Journal of Plant Sciences
2014; 2(1): 23-30
Published online February 20, 2014 (http://www.sciencepublishinggroup.com/j/jps)
doi: 10.11648/j.jps.20140201.15

Molecular mechanisms and mediators of the immune response in plants

Anna Boyajyan[1, *], Hakob Devejyan[1], Vardan Haykazyan[2], Gevorg Avetisyan[1], Donara Khanoyan[1]

[1]Institute of Molecular Biology NAS RA, Yerevan, Armenia
[2]Republican Agriculture Support Centre of the Ministry of Agriculture RA, Yerevan, Armenia

Email address:
aboyajyan@sci.am (A. Boyajyan)

To cite this article:
Anna Boyajyan, Hakob Devejyan, Vardan Haykazyan, Gevorg Avetisyan, Donara Khanoyan. Molecular Mechanisms and Mediators of the Immune Response in Plants. *Journal of Plant Sciences.* Vol. 2, No. 1, 2014, pp. 23-30. doi: 10.11648/j.jps.20140201.15

Abstract: The purpose of this review article is to analyze, sum up and discuss the existing knowledge and recent concepts on the plant immune system based on the available literature. The main attention is focused on the major molecular players, mediators and regulators of this system, as well as on mechanisms of generation and progression of the different types of the immune response playing an important role in plant physiology, regeneration, resistance, and defense against a broad number of pathogens.

Keywords: Plant Immune System, Gene-for-Gene Resistance, Basal Immune Response, Hypersensitive Response, Systemic Acquired Resistance, Jasmonic Acid/Ethylene Pathway, Non-Host Resistance

1. Introduction

The plant has well-developed immune system. The immune responses in plant include basal response through transcription of genes in response to pathogen-associated molecular pattern recognition, hypersensitive response through apoptosis of cells at the site of infection, systemic acquired resistance making the entire plant resistant to infection, jasmonic acid response (jasmonic acid/ethylene pathway) through which the entire plant and neighboring plants develop resistance to herbivores, and non-host immunity. Many plant-associated microbes are pathogens that impair plant growth and reproduction. Among organisms that infects plants are bacteria, fungi, nematodes and insects as well as viruses. All microbes possess a suite of conserved molecules, pathogen-associated molecular pattern/microbe-associated molecular patterns (PAMPs/ MAMPs) that can be recognized by plants, often via receptor kinase located in the plant plasma membrane [1-3].

There are two branches of the plant innate immune system. One uses transmembrane pattern recognition receptors (PRRs) that respond to slowly evolving PAMPs, such as flagellin. The second acts largely inside the cell, using the polymorphic nucleotide-binding site leucine-rich repeat (NB-LRR) protein products encoded by most r*esistance* (*R*) genes. *R genes* are genes in plant genomes that convey plant disease resistance against pathogens by producing R proteins. One type of important plant disease resistance, gene-for-gene resistance [4], is resulted from the interactions between products of the pathogen avirulence (*Avr*) genes and their matching plant *R* genes. *Avr* genes have been cloned from a variety of pathogens including fungi, bacteria, viruses and oomycetes. No significant homology is found between sequences of the most cloned *Avr* genes and those of known proteins or between those of themselves. However, significant homology has been found between sequences of the cloned *R* genes and those of known proteins or between those of themselves. R proteins consist of similar domains. It has been reported that hypersensitive cell death and resistance, which are induced by interactions between products of different Avr/R gene pairs consisting of similar R genes but different Avr genes, are distinct in development speed, strength, and organ and tissue specificity. Avr genes have dual functions: Pathogens containing *Avr* genes are avirulent to plants carrying the matching *R* genes, while they are virulent in race, strain, pathovar or species-specific way to plants without carrying the matching *R* genes (Fig. 1) [5, 6].

As it was mentioned above, plants respond to infection using a two-branched innate immune system. The first branch recognizes and responds to molecules common to many classes of microbes, including non-pathogens. The second responds to pathogen virulence factors, either

directly or through their effects on host targets. In addition plants have developed immune methods of dealing with herbivores. Differences between plants and animals in general are that plants have no antibody/T cell response; there are no circulating immune cells in a plant. Plants, unlike mammals, lack mobile defender cells and a somatic adaptive immune system. Instead, they rely on the innate immunity of each cell and on systemic signals emanating from infection sites [1, 7-9].

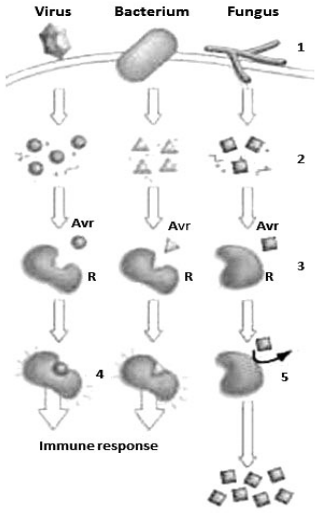

Figure 1. *Gene-to-gene resistance mechanisms.*

1 - pathogens enter plant cell; 2 - Avr gene encoding proteins (Avr) are released from pathogens; 3 - R gene encoding proteins (R) bind to Avr; 4 - binding of R to Avr triggers protective immune response; 5 - when R and Avr do not match, no response occurs, and plant succumbs to disease [5, 6].

