PAPER - IX GENETICS, CYTOGENETICS AND PLANT BREEDING
UNIT- 1

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Mendel's Experiments

Mendel experimented on a pea plant and considered 7 main contrasting traits in the plants. Then, he conducted both the experiments to determine the aforementioned inheritance laws. A brief explanation of the two experiments is given below.

Monohybrid Cross

In this experiment, Mendel took two pea plants of opposite traits (one short and one tall) and crossed them. He found the first generation offsprings were tall and called it F1 progeny. Then he crossed F1 progeny and obtained both tall and short plants in the ratio 3:1.

Mendel even conducted this experiment with other contrasting traits like green peas with yellow peas, round with wrinkled, etc. In all the cases, he found that the results were similar. From this, he formulated the **laws of Segregation and Dominance.**

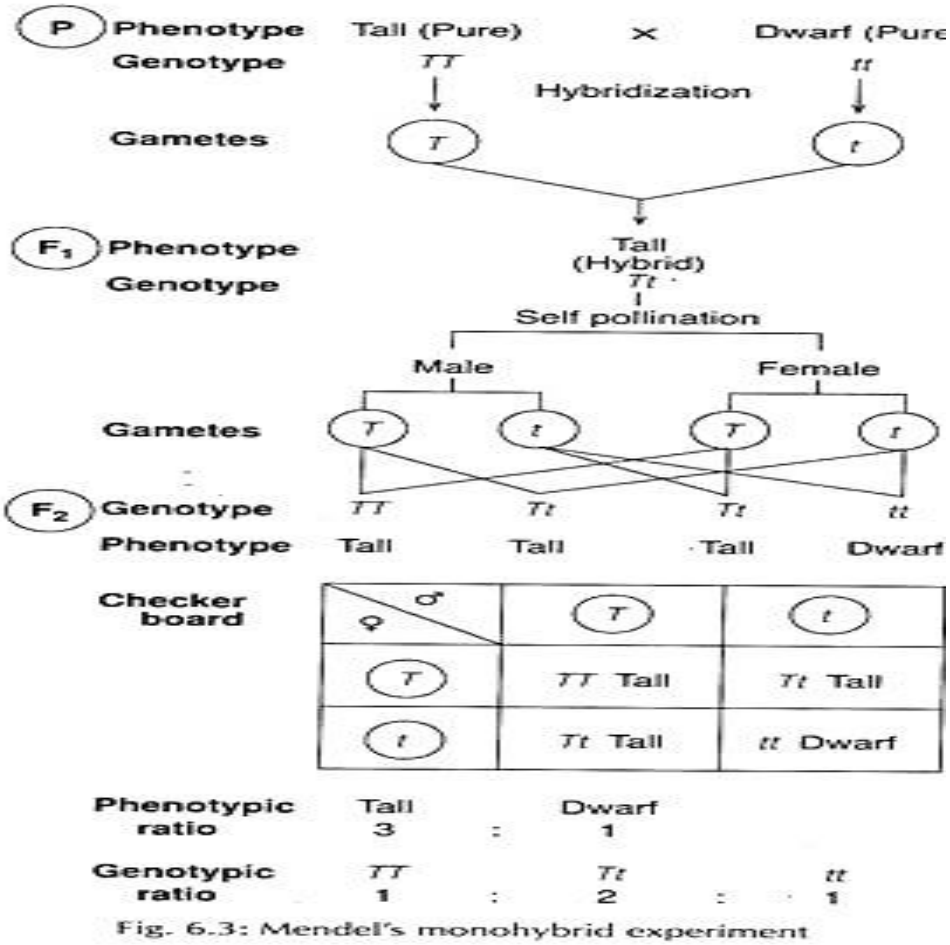
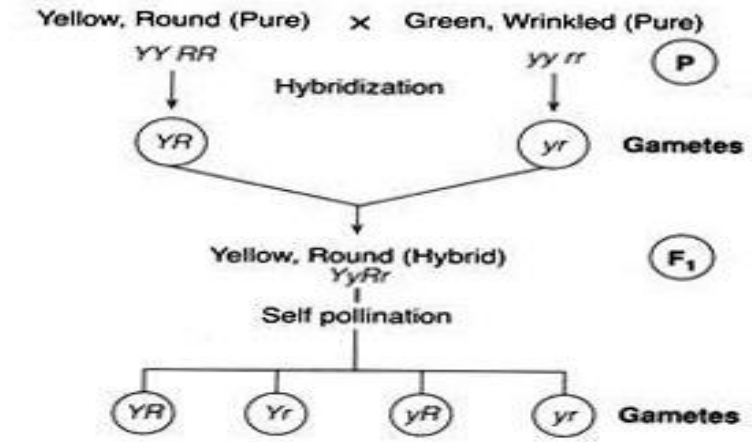


Fig. 6.3: Mendel's monohybrid experiment

Dihybrid Cross

In a dihybrid cross experiment, Mendel considered two traits, each having two alleles. He crossed wrinkled-green seed and round-yellow seeds and observed that all the first generation progeny (F1 progeny) were round-yellow. This meant that dominant traits were the round shape and yellow colour.

He then self-pollinated the F1 progeny and obtained 4 different traits wrinkled-yellow, round-yellow, wrinkled-green seeds and round-green in the ratio 9:3:3:1.



♂ / ♀	YR	Yr	yR	yr
YR	YYRR	YYRr	YyRR	YyRr
Yr	YYRr	YYrr	YyRr	Yyrr
yR	YyRR	YyRr	yyRR	yyRr
yr	YyRr	Yyrr	yyRr	yyrr

F2

Phenotype	Genotype	Genotypic ratio	Phenotypic ratio
Yellow, Round	YYRR	1	9
	YYRr	2	
	YyRR	2	
	YyRr	4	
Yellow, Wrinkled	YYrr	1	3
	Yyrr	2	
Green, Round	yyRR	1	3
	yyRr	2	
Green, Wrinkled	yyrr	1	1

Fig. 6.4: Mendel's dihybrid experiment

After conducting for other traits, the results were found to be similar. From this experiment, Mendel formulated his second law of inheritance i.e law of Independent Assortment.

