Biological Forum – An International Journal **13(1): 48-53(2021)**

ISSN No. (Print): 0975-1130
ISSN No. (Online): 2249-3239

Molecular Perspectives of Plant-Pathogen Interactions: An Overview on Plant Immunity

Gagan Kumar and Satyabrata Nanda
MS Swaminathan School of Agriculture,
Centurion University of Technology and Management, Odisha, India.

(Corresponding author: Satyabrata Nanda)
(Received 12 November 2020, Accepted 15 February, 2021)
(Published by Research Trend, Website: www.researchtrend.net)

ABSTRACT: In nature, there is a constant arm race is going on in between plants and pathogens. These plant-pathogen interactions are complex and multifaceted. To tackle the invading pathogens, plants have developed multiple resistance responses at several levels. On the contrary, adapting capabilities and evolution of new effector molecules help the phytopathogens to outrun plant defenses and proliferate in the host cells. 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-pathogen interaction models and delineate their intricacy related to plant protection. In this review, two crucial pathways of plant immune response, including the pathogen-associated molecular patterns (PAMP)-triggered immunity (PTI) and effector-triggered immunity (ETI) has been discussed elaborately. As both PTI and ETI are the major pathways involved in plant immunity, understanding their details and the key players involved in these cascades will be highly beneficial. In addition, a comparative discussion of the different models proposed for understanding the ETI has been presented. Understanding of these complex interactions can facilitate the unravelling of the involvement of different plant resistance pathways. Moreover, the review will serve as a basic layout to have an overview of the molecular mechanisms of plant immune responses against phytopathogens.

Keywords: Plant immunity, PTI, ETI, plant-pathogen interactions

INTRODUCTION

Being sessile, plants are constantly exposed to an array of biotic stresses including bacteria, fungi, and nematodes. The plant homeostasis is challenged by these pathogen invasions (Sharma and Gautam, 2019). However, plants don't possess a well characterized and systematic immune system, like in animals to overcome such stresses. Plans employ their survival strategies against the biotic stresses, which is further fine-tuned by several lines of defense. Epidermis, the outermost layer of plants, operates as a corporal wall for the external stress and threats. Further, deposition of lignin, resins or silica on the epidermal layer, and/or development of modified leaves such as trichomes, spines, thrones and prickles restrict pathogen invasion. Plants deploy the use of secondary metabolites as the second line of defense against the invading pathogens and their effector molecules. Hypersensitive responses (HR), programmed cell death, tissue reinforcement at the site of infection and expression of defense-related proteins are often regarded as the third line of defense by plants in response to pathogen or herbivore attacks (Nanda *et al.,* 2021). The induced local responses at the site of infection followed by the establishment of immune response throughout the plant known as the systemic acquired resistance (SAR). SAR gives the plant the long lasting and broad-spectrum pathogen defense capability (Klessig *et al.,* 2018). In addition to this, plant defense is significantly monitored by the resistance genes (*R* gene). The pathogen attacks result in oxygen burst inside the cell thereby releasing intermediate signal molecules such as reactive oxygen species (ROS), superoxides (O_2^-), nitric oxide (NO) and hydrogen peroxide (H_2O_2), which in turn induces the defense responses through activation of downstream targets (Wang *et al.,* 2013). Similarly, several phytohormones like abscisic acid (ABA), salicylic acid (SA), jasmonic acid (JA), ethylene (ET) and gibberellins (GA) also regulate the defense responses and modulate the expression of several downstream target genes (Davière and Achard, 2013; Shin *et al.,* 2014). Additionally, calcium-dependent protein kinase (CDPK), cyclin-dependent protein kinase (CDK) and mitogen activated protein kinase (MAPK) serve as an important component of the defense signaling cascades (Kitsios and Doonan, 2011; Hettenhausen*et al.,* 2015). By the fine tuning of all these defense responses plants tackle the pathogen invasions.

