JIPB Journal of Integrative Plant Biology

Journal of Integrative Plant Biology 2011, **53** (6): 429–445

Invited Expert Review

# Auxin-Oxylipin Crosstalk: Relationship of Antagonists[F]

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[F] Articles can be viewed online without a subscription.

Available online on 26 May 2011 at www.jipb.net and www.wileyonlinelibrary.com/journal/jipb

doi: 10.1111/j.1744-7909.2011.01053.x

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## Abstract

**Phytohormones regulate a wide array of developmental processes throughout the life cycle of plants. Herein, the various plant hormones may interact additively, synergistically, or antagonistically. By their cooperation they create a delicate regulatory network whose net output largely depends on the action of specific phytohormone combinations rather than on the independent activities of separate hormones. While most classical studies of plant hormonal control have focused mainly on the action of single hormones or on the synergistic interaction of hormones in regulating various developmental processes, recent work is beginning to shed light on the crosstalk of nominally antagonistic plant hormones, such as gibberellins and auxins with oxylipins or abscisic acid. In this review, we summarize our current understanding of how two of the first sight antagonistic plant hormones, i.e. auxins and oxylipins, interact in controlling plant responses and development.**

**Keywords:** Auxin; biosynthesis; crosstalk; hormone; jasmonic acid.

**Hoffmann M, Hentrich M, Pollmann S** (2011) Auxin-oxylipin crosstalk: Relationship of antagonists. *J. Integr. Plant Biol*. **53**(6), 429–445.

## Introduction

The lifestyle of plants is largely determined by growth anchored in one place and thus not being able to escape often hostile conditions in their ever-changing environment. Because of this fact, plants must respond and adjust to a multitude of different external cues and coordinate their growth and developmental program accordingly. To cope with problems arising from their sessile lifestyle, they have evolved complex solutions to ensure their survival. Over the course of time, plants have developed a remarkable repertoire of developmental means to shape their basic body plan and optimize their metabolism to given environmental demands. This developmental plasticity involves the permanent activity of meristematic tissues, *de novo* organogenesis, a notable capability for regeneration, and direct growth responses to external stimuli (Tanaka et al. 2006). Likewise, the coordinated course of physiological processes requires the efficient communication between concerned single cells and whole tissues, respectively (Lau et al. 2010; Lehesranta et al. 2010).

Thus far, it is not fully understood how external triggers are converted into adequate developmental changes and how these are internally coordinated in order to regulate plant developmental plasticity. Like animals, plants utilize a chemical-based regulatory network to coordinate their development and ensure communication across the cellular level. Central to this sophisticated network are a limited number of structurally unrelated small bioactive molecules, collectively referred to as phytohormones.

Since the pioneer days in the field of plant hormone physiology, in the first half of the twentieth century, a considerable

amount of phytohormones have been identified, including the five 'classical' plant hormones, i.e. auxin, abscisic acid (ABA), cytokinin (CK), gibberellin (GA), and ethylene (ET) (Davies 2004; Santner et al. 2009), and some additional substances more recently recognized to have hormonal properties, such as brassinosteroids (BRs), jasmonates, salicylic acid (SA), and strigolactones (Vert et al. 2005; Loake and Grant 2007; Wasternack 2007; Gomez-Roldan et al. 2008; Umehara et al. 2008; Browse 2009). Very much like their animal counterparts, plant hormones act at very low, sub-micromolar concentrations. In general, there are striking similarities but also differences in the mode of action of plant and animal hormones. For instance, animal hormones often directly affect physiological outcomes, e.g. the pulse rate in mammals, whereas phytohormones mostly influence the developmental program of a given tissue by triggering transcriptional reprogramming of affected cells. In addition, the biosynthesis of plant hormones is not restricted to specialized tissues, as is the case for animals where for the most part specific glands are responsible for hormone production.

Over recent years, mounting evidence led to the widely accepted concept that plant hormone action is not the read-out of linear pathways, but determined by the extensive combinatorial activity of the signaling molecules and the integration of their signaling pathways, both in terms of regulating growth and development and in adapting to external stimuli (Kazan and Manners 2008; Wolters and Jürgens 2009). Further complicating the scenario, the triggered physiological processes are not only dependent on the perceived stimulus, but also on the specific properties of the responding tissue in terms of sensitivity and responsiveness to a given signaling molecule class (Trewavas 1982; Bennett et al. 2005).

The plant hormone network can affect plant development and physiological responses on several different levels involving for example, control of mRNA and protein synthesis, configuration, modification, and turnover of proteins, as well as by hormone transport and reversible or irreversible inactivation of active signaling molecules (**Figure 1**). For instance, directed polar auxin transport and spatiotemporally defined auxin maxima, and the therewith coupled control of gene expression and gene signaling hierarchies, coordinate organogenesis and axis formation during embryogenesis and patrol plant development (Bayer et al. 2009; Möller and Weijers 2009; Zhao 2010). Another example is the modification of plant hormones by, e.g., glycosylation, methylation, or amino acid conjugation, which either serve to reversibly modulate their activity or confer the first step in their irreversible metabolism (Bajguz 2007; Loake and Grant 2007; Ludwig-Müller 2011). In addition, plant hormones can also directly impact the synthesis or degradation of other signaling molecules, as has been shown, amongst other relationships, for ET production, which is induced both by auxin and BR (Swarup et al. 2002; Arteca and Arteca 2008).

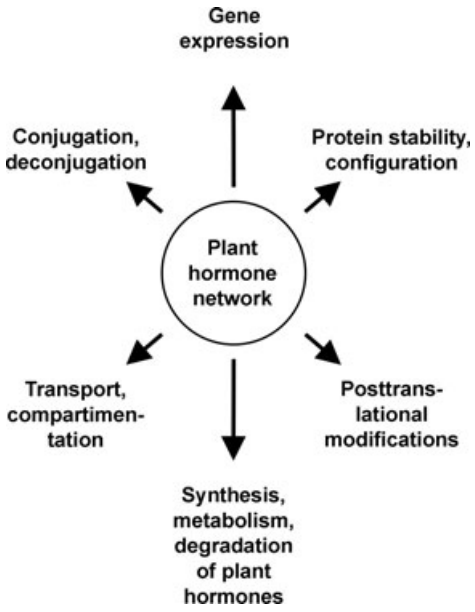


**Figure 1. The plant hormone network affects plant development on several different levels.**

A plethora of different physiological and biochemical processes constitute possible targets of phytohormone action. They reach from the regulation of gene expression and protein abundance over posttranslational modifications of proteins and the influence of plant hormone homeostasis by affecting synthesis, metabolism, and conjugation of signaling compounds to the impact on hormone transport and compartmentation.

Also, posttranslational modifications of proteins can be a target for modulation by plant hormones, as is the case in CK, ABA, and ET signaling which involve phosphorylation of downstream components of the signal transduction machinery (Stepanova and Alonso 2009; Argueso et al. 2010; Umezawa et al. 2010).

