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Popular Article

Fusarium wilt: an insight into molecular mechanism of host pathogen interactions

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Abstract

Fusarium wilt is a common plant disease brought on by several strains of the fungus *Fusarium oxysporum*, which lives in the soil as well as in seeds of the host crop plants. Numerous plant species, including commercially significant food crops like Banana and pulse crops like Chickpeas, are vulnerable. As the pathogen is typically a soil borne type and it can survive in soil for several years, the development and cultivation of resistant variety of crop plant is an efficient way to overcome the yield loss brought on by *Fusarium* wilt. Thus, researchers across the world are working continuously to develop the crop plants resistant against different strains of *F. oxysporum*. However, in order to develop resistant crop plants, it is inevitable to understand the host pathogen interaction and molecular basis of resistance mechanism. This popular article highlights the important aspects *F. oxysporum* specific host pathogen interaction and general molecular mechanisms involved during *F. oxysporum* specific signal perception and resistance response by the host plants

Keywords: Wilt, *Fusarium oxysporum*; Pathogen, Host-pathogen interaction, Virulence, Resistance

Introduction

Fusarium is a globally distributed genus of fungi species that are significant to the global agricultural economy. Twenty monophyletic species complexes, comprising up to sixty species, are formed by nine of the species. Among all, *F. oxysporum* is a common soil born plant pathogen that is responsible for causing catastrophic wilt disease in over 100 plant species. It is a major danger to many commercially significant crops, including tomato, cotton, bananas and melon. It is regarded as species complex in which various species may be nonpathogenic or pathogenic to various hoists. Pathogen isolates that are harmful to the same host are categorized in to same forma specialis (for example, chickpea pathogen' *F. oxysporum*

f. sp. *ciceris*), and all formae speciales share a common method of infection. Farmer all around the world is facing a threat from *F. oxysporum*, which is causing devastating vascular wilt that is reducing the yield of economically significant crops (Bhutia *et al*., 2023). The pathogen become even more lethal as it expands hosts range of horizontally acquiring lineage-species (LS) genomic sections from several *F. oxysporum* species. It is now consider to being a human pathogen and is ranked fifth out of the top ten fungal pathogens.At first, pathogen enters roots asymptomatically. After that, it invades vascular tissue, causing widespread chlorosis, necrosis and wilting of crop plants particularly in the aerial parts. The isolates of *F. oxysporum* have host specificity of high degree when compared to other *Fusarium* species (Rana *et al*., 2017). When *F. oxysporum* detect a host nearby, it initiates an intricate network of overlapping and interrelated signaling channels. As per several reports, these routes include the cAMP pathway, the G-protein and mitogen-activated protein (MAP) kinase and their downstream signaling components and pathways, the velvet (LaeA/VeA/VelB) complex components, etc. The element of many pathways controls the expression of various genes involved in pathogenecity, giving *Fusarium oxysporum* its virulence.

As will be detailed below, several plants have developed complex and sensitive defense mechanism to fend off the attack of *F. oxysporum*. These mechanisms entail complex cellular processes that are set off by wilt infection and that transducer signals to switch on the response mechanism to defend the cells from infections. To understand the gene network involved during *F. oxysporum* infection in modulating the resistance mechanism, model crop Arabidopsis thaliana and an important host crop of *F. oxysporum i.e.* tomato have been employed. Thus a comprehensive grasp of the molecular mechanism behind the pathogenesis of *F. oxysporum* as well as the ability to identify the type of resistance present in resistant cultivars will be necessary for the proper management of the wilt diseases (Swarupa *et al*., 2014). Aspects of the genetics of plant signaling network and *F. oxysporum* that effectively evade wilt are outlined in this popular article.

Signal Perception and activation of defense mechanism by plants against *F. oxysporum*.

Plants are able to defend themselves against *Fusarium* attack. By monitoring cellular integrity, they are able to detect a variety of nonself chemicals released by *F. oysporum* in addition to autocrine signals. The underlying mechanisms of host defense vary amongst crops. The *F. oxysporum* faces the plant cell wall as first barrier during an attack, which establishes the fundamental strength of the plant and acts to impede it. The cell wall of some resistant banana cultivars confers resistance to *F. oxysporum* Foc TR4 strains prior to colonization

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(Chen *et al*., 2019). When compared to sensitive varieties, the quality of spores adhered to the roots of resistant banana cultivars is significantly lower. Many genes play vital role in separate steps of the cellulose biosynthesis pathways and shikimate phynylpropanoid-lignin biosynthesis pathways - such as 4-coumarate-CoA ligase, 3-deoxy-d-arabino-heptulosonate synthase, glutathione S-transferase (GST), polyphenol oxidase, cellulose synthase and UDP-glucuronic acid decarboxylase have been reported to be upregulated in crops like wilt resistant banana varieties and found be involved in strengthening the cell wall. The increase resistance and decrease spore attachment may be due to the stronger cell wall.

