M.Sc. (Ag) Previous year II semester
Plant Breeding and Genetics
Name of Subject: GP 510- Breeding for biotic and abiotic stress resistance
Unit I- Lecture: Mechanism for the generation of variability in pathogen
Name of course Teachers- Dr. M.K.Shrivastava, Dr. Stuti Sharma, Mr. Pawan Amrete

Mechanism for the generation of variability in pathogen

Pathogens have a wide range of flexible mechanism for producing variability. New variations in pathogens may be created by following ways in case of fungi:

1.Sexual reproduction (recommendation):

Sexual reproduction is a common feature of the most of the pathogenic fungi. In this phenomenon two haploid cells get fused which may or may not be sexually differentiated to produce a cell containing two nuclei, a dikaryon. The duration of dikaryon on varies from very brief period (phycomycetes and primitive ascomycetes) to entire life cycle except for brief diploid and haploid generations (basidomycetes (smuts)). The two nuclei of a dikaryon ultimately fuse to produce a diploid nucleus (zygote) which undergoes meiosis to produce four haploid nuclei. The haploid nuclei may or may not divide mitotically before they produce haploid sexual spores which on germination give rise to the haploid phase of life cycle. Thus life cycle of fungi contains one or three phases i.e. haploidy (n), diploidy (2n) and dikaryotic (n+n) stages. The diploid and dikaryon manifest similar properties in expression. Most of the pathogenic species of fungi however, divide most of their life cycle between the haploid and dikaryotic phase and spend balance in the diploid phase. In such case the pathogenic phase may be haploid or dikaryotic or both. The diploid phase ends with completion of meiosis.

The two haploid cells fusing to from a dikaryon may belong to the same hypha, such fungus species homothallic. In heterothalic fungi haploid cells from two different hyphae (+ and – mating types) fuse Heterothalism ensures the fusion of genetically two dissimilar nuclei to produce the dikaryon. The heterothalism provides chance of new recombinants or variability. (Examples rust, smuts, powdery mildew and potato blight).

2. HeteroKaryoses:

In many fungi e.g. fungi imperfecti (fungi that are not known to produce sexual spores) the hyphae are multi-nucleate during the active growth. These nuclei may be genetically identical or dissimilar. Heterokaryoses is produced by the fusion of vegetative hyphae which are genetically dissimilar; the fusion is not affect by mating types. Heterokaryoses occurs in nature and it has adaptive value. This also undergoes meiosis and may create genetic variability as a result of recombination (Example M. lini)

3. Para sexual reproduction:

In such phenomenon diploid nuclei are produced in vegetative cells from which haploid nuclei are produced through mitotic irregularities (mitotic crossing over) but not through meiosis. Diploid cells occur with very low frequency 10^{-7} among heterokaryon and production of haploid cells occurs 10^{-3} as a result of separation of two nuclei. The pathogenic variability or may be produced by new pathogenic races either through interaction among genes present in the two nuclei of a heterokaryon or through parasexual recombination of genes of two nuclei as well.

4. Mutation:

Spontaneous mutations are ultimate source of all genetic variations present in the biological world. The rate of spontaneous mutation is rather very low (10^{-5} to 10^{-6}). But in case of fungi or pathogen this rate is large enough when it is considered with astronomical number of sexual and asexual spores produced by fungus. In case of *Puccinia recondita* (wheat leaf rust) number of mutant a locus produced per day per hectare estimated as ---1,00,000 at the mutation frequency 10^{-6}. In case of *Puccinia graminis tritici* (stem rust of wheat) the rate of mutation for virulence to Sr5, Sr15, Sr21 and Sr92 were lugh.

5. Heteroploidy:

Heteroploidy is an endogenous system linked to variability and cellular differentiation in various eukaryotes including the fungi. It is well evident in several organisms that the change in chromosome number plays an important role in variability between individuals.

