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Review Article

Jasmonic Acid Pathway in Plants: In Response to Wounding and Insect Attack

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Abstract

In response to external wounding or insect attack jasmonic acid is produced by JA-pathway. Actually the herbivore attack or the wounding induces the lipids of the membrane to release linolenic acid which is then converted into JA. The JA causes the transcriptional changes for the production of proteinase inhibitor (PIs) and formation of enzymes responsible for secondary metabolites or volatile compounds such as nicotine, phenolics and many defence associated compounds. This review tries to cover the biochemical pathway induced for the production of defense responses in plants against the attack of chewing insects. Jasmonate through jasmonate pathway results in gene expression for the production of defense proteins. Insects feed both above and below the ground level. As majority of insects feed above ground level therefore, this review focuses on the chewing insect above the ground level.

Keywords: *JA-Pathway; Octadecanoid Pathway and Hexadecanoid Pathway*

Introduction

External stimulus of insect attack and wounding results in signal transduction pathway sited at parenchyma and companion cells of phloem. By the induction of insect attack prosystemin catalyzed systemin bind with the SR160 receptor of companion cell to initiate the intracellular signaling cascade [1]. The cascade consisted of the MAP kinase and number of unknown intermediates which together with the enzyme phospholipase results in the release of polyunsaturated fatty acid (PUFA's) from the plasma membrane. Within the chloroplast and peroxisome the synthesis of JA took place which induced target gene expression within the companion cell and can also be transported to other target cell via sieve element as represented in figure 1. Thus JA or other JA elements activates gene expression in other targeted undamaged leaves. By analyzing JA- pathway in varieties of plants and its respective induction of target genes, can revolutionize the production of resistant varieties against pathogenic insects.

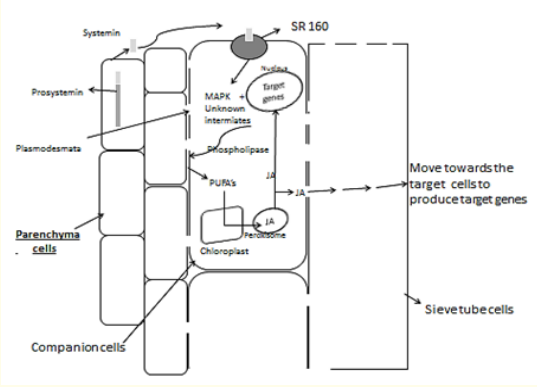

Figure 1: *Model of Systematic signaling showing the Signal generation, Signal transport and Signal recognition.*

Production of jasmonates for JA-pathway induction

In chloroplast there are two pathways, Octadecanoid pathway and Hexadecanoid pathway that yield Jasmonate. In case of Octadecanoid pathway the chloroplast membrane contained linoleic acid (18:2) is transformed into linoleic acid (18:3) by ω-3 fatty acid, desaturases (FADs) [2] and/or phospholipase A2 (PLA2) [3]. In Hexadecanoid pathway Hexadecatrionic acids (16:3) are released from the membrane. The lipoxygenase (13-LOX) [4] enzyme through oxygenation converts linoleic acid (18:3) and Hexadecatrionic acid (16:3) into 13-(S)-hydroxyperoxy-octadecadi(tri)enoic acid (13-HPOT) and 11-(S)-hydroperoxy-hexadeca(tri)enoic acid (11-HPHT) which at least through six pathways was converted in JA [5,6]. The (13-HPOT) acted as a precursor for the octadecanoid pathway and (11-HPHT) acted as a precursor for Hexadecanoid pathway, the two pathways then run parallel to each other [7]. In first step allene-oxide synthase (AOS) [8] formed the unstable 12,13-(S)-epoxy-octadecatrienoic acid (12,13-EOT) and 10,11(S)-epoxy-hexadeca(tri)enoic acid (10,11-EHT) as elaborated in figure 3. The enzyme alleneoxide cyclase (AOC) [9] produces cis-OPDA in octadecanoid pathway and dinor OPDA (dnOPDA) in hexadecanoid pathway. The mechanism through which these OPDAs entered the peroxisome is still unresolved but it is known that in *Arabidopsis* the ATP-binding cassette (ABC) transporter COMATOSE (CTS/PXA1/PED3) had been shown to catalyze the ATP-dependent entrance of fatty acids inside peroxisomes that acted as an agent for β-oxidation [10]. Yet, it is the challenge for scientists to search for other pathways for the entrance of OPDA into peroxisome.

