

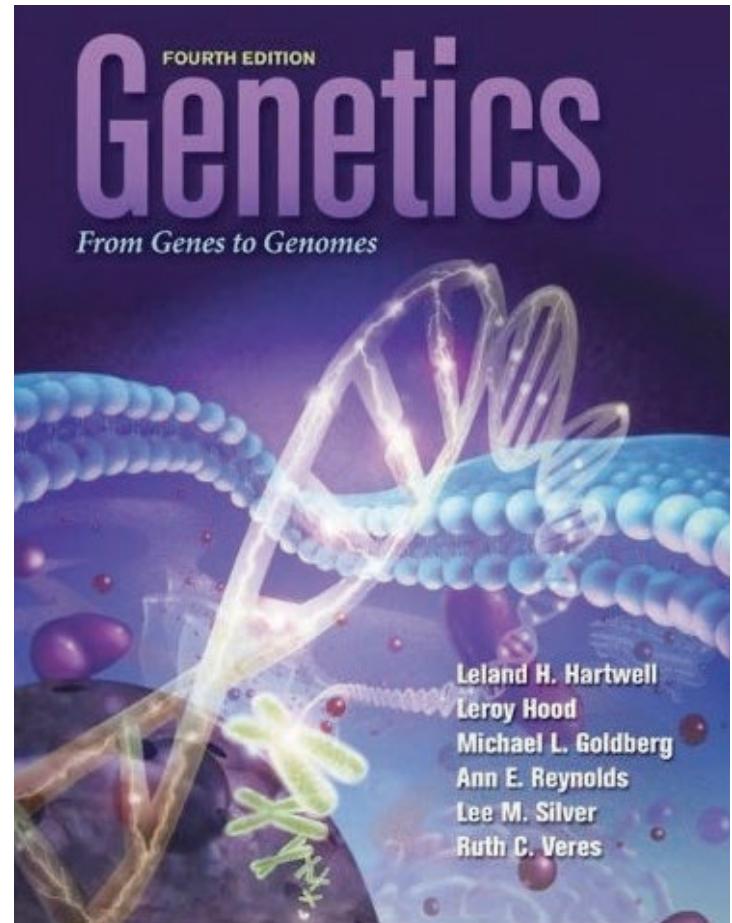
**PowerPoint to accompany**

# **Genetics: From Genes to Genomes**

## **Fourth Edition**

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## Extensions to Mendel's laws



## CHAPTER OUTLINE

- **Extensions to Mendel's laws**
- 3.1 Extensions to Mendel for Single-Gene Inheritance
- 3.2 Extensions to Mendel for Multifactorial Inheritance

# Some phenotypic variation poses a challenge to Mendelian analysis

**Example: Lentils come in an array of colors and patterns**

**Crosses of pure-breeding lines can result in progeny phenotypes that don't appear to follow Mendel's rules**

**Explanations for some traits:**

- No definitively dominant or recessive allele
- More than two alleles exist
- Multiple genes involved
- Gene-environment interactions



Fig. 3.1

# Extensions to Mendel for single-gene inheritance

Dominance is not always complete

- Incomplete dominance – e.g. snapdragon flower color
- Codominance – e.g. lentil coat patterns, AB blood group in humans

A gene may have >2 alleles – e.g. lentil coat patterns, ABO blood groups in humans, histocompatibility in humans

Pleiotropy - one gene may contribute to several characteristics

- Recessive lethal alleles – e.g.  $A^Y$  allele in mice
- Delayed lethality

# Summary of different dominance relationships

The phenotype of the heterozygote defines the dominance relationship of two alleles

**Complete dominance:** Hybrid resembles one of the two parents

**Incomplete dominance:** Hybrid resembles neither parent

**Codominance:** Hybrid shows traits from both parents

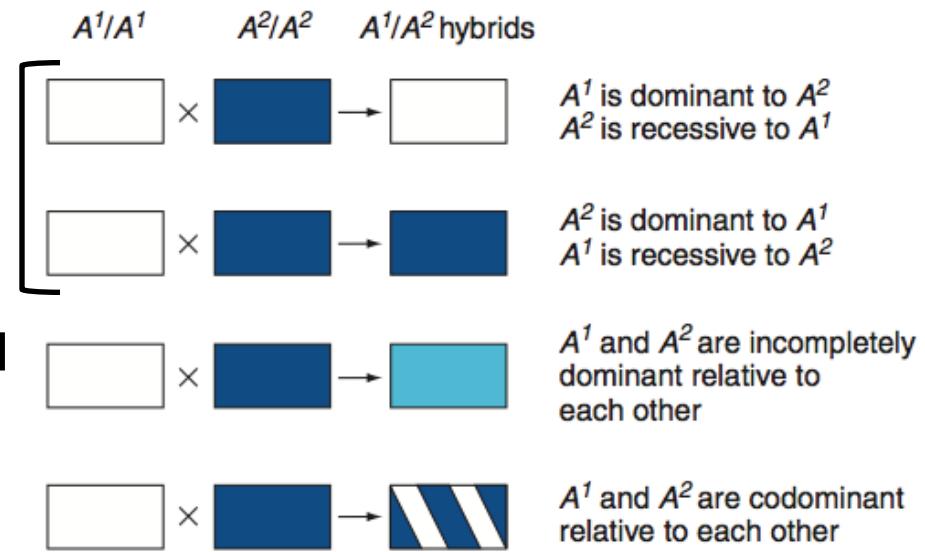


Figure 3.2

# Flower color in snapdragons is an example of incomplete dominance

Crosses of pure-breeding red with pure-breeding white results in all pink F<sub>1</sub> progeny



Figure 3.3a

# Pink flowers in snapdragons are the result of incomplete dominance

**F<sub>2</sub> progeny ratios:**

1 red (AA)

2 pink (Aa)

1 white (aa)

**Phenotype ratios reflect the genotype ratios**

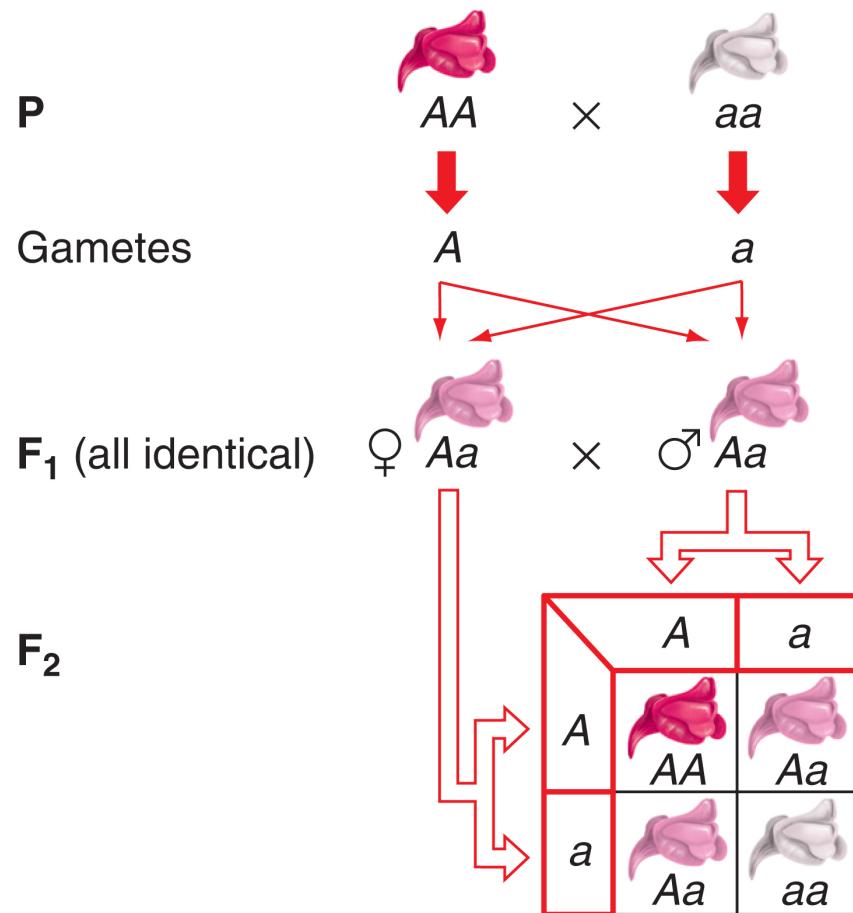


Figure 3.3b

# In codominance, the F<sub>1</sub> hybrids display traits of both parents: e.g. lentil coat patterns

SSpotted ( $C^S C^S$ ) x dotted ( $C^D C^D$ )

All F<sub>1</sub> progeny are spotted and dotted ( $C^S C^D$ )

F<sub>2</sub> progeny ratios:

1 spotted ( $C^S C^S$ )

2 spotted and dotted ( $C^S C^D$ )

1 dotted ( $C^D C^D$ )

