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Resistance (R) Genes and Their Role in Host Resistance

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SUMMARY

Plants have evolved resistance genes (R genes) whose products mediate resistance to specific virus, bacteria and fungi. R protein based resistance of plants to various pathogens is a main area of interest in plant innate immunity. Various classes of effector R proteins are known and their sequences suggest roles in both effector recognition and signal transduction. The resistance can be conveyed through a number of mechanisms including gene-for-gene relationship, guard hypothesis, Pathogen Associated Molecular Pattern (PAMP) or Microbe Associated Molecular Pattern (MAMP) and degrades a toxin produced by a pathogen. Numerous resistance genes conferring resistance against a range of pathogens have been successfully used in development of transgenic crops.

INTRODUCTION

Plants are attacked by a wide spectrum of pathogens, being the targets of viruses, bacteria and fungi. Attacked plants developed numerous defense mechanisms including the chemical and physical barriers that are constitutive elements of plant cell responses locally and/or systemically. There must be some natural phenomenon of induced resistance to protect plants from disease. One of the best studied defense mechanisms is based on gene for gene resistance through which plants, harbouring specific resistance (R) genes, specifically recognise pathogens carrying matching avirulence (Avr) genes. However, the modern approach in plant sciences focuses on the evolution and role of plant protein receptors corresponding to specific pathogen effectors (Jatwa *et al*., 2017).

Resistance (R) genes

R genes are present in plant genome that convey plant disease resistance against pathogens by producing R protein. R gene products are proteins that allow recognition of specific pathogen effectors, either through direct binding or by recognition of the effector's alteration of a host protein. Avirulence (Avr) genes are genes of the pathogen that govern its specific recognition by particular plant genotypes. The pathogen Avr gene must have matched specificity with the R gene for that R gene to confer resistance, suggesting a receptor/ligand interaction for Avr and R genes.

Function of R Protein

The two main domains of plant R proteins NBS and LRR seem to be the most crucial in the pathogen recognition process and the activation of signal transduction in the response to pathogen attack.

The LRR domain

The essential structural element of the LRR domain is the tandem repeat of 20-30 amino acids. The tertiary structure of a single LRR domain is usually a horseshoe shaped super helix and each repeat forms other coils of the super helix. It is believed that LRR domains constitute a platform for protein protein interactions.

The NBS domain

This domain is characterized by NTPase activity and it is suggested to play a crucial role as a molecular switch activating signal transduction. In the signalling pathway, changes in the conformation of the NBS domain occur, caused by reversible nucleotide binding, which leads to the activation/deactivation of the whole receptor (Głowacki *et al*., 2010).

Other domains

The Toll/interleukin-1 receptor/resistance protein (TIR) domain is a protein protein interaction domain consisting of 125–200 residues. The TIR domain included in TLRs seems to be crucial for interactions with adaptor molecules mediating the initiation of the further steps of signal transduction. The coiled-coil (CC) domain is composed of two or more α-helices that are usually twisted super helically around each other in the parallel and antiparallel orientations.

Classification of R genes

Based on the structural characteristics of their protein product

There are five classes of R genes based on the structural characteristics of their protein product. The first class codes for serine/threonine kinase with the Pto gene as the only known member. It confers resistance to bacterial speck in tomato and encodes a serine/threonine protein kinase with no leucine rich repeat (LRR). The second class includes R genes coding for receptor like protein with a transmembrane domain and an extracellular LRR domain. Members of this class include *Cf* gene product mainly found in solanaceous species and specific for leaf mold resistance and *HS1* specific for nematode resistance.

The third class of R genes codes for receptor-like kinase with an extracellular LRR, a membrane spanning region and an intracellular protein kinase domain. This class is exemplified by Xa21 and Xa26 in rice. The fourth class includes the vast majority of R genes known as the nucleotide binding-site leucine rich repeat (NBS-LRR) resistance genes. The last class includes all R genes which cannot be fitted in any of the above four classes due to their different structure. An example is the powdery mildew resistance gene, RPW8 in Arabidopsis. It codes for a small protein with only an amino-terminal transmembrane domain and a coiled coil domain and it confers an unusually broad spectrum of resistance.

Based on their amino acid motif organization and their membrane spanning domains

Plant resistance genes can be broadly divided into eight groups based on their amino acid motif organization and their membrane spanning domains. First major class of R genes include the genes encoding for cytoplasm proteins with a nucleotide-binding site (NBS), a C terminal leucine rich repeat (LRR) and a putative coiled coil domain (CC) at the N- terminus. The examples of this class of resistance genes include the *P. syringae* RPS2 and RPM1 resistance genes of Arabidopsis. The second class of resistance genes consists of cytoplasmic proteins which possess LRR and NBS motifs and an N-terminal domain with homology to the mammalian toll-interleukin-1- receptor (TIR) domain. The tobacco N gene, flax L6 gene and RPP5 gene are a few examples categorized under this class.

