RAMA UNIVERSITY

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FACULTY OF AGRICULTURE SCIENCES AND ALLIED INDUSTRIES

(Breeding for Biotic and Abiotic stress Resistance)

For

M.Sc. Ag (GPB)

Course Instructor

Dr Shiv Prakash Shrivastav

FASAI(Genetics and Plant Breeding)

Rama University, Kanpur

GENE FOR GENE HYPOTHESIS

For each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite"

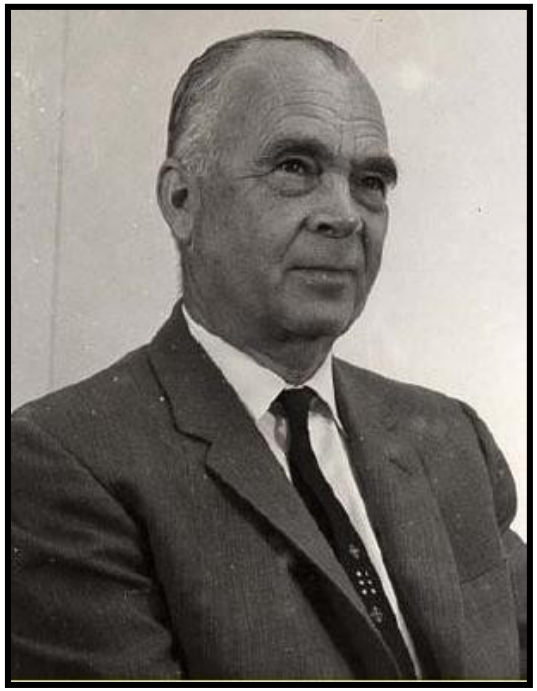
"

states that during their evolution host and parasite developed complementary genic systems

- **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*).



"for each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite"

Pathogen genotype	Host genotype	
	R1	r1
Avr1	-	+
avr1	+	+
-		

= Incompatible reaction

\+ = Compatible reaction

INTERACTION BETWEEN TWO 'R' AND 'AVR' GENES

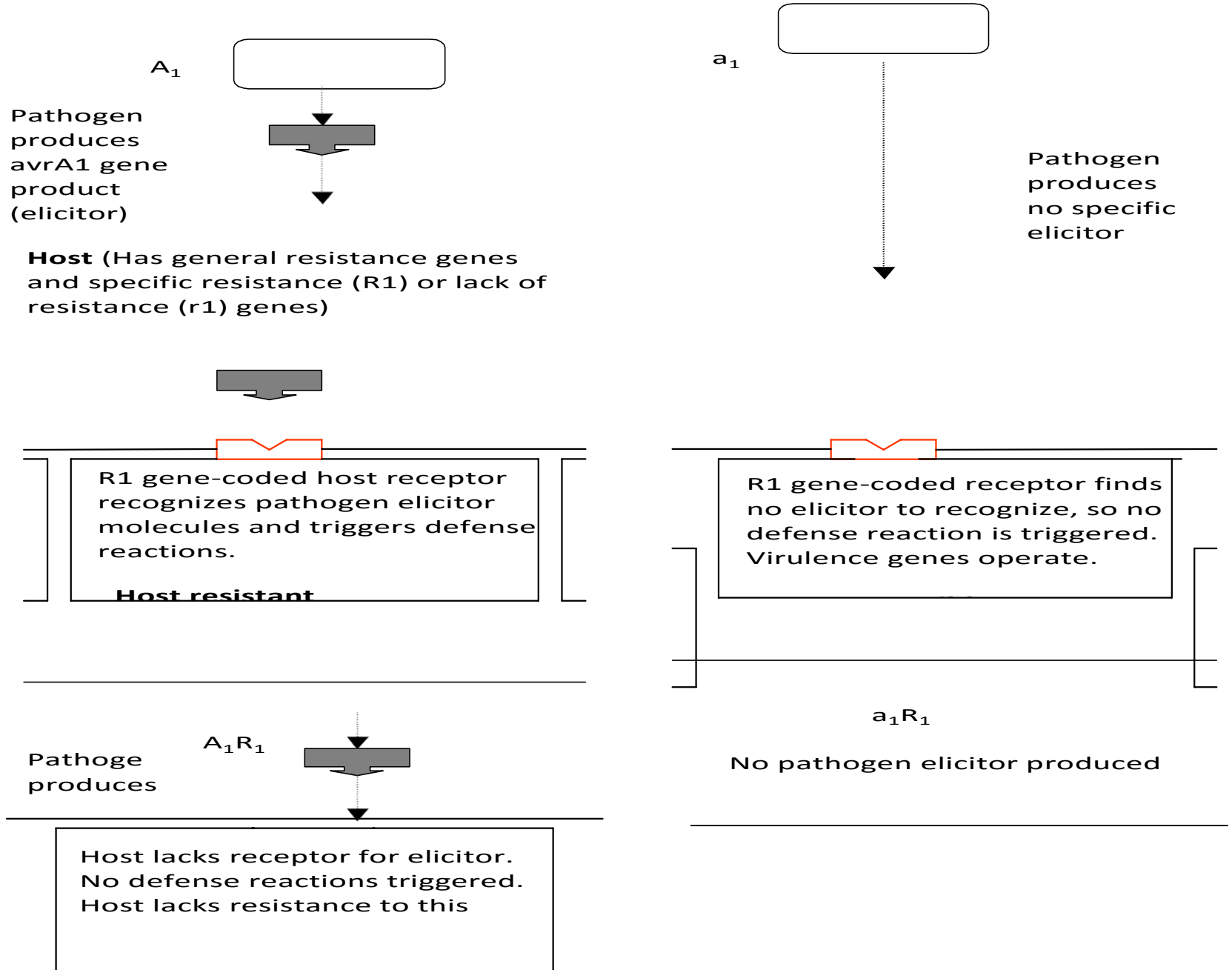
PATHOGEN/HOST	R1R2	R1r2	r1R2	r1r2
A1A2	-	-	-	+
A1a2	-	-	+	+
a1A2	-	+	-	+

