

R (Resistance)-gene mediated host
resistance

Genetic basis of plant disease resistance

- Plant basal disease resistance
- R-gene mediated pathogen resistance
 - Qualitative and quantitative disease resistance
 - Major genes for disease resistance (Qualitative)
 - confers complete resistance to a specific pathogen or pathogen race
 - R genes can be rapidly overcome by new virulent pathogens
 - Quantitative loci for disease resistance
 - Quantitative disease resistance (QDR) is controlled by multiple genes, each contributing to partial resistance QDR leads to lower selection pressure against pathogen variants.
 - Thus, quantitative disease resistance tends to be more durable than R gene-mediated resistance

Mechanisms underlying plant resistance to pathogens

- Plant pathogens are diverse: fungi, bacteria, virus
- Plant genes conferring resistance to different pathogens are also different

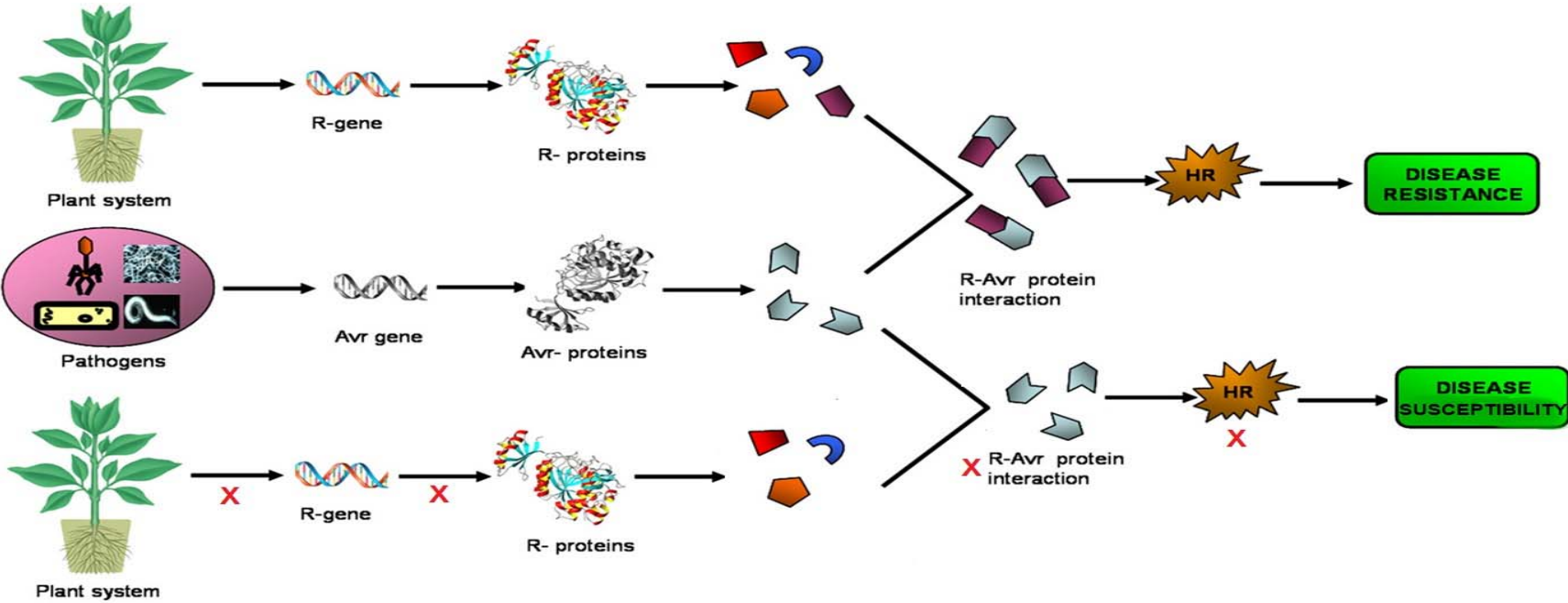
Bacterial pathogens and interacting Avr-genes and R-genes.