2. The Basal Immune Response

The basal immune response is the response induced by PAMP elicited signaling. Plant cells can recognize PAMPs and the effectors of this response, PRRs, are currently being characterized. Among those the most well studied is the leucine-rich repeat transmembrane receptor-like kinase Flagellin sensitive2 (Fls2), which is a receptor to a globular protein flagellin arranged in a hollow cylinder to form the filament in bacterial flagellum. Flagellin is the main building block of the bacterial flagellum and acts as a PAMP triggering the innate immune response in animals and plants. Flg22, a peptide sequence corresponding to the amino terminal of bacterial flagellin, is sufficient to trigger an immune response in plant cells. The binding of flg22 to FLS2 induces the heterodimerization of FLS2 with the receptor-like kinase BAK1, which in turn interacts with the receptor kinase BRI1 to regulate brassinosteroid signaling and alert the immune system (Fig. 2) [10-14].

Loss of function mutations in the gene encoding Fls2 sensitizes the plants to infection. The structure of Fls2 is reminiscent of mammalian Toll-like receptors in that the extracellular domain of the protein contains leucine-rich repeats. The intracellular domain contains a serine threonine kinase. As in animals, there are many PRRs in plants that presumably can recognize microbes by more than one PAMP. Signaling is transduced through a mitogen-activated protein (MAP) kinase cascade and activates transcription factors in the WRKY transcription factor family. Forced expression of the MAP kinases or WRKY29 forces the activation of the pathway and protects the plant from fungal and bacterial infections. This PAMP activated pathway is required for fighting bacterial and fungal infections [15, 16].

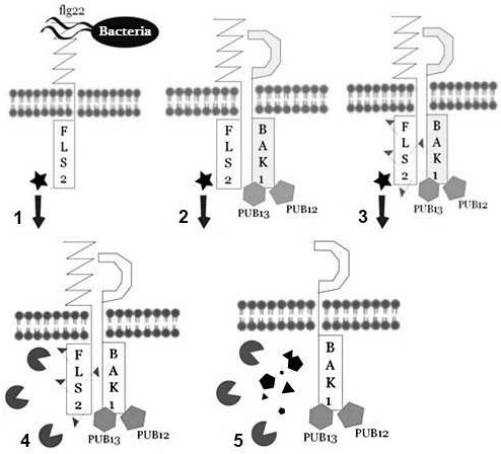

Figure 2. *Fls2-induced mechanism leading to basal immune response.*

1 - the FLS2 PRR detects bacterial flg22. and this triggers defensive responses such as callose deposition and hydrogen peroxide production; 2 - BAK1 associates with FLS2. BAK1 along with the associated enzymes PUB12 and PUB13 attach to FLS2. FLS2 phosphorylates the PUB proteins to activate them; 3- FLS2 becomes tagged for destruction. The activated PUB proteins tag the FLS2 sensor with a marker called ubiquitin. This attracts proteosomes, which then degrade the cytosolic side of FLS2; 4 - proteosomes recognize the marker and move in to destroy FLS2; 5 - the alarm signal is silenced. FLS2 is destroyed so the alarm is silenced. BAK1 is left untouched which is important as it is a common signaling partner of many membrane receptors involved in both immunity and development [10-14].

3. Hypersensitive Response

Hypersensitive response is a rapid apoptosis response that kills cells in the area of infection. Host resistance and parasite ability to cause disease is controlled by pairs of matching genes. One belongs to a plant *R* genes and the other to a parasite Avr genes. Plants producing a specific *R* gene product are resistant towards a pathogen that produces the corresponding *Avr*-gene product. Hypersensitive response can be induced by the interaction of an *R* gene carrying plant with an *Avr* carrying microbe (Fig. 1, 3) [4, 17]. Infiltration of bacteria into the whole leaf *in vitro*, causes a massive cell death response but *in vivo* the hypersensitive response is likely tiny and limits the growth of microbes to a small area on the leaf to stop the growth of biotrophic pathogens that require living tissue in order to survive. Nitric oxide and hydrogen peroxide regulate the response [2, 5, 18].

Cell death caused by pathogen infection is frequently associated with plant resistance. There appear to be two

types of plant cell death associated with pathogen infection: a rapid, hypersensitive cell death localized at the site of infection during an incompatible a resistant plant and an avirulent pathogen, and a slow, "normosensitive" plant cell death that spreads beyond the site of infection during some compatible interactions involving a susceptible plant and a virulent, necrogenic pathogen. Hypersensitive cell death is accompanied by the induction of multifaceted defense responses, including production of active oxygen species and antimicrobial compounds (phytoalexins), rapid cross-lin- king of cell-wall proteins, and, ultimately, resistance to pathogens [19, 20]. Consequently, hypersensitive cell death is considered to be a sacrifice of locally infected tissue (sometimes only one or a few cells) to protect against the spread of the pathogen into healthy plant tissues. In contrast, the slow, normosensitive plant cell death does not effectively prevent pathogen multiplication or spread and is therefore not associated with local resistance. It has long been observed that diverse plant pathogens, from multicellular organisms such as fungi and worms to simple parasites such as viruses, can cause superficially similar hypersensitive cell death in resistant plants [20]. Therefore, hypersensitive cell death has been considered to be a conserved mechanism in higher plants for rapidly self-elimi- nating cells doomed to die, and, in the process of doing so, activating other local and systemic resistance responses either causally or simultaneously. In the past decades, steady progress has been made in understanding the mechanism by which pathogens elicit hypersensitive cell death and the mechanism of signal perception and transduction in the plant cell during hypersensitive cell death.

