Review Article

How Auxin May Contribute to the Regulation of Plant Defense Responses against Herbivory

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Abstract

Numerous studies have indicated that investing in defense usually comes at the expense of plant growth. A phenomenon known as the "growth-defense tradeoff". Nevertheless, recent research puts emphasize on the fact that the degree of cooperation and crosstalk between phytohormones is of key importance in driving plant responses to their ever-changing environment. In this context, the growth hormone indole-3-acetic acid, the most common auxin in the plant kingdom, makes no exception. Several lines of evidence indicate that the relationship between auxin and jasmonates is of particular relevance for the control of plant defenses responses. In this review we discuss multiple levels at which auxin homeostasis can intimately regulate plant defense against biotic foes, paying special attention to responses towards phytophagous predators.

Keywords: Phytohormones; Phytophagous pathogens; Direct/indirect defense; Indole-3-acetic acid; Jasmonates
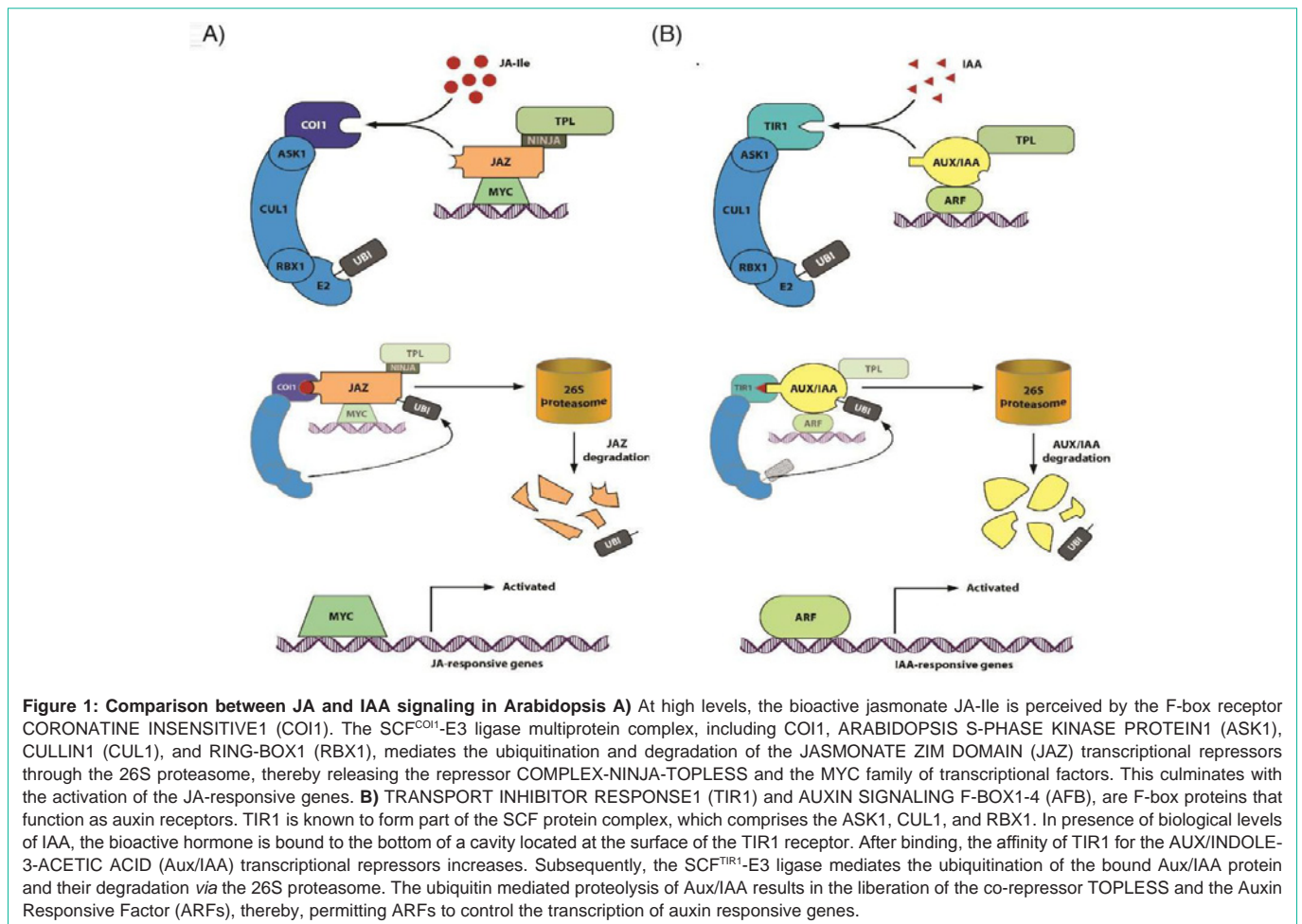
Abbreviations