Phenotype ratios reflect the genotype F<sub>2</sub> ratios

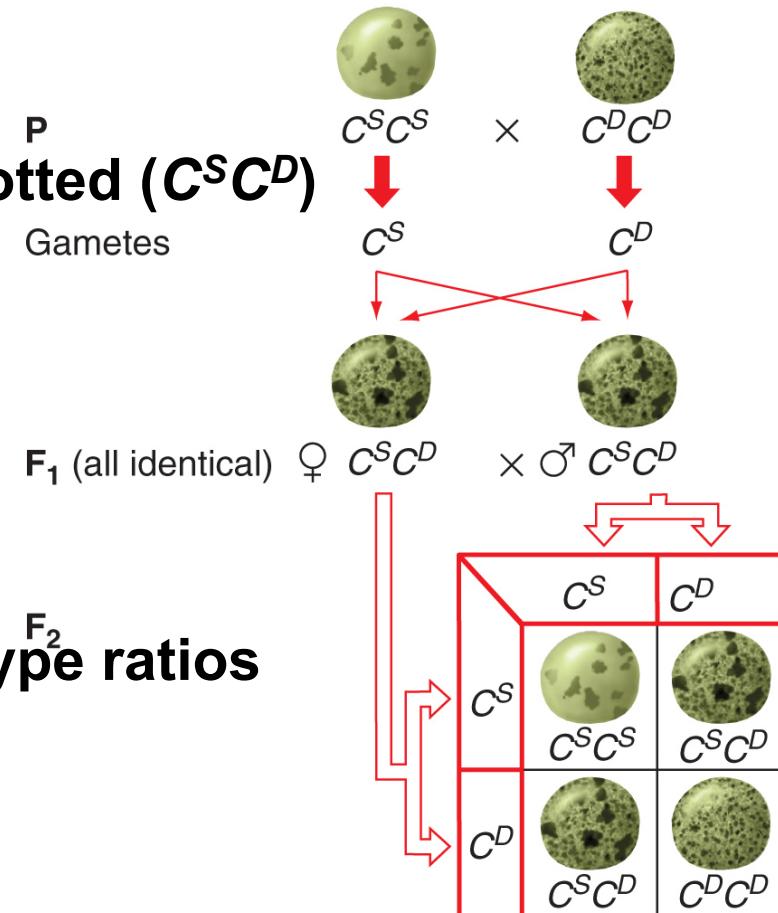


Figure 3.4a

# In codominance, the F<sub>1</sub> hybrids display traits of both parents: e.g. AB blood group

Gene *I* controls the type of sugar polymer on surface of RBCs

Two alleles, *I<sup>A</sup>* and *I<sup>B</sup>*, result in different sugars

- *I<sup>A</sup> I<sup>A</sup>* individuals have A sugar
- *I<sup>B</sup> I<sup>B</sup>* individuals have B sugar
- *I<sup>A</sup> I<sup>B</sup>* individuals have both A and B sugars

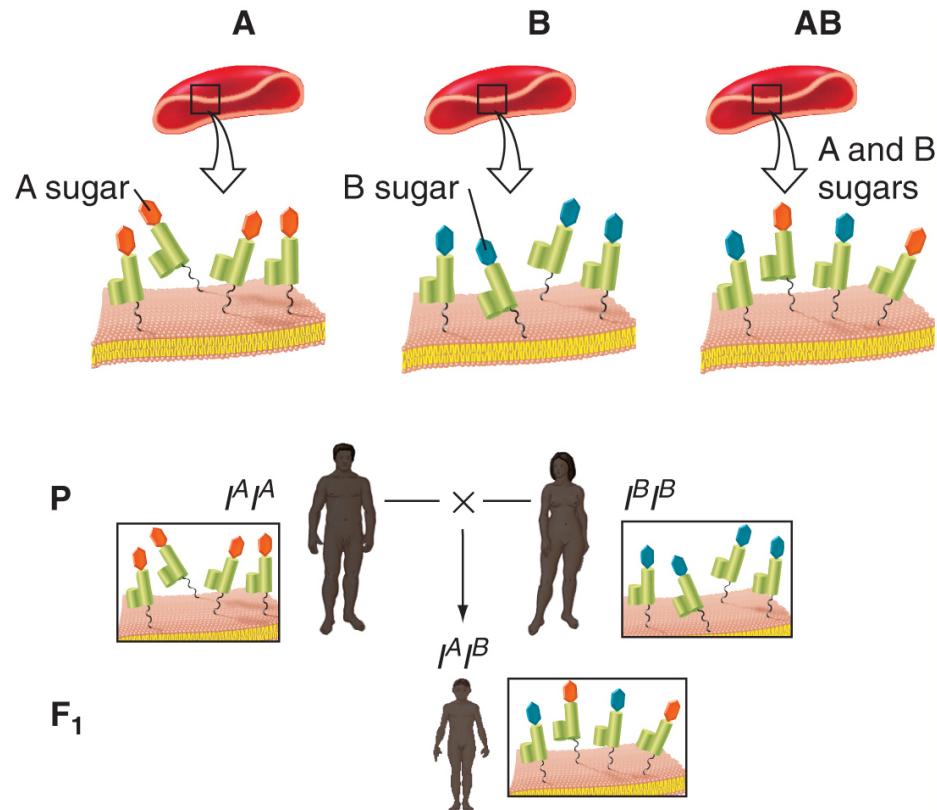


Figure 3.4b

# **Dominance relations between alleles do not affect transmission of alleles**

**Type of dominance (complete, incomplete dominance, codominance) depends on the type of proteins encoded and by the biochemical functions of the proteins**

**Variation in dominance relations do not negate Mendel's laws of segregation**

**Alleles still segregate randomly**

**Interpretation of phenotype/genotype relations is more complex**

# **A gene can have more than two alleles**

**Multiple alleles of a gene can segregate in populations**

**Each individual can carry only two alleles**

**Dominance relations are always relative to a second allele  
and are unique to a pair of alleles**

# ABO blood types in humans are determined by three alleles of one gene

$I^A$  allele → A type sugar

$I^B$  allele → B type sugar

$i$  allele → no sugar

Genotypes	Corresponding Phenotypes: Type(s) of Molecule on Cell
$I^A/I^A$ $I^A/i$	A
$I^B/I^B$ $I^B/i$	B
$I^A/I^B$	AB
$ii$	O

Six genotypes produce four blood types

Fig. 3.5 a

Dominance relations are relative to a second allele

- $I^A$  and  $I^B$  are codominant
- $I^A$  and  $I^B$  are dominant to  $i$

# Medical and legal implications of ABO blood group genetics

**Antibodies are made against type A and type B sugars**

- **Successful blood transfusions occur only with matching blood types**
- **Type AB are universal recipients, type O are universal donors**

Blood Type	Antibodies in Serum
A	Antibodies against B
B	Antibodies against A
AB	No antibodies against A or B
O	Antibodies against A and B

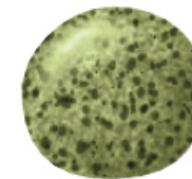
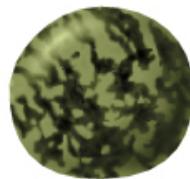
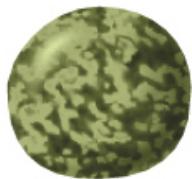
Blood Type of Recipient	Donor Blood Type (Red Cells)			
	A	B	AB	O
A	+	-	-	+
B	-	+	-	+
AB	+	+	+	+
O	-	-	-	+

Figure 3.5 b,c

# Seed coat patterns in lentils are determined by a gene with five alleles

Five alleles for C gene: spotted ( $C^S$ ), dotted ( $C^D$ ), clear ( $C^C$ ), marbled-1 ( $C^{M1}$ ), and marbled-2 ( $C^{M2}$ )

Reciprocal crosses between pairs of pure-breeding lines is used to determine dominance relations (see Fig 3.6)



marbled-1 > marbled-2 > spotted = dotted > clear

Fig. 3.6

# Dominance relations are established between pairs of alleles

## Three examples from Figure 3.6

- 1) marbled-1 ( $C^{M1}C^{M1}$ ) x clear ( $C^cC^c$ )  $\diamond$  all  $F_1$  marbled-1 ( $C^{M1}C^c$ )  $F_2$  progeny: 798 marbled-1 ( $C^{M1}—$ ) and 296 clear ( $C^cC^c$ )
- 2) marbled-2 ( $C^{M2}C^{M2}$ ) x clear ( $C^cC^c$ )  $\diamond$  all  $F_1$  marbled-2 ( $C^{M2}C^c$ )  $F_2$  progeny: 123 marbled-1 ( $C^{M2}—$ ) and 46 clear ( $C^cC^c$ )
- 3) marbled-1 ( $C^{M1}C^{M1}$ ) x marbled-2 ( $C^{M2}C^{M2}$ )  $\diamond$  all  $F_1$  marbled-1  
 $F_2$  progeny: 272 marbled-1 ( $C^{M1}—$ ) and 72 marbled-2 ( $C^{M2}C^{M2}$ )  
3:1 ratio in each cross indicates that different alleles of the same gene are involved

Dominance series:  $C^{M1} > C^{M2} > C^c$

# **Human histocompatibility antigens are an extreme example of multiple alleles**

**Three major genes (*HLA-A*, *HLA-B*, and *HLA-C*) encode histocompatibility antigens**

- **Cell surface molecules present on all cells except RBCs and sperm**
- **Facilitates proper immune response to foreign antigens (e.g. virus or bacteria)**

**Each gene has 20-to-100 alleles each**

- **Each allele is codominant to every other allele**
- **Every genotype produces a distinct phenotype**
- **Enormous phenotypic variation**

# Mutations are the source of new alleles

**Chance alterations of genetic material arise spontaneously**

**If mutations occur in gamete-producing cells, they can be transmitted to offspring**

**Frequency of gametes with mutations is  $10^{-4}$ - $10^{-6}$**

**Mutations that result in phenotypic variants can be used by geneticists to follow gene transmission**

**Molecular basis of mutations described in Chapter 7**

# Nomenclature for alleles in populations

**Allele frequency** is the percentage of the total number of gene copies for one allele in a population

Most common allele is usually the wild-type (+) allele

Rare allele is considered a mutant allele

Gene w/ only one common wild-type allele is **monomorphic**

- *Agouti* gene in mice – only one allele in wild populations, many alleles in lab mice

