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Review

Influence of hormones on microbial plant infection: A defence mechanism

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Plant produce a host range of organic compounds which are hormones in nature including organic acids and vitamins in complex networks to balance the response to developmental and environmental cues, which could serve as nutrients or signals by microbial populations. Pathogens release phytohormones, small molecules or volatile compounds which act directly or indirectly to activate plant immunity or regulate plant growth. Phytohormones pathways which are linked to each other in a complex network; ABA, ET, gibberellins auxins, cytokinius, pathways are seen as hormone modulators of the SA-JA signaling back bone. Plants employ specific recognition and signaling systems enabling the rapid detection of pathogen invasion and initiation of vigorous defensive responses. Evidence has accumulated showing that classic plant signals such as auxins and cytokinins can be produced by microorganisms to efficiently colonize the root and modulate root system. Plant hormones are able to control some pathogen bacteria through microbial antagonism, which is achieved by competing with the pathogens for nutrients, producing antibiotic, and the production of anti-fungal metabolites. Mutual interactions between stress-specific hormones such as salicyclic acid and jasmonic acid/ethylene SA-JA (ET) are regarded as the central back bone of the immunity in plants. However, growth-promoting hormones (auxins, cytokinins, gibberellic acid, and abscisic acid) either inhibit or potentiate this balance in mediating the protection or susceptibility of the plant against the invading pathogen.

Key words: Plant hormones, abscisic acid, auxins, microbes, jasmonic acid.

INTRODUCTION

Hormones are tuners of plant responses to biotic and abiotic stresses. They are involved in various complicated networks, through which they modulate responses to different stimuli (Dong-Lei et al.,2013). Four hormones primarily regulate plant defense to pathogens: salicylic acid (SA), jasmonic acid (JA), ethylene (Et) and abscisic acid (ABA). In susceptible plants, viral infections result in hormonal disruption, which manifests as the simultaneous induction of several antagonistic hormones. However, these antagonistic hormones may exhibit some sequential accumulation in resistant lines. Virus propagation is usually restricted by the activation of the small interfering RNA (siRNA) antiviral machinery and/or SA signaling pathway. Several studies have investigated these two systems, using different model viruses.

ABSCISIC ACID

The plant hormone abscisic acid is involved in the initiation of stress-adaptive responses to various environmental cues. ABA is a pivotal determinant in the outcome of plant Pathogen interactions. For instance,

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ABA primes for callose deposition and thereby enhances basal defense against the powdery mildew fungus *Blumeria graminis* and the necrotrophic fungus *Alternaria brassicicola*, and also activates JA– mediated resistance against the Oomycete *Pythium irregulare*. ABA acts as a negative regulator of disease resistance with inhibition of ABA biosynthesis and/or signal transaction commonly resulting in enhanced disease resistance to a wide variety of bacterial, fungal and Oomycete pathogens exhibiting distinct parasitic habits. It was shown that *Pseudomonas syringae* hijacks the ABA biosynthetic and response machinery to cause disease, indicating that ABA is a susceptibility factor for this bacterium. Similarly, Jiang et al. (2010) reported transiently elevated ABA titers in rice plants attacked by the blast fungus *Magnaporthe oryzae*. Previous work has shown that ABA enhances basal resistance against the rice brown spot pathogen. ABA plays both positive and negative roles in rice disease resistance depending on the type of pathogen and stage of infection (Ching-Hong, 2013).

ABA, a sesquiterpene compound strongly regulates several Plant hormone–virus interactions in 3 developmental stages, including seed germination and fruit ripening, and is considered as the key hormone in the modulation of plant responses to many abiotic stresses (Atkinson and Urwin, 2012; Rajjou et al., 2012; Sung and Luan, 2012). In addition to its antagonistic roles in defense of hormone pathways, such as SA and JA/Et, ABA appears to have multifaceted roles against the same pathogen, depending on the stage of infection. ABA can positively regulate plant defense at the early stages of infection by the mediation of stomatal closure against invaders, or induction of callus deposition if the pathogen evades the first line of defense (Leustek, 2002).

The strong antagonism with SA suggests that either of the two could prevail under certain circumstances. The SA pathway is induced to various levels under both compatible and incompatible interactions with many viral infections. However, ABA is also induced during some viral infections. Simultaneous up-regulation of ABA and SA pathways has been reported for TMV and BaMV infections (Alazem et al., 2014). Other studies have reported the induction of either pathways without masking the other (Van Loon et al., 2006; Flors et al., 2009). This particular phenomenon, in which these two antagonistic pathways are induced following infection of certain RNA viruses, may be a common occurrence.