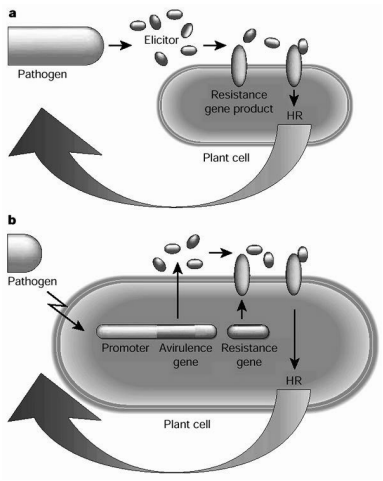

Figure 3. *Hypersensitivity response*

a. The hypersensitive response (HR) is triggered by the highly specific recognition of a pathogen-derived elicitor by a plant resistance gene product. The powerful and concerted defense that constitutes the hypersensitive response stops the pathogen; b. The components involved in the basic switch of the hypersensitive response can be used to create a more nonspecific defense system. A plant-derived pathogen-inducible promoter drives expression of a pathogen elicitor gene. The elicitor formed will trigger the hypersensitive response if the plants also contain the resistance gene [17].

4. Systemic Acquired Resistance (SAR)

One reaction of plants to pathogen infection is the induction of a systemic long-lasting broad-spectrum acquired immune response known as SAR. SAR is effective against subsequent infection by the same or different pathogens. Both hypersensitive and normosensitive cell death can lead to SAR. In a variety of plant species, the development of both necrotic lesions in response to pathogen infection or accelerated apoptosis rate leads to induction of this generalized disease resistance in uninfected tissues. SAR appears to be distinct from preexisting resistance mechanisms such as physical barriers or protein cross-linking and also from other inducible resistance mechanisms such as phytoalexins biosynthesis, the hypersensitive response, and ethylene-induced physiological changes. Furthermore, SAR is not related to responses induced by wounding or osmotic stress. Challenge a leaf with an infectious agent, bacteria, fungi or viruses, and distal tissues become resistant. The distal tissues have broad resistance not only just to the original pathogen. This can be induced by cell hypersensitive or normosensitive cell death. SAR is heightened state of resistance against a broad spectrum of pathogens activated in the uninoculated systemic tissue of a pathogen-infected plant [21, 22].

The activation of SAR requires the accumulation of endogenous salicylic acid [21, 22] and communication by the primary infected tissues with the distal organs. For systemic protection to be initiated, a mobile signal that is produced at the site of primary infection needs to travel through the plant. Several plant-derived substances have been proposed to participate in these long-distance signaling. These involve the putative lipid transfer protein defective in induced resistance1 (DIR1), the methyl ester of salicylic acid, glycerol-3-phosphate, the diterpenoid dehydroabietinal, the dicarboxylic acid azelaic acid, and the Lys catabolite pipecolic amino acid (Pip) [23-29]. Thus, the pathogen-induced salicylic acid signal travels through the plant, activates a molecular signal transduction pathway, which triggers a coordinate expression of a number of genes inducing SAR, and increases the resistance of the plant to further infection (Fig. 4) [30-32]. Genome-wide microarray analyses revealed that during biological activation of SAR in *Arabidopsis*, the transcript levels of several hundred plant genes were consistently up-regulated (SAR^+ genes) in systemic, non-inoculated leaf tissue [33]. This transcriptional reprogramming fully depended on the SAR regulator flavin-dependent monooxygenase 1, which, most possibly, synthesize yet unclear metabolite required for the transduction or amplification of a signal during the early phases of SAR establishment in systemic leaves [34]. Alignment of the SAR expression data with other microarray information allowed defining three clusters of SAR^+ genes. Cluster I consists of genes tightly regulated by salicylic acid. Cluster II genes can be expressed independently of salicylic acid, and this group is moderately enriched in $H_2O_2^-$ and abscisic acid-responsive genes. The expression of the cluster

III SAR^+ genes is partly salicylic acid -dependent. The expression of the cluster III SAR^+ genes is partly SA-dependent. It is proposed that salicylic acid -independent signaling events in early stages of SAR activation enable the biosynthesis of salicylic acid and thus initiate salicylic acid-dependent SAR signaling. Both salicylic acid-independent and salicylic acid-dependent events tightly co-operate to realize SAR [33, 4].

