GROUPS OF PESTS TARGETED BY PLANT BREEDERS

- Plant diseases are caused by pathogens that vary in nature and may be microscopic or readily visible (e.g., virus, plant, animal).

- Six general groups of causal agents of disease, which represent six general approaches to breeding for pest resistance, may be identified as: **airborne fungi**, **soil-borne fungi**, **bacteria**, **viruses**, **nematodes**, and **insects.**

- Through an understanding of the biology, epidemics, spread, and damage caused by these organisms in each category, breeders have developed certain strategies and methods for breeding cultivars to resist certain types of biotic stress in plant production.

- Plant species vary in their susceptibility to diseases caused by pathogens or pests in each group.

- Cereal crops tend to have significant airborne fungal disease problems, while solanaceous species tend to experience viral attacks.

- Breeding for resistance to fungi, especially airborne fungi, is the most prominent resistance breeding activity.

- N. W. Simmonds has suggested that the relative importance of the six groups of pathogens of importance to plant breeders, might be something like this: airborne fungi > soil-borne fungi > viruses > bacteria = nematodes= insects.

BIOLOGICAL AND ECONOMIC EFFECTS OF PLANT PESTS

- **1 Complete plant death**. Certain parasites sooner or later will completely kill the afflicted plant; include those that cause mildews, vascular wilts, and insects such as cutworms that cause a seedling to fall over and die.

- **2 Stunted growth**. Viruses are known to reduce the metabolic performance of plants without killing them outright.

- **3 Partial plant death**. Some diseases that afflict adult plants do not completely kill them. Rather, only certain parts of the plant (e.g., branches) are killed(e.g., as observed in fungal diebacks).

- **4 Direct product damage** Some pests directly injure these products completely (e.g., by causing rotting of tissue) or reducing quality (e.g., by causing blemishes, holes).

Overview of the methods of control of plant parasites

1. **Exclusion of pathogen from the host.** This strategy may use methods such as legislation (plant quarantine, crop inspection) or crop isolation to prevent the pathogen or pest from making initial contact with the host plant.

2. **Reduction or elimination of the pathogen's inoculum**. A method such as crop rotation reduces disease buildup in the field, while observance of sanitation (e.g., removing diseased plants and burning them) reduces the spread of the pathogen.

3. **Improvement of host resistance**. This is the strategy of most concern to plant breeders. It entails breeding to introduce genetic resistance into adapted cultivars

4. **Protection of the host**. Economic plants may be protected from parasites by using chemicals (pesticides).

PATHOGEN AND HOST

- The **pathogen** is a living organism that is capable of inflicting a distinct disease or disorder in another organism (the host).
- The capacity of the pathogen to cause disease or disorder in a member of a host species is called its **pathogenecity**.
- The extent of disease development pathogen causes is its **virulence**.
- The pathogenecity and virulence of a pathogen vary among pathogen types (**races** or **pathotypes**).

- Races or pathotypes that fail to cause disease symptoms or successfully attack a given host are said to be **avirulent**.

- A third factor – favorable environment – is needed, the trio (pathogen plus susceptible host plus favorable environment) referred to as the **disease triangle**.

- Pathotypes or races of pathogens may also be described in terms of **aggressiveness** or **non-aggressiveness** in relation to the rate at which they produce disease symptoms.

- **The host**

The host (genotype, plant) is the organism in which a pathogen may produce disease symptoms.

- A susceptible host is one in which a pathotype or race can manifest a disease symptom.
- A host may employ one of several mechanisms (defense mechanisms) to resist pathogens.

1 Pre-existing defense mechanisms. These include morphological features that pose as barriers to the penetration of the pathogen into the plant (e.g., presence of lignin, cork layer, callose layers), or secondary metabolites (phenols, alkaloids, glycosides) that have antimicrobial properties.

2 Infection-induced defense mechanisms. Upon infection, the host quickly produces chemical products (e.g., peroxidases, hydrolases, phytoalexins, etc.) to combat the infection.

Mechanisms of defense in plants against pests

- **Mechanisms of defense in plants against pests** Plants exhibit a wide variety of strategies and mechanisms of defense against pathogens and insects pest that may be classified into three major groups – **avoidance**, **resistance**, and **tolerance**.