Mendel's laws

The two experiments lead to the formulation of Mendel's laws known as laws of inheritance which are:

Law of Dominance

This is also called Mendel's first law of inheritance. According to the law of dominance, hybrid offsprings will only inherit the dominant trait in the phenotype. The alleles that are suppressed are called as the recessive traits while the alleles that determine the trait are known as the dormant traits.

Law of Segregation

The law of segregation states that during the production of gametes, two copies of each hereditary factor segregate so that offspring acquire one factor from each parent. In other words, allele (alternative form of the gene) pairs segregate during the formation of gamete and re-unite randomly during fertilization. This is also known as Mendel's third law of inheritance.

Law of Independent Assortment

Also known as Mendel's second law of inheritance, the law of independent assortment states that a pair of trait segregates independently of another pair during gamete formation. As the individual heredity factors assort independently, different traits get equal opportunity to occur together.

Back Cross & Test Cross:

Crossing of F_1 organism with either of the parents is called back cross (Fig. 6.6). When an organism is crossed with other organism having recessive phenotypic trait (recessive homozygous genotype) is called test cross. This is called test cross because it helps to test the genotype of an organism. In monohybrid cross, tall pea plant of F_2 may be homozygous (TT) or heterozygous (Tt). Test cross results confirm it (Fig. 6.7).

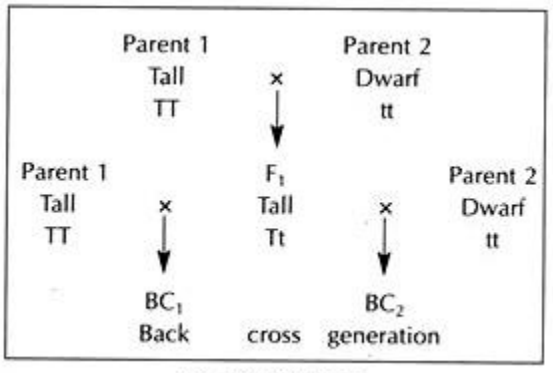


Fig. 6.6: Back cross

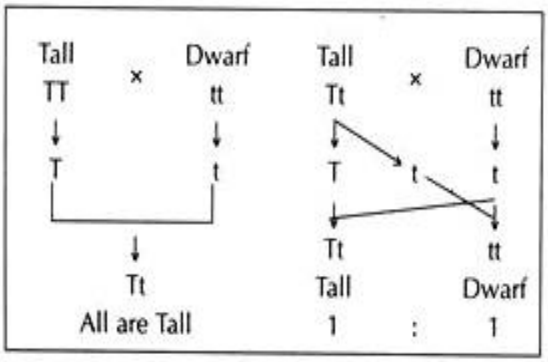


Fig. 6.7: Test cross to confirm the genotype of tall plant.

In monohybrid test cross, the ratio is 1:1, while in di-hybrid test cross, the expected ratio is 1 : 1 : 1 : 1 (Fig. 6.8).

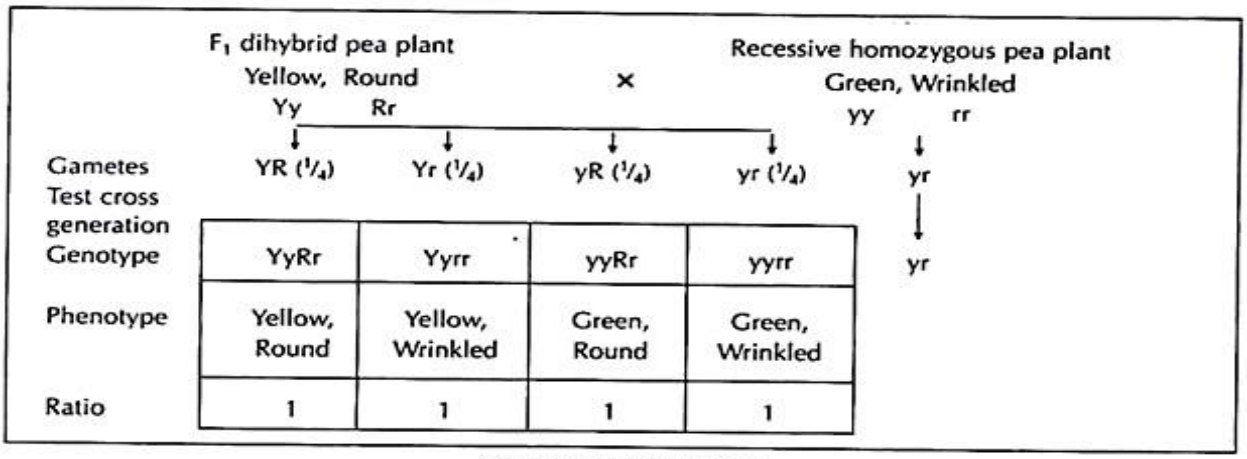


Fig. 6.8: Dihybrid test cross

Gene Interaction:

Mendelian genetics does not explain all kinds of inheritance for which the phenotypic ratios in some cases are different from Mendelian ratios (3:1 for monohybrid, 9:3:3:1 for di-hybrid in F2). This is because sometimes a particular allele may be partially or equally dom-inant to the other or due to existence of more than two alleles or due to lethal alleles. These kinds of genetic interactions between the alleles of a single gene are referred to as allelic or intra- allelic interactions.

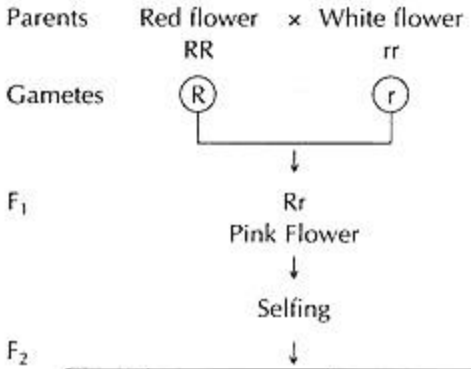
Allelic Gene Interactions:

The alleles of the same gene interact in such a way to produce the new character. The kinds of genetic interactions between the alleles of a single gene are referred to as allelic or intra- allelic interactions.