A. Mechanism of plant-pathogen interactions

The specificity of plant-pathogen interactions starts even before a pathogen actually invades or attacks upon a plant. The pathogens follow host specificity, and mostly attack 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 an apt 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. In due course of evolution, both plants and pathogen have developed immaculate machinery including metabolites, signaling molecules, and genes fitting for these interactions. 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, such as glycans, flagellin in bacteria, and chitin in fungi, those can be recognized by plant receptors and thus, a defense response can be induced (Boller and Felix, 2009; Maverakis *et al.,* 2015). 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" (Boyd *et al.,* 2013). Both these plant immune responses and their roles during the plant-pathogen interactions are discussed in detail in the followings.

B. Plant-pathogen interaction and PTI

Among the extensive microbial species, some of the comprehensively explored PAMPs or microbial-associated molecular patterns (MAMPs) are flg22 and elf18 of bacterial origin, and glucans and chitin from the 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 pattern-recognition 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). The RKs possess a ligand-binding ectodomain, a single-pass transmembrane domain, and an intracellular kinase domain, whereas RLPs lack an intracellular kinase domain but have the other domains. Due to the non-availability of any intracellular signaling domains, RLPs mostly function in association with RKs to transduce the perceived signal further (Zipfel, 2014).Flagellin Sensing2 (FLS2), a Leucine repeat receptor kinase (LRR-RK) from *Arabidopsis* that recognizes and binds to the bacterial PAMP flg22 (Boller and Felix, 2009). Perception of flg22 from the invading pathogen activates immune responses, including H_2O_2 generation, hypersensitive cell death and pathogenesis-related (PR) gene expression (Yu*et al.,* 2017). Similarly, one more LRR-RK, EF-Tu Receptor (EFR) of *Arabidopsis* recognizes EF-Tu and triggers the immune responses via PTI (Zipfel *et al.,* 2006). Plant PRRs also possess the ability to detect the cell wall components or peptides as PAMP signatures during the pathogen attacks (De Lorenzo *et al.,* 2011). Chitin, a major compound of fungal cell walls is recognized by Chitin-Elicitor Binding Protein (CEBiP) in rice. CEBiP is a LysM domain-containing receptor-like protein (RLP) which requires the RLK Chitin Elicitor Receptor Kinase1 (CERK1) to activate PTI (Miya *et al.,* 2007). The chitin-CEBiP interactions result in activation of defense responses, including reactive oxygen species (ROS) generation, PR gene expression, and phytoalexin biosynthesis. Rice cells having lower CEBiP expression exhibit decreased response to chitin, signifying the pivotal role of CEBiP in chitin perception and subsequent downstream signal transduction (Chen and Ronald, 2011).

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) (Muthamilarasan and Prasad, 2013). The first plant DAMP receptor, PEP receptor1 (PEPR1), has been identified from *Arabidopsis* belongs to the LRR-RK family (Yamaguchi *et al.,* 2010). PEPR1 and PEPR2 detect AtPep1, a danger signal peptidic DAMP in *Arabidopsis*. AtPep1 is a 23-amino-acid peptide generated from the C-terminus of a wound-induced protein PROPEP1, and upon recognition by PERP1/2, it induces the downstream defense signaling. The activation of immune responses by DAMP are very similar to that of PAMPs, which suggests the possible connection between PAMP and DAMP signaling.Additionally, the perception of PAMPs/MAMPs by the PRRs generate some immune receptor complexes that initiate signal transductions triggering PTI. Upon recognition of a PAMP at the cell membrane, the immune receptor complexes are formed and induce several auto- and trans-phosphorylation reactions downstream. Post-recognition of a PAMP/MAMP, BRI1 associated receptor kinase1 (BAK1), Botrytis-induced kinase1 (BIK1) and PBL (PBS1-like) kinases bind to FLS2 and EFR, get rapidly phosphorylated and then get dissociated from the PRR complexes (Zhang *et al.,* 2010). One of the earliest physiological responses upon PAMP/MAPM detection is calcium (Ca^{2+}) and oxidative bursts (Jeworutzki *et al.,* 2010). Ca^{2+} burst is initiated by the influx of extracellular Ca^{2+} ions into the cytosol, which occurs within minutes of PAMP perception by the PRRs. The Ca^{2+}burst further stimulates the opening of other membrane-bound transporters such as NO_3^-, H^+, K^+, resulting in depolarization of the cell membrane (Jeworutzki *et al.,* 2010). In *Arabidopsis*, the membrane-bound *Arabidopsis*-autoinhibited Ca^{2+}-ATPase8 (ACA8) makes a complex with FLS2 and fine tune the intracellular Ca^{2+} levels during MAMP-responsive signal transductions (Frei dit Frey *et al.,* 2012). Similarly, in response to PAMP/MAMP detection, the oxidative burst is produced by nicotinamide adenine dinucleotide phosphate (NADPH)