- **Avoidance** Also described as **escape**, avoidance is a mechanism that reduces the probability of contact between pathogens or insect pests and the plant.

- **Resistance** The mechanism of resistance manifests after a host has been attacked by a pathogen or insect pest. The mechanism operates to curtail the invasion or to reduce the growth and/or development of the pathogen.

- **Tolerance** Unlike avoidance and resistance mechanisms that operate to reduce the levels of infection by the pathogen or pest, tolerance (or endurance) operates to reduce the extent of damage inflicted. The afflicted host attempts to perform normally in spite of the biotic stress.

Types of genetic resistance

- The complexity of host–pathogen interaction makes it difficult to categorize resistance into finite types.
- A large number of host–pathogen interaction systems occur at various stages of coevolution.
- Resistance reactions may be generally categorized into two major kinds – **vertical** or **horizontal** – based on their epidemiological status and stability of resistance.

VERTICAL RESISTANCE / RACE OR PATHOTYPE-SPECIFIC RESISTANCE/ OLIGOGENIC RESISTANCE /NONDURABLE /QUALITATIVE RESISTANCE/NON-UNIFORM RESISTANCE

- This reaction is said to occur when a race of a pathogen produces disease symptoms on some cultivars of a host but fails to do so on others.
- This type of resistance is relatively easy to breed because the major genes are easy to identify and transfer through simple crosses.
- These genes control specific races or genotypes of pests and hence do not protect against new races of the pests.

HORIZONTAL RESISTANCE/PARTIAL RESISTANCE/ RACE-NON-SPECIFIC RESISTANCE/MINOR GENE REACTIONS/ POLYGENIC RESISTANCE.

- The resistance is effective against all genotypes of the parasite species without cultivar × isolate interaction (i.e., race-non-specific).
- Horizontal resistance is controlled by polygenes. Each of the genes that condition the disease contributes toward the level of resistance, and hence resistance is also called **minor gene resistance**.
- Breeding polygenic resistance is more challenging. The many minor genes cannot be individually identified and consequently cannot be transferred through crossing in a predictable fashion.

GENETICS OF HOST–PATHOGEN REACTIONS

- R. H. Biffen is credited with providing the first report on the genetics of resistance. Working on stripe rust (*Puccinia striiformis*), he reported that resistance to disease was controlled by a single Mendelian gene.
- However, it is known that resistance may be controlled by any number of genes whose effects may be large or small.
- Further, the genes may interact epistatically or additively.

GENE-FOR-GENE REACTIONS (GENETICS OF SPECIFICITY)

- Working on flax rust (caused by *Melamspora lini*), H. H. Flor discovered that the major genes for resistance in the host interacted specifically with major genes for avirulence in the pathogen.
- For each gene conditioning resistance in the host, there is a specific gene conditioning virulence in the parasite.
- In the host the genes for resistance are Dominant(R) , however gene for susceptibility are recessive(r).
- In the pathogen genes for avirulence (inability to infect) are usually dominant(A) whereas genes for virulence are recessive(a).

Quadratic Check of Gene Combinations and Disease Reaction Types in a Host–Pathogen System in Which the Gene-for-Gene Concept for One Gene Operates[a]

Virulence or avirulence genes in the pathogen	Resistance or susceptibility genes in the plant	
	R (resistant) dominant	r (susceptible) recessive
A (avirulent) dominant	AR (–)	Ar (+)
a (virulent) recessive	aR (+)	ar (+)

[a]Minus signs indicate incompatible (resistant) reactions and therefore no infection. Plus signs indicate compatible (susceptible) reactions and therefore infection develops.

General considerations for breeding resistance to parasites

1. Breeding is an expensive and long-duration undertaking that makes it only justifiable for major pests that impact crops that are widely produced or have significant benefit to society.

2. Natural resistance is not available for all pests. Sometimes, the resistance is available in unadapted gene pools, requiring additional costs of prebreeding.

3. Breeding for resistance varies in ease and level of success from one pest to another. Resistance to vascular pathogens, viruses, smuts, rusts, and mildews is relatively easier to breed than breeding against pathogens that cause rots (root rot, crown rot, storage rot) and ectoparasitic nematodes. Similarly, it is relatively easier to have success with breeding resistance to aphids, green bugs, and hoppers, than to breed resistance to root-chewing or grain-storage pests.

