A Review: Molecular Mechanisms of Plant Immunity

Shousen Guo[1,a], Baojun Li[2,b], Le Su[1,c], Fujia Li[2,d], Orlando Borrás-Hidalgo[1,e], Xin Sun[1,f], Qun Su[3,g], Lei Sun[3,h], Chen Zhao[1,i]*, Lin Zhao[1,j]*

[a]1815317702@qq.com, [b]13869149205@163.com, [c]sule@sdu.edu.cn, [d]3191889@qq.com, [e]orlando@yotabio.com, [f]cindy_sun82@163.com, [g]su820525@163.com, [h]sunlei@baoyuan-bio.com, [i*]zhaochen_bioeng@163.com, [j*]sdilizhaolin@163.com

[1]State Key Laboratory of Biobased Material and Green Papermaking, Shandong Provincial Key Laboratory of Microbial Engineering, Qilu University of Technology (Shandong Academy of Sciences), Jinan, China
[2]Shandong Zhuoran Biotechnology Co., Ltd., Jinan, China
[3]Shandong Baoyuan Biotechnology Co., Ltd., Yantai, China

Abstract: Plant immunity represents a rapidly developing field in environmental protection and food safety. In this paper, we discuss the molecular mechanisms of plant immunity and describe how they promote plant health. Different infection patterns of pathogen and different defense responses of plant including pattern-triggered immunity (PTI) and effect-triggered immunity (ETI) are mainly talked. This review presents an integral understanding of plant-pathogen interactions and will help to gain our knowledge of plant microbial ecology.

Keywords: Plant immunity; plant-pathogen interactions; pattern-triggered immunity (PTI); effect-triggered immunity (ETI)

1. Introduction

Plants face various threats from the environment during their growth and development. When plants are stressed, the immune system will initiate a defense mechanism to ensure the normal survival of plants. The pathogens of invasive plants are roughly divided into the following categories. One is the necrotic vegetative body that feeds on nutrients at the expense of the host. The other is the biological nutrient body that relies on the surviving host to provide nutrients. The third category is the semi-biological nutrient body that initially needs to survive the host but is killed in the late stage of infection. [1] Pathogens enter plant cells through stomata, water holes or wounds, and proliferate in the intercellular space (exosomes). Aphids and nematodes obtain nutrients by penetrating into plant cells through stigma. Fungi infect plants in a variety of ways, not only directly invade epidermal cells, but also by infecting plants on plant cells, between cells, or by extending hyphae. Mammals and plants have different ways of immunity. Mammals rely on the migration of immune cells in the body to kill pathogens, while plants need to rely on the innate immunity of each cell and signals from the infected site to activate their own immune response to kill invading pathogens. [2][3] Plant immune system is mainly divided into two categories. One is PTI (pattern-triggered immunity), which works by directly recognizing pathogens through transmembrane pattern recognition receptors (PRRs) located on plant cell membranes, known as elicitors of pathogen-associated molecular patterns (PAMPs). [4] The other is ETI (effector-triggered immunity), which contains a nucleotide-

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leucine-rich repeat sequence (NB-LRR). It is a receptor kinase that can recognize effectors from different pathogens and activate the corresponding defense response. [5] At present, the more recognized understanding of plant systems is the four-stage " zhi " -shaped model. Pattern recognition receptors first recognize PAMP, and then PTI immunity in plants is activated. Subsequently, the immune response of PTI will be affected by the effector molecules released by the pathogen. The release of pathogenic effectors will be recognized by NB-LRR in plant cells, which will cause ETI immune response in plants. The ETI reaction is stronger than the PTI reaction, because the ETI reaction is a further immunity of plants to the invasion of pathogens. Whether it is the outbreak of ROS, the up-regulation of defense genes or the secretion of plant hormones will be more intense. The two work together to enhance plant disease resistance.