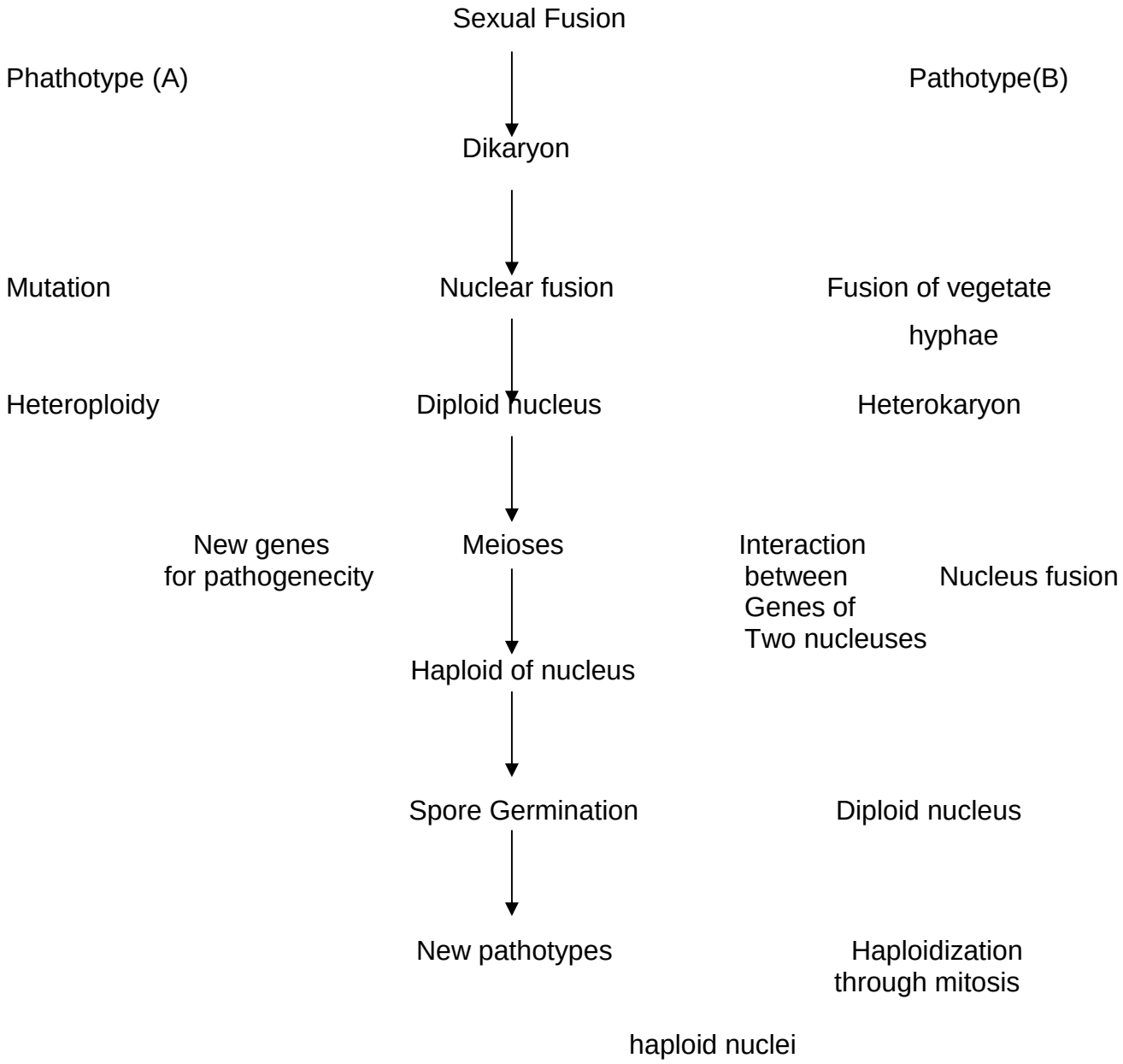


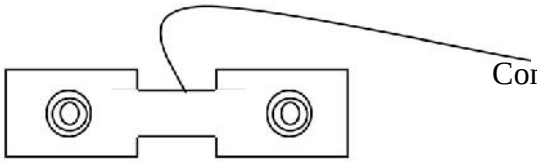
Fig. The possible pathogen for production of new pathogens in pathogenic fungi

Variation in Bacteria:

1. Conjugation 2. Transformation 3. Transduction

1. Conjugation:

During the conjugation DNA is transferred from the donor cell (sometimes called male) to the recipient cell (also called female cell F^-) through a conjugation formed between then. The F factor can integrate at several sites in the bacterial chromosome. This determines the site of origin of transfer in the bacterial chromosome.



Hfr cell - High frequency cell

2. Transformation:

In transformation bacterial cells are transformed genetically by absorbing and incorporating their own cells genetic material secreted by, or released during rupture of other bacteria. The DNA of other bacteria replaces the homologous segments of the chromosomes of recipient cells. The uptake of DNA nodules is an active process requiring energy. Therefore, transformation occurs in only those species of bacteria that possess the enzymes for active transport. In a culture only some cells are capable of transformation called competent.

