

Heredity

AP Biology • Unit 5

AP Biology

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Heredity

What we will cover

- 1 Meiosis
- 2 Meiosis and genetic diversity
- 3 Mendelian genetics
- 4 Non-Mendelian patterns
- 5 Environment and phenotype

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1 Meiosis

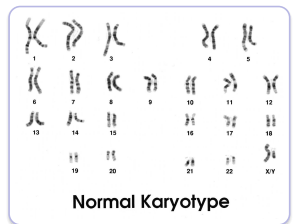
Heredity

Meiosis

Meiosis 减数分裂 turns one **diploid** 二倍体 cell into four **haploid** 单倍体 **gametes** 配子 – halving the chromosome number.

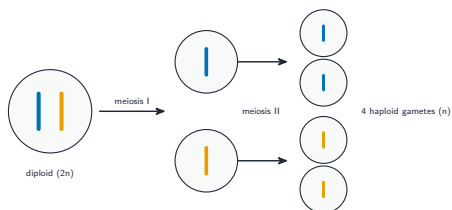
- **meiosis I** separates **homologous** 同源染色体 pairs (halving)
- **meiosis II** separates the copies

Two haploid gametes join at fertilization to restore the diploid number.



Meiosis halves chromosome sets for gametes

Meiosis halves the chromosome number in two



Meiosis halves the chromosome number in two divisions

2 Diversity

Meiosis and genetic diversity

Three shuffles make every gamete unique:

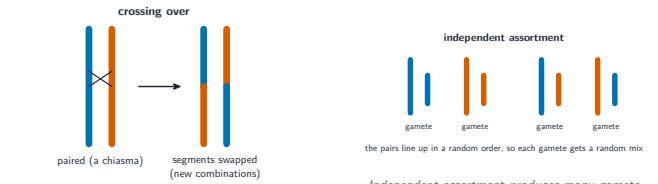
- **Crossing over** 交叉互换 – homologs swap pieces
- **Independent assortment** 自由组合 – pairs line up at random
- **Random fertilization** 随机受精 – any sperm, any egg

Independent assortment alone gives 2^{23} human gamete types – **genetic diversity** 遗传多样性 is insurance.



Variation among offspring is not noise – it is insurance

Crossing over swaps segments between homologous



Crossing over swaps segments between homologous chromosomes

Independent assortment produces many gamete combinations

Crossing over swaps segments between homologous chromosomes

Where meiosis gets its variation

crossing over 交叉互换	homologous chromosomes swap segments in prophase I – new combinations <i>within</i> a chromosome
independent assortment 自由组合	each homologous pair lines up independently at metaphase I – 2^{23} combinations in humans
random fertilisation	any sperm with any egg, multiplying the possibilities again
together	these create enormous variation – the raw material evolution acts on
homozygous 纯合 / heterozygous 杂合	two identical alleles, or two different ones, at a locus

Mutation *creates* new alleles; meiosis *recombines* the existing ones. Both are needed, and they are not the same source.

3 Mendelian

Mendelian genetics

The **law of segregation** 分离: alleles separate, so each gamete carries one. A **Punnett square** 庞纳特方格 predicts the cross.

- **genotype** 基因型 (Bb) vs **phenotype** 表型 (trait)
- **dominant** 显性 (capital) hides **recessive** 隐性

$Bb \times Bb \Rightarrow$ phenotype ratio 3:1. Test the fit with $\chi^2 = \sum \frac{(O - E)^2}{E}$.

