DEVIATIONS FROM MENDELISM (GENE INTERACTION)

Compiled by Srimanta Kanji

Mendel success:

7 genes: all present in **autosome** and all show **dominant and recessive relationship**.

In monohybrid cross- phenotypic ratio 3:1 and genotypic ratio 1:2:1 reason: 1. allele are separate each other at gamete formation time. 2. dominant and recessive relationship between two alleles of a gene.

In dihybrid cross- phenotypic ratio 9:3:3:1, reason: dominant and recessive relationship between gene and do not contamination among the genes. Genotypic ratio 1:2:1: 2:4:2: 1:2:1 reason: allele segregation and independent assortment.

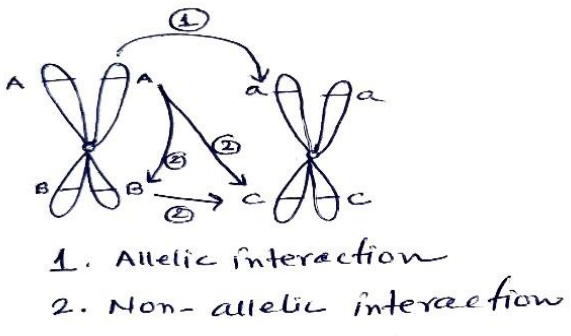
Each of the seven pairs of characters of pea plant which Mendel had chosen was controlled by a single pair of **genes or factors**.

BUT: In nature sometimes two alleles of a gene may show other relationship than dominant recessive relationship and also there is no independent assortment. In few cases, hence, genotypic and phenotypic ratio are change. There are many characters of various organisms which are controlled by two or more of genes located on different pairs of chromosomes and they interact in some complex fashion to give completely novel phenotypes. This phenomenon of two or more pairs of genes interacting with each other in various ways for the expression of a single character is called "**gene interaction**'. As a result of gene interaction, the typical mono- and dihybrid ratios 9:3:3:1 & 3:1 **will not be obtained**. Such exceptions, however, never deviate from Mendel's principle of independent assortment but simply extend and develop the ratios.

Definition: when two or more genes present on same or same or different chromosome but affect the expression of each other in the development of phenotype, known as **gene interaction**.

Gene interaction is two types:

A. **Allelic interaction/Intragenic interaction**
B. **Non allelic interaction/Intergenic interaction**



ALLELIC INTERACTION/INTRAGENIC INTERACTION:

Allelic interaction takes place between allele of **same gene** which are present at **same locus**.

*** In **case of Mendel law: one allele interacts with its second allele.**

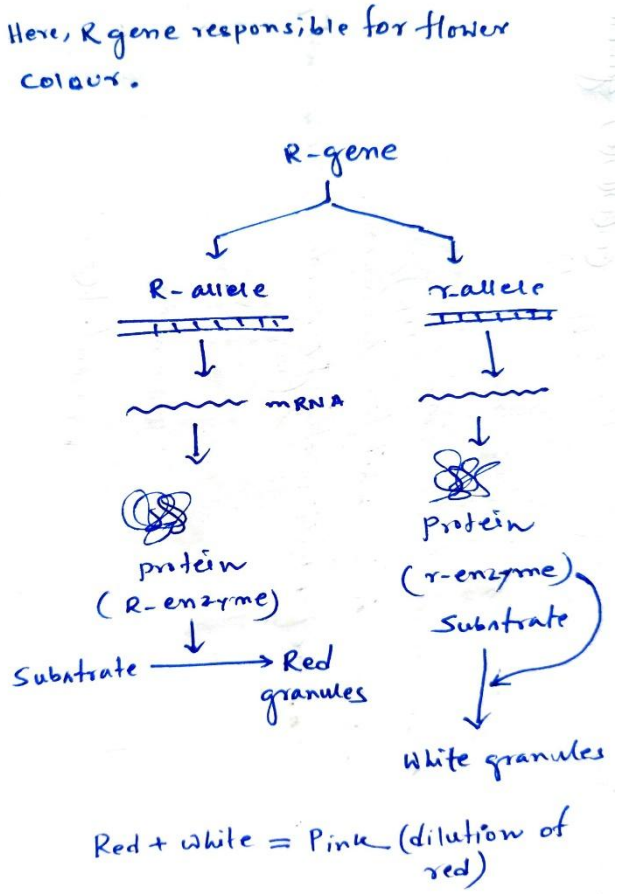
Example of Allelic interaction are as follows:

INCOMPLETE DOMINANCE OR SEMIDOMINANCE OR PARTIAL DOMINANCE:

Definition: Individuals with contrasting features when produce **some intermediate phenotype**, the phenomenon is called **incomplete dominance**.

Incomplete Dominance (**Indeterminate Inheritance**) is the phenomenon where **none** of the two contrasting alleles or factors is dominant. The expression of the character in a hybrid individual is **intermediate** or a **fine mixture** of the expression of the two factors.

According to Mendel's law of dominance, dominant character must be present in F_1 generation. But in some organisms, F_1 generation is different from the both parents. Both factors such as dominant and recessive are present in incomplete dominance but **dominant factor is unable to express its character completely**, resulting intermediate type of generation is formed which is different from the both parents. Some examples are –



1. **Flower colour in *Mirabilis jalapa*:** Incomplete dominance was first discovered by **Correns** in *Mirabilis jalapa*. This plant is called as '**4 O'clock plant** 'or' **Gul-e-Bans**'. Three different types of plant are found in Mirabilis on the basis of flower colour, such as **red, white** and **pink**.
   When plants with red flowers is crossed with white flower, plants with pink flower obtained in F1 generation. The reason of this is that the genes of red colour is incompletely dominant over the genes of white colour.
   When, F1 generation of pink flower is self-pollinated then the phenotypic ratio of F_2 generation is red, pink, white is **1:2:1** in place of normal monohybrid cross ratio **3:1**.
   The ratio of phenotype and genotype of F_2 generation in incomplete dominance is **always same**.
   N.B.: Here, genotypic ratio is 1:2:1 because alleles are normally segregated, so, no problem. But if **linkage** is present then genotypic ratio may be changed.

2. **Flower colour in *Antirrhinum majus*:** Incomplete dominance is also seen in flower colour off plant. This plant is also known as '**Snapdragon**' or '**Dog flower**'. Incomplete dominance is found in this plant which is the same as Mirabilis.
3. **Egg plant (brinjal):** purple, white and intermediate is violet colour.

4. **Feather colour in *Andalusian* Fowls:** Incomplete dominance is present for their feather colour, when a black colour fowl is crossed with a white colour fowl, the colour of F_1 generation is blue.

Red × White

R R ↓ r r

F_1 generation → R r (Pink)

↓ Self pollination

F_2 – Generation →

	R	r
R	RR	Rr
r	Rr	rr

RR = Red - 1

Rr = Pink - 2

rr = White - 1

CO-DOMINANCE:

A condition where expression of both alleles in **heterozygous** condition are **visible together**. There is no complete dominant recessive relationship.

In this phenomenon, both the gene expressed **for a particular character** in F1 progeny. There is no blending of characters, whereas both the characters expressed equally.



Examples- Co-dominance is seen in animals for coat colour. When a black parent is crossed with white parent, a **roan** colour F_1 progeny is produced. When we obtain F_2 generation from the F_1 generation, the ratio of black; black-white (Roan); white animals are **1:2:1**.

Note: F_2 generation is obtained in animals by sib-mating cross.

BLACK × WHITE

R_1R_1 ↓ R_2R_2

F_1 generation R_1R_2 (Roan)

↓ Sib-mating cross

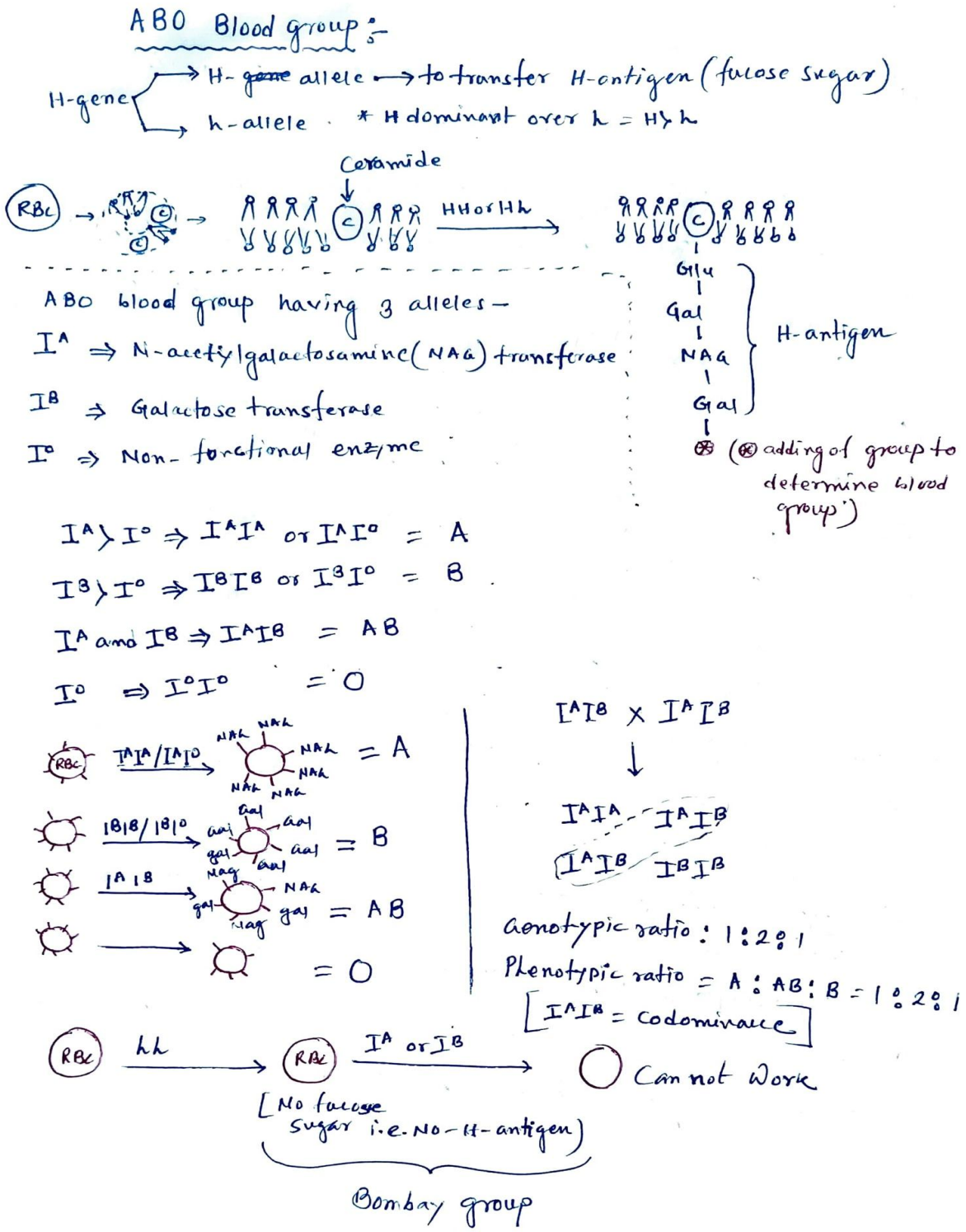
	R_1	R_2
R_1	R_1R_1	R_1R_2
R_2	R_1R_2	R_2R_2

