Novelty Journals

ISSN 2394-966X

International Journal of Novel Research in Life Sciences
Vol. 7, Issue 1, pp: (64-77), Month: January - February 2020, Available at: www.noveltyjournals.com

THE ROLE OF PLANT HORMONES IN PLANT DISEASE DEFENSE RESPONSES: A REVIEW

Worku Abebe

Ethiopian Institute of Agricultural Research, Holleta Agricultural Research Center (HARC), P.O. Box 2003, Holeta, Ethiopia

E-mail: workuabebe2005@gmail.comTel: +251913317619

Abstract: **In addition to regulating growth processes, Plant hormones play important roles in signalling networks involved in plant responses to a wide range of biotic and abiotic stresses. Plants are able to protect themselves against the attack of microbial pathogens via their disease defense mechanisms. Plant defense mechanisms are usually complex and composed of multiple layers of defense that are effective against diverse array of pathogens. Of these defense mechanisms, plant hormones are the major ones. Salicylic acid (SA), jasmonates (JA) and ethylene (ET) are well known to play crucial roles in plant disease and pest resistance. However, the roles of other hormones such as abscisic acid (ABA), auxin, gibberellin (GA), cytokinin (CK) and brassinosteroid (BL) in plant defence are less known. The objective of this study was to review recent knowledge on the role of plant hormones in plant disease defense response. In this paper, the roles of Auxins, Gibberellins (GA), Abscisic acid (ABA), Cytokinins (CK), Salicylic acid (SA), Ethylene (ET), Jasmonates (JA), Brassinosteroids (BR) and Peptide hormones have been thoroughly reviewed.**

Keywords: **Disease, Defense Response, Disease, Plant, plant Hormones, Signaling Networks.**

1. INTRODUCTION

In their natural environment, plants encounter a vast array of pathogenic microorganisms such as fungi, bacteria, viruses and nematodes. These diverse pathogens deliver effecter molecules (virulence factors) into the plant cell to promote virulence and cause disease. Despite the presence of a large number of microorganisms in the surroundings of plants, few microorganisms are able to attack any particular plant species, because they are able to protect themselves against the attack of microbial pathogens through their disease defense mechanisms. Plant defense mechanisms are usually complex and composed of multiple layers of defense that are effective against diverse array of pathogens. Plants utilize preformed physical and chemical barriers such as waxy cuticle, the plant cell wall and phenolics that hinder pathogen entry and infection. In addition, plants have evolved a wide variety of inducible defense mechanisms that are triggered upon pathogen recognition. These inducible defenses include multifaceted molecular, biochemical, and morphological changes, such as oxidative burst, expression of defense-related genes, production of antimicrobial compounds, and/or programmed cell death (van Loon *et al*. 2006). Plants defend themselves against most potential microbial pathogens through a basal defence mechanism. The current view of the plant immune system has been represented by a model in which the perception of pathogen-associated molecular patterns (PAMPs) by host encoded pattern recognition receptors (PRRs) results in PAMP triggered immunity (PTI). Successful pathogens secrete effectors that suppress PTI and thus induce disease, resulting in effector triggered susceptibility (ETS). As a counter defense strategy, in which some of the defense mechanisms are mediated or activated by plant hormones such as Salicylic Acid, Jasmonic Acid, Ethylene, etc., Plants recognize a given effector either directly or indirectly and activate effector-triggered immunity (ETI) resulting in disease resistance (Chisholm *et al.* 2006; Jones and Dangl 2006). Here, the plant hormones play a role as signaling molecules

through which inducible defense mechanisms are activated. The activation of PTI or ETI enhances plant disease resistance and restricts pathogen growth. Hence, the timely recognition of an invading microorganism coupled with the rapid and effective induction of defense responses appears to make a key difference between resistance and susceptibility. This is to say, rapid recognition of the pathogen and induction of defense response by the invaded host, result into resistance reaction while the reverse host-pathogen interaction result into susceptibility reaction of the host plant.

2. OBJECTIVE

To review recent knowledge on the role of plant hormones in plant disease defense response.