ABA may have an important role in incompatible interactions with viruses. A recent study has proposed a role for ABA in controlling the localization of temperature-sensitive *R* genes (Mang et al., 2012). ABA deficiency promoted the activity and nuclear localization of temperature-sensitive *SNC1* (*SUPPRESSOR OF NPR1-1, CONSTITUTIVE 1*) and *RPS4* (*RESISTANCE TO PSEUDOMONAS SYRINGAE 4*) *R* genes, which function against *P. syringae*. Such localization is essential for these proteins to function at low and high temperatures, whereas, inWT plants, these proteins function only at low temperatures. ABA also affects plant defenses at the level of the RNA silencing machinery, which is considered to be a broader defense system against viruses when compared with *R*-gene-specific resistance.

RNA silencing affects both the local accumulation and systemic movement of a wide range of viruses, and is considered to be the cause of non-host resistance for some viruses, such as PVX (Jaubert et al., 2011; Lewsey et al., 2008; Ruiz-Ferrer and Voinnet, 2009). ABA seems to have direct and indirect links with this system. For example, ABA partially controls levels, which are significantly increased in *aba1-5* (Li et al., 2012).

JASMONIC ACID

Jasmonic acid (JA) plays an important role in rice basal defense against bacterial and fungal infection and may be involved in the Salicylic acid (SA) -mediated resistance. Most of these compounds, for instance JA function as hormone-like signals that regulate such processes as sexual and asexual spore development and toxin production. For instance, fungal pathogens can exploit host oxylipins to increase their own virulence, while plant oxylipins such as the jasmonates function in the opposite side to resist the attack of fungal pathogens. Jasmonates also promotes fungi and nitrogen-fixing bacteria.

JA is an oxygenated fatty acid (oxylipin) involved in resistance to necrotrophic pathogens and insect infestation (Thaler et al., 2004). Together with Et, JA regulates induced systemic resistance (ISR), which is invoked by non-pathogenic microbes, such as rhizobacteria. A study has shown that rhizobacterium-mediated induction of JA reduces the symptoms of CMV infection in *Col-0* (Ryu et al., 2004). Several later studies supported the positive roles of JA in compatible interactions, but in a phase-specific Mode. JA treatment at early stages of PVY–PVX double infection enhanced resistance, but later application increased susceptibility, probably as a result of the antagonistic effect of JA on SA (Garcia-Marcos et al., 2013). Similar studies have shown that JA-responsive genes are modulated at early stages of infection, e.g. in CaMV, *A. thaliana* and *Panicum mosaic* virus and its satellite virus in the monocot plant *Brachypodium distachyon* (Love et al., 2005, 2012; Mandadi and Scholthof, 2012). Recently, Zhu et al. (2014) showed that the treatment of *Nicotiana benthamiana* plants with JA or SA enhances systemic resistance to TMV, and that resistance is further enhanced by pretreatment with JA followed by SA. Remarkably, plants impaired in the JA pathway failed to accumulate SA in the leaves or phloem, and exhibited increased susceptibility, whereas impairment of the SA pathway did not affect JA levels, but increased susceptibility (Zhu et al., 2014). JA may modulate early

components of the SA pathway, but how JA regulates SA biosynthesis and resistance in compatible interactions is still unknown.

ETHYLENE

Depending on the pathogen type and environmental conditions, studies have demonstrated that ET could act as a positive or negative modulator of disease resistance (Broekaert et al., 2006). It plays an important role in rice basal pond to the presence of phytopathogens by synthesizing stress ethylene that exacerbates the effects of the stress on the plant. Thus, to decrease the damage to plants caused by a wide range of phytopathogens is to lower the plants ethylene response, by treating plants with ACC deaminase-continuing plant growth promoting hormone (PGPB). ET is involved in certain developmental stages, such as senescence, as well as in the defense response to necrotrophic pathogens (Van der Ent and Pieterse, 2012; Graham et al., 2012). ET does not appear to be essential for plant resistance against viruses, with only a few studies describing an involvement of ET in symptom development. Although ET may support symptom development in the case of CaMV infection and systemic movement in the case of TMVcg infection, an interesting, opposing study demonstrated the importance of ET to the ER against TBSV in tobacco plants. TBSV accumulates in tobacco plants insensitive to ET (ETR line), but not in WT plants (Sansregret et al., 2013). It remains to be determined how ET positively regulates ER in response to TBSV in this case. Although endogenous JA and ET have antagonistic effects on SA-mediated defense against viruses, these findings imply that the timing of treatment greatly affects plant defense against viral infection.

AUXINS

Increasing evidence shows that auxins stimulates disease susceptibility in the model plants; Arabidopsis and rice. Cytokinins and auxins are produced by bacteria involved in nitrogen fixing associations and mycorrhizal fungi have the ability to produce microbes that can synthesize cytokinins and/or auxins which are exclusively produced by microbial symbionts of plants. IAA (indole - 3- acetic-acid) have clearly been shown to be a signaling molecule in microorganisms, in both IAA producing and IAA-non producing species. Diverse bacterial species posses the ability to produce auxin phyto-hormone, IAA. The amount of bacteria auxin produced and the contrasting effects of IAA on plant development are linked to the sensitivity of the host itself.