SAR is long lasting and acts against a broad range of pathogens while its maintenance does not significantly affect plant yield. The latter characteristic makes that SAR signals are now generally considered good candidates for protection of crop plants from disease. Systemic Acquired Resistance

SAR-response has found a use in agriculture in the form of inducers of SAR like Actigard, Messenger and Vacciplant [35].

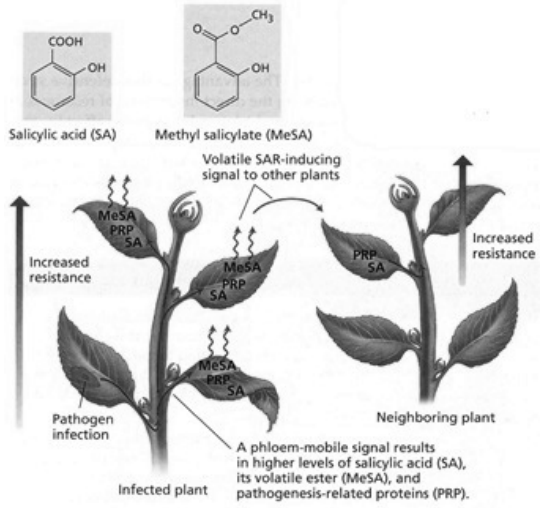

Figure 4. *Initial pathogen infection increases resistance to future pathogen attack through development of SAR [34].*

5. Jasmonic Acid/Ethylene Pathway

Plants respond to infection with necrotrophic pathogens with the synthesis of phytohormones - jasmonic acid and ethylene. The combination of both hormones elicits an anti-microbial defense program. Jasmonic acid is a volatile plant hormone involved in regulation of immunity. Jasmonic acid and its derivative jasmonoyl-isoleucine accumulate upon wounding (e.g. herbivory) and upon attack with necrotrophic pathogens. However, different responses are elicited depending on whether the plant is injured by insects or whether it is infected by a pathogen. This differential reaction is due to higher synthesis of ethylene after pathogen attack than after herbivory [36]. Jasmonic acid synthesis is triggered upon herbivory and further induces the transcription of a number of genes that are anticipated to reduce the digestion of the herbivore. An example is the induction of the arginase encoding gene in tomato plants. This reduces the availability of arginine to the insect gut and reduces growth of the caterpillar.

Herbivore attack leads to the profound changes in plant metabolism. Metabolic genes activated by hormone signaling produce large amount of secondary metabolites that function as defense shield against herbivores. The jasmonic acid pathway is activated through COI1 leading to the degradation of jasmonate ZIM-domain (JAZ)-proteins, which belong to a family of transcriptional regulators (Fig. 5) [37]. The jasmonic acid-dependent defense program is repressed by JAZ-proteins, which bind to the transcriptional activator MYC2. After pathogen attack or wounding, the jasmonic acid derivative jasmonoyl-isoleucine accumulates and binds to the receptor protein coronatine insensitive 1 (COI1). Upon binding of jasmonoyl-isoleucine to COI1, JAZ-proteins become degraded through the 26S proteasome so that MYC2 is free to function [38]. Consistent with this model, JAZ proteins are not degraded in *coi1* and JA biosynthesis mutants leading to the permanent inactivation of the pathway. In the presence of ethylene, which is associated with pathogen attack rather than wounding, the cascade is modified. JAZ-proteins do not only repress MYC2, but also transcription factor ethylene insensitive 3 (EIN3). EIN3 accumulates only when the ethylene signaling cascade is activated. Ethylene inactivates the ethylene receptor, which represses the ethylene cascade. Thus, ethylene induces the cascade finally leading to the stabilization of EIN3. EIN3 activates the promoter of the transcription factor ORA59, which is a regulator of the jasmonic acid/ethylene-activated defense program. TGA transcription factors are required for the activation of the jasmonic acid/ethylene pathway. TGA transcription factors belong to the group of bZIP transcription factors, which are found in all eukaryotes. TGA factors bind specifically to variants of the palindrome TGACGTCA. Two of these sequences separated by 4 bps are called an activation sequence-1 (as-1) [39].

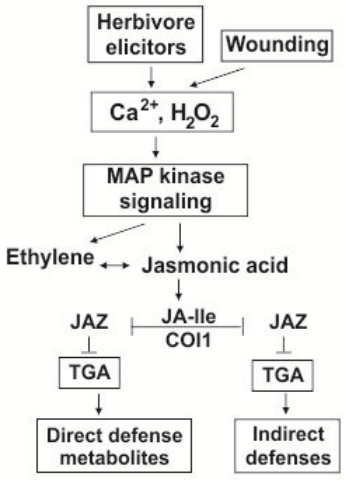

Figure 5. *Activation of jasmonic acid/ethylene pathway*

Herbivore attack induces burst of jasmonic acid which is converted to jasmonoyl-isoleucine (JA-Ile) that mediates COI1-dependent degradation of JAZ-proteins, transcriptional reprogramming and activation of defense responses against insect herbivores in plants [37-39].