3. THE ROLE OF PLANT HORMONES IN PLANT DISEASE DEFENSE RESPONSES

Plants produce a wide variety of hormones, which include Auxins, Gibberellins (GA), Abscisic acid (ABA), Cytokinins (CK), Salicylic acid (SA), Ethylene (ET), Jasmonates (JA), Brassinosteroids (BR) and Peptide hormones. Recently, Strigolactones are identified as a new class of plant hormones (Gomez-Roldan *et al*. 2008; Umehara *et al.* 2008). They play important roles in diverse growth and developmental processes as well as various biotic and aboitic stress responses in plants. Infection of plants with diverse pathogens results in changes in the level of various phytohormones (Adie *et al.* 2007; Robert-Seilaniantz *et al.* 2007). The identification and characterization of several mutants affected in the biosynthesis, perception and signal transduction of these hormones has been instrumental in understanding the role of individual components of each hormone signaling pathway in plant defense response. Substantial progress has been made in understanding individual aspects of phytohormones perception, signal transduction and influence on gene expression. However, the underlying of molecular mechanisms by which plants integrate stress induced changes in hormone levels and initiate adaptive responses are poorly understood. In counteract strategy; microbial pathogens have also developed the ability to manipulate the defense-related regulatory network of plants by producing phytohormones or their functional mimics. This results in hormonal imbalance and activation of inappropriate defense responses (Robert-Seilaniantz *et al.* 2007). For example, production of coronatine, a jasmonic acid mimic by Pseudomonas syringae pv. tomato (Pst) bacteria, triggers the activation of JA-dependent defense responses leading to the suppression of SA-dependent defense responses and promotion of disease symptoms (Cui *et al*. 2005; Laurie-Berry *et al.* 2006). In addition, coronatine has been shown to prevent pathogen-induced stomata closure which facilitates bacterial entry into the leaf (Melotto *et al.* 2006). However, there is still a limited knowledge on complex regulatory networks where multiple hormonal pathways interact and influence plant defense responses, eventhough major recent advances were made in the identification of different hormonal components involved in defense responses of plants against various pests and diseases.

3.1. The Role of Salicylic Acid (SA), Jasmonic Acid (JA) and Ethylene (ET) in Plant Disease Defense response

Three phytohormones, SA, JA and ET, are known to play major roles in regulating plant defense responses against various pathogens, pests and abiotic stresses such as wounding and exposure to ozone (Glazebrook 2005; Lorenzo and Solano 2005; Broekaert *et al.* 2006; Loake and Grant 2007; Balbi and Devoto 2007). On the basis of the interactions that have been studied, a general rule for hormonal action has been proposed in which resistant responses to biotrophs and Hemibiotrophs require SA, whereas responses to necrotrophs require JA and Ethylene (Feys and Parker, 2000). In other words, SA plays a crucial role in plant defense and is generally involved in the activation of defense responses against biotrophic and hemi-biotrophic pathogens as well as the establishment of systemic acquired resistance (SAR) (Grant and Lamb 2006). For example, Mutants that are insensitive to SA showed enhanced susceptibility to biotrophic and hemi-biotrophic pathogens. Recently, it has been shown that, methyl salicylate, which is induced upon pathogen infection, acts as a mobile inducer of Systemic Aquired Resistance (SAR) in tobacco (Park *et al*. 2007). SA levels increase in pathogen-challenged tissues of plants and exogenous applications result in the induction of pathogenesis related (PR) genes and enhanced resistance to a broad range of pathogens.

By contrast, JA and ET are usually associated with defense against necrotrophic pathogens and herbivorous insects. This suggests that the defense signaling network activated and utilized by the plant is dependent on the lifestyles of the pathogen (Adie *et al*. 2007). Although, SA and JA/ET defense pathways are mutually antagonistic, evidences of synergistic interactions have also been reported (Schenk *et al*. 2000; Kunkel and Brooks 2002; Beckers and Spoel 2006; Mur *et al*. 2006).

Eventhough JAs are involved in diverse processes such as seed germination, root growth, tuber formation, tendril coiling, fruit ripening, leaf senescence and stomatal opening, they play crucial roles in plant defense responses against insects and microbial pathogens. Several studies have demonstrated that concentrations of JA increase locally in response to pathogen infection or tissue damage and exogenous application of JA induced the expression of defense-related genes (Lorenzo and Solano 2005;Wasternack 2007). Over the past decade, several mutants affected in JA signal perception and transduction have been isolated and characterised. Three main JA-signaling components include: coronatine insensitive 1 (COI1), jasmonate resistant 1 (JAR1) and Jasmonate insensitive (JIN1). COI1 encodes protein involved in the protein degradation by the proteasome and is required for most JA-mediated responses (Xie *et al.* 1998). JAR1 encodes a JA amino acid synthetase involved in the conjugation of isoleucine to JA (JA-Ile) which is considered to be the bioactive JA molecule perceived by plants (Staswick and Tiryaki 2004; Thines *et al.* 2007). JIN1 encodes a transcription factor involved in the transcriptional regulation of some JA responsive gene expression (Lorenzo *et al.* 2004).