3. Transduction :

Transfer of DNA from one bacteria strain into another by bacteriophages or viruses and the subsequent recombination below on the chromosomes of recipient cells and the introduced DNA, is known as transduction. Transduction may be either generalized or specialized. In generalized transduction, a random or nearly random segment of the bacterial chromosomes is transferred by the virus. In specialized transduction the recombination occurs at specific attachment sites present in both phase and host chromosomes.

Variation in Viruses:

1. Mutation and 2. Recombination in viruses
2. Recombination: Viral chromosomes undergo recombination during their reproduction in host cells. Therefore a host cell must be simultaneously infected by two different strains/ genotypes of a virus in order to permit their chromosomes to undergo recombination. This is achieved by infecting host cells with a mixture of two selected virus strains. The

density of virus intensity is kept high so that on an average each host cell is get infected by two or more virus particles.

Physiological races and pathotypes

The concept of physiological races introduced by Barrus in 1911. Physiological races are strains of a single pathogen species differing in their ability to attack different varieties of the same host species. The varieties of a host species used to identify physiological races of a pathogen are known as differential hosts or host testers. Differential hosts are chosen on the basis of differences in their resistance to the pathogen, but the genes for resistance present in them are usually not known. Ideally each of the different hosts should possess a single resistance gene different from those present in others. Such a set of differentials is known as ideal differentials. In such case n differentials hosts would be able to identify 2^n physiological races if there were only two types of host pathogen interaction e.g. resistant and susceptible. It however more types of reaction available as in the case in stem rust of wheat; a still larger number of physiological races can be identified. The number of race identified would be r^n.

R= number of reaction types identifiable

N= number of differential hosts with a single unique resistance gene

Identification of physiological races can be explained using bunt resistance of Martin and Turkey wheat varieties. A set of 18 differential hosts each carrying a single gene available for linseed stem rust and a set of 16 differentials is available for late blight in potatoes.

Identification of 4 physiological races of Tilletia sp. producing wheat bunt using for differential wheat varieties.

Differential host	**Reaction to bunt**			
	Race-I	**Race-II**	**Race-III**	**Race-IV**
Martin	R	R	S	S
Turkey	R	S	R	S

A pathogen race capable of attacking a host strain carrying a specific resistance gene said to be virulent towards that gene while those unable to attack this strain are known as avirulent. When the strains of a pathogen are classified on the basis of their virulence to know resistance genes present in the host they are referred to as pathotypes.

Pathotype classification is more precise than that of the physiological races.

Pathotype classification based on 12 differential hosts of phytopthera infestans (Late blight of potato)

Resistance gene in differential host	Pathotype of potato late blight fungus											
	P1	**P2**	**P3**	**P4**	**P5**	**P6**	**P7**	**P8**	**P9**	**P10**	**P11**	**P12**
R1	s											
R2		s										
R3			s									
R4				s								
R5					s							
R6						s						
R7							s					
R8								s				
R9									s			
R10										s		
R11											s	
R12												s

If the differential hosts have single and distinct resistance gene than identified physiological races will be infect pathotypes.

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Unit I- Lecture-6: *Concept in Pathogen Resistance*
Name of courseTeachers- Dr. M.K.Shrivastava, Dr. Stuti Sharma, Mr. Pawan Amrete

Concept in Pathogen Resistance

"Resistance refers to any inherited character of a host plant which lessens the effect of Parasitism" (Russel, 1978).
"Robinson (1969) defined resistance as the ability of the host to hinder a pathogen or disease causing agent."
Disease resistance in host involves a restriction on the establishment and, particularly, multiplication of the pathogen.

Classification of resistance:
There are different basis of classification:

A. Based on existence:
1. Preformed or pre-existing or passive or constitutive
2. Induced or active or acquired: a. localized acquired resistance
   b. systemic acquired resistance

B. Based on type of host response:
1. Immune
2. Resistant
3. Tolerant

C. Based on genetic control or genetic basis:
1. Oligogenic
2. Polygenic
3. Cytoplasmic

D. Based on mechanism of resistance:
1. Mechanical
2. Biochemical: a. Hypersensitive or phytoalexins
   b. Resistance to phytotoxins
3. Nutritional

E. Based on degree or range of resistance:
1. Vertical resistance
2. Horizontal resistance

F. Based on persistence or duration:
1. Durable
2. Non durable

A. Based on existence

1. Preformed or preexisting or passive or constitutive:

When the resistance is already present in the plant in the absence of pathogen it is known as preformed. Such resistance is governed by two types of defense mechanical ie. Pre-existing defense structures and pre-existing chemical defense. The defense structures includes the amount and quality of morphological characters as thick cuticle, presence of wax, thick mat of hairs, thickness and toughness of outer wall of epidermis cells hinder the penetration of pathogen. The properties of stomata also hinder pathogen. In case of stem rust of wheat if stomata open late, rust spores do not germinate as dew evaporates till that time. Narrow stomata opening, broad and elevated guard cell tips confer resistance against certain bacterial pathogens.

