Deviations from Mendelian ratios

Contents: Allelic interactions and deviations from Mendelian monohybrid ratio; Examples of allelic interactions; Non-allelic interactions and deviations from Mendelian dihybrid ratio; Examples of non-allelic interactions; Suggested reading.

Allelic interactions and deviations from Mendelian monohybrid ratio

The interactions of genes or alleles located on the same or different chromosomes of an organism are referred to as gene interactions. According to the definition, therefore, gene interactions could be categorized into either (a) allelic or (b) non-allelic.

We know that the classical monohybrid ratio is 3: 1, where T is completely dominant over t (*i.e.* T > t). Deviations from monohybrid ratio are due to allelic interactions, which are of the following three main types:

1. Incomplete dominance (T is not > t): The ratio is 1: 2: 1;
2. Codominance (T = t): The ratio is also 1: 2: 1; and
3. Lethal gene (homozygotes TT or tt die): The ratio is 2: 1.

Examples of allelic interactions

1. Incomplete dominance: The condition in heterozygotes where the phenotype is a blending *i.e*. intermediate between the two homozygotes. For example, pink flower progenies from the red and white flower parents.

2. Codominance: The condition in heterozygotes where both members of an allelic pair contribute to a phenotype, resulting in a mixture of both homozygotes. For example, roan offspring possess both red and white hairs of their parents.

3. Lethal gene: A gene whose phenotypic effect is sufficiently drastic to kill the bearer. The gene may be dominant, incompletely dominant or recessive.

Examples of the aforesaid allelic interactions are described in the following paragraphs.

Incomplete dominance in the Snapdragon flower, *Antirrhinum majus*

P: Red flower (r_1r_1)	×	White flower (r_2r_2)	
F_1:	r_1r_2 (all pink flowers; blending to a single colour)		
$F_1 \times F_1$:	r_1r_2	×	r_1r_2
F_2:	r_1r_1	r_1r_2, r_1r_2	r_2r_2
F_2:	¼ Red:	½ Pink:	¼ White
Phenotypic ratio	1 Red:	2 Pink:	1 White
Genotypic ratio	1:	2:	1

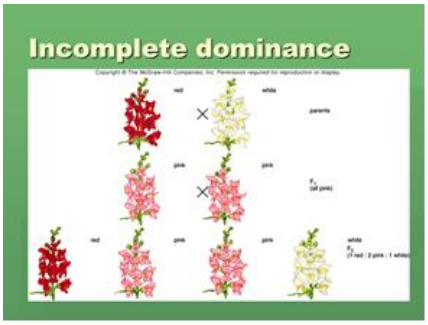

Fig. 3.1 Incomplete dominance for flower colour in the Snapdragon, *Antirrhinum majus*

Codominance in the Shorthorn cattle, *Bos taurus*

P:	Red ox (RR)	×	White cow (rr)
F_1:		Rr (all roan cattle)	

Roan= reddish grey; presence of both red and white hair side by side

$F_1 \times F_1$:	Rr	×	Rr
F_2:	RR	Rr, Rr	rr
F_2:	¼ Red:	½ Roan:	¼ White
Phenotypic ratio	1 Red:	2 Roan:	1 White
Genotypic ratio	1:	2:	1

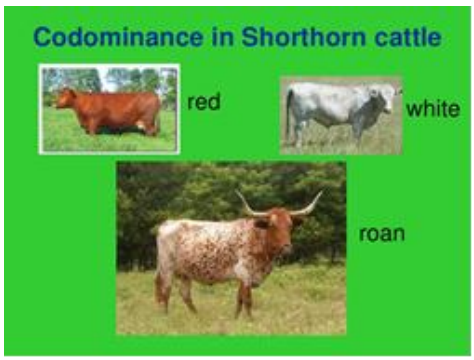

Fig. 3.2 Codominance for coat colour in the Shorthorn cattle

Example of lethal gene in maize/Indian corn, *Zea mays*

P:	Pale green (Gg)	×	Pale green (Gg)
F_1:	gg	Gg, Gg	GG
	¼ white (dies):	½ Pale green:	¼ Deep green
Phenotypic ratio		2 Pale green:	1Deep green
Genotypic ratio		2:	1

Fig. 3.3 Deep green (left), white (middle) and pale green (right) leaves of maize

Note: Crosses between a deep green (GG) and white or albino (gg) maize plants are not possible because the white seedlings, due to the lack of chlorophyll, die before maturity.