Pathogen	Host	Avr-gene	R-gene
<i>Xanthomonas campestris</i>	<i>Capsicum annum</i>	<i>Avr-Bs2</i>	<i>Bs2</i>
<i>Xanthomonas oryzae</i>	<i>Oryza sativa</i>	– <i>Avr-Xa1</i> <i>Avr-Xa21</i>	<i>NPR1</i> <i>Xa1</i> <i>Xa21</i>
<i>Pseudomonas syringae</i> pv <i>tomato</i>	<i>Lycopersicon esculentum</i>	<i>Avr-Pto</i> , <i>Avr-PtoB</i>	<i>Pto</i>
<i>P. syringae</i>	<i>Arabidopsis thaliana</i>	<i>AvrRpm1</i> , <i>AvrB</i> <i>AvrRpt2</i> <i>AvrPphB</i> <i>AvrRps4</i>	<i>RPM1</i> <i>RPS2</i> <i>RPS5</i> <i>RPS4</i>

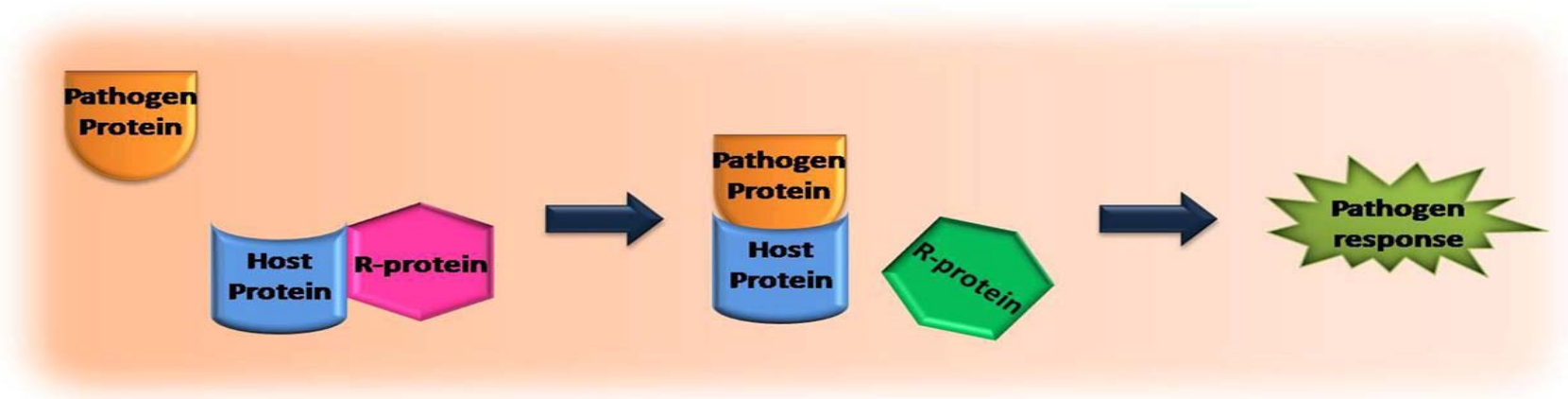
Fungal pathogens and interacting R-genes.

Pathogen	Host	Avr- gene	R-gene
<i>Blumeria graminis</i>	<i>Hordeum vulgare</i>	<i>AvrMla</i>	<i>Mla</i>
		–	<i>Mlo</i>
<i>Cochliobolus carbonum</i>	<i>Zea mays</i>	–	<i>Hm1</i>
<i>Cladosporium fulvum</i>	<i>Lycopersicum esculentum</i>	<i>Avr2</i>	<i>Cf-2</i>
		<i>Avr4</i>	<i>Cf-4</i>
		<i>Avr5</i>	<i>Cf-5</i>
		<i>Avr9</i>	<i>Cf-9d</i>
<i>Erysiphe orontii</i> , <i>E. cichoracearum</i> and <i>Oidium lycopersici</i>	<i>Arabidopsis thaliana</i>	–	<i>RPW8.1</i> , <i>RPW8.2</i>
<i>Fusarium oxysporium</i>	<i>Lycopersicum esculentum</i>	<i>Avr1</i>	<i>I2</i>
<i>Melampsora lini</i>	<i>Linum usitatissimum</i>	<i>AyrL</i>	<i>L</i>
		<i>AvrM</i>	<i>M</i>
		<i>AvrN</i> <i>AvrL567</i> genes, whose products are recognized by the <i>L5</i> , <i>L6</i> , and <i>L7</i>	<i>N</i>
<i>Magnaporthe grisea</i>	<i>Oryza sativa</i>	<i>Avr-Pita</i>	<i>Pi-ta</i>
<i>Puccinia sorghi</i>	<i>Zea mays</i>	<i>AvrRP-I-D</i>	<i>Rp1</i>
<i>Puccinia triticina</i>	<i>Triticum aestivum</i>	–	<i>Lr46</i>
<i>Puccinia graminis</i> f.sp. <i>tritici</i>	<i>Hordeum vulgare</i>	<i>Avr-Rpg1</i>	<i>Rpg1</i> , <i>Rpg4</i> , <i>Rpg5</i>
<i>Verticillium albo-atrum</i>	<i>Lycopersicum esculentum</i>	–	<i>Ve1</i> , <i>Ve2</i>
	<i>Mentha arvensis</i>		<i>mVe1</i>
	<i>Mentha longifolia</i>		
<i>Verticillium dahliae</i>	<i>Lycopersicum esculentum</i>	–	<i>Ve1</i>