6. Non-Host Resistance (NHR)

NHR is resistance exhibited by an entire plant species to all genetic variants of a non-adapted pathogen species (or bacterial pathovar or fungal forma specialis and represents the most robust and durable form of plant resistance in nature [40]. The presence of this defense system explains why plants are immune to the vast majority of potential pathogens and normally healthy. Molecular mechanisms underpinning NHR remain relatively unexplored.

NHR is a broad-spectrum plant defense that provides immunity to all members of a plant species in field conditions against all isolates of a microorganism that is pathogenic to other plant species. Upon landing on the surface of a non-host plant species, a potential bacterial pathogen initially encounters preformed and, later, induced plant defenses. One of the initial defense responses from the plant is PAMP-triggered immunity. Non-host plants also have mechanisms to detect non-host-pathogen effectors and can trigger a defense response referred to as effector-triggered immunity. This NHR response often results in a hypersensitive response at the infection site [41-43].

NHR against bacteria, fungi and oomycetes can be divided into two types [42]. Type I NHR does not produce visible symptoms whereas type II NHR results in a rapid hypersensitive response with cell death [42]. Type I NHR is much more common than type II NHR, and NHR of plants against the majority of unadapted pathogens belongs to Type I. Plants have evolved sophisticated mechanisms to exclude unadapted pathogens. An obvious initial requirement for plant disease is basic compatibility where appropriate physical and chemical signals from the plant are required for inducing cell differentiation and expressing essential pathogenicity genes [44, 45]. Presence of preformed plant physical and chemical barriers, including plant cell wall and plant surface antimicrobial enzymes and secondary metabolites, are often considered the first line of defense in plants against a pathogen before penetration [45]. Constitutive barriers are more likely to contribute to NHR to pathogens of other plant families than to pathogens of related plant species [46]. After these constitutive barriers are breached, plants have evolved inducible defense mechanisms against invading pathogens. An example of an inducible structural barrier is the formation of papillae. This local cell wall fortification is formed on the inner side of plant cell walls at the penetration site. The plant primary innate immune responses are mediated by transmembrane PAMP-triggered immunity that can halt further colonization of the pathogen [2]. However, effector triggered immunity is not just confined to adapted pathogen recognition and may also play a role in NHR, particularly against pathogens that colonize plant species closely related to non-host species [47].

Obligate biotrophic pathogens, with a specific lifestyle that keeps plant cells alive and minimizes tissue damage in susceptible hosts, are suitable for NHR studies [48]. *Arabidopsis* NHR to non-adapted biotrophic powdery mildews is based upon two successive, multicomponent and independently effective defense systems: *PEN* gene-mediated pre-invasion resistance and *EDS1*/*PAD4*/*SAG101* - controlled post-invasion immunity [40, 49-51]. Compared to powdery mildew fungi, the understanding of NHR mechanisms to rust fungi has lagged behind. *Puccinia* and *Uromyces* represent two large and important genera of rust fungi, which have damaged cereals and legumes, respectively, around the globe throughout history [52]. The emergence of Ug99, a new pathotype of the wheat stem rust pathogen that threatens global wheat production, is a reminder of the need for durable rust resistance in cereals [53, 54]. Much effort has been taken to study NHR to rust with non-host pathosystems of *Puccinia-Gramineae* and *Uromyces-dicotyledons* at histological and cytological levels, demonstrating that the majority of rust pathogens are arrested immediately after the formation of the first haustorium mother cell (HMC) in most non-host plant species [55-60]. Several recent studies have investigated the interaction of rust pathogens on non-host plants mainly at molecular levels, including growth of *U. vignae*, *P. triticina*, *Hemileia vastatrix* on *Arabidopsis* [48, 61, 62], *P. hordei* and *U. fabae* on wheat [63, 64], *P. triticina*, *P. hordei- murini*, *P. hordei-secalini*, *P. persistens* on barley [65], and *P. graminis*, *P. triticina*, *P. striiformis*, *P. hordei, Melampsora lini* on rice [44, 66]. These studies demonstrated that NHR to rust fungi is polygenically inherited and is an active response involving salicylic acid signaling.

7. Conclusive Remarks

Plants have a wide range of invaders to deal with including viruses, bacteria, fungi, insects and nematodes. Individual plant cells express receptors that recognise pathogen molecules and then trigger defence responses, which can include cell wall thickening, production of anti-microbial compounds and host cell death.

Plants have evolved multiple defense strategies for combating invading pathogens. The exterior surfaces of plants have waxy cuticles and preformed antimicrobials to prevent the entry of many would-be invaders. Cell walls provide an effective second barrier to any invaders that are able to gain access to interior spaces. Any invaders that overcome both barriers must still face the formidable task of overcoming the plant immune response. Plant immunity can be broken down into two components operating on different time scales. The basal defense system appears early in pathogen interaction, while the ***R*** gene-mediated defense operates on the time scale of hours.

The early basal response is mediated by PAMPs, which include lipopolysaccharide, peptidoglycan, bacterial flagellin, elongation factor EFTu, and mannans of yeast. PAMPs are recognized by PRR receptors located in the plasma membrane, activating a phosphorylation cascade upon binding, leading to the induction of early basal

resistance that plays a role in preventing colonization by nonpathogenic bacteria. Typically, this PAMP-triggered immunity is enough to halt infection before the microbe becomes established. Indeed, a connection between curbing of pathogen growth and the recognition of the PAMP flagellin by the receptor FLS2 has been demonstrated.

