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Review

Auxins in defense strategies

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Abstract: Plant hormones operate in a very complex network where they regulate and control different vital mechanisms. They coordinate growth, development and defense via signaling involving different interactions of molecules. Activation of molecules responsible for regulation of plant immunity is mainly provided by salicylic and jasmonic acid signaling pathways. Similar to the signaling of these defense-associated plant hormones, auxin can also affect resistance to different pathogen groups and disease is manifested indirectly through the effects on growth. The various ways in which auxin regulate growth and plant development and might be closely connected to plant defense, are discussed in this review.

Key words: auxin; defense responses; JA; SA; hormonal crosstalk

Introduction

Defense mechanisms manifested in plants with innate or induced resistance in relation to how the challenged plants may utilize various signaling pathways to restrict pathogen development and/or invasion strategies, will be discussed. One important approach to understand particular reaction to infection, is observing biomolecules that are biologically relevant to defense.

The efficiency of defense reactions in plants relates to both the host and pathogen and depends on a number of complicated mechanisms of molecular recognition principles and signal transduction pathways. There are three ways of plant-pathogen relationship associated with resistance. Firstly, plants are equipped to identify various pathogen-associated molecular patterns (PAMPs) via proteins expressed by cells of the innate immune system- pattern-recognition receptors (PRRs). Activation of this mechanism results in PAMPs-triggered immunity (PTI), which is sufficient to defend against nonpathogenic microorganisms only (Jones & Dangl 2006). Secondly, plants have developed genes of resistance to compete against pathogens inhibiting PTI by distributing virulence effectors proteins into host cells. These genes recognize specific pathogen effectors and trigger effectors immunity (ETI) (Jones & Dangl 2006). Downstream of these plant advisory systems activate appropriate defense responses through crosstalk between specific hormonal signaling pathways (Chung et al. 2008; Koornneef & Pieterse 2008; Qi et al. 2012).

Plants coordinate their growth, development, reproduction, defense and death via complex hormonal signaling. The plant hormones jasmonic acid, salicylic acid and ethylene are not only important signaling molecules, but also play a critical role in the regulation of plant immune responses in many cases. Furthermore, other plant hormones (also known as phytohormones), such as auxins, cytokinins, abscisic acid, gibberellins and brassinosteroids, primarily regulating cellular processes targeted on growth and development of plants, have been shown as crucial regulators of plant immunity (Denancé 2013). Interaction between 'defense hormones' and 'growth hormones' during plant defense have been at large overlooked so far. On the other hand, it is clear that physiological processes are regulated in a multiple way through their crosstalk (Munné-Bosch & Müller 2013).

This article summarizes recent studies about auxin and its role in plant defense. The auxin signaling pathway represents an essential piece of the complex network working against biotic and abiotic stress. Additionally, understanding the specific character auxin plays in plant defense can finally lead to a better understanding of the plant adaptation to stress conditions.

Role of auxin(s) in plant defense

Auxins, represented by IAA (indole-3-acetic acid), as the most abundant native auxin, are the group of signaling molecules that are necessary for almost any aspects of plant growth and development, such as phototropism, gravitropism and growth responses to the environment in general, apical dominance, formation,

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emergence and shape determination of root hairs, lateral roots, leafs, or flowers, generation and development of reproduction organs (Aloni et al. 2005; Pagnussat et al. 2009) or development of plant vasculature (reviewed in Tanaka et al. 2006; Petrášek & Friml 2009). Cell-to-cell auxin directional (polar) transport is crucial in building up the spatial auxin maxima and minima, also called auxin gradient (Friml & Palme 2002; Friml 2003; Zažímalová 2007). The contribution of this polar transport in creating the hormone gradients that control the above list of developmental processes has been found to have a role in plant defense, described later.

To generate biological response, auxin present in a cell must be firstly perceived by plant and transformed into a signal. The amount of auxin in any given cell is intricately regulated by complex of homeostatic mechanisms. There are known four receptor systems ensuring auxin response pathway in this signaling system.