Auxins play a key role in plant growth and development by maintaining apical dominancy, and mutants in the auxin signaling pathway or responsive factors display as an aberrant growth phenotype (Benjamins and Scheres, 2008). Many viral infections result in aberrant phenotypes, such as stunting, leaf curl and loss of apical dominance, which resemble those of mutants with compromised Aux biosynthesis and/or signaling (Kazan and Manners, 2009).

CYTOKININS

CKs (cytokinins) are mainly produced in the meristemic zones of shoots and translocated to actively growing areas. They promote cell proliferation and elongation, and are involved in various developmental processes, including transduction of nutritional signals and delay of senescence (Aloni et al., 2005; Sakakibara, 2006). In addition, some bacterial and fungal pathogens produce CKs. Much like auxins, CKs suppress defense responses (such as HR) to *Pseudomonas savastanoi* (Robert-Seilaniantz et al., 2007).

GIBBERELLIC ACID

GA (gibberellic acid) induces seed germination, promotes stem elongation and modulates flowering (Sun and Gubler, 2004). This hormone promotes plant growth by inhibiting DELLA proteins, which are negative regulators of plant growth (Robert-Seilaniantz et al., 2007). GA seems to have a negative role in plant defence. Loss of function mutants of DELLA increase plant resistance to biotrophs, but exhibit hyper-susceptibility to infection with necrotrophs. GA may serve to facilitate defenses to biotrophs or necrotrophs by partially modulating the balance between SA and JA/Et-mediated signaling pathways (Robert-Seilaniantz et al., 2007).

BRASSINOSTEROIDS

BRs (brassinosteroids) are a class of polyhydroxysteroids that affect many cellular processes, including elongation, proliferation, differentiation, membrane polarization and proton pumping (Xia et al., 2010). They also affect disease resistance at several levels in tobacco and rice (Nakashita et al., 2003). In potato, BRs can reduce viral infection in starting plant materials at various stages of development until the second tuber generation.

In addition, BR treatment decrease the levels of TMV and other biotrophs in tobacco plants (Hayat et al., 2011). Studies using additional viruses may unveil novel strategies by which plants tolerate or resist viral infections. However, the roles of hormones other than SA, especially those with antagonistic properties, such as ABA, have been neglected. In this review, we summarize recent findings on the different roles of hormones in the regulation of plant–virus interactions, which are helping in

elucidating the fine tuning of viral and plant systems by hormones.

In natural systems, plants face a plethora of antagonists and thus posses a myriad of defense and have evolved multiple defense mechanisms by which they are able to cope with various kinds of biotic and abiotic stress (Ballhorn et al., 2009). Besides antimicrobial nature, some of which are produced and some of which are induced by infection will improve their resistance against microbial pathogens as well as various environmental stresses. There are various other modes of defense: the construction of polymeric barriers to pathogen penetration and the synthesis of enzymes that degrade pathogen cell wall. In addition, plants employ specific recognition and signaling systems enabling the rapid detection of pathogen invasion and initiation of vigorous defensive responses. Once infected, some plants also develop immunity to subsequent microbial attacks.

Plants produce a large variety of secondary products that contain a phenol group, a hydroxyl functional group on an aromatic ring called Phenol, a chemically heterogeneous group also. They could be an important part of the plants defense system against pests and diseases including root parasitic nematodes (Wuyts et al., 2006; Saviranta et al., 2010).

Coumarin: They are simple phenolic compounds, widespread in vascular plants and appear to function in different capacities in various plant defense mechanisms against fungi. They are derived from the shikimic acid pathway, common in bacteria, fungi and plants but absent in animals. Also, they are a highly active group of molecules with a wide range of anti-microbial activity against both fungi and bacteria (Brooker et al., 2008). It is believed that these cyclic compounds behave as natural pesticidal defense compounds for plants and they represent a starting point for the exploration of new derivatives possessing a range of improved antifungal activity.

Isoflavonoids: Isoflavonoids are derived from a flavonone intermediate, naringenin, ubiquitously present in plants and play a critical role in plant developmental and defense response. They are secreted by the legumes and play an important role in promoting the formation of nitrogen-fixing nodules by symbiotic rhizobia (Sreevidya et al., 2006).

SULPHUR CONTAINING SECONDARY METABOLITES

They include GSH, GSL, phytoalexins, thionins, defensins and allinin which have been linked directly or indirectly with the defense of plants against microbial pathogens (Crawford et al., 2000; Leustek et al., 2000; Saito, 2004; Grubb and Abel, 2006; Halkier and Gershenzon, 2006), and a number of them thought to be involved in the SIR (ElkeBloem et al., 2005).