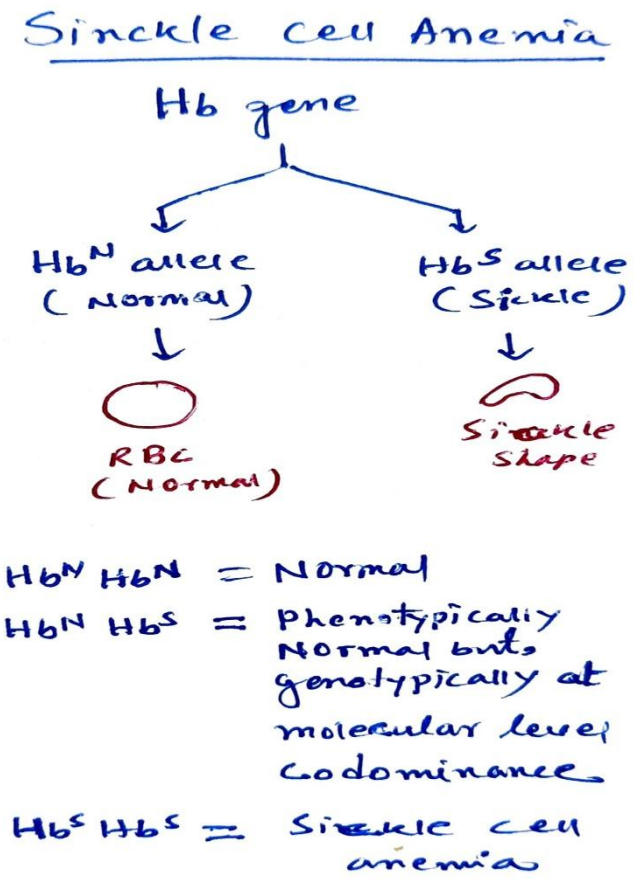
R_1R_1 = Black -1 R_1R_2 = Roan - 2 R_2R_2 = White - 1

It is obvious by above analysis that the ratio of phenotype as well as genotype is 1:2:1 in co-dominance.

N.B.: In incomplete dominance, characters are **blended phenotypically**, while in co-dominance, **both genes of a pair exhibit both the characters side by side** and effect of both the character is independent each other.

Other Examples of Co-dominance: (ii) AB blood group inheritance **(I^AI^B)** (iii) Carrier of Sickle cell anaemia **($Hb^A Hb^S$)**.



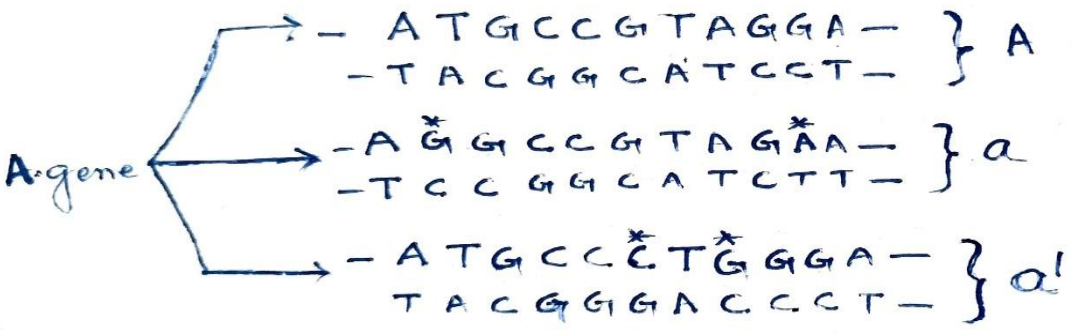


OVERDOMINANCE:

Sometimes the phenotype of F_1 heterozygote is **more extreme than that of either parent**. The amount of **fluorescent eye pigment** in heterozygous white eyes of *Drosophila* exceeds that found in either parent.

MULTIPLE ALLELE: MULTIPLEALLILISM: (More detail Hait book 137-139 pages)

An allele is a specific form of a gene and may be **mutated** producing an altered phenotype. Such mutation produces more than two alleles of a single gene (**cistron**), each governing a distinct form of the concerned trait in the population, called **multiple alleles. So, more than 2 alternative forms of same gene** called as **multiple alleles**. The phenomenon is known as **multipleallilism**.



Or, multiple alleles are also known as allelic series, when more than two alternative forms of a gene are present in a population, the condition is known as multiple alleles. Example- **A, a, a'** or **IA, IB, IO** etc.

In this case **only the monohybrid ratio** and **only two distinct phenotypes are observed in F_2 generation**. Some classical examples of multiple alleles are - (1) human blood group and Rh factor (3 alleles), (2) human haemoglobin, (3) self-incompatibility in plants, (4) coat colour in rabbits (4 alleles), (5) white (w) and rosy (ry) eye colour and lozenge eye shape gene of Drosophila, (6) waxy (wx) gene of maize etc.

- Multiple alleles are formed **due to mutation**.
- They are may be dominant recessive relationship or codominance between the alleles of a gene.
- Multiple alleles located on **same locus of homologous chromosome.**
- A **diploid** individual contains **two** alleles like **AA/Aa/Aa'/aa/aa'/a'a'** and **gamete** contains **one** allele for a character.
- Multiple alleles are of two types - (1) **true multiple alleles** i.e., more than two alleles of a single gene, or (2) **pseudoalleles**.
- **Pseudoalleles:** Two alleles each of **two or more tightly-linked genes** affecting the **same function** as a result of which they appear as multiple alleles in a **complex locus**, are called **pseudoalleles**. It is only a hypothesis, and not widely accepted.
- Multiple alleles are studied in **population.**
- **Lewis's effect:** 139 pages of Hait book.

If n is the number of allele of a gene then number of different possible genotype = $\boxed{\frac{n(n+1)}{2}}$

Example of multiple allele :

1. **ABO blood group** → ABO blood groups are determined by allele I^A, allele I^B, allele I^O

   I^A = dominant

   I^B = dominant

   I^O = recessive

   Possible phenotypes - A, B, AB, O

Blood group	Genotype	Antigen or agglutinogen	Antibody or agglutinin
A	I^AI^A, I^AI^O	A	b
B	I^BI^B, I^BI^O	B	a
AB	I^AI^B	A & B	None
O	I^OI^O	none	a & b

Possible genotype number = $\frac{3(3+1)}{2} = 6$ genotype

2. **Coat colour in rabbit** → Four alleles for coat colour in rabbit

Wild type = Full coloured = agouti = C^+

Himalayan [white with black tip on extremities (like nose, tail and feet)] = c^h

Chinchilla [mixed coloured and white hairs] = c^{ch}

albino = Colourless = c^a

These alleles show a gradient in dominance $C^+ > c^{ch} > c^h > c^a$

Possible genotypes –

Coloured	=	C^+C^+, C^+c^{ch}, C^+c^h C^+c^a
Chinchilla	=	$c^{ch}c^{ch}$, $c^{ch}c^h$, $c^{ch}c^a$
Himalayan	=	c^hc^h, c^hc^a
Albino	=	c^ac^a

Possible genotype $= \frac{4(4+1)}{2} = 10$ genotypes

Eg- Duck feather pattern

M gene = 3 allele = M^R → Restricted pattern; M → Mallard pattern; M^d → dusky pattern

$M^R > M > M^d$

∴ Maximum possible genotype = $\frac{n(n+1)}{2}$

n = no. of allele

So, $\frac{3(3+1)}{2} = 6$

$M^R M^R$, $M^R M$, $M^R M^d$ } Restricted

$M M$, $M M^d$ } Mallard

$M^d M^d$ } dusky

Self-sterility in tobacco: Self-sterility in tobacco is determined by the gene with many different allelic forms. If there are only three alleles (S_1, S_2, S_3), the possible genotypes of plants are S_1S_2, S_1S_3, S_2S_3 (**always heterozygous**), homozygous genotypes (S_1S_1, S_2S_2, S_3S_3) are **not possible** in a self-incompatible species. In this case, pollen carrying an allele different from the two alleles present in the female plant will be able to function resulting in restriction of fertility (Fig 7.4). More detail Hait book 138 page.

Male parent		S_1S_2		S_1S_3		S_2S_3	
Pollens		S_1	S_2	S_1	S_3	S_2	S_3
Female parent	S_1S_2	—	—	—	S_1S_3	—	S_1S_3
		Fully incompatible			S_2S_3		S_2S_3
	S_1S_3	—	S_1S_2	—	—	S_1S_2	—
			S_2S_3	Fully incompatible		S_2S_3	
	S_2S_3	S_1S_2	—	S_1S_2	—	—	—
		S_1S_3		S_1S_3		Fully incompatible	

Fig. 7.4: Compatibility in self-sterile plants in tobacco

Multiple allele in population :—

⇒ When alleles are present in X chromosome.

A gene ⟷ A, a

♀ | ♂

X^AX^A, X^AX^a, X^aX^a | 3 Types (geno-) | X^AY, X^aY | 2 types

⇓ | ⇓

$\frac{n(n+1)}{2}$ | only n formula

$= \frac{2(2+1)}{2}$ | So, number allele = number of types

= 3 genotypes | So, n = 2 genotypes

So, in population = ♀ + ♂

$= \frac{n(n+1)}{2} + n$

So, total population genotypes

= 3 + 2

= 5

eg- Rh blood group

Rh protein = Amonium transporter

↳ Present in RBC plasmamembrane

↳ 12 times span the membrane

⇒ Rh factors blood group system have 35 types in human.

⇒ The most famous blood group system is ABO group system and second most famous is Rh factor blood group system.

⇒ Rh system is consists of 50 antigens. Out of 50 only 5 are important C, c, D, E, e (There is no d antigen)

⇒ If D present = Rh+

If D absent = Rh−

* ⇒ Rh group is controlled by 8 alleles (present in 1st chromosome at $1p^{34-36}$ band).

