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MOLECULAR BASIS OF PLANT-FUNGAL PATHOGEN INTERACTION AND ITS SIGNIFICANCE IN MANAGEMENT OF PLANT DISEASES: A REVIEW

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ABSTRACT

Plant-pathogen interactions are very complex and multifaceted and plant-fungal pathogen interactions are one of them. These are two way interactions between the attacking fungal pathogen and the host plant. In plant- fungal pathogen interaction, different studies revealed that plants have developed multiple resistance responses at several levels against the invading pathogen in due course of time and on the contrary, fungal pathogens also adapt and produce different molecules to counteract plant defense mechanism and proliferate into the host cells. There are two crucial pathways of plant immune response, which includes the pathogen-associated molecular patterns (PAMP)-triggered immunity (PTI) and effector-triggered immunity (ETI). Since PTI and ETI are the two main routes underlying plant immunity, it would be highly useful to comprehend their specifics and the significant components in these cascades. Although, many theories and models have been proposed to address these interactions, none of them are exhaustive and fully understood. Therefore, it is essential to make a comprehensive summery of the existing plant-fungal pathogen interaction models and map out their involvement related to plant protection. This review focuses to understand these complex interactions, which can facilitate the unraveling of the involvement of different plant resistance pathways and the molecular mechanisms of plant immune responses against fungal-pathogens.

Key words : Fungal pathogen, Interaction, PAMP, PTI, ETI.

Introduction

A very adaptable class of eukaryotic heterotrophic organisms, fungi has effectively colonized the majority of natural habitats. However, phytopathogenic fungi are one of the primary cause of infectious diseases that affect crop plants. They not only cause devastating epidemics but also cause persistent and significant annual crop yield losses, because of whichfungal plant diseases have been considered as a serious economic factor by the scientists, researchers, plant breeders, as well as farmers. Approximately 300,000 species of flowering plants are attacked by pathogenic fungi. Fungal pathogens colonize plants in different ways to obtain nutrients. Biotrophic fungi feed only in living plant tissues anddo not kill the host, preventing cell death and manipulating plant metabolism by secreting effector molecules whereas necrotrophic fungi infect living tissues, continuously produce hydrolytic enzymes and secrete toxins to kill plant cells and acquire nutrients. Hemibiotrophs initially extract nutrients from living tissues before switching to a necrotrophic phase (Tang *et al*., 2022).

Fungi generally secrete numbers of hydrolytic enzymes, including cutinases, cellulases, pectinases, and proteases to access plant tissue. Because these enzymes are also required for the saprophytic lifestyle, they are unlikely to represent the tools specifically developed by fungi to implement pathogenesis, and each individual hydrolytic enzyme may not be absolutely necessary for penetration (Martin *et al*., 2017). The secreted molecules, including secondary metabolites are critical for intercellular communication, it is likely that both plants and their associated fungi have evolved to adopt identical

secretion processes, namely conventional and unconventional mechanisms, allowing them to combat and counterattack during pathogenesis (Vincent *et al*., 2020).

Principle and Mechanism of Plant-Fungal Pathogen Interaction

The specificity of plant-fungal pathogen interactions starts even before a fungal pathogen actually invades or attacks upon a plant. The fungus follows host specificity, and mostly attacks those plants which fall within their compatible range. The plant-pathogen interactions are complex and fine-tuned biochemical processes occurring inside both plants and pathogens. Thus, almost all of these interactions are two-way communications between the attacking pathogen and the host plant (Boyd *et al*., 2013). The invading pathogen tries to escape or out run the plant defense responses and thus, creating a suitable environment for the disease progression. On the contrary, the host plant tries to trigger the defense responses by recognizing the pathogen or its effector molecules to neutralize the pathogen attack. The communications during the pathogen invasions and the triggered plant immunity against them are mainly divided into two types such as pathogen-associated molecular patterns (PAMP)-triggered immunity (PTI) and effector triggered immunity (ETI) (Jones and Dangl, 2006). PAMPs are usually highly conserved; vital components of pathogens, chitin in fungi, can be recognized by plant receptors and thus, a defense response can be induced (Boller and Felix, 2009). However, ETI is triggered by recognizing the effector molecules, often regarded as the avirulence (Avr) proteins secreted by the pathogen, by the resistant (R) genes of the plants. Thus, ETI works on the basis of R-Avr gene interactions which is commonly referred as "gene-for-gene resistance" (Kumar *et al*., 2021).