An effective virulence strategy of plant pathogens is to secrete effector proteins or DNA into the host cell to attempt to overcome plant defense systems. space form a pilus to inject

Gene-for-gene-mediated defense is inherited and is specific to a particular pathogen. Plants have dominant *R* genes whose products recognize those of the pathogen's complementary *Avr* alleles. Avr proteins are effector proteins secreted into the plant cell to promote pathogen virulence and to overcome host defenses. Localized programmed cell death, the hypersensitive response, is a hallmark of *R* gene-mediated defense and also a target of effector proteins.

References

[1] Dangl, J.L. and Jones, J.D.G. Plant pathogens and integrated defense responses to infection. Nature 411: 826-833, 2001

[2] Jones, J.D.G. and Dangl, J.L. The plant immune system. Nature 444: 323-329, 2006.

[3] Zipfel, C. Pattern-recognition receptors in plant innate immunity. Current Opinion in Immunology 2008, 20: 10-16.

[4] Yamada, T. Perception of pathogen signals and gene-for-gene hypothesis. Tanpakushitsu Kakusan Koso 44(15 Suppl): 2305-2307, 1999.

[5] Cai, X.Z., Xu, Y.P. and Zheng, Z. Avirulence genes of plant pathogens. Sheng Wu Gong Cheng Xue Bao 18(1): 5-9, 2002.

[6] Gururani, M.A., Venkatesh, J., Upadhyaya, C.P., Nookaraju, A., Pandey, S.K. and Park, S.W. Plant disease resistance genes: current status and future directions. Physiological and Molecular Plant Pathology 78: 51-65, 2012.

[7] Ausubel, F.M. Are innate immune signaling pathways in plants and animals conserved? Nature Immunology 6: 973-979, 2005.

[8] Zipfel, C. and Felix, G. Plants and animals: a different taste for microbes? Current Opinion in Plant Biology 8: 353-360, 2005.

[9] Chisholm, S.T., Coaker, G., Day, B. and Staskawicz, B.J. Host–microbe interactions: shaping the evolution of the plant immune response. Cell 124: 803-814, 2006.

[10] Lu, D., Lin, W., Gao, X., Wu, S., Cheng, C., Avila, J., Heese, A., Devarenne, T.P., He, P. and Shan L. Direct ubiquitination of pattern recognition receptor FLS2 attenuates plant innate immunity. Science 332(6038): 1439-1442, 2011.

[11] Danna, C. H., Millet, Y. A., Koller, T., Han, S.-W., Bent, A. F., Ronald, P. C. and Ausubel, F. M. The Arabidopsis flagellin receptor FLS2 mediates the perception of Xanthomonas Ax21 secreted peptides. Proceedings of the National Academy of Sciences USA 108 (22): 9286-9291, 2011.

[12] Sun, Y., Li L, Macho, A.P., Han, Z., Hu, Z., Zipfel, C., Zhou, J.M. and Chai J. Structural basis for flg22-induced activation of the Arabidopsis FLS2-BAK1 immune complex. Science 342(6158): 624-628, 2013.

[13] Garcia, A.V., Charrier, A., Schikora, A., Bigeard, J., Pateyron, S., de Tauzia-Moreau, M.L., Evrard, A., Mithöfer, A., Martin-Magniette, M.L., Virlogeux-Payant I. and Hirt H. Salmonella enterica flagellin is recognized via FLS2 and activates PAMP-triggered immunity in Arabidopsis thaliana. Molecular Plant [Epub ahead of print], 2013.

[14] Wu, K.L., Guo, Z.J., Wang, H.H. and Li J. The WRKY family of transcription factors in rice and Arabidopsis and their origins. DNA Research 12(1): 9-26, 2005.

[15] Pandey, S.P. and Somssich, I.E. The role of WRKY transcription factors in plant immunity. Plant Physiology 150 (4): 1648-1655, 2009.

[16] Stuiver, M.H. and Custers, J.H. Engineering disease resistance in plants. Nature 411: 865-868, 2001.

[17] He, S.Y. Elicitation of plant hypersensitive response by bacteria. Plant Physiology 11(2): 865-869, 1996.

[18] Dixon, R.A., Harrison, M.J. and Lamb, C.J. Early events in the activation of plant defense responses. Annual Reveiw of Phytopathology 32: 479-501, 1994.

[19] Goodman, R.N. and Novacky A. The hypersensitive reaction in plants to pathogens. A resistance phenomenon. American Phytopathological Society Press, USA, 1994.

[20] Ryals, J., Uknes, S. and Ward, E. Systemic acquired resistance. Plant Physiology 104: 1109-1112, 1994.

[21] Conrath, U. Systemic acquired resistance. Plant Signaling and Behavior 1(4):179-184, 2006.