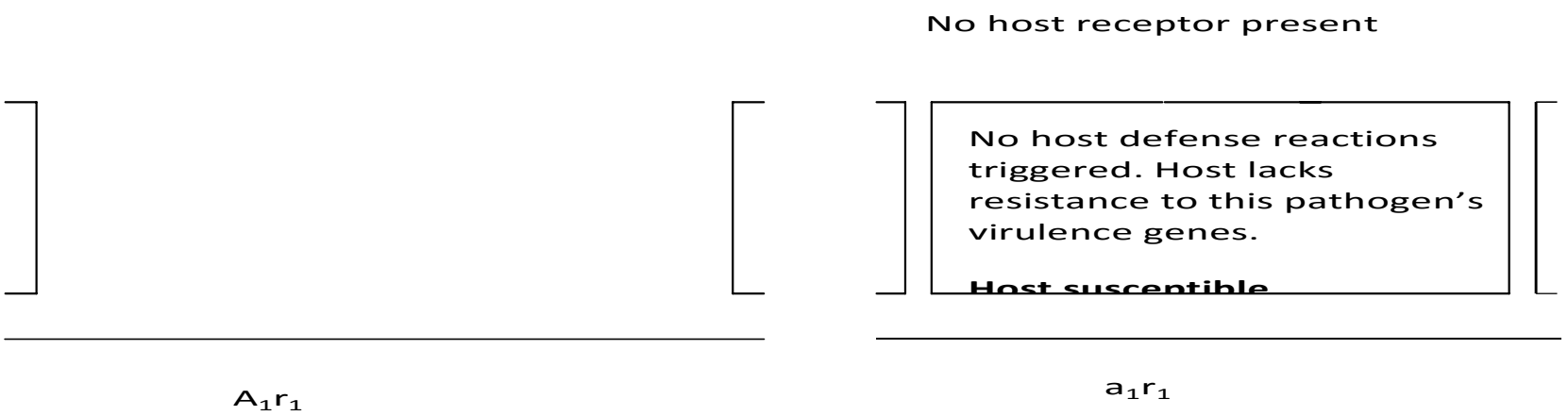
Biotrophy and gene –for –gene systems

- 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)





Basic interactions of pathogen avirulence (A)/ virulenc (a) genes with host resistance (R)/ suscptibilty (r) genes in a gene-for-gene relationship, and the final outcomes of the interactions.

Biochemical basis of Gene for Gene Hypothesis

- There are two different schools of thought pertaining to biochemical basis of gene-for -gene interactions.
  - *According to first specificity in gene- for- genesystemsliesinsusceptibility(Vander Plank,1978)*
  - whereas to other specificity lies in resistance (Ellingboe, 1981).

Biochemical basis of Gene for Gene Hypothesis

- 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. The diagonal check for specificity in a gene-for gene relationship [a]

Pathogen	Plant				
	R_1R_1 [b]	R_2R_2	R_3R_3	R_4R_4	R_5R_5
v_1v_1	S	R	R	R	R
v_2v_2	R	S	R	R	R
v_3v_3	R	R	S	R	R
v_4v_4	R	R	R	S	R
v_5v_5	R	R	R	R	S

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

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 copolymenizes 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.

- **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 ahypothesis 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, R1, R2, R3 ---------- R10.
- Of these, the first four R1---R4 have been well studied. These genes have not been found of equal importance and strength.
  - From the reports available in the literature, R4 has not been successfully used on its own by breeders although it has occasionally been used in combination with other genes.
  - The R1 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 R4 preexisted in population of *Phytophthora infestans* whereas virulences on R1 don't (Van der Plank, 1975).
  - The ratio for virulence between R1 and R4 genes has been

found to differ significantly. Thus there is difference in the quality of resistance genes R1 and R4.

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 resistanceis 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 tome
- 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)

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

Pathogenicity is inherited in Mendelian fashion (Newton,1929)

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

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

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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)

AvrPTO - PTO physically interact (Tang *et. al,* 1996 Scofield

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)