RNAi: RNA interference

Auxin: An Overview of the Growth-Defense Dilemma

Excluding seed dispersal, the entire life cycle of higher plants proceeds anchored to a fixed place. Due to this fact, the rapid integration of environmental signals is of key importance to develop effective mechanisms to cope with changes including the defense against abiotic and biotic stresses [1,2]. Plant hormones or phytohormones, are small chemical messengers derived from secondary metabolism that take center stage in the integration and translation of environmental demands into physical plant responses [3,4]. The major hormones produced by plants include the "classical" five, i.e. auxins, gibberellins (GA), cytokinins (CK), abscisic acid (ABA), ethylene (ET), and the relatively new compounds salicylic acid (SA), jasmonates, brassinosteroids (BR) and strigolactones [5-7]. Regarding their function, auxins, BR, CK, GA and strigolactones are known to play decisive roles in the orchestration of growth and development [8]. In contrast, ABA, SA, jasmonates and ET are considered to play crucial roles in mediating plant defense responses against pathogens and abiotic stresses, such as drought, light, salinity, or high temperatures [6,9]. Historically, it has been assumed that these two functional blocks act antagonistically and, therefore, growth and defense-related hormones work in separate modules. Supporting this hypothesis, jasmonic acid (JA) and its metabolites, collectively known as jasmonates (JAs), are plant growth inhibitors, mainly involved in counteracting stress against herbivore and pathogen attack [10-12]. *In vivo*-analyses demonstrated that induction of endogenous JAs in Arabidopsis is sufficient to impair primary root growth and leaf expansion [13]. In leaves, JAs exert their growth inhibitory effect through the suppression of mitosis, by arresting the cell cycle in G1 prior to the S transition [14]. In contrast to the growth inhibitory effect triggered by JAs, indole-3-acetic acid (IAA) constitutes the most important plant growth hormone [15-18]. In agreement with the antagonistic relationship between growth and defense, Mutka and co-workers [19] demonstrated that elevation of the endogenous content of IAA, driven by the constitutive overproduction of the Auxin biosynthetic gene *YUC1*, increased the susceptibility of the Arabidopsis mutant against the plant pathogen *Pseudomonas syringae*. Moreover, it has been observed that inactivation of IAA-mediated processes, i.e. cell expansion and plant cell wall relaxation, activates *Nicotiana attenuata* defense in response to the herbivore *Manduca sexta* oral secretion elicitation [10]. Nonetheless, despite all this insight suggesting a negative impact of auxin in plant defense, a number of recent studies have called this assumption into question. For example, it has been observed that several herbivory insects are capable of manipulating IAA biosynthesis to induce abnormal tissue formation, such as galls [20]. In view of these evidences, we specifically try to summarize and discuss recent findings concerning the contribution of auxin in the regulation of plant defense against phytophagous predators in this mini-review. Furthermore, we try to highlight new molecular targets for plant bioengineering that may facilitate increased crop resistance against herbivore foes. The intricacies of IAA biosynthesis and signaling will not be considered here, and readers interested in this aspect should consult the recent reviews on this topic [15,16,21,22].