Example of lethal gene in the laboratory mouse, *Mus musculus*

P:	Yellow (A^YA) ×	Yellow (A^YA)	
F_1:	A^YA^Y	A^YA, A^YA	AA
	¼ Dies:	½ Yellow:	¼ Agouti
Phenotypic ratio		2 Yellow:	1 Agouti
Genotypic ratio		2:	1

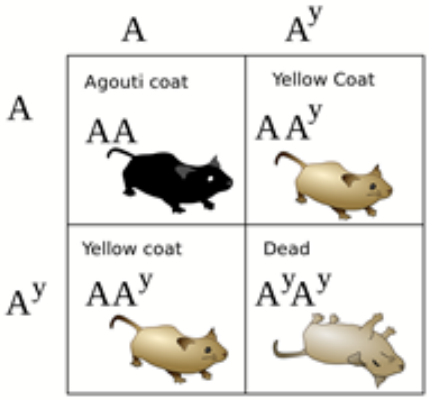

Fig. 3.4 Lethal gene in lab mice; ¼ mice of the genotype A^yA^y die due to lethal gene (A^y) in homozygous condition

Example of lethal gene in the chicken, *Gallus domesticus*

P:	Creeper (Cr/+)	×	Creeper (Cr/+)
F_1:	Cr/Cr	Cr/+, Cr/+	+/+
	¼ Dies:	½ Creeper:	¼ Normal
Phenotypic ratio		2 Creeper:	1 Normal
Genotypic ratio		2:	1

Note: Homozygous creeper (Cr/Cr) is embryonic lethal.

Example of lethal gene in the fruit fly, *Drosophila melanogaster*

P: Curly wing (Cy/+) × Curly wing (Cy/+)

F_1: Cy/Cy Cy/+, Cy/+ +/+

¼ Dies: ½ Curly wing: ¼ Normal wing

Phenotypic ratio 2 Curly wing: 1 Normal wing

Genotypic ratio 2: 1

Note: Homozygous curly (Cy/Cy) is embryonic lethal.

Non-allelic interactions and deviations from Mendelian dihybrid ratio

A classic example of non-allelic gene interaction came from the experiment of Bateson & Punnett on the size of combs in chickens. The *rose* comb is found in Wyandotte, the *pea* comb in Brahma and the *single* comb in Leghorn. A cross between Wyandotte × Brahma breeds gave rise to the *walnut* comb in F_1 generation, which was not found in any breed of chickens describe above. Then $F_1 \times F_1$ resulted in 9/16 *walnut*, 3/16 *rose*, 3/16 *pea* and 1/16 *single* combs among the F_2 progenies. However, it is to be kept in mind that neither *walnut* nor *single* combs are ever found in the pure breeds of Wyandotte or Brahma chickens.

Explanation

It is apparent that the *walnut* and *single* combs in chickens were due to an interaction of genes. Bateson & Punnett assumed that R and P genes controlled the expression of the *rose* and *pea* combs, respectively. The *walnut* comb was due to a heretozygote condition (R-P-), whereas the *single* comb was due to a recessive condition (rrpp). Accordingly, Bateson & Punnett considered that the genotype for the *rose* comb in Wyandotte was RRpp and that for the *pea* comb in Brahma was rrPP. The peculiar nature of the inheritance of comb trait in chickens is shown below.

P: Wyandotte (RRpp) × Brahma (rrPP)

(*rose* comb) (*pea* comb)

F_1: *walnut* comb

$F_1 \times F_1$: *walnut* comb × *walnut* comb

F_2: 9/16 *walnut* comb: 3/16 *rose* comb: 3/16 *pea* comb: 1/16 *single* comb

A couple of interesting points are to be note here. Firstly, even though the F_2 ratio was 9: 3: 3: 1, all the F_1 offspring had *walnut* comb, a trait that was not present in their parents. Secondly, *walnut* and *single* combs were found among F_2 progenies, while the traits were not present in their parents. So, it is obvious from the above explanation that interaction between a pair of non-allelic genes (R and P) resulted in the modified and exceptional combs in chickens, even though the dihybrid cross ratio was still like the classical 9: 3: 3: 1.