Gene w/ more than one common allele is **polymorphic**

- High-frequency alleles of polymorphic genes are common variants
- Extreme example – 92 plant incompatibility alleles (Fig. 3.8)

# The mouse *agouti* gene controls hair color: One wild-type allele, many mutant alleles

Wild-type *agouti* allele (*A*) produces yellow and black pigment in hair

14 different *agouti* alleles in lab mice, but only *A* allele in wild mice

e.g. mutant alleles *a* and *a<sup>t</sup>*

- ***a* recessive to *A***
  - *aa* has black only
- ***a<sup>t</sup>* dominant to *a* but recessive to *A***
  - *a<sup>t</sup>a<sup>t</sup>* mouse has black on back and yellow on belly

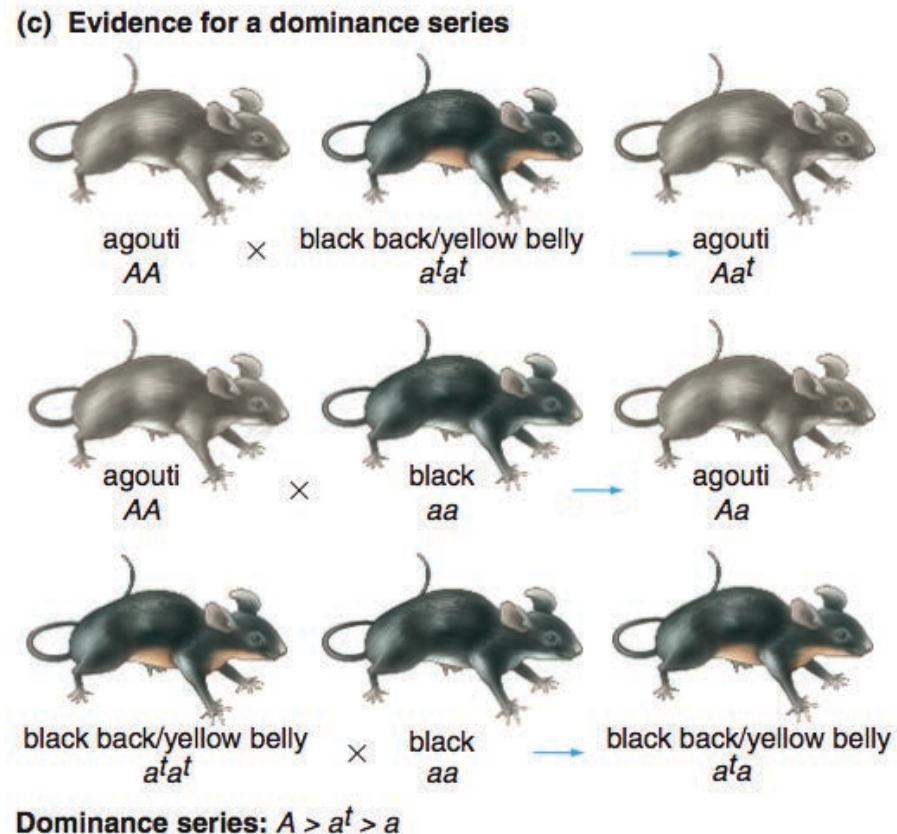


Fig. 3.7c

# One gene may contribute to several characteristics

Pleiotropy is the phenomenon of a single gene determining several distinct and seemingly unrelated characteristics

- e.g. Many aboriginal Maori men have respiratory problems and are sterile
  - Defects due to mutations in a gene required for functions of cilia (failure to clear lungs) and flagella (immotile sperm)

With some pleiotropic genes

- Heterozygotes can have a visible phenotype
- Homozygotes can be inviable (e.g. AY allele of agouti gene in mice, see Fig 3.9)

# The $A^Y$ allele produces a dominant coat color phenotype in mice

$A^Y$  allele of *agouti* gene causes yellow hairs with no black

Cross agouti x yellow mice

- Progeny in 1:1 ratio of agouti to yellow
- Yellow mice must be heterozygous for  $A$  and  $A^Y$
- $A^Y$  is dominant to  $A$

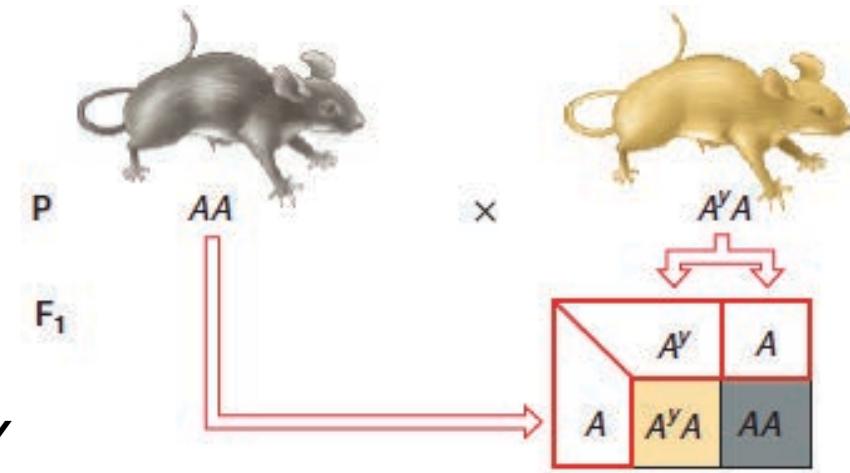


Fig. 3.9a

# The $A^Y$ allele is a recessive lethal allele

$A^Y$  is dominant to  $A$  for hair color, but is recessive to  $A$  for lethality

Cross yellow x yellow mice

- $F_1$  mice are 2/3 yellow and 1/3 agouti

2:1 ratio is indicative of a recessive lethal allele

- Pure-breeding yellow ( $A^Y A^Y$ ) mice cannot be obtained because they are not viable

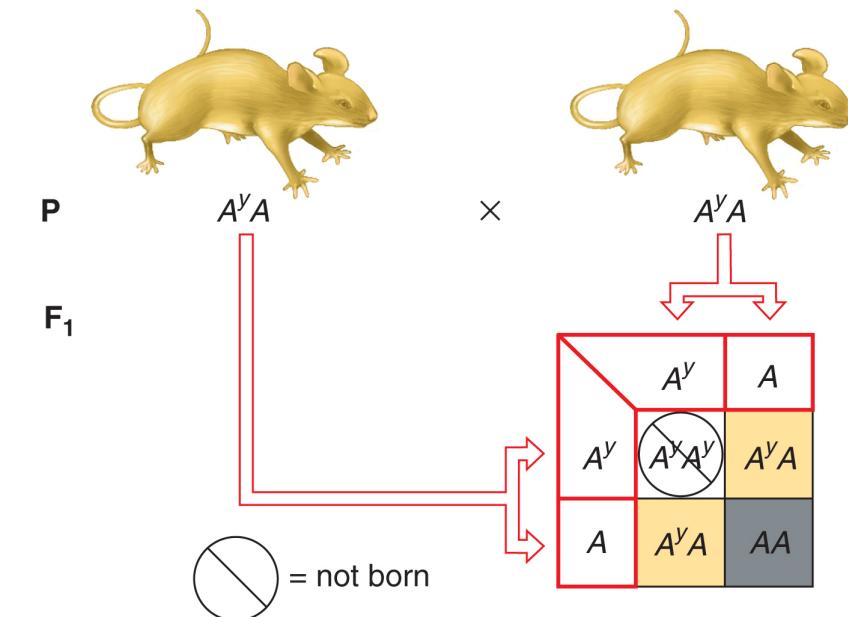


Figure 3.9b

# Extensions to Mendel's analysis explain alterations of the 3:1 monohybrid ratio

What Mendel Described	Extension	Extension's Effect on Heterozygous Phenotype	Extension's Effect on Ratios Resulting from an $F_1 \times F_1$ Cross
Complete dominance	Incomplete dominance Codominance	Unlike either homozygote	Phenotypes coincide with genotypes in a ratio of 1:2:1
Two alleles	Multiple alleles	Multiplicity of phenotypes	A series of 3:1 ratios
All alleles are equally viable	Recessive lethal alleles	No effect	2:1 instead of 3:1
One gene determines one trait	Pleiotropy: one gene influences several traits	Several traits affected in different ways, depending on dominance relations	Different ratios, depending on dominance relations for each affected trait

Table 3.1

# A comprehensive example: Sickle-cell disease

Hemoglobin transports oxygen in RBCs

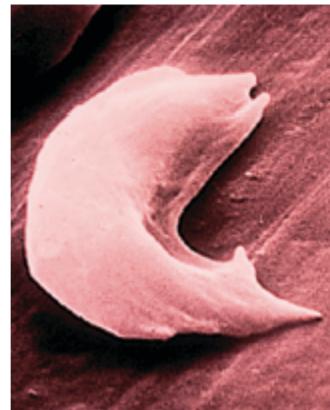
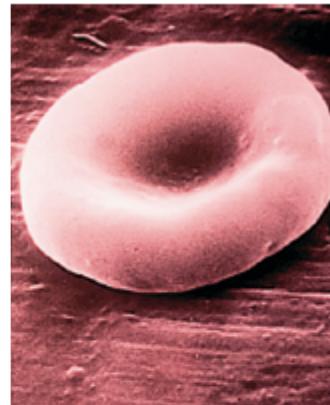
- Two subunits – alpha ( $\alpha$ ) globin and beta ( $\beta$ ) globin