2. Pathogen Entry and Infection Sites

The invasion of microbial pathogens into plant tissues is the main cause of plant disease, and the virulence of pathogens is also manifested in tissues. The entry of pathogens into plant tissues is mainly achieved by directly penetrating the surface of plants, or through natural openings such as physical wounds or stomata of plants. Bacteria can pass through trichomes; lenticels; stomata; hydathodes; lateral root; wounds enter into the plant tissue and survive in the plastids. [6]-[11] The way pathogenic fungi infect plants is mainly through the modification of mycelium-specific infection of plant tissue. Fungi attach to the cuticle of the plant, so that the embryo tube on the surface of the plant grows, and then forms an appressorium attached to the embryo tube, [12] The pressurizer carried by fungi will produce higher expansion pressure to support its invasion process. In addition, some fungi only survive in the plastids after invading plants. For example, Cladosporium fulvum does not form haustoria, but completely grows in the plastids and depends on the nutrients leaked by plants to survive. [13] For viruses, they can only enter the interior of plants through environmental factors or plant trauma caused by the outside world. Arthropods that suck juices contribute to vector-mediated viral infections. They can directly transport viral particles to the vascular system, thereby rapidly spreading the virus to the entire plant causing plant lesions.

3. Plant Defense Mechanisms

Pathogens entering plants through different channels will induce the response of plant immune system. The PTI response occurs when the pathogen carries PAMP or DAMP (damage-related molecular patterns) through the cell surface and is recognized by PRR receptors located in the first layer of the immune system. PTI plays an important role in killing invading pathogens or maintaining the stability of microbial populations in plant leaves PTI plays an important role in killing invading pathogens or maintaining the stability of microbial populations in plant leaves. With the evolution of species, in order to enhance invasion and proliferation, plant cells or exosomes can sense the effector molecules secreted by T3SS (bacterial type III secretion system) and perceive the virulence of pathogens, thereby inhibiting plant immunity. [14] After the first layer of immune failure, in order to resist the virulence of pathogens, plants will have NLR receptors to directly or indirectly identify pathogen effectors, and then activate the second usually stronger immune signal, called ETI [15]. The ' zigzag ' model proposed in 2006 triggered

a strong response. In this model, PAMP is first recognized by the recognition receptor in the plant, thereby activating the PTI immunity of the plant, and then the pathogen will release the pathogenic effector to eliminate the immune response of PTI. The release of the pathogenic effector will be recognized by the NB-LRR in the plant cells, which in turn causes the ETI immune response of the plant.

PTI initially activates the Ca2+ influx of plant cells, and the Ca2+ channel on the plasma membrane is activated by phosphorylation of PRRs. Calcium-dependent protein kinase is the receptor of Ca2+, which is the main medium for signal transmission and is responsible for transmitting signals to the next level. [16] Ca2+ influx can cause stomatal closure and the accumulation of ROS near the stomata. Stomatal closure is to prevent pathogens from entering the plant through the stomata, and ROS is thought to directly kill microorganisms, and enhance the cell wall structure through the oxidative cross-linking of glycoproteins, and induce the synthesis of intracellular salicylic acid and the cascade activation of mitogen-activated protein kinase (MAPK). [17]. Finally, PTI increases the synthesis of salicylic acid and jasmonic acid in plants, both of which act as signaling molecules to transmit immune activation signals, thereby inducing further immune responses. In order to further infect plants and resist the PTI reaction produced by plants, pathogens will release effectors into cells to inhibit the PTI reaction. Effector factors act as transcription factors in cells, directly affect the transcription of host cells, affect the assembly of histones and chromosomes, inhibit the activity of host transcription factors, and promote the colonization of pathogens and the release of required nutrients through the above means. However, the NB-LRR of plants recognizes the effectors released by pathogens in a highly specific manner, thereby activating the ETI response of plants. The defense methods activated by ETI response and PTI response are basically the same, and the difference is that the immunity activated by ETI response is more intense and lasting [18]. Although there are different ligand perception and activation patterns in PTI and ETI, more and more evidence shows that these two signaling branches are functionally connected.

4. Immune System Recognition Receptor Prr and Nlr

PRR and NLR immune receptor genes were first isolated in 1994. With the deepening of research, it has been found that both NLR and PRR require other functionally connected NLR and PRR as auxiliary / auxiliary receptors to initiate immune responses. In recent years, with the introduction of the concept of “receptor network”. In 2017 and 2018, the term NLR and PRR networks were proposed successively. [19] In addition, there is also a certain relationship between different plant hormones.