Direct Plant Defense Activates Auxin Biosynthesis and Signalling

To understand plant defense responses against phytophagous pathogens we should establish two different levels of action, known as direct and indirect defense. Direct defenses involve all the appliances used by the host plant to counteract a specific attack from predators [23-27]. A good example of this is the mechanical protection of the plant surface by the development of spines, thorns and trichomes [28,29]. Focusing on leaf trichomes, studies performed in several plant species have consistently demonstrated that the formation of

Figure 1: Comparison between JA and IAA signaling in Arabidopsis A) At high levels, the bioactive jasmonate JA-Ile is perceived by the F-box receptor CORONATINE INSENSITIVE1 (COI1). The SCF^{COI1}-E3 ligase multiprotein complex, including COI1, ARABIDOPSIS S-PHASE KINASE PROTEIN1 (ASK1), CULLIN1 (CUL1), and RING-BOX1 (RBX1), mediates the ubiquitination and degradation of the JASMONATE ZIM DOMAIN (JAZ) transcriptional repressors through the 26S proteasome, thereby releasing the repressor COMPLEX-NINJA-TOPLESS and the MYC family of transcriptional factors. This culminates with the activation of the JA-responsive genes. **B)** TRANSPORT INHIBITOR RESPONSE1 (TIR1) and AUXIN SIGNALING F-BOX1-4 (AFB), are F-box proteins that function as auxin receptors. TIR1 is known to form part of the SCF protein complex, which comprises the ASK1, CUL1, and RBX1. In presence of biological levels of IAA, the bioactive hormone is bound to the bottom of a cavity located at the surface of the TIR1 receptor. After binding, the affinity of TIR1 for the AUX/INDOLE-3-ACETIC ACID (Aux/IAA) transcriptional repressors increases. Subsequently, the SCF^{TIR1}-E3 ligase mediates the ubiquitination of the bound Aux/IAA protein and their degradation *via* the 26S proteasome. The ubiquitin mediated proteolysis of Aux/IAA results in the liberation of the co-repressor TOPLESS and the Auxin Responsive Factor (ARFs), thereby, permitting ARFs to control the transcription of auxin responsive genes.

these structural barriers is controlled by the combined action of JA, GA, and CK [30-33]. Interestingly, Auxin Response Factors (ARFs), which are key-components of auxin signaling [34,35], have been described to be required for this process. Fahlgren et al. [36] reported that an increased accumulation of ARF3 transcripts promote the formation of abaxial tracheas in *Arabidopsis thaliana*. Likewise, in tomato (*Solanum lycopersicum*) is has been observed that *SlARF3* down-regulation by applying a RNAi approach significantly reduced the trichome density in leaves [37]. On the other hand, plants also limit phytophagous attacks by increasing leaf rigidity and stem strength through the lignification of their cell walls. In this regard, wounding and JA have been associated with lignin biosynthesis through the transcriptional regulation of a series of wound-induced genes, as for example the *CAFFEIC ACID-O-METHYL TRANSFERASE* (*COMT*) [38], which encodes an enzyme implicated in the synthesis of G-lignin [39]. Alternatively, quite some time ago, Vanholme et al. [40] reported that auxin stimulation induced lignin formation in the secondary xylem of *Coelus blumei*. In Arabidopsis, the constitutive overexpression of YUC8 and YUC9, two key enzymes in IAA biosynthesis, has been reported to translate into enhanced lignification [41]. Further underlining the role of increased IAA levels, the tobacco auxin overproducer mutant 35S-iaaM/iaaH has been described to show increased peroxidase gene transcription levels [42]. Peroxidases are known to be involved in the polymerization of lignin

