flower and seed development, jasmonic acid (JA) and a remarkable number of different metabolites accumulate organ- and tissue specifically. The accumulation is accompanied with expression of jasmonate-inducible genes. Among these genes there are defense genes and developmentally regulated genes. The profile of jasmonate compounds in flowers and seeds covers active signaling molecules such as JA, its precursor 12-oxophytodienoic acid (OPDA) and amino acid conjugates such as JA-Ile, but also inactive signaling molecules occur such as 12-hydroxy-JA and its sulfated derivative. These latter compounds can occur at several orders of magnitude higher level than JA. Metabolic conversion of JA and JA-Ile to hydroxylated compounds seems to inactivate JA signaling, but also specific functions of jasmonates in flower and seed development were detected. In tomato OPDA is involved in embryo development. Occurrence of jasmonates, expression of JA-inducible genes and JA-dependent processes in flower and seed development will be discussed.

1. Introduction

Jasmonic acid (JA) and its metabolites collectively called jasmonates are lipid-derived signaling molecules. They are active in plant development and plant stress responses together with other plant hormones. Among developmental processes jasmonates modulate root growth, flower development, senescence and tendril coiling [1]. Jasmonates increase upon biotic stress such as herbivory or pathogen attack and upon abiotic stress such as wounding, ozone or UV light [2–4]. The rise in jasmonates leads to dramatic reprogramming of expression of genes involved in flower development [5], defense against herbivores [6,7] or formation of secondary metabolites [8–10] and other metabolic pathways [11].

The biosynthesis of jasmonates is initiated by the release of α-linolenic acid (α-LeA) (18:3) from plastid membranes by a galactolipase. Upon oxygenation by a 13-lipoxygenase (13-LOX), the 13(*S*)-hydroperoxyoctadecatrienoic acid (13(*S*)-HPOT) is converted to an epoxide by a 13-allene oxide synthase (AOS) and cyclized to the cyclopentenone (*cis*)-12-oxophytodienoic acid (OPDA) by an allene oxide cyclase (AOC) [4,12]. In this step the enantiomeric structure of the naturally occurring (+)-7-*iso*-JA ((3*R*,7*S*)-JA) is established. Galactolipase, 13-LOX, AOS, and AOC are located in plastids. The subsequent reduction of the cyclopentenone ring by an OPDA reductase (OPR3) and three cycles of β-oxidation of the carboxylic acid side chain by fatty acid β-oxidation enzymes take place in peroxisomes. The final product (+)-7-*iso*-JA may equilibrate to the more stable (−)-JA ((3*R*,7*R*)-JA). Mechanistic insights into catalysis of JA biosynthesis enzymes were found after crystallization of 13-LOX, 13-AOS, AOC, OPR3 and ACX1 [12].

Mutants in JA biosynthesis and signaling have been contributed notably in elucidating jasmonate-dependent processes [13,14]. Most prominent examples for mutants of JA biosynthesis are the triple mutant *fad3–2fad7–2fad8* which is affected in formation of α-LeA and *opr3* affected in the reduction of OPDA by OPR3. The *opr3* mutant is JA-deficient but can form OPDA, whereas the mutant *fad3–2fad7–2fad8* lacks JA and OPDA. A constitutive JA response occurs in *cev1*, in which a gene encoding CES3, a member of the cellulose synthase complex, is mutated. Among the numerous mutants with reduced or even lack of sensitivity to JA, *coi1* is the most prominent member. CORONATINE (a molecular mimic of jasmonates) INSENSITIVE protein COI1 is an F-box protein [15], a central component of jasmonate perception (cf. below). Characteristic phenotypes of JA deficiency or insensitivity are reduced root growth inhibition,

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http://dx.doi.org/10.1016/j.biochi.2012.06.005

male sterility (Arabidopsis), female sterility (tomato), enhanced sensitivity to necrotrophic pathogens or diminished synthesis of secondary compounds such as anthocyanins.

In long-term processes, such as development or long-lasting and repeatedly performed wounding, JA biosynthesis is regulated by a positive feedback loop [4,16,17]. All genes encoding JA biosynthesis enzymes are JA-inducible, and JA-deficient mutants show decreased expression of those genes such as AOC [4,17]. Numerous studies showed that JA formation takes place very rapidly within some minutes after an external stimulus such as wounding [18,19]. All biosynthetic enzymes analyzed so far (LOX, AOS and AOC) occur constitutively and abundantly in leaves. Following a stimulus, JA is formed then upon release of α-LeA, the substrate of JA biosynthesis. Consistently, transgenic lines over-expressing *AOS* or *AOC* did not show increased JA levels without external stimuli [20,21]. These facts clearly indicate that substrate availability is a regulatory factor of JA biosynthesis [4]. A putative enzyme activity regulation, however, is poorly understood. Another factor of regulation of JA biosynthesis is given by cell- and tissue-specific occurrence of JA biosynthetic enzymes [22], thereby attributing to localized generation of JA, e.g. during wounding [3,23].