Pre-existing defense chemicals also provide resistance against pathogens produced by secondary metabolites. Such chemicals include phenolics of varying structural sophistication, terpenoides and steroids.

Some compounds are directly toxic to pathogen while other exists as conjugates such as glycosides which are not directly toxic but become toxic following disruption of the conjugate. For instances plant glycosides are often hydrolyzed following pathogen ingress that releases vacuolar glycosidases, for examples preformed saponine glycoside, avenacin in oat plants inhibit the growth of root fungus. Similarly tomatine in tomato has been found antifungal.

2. Induced systemic resistance or active resistance or acquired resistance:

When plant comes into the contact of pathogen, resistance is induced. Inducible resistance mechanisms are active, energy requiring systems typified by specific recognition of an invader that ultimately leads to production of proteins or metabolites that are antagonistic to the invaders. Such active resistance may be localized at the site of the infection or it can be systemic. Various pathogens especially, Fungi and bacteria releases substances such as glycoprotein, carbohydrates fatty acids and peptides. Some of these substances directly act as elicitors (प्रकाश में लाना, निकालना) but in many cases host enzymes breakdown a portion of the polysaccharides making up the pathogen surface, or pathogen enzymes breakdown a portion of plant surface polysaccharides, the released monomers (a molecule that can be bonded to other identical molecules to form a polymer) of the polysaccharides acts as recognition elicitors for the plant. The receptors in the plant recognized the elicitors of pathogens. Following recognition, plants have series of structural and biochemical changes.

Among the structural changes cytoplasmic defense reaction, cell wall defense structures, histological defense (formation of cork layers) and necrosis of affected cell. (Glyco proteins)

Among the biochemical defense mechanism, there is production of proteins or metabolites that are antagonistic to the invaders (घुसने वाला, आक्रमण करने वाला) such as glucanses, lycozyme active proteins. The induced resistance is further divided into two mechanisms. Local acquired resistance and systemic acquired resistance.

a. Local acquired resistance:

The infection by fungi, bacteria or viruses will elicit a set of localized response in and around the infected host cells. These responses include an oxidative burst (Lamb and Dixon, 1997) which can lead to cell death (Kombrink and Schmetzer, 2001). Thus the pathogens may be trapped in dead cells and appears to be prevented from the site of initial infection further local responses in the surrounding cells include changes in the cell wall composition through cell wall strengthening proteins such as hydroxyprotein rich glycoprotiens and response proteins may also work for lignifications (it becomes harder) of cells wall. This inhibits the penetration of pathogens. The denovo synthesis (नए सिरे से) of phytoalexins and pathogesis related (PR) proteins cause inhibition in the spread of infection.

b. Systemic Acquired Resistance:

Such phenomenon is already recognized long back in 1901 by *Ray and Beauvene* working with *Botrytis cinerea* (Gray mold). In 1933 Chester reviewed 200 publications and found that scientists of at that time believed they were investigating a phenomenon analogous to the immune response in mammals. In retrospect (a survey or review of past course of events or period of time), three different process were being called acquired immunity; viral cross protection, antagonism and what we now referred to as systemic acquired resistance (SAR). The first systematic study of SAR in published by A. Frank Ross in 1961 who demonstrated that inoculation of single leaf of tobacco with tobacco mosaic virus (TMV) reduced severity of subsequent infection on other leaves. He coined the term SAR. SAR differs from viral cross protection in being non specific and from antagonism in being based on an active plant processes. The SAR is the phenomenon where by a plant on defense mechanisms are induced by prior treatment with either a biological or chemical agent. It is a long lasting systemic resistance effective against viral, bacterial and fungal pathogens.

Mechanism of SAR: Van Loon and Van Kammen (1970) and Gianinazzi et, al. (1970) showed that viral infection of tobacco induced the accumulation of distinct set of pathogenesis related proteins (PR proteins). Ward et al. (1991) demonstrated nine gene families now known as SAR genes which are responsible for production of anti-microbial substances and proteins as P-1,3 glyconases, chitinases, cystein rich proteins and PR-1 proteins. In cucumber, a class III chitinase is the most highly induced SAR gave while in tobacco and arabidopsis PR-1 and NPR-1 are the predominant gens.