4. Instability of pest resistance is a key consideration breeding for pest control because diseases and insect.pests continue to change. New pathogenic races may arise, or the cultural environment may modify the resistance of the cultivar.

5. The techniques of biotechnology may be effective in addressing some breeding problems more readily than traditional methods.

6. After being satisfied that breeding for disease resistance is economical, the breeder should select the defense mechanism that would be most effective for the crop, taking into account the market demands. For example, horticultural products and produce for export usually require that the product to be free from blemish. In these cases, breeding for major gene resistance with complete expression is desirable. It is also easy to breed for this type of reaction. However, the breeders should note that this resistance is not durable.

7. When a crop is grown for food or feed, breeding for mechanisms that increase the levels of chemical toxins in plant tissues is not suitable.

RESISTANCE BREEDING STRATEGIES

- Disease breeding is a major objective for plant breeders.
- It is estimated that 95–98% of cultivars of small grains grown in the USA have at least one gene for disease resistance.
- It should be pointed out that a combination of traits rather than just one trait, makes a cultivar desirable.
- Yield and resistance to disease are top considerations in breeding programs.

1. SPECIFICITY IN THE PARASITE

- When pathogen genotypes share a group of cultivars to which they are virulent, they are said to belong to a **physiological race**.
- Physiological races of pathogens occur in rusts, powdery mildew, and some insects.
- The physiological races may be identified by using **differential cultivars** (contain known genes for disease reaction).
- Breeders use a series of differentials to determine what genes would be most effective to incorporate into a cultivar.
- The concept of differentials stems from the ability of a cultivar to differentiate between races of a parasite on the basis of disease reaction.
- If a cultivar has resistance to one race but is susceptible to another, it has differential properties to identify two races of a pathogen and hence is called a **differential**.

- In that case, four races of a pathogen can be differentiated

		Races			
		R_1	R_2	R_3	R_4
Cultivars	C_1	−	−	+	+
	C_2	−	+	−	+

- The ideal set of differential cultivars is one in which each cultivar carries a gene for resistance to only one race.

- The differential cultivars provide some information on the virulence characteristics present in resistance to which the pathogen population carries avirulent genes, and, similarly, the genes for resistance of the host that would fail because the pathogen possesses the necessary genes for virulence.

2. Planned release of resistance genes

- It is recommended to have a planned release (consecutive release of different resistance genes) of resistance genes so that only one or a few are used in agricultural production at one time.

- Once a current cultivar succumbs to a new race of a pathogen (i.e., a new race that is virulent with the resistance gene in use), breeders then release new cultivars that carry another effective gene. This way, plant breeding stays ahead of the pathogen

3. Application of gene pyramiding

- The concept of transferring several specific genes into one plant is called **gene pyramiding**.

- Because there are different races of pathogens, plant breeders may want to transfer a number of genes for conferring resistance to different races of a disease into a cultivar.

- Functional stacking of three resistance genes against *Phytophthora infestans* in potato.

Three broad spectrum potato R genes (Rpi), Rpi-sto1 (Solanum stoloniferum), Rpi-vnt1.1 (S. venturii) and Rpi-blb3 (S. bulbocastanum) were selected, combined and transformed into the susceptible cultivar to exhibit durable resistance.

4. BREEDING FOR HORIZONTAL RESISTANCE

- It is suitable for both annuals and perennials and is applicable to all pathogens.
- Breeding for general resistance is more challenging because many genes with minor effects are involved.
- It is laborious to develop breeding stocks with horizontal resistance.
- However, it is easy to improve on the very low level of horizontal resistance that normally underlies a failed vertical resistance.
- Such improvements may be accomplished by using recurrent selection methods.

5. Breeding for vertical resistance

- Vertical resistance is pathotype- specific and easy to breed.

- Boom and bust cycles

Arise when major genes for vertical resistance against a major economic race of a pathogen are used in cultivar development for a region, leading to widespread adoption of the resistant cultivars by most producers in the region (**boom phase**).

Selection pressure on the races of the pathogen present in the cultivars reduces the virulent ones.

However, the less virulent one against which the cultivars carry no major genes continues to increase until it becomes epidemic in the vast region of production of the crop (**bust phase**).