Biotic stress results in changes in different phytohormones levels. Alterations in plant hormone levels results in the changes in the expression of defense related genes and activation of defense responses. In the above diagram, a plus sign (+) indicates positive interaction between the plant hormones whereas a minus (-) sign indicates negative interaction between them.

The recent discovery of jasmonate ZIM-domain (JAZ) proteins has advanced the understanding of the molecular mechanisms of JA signaling in plants. It has been reported that COI1 or COI1-JAZ complex acts as a receptor for JA-Ile in Arabidopsis (Katsir *et al.* 2008). JAZ proteins are repressors of JA signaling which have been shown (JAZ1 and JAZ3) to interact with JIN1 and inhibit the expression of JA-responsive genes. JA promotes interaction between JAZ proteins and the COI1 ubiquitin ligase, leading to the ubiquitination and subsequent degradation of JAZ proteins by the proteasome. The degradation of JAZ proteins allows transcription factors to activate the expression of JA-responsive genes (Chini *et al.* 2007; Thines *et al.* 2007).

Recently, JA signaling has been implicated in the long-distance information transmission leading to systemic immunity in Arabidopsis (Truman *et al.* 2007). Rapid accumulation of JA in phloem exudates of leaves challenged with an avirulent strain of Pst and increased accumulation of JA biosynthetic gene transcripts as well as JA levels in systemic leaves suggests that JA could act as a mobile signal in Arabidopsis pathogen immunity (Truman *et al.* 2007). JA signaling plays a prominent role in promoting plant defense responses to many herbivores including caterpillars, beetles, thrips, leafhoppers, spider mites, fungal gnats and mired bugs (Browse and Howe 2008). For example, JA signaling is activated in response to attack by insects. However, not all herbivores activate JA signaling in plants. For example, the silverleaf whitefly *Bemisia tabaci* activates SA signaling and suppresses JA signaling in Arabidopsis (Kempema *et al.* 2007) indicating that SA and other hormones are also important for the resistance of plants against some herbivores. However, compared to JAs, the contribution of other phytohormones to host resistance against herbivores appears to be relatively minor (Bodenhausen and Reymond 2007; Koornneef and Pieterse 2008; Zheng and Dicke 2008). Treatment of plants with JA results in enhanced resistance to herbivore challenge (Howe and Jander 2008). These results indicate that JA plays a dominant and conserved role in plant resistance to herbivore attack.

3.2. Interaction between Defense Signaling Pathways

Interaction between defense signaling pathways is an important mechanism for regulating defense responses against various types of pathogens. In the recent years, several components regulating the cross-talk between SA, JA and ET pathways have been identified. Some of the important components mediating the crosstalk between defense signaling pathways are described below.

3.2.1. Interactions between SA, JA and ET signaling pathways

3.2.1.1. SA and JA interaction

One of the important regulatory components of SA signaling is non-expressor of PR genes 1 (NPR1), which interacts with transcription factors that are involved in the activation of SA-responsive PR genes (Dong, 2004). Arabidopsis npr1 plants are compromised in the SA-mediated suppression of JA responsive gene expression indicating that NPR1 plays an important role in SA-JA interaction (Spoel *et al.* 2007). Downstream of NPR1 and several SA transcription factors play important roles in the regulation of SA-dependent defense responses in plants (Wang *et al.* 2006; Eulgem and Somssich

2007). The Arabidopsis transcriptional factor/gene (WRKY70) has been found to regulate the antagonistic interaction between SA-and JA-mediated defenses. Overexpression of WRKY70 resulted in the constitutive expression of SA-responsive PR genes and enhanced resistance to the biotrophic pathogen *Erysiphe cichoracearum* but repressed the expression of JA-responsive marker gene PDF1.2 and compromised resistance to the necrotrophic pathogen *Alternaria brassicicola (A. brassicicola)* (Li *et al*. 2004, 2006).

