# BREEDING FOR BIOTIC STRESS RESISTANCE
# DISEASE RESISTANCE

**Stress:** Constraining influence, force, pressure or adverse conditions for crop growth caused by biological or environmental factors.

**Biotic (living) :** Adverse effects due to pests and diseases abiotic stresses

**Abiotic (non living) :** Adverse effects on host due to environmental factors

eg: Drought, water logging, heat, cold, salinity, alkalinity and air pollution etc.

**Host** : Plant effected by a disease or which can accommodate pathogen.

**Pathogen** : An organism that produces the disease

**Disease** : an abnormal conditions in the plant caused by an organism (pathogen)

**Pathogenicity** : The ability of a pathogen to infect a host strain

**Virulence :** Capacity of a pathogen to incite a disease

**Avirulence** : The inability of a pathogen to cause or incite a disease

**Physiological race** : Strains of a single pathogen species with identical or similar morphology but differ in pathogenic capabilities.

**Pathotype** : Strains of a pathogen classified on the basis of their virulence to known resistance genes present in the host.

**Epidemic** : Severe and sudden out break of disease beginning from a low level of infection.

**Variability in fungal pathogens:**

a) **Hybridization:** Recombination of genes of the two parental nuclei takes place in the zygote, and the haploid nuclei or gametes resulting after meiosis are different both from gametes that produced the zygote and from each other.

Thus every diploid pathogen individual is genetically different from any other pathogen even within the same species and variability of the new individual pathogens is continued indefinitely.

*e.g., Phytophthora infestans.*

**Heterokaryosis:** Condition in which fungal hyphae that are genetically different come together in the same cell to form heterokaryons.

**Parasexualism** : Parasexuality – re-assortment of genetic material both in haploid and diploid condition, ready for natural and artificial selection.

Mixtures of races grown together on a susceptible host combine genetically to produce new races e.g. *phytophthora infestans*

**Mutation:** The rate at which new variants of a pathogen are produced will depend on the mutation rate of the genes at a particular locus. The mutation rate varies from gene to gene and from pathogen to pathogen.

e.g. *Melampsora lini* – new race produced with UV rays (Flor 1956)

**Cytoplasmic adaptation:** There are several examples of cytoplasmic inheritance of important characteristics such as growth rate and virulence (Jinks 1966).

Virulence of *P.graminis f. sp. Avenae*, carrying gene E, is maternally inherited and may be controlled by single plasma gene (Johnson *et al* 1967)

**MECHANISMS OF DISEASE RESISTANCE:**

There are different ways of disease resistance *viz.,* disease escape, disease endurance or tolerance disease resistance and immunity

**Disease escape** : The ability of susceptible host plants to avoid attack of disease due to environmental conditions factors, early varieties, charge in the date of plating, change in the site of planting; balanced application of NPK etc.

Eg. Early varietie s of groundnut and potato may escape 'Tikka' and 'Late blight' diseases respectively since they mature before the disease epidemic occurs.

Changing planting season in sugarcane from June to October has successfully escaped leaf-rust.

Virus free seed potato is produced by sowing the crop in October in Jullundher and other places instead of November, the normal planting time.

**Disease endurance or tolerance** : The ability of the plants to tolerate the invasion of the pathogen without showing much damage. This endurance is brought about by the influence of external characters. Generally, tolerance is difficult to measure since it is confounded with partial resistance and disease escape. To estimate tolerance the loss in yield and some other trait of several host varieties having the same amount of disease eg., leaf area covered by disease etc., is compared.

Eg. In Barley the variety Proctor shows 13% yield loss as compared to 20% loss in the varieties Zephy and Sultan.

Wheat varieties when fertilized with potash and phosphorus are more tolerant to the rust and mildew infection.

The Rice crop fertilized with silicate is resistant to blast infection in Japan.

**Disease Resistance** : The ability of plants to withstand, oppose or overcome the attack of pathogens. Resistance is a relative term and it generally refers to any retardation in the development of the attacking pathogen. In case of resistance, disease symptoms to develop and the rate of reproduction is never zero i.e., r? o but it is sufficiently lower than 1 (the rate of reproduction on the susceptible variety) to be useful. The inhibition of growth of the pathogen is believed to be nutrional in nature and in some cases chemical growth inhibitors may be involved.