⇒ Weiner Hypothesis → all 8 alleles belongs to same locus, hence, they are example of multiple allele.

⇒ Fisher and Race hypothesis : 3 alleles belong to the 3 different position, hence, they are called as Pseudoallele.

* Multiple allele :— Same function with same position.

Pseudoallele :- Same function but different position.

PLEIOTROPY:

Gene which controls **more than one character** is called **pleiotropic gene**. This shows multiple phenotypic effect.

For example:

(1) **In Pea plant:** Single gene influences i. the seed coat colour ii. Red spot-on leaf and iii. Flower colour.

(2) **In *Drosophila*** recessive gene of vestigial wings also influence the some another characters- **i.** Structure of reproductive organs **ii.** Longevity (Length of Body) **iii.** Bristles on wings **iv.** Reduction in egg production.

Examples of pleiotropic gene in human.

i. **Sickle cell anaemia** - Gene Hb^{S}_{β}, provide a classical example of pleiotropy. It not only causes haemolytic anaemia but also results increased resistance to one type of malaria that caused by the parasite *Plasmodium falciparum*. The sickle cell Hb^{S}_{β}, allele also has pleiotropic effect on the development of many tissues and organs such as bone, lungs, kidney, spleen, heart.

ii. **Cystic fibrosis**- Hereditary metabolic disorder that is controlled by a single autosomal recessive gene. The gene specifies an enzyme that produces a unique **glycoprotein**. This glycoprotein results in the production of **mucous**. More mucous interfere with the normal functioning of several **exocrine glands** including those in the skin, lungs, liver and pancreas.

iii. **Phenylketonuria-** Phenylketonuria (PKU) is a genetic disorder where the body can't process phenylalanine, an amino acid found in protein-rich foods. This leads to a build-up of phenylalanine in the blood, which can cause intellectual disability and other health problems if left untreated.

iv. In human, an autosomal dominant pleiotropic disease, **Marfan Syndrome**, the genetic defect affects the elastic connective tissue protein called **fibrillin** and this protein product of this pleiotropic gene also affects **eye lens**, that may dislocate **aorta** and **limb bones** and **fingers**. Limbs would be long and fingers become **spindle**. The most serious symptom is a life-threatening weakening in the aorta wall which sometimes may suddenly burst.

v. Another autosomal dominant, Pleiotropic disease, in human, ***Porphyria***, for example, had effects on the Royal families of Europe, particularly **King George III** of England. At age 50, he first felt abdominal pain and constipation, followed by week limbs, fever, fast pulse and dark red urine. Next nervous system symptoms began, including insomnia, headaches, visual problems, restlessness, delirium, convulsions and stupor. Parliament and Court were convinced that the king was mad. But he mysteriously recovered and again relapsed 13 years later and thereafter 3 years later, finally an attack in 1811 placed George in a prolonged stupor.

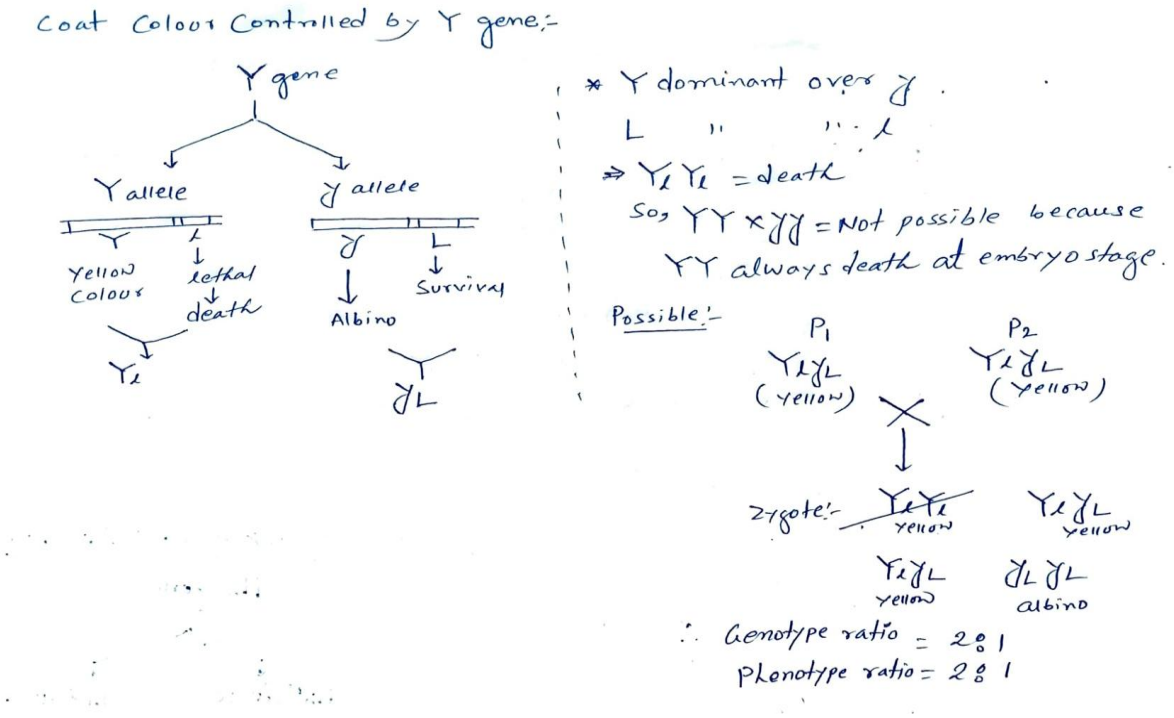
LETHAL GENE:

Lethal genes are the genes which are present at certain locus in the genome and they cause death of an organism upon mutation at certain stage of life. Actually, they are the normal genes, required for normal growth and development (like hexokinase genes in glycolysis), but upon mutation (hexokinase mutation glycolysis checked so, no ATP or energy produced for survival) they can cause death; hence, they are known as **lethal gene**.

Gene which causes death of individual in early stage when it comes in **homozygous condition** called **lethal gene**.

1. Lethal gene was discovered by **Lucien Cuenot** in coat colour of mice.

Yellow body colour**(Y)** was dominant over normal brown or albino colour**(y)**. **Gene of yellow body colour is lethal.** So homozygous yellow mice are **never obtained** in population. It dies in embryonal stage.



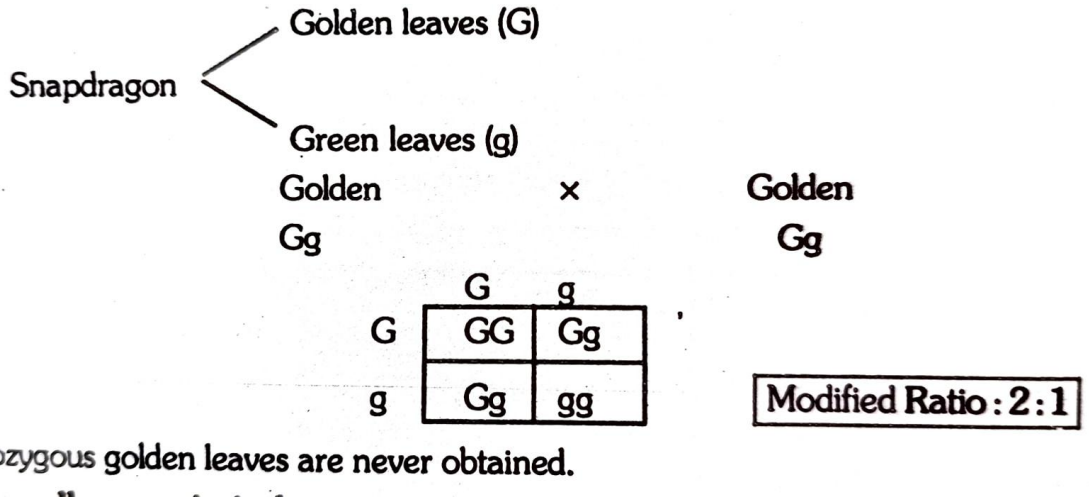
When yellow mice were crossed among themselves segregation for yellow and brown body colour was obtained in **2: 1** ratio.

Yy × Yy

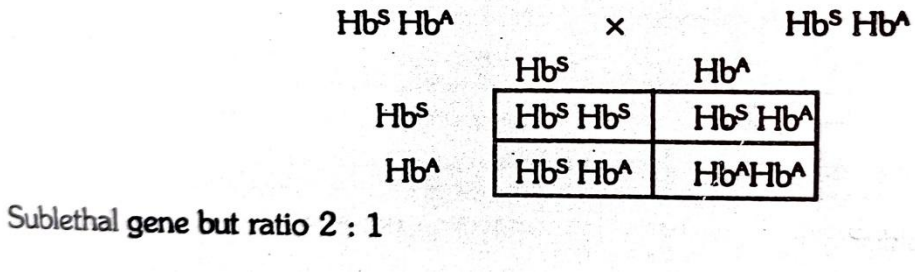
	Y	y
Y	YY	Yy
y	Yy	yy

YY - death in embryonal stage modified ratio = 2 : 1

2. In plant lethal gene was first discovered by **E. Baur** in **Snapdragon** (*Antirrhinum majus*)



3.Sickle cell anaemia in human: In human, gene of sickle cell anaemia Hb^S is the example of lethal gene. When two carrier individuals of sickle cell anaemia are crossed then offspring are obtained in **2: 1** ratio.



- Lethal gene may be dominant, recessive and conditional, but **mostly recessive for lethality**.
- **Dominant lethal:** AA= Death, e.g., all autosomal and X- linked dominant allele. Dominant lethal are lost from the population because they cause death of the organism **even in a heterozygous state**, e.g., ***epiloia*** gene in human beings.
- **Recessive lethal:** aa= death, example- all autosomal and X- linked recessive allele. **Mostly recessive for lethality.**
- **Conditional lethal: Conditional lethal** are those genes which are lethal in one condition and normal in other condition**. Conditional lethal** require a specific condition for their lethal action, e.g., temperature sensitive mutant of barley (**lethal effect at low temperature**). Another example is favism (haemolytic anaemia).
- **Balanced lethal** are all heterozygous for the lethal genes; both dominant and recessive homozygotes will die, e.g., balanced lethal system in *Oenothera*.
- Gametic lethals make the gametes **incapable of fertilization**, e.g., **segregation distorter** gene in male *Drosophila*.
- Many of these genes which do not cause definite lethality are called **semi lethal**. In semi lethal gene death occurs in **late stage**, e.g., *xentha* mutants in some plants.