The TRANSPORT INHIBITOR RESPONSE 1 (TIR1) family of F-box proteins (AFBs) (Dharmasiri et al. 2005; Kepinski & Leyser 2005), directly modulates gene expression in response to auxin. It acts as a part of the SCF-type E3 complex and can interact with a specific domain of transcriptional co-factors of the AUXIN/INDOLE-3-ACETIC ACID (AUX/IAA) family (Gray et al. 2001; Kepinski & Leyser 2004; Dharmasiri et al. 2005). This auxin mediated interaction triggers the ubiquitination of AUX/IAA repressors and their 26S proteasome-mediated degradation (Tiwari et al. 2003; Tan et al. 2007; Szemenyei et al. 2008). Consequently, AUXIN RESPONSE FACTORs (ARF) are released from repression and can activate transcription of auxin-responsive genes (Gray et al. 2001; Dharmasiri et al. 2005; Kepinski & Leyser 2005).

INDOLE-3-BUTYRIC ACID RESPONSE (IBR5) is another putative receptor promoting auxin responses, including auxin dependent gene expression. Genetic analyses of the double mutant *tir1 ibr5* showed that IBR5 regulates auxin responses differently than the TIR1-AUX/IAA pathway (Strader et al. 2008) as the dual specificity MAPK phosphatase by dephosphorylation of MPK12 – negative regulator of auxin signaling, which influences auxin-triggered fast increase of MAPK (mitogen-activating protein kinase) activity (Lee et al. 2009).

Very rapid auxin responses at plasma membrane utilize another auxin receptor – AUXIN BINDING PROTEIN 1 (ABP1) (Senn & Goldsmith 1988; Shishova & Lindberg 2004; Sauer & Kleine-Vehn 2011). ABP1 is associated to cell polarity-generating mechanism in which it activates Rho-GTPases involved in regulation of endocytosis and cytoskeleton reorganization (Xu et al. 2010). The loss of the single *Arabidopsis* ABP1 gene confers an embryo-lethal phenotype suggesting its function is cardinal during embryonic development (Chen et al. 2001). Recently Robert et al. (2010) have demonstrated that auxin binding to ABP1 at the plasma membrane inhibits clathrin dependent endocytosis, this affects also vesicle trafficking of PIN-FORMED (PIN) auxin efflux carriers. PIN proteins are polar localized transmembrane transporters acting in efflux of the auxin molecule from cells (Křeček et al. 2009; Depuydt & Hardtke 2011) as described below. Importantly, this leads to ABP1-dependent auxin level feedback regulation (Paciorek et al. 2005).

In the last case auxin binds directly and specifically to S-PHASE KINASE-ASSOCIATED PROTEIN 2A (SKP2A), the regulator of cell cycle, where positively regulates degradation of key transcription factors DPB and E2FC (Del Pozo et al. 2006; Jurado et al. 2010). High levels of auxin trigger degradation of SKP2A itself by unknown mechanism (Sauer et al. 2013).

Another level of complexity affecting auxin signaling is resulting from intricate management of auxin levels in every cell. That includes both regulation of transport in and out from the cell and metabolic regulation to and from its active forms (reviewed in Rosquete et al., 2012).

It has been reported that upon pathogen infections the endogenous IAA level significantly increases and expression of some auxin-regulated genes is induced. Auxin transiently causes transcription of three primary gene families: SMALL AUXIN UP RNA (SAUR), AUX/IAA and GRETCHEN HAGEN 3 (GH3) (Guilfoyle 1999; Hagen & Guilfoyle 2002).