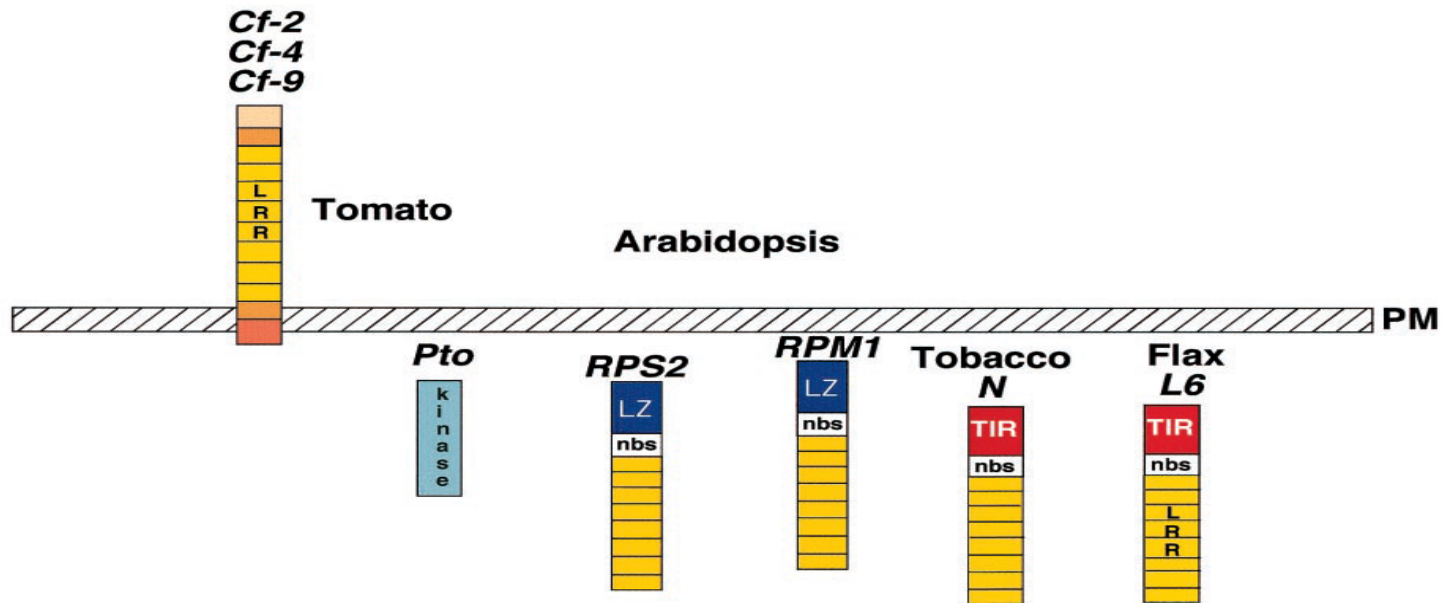
R vs. Avr gene interaction



Guard Model for disease resistance

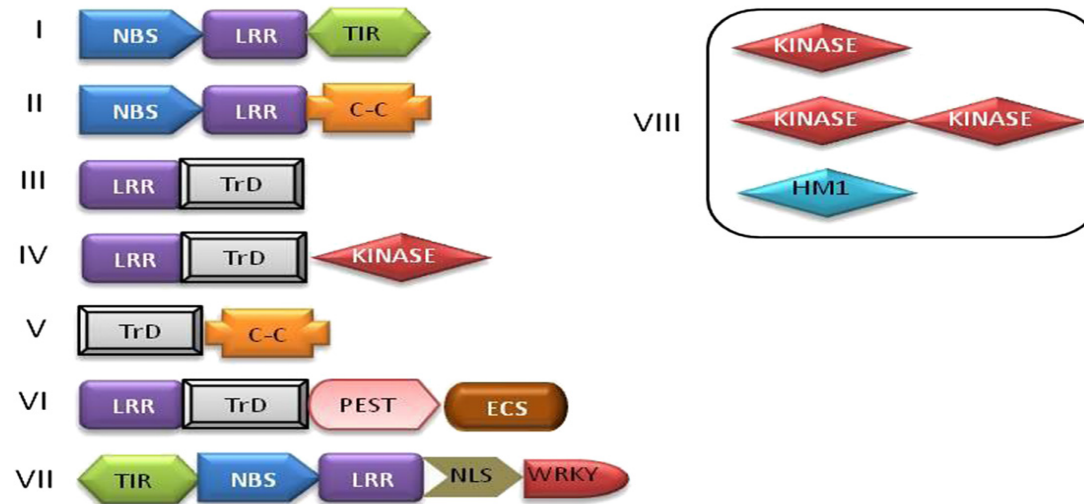


Plant Disease Resistance Proteins



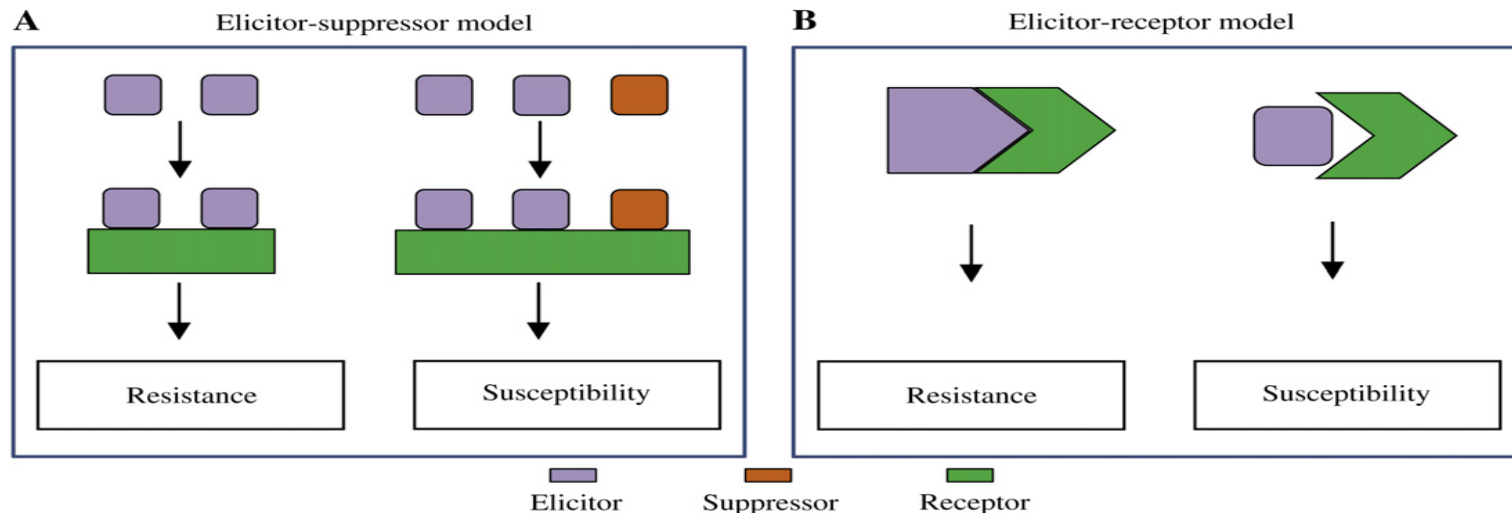
Characterization of several of the first disease resistance proteins cloned revealed the presence of common motifs.

Classes of R-genes



Major classes of plant resistance (R) genes based on the arrangement of the functional domains. LRR - Leucine rich repeats; NBS - Nucleotide-binding site; TIR/Toll/ Interleukin-1- receptors; C-C -Coiled coil; TrD - Transmembrane domain; PEST - Protein degradation domain (proline-glycine-serine-threonine); ECS - Endocytosis cell signaling domain; NLS - Nuclear localization signal; WRKY - Amino acid domain; HM1 - *Helminthosporium carbonum* toxin reductase enzyme.