The past has taught us that, due to the high level of complexity, molecular "bottom-up" approaches are seemingly not suited to explain plant growth and development as a whole (Morandini and Salamini 2003). Those kind of approaches to understanding sophisticated adaptive systems such as plants are, on their own, likely to struggle and stumble in the close-meshed network of physiological and biochemical relationships. Consequently, those observations initiated a shift in thinking from the level of isolated pathways and hormone actions to that of integrated plant hormone networks, which very much promoted the engagement of integrative systems biology "top-down" approaches for attempts to explain complex regulatory processes both in plant stress responses and development (Hammer et al. 2004).

The term crosstalk is often used indiscriminately to describe the influences of two or more signaling pathways that are functionally defined by biochemistry or genetics, in triggering

one response. Such general usage of the term is in vogue because it encompasses a multitude of different processes, e.g. positive and negative signaling, or multi-layered gene regulation. In other places, crosstalk is occasionally defined as particular components shared between more than one distinct signaling pathway (Munday et al. 2006). However, several lines of evidence highlight that especially the first mentioned usage of the terminology can be misleading: it is possible that several hormones influence one particular physiological response without the respective pathways interacting at all. In such a case one would have to speak about co-regulation, rather than crosstalk. Therefore, the not precisely defined usage of the term demands a consolidation of the discourse used. The word crosstalk implies both exchange of information and mutual interference, which culminates in a certain reaction or response. Plant hormone crosstalk may be defined as the result of a mutual positive influence on a particular plant response that is essentially based on the integration of multiple incoming stimuli and the triggering and fine-tuning of appropriate developmental responses according to given environmental demands on a tissue-specific as well as temporal basis. A recent publication suggested the division of crosstalk into three distinguishable levels of either direct or indirect crosstalk and co-regulation (Chandler 2009); a picture that makes a lot of sense.

In this framework, direct crosstalk describes molecular processes where different hormone signaling pathways co-regulate gene expression of common targets by acting on their promoters or by affecting the same target proteins. In this type of crosstalk, single components of the hormone-signaling machineries, such as AXR1 or AXR6 (Tiryaki and Staswick 2002; Ren et al. 2005), often act as hubs, involved in integrating incoming signals (**Figure 2A**). Indirect crosstalk is mediated by the modulation of a hormone-signaling pathway by another hormone signaling pathway, which may involve processes such as, for example, alteration of hormone sensitivity/perception (Lackman et al. 2011), hormone abundance/metabolism (Arteca and Arteca 2008; Zhang et al. 2010), and hormone transport/sequestration (**Figure 2B**) (Sun et al. 2011). By contrast, co-regulation denotes a process that is controlled by more than one signaling molecule, with the overall response being determined by the merged outputs of the independent reactions, which can culminate in an additive, synergistic, or antagonistic manner. However, with respect to co-regulated processes, it has to be noticed that there is no necessity of any kind of exchange or interaction between the signaling pathways (**Figure 2C**). As an example for such kind of process, one may quote the synergistic control of root development by CK and auxin (reviewed by Bishopp et al. 2011).

In general, investigation of hormonal crosstalk is challenging, as for a long time hormone functions have largely been studied separately, which makes operational data integration difficult to achieve. For example, it wasn't until the beginning of this century that the plant scientific community was able to answer the need for simultaneous plant hormone analysis technologies (Müller et al. 2002; Chiwocha et al. 2003; Schmelz et al. 2003; Durgbanshi et al. 2005), which was, in fact for quite some time largely hampered by inadequate analytical means. Nowadays, the available technologies, whether it is transcript profiling at a whole-genome scale, or untargeted metabolite profiling by cutting-edge mass spectrometric techniques, have greatly improved. Thus, major breakthroughs in deciphering interaction nodes within the hormonal network and in resolving the intricate regulatory patterns in this framework may be expected within the next five to ten years. This will likely include the discovery of distinct responses of the different signaling compounds to defined internal and external stimuli, and how these signals are integrated on the molecular level. In order to achieve this goal, a profound understanding of feedback and feed-forward loops, as well as of spatiotemporal regulation of hormone contents at the cellular, tissue, and organ level is required.

In this context, the aim of this review is to summarize current knowledge on the mechanistic and conceptual crosstalk between auxin and oxylipins, in particular between indole-3-acetic acid (IAA) and jasmonic acid (JA); two plant hormones that, at first glance, appear to have not much in common.

## Shared Components of Auxin and Jasmonic Acid Signaling Pathways

Plant hormone crosstalk is suggested to mainly occur at the gene regulatory level, rather than on the signal transduction level involving shared pathway components, as the phytohormone signaling pathways are often very short and use non-canonical signaling modules (Jaillais and Chory 2010). However, there are also a number of good examples of hormonal crosstalk on the signal transduction level in plants. Over the past few years, numerous genetic screens uncovered many of the proteins that contribute to plant hormone signaling, thereby furthering the current understanding of phytohormone action. One particularly important finding was the identification of receptors and perception mechanisms for auxin (Dharmasiri et al. 2005; Kepinski and Leyser 2005; Tan et al. 2007), GA (Ueguchi-Tanaka et al. 2005), jasmonate (Chini et al. 2007; Thines et al. 2007; Yan et al. 2009; Sheard et al. 2010), and ABA (Ma et al. 2009; Pandey et al. 2009; Park et al. 2009; Santiago et al. 2009a; 2009b; Melcher et al. 2010). Taken together, those results provided a greatly improved picture of hormone perception and facilitated comparisons between different hormone signaling pathways. From these it is clear that a number of hormones, including auxin, JA, GA, and ethylene (ET), share a mechanistically conserved perception mechanism that makes use of the

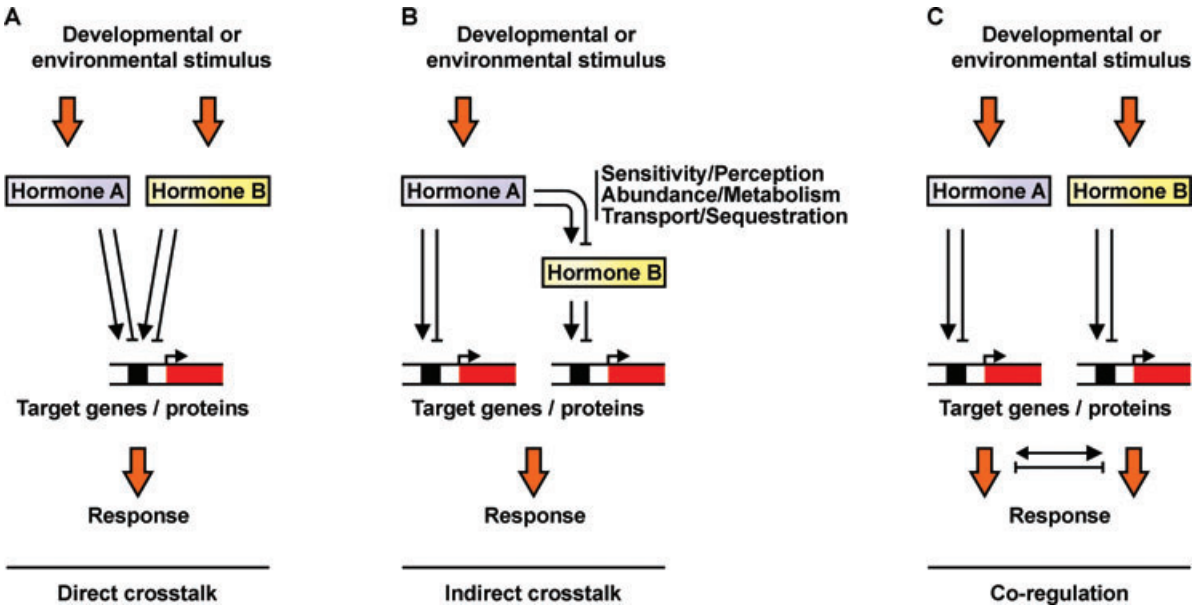


**Figure 2. Schematic overview of the different levels of plant hormone crosstalk and co-regulation, respectively. Three different operational modes can be distinguished:**

**(A)** Direct crosstalk is given when one (developmental or environmental) stimulus affects the homeostasis of two or more phytohormones, which, in turn, act on defined target genes to trigger one response. The same holds true in the matter of two proteins that interact to directly affect a common response, or to create a node in the signal transduction network.