[22] Maldonado, A.M., Doerner, P., Dixon, R.A., Lamb C.J. and Cameron R.K. A putative lipid transfer protein involved in systemic acquired resistance signalling in Arabidopsis. Nature 419: 399-403, 2002.

[23] Park, S.-W., Kaimoyo, E., Kumar, D., Mosher, S. and Klessig, D.F. Methyl salicylate is a critical mobile signal for plant systemic acquired resistance. Science 318: 113-116, 2007.

[24] Jung, H.W., Tschaplinski, T. J., Wang, L., Glazebrook, J. and Greenberg J.T. Priming in systemic plant immunity. Science 24: 89-91, 2003.

[25] Chanda, B., Xia, Y., Mandal, M. K., Yu, K., Sekine, K.-T., Gao, Q.-M., Selote, D., Hu, Y., Stromberg, A., Navarre, D., Kachroo, A. and Kachroo, P. Glycerol-3-phosphate is a critical mobile inducer of systemic immunity in plants. Nature Genetics 43: 421-427, 2011.

[26] Chaturvedi, R., Krothapalli, K., Makandar, R., Nandi, A., Sparks, A., Roth, M.R., Welti, R. and Shah J. Plastid ω-3 desaturase-dependent accumulation of a systemic acquired resistance inducing activity in petiole exudates of Arabidopsis thaliana is independent of jasmonic acid. Plant Journal 54: 106-117, 2008.

[27] Shah, J. Plants under attack: systemic signals in defense. Current Opinion in Plant Biology 12: 459-464, 2009.

[28] Návarová, H., Bernsdorff, F., Döring, A.-C. and Zeier, J. Pipecolic acid, an endogenous mediator of defense amplification and priming, is a critical regulator of inducible plant immunity. Plant Cell 24: 5123-5141, 2012.

[29] Dempsey, D.A. and Klessig, D.F. SOS – too many signals for systemic acquired resistance? Trends in Plant Science 17: 538-545, 2012.

[30] De Vos, M., Van Oosten, V.R., Van Poecke, R.M., Van Pelt, J.A., Pozo, M.J., Mueller, M.J., Buchala, A.J., Métraux, J.P., Van Loon, L.C., Dicke, M. and Pieterse, C.M. Signal signature and transcriptome changes of Arabidopsis during pathogen and insect attack. Molecular Plant-Microbe Interactions 18(9): 923-937, 2005.

[31] Shah, J. and Zeier, J. Long-distance communication and signal amplification in systemic acquired resistance.Frontiers in Plant Science 4: 30, 2013.

[32] Gruner, K., Griebel, T., Návarová, H., Attaran, E. and Zeier, J. Reprogramming of plants during systemic acquired resistance. Frontiers in Plant Science 4: 252, 2013.

[33] Mishina, T.E. and Zeier, J. The arabidopsis flavin-dependent monooxygenase fmo1 is an essential component of biologically induced systemic acquired resistance. Plant Physiology 141(4): 1666-1675, 2006.

[34] Taiz, L. and Zeiger, E. Plant Physiology. Sinauer Associates, Inc., Publishers, 5th ed. USA, 2010.

[35] Grunwald, N.J., Navarre, D.A. and Coffman, V.A. Evaluation of plant activators for management of Fusarium root rot on green pea. Fungicide and Nematicide Tests, 58: V001, 2003.

[36] Santner, A. The JAZ proteins link jasmonate perception with transcriptional changes. Plant Cell 19 (12): 3839-3842, 2007.

[37] Pauwels, L. Goossens, A. The JAZ proteins: a crucial interface in the jasmonate signaling cascade. Plant Cell 23(9): 3089-3100, 2011.

[38] Zander, M., La Camera, S., Lamotte, O., Métraux, J.P. and Gatz, C. Arabidopsis thaliana class-II TGA transcription factors are essential activators of jasmonic acid/ethylene-induced defense responses. Plant Journal 61(2): 200-2010, 2010.

[39] Stotz, H.U., Mueller, S., Zoeller, M., Mueller, M.J. and Berger, S. TGA transcription factors and jasmonate-independent COI1 signalling regulate specific plant responses to reactive oxylipins. Journal of Experimental Botany 64(4): 963-975, 2013.

[40] Lipka, U., Fuchs, R., Kuhns, C., Petutschnig, E. and Lipka, V. Live and let die-Arabidopsis nonhost resistance to powdery mildews. European Journal of Cell Biology 89: 194-199, 2010.

[41] Heath, M.C. Nonhost resistance and nonspecific plant defenses. Current Opinion in Plant Biology 3(4): 315-319, 2000.

[42] Mysore, K.S. and Ryu, C.M. Nonhost resistance: how much do we know? Trends in Plant Science 9(2): 97-104, 2004.

[43] Senthil-Kumar, M. and Mysore, K.S. Nonhost resistance against bacterial pathogens: retrospectives and prospects. Annual Review of Phytopathology 51: 407-427, 2013.

[44] Ayliffe, M., Jin, Y., Kang, Z., Persson, M., Steffenson, B., Wang, S. and Leung, H. Determining the basis of nonhost resistance in rice to cereal rusts. Euphytica 179: 33-40, 2011.

