

International Journal of Plant Science and Horticulture

Mini Review

Open Access

Plant Virus Interaction: Layers of Plant Antiviral Immunity

Pedro Filho Noronha Souza

Department of Biochemistry and Molecular Biology, Center of Science, Federal University of Ceara, Fortaleza, Ceara, Brazil

*Correspondig Author: Pedro F. N. Souza, Department of Biochemistry and Molecular Biology, Center of Science, Federal University of Ceara, Fortaleza, Ceara, Brazil, Tel.: +1(402) 416-6370; +55 (85) 3366-9823; Email: pedro.de-souza@unl.edu; pedrofilhobio@gmail.com

Received Date: Apr 12, 2019 / Accepted Date: Apr 22, 2019 / Published Date: Apr 24, 2019

Abstract

Plant viruses are wise pathogens. The prevention of viral infection is a hard task for plants. However, during the evolutionary time, the "arms race" plants learn somehow to perceive and counterattack viral infection. Up to date is known plants have at least three important way to defend themselves from the viral infection, (1) dominant resistant; (2) recessive resistance; and (3) RNA silencing. Right after recognition of virus plants can begin a quite complex signaling pathway resulting in activation of many defense proteins and RNA silencing. Also, the plants could make some changes in essential proteins to viral infection, and then prevent viral replication and disease establishment. Here, will be reviewed the layers of antiviral immunity and they work together to establish a defense against the virus. Therefore, this knowledge can be beneficial to gene editing to engineer resistance to plant viruses.

Keywords: Plant Defense; Plant Viruses; Antiviral Immunity; Plant defense mechanism

Cite this article as: Pedro Filho Noronha Souza. 2019. Plant Virus Interaction: Layers of Plant Antiviral Immunity. Int J Plant Sci Hor. 1: 89-94.

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Introduction

Plant defense mechanisms are divided into two phases; (1) the Pathogen-Triggered Immunity (PTI), which is basal resistance and; (2) Effector-Triggered Immunity (ETI) or induced resistance, the second line of defense of plants [1,2]. The PTI response is rapidly active by plants after recognizing pathogens effectors, which could be MAMPS or PAMPs (Microbe/pathogen-associated molecular patterns, e.g., bacterial flagellin), DAMPs (Damage-associated molecular patterns, e.g., fungal haustorium), and VAMPs (Viralassociated molecular patterns, e.g., doublestranded RNA of viruses). The recognition of pathogens effectors is performed by Pattern Recognition Receptors (PRR) [3-6].

PRRs are proteins with an extracellular receptor domain, a transmembrane domain holding protein anchored into the plasma membrane, and a cytoplasmic domain with a kinase function necessary to trigger the signaling of defense responses within the cell [3]. After



IJPSHPlant Virus Interaction: Layers of Plant Antiviral ImmunityDOI: https://doi.org/10.36811/ijpsh.2019.110010IJPSH: April-2019: Page No: 89-94

recognition of molecular patterns by PRRs, several events are induced to prevent infection. Among those events, are included Ca²⁺ ion signaling and oxidative burst, mediated by Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS), as well as activation of different proteins such as mitogen-activated kinase (MAPKs) and calcium-dependent kinase (CDPK), which induce reprogramming of the expression of genes related to pathogenesis (PR) (Pathogenesis-Related Genes) [7,8].

In order to overcome the PTI, during the evolution time, pathogens acquired the ability to produce effector molecules to suppress PTI response, denominated Avr (avirulence proteins). In turn, plants co-evolved and started to synthesize proteins called Nucleotide Binding site Leucine-Rich Repeat (NBS-LRR), known as *R* proteins, which recognize these pathogens' effector molecules [1,2,9].

After recognition of pathogens' effector molecules, several biochemical responses are involving signal induced, transduction, resulting in local and systemic responses. These initial responses are based on the oxidative burst, which releases ROS and RNS, resulting in the HR and programmed cell death (PCD) and induction of the expression of defense genes, systemic acquired resistance (SAR) [10]. SAR is essential in the defense of the plant for 4 important reasons: 1) development of resistance at sites far from the site of infection [11]; 2) activity against a broad spectrum of pathogens, including viruses, fungi, bacteria and nematodes [10]; 3) long-lasting protection; and 4) expression of genes related to the synthesis of PR-proteins (Pathogenesis-Related Proteins) Based on the subject abovementioned, in this review will be addressed some specific aspects of plant-virus interaction. During plant-virus interaction many layers of resistance are active and combined could result in plant resistance to viruses.

Plant-Virus Interaction

Plant-virus interaction has some unique characteristics compared to other plant-pathogen interactions. Hull [12], discusses the existence of several types of resistance in plants toward viruses. Also, Nicaise [13], argues that plant resistance against viruses is composed by dominant, recessive and RNAi-mediated resistance, which are complementary in such way and divided into a temporal scale and spatial always targeting the virus-derived molecules [13].

Dominant Resistance

The dominant resistance is antiviral immunity of plant defense mediated by the NBS-LRR proteins, encoded by dominant resistance (R) genes, which recognize avirulence (Avr) gene products, initiating FTI defense mechanisms, and avoiding [14-16]. Several NBS-LRR proteins have been described as playing a crucial role in the defense against viral infection. These responses may be mediated by recognition, directly or indirectly, of viral effectors, or interaction with proteins that were modified by viral effectors.

The defense responses mediated by NBS-LRR proteins are well described in Nicotiana tabacum plants, which have a well-studied dominant *R*-resistance gene encoding a TIR-NBS-LRR protein that interacts with the replicase from TMV (Tobacco Mosaic Virus, Tobamovirus), triggering defense responses [17]. In addition, in Solanum tuberosum plants, the Rx1 gene encodes another type of CC-NB-LRR type protein, which recognize the PVX viral capsid protein (Potex virus X, Potexvirus), inducing plant defense responses [18]. In both N. tabacum and S. tuberosum, activation of NBS-LRR initiates a cascade of MAPK proteins followed by an increase in Ca2 + influx, inducing HR and consequent PCD in order to prevent the virus (biotrophic organisms) spread to new healthy local and systemic tissue [19].