Incomplete Dominance or Blending Inheritance (1:2:1):

A dominant allele may not completely suppress other allele; hence a heterozygote is phenotypically distinguishable (intermediate phenotype) from either homozygotes.

In snap-dragon and Mirabilis jalapa, the cross between pure bred red-flowered and white-flowered plants yields pink-flowered F1 hybrid plants (deviation from parental phenotypes), i.e., intermediate of the two parents. When F1 plants are self-fertilized, the F2 progeny shows three classes of plants in the ratio 1 red: 2 pink: 1 white instead of 3:1 (Fig. 7.1).



♂ \ ♀	R	r
R	RR Red	Rr Pink
r	Rr Pink	rr White

Genotype	Phenotype	Ratio
RR	Red	1
Rr	Pink	2
rr	White	1

Fig. 7.1: Inheritance of flower colour in snapdragon

Therefore, a F1 di-hybrid showing incomplete dominance for both the characters will segregate in F2 into (1 :2 : 1) X (1 :2 : 1) = 1 :2 : 1 : 2 : 4 : 2:1 : 2 : 1. And a F1 di-hybrid showing complete dominance for one trait and incomplete domi-nance of another trait will segregate in F2 into (3:1) x (1 :2:1) = 3:6:3:1 :2:1.

Co-dominance:

Here both the alleles of a gene express themselves in the heterozygotes. Phenotypes of both the parents appear in F1 hybrid rather than the intermediate phenotype. In human, MN blood group is controlled by a single gene.

Only two alleles exist, M and N. Father with N blood group (genotype NN) and mother with M blood group (genotype MM) will have children with MN blood group (genotype MN). Both phenotypes are identifiable in the hybrid. F2 segregates in the ratio 1M blood group: 2 MN blood group: 1 N blood group.

Lethal Factor (2:1):

The genes which cause the death of the indi-vidual carrying it, is called lethal factor. Recessive lethals are expressed only when they are in homozygous state and the heterozygotes remain unaffected. There are genes that have a dominant phenotypic effect but are reces-sive lethal, e.g.,

gene for yellow coat colour in mice. But many genes are recessive both in their phenotypic as well as lethal effects, e.g., gene producing albino seedlings in barley (Fig. 7.2).

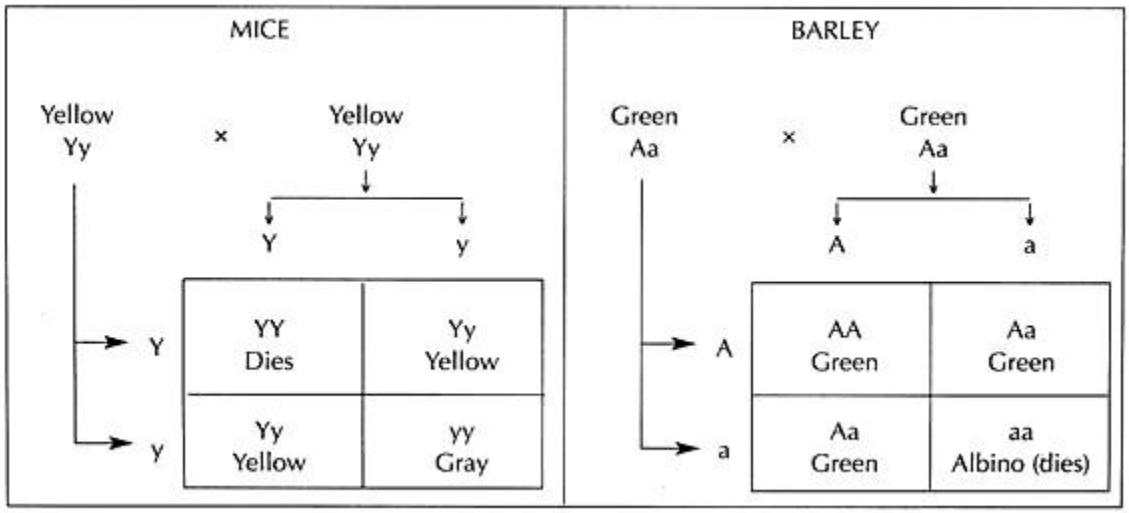


Fig. 7.2: Inheritance of lethal gene in mice and barley

Dominant lethals are lost from the popula-tion because they cause death of the organism even in a heterozygous state, e.g., epiloia gene in human beings. **Conditional lethals** require a specific condi-tion for their lethal action, e.g., temperature sensitive mutant of barley (lethal effect at low temperature). **Balanced lethals** are all heterozygous for the lethal genes; both dominant and recessive homozygotes will die, e.g., balanced lethal sys-tem in Oenothera. **Gametic lethals** make the gametes incapable of fertilization, e.g., segregation distorter gene in male Drosophila. **Semi-lethal** genes do not cause the death of all the individuals, e.g., xentha mutants in some plants.