Morphological and biochemical changes in SAR protects plants through lignifications in response to peroxidase activity, other changes are the increase level of glucose and fructose concentration in systemic tissue, accumulations of fungitoxic substance, induction of lipoxygenase, antimicrobial fatty acids, phytoalexins and hydroxyproline rich glycol protein. SAR is accompanied by elevated levels of salicylic acid.

Induction of SAR: (by biological agent)

Kessmann et al. (1994) reported that an extract of Rheynoutria Sachatinenses gave good control of powdery mildew and other crop pathogens. Extract of Bacillus subtilis have been reported to induce resistance in barley, especially against powdery mildew. Penicillium janczewskii or its culture filtrate into melon and cotton increased perosidase activities and resulted in increased protection against *Rhizoctonia solani* and elimination of the incidence of damping off systems. Similarly, there are several reports on biological organisms like Pseudomonas and Bacillus have been found but interestingly no commercial biological products with an SAR mode of action has been introduced into market.

By chemicals: Development of SAR involves the accumulation of salicylic acid. Exogenous application of salicylic acid to the leaves of tobacco induces resistance to the same spectrum of pathogens and the expression of the same set of SAR genes as does the pathogenic induced SAR. Some reports indicate that exogenous spray of SA does not translocate effectively in total plant system and it is short term response.

SAR is also induced by synthetic chemicals viz. salicylic acid, 2, 6-dichloroisonicotinic acid (INA) and benzo (1,2,3) thiadiazole-7-carbothioic acid-s-methyl ester (BTH) in both monocot and dicots. Application of Jasmonic acid (JA) has been also found showing SAR response. Bio-pesticides " Messenger" based on Harpin protection obtained from Erwinia amylovora induced SAR in crop plants against bacteria, fungi, viruses, insect pests and nematodes. Application of Messenger improves the yield, quality and overall plant performance.

SAR may be one of the main reasons for abnormal segregation for disease resistance observed in field studies.

B. Based on type of host response or Host Parasite reaction:

1. Immune 2. Resistant 3. Tolerant

1. Immune:

It is an extreme form of resistance, when a host does not show any symptoms of a disease. Immunity may result from the prevention of pathogen to reach the appropriate parts of the host e.g. exclusion of the spores of ovary infecting fungi by closed flowering habit of wheat and barley. But more generally it is produced by hypersensitive reaction of the host, usually, immediately after the infection has occurred. In hypersensitive reaction a group of host cells around the point of infection dies. This severity restricts the establishment of pathogen and eliminates its reproduction. Thus in immune reaction the rate of reproduction of the pathogen is zero, that is, r=0.

2. Resistance:

Resistant denotes less disease development in a genotype than that in the susceptible variety and is a relative attribute. Infection and establishment do take place but the growth of pathogen in the host tissue is restricted. This results in smaller spots or pustules than in the

susceptible variety. Generally the rate of reproduction is considerably reduced which limits the spread of disease. In case of resistance the disease symptoms do develop and the rate of reproduction is never zero, e.g. r>0, but sufficiently lower than 1 as incase of susceptible reaction.

3. Tolerance:

In such reaction, host is attacked by the pathogen and reaction is similar to susceptible variety but there is little or no loss in biomass production or yield. In certain situations this may be so but often this term is used without sufficient evidence. Generally tolerance is difficult to measure because it is confounded with partial resistance and escape. To estimate tolerance, the loss in yield or some other traits of several host varieties having the same amount of disease, leaf area covered by disease is compared. An example of true tolerance is known in the barley variety proctor to powdery mildew. Virologists use the term tolerance to denote the lack of symptoms even in the presence of the virus.