Mutations in  $\beta$ -globin gene cause  $\beta$ -thalassemia

Most common mutation of  $\beta$ -globin (Hb $\beta$ S) causes sickle-cell disease

- Pleiotropic – affects >1 trait (deformed RBCs, anemia, heart failure, resistance to malaria)
- Recessive lethality – heart failure
- Different dominance relations for different phenotypic aspects of sickle-cell disease (see Figure 3.10)

# Pleiotropy of sickle-cell anemia: Dominance relations vary with the phenotype under consideration



Phenotypes at Different Levels of Analysis	Normal <i>Hb<math>\beta</math>A</i> <i>Hb<math>\beta</math>A</i>	Carrier <i>Hb<math>\beta</math>A</i> <i>Hb<math>\beta</math>S</i>	Diseased <i>Hb<math>\beta</math>S</i> <i>Hb<math>\beta</math>S</i>	Dominance Relations at Each Level of Analysis
$\beta$ -globin polypeptide production				<i>Hb<math>\beta</math>A</i> and <i>Hb<math>\beta</math>S</i> are codominant
Red blood cell shape at sea level				<i>Hb<math>\beta</math>A</i> is dominant <i>Hb<math>\beta</math>S</i> is recessive
Red blood cell concentration at sea level				<i>Hb<math>\beta</math>A</i> and <i>Hb<math>\beta</math>S</i> show incomplete dominance
Red blood cell shape at high altitudes				<i>Hb<math>\beta</math>S</i> is dominant <i>Hb<math>\beta</math>A</i> is recessive
Red blood cell concentration at high altitudes				
Susceptibility to malaria				

Fig. 3.10

## CHAPTER OUTLINE

- **Extensions to Mendel's laws**
- 3.1 Extensions to Mendel for Single-Gene Inheritance
- 3.2 Extensions to Mendel for Multi-factorial Inheritance

# Extensions to Mendel for multi-factorial inheritance

Two genes can interact to determine one trait

- **Novel phenotypes** can result from gene interactions, e.g. seed coat in lentils
- **Complementary gene action**, e.g. flower color
- **Epistasis**, e.g. dog fur, Bombay phenotype in humans, squash color, chicken feather color

In all of these cases,  $F_2$  phenotypes from dihybrid crosses are in a **variation of the 9:3:3:1 ratio** expected for independently assorting genes

# Novel phenotypes resulting from gene interactions, e.g. seed coat in lentils

Dihybrid cross of lentils,  
tan x gray

All F<sub>1</sub> seeds are brown

F<sub>2</sub> progeny:

- 9/16 brown
- 3/16 tan
- 3/16 gray
- 1/16 green

9:3:3:1 ratio in F<sub>2</sub> suggests two independently assorting genes for seed coat color

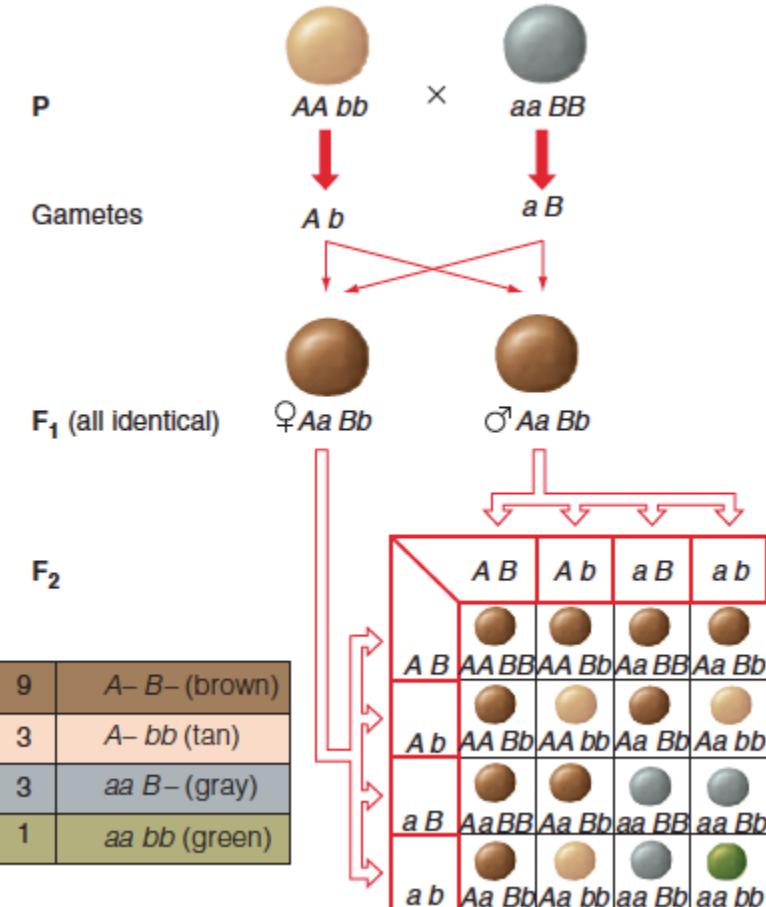


Fig. 3.11a

# Results of self-crosses of $F_2$ lentils supports the two-gene hypothesis

Phenotypes of $F_2$ Individual	Observed $F_3$ Phenotypes	Expected Proportion of $F_2$ Population*
Green	Green	1/16
Tan	Tan	1/16
Tan	Tan, green	2/16
Gray	Gray, green	2/16
Gray	Gray	1/16
Brown	Brown	1/16
Brown	Brown, tan	2/16
Brown	Brown, gray	2/16
Brown	Brown, gray, tan, green	4/16

Figure 3.11b

\*This 1: 1: 2: 2: 1: 2: 2: 4  $F_2$  genotypic ratio corresponds to a 9 brown: 3 tan: 3 gray: 1 green  $F_2$  phenotypic ratio

# Sorting out the dominance relations by select crosses of lentils

**F<sub>2</sub> phenotypes from dihybrid crosses will be in 9:3:3:1 ratio  
only when dominance of alleles at both genes is complete**

Seed Coat Color of Parents	F <sub>2</sub> Phenotypes and Frequencies	Ratio
Tan × green	231 tan, 85 green	3:1
Gray × green	2586 gray, 867 green	3:1
Brown × gray	964 brown, 312 gray	3:1
Brown × tan	255 brown, 76 tan	3:1
Brown × green	57 brown, 18 gray, 13 tan, 4 green	9:3:3:1

Figure 3.11c

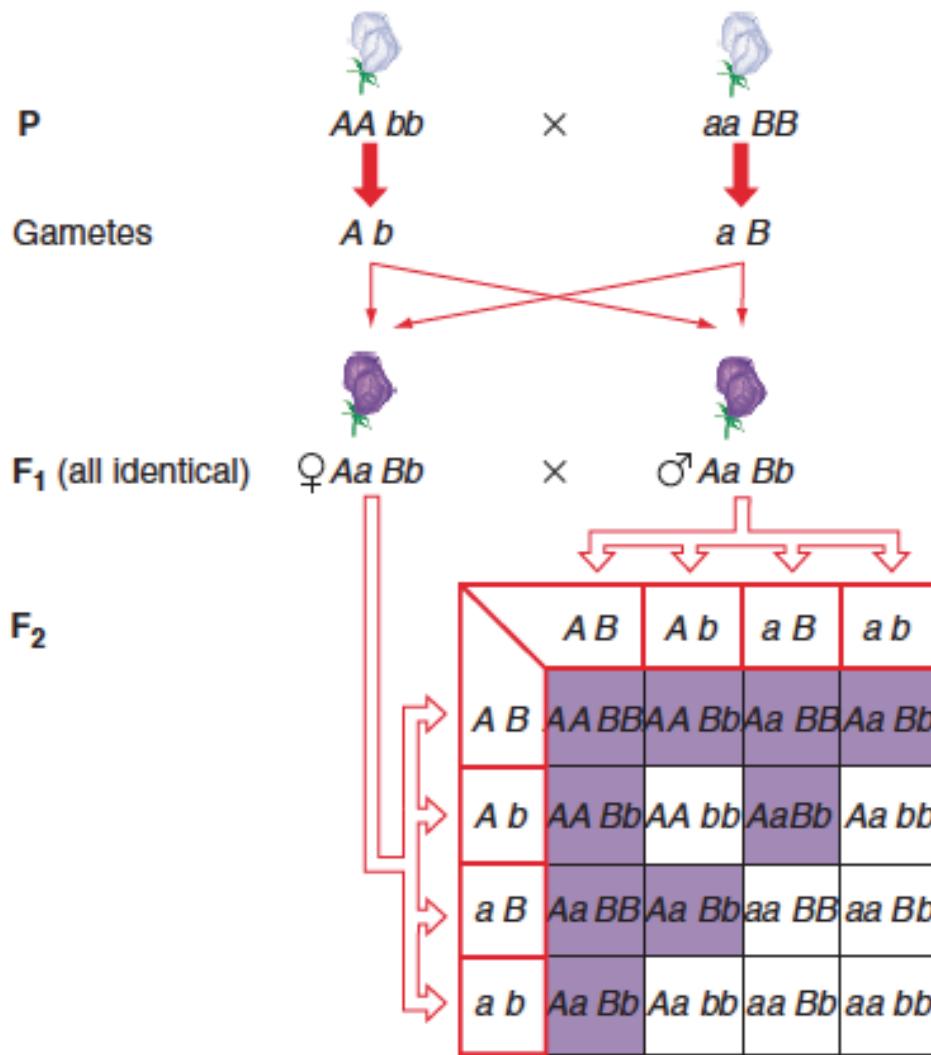
# Complementary gene action in sweet peas

