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Phytoharmones Interaction Defense Against Necrotrophic Fungus (*Botrytis cinerea*)

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ABSTRACT: The phytoharmones essential for growth, reproduction, cell differentiation changes in our plant ecosystem. The exogenous application of auxins and gibberellins can reduce disease development, where ABA and ethylene promote disease spread. Hormones were well known in interaction with botrytis infection. The fungus was infected by releasing different hormones so that to invade into the plant host and possible ways to study different pathways to knock out the genes. Deficient mutants seem to enhance resistance towards botrytis infection like ethylene, ABA Abscisic acid. The entire process of fungal development, host-pathogen interaction have been being reported. The review summarize the current state of knowledge about Botrytis – host interaction, whereby nectrophs utilizes the plant harmones or metabolic enzymes to suppress the plant immune system. In this study strongly suggested that the involvement of plant resistance pathways in association with fungal effectors, metabolites can shape to maintain against botrytis phytopathogen. In this article, we review our current understanding haromonal regulatory networks involving SA, JA, ET and ABA. Finally the role of phtyoharmones interaction as complementary and alternative disease management will be over viewed.

Keywords: Phytoharmones Interaction, Necrotrophic fungus, ABA

INTRODUCTION

Botrytis cinerea is a necrotrophic pathogen and a wide host range of crops, especially infect on fruits. The pytoharmones like auxin, cytokinin, brassinosteroids plays a major role in defense response either in upregulation or downregulating against the *B.cinerea*. The mostly gene-gene hypothesis was studied in biotrophic pathogens including other organisms leading to compatible interaction between R gene (plant) and AVR gene (pathogen) resulting in multiple changes of physiological and biochemical changes, necrotic cell death leads to hypersensitive response (HR) (Flor,1971; Govin and Levine, 2000). Likewise, in the necrotrophic pathogen is mediated by a single host resistance gene, where plant identifies elicitors has PAMP recognition to activate defense responses (Nurnberger and Brunner, 2002). The pathogen produces enzymes like PGs (endoploygalacturonases) released a by-product homogalacturonan as pectin component, play important role in virulence factor in Botrytis infection and showed in defense response in other a pathogens. (tenHAVE *et al.,* 2001; De Lorenzo *et al.,* 1997). The pathogenic microbes were attached on adherent surfaces on leaves to absorption required nutrients and for further survival. This causes symptom development and losses of yield. The disease occurrence leads to a reduced level of plant hormones, scavengers of free radicals, high amount of calcium content. Host tissue defeat in battle war to challenge against pathogen-related proteins like PR proteins, Phytoalexins, Polygalacturonase, Secondary metabolites, and lignifications of the cell wall. The mechanism of these compounds was not clearly understood. Different possibilities were reduced levels of disease growth (Elad, 1997). Furthermore, the transcriptional factors were useful for disease suppression. For example, the Arabidopsis WRKY33 transcription gene down-regulated of ZIM domain genes in Jasomonic acid pathways, reduction level of salicylic acid, higher-level post-infection, wrky33 susceptibility in *B.cinerea* infection. However, the, management by fungicides are not safe to environment and humans. Therefore, it is important to understand the signaling pathways and develop new strategies to prevent botrytis infection and functions of these pathogensis –related proteins genes are untangle. Botrytis infection where, pathogen genotypes were vary and plant signaling networks that lead resistance is not constant. In this review, we mainly introduce the latest information about screening of varied mutants complementary phenotypes on plant hormones and advances of transcriptomics and mutagenic are helpful to study fungal effectors.

A. The role of auxin mutants

However, auxin mutants like aux 1 decrease the auxin influx, lack of systemic resistance, and signaling mutants axr1, axr2,axr6 are showed more susceptible compare to wild type (Korolev *et al.,* 2008). In the model plant, Arabidopsis mutants the level of infection was increased in *B. cinerea*. The changes occur in signaling mutants due to activation of salicylic acid and jasmonic acid. In a higher level of precursor auxin the Indole acetic acid converts into Indole acetic acid - aspartic acid. Induction of ethylene leads to resistance against the *B. cinerea* (González-Lamothe *et al.,* 2012).