GSH: It is one of the major forms of organic S in the soluble fraction of plants and has an important role as a mobile pool of reduced S in the regulation of plant growth and development, and as a cellular anti-oxidant in stress responses. Kang and kim (2007) and Noctor et al. (1998) reported it as a signal of plant S sufficiency that down regulates S-assimilation and S-uptake by roots. Similar conclusion was reached by Lappartient and Touraine (1997) and Lappartient et al. (1999). Specialized cells such as Trichomes exhibit high activities of enzymes for synthesis of GSH and other phytochelatins necessary for detoxification of heavy metals (Gutierez-Alcala et al., 2000; Choi et al., 2001). The GSH content varies between 3 to 10 mM and is present in the major cellular compartments of the plant. To mitigate oxidative stress, GSH functions as a direct anti oxidant and also as a reducing agent for other anti oxidants such as ascorbic acid (Nocito et al., 2002), as well as an integral weapon in the defense against ROS generated by O_3 (Pasqualini et al., 2002; Conklin et al., 2004) or as a reaction to biotic and abiotic stress.

Additionally, GSH is also involved in the detoxification of xenobiotics and cytotoxins by targeting them into vacuole (Rea et al., 1998). GSH rapidly accumulate after fungal attack, and may act as systemic messenger carrying information concerning the attack to non-infested tissues (Foyer and Rennenberg, 2000).

In several studies, the switch from single low to double low oil seed rape varieties led to increasing infestation of oil seed rape with fungal diseases such as light leaf spot (*Pyrenopeziza brassicae*), sclerotina stem rot (*Sclerotinia sclerotiorum*) and Alterneria (*Alternaria brassicae*). (Geu-Flores *et al*., 2009; Ratzka *et al*., 2002; Zukalova and Vask, 2002).

Phytoalexins: Phytoalexins are synthesized in response to bacterial or fungal infection or other forms of stress that help in limiting the spread of the invading pathogens by accumulating around the site of infection, appears to be a common mechanism of resistance to pathogenic microbes in a wide range of plant. Most plant families produce organic phytoalexins of diverse chemistry; these groups are often associated with a family, for example sesquiterpenoids of Solanaceae, isoflavonoids of Leguminosae, while phytoalexins from *Brassica* have an indole or related ring system and one S atom as common structural features. Cruciferous crops are cultivated worldwide because they are extremely valuable and for the last decades, various research groups have investigated cruciferous phytoalexins (Monde et al., 2000) as well as their biological activity.

Defensins, thionins and lectins: All these are S-rich non-storage plant proteins synthesize and accumulate

after microbial attack and such related situations. All of which inhibits the growth of a broad range of fungi (Thomma et al., 2002). Some defensins are antifungal or occasionally anti-bacterial (Thomma et al., 2002). The components seem to be involved in the natural defense system of plants as they can be highly toxic to microorganisms. Accumulation of thionins in the cell wall of infected wheat spikes of resistant wheat cultivars indicates that the accumulation of thionins may be involved in defense responses to infections and in spreading of *Fusarium culmorum* (Kang and Buchenauer, 2003). Within a species, individual plants often differ greatly in their resistance to microbial pathogens.

CONCLUSION

Plants have evolved multiple defense mechanisms against microbial pathogens and various types of environmental stress. Besides anti-microbial secondary metabolites, some of which are produced and some of which are induced by infection will improve their resistance against microbial pathogens as well as various environmental stresses.

Viral infections disrupt many processes, resulting in temporal changes in hormone signaling and responses, metabolites, and transcriptomic and small RNA profiles. The roles of hormones in plant virus interactions and cross-talk among hormone pathways will help to determine the molecular mechanisms by which plants resist infection. The interactions that occur between plants and their associated microorganisms have long been of interest, as knowledge of these processes could lead to the development of novel agricultural applications. Plants produce a wide range of organic compounds including sugars, organic acids and vitamins, which can be used as nutrients or signals by microbial populations. On the other hand, microorganisms release phytohormones, small molecules or volatile compounds, which may act directly or indirectly to activate plant immunity or regulate plant growth and morphogenesis. In this review, we focused on the roles of some plant hormones in microbial infection e.g phytoaelexins, jasmonic acid, auxin, cytokinins etc. and how they interact with plants in harmful or beneficial ways. The inducing bacteria for instance, triggers a reaction in the roots that creates a signal that spreads throughout the plant which results in the activation of defense mechanisms, such as reinforcement of plant cell wall, production of anti-microbial phytoalexius, and the synthesis of pathogen related proteins.

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