In contrast, suppression of WRKY70 gene expression caused an increase in PDF1.2 transcript levels and enhanced resistance to *A. brassicicola* (Li *et al*. 2006). These results suggest that WRKY70 gene acts as a positive regulator of SA-dependent defenses and a negative regulator of JA-dependent defenses and plays a pivotal role in determining the balance between these two pathways. Recently, WRKY62 has been reported to be induced by JA and SA synergistically. In addition, the analysis of loss and gain of function mutants in Arabidopsis plants revealed that WRKY62 gene downregulates JA-responsive LOX2 and VSP2 genes. These results suggest potential involvement of WRKY62 in the SA-mediated suppression of JA-responsive defense in Arabidopsis (Mao *et al.* 2007).

Mitogen activated protein kinase 4 (MPK4) has been identified as another key component involved in mediating the antagonism between SA-and JA-mediated signaling in Arabidopsis. The Arabidopsis mpk4 mutants show elevated SA levels, constitutive expression of SA responsive PR genes and increased resistance to Pst. In contrast, the expression of JA responsive genes and the resistance to A. brassicicola were found to be impaired in mpk4 mutants (Petersen *et al*. 2000; Brodersen *et al*. 2006). These results indicate that MPK4 acts as a negative regulator of SA signaling and positive regulator of JA signaling in Arabidopsis.

Another important regulator identified to affect antagonism between SA and JA mediated signaling is a glutaredoxin, GRX480. Glutaredoxins are disulfide reductases which catalyze thiol disulfide reductions and are involved in the redox regulation of protein activities involved in a variety of cellular processes (Meyer *et al*. 2008). Recently, GRX480 has been shown to interact with TGA transcription factors involved in the regulation of SA responsive PR genes (Ndamukong *et al.* 2007). The expression of GRX480 is induced by SA and requires TGA transcription factors and NPR1. Furthermore, the expression of JA responsive PDF1.2 gene was inhibited by GRX480 (Ndamukong *et al*. 2007). These findings suggest that SA-induced NPR1 activates GRX480, which forms a complex with TGA factors and suppresses the expression of JA-responsive genes. A recent identification of a senescence specific transcription factor WRKY53 represents an additional component involved in mediating the cross-talk between SA and JA signaling (Miao and Zentgraf 2007). WRKY53 has been shown to interact with the JA-inducible protein epithiospecifying senescence regulator (ESR). More importantly, the expression of these genes is antagonistically regulated in response to JA and SA suggesting that WRKY53 and ESR mediate negative cross-talk between pathogen resistance and senescence in Arabidopsis (Miao and Zentgraf 2007). The JA-responsive transcription factor JIN1 acts as a negative regulator of SA signaling during Pst DC3000 infection in Arabidopsis. The jin1 mutant plants showed increased accumulation of SA, enhanced expression of PR genes and increased resistance to Pst DC3000 compared to the wild type plants (Laurie-Berry *et al.* 2006).

3.2.1.2. JA and ET Interaction

Several studies indicate that JA-and ET-signaling often operate synergistically to activate the expression of some defense related genes after pathogen inoculation (Penninckx *et al.* 1998; Thomma *et al*. 2001; Glazebrook 2005). Microarray analysis of defense related genes revealed significant overlap in the number of genes induced by both JA and ET (Schenk *et al.* 2000). Furthermore, the induction of PDF1.2 gene by A. brassicicola was found to be inhibited in both Jasmonate insensitive mutant coi1 and ethylene insensitive mutant ein2 (Penninckx *et al*. 1998; Thomma *et al*. 2001). Recently, several members of ERF family have been shown to play important role in mediating defense responses in Arabidopsis (McGrath *et al.* 2005). It is becoming evident that plants modulate the relative abundance of SA, JA and ET levels, modify the expression of defense-related genes and coordinate complex interactions between defense signaling pathways to activate an effective defense response against attack by various types of pathogens and pests.

3.4. The Role of Auxin in Plant Disease Defense Response

Exogenous application of auxin has been shown to promote disease caused by Agrobacterium tumefaciens (Yamada 1993), Pseudomonas savastanoi (Yamada 1993) and Pst DC3000 (Navarro *et al.* 2006; Chen *et al*. 2007). Similarly, co-inoculation of P. syringae pv. maculicola (Psm) 4326 and auxin has been found to promote both disease symptom and

pathogen growth in tomato (Wang *et al*. 2007). These results indicate that auxin is involved in the inhibition of defense responses in plants. In contrast, blocking auxin responses has been shown to increase resistance in plants.