**(B)** Indirect crosstalk describes a scenario in which the homeostasis of one plant hormone is altered by a developmental or environmental trigger, which subsequently affects, besides the expression of particular target genes, the sensitivity/perception, abundance/metabolism, or transport/sequestration of a second phytohormone. The effect on the homeostasis of the second hormone can be either positive or negative. The mechanism of indirect plant hormone interactions can show a domino effect or have a snowball effect, depending on the ability of the initially induced hormone to affect one ore more plant hormone signaling pathways. It is important to notice that the involved plant hormones corporate in regulating one common response.

**(C)** Co-regulation can be regarded as an event where a given stimulus is able to activate two or more signaling pathways, which control separate outcomes that join together in either an additive, synergistic, or antagonistic manner to give rise to a combined response. While in this setting the different outcomes add up to one common response, the plant hormone signaling pathways might not interact at all.

ubiquitin-26S proteasome system (UPS) (Dreher and Callis 2007; Vierstra 2009). Herein, hormone-governed gene activation is achieved through the targeted degradation of repressor proteins via the UPS.

In very simplified terms, the activity of transcription factors, such as Auxin Response Factors (ARFs) and basic helix-loop-helix (bHLH) transcription factors of the MYC family, is blocked through binding to specific repressor proteins (**Figure 3**). In the given examples, these repressors are so-called Auxin/Indole-3-acetic acid (Aux/IAA) and Jasmonate ZIM-domain (JAZ) proteins, respectively. Once internal or external cues trigger the accumulation of endogenous signaling molecules, receptor proteins, such as Transport Inhibitor Response 1 (TIR1) and Coronatine Insensitive 1 (COI1), which serve as components of bigger ubiquitin protein ligase (E3) complexes and determine their specificity, perceive the alteration. This finally leads to a transfer of ubiquitin to the repressor proteins mediated by the E3 protein ligase complex, which marks them for protein degradation by the 26S proteasome, thereby releasing the transcription factors from repression.

Of course, the *in vivo* situation is much more complex than this very superficial summary. A more detailed description will be given in the following. While a family of bric-a-brac-tramtrack-broad complex (BTB)-type E3 ligases are responsible for ET perception, S-Phase Kinase-associated Protein 1 (SKP1)-Cullin 1 (CUL1)-F-box SCF-type Cullin-Ring ligases mediate perception of both IAA and JA. SCF-type ubiquitin protein ligase (E3) complexes share a highly conserved core structure (**Figure 3**), each of which is completed by one of about 700 F-box proteins that confer substrate specificity to the multi-subunit complex (Gagne et al. 2002; Risseeuw et al. 2003; Takahashi et al. 2004). With regard to auxin perception, the

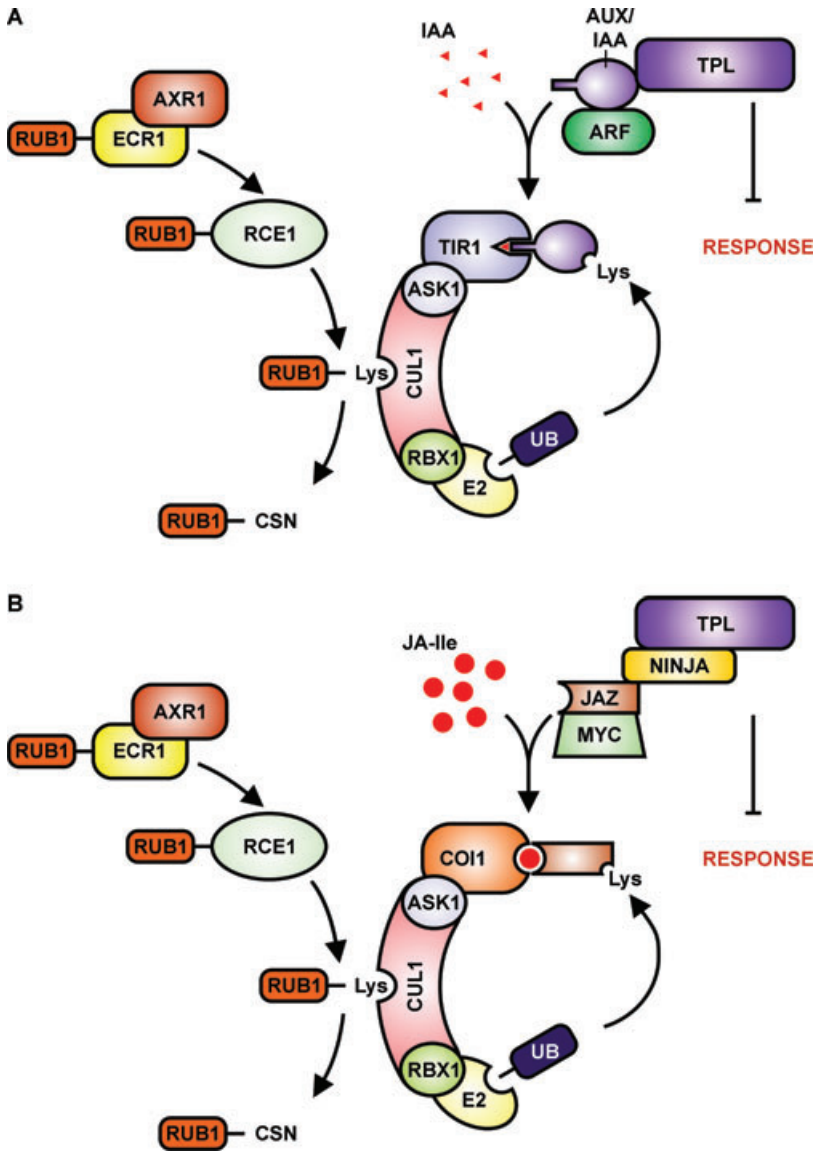


**Figure 3. Composition of $SCF^{TIR1}$ and $SCF^{COI1}$ E3 protein ligase complexes contributing to IAA and JA-Ile perception.**

**(A)** The IAA signaling pathway involves the ubiquitylation of the Auxin/Indole-3-acetic acid (Aux/IAA) proteins by an SCF-type E3 ligase complex containing the F-box protein Transport Inhibitor Response 1 (TIR1). Aux/IAA repressors are ubiquitin-tagged by the $SCF^{TIR1}$ complex, marking them for targeted protein degradation by the 26S proteasome. Thereby, Auxin Response Factor (ARF) transcriptional regulators, which directly interact with Topless (TPL) co-repressors, are released from repression.