Purple F<sub>1</sub> progeny are produced by crosses of two pure-breeding white lines



Figure 3.12a

# Complementary gene action generates purple flower color in sweet peas

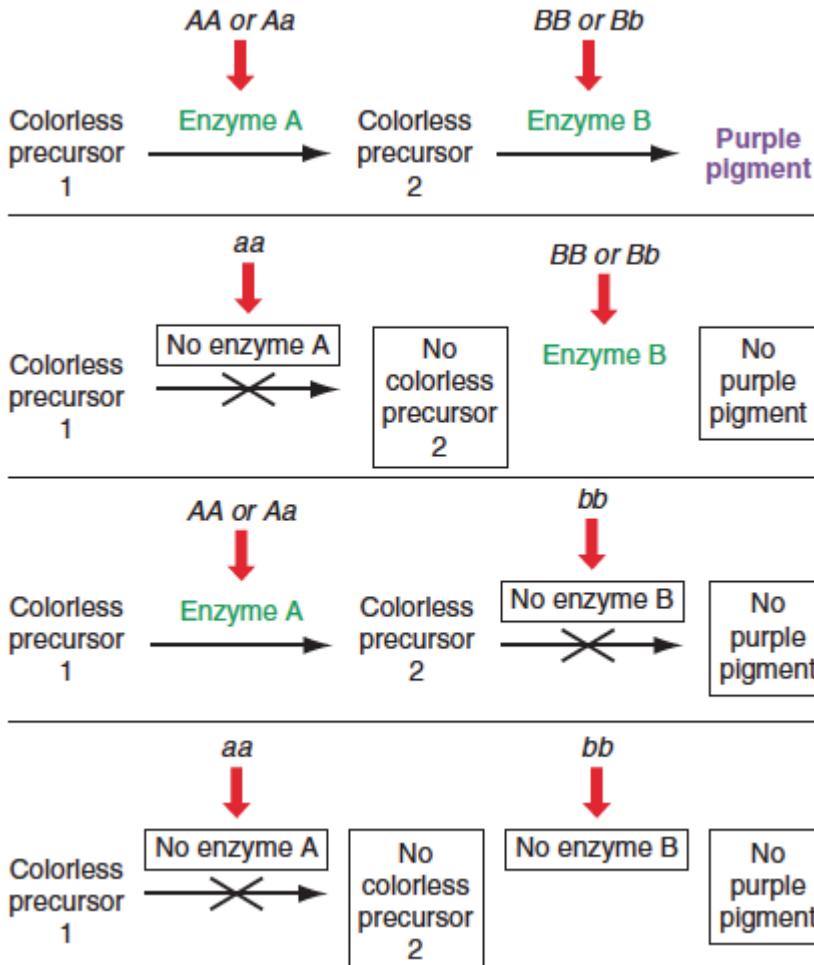


Dihybrid cross generates  
9:7 ratio in F<sub>2</sub> progeny

9/16 purple ( $A\ B\ —$ )  
7/16 white ( $A\ —\ bb$ ,  $aa\ B\ —$ ,  
 $aa\ bb$ )

Figure 3.12b

# Possible biochemical explanation for complementary gene action for flower color in sweet peas



**One pathway has two reactions catalyzed by different enzymes**

- At least one dominant allele of both genes is required for purple pigment
- Homozygous recessive for either or both genes results in no pigment

Figure 3.13

# Epistasis results from the effects of an allele at one gene masking the effects of another gene

The gene that does the masking is **epistatic** to the other gene

The gene that is masked is **hypostatic** to the other gene

Epistasis can be recessive or dominant

- Recessive – epistatic gene must be homozygous recessive (e.g. ee)
- Dominant – epistatic gene must have at least one dominant allele present (e.g. *E*—)

# Recessive epistasis in Golden Labrador dogs

9:3:4 ratio in  $F_2$  progeny of dihybrid crosses indicates recessive epistasis

9/16 black ( $B-$   $E-$ )

3/16 brown ( $bb$   $E-$ )

4/16 yellow ( $B-$   $ee$ ,  $bb$   $ee$ )

Genotype  $ee$  masks the effect of all  $B$  genotypes

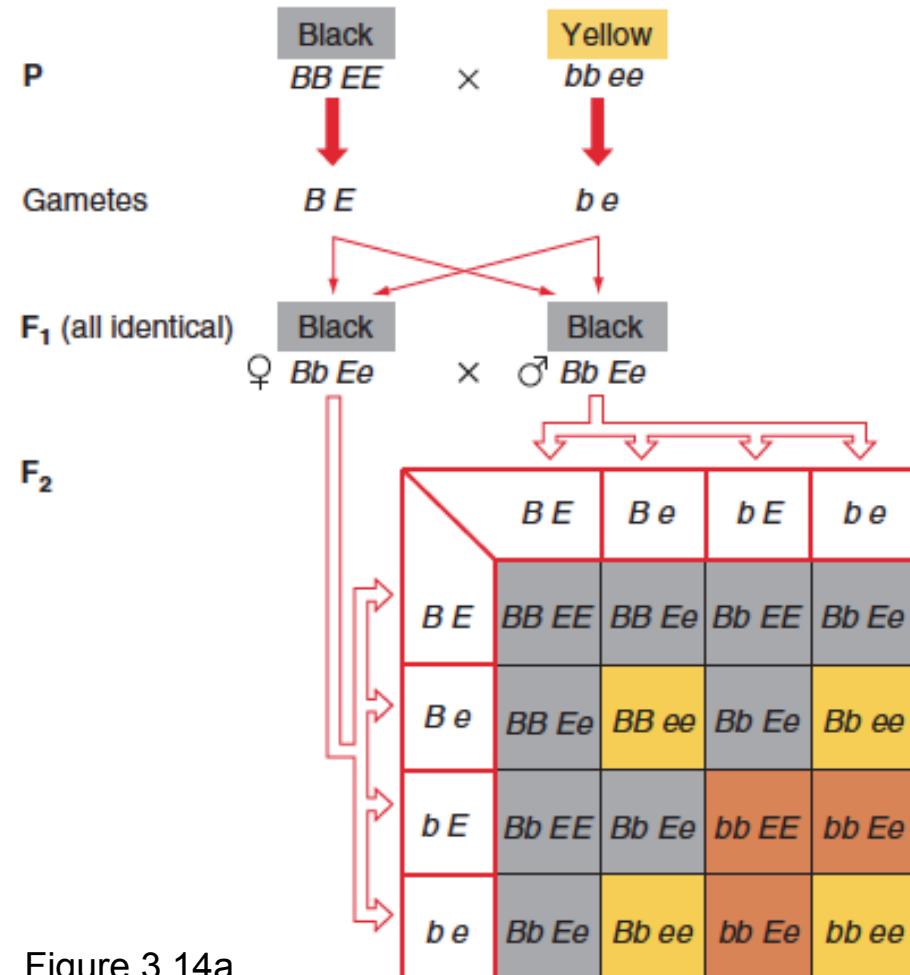


Figure 3.14a

# Recessive epistasis in humans with a rare blood type

Gene for substance H is epistatic to the ABO gene

- Without the H substance, there is nothing for the A or B sugar to attach to

All type A, type AB, type B, and type O people are *H*—

People with *hh* genotype will appear to be type O

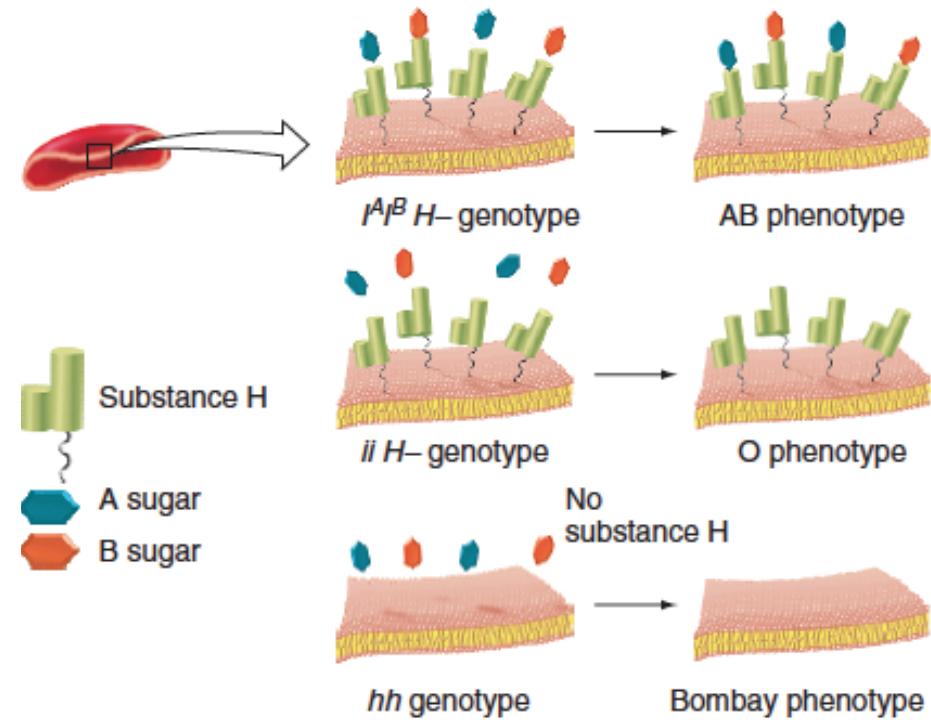


Figure 3.14b

# Dominant epistasis I in summer squash

12:3:1 ratio in  $F_2$  progeny of dihybrid crosses indicates dominant epistasis I