Third major class of resistance genes family devoid of NBS motif consists of extra cytoplasmic leucine rich repeats (eLRR), attached to a transmembrane domain (TrD). The *C. fulvum* resistance genes (Cf-9, Cf-4 and Cf-2) having an extracellular LRR (eLRR), a membrane spanning domain and a short cytoplasmic C terminus are some examples of this class of resistance genes. The rice Xa21 resistance gene for *Xanthomonas* is an example of the fourth class of resistance genes which consists of an extracellular LRR domain, a transmembrane domain (TrD) and an intracellular serine threonine kinase (KIN) domain. The fifth class of resistance genes contain the putative extra cellular LRRs, along with a PEST (Pro-Glu-Ser-Thr) domain for protein degradation (found only in Ve2 and not Ve1) and short proteins motifs (ECS) that might target the protein for receptor mediated endocytos (e.g. tomato Ve1 and Ve2 genes). The Arabidopsis RPW8 protein is an example of the sixth major class of resistance genes which contains a membrane protein domain (TrD), fused to a putative coiled coil domain (CC). Whereas, the seventh major class of resistance genes includes the Arabidopsis RRS1-R gene conferring resistance to the bacterial phytopathogen *Ralstonia solanacearum* and it is a new member of the TIR NBS LRR R protein class. RRS1-R has a C-terminal extension with a putative nuclear localization signal (NLS) and a WRKY domain. The eighth major class of resistance genes includes the enzymatic R-genes which contain neither LRR nor NBS groups. For example the maize Hm1 gene which provides protection against southern corn leaf blight caused by the fungal pathogen *Cochliobolus carbonum* (Gururani *et al*., 2015).

Resistance Mechanisms

Resistance can be conveyed through a number of mechanisms including,

- The R protein interacts directly with an Avr gene (Avirulence gene) product of a pathogen (Gene-for-Gene relationship).
- The R protein guards another protein that detects degradation by an Avr gene (Guard hypothesis).
- The R protein may detect a Pathogen Associated Molecular Pattern or PAMP (alternatively called MAMP for microbe associated molecular pattern).
- The R protein encodes enzyme that degrades a toxin produced by a pathogen.

1. Gene-for-Gene relationship

The gene for gene relationship was discovered by the late Harol Henry Flor (1956) who was working with rust (*Melampsora lini*) of flax (*Linum usitatissimum*). He stated that, for each resistance gene in the host there is a corresponding avirulence in the pathogen conferring resistance and vive versa. One is a plant gene called the resistance (R) gene. The other is a parasite gene called the avirulence (Avr) gene. Plants producing a specific R gene product are resistant towards a pathogen that produces the corresponding Avr gene product. When R gene in host or Avr gene in pathogen is missing then there is no recognition and disease occurs. When matching of proteins of R gene in host or Avr gene in pathogen then disease resistance occurs.

2. Guard Hypothesis

According to this model, the target protein of the pathogen effector (guardee) is "guarded" by a suitable guard protein, namely an NBS-LRR receptor. Thus, direct detection of the pathogen effector molecules does not occur. In only some cases is there direct interaction between the R gene product and the Avr gene product. This model proposes that the R proteins interact or guard, a protein known as the guardee which is the target of the Avr protein. When it detects interference with the guardee protein, it activates resistance. E.g. Yeast two hybrid studies of the tomato Pto/Prf/AvrPto interaction showed that the avirulence protein, AvrPto, interacted directly with Pto despite Pto not having an LRR. This makes Pto the guardee protein, which is protected by the NBS-LRR protein Prf. However, Pto is a resistance gene alone, which is an argument against the guard hypothesis.

3. PAMP/MAMP Mechanism in Plants against Pathogens

These are molecules associated with groups of pathogens that are recognized by cells of the innate immune system. These molecules can be referred to as small molecular motifs conserved within a class of microbes. They are recognized by Toll-like receptors (TLRs) and other pattern recognition receptors (PRRs) in both plants and animals. A vast array of different types of molecules can serve as PAMPs, including glycans and glycoconjugates. PAMPs activate innate immune responses, protecting the host from infection, by identifying some conserved nonself molecules. These defense genes releases several gene products *viz.*, secondary metabolites (abiotic stress), pathogenesis related (PR) proteins, Phytoalexins and chitinases (biotic stress) which help in primary immune response of plants.

Elicitors like salicylic acid (SA) and Jasmonic acid (JA) activates their respective dependent pathways i.e., SA-dependent pathway and JA-dependent pathway. SA results in systemic acquired resistance (SAR) i.e. when there is a pathogen attack in one part of the plant then the remaining parts of the plant become resistant to further pathogen attack. SA activates NPR1 (Non-expresser of pathogenesis related genes) which releases PR-1, 2, 5 (pathogenesis related proteins) that causes SAR. JA results in localized acquired resistance (LAR) i.e. when there is a pathogen attack JA helps in the death of the affected tissue thus preventing further attack of the pathogen. JA activates MYC2 (mycelocytomatosis-2) a transcription factor which releases PR-3, 4 and PDF 1.2 (Protodermal factor 2) that causes LAR.

CONCLUSION

The ability of plant species to survive over evolutionary time might depend on their ability to generate useful diversity at resistance gene loci. Disease development depends upon successful host pathogen interaction. Susceptibility and resistance of a host against various pathogens is predominantly decided by resistance gene. R proteins detect and recognize pathogen effectors.

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