SAUR genes were originally identified in auxin-treated soybean elongating hypocotyl sections (Mcclure & Guilfoyle 1987). Members of this class were also isolated from mung bean (Yamamoto et al. 1992), *Arabidopsis* (Gil et al. 1994), tobacco (Roux et al. 1998), maize (Knauss et al. 2003) and rice (Jain et al. 2006). *Arabidopsis* genome comprises of over 70 SAUR genes from which 58 members were identified in rice with no intron in coding sequences (Hagen & Guilfoyle 2002; Jain et al. 2006). In addition, the connection between Ca^{2+}/calmodulin second messenger system and auxin signaling was demonstrated by calcium-dependent *in vitro* binding of SAUR proteins with calmodulin (Yang & Poovaiah 2000; Knauss et al. 2003). However, there was no report relevant to involvement of SAUR in plant immune responses till the last year. The study of chickpea SAUR gene *CaSAUR1* in response to *Fusarium* wilt provides basic genomic information about its role in plant immunity. Expression pattern showed higher level of *CaSAURl* expression in resistant genotype, suggesting its function in defense against necrotrophs (Nasheeman 2013).

AUX/IAA proteins, characterized as repressors of auxin response, appear to be common targets for pathogen strategy. In *Arabidopsis*, yokonolide B, a spiroketal-macrolide isolated from *Streptomyces diastatochromogenes*, inhibits the expression of auxin-responsive genes by blocking AUX/IAA protein degradation (Hayashi et al. 2003), see above.

In *Arabidopsis* plants, large number of auxin-dependent genes is transcriptionaly reprogrammed under the influence of tobacco mosaic virus infection. This is accomplished through the interaction between the tobacco mosaic virus replicase protein and the *Arabidopsis* AUX/IAA26 protein, which prevent the localization

of AUX/IAA to the nucleus (Padmanabhan et al. 2008) and its function there. Also in *Arabidopsis,* it has been shown that virulence protein AvRpt2 of *Pseudomonas syringae* supports auxin response by modulating stability of the negative regulator IAA7 and IAA17 proteins of the AUX/IAA family (Chen et al. 2007; Cui et al. 2013).

Another group of auxin upregulated genes represented by GH3 family of acyl-acid-amido synthetases plays important role in catalysis of conjugation reaction in a number of plants species (Terol et al. 2006). These crucial regulators of auxin homeostasis are involved in many pathogen-plant related interactions. These enzymes are involved in adenylation and transferase activities with the aim to conjugate acyl acid groups of various plant hormones to amino acid (Westfall et al. 2012). For example, *Arabidopsis thaliana* and rice encode 19 and 13 GH3 proteins, respectively (Hagen & Guilfoyle 1985; Westfall et al. 2010; Okrent & Wildermuth 2011). Each of GH3 genes encode proteins with estimated molecular masses of 65–70 kDa. Plant GH3 proteins can be divided into three major classes, identified as Groups I, II, and III (Staswick et al. 2002; Felten et al. 2009). In *Arabidopsis*, the group I consists of two proteins, AtGH3.11 (JAR1), which is involved in adenylation of jasmonic acid (JA) *in vitro* and displayed JA-amino synthetase activity, and AtGH3.10 (DFL2) (Staswick & Tiryaki 2004). The group II contains most of the members, including AtGH3.2 (YDK), AtGH3.5 (AtGH3a), AtGH3.6 (DFL1), and AtGH3.17, which adenylate IAA and subsequently catalyzes its conjugation to amino acids through amide bonds (Staswick et al. 2002, 2005). In addition, AtGH3.6, AtGH3.5 and AtGH3.17 are possible targets of the ARF8 auxin response factor (Tian et al. 2004).

GH3 genes also show different responses to diverse types of stimulus (Okrent & Wildermuth 2011). *Arabidopsis* mutants of the *AtGH3.12/PBS3* gene display increased disease susceptibility to virulent and avirulent forms of the pathogen *Pseudomonas syringae* (Nobuta et al. 2007). Overexpression of GH3.12 results in a SA-related (dwarf) phenotype along with other traits indicate alternation in auxin signaling (Ding et al. 2008; Zhang et al. 2009; Takase et al. 2004; Nakazawa et al. 2001; Park et al. 2007; Khan & Stone 2007; Westfall et al. 2012). Moreover, while overexpression of*AtGH3.5* increases level of IAA accumulation (Zhang et al. 2007), the *gain-of-function* mutant of *AtGH3.5* also elevates SA accumulation and increases expression of PATHOGENESIS-RELATED PROTEIN 1 (PR-1) in response to biotic stress.