Resista nce is largely controlled by inherited characters i) may be controlled by single dominant gene in Ottawa 770 B, Newland flax variety, wheat all rusts NP 809

**Immunity**: When the host does not show the symptoms of disease it is known as immune reaction. Immunity may result from prevention of the pathogen to reach the appropriate parts of the host e.g. exclusion of spores of ovary infecting fungi by closed flowering habit of wheat and barley. It is more generally produced by hypersensitive reaction of the host usually immediately after the infection was occurred. In immune reaction the rate of reproduction in zero i.e. r = 0

**Hypersensitivity:** Immediately after the infection several host cells surrounding the point of infection are so sensitive that they will die. This leads to the death of the pathogen because the rust mycelium cannot grow through the dead cells. This super sensitivity (hypersensitivity) behaves as a resistant response for all practical purposes. Phytoalexins are specific polyphenolic or terpenoid chemicals and are produced by the host in response to the infe ction by a pathogen. More than 30 different phytoalexins have been identified. Phytoalexins are either fungicidal or fungistatic. Eg. Rust fungi and virus attack.

**Factors for disease resistance (Causes of Disease resistance)**

The disease resistance may be caused due to

Morphological, structural and functional characteristics which prevents the entrance of the pathogen *i.e.* prevents the first stage of infection.

Biochemical or anatomic al properties of tissue which prevent the establishment of parasitic relationship.

**a . Morphological characters**

Certain morphological features of the host may prevent infection.

Eg. Resistance to Jassid attack in cotton has been shown to be correlated with the hariness of varieties : hairy type resists the attack more, than glabrous types.

Failure to germinate rust spores on the leaves of the barley due to waxy coating.
Young sugarbeet leaves practically immune to attack of the circos pora because the stomata size is very small.

**b. Physiological characters**

**Protoplasmic factors or chemical interactions :**
By virtues of its chemical composition the protoplasm may exert an inhibitory influence on the pathogen bringing about the desired resistance in the plant.
Eg. : Resistance of grape to powdery mildew is highly correlated with the acidity of cell sap. Presence of toxic substance in the red pigment in the coloured onions. The outer scales resist the smudge fungus attack when the scales are removed they become susceptible.

**c. Anatomical:** More secondary thickening of the cell walls of resistant potato varieties which resists the mechanical puncture of the invading *Pythium* pathogen.

**Nutritional factors :** Reduction in growth and in spore production is generally supposed to be due to unfavourable physiological conditions within the host. Most likely a resistant host does not fulfill the nutritional requirements of the pathogen and thereby limits its growth and reproduction.

**e. Environmental factors :** In addition to the above the environmental factors have marked effect on the pathogen attack. Temperature, moisture, humidity and soil $P^H$ and fertility status of the soil effects the pathogen reaction greatly.

**Genetic basis of disease resistance**

The first study on genetics of disease resistance was done by Biffen in 1905. He reported the inheritance of resistance to leaf rust of wheat variety Rivet in crosses with some susceptible varieties. In $F_2$ there were 3 susceptible : 1 resistant plants indicating that resistance was controlled by a single recessive gene. Most of the earlier studies were conducted without taking into consideration the physiological specialization (pathotype differentiation) of the pathogen which can materially influence the conclusions drawn. It is now recognized that disease resistance may be inherited in three different ways :

Oligogenic

Polygenic and

Cytoplasmic inheritance

**Oligogenic inheritance:**

The disease resistance is governed by one or few major genes and resistance is generally dominant to the susceptible reaction. The action of major resistance genes may be altered by modifying genes in many cases. Eg. bunt resistance in Wheat. Oligogenes generally produce immune reaction. The chief characteristic of the oligogenic disease resistance is pathotype - specificity, i.e. resistant gene is effective against some pathogens, while it is ineffective against the others. In most cases, there are a number of major genes that determines resistance to a particular disease Eg. more than 20 different resistance genes are known for leaf rust of wheat, while those for stem rust resistance exceed 30. The genetics of oligoganic resistance has advanced by two events *viz.,*

Discovery of a resistance gene to the prevalent pathotype and

Evolution of a pathotype virulent to the new resistance

gene. Oligogenic resistance is synonymous to vertical resistance.