In the last couple of years several breakthroughs improved our knowledge on regulation of JA signaling and therefore also JA biosynthesis:

(i) jasmonate-ZIM-domain (JAZ) proteins were discovered and identified as repressors of JA-induced gene expression [24,25];
(ii) (+)-7-*iso*-JA-Ile was identified as the endogenous bioactive jasmonate [26];
(iii) COI1 of Arabidopsis was identified as constituent of the jasmonate co-receptor complex [27];
(iv) The COI1–JAZ co-receptor complex was crystallized and shown to be potentiated in jasmonate perception by inositol-5-phosphate [28];
(v) NINJA was shown to connect the co-repressor TOPLESS to JAZ proteins [29];
(vi) Similar signaling modules are components in regulation of gene expression of jasmonate-, gibberellic acid-, ABA- and auxin-induced processes. They include transcription factors, transcriptional repressors, and a co-receptor complex connecting the repressor and the SCF-complex upon hormone binding [30–32].

JA-Ile is the most bioactive jasmonate compound [26]. Numerous other JA metabolites have been identified being formed by decarboxylation, glucosylation, or hydroxylation of the pentenyl side chain or by sulfation of the hydroxylated derivatives [4,12]. Some of these compounds such as 12-OH-JA were identified to be inactive suggesting a switch off in JA signaling by metabolic conversion [33]. There are, however, distinct developmental processes such as tuber formation or nyctinastic leaf movement, where these metabolites have biological activity [34,35].

Here, we will discuss occurrence and putative functions of jasmonates in flower and seed development. After a descriptive overview on various jasmonate compounds detected in flowers and seeds, jasmonate-responsive gene expression in flower organs will be discussed in terms of putative functions. A new example on OPDA specific effects will be given by describing its role in embryo development of tomato.

2. Occurrence of jasmonate and its derivatives in flowers and seeds

Although there is no systematic study, the available data indicate that the content and number of JA compounds can differ extremely in various plants and during flower and seed development. In monocotyledonous plants such as *Hordeum vulgare* OPDA, JA, 12-OH-JA, the sulfated derivative 12-HSO_4-JA and the glucoside 12-*O*-Glc-JA occur with a basal level of about 150 to 2.100 pmol g^{-1} FW in green and white caryopses, but in *Zea mays* the corresponding levels of these compounds range from 1.700 to 94.500 pmol g^{-1} FW in tassels, silks and pollen [33]. Similarly, high levels were found in the pericarp of *Glycine max* and *Vicia faba*, whereas the pericarp of *Cucumis sativa* contains only residual amounts of these compounds. The dominant occurrence of OPDA compared to JA may indicate its role during flower and seed development as recently shown for tomato flowers and seeds [36], (cf. *chapter 5*). The extremely high levels of 12-OH-JA, 12-HSO_4-JA and 12-*O*-Glc-JA in some flowers organs may indicate a role as an inactive or storage form of JA [33].

In the early days of JA research numerous JA compounds were identified in flower and seed tissues without any hint on putative functions (Table 1). These data go back to first identification of JA and JA-Me as odorant constituents of flowers of *Jasminum grandiflorum* [37]. First physiological effects of jasmonates were shown to be senescence promotion in several plant species [38] and pericarp growth inhibition in *V. faba* [39]. Using the present knowledge on the various facets in JA signaling generated by metabolic conversion, the data on occurrence of the various JA compounds may help to ask for distinct roles in different organs and tissues.

3. JA biosynthesis in flower tissues

Fundamental contributions were done on involvement of JA in flower development by phenotype analyses of mutants. In Arabidopsis several mutants of JA biosynthesis are male sterile due to delayed anther development and/or reduced filament elongation (cf. reviews in [13,14]). Among them are all JA-deficient mutants, such as *dad1* (defective in a phospholipase A1), *fad3–2fad7–2fad8*

Table 1
Early detection of jasmonates in flowers of various mono- and dicotyledonous plant species.

Species	Organ/tissue	Compound	Reference
Petunia hybrida	Pollen	N-((−)-jasmonoyl) -tyramin	[76]
Pinus mugo	Pollen	N-((−)-jasmonoyl) -(*S*)-*iso*-leucine N-(7-*iso*-cucurbinoyl) -(*S*)-*iso*-leucine	[77]
Juglans regia	Female flowers	(−)-JA 6-epi-7-*iso*-cucurbic acid 6-epi-cucurbic acid	[78]
Cymbidium faberi	Flowers	(−)-JA-Me (+)-7-*iso*-JA-Me	[79]
Jasminum grandiflorum	Flowers	*cis*-jasmone (+/−)-JA	[37,80]
Cymbidium kanran	Flowers	JA-Me (+)-7-*iso*-JA-Me	[81]
Vicia faba	Flowers	(−)-JA (−)-JA-Me (+)-7-*iso*-JA	[82]
Cattleya luteola	Flowers	*cis*-jasmone	[83]
Equisetum sylvaticum	Fertile fronds	6-epi-7-*iso*-cucurbic acid	[84]
V. faba	Flowers	N-((−)-jasmonoyl) -*S*-tryptophan N-((+)-cucurbinoyl) -*S*-tryptophan	[85]
Aglaia odorata	Flowers	JA-Me	[86]
Camelliia sinensis	Anthers, pollen	(+/−)-JA (+/−)-JA-Me	[87]
Phaseolus vulgaris and other *Fabaceae*	Immature pericarp	(+/−)-JA-Me	[88]