NON-ALLELIC INTERACTION/INTERGENIC INTERACTION:

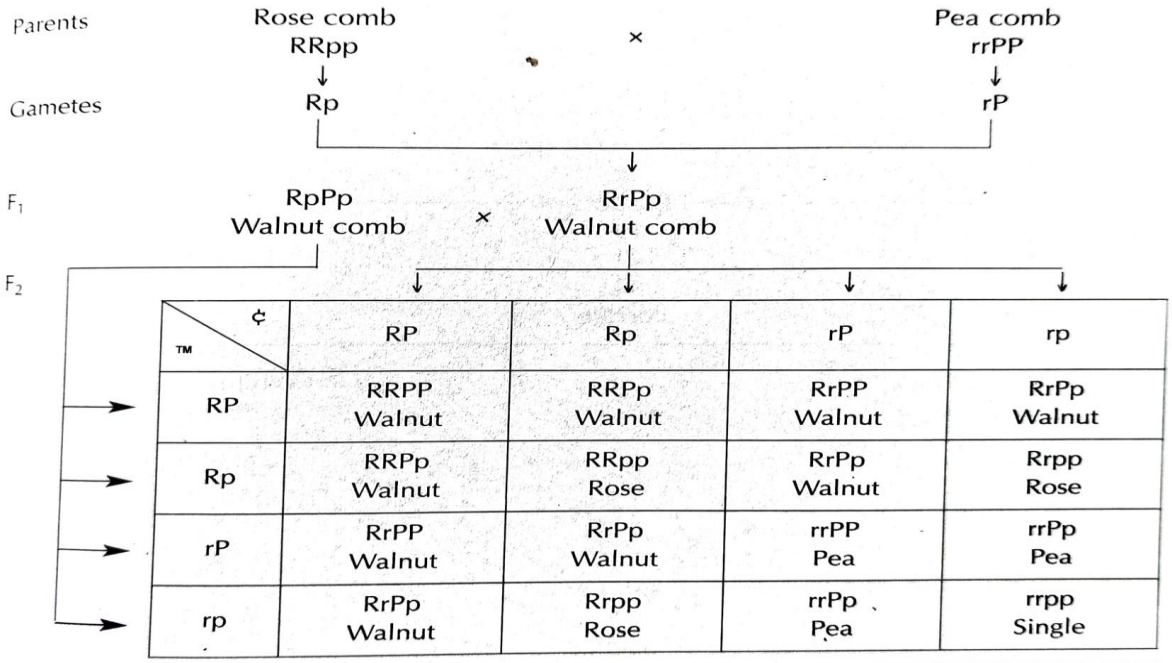
In Mendelian interaction two alleles of the same genes affect each other, so, they are known as allelic interaction. But, here two interacting genes are not the same allele of each other, hence it is known as **non-allelic gene interaction**.

Definition: When interaction takes place **between non allele** is called **non allelic gene interaction**. It changes or modifies other non-allelic gene.

SIMPLE INTERACTION (9:3:3:1):

In this case, two non-allelic gene pairs affect the same character. The dominant allele of each of the two factors produces separate phenotypes when they are alone. When both the dominant alleles are present together, they produce a distinct new phenotype. The absence of both the dominant alleles gives rise to yet another phenotype. The interaction is called **Simple interaction.**

1. The inheritance of **comb types** in fowls is the best example where **R** gene gives rise to **rose comb** and **P** gene gives rise to **pea comb**; both are dominant over **single comb**; the presence both the dominant genes result in **walnut comb** (Fig. 7.5).



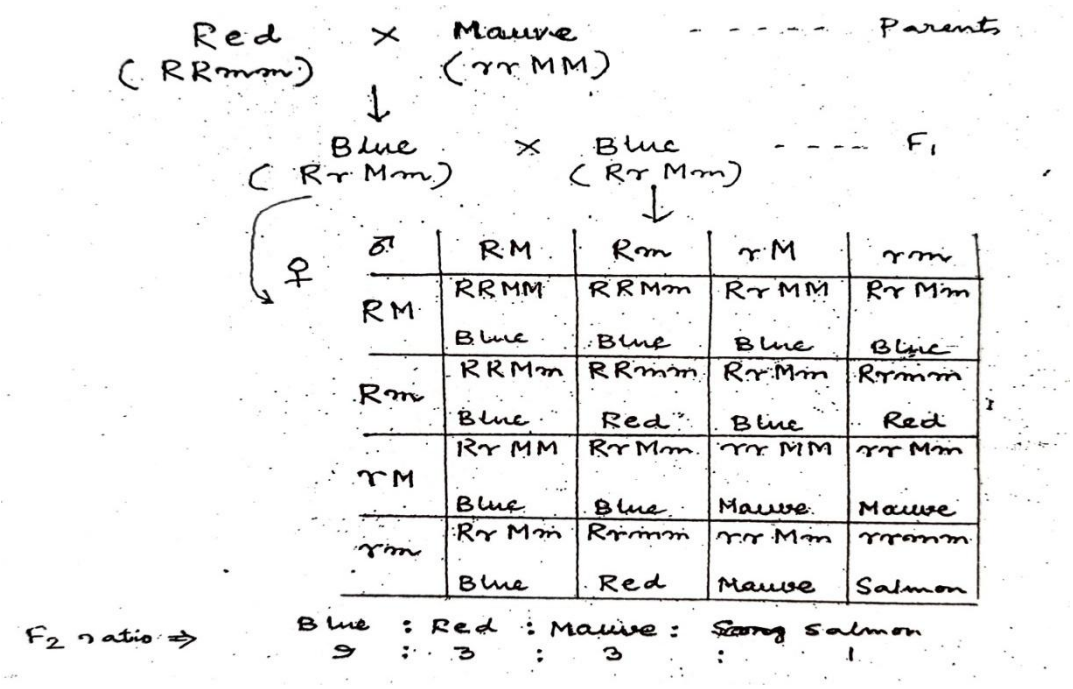
♂ / ♀	RP	Rp	rP	rp
RP	RRPP Walnut	RRPp Walnut	RrPP Walnut	RrPp Walnut
Rp	RRPp Walnut	RRpp Rose	RrPp Walnut	Rrpp Rose
rP	RrPP Walnut	RrPp Walnut	rrPP Pea	rrPp Pea
rp	RrPp Walnut	Rrpp Rose	rrPp Pea	rrpp Single

Genotype	Phenotype	Ratio
R-P-	Walnut	9
R-pp	Rose	3
rr P-	Pea	3
rr pp	Single	1

Fig. 7.5: Inheritance of comb types in fowl

2. **Simple interaction in plants:** The inheritance of flower colour in *Streptocarpus* follow **simple gene interaction**. A cross between true breeding **red flowered** variety and true breeding **mauve flowered** variety gives all **blue**

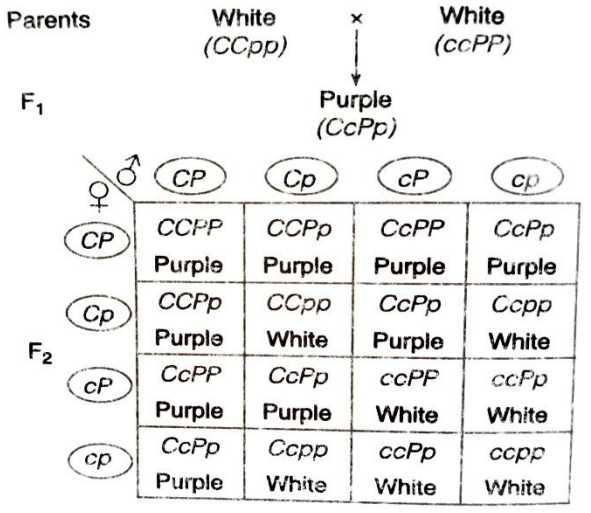
flowered in F1. The selfing of blue flowered variety give 4 phenotypes -bine, red, mauve & Salmon in a F2 ratio of **9:3:3:1**. The inheritance is as follows-



COMPLEMENTARY FACTOR (9:7): duplicate recessive epistasis

Certain characters are produced by the **interaction 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.

So, when two genes are involved in same biochemical pathway then both genes must be functional to produce a final phenotype or product. If any gene present in its **recessive homozygous condition the final phenotype is not expressed** and there will be mutant phenotype.



Phenotypic ratio = 9 Purple : 7 White

Fig. 1.6.11: Complementary gene interaction showing 9 : 7 ratio in the development of flower colour of sweet pea in F_2

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).

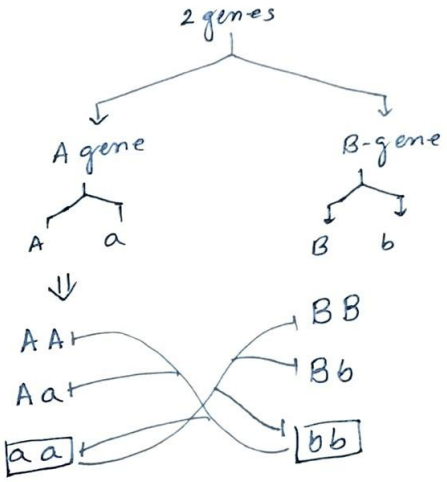
Involvement of more than two complementary genes is possible, e.g., three complementary genes governing. aleurone colour in maize.