While experiments with above mutants demonstrate that amino acid conjugates of SA by auxin upregulated GH3 proteins can act as a bioactive trigger of plant pathogen defense responses (Park et al. 2007).

It has also been confirmed, that auxin increases disease development generally (Zhang et al. 2007; Fu et al. 2011). In rice activation of *OsGH3.2* caused auxin-deficient morphological phenotypes and conferred broad-spectrum resistance against phytopathogens. Overexpression of *OsGH3.1* and *OsGH3.8* results also in auxin content decrease and inhibition of plant growth and development, abnormal plant morphology, and enhanced pathogen resistance with activation of defense-related genes (Ding et al. 2008; Domingo et al. 2009). In general, GH3 proteins seem to be important mediators in defense mechanisms via conversion of IAA to its amide metabolites, to attenuate the auxin-underlined developmental program and to give defense mechanisms priority instead.

Auxins can influence regulation of pathogen resistance responses in plants on both direct and indirect ways. Indirect effects, because of development-associated processes of auxins, regulate cell wall architecture, root morphology, and stomata pattern, and also lead to cell wall loosening in treatment of rice with IAA disturbed the resistance to *Xanthomonas oryzae* (Ding et al. 2008; Kazan & Manners 2009; Denancé et al. 2013). On the other hand, plant immunity is directly triggered through the PTI machinery, which represses auxin signaling (Yang et al. 2013). Then auxin itself negatively regulates plant defense by interfering with other hormone signaling pathways (Robert-Seilaniantz et al. 2011a; Yang et al. 2013). The PAMPs such as bacterial flagellin-peptide flg22 induces an *Arabidopsis* microRNA (miR393) receptors, which leads to the auxin receptor genes (TIR1, AFB2, and AFB3) be targeted for cleavage, resulting in the suppression of auxin signaling and increased resistance to the bacterium *Pseudomonas syringae* (Navarro et al. 2006; Robert-Seilaniantz et al. 2011b). The flg22-induced resistance to this biotrophic pathogen was interpreted by the antagonism of auxin signaling to SA-dependent defenses, for details see later. To prove the hypothesis, treatment of *Arabidopsis* leaves with flg22 was found to induce SA accumulation (Tsuda et al. 2008). Although overexpression of miR393 causes significant resistance of plants to biotrophs, it shows increased susceptibility to necrotrophs, suggesting that suppression of auxin signaling also affects SA-JA cross talk. Furthermore, inhibition of auxin signaling by miR393 redirects the metabolic flow of the tryptophan metabolic pathway, which is responsible for auxins and antimicrobial indole glucosinolates and camalexin synthesis. As a result, auxin-suppressed plants produce greater amount of indole glucosinolates, which are implicated in biotrophic resistance, while the production of camalexin, which is more effective against necrotrophic fungi, is reduced (Robert-Seilaniantz et al. 2011b).

Jasmonic acid (JA) and its derivatives

JA and its structurally related metabolites are lipid-derived compounds from the enzymatic oxygenation of 18 and 16-carbon tri-unsaturated fatty acids (Wasternack & Kombrink 2010) and can also be synthesized rapidly via the oxylipin biosynthesis pathway upon pathogen or insect attack (Gfeller et al. 2010). The best known jasmonates are jasmonic acid (JA) and its derivates methyl jasmonate (MeJA) and jasmonoyl

isoleucine (JA-Ile) (Acosta & Farmer 2010). After synthesis, JA can be metabolized by the JA carboxyl methyltransferase (JMT) to MeJA (Seo et al. 2001). It's conjugation via JA conjugate synthase (JAR1) to amino acids isoleucine (Ile) results in a biologically highly active enantiomer JA-Ile (Staswick & Tiryaki 2004; Fonseca et al. 2009). JA signaling regulates many aspects of growth development, as well as abiotic and biotic stresses, especially defense to herbivores and necrotrophic pathogens (Browse 2009).

JA and auxin signaling share many commonalities and interact positively in the most instances. Similarly to auxin signaling, JA signaling is known to be generally antagonistic to SA signaling (Kazan & Manners 2008).