**(B)** JA signaling involves the ubiquitylation of JA-ZIM-domain (JAZ) proteins by an E3 SCF multi-enzyme complex containing the F-box protein Coronatine-Insensitive 1 (COI1). Like the Aux/IAA proteins, JAZ repressors get ubiquitylated at a specific lysine (Lys) residue, which marks them for protein degradation by the 26S proteasome. Note, in contrast to ARFs, JAZ proteins recruit TPL co-repressors not directly, but through Novel Interactor of JAZ (NINJA) adapter proteins. It has to be stressed that the two perception machineries share several SCF core components, e.g. Arabidopsis S-Phase Kinase-associated Protein 1 (ASK1), Cullin 1 (CUL1), and Ring-Box 1 (RBX1). Notably, normal activity of both SCF-type E3 protein ligase complexes depends on the reversible rubylation of CUL1. Rubylation of CUL1 is mediated by further shared components of the two signaling pathways. In a first step, Related to Ubiquitin 1 (RUB1) is activated through a heterocomplex composed of either Auxin Resistant 1 (AXR1) or Auxin Resistant-like proteins (AXL) and E1 C-Terminal-related 1 (ECR1). Activated RUB1 is then passed on to Related to Ubiquitin 1 (RUB1) Conjugating Enzyme 1 (RCE1) that transfers RUB1 to a particular lysine (Lys) residue of CUL1. Derubylation of CUL1 both in $SCF^{TIR1}$ and $SCF^{COI1}$ involves the active participation of the Constitutive Photomorphogenesis 9 (COP9) signalosome (CSN).

underlying molecular mechanism is well characterized. The active hormone, free IAA, is bound at the bottom of a cavity at the surface of a specific F-box protein, either TIR1 or of the related family of Auxin Signaling F-Box (AFB) proteins (Parry et al. 2009; Calderon-Villalobos et al. 2010). In case of TIR1, it is known that the bottom of the surface binding pocket is decorated with an inositol hexakiphosphate ($InsP_6$) co-factor (Tan et al. 2007). Through the binding of IAA the affinity of TIR1 for its substrate, the Aux/IAA transcriptional repressor proteins, is greatly enhanced. Ultimately, this culminates in the tagging of the Aux/IAA proteins by ubiquitin and their subsequent degradation through the 26S proteasome. Thereby, active gene regulation is accomplished by unblocking the auxin specific transcription factors of the ARF family (Kieffer et al. 2010). With the exception that the bottom of the surface binding pocket of COI1 contains inositol pentakiphosphate ($InsP_5$) as co-factor instead of $InsP_6$ (Sheard et al. 2010; Mosblech et al. 2011), jasmonates, i.e. (+)-7-*iso*-jasmonoyl-L-isoleucine (Fonseca et al. 2009), trigger the regulated degradation of JAZ repressor proteins via the $SCF^{COI1}$ complex in a very similar way (Chico et al. 2008; Chini et al. 2009). After the release of the transcription factors from repression, they are able to form hetero- or homodimers, which likely modulates their transcription regulatory activity (Chini et al. 2007; Guilfoyle and Hagen 2007; Chung and Howe 2009), and can develop their gene regulatory abilities.

As a slight variation of this theme, the integration of GA-dependent signals is based on the comparable concept of repressor protein degradation via the UPS in a hormone-dependent manner. Here, so-called DELLA proteins constitute targets of $SCF^{GID2/SLY1}$ multi-subunit E3 ligase complexes (McGinnis et al. 2003; Sasaki et al. 2003). However, in this case, hormone binding does not occur through the F-box protein itself, but is mediated by an additional receptor protein, GID1 (Ueguchi-Tanaka et al. 2005; Nakajima et al. 2006), that interacts with a DELLA protein in a GA-dependent manner. Subsequently, this results in the interaction of the GID1-DELLA heteromer with the SCF complex to tag the DELLA protein for degradation (for review see: Ueguchi-Tanaka and Matsuoka 2010).

Based on the common composition of basic elements, it is obvious that mutations in core components such as, for instance, CUL1 translate into highly pleiotropic phenotypes affecting IAA and JA perception at the same time (Quint et al. 2005; Moon et al. 2007). To facilitate normal function of the SCF complex, it is necessary that CUL1 is post-translationally modified; a task that is accomplished by the conjugation of so-called Related to Ubiquitin (RUB) proteins. This process requires the action of two enzymes, a RUB-activating and a RUB-conjugating one. In Arabidopsis, the first step in the RUB conjugation pathway is the assembly of bipartite complexes of AXR1 and AXL proteins with ECR1 (Dharmasiri et al. 2007). Those heterodimers are capable of activating Related to Ubiquitin 1 (RUB1) and, in conjunction with the Related to Ubiquitin 1 (RUB1) Conjugating Enzyme (RCE1), promote the reversible rubylation of the CUL1 subunit (del Pozo and Estelle 1999; Hotton and Callis 2008; Zhang et al. 2008). Derubylation of CUL1 requires the functionally still enigmatic Constitutive Photomorphogenesis 9 (COP9) signalosome complex, which was described initially as a key regulator of photomorphogenesis (Feng et al. 2003; Wei and Deng 2003). Since both IAA and JA perception depends on CUL1 and its post-translational modification, this illustrates why both the *axr1* and *axr6* mutation results in insensitivity both to IAA and JA (Tiryaki and Staswick 2002; Ren et al. 2005).

Apart from these striking evidence for component sharing in hormone perception, some recent publications also pointed to a possible sharing of downstream components involved in directing target gene expression. As briefly outlined before, a specific class of Aux/IAA proteins represses ARF transcriptional regulators. For a long time it was assumed that the repression of ARFs was solely based on heterodimerization with their specific repressors, which was thought to be sufficient to block their gene regulatory activity (Tiwari et al. 2003; Guilfoyle and Hagen 2007). However, recent findings highlighted that in the case of the Aux/IAA protein IAA12/Bodenlos, which is a repressor of ARF5/Monopteros, binding of the additional Topless (TPL) co-repressor protein (**Figure 3A**) through an Ethylene Response Factor (ERF)-associated amphiphilic repression (EAR) motif to IAA12/Bodenlos is required for the transcriptional repression of downstream auxin response genes (Szemenyei et al. 2008). Intriguingly, the TPL co-repressor is also found in JAZ/MYC complexes (**Figure 3B**). The major difference is that TPL does not directly bind to the JAZ repressor proteins. Instead, it is recruited through the EAR domain of an additional Novel Interactor of JAZ (NINJA) adaptor protein that has been shown to physically interact with JAZ (Pauwels et al. 2010). Detailed sequence analysis of NINJA-related AFP proteins, involved in ABA signaling (Garcia et al. 2008), uncovered a wide distribution of the EAR motif previously identified in NINJA and the Aux/IAA proteins, suggesting that TPL possibly acts as a further point of convergence or integrator in several plant hormone signaling pathways.