The recognition of virus infection by plant also result in activation of many defense enzymes and deposition of phenolic compounds that can be useful in plant defense [20-22]. To support all the modification required to plants cope with the viral infection a high demand of energy is required, which is supported by the increase in photosynthesis index [20,23]. For example, cowpea (*Vigna unguiculata*) resistant to Cowpea severe mosaic virus (CPSMV) infection increase the activity of many enzymes such as Superoxide dismutase, phenylalanine ammonium lyase, guaiacol peroxidase as well as high photosynthetic rates [20,23].

Recessive Resistance

Recessive resistance (immunity) is characterized by the virus inability to infect the plant, due to the lack of compatibility between proteins encoded by the virus and plant proteins, a process necessary for viral replication and infection establishment [15]. Several genes involved in recessive resistance in plants have been identified, cloned and characterized in many plant cultures, all encoding proteins belonging to families of translation initiation factors (eIF) which are proteins necessary for viral replication in the host, indicating that type of resistance is more common against viruses [7,24].

Recessive resistance occurs preferentially in inoculated leaf protoplasts, where the viral replication process occurs, impairing virus multiplication in the cell as well as cell-to-cell movement [7,24]. The most studied recessive genes are those encoding translation initiation factors of the 4E (eIF4E) and 4G (eIF4G) families that together form the eIF4F complex, and their respective isoforms, eIF(iso)4E and eIF(iso)4G, which are essential in the translation process [25].

The eIF4E subunit is a 24 kDa protein, which helps the interaction between the mRNA with ribosomes in the plant [26]. The eIF4G (~200 kDa) interacts with eIF4E and other initiation factors, including eIF4A, eIF3, and the polyadenine chain binding protein to stabilize the mRNA and the complex important for protein synthesis [15,27].

Studies with Potyviruses have shown that plant resistance may be associated with the inability of VPg (viral-genome linked protein) to bind to either eIF4E or eIF(iso)4E of plants, without this interaction virus cannot replicate in plant cells [28]. This inability to bind is the result of amino acid mutations exposed on the surface of the eIF4E preventing the recognition of eIF4E or eIF(iso)4E by VPg.

RNA Silencing

Recently, a new induced defense mechanism, which is present in plants, and virtually in all kingdoms, has been discovered, called RNA silencing (RNAi). RNAi is a small non-coding RNA molecule (sRNA) ranging in size from 20-30 nucleotides. RNAi is a specific mechanism gene expression of inhibition. RNAi is involved in the regulation of various physiological processes of plants, is the main route of defense of plants against viruses [29].

The classification of sRNAs in plants is divided into two groups, according to their precursor and biogenesis pathway: miRNA (microRNA) and siRNA (small interfering RNA). The miRNAs are 21-24 nucleotides long and are originated from endogenous imperfect base pairing RNA molecules. The siRNAs have 23-30 nucleotides of size and are generated from exogenous dsRNA (double-stranded RNAs), and require the activity of the RNA-dependent RNA polymerase enzyme [30,31].

The siRNAs control gene expression in plants or pathogens by two distinct mechanisms, posttranscriptional gene silencing (PTGS) or transcriptional gene silencing (TGS). Both miRNA and siRNA induce PTGS through cleavage / degradation or inhibition of translation by the so-called RNA-Induced Silencing Complex (RISC) [32,33], which is



formed by ribonucleoproteins, Dicers, TRBP (three double-stranded RNA-binding domains protein), AGO protein (RNase function) represents the center of the complex, and siRNA or miRNA.

After recognition of dsRNA, which is exclusive from virus replication, Dicers enzymes degrade the dsRNA in small duplex fragments that are incorporated into the RISC cytoplasmic complex to degrade mRNA targeted. On the other hand, TGS acts on gene regulation through DNA methylation, histone modification or even chromatin modification, which is usually performed by siRNA, although some specific classes of miRNAs can perform this process [34].

Several studies cite the importance of RNAi silencing in plant defense against viruses. Recently, Garcia-Ruiz et al. [35], have shown that silencing by RNAi is essential for the defense of Arabidopsis plants against TuMV (Turnip Mosaic Virus, genus Potyvirus). Cruz and Aragão [36] demonstrated that transgenic bean plants, capable of inhibiting the production of the viral protease cofactor via RNAi, showed resistance to CPSMV when compared to untransformed plants susceptible to the virus.

Conclusion

As discussed above, the plant-virus interaction is quite complex involving many defense layers that work together to improve plant antiviral immunity. Although plants have defense mechanisms most case, they seem to be not enough to result in plant defense. Because in many case plants suffer from severe viral infection. Up to date, viral infections lead to millions of dollars worldwide every year.

The understanding of plant defense mechanisms is important to produce mutant resistant plants that can be applied to reduce the losses caused by viral infection. As discussed above, the most promising to mutant production are recessive resistance and RNAi. The first can be done by producing plants mutant in the translation initiation factors. The second could be applied in the production of plants expressing some siRNA that target viral proteins.

Acknowledgment

This study was supported by CAPES (Coordination of Improvement of Higher Education. Toxinology Project, Process number: 431511/2016-0).

Author contribution

Pedro F. N. Souza conceived and wrote this manuscript.

Compliance with ethical standards Competing interests

The author declares that no financial or any other conflicting interests exist.

Ethical approval

The author did not perform any study with animals or humans' participants to produce this manuscript

Informed consent

The author reads and approves this manuscript.

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