12/16 white ( $A-$ ,  $B-$ ,  $aa\ B-$ )

3/16 yellow ( $A-\ bb$ )

1/16 green ( $aa\ bb$ )

The dominant allele of one gene **masks both alleles of another gene**

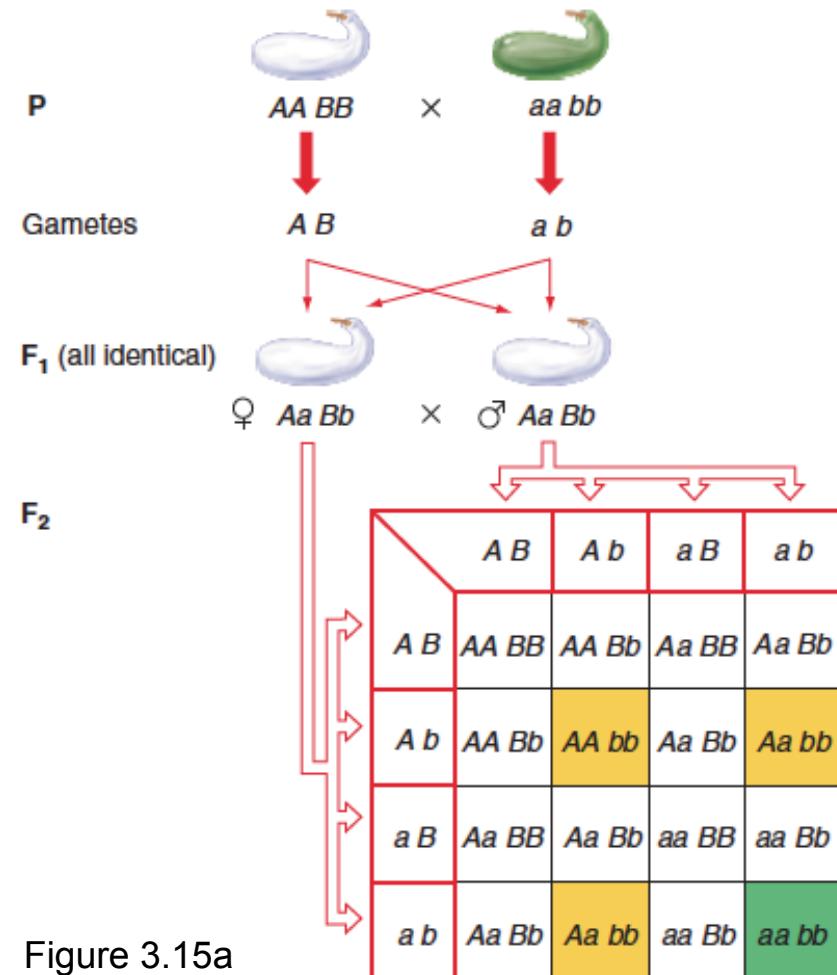


Figure 3.15a

# Dominant epistasis II in chickens

13:3 ratio in  $F_2$  progeny of dihybrid crosses indicates dominant epistasis II

13/16 white ( $A-$ ,  $B-$ ,  $aa\ B-$ ,  $aa\ bb$ )

3/16 colored ( $A-\ bb$ )

The dominant allele of one gene **masks the dominant allele of another gene**

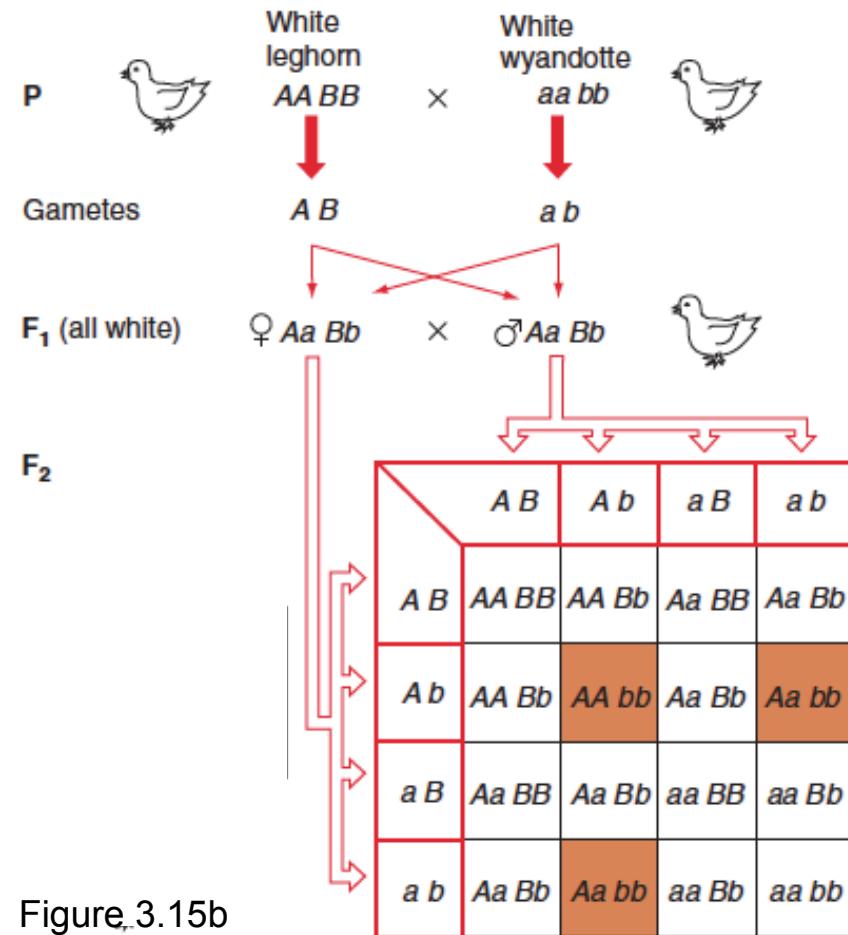


Figure 3.15b

# Summary of gene interactions discussed in this chapter

Observing the  $F_2$  ratios below is diagnostic of the type of gene interaction

- These  $F_2$  ratios occur only in dihybrid crosses where there is complete dominance

Gene Interaction	Example	F <sub>2</sub> Genotypic Ratios from an F <sub>1</sub> Dihybrid Cross				F <sub>2</sub> Phenotypic Ratio
		A- B-	A- bb	aa B-	aa bb	
None: Four distinct F <sub>2</sub> phenotypes	Lentil: seed coat color (see Fig. 3.11a)	9	3	3	1	9:3:3:1
Complementary: One dominant allele of each of two genes is necessary to produce phenotype	Sweet pea: flower color (see Fig. 3.12b)	9	3	3	1	9:7
Recessive epistasis: Homozygous recessive of one gene masks both alleles of another gene	Retriever coat color (see Fig. 3.14a)	9	3	3	1	9:3:4
Dominant epistasis I: Dominant allele of one gene hides effects of both alleles of another gene	Summer squash: color (see Fig. 3.15a)	9	3	3	1	12:3:1
Dominant epistasis II: Dominant allele of one gene hides effects of dominant allele of another gene	Chicken: feather color (see Fig. 3.15b)	9	3	3	1	13:3

# Heterogeneous traits and the complementation test

**Heterogeneous traits** have the same phenotype but are caused by mutations in different genes

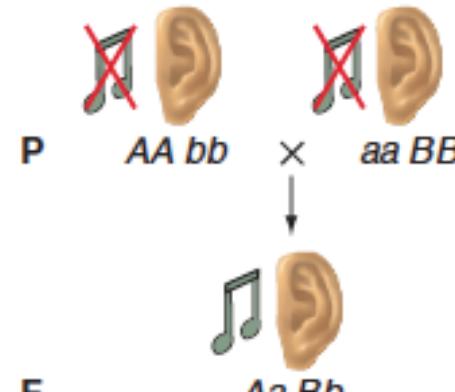
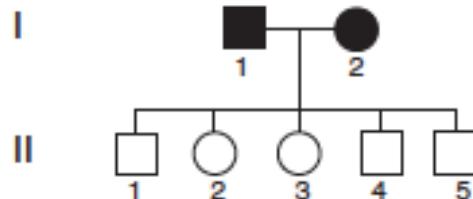
- e.g. deafness in humans can be caused by mutations in ~ 50 different genes

**Complementation testing** is used to determine if a particular phenotype arises from mutations in the same or separate genes

- Can be applied only with recessive, not dominant, phenotypes
- Discussed more in Chapter 7

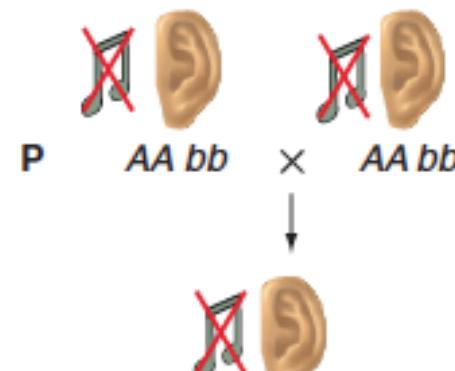
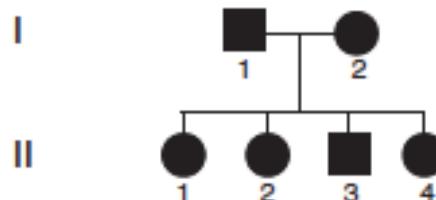
# Genetic heterogeneity in humans: Mutations in many genes can cause deafness