C. Based on genetic control or genetic basis:

1. Oligogenic 2. Polygenic 3. Cytoplasmic

1. Oligogenic:

In such case disease resistance is governed by one or few major genes and resistance may be dominant and recessive. In such case generally two distinct reactions either resistant or susceptible reaction is expected. The chief characteristic of olegogenic inheritance of disease is its pathotype specificity that is particular resistance gene is effective against some pathotypes and ineffective against other. For example in wheat leaf rust more than 20 and against stem rust more than 30 resistance genes are known (such genes are less affected by environments). The genetics of oligogenic resistance advanced by two events occurring repeatedly which are (1) Search of a new resistance gene to the prevalent pathotype and (2) the evolution of a pathotype virulent towards new resistance gene. Oligogenic resistance is synonymous to vertical resistance. For example Anthracnose resistance in *Phaseolus vulgaris* (Bean)

Pathotype	**Rajma variety white marrow AABB**	**Robust aaBB**	**F_1**	**Ratio in F_2**	
				Resistant	**Susceptible**
Alpha (Pb)	R	S	R	3	1
Beta (Pa)	S	R	R	3	1
Avirulent (Po)	R	R	R	15	1
Alpha+Beta Mixture (Pa+Pb)	S	S	R	9	7

2. Polygenic resistance:

When resistance is governed by many genes having small effects. Disease reaction in this case shows continuous variation and no distinct class is found. Polygenes show both additive and non additive effects and largely affected by environments as in case of quantitative traits.

The mechanism of resistance is not clearly known but show growth of pathogen and slow spore production leads to resistance against infection.

Polygenic resistance does not show any race specificity. It is synonym to horizontal resistance or partial resistance for example bacterial blight resistance in cotton.

3. Cytoplasmic Resistance:

In some cases resistance is governed by cytoplasmic gene (s) or plasma gene (s). For example maize strain having Texas (T) male sterile cytoplasm (cms-T) are extremely susceptible to *Helminthosporium* leaf blight. Where as normal or non T cytoplasm strains show resistance. Such resistance is rare in nature and this one is good and classical example. Contrary to this C or S cytoplasm are also male sterile but are resistance to T race of the pathogen.

D. Based on mechanisms of resistance:

i. Mechanical ii. Biochemical iii. Nutritional

i. Mechanical:

Certain mechanical and or anatomical (structural) features of the host may prevent infection. First line of defense mechanism is surface of plant parts like thick hair, hard cuticle, wax on stem and leaves check the contact, germination, and entry of pathogen in host tissues.

ii. Biochemical or physiological:

A series of different reactions are produced in the host following are attack by the pathogen. Such reaction can be divided into two types:

(a). Hypersensitive reaction (phytoalexins):

In large number of cases a necrotic spot (fleck) is marked on host tissues and typical immunity is the result of hypersensitive reaction. Ward (1902) focused attention on the hyper sensitive reaction as potential defense mechanisms of plants. Such reaction is due to increased sensitivity of host cells in the regions of infection site and expressed as necrotic area. This is due to increased oxydative enzymes or phenolic compounds (phytoalexins). Mullar and Borger (1939) formulated the phytoalexin theory. According to him an incompatible host parasite relationship, the host tissue releases the toxic substances that prevent the further development of parasite. Phytoalexins are fungicidal or fungistatic. Mullar (1959) described hypersensitive as “All morphological and histological changes when produced by an infectious agent elicit the premature death (necrosis) of infected tissues. Phytoalexin productions are also induced by certain microbial compounds named as elicitors (carbohydrate or glycoproteins) produced by pathogens. Phytoalexins are phenolics or terpenoid (low molecular weight compound) for example:

Isofavonoid in leguminosae, Terpenoid in solanceae

Overthirty phytoalexins are known: In pea- Pisatin, Bean- Phaseolin, Potato- rishitin.
The best example of phytoalexin is found in potato against phytopthora infestans.

(b). Resistance to phytotoxins:

Some pathogens produce toxins to kill the host tissue. In resistant genotypes non sensitive responses are found against the toxin produced by pathogens.

Holden (1980) conferred the role of avanacin in oat as antifungal compound against. G. *gramins*, *Phialophora radicicola*, *Leptisphaeria normasi*. Similar, response to toxins of *Exserohilum turcicum* (Basan and Levey, 1992) *H. Carbonum* (Wolf and Earley, 1991) in maize has been reported.

iii. Nutritional:

Until the host tissue supplies all the nutrients necessary for the growth of pathogen is available the later will grow and cause the infection. Walker and Stahmann (1955) suggested in some pathogens the availability of highly elaborate growth factors such as vitamins, polypeptides, amino acids, proteins and enzymes are perquisites for normal parasitism.

E. Based on degree and range of resistance (it is also genetic mechanism):

(Van der Plank (1963) coined the term vertical &horizontal persistence)

1. Vertical resistance (VR) or perpendicular:

Synonyms: Differential, hypersensitivity, pathogen specific, race specific, specific, non uniform, major/ oligogenic seedling resistance (Abdalla and Hermsen 1971).