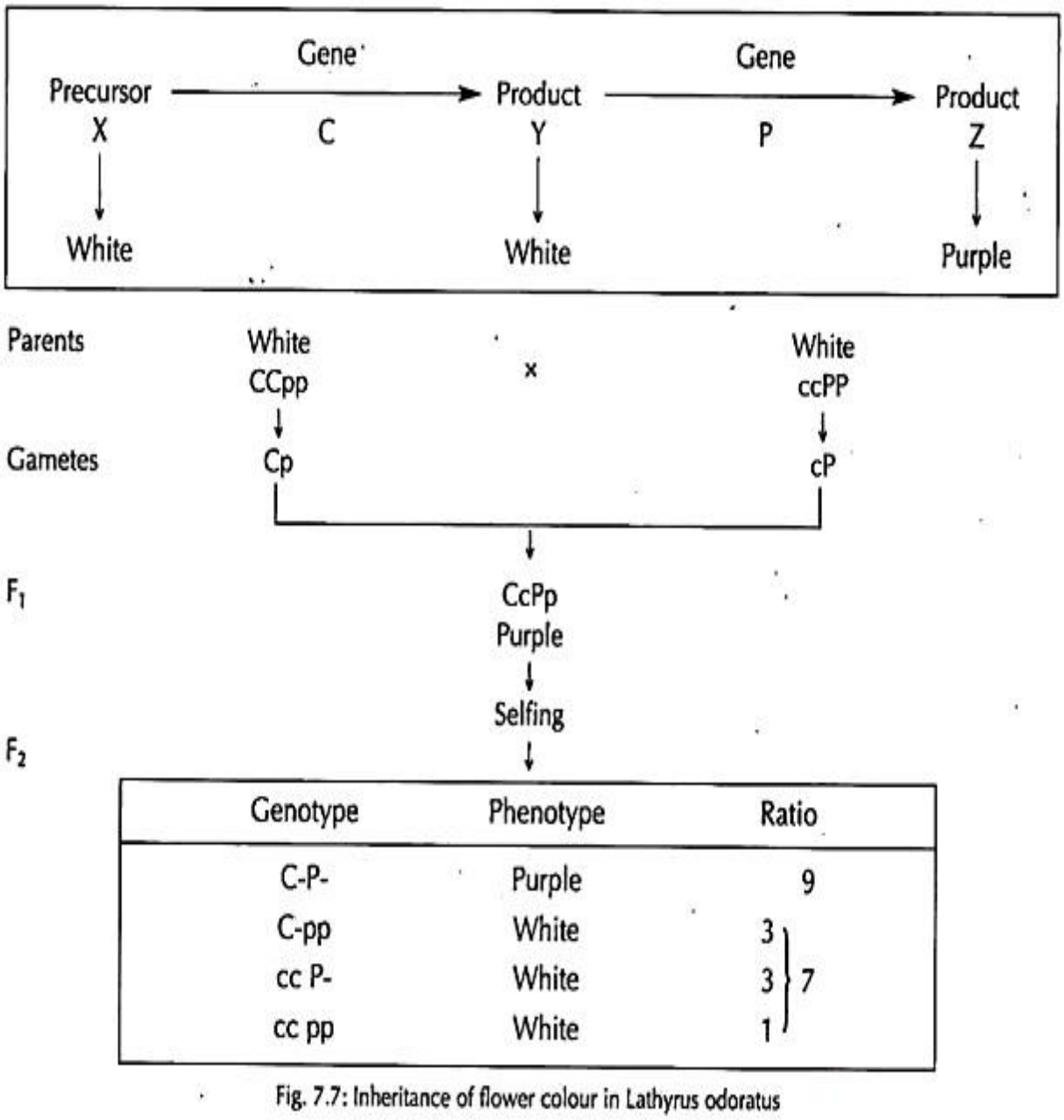
Non Allelic Gene Interactions:

Non-allelic or inter-allelic interactions also occur where the development of single character is due to two or more genes affecting the expression of each other in various ways.

Complementary Factor (9:7):

Certain characters are produced by the inter-action between two or more genes occupying different loci inherited from different parents. These genes are complementary to one another, i.e., if present alone they remain unexpressed, only when they are brought together through suitable crossing will express.

In sweet pea (Lathyrus odoratus), both the genes C and P are required to synthesize anthocyanin pigment causing purple colour. But absence of any one cannot produce anthocyanin causing white flower. So C and P are complementary to each other for anthocyanin formation (Fig. 7.7).



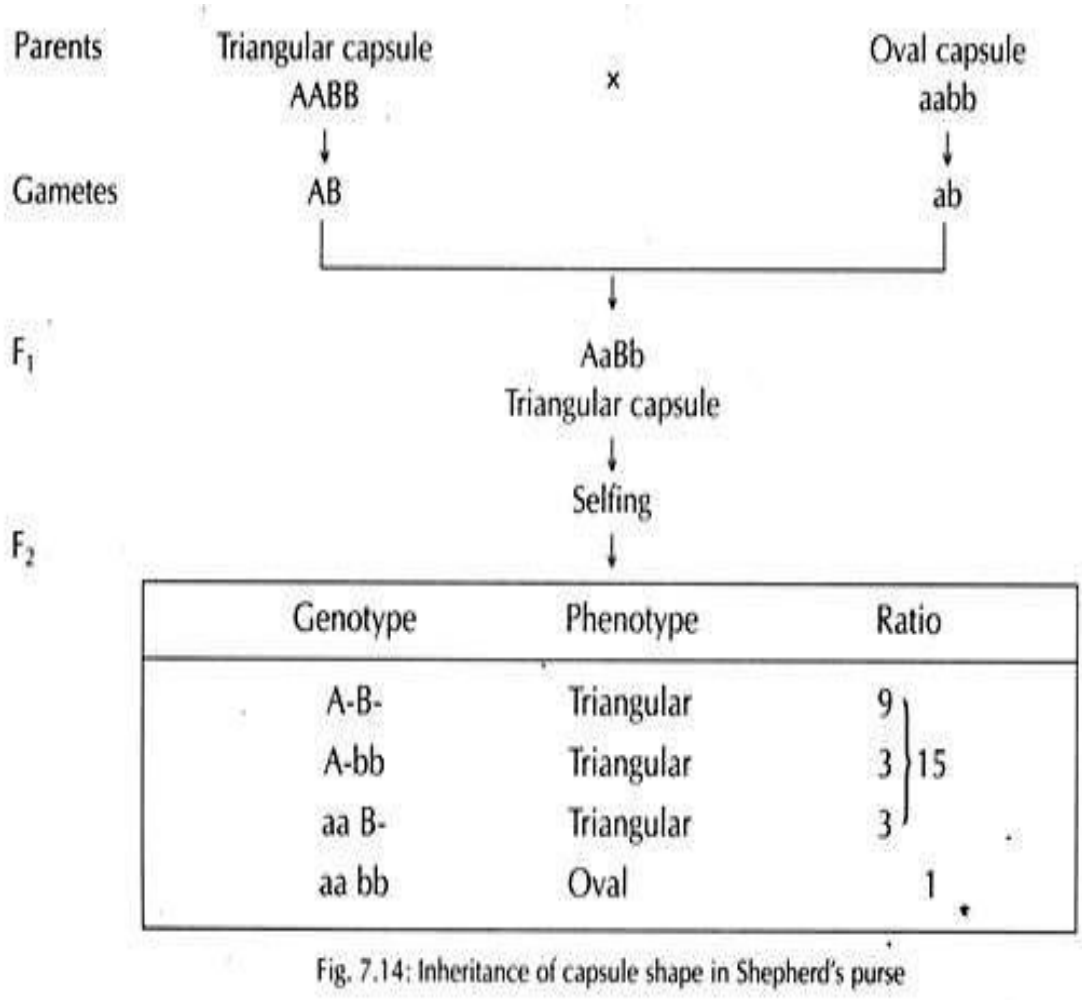
Genotype	Phenotype	Ratio
C-P-	Purple	9
C-pp	White	3
cc P-	White	3 } 7
cc pp	White	1

