Chapter 12: Patterns of Inheritance

I. Genes
II. Gregor Mendel & Mendelian Genetics
III. How do genes relate to traits?
IV. How are multiple traits inherited?
V. How is sex determined?
VI. Variations on Mendelian genetics
VII. Genetic Disorders

I. Genes

A. Gene’s location on chromosome = Locus
   - Homologous chromosomes (homologues) carry same genes at same loci

Humans have 23 homologous pairs
I. Genes

A. Gene’s location on chromosome = **Locus**
   - Homologous chromosomes (homologues) carry same genes at same loci

B. **Alleles** = similar sequences at same locus on homologous chromosomes (= alternative forms of a gene)
   - **Homozygous** individual: same allele at a given gene locus
   - **Heterozygous** individual: different alleles at a given locus

Examples:

- **M locus**: carries M gene: leaf color
  - this plant is **homozygous** at the M locus

- **D locus**: carries D gene: plant height
  - this plant is **homozygous** at the D locus

- **Bk locus**: carries Bk gene: fruit shape
  - this plant is **heterozygous** at the Bk locus
II. Gregor Mendel & Mendelian Genetics

“Father” of modern Genetics

- Attended the University of Vienna, where he failed his qualifying exams for teacher certification
- Austrian Monk
- Studied the Garden Pea at the monastery
- First quantitative experiments in genetics
- Conducted during the mid 1800’s
- No knowledge of chromosomes, genes, DNA, cellular patterns of inheritance, or meiosis
- Mendel’s work not “discovered” until after his death in 1900

II. Gregor Mendel & Mendelian Genetics

A. Study species: pea plants:
   - can self-fertilize
   - can also be cross-fertilized
   - studied individual heritable traits i.e. flower color

B. Mendel grew varieties that were true-breeding for traits:

   purple flowers X purple flowers = purple flowers
   white flowers X white flowers = white flowers
Traits used by Mendel had 2 Contrasting Forms

The seven pea characteristics studied by Mendel

<table>
<thead>
<tr>
<th>Flower color</th>
<th>Purple</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flower position</td>
<td>Axial</td>
<td>Terminal</td>
</tr>
<tr>
<td>Seed color</td>
<td>Yellow</td>
<td>Green</td>
</tr>
<tr>
<td>Seed shape</td>
<td>Round</td>
<td>Wrinkled</td>
</tr>
<tr>
<td>Pod shape</td>
<td>Inflated</td>
<td>Constricted</td>
</tr>
<tr>
<td>Pod color</td>
<td>Green</td>
<td>Yellow</td>
</tr>
<tr>
<td>Stem length</td>
<td>Tall</td>
<td>Dwarf</td>
</tr>
</tbody>
</table>
Mendel’s Research

What happened to the white color?

Mendel’s Research

Pollen transferred from white flower to stigma of purple flower

Parental generation

Anthers removed

All purple flowers result

F1 generation

Mendel’s Research

self-fertilize

First-generation offspring ($F_1$)

3/4 purple

Second-generation offspring ($F_2$)

1/4 white

Ratio = 3 purple: 1 white
### Results of Mendel’s Crosses

<table>
<thead>
<tr>
<th>Trait</th>
<th>Dominant vs. recessive</th>
<th>F&lt;sub&gt;2&lt;/sub&gt; generation</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Dominant form</td>
<td>Recessive form</td>
</tr>
<tr>
<td>Flower color</td>
<td>Purple X White</td>
<td>705</td>
<td>224</td>
</tr>
<tr>
<td>Seed color</td>
<td>Yellow X Green</td>
<td>6,022</td>
<td>2,001</td>
</tr>
<tr>
<td>Seed shape</td>
<td>Round X Wrinkled</td>
<td>5,474</td>
<td>1,850</td>
</tr>
<tr>
<td>Pod color</td>
<td>Green X Yellow</td>
<td>428</td>
<td>152</td>
</tr>
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### Results of Mendel’s Crosses