## Gene for gene hypothesis:

The concept of gene for hypothesis was first developed by Flor in 1956 based on his studies of host pathogen interaction in flax rust caused by *Malampsora lini.* The gene for gene hypothesis states that for each gene controlling resistance in the host, there is a corresponding gene controlling pathogenicity in the pathogen. The resistance of host is governed by dominant genes and virulence of pathogen by recessive genes. The genotype of host and pathogen determine the disease reaction. When genes in host and pathogen match for all the loci, then only the host will show susceptible reaction. If some gene loci remain unmatched, the host will show resistant reaction. Now gene-for -gene relationship has been reported in several other crops like potato, *Sorghum,* wheat etc. The gene for gene hypothesis is known as "Flor Hypothesis".

**A simple scheme to explain gene for gene relationship hypothesis (Fehr, 1987)**

| *Varieties* | *Host genotype* | *Pathogen genotypes* | *Disease Reaction* |
|---|---|---|---|
| 1. One gene pair | AA | aa | Susceptible |
| | Aa | | |
| | BB | bb | Susceptible |
| | Bb | | |
| 2. Two gene pair | AA CC | aa | Resistant |
| | Aa CC | cc | Resistant |
| | Aa Cc | aacc | Susceptible |
| 3. Three gene pair | AA BB CC | aa bb | Resistant |
| | AA BB CC | aa cc | Resistant |
| | Aa Bb Cc | aabbcc | susceptible |

Note: Dominant genes in the host are responsible for resistance and recessive genes in the pathogen for virulence.

**vertifolia Effect** : Vander plank introduced the term vertifolia effect and refers to epidemic development in a variety carrying vertical resistance genes (oligogenes) leading to heavy economic losses. Total failure of vertical resistance leading to a disease epidemic is known as vertioalia effect. This failure occurs because of two reasons :

The level of horizontal resistance in varieties carrying oligogenes is usually low and The pathogen is able to evolve new virulent pathotypes

**Polygenic inheritance**

In this type the disease resistance is governed by many genes with small effects and a continuous variation for disease reaction is produced. The genes show additive and non additive effects and the environmental effect is also observed. The polygenic resistance does not show pathotype-specificity as against the oligogenic resistance. It is almost same as horizontal resistance. In some cases the polygenic inheritance may have a oligogenic component, the oligogenes acting in an additive manner eg. bacterial blight resistance in cotton

**Cytoplasmic inheritance :**

Resistance in some cases is determined by cytoplasmic genes or plasma gene(s).

Eg. The T-male sterilizy cytoplasm (cms-T) in maize is extreamly susceptible to *Helminthosporium* leafblight, while the non-T cytoblasms are resistant to this disease.

## Vertical and Horizontal Resistance (Vander plank)

| *Feature* | *Vertical resistance* | *Horizontal resistance* |
|---|---|---|
| 1. Pathotype – specificity | Specific | Non specific |
| 2. Nature of gene action | Oligogenic | Polygenic; rarely oligogenic |
| 3. Response to pathogen | Usually, hypersensitive | Resistant response |
| 4. Phenotypic expression | Qualitative | Quantitative |
| 5. Stage of expression | Seedling to maturity | Expression increases as plant matures |
| 6. Selection and evaluation | Relatively easy | Difficult |
| 7. Host pathogen interaction | Present | Absent |
| 8. Commonly used, synonyms | Major: gene, race -specific seedling, monogenic, pathotype specific resistance | Polygenic, race nonspecific, pathotype-nonspecific, mature plant, adult plant, field uniform resistance |
| 9. Efficiency | Highly efficient against specific races | Variable, but operates against all races |

**Sources of Disease Resistance**

Resistance to diseases may be obtained from four different sources :

A known variety

Germplasm collection

Related species

Through mutation

**A known variety:** Disease reactions of most of the cultivated varieties are documented and a breeder may find the resistance he needs in a cultivated variety. Resistant plants were also lated from commercial varieties as in the case of cabbage yellows in cabbage curlytop resistance etc. These provide the basis for new resistance varieties

**Germplasm collection :** When resistance to a new disease or a new pathotype of a disease is not known in a cultivated variety germplasm collection should be screened. Several instances disease resistance were found from the germplasm collections.

Eg. resistance to neckblotch in barley resistance to wilt in watermelon

**Related species :** Often the resistance to a disease may be found in related species and transferred through interspecific hybridization.

Eg. Resistance to stem, leaf & stripe rusts of wheat

**Mutation :** Resistance to diseases may be obtained through mutation arising spontaneously or induced through mutagenic treatments.

Eg.