oxidases. The perception of PAMP/MAMP signatures in apoplast activates the respiratory burst oxidase homolog D (RBOHD), which generates ROS or superoxide (O_2^-) ions (Zhang *et al.*, 2007). Detection of PAMP/MAMP then promotes phosphorylation of RBOHD on different residues by CDPKs and BIK1, making the NADPH oxidase fully activated (Kadota *et al.*, 2014). Both ROS and H_2O_2 has the capacity to regulate the intracellular Ca^{2+} levels and can induce downstream signaling cascades like CDPK or MAPK mediated defense responses.

C. Plant-pathogen interaction and ETI

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. The "zig-zag model" proposed by Jones and Dangl (2006) describes this communication between the pathogen effector molecules and the plant R proteins. 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. The *Avr* gene products, when enter into a plant cell destabilize the cell homeostasis. Subsequently, the plant R proteins perceive these effectors and trigger immune responses referred as *R*-gene mediated pathogen resistance (Nimchuk *et al.*, 2003). These Avr-R protein interactions were first proposed by Flor (1971) and coined as gene-for-gene relationships. Once an effector molecule is detected either by an appropriate R protein, it usually triggers the HR response leading to programmed cell death (PCD) (Nimchuk *et al.*, 2003). The activation of HR or PCD often accompanied by Ca^{2+} burst or oxidative burst (ROS), and defense responsive gene expressions ultimately leading to local and systemic acquired resistance (SAR) (Gururani *et al.*, 2012). The exact mechanism of interaction of pathogenic effectors and plant resistant genes or specific receptor in ETI is debatable. However, some interesting models have been put forwarded to understand these interactions occurring during ETI. To illustrate the R-effector interactions, one such model is the "direct interaction" model. Here, the effector physically interacts and binds to the receptor protein or NB-LRR resistance proteins and triggers defense signals via ETI. The recognition of the effector Avr9 from fungus *Cladosporium fulvum* by the tomato Cf-9 (R protein), supports the direct interaction hypothesis. However, in many cases, the association of R-Avr proteins are not direct, and often assisted by the accessory proteins. In indirect interactions, the effector-R protein binding is facilitated by an accessory protein, which also happens to be a pathogen virulence target or a structural analog. After entering into a plant cell, the effector persuades structural changes of the accessory protein, which is later recognized by the R protein (van der Hoorn and Kamoun, 2008). Another model named as the 'guard' model was proposed to illustrate the R-Avr indirect interactions. According to this model, the R or NB-LRR proteins safeguard an accessory protein referred as "guardee" which is targeted and modified by the pathogen effectors (Dangl and Jones, 2001). Defense signals leading to ETI are generated once the R protein perceives any structural change of its guardee or any attacks on it (McDowell and Woffenden, 2003). The interaction of *Arabidopsis* RPM1 interacting protein4 (RIN4) with RPM1 and Resistance to *Pseudomonas syringae*2 (RPS2) elucidate the guard model. However, the guard model couldn't stand universal for all the indirect interaction of R-Avr proteins and lacked the evolutionary aspects of plant R proteins (Dodds and Rathjen, 2010). Yet another model, describing the possible interaction strategies of R-Avr proteins came up, known as the "decoy" model (van der Hoorn and Kamoun, 2008). As per this model, the independent progression of the target analog or duplication of the target gene will favor the sole participation of the accessory protein in effector recognition. The mechanism of interaction between tomato R protein Prf and pathogenic effector AvrPto supports the postulates of this model. During the interaction between Prf-AvrPto, the NB-LRR protein Prf makes a complex with the accessory protein Pto kinase. Though the decoy model exemplified the role of accessory protein in recognition of effector by the R proteins, it didn't explain its role in activation of R proteins. To further clarify the interactions of effectors and plant R proteins, the "bait and switch" model was proposed (Collier and Moffett, 2009). In this concept, the recognition of an effector molecule is carried away in two steps: first, the effector binds to an accessory protein (bait) associated with a R protein, secondly, the effector is recognized by a NB-LRR R protein triggering the downstream signaling events. Thus, according to this model the R protein directly interacts with the accessory protein or the effector target, rather than interacting with the modified accessory protein. All these afore-discussed models are proposed according to some of the interactions between R-Avr proteins leading to ETI. However, none of the models are universally acceptable and fully understood.