Several studies have shown that pathogen infection results in imbalances in auxin levels as well as changes in the expression of genes involved in auxin signaling. For example, infection with Pst DC3000 resulted in increased IAA levels in Arabidopsis (O'Donnell *et al.* 2003). Interestingly, the bacterial type III effector avrRpt2, which encodes a cysteine protease, has been shown to modulate host auxin physiology to promote pathogen virulence and disease development in Arabidopsis (Chen *et al*. 2007). Global gene expression analysis using microarrays revealed that Pst DC3000 induces auxin biosynthetic genes and represses genes belonging to Aux/IAA family and auxin transporters. Thus, Pst DC3000 activates auxin production, alters auxin movement and derepresses auxin signaling thereby modulating auxin physiology in Arabidopsis (Thilmony *et al*. 2006). This suggests that auxin promotes disease susceptibility and repression of auxin signaling could potentially result in enhanced resistance in plants. Indeed, down-regulation of auxin signaling has been shown to contribute to plant induced immune responses in Arabidopsis.

Navarro *et al*. (2006) showed that down-regulation of auxin receptor genes by overexpression of a micro RNA (miR393), which targets auxin receptors, increased resistance against Pst DC3000 in Arabidopsis. In contrast, activation of auxin signaling through over expression of an auxin receptor that is partially refractory to miR393-mediated transcript cleavage, enhanced susceptibility to Pst DC3000 (Navarro *et al*. 2006). These results suggest that auxin promotes susceptibility to bacterial disease, and that down-regulation of auxin signaling is part of the plant induced immune response.

Recently, Llorente *et al*. (2008) reported that repression of auxin signaling either through mutations in the auxin signaling components or interference with auxin transport, compromises resistance of Arabidopsis plants to the necrotrophic fungi Plectosphaerella cucumerina (P. cucumerina) and Botrytis cinerea (B. cinerea). Moreover, infection of virulent necrotrophs such as P. cucumerina results in the down-regulation of auxin response genes in Arabidopsis (Llorente *et al*. 2008). This suggests that auxin signaling is an important component involved in modulating plant responses to necrotrophic fungi. Viral pathogens also manipulate auxin signaling components to promote virulence and cause disease. For example, the interactions of tobacco mosaic virus (TMV) replicase with Aux/IAA proteins affect the transcriptional activation of auxin-responsive genes and promote the development of disease symptoms in tomato (Padmanabhan *et al.* 2005, 2006, 2008).

Generally, emerging evidence suggests that auxin acts as an important component of hormone signaling network involved in the regulation of defense responses against various biotrophic and necrotrophic pathogens.

3.5. The Role of Abscisic acid (ABA) in Plant Disease Defense Response

Several recent papers have reported that ABA plays important roles in plant defense responses (Mauch-Mani and Mauch 2005; Mohr and Cahill 2007; de Torres-Zabala *et al.* 2007; Adie *et al.* 2007). However, the role of ABA in plant defense appears to be more complex, and vary among different types of plant-pathogen interactions.

Exogenous application of ABA enhances susceptibility of various plant species to bacterial and fungal pathogens. For example, application of ABA enhanced the susceptibility of Arabidopsis plants to Pst (de Torres-Zabala *et al.* 2007), soybean plants to *Phytophthora sojae* (Mohr and Cahill 2001) and rice plants to *Magnaporthe grisea* (Koga *et al.* 2004). Recently, Yasuda *et al.* (2008) reported that ABA treatment suppressed systemic acquired resistance (SAR) induction indicating that there is an antagonistic interaction between SAR and ABA signaling in Arabidopsis. Generally, these results suggest that ABA acts as a negative regulator of defense responses in various plant pathosystems. However, the role of ABA as a positive regulator of defense has also been reported (Mauch-Mani and Mauch 2005). ABA activates stomatal closure that acts as a barrier against bacterial infection (Melotto *et al.* 2006). As a result, ABA deficient mutants show more susceptibility to Pst.