(a) Complementation: mutations in two different genes



Genetic mechanism of complementation

(b) Noncomplementation: mutations in the same gene



Genetic mechanism of noncomplementation

Fig. 3.16

# Breeding studies help determine inheritance of a trait

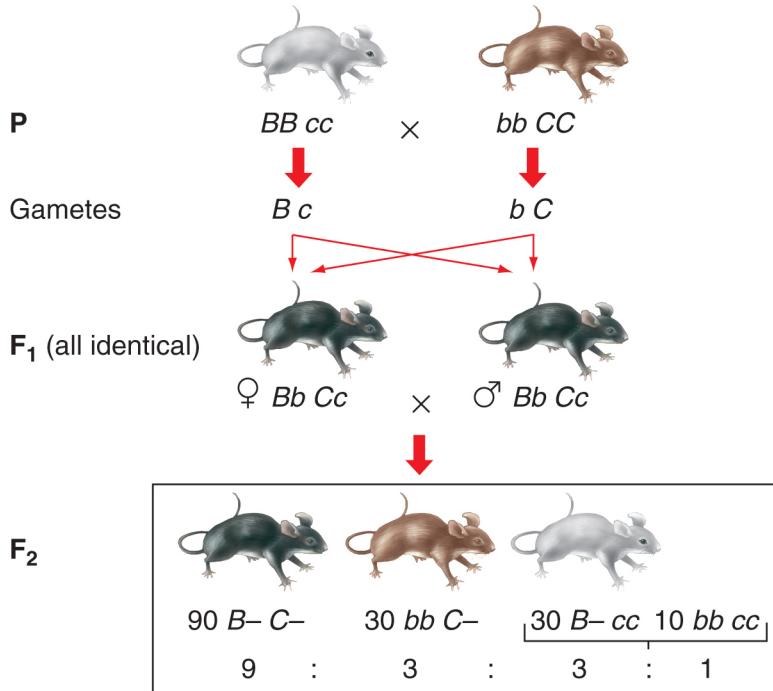
How do we know if a trait is caused by one gene or by two genes that interact?

Example: dihybrid cross of pure-breeding parents produces three phenotypes in  $F_2$  progeny

- If single gene with incomplete dominance, then  $F_2$  progeny should be in 1:2:1 ratio
- If two independently assorting genes and recessive epistasis, then  $F_2$  progeny should be in 9:3:4 ratio
- Further breeding studies can reveal which hypothesis is correct

# Two hypotheses to explain phenotypes in F<sub>2</sub> progeny of mice with different coat colors

(a) Hypothesis 1 (two genes with recessive epistasis)



(b) Hypothesis 2 (one gene with incomplete dominance)

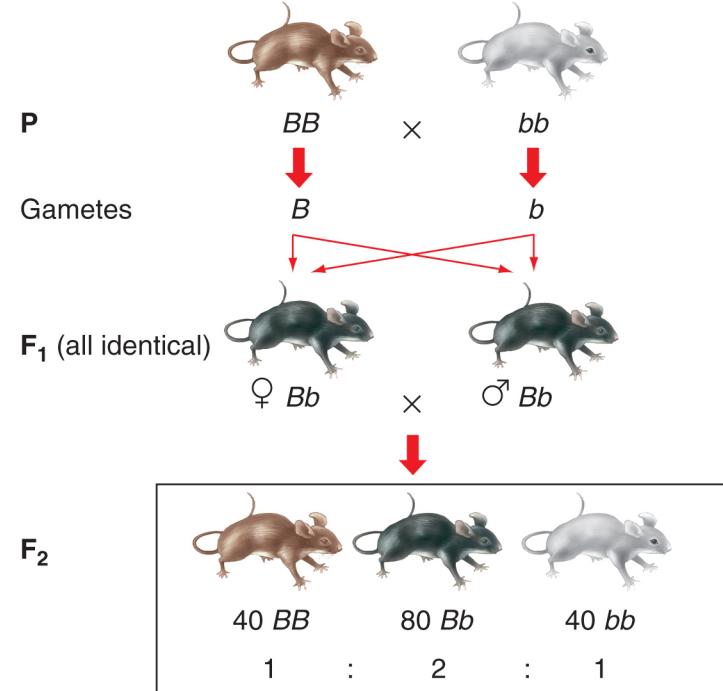


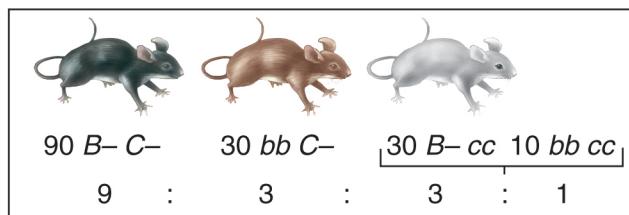
Fig. 3.18  
(top)

Are these F<sub>2</sub> progeny in a ratio of 9:3:4 or 1:2:1?

# Specific breeding tests can help decide between two hypotheses

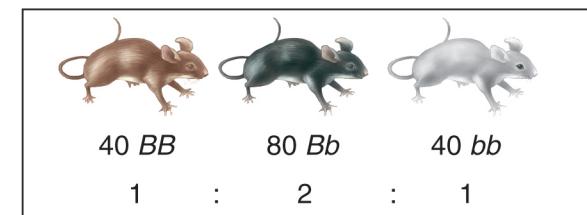
## Hypothesis 1 – two genes with recessive epistasis

$F_2$

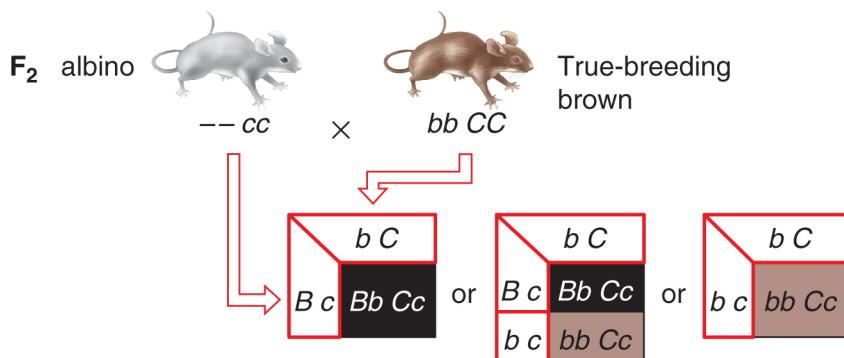


## Hypothesis 2 – one gene with incomplete dominance

$F_2$



If two-gene hypothesis is correct:



If one-gene hypothesis is correct:

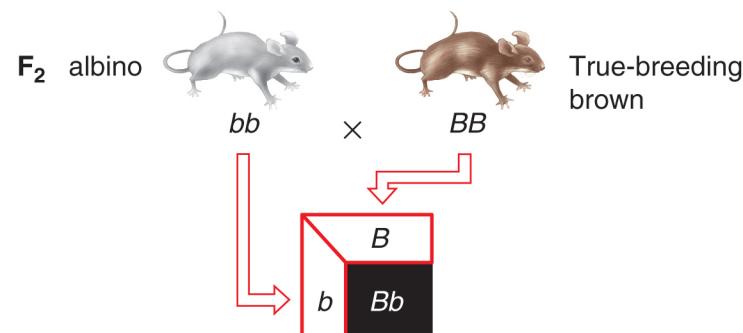


Figure 3.18 (bottom)

# **The same genotype does not always produce the same phenotype**

**In all of the traits discussed so far, the relationship between a specific genotype and its corresponding phenotype has been absolute**

**Phenotypic variation for some traits can occur because of:**

- Differences in penetrance and/or expressivity**
- Effects of modifier genes**
- Effects of environment**
- Pure chance**

# Phenotype often depends on penetrance and/or expressivity

**Penetrance** is the percentage of a population with a particular genotype that shows the expected phenotype

- Can be complete (100%) or incomplete (e.g. penetrance of retinoblastoma is 75%)

**Expressivity** is the degree or intensity with which a particular genotype is expressed in a phenotype

- Can be variable or unvarying

# Some traits result from different genes that do not contribute equally to the phenotype

**Modifier genes** alter the phenotypes produced by alleles of other genes

- Can have major effect or more subtle effects

Example: T locus of mice

- Mutant *T* allele causes abnormally short tail
- In some inbred strains, mice with *T* allele have tails that are 75% the length of normal tails
- In other inbred strains, mice with the same *T* mutation have tails that are 10% the length of normal tails
- Different inbred strains must carry alternative alleles of a modifier gene for the *T* mutant phenotype

# Environmental effects on phenotype

**Temperature is a common element of the environment that can affect phenotype**

- Example 1: Coat color in Siamese cats
  - Extremities are darker than body because of a temperature sensitive allele
- Example 2: Survivability of a *Drosophila* mutant
  - *Shibire* mutants develop normally at  $< 29^{\circ}\text{C}$  but are inviable at temperatures  $> 29^{\circ}\text{C}$

**Conditional lethal** mutations are lethal only under some conditions

- **Permissive conditions** - mutant allele has wild-type functions
- **Restrictive conditions** - mutant allele has defective functions