Table 1: Phytoharmones with different combination of mutant genes in role of defense response towards *B. cinerea.*

Sr. no.	Phytoharmones	Crop	Mutant genes	Reference
I.	Ethylene	Tomato	*Never ripe*-wound signaling resistance	Dıaz *et al.,* 2002
II.	JA(Jasmonic acid) /ET	*Arabidopsis thaliana*	Bos 1 gene encodes R2r3MYB transcription factor – biotic and abiotic stress	Mengiste *et al.,* 2003
III.	SA/camlexin	*Arabidopsis thaliana*	Overexpression SA leads to decrease the lesion size through the NPR1 dependent mechanism	Ferrari *et al.,* 2003
IV.	JA	*Arabidopsis thaliana*	ANAC019 AND ANAC055 (non-mutant) regulates defense responses	Bu *et al.,* 2008
V.	JA/IAA	*Arabidopsis thaliana*	Overexpression of P450 protein CYP82C2 – increased resistance	Liu *et al.,* 2010
VI.	JA	*Arabidopsis thaliana*	*jah2* mutant defense -hypersenetive respone	Wei *et al.,* 2012.
VII.	D-galacturonic catabolism acid (pectin residue)	*Arabidopsis thaliana* And *Nicotiana benthamiana*	*lgd1*- galactonate dehydratase gene –defense related genes	Zhang *et al.,* 2012
VIII.	JA and ethylene	*Arabidopsis thaliana*	etr1-1-insensitivity to petunia, delayed senescence	Wang *et al.,* 2013
IX.	ABA	Tomato	*Sitiens* –enhance resistance	Sivakumaran *et al.,* 2016
X.	JA/ET with AR156 *Bacillus cereus*	*Arabidopsis thaliana*	Bacillus cereus triggers JA/ET in *NahG* and sid2-2 mutants-signaling PAMP immunity	Nie *et al.,* 2017
XI.	JA and ethylene/SA	*Arabidopsis thaliana*	SR1 –transcrptional factor regulates cell death, plant defensin genes	Vuorinen *et al.,* 2021
XII.	Ethylene	Tomato	*Never ripe*-wound signaling resistance	Dıaz *et al.,* 2002
XIII.	JA(Jasmonic acid) /ET	*Arabidopsis thaliana*	Bos 1 gene encodes R2r3MYB transcription factor – biotic and abiotic stress	Mengiste *et al.,* 2003
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XX.	ABA	Tomato	*Sitiens* –enhance resistance	Sivakumaran *et al.,* 2016
XXI.	JA/ET with AR156 *Bacillus cereus*	*Arabidopsis thaliana*	Bacillus cereus triggers JA/ET in *NahG* and sid2-2 mutants-signaling PAMP immunity	Nie *et al.,* 2017
XXII.	JA and ethylene/SA	*Arabidopsis thaliana*	SR1 –transcrptional factor regulates cell death, plant defensin genes	Vuorinen *et al.,* 2021

In the arabidopsis plant, the role of indole acetic acid upregulated the biosynthetic genes (Windram *et al.,* 2012). Notably, *B. cinerea* helps to down-regulate the auxin signaling genes. It is noteworthy that exogenous application of oligogalacturonides enhances defense response in leaves of tobacco, grapes, transgenic plants, and also Arabidopsis plants (Aziz *et al.,* 2004). Interestingly, oligogalacturonides showed antagonized by Indole acetic acid and vice-versa but the mechanism was not fully studied (Branca *et al.*, 1998). The pectin is an extracellular component it crosslinks the plant cell wall, it was expressed and induced by auxin. In auxin treatment, the plant express pectin methylesterase and expansin in roots (Laskowski *et al.*, 2006). The plant express polygalacturonases inhibiting proteins to reduce the activity of *B. cinerea* and slow down the homogalacturonan molecules (Ferrari *et al.*, 2013). In the Arabidopsis plant pectin methylesterase like AtPME-1 or AtPME-2 promotes increase resistance against *B. cinerea* due to the esterification process and reduction level of Salicylic acid (Raiola *et al.,* 2004). Hence, the auxin plant hormones induce resistance over *B. cinerea* fungal pathogen.

B. Cytokinin (CKs)

The cytokinin promotes cell growth, cell differentiation, and nutrient assimilation. At present cytokinin appeared in the form of Zeatin, Kinetin, and 6-benzyl amino purine. They were few were reported on the cytokinin towards *B.cinerea*. The low levels of cytokinin lead to downregulation, susceptibility of *B. cinerea*. (Simens *et al.*, 2006). Likewise, the application of increased levels of cytokinins exhibits resistance with late leaf senescence in transgenics tomato plants (Swartzberg *et al.,* 2008; Grosskinsky *et al.,* 2011). However, the cytokinin-induced genes will reduce the auxin level in pathogens showed a defense response, unlike the mutant plants that showed susceptibility in pathogens (Lee *et al.,* 2009). Experimental evidence suggested that the increased growth concentration (0-10M) of kinetin inhibits the mycelia growth of *Botrytis* spp. (Elad, 1995; Russo and Pappelis,1993) rose flowers.