General strategies may be used to make vertical resistance a success

1. **Temporal deployment**. A strategy for enhancing the success of vertical resistance is to develop and release several cultivars in successive or cyclical fashion.

2. **Geographic deployment**. The application is of limited practical use because it requires a special circumstance that one crop population protects another by acting as a filter to delay the advance of disease.

3. **Spatial deployment**. In a situation where virulent pathogens are spatially localized, a cultivar diversification strategy whereby the fields of different farmers are planted to different cultivars , containing two or more virulent genes, would slow down disease epidemics.

4. **Multiline deployment**. A multiline consisting of genotypes carrying different major genes would also put a damping effect on epidemics just like in the case of spatial deployment.

5. **Mixture deployment**. A mixture of distinct cultivars with complementary vertical resistance genes can be deployed.

6. Combining vertical and horizontal resistance

- It is tempting to think that combining vertical resistance and horizontal resistance will provide the best of two worlds in the protection of plants.

- The erosion of horizontal resistance while breeding for race-specific vertical resistance is called the **vertifolia effect** (after the potato cultivar “Vertifolia”) in which the major gene is so strong that while the breeder focuses on vertical resistance, no evaluation and selection for horizontal resistance is possible, eventually leading to the loss of horizontal resistance.

- The vertifolia effect is not of universal occurrence.

- Some researchers have reported race-specific resistance in addition to high-level polygenic resistance to leaf rust in barley.

- To reduce the incidence of Vertifolia effect, some suggest that breeders select and discard susceptible plants in segregating populations, rather than selecting highly resistant genotypes.

- Also, others suggest to first breed for a high level of horizontal resistance in a genotype then cross it with one that has high vertical resistance.

7. Role of wild germplasm in resistance breeding

- The success and effectiveness of introgression of disease-resistance genes into crop species from wild relatives varies by crop.
- The resistance to the devastating late blight of potato was found in a wild species.
- Similarly, resistance to the root knot nematode in peanut was obtained from three wild species.
- A wild relative of rice, *Oryza nivara*, growing in the wild in Uttar Pradesh was found to have one single gene for resistance to the grassy stunt virus, a disease that devastated the crop in South and South East Asia in the 1970s.

8. Applications of biotechnology in resistance breeding

A. **Tissue culture**:

- Meristem tip culture can be used to produce virus free plants.

- Haploid production has been used for introduction of disease resistance into the cultivars.

eg: Resistance to barley YMV has been introduced into susceptible breeding lines by haploid breeding,

Medium late maturing rice variety Hwacheongbyeo derived from anther culture showed resistance to brown plant hopper.

- Somatic Hybridization : disease resistance genes like potato leaf roll virus , leaf blight, have been transferred to *Solanum tuberosum* from other species.

- Somaclonal variation: resistance to *Phytophthora infestans* in potato somaclones.

B. TRANSGENICS

Table 25.1: A list of few transgenic plants conferring resistance against insects

Crop	Gene transferred	Insect(s) controlled
Tobacco	Bt from *B. thuringiensis*	*Manduca Sexta*
	Truncated *cryI* from *B. thuringiensis*	*M. Sexta*
	Bt	*M. Sexta*
	CpTI	*Heliothis armigera*
	CpTI	*M. Sexta*
	Insecticidal protein from *Streptomyces*	Boll weevil *(Spodoptera litura)*
	Sweet potato trypsin inhibitor gene	*Spodoptera litura*
Tomato	*Bt*	*Heliothis armigera*
Potato	*cry III* from *B. thuringiensis*	Colorado potato beetle *(Leptinotarsa decenlineata)*
	Modified *cry III* gene	*L. decenlineata*
	Snowdrop lectin (GNA)	Tomato moth *(Lacanobia oleracea)*
Cotton	*Bt*	*Spodoptera*
	Bt	Cotton boll worm
Pea	*αAI* from bean	Bruchus beetle
	αAI from bean	Pea weevil
Rice	*Bt*	-
	Bt cryI gene of *B. thuringiensis*	Striped stem borer
	Corn cysteine gene	Coleopteran *(Sitophilus zeamis)*
Maize	*Bt cry II*	European corn borer
Sugarcane	*Bt cry I*	Sugarcane borer *(Diatracea saccharis)*

