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PLANTS DEFENSE RESPONSE AGAINST RECOGNITION OF A VIRAL PATHOGEN

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Abstract

Plant viruses are infective particles considered obligate intracellular parasites usually composed of positive single-stranded ribonucleic acid (ssRNA) and only in a few cases by single stranded or double-stranded deoxyribonucleic acid (ssDNA and dsDNA, respectively). Viruses can only enter the plant cell passively through wounds caused by physical injuries due to environmental factors or by vectors. Among vectors, several species of insects, mites, nematodes and some soil inhabitant fungi can transmit specific viruses. In the cytoplasm, the RNA disassembles replicates, converts its mRNA to proteins, and mobilizes locally and systemically.

Keywords : Plant immune, Infection, defense, pathogens.

Introduction

Viruses use energy and proteins from the host cell to perform these processes. If the viral particle is not recognized by the host plant, a compatible interaction between the plant and the virus is established. However, if the plant recognizes the viral particle, an incompatible interaction that is unfavorable for the virus is established. It is known that plants can recognize the virus, limiting it to the site of the infection. A series of complex cascade defense reactions can be induced, limiting virus replication and virus movement within the host plant (Hammond-Kosack and Jones, 2000).

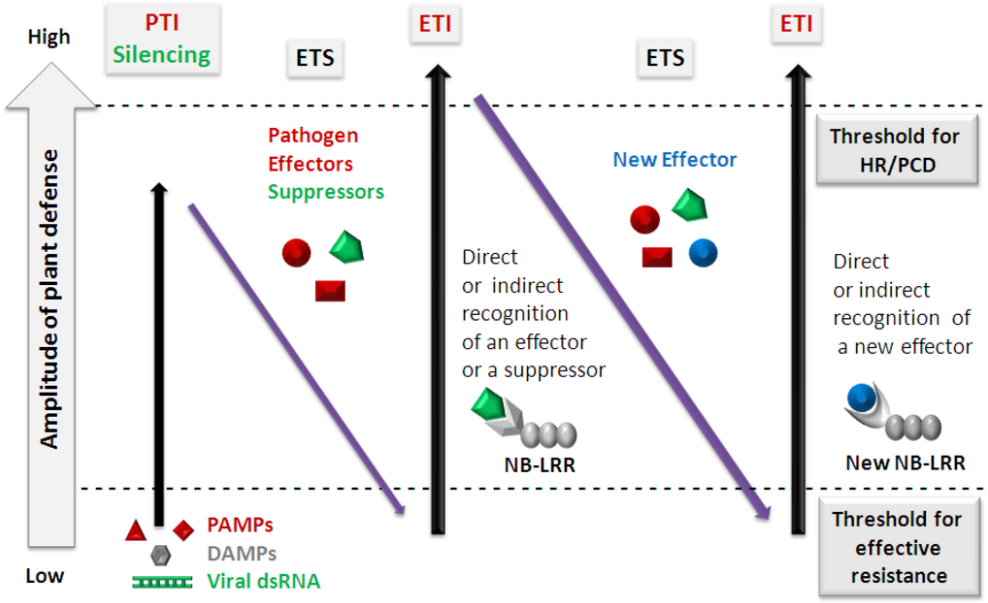
Plant virus recognition

All plant cells are naturally and frequently exposed to microorganisms. To cope with invading pathogens, plant cells evolved a sophisticated immune system under constant pressure for dominance over the pathogens' virulence strategies, which in turn coevolved to escape the host recognition system (Jones and Dangl, 2006). Plants have developed recognition mechanisms that allow them to defend themselves against parasites (parasitic plants, insects, and some invertebrate animals) and pathogenic agents like viruses, viroids, bacteria, phytoplasms, fungi, and nematodes. Some of these mechanisms act as physical and chemical barriers that prevent infection by pathogens. Compatibility and incompatibility reaction Plants have developed a defence mechanism at the molecular level based on the gene for gene theory described by Flor (1971). This model is defined by the expression of a resistance gene (R) in the plant, which can bind directly or indirectly to the product of the avirulence gene(avr) of the pathogen (Ellis *et al.*, 2000a). R proteins act as receptors and AVR ligands as elicitor proteins (Ellis *et al.,* 2000b).