Fig. 7.7: Inheritance of flower colour in Lathyrus odoratus

Involvement of more than two complemen-tary genes is possible, e.g., three complementary genes governing aleurone colour in maize.

Duplicate Gene (15:1):

Sometimes a character is controlled by two non-allelic genes whose dominant alleles produce the same phenotype whether they are alone or together. In Shepherd's purse (Capsella bursa-pastoris), the presence of either gene A or gene B or both results in triangular capsules; when both these genes are in reces-sive forms, the oval capsules produced (Fig. 7.14).



Genotype	Phenotype	Ratio
A-B-	Triangular	9 | 15
A-bb	Triangular	3 |
aa B-	Triangular	3 |
aa bb	Oval	1

Fig. 7.14: Inheritance of capsule shape in Shepherd's purse

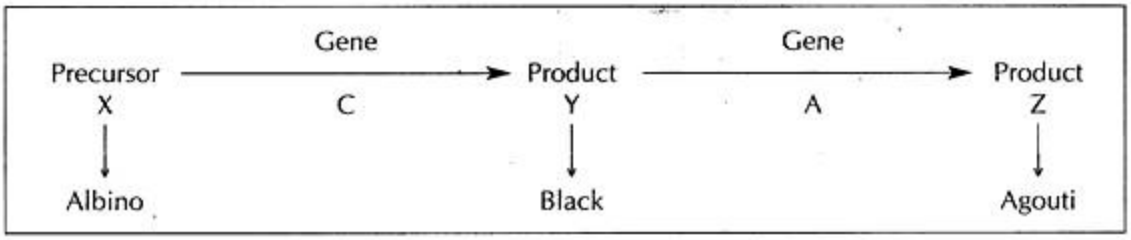
Epistasis:

When a gene or gene pair masks or prevents the expression of other non-allelic gene, called epistasis. The gene which produces the effect called epistatic gene and the gene whose expres-sion is suppressed called hypostatic gene.

Recessive Epistasis or Supplementary gene interaction (9:3:4):

In this case, homozygous recessive condi-tion of a gene determines the phenotype irres-pective of the alleles of other gene pairs, i.e., recessive allele hides the effect of the other gene. The coat colour of mice is controlled by two pairs of genes.

Dominant gene C produces black colour, absence of it causes albino. Gene A pro-duces agouti colour in presence of C, but cannot express in absence of it (with cc) resulting in albino. Thus recessive allele c (cc) is epistatic to dominant allele A (Fig. 7.8).



Parents: Black CCaa × Albino ccAA

Gametes: Ca, cA

F_1: CcAa Agouti × CcAa Agouti

F_2:

Genotype	Phenotype	Ratio
C-A-	Agouti	9
C-aa	Black	3
cc A-	Albino	3 } 4
cc aa	Albino	1 }

Fig. 7.8: Inheritance of coat colour in mice

The grain colour in maize is governed by two genes — R (red) and Pr (purple). The reces-sive allele rr is epistatic to gene Pr (Fig. 7.9).

Parents: Red RRprpr × White rrPrPr

Gametes: Rpr, rPr

F_1: RrPrpr Purple

Selfing

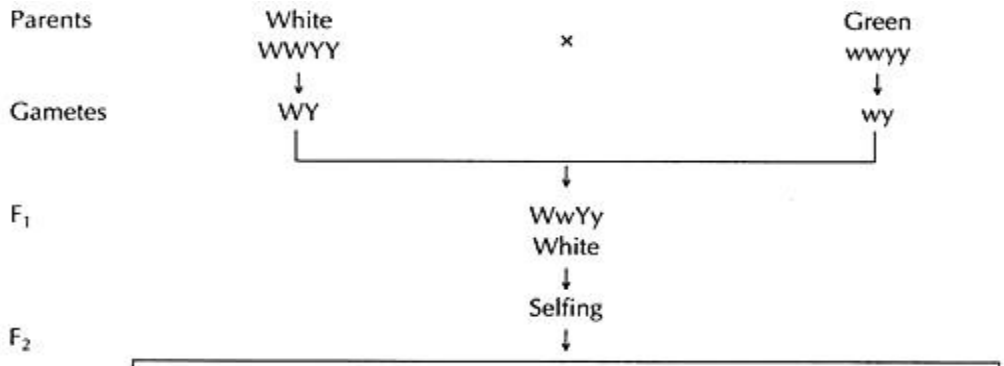
F_2:

Genotype	Phenotype	Ratio
R-Pr-	Purple	9
R-prpr	Red	3
rr Pr-	White	3 } 4
rr prpr	White	1 }

Fig. 7.9: Inheritance of grain colour in maize

Dominant Epistasis (12:3:1):

Sometimes a dominant gene does not allow the expression of other non-allelic gene called dominant epistasis. In summer squash, the fruit colour is governed by two genes. The dominant gene W for white colour, suppresses the expres-sion of the gene Y which controls yellow colour. So, yellow colour appears only in absence of W. Thus W is epistatic to Y. In absence of both W and Y, green colour develops (Fig. 7.10).