4.1 Prrs Involved in Pathogen Recognition

Plant PRR includes receptor kinase (RLP) or receptor-like protein (RLK). Both of them cannot independently transduce immune signals and require the assistance of auxiliary receptors. RLK is mainly composed of extracellular region, transmembrane region and cytoplasmic kinase region, while no cytoplasmic kinase region was found in RLP. PRR is anchored to PM by transmembrane α-helix or glycophosphatidylinositol (GPI). Both RLP and RLK sense ligands through a series of extracellular domains. [20] They are mainly leucine-rich repeat (LRR), lectin, apple lectin, lysine motif (Lys M) and EGF-like domain. [20] It has been reported that

RLK accounts for 60 % of the kinases in the Arabidopsis genome and can be divided into 44 subfamilies according to the kinase domain. [20] RLK is not present in fungi, but is widely present in animals and plants. In 1994, Avr9, which can recognize extracellular effectors from fungi, was found in tomato and then identified as a gene encoded by PRR. Cladosporium fulvum. Since then, multiple RLPs that recognize extraplasmic effectors have been discovered,[21] For example, Cf-4 and Cf-2. PRRs can recognize PAMPs / MAMPs / HAMPs (herbivore-associated molecular patterns) from a variety of sources, including bacteria, fungi, oomycetes, plants and animals. Some of these PRRs can also recognize their own molecules. [22]

Plants can sense PAMPs from bacteria. For example, peptides (lipids) from bacteria have effects on Arabidopsis, tomato, and rice. [23] For fungi, they can be perceived by a variety of PRRs because their cell walls include chitin and oligogalacturonates (OGs). Not only the fungal cell wall can be recognized by LRR-RLPs, but also the extracellular effectors of fungal pathogens can be identified. [24] The cell wall of oomycetes is also composed of chitin, endoPGs and OGs, so it can also be recognized by PRRs, causing an immune response. Some of these PRRs can also recognize specific PAMPs from oomycetes. Plants can also activate immunity by sensing PAMPs from parasitic plants, and tomato LRR-RLP SlCuRe1 can sense the peptide Crip21 of Cuscuta spp. Crip21 from Cuscuta chinensis is a cell wall protein rich in glycine. Activation of SlCuRe1 by Cris21 triggers tomato cell death and defense responses. As shown in Figure 1 Heterodera schachtii (sugarbeet nematode) extracts can be sensed by Arabidopsis LRR-RLK AtNILR1. Picture making By Figdraw. Most of the characterized PRRs have been shown to bind to a specific ligand, leading to the activation of PTI.

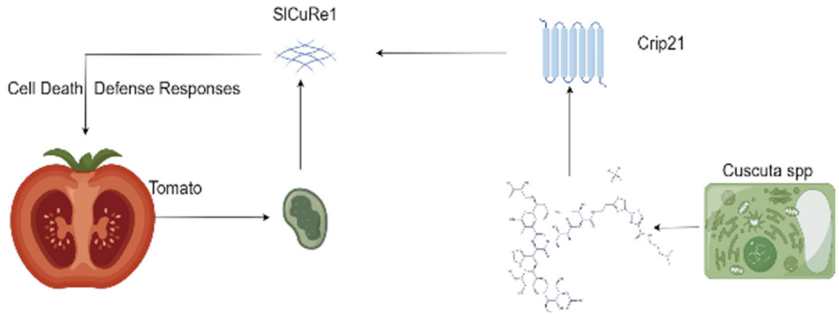

Fig. 1 The process of tomato perceiving the parasitic plant dodder-

4.2 Nlrs Involved in Pathogen Recognition

NLR genes can be found in the genomes of all terrestrial plants, NLR can be divided into three categories by distinguishing the terminal domain: spiral (CC) NLR (CNLs), Toll / interleukin-1 receptor / resistance (TIR) protein NLR (TNLs) and RPW8-like CC domain (RPW8) NLR (RNLs). CNL, TNL and RNL are widespread in basal angiosperm species such as Ambarellas and Nymphaea. The loss of TNL gene may occur before the differentiation of mono-and di-ovary plants, which also leads to the absence of TNL in most mono-ovary plants, and the loss of this gene also leads to the interruption of TNL-containing signaling pathways. There are NB-arc domain and LRR domain in plant NLRs. Due to the difference of NLR, there are differences between domains. NB-arc domain can activate NLR and has ATP binding activity.

