

How Plants Face the Viruses Challenge?

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ARTICLE INFO

Received: 📅 March 15, 2022

Published: 📅 April 05, 2022

Citation: José Efraín González Ramírez, Katia Ojito Ramos, Orelvis Portal Villafaña. How Plants Face the Viruses Challenge?. Biomed J Sci & Tech Res 43(1)-2022. BJSTR. MS.ID.006850.

ABSTRACT

Abbreviations: MeSA: Methyl Salicylic Acid; AzA: Azelaic Acid; DA: Dihydroabietinal; G3P: Glycerol-3-Phosphate; PIP: Pipecolic Acid; TEV: Tobacco Etch Virus; NSP: Nuclear Shuttle Protein

Mini Review

The interaction of virus plants is marked by the immobility of the first. This fact makes them impossible to "avoid" the viruses arrival. For their defense, plants have a group of physical barriers and a group of biochemical mechanisms Jones and Dangl [1-3]. Viruses have the help of vectors to overcome the plants physical barriers. Therefore, the final battle between plant and viruses is defined at cell and tissue level. In this sense, the biotrophic character of viruses is of particular interest, because they completely depend on the machinery of plant cells to complete their cycle, an aspect that determines the joint evolution of biochemical attacks and counterattacks between plants and viruses. Plants constantly face potentially pathogenic agents; for this, antiviral defense system is, in several aspects, more complex than that facing fungi and bacteria Liu, et al. [4,5]. At each stage of the virus-plant confrontation, complex responses and counter-responses are developed during the evolutionary process Zvereva, et al. [6-8]. General regulatory systems at the cellular level, less specific, but with a broad action

spectrum and specific molecular mechanisms combine to prevent viral replication, cell-cell passage and long-distance movement within the host. Six molecular mechanisms have been described in plants to deal with viruses.

Innate Antiviral Immunity

Plants recognize compounds of viral origin produced during infection that act as PAMP (Pathogen-Associated Molecular Pattern) or other effectors (capsid protein, movement proteins and replicase) as avirulence factors that activate PTI responses (from English: PAMP-Triggered Immunity) and ETI (Effector-Triggered Immunity), respectively Zvereva, et al. [6,9,10]. Both mechanisms result in the establishment of resistance in distant and non-infested tissues called SAR (Systemic Acquired Resistance) Glazebrook [4,11]. SAR can be activated by phytohormones such as methyl salicylic acid (MeSA), azelaic acid (AzA), dihydroabietinal (DA), glycerol-3-phosphate (G3P) and pipecolic acid (PIP) Shine, et al. [12,13].

RNA Silencing

Virus-infected plants accumulate dsRNAs (Virus-Derived Double-Stranded RNA), which induce the mechanism of RNA silencing or RNA interference. The dsRNAs are recognized by the DCL (Dicer Like) proteases that cut them into small pieces of 21 to 24 nt called vsiRNAs (virus-derived short-interfering small RNAs). VsiRNAs regulate antiviral defense by inducing transcriptional and post-transcriptional silencing of viral genes Ceniceros-Ojeda, et al. [9,14,15]. The visRNAs associate with the AGO (Argonaute) proteins that are part of the RISC (RNA-Induced Silencing Complex) that cut into the viral single-stranded RNA. This recognition signal is transmitted to cells far from the initial point of infection, enhancing defensive responses in them Yang, et al. [16,17]. The RNA silencing mechanism is dependent on environmental factors such as temperature Rosa, et al. [18] and can explain various types of virus-plant interactions i.e. cross protection, phenotype recovery, non-host resistance Prasad, et al. 2019 and reversion of viral infection Gibson, et al. [19,20].

Suppression of Protein Translation

Plants strictly regulate protein biosynthesis. In this sense, the antiviral activity of RIPs (Ribosome-Inactivating Proteins) characterized in *Phytolacca americana* L. against the potyvirus tobacco etch virus (TEV), PVY, among others, has been demonstrated. The expression of RIPs increases under stress conditions Domashevskiy, et al. [21]. Another mechanism for the suppression of protein synthesis is NIK1 (from the English: Nuclear Shuttle Protein (NSP) Interacting Kinase 1) and its homologues NIK2 and NIK3; these appear anchored to the cell membrane that can interrupt the global synthesis of proteins in the plant Zorzatto, et al. [22].

Atypical Dominant Viral Resistance

The exploration of resistance mechanisms against viruses has resulted in the discovery of dominant resistance genes that are independent of those involved in the innate antiviral response. The products of the former, distinctively, act directly on viral proteins, preventing their activity and are referred to as ADVRPs (Atypical Dominant Viral Resistance Proteins). The most important ADVRPs are lectins e.g. RTM (Restricted TEV Movement) with activity against several potyviruses such as TEV, lettuce mosaic virus and plum pox virus, preventing their long-distance movement Choi, et al. [23]. Other ADVRPs have demonstrated their effect against other potyviruses such as the sugarcane mosaic virus Wu, et al. [24]. Protein degradation through ubiquitination and autophagy Ubiquitination is the major regulatory mechanism of protein synthesis in plants and has been shown to be effective against the

proliferation of the tomato yellow leaf curl China virus, the turnip yellow mosaic virus and the tobacco mosaic virus. On the other hand, plants use a selective autophagy mechanism to degrade viral proteins, riboproteins Li, et al. [25] demonstrated for the NIB protein of potyviruses Shen, et al. [26].

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Protein Degradation Through Ubiquitination and Autophagy

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Regulation of RNA Translation

Plants have three mechanisms for regulating the translation of mRNAs i.e. NGD (from English: No-Go Decay), NSD (Non-Stop Decay) and NMD (Nonsense-Mediated mRNA Decay) Navickas, et al. [27]; Powers, et al. 2020. These mechanisms allow the degradation of viral RNAs Li, et al. [28] and have shown a predilection for visRNA Paudel, et al. [29]. Both NGD and NSD are effective against viruses with long adenine chains Szádeczky-Kardoss, et al. [30], while NMD has demonstrated its action against single-chain RNA + viruses Li, et al. [28].

Final Consideration

The interaction of these molecular mechanisms increases the defensive response of plants to face viruses Wu, et al. [24,30]. In order to develop diseases, viruses must counteract, at least in part, these barriers Rodamilans, et al. [5,31,32]. However, the development of the disease is not the most common result of the virus and plant interaction.

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ISSN: 2574-1241

DOI: 10.26717/BJSTR.2022.43.006850

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