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<td>Pod shape</td>
<td>Round X Constricted</td>
<td>882</td>
<td>299</td>
</tr>
<tr>
<td>Flower position</td>
<td>Axial X Top</td>
<td>651</td>
<td>207</td>
</tr>
<tr>
<td>Plant height</td>
<td>Tall X Dwarf</td>
<td>767</td>
<td>277</td>
</tr>
</tbody>
</table>
1. Each trait determined by genes. These are transmitted from parent to offspring.
   - Individuals have 2 alleles for each gene
   - one on each homologous chromosome.
2. Each individual receives two genes that encode each trait.
   - Alleles (on homologs) segregate during meiosis
3. Not all alleles are the same. Which allele gets included in a gamete is determined by chance (homologues separate randomly)
4. Alleles do not influence each other. They remain discrete and do not blend.
5. The presence of an allele does not ensure it will be expressed. This is the Law of Dominance.
   - Dominant allele (purple) may mask recessive allele (white).
   - Dominant alleles = Upper case
   - Recessive Allele = Lower case
     - \( PP \rightarrow \) purple
     - \( pp \rightarrow \) white
     - all gametes of a \( PP \) parent carry the \( P \) allele
     - all gametes of a \( pp \) parent will carry the \( p \) allele
Examples of inherited traits in humans

**Recessive Traits**

1. Cystic fibrosis
   An autosomal recessive genetic condition, which causes the body to produce excessively thick, sticky mucus that clogs the lungs and pancreas, impairing breathing and digestion

2. Sickle cell anemia

**Dominant Traits**

1. Huntington Disease
   A genetic disorder of the nervous system, characterized by involuntary movements and progressive mental deterioration. It usually starts between ages 30 and 50 and slowly progresses to death.

2. Hypercholesterolemia

---

II. Gregor Mendel & Mendelian Genetics

**Mendel’s Laws**

**Mendel’s First Law of Heredity: Segregation**

1. The two alleles for a gene segregate during gamete formation and are rejoined at random during fertilization

   → disjunction of homologs in Anaphase I

**Mendel’s Second Law of Heredity: Independent Assortment**

1. Fate of one pair of alleles associated with one trait (gene) does not influence the fate of another pair of alleles associated with a different trait (another gene)

2. Independent alignment of different homologous pairs during metaphase I
III. How do genes relate to traits?

A. Terminology

Genotype = Combination of alleles: \( PP \) or \( Pp \) or \( pp \)

Phenotype = appearance, blood type, etc.

Genotype determines phenotype

B. How can you predict genotypes and phenotypes?

Punnett square method

Determines approximate proportion of genotypes and phenotypes for potential offspring

Based on laws of probability

Phenotypic Ratio = 3:1
Genotypic Ratio = 1:2:1
C. How can you determine an individual’s genotype?

Test Cross: Confirmation of Segregation

Alternative 1

All offspring purple; therefore unknown flower was homozygous

Homozygous recessive (white)

Dominant phenotype (allele distribution unknown)

if PP

if Pp

Alternative 2

Half of offspring white; therefore unknown flower was heterozygous

Homozygous recessive (white)

pairs of alleles on homologous chromosomes in diploid cells

chromosomes replicate

replicated homologues pair during metaphase of meiosis I,

orienting like this or like this

meiosis I

meiosis II

independent assortment produces four equally likely allele combinations during meiosis
D. What does it mean for a trait to be “dominant”?
ALL TRAITS HAVE A MOLECULAR BASIS! →
The molecular basis is coded in our genes (alleles are variants of one gene) →
Our genes code for proteins (perhaps enzymes) →
Proteins can be involved in complex metabolic pathways →

Ex: Round vs. Wrinkled Pea
RR or Rr → R gene codes for a starch-branching enzyme
rr → Peas that are rr have an inactive form of the enzyme due to a gene defect

If the enzyme is present, the starch grains in the pea are large and spherical.
RR and Rr starch grains allow the seeds to retain water and shrink uniformly
In rr seeds, the starch grains are irregular in shape, they lose water too rapidly and the seeds shrink unevenly, causing the wrinkled seed.

IV. How are multiple traits inherited?

A) Traits on separate chromosomes
Mendel:
Cross-bred homzygous plants that differed in two traits
e.g. seed color and seed shape…
SSYY x ssyy
What results should he get?
What did he find?