Complementory gene interaction / duplicate recessive epistasis :-

Substrate 1 $\xrightarrow{\text{gene-1}}$ Substrate 2 $\xrightarrow{\text{gene-2}}$ Product

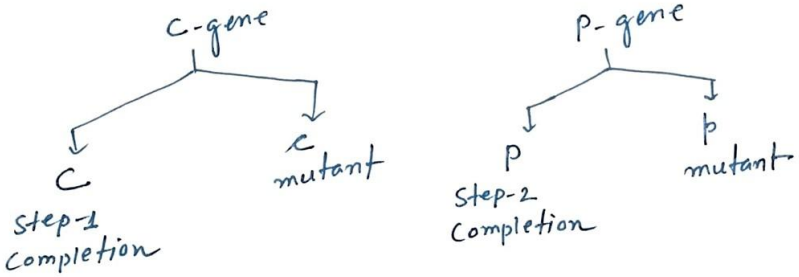
Like, $S_1 \xrightarrow[\text{A-gene}]{aa} S_2 \xrightarrow[\text{B-gene}]{bb} P$

(AA or Aa) (BB or Bb)



Example:- Sweet Pea flower colour

Substrate 1 (white) $\xrightarrow[CC/Cc]{\text{C gene}}$ Substrate 2 (white) $\xrightarrow[PP/Pp]{\text{P-gene}}$ purple colour (wild type)



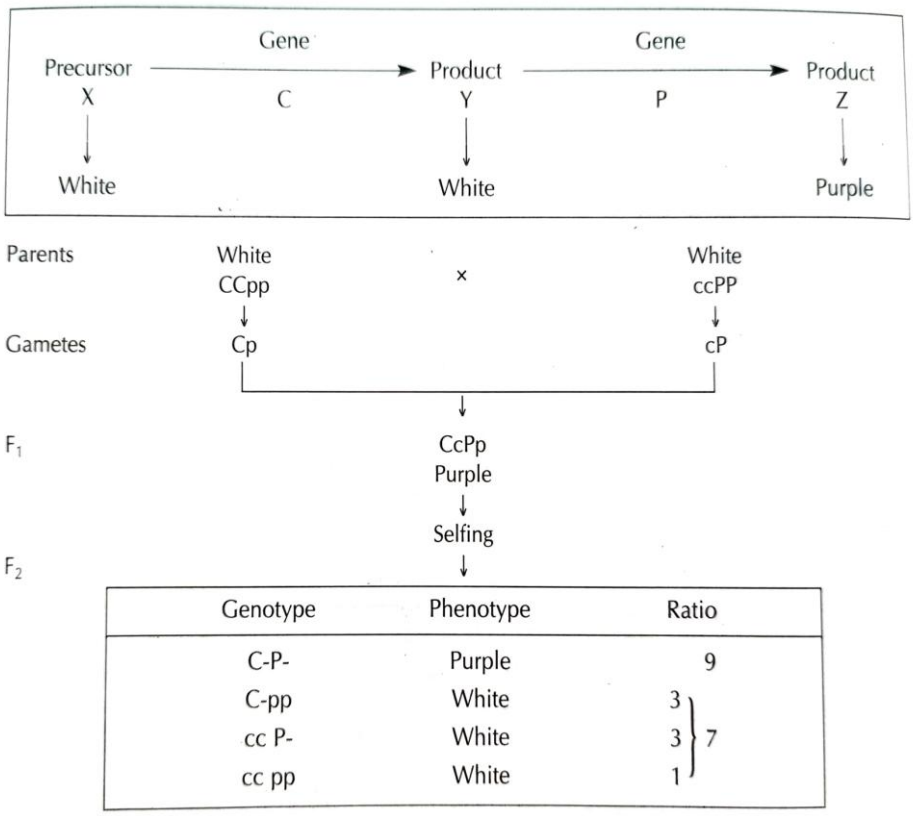
P_1 (CCpp, step-1 ✓ step-2 ✗, white flower mutant) × P_2 (ccPP, step-1 ✗ ×, white mutant)

$F_1 \rightarrow$ CcPp (selfing) (purple wild Normal type)

step-1 ✓
step-2 ✓

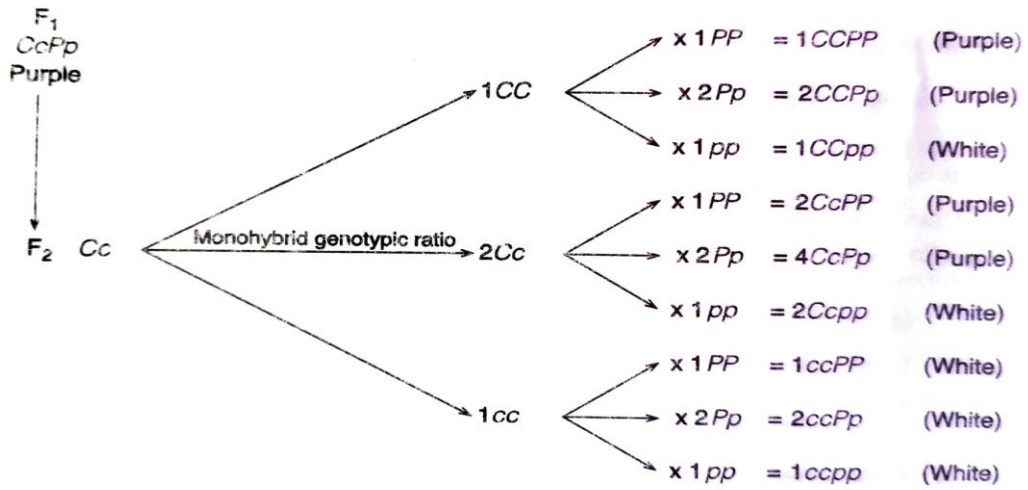
$F_2 \rightarrow$ C_P_ (9 purple) | C_pp (3 white) | ccP_ (3 white) | ccpp (1 white)

∴ purple : white = 9 : 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



Phenotypic ratio = 9 Purple : 7 White

Fig. 1.6.12: Complementary gene interaction showing 9 : 7 ratio in F_2 through forked line method

EPISTASIS:

When, a gene prevents the expression of another non-allelic gene i.e. When one gene at one locus hides the expression of another gene at different locus when present together, then it is known as **epistatic gene** and this phenomenon is known as **Epistasis**.

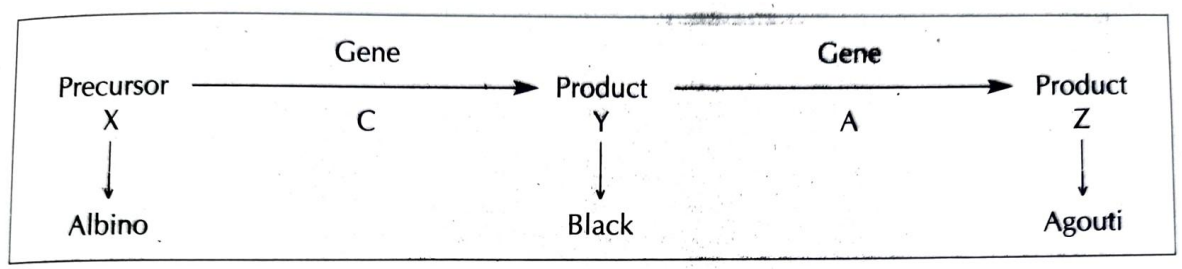
DOMINANCE	EPISTASIS
Allelic interaction.	Non-allelic interaction.
Dominant allele hides the expression of recessive allele.	Epistatic gene hides the expression of hypostatic gene.

Gene which inhibit the expression of another non allelic gene is called **epistatic gene** and expression of gene which is suppressed by epistatic gene called **hypostatic gene.** It is of two types- **1. Recessive epistasis and 2. Dominant epistasis**.

Recessive epistasis (Supplementary factor, 9:3:4):

When a recessive gone masks the expression of a non-allelic dominant gene, the interaction is termed as **recessive epistasis**.

In mice, the wild body colour '**agouti** (greyish) is controlled by a dominant gene '**A**' which is dominant over another dominant gene '**C**' which gives **black colour**. The recessive gene **c**' is epistatic to agouti gene '**A**' and the colour will be **albino**. So, '**A**' **gives rise agouti colour in the absence of** '**c**'. Similarly, 'C' gives rise to black colour only in the absence of 'A'. In the absence of both the dominant gene 'C' and 'A' the colour will be **albino**. When a black mice (CCaa) crossed with albino (cc AA) all the f1 progenies are agouti (Cc Aa). When F1 agouti mice are crossed among themselves agouti, black and albino mice are formed in a F2 ratio **9:3:4**. The inheritance is as follows-



Parents: Black CCaa × Albino ccAA

Gametes: Ca, cA

F_1: CcAa Agouti × CcAa Agouti

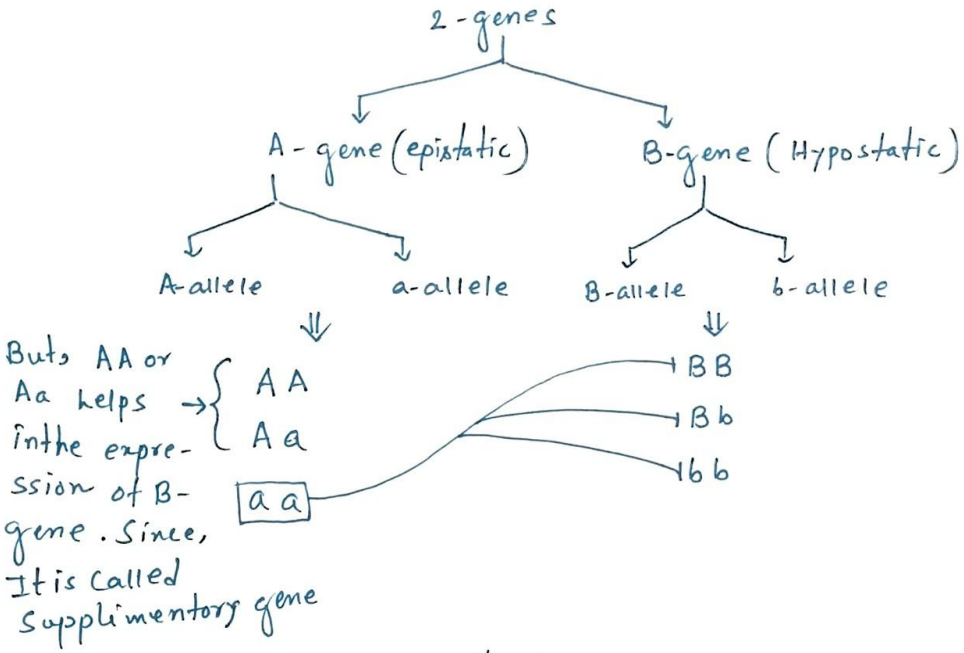
F_2

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

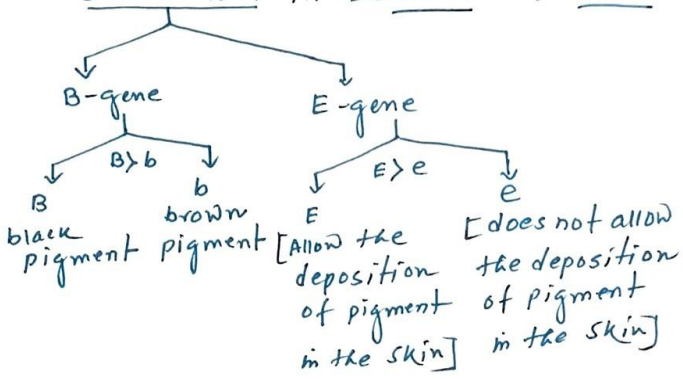
Recessive epistasis / Supplimentory gene interaction :-

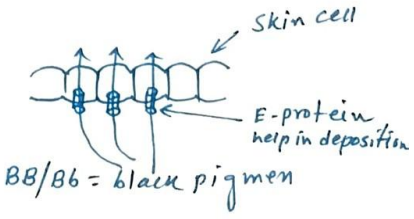
↳ when a gene is present in recessive homozygous condition it has the capacity to suppress the expression of other gene.