Table 25.3: Virus resistant transgenic plants generated in various crops for pathogen derived resistance

Crop	Transgene	Transgene mode of action	Virus	Reference
Potato	Replicase protein	Competition for enzyme	PVY	Audy *et al.* (1994)
Pea	Replicase protein	Competition for enzyme	PSbMV	Jones *et al.* (1998)
Rice	Replicase protein	Competition for enzyme	RYMV	Pinto *et al.* (1999)
Tobacco	Movement protein	Interference with transport	TMV	Cooper *et al.* (1995)
Potato	Movement protein	Interference with transport	PLRV, PVX, PVY	Tacke *et al.* (1996)
Tobacco	Transport protein	Interference with transport	TEV	Cronin *et al.* (1995)
Potato	Viral protease	Polyprotein processing	PVY	Vardi *et al.* (1993)
Potato	Antisense RNA	Blocks viral RNA and prevents translation	PLRV	Kawchuk *et al.* (1991)
Tomato	Ribozyme	Cleaves viral RNA	CEVd	Atkins *et al.* (1995)
Tobacco	Satellite RNA	Competes for capsids	CMV	Harrison *et al.* (1987)
Potato	Antiviral protein ribonuclease	Degrades ds-RNA (viroids)	PSTV	Sano *et al.* (1997)
Tobacco	Pokeweed antiviral protein	Inhibits rRNA of 60S subunit	TMV, PVX	Wang *et al.* (1998)
Tobacco	2′,5′ oligoadenylate antiviral protein	Degrades ds-RNA	CMV	Ogawa *et al.* (1996)

Table 25.4: Transgenic plants generated in various crops for resistance to fungal and bacterial diseases

Crop	Gene transferred	Controlled pathogen
PR proteins		
Tobacco	Bacterial chitinase from *Serratia marcescens*	*Alternaria longipes*
	Bean chitinase gene	*Rhizoctonia solani*
	PR-1-a gene	*Peronospora tabacina* Phytophthora *parasitica* var. *nicotianae*
	Chitinase	*Sclerotinia sclerotiorum*
	Chitinase	*Rhizoctonia solani*
	Chitinase and 1,3-β glucanase	*Cercospora nicotinae*
Tomato	Chitinase and 1,3-β glucanase	*Fusarium oxysporum lycopersici*
Brassica napus	Chitinase	*Rhizoctonia solani*
Brassica napus var. *oleifera*	Chitinase	*Cylindrosporium concentricum*, Phoma lingam, *Sclerotinia sclerotiorum*
Rice	Chitinase	*Rhizoctonia solani*
Carrot	Chitinase and 1,3-β glucanase	*Alternaria dauci*, *Alternaria radicina*, Cercospora *carotae*, *Erysiphe heraclei*
Potato	PR5	*Phytopthora infestans*
Potato	1,3-β glucanase	*Phytopthora infestans*
Kiwi fruit	1,3-β glucanase	*Botrytis cinerea*
Anti-microbial proteins		
Tobacco	Barley RIP (ribosome inactivating protein)	*Rhizoctonia solani*
Tomato	Prohevein from *Hevea brassiliensis*	*Trichoderma hamatum*
Tobacco	Defensin - Rs AFP2 from radish	*Alternaria longipes*
Tobacco	Barley α thionin gene	*Pseudomonas syringae* pv. tabaci; *P. syringae* pv. *syringae*
Tobacco	Cecropin	*P. syringae* pv. *tabaci*
Rice	Cecropin	Bacterial pathogen
Potato	Bacteriophage T4 lysozyme	*Erwinia carotovora* subsp. *Atroseptia*
Tobacco	Hen egg white lysozyme (HEWL)	*Botrytis cinerea*, *Verticillium albo-atrum*, *Rhizoctonia solanum*
Tobacco	Lysozyme from human being	*Pseudomonas syringae* pv. *tabaci*, *Erysiphe cichoracearum*
Potato	H_2O_2 gene for glucose oxidase	*Verticillium dahlea*, *Phytophthora*; *Erwinia carotovora*
Tobacco	Cryptogein from *Phytophthora cryptogea*	*Erysiphe cichoracearum*, *Botrytis cinerea*
Phytoalexins		
Tobacco	Stilbene synthase	*Botrytis cinerea*
Brassica napus	Stilbene synthase	—
Rice	Stilbene synthase	*Pyricularia oryzae*

C. Marker Assisted Selection

There are five main considerations for the use of DNA markers in MAS:

1. Reliability. Markers should be tightly linked to target loci, preferably less than 5 cM genetic distance. The use of flanking markers or intragenic markers will greatly increase the reliability of the markers to predict phenotype.