All F₁ offspring: smooth & yellow:

- **SsYy** (as we would expect)

Then he self-fertilized those: SsYy x SsYy

This is a **DIHYBRID CROSS**

Expecting:

- ¾ yellow & ¼ green
- ¾ smooth & ¼ wrinkled

Which is what he found…

For each trait

---

What did this mean?

- Genes for seed color and seed shape were inherited independently (each behaved as if by itself)
  - Had to be on separate chromosomes

**Mendel’s Second Law of Heredity: Independent Assortment**
Recombination can create new combinations of linked alleles

Duplicated homologues starting Meiosis I

Crossing over can create new allele combinations!

Recombination between linked alleles is less frequent the closer on a chromosome the loci.

Maize

<table>
<thead>
<tr>
<th>Genotype of all gametes formed by csh parent</th>
<th>Genotypes of gametes formed by heterozygous (C,c,Hh,sh) parent</th>
</tr>
</thead>
<tbody>
<tr>
<td>csh parent</td>
<td>csh, Csh, csh, Csh, csh, Csh, csh, Csh, Csh, csh, Csh</td>
</tr>
<tr>
<td>Appearance (phenotype)</td>
<td>Colored, smooth, Colorless, wrinkled, Colored, smooth</td>
</tr>
<tr>
<td>If independent assortment</td>
<td>25% 25% 25% 25%</td>
</tr>
<tr>
<td>Actual results</td>
<td>48.6% 48.6% 1.4% 1.4%</td>
</tr>
</tbody>
</table>
Recombination between linked alleles is less frequent the closer on a chromosome the loci.

Maize

<table>
<thead>
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<th>Genotype of all gametes formed by c,s,h,sh parent</th>
<th>Genotypes of gametes formed by heterozygous (c,c,s,h,sh) parent</th>
</tr>
</thead>
<tbody>
<tr>
<td>c,s,h,sh</td>
<td>Csh</td>
</tr>
<tr>
<td>c,s,h,sh</td>
<td>csh</td>
</tr>
<tr>
<td>Appearance (phenotype)</td>
<td>Colored, smooth</td>
</tr>
<tr>
<td>Genotypes</td>
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<td>48.6%</td>
</tr>
</tbody>
</table>

Chromosome 9

IV. How are multiple traits inherited?

A) Traits on separate chromosomes

B) Traits on the same chromosome

- Genetically linked: on same double helix
  - Do not assort independently
  - Get inherited together

Gametes will have either:

- Purple flower allele & long pollen allele
  - or

- Red flower allele & round pollen allele
VI. Variations on Mendelian genetics

1) Incomplete dominance

- Heterozygote phenotype is intermediate between phenotypes of homozygotes

In Japanese Four O’Clock

In Snapdragon

Incomplete dominance in Snapdragon's
Incomplete dominance in human hypercholesterolemia
Abnormal form of cholesterol cell surface receptor (they lack hydrophobic
tails); cholesterol remains in the blood stream; leads to heart disease

<table>
<thead>
<tr>
<th>Genotypes</th>
<th>Phenotypes</th>
</tr>
</thead>
<tbody>
<tr>
<td>hh</td>
<td>Homozygous for ability to make LDL receptors</td>
</tr>
<tr>
<td>Hh</td>
<td>Heterozygous</td>
</tr>
<tr>
<td>HH</td>
<td>Homozygous for inability to make LDL receptors</td>
</tr>
</tbody>
</table>

VI. Variations on Mendelian genetics

2) Multiple alleles for a single gene & Codominance
ex.: Blood types; Gene: I; Alleles: I^A, I^B, i; Phenotypes: A, B, AB, O

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Type A</th>
<th>Type B</th>
<th>Type AB</th>
<th>Type O</th>
</tr>
</thead>
<tbody>
<tr>
<td>I^A I^A or I^A i</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I^B I^B or I^B i</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I^A I^B</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ii</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Gene encodes an enzyme that adds sugar molecules to an existing glycolipid on the surface of red blood cells
Multiple Alleles