Genotype	Phenotype	Ratio
W-Y-	White	9 } 12
W-yy	White	3 }
ww Y-	Yellow	3
ww yy	Green	1

Fig. 7.10: Inheritance of fruit colour in summer squash

Multiple alleles in Corn:

In Maize, three of the four kernel colors-yellow, red and purple-are produced by pigments synthesized from one of two metabolic pathways, the carotenoid (yellow pigments) or the anthocyanin (red and purple pigments) pathway. White, the fourth color, results from the lack of pigments produced from either pathway. The synthesis of each pigment requires numerous genes, including those that are structural (coding for enzymes) and regulatory (called transcription activators and coding for proteins that control the transcription of structural genes).

Table 1. Summary of the four genes, *Pr1, R1, C1* and *Y1*, influencing kernel color.

Biological Supply[1]		*Maize Database*[2]		*Biochemical*	*Metabolic*			*Chromosomal*
Gene Name	*Symbol*	*Gene Name*	*Symbol*	*Phenotype*	*Pathway*	*Protein Role*	*Protein Name*	*Location*[3]
Color gene	*Pr*	Red	*Pr1*	Purple aleurone[4]	Anthocyanin	Structural	Flavonoid 3′-hydroxylase	5L
	pr	Aleurone1	*pr1*	Red aleurone				
Color gene	*R*	Colored1	*R1 (R-r)*	Colored aleurone[4]	Anthocyanin	Regulatory	Transcription factor	10L
	r		*r1 (r-r)*	Colorless aleurone[5]				
Color gene	*C*[1]	Colored	*C1-I*	Colorless aleurone[5]	Anthocyanin	Regulatory	Transcription factor	9S
	C	Aleurone1	*C1*	Colored aleurone[4]				
	c		*c1*	Colorless aleurone[5]				
Color gene	*Y*	White1	*Y1*	Yellow endosperm[6]	Carotenoid	Structural	Phytoene synthetase	6L
	y		*y1*	White endosperm[6]				

In monohybrid cross, purple and yellow kernels-provides an introduction to explain the influence of genetic background on phenotype (Table 3). For example, the phenotypic ratio of 3 purple: 1 yellow for the segregation of Ri depends on the presence of Prl, Cl and Y1 in addition to RI. Since the kernels are homozygous for the dominant alleles for the other two genes (Prl and Cl) in the anthocyanin pathway, the purple pigment is synthesized only when RI is present. Otherwise, the aleurone is colorless and the starchy endosperm's yellow pigment, which is present in all kernels including the purple ones, is observed. Since the yellow endosperm is present whether or not the anthocyanin is produced, the phenotypic ratio for this cross is expressed more precisely as 3 colored: 1 noncolored aleurone. Crosses with more than one gene pair, dihybrid and trihybrid, provide inheritance patterns that are challenging to explain. Because these four genes are nonlinked or located on different chromosomes, the phenotypic ratios illustrating gene interactions for the dihybrid crosses (e.g. 9:7, 9:3:4, 13:3 and 12:3:1) are variations of the 9:3:3:1 ratio and the trihybrid cross (9:3:3:1) represents a variation of the 27:9:9:9:3:3:3:1:1:1 ratio. The five dihybrid crosses listed in Table 3 include examples of both recessive and dominant epistasis as well as more complex patterns of duplicate recessive epistasis and dominant epistasis coupled with recessive epistasis. The trihybrid cross is a case of simple recessive epistasis.

Table 3. Phenotypic ratios of eight crosses illustrating inheritance of kernel color in corn including the genetic background and type of interaction.

Category of Cross	*F_2 Ratio*	*Segregating Alleles*	*Genetic Background*[1]	*Type of Interaction*
Monohybrid	3 purple 1 yellow	*R1* and *r1*	*Pr1Pr1/Y1Y1/C1C1*	None
	3 purple 1 yellow	*C1* and *c1*	*Pr1Pr1/Y1Y1/R1R1*	None
Dihybrid	9 red 7 white	*C1* and *c1* *R1* and *r1*	*pr1pr1/y1y1*	Duplicate recessive epistasis
	9 purple 3 red 4 white	*Pr1* and *pr1* *R1* and *r1*	*y1y1/C1C1*	Recessive epistasis
	9 yellow 3 purple 4 white	*C1-I* and *C1* *Y1* and *y1*	*Pr1Pr1/R1R1*	Dominant epistasis
	12 purple 3 yellow 1 white	*Y1* and *y1* *R1* and *r1*	*Pr1Pr1/C1C1*	Recessive epistasis
	13 yellow 3 purple	*C1-I* and *C1* *R1* and *r1*	*Pr1Pr1/Y1Y1*	Dominant & recessive epistasis
Trihybrid	9 purple 3 red 3 yellow 1 white	*C1* and *c1* *Pr1* and *pr1* *Y1* and *y1*	*R1R1*	Recessive epistasis

[1]The genetic background includes other genes that influence the phenotypic ratio.

Epistasis can be attributed to the blocking of the biochemical pathway for a particular pigment because enzymes critical to the pathway are either missing or nonfunctional. Each allele can be assigned a mechanism of action for its epistasis effect, either dominant or recessive. The two mutant alleles of Cl (Cl-I and cl) exhibit different types of epistasis yet produce the same phenotype (colorless aleurone). The Cl-I allele, illustrating dominant epistasis, is dominant to Cl because the Cl-I mutant protein competes with the Cl wildtype protein for the regulatory sites of structural genes associated with the pathway, thereby blocking synthesis of the corresponding enzymes required for pigment production synthesis (Paz-Ares et al. 1990). In contrast, the gene (cl) corresponding to recessive epistasis is not transcribed (Scheffler et al. 1994), and in heterozygotes (Clcl), one copy of the wildtype gene is sufficient for producing enough regulatory protein to produce the purple color.

Multiple alleles in Tobacco

In plants, multiple alleles have been reported in association with self-sterility or self-incompatibility. Self-sterility means that the pollen from a plant is unable to germinate on its own stigma and will not be able to bring about fertilization in the ovules of the same plant. **East (1925)** observed multiple alleles in ***Nicotiana*** which are responsible for self-incompatibility or self-sterility. The gene for self-incompatibility can be designated as S, which has allelic series S_1, S_2, S_3, S_4 and S_5 (Figure 3.17).

