

Genetic Analysis of Resistance Gene Against Plant Diseases

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Due to the severity of diseases outbreaks around the world, plant diseases can significantly reduce crop production. Therefore, one of the primary goals of any crop improvement programme has always been the management of plant diseases. Genes for plant diseases resistance can recognise and fight against a pathogen and assist in a counter attack. Numerous plant R –genes have been employed in crop improvement programme in the past with varying degrees of success, and many of them are still being used today. Recent advances in genomic, bioinformatics and molecular biology techniques make it possible to manage R-genes effectively for the treatment of plant illnesses brought on by pathogens. The recent uses and future promise on R-genes in crop diseases management are outlined in this paper. Resistance to different pathogens that is induced by the R protein is often race-specific and only effective against pathogen strains that produce the cognate effector protein (Avr protein) that the R protein recognises. Frequently, this resistance is accompanied by a hypersensitive reaction (HR), which manifests as the rapid death of the invaded cell and occasionally, a few adjacent cells. It is important to analyse the structural and functional properties of plant resistance genes and R-gene loci in order to efficiently assemble a variety of resistance sources.

The activation of signals during plant pathogen interactions, which can occasionally lead to a quick defence reaction against a variety of plant infections, is a well-known process. This reaction aids the host plant in protecting itself against the disease's spread. The production of specialised host genes known as R-genes recognises certain pathogen effectors to initiate plant defence signalling. Many distinct plant resistance (R) genes have previously been identified and are effectively exploited in crop enhancement research projects. It is practical to use plant resistance genes instead of other means, such as

pesticides or other chemical control approaches, to create disease-resistant types of plants.

Use of plant resistance genes in resistance breeding programmes has several advantages, including effective pathogen growth reduction, minimal host plant damage, no farmer input of pesticides, and most importantly, environmentally friendly nature of the process. Similar plants. However, in the case of conventional breeding for resistance, the introduction of resistance genes from one species into the gene pool of another via repeated backcrossing is a lengthy process and typically occurs after several hybrid generations. It is anticipated that thorough functional analyses, cloning, characterisation, and genetic transformation of plant resistance genes will aid researchers in quickly finding solutions to these issues.

Molecular isolation of resistance gene

Resistance caused by genes (reviewed in Gabriel and Rolfe, 1990). According to this theory, the elicitors that the Avr genes produce act as ligands for the R genes' receptors. The isolation of R genes or their gene products wasn't made any easier by this paradigm, even though it may prove to be accurate in some situations (described below). It took the advent of tools for plant gene cloning with unknown structural or molecular functions to be successful in isolating R genes. The first real chance for success came from maize transposable elements, but early attempts failed due to a high incidence of spontaneous mutations in the R genes that were being targeted, including maize Rp1 (Bennetzen et al., 1988). Nevertheless, these efforts led to the development of a body of research that is still in its early stages on the biological importance of R gene mutability in the creation of new resistance specificities (e.g., Sudapak et al., 1993; see also Crute and Pink, 1996, in this issue, and the discussion below).

Instead, transposon tagging of the R gene Hm7 from maize, which differs functionally from

traditional Avr gene-dependent R genes, led to the first R gene cloning. The fungus pathogen *Cochliobolus carbonum* Race 1 strains are resistant to Hm1 (Johal and Briggs, 1992). Hm1 was discovered to encode a NADPH-dependent reductase that inactivates the powerful plant toxin generated by these fungal strains (Johal and Briggs, 1992; Meeleyeta, 1992) in a conclusive series of investigations. Because Hm1's toxin-degrading method lacks the pathogen Avr genes, the triggering of hypersensitive plant cell death, or other markers of gene-for-gene interactions, studies of Hm1 unfortunately did not suggest a structure or function for classically described R genes. However, work on Hm1 revealed a crucial mechanistic paradigm for naturally occurring or manufactured plant disease resistance, despite the fact that toxin generation is a highly prevalent aspect of pathogen pathogenicity (see Walton, 1996, in this issue).

Numerous isolated R genes have now been successfully identified thanks to the development of positional cloning (chromosome walking) and heterologous transposon tagging technologies (this topic is also covered in Lamb, 1994; Briggs, 1995; Dangl, 1995; Michelmore, 1995; and Staskawicz et al., 1995). The cloned R genes for which published reports are available. The first Avr gene-specific R gene to be isolated was Pto of tomato, which confers resistance against *Pseudomonas syringae* pv tomato bacteria expressing the Avr gene (Martin et al., 2004). After Pto was isolated using a positional cloning technique, it was discovered that this gene encodes a protein with properties resembling those of serine-threonine protein kinases.

R-gene mediated pathogen resistance

The first step of infection involves the delivery of specific chemicals produced by phytopathogens known as "effectors"—encoded by the Avr (avirulence) genes—directly into the plant cells. In this case, the effectors in order to facilitate pathogen colonisation, host plant physiological states are altered, or host plant defences are interrupted. However, plants have since evolved a defence mechanism known as R-gene mediated pathogen

resistance that is based on how host resistance proteins perceive these proteins.