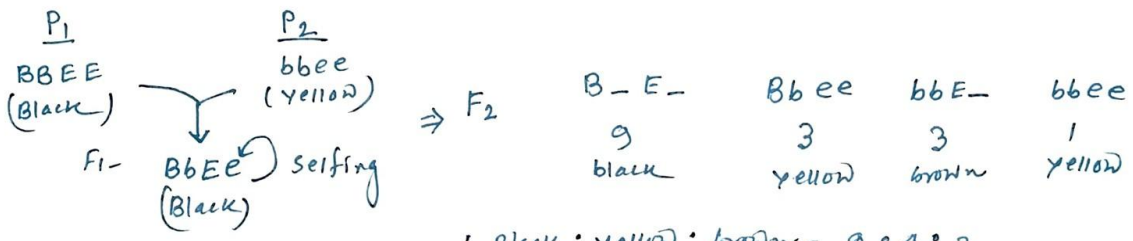


Example-of recessive epistasis :-

Coat colour in Labrador retrivers







∴ Black : yellow : brown = 9 : 4 : 3

White × Green — Parent

WWYY × wwyy

↓ ↓

WY wy — Gametes

WwYy (White) — F1

↓ Selfing

F1 × F1

WwYy × WwYy

Ww × Ww

$\frac{1}{4}$WW, $\frac{2}{4}$Ww, $\frac{1}{4}$ww

Yy × Yy

$\frac{1}{4}$YY, $\frac{2}{4}$Yy, $\frac{1}{4}$yy

$\frac{1}{4}$WW × $\frac{1}{4}$YY = $\frac{1}{16}$WWYY white

× $\frac{2}{4}$Yy = $\frac{2}{16}$WWYy white

× $\frac{1}{4}$yy = $\frac{1}{16}$WWyy white

$\frac{2}{4}$Ww × $\frac{1}{4}$YY = $\frac{2}{16}$WwYY white

× $\frac{2}{4}$Yy = $\frac{4}{16}$WwYy white

× $\frac{1}{4}$yy = $\frac{2}{16}$Wwyy white

$\frac{1}{4}$ww × $\frac{1}{4}$YY = $\frac{1}{16}$wwYY yellow

× $\frac{2}{4}$Yy = $\frac{2}{16}$wwYy yellow

× $\frac{1}{4}$yy = $\frac{1}{16}$wwyy green

∴ white : yellow : green = 12 : 3 : 1

Fig:- Dominant epistasis inheritance of fruit colour in Gummer Squash.

Black × Albino
CC aa × cc AA

↓

F_1 - Generation → Cc Aa [Agouti]

	C A	C a	c A	c a
C A	CC AA Agouti	CC Aa Agouti	Cc AA Agouti	Cc Aa Agouti
C a	CC Aa Agouti	CC aa Black	Cc Aa Agouti	Cc aa Black
c A	Cc AA Agouti	Cc Aa Agouti	cc AA Albino	cc Aa Albino
c a	Cc Aa Agouti	Cc aa Black	cc Aa Albino	cc aa Albino

Thus, Recessive epistasis or supplementary gene ratio in F_2 - Agouti : Black : Albino
9 : 3 : 4

Example of recessive epistasis :-

Bombay phenotype blood group

H-gene

I-gene

H-allele [help in formation of fucose sugar i.e. H-antigen)

h allele [cannot help in fucose sugar i.e. H-antigen formation]

HH

Hh

hh

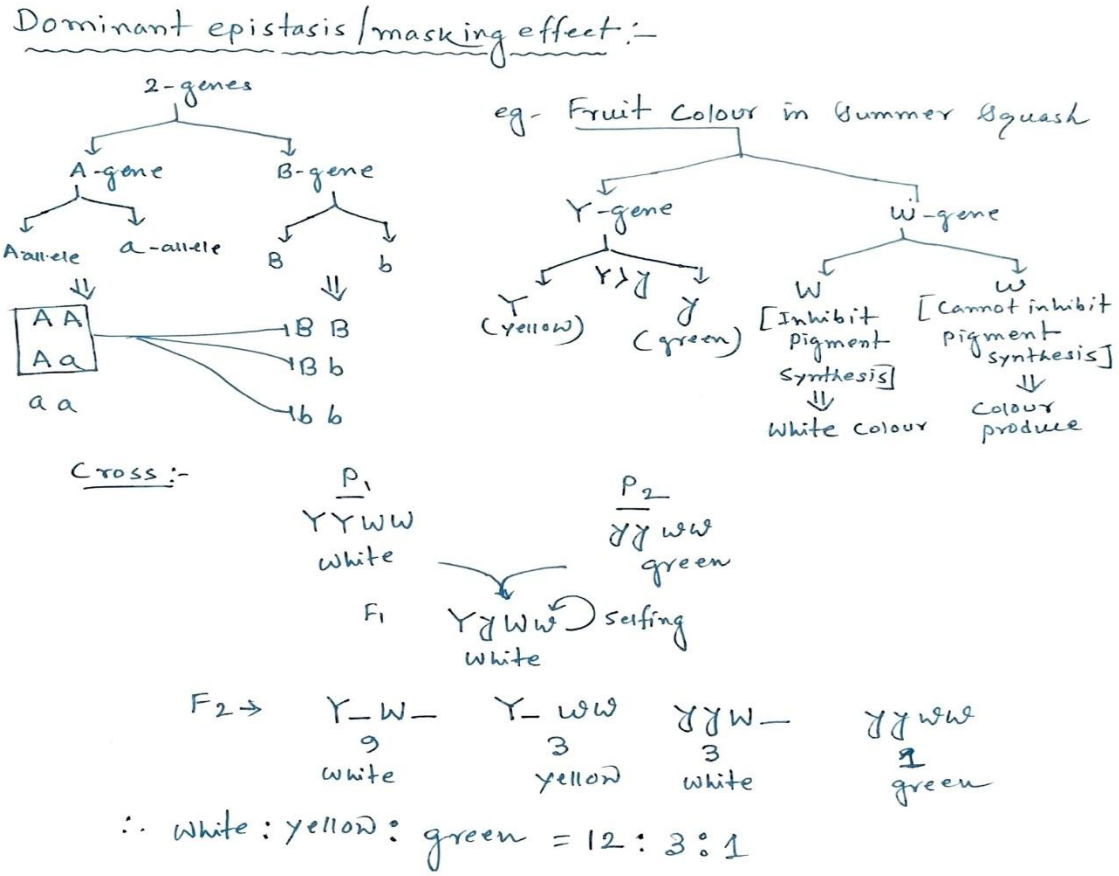
I^AI^A or I^AI^O

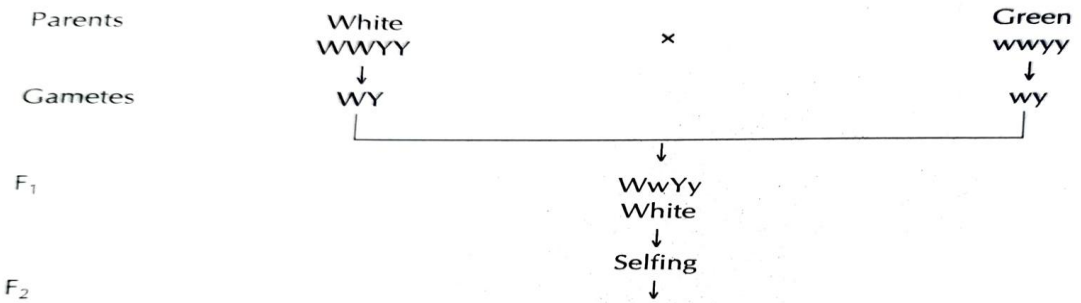
I^BI^B or I^BI^O

I^AI^B or I^OI^O

Dominant epistasis (12:3:1): Masking effect

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 expression 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

White $\times$ Green — Parent

$WWYY \times wwyy$

$\downarrow \quad \downarrow$

$WY \quad wy$ — Gameten

$WwYy$ (White) — F_1

$\downarrow$ Selfing

$F_1 \times F_1$

$WwYy \times WwYy$

$Ww \times Ww$ $\qquad$ $Yy \times Yy$

$\frac{1}{4}WW, \frac{2}{4}Ww, \frac{1}{4}ww$ $\qquad$ $\frac{1}{4}YY, \frac{2}{4}Yy, \frac{1}{4}yy$

$\frac{1}{4}WW \times \frac{1}{4}YY = \frac{1}{16}WWYY$ white

$\times \frac{2}{4}Yy = \frac{2}{16}WWYy$ white

$\times \frac{1}{4}yy = \frac{1}{16}WWyy$ white

$\frac{2}{4}Ww \times \frac{1}{4}YY = \frac{2}{16}WwYY$ white

$\times \frac{2}{4}Yy = \frac{4}{16}WwYy$ white

$\times \frac{1}{4}yy = \frac{2}{16}Wwyy$ white

$\frac{1}{4}ww \times \frac{1}{4}YY = \frac{1}{16}wwYY$ yellow

$\times \frac{2}{4}Yy = \frac{2}{16}wwYy$ yellow

$\times \frac{1}{4}yy = \frac{1}{16}wwyy$ green

$\therefore$ white : yellow : green = 12 : 3 : 1

Fig:- Dominant epistasis inheritance of fruit colour in Gummer Squash.

INHIBITORY FACTOR (13:3): SUPRESSOR GENE?

Inhibitory factor is such a gene which **itself has no phenotypic effect** but **inhibits the expression of another non-allelic gene**. In rice, purple leaf colour is due to gene **P**, and **p** causing green colour. Another non-allelic **dominant gene I inhibits the expression of P** but is **ineffective in recessive form** (**ii**). Thus, **the factor I has no visible effect of its own but inhibits the colour expression of P** (Fig. 7.11).

