

Emerging Concepts in Effector Biology of Plant-Associated Organisms

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What is an “effector”?

- ☞ **Effector:** pathogen molecule that manipulates host cell structure
 - ☞ facilitate infection and/or
 - ☞ trigger defense responses

Kamoun Annu Rev Phytopath 2006

- ☞ Richard Dawkins, 1999. “The extended phenotype: The long reach of the gene.”
 - ☞ extended phenotype: “genes whose effects reach beyond the cells in which they reside”

“Avirulence gene”: the old name for “effector”

☞ H. H. Flor “Gene-For-Gene” model (1940s)

	pathogen		
		Avr	no Avr
R		HR	disease
no R		disease	disease

http://www.plantpath.com/edu/labs/Colmer_AResearch.html

Adoption of term "Effector"

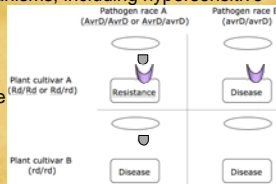
- ∞ pathogen molecule that manipulates host cell structure
- ∞ facilitate infection and/or
- ∞ trigger defense responses
- ∞ "avirulence", "elicitor", "toxin", "virulence":
 - ∞ positive or negative outcome in host/pathogen interaction
 - ∞ "effector" is a neutral term



R/Avr genes: direct recognition or indirect perception?

- ∞ **Direct recognition:** R genes products are receptors for ligands encoded by Avr genes
- ∞ Ligand (Avr) binding to R receptor triggers a signaling cascade leading to inactivation of defense mechanisms, including hypersensitive response (HR)

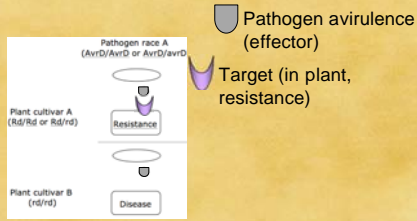
- ∞ Pathogen avirulence
- ∞ Plant resistance gene = effector target



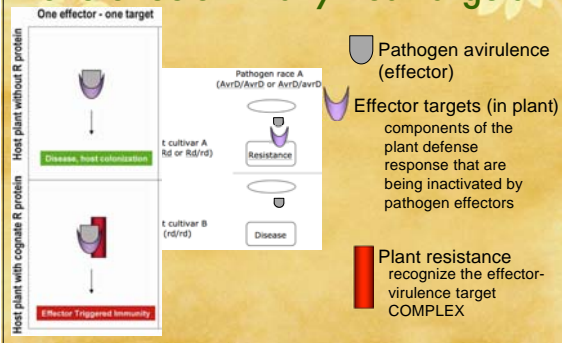
R/Avr genes: direct recognition or indirect perception?

- ∞ **Direct recognition:** R genes products are receptors for ligands encoded by Avr genes
- ∞ Ligand (Avr) binding to R receptor triggers a signaling cascade leading to inactivation of defense mechanisms, including hypersensitive response (HR)
- ∞ **Indirect perception:** "guard model"
 - ∞ no direct physical interactions between R and Avr proteins
 - ∞ detect the biochemical perturbations by Avr
 - ∞ Avr and avr both cause perturbations, which trigger plant response or not -- "effector"

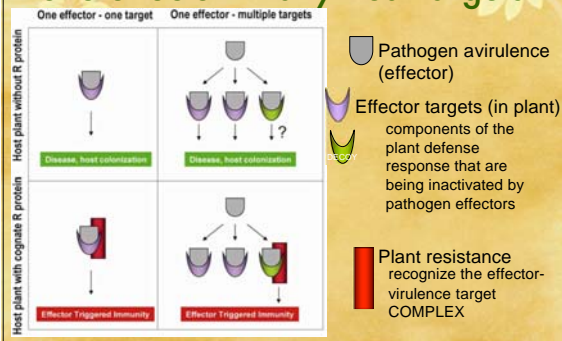
Indirect perception ramifications: one effector—many host targets



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Indirect perception ramifications: one effector—many host targets



Sites of action

- ☞ Delivered into host cells

- ☞ Apoplast

Pathogen structures
Plant structures
Apoplastic effectors
and effector targets
Cytoplasmic effectors

Kamoun, S. 2006. A catalogue of the effector secretome of plant pathogenic oomycetes. Annual Review of Phytopathology, 44: 41-60.

Sites of action

- ☞ Delivered into host cells
 - ☞ bacteria: T3SS
 - ☞ fungi & oomycetes: haustoria
 - ☞ nematodes: stylet

- ☞ Apoplast
 - ☞ cysteine-rich
 - ☞ inhibit plant hydrolases and proteases

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Many effectors suppress plant immunity

- ☞ Effectors target key plant processes to plant immunity
 - ☞ Protein turnover
 - ☞ RNA homeostasis
 - ☞ Phosphorylation pathways
- ☞ One scenario:
 - ☞ Pathogen-associated molecular patterns (PAMPs) have conserved epitopes
 - ☞ The plant recognizes these PAMPs and triggers basal defense system
 - ☞ The pathogen suppresses basal plant defense

Many effectors suppress plant immunity

Plant molecule **Pathogen molecule**

A **JA-Ile** **Coronatine**

jasmonoyl-isoleucine (JA-Ile) regulates plant defense responses

B **AtPub14** **AvrPtoB**

AtPub14 regulates protein degradation in plants

AvrPtoB effector has anti-programmed cell death activity and mimics E3 ubiquitin ligases

Many effectors suppress plant immunity

unknown transcription factor

C

TFx **AvrBs3**

upa21 *upa20* *upa22* *Bs3*

cell hypertrophy hypersensitive response

promoter with upa-box

Compatible interaction (disease):

- AvrBs3 induces expression of *upa20* and other *upa* genes -> hypertrophy

Incompatible interaction:

- AvrBs3 also binds the promoter of the resistance gene *Bs3* -> hypersensitive response

Some effectors alter plant behavior and development

A *P. syringae*

flagellin CORONATINE entry

Reopening of stomata

B *X. citri*

pthA

Hypertrophy, necrosis, canker formation

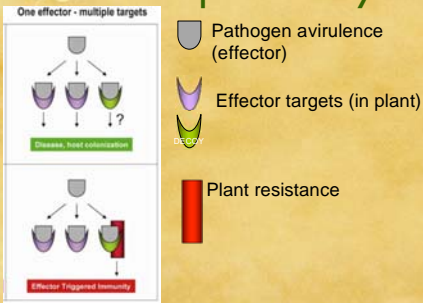
C

G. fujikuroi Gibberellin seedling growth

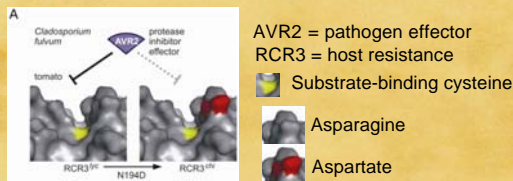
Rice Rice + foolish seedling

G. fujikuroi spores

Effector targets can evolve to evade manipulation by effectors



Effector targets can evolve to evade manipulation by effectors



Decreased interaction between effector and host protein
Effector cannot suppress cysteine protease RCR3
RCR3 is free to degrade pathogen proteins
Property may be used for *durable* disease resistance breeding

Conclusions

- ☞ Pathogen effectors may
 - ☞ directly interact with plant resistance genes
 - OR
 - ☞ cause perturbations sensed by plant resistance genes
- ☞ Effectors may act *inside* or *outside* the cell
- ☞ Pathogens mimic or suppress plant components to be successful