The interactions between effector and R proteins initiates a signal transduction cascade leading to the activation of defense responses (Rout *et al.*, 2014). The plant R proteins are highly polymorphic which helps in recognizing diverse Avr proteins from the invading pathogens. The rice resistant allele Xa27 exhibits induced expression when challenged by bacteria containing the effector AvrXa27 (Gu *et al.*, 2005). Similarly, the resistance gene Ve1 in tomato differs from the closely linked Ve2, in providing defense responses against *Verticillium* spp. (Fradin *et al.*, 2009). Thus, the sequence variations in R genes can lead to

plant resistance or susceptibility against a particular pathogen. The activated R proteins or NLRs instigate a set of immune responses including oxidative burst, ion fluxes, MAPK cascades, accumulation of phytohormones, and transcriptional reprogramming (Buscaill and Rivas, 2014; Nanda *et al.*, 2016). For example, interactions between barley MLA10 protein and transcription factors HvWRKY1 and HvWRKY2 resulted in immunity against powdery mildew infection (Shen *et al.*, 2007). Further, MLA10 also interacts with HvMYB6, a positive regulator of resistance to powdery mildew via its CC domain. MLA10 releases HvMYB6 from HvWRKY1 and promotes its DNA binding activity, thus enhancing the immunity against powdery mildew (Chang *et al.*, 2013). Similarly, in rice, ETI is regulated by the CNL receptor Pb1, which interacts with OsWRKY45 to provide resistance against rice blast fungi (Inoue *et al.*, 2013). During ETI, the recognition of pathogenic effectors is often associated with activation of HR and/or generation of ROS. The generation of ROS during plant-pathogen interactions was first studied in *Phytophthora infestans*—potato interactions. Till date, numerous studies have revealed the role of ROS and HR in plant defense during plant-pathogen interactions. The WRKY53 transcriptional network mediates ROS generation and oxidative responses during interactions between AvrRxo1 and R protein in *N. benthamiana* plants (Triplett *et al.*, 2016). Albeit defense responses like ROS production, Ca^{2+} bursts, and protein kinase signaling are shared by PTI and ETI, but the kinetics of these responses are way too prolonged in ETI (Gao *et al.*, 2013). Furthermore, the downstream defense gene expression patterns are mostly similar in PTI and ETI, however, the magnitude is higher during the ETI responses. Additionally, the defense signaling in ETI are robust and flexible against pathogen effector alterations in compared to PTI. For example, in *Arabidopsis*, the prolonged MAPK activation during ETI resulted in robust immune responses and expression of defense-specific genes like PR1 (Tsuda *et al.*, 2013). Further, during ETI many of the SA-dependent genes could be controlled in a SA-independent manner. In addition, the CNL RPM1 or RPS2-mediated continual activation of Ca^{2+}-dependent protein kinases (CPKs) in *Arabidopsis* resulted in the phosphorylation of several WRKY transcription factors ultimately achieving transcriptional reprogramming (Gao *et al.*, 2013). Conclusively, the robustness and flexibility of ETI varies from that of PTI and can be controlled both quantitatively and qualitatively by several factors.