Parents: Purple PPii × Green ppII

Gametes: Pi × pI

F_1: PpIi, Green

Selfing

F_2:

Genotype	Phenotype	Ratio
P-I-	Green	9 } 13
ppI-	Green	3 } 13
ppii	Green	1 } 13
P-ii	Purple	3

Fig. 7.11: Inheritance of leaf colour in rice

P: Purple (*PPii*) × Green (*ppII*)

F_1: Green (*PpIi*)

F_2:

♀ \ ♂	*PI*	*Pi*	*pI*	*pi*
PI	*PPII* Green	*PPIi* Green	*PpII* Green	*PpIi* Green
Pi	*PPIi* Green	*PPii* Purple	*PpIi* Green	*Ppii* Purple
pI	*PpII* Green	*PpIi* Green	*ppII* Green	*PPIi* Green
pi	*PpIi* Green	*Ppii* Purple	*ppIi* Green	*ppii* Green

Phenotypic ratio = 13 Green : 3 Purple

Fig. 1.6.17: Inhibitory gene interaction showing 13 : 3 ratio in the development of leaf colour in rice in F_2

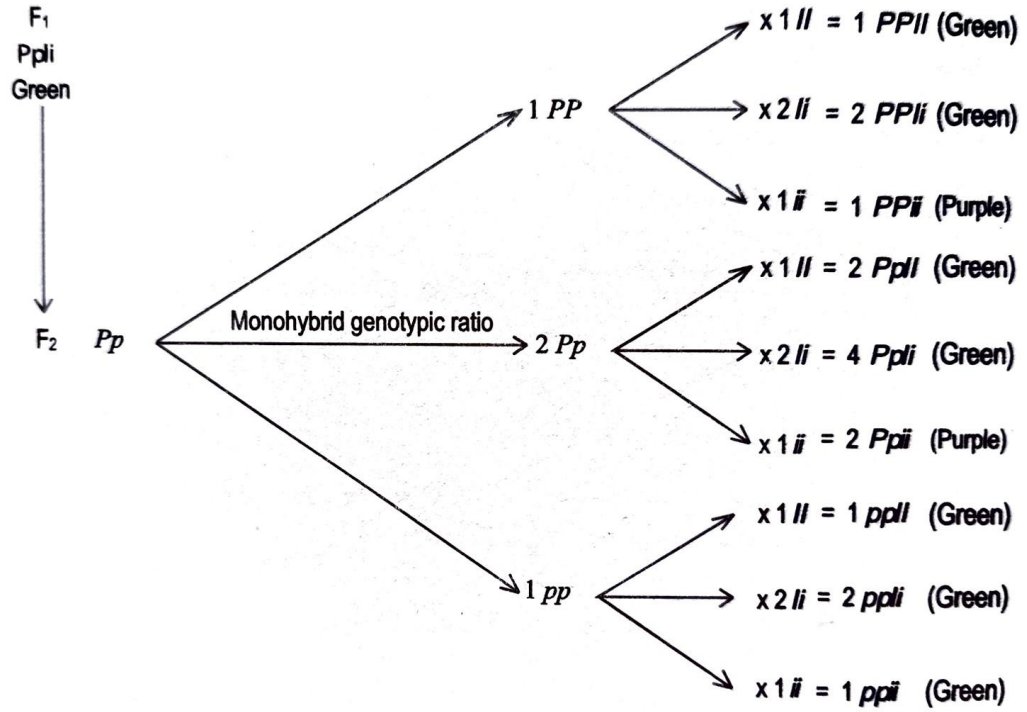
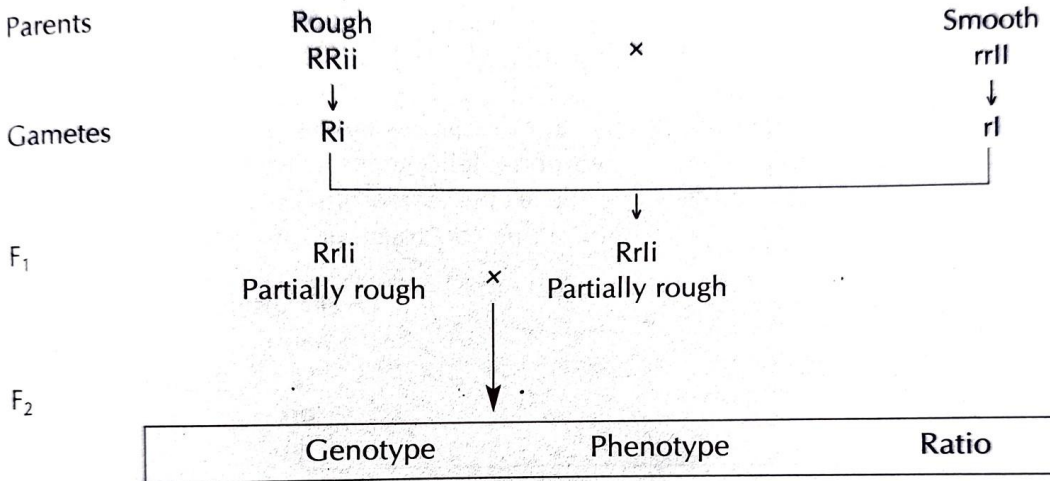


Fig. 1.6.18: Inhibitory gene interaction showing leaf colour of rice in 13 : 3 ratio in F_2 through forked line method

Inhibitory Factor with Partial Dominance (7:6:3): Sometimes an inhibitory gene shows incomplete dominance thus allowing the expression of other gene **partially**. In guineapig, hair direction is controlled by two genes. **Rough (R)** hair is dominant over **smooth (r**) hair, other gene I is inhibitory to **R at homozygous state (II**) **but in heterozygous state (Ii) causes partially rough** (Fig. 7.12).



Genotype	Phenotype	Ratio
R-II	Smooth	3 } 7
rr - -	Smooth	4 } 7
R-Ii	Partially rough	6
R-ii	Rough	3

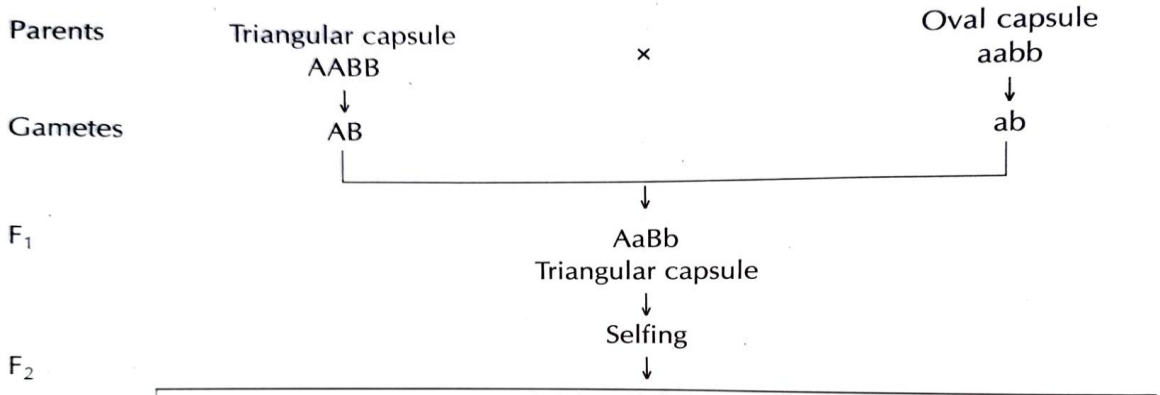
Fig. 7.12: Inheritance of hair direction in guineapig

DUPLICATE GENE (15: 1): Duplicate dominant epistasis or POLYMERIC GENE

Sometimes a character is controlled by two non-allelic genes whose dominant alleles produce the same phenotype whether they are alone or together.

Duplicate gene also example of **pseudo allele** (function same but position different).

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 recessive forms, the oval capsules produced (Fig. 7.14).



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

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

Duplicate gene interaction / duplicate dominant epistasis :-

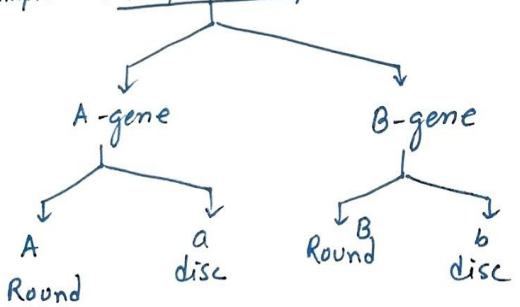
↳ This is example of pseudo allele [Same function but different position]

A-gene ⇒ Phenotype x type

B-gene ⇒ Phenotype x type

A_B_ ⇒ Phenotype x type

Example - Endosperm shape in maize.



Cross :- P1 × P2

AAbb (Round) × aaBB (Round)

↓

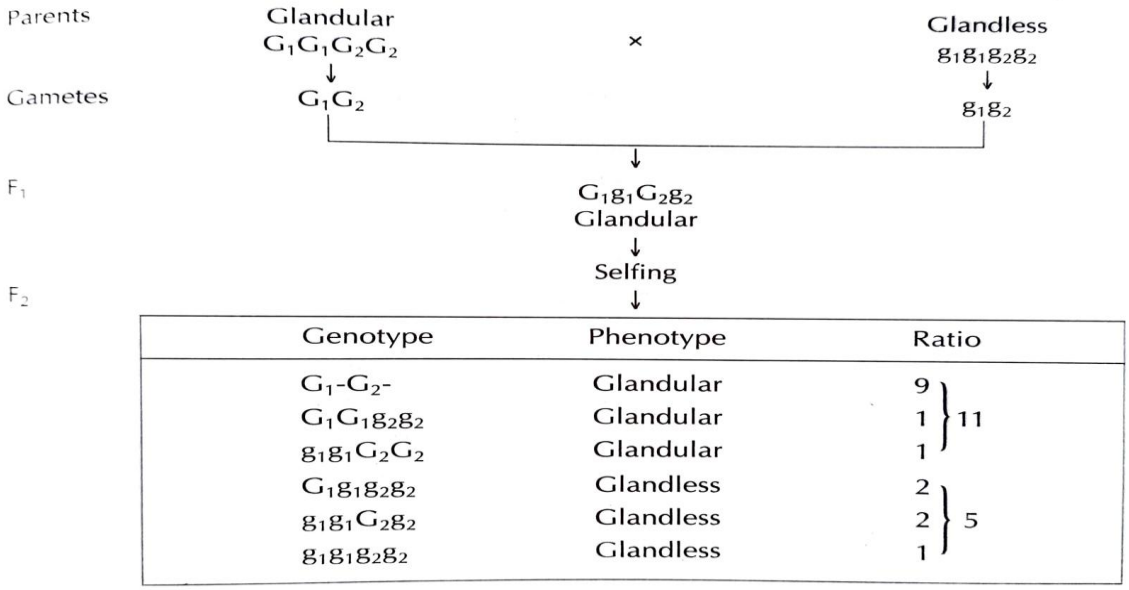
F1 - AaBb (Round) ↻ selfing

F2 ⇒ A-B- (9 Round) | A-bb (3 Round) | aaB- (3 Round) | aabb (1 disk)

∴ Round : disc = 15 : 1

Duplicate Gene with Dominance Modification (11:5):

A character controlled by two gene pairs showing dominance **only if two dominant alleles are present**. Dominant phenotype will thus be produced only when two non-allelic dominant alleles or two allelic dominant alleles are present. Such a case is found in pigment glands of cotton (Fig. 7.15).