[45] Thordal-Christensen, H. Fresh insights into processes of nonhost resistance. Current Opinion in Plant Biology 6: 351-357, 2003.

[46] Niks, R.E. and Marcel, T.C. Nonhost and basal resistance: how to explain specificity? New Phytology 182: 817-828, 2009.

[47] Schulze-Lefert, P. and Panstruga, R.A. Molecular evolutionary concept connecting nonhost resistance, pathogen host range, and pathogen speciation. Trends in Plant Science 16: 117-125, 2011.

[48] Mellersh, D.G. and Heath, M.C. An investigation into the involvement of defense signaling pathways in components of the nonhost resistance of Arabidopsis thaliana to rust fungi also reveals a model system for studying rust fungal compatibility. Molecular Plant-Microbe Interactions 16: 398-404, 2003.

[49] Collins, N.C., Thordal-Christensen, H., Lipka, V., Bau, S., Kombrink, E., Qiu, J.L., Hückelhoven, R., Stein, M., Freialdenhoven, A. and Somerville C. SNARE-protein -mediated disease resistance at the plant cell wall. Nature 425: 973-977, 2003.

[50] Lipka, V., Dittgen, J., Bednarek, P., Bhat, R., Wiermer, M., Stein, M., Landtag, J., Brandt, W., Rosahl, S. and Scheel D. Pre- and postinvasion defenses both contribute to nonhost resistance in Arabidopsis. Science 310: 1180-1183, 2005.

[51] Stein, M., Dittgen, J., Sánchez-Rodríguez, C., Hou, B.H., Molina, A., Schulze-Lefert, P., Lipka, V. and Somerville, S. Arabidopsis PEN3/PDR8, an ATP binding cassette transporter, contributes to nonhost resistance to inappropriate pathogens that enter by direct penetration. Plant Cell 18: 731-746, 2006.

[52] Voegele, R.T. Uromyces fabae: development, metabolism, and interactions with its host Vicia faba. FEMS Microbiology Letters 259: 165-173, 2006.

[53] Ayliffe, M., Singh, R. and Lagudah, E. Durable resistance to wheat stem rust needed. Curr. Opin. Plant Biol., 2008, 11, 187-192.

[54] Stokstad, E. Plant pathology. Deadly wheat fungus threatens world's breadbaskets. Science 315: 1786-1787, 2007.

[55] Heath, M.C. Light and electron microscope studies of the interactions of host and non-host plants with cowpea rust-Uromyces phaseoli var. vignae. Physiology and Plant Pathology 4: 403-408, 1974.

[56] Heath, M.C. Partial characterization of the electron-opaque deposits formed in the non-host plant, French bean, after cowpea rust infection. Physiology and Plant Pathology 15: 141-144, 1979.

[57] Heath, M.C. Resistance of plants to rust infection. Phytopathology 71: 971-974, 1981.

[58] Niks, R. Comparative histology of partial resistance and the nonhost reaction to leaf rust pathogens in barley and wheat seedlings. Phytopathology 73: 60-64, 1983 .

[59] Niks, R. Haustorium formation by Puccinia hordei in leaves of hypersensitive, partially resistant, and nonhost plant genotypes. Phytopathology 73: 64-66, 1983.

[60] Hoogkamp, T., Chen, W.Q. and Niks, R. Specificity of prehaustorial resistance to Puccinia hordei and to two inappropriate rust fungi in barley. Phytopathology 88: 856-861, 1998.

[61] Shafiei, R., Hang, C., Kang, J.G.U. and Loake, G.J. Identification of loci controlling non-host disease resistance in Arabidopsis against the leaf rust pathogen Puccinia triticina. Molecular Plant Pathology 8: 773-784, 2007.

[62] Azinheira, H.G., Silva, M.C., Talhinhas, P., Medeira, C., Maia, I., Petitot, A.-S. and Fernandez D. Non-host resistance responses of Arabidopsis thaliana to the coffee leaf rust fungus (Hemileia vastatrix). Botany 88: 621-629, 2010.

[63] Zhang, H., Wang, C., Cheng, Y., Wang, X., Li, F., Han, Q., Xu, J., Chen, X., Huang, L. and Wei G. Histological and molecular studies of the non-host interaction between wheat and Uromyces fabae. Planta 234: 979-991, 2011.

[64] Prats, E., Martinez, F., Rojas-Molina, M. and Rubiales D. Differential effects of phenylalanine ammonia lyase, cinnamyl alcohol dehydrogenase, and energetic metabolism inhibition on resistance of appropriate host and nonhost cereal-rust interactions. Phytopathology 97: 1578-1583, 2007.

[65] Jafary, H., Albertazzi, G., Marcel, T.C. and Niks R.E. High diversity of genes for nonhost resistance of barley to heterologous rust fungi. Genetics 178: 2327-2339, 2008.

[66] Ayliffe, M., Devilla, R., Mago, R., White, R., Talbot, M., Pryor, A. and Leung, H. Nonhost resistance of rice to rust pathogens. Molecular Plant-Microbe Interactions 24: 1143-1155, 2011.