Gene-for-gene hypothesis

PI Path 604
“For each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite”

For each resistance gene in the host there is a corresponding gene for avirulence in the pathogen conferring resistance and vice versa.

(Flor, 1942. *Phytopathology*)
Flor (1946,47) showed correlation between inheritance of pathogenicity and resistance to linseed rust caused by *Melampsora lini* which is now commonly known as gene-for-gene hypothesis. That “for each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite.

The concept has been applied with varying degree of proof to other host pathogen combinations including viruses, bacteria, fungi, nematodes, insects and a flowering plant (*Orobanche*).
Gene for Gene Concept

<table>
<thead>
<tr>
<th>Pathogen genotype</th>
<th>Host genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R1</td>
</tr>
<tr>
<td><strong>Avr1</strong></td>
<td>-</td>
</tr>
<tr>
<td><strong>avr1</strong></td>
<td>+</td>
</tr>
</tbody>
</table>

- = Incompatible reaction
+ = Compatible reaction

“for each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite”
### Interaction Between Two ‘R’ and ‘AVR’ Genes

<table>
<thead>
<tr>
<th>Pathogen/Host</th>
<th>R1R2</th>
<th>R1r2</th>
<th>r1R2</th>
<th>r1r2</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1A2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>A1a2</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>a1A2</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>a1a2</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

(Source: Agrios, 2006. *Plant Pathology*)
Biotrophy and gene –for –gene systems

All the parasites in which gene for gene relationship has been proved are essentially biotrophic or biotrophs at least for some time after start of infection.

*(Xanthomonas campestris pv. malvacearum, Phytophthora infestans, Venturia inaequalis* *(Vander Plank, 1978).*

The genes-for-gene systems thus involve biotrophy.

But the converse is not necessarily true. For example, *Plamodiophora brassicae*, the cause of club root of crucifers, is biotrophic but no evidence has yet been presented in the literature to suggest that host-pathogen interaction in them is based on a gene-for–gene systems.
**HOW CAN WE EXPLAIN THIS BIOCHEMICALLY?**

**PATHOGEN** (Has general pathogenicity genes and specific avirulence \(A_1\) or virulence \(a_1\) gene)

Pathogen produces

\[ \text{avr}A_1 \text{ gene product (elicitor)} \]

**Host** (Has general resistance genes and specific resistance \(R_1\) or lack of resistance \(r_1\) genes)

R1 gene-coded host receptor recognizes pathogen elicitor molecules and triggers defense reactions.

**Host resistant**

**Basic interactions of pathogen avirulence (A)/virulence (a) genes with host resistance (R)/susceptibility (r) genes in a gene-for-gene relationship, and the final outcomes of the interactions.**
There are two different schools of thought pertaining to biochemical basis of gene-for-gene interactions.

- According to first specificity in gene-for-gene systems lies in susceptibility (Van der Plank, 1978)
- whereas to other specificity lies in resistance (Ellingboe, 1981).
According to Van der Plank (1978), specificity in gene–for-gene relationships lies in susceptibility.

- He explains it with the help of interactions of five host and five pathogens attacking them specifically.
- Suppose there are five host varieties with five different R genes; R₁, R₂, R₃---------R₅. A plant with resistance gene R₁ is attacked by a pathogen having virulence gene v₁ and not to pathogen without this particular resistance gene irrespective of how many the virulence genes it may have.
<table>
<thead>
<tr>
<th>Pathogen</th>
<th>(R_1 R_1)</th>
<th>(R_2 R_2)</th>
<th>(R_3 R_3)</th>
<th>(R_4 R_4)</th>
<th>(R_5 R_5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(v_1 v_1)</td>
<td>S</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td>(v_2 v_2)</td>
<td>R</td>
<td>S</td>
<td>R</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td>(v_3 v_3)</td>
<td>R</td>
<td>R</td>
<td>S</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td>(v_4 v_4)</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>S</td>
<td>R</td>
</tr>
<tr>
<td>(v_5 v_5)</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>S</td>
</tr>
</tbody>
</table>

a. Plant reaction when resistance gene \(R_1, R_2, R_3, R_4, R_5\) at five loci interact with virulence genes \(v_1, v_2, v_3, v_4, v_5\) at five loci in the pathogen

b. Resistance is assumed to be dominant and RR can be replaced by Rr. Virulence is assumed to be recessive. However, recessive resistance and dominant virulence are also known.