- Crop plants are endowed with numerous defense genes which encode various proteins responsible for synthesis and accumulation of defense arsenals in plants.
- The defense genes are involved in production of antimicrobial compounds like phenolics, phytoalexins, pathogenesis related proteins and active oxygen species or involved in reinforcement of cell wall by accumulating hydroxyproline-rich glycoproteins, callose, lignin and wall-bound phenolics.

When a fungal pathogen come into contact with the plants the defense genes are activated by releasing signals. These signals are transferred to the plant nucleus through signal transduction pathway activating defense genes.

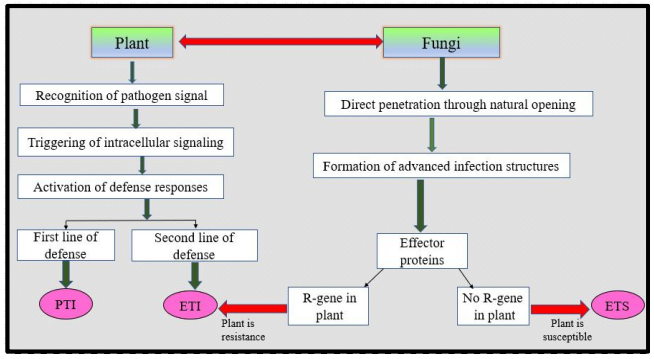

Fig. 1 : A flow chart of the interaction between the fungal pathogen and plant immune response.

Defense Strategies

Plant Defense Strategies

First line of defense (Non-specific defense response): Glucans and chitin are the, comprehensively explored pathogen associated molecular patterns (PAMPs) or microbial- associated molecular patterns (MAMPs) of plant pathogenic fungi (Boyd *et al.*, 2013). One of the initial events of PTI is perceiving the stimulus of pathogen attack by the recognition of PAMPs/MAMPs *via* plant patternrecognition receptors (PRRs) (Bigeard *et al.*, 2015). PRRs are localized on the surface of plant cells and function as immune receptors. PRRs of plant cells are usually either receptor kinases (RKs) or receptor-like proteins (RLPs) (Zipfel, 2008). To penetrate through different structural barriers of plants, pathogens secret lytic enzymes that degrade plant cell components. These cell wall fragments act as endogenous elicitors and induce plant defense responses and termed as damage-associated molecular patterns (DAMPs). Upon recognition of a PAMP at the cell membrane, the immune receptor complexes are formed and induce several auto and trans-phosphorylation reactions downstream. One of the earliest physiological responses upon PAMP/ MAPM detection is calcium (Ca^{2+}) and oxidative bursts. Ca^{2+} burst is initiated by the influx of extracellular Ca^{2+} ions into the cytosol, which occurs within minutes of

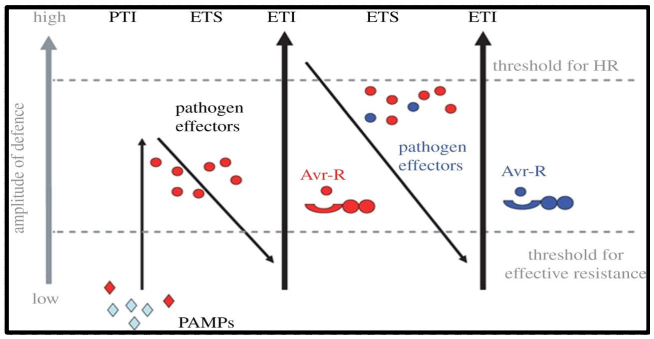

Fig. 2 : A zigzag model of the plant immune system (Shen *et al*., 2017).

Table 1 : Classes of antifungal proteins.