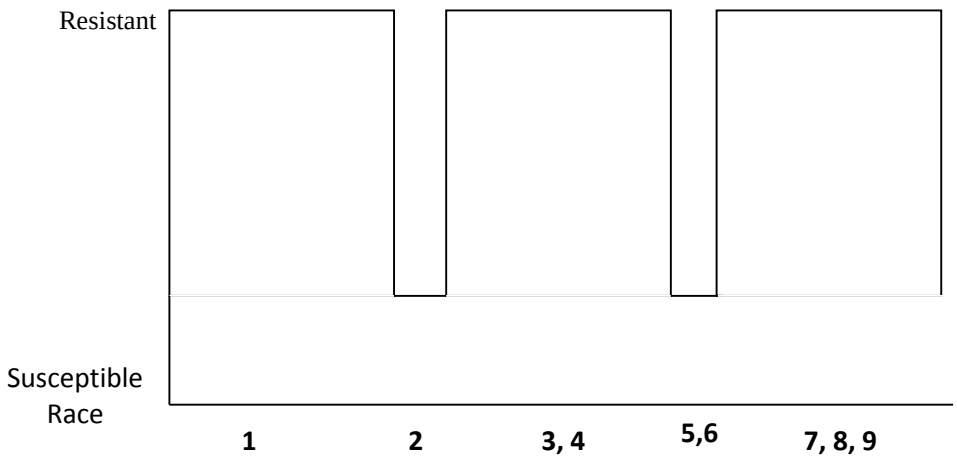
When a variety shows resistance against one or some specific races or pathotypes of a pathogen but not against others race of the same pathogen. Such resistance is qualitative produces distinct classes of reaction either resistant or susceptible. It shows hypersensitive reaction against diseases. Gene for gene relationship can be established. Vertical resistance is temporary which changes with change of races or environment (location). Differential reactions are shown by varieties against different races. In such case there is no development of disease symptoms. Means r=0 or close to O. but in case of attack of virulent races susceptible reaction will be result and r=1. It is suitable for annuals but not for perennials. It is suitable for immobile pathogens e.g. soil pathogens but not for air borne pathogens as smut, rusts.

ii. Horizontal resistance (HR) or lateral resistance (LR):

Synonyms: Partial, slow rusting, fied, generalized, race or pathogen non specific, uniform, minor gene or polygenic, quantitative, Adult plant resistance (Abdalla Hermson, 1971).

This type of resistance is characterized as race non specific means there is similar resistance reaction against all the races or pathotypes. Such resistance is known as quantitative and governed by Polygenes; hence no distinct class is visible means continuous variations. HR does not establish gene for gene relationship. It is difficult to handle. It is stable, durable and frequently found in annual and perennials both. Horizontal resistance is based on capacity of the plant cell to limit infection and reduces the rate of development of the pathogens. Hence, it is considered to be true resistance. HR is generally low level and has low heritability. Each gene

has additive and small effects and interaction among the genes also works. As per Perlevliet (1981) so called HR is difficult to separate from VR.

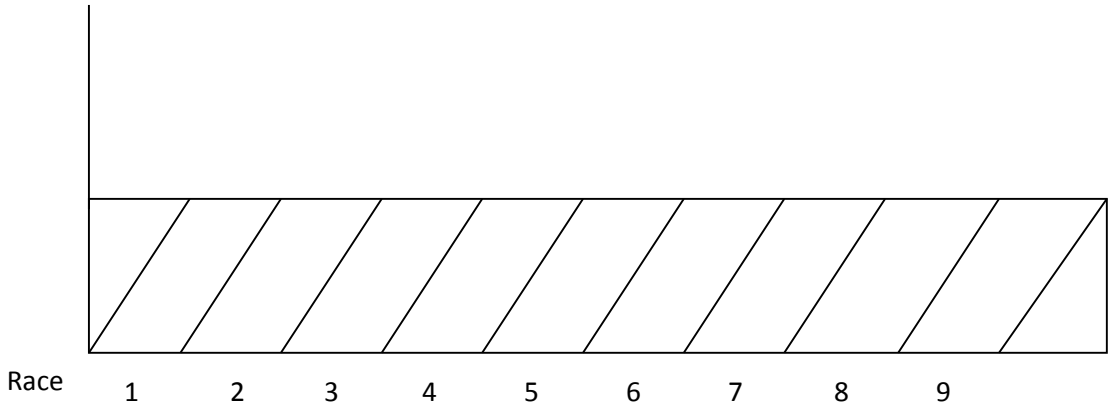


Pathotype Specific

	P_1	P_2	P_3
R_1	S(1)	I(0)	I(0)
R_2	I(0)	S(1)	I(0)
R_3	I(0)	I(0)	S(1)