Some very recent publications underlined an intimate crosstalk of JA and ABA signaling. On the one hand, it has been demonstrated that MeJA treatment transcriptionally affects the expression of the tobacco ABA receptor *NtPYL4* that shares high homology with members of the Arabidopsis PYR/PYL/RCAR ABA receptor family, in particular with *PYL4*. In addition, MeJA modulates *NtT172* expression, a gene that encodes a tobacco type 2C Protein Phosphatase (PP2C) with homology to the Arabidopsis HAB1 phosphatase (Lackman et al. 2011). PYR/PYL/RCAR ABA receptor family members act as repressors of PP2Cs that function as global negative

regulators of ABA signaling (for a more detailed introduction into ABA perception and signaling see: Cutler et al. 2010; Umezawa et al. 2010). Mainly from this simultaneous transcriptional regulation of *NtPYL4* and the *NtT172* PP2C encoding tobacco gene and alterations in anthocyanin accumulation and biomass production in wild type and *pyl4* and *pyl5* null mutants of Arabidopsis, the authors infer that ABA – JA crosstalk serves as a mechanism to balance a compromise between plant growth and defense. On the other hand, it has been demonstrated that the JA precursor 12-oxo-phytoyienoic acid (OPDA) operates in combination with ABA to regulate seed germination in Arabidopsis (Dave et al. 2011).

Coming back to the crosstalk between auxin and JA signaling pathways, interaction through the transcriptional repressors themselves may not be ruled out *a priori*. While such kind of physical interaction has not yet been demonstrated for Aux/IAA and JAZ proteins, DELLA proteins that are considered to play a key role in integrating plant responses to a multitude of developmental and environmental stimuli (Achard et al. 2006; Feng et al. 2008; Navarro et al. 2008) have been found to manipulate MYC2 transcription factor activity through competitive binding to the JAZ1 repressor protein, which ultimately modulates JA signaling (Hou et al. 2010). Whether such modulations of transcription factor activities by the interaction of DELLA with JAZ proteins does also apply to other targets of JAZ proteins, such as MYC3 and MYC4 (Fernández-Calvo et al. 2011) or MYB21 and MYB24 that are attributed to be key-regulators of male fertility (Song et al. 2011), is yet to be demonstrated. At least, it is known that GA can promote JA synthesis through DELLAs to control the transcription levels of MYB21 and MYB24 (Cheng et al. 2009; Peng 2009).

At present, it seems likely that additional insight into the different hormone signaling pathways will provide further evidence for specific shared components in multiple hormone signal transduction cascades. In general, it may be concluded that such additional components add more features to the signaling pathways, thereby increasing the means to fine-tune hormonal crosstalk on the signal transduction level.

## Auxin and JA Pathways Crosstalk through the Action of Auxin Response Factors

As already broached above, expression of the majority of auxin responsive genes is directed by a small group of DNA-binding transcription factors of the ARF family. In Arabidopsis, the *ARF* gene family consists of 23 members, comprising 22 full-length *ARF* genes and one truncated gene (*ARF23*), having a premature stop codon in the first homology domains (DBD) (Guilfoyle and Hagen 2007; Lokerse and Weijers 2009). When judged by their sequence homology, most of the ARF proteins fall in either sister pairs, including ARFs 1-4, 6-8, 11, and 18-19, or in triplets as in the case of ARF10, 16, and 17 (Remington et al. 2004). However, this clustering does not necessarily give an account of their functional relationships, which are still poorly understood. While ARF6 and ARF8 fall into one sister pair and show functional interaction in regulating one particular physiological response (Nagpal et al. 2005), plant literature also contains examples of non-paired ARF cooperativity, e.g. ARF5/ARF7 (Hardke et al. 2004).

With regard to the interplay of auxin and JA signaling, ARF6 and ARF8 are of particular interest. In addition to an impaired auxin response, *arf6arf8* double mutants exhibit aberrant flower maturation, and both JA deficiency and reduced transcription of JA biosynthesis genes in their flowers. This is a good example for direct but spatiotemporally restricted crosstalk of auxin and JA in the regulation of a set of target genes (JA biosynthesis genes) during flower development (Nagpal et al. 2005). Moreover, it also highlights the cooperative function of auxin and JA in controlling flower development and fertility. The essential contribution of JA to conferring stamen development, pollen fertility, and anther dehiscence has already been documented (Ishiguro et al. 2001). The group of Alain Goossens has recently made another very intriguing observation concerning the crosstalk between auxin and JA signaling. Their findings strongly suggest that the expression of *JAZ1*/*TIFY10A* is not exclusively induced by JA, but that it is also an early auxin responsive gene. By analyzing auxin-induced *JAZ1*/*TIFY10A* expression level in the *arf6* and *arf8* single mutants and an *arf6arf8* sesquimutant (*arf6*/*arf6*;*arf8*/*ARF8*), they were able to demonstrate that both ARF6 and ARF8 contribute to the auxin-dependent induction of *JAZ1*/*TIFY10A* (Grunewald et al. 2009). Thus, providing further strong evidence for a tight molecular interconnection of auxin and JA signaling.

On top of that, the yet discussed molecular mechanisms in phytohormone crosstalk did not encompass possible post-translational modifications of respective transcription factors, which may contribute to render them active or inactive, depending on their nature and the type of modification. For example, evidence has been provided supporting ARF2 phosphorylation by the GSK3-type kinase BIN2. This has been shown to alleviate ARF2 DNA-binding and repressor activity (Vert et al. 2008). Respective modifications both of ARF and MYC transcription factors, mediated by commonly shared components such as kinases, may emerge as a general theme in plant hormone crosstalk and add another layer of complexity to the sophisticated network of auxin-oxylipin interaction.

Although on the signaling level currently not many more convergence points in auxin-oxylipin crosstalk are known, it is becoming increasingly clear that several transcription factors are equally regulated by multiple plant hormones (Peng et al. 2009). In addition to this finding, the Arabidopsis Hormone Database project (http://ahd.cbi.pku.edu.cn) identified 17% of all hormone-responsive genes to be controlled by more than

one hormone. This observation is supported by further studies on the synthetic *DR5* promoter, which basically consists of the tandemly arranged TGTCTC *cis*-regulatory element that is recognized as an auxin response element (ARE) (Ulmasov et al. 1995; Ulmasov et al. 1997). It has been reported that *DR5* is also responsive both to BR and methyl jasmonate (MeJA) (Nakamura et al. 2003; Sun et al. 2009), implying intimate crosstalk of numerous phytohormone signaling pathways using common sets of *cis*-elements. BR and JA pathways also directly interact with one another, although the underlying molecular mechanism may be very complex and has yet to be uncovered. However, several lines of evidence suggested that BR is involved in JA signaling and repress JA-governed inhibition of root growth (Ren et al. 2009). Summarized, this puts forward the concept that *cis*-regulatory elements in the promoters of hormone responsive genes are primary crosstalk nodes that play a key role in plant hormone interactions. Likewise, it stresses the importance of projects that are dedicated to identifying specific transcription factor binding sites in promoters on a whole-genome scale, no matter whether it is achieved by bioinformatics strategies, such as the AGRIS or AthaMap Database projects (Davuluri et al. 2003; Bülow et al. 2009), or by improved microarray approaches (Godoy et al. 2011).