Cass	Occurrence	Major characteristic	Mechanism of Action
PR-1 proteins	Plants	Molecular masses of 15–17 kDa. Homology to the superfamily of cysteine-rich proteins	Unknown
β-Glucanases	Microorganisms, plants, invertebrates and vertebrates	1,3-β-Endoglucanase activity	Hydrolysis of the structural 1,3-β glucan present in the fungal cell wall
Chitinases	Viruses, bacteria, fungi, snails, fish, plants, insects, mammals and amphibians	Chitinase activity. Molecular masses of 26–43 kDa	Cleave cell wall chitin polymers *in situ*
Chitin-binding proteins	Bacteria, plants, insects and crustaceans	Molecular masses of 3.1–20 kDa. Chitin-binding proteins	Binding to chitin
Thaumatin-like proteins	Plants	Molecular masses ~22 kDa. Share significant amino acid homology to thaumatin	Not completely understood. Some cause fungal cell permeability changes, others bind to 1,3-β-glucan and exhibit 1,3-β-glucanase activity
Defensins/thionins	Mammals, fungi, insects and plants	Low-molecular-mass, cysteine-rich proteins	Fungal inhibition probably occurs through an ion efflux mechanism
Cyclophilin-like proteins	Bacteria, plants, animals and fungi	Example: mungin	Unknown
Glycine/histidine-rich proteins	Insects	Extremely rich in glycine and histidine, which may comprise as much as 80% of the amino acids	Unknown
Ribosome-inactivating Proteins (RIPs)	Fungi and plants	RNA N-glycosidases that depurinate rRNA	Inactivate fungal ribosomes in vitro and, presumably, *in situ*
Lipid transfer proteins (LTPs)	Mammals, plants, fungi and bacteria	Molecular masses of ~8.7 kDa	Unknown

Ferreira *et al.* (2007).

PAMP perception by the PRRs(Jeworutzki *et al*., 2010).

Second line of defense(Specific defense response): The membrane-bound PRRs perceive the invading pathogens or PAMPs and trigger PTI, which seizes further colonization or spreading of infection. Conversely, at times, pathogens can successfully dodge the PTI responses and deploy effectors those contribute to pathogen virulence (Jones and Dangl, 2006). This results in the pathogen proliferation causing the effector-triggered susceptibility (ETS). On the other hand, plants have evolved different sets of receptors such as resistance (R) proteins which can efficiently detect the pathogen-generated effectors and initiate immune responses. These cytosolic immune receptors usually contain nucleotide binding (NB) and leucine rich repeat (LRR) domains (NLRs) and recognize the pathogen-delivered effector proteins and trigger effector-triggered immunity (ETI) (Elmore *et al*., 2011). The effector molecules produced by pathogens are encoded by specific sets of genes known as avirulence (Avr) genes.

Fungal Defense Strategies

Plant pathogenic fungi employ various strategies to avoid triggering host defense response; *e.g.,* formation of the advanced infection structures and limited secreted amounts of lytic enzymes. In plant-pathogen interactions, the ability of the fungal spore to adhere to the host plant is considered a fundamental requirement for spore germination, germ tube elongation, and appressorium formation (Tucker and Talbot, 2001). Chemical or physical signals from the host plant including ethylene signals (Sharma and Gautam, 2019), topographic signals (Jones *et al*., 2001), cutin monomers and substratum

hydrophobicity (Kamakura *et al*., 2002) trigger the formation of appressoria. For example, in wheat rust the dikaryotic urediospores germinate within a short time once they land on the wheat epidermis when the humidity meets the germination requirements and the germ tube extends perpendicular to the vein and is directed toward stomatal cells of the leaf surface. An appressorium then forms above the stomata, followed by the development of a penetration peg to invade the host (Voegele *et al*., 2009).

Secretes Effector Proteins: For successful infection and establishment of compatible interactions leading to proliferation, pathogenic fungi must counteract PTI (Dangl and Jones, 2001) to evade the host defense system and to facilitate this, the pathogen secretes several effector proteins. The pathogen's effector proteins are distinctive recognition targets for the host defense system, whereas components of the host defense signals or their receptors are the main target of effectors during infection. Effectors are considered as a group of proteins without mutual conservation, that are uniquely responsible for some crucial functions such as tethering the host defense machinery by producing cytotoxicity to facilitate pathogen progression (Jaswal *et al*., 2020).

Importance of Plant Fungal Pathogen interaction in Plant Disease Management

Numerous individual plant resistance (R) genes have already been characterized and are being efficiently used in crop improvement research programs. Using plant resistance genes for developing disease resistant varieties is a convenient alternative for chemical control methods employed to protect crops from diseases. Application of metabolomics in plant pathogenic fungi research can help to understand the pathogenesis of pathogenic fungi as well as plant defense mechanisms and eventually help to develop new strategies for fungal diseases management (Chen *et al*., 2019). Proteomics analysis has been widely subjected to understand defense mechanism between crops (wheat, tomato, strawberry and mint etc.) and their fungal pathogen interaction. In recent years, the RNA interference (RNAi) mechanism has become one of the

Table 2 : Biotrophic plant fungal pathogens effectors involved in the manipulation of host reaction.