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4 Non-Mendelian

Heredity

└ Non-Mendelian

Non-Mendelian patterns

Many traits break Mendel's simple dominant/recessive rule:

Incomplete dominance
不完全显性: red+white → pink

Codominance 共显性: AB blood (both show)

Multiple alleles 复等位基因:
 I^A, I^B, i

Sex-linked 伴性遗传: colour blindness

Polygenic 多基因遗传 & epistasis 上位效应: a smooth range

Heredity

└ Non-Mendelian

Non-Mendelian patterns, continued

father:

X^B

Y

mother

X^B

X^b

$X^B X^B$

$X^B X^b$

$X^B Y$

$X^b Y$

carrier daughter

colour-blind son

- Incomplete dominance** 不完全显性: heterozygotes are a blend (red × white → pink).
- Codominance** 共显性: both alleles show fully (AB blood type).
- Multiple alleles, polygenic** 多基因 traits (many genes, like height), **pleiotropy** (one gene, many effects), and **sex-linked** 伴性 genes (on the X chromosome) all give more complex ratios.

Heredity

└ Non-Mendelian

Worked example – the chi-square test

A monohybrid cross predicts 3 : 1. Of 80 offspring, 55 show the dominant trait and 25 the recessive. Do the data fit?

- expected: 60 and 20
- $\chi^2 = \sum \frac{(o - e)^2}{e} = \frac{25}{60} + \frac{25}{20} = 0.42 + 1.25 = 1.67$
- $df = 2 - 1 = 1$; the critical value at $p = 0.05$ is 3.84
- $1.67 < 3.84$, so **do not reject** the null hypothesis – the data fit

The null hypothesis is that any difference is chance alone. Failing to reject it is not proof the ratio is right, only that the deviation is unremarkable.

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Environment

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└ Environment

Environment and phenotype

Genes are not the whole story – the **environment** 环境 shapes the phenotype too.

- temperature (dark fur on cool paws)
- nutrition, light

Phenotypic plasticity 表型可塑性: one genotype, a **range** of phenotypes – nature sets the options, the environment chooses.

same genotype

blue (acid soil)

pink (basic soil)

Hydrangea: the soil decides the colour.

Heredity

└ Environment

Phenotype is not set by genes alone

the claim

the **environment** also shapes how a genotype is expressed

temperature

Siamese cat coat colour, and temperature-dependent sex determination in some reptiles

nutrition

height and mass in almost every organism

density and crowding

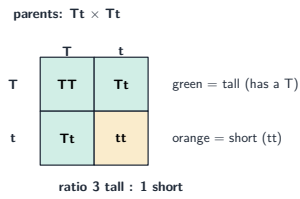
can change growth form in plants and behaviour in animals

the general point

a genotype specifies a **range** of possible phenotypes, not one outcome

Identical twins are the standard evidence: the same genome, and measurably different phenotypes. That is what the term *norm of reaction* names.

Mendelian Genetics, continued



A monohybrid Punnett square giving a 3:1 ratio

Exam tips

- Contrast mitosis (2 identical, full chromosome number) with **meiosis** (4 non-identical gametes, half the number).
- Explain variation from **crossing over**, **independent assortment**, and **random fertilisation**.
- Use the multiplication rule for dihybrid crosses (each gene's 3:1 multiplied), and a **chi-square** test ($\chi^2 = \sum \frac{(o-e)^2}{e}$) to judge observed vs expected ratios.
- Keep **genotype** (the alleles) separate from **phenotype** (what you see) – AA and Aa can look the same.
- Recognise non-Mendelian patterns: incomplete dominance, codominance, and sex linkage.

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Recap

Unit 5 – key takeaways

1 Meiosis

diploid → four haploid gametes;
halves chromosomes

2 Diversity

crossing over, independent assortment,
random fertilization

3 Mendel

$Bb \times Bb \Rightarrow 3:1$; test fit with χ^2

4 Beyond Mendel

incomplete dominance, codominance,
plasticity

Check yourself

- 1 How many cells, and what ploidy, does meiosis produce?
- 2 What phenotype ratio comes from $Bb \times Bb$?
- 3 Red + white flowers give pink – which inheritance pattern?
- 4 Two identical clones differ in colour – what is this called?

BB	Bb
Bb	bb

Answers 1. four haploid cells 2. 3:1 3. incomplete dominance 4. phenotypic plasticity