CONCLUSION

The interactions between plant-pathogen are complex and multi-faceted. Innumerable studies have been carried out from last decade to the present day to unveil the mechanism of these interactions. When exploring the avenues of plant-pathogen interactions, mainly two broad nodes come into pictures such as PTI and ETI. The former one is based on the strategic detection and neutralization of conserved PAMP or MAPM signatures, whereas the later one relies on the plant resistance genes to confer immunity. Like plants employ PTI or ETI to get rid of the invading pathogens, some of the pathogens can produce potent effectors that can dodge the plant patrolling and spread further infection and pathogen colonization. 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. This review, serving as a comprehension of all such investigations, will help to understand and interpret the several mechanisms of the plant-pathogen interactions.

ACKNOWLEDGEMENT

Authors are thankful to the Centurion University of Technology and Management for necessary facilities and support.

REFERENCE

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 receptors. *Annual Review of Plant Biology*,**60**: 379–407.

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**: 233–240.

Buscaill, P., and Rivas, S. (2014). Transcriptional control of plant defense responses. *Current Opinion in Plant Biology*, **20**: 35–46.

Chang, C., Yu, D., Jiao, J., Jing, S., Schulze-Lefert, P.,and Shen, Q.H.(2013). Barley MLA immune receptors directly interfere with antagonistically acting transcription factors to initiate disease resistance signaling. *Plant Cell*, **25**: 1158–1173.

Chen, X., and Ronald, P.C. (2011). Innate immunity in rice. *Trends in Plant Science*, **16**: 451–459.

Collier, S.M., and Moffett, P. (2009). NB-LRRs work a "bait and switch" on pathogens. *Trends in Plant Science*, **14**: 521–529.

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

Davière, J.M., and Achard, P. (2013). Gibberellin signaling in plants. *Development*,**140**: 1147–1151.

De Lorenzo, G., Brutus, A., Savatin, D.V., Sicilia, F., and Cervone, F. (2011). Engineering plant resistance by constructing chimeric receptors that recognize damage-associated molecular patterns (DAMPs). *FEBS Letters*, **585**: 1521–1528.

dit Frey, N.F., Mbengue, M., Kwaaitaal, M., Nitsch, L., Altenbach, D., Häweker, H., Lozano-Duran, R., Njo, M.F., Beeckman, T., Huettel, B., Borst, J.W., Panstruga, R., and Robatzek, S. (2012). Plasma membrane calcium ATPases are important components of receptor-mediated

signaling in plant immune responses and development. *Plant Physiology*, **159**: 798–809.

Dodds, P.N., and Rathjen, J.P. (2010). Plant immunity: towards an integrated view of plant–pathogen interactions. *Nature Reviews Genetics*, **11**: 539–548.

Elmore, J.M., Lin, Z.J.D., and Coaker, G. (2011). Plant NB-LRR signaling: Upstreams and downstreams. *Current Opinion in Plant Biology*, **14**: 365–371.

Flor, H.H. (1971). Current Status of the Gene-For-Gene Concept. *Annual Reviews in Phytopathology*, **9**:275–296.

Fradin, E.F., Zhang, Z., Ayala, J.C.J., Castroverde, C.D.M., Nazar, R.N., Robb, J., Liu, C.M., and Thomma, B.P.H.J. (2009). Genetic dissection of verticillium wilt resistance mediated by tomato ve1. *Plant Physiology*, **150**: 320–332.

Gao, X., Chen, X., Lin, W., Chen, S., Lu, D., Niu, Y., Li, L., Cheng, C., McCormack, M., Sheen, J., Shan, L., and He, P. (2013). Bifurcation of Arabidopsis NLR Immune Signaling via Ca2+-Dependent Protein Kinases. *PLoS Pathogens*, **9**.

Gu, K., Yang, B., Tian, D., Wu, L., Wang, D., Sreekala, C., Yang, F., Chu, Z., Wang, G.L., White, F.F., and Yin, Z. (2005). R gene expression induced by a type-III effector triggers disease resistance in rice. *Nature*, **435**: 1122–1125.

Gururani, M.A., Venkatesh, J., Upadhyaya, C.P., Nookaraju, A., Pandey, S.K., and Park, S.W. (2012). Plant disease resistance genes: Current status and future directions. *Physiological and Molecular Plant Pathology*, **78**: 51–65.