## Indirect Auxin-Oxylipin Crosstalk Mutually Affects Hormone Homeostasis and Transport

Indirect hormone crosstalk is likely to be the most common method of phytohormone interaction. There is a wealth of information in the literature on the interactivity of plant hormones. Herein, the interaction of auxin and oxylipins makes no exception. The question is no longer whether crosstalk takes place, but rather how it is spatiotemporally controlled in the developmental and environmental context, which is, for instance, reflected by a shift in the literature from phenomenological descriptions of single crosstalk events towards integrative attempts in understanding and modeling how things are interconnected with each other (Liu et al. 2010). This is, however, a very arduous and intricate task, due to the intriguing complexity of plant hormone networking, which often takes place in dose-, species-, tissue-, and inducer-specific manner (Kazan and Manners 2008).

Thus far, the interaction between auxin and JA is still only poorly investigated, but there is growing evidence for substantial indirect crosstalk between these two signaling molecules. Auxin formation in Arabidopsis is enhanced by JA-mediated induction of auxin biosynthesis genes (Dombrecht et al. 2007). Several lines of evidence indicated a MYC2-regulation of L-tryptophan (Trp) metabolism during JA signaling. Both the expression of Trp biosynthesis genes, such as *ASB1*, *IPGS*, *TSA1*, *TSB2*, and that of indole glucosinolate biosynthesis genes, like for example *CYP83B1* and *CYP71B15*, have been shown to be substantially impaired in the *myc2/jin1* null mutant. In addition, the jasmonate-mediated induction of *ASA1, NIT3, YUC2* and the general induction of auxin efflux and influx carriers, such as *Pin-formed 1* (*PIN1*), *PIN2*, and *AUX1*, have been reported to proceed in an $SCF^{COI1}$-dependent manner (Sun et al. 2009; Sun et al. 2011). The induced expression of *ASA1*, *ASB1*, *IPGS*, *TSA1*, and *TSB2* is suggested to direct the substrate flux via enhanced anthranilate synthesis to an increased production of Trp. According to present knowledge, Trp is the major starting point both of auxin (Lehmann et al. 2010; Zhao 2010) and indole glucosinolate (IG) biosynthesis (Sønderby et al. 2010). The differential expression of Trp and IG biosynthesis genes in combination with an observed increase in IG levels suggested stress-mediated metabolite flux to be directed towards IGs that might occur at the expense of auxin production. A possible decrease of IAA levels would be in agreement with the reduced expression of *ILR1*, encoding an IAA amidohydrolase (Bartel and Fink 1995; Campanella et al. 2003), in the *myc2/jin1* background, which is suggested to be involved in the release of free IAA from IAA-amino acid conjugates (Dombrecht et al. 2007). Arguing against this hypothesis, *ILR1* as well as *IAR3* and *ILL5*, encoding two other IAA conjugate hydrolases, appeared to be up-regulated by either different oxylipins, i.e. OPDA, JA, and MeJA, or coronatine, a mimicry of the active jasmonate JA-Ile (Fonseca et al. 2009), in other studies (Taki et al. 2005; Uppalapati et al. 2005). Moreover, the Anthranilate Synthase $\alpha$1 (*asa1-1*) null mutant shows decreased IAA production in response to MeJA treatment (Sun et al. 2009), and coronatine has been observed to increased free IAA levels in an $SCF^{COI1}$-dependent manner (Uppalapati et al. 2005). During clubroot disease of Chinese cabbage, caused by the obligate biotroph *Plasmodiophora brassicae*, both endogenous JA and IAA levels were found to be elevated. Associating these two events, it has further been shown that the increased JA levels caused the induction of a sub-set of auxin biosynthesis-related genes, including myrosinase and nitrilase, two gene products involved in the decomposition of IGs to free IAA (Grsic et al. 1999). In addition, a more recent study highlighted that *P. brassicae*-induced IAA production occurs independent of IG biosynthesis (Siemens et al. 2008). From this we may instead conclude that oxylipins are capable of inducing auxin and IG production to equal extents.

Looking into the opposite direction, auxin has also been recognized to induce JA biosynthesis (Tiryaki and Staswick 2002). At least two of the JA biosynthesis genes, *LOX2* and *AOS*, appeared to be considerably induced by IAA in the respective study, which led the authors to the suggestion that this kind of indirect crosstalk may provide a mechanistic link to amplify the JA signal. Taken together, the presented pieces of evidence strongly infer that oxylipin and auxin homeostasis is closely

interlinked, possibly forming a positive feedback loop that is likely connected with metabolic side reactions, including signal molecule conjugation and decay. In this context, the formation of JA-Trp and IAA-Trp conjugates is worth particular mention (Staswick 2009). Unlike JA-Ile, Trp conjugates of JA do not act through $SCF^{COI1}$ and do not have any intrinsic JA signaling molecule character. Instead, it has been shown that JA-Trp and IAA-Trp are capable of suppressing auxin responses in Arabidopsis roots. They may determine an additional negative regulatory loop, possibly by altering the balance between JA and IAA pools, thereby adding a further layer of physiological checkpoints to the network.