\(I^A = \) galactosamine antigen on RBC surface
\(I^B = \) galactose antigen on RBC surface
\(i = \) no antigens on RBC surface

<table>
<thead>
<tr>
<th>Phenotype (Blood Type)</th>
<th>Genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>(I^A)(I^A) (\text{or} \ I^A)(i)</td>
</tr>
<tr>
<td>B</td>
<td>(I^B)(I^B) (\text{or} \ I^B)(i)</td>
</tr>
<tr>
<td>AB</td>
<td>(I^A)(I^B)</td>
</tr>
<tr>
<td>O</td>
<td>(i)(i)</td>
</tr>
</tbody>
</table>

Possible alleles from male

Possible alleles from female

Blood types: A, AB, B, O
Multiple alleles for the ABO blood groups

<table>
<thead>
<tr>
<th>Blood Group (Phenotype)</th>
<th>Genotypes</th>
<th>Antibodies Present in Blood</th>
<th>Reaction When Blood type Below is Mixed with blood type on far left column</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>ii</td>
<td>Anti-A</td>
<td>Anti-B</td>
</tr>
<tr>
<td>A</td>
<td>$i^A i^A$ or $i^A i^J$</td>
<td>Anti-B</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>$i^B i^B$ or $i^B i^J$</td>
<td>Anti-A</td>
<td></td>
</tr>
<tr>
<td>AB</td>
<td>$i^A i^B$</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

= agglutination  = no agglutination

Blood Type Distribution, General Population

Percentage of B Blood Allele
- 5-10%
- 10-15%
- 15-20%
- 20-25%
- >25%

Page 18
VI. Variations on Mendelian genetics

3) Polygenic inheritance
• two or more genes contribute to single phenotype

Eye color:
• Determined by melanin pigment
• Four (+) genes for melanin synthesis
• All show incomplete dominance

3) Polygenic inheritance
Continuous Variation
Polygenic Inheritance
Height in People

(a)

(b)
3) Polygenic inheritance

A model for polygenic inheritance of skin color

P generation

\[ aabbcc \quad \text{(very light)} \] × \[ AABBCC \quad \text{(very dark)} \]

F\(_1\) generation

\[ AaBbCc \times AaBbCc \]

Sperm

F\(_2\) generation

Eggs

Continuous Variation
Skin Color & Polygenic Inheritance

VI. Variations on Mendelian genetics

4) Environmental influence
i.e. height can be reduced by poor nutrition

\[ P = G + E \]
5) Epistasis: one gene affects the expression of another gene

<table>
<thead>
<tr>
<th>ee</th>
<th>E_</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No dark pigment in fur</strong></td>
<td><strong>Dark pigment in fur</strong></td>
</tr>
<tr>
<td>Yellow Lab</td>
<td>Yellow Lab</td>
</tr>
<tr>
<td>eeBB</td>
<td>eeBB</td>
</tr>
<tr>
<td>Yellow fur</td>
<td>Yellow fur</td>
</tr>
<tr>
<td>eeBB</td>
<td>eeBB</td>
</tr>
<tr>
<td>Chocolate Lab</td>
<td>Chocolate Lab</td>
</tr>
<tr>
<td>E_bb</td>
<td>E_bb</td>
</tr>
<tr>
<td>Brown fur</td>
<td>Brown fur</td>
</tr>
<tr>
<td>E_B_B_</td>
<td>E_B_B_</td>
</tr>
<tr>
<td>Black fur</td>
<td>Black fur</td>
</tr>
</tbody>
</table>

E gene determines whether Melanin will be deposited; B gene determines how dark the pigment will be.