In addition, treatment with ABA protects plants against *A. brassicicola* and *P. cucumerina* indicating that ABA acts as a positive signal for defense against some necrotrophs (Ton and Mauch-Mani 2004). In contrast, mutants deficient in ABA are more sensitive to infection by the fungal pathogens *A. brassicicola*, *Pythium irregulare* (*P. irregulare*) (Adie *et al.* 2007) and *Leptosphaeria maculans* (Kaliff *et al*. 2007). These results demonstrate that ABA is not a positive regulator of plant defense against all necrotrophs and its role depends on individual plant pathogen interactions.

3.6. The Role of Gibralic Acid (GA) in Plant Disease Defense Response

Exogenous application of GA resulted in enhanced resistance to Pst DC3000 and susceptibility to *A. brassicicola* in Arabidopsis indicating that GA acts as a virulence factor for necrotrophic pathogens. These results suggest that Gibberella might secrete GA as a virulence factor to promote the degradation of DELLA proteins and attenuate JA-dependent defense responses resulting in the loss of DELLA-mediated growth restraint.

Recently, it has been shown that DELLA proteins promote the expression of genes encoding ROS detoxification enzymes thereby regulating the levels of ROS after biotic or abiotic stress (Achard *et al.* 2008). In consistence with this, della penta mutants (that lack all five DELLA genes) accumulate higher levels of ROS after biotic stress and show down regulation of ROS detoxification enzymes compared to wild type plants (Bari and Jones, 2008). Thus, it seems that DELLA proteins regulate plant defense responses against various biotrophic and necrotrophic pathogens at least in part through the modulation of ROS levels in plants.

Mutants affected in GA perception have been shown to affect defense responses in plant. It has been demonstrated that gid1 mutant of rice, defective in GA receptor, accumulates higher GA levels and shows enhanced resistance to the blast fungus *Magnaporthe grisea* compared to wild type plants (Tanaka *et al.* 2006). In addition, the expression of a GA inducible protein PBZ1 (probenazole inducible 1) was found to be elevated in gid1 mutants. Probenazole is a fungicide which is effective against blast disease in rice (Midoh and Iwata 1996). Furthermore, the expression of PBZ1 is induced by rice blast infection. Since, gid1 mutants accumulate high amounts of GA, PBZ1, and show increased resistance to the blast fungus, the accumulation of PBZ1 appears to play important role in resistance against blast in rice. This indicates that GA signaling components play roles in defense signaling in rice (Tanaka *et al.* 2006).

Modulation of bioactive GA levels through GA deactivating enzymes has been shown to affect disease resistance in plants. Recently, Yang *et al.* (2008) reported that a GA deactivating enzyme called Elongated Uppermost Internode (EUI) regulates bioactive GA levels and is involved in disease resistance against bacterial and fungal pathogens in rice. The loss of function eui mutants accumulate high levels of GAs and show compromised resistance whereas EUI overexpressors accumulate low levels of GAs and show increased resistance to Xoo and M. oryzae in rice (Yang *et al*. 2008). Consistent with this, eui plants treated with a GA biosynthesis inhibitor, uniconazole, restored resistance whereas exogenous application of GA to EUI overexpressors compromised resistance to Xoo. These results indicate that GA plays a negative role in basal disease resistance in rice.

Viral proteins have also been shown to affect GA signaling components in plants. For example, expression of a GA biosynthetic enzyme, ent-kaurene oxidize, was repressed in rice plants infected with rice dwarf virus (RDV) resulting in a dwarf phenotype (Zhu *et al.* 2005). It has been shown that P2 protein of RDV interacts with rice ent-kaurene oxidizes and affects the production of GA. RDV infected rice plants showed significant reduction in GA level and treatment of infected plants with GA restored normal growth phenotype (Zhu *et al.* 2005). Infection of rice plants with RDV results in stunting and dark leaves, symptoms that are characteristic of GA-deficient rice mutants. These observations indicate that RDV modulates GA metabolism to promote disease symptoms in rice. Accumulating evidence indicates that GA and its signaling components play important roles in regulating defense responses against various biotrophic and necrotrophic pathogens.