2. DNA quantity and quality. Some marker techniques require large amounts and high quality of DNA, which may sometimes be difficult to obtain in practice, and this adds to the cost of the procedures.

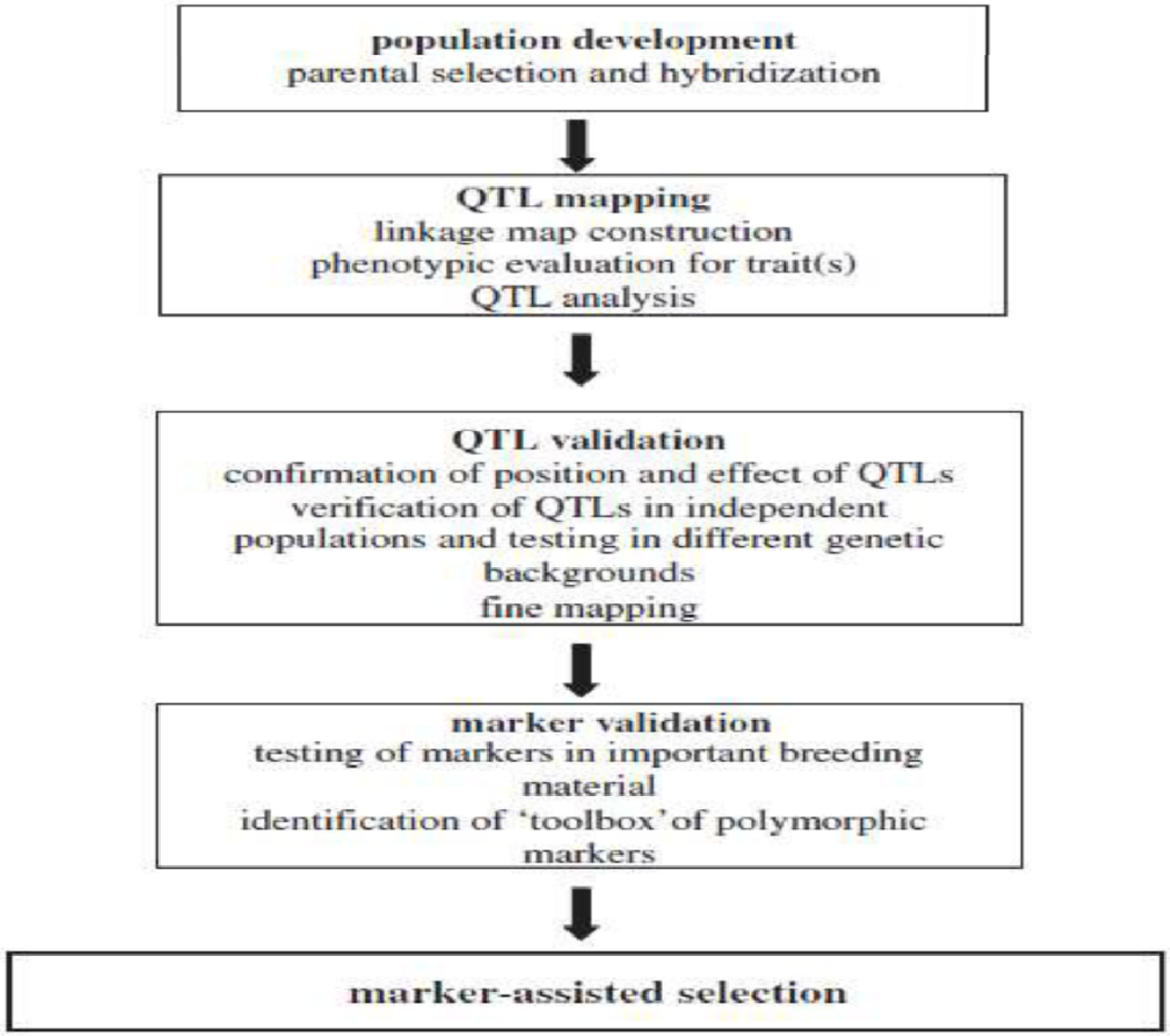
3. Technical procedure. The level of simplicity and the time required for the technique are critical considerations. High-throughput simple and quick methods are highly desirable.

