The Ultimate AP® Biology Unit 5 Cheat Sheet

5.1 Meiosis

Key Terms

Diploid (2n): 2 sets of chromosomes (human 2n = 46).
Haploid (n): 1 set (human gametes, n = 23).
Homologous chromosomes: same genes, same loci, same order; 1 from mom, 1 from dad; may carry diff. **alleles**.
Sister chromatids: identical DNA copies made in S phase, joined at the centromere; separate in meiosis II.

Meiosis I: Homologs Separate

Prophase I: homologs pair up (**synapsis**) → **tetrad**; **crossing over** at chiasmata swaps DNA segments.
Metaphase I: homologous PAIRS line up at the plate; each pair's orientation is random (indep. assortment).
Anaphase I: homologs pulled to opposite poles; sister chromatids stay joined at the centromere for now.
Telophase I + cytokinesis → 2 haploid cells (each chrom. still 2 chromatids). No DNA replication follows.

Meiosis II: Like Mitosis

Meiosis II: no S phase before it; sister chromatids separate → 4 haploid cells, each one genetically unique.

Stage	Chrom. #	DNA
G_1 (start)	2n = 46	2c
After S	2n = 46	4c
After meiosis I	n = 23	2c
After meiosis II	n = 23	1c

Gametes: sperm (4 per meiosis) & egg (1 egg + 2–3 polar bodies). Fertilization → zygote, restores 2n.
Life cycles: animals → meiosis makes gametes; plants & fungi → meiosis makes spores (plants alternate generations).

⚠ Chromosome # halves in MEIOSIS I, not meiosis II. Meiosis II only splits sister chromatids (like mitosis).

✓ Diagram Q: pairs lined up = metaphase I; chromosomes in single file = mitosis or metaphase II of meiosis.

5.2 Meiosis & Genetic Diversity

Three Sources of Variation

1) Crossing over (prophase I): homologs swap segments → **recombinant** chromatids w/ new allele combos.
2) Independent assortment (metaphase I): each pair orients randomly → 2^n combos. Human: $2^{23} \approx 8.4$ million.
3) Random fertilization: any sperm + any egg → $2^{23} \times 2^{23} \approx$ 70 trillion zygotes (before crossing over).
Why it matters: variation → raw material for natural selection → populations can adapt to a changing environment.

Mitosis vs Meiosis

Feature	Mitosis	Meiosis
Divisions	1	2
Daughters	2, diploid	4, haploid
Genetics	Identical	Unique
Synapsis	No	Yes (prophase I)
Crossing over	No	Yes
Purpose	Growth, repair	Gametes

Errors & Evidence

Nondisjunction: homologs or chromatids fail to separate → gametes n+1 or n−1 → **aneuploidy** (see 5.6).
Meiosis I error → all 4 gametes abnormal (2 are n+1, 2 are n−1). **Meiosis II error** → only 2 of 4 abnormal.
Common ancestry: meiosis & sexual repro. shared across eukaryotes → evidence of shared origin.
Asexual repro: offspring are clones; no new allele combos → fast, but risky when environment changes.

⚠ Crossing over is between HOMOLOGS (non-sister chromatids), not between sister chromatids.

✓ FRQ: name the event (crossing over, independent assortment) + the phase + how it adds variation.

5.3 Mendelian Genetics

Vocabulary

Genotype = alleles (AA, Aa, aa). **Phenotype** = trait shown.
Homozygous = same 2 alleles; **heterozygous** = 2 diff.
Dominant allele masks **recessive** in a heterozygote. Dominant ≠ more common in a population, and ≠ "better."
P = parents; $\mathbf{F_1}$ = their offspring; $\mathbf{F_2}$ = offspring of $F_1 \times F_1$.

Mendel's Laws

Law of segregation: the 2 alleles of a gene separate in meiosis (anaphase I) → each gamete gets just 1.
Law of independent assortment: genes on diff. chromosomes sort independently (metaphase I).

Ratios to Know

Cross	Genotype	Phenotype
Aa × Aa	1:2:1	3:1
Aa × aa	1:1	1:1
AA × aa	All Aa	All dom.
AaBb × AaBb	—	9:3:3:1
AaBb × aabb	—	1:1:1:1

Testcross: unknown dominant × homozygous recessive. Any recessive offspring → unknown was heterozygous.
Monohybrid cross = 1 gene; **dihybrid** cross = 2 genes. **True-breeding** = homozygous for the trait in question.
Mendel's success: peas (many offspring, controlled crosses, clear either/or traits); counted large samples.
Punnett square: gametes of one parent on top, other on side; each box = ¼ (mono) or 1/16 (dihybrid).
Gametes from AaBb: AB, Ab, aB, ab (¼ each) b/c of independent assortment of the 2 pairs in meiosis I.

⚠ Ratios are probabilities. Small samples (e.g., a family with 4 kids) often don't match 3:1 exactly; big ones do.

✓ Unknown dominant genotype? Do a testcross with the homozygous recessive; any recessive offspring → Aa.

5.3 Probability & Pedigrees

Probability Rules

Product rule (AND, independent events): P(A and B) = P(A) × P(B). e.g., P(aa) from Aa × Aa = ½ × ½ = ¼.
Sum rule (OR): P(A or B) = P(A) + P(B) if exclusive. e.g., P(heteroz.) = P(Aa) + P(aA) = ¼ + ¼ = ½.
Split genes: AaBbCc × AaBbCc → P(aabbcc) = ¼ × ¼ × ¼ = 1/64. Faster than a big Punnett square.
Already dominant: P(carrier | unaffected child of Aa × Aa) = 2/3, not ½, b/c the aa outcome is already ruled out.
Dihybrid shortcut: P(dominant A, bb) from AaBb × AaBb = ¾ × ¼ = 3/16. Split by gene, then multiply.

Pedigrees

Symbols: □ male, ○ female, shaded = affected; half-shaded = carrier (sometimes); horizontal line = mating.

Pattern	Clue
Auto. dominant	Every gen.; affected parent
Auto. recessive	Skips gen.; unaffected parents
X-linked rec.	Mostly males; via mothers
X-linked dom.	Affected dad → all daughters
Mitochondrial	Mom → all kids

Auto. recessive examples: cystic fibrosis, sickle cell, Tay-Sachs. **Auto. dominant**: Huntington's, achondroplasia.
Rule out: 2 unaffected parents w/ affected kid → recessive. Affected son of unaffected dad → not Y-linked.
Carriers: unaffected heterozygotes in recessive pedigrees; label a parent Aa once any of its kids is aa.
Generations = Roman numerals (I, II, III); individuals numbered left → right (e.g., II-3 = gen. II, #3).

⚠ Add probabilities for OR; multiply for AND. Mixing the two up is the #1 math error on genetics FRQs.

✓ Pedigree Q: test each pattern in turn; eliminate any pattern that a single family in the chart contradicts.

5.3 Chi-Square Analysis

When & Why

Chi-square (χ^2): tests if observed counts differ from expected more than chance would explain.
Null hypothesis (H_o): no real difference; data fit the expected ratio (e.g., 9:3:3:1); any gap = random chance.
Alternative hypothesis: the difference is real; some factor other than chance (e.g., gene