## What is the Added Value of Auxin-Oxylipin Crosstalk?

The previous paragraphs gave an overview of nodes and hubs connecting auxin with oxylipin signaling pathways and provided strong arguments for extensive and intimate crosstalk between the two pathways. However, when looking at their general characteristics, the value of their interaction is not obviously recognizable. JA is a well-characterized inhibitor of plant growth mainly involved in plant defense but also in plant developmental processes (Browse 2005; Wasternack 2007; Browse 2009; Yang et al. 2009). Early observations on the inhibitory effect of jasmonates on auxin-enhanced coleoptile growth of intact monocotyledonous seedlings attributed the negative impact of JA to the inhibition of cell wall polysaccharide synthesis (Ueda et al. 1994; Miyamoto et al. 1997) and to the shift of the intracellular pH (Irving et al. 1999). Meanwhile, it has been found that JA exerts its growth inhibitory effects also through the suppression of mitosis (Zhang and Turner 2008). By contrast, IAA is one of the most prominent and versatile growth promoters found in plants, involved in an extremely broad array of different physiological processes (Woodward and Bartel 2005; Zhao 2010). However, this simplified picture does not generally prove correct, since high auxin concentrations often turn out to be inhibitory, being the reason why the maximum endogenous concentration has to be strictly controlled. Different plant tissues show varying dose-dependencies towards auxin; a circumstance that has early been highlighted, e.g., in a famous classical plant physiological study by Kenneth Thimann (1938), one of the founders of modern plant hormone biology. Nevertheless, the generally used classification system of growth inhibiting and growth promoting plant hormones, of course, provokes the obvious question of how and in what framework the putative antagonists may interact. In the following, a few examples for possible modes of interaction will be discussed against a backdrop of defined physiological circumstances. First, we will address some very interesting examples of the intimate interaction of auxin and JA came from Peter Nick's laboratory. Studies on the crosstalk between light, hormone signaling, and photomorphogenic responses in rice coleoptiles, led to the isolation of the *hebiba* mutant showing an altered growth response to red light. While wild type seedlings were shown to respond to red light irradiation with a transient increase of OPDA levels in the basal coleoptile region after 30 min and a significant increase of JA contents in that tissue, the *hebiba* mutant was detected to be impaired in this response. Neither light treatment nor wounding was able to induce JA formation in the mutant. Additional analysis of plant hormone contents in the *hebiba* mutant displayed neither a considerable impact on endogenous auxin nor on ABA contents, but an enhanced responsiveness to IAA that has been demonstrated in classical segment assays. Overall, the findings suggested a picture in which red light negatively affects the auxin responsiveness of growth through the light-triggered production of JA. Moreover, the comparison of JA and IAA contents in wild type and *hebiba* coleoptiles indirectly indicated a slight stimulation of auxin biosynthesis by jasmonates (Riemann et al. 2003). A more recent follow-up to the respective initial study on the *hebiba* mutant provided evidence for a role of jasmonate in the photodestruction of Phytochrome A (PhyA), whereby the sensitivity of phytochrome-governed growth responses to red and far-red light, e.g. the auxin responsiveness, may be affected (Riemann et al. 2009). A further example of auxin-oxylipin crosstalk came from the investigation of the role of different plant hormones in gravitropism of rice coleoptiles. It has been shown that IAA gradients necessary to trigger asymmetric growth of the coleoptiles are accompanied by reciprocally oriented JA gradients. Seemingly, the gradients develop independently from one another, which, in fact, refers to a co-regulation event rather than to crosstalk. The effect of the IAA gradient is further enhanced by a parallel gradient of either auxin responsiveness or sensitivity, or both, which could not be distinguished by the utilized split-coleoptile assay (Gutjahr et al. 2005). The molecular mechanism by which the additional gradient of IAA responsiveness is brought about and whether it can be associated with the opposing JA gradient is yet not entirely understood. However, the authors draw the attention to shared components of auxin and JA signaling pathways, in particular AXR1, which might reduce auxin responsiveness through the recruitment of such shared components by JA, thus making them a limiting factor for auxin-mediated growth responses. With regard to the formation of gravity induced IAA gradients in Arabidopsis, polar auxin transport mediated by PIN3 plays an essential role (Kleine-Vehn et al. 2010). Intriguingly, jasmonates have been shown to directly contribute to the regulation of directional IAA transport. It has been demonstrated that jasmonates are capable of inducing the expression of *PIN1* and *PIN2* (Sun et al. 2009), and to modulate PIN2 accumulation in the plasma membrane and its recycling via endocytosis in a concentration-dependent manner (Sun et al. 2011). However, it remains unclear if this mechanism

of molecular interaction can be generally attributed to all PIN proteins or whether it may be restricted to a sub-set of PIN family members.

By contrast, thus far, there is no evidence for directed transport of JA. Hence, although JA transport cannot be ruled out *per se*, JA gradients may originate from gradually increased local biosynthesis or by shifting the balance between JA breakdown and synthesis towards the latter. Derived from these observations the authors suggested two concepts: i) in the context of photomorphogenic growth responses of coleoptiles, red-light treatment induces the formation of JA, which in turn reduces the IAA responsiveness in basal regions of the coleoptile known to be the site where elongation growth occurs. Thereby, auxin-governed growth gets retarded and inhibited, respectively (**Figure 4A**). ii) With respect to gravitropic responses of coleoptiles they suggested a scenario in which growth is additionally inhibited by JA in places where IAA already only poorly promotes it (**Figure 4B**). In their model, the effect of JA is not a prerequisite and by itself also not sufficient to cause gravitropic bending, but JA is suggested to contribute to an amplification and acceleration, respectively, of the auxin-mediated response. Moreover, the JA gradient is associated with the establishment of a gradient of IAA responsiveness that is seemingly necessary to accelerate bending. Interestingly, the experiments also provided evidence for an OPDA gradient in the coleoptiles that paralleled the IAA gradient, perhaps underlining a differential and independent function of the JA precursor in the regulation of plant growth responses, which has been discussed in the literature before (Böttcher and Pollmann 2009).

A third model for the mechanism of IAA – JA interaction can be derived from the impact of JA on auxin formation and transport during lateral root formation. The experiments presented by Sun and co-workers (2009) showed that several auxin biosynthesis, metabolism, and transport related genes are differentially regulated by jasmonates. In addition, it has been reported that jasmonate can both influence *DR5* activity in root tips and increase lateral root density; the latter being a COI1-dependent effect that has shown to be impaired in the *jdl1*/*asa1-1* mutant (Sun et al., 2009; supplemental Figures 4 and 5). This suggests a model in which JA contributes to the fine-tuning of spatiotemporal IAA accumulation and auxin signaling, respectively, in root basal meristems that is required for lateral root formation (**Figure. 4C**). Likewise, the described impact of MeJA on *DR5* activity possibly indicates that jasmonates can impact lateral root formation on two levels. First, during priming of pericycle cells to become lateral root founder cells, which takes place in the apical meristem (De Rybel et al. 2010), and, secondly, during the initiation of lateral formation in the basal meristem (De Smet et al. 2007; Overvoorde et al. 2010). In any case, the underlying model constitutes a good example for indirect crosstalk of the signaling molecules in the defined

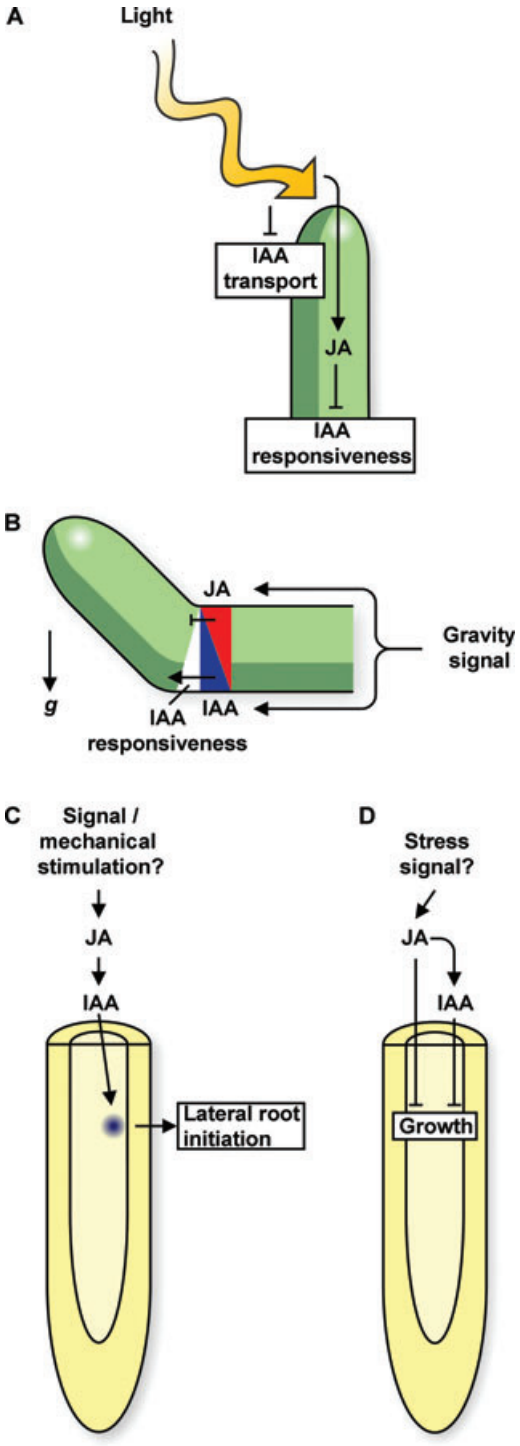


**Figure 4. Schematic representation of conceivable modes of interaction of IAA and JA signaling pathways.**

**(A)** The photomorphogenic inhibition of growth in aerially grown coleoptiles is based on the rapid inhibition of cell elongation growth, which goes in line with a block of basipetal auxin transport. Analysis of the *hebiba* mutant of rice, impaired in proper light response,