Pathogen	Effector protein	Host	Localization	Function in virulence
Pgt	PgtSR1	Wheat	Unknown	Suppresses RNA silencing in plants and impedes basal plant defense by altering the abundance of small RNAs that serve as defense regulators
	AvrSr35	Wheat	Colocalize in the ER	Suppresses cell death signaling activities
	AvrSr50	Wheat	Cytosol and nucleus	Suppresses cell death signaling activities
Pst	Pst-4	Wheat	Cytoplasm	Disrupts sorting of chloroplast protein TaISP thereby suppressing host ROS accumulation
	Pst55	Wheat	Cytoplasm	Disrupts sorting of chloroplast protein TaISP, thereby suppressing host ROS accumulation
	PSEC2	Wheat	Cytoplasm and nucleus	Suppresses PTI-related callose deposition
	PSEC17	Wheat	Cytoplasm and nucleus	Suppresses PTI-related callose deposition
	PEC6	Barley	Cytoplasm and nucleus	Interacts with adenosine kinases (ADKs) with generic functions to suppress PTI
Pt	Pt3 and Pt27	Wheat	Unknown	Function in avirulence against wheat leaf rust in resistant genotypes
	Pt18906	Wheat	Nucleus and cytoplasm	Acts in the cytoplasm and may cause accumulation of reactive oxygen species and callose in TcLr10 + 27 + 31
Bgh	CSEP0027	Barely	Cytosol and nucleus	Interacts with HvCAT1 to regulate host immunity regulation and probably ROS homeostasis to promote virulence during infection
	BEC1019	Barley	Cytosol and nucleus	Essential for virulence
		Wheat		Essential for haustorial formation
Bgt	AvrPm2	Wheat	Cytoplasm and nucleus	Suppresses the recognition of Avr
	SvrPm3a1/f 1	Wheat	Cytoplasm and nucleus	Facilitates evasion of pathogen recognition by R genes

Mapuranga *et al*. (2022)

main areas of focus in plant-fungal pathogen interaction studies and has been implicated as one of the major contributors to comprehend fungal pathogenicity (Jesenicnik *et al.*, 2022). Rapid activation of localized defense responses at the site of pathogen infection, often associated with a hypersensitive response (HR), is the most prevalent and effective mechanism used by plants to minimize pathogen attack. By combining R and Avr gene expression in a single plant genotype, it is possible to engineer a 'trigger' for hypersensitive response (HR).

Recent investigations of plant-fungus interactions demonstrate two parallel but complementary disease management strategies: firstly, beneficial fungi (e.g., *Trichoderma* spp.) that colonize roots can prime the host for systemic induced resistance (ISR) by elevating defence-related gene expression, phenolic compounds and PR proteins. Secondly, classical R-gene mediated resistance (ETI) against fungal pathogens continues to underpin breeding based control of major diseases such as vascular wilts. Integrating these approaches, for example by combining biocontrol or priming agents with R-gene bearing cultivars, may enhance durability of resistance, broaden spectrum and reduce reliance on fungicides.

Beneficial fungus inducing ISR against a fungal pathogen: In a glass house study, seed treatment of rice with isolates of *Trichoderma harzianum* and *Trichoderma virens* significantly suppressed sheath blight disease caused by *Rhizoctonia solani* and concurrently increased total phenol content of plant tissues. For example, one isolate (PB 22) achieved a disease severity of only 13.6% with a phenol content of 394 µl/g (168 h after pathogen inoculation), while other effective isolates achieved phenols ~ 440 - 466 µl/g and >34 % disease reduction. The elevated phenolic levels imply activation of plant defence metabolism, consistent with induction of systemic resistance (ISR) by the *Trichoderma* treatment (Sharma, 2017). This demonstrates that root or seed applied beneficial fungi can prime plant defence responses (e.g., increased phenolics, possibly PR proteins) and thereby reduce incidence or severity of fungal disease. Incorporating such a biocontrol and priming approach offers a sustainable disease management strategy complementing chemical fungicides.