monomers [39]. Thus, it appears tempting to speculate that JA-IAA crosstalk can mediate lignin biosynthesis in response to challenges by biotic foes. The positive interplay between mechanical defense and auxin becomes also evident during the process of herbivore recognition. Perception of a predator challenge mainly occurs after egg deposition or feeding. For pea (*Pisum sativum*) it is known that ovoposition stimulates cell division and neoplasm formation at the egg location, with the intention of impeding the entry of larvae [43]. In agreement with this, the positive effect of auxin in cell division has been well documented [15,44]. Moreover, experiments with the gall-inducing caterpillar *Gnorimochema gallaesolidaginis* showed a significant increment of IAA contents in galls. Thus, although further elucidation is required, it can be hypothesized that egg perception can activate auxin biosynthesis in the host plant. On the other hand, phytophagous predator herbivory commonly entails disruption of plant tissue integrity. It is well characterized that after wounding or oral elicitation the production of the bioactive JA, jasmonoyl-L-isoleucine (JA-Ile), is stimulated [45]. Subsequently, synthetized JA-Ile is perceived by the F-box protein CORONATINE INSENSITIVE 1 (COI1) [46]. As shown in (Figure 1A), the perception of JA-ILE enables the SCFCOI1 complex to bind and ubiquitinate specific repressor proteins, the so-called Jasmonate ZIM Domain (JAZ) family of transcription repressors. The JAZ repressors are, in turn, labeled for degradation by the 26S proteasome, thereby relieving
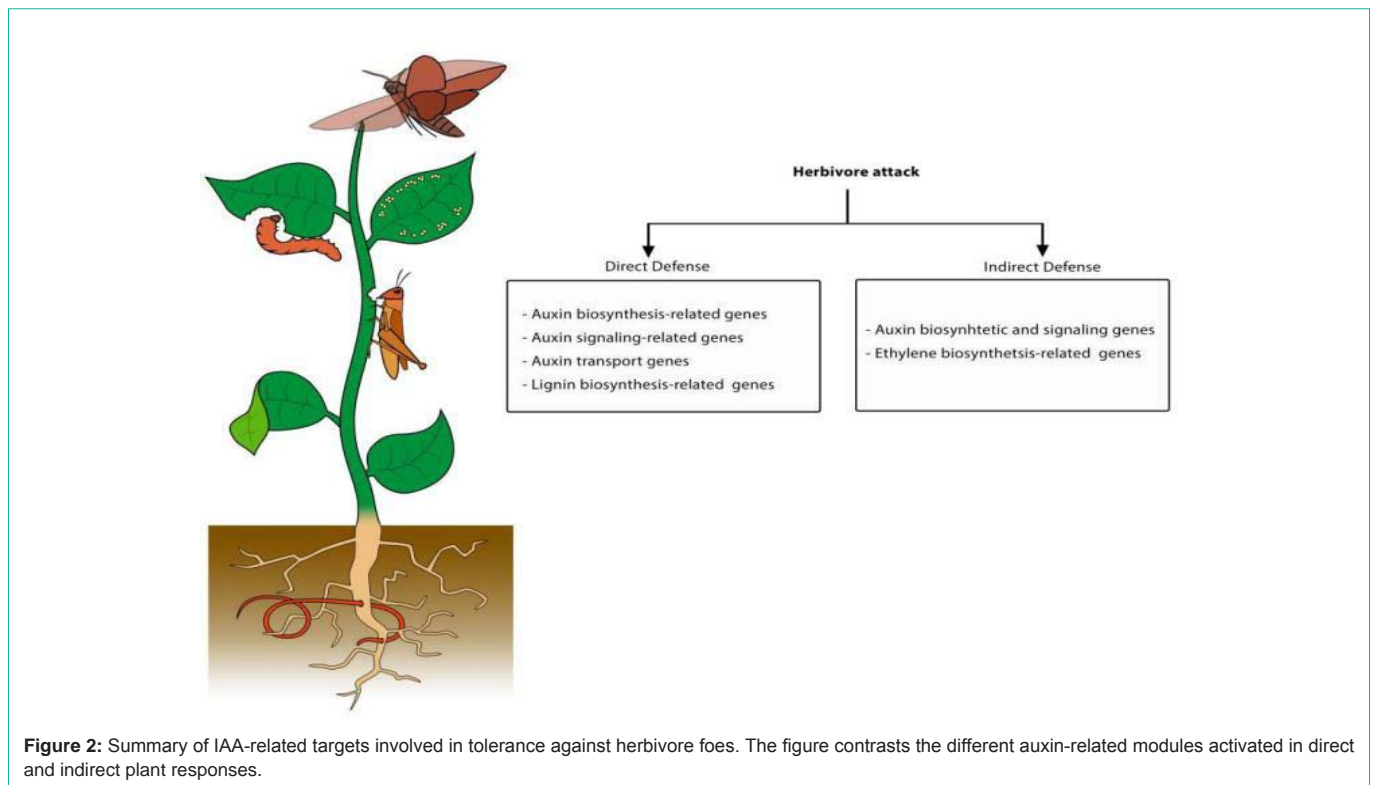
Figure 2: Summary of IAA-related targets involved in tolerance against herbivore foes. The figure contrasts the different auxin-related modules activated in direct and indirect plant responses.