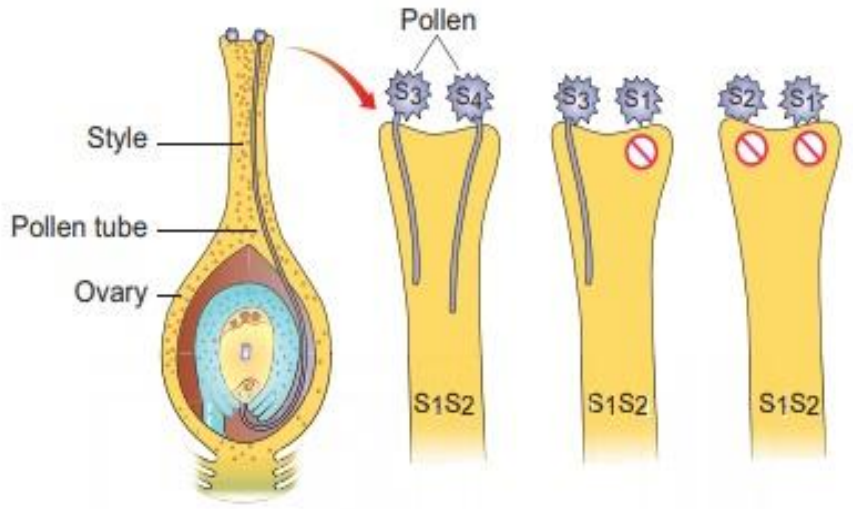


Figure: 3.17 The self-incompatibility in relation to its genotype in tobacco

The cross-fertilizing tobacco plants were not always homozygous as S_1S_1 or S_2S_2, but all plants were heterozygous as S_1S_2, S_3S_4, S_5S_6. When crosses were made between different S_1S_2 plants, the pollen tube did not develop normally. But effective pollen tube development was observed when crossing was made with other than S_1S_2 for example S_3S_4.

When crosses were made between seed parents with S_1S_2 and pollen parents with S_2S_3, two kinds of pollen tubes were distinguished. Pollen grains carrying S_2 were not effective, but the pollen grains carrying S_3 were capable of fertilization. Thus, from the cross $S_1S_2XS_3S_4$, all the pollens were effective and four kinds of progeny resulted: S_1S_3, S_1S_4, S_2S_3 and S_2S_4. Some combinations are showed in the table-3.5.

Female parent (Stigma spot)	Male parent (Pollen source)		
	S_1S_2	S_2S_3	S_3S_4
S_1S_2	Self Sterile	S_3S_2 S_3S_1	S_3S_1 S_3S_2 S_4S_1 S_4S_2
S_2S_3	S_1S_2 S_1S_3	Self Sterile	S_4S_2 S_4S_3
S_3S_4	S_1S_3 S_1S_4 S_2S_3 S_2S_4	S_2S_3 S_2S_4	Self Sterile

Table: 3.5. Different combinations of progeny in self-incompatibility

Multiple factors

The inheritance due to multiple factors or polygenes can be illustrated with the help of three examples : **(i)** kernel colour in wheat (studied by **H. Nilsson-Ehle). (ii)** Skin colour in Humans (studied by **C.B. Davenport)** and **(iii)** corolla length in *Nicotiana longiflora* (studied by E.M. East).

Kernel colour in wheat:

Kernel colour in wheat is a quantitative character and was studied by **H. Nilsson-Ehle** for the first time in 1908. It was argued that if one gene was considered or in other words, if the two parents differed due to one gene only, a 3 : 1 ratio for red and white kernels was obtained in F_2 generation. However, out of three red, one was as red as one of the parents and two were lighter and were comparable to F_1 individuals. This indicated that the dominant alleles had a cumulative effect. If 'K' stands for red colour and V for white, the two parents could be designated as *RR* and *rr*, the F_1 could be designated as *Rr* and F_2 would be obtained in 1*RR* : 2*Rr* : 1*rr* ratio. In these three classes, *RR* should be red, *Rr* should be intermediate in colour and *rr* should be white. In case there were two genes involved, a 15: 1 ratio (15 coloured : 1

white) would be obtained (Table 4.1). If different shades are taken into account, 1:4:6:4:1 ratio will be obtained, provided R_x and R_2 contribute equally to the colour (Fig. 4.1). However, it is known now that there are three genes involved in kernel colour in wheat. Obviously if the two parents differ in all three genes, in F_2 63: 1 or 1: 6: 15: 20: 15: 6: 1 ratio will be obtained (Fig. 4.2). By the study of kernel colour in wheat, **Nilsson-Ehle** reached the conclusion that the effect of each dominant allele was cumulative and hence forwarded his multiple factor hypothesis. The hypothesis states that for a given quantitative trait there could be several genes, which were independent in their segregation, but had cumulative effect on phenotype.

Table 4.1. **Genotypic and phenotypic ratios in F_2 obtained due to a cross $R_1R_1R_2R_2 \times r_1r_1r_2r_2$.**

Genotype	Genotypic ratio	Phenotype	Phenotypic ratio	
$R_1R_1R_2R_2$	1	red (like) P_1	1	coloured ($^{15}/_{16}$)
$R_1R_1R_2r_2$	2	darker than F_1 and lighter than P_1	4	
$R_1r_1R_2R_2$	2			
$R_1r_1R_2r_2$	4	like F_1 (intermediate between P_1 and P_2)	6	
$R_1R_1r_2r_2$	1			
$r_1r_1R_2R_2$	1			
$R_1r_1r_2r_2$	2	lighter than F_1	4	
$r_1r_1R_2r_2$	2			
$r_1r_1r_2r_2$	1	colourless	1	colourless ($^{1}/_{16}$)

Skin colour in Man:

It was idea of Davenport (1913) that the multiple factor hypotheses explains the mode of inheritance of skin colour in man. His assumption was that the Negroes differ from the whites in having 2 pairs of colour forming genes that do not show complete dominance. He carried on his studies in Jamaica and Bermuda where intermarriages between coloured and white people were very common.