The pathogens recognized by NLR include viruses, bacteria, fungi, oomycetes, parasitic plants and herbivores. NLR can act as an auxiliary or common receptor to transduce immune signals after recognizing pathogens, thereby activating the immune response. Since bacterial infection of plants targets contact with PRR and inhibits the immune response of plants, plants have evolved a variety of NLRs to protect the host 's immune components and induce ETI immune response. Plant NLR can sense a variety of effectors and molecules from fungal pathogens. LOV1 of Arabidopsis thaliana and Phaseolus vulgaris can recognize a secondary metabolite Victorin;[25] Melon CNL cmform-2 can recognize AvrFom2 of Fusarium oxysporum. In the process of plant growth, the harm of oomycetes to plants is also fatal, and it has also caused huge economic losses worldwide. Oomycetes themselves carry a large number of plant pathogenic pathogens belonging to Phytophthora. NLRs of many species in Solanaceae can recognize the effectors of pathogens. For viruses, their movement proteins and RNA-dependent RNA polymerase can be recognized by NLR when they infect plants, but no NLR receptors involved in self-recognition have been found in plants. In addition, NLRs have also been shown to be resistant to herbivores. NLRs involved in nematode resistance include TIR-NB-LRR to AtDSC1 and AtWRKY19, tauschii AtaCD3.1, CaMi, Prunus cerasifera PcMa, SlHero, StGpa-2 and StGro1-4. In addition, resistance to aphids and whiteflies, and arthropod brown planthoppers have also been demonstrated. For auxiliary NLR, although there is a part that does not require auxiliary NLR, many NLRs need to work with auxiliary NLR to transduce immune signals. Although the mechanism of individual NLR activation has been extensively studied, it is not clear whether the activation of NLR will affect other NLRs.

5. Plant Immune Inducer

At present, in the study of oligosaccharide plant immune inducers, chito-oligosaccharides are more studied and have better effects. Chito-oligosaccharides are one of the first oligosaccharide inhibitors studied, and in the study of fungal plant pathogens, Zhang Wenge et al. studied the prevention effect of chito-oligosaccharides on wheat blight at indoor and field levels, and its defense effect reached 88.40%-90.60%. Xu Junguang et al. used chito-oligosaccharide to treat chili pepper to study its disease resistance to Phytophthora mildew, and the results showed that the use of chito-oligosaccharide to pretreat chili pepper in the field could enhance the disease resistance of chili pepper to Phytophthora to a certain extent, and the prevention effect of 40 mg/L chito-oligosaccharide against pepper blight was as high as 73.20%. Yin et al. found that 50 mg/L chito-oligosaccharide was the optimal dose to induce resistance to tobacco mosaic virus in Arabidopsis, and the expression of the salicylic acid pathway marker gene pr1 in Arabidopsis thaliana was up-regulated, and the content of salicylic acid was significantly increased. On plant bacterial pathogens, the plant protection station of Hainan Province studied the effect of chito-oligosaccharide in the prevention and control of soft rot in cabbage, and they found that 60 mg/L chito-oligosaccharide pretreatment of cabbage could achieve 85% control effect, while the control group streptomycin sulfate 3000-fold dilution control effect was 74.99%. The results of field experiments showed that after 5 consecutive treatments before cabbage disease, the prevention efficacy of chito-oligosaccharides at 60 mg/L, 50 mg/L and 40 mg/L was 66.90%, 62.26% and 47.60%, respectively, after 10 days.

6. Conclusion

The defense of plants is not only PTI and ETI reactions, but also different defense measures when resisting pathogen invasion. The cuticle of the plant surface acts as a physical barrier and also plays a certain defensive role. Recent studies have found that the cuticle also activates related immune pathways when plants are endangered. In addition, plants also secrete specific proteolytic enzyme inhibitors to resist the proteolytic enzymes secreted during pathogen invasion. Plants will also produce low molecular weight secondary metabolites with antibacterial activity after stress damage. These products will be de novo synthesized in their bodies, collectively referred to as plant antitoxins. The immune response of plants is a complex process, in which there are a variety of signaling pathways. There are both independent transmission and mutual assistance between them, which play an important role in the immune response.

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References

[1] Zhou, J.M., Zhang, Y. (2020) Cell., 181(5): 978-989. DOI: 10.1016/j.cell.2020.04.028.

[2] Ausubel, F.M. (2005) Nature Immunol., 6: 973–979. DOI: 10.1038/ni1253.

[3] Chisholm, S.T., Coaker, G., Day, B. & Staskawicz, B. J. (2006) Cell., 124, 803–814. DOI: 10.1016/j.cell.2006.02.008.

[4] Durrant, W.E., Dong, X. (2004) Annu Rev Phytopathol., 42: 185-209. DOI: 10.1146/annurev.phyto.42.040803.140421.