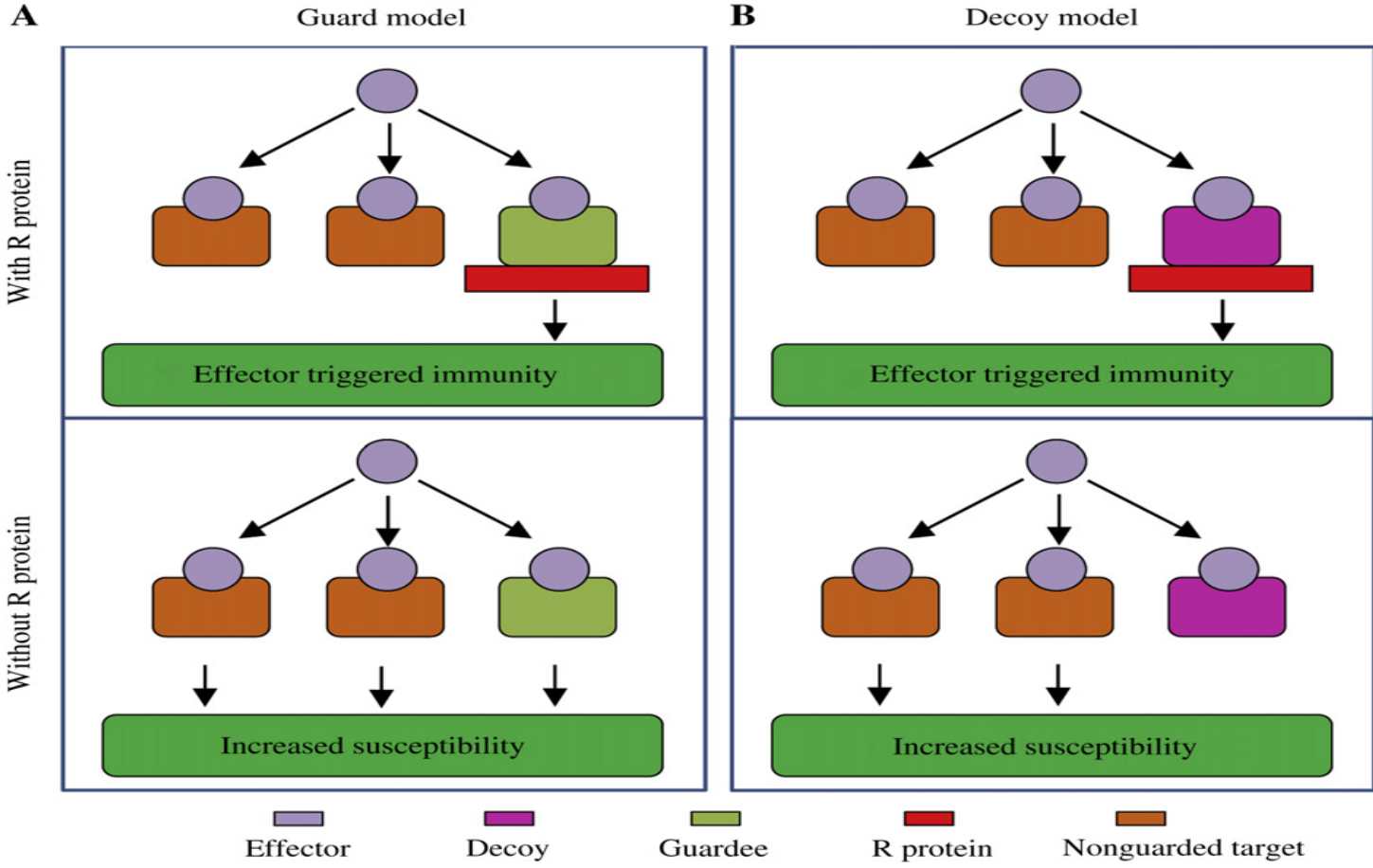
Comparison of the elicitor-suppressor model and the elicitor-receptor model



Elicitor initiates plant defense reaction (resistance) until appearance of a specific suppressor in a particular pathogen race, which leads to failure of defense reaction (susceptibility).

Protein encoded by avirulence gene is recognized by a specific plant receptors, which then triggers the resistance response. If the receptor does not fit the avirulence protein, this would inevitably lead to susceptibility.

Comparison of the guard model and the decoy model



Summary - Molecular basis of plant resistance to pathogens