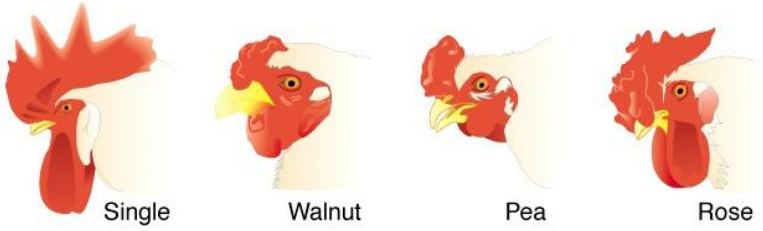

Fig. 3.5 Changes in the size of comb in chickens due to interaction of no-allelic genes

Similar to comb in chickens, genes for the coat colour in mice also exhibit non-allelic interactions. Here, the normal (wild-type) agouti or grey colour is modified to black, piebald, albino and Himalayan (Fig. 3.6). However, more examples of non-allelic interactions are cited under the modifications of dihybrid ratio in the next lecture.

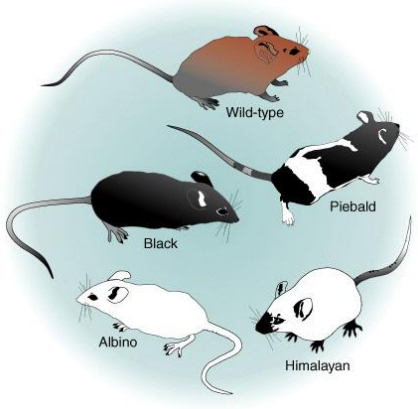

Fig. 3.6 Coat colour in mice is due to non-allelic interaction of genes

Examples of non-allelic interactions

We know that the classical dihybrid ratio is 9: 3: 3: 1, where T and R genes are completely dominant over t and r genes, respectively. Deviations from dihybrid ratio are due to non-allelic interactions (see Table and Fig. 3.7 below), which are mainly due to the phenomena of epistasis and hypostasis as explained below.

1. Epistasis: The masking of the phenotypic effect of either or both members of a pair of alleles by a gene of a different locus. Such gene is called an epistatic gene. For example, the inhibitory genes II and Ii are epistatic to the colour producing genes CC or Cc, resulting in white feathers in White Leghorn chickens.

2. Hypostasis: When in the presence of an epistatic gene, the expression of an allele at a different locus is suppressed, the phenomenon is called hypostasis. Such gene is known as a hypostatic gene. For example, the genes for deaf-mutism in man (dd or mm) are hypostatic to the normal hearing and speaking genes D- or M-.

Classical/standard	Ratio= 9: 3: 3: 1	BBSS × bbss in guinea pig
Interactions	Modified ratios	Examples
1. Recessive epistasis (Supplementary gene/factor)	9: 3: 4	Lab mice, *Mus musculus*
2. Dominant epistasis	12: 3: 1	Dog, *Canis familaris*
3. Duplicate interaction (Duplicate gene with cumulative effect)	9: 6: 1	Pig, *Sus domesticus*
4. Duplicate recessive epistasis (Complementary gene/factor)	9: 7	Man, *Homo sapiens*
5. Duplicate dominant epistasis	15: 1	Chicken, *Gallus domesticus*
6. Dominant and recessive epistasis (Inhibitory gene/factor)	13: 3	Chicken, White Leghorn

Summary of epistatic interaction

GENOTYPES	*A-B-*	*A-bb*	*aaB-*	*aabb*
Classical ratio	9	3	3	1
Dominant epistasis	12		3	1
Recessive epistasis	9	3	4	
Duplicate genes with cumulative effect	9	6		1
Duplicate dominant genes	15			1
Duplicate recessive genes	9	7		
Dominant and recessive interaction	13		3	

Fig. 3.7 Summary of epistatic interactions showing how the classical ratio 9: 3: 3: 1 is modified into various combinations such as 12: 3: 1, 9: 3: 4 and 13: 3

An example of recessive epistasis in lab mouse, *Mus musculus*

P: ♂♂Black (aaCC) × ♀♀Albino (AAcc)
F_1: All Agouti (AaCc)
$F_1 \times F_1$: AaCc × AaCc
Phenotypic ratio in F_2: 9 Agouti (A-C-)
3 black (aaC-)
4 albinos (A-cc, aacc)

Punnett square or checker board:

♂♂/♀♀	AC	Ac	aC	ac
AC	AACC	AACc	AaCC	AaCc
Ac	AACc	AAcc	AaCc	Aacc
aC	AaCC	AaCc	aaCC	aaCc
ac	AaCc	Aacc	aaCc	aacc