In the absence of the JA-Ile, JASMONATE ZIM DOMAIN (JAZ) proteins, similarly to IAA/AUXs, act as transcriptional repressors of JA signaling by binding to positive transcriptional regulators – the basic helix-loop-helix leucine zipper proteins MYC TFs 2, 3, and 4 (Chini et al. 2007; Fernandez-Calvo et al. 2011; Niu et al. 2011), in paralel to ARFs. Repressor function is performed by ZIM domain of JAZs proteins (Vanholme et al. 2007). JAZ interacts through ETHYLENE RESPONSE FACTOR (ERF)-associated amphiphilic repression (EAR) motif with adaptor protein NOVEL INTERACTOR OF JAZ (NINJA). This adaptor protein is necessary for recruitment of the corepressor TOPLESS (TPL) and the resulting complex has to be bound to MYC transcription factors to repress the JA pathway (Szemenyei et al. 2008; Pauwels et al. 2010). Similarity in the signaling pathway to auxin continues on the hormone reception level, with SCF-type E3 ubiquitin ligase with a specific F-box protein for jasmonate as a receptor-CORONATINE INSENSITIVE 1 (COI1) (Dharmasiri et al. 2005). SCF-COI1 in presence of JA-Ile complex together with JAZs, which are targeted to ubiquitinylation and subsequent degradation of JAZ proteins via the proteasome (Sheard et al. 2010; Yang et al. 2009; Pauwels & Goossens 2011), which consequently release jasmonate responsive gene expression from repression.

JA-auxin crosstalk has been reported to occur through the signaling components and interaction of auxin- and JA-related regulators of gene expression. The best known proteins to modulate homeostasis of auxin and JA are GH3 proteins (Hagen et al. 1984; Hagen & Guilfoyle 1985). The group I of GH3 proteins (JAR1/GH3.11) conjugates JA to Ile (Staswick & Tiryaki 2004; Staswick 2009). GH3.9 belongs to the group II which can conjugate IAA with amino acid and negatively influence primary root growth. Mutation in *GH3.9* is reason to increased auxin sensitivity and primary root length and this change also altered IAA and MeJA mediated inhibition of root growth (Khan & Stone 2007). It was found that auxin-mediated adventious root initiation is aided by reduction of JA level through GH3.3, GH3.5, GH3.6 (Gutierrez et al. 2012). JA signaling can also regulate biosynthesis of auxin by MYC2, which negatively influences secondary metabolism of thryptophan (Trp) (Dombrecht et al. 2007; Acosta & Farmer 2010). MYC2 transcription factors are responsible for root stem cell activity by binding to the promoters of PLETHORA 1 (PLT1) and PLT2 and inhibit their expression (Galinha et al. 2007; Dhonukshe et al. 2012; Qi et al. 2012; Chen & Baluška 2013).

Interaction in the level of signaling components shows that auxin-inducible expression of JAZ1/TIFY10A is controlled by IAA-ARFs and does not depend on the JA signaling pathway (Grunewald et al. 2009; Chen & Baluška 2013; Cuéllar Pérez et al. 2014). On the other side JA mediates the expression of some of the auxin biosynthetic genes, such as ANTHRANILATE SYNTHASE A1 (ASA1) and two members of the YUCCA family (YUC8/YUC9) (Dombrecht et al. 2007; Sun et al. 2009; Hentrich et al. 2013).

Investigation of intracellular trafficking of PIN2 showed that low concentration of JA had inhibitory effect on PIN2 endocytosis through ASA1-dependent auxin biosynthesis and SCFTIR1/AFBs-dependent auxin signaling. Characterization of the *Arabidopsis asa1-1* mutant, demonstrated negative regulation of auxin transport through the reduction of PIN1 and PIN2 protein levels in the plasma membrane. On the other hand, higher concentration of JA down-regulates levels of PIN2 protein in the plasma membrane through a various mechanism, independently of the auxin pathway. This subcellular management of PIN2 distribution and abundance induces redistribution of lateral auxin during the root gravitropic response (Sun et al. 2011).