The first layer of the plant immune system is represented by the pattern recognition receptors (PRRs) at the cell surface, which recognize either conserved signature molecules produced by the pathogens, designated pathogen associated molecular patterns (PAMPs), or endogenous damage/danger signals associated with pathogen invasion, designated danger or damage associated molecular patterns (DAMPs) (Choi and Klessig, 2016; Ma *et al.*, 2016). The sensing of PAMPs by PRRs activates PAMP triggered immunity (PTI), leading to a rapid, non-specific response to a broad range of pathogens (Ma *et al.*, 2016). To counterattack this first layer of defence, adapted

pathogens deliver virulence effectors in the host cell cytoplasm, which prevent the activation of PTI and elicit effector triggered susceptibility (ETS) Wang and Wang, 2018.

The plants are respond by the activation of defense gene, the formation of reactive oxygen species (ROS), the synthesis of pathogenesis-related (PR) proteins, localized cell wall reinforcement and the production of antimicrobial compounds. Recognition of a pathogen often triggers a localized resistance reaction, which is knows as hypersensitive response (HR), which is characterized by rapid cell death at the site of infection (Hammond-Kosack and Jones, 1997). During the HR, chemical oxidant species are produced (Lamb and Dixon, 1997), cellulose (Shimomura and Dijkstra, 1975) and lignin are synthesized, the levels of salicylic acid increase (Naylor *et al*., 1998) and pathogenesis related proteins are produced (Yalpani *et al.,* 1991). In response, plant cells have evolved intracellular nucleotide binding leucine rich repeat (NLR) receptors, which recognize the virulence effectors in a highly specific manner to activate the second level of plant defence and are designated as effector triggered immunity (ETI; Jones and Dangl, 2006). As a result, plants limit the short and long-distance movement of the pathogen.



plant defensive model against virus pathogen Zvereva & Pooggin, 2012

Systemic Necrosis Responses

The resistant (or incompatible) host–virus interactions, most susceptible (or compatible) virus infections do not trigger hypersensitive response (HR) and do not produce localized necrotic lesion phenotypes to limit the virus spread in the host plants. However, a similar or dissimilar form of necrosis, termed systemic necrosis, is observed in susceptible interactions. For example, systemic necrosis was reported in *Nicotiana benthamiana* with mixed infections of potexviruses, PVX, or Plantago asiatica mosaic virus (PLAMV) isolate Li1 and Potato virus Y (Ozeki *et al.,* 2006); Cucumber mosaic virus (CMV) and satellite RNA-D infected tomato (Xu and Roossinck, 2000); and Panicum mosaic virus (PMV) and its satellite virus (SPMV) infected *Brachypodium distachyon* and millet species (*Panicum miliaceum*, *Pennisetum glaucum,* and *Setaria italica*) Mandadi and Scholthof, 2012.

Systemic necrosis resembles necrosis commonly observed in lesion mimic mutants, resulting either from constitutive or uncontrolled cell death (Moeder and Yoshioka, 2008). Systemic necrosis is thought not to preclude virus multiplication or its systemic movement, thereby resulting in a

susceptible infection. The relatively well understood mechanisms leading to HR and associated necrosis, we are just beginning to understand the molecular processes that underlie systemic necrosis responses and how systemic necrosis responses relate to antiviral immunity. Recent findings revealed that despite the differing roles or outcomes, systemic necrosis and HR-associated necrosis share remarkable similarities at the biochemical and molecular level. For example, both systemic necrosis and HR-associated necrosis involve programmed cell death, alter expression of similar defense-related genes, and trigger ROS accumulation (Xu *et al.,* 2012). Komatsu *et al*. (2010) investigated the molecular determinants leading to systemic necrosis elicited by infection with PLAMV, a potexvirus, in *N. benthamiana*.

Systemic Acquired Resistance

SAR is triggered during an incompatible interaction involving Avr and R proteins in the primary infected cells. The resistance is transduced to the noninfected distant tissues. Although the exact mechanisms of SAR are not defined, it is initiated through a local interaction among Avr and R proteins and results in accumulation of phytohormones such as SA and JA in the distant tissues (Vlot *et al.,* 2008).