Hettenhausen, C., Schuman, M.C., and Wu, J. (2015). MAPK signaling: A key element in plant defense response to insects. *Insect Science*, **22**: 157–164.

Inoue, H., Hayashi, N., Matsushita, A., Xinqiong, L., Nakayama, A., Sugano, S., Jiang, C.J., and Takatsuji, H. (2013). Blast resistance of CC-NB-LRR protein Pb1 is mediated by WRKY45 through protein-protein interaction. *Proceedings of the National Academy of Sciences of the United States of America*, **110**: 9577–9582.

Jeworutzki, E., Roelfsema, M.R.G., Anschütz, 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 Ca2+-associated opening of plasma membrane anion channels. *Plant Journal*, **62**: 367–378.

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

Kadota, Y., Sklenar, J., Derbyshire, P., Stransfeld, L., Asai, S., Ntoukakis, V., Jones, J.D., Shirasu, K., Menke, F., Jones, A., and Zipfel, C. (2014). Direct Regulation of the NADPH Oxidase RBOHD by the PRR-Associated Kinase BIK1 during Plant Immunity. *Molecular Cell*, **54**: 43–55.

Kitsios, G., and Doonan, J.H. (2011). Cyclin dependent protein kinases and stress responses in plants. *Plant Signaling and Behavior*, **6**: 204–209.

Klessig, D.F., Choi, H.W., and Dempsey, D.A. (2018). Systemic Acquired Resistance and Salicylic Acid: Past, Present, and Future. *Molecular Plant Microbe Interactions*, **31**: 871-888.

Maverakis, E., Kim, K., Shimoda, M., Gershwin, M.E., Patel, F., Wilken, R., Raychaudhuri, S., Ruhaak, L.R., and Lebrilla, C.B. (2015). Glycans in the immune system and The Altered Glycan Theory of Autoimmunity: A critical review. *Journal of Autoimmunity*, **57**: 1–13.

McDowell, J.M., and Woffenden, B.J. (2003). Plant disease resistance genes: Recent insights and potential applications. *Trends in Biotechnology*, **21**: 178–183.

Miya, A., Albert, P., Shinya, T., Desaki, Y., Ichimura, K., Shirasu, K., Narusaka, Y., Kawakami, N., Kaku, H., and Shibuya, N. (2007). CERK1, a LysM receptor kinase, is essential for chitin elicitor signaling in Arabidopsis. *Proceedings of the National Academy of Sciences of the United States of America*, **104**: 19613–19618.

Muthamilarasan, M., and Prasad, M. (2013). Plant innate immunity: An updated insight into defense mechanism. *Journal of Bioscience*, **38**: 433–449.

Nanda, S., Mishra, R., and Joshi, R.K. (2021). Molecular basis of insect resistance in plants: current updates and future prospects. *Research Journal of Biotechnology*, **16**: 194-205.

Nanda, S., Rout, E., and Joshi, R.K. (2016). *Curcuma longa* Mitogen-Activated Protein Kinase 6 (ClMPK6) Stimulates the Defense Response Pathway and Enhances the Resistance to Necrotrophic Fungal Infection. *Plant Molecular Biology Reporter*, **34**: 886–898.

Nimchuk, Z., Eulgem, T., Holt, B.F., and Dangl, J.L. (2003). Recognition and Response in the Plant Immune System. *Annual Reviews in Genetics*, **37**: 579–609.

Rout, E., Nanda, S., Nayak, S., and Joshi, R.K. (2014). Molecular characterization of NBS encoding resistance genes and induction analysis of a putative candidate gene linked to *Fusarium* basal rot resistance in *Allium sativum*. *Physiological and Molecular Plant Pathology*, **85**: 15–24.

Sharma, N. and Gautam, A.K. (2019). Early Pathogenicity events in Plant Pathogenic Fungi: A Comprehensive Review. *Biological Forum – An International Journal*, **11**: 24-34.

