AGRIBLOSSOM
ISSN-2582-8258
A monthly peer reviewed e-magazine for Agriculture & allied Sciences

Protein for Protein concept: Foundation of Immunization basis in plant

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Introduction

Effector proteins encoded by the pathogen's avirulence (avr) gene are delivered directly into plant cells. These effector molecules either alter the physiology of the host plant cell to facilitate pathogen colonisation or interfere with the initiation of the host plant defence response. On the basis of a very precise interaction between the R gene products (R proteins) and the effector chemicals, hot plants have produced resistance genes (R genes) to specifically counteract the effector molecules. Several R genes that give resistance to fungus, bacteria, viruses, nematodes, and insects have been cloned and studied functionally and molecularly. Many of these genes encode R proteins, which have been the topic of extensive research. R proteins are a diverse set of proteins with various functional domains that are divided into eight major classes based on the domains they contain. The leucine-rich repeat (LRR) domain is found in the majority of R proteins (six out of eight) and is involved in pathogen effector protein specific recognition. The transmembrane domain (TrD) is the second most prevalent domain in R proteins, appearing in four of the eight classes. The eighth class of R proteins includes host enzymes that aid in disease resistance. For example, the HC toxin reductase activity of the protein Hm1 expressed by the maize gene Hm detoxifies the HC toxin (a cyclic tetrapeptide) produced by the fungal disease *Cochliobolus carbonum* (previously, *Helminthosporium maydis*), the cause of Southern corn leaf blight. The interactions between the R proteins and the effector proteins are basically of the following two types: (i) gene-for-gene interactions and (ii) host target protein (guardee)-mediated interactions.

(I) Gene-for-gene interaction

In gene-for-gene interactions, the pathogen avirulence genes' effector proteins interact directly and in a very precise manner with the host R proteins expressed by the Avr alleles. This contact triggers the

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HR response, which results in programmed cell death at the pathogen's entry point. The tomato-*Cladosporium fulvum* host-pathogen system is an excellent example of gene-for-gene interaction. The Avro peptide is encoded by the fungal pathogen's Avr gene, and it is recognised by the R protein produced by the tomato's Cf-9 gene in a highly precise manner. HR was seen when tomato plants with the C-9 gene or transgenic tobacco plants expressing this gene were treated with the AVT9 peptide. The rice-fungal pathogen *Mangnoporthe grisea* (rice blast) system is another example of a well-studied gene-for-gene relationship. This virus produces the AVT-Pita effector protein, which binds to the rice Pita R protein in a highly precise manner, causing HR response.

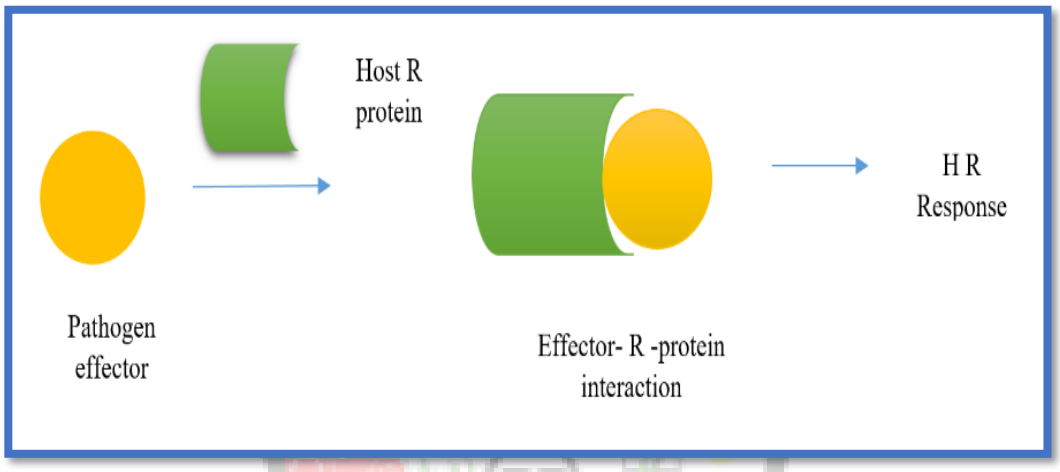

Figure 1: R proteins from the host plant recognise pathogen effectors in models. R protein receptor recognises pathogen effectors directly (gene-for-gene-model). Source: (Singh, B. 1995)

(II) Host target protein (guardee) - mediated interactions

As a biochemical explanation of gene for gene interaction, Vander Plank (1978) developed the protein for proteins hypothesis. According to the protein for protein hypothesis, in gene-for-gene diseases, the mutual recognition of the host and pathogen is mediated by their coded proteins rather than the genes themselves. The pathogen effector protein does not bind directly with the host R protein in the host target protein-mediated interaction. Instead, it interacts with the guardee protein, which is the target host protein. The contact with the effector protein either causes the host target protein to alter shape or causes it to degrade. The matching R protein (commonly referred to as the guard) detects these changes in the host target protein (the guardee), triggering the signalling cascade that leads to HR. The guard hypothesis describes the scheme for this three-component interaction (effector protein, host target