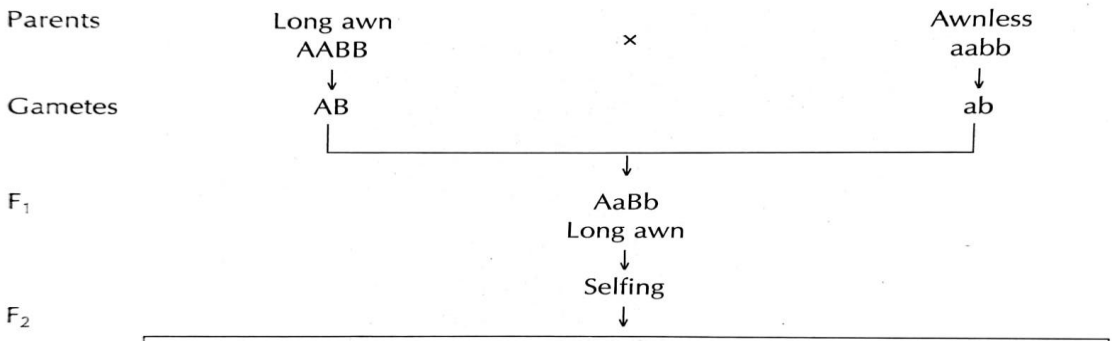


Genotype	Phenotype	Ratio
G_1-G_2-	Glandular	9 } 11
$G_1G_1g_2g_2$	Glandular	1 } 11
$g_1g_1G_2G_2$	Glandular	1 } 11
$G_1g_1g_2g_2$	Glandless	2 } 5
$g_1g_1G_2g_2$	Glandless	2 } 5
$g_1g_1g_2g_2$	Glandless	1 } 5

Fig. 7.15: Inheritance of pigment glands in cotton

POLYMORPHIC GENE (9:6:1):

Here two non-allelic genes controlling a character produce identical phenotypes when they are alone, but when **both the genes are present together their phenotypic effect is enhanced due to cumulative effect**. In barley, two genes **A** and **B** affect the length of awns. Gene A or B alone gives rise to awns of **medium** length (the effect of A is same as B); but when both present, **long** awn is produced; absence of both results **awnless** (Fig. 7.13).



Genotype	Phenotype	Ratio
A-B-	Long awn	9
A-bb	Medium awn	3 } 6
aa B-	Medium awn	3 } 6
aa bb	Awnless	1

Fig. 7.13: Inheritance of awns in barley

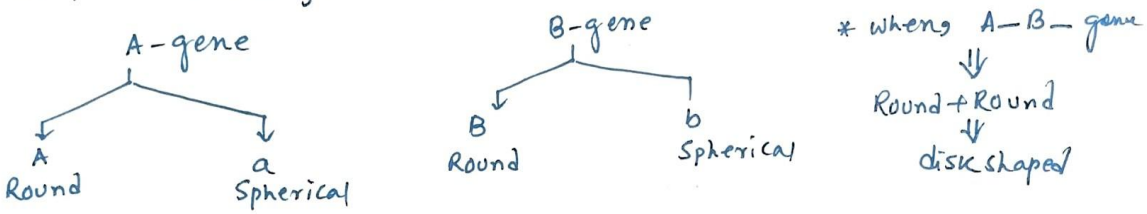
Polymorphic gene interaction/additive interaction/polygenic inheritance :-

A-gene = Phenotype X

B gene = Phenotype X

A–B– gene = phenotype x + phenotype X

Example – Pumkin fruit shape



Cross :- P_1 AAbb Round × P_2 aaBB Round

↓

AaBb disc ↻ selfing

$F_2 \Rightarrow$

A—B—	A–bb	aaB–	aabb
9	3	3	1
disc	Round	Round	spherical

∴ Disk : Round : spherical = 9 : 6 : 1

ADDITIVE GENE EFFECT:

In additive gene effect if non allelic gene **separately in dominant stage phenotype is same but both gene come dominant stage together phenotype is change** due to additive effect.

eg. Fruit shape in cucumber

A – bb → spherical

aa B → spherical

A – B – discoid (new phenotype)

aa bb – cylinderical

A – B –	A – bb	a – BB	aa bb
9	3	3	1
9 :	6 :	1	New phenotypic ratio
discoid	Spherical	Cylinderical	

COLLABORATORY GENE:

Two pairs of non-allelic gene interacting together to produce **a new phenotype**.

In this interaction two genes at two loci control a single character. Normally, two genes expressed two different phenotypes when present individually but, when two genes in their **dominant form** and come together they produce a **third phenotype**.

Definition: The two pairs of genes which though present on separate locus, **interact to produce a new trait or phenotype that neither of the genes could produce when present alone**.

Comb types in poultry is an example of collaborative genes. The gene '**R**' gives rise to **rose comb** while the gene '**P**' gives rise to **pea comb** when present singly. But, the genes 'R' and 'P' when present together produce a **new** '**walnut comb**'. The phenotypic ratio obtained is **9:3:3:1**.

If, **RR** = For Rose comb

PP = For Pea comb

Both **R & P** = For walnut comb

rr pp = for single comb

A new type of phenotype walnut (**Rr Pp**) comb is produced by the cross in between rose comb (**RR pp**) and pea comb (**rr PP**).

Rose comb RR pp × Pea comb rr PP

↓

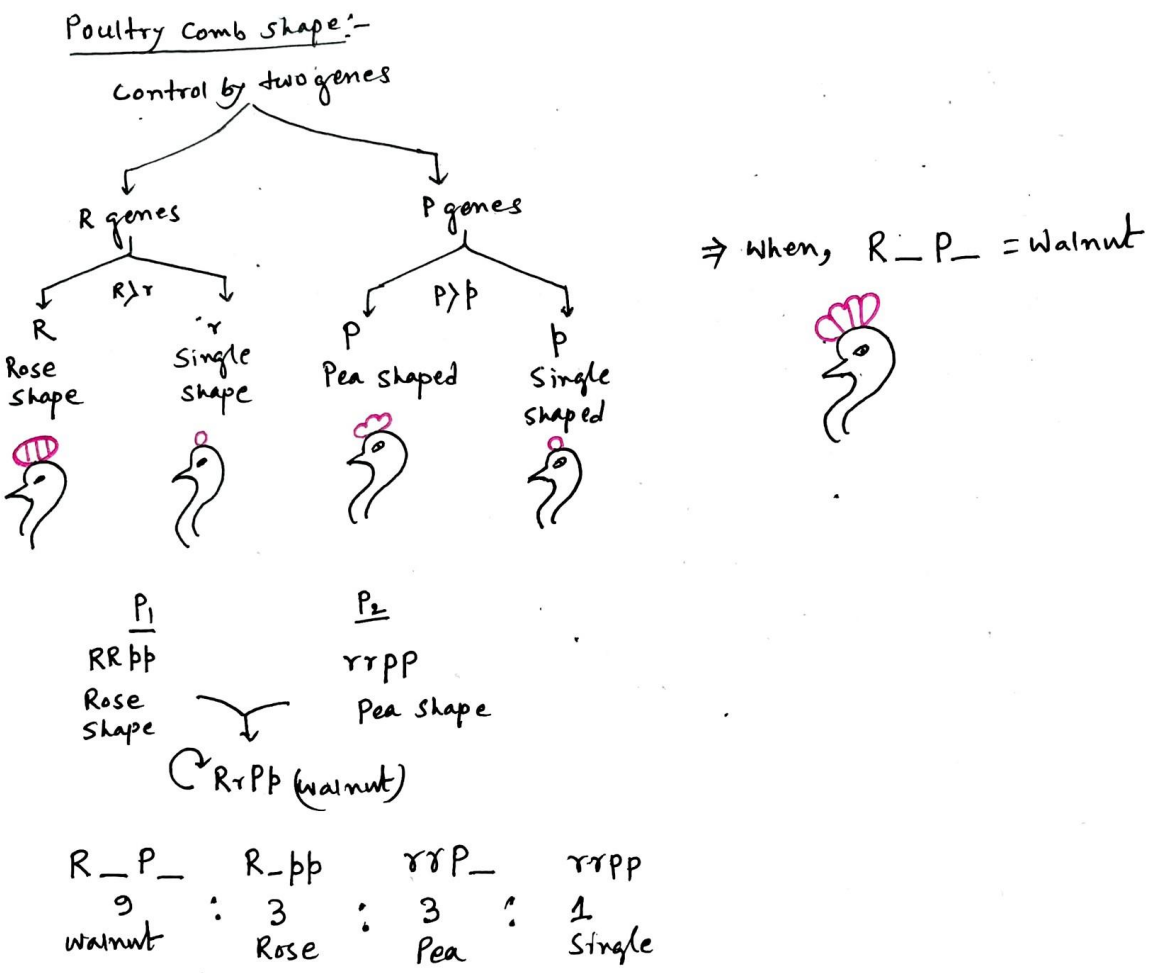
F_1-generation → Rr Pp = (Walnut comb)

↓

F_2

	R P	R p	r P	r p
R P	RR PP Walnut	RR Pp Walnut	Rr PP Walnut	Rr Pp Walnut
R p	RR Pp Walnut	RR pp Rose	Rr Pp Walnut	Rr pp Rose
r P	Rr PP Walnut	Rr Pp Walnut	rr PP Pea	rr Pp Pea
r p	Rr Pp Walnut	Rr pp Rose	rr Pp Pea	rr pp single

Thus, phenotypic ratio of collaboratory gene = Walnut : Rose : Pea : Single = 9 : 3 : 3 : 1



MODIFIERS:

Genes which modify the phenotypic effect of a major gene called **modifying gene**. They reduce or enhance the effect of other gene in **quantitative manner**, e.g., genes responsible for dilution of body colour.

SUPPRESSORS:

Genes which will not allow mutant allele of another gene to express resulting in wild phenotype called **suppressor gene**, e.g., **Su-s** in *Drosophila* suppresses the expression of dominant mutant gene star eye**(s)**.

ATAVISM:

The appearance of offsprings which resemble their **remote ancestors** called **atavism**.

Exceptions to Mendel's laws of inheritance:

Exceptions of law of dominance:

1. Incomplete dominance (indeterminate inheritance)
2. Codominance
3. Pleiotropy
4. Multiple allelism

Exception to law of segregation:

1. Complementary genes
2. Epistasis

Exceptions of law of independent assortment:

1. Supplementary genes
2. Duplicate genes
3. Collaborator genes

Deviation from Monohybrid ratio:

1. Incomplete dominance
2. Co-dominance
3. Over dominance
4. Lethal gene

Deviation from dihybrid ratio:

1. Complementary
2. Epistasis
3. Duplicate