Both ASA1 and the YUCCA genes affect IAA levels, but ASA1 might play a more general role and act on the formation of L-Trp, a primary building block in protein synthesis. On the contrary, study of YUCCA genes shows a very tight spatio-temporal regulation of their expression (Zhao 2008). This characteristic expression pattern could be the reason why mutation in YUC8 and YUC9 cause changes in inhibitory effect of MeJA on primary root elongation. MeJA induces the overexpression of YUC9/YUC8 and increased YUC levels cause transient IAA overproduction (Hentrich et al. 2013). Thereafter, elevated levels of IAA result in altered lateral root development. Overexpression of YUC assists in inhibition of primary root elongation and shows that both genes participate in the induction of the auxin synthesis by MeJA (Hentrich et al. 2013).

The dominant role of JA pathway is regulation of resistance to necrotrophic pathogens. *Alternaria brassicicola* infection activates the transcription of auxin biosynthetic genes and therefore elevates synthesis of auxin in host plants. Infection then reduces auxin transport capacities by reduction of PIN protein levels. These effects together, lead to an increased auxin response in the host plants (Glazebrook 2005; Qi et al. 2012).

Emerging evidence by the use of mutants and accordingly by the application of auxin transport inhibitors indicates that the inhibition of auxin transport also differentially affects resistance to different pathogen groups. Disruption of natural auxin trans-

port by auxin transport inhibitor 2,3,5-triiodobenzoic acid (TIBA) prior to inoculation by the leaf-infecting necrotroph *Plectosphaerella cucumerina* and root-infecting hemibiotrophic pathogen *Fusarium oxysporum* increases the susceptibility of plants to this pathogens (Davis et al. 1953; Llorente et al. 2008). In wild-type *Arabidopsis*, TIBA treatment leads to increased expression of PR1 protein what is in concert with a proposed negative cross-talk among SA and both the auxin and JA pathways (Thomma et al. 1998; Wang et al. 2007; Llorente et al. 2008). In contrast, pathogen-dependent expression of plant defensin PDF1.2, a marker gene for the JA-induced pathway, was suppressed by TIBA. Negative effects of TIBA on JA-responsive defense gene expression provide an explanation why TIBA-treated plants show an increased susceptibility to necrotrophic pathogen. Those data suggest role of JA dependent pathway in the regulation of auxin transport affecting both development and defense responses and that undisrupted auxin flow seems to be important in resistance to necrotrophic pathogens.

Salicylic acid (SA) signaling

While JA may positively modulate the auxin pathway in plant resistance to necrotrophic pathogens, well-recognized antagonistic crosstalk between SA and auxin during plant resistance is used against biotrophic pathogens (Kazan & Manners 2009; Qi et al. 2012). The repression of SA pathways by auxin is used to enhance susceptibility of the host using pathogen-associated molecular patterns: miR393 and flg22 (Navarro et al. 2006; Chen et al. 2007; Robert-Seilaniantz et al. 2011b).

The key role in plant immunity against microbial infections belongs to SA and its detection via different ligand/receptor-binding methods (Gimenez-Ibanez & Solano 2013). SA induces defense-related genes by NON-EXPRESSOR OF PR GENES 1 (NPR1) receptor or NPR1-related proteins in *Arabidopsis* (Fu et al. 2012; Wu et al. 2012). NPR1-LIKE PROTEIN 3 (NPR3) and NPR1-LIKE PROTEIN 4 (NPR4; NPR1-related proteins) seem to act as Cullin 3 (CUL3) adaptors for the degradation of NPR1 via 26S-proteasome. Yeast two hybrid system and pull-down assays show that interaction between NPR1 and NPR3 occurs only in the presence of SA. On the other hand, NPR4-NPR1 interaction occurs in the deficiency of SA (Fu et al. 2012; Gimenez-Ibanez & Solano 2013). NPR1 is originally localized at the cytosol as an oligomer, and only in the presence of SA, dissociation of NPR1 complex as a result of concurrent redox changes leads to the translocation of the corresponding monomers to the nucleus. There, NPR1 protein activates the transcription of defensive genes, such as *pathogen related proteins* (PR) by interacting with TGACG MOTIF-BINDING FACTOR (TGA) transcription factors (Dong 2004; Wang et al. 2006; Tada et al. 2008; Robert-Seilaniantz et al. 2011a). *PR* genes include various groups, but few encode proteins with antimicrobial activity (van Loon et al. 2006). Transcription factors responsible for activation or inhibition of SA defense responses are WRKY factors, which underline their role in both SA-mediated resistance and feedback control of its signaling pathways (Wang et al. 2006; Rushton et al. 2010).