protein, and R protein). The *Arabidopsis thaliana* and the bacterial disease *Pseudomonas syringae* pv. tomato system is one example of such an interaction. Two Arabidopsis genes, RPMI and RPS2, offer resistance to this disease. The Avr protein, a bacterial effector protein, has no interaction with the RPMI and RPS2 gene products. Rather, it interacts with RIN4, another host protein (RPM1-interacting protein 4). RIN4, the guardee, undergoes a conformational shift as a result of this contact, which is recognised by the R proteins RPMI and RPS2. Resistance to P. syringae Pv. tomato is also conferred by the A. thaliana RPSS gene. The cysteine protease expressed by the gene AvrPphB is the effector protein generated by the bacterial pathogen in this scenario. This protease cleaves the PBSI kinase, which is generated by *A. thaliana* and serves as the guardian in this situation. This cleavage is detected by the R protein produced by the RPSS gene, which becomes activated, resulting in HR.The Avr protein associated with RIN4 decides which R protein will be activated by the conformational change, and this determines which R protein will be triggered by this change.

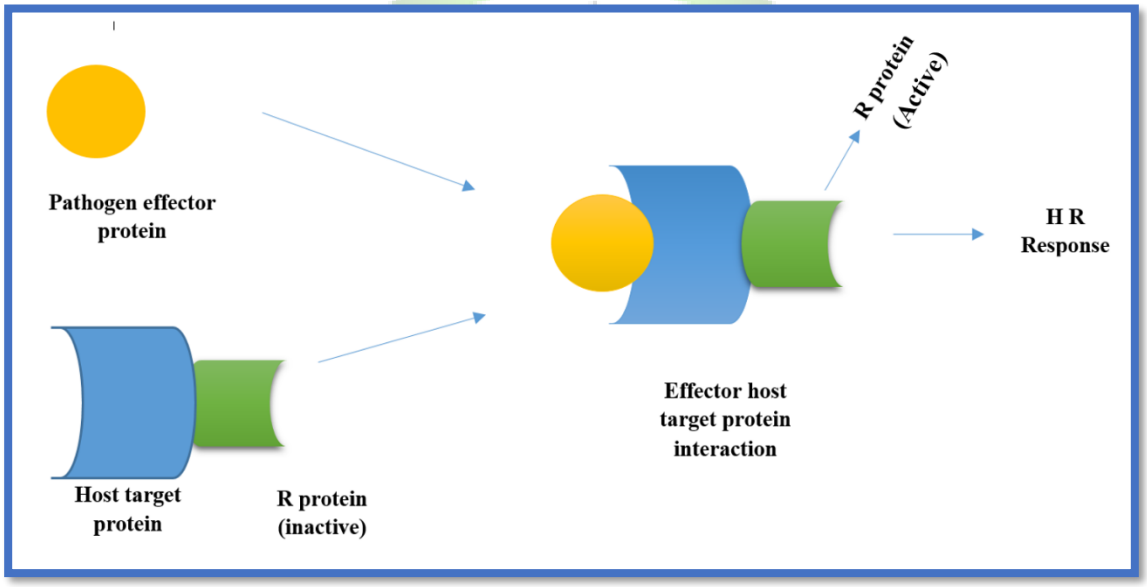

Figure 2: Model for Host target protein (guardee)-mediated interactions. The R protein is linked to a target protein encoded by the host that interacts with the pathogen effector protein. This interaction produces a conformational change in the host target protein, resulting in R protein activation and the initiation of the HR response. Source: (Singh, B. 1995)

Plants have a protection system that is similar to animals' innate defence reaction. Microbe associated molecular patterns (MAMPS) contained in the elicitor molecules produced by R proteins are used to

detect infections. Many R proteins work similarly to animal Toll-like receptors (TLRs), but the response mechanism is different. The activation of a mitogen-activated protein kinase (MAPK) cascade that creates HR is triggered when an R protein recognises a specific MAMP. In a given host, many distinct pathogens can activate the same MAPK cascade. Other genes are implicated in the control of resistance gene function, particularly in the generation of multiple pathogen resistance, in addition to the R genes. The gene Sgt l, for example, is known to be required for broad-spectrum resistance to potato late blight. Similarly, the powdery mildew pathogen *Blumeria graminis* f. sp. hordei is resistant to the gene Rar l, which is controlled by the gene Mla12. Surprisingly, the molecular chaperone Hsp90 (heat shock protein 90) was discovered to be essential for race-specific resistance to powdery mildew mediated by the gene Mla13.

In susceptibility, the pathogen excretes a protein (virulence for product) into the host cell, which copolymerizes with a complementary host protein, according to Vander Plank (1978). (Resistance gene product). This co-polymerization disrupts one auto regulation of the protein-coding host gene, causing the gene to turn on and make more protein. In resistance, the pathogen's avirulence gene specifies a protein that is ejected into the host and does not polymerize with the resistance gene's protein. The host does not recognise it at all. This created the foundations for 'Immunization.' Natural or artificial immunisation, i.e., a sub minimal natural infection with the pathogen or an artificial injection of pathogen proteins and other antigenic substances, is frequently used to activate pathogen defences in humans and animals. Both events result in the production of antibodies against the pathogen, which provide the human or animal with long-term protection (immunity) against infection by subsequent pathogen attacks. (Singh, B. 1995)

References

Singh, B. (1995). In *Plant Breeding; principal and methods* (pp. 466–468). essay, Kalyani Publishers.