SAR is a long-lasting immune response primed to provide distant tissue resistance against subsequent infections. In the case of TMV-triggered SAR, the response persists up to 3 weeks (Ross, 1961). Interestingly, the transgenerational stability of SAR requires NPR1, as progeny of the SA-insensitive npr1-1 mutant plants failed to possess SAR in the next generation. This induced resistance phenomena is also triggered in the progeny of plants exposed to caterpillar herbivory (Rasmann *et al.,* 2012). In this case, the stable resistance response is dependent on intact JA signaling and requires the biogenesis of short interfering RNA that could mediate the epigenetic chromatin modifications (Rasmann *et al.,* 2012).

In viral infections, in addition to the dominant R gene–related resistance responses, another form of recessive resistance exists that is typically derived by a loss of function in host proteins critical for the establishment of disease (Gururani *et al*., 2012). For example, amino acid mutations in the eukaryotic translation initiation factor, eIF4E, mediates resistance against several viruses in Arabidopsis, tomato, pepper, pea (*Pisum sativum*), melon (*Cucumis melo*), and barley (*Hordeum vulgare*) (Piron *et al*., 2010).

Hypersensitive response

The term hypersensitivity indicates that the host cells are 'over-sensitive' to the presence of the pathogen. Host cells suicide in the presence of the pathogen, preventing further spread of the infection. Virus-associated chlorotic lesions or spots, ringspots, and necrotic lesions on leaves, stems, and fruits are various symptomatic manifestations of host immune responses triggered in the infected cells. In the instances of HR and necrosis, virus accumulation is limited to a few hundred infected cells. Classically, HR-mediated resistance is known to be triggered when a pathogen-encoded avirulence factor (Avr) is recognized in plants by a host R gene product.

According to current plant immunity descriptions, there are two layers of plant immune responses against microbial pathogens. First, the recognition of certain conserved pathogen- or microbe-associated molecular patterns (P/MAMPs) by plant pattern recognition receptors (PRRs) initiates the so called P/MAMP-triggered immune (PTI) response, which may occasionally result in HR. As a counter-response to plant PTI defenses, adapted microbes deliver specific effector proteins into plant cells, which compromise PTI defenses and interfere with host defense signaling. To further defend the action of the microbial effectors, plants evolved specific surveillance systems involving receptor-like proteins (R proteins) that directly or indirectly recognize the microbial effectors or

monitor their activities in the cell to trigger the so-called effector-triggered immune (ETI) response. Paradoxically, an effector protein can also be the elicitor of ETI defense. Whether the effector or elicitor role of an effector protein prevails is primarily predicated on the presence of the complementing R gene in the plant. The ETI responses, and to a somewhat lesser extent the PTI responses, are closely associated with or even culminate in HR, thus imparting resistance against the invading pathogen (Jones and Dangal, 2006).

HR and necrotic responses impart resistance against diverse plant pathogenic fungi, bacteria, and viruses, and, to some extent, use similar mechanisms. During a viral infection, in a manner similar to nonviral infections, an HR response is initiated by Avr/R protein interactions that lead to metabolic changes in defense hormone levels, such as salicylic acid (SA), jasmonic acid (JA), and nitric oxide (NO), and the accumulation of reactive oxygen species (ROS), such as $O^{2-}$and hydrogen peroxide, both in the infected and noninfected tissues (Mandadi and Scholthof, 2012). At the cellular level, HR affects calcium (Ca^{2+}) ion homeostasis and alters membrane potential and permeability (Mur *et al*., 2008). For example, TMV and turnip crinkle virus (TCV) infections both induce cellulose deposition at the plasmodesmata and alter membrane permeability permitting electrolyte leakage in tobacco and Arabidopsis, respectively (Zavaliev *et al*., 2011).

Summary

Several host plant proteins participate during the viral cycle. Some of these proteins (i.e.microtubules, filaments of actin/myosin, calreticulin) facilitate the infective process and virus movement through the plant. Others, like the receptors encoded by resistance genes, interact with viral proteins in the virus recognition process. The recognition of the pathogen by the host plant induces a hypersensitivity reaction (HR) and a systemic defence. This is unfavorable for the development of the virus cycle, avoiding massive and systemic virus dissemination in the host plant. If host not able to recognized the pathogen then this is favorable condition for pathogen, pathogen easily spread infection in host plant and cause disease to plant and ultimately plant died.

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