A plant with a resistance gene fights pathogen races with the appropriate effectors in gene-for-gene interactions. A host plant develops a resistance response to a plant pathogen as a result of the effectors that are present in bacteria, viruses, nematodes, fungi, oomycetes, and insects

Major classes of R protein

In general, plant resistance genes can be categorised into eight classes based on the arrangement of their amino acid motifs and membrane structure cover multiple domains. The bulk of R proteins contain the LRRs (Leucine Rich Repeats), which are components with a significant impact on recognition specificity.

Genes for cytoplasmic proteins with a nucleotide-binding site (NBS), a leucine-rich repeat (LRR), and a putative coiled coil domain (CC) at the N-terminus make up the first major class of R-genes. Examples of this type of resistance genes include the tomato *Fusarium oxysporum* resistance gene I2, the *Arabidopsis* *P. syringae* RPS2 and RPM1 resistance genes, and the *P. syringae* RPS2 and RPM1 resistance genes.

Cytoplasmic proteins with LRR and NBS motifs and an N-terminal domain that is homologous to the mammalian toll-interleukin-1 receptor (TIR) domain make up the second class of resistance genes. The cigarette Examples of this class include the N gene, flax L6 gene, and RPP5 gene. Extra cytoplasmic leucine rich repeats (eLRR), connected to a transmembrane domain (TrD), make up the third major class of resistance genes family lacking the NBS motif. Even though they are not directly engaged in pathogen identification and the activation of defence genes, eLRRs are known to play a significant function for some defence proteins, such as polygalacturonase inhibiting proteins (PGIPs). This class of resistance genes includes the *C. fulvum* resistance genes (Cf-9, Cf-4, and Cf-2) with an extracellular LRR (eLRR), a membrane spanning domain, and a short cytoplasmic C terminus. The fourth category includes the rice Xa21 *Xanthomonas* resistance gene. class of resistance

genes, which are made up of an intracellular serine-threonine kinase (KIN) domain, a transmembrane domain (TrD), and an extracellular LRR domain [252]. The putative extracellular LRRs, a PEST (Pro-Glu-Ser-Thr) protein degradation domain (found only in Ve2, not Ve1), and short proteins motifs (ECS) that may target the protein for receptor-mediated endocytosis are all components of the fifth class of resistance genes (e.g., tomato Ve1 and Ve2 genes). However, it has recently been suggested that these Ve1 and Ve2 proteins are PAMP receptors.

Challenges and future direction

ESTs, whole genome sequences, data on gene expression, and other types of experimental data have all been produced in great quantities by researchers thanks to the development of high throughput techniques and effective genomic approaches. Even so, there has been very modest advancement in our knowledge of how resistance gene's function. For instance, the structural underpinnings of pathogen identification are poorly understood. A reference set of sequences that can be utilised as a model for resistance genes, which typically cluster in genomic regions with a large number of homologs and pseudogenes, is currently insufficient. Another barrier is the challenges in conducting investigations on plant-pathogen interactions.

In addition to improving our understanding of plant defence signalling, the application of functional genomics methods for disease resistance may also provide new insights into the relationships between these signalling pathways and other plant processes. It would be foolish to anticipate a significant advance in impermeable broad-spectrum resistance, even as research into plant defence mechanisms as a whole is moving forward at a rapid rate. However, it is wise to plan for a variety of extremely helpful instruments working in conjunction with other control mechanisms to provide enough protection in specific situations.

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Conflicts of Interest

The authors declare no conflict of interest

References

- Abramovitch RB, Kim YJ, Chen S, Dickman MB, Martin GB. *Pseudomonas* type III effector AvrPtoB induces plant disease susceptibility by inhibition of host programmed cell death. *EMBO J* 2003; 22:60e9.
- Acciarri N, Rotino GL, Tamietti G, Valentino D, Voltattorni S, Sabatini E. Molecular markers for Ve1 and Ve2 *Verticillium* resistance genes from Italian tomato germplasm. *Plant Breed* 2007; 126:617e21.
- Albar L, Bangratz-Reyser M, Hebrard E, Ndjiondjop MN, Jones M, Ghesquiere A. Mutations in the eIF (iso)4G translation initiation factor confer high resistance of rice to Rice yellow mottle virus. *Plant J* 2006; 47:417e26.
- Alfano JR, Collmer A. Bacterial pathogens in plants: life up against the wall. *Plant Cell* 1996; 8:1683e98.
- Allen RL, Bittner-Eddy PD, Grenville-Briggs LJ, Meitz JC, Rehmany AP, Rose LE, et al. Host- parasite co-evolutionary conflict between *Arabidopsis* and downy mildew. *Science* 2004; 306:1957e60.
- Armstrong MR, Whisson SC, Pritchard L, Bos JIB, Venter E, Avrova AO, et al. An ancestral oomycete locus contains late blight avirulence gene *Avr3a*, encoding a protein that is recognized in the host cytoplasm. *Proc Natl Acad Sci USA* 2005;102:7766e71.
- Austin MJ, Muskett P, Kahn K, Feys BJ, Jones JD, Parker JE. Regulatory role of SGT1 in early R-gene-mediated plant defenses. *Science* 2002;295(5562): 2032e3.
- Baker CM, Chitrakar R, Obulareddy N, Panchal S, Williams P, Melotto M. Molecular battles between plant and pathogenic bacteria in the phyllosphere. *Braz J Med Biol Res* 2010;43:698e704.
- Baldauf SL, Roger AJ, Wenk-Siefert I, Doolittle WF. A kingdom-level phylogeny of eukaryotes based