Resistance to Victoria blight in oats was induced by irradiation with x-rays or thermal neutrons / also produced spontaneously

Resistance to stripe rust in wheat

Resistance to brown rust in oats

Resistance to mildew in barley

Resistance to rust in linseed

Resistance to tikka leaf spot and stem root in groundnut

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**Vertical and Horizontal Resistance (Van der plank)**

Vertical Resistance is generally d etermined by major genes and is characterized by pathotype specificity. Clearly immune or susceptible response in the case of vertical resistance depends on the presence of virulent pathotype. When virulent pathotype becomes frequent, epidemics are common in the cases of vertical resistance. Thus an avirulent pathotype will produce an immune response i.e. r=0 or close to 0 but the virulent pathotype will lead to susceptible reaction i.e. r=1. It is also known as race specific, pathotype specific or simply specific resistance.

**Horizontal Resistance**

Race non-specific, pathotype -nonspecific and partial, general or field resistance. Horizontal resistance is generally controlled by polygenes i.e. many genes with small effects and it is pathotype nonspecific. In this case, the reproduction rate is not zero but it is less than one. Poly genes, govern horizontal resistance.

**Methods of Breeding for Disease Resistance**

The methods of breeding for disease resistance are essentially same as those used for other agronomic traits. They are :

Introduction
Selection
Hybridization
Budding & Grafting
Mutation Breeding
Biotechnological methods.

**Introduction :** Resistant varieties may be introduced for cultivation in a new area. Eg.
Early varieties of groundnut introduced from USA have been resistant to leaf spot (Tikka)

Kalyanasona and Sonalika wheat varieties originated from segregating material introduced from CIMMYT, Mexico, were rust resistant.
African bajra introductions have been used in developing downy mildew resistant cms lines.

**Selection :** Selection of resistant plants from commercial varieties is easiest method.
Eg.Kufri Red potato is selection from Darjeeling Red round
Pusa Sawani behind (yellow mosaic) selection from a collection obtained from Bihar
MCU I was selection from CO4 for black arm resistance in cotton

**Hybridization :** Transfering disease resistance from one variety or species to the other.

a. Pedigree method is quite suitable for horizontal resistance. Artificial disease epiphytolics are produced to help in selection for disease resistance. Eg. In wheat Kalyana Sona, Sonalaka, Malvika 12
Malvika 37, Malavika 206, Malavika 234
Laxmi in Cotton (Gadag 1 x CO2) for leaf blight resistance

b. Backcross method is used to transfer resistance genes from an undersirable agronomic variety to a susceptible, widely adoptable and is agronomically highly desirable variety.
If the resistant parent is a wholly unadapted variety, backcross method is a logical choice.
If resistant variety also possess some good qualities then chose pedigree method of handling segregating material.

**Budding & Grafting :** The disease resistance in vegetatively propogated material is transferred by adopting either by budding or grafting. By grafting or budding the resistant material, the resistance can be transferred.

**Mutation Breeding :** When adequate resistance is not available in the germplasm ; Mutation breeding is resorted to induce resistance. This is also us ed to break the linkages between desirable resistant genes and other desirable genes.

**Precautions**

The donor parent must possess the required amount of resistance
It must be simply inherited without any linkage
The recovery in the recipient parent should be more
Proper condition for full expression of the resistant genes has to be provided

**Advantages with breeding for disease resistance**

Helps in reducing the losses caused by patogens
Reduces the high cost of disease control by chemical treatment
Helps to avoid the use of poisonous fungicides
Only method available to some specific diseases like viruses, wilt etc.

**Limitations**

Linkage of resistant genes with genes of inferior quality

Occurrence of physiological races of varying capacities

Self sterility in host plants

**Utilization and achievements**

1. Rice ADT 10 x Co4 (resistant to blast)
2. Potato *Solanum* tuberosum x *Solanum* demissum

   (susceptible to late blight) (wild resistant to late blight)

   $F_1$ backcrossed with *Sol. tuberosum*

   Resistant variety

**Varieties resistant to different diseases**

| | |
|---|---|
| Rice : Blast | Co25, Co26, |
| Wheat : all three rusts | : NP 809 |
| Yellow rust | : NP 785, NM86 |
| Black rust | : NP 789 |
| Brown rust | : NP 783, NP 784 |
| Sugarcane : Red rot | Co 419, Co 421, Co 527 |
| Cotton : Wilt | Vijay, Kalyan, Suyog |
| Groundnut : Tikka leafspot | Ah 45 |
| Chilli : Mosaic resistant | Pusa red, Pusa orange |