[5] Jones, J.D., Dangl, J.L. (2006) Nature., 16;444(7117): 323-9. DOI: 10.1038/nature05286.

[6] Layne, REC. (1967) Phytopathology., 57: 981–985. https://api.semanticscholar.org/CorpusID:83617370.

[7] Fox, R.T.V., Manners, J.G. and Myers, A. (1971) Potato Res., 14: 61–73. https://doi.org/10.1007/BF02355930.

[8] Getz, S., Fulbright, D.W and Stephens, C.T. (1983) Phytopathology., 73: 39–43. https://api.semanticscholar.org/CorpusID:86817343.

[9] Melotto, M., Underwood, W., Koczan, J., Nomura, K., He, S.Y. (2006) Cell., 126(5): 969-80. DOI: 10.1016/j.cell.2006.06.054.

[10] Mew, T.W., Mew, I.C. and Huang, J.S., (1984) Phytopathology., 74: 681–641. https://api.semanticscholar.org/CorpusID:85154635.

[11] Huang, J.S (1986) Phytopathol., 24: 141–157. https://doi.org/10.1146/annurev.py.24.090186.001041.

[12] Howard, R.J., Ferrari, M.A., Roach, D.H. and Money, N.P. (1991) Proc. Natl. Acad. Sci. USA., 88: 11281–11284. DOI: 10.1073/pnas.88.24.11281.

[13] Thomma, B.P.H.J., Esse, H.P.V., Crous, P.W. and DE, Wit. PJ. (2005) Mol. Plant Pathol. 6: 379–393. DOI: 10.1111/j.1364-3703.2005.00292.x.
[14] Xin, X.F., Kvitko, B., He, S.Y. (2018) Nat Rev Microbiol., 16(5): 316-328. DOI: 10.1038/nrmicro.2018.17.
[15] Yuan, M., Ngou, B.P.M., Ding, P., Xin, X.F. (2021) Curr Opin Plant Biol., 62:102030. DOI: 10.1016/j.pbi.2021.102030.
[16] Nomura, H., Komori, T., Uemura, S., et al. (2012) Nature Communications., 3: 926. DOI: 10.1038/ncomms1926.
[17] Ali, R., Ma, W., Lemtiri-Chlieh, F., et al. (2007) Plant Cell., 19(3): 1081-1095. DOI: 10.1105/tpc.106.045096.
[18] Mishina, T.E., Zeier, J. (2007) Plant Journal., 50(3): 500-513. DOI: 10.1111/j.1365-313X.2007.03067.x.
[19] Smakowska-Luzan, E., Mott, G.A., Parys, K., Stegmann, M., Howton, T.C., Layeghifard, M., Neuhold, J., Lehner, A., Kong, J., Grünwald, K., Weinberger, N., Satbhai, S.B., Mayer, D., Busch, W., Madalinski, M., Stolt-Bergner, P., Provart, N.J., Mukhtar, M.S., Zipfel, C., Desveaux, D., Guttman, D.S., Belkhadir. Y. (2018) Nature., 18;553(7688):342-346. DOI: 10.1038/nature25184.
[20] Boutrot, F., Zipfel, C. (2017) Annu Rev Phytopathol., 55: 257–286.DOI: 10.1146/annurev-phyto-080614-120106.
[21] Ngou, B.P.M., Jones, J.D.G., Ding, P. (2022) Trends Plant Sci., 27(3):255-273. DOI: 10.1016/j.tplants.2021.08.012.
[22] Hou, S., Liu, D., He, P. (2021) Stress Biol., 1: 8. DOI: 10.1007/s44154-021-00009-y.
[23] Pruitt, R.N., Schwessinger, B., Joe, A., Thomas, N., Liu, F., Albert, M., Robinson, M.R., Chan, L.J.G., Luu, D.D., Chen, H., et, al. (2015) Sci Adv., 1: e1500245. DOI: 10.1126/sciadv.1500245.
[24] Dixon, M.S., Jones, D.A., Keddie, J.S., Thomas, C.M., Harrison, K., Jones, J.D. (1996) Cell., 84: 451–459. DOI: 10.1016/s0092-8674(00)81290-8.
[25] Sweat, T.A., Lorang, J.M., Bakker, E.G., Wolpert, T.J. (2008) Mol Plant Microbe Interact., 21: 7–19. DOI: 10.1094/MPMI-21-1-0007.