4. Level of polymorphism. Ideally, the marker should be highly polymorphic in breeding material (i.e. it should discriminate between different genotypes), especially in core breeding material.

5. Cost. The marker assay must be cost-effective in order for MAS to be feasible.

QTL MAPPING AND MAS

- The detection of genes or QTLs controlling traits is possible due to genetic linkage analysis, which is based on the principle of genetic recombination during meiosis.
- Construction of linkage maps composed of genetic markers for a specific population. Segregating populations such as F2, F3 or backcross (BC) populations are frequently used.
- Using statistical methods such as single-marker analysis or interval mapping to detect associations between DNA markers and phenotypic data, genes or QTLs can be detected in relation to a linkage map.
- Once tightly linked markers that reliably predict a trait phenotype have been identified, they may be used for MAS.


population development
parental selection and hybridization
QTL mapping
linkage map construction
phenotypic evaluation for trait(s)
QTL analysis
QTL validation
confirmation of position and effect of QTLs
verification of QTLs in independent populations and testing in different genetic backgrounds
fine mapping
marker validation
testing of markers in important breeding material
identification of 'toolbox' of polymorphic markers
marker-assisted selection

Advantages of MAS

- It may be simpler than phenotypic screening, which can save time, resources and effort.
- Selection can be carried out at the seedling stage. This may be useful for many traits, but especially for traits that are expressed at later developmental stages. Therefore, undesirable plant genotypes can be quickly eliminated.
- With MAS, individual plants can be selected based on their genotype. For most traits, homozygous and heterozygous plants cannot be distinguished by conventional phenotypic screening.

MARKER-ASSISTED BACKCROSSING

- Three general levels of marker-assisted backcrossing (MAB) can be described.
- In the first level, markers can be used in screening for the target gene or QTL.
- This is referred to as 'foreground selection' .
- It can also be used to select for reproductive-stage traits in the seedling stage, allowing the best plants to be identified for backcrossing.
- Furthermore, recessive alleles can be selected, which is difficult to do using conventional methods

- The second level involves selecting BC progeny with the target gene and recombination events between the target locus and linked flanking 'recombinant selection'.

- The purpose of recombinant selection is to reduce the size of the donor chromosome segment containing the target locus (i.e. size of the introgression).

- By using markers that flank a target gene (e.g. less than 5 cM on either side), linkage drag can be minimized.

- The third level of MAB involves selecting BC progeny with the greatest proportion of recurrent parent (RP) genome, using markers that are unlinked to the target 'background selection'.

- Background selection refers to the use of tightly linked flanking markers for recombinant selection and unlinked markers to select for the RP .

- Background markers are markers that are unlinked to the target gene/QTL on all other chromosomes, in other words, markers that can be used to select against the donor genome.

- This is extremely useful because the RP recovery can be greatly accelerated.