MYC transcription factors from repression and triggering the expression or different subsets of JA-responsive genes [47-49]. Analogously, auxin perception shares a conserved signal transduction mechanism that also uses the 26S-proteasome apparatus (Figure 1B). Importantly, it has been observed that a point mutation of a SCF subunit of Arabidopsis resulted not only in reduced auxin response, but also in a diminished expression of several specifically JA-induced genes, indicating a reduction in JA sensitivity [50]. This evolutionary connection emphasized the value of the synergistic interaction between the two plant hormones, JA and IAA, for the fine-tuning of plant stress responses. As aforementioned, elicitation by oral secretion or wounding play prominent roles in herbivore attack perception. In relation to this, the polymer of *N*-acetyl-*β*-D-glucosamine, commonly termed chitin, is a structural component of insect and spider mite exoskeletons, as well as fungal cell walls and nematode egg [51-54]. However, it has never been reported in plant cell walls. Chitin is a recognized elicitor of plant defense responses [53,55,56], being an important activator of JA-signaling [57]. Recently, Lopez-Moya et al. [58] demonstrated that chitosan, a deacetylated form of chitin, which is widely used in agriculture, stimulates the production of auxin through the modulation of two auxin biosynthetic genes, *YUC2* and *AMI1*, in *A. thaliana*. Remarkably, chitosan also induces the expression of *MYC2*. In addition, Hentrich et al. [59] further reported the induction of two IAA biosynthetic genes, *YUC8* and *YUC9*, after the application of several oxylipins. In their work the authors also described the activation of *YUC9* after mechanical wounding. This observation has recently been confirmed [60], showing the activation of *YUC8* after *Plutella xylostella* larvae attack in *A. thaliana*. *YUC8* induction was accompanied by an elevation of IAA levels in the place of damage, but also in distal parts, such as roots. Therefore, further

elucidation of the molecular mechanism underlying auxin transport upon herbivory attack will help to shed light onto the role of auxin in tolerance against pathogens. In the same way, the exposition of *N. attenuata* plants to *M. sexta* herbivory leads to a significant increase of IAA levels in leaves and roots of attacked plants [61]. In a later study, Machado et al. [62] reported that the *M. sexta*-mediated increase of IAA in *N. attenuata* correlates with a rapid induction of several *YUC*-like genes. Additionally, the authors demonstrated that the simultaneous application of IAA and methyl jasmonte (MeJA) induces the production of anthocyanin in *N. attenuate*, which is believed to acts as a chemical repellent [63]. This observed induction was further reproduced under real or simulated *M. sexta* attack using *A. thaliana*.

A Role of Auxin in Indirect Defense

Indirect defense is based on the capability of attacked plants to emit volatile organic compounds (VOCs), such as terpenoids, glucosinolates, fatty acid derivatives and ET, to attract natural enemies of herbivores [23-25]. A broad number of genes relates to VOC production are induced by JAs [26]. However, earlier studies indicated that IAA is capable of stimulating ET production through the activation of specific ACC-synthase genes, which encode enzymes involved in a rate- limiting step in this process [64-66]. In Arabidopsis, it has been demonstrated that the overproduction of auxin mediated by YUC8 and YUC9 likely results in increased ET production, as these lines are less sensitive to the ET biosynthesis inhibitor 2-aminoisobutyric acid [41]. Thus, it may be concluded that a hormonal cascade employing JA, IAA, and ET contributes to plant defense responses. The main role of VOC emission is to prepare (prime) distal tissues or neighboring plants for a possible imminent

attack. Such a VOC-mediated induction of the defense machinery can provide substantial advantages to responding plants, providing time for the production a battery of secondary metabolites to deter potential aggressors. Interestingly, the chemical communication between plants seemingly involves the canonical auxin perception pathway, too. Sweeney et al. [67] reported that mechanical wounding stimulates both root growth and the activation of auxin signaling, as analyzed by using the DR5: GUS reporter line, in neighboring unwounded *A. thaliana* plants.

Conclusion

Taking all these observation into account, we conclude that although elicitation of the JA-mediated response is the primary event, reconfiguration and adjustment, respectively, of IAA biosynthesis and signaling is likely to contribute to direct/indirect plant resistance against herbivory (Figure 2). However, many questions still remain elusive and require more detailed investigations in the future.

Acknowledgement

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