It is known, that SA can down-regulate a number of auxin-related genes at the molecular level. Treatment of *Arabidopsis* plants with SA functional analog, benzothiadiazole S-methyl ester (BTH) resulted in the repression of auxin responsive genes including an auxin influx carrier AUX1, an auxin efflux carrier PIN7, auxin receptors TIR1 and AFB1, and genes belonging to auxin inducible SAUR and AUX/IAA family (Wang et al. 2007). SA increases level of AUX/IAA repressors primarily by limiting auxin receptors TIR1/AFB1, which are needed for degradation of AUX/IAA proteins (Wang et al. 2007; Bari & Jones 2009). In addition, it was found that a vast majority of the above genes highly induced by auxin was also repressed in systemic tissues after stimulation of *systemic acquired resistance* (SAR). In the microarray assays for NPR1 direct transcriptional targets, many of these genes had low signal levels, suggesting that these auxin-pathway genes are likely to be indirectly regulated by NPR1. In addition, SA has been shown to inhibit expression of auxin inducible reporter DR5::*β-glucuronidase* (GUS). This result was confirmed with *in situ* staining of DR5 reporter in roots, where its promoter is the most active (Wang et al. 2007).

Studies on *Arabidopsis* activation-tagged mutant, *bud1* (BUSHY DWARF 1), in which the expression of the *MAP kinase kinase 7* (*AtMKK7*) gene is increased showed the antagonism of SA and auxin in defense responses. The *bud1* mutant plants accumulate elevated levels of SA and display constitutive *pathogenesis-related* (*PR*) gene expression and enhanced resistance to pathogens *Pseudomonas syringae* and *Hyaloperonospora parasitica* (Zhang et al. 2007). The increased expression of *AtMKK7* in *bud1* causes deficiency in polar auxin transport (PAT) with significant reduction in free auxin (IAA) levels in the mutant plants (Dai et al. 2006; Zhang et al. 2008) indicating that AtMKK7 negatively regulates auxin signaling. Given that SA is a positive regulator of defense responses, whereas auxin is likely a negative regulator of defense responses.

The relationship between auxin levels and auxin transport is complicated and so is the relationship between SA and auxin transport, as might be illustrated by the works above. Auxin transport generates characteristic patterns of patches with high and low auxin levels within the plant body, which determines future growth. If normal auxin transport is compromised it ends up in unnatural concentrations of auxin in different position and it seems that effectivity of SA signaling and resistance to biotrophic pathogens is determined directly by auxin levels in any of the cell undergoing defense response rather than the transport process.

Summary

The integration of multiple signaling pathways of phy-

tohormone responses determines how plants develop, grow and react on different environmental stimuli. However, the perception of small molecule signals by receptors is only one piece of these complex biological tasks. 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 is causative for correct function of this cellular machinery. Recent efforts summarized in the review provide new molecular insights into how large enzyme families catalyze similar types of modifications on chemically diverse plant growth regulators and pathogen-released molecules to alter their biological functions. The effects described above suggest role of JA dependent pathway in the regulation of auxin transport affecting both development and defense responses and undisrupted auxin flow seems to be important in resistance to necrotrophic pathogens. Further, we consider the activation of auxin signaling as part of SA mediated disease resistance mechanism. Changes in auxin homeostasis seem to influence SA signaling result in resistance to biotrophic pathogens.

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