A marriage between a Negro (P1P1P2P2) and a white (p1p1p2p2) results in children having intermediate shade and has only 2 colour forming genes (P1p1P2p2). These are mulattoes. When 2 mulattoes marry they may have children showing different degrees of colouration ranging from pure black to white.

In the F2 1/16 will be as dark as the Negro grandparent having 4 colour genes. The rest 14/16 will show intermediate shades depending upon the number of colour genes contained by them. But all these gradations could be seen only when a large number of children are born. In a small family the mulattoes parents will produce a completely black or white child.

Parents	Negro	×	White
	$P_1P_1P_2P_2$	×	$p_1p_1p_2p_2$
Gametes	P_1P_2	↓	p_1p_2
F_1		Mulattoes	
		$P_1p_1P_2p_2$	

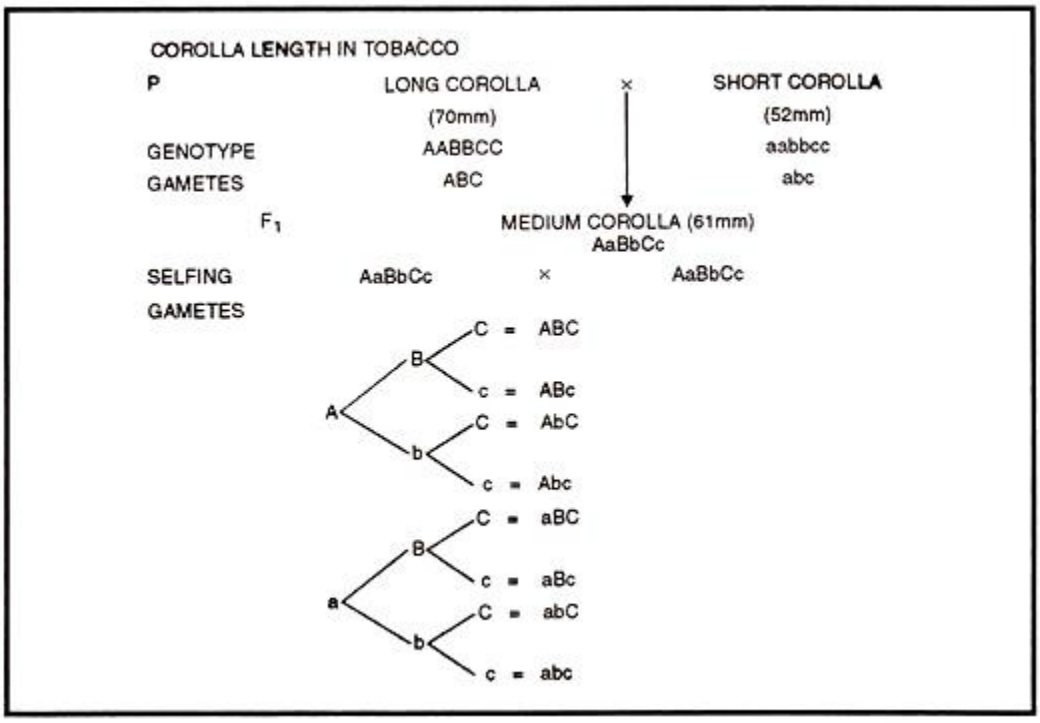
F_2	♂ gametes	P_1P_2	P_1p_2	p_1P_2	p_1p_2	
♀ gametes	P_1P_2	$P_1P_1P_2P_2$	$P_1P_1P_2p_2$	$P_1p_1P_2P_2$	$P_1P_1P_2p_2$	Summary :
	P_1p_2	$P_1P_1P_2p_2$	$P_1P_1p_2p_2$	$P_1p_1P_2p_2$	$P_1p_1p_2p_2$	15/16 Dark
	p_1P_2	$P_1p_1P_2P_2$	$P_1p_1P_2p_2$	$p_1p_1P_2P_2$	$p_1p_1P_2p_2$	1/16 White
	p_1p_2	$P_1p_1P_2p_2$	$P_1p_1p_2p_2$	$p_1p_1P_2p_2$	$p_1p_1p_2p_2$	

It has been proposed that the difference in the skin colour between Negros and whites is due to the presence of more than two pairs of colour forming genes bringing about a considerable variation in the skin colour. Gates has suggested for three where as Stern's for four, five or six pairs of genes. Other geneticists have estimated the colour genes number from two to twenty pairs but the exact number involved is till unknown.

Corolla length in tobacco (*Nicotiana*):

East (1916) reported his studies on the inheritance of corolla length in *Nicotiana longiflora*, a self-pollinated variety of tobacco. This character is governed by multiple genes. He crossed a variety, the corolla of which had an average length of 52 mm, to a variety, the corolla of which had an average length of 70 mm. Both these varieties had long been inbred and therefore were homozygous. The marked differences in corolla lengths were heritable pointing out that they are controlled by genes rather than environmental. East found that F1 was intermediate with mean corolla length of 61 mm.

In F2 a much larger variation for corolla length than F1 was observed. The variation was continuous as well. East raised 444 F2 plants and failed to get even a single plant like either of the parents. This pointed out that more than 4 pair of genes are involved in determining the length of corolla in *Nicotiana longiflora.*



ABC, ABc, AbC, Abe, aBC, aBc, abC, abc X ABC, ABc, AbC, Abc, aBC, aBc, abC, abc

F_2 generation.

Genotype	Frequency	Number of dominant factors	Length	Frequency
AABBCC	1	6	70mm.	1
AABBCc AABbCC AaBBCC	2 2 2	5	67mm.	6
AABbCc AaBBCc AaBbCC AABBcc AAbbCC aaBBCC	4 4 4 1 1 1	4	64mm.	15
AaBbCc AABbcc AaBBcc AAbbCc AabbCC aaBBCc aaBbCC	8 2 2 2 2 2 2	3	61mm.	20
AaBbcc AaBbCc aaBbCc AAbbcc aaBBcc aabbCC	4 4 4 1 1 1	2	58mm.	15
Aabbcc aaBbcc aabbCc	2 2 2	1	55mm.	6
aabbcc	1	0	52mm.	1