Shen, Q.H., Saijo, Y., Mauch, S., Biskup, C., Bieri, S., Keller, B., Seki, H., Ülker, B., Somssich, I.E., and Schulze-Lefert, P. (2007). Nuclear activity of MLA immune receptors links isolate-specific and basal disease-resistance responses. *Science*, **315**: 1098–1103.

Shin, S., Lv, J., Fazio, G., Mazzola, M., and Zhu, Y. (2014). Transcriptional regulation of ethylene and jasmonate mediated defense response in apple (*Malus domestica*) root during *Pythium ultimum* infection. *Horticulture Research*, **1**.

Triplett, L.R., Shidore, T., Long, J., Miao, J., Wu, S., Han, Q., Zhou, C., Ishihara, H., Li, J., Zhao, B.,

and Leach, J.E. (2016). AvrRxo1 Is a Bifunctional type III Secreted effector and toxin-antitoxin system component with homologs in diverse environmental contexts. *PLoS One*, **11**.

Tsuda, K., Mine, A., Bethke, G., Igarashi, D., Botanga, C.J., Tsuda, Y., Glazebrook, J., Sato, M., and Katagiri, F. (2013). Dual Regulation of Gene Expression Mediated by Extended MAPK Activation and Salicylic Acid Contributes to Robust Innate Immunity in *Arabidopsis thaliana. PLoS Genetics*, **9**.

Van Der Hoorn, R.A.L., and Kamoun, S. (2008). From guard to decoy: A new model for perception of plant pathogen effectors. *Plant Cell*, **20**: 2009–2017.

Wang, Y., Loake, G.J., and Chu, C. (2013). Cross-talk of nitric oxide and reactive oxygen species in plant programed cell death. *Frontiers in Plant Science*, **4**.

Yamaguchi, M., Ohtani, M., Mitsuda, N., Kubo, M., Ohme-Takagi, M., Fukuda, H., and Demura, T. (2010). VND-INTERACTING2, a NAC domain transcription factor, negatively regulates xylem vessel formation in Arabidopsis. *Plant Cell*, **22**: 1249–1263.

Yu, X., Feng, B., He, P., and Shan, L. (2017). From Chaos to Harmony: Responses and Signaling upon Microbial Pattern Recognition. *Annual Review in Phytopathology*, **55**:109-137.

Yue, J.X., Meyers, B.C., Chen, J.Q., Tian, D., and Yang, S. (2012). Tracing the origin and evolutionary history of plant nucleotide-binding site-leucine-rich repeat (NBS-LRR) genes. *New Phytologist*, **193**: 1049–1063.

Zhang, J., Li, W., Xiang, T., Liu, Z., Laluk, K., Ding, X., Zou, Y., Gao, M., Zhang, X., Chen, S., Mengiste, T., Zhang, Y., and Zhou, J.M. (2010). Receptor-like cytoplasmic kinases integrate signaling from multiple plant immune receptors and are targeted by a *Pseudomonas syringae* effector. *Cell Host and Microbe*, **7**: 290–301.

Zhang, J., Shao, F., Li, Y., Cui, H., Chen, L., Li, H., Zou, Y., Long, C., Lan, L., Chai, J., Chen, S., Tang, X., and Zhou, J.M. (2007). A *Pseudomonas syringae* Effector Inactivates MAPKs to Suppress PAMP-Induced Immunity in Plants. *Cell Host and Microbe*, **1**: 175–185.

Zipfel, C. (2008). Pattern-recognition receptors in plant innate immunity. *Current Opinion in Immunology*, **20**: 10–16.

Zipfel, C. (2014). Plant pattern-recognition receptors. *Trends in Immunology*, **35**: 345–351.

Zipfel, C., Kunze, G., Chinchilla, D., Caniard, A., Jones, J.D.G., Boller, T., and Felix, G. (2006). Perception of the Bacterial PAMP EF-Tu by the Receptor EFR Restricts *Agrobacterium*-Mediated Transformation. *Cell*, **125**: 749–760.

How to cite this article: Kumar, G. and Nanda, S. (2021). Molecular Perspectives of Plant-Pathogen Interactions: An Overview on Plant Immunity. *Biological Forum – An International Journal*, **13**(1): 48-53.