Soon the respective OPDA's enter the peroxisome it is reduced to respective oxophytodienoic acid reductase (OPR) [11] which than leaded toward three steps of ß-oxidation for the formation of JA [12]. The 9S,13S-OPDA also termed as cisOPDA is reduced by an enzyme 12-oxophytodienoate reductase (OPR3) for the production of 3-oxo-2-(2'(Z)-pentenyl) cyclopentane-1-octanoic acid (OPC-8:0), and dnOPDA was also reduced respectively to its equivalent hexanoic acid derivative (OPC-6:0) which is elaborated briefly in figure 2.

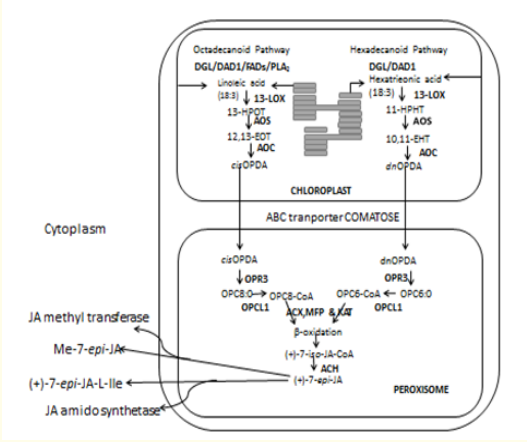

Figure 2: *Synthesis of Jasmonic acid in chloroplast and peroxisome by octadecanoid and hexadecanoid pathway.*

The activation of OPC-8:0 and OPC-6:0 then took place through CoA esterification of the carboxylic moiety and OPC-8:0 CoA ligase 1 (OPCL1) but exact ligase is still unknown for OPC-6:0. The hexanoic and octanoic side chains such as OPC-8:0 and OPC-6:0 respectively are shortened by two or three steps of β-oxidation. The process of β-oxidation involved three enzymes; acyl-CoA oxidase (ACX), a multifunctional protein (MFP, comprising enoyl-CoA hydratase and β-hydroxy-acyl-CoA dehydrogenase activities) and 3-ketoacyl-CoA thiolase (KAT) forming JA-CoA. The last step took place with the help of an enzyme acyl-thioesterase (ACH) resulted in the production of (+)-7-*epi*-jasmonic acid that than epimerized into the more stable (−)-7-*epi*-jasmonic acid [13]. Upon the release to the cytoplasm, it was further transformed into methyl-(+)-7-*epi*-jasmonate (Me-7-*epi*-JA) with the help of JA methyl transferase and into (+)-7-*epi*-jasmonyl-L-isoleucine ((+)-7-*epi*-JA-L-Ile) which was catalyzed by a JA amido synthetase or other derivatives [14]. Thus, the production of Jasmonic

acid derivatives results in the regulation of targeted gene expression [15]. The products produced by gene regulation in sequential manner as seen in figure 3, acts as defensive protein against the external attacking entities.