Plants are able to identify chemicals produced from microbial surface that are known as microbes or pathogen associated molecular patterns (MAMP or PAMP, respectively) basically through PRR *i.e.* pattern recognition receptors. When PAMP is bound to specific PRR, these receptors become active, transmitting the signal to pathways of convergent signaling that in turn cause broad- spectrum immunity. By initiating the generation of ROS (reactive oxygen species) and by encoding PR (pathogenesis-related) proteins as well as other molecules involved in defense mechanism, these PRR stimulate the plant's baseline resistance response. Against *F. oxysporum,* the initial defense response is the release of nonspecific elicitors (endogenous and exogenous origin), such as monomeric or oligomeric pieces of the cell wall and the cuticle, which trigger defense responses (innate). When *F. oxysporum* penetrates pectin layers, pieces containing galacturonic acid are released. By fortifying defensive barriers, these pieces serve as molecules of signaling that regulates defense mechanism and transmit resistance. The defense signaling pathway's CERK1 (Chitin elicitor receptor kinase) and the CEBiP (Chitin elicitor-binding protein) components are responsible for recognizing chitin oligosaccharides; resistance crop types have elevated expression of these proteins' genes. As part of the defense reaction to pathogens' cell wall derived elicitors, the phenolic pathway is also triggered. Phenylalanine ammonia lyase activity is induced by *F. oxysporum* oligosaccharide derivatives specifically. After infection by *F. oxysporum*, Arabidopsis plants produce a number of defense genes, including cystien rich, basic, small antimicrobial protein called thionins like Thi2.1 the plant defensin gene Pdf1.2 and PR proteins such as PR-1, PR-5, etc.

Perception of host by pathogen and the initiation of virulence

For *F. oxysporum* to cause a vascular infection, the host must be perceived. Once it has identified a suitable host, the pathogen enters plant tissues and begins to grow. During penetration, it effectively avoids basal defense and plant nonself-recognition. Cutin's

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distinctive dihydroxy-C16 and trihydroxy-C18 acids function as particular inducers, providing the fungus with a failsafe way to detect when it is in touch with the plant (Husaini *et al.*, 2018). *Fusarium* species sense chemical and physical cues from their hosts and react by undergoing the necessary morphogenetic and metabolic changes to become pathogenic. Moreover, glucanase and chitinases, two host-derived hydrolases, produces an elicitor, signaling the fungus to produce the cutinases, endopolygalacturonases, pectinases, etc., necessary for host penetration. During host infection, *Fusarium* spp. activates many signaling cascades. Their primary mechanism of activation is the perception of signals by various receptor proteins. The infection mechanisms in *F. oxysporum* are regulated by the membrane proteins Sho1 and Msb2. By controlling the Fmk1 pathway, the transmembrane proteins Msb2, which is highly glycosylated, and Sho1, which is tetraspan, contribute to plant infection. Sho1 and Msb2 work together upstream of Fmk 1 and are necessary for the complete phosphorylation of Fmk 1. Sho1 and Msb2 have many roles in virulence: they (i) contribute to stress response by the cell wall (ii) increase the function Fmk 1-dependent virulence and (iii) upregulate expression of the genes involved in cell wall synthesis which are regulated Fmk1. Along with Fmk1, membrane proteins Sho1 and Msb2 are necessary for the up-regulation of the genes fks1, chsV and gas involved in cell wall synthesis.*Fusarium spp.* 's Rho1 RHO-type GTPase is necessary for both virulence and proper cell wall construction. Rho1 plays a crucial function in preserving the hyphal architecture, which keeps the glucan synthase from being detected by the host, and it also favorably modulates the posttranslational activity of the enzyme. Essential elements that mediate cellular responses to external stimuli are GTP-binding proteins. The G protein coupled receptor can activate G protein by changing its shape in response to an external signal molecule attaching to receptor. The G protein is affixed to the plasma membrane's cytoplasmic face. Genes encoding β subunit (Gβ proteins FGB1, FGA1 and FGA 2) of G protein are present in *F. oxysporum*. Pathogenicity is caused by changes in cAMP levels and adenyl cyclase activity, which are regulated by FGA1 and FGA2. When FGA1 and FGA2 are disrupted, pathogenicity is decreased and colony form is changed.

Conclusion

Fusarium wilt caused by several strains of *Fusarium oxysporum* is one of the devastating diseases of crop plants. The pathogen survives in the soil and disseminates its infection into the host plants. During the process of infection, complex network of host pathogen interactions takes place leading to susceptibility or resistance mechanism activation of the host plants. Several genes encoding different type's proteins and enzymes in host plant

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cells are activated upon infection that are involved from perception to response mechanisms in host cells. Understanding of these complex mechanisms imparting resistance against Fusarium wilt is vital in developing crop plants resistant to this pathogen.

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