context of root system architecture control. It is tempting to speculate that the increase in JA levels is perhaps induced by mechanical stimulation, which has been reported to promote lateral root formation (Ditengou et al. 2008). The vital role of oxylipins, namely OPDA, as a signal transducer of *Bryonia dioica* and *Phaseolus vulgaris* mechanotransduction has been confirmed by a number of investigations (Falkenstein et al. 1991; Weiler et al. 1993; Stelmach et al. 1998; Blechert et al. 1999). However, JA-mediated regulation of auxin biosynthesis and transport in the context of lateral root initiation has to be strictly controlled to ensure perpetuation of the delicate balance between the two hormones. Excessive JA-triggered overproduction of IAA is likely to shift the outcome to its reverse, as a recent in-depth study reported substantial inhibition of lateral root initiation by elevated IAA contents (Ivanchenko et al. 2010).

Expanding this thought-provoking scenario in which the JA signal may cause a general and locally not defined auxin overproduction implies the translation into general growth repression and inhibition of lateral root formation. Having the dose-dependent effect of auxin, in particular in roots, in mind, a locally unrestricted flooding of the system by auxin may be able to amplify the intrinsic growth-inhibiting JA effect by adding a second layer of inhibition (**Figure 4D**). On the other hand, one has to bear in mind that strongly elevated JA concentrations may not directly affect auxin contents, as has been described above for the rice coleoptile system, but rather impact auxin sensitivity or responsiveness. Thus, differential concentration-dependent effects of JA may have to be considered in this connection. Our own data, which will soon be published elsewhere, provides several lines of evidence showing that root growth inhibition triggered by applied MeJA is not solely based on the effect of JA, but also relies partly on JA-induced auxin formation in the root (personal communication, Mathias Hentrich).

## Prospectives

It is an exciting time to study plant hormone crosstalk. Taking the steady increasing collection of plant mutants, the next generation genomic technologies, the greatly improved bioanalytical techniques, and the quickly developing amount and quality of datasets in publicly available databases into account, it seems to be just a matter of time until further major breakthroughs in deciphering hormonal crosstalk both during plant development and plant stress responses will be made. With respect to auxin-oxylipin crosstalk, this could involve available IAA- and JA-biosynthesis, metabolism, and response mutants in state-of-the-art "-omics" approaches combined with thorough cell biological and phenotypic analyses to tackle both the elucidation of direct and indirect crosstalk between the two signaling pathways. For instance, appropriate inducible gain-of-function mutants that facilitate tissue-specific expression of a phytohormone of interest might be valuable tools well suited to the task of disclosing gene regulatory circuits on a spatiotemporally defined basis in transcriptomics approaches on a genome-wide scale. In addition, metabolomics will likely be useful to gain deeper insight into alterations in plant hormone levels in selected biosynthesis mutants. At the same time, such experiments are likely to reveal even complex interactions of one plant hormone with several other biosynthetic pathways. These kinds of studies promise to yield intriguing new insights

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revealed enhanced IAA responsiveness but no considerably elevated IAA contents in the mutant. As a consequence, this suggests a scenario in which red light irradiation induces jasmonate production, which presumably represses the responsiveness to auxin.

**(B)** Gravitropism of rice coleoptiles involves the formation of IAA and JA gradients across the lateral axis of the coleoptile, oriented reciprocally to one another. Formation of these diametrically opposed gradients is suggested to occur independent of each other, initiated by gravity stimulation. The IAA gradient is paralleled by a gradient of IAA responsiveness. Being an example for co-regulation, the signaling pathways trigger distinct outcomes that assist each other in controlling gravitropic coleoptile bending. While the JA cascade presumably blocks cell division in the upper part of the coleoptile by inhibiting mitosis, the increased IAA contents in the lower part of the coleoptile promote cell elongation growth. However, an additional layer of direct interaction might also be conceivable in this context, as the JA gradient perhaps negatively affects IAA responsiveness by competing with auxin for shared signaling components.

**(C)** In Arabidopsis roots, JA and auxin signaling pathways crosstalk indirectly in initiating lateral root formation. JA has been shown to induce auxin biosynthesis through the up-regulation of a number of Trp and IAA biosynthesis genes. Mutation of one of those genes, *Anthranilate Synthase* $\alpha$*1* (*ASA1*), translates into impaired auxin formation and a decreased number of lateral roots. In the respective model, the signaling cascade is considered to trigger spatiotemporal restricted auxin biosynthesis, which subsequently results in lateral root initiation. The endogenous or exogenous cue responsible for launching that process is yet to be identified. Possibly, mechanical stimulation, such as root bending, plays a role in this context.

**(D)** Hypothesized that JA-mediated IAA production extends over wider areas of the root rather than being locally defined, this may result in presumably stress-induced general auxin overproduction. Together with the proven growth inhibitory effect of JA on root growth, and having in mind the dose-dependent effect of IAA, the increased auxin levels may cause further growth inhibition, as roots are recognized to be particularly sensitive to high IAA doses.

into one of the most important and complex fields of plant biology: the chemical-based control of plant growth and development. However, the ultimate goal must be the acquisition of a likewise flexible and robust networking model that helps to understand the sophisticated relationships between the various phytohormones in controlling plant growth and development. In order to be able to do so, one has to take on one major scientific challenge that concerns handling and processing of the vast amount of data generated by those type of cutting-edge '-omics' technologies. In particular, the heterogeneity of the data obtained demand the development of integrative, powerful and high quality bioinformatics solutions. Anyhow, practical applications addressing problems ranging from losses in plant biomass production and crop yield, due to salinization of available soils, to the negative effect of the climate change on plant growth will all rely, at least to some degree, on furthering our basic understanding of the interlinkage of plant hormone-mediated processes, hence, emphasizing the great and immediate significance of this field of research for the general public.

## Acknowledgements

The authors are grateful to all of the members of the Department of Plant Physiology at the Ruhr University Bochum (RUB), Germany and of the Centro de Biotecnología y Genómica de Plantas (UPM-INIA), Spain, who contributed in numerous stimulating discussions to this work. The work was supported by DFG grant PO1214/3-2 and SFB480/A10 to SP. MH was supported by a fellowship from the German National Academic Foundation and a membership of the Research School of the RUB.

Received 22 Mar. 2011 Accepted 10 May 2011

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(Co-Editor: Yunde Zhao)