Reason: cc is epistatic to both A and a alleles

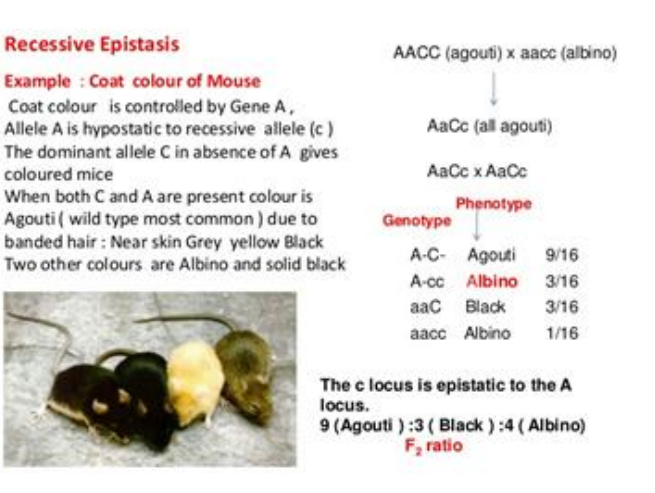

Fig. 3.8 An example of recessive epistasis in mice, in which the classical dihybrid ratio 9: 3: 3: 1 is modified to 9: 3: 4

An example of duplicate interaction in domestic pig, *Sus domesticus*

P: ♂♂Sandy (RRss) × ♀♀Sandy (rrSS)

F_1: All Red (RrSs)

$F_1 \times F_1$: RrSs × RrSs

Phenotypic ratio in F_2: 9 Red (R-S-)

6 sandy (R-ss, rrS-)

1 white (rrss)

♂♂/♀♀	RS	Rs	rS	rs
RS	RRSS	RRSs	RrSS	RrSs
Rs	RRSs	RRss	RrSs	Rrss
rS	RrSS	RrSs	rrSS	rrSs
rs	RrSs	Rrss	rrSs	rrss

Reason: rr and ss are epistatic to both R- and S-

Fig. 3.9 Red and sandy pigs

An example of duplicate recessive epistasis in man

P: ♂♂Normal hearing and speaking (DDMM) × ♀♀Deaf-mute (ddmm)

F_1: All normal hearing and speaking (DdMm)

$F_1 \times F_1$: DdMm × DdMm

Phenotypic ratio in F_2: 9 Normal hearing and speaking (D-M-)

7 deaf-mute (D-mm, ddM-, ddmm)

Punnett square

♂♂/♀♀	DM	Dm	dM	dm
DM	DDMM	DDMm	DdMM	DdMm
Dm	DDMm	DDmm	DdMm	Ddmm
dM	DdMM	DdMm	ddMM	ddMm
dm	DdMm	Ddmm	ddMm	ddmm

Reason: dd epistatic to M and m; mm epistatic to D and d

An example of dominant and recessive epistasis in chickens

P: ♂♂White Leghorn (CCII) ×♀♀White Plymouth Rock (ccii)

F_1: All White (CcIi)

$F_1 \times F_1$: CcIi × CcIi

Phenotypic ratio in F_2: 13 White (C-I-, ccI-, ccii)

3 coloured (C-ii) (Checkerboard shown below)

♂♂/♀♀	CI	Ci	cI	ci
CI	CCII	CCIi	CcII	CcIi
Ci	CCIi	CCii	CcIi	Ccii
cI	CcII	CcIi	ccII	ccIi
ci	CcIi	Ccii	ccIi	ccii

Reason: C epistatic to I and i; ii epistatic to C and c

White Leghorn ×

Brown Leghorn

- 13 (A___ or __bb) : 3 aaB_
- White Leghorn vs White Wyandotte chickens

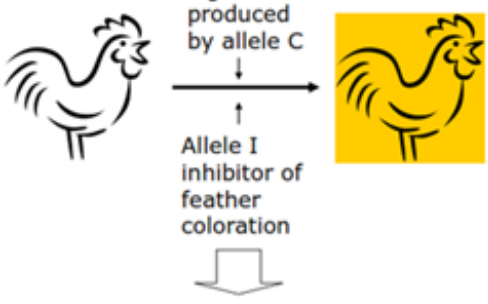

Fig. 3.10 An example of dominant and recessive epistasis in chickens, in which the classical dihybrid ratio 9: 3: 3: 1 is modified ti 13: 3

Suggested reading:

Ayala & Kiger, 1980.
Burns, GW. 1980.
Gardner *et al*. 1991.
Islam, MS. 2018.
Sinnott *et al*. 1973.
Winchester, AM. 1966.
Internet sources, 2018.
ইসলাম, ম.সা., খান, হা.সা. ও রানা, ম.হা.তা. ২০১৭।