Beneficial fungus triggers defence gene up regulation in tomato: Root or soil application of *Trichoderma pubescens* strain Tp21 to tomato plants challenged with *Rhizoctonia solani* (strain R11) significantly reduced the disease index (DI) to 16% versus 78.7% in untreated infected controls. At 15 days post inoculation, the relative expression levels of three defence related genes, PAL (phenylalanine ammonia lyase), CHS (chalcone synthase) and HQT (hydroxycinnamoyl-CoA quinate hydroxycinnamoyl transferase) were increased by 2.72, 4.44 and 3.72 fold, respectively, compared to control (Behiry *et al.*, 2023). This study illustrates a mechanistic link between beneficial fungus colonization and induction of host defence gene expression (presumably SAR or ISR pathways) that contributes to enhanced resistance to a fungal pathogen. It supports the argument that beneficial fungi mediated priming (including up regulation of defence genes) is a valid disease management tool.

R-gene mediated resistance in tomato against a fungal vascular pathogen: The tomato (*Solanum lycopersicum*), *Fusarium oxysporum* f. sp. *lycopersici* (Fol) vascular wilt system provides a robust example of gene for gene

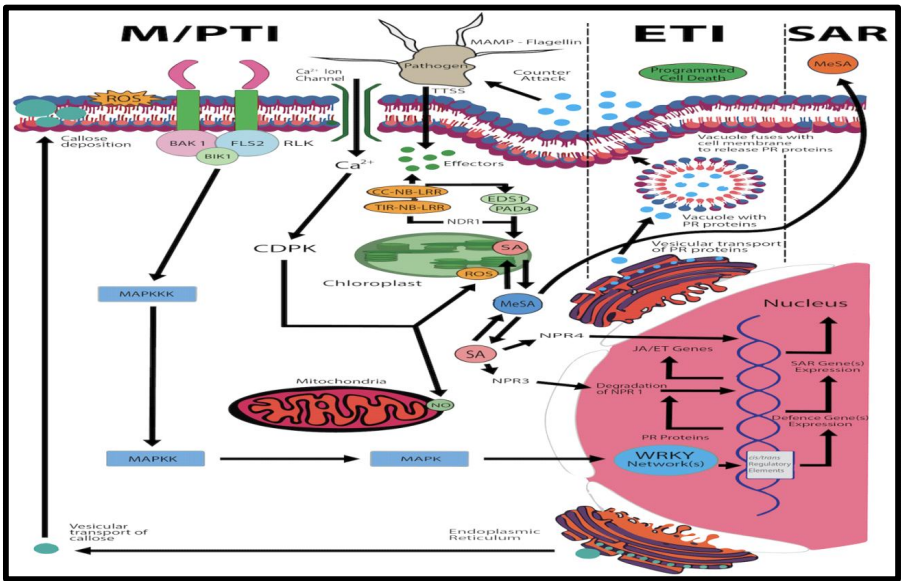

Fig. 3 : Diagram showing the cellular dynamics of the plant immune response (César J. Chiquito-Contreras *et al.*, 2024).

resistance. Four R genes (I-1, I-2, I-3, I-7) have been identified, expressed in roots, which upon recognition of cognate fungal Avr effectors initiate immunity. Recent study also showed that an I-2 resistant rootstock able to protect a susceptible scion from *Fusarium* wilt symptoms even though the fungus colonized the scion's xylem vessels. Proteomic analysis of xylem sap revealed accumulation of a common set of four PR (pathogenesis related) proteins (PR-5x, PR-P2 and two glucan endo-1,3 β D glucosidases) across all four R gene lines (Simkovicova *et al.*, 2024). Another earlier report demonstrated that Fol can colonize stem tissues even in incompatible (resistant) interactions, but the extent of colonization is less in plants carrying I or I-3 compared to I-2, suggesting differences among R genes in restricting pathogen spread (de Lamo *et al.*, 2018). This case underlines how R gene mediated (effector triggered immunity, ETI) mechanisms can limit fungal colonization and disease development, forming a cornerstone of disease management breeding strategies. Furthermore, the finding of mobile root derived defence proteins in xylem underscores systemic aspects of immunity beyond local HR.