R= resistant  S= susceptible
Vander Plank (1978) elaborated the protein for proteins hypothesis as a biochemical explanation of gene for gene interaction.

- The protein for protein hypothesis states that in gene-for-gene diseases the mutual recognition of host and pathogen is not by the genes themselves but by their coded proteins.
Vander Plank (1978) hypothesized that in susceptibility the pathogen excretes a protein (virulence for product) into the host cell which copolymerizes with a complementary host protein (resistance gene product). This co-polymerization interferes with one auto regulation of the host gene that codes for the protein and by so doing turns the gene on to produce more protein.

In resistance, the protein specified by the gene for avirulence in the pathogen and excreted into the host does not polymerize with the protein coded for by the gene for resistance. It is not recognized by the host at all.
According to 2nd school of thought, comprising of Ellingboe (1981), Keen (1992), Day (1984) and Gabriel (1990), the biochemical explanation of gene for gene systems is based on the fact that specificity lies in resistance and not in susceptibility as proposed by Vander Plank (1978).
Flor’s gene –for- gene hypothesis is purely a hypothesis of identities.

The resistance gene in the host and the corresponding virulence gene can be identified by this hypothesis.

But it does not tell us about the gene quality. A second gene –for -gene hypothesis, which is an extension of Flor’s hypothesis, tells us about the quality of genes.
The quality of resistance gene in the host determines the fitness of matching gene in the pathogen to survive, when this gene for virulence is unnecessary.

**Unnecessary gene means** - a gene for virulence in the pathogen population against which matching resistance gene in the host is not present.

Reciprocally, the fitness of the virulence gene in the parasite to survive when it is unnecessary determines the quality of matching resistance gene in the host.
For instance, there are ten or more genes in the host for resistance to late blight of potato, $R_1$, $R_2$, $R_3$---------$R_{10}$.

- Of these, the first four $R_1$---$R_4$ have been well studied. These genes have not been found of equal importance and strength.
- From the reports available in the literature, $R_4$ has not been successfully used on its own by breeders although it has occasionally been used in combination with other genes.
- The $R_1$ gene has often been used alone and it has given protection to the varieties against blight. The difference between these $R$ genes is that virulences on $R_4$ preexisted in population of *Phytophthora infestans* whereas virulences on $R_1$ don’t (Van der Plank, 1975).
- The ratio for virulence between $R_1$ and $R_4$ genes has been found to differ significantly. Thus there is difference in the quality of resistance genes $R_1$ and $R_4$. 
From a practical point of view, gene-for-gene relationship can be used to study the following:

- The source of pathogenic variability in pathogens
- The mutability of resistance and virulence genes
- Why host resistance is expressed under one set of conditions and not others
- Prediction of putative genotypes
- Race nomenclature
- Genetic dissection of complex loci
- Cataloguing and storing of R genes in the form of plant seeds or cuttings and V genes in the form of pathogen strains
- Management and deployment of resistance genes in space and time
- Detection of linkage and allelic relationship
- Geographic distribution of R and V genes
- Synthesis of multilines and multigene cultivars.
HISTORICAL OVERVIEW

Resistance in Mendelian fashion (Biffen, 1905)

Pathogenicity is inherited in Mendelian fashion (Newton, 1929)

Correlation between inheritance of pathogenicity \((Melampsora lini)\) and resistance (Linseed) to (Flor, 1942, 1947, 1971) GENE FOR GENE HYPOTHESIS

Surface Carbohydrate elicitor - receptor model (Albersheim and Anderson Prouty, 1975)

Modified as elicitor - receptor model (Keen and Bruegger, 1977)

Protein - Protein interaction (Vanderplank, 1978)

Genetic and physiological evidences elicitor-receptor models (N T Keen, 1982)

Dimer Model (Ellingboe, 1982)

Ion channel defense model (Gabreil, 1984)
First Avr gene cloned from *Pseudomonas syringae* (Staskawicz *et al.*, 1990)

First R gene (Hm1) was cloned (Johal and Briggs, 1992)

R gene (PTO) cloned (Martin, G.B. *et al.*, 1993)


Guard hypothesis (Van-der –biezen and Jones, 1998)

R proteins are dynamic and subject to intra-molecular interactions (Moffet *et al.*, 2002)

Several host proteins as pathogen virulence targets were discovered (Mackney *et al.*, 2003, Axtel *et al.*, 2003, Rooney *et al.*, 2005)

The soft wired model to explain the interaction of NBS-LRR domains (Bekhaldir *et al.*, 2004)