VII. Genetic Disorders

A. Most genetic disorders caused by recessive alleles

**Albinism** → defects in melanin production
  - homozygous recessive → **albinism**

**Sickle Cell Anemia**

defects in hemoglobin gene
  - “Sickle” cells
  - homozygous recessive (gene mutation)

B. Some genetic disorders caused by dominant alleles

- at least one parent must suffer the disease (& still reproduce)

**Huntington disease:** (onset at 40-50 yrs of age)
- destroys nerve tissue

VII. Genetic Disorders

Genetic Counseling

Counselors can look for three things in cell cultures in search of genetic disorders:
1. alterations in chromosome number/structure
2. proper enzyme functioning
3. association with known genetic markers
   (a method of screening for abnormalities)

When?
→ Before birth
→ After birth
→ Adult

VII. Genetic Disorders

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Symptom</th>
<th>Defect</th>
<th>Dominant/Recessive</th>
<th>Frequency Among Human Births</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystic fibrosis</td>
<td>Mucus dogs lungs, liver, and pancreas</td>
<td>Failure of chloride ion transport mechanism</td>
<td>Recessive</td>
<td>1/2500 (Caucasians)</td>
</tr>
<tr>
<td>Sickle cell anemia</td>
<td>Blood circulation is poor</td>
<td>Abnormal hemoglobin molecules</td>
<td>Recessive</td>
<td>1/625 (African Americans)</td>
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<td>Muscular dystrophy</td>
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<td>Sex-linked recessive</td>
<td>1/1700 (males)</td>
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<tr>
<td>(Duchenne)</td>
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Sickle-cell disease, multiple effects of a single human gene

Individual homozygous for sickle-cell allele
Sickle-cell (abnormal) hemoglobin
Red blood cells to become sickle-shaped

Genotypes:
- SS X SS
- Ss X SS
- Ss X Ss
- Ss X ss
- SS X ss
- ss X ss

Alleles: S = Normal, s = Sickle cell

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Frequency of Sickle Cell Allele  Distribution of Malaria

SS = wildtype and susceptible to malaria
Ss = modest anemia and resistant to malaria
Ss = severe, lethal anemia and resistant to malaria

VII. Genetic Disorders

Prenatal Diagnosis: Autosomal Nondisjunction or Aneuploidy
VII. Genetic Disorders

Adult Screening: Hexosaminidase and Tay-Sachs Disease; autosomal recessive

Phenotypes: Carrier X Carrier

Alleles: T = normal allele
t = Tay Sachs allele

Genotypes: 
\[
\begin{array}{ccc}
T & t & t \\
T & T & Tt \\
t & Tt & tt \\
\end{array}
\]

Infants with Tay-Sachs disease appear to develop normally for the first six months of life. Then, as nerve cells become distended with gangliosides, a relentless deterioration of mental and physical abilities occurs. The child becomes blind, deaf, and unable to swallow. Muscles begin to atrophy and paralysis sets in. Death usually occurs before the age of 4 or 5 (Juvenile T-S).

V. How is sex determined?

By sex chromosomes:

Female = XX & Male = XY
(all other body chromosomes are autosomes)

Sex chromosome carried by sperm determines sex of offspring

Sex linked genes: on sex chromosomes
Y: ~75 genes
X: >1400 genes
(In males): MOST genes on X have no counterpart on Y

→ expressed regardless of dominance
(i.e. color blindness- more common in males)
VII. Genetic Disorders

Some Important Genetic Disorders

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VII. Genetic Disorders: Can be traced with Pedigrees

- Mutant alleles often recessive
- Heterozygotes (carriers) basically normal
- Diseases → homozygous recessive individuals

Most people carry 5-15 defective recessive alleles

low odds for double recessive genotype (in offspring)

UNLESS PARTNERS ARE RELATED....
Testing a fetus for genetic disorders

Amniocentesis

- Needle inserted through abdomen to extract amniotic fluid
- Ultrasound monitor
- Fetus
- Placenta
- Uterus
- Cervix

Chorionic villus sampling

- Extract tissue from chorionic villi
- Ultrasound monitor
- Fetus
- Placenta
- Chorionic villi
- Uterus
- Cervix

Fetal cells

Centrifugation

Several weeks

Tests

Several hours

Karyotyping

Barr body, X-inactivation

- Allele for black fur is inactivated
- Allele for orange fur is inactivated
- X chromosome allele for orange fur
- Inactivated X chromosome becomes Barr body
- X chromosome allele for black fur
- Inactivated X chromosome becomes Barr body
- Nucleus

Second gene causes patchy distribution of pigment:
white fur = no pigment, orange or black fur = pigment

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