Conclusion

In-depth research on diverse plant-pathogenic fungal species is the basis for understanding plant diseases and studies on pathogenic mechanisms, disease infection cycles, and effective disease control. However, there is need for systematic research on identification of signaling mechanism and defense-response activation processes, and little is known about the disease-resistance characteristics and mechanisms of major crops. Thorough understanding and characterization of the different physiological and genetic processes involved in plant-pathogen interaction and exploring more on the phyto-pathosystems will pave ways for exploiting these phenomena in crop protection and improvement. Efficient application of functional genomics tools for disease resistance will not only help better understanding the plant defense signaling, but also reveal novel insights on the interactions between these signaling pathways and other plant processes.

Future Prospects

The continuous increase in the human population and public concern over the generalized use of chemical fungicides are associated with the increasing number of obsolete fungicides that derive from the development of fungal resistance and this demands alternative ways for disease management. Emerging evidence suggests that during a plant-fungal pathogen interaction, the pathogen may take over selected aspects of plant gene expression to its own benefit. Therefore, it is likely that the fungus may induce the expression of some components required for the infection or development processes or repress components of the host defense system. In this context, a detailed understanding of the molecular events that take place during a plant-fungal pathogen interaction is a prerequisite for boosting the natural inherent defenses of plants, and to transfer defensive traits into the genome of economically important crops. To effectively control the occurrence of and harm from fungal diseases in plants, research needs to be continued on a number of related topics. Identification of characteristics and signaling pathways, regulation of defense response activation processes, and isolation and functional verification of pathogenic fungal effectors will surely lay a theoretical foundation for the development of new approaches for controlling pathogen damage and selecting resistant varieties. Genome of disease-resistant plant and genome-wide association studies are the need of the hour. With the efficient identification of disease resistance genes, their diversity can be fully utilized for the breeding of plants for disease resistance. Studying fungal pathogenic toxins and their mechanisms of pathogenicity is of great significance for understanding the interaction between plant hosts and pathogens, as well as for the use of pathogenic toxins to identify plant disease resistance, to screen for disease-resistant mutants and to manage diseases.

References

Behiry, S., Soliman S.A., Massoud M.A., Abdelbary M., Kordy A.M., Abdelkhalek A. and Heflish A. (2023). *Trichoderma pubescens* elicit induced systemic resistance in tomato challenged by *Rhizoctonia solani*. *J. Fungi*, **9(2)**, 167.

Bigeard, J., Colcombet J. and Hirt H. (2015). Signaling mechanisms in pattern-triggered immunity (PTI). *Molecular Plant*, **8**, 521–539.

Boller, T. and Felix G. (2009). A renaissance of elicitors: perception of Microbe–associated molecular patterns and danger signals by pattern-recognition. *Ann. Rev. Plant Biol.*, **60**, 379-406.

Boyd, L.A., Ridout C., O'Sullivan D.M., Leach J.E. and Leung H. (2013). Plant–pathogen interactions: disease resistance in modern agriculture. *Trends in Genetics*, **29(4)**, 233-240.

Chen, F., Ma R. and Chen X.L. (2019). Advances of metabolomics in fungal pathogen–plant interactions. *Metabolites*, **9(8)**, 169.

Chiquito-Contreras, C.J., Meza-Menchaca T., Guzmán-López O., Vásquez E.C. and Ricano-Rodriguez, J. (2024). Molecular insights into plant–microbe interactions: A comprehensive review of key mechanisms. *Frontiers in Bioscience-Elite*, **16(1)**, 9.

Dangl, J.L. and Jones J.D.G. (2001). Plant pathogens and integrated defense responses to infection. *Nature*, **411**, 826–833. doi: 10.1038/35081161

de Lamo, F.J., Constantin M.E., Fresno D.H., Boeren S., Rep M. and Takken F.L. (2018). Xylem sap proteomics reveals distinct differences between R gene-and endophyte-mediated resistance against Fusarium wilt disease in tomato. *Front. Microbiol.*, **9**, 2977.

Elmore, J.M., Lin Z.J.D. and Coaker G. (2011). Plant NB-LRR signaling: Up streams and down streams. *Curr. Opin. Plant Biol.*, **14**, 365–371.

Ferreira, R.B., Monteiro S.A.R.A., Freitas R., Santos C.N., Chen Z., Batista L.M. and Teixeira A.R. (2007). The role of plant defense proteins in fungal pathogenesis. *Mole. Plant Pathol.*, **8(5)**, 677-700.