3.7. The Role of Cytokinin (CK) in Plant Disease Defense Response

Although, the role of CK in plant defense is poorly understood, there are indications that CK is involved in the regulation of plant defense responses against some pathogens. CK plays an important role in the development of club root disease caused by *Plasmodiophora brassicae* in Arabidopsis (Siemens *et al.* 2006). Global gene expression analysis of *P. brassicae* infected Arabidopsis resulted in differential expression of more than 1,000 genes compared to control plants. Interestingly, genes involved in cytokinin homeostasis were strongly downregulated. Transgenic plants overexpressing cytokinin oxidase/dehydrogenase genes showed resistance against *P. brassicae* infection suggesting that cytokinin acts as a key factor in the development of club root disease in Arabidopsis (Siemens *et al.* 2006). Recently, infection with *Rhodococcus fascians* has been shown to modulate cytokinin metabolism in Arabidopsis (Depuydt *et al.* 2008). It has been shown that *A. tumefaciens* modifies CK biosynthesis by sending a key enzyme into plastids of the host plant to promote tumorigenesis (Sakakibara *et al.* 2005). Constitutive activation of a resistance (R) protein in Arabidopsis has been shown to display morphological defects through the accumulation of CK indicating the involvement of CK pathway in some R protein-mediated responses (Igari *et al.* 2008).

3.8. The Role of Brassinosteroids (BRs) in Plant Disease Defense Response

Brassinosteroids (BRs) are a unique class of plant hormones that are structurally related to the animal steroid hormones. Although, BRs are known to influence abiotic stress responses in plants, very little is known about their role in plant responses to biotic stresses. However, emerging evidence indicates that BRs are involved in the regulation of plant defense responses. It has been reported that BR enhances resistance to TMV, Pst and *Oidium sp*. in tobacco. Similarly, BR was shown to increase the resistance of rice plants against *M. grisea* and *Xanthomonas oryzae* infection (Nakashita *et al.* 2003).

3.9. The Role of Peptide Hormones in Plant Disease Defense Response

Peptide hormones comprise a new class of hormones and are involved in the regulation of various aspects of plant growth and development including defense responses against attacking pathogens and pests (Matsubayashi and Sakagami 2006; Farrokhi *et al*. 2008). Defense-related peptide hormones include systemin (Pearce *et al.* 1991), hydroxyproline-rich glycopeptide systemins (Pearce *et al*. 2001, 2007; Pearce and Ryan 2003) from solanaceous plants and AtPep1 peptide from Arabidopsis (Huffaker *et al*. 2006). These peptides play roles in the activation of local and systemic responses against wounding and pest attack.

Generally, defense-signaling peptides play important roles in the activation of defense against invaders probably by amplifying the signal initiated by wounding and elicitors. However, the underlying molecular mechanism involved in the activation of these peptide hormones in regulating plant defense remains elusive.

4. SUMMARY AND CONCLUSION

Plant hormones regulate complex signaling networks involving developmental processes and plant responses to environmental stresses including biotic and abiotic stresses. Significant progress has been made in identifying the key components and understanding plant hormone signaling (especially SA, JA and ET) and plant defense responses. Several recent studies provide evidence for the involvement of other hormones such as ABA, auxin, GA, CK and BR in plant defense signaling pathways. Treatment of plants with some hormones results in the reprogramming of the host metabolism, gene expression and modulation of plant defense responses against microbial challenge. Depending on the type of plant-pathogen interactions, different hormones play positive or negative roles against various biotrophic and necrotrophic pathogens. Plant hormone signaling pathways are not isolated but rather interconnected with a complex regulatory network involving various defense signaling pathways and developmental processes. To understand how plants coordinate multiple hormonal components in response to various developmental and environmental cues is a major challenge for the future.

In addition to the production of hormones by plants, several plant pathogens also produce phytohormones or their functional mimics to manipulate defense-related regulatory network of plants. Emerging evidence suggests that plant pathogens manipulate components of hormone biosynthesis and signaling machinery leading to hormone imbalances and alterations in plant defense responses. This is one of the strategies used by some pathogens to confer virulence and cause disease. However, we have very limited knowledge on how pathogen effectors confer virulence by modulating hormone signaling components. Recent global expression profiling studies in response to pathogen challenges are providing useful information about different components involved in the complex interactions between hormone-regulated defense signaling pathways. However, additional studies are necessary to extend our understanding of the complex regulatory mechanisms operating between plant hormone signaling and plant defense responses. A better understanding of phytohormone- mediated plant defense responses is important in designing effective strategies for engineering crops for disease and pest resistance.

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