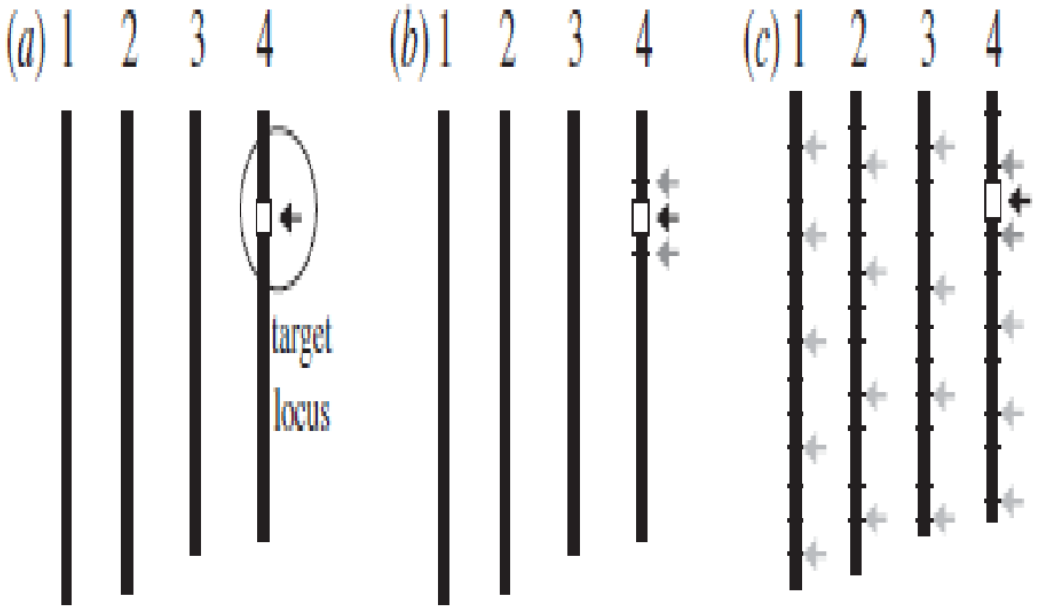


Figure 3. Levels of selection during marker-assisted backcrossing. A hypothetical target locus is indicated on chromosome 4. (*a*) Foreground selection, (*b*) recombinant selection and (*c*) background selection.

species	trait(s)	gene/QTLs	foreground selection	background selection	reference
barley	barley yellow dwarf virus	*Yd2*	STS	not performed	Jefferies *et al.* (2003)
barley	leaf rust	*Rphq6*	AFLP	AFLP	van Berloo *et al.* (2001)
barley	stripe rust	QTLs on 4H and 5H	RFLP	not performed	Toojinda *et al.* (1998)
barley	yield	QTLs on 2HL and 3HL	RFLP	RFLP	Schmierer *et al.* (2004)
maize	corn borer resistance	QTLs on chromosomes 7, 9 and 10	RFLP	RFLP	Willcox *et al.* (2002)
maize	earliness and yield	QTLs on chromosomes 5, 8 and 10	RFLP	RFLP	Bouchez *et al.* (2002)
rice	bacterial blight	*Xa21*	STS[a]	RFLP	Chen *et al.* (2000)
rice	bacterial blight	*Xa21*	STS[a]	AFLP	Chen *et al.* (2001)
rice	bacterial blight	*xa5*, *xa13* and *Xa21*	STS, CAPS	not performed	Sanchez *et al.* (2000)
rice	bacterial blight	*xa5, xa13 and Xa21*	STS	not performed	Singh *et al.* (2001)
rice	bacterial blight + quality	*xa13, Xa21*	STS and SSR	AFLP	Joseph *et al.* (2004)
rice	blast	*Pi1*	SSR	ISSR[b]	Liu *et al.* (2003)
rice	deep roots	QTLs on chromosomes 1, 2, 7 and 9	RFLP and SSR	SSR	Shen *et al.* (2001)
rice	quality	waxy	RFLP[a]	AFLP	Zhou *et al.* (2003*a*)
rice	root traits and aroma	QTLs on chromosomes 2, 7, 8, 9 and 11	RFLP and SSR	RFLP and SSR	Steele *et al.* (2006)
rice	submergence tolerance	*Sub1* QTL	phenotyping and SSR[a]	SSR	Mackill *et al.* (2006)
rice	submergence tolerance, disease resistance, quality	*Subchr9* QTL, Xa21, Bph and blast QTLs and quality loci	SSR and STS	not performed	Toojinda *et al.* (2005)
wheat	powdery mildew	22 *Pm* genes	phenotyping	AFLP	Zhou *et al.* (2005)

[a] Indicates recombinant selection performed to minimize linkage drag around target locus.
[b] ISSR and inter SSRs.

LOW IMPACT OF MARKER-ASSISTED SELECTION

a) Still at the early stages of DNA marker technology development.

b) Reliability and accuracy of quantitative trait loci mapping studies.

c) Insufficient linkage between marker and gene/ quantitative trait locus.

d) Quantitative trait loci and environment effects.

e) High cost of marker-assisted selection.

f) 'Application gap' between research laboratories and plant breeding institutes.

g) 'Knowledge gap' among molecular biologists, plant breeders and other disciplines