- on combined protein data. *Science* 2000;290: 972e7.
- Ballvora A, Ercolano MR, Weiß J, Meksem K, Bormann C, Oberhagemann P, et al. The R1 gene for potato resistance to late blight (*Phytophthora infestans*) belongs to the leucine zipper/NBS/LRR class of plant resistance genes. *Plant J* 2002;30:361e71.
- Bar-Joseph M, Marcus R, Lee A. The continuous challenge of citrus tristeza virus control. *Annu Rev Phytopathol* 1989;27:291e316.
- Belfanti E, Silfverberg-Dilworth E, Tartarini S, Patocchi A, Barbieri M, Zhu J, et al. The HcrVf2 gene from a wild apple confers scab resistance to a transgenic cultivated variety. *Proc Natl Acad Sci USA* 2004;101:886e90.
- Belkhadir Y, Subramaniam R, Dangl JL. Plant disease resistance protein signaling: NBS- LRR proteins and their partners. *Curr Opin Plant Biol* 2004;7: 391e9.
- Bendahmane A, Kanyuka K, Rouppe van der Voort J, Van der Vossen E, Baulcombe DC. A high resolution molecular map around the Rx locus of potato: analysis of a complex locus in a tetraploid background. *Theor Appl Genet* 1999;98:679e89.
- Bendahmane A, Kohm B, Dedi C, Baulcombe D. The coat protein of potato virus X is a strain-specific elicitor of Rx-mediated virus resistance in potato. *Plant J* 1995;8:933e41.
- Bendahmane A, Querci M, Kanyuka K, Baulcombe DC. *Agrobacterium* transient expression system as a tool for disease resistance genes isolation: application to Rx2 locus in potato. *Plant J* 2000;21(1):73e81.
- Bent AF, Kunkel BN, Dahlbeck D, Brown KL, Schmidt R, Giraudat J, et al. RPS2 of *Arabidopsis thaliana*: a leucine-rich repeat class of plant disease resistance genes. *Science* 1994;265:1856e60.
- Bent AF. Plant disease resistance: function meets structure. *Plant Cell* 1996; 8:1757e71
- Büschges R, Hollricher K, Panstruga R, Simons G, Wolter M, Frijters A, et al. The barley Mlo gene: a novel control element of plant pathogen resistance. *Cell* 1997;88:695e705.
- Cai D, Kleine M, Kifle S, Harloff HJ, Sandal NN, Marcker KA, et al. Positional cloning of a gene for nematode resistance in sugar beet. *Science* 1997;275: 832e4.
- CavaliereSmith T, Chao EE. Phylogeny and megasystematics of phagotrophic heterokonts (kingdom Chromista). *J Mol Evol* 2006;62:388e420.
- CavaliereSmith T. Membrane heredity and early chloroplast evolution. *Trends Plant Sci* 2000;5:174e82.
- Champouret N, Bouwmeester K, Rietman H, van der Lee T, Maliepaard C, Heupink A, et al. *Phytophthora infestans* isolates lacking class I ipiO variants are virulent on Rpi-blb1 potato. *Mol Plant-Microbe Int* 2009;22:1535e45.
- Chen RG, Li HX, Zhang LY, Zhang JH, Xiao JH, Ye ZB. CaMi, a root-knot nematode resistance gene from hot pepper (*Capsicum annuum* L.) confers nematode resistance in tomato. *Plant Cell Rep* 2007;26:895e905.
- Chen W, Provart NJ, Glazebrook J, Katagiri F, Chang HS, Eulgem T, et al. Expression profile matrix of *Arabidopsis* transcription factor genes suggests their putative functions in response to environmental stresses. *Plant Cell* 2002;14:559e74.
- Chern MS, Fitzgerald HA, Yadav RC, Canlas PE, Dong X, Ronald PC. Evidence for a disease-resistance pathway in rice similar to the NPR1-mediated signaling pathway in *Arabidopsis*. *Plant J* 2001;27:101e13.

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