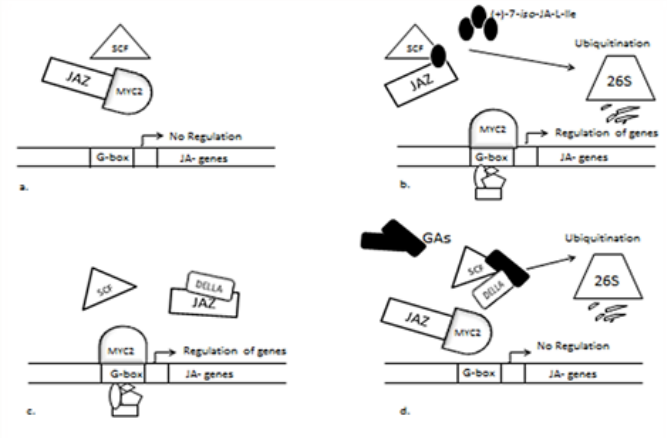

Figure 3: *Jasmonate regulation in plants. a, In resting stage no regulation take place because MYC2 (transcription factor) is bonded with JAZ protein. b, Ubiquitination of JAZ protein took place by proteasome 26S, this results in the escape of MYC2, which bonded with the G-box region together with other transcription factor for production of the JA responsive gene and this stage is termed as active stage. c, In the absence of GA the DELLA got bonded with JAZ , this resulted in the escape of MYC2 which bonded with the G-box for regulating JA genes. d, the presence of GA, thus resulted in the ubiquitination of DELLA protein which resulted in binding of MYC2 with JAZ, thus repression of JA genes occurred.*

Effect of chewing insect feeding on rate of photosynthesis

Plant insect interaction results in 50 % of the decrease in the rate of photosynthesis. Different feeding guilds show differential responses as chewing insect results in defoliation usually increases the rate of photosynthesis [16]. In contrast to this it was proposed that defoliation might result in the increased [17], decreased [18] or remained same with no change of photosynthetic rate [19]. By the attack of chewing insect down regulation of photosynthetic related genes was proved through transcriptomic analysis [20]. The attacking herbivore inhibit the transcription of an enzyme ribulose-1,5-bisphosphate carboxylase (RuBPCase) [21]. Inhibition of RuBPCase activase (RCA) was seen in N. attenuata attacked by Manduca sexta. It was proved that saliva of Manduca sexta contains fatty acid-amino acid conjugates (FACs) that not only triggered the change at transcriptional level but also resulted in the change of plant proteomics such as the reduction of RuBPCase activase (RCA) [22]. RCA was responsible for controlling the activity of RuBPCase, it is an enzyme that is responsible for carbamylation of lysine amino acids at active sites which resulted in activation of the enzymes [23]. Moreover, the jasmonate induced by insect attack resulted in the reduction of growth and photosynthesis as proved by the incorporation of methyl Jasmonate which resulted in the formation of shorter petioles than normal control plants [24]. Shorter growth of petioles was also seen in mutant Arabidopsis, which was specialized for more JA production comparable to its wild type [25]. Jasmonates show the suppression of growth by inhibiting the mitosis in apical meristematic tissue [26], it not only inhibit photosynthesis but also show effects on many energy generating processes [27] and trigger the expression of defensive genes in plants against the attacker [28]. Thus, plant maintains the balance between the defense and growth according to the change applied by a biotic stress. The herbivore attack on *Nicotiana attenuata* wild type plant when compared with the mutant plant deficient in jasmonate synthesis, concluded in the reduction of photosynthesis more in wild type plant than mutant plant because in wild type plant the lipoxygenase signaling pathway effect the photosynthetic electron transport chain which result in the reduction of photosynthesis [29].

Conclusion

The JA signaling pathway is induced by stress stimulus of both wounding and chewing insect attack. And JA is not only present at the site of stress applied but may also present at the site of unwounded region, thus showing the systemic responses. There are many defense gene identified by microarray analysis triggered by wounding or chewing insect attack. There are certain group of plants and insect species which are being focused for such study. The plants mostly included are *Arabidopsis*, tomato, potato, maize and tobacco and the chewing insect larvae belonging to family Lepidoptera. Not all plants stimulate the same kind of chemical defense responses against a single herbivore. In the same way not all insects inject the same kind of elictors inside the plants through their saliva during feeding. It would therefore be of interest to study more variety of plants and insect herbivory to investigate the chemical induction and responses of such interaction. The better variety of plant can be engineered by biotechnologists by understanding the defense systems.

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