C. Ethylene (ET)

In laboratory experiment proved that ethylene production by *B. cinerea* in shake cultures and enzymes involved ethylene biosynthesis pathway (Qadir *et al.*, 1997). It is light-dependent culture growth, ethylene release in light condition (Chague *et al.,* 2002). These three pathways were involved in the ethylene biosynthesis pathway namely, Methionine Dependent Pathway (MDP) produced compounds like ACC and SAM (S-Adenosyl Methionine) in higher plants, Ethylene-Forming Enzyme (EFE) the intermediate is 2-oxoglutarate, deamination and spontaneous oxidation of KMBA(D-keto Jmethylthiobutyic acid) to produce the ethylene (Cristescu *et al.,* 2002). The ethylene response factor (ERF) like ERF 5 or ERF 6 showed resistance against *B. cinerea* and single mutants showed to negative regulation and susceptible action of the pathogens. But the double mutants (*er5* and *er6*) were declared as a positive response in susceptibility, and crosstalk with SA/JA pathways (Moffat et al., 2012).

D. Brassinosteroids (BRs)

It is a steroid hormone. The functions of BRs are reproduction processes, seedling growth, second organogenesis (Ryu and Hwang, 2013) resistance to biotic, biotic stresses, and increase the crop yield, ion balance level (Khripach *et al.,* 2000). Current evidence that BRs homeostasis in the cell wall, cellulose production to fight against host pathogens system and directly involved in pectin esterification process. As per interest concern the reduction of *B. cinerea* mycelia growth overexpression of enzymes like 3-hydroxy-3-methylglutaryl-CoA, isoprenoids involve BR biosynthesis pathway. In lower expression of these enzymes leads to susceptibility and resistant plants induced antioxidants for stoppage of pathogen growth (Zhu *et al.,* 2013). Additionally, the transcriptional factors i.e., *WRKY, MYB, ERF, NAC* and *bHLH* families with 188 encoding genes were upregulated, dependent on resistance against the *B. cinerea*. The exogenous application of BRs showed a defense response in the rose petals (Lv *et al.,* 2018). In Arabidopsis, THE1 is a transcription factor that positively upregulated with interaction with GEF 4 of BR's resistance towards the *B. cinerea* (Qu *et al*., 2017).

E. Abscisic acid (ABA)

ABA interplays a role in callose formation, cell wall preamblility, release of antioxidants, produces EXPANSION-LIKE genes, and negative regulation in plants. The ABA mutants enhance more resistance towards *B. cinerea*. ABA treatment mutants of Arabidopsis plant alter the properties of the cell wall, cell to cell adhesion, thickening of the cell wall (Oide *et al.,* 2013). Furthermore, lack of ABA content mutant of tomato variety showed resistance to *B.cinerea* (Curvers *et al.,* 2010). The cuticular mutants, ABA biosynthetic *aba 2*, *aba 3* mutants enhance resistance to *B. cinerea* (L'Haridon *et al.,* 2011). It was suggested that it triggers Reactive Oxygen Species production to alter the cell wall modification genes, decrease the level of endo-arabinan enzymes, pectin enzymes (Abuqamar *et al*., 2013). The phytohormones represent as good horizontal gene transfer. Higher content of ABA, pathogencity occurs in the plant. Interestingly, for a detailed study of the host–pathogen interaction, research would be done for biological purposes and find out different pathways. Genomic data is available in the NCBI website, to clone ABA pathway genes. In this pathway first, step for in FFP Including P-450 Monooxyenase to determine whether ABA genes were

present or not. Non-found ABA genes non-significant infection growth on higher plants. Likewise, this pathway is 1,4 transdiol-ABA pathway *Cercospora densiflorae* (Hirai *et al.,* 1986).

F. Gibberlic acid (GA)

Gibberellic acid was first identified in paddy by Bakanne disease (*Gibberella fujikuroi*) to stem elongation, GA biosynthesis was studied to examines various pathways and production of several stains use to knock out the genes. Furthermore, the same pathway was found in *B.cinerea* like GA in paddy a gene as entcopalyl diphosphate synthase. (bifunctional encoding enzyme) still, the mechanism is not known. (Tudzynski and Sharon, 2002).

CONCLUSION AND FUTURE SCOPE

B.cinerea utilizes the metabolites from plants to causes pathogenesis on host-pathogen produce several plant hormones mainly ethylene production, low level of ABA, IAA, and cytokinins. In the hormonal cross talk, auxin and gibberellins suppress the pathogen spread but in ethylene leads to susceptibility. The main concept of the pathogenic infection that it utilizes phytohormones for proper colonization and occurrence of more survival. Present studies that exogenous application has a chance to reduce the disease development, it employs for varied biosynthetic pathways and also finds out intermediate compounds so that we can block the pathways by the construction of deficient mutants. In botrytis infection, we can able to study ethylene production. Nevertheless, by cumulative data farmers should apply the right time of application of phytohormones to prevent Botrytis infection. For future understanding the physiological and genetic processes includes plant –pathogen interaction, pave ways for exploring the phenomenon in crop improvement and protection. Molecular mechanism of *B.cinerea* pathogencity will develop to control the technologies. However, more research effort is needed for mechanism and regulatory networks in aspects of host –pathogen interaction.

Conflicts of Interest. The authors declared no conflict of interest.

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