# A temperature sensitive mutation affects coat color in Siamese cats

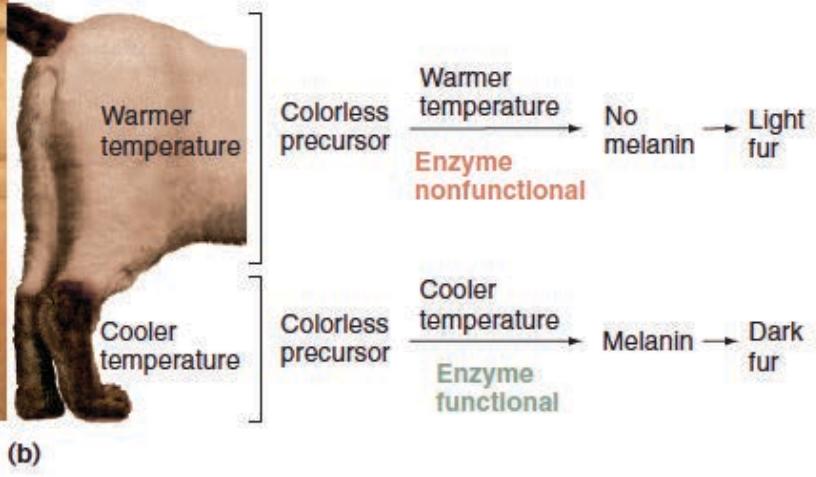


Fig. 3.20

# Other effects of environment on phenotype

**Phenocopy** - phenotype arising from an environmental agent that mimics the effect of a mutant gene

- Not heritable
- Can be deleterious or beneficial
- Examples in humans
  - Thalidomide produced a phenocopy of phocomelia, a rare dominant trait
  - Children with heritable PKU can receive a protective diet
  - Genetic predisposition to cardiovascular disease can be influenced by diet and exercise
  - Genetic predisposition to lung cancer is strongly affected by cigarette smoking

# Mendelian principles can also explain continuous variation

**Discontinuous traits** give clear-cut, "either-or" phenotypic differences between alternative alleles

- Example: All of the traits Mendel studied in peas were discontinuous

**Continuous traits** are determined by segregating alleles of many genes that interact together and with the environment

- Examples in humans: height, weight, skin color
- Often appear to blend and "unblend"
- Also called **quantitative traits** because the traits vary over a range that can be measured
- Usually **polygenic** – controlled by multiple genes

# Two continuous traits in human

Height is a continuous trait



Skin color is a continuous trait

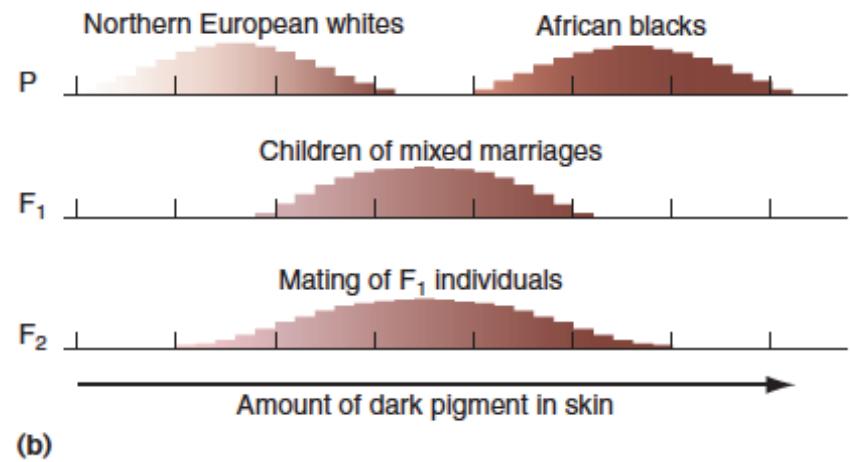


Fig. 3.21

# Mendelian explanation of continuous variation

The more genes or alleles,  
the more possible  
phenotypic classes and the  
greater the similarity to  
continuous variation

In these examples, all of the  
alleles are incompletely  
dominant and have additive  
effects

	$A^1$	$A^0$
$A^1$	2	1
$A^0$	1	0

(a) 1 gene with  
2 alleles  
yields 3  
phenotypic  
classes.

$A^1B^1$	$A^1B^0$	$A^0B^1$	$A^0B^0$
$A^1B^1$	$A^1B^0$	$A^0B^1$	$A^0B^0$
$A^1B^1$	$A^1B^0$	$A^0B^1$	$A^0B^0$
$A^1B^0$	4	3	3
$A^1B^0$	3	2	2
$A^0B^1$	3	2	2
$A^0B^0$	2	1	1

(b) 2 genes with 2  
alleles apiece  
yield 5  
phenotypic  
classes.

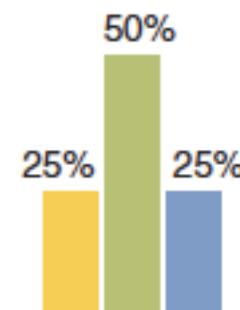
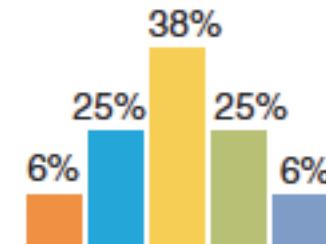


Fig. 3.22 (partial)



# Mendelian explanation of continuous variation (continued)

The more genes or alleles, the more possible phenotypic classes and the greater the similarity to continuous variation

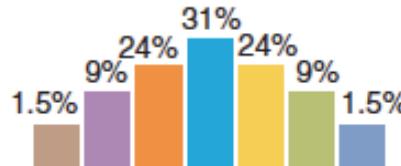
In these examples,  
all of the alleles are  
incompletely  
dominant and have  
additive effects

[END]

Fig. 3.22 (partial)

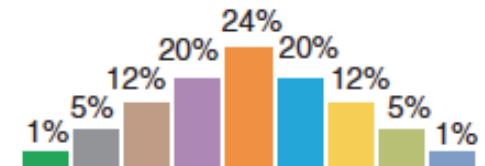
A <sup>2</sup> B <sup>2</sup> C <sup>2</sup>	A <sup>2</sup> B <sup>2</sup> C <sup>1</sup>	A <sup>2</sup> B <sup>2</sup> C <sup>0</sup>	A <sup>1</sup> B <sup>2</sup> C <sup>2</sup>	A <sup>1</sup> B <sup>2</sup> C <sup>1</sup>	A <sup>1</sup> B <sup>2</sup> C <sup>0</sup>	A <sup>0</sup> B <sup>2</sup> C <sup>2</sup>	A <sup>0</sup> B <sup>2</sup> C <sup>1</sup>	A <sup>0</sup> B <sup>2</sup> C <sup>0</sup>
A <sup>2</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>2</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>2</sup> B <sup>1</sup> C <sup>0</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>0</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>0</sup>
A <sup>2</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>2</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>2</sup> B <sup>0</sup> C <sup>0</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>0</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>0</sup>
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A <sup>2</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>2</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>2</sup> B <sup>1</sup> C <sup>0</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>0</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>0</sup>
A <sup>2</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>2</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>2</sup> B <sup>0</sup> C <sup>0</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>0</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>0</sup>
A <sup>2</sup> B <sup>2</sup> C <sup>2</sup>	A <sup>2</sup> B <sup>2</sup> C <sup>1</sup>	A <sup>2</sup> B <sup>2</sup> C <sup>0</sup>	A <sup>1</sup> B <sup>2</sup> C <sup>2</sup>	A <sup>1</sup> B <sup>2</sup> C <sup>1</sup>	A <sup>1</sup> B <sup>2</sup> C <sup>0</sup>	A <sup>0</sup> B <sup>2</sup> C <sup>2</sup>	A <sup>0</sup> B <sup>2</sup> C <sup>1</sup>	A <sup>0</sup> B <sup>2</sup> C <sup>0</sup>
A <sup>2</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>2</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>2</sup> B <sup>1</sup> C <sup>0</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>1</sup> B <sup>1</sup> C <sup>0</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>2</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>1</sup>	A <sup>0</sup> B <sup>1</sup> C <sup>0</sup>
A <sup>2</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>2</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>2</sup> B <sup>0</sup> C <sup>0</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>1</sup> B <sup>0</sup> C <sup>0</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>2</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>1</sup>	A <sup>0</sup> B <sup>0</sup> C <sup>0</sup>

(c) 3 genes with 2 alleles yield 7 phenotypic classes.



A <sup>2</sup> B <sup>2</sup>	A <sup>2</sup> B <sup>1</sup>	A <sup>2</sup> B <sup>0</sup>	A <sup>1</sup> B <sup>2</sup>	A <sup>1</sup> B <sup>1</sup>	A <sup>1</sup> B <sup>0</sup>	A <sup>0</sup> B <sup>2</sup>	A <sup>0</sup> B <sup>1</sup>	A <sup>0</sup> B <sup>0</sup>
8	7	7	7	6	6	6	5	4
7	6	6	5	5	5	5	4	3
7	6	6	5	5	5	5	4	3
6	5	5	4	4	4	3	3	2
6	5	5	4	4	4	3	3	2
6	5	5	4	4	4	3	3	2
6	5	5	4	4	4	3	3	2
5	4	4	3	3	3	3	2	1
5	4	4	3	3	3	3	2	1
4	3	3	2	3	2	2	2	1
4	3	3	2	3	2	2	2	1
3	2	2	1	2	1	1	1	0

(d) 2 genes with 3 alleles apiece yield 9 phenotypic classes.



# Chapter 3 Questions