S = Susceptible
R = Resistant
I = Immune

Vertical Resistant



Pathotype non specific

R	**R**	**R**
<1	**<1**	**<1**

Horizontal Resistant

Several Hypotheses has been accounted for HR:

1. Ghost gene hypothesis: According to this HR represents the cumulative effect of VR genes which have been succumbed to the corresponding virulence genes of the pathogen i.e. ghost or residual effects of VR genes.
2. HR may be due to the modifying effects of the genetic background on VR genes and due to several VR genes being present together. This is similar to the ghost hypothesis.
3. HR is governed by polygenes. Oligogenes act as regulators of HR making the action of polygenes race specific.

4. The polygenes show genes for genes relationship with minor virulence genes of the pathogen, which means that they are race specific. But their effect appears to be race non specific HR due to interaction effects due to relatively larger error variance.

F. Based on duration or persistence:

(* portion mentioned before non durable resistance)

(i) Durable resistance:

It is defined as resistance that has remained effective for a considerable time while a cultivar possessing it has been widely cultivated in an environment favouring disease (Johnson and Law, 1973; Johnson 1979, 1981). It is a description based on observation not on assumption. It does not imply that resistant will remain permanently effective. In this sense it is a quantitative concept. The Rpg1 gene resistant for stem rust of wheat was found durable but did not last forever. In the evolutionary sense, no resistance will last forever. The most powerful test for detection of durable resistance occurs when a variety is widely grown commercially for several years. It is possible to discern three groups of resistance that are predominantly durable:

1. Resistance to pathogens with a wide host range, generalists are usually of a quantitative nature (Bruehl, 1983) and nearly always durable (Parlevliet, 1993). But there are exceptions, such as major resistance genes Mi in tomato and Rk in cowpea against the root knot nematode, M incognita (Roberts, 1995)

* Generalists- the pathogen that has wide host range.

*Specialists- the pathogen that has narrow host range.

2. Quantitative resistance (QR) against specialists (narrow host range) and based on some to several genes with additive effects seems durable.

In few cases QR broke down seems to be monogenic like fold resistance against rice blast M. grisea in Rice cultivar St-1. The resistance become ineffective within a few years appears to be based on single dominant gene.

3. Monogenic resistance of a non hypersensitive nature: Such resistance is often quite durable. The non hypersensitive resistance genes Rpg 2 (sr=2) and Rpr 34 (Lr-34) of wheat to stem rust and leaf rust, respectively and mlo gene of barley to powdery mildew have already lasted for a considerable time.

The mechanism of resistance may be race specific or non race specific. Polygenic resistance usually considered to be non race specific and durable. However, durability were also demonstrated where polygenic race specific systems are operated (Parlevliet, 1997). It has been reported that polygene for polygene system was more durable.

The ability of cultivar for durable resistance is dependent on:

1. The inherent durability of its resistance.
2. Inherent variability in pathogenecity.
3. Life cycle of host and pathogen.
4. The manner and extent of deployment of the cultivar and of the other resistant and susceptible host cultivars or host species.
5. Epedemiological conditions i.e. climate & weather.

Keeping all other factors as the generalized or horizontal resistances are considered as durable resistance, however evidence of durable resistance in other systems also prevails. It will be better to combine breeding for horizontal and vertical resistance into one programme for durable resistance.

Lutescence 62 a Russian variety remains resistant for 45 years. Cappelle- Desprez has been found having durable resistance against yellow rust.

(ii) Non durable resistance:

In nature there is a constant confrontation between parasite and host. In the evolutionary sense all the resistance is transitory. Resistance may already be neutralized in the last stage of the breeding programme, and may still be effective after more than 130 years and wide exposure as the case of the Phylloxera aphid resistance of grape root stocks (Niks et. al, 1993).

There are many types of resistance that appears highly elusive, the effective time ranging from less than one year to several years. A major gene including the hypersensitive reaction almost invariably controls this clearly non durable resistance. These genes operate in a gene for gene with avirulence genes in the pathogen. The pathogens are mostly specialized fungi and bacteria such as the cereal rusts, cereal powdery mildew, flax rust of flax, rust of maize, late blight of potato.

However, not all the major gene resistance are elusive and some major gene resistance of non hypersensitive are also elusive.