Jaswal, R., Kiran K., Rajarammohan S., Dubey H., Singh P.K. and Sharma Y. (2020). Effector biology of biotrophic plant fungal pathogens: current advances and future prospects. *Microbiol. Res.*, **241**, 126567. doi: 10.1016/j.micres.2020. 126567.

Jesenicnik, T., Stajner N., Radisek S., Mishra A.K., Kosmelj K., Kunej U. and Jakse J. (2022). Discovery of microRNA-like small RNAs in pathogenic plant fungus Verticillium nonalfalfa using high-throughput sequencing and qPCR and RLM-RACE validation. *Int. J. Mole. Sci.*, **23(2)**, 900.

Jeworutzki, E., Roelfsema M.R.G., Anschutz U., Krol E., Elzenga J.T.M., Felix G., Boller T., Hedrich R. and Becker D. (2010). Early signaling through the Arabidopsis pattern recognition receptors FLS2 and EFR involves Ca_2^+-associated opening of plasma membrane anion channels. *Plant J.*, **62**, 367–378.

Jones, H., Whipps J. and Gurr S. (2001). The tomato powdery mildew fungus *Oidium neolycopersici*. *Mol. Plant Pathol.*, **2**, 303–309.

Jones, J.D.G. and Dangl J.L. (2006). The plant immune system. *Nature*, **444**, 323–329

Kamakura, T., Yamaguchi S., Saitoh K., Teraoka T. and Yamaguchi I. (2002). A novel gene, CBP1, encoding a putative extracellular chitin-binding protein, may play an important role in the hydrophobic surface sensing of Magnaporthe grisea during appressorium differentiation. *Mol. Plant Microbe Interact*, **15**, 437–444. doi: 10.1094/mpmi.2002.15.5.437

Kumar, G. and Nanda S. (2021). Molecular perspective of plant-pathogen interaction: An overview on plant immunity. *Biological Forum – An Int. J.*, **13(1)**, 48-53.

Mapuranga, J., Zhang N., Zhang L., Chang J. and Yang W. (2022). Infection strategies and pathogenicity of biotrophic plant fungal pathogen. *Front. Microbiol.*, **13**, 799396.

Martin, F.M., Uroz S. and Barker D.G. (2017). Ancestral alliance: Plant mutualistic symbioses with fungi and bacteria. *Science*, **356** (6340), 9.

Sharma, K.K. (2017). Induction of systemic resistance (ISR) against sheath blight of rice caused by *Rhizoctonia solani* Kuhn using biological seed treatment with Trichoderma. *J. Appl. Nat. Sci.*, **9(2)**, 123-130.

Sharma, N. and Gautam A. (2019). Early pathogenicity events in plant pathogenic fungi: A comprehensive review. *Biol. Forum Int. J.*, **11**, 24–34.

Shen, Y., Liu N., Li C., Wang X., Xu X., Chen W., Xing G. and Zheng W. (2017). The early response during the interaction of fungal phytopathogen and host plant. *Open Biology*, **7(5)**, p.170057.

Simkovicova, M., Kramer G., Rep M. and Takken F.L. (2024). Tomato R-gene-mediated resistance against *Fusarium* wilt originates in roots and extends to shoots via xylem to limit pathogen colonization. *Front. Plant Sci.*, **15**, 1384431.

Tang, Y., He G., He Y. and He T. (2022). Plant Resistance to Fungal Pathogens: Bibliometric Analysis and Visualization. *Toxics*, **10(10)**, 624.

Tucker, S.L. and Talbot N.J. (2001). Surface attachment and pre-penetration stage development by plant pathogenic fungi. *Annu. Rev. Phytopathol.*, **39**, 385–417.

Vincent, D., Rafiqi M. and Job D. (2020). The multiple facets of plant–fungal interactions revealed through plant and fungal secretomics. *Front. Plant Sci.*, 10- 1626.

Voegele, R.T., Hahn M. and Mendgen K. (2009). The uredinales: "cytology, biochemistry, and molecular biology". *Plant Relationships*, 69-98.

Zipfel, C. (2008). Pattern-recognition receptors in plant innate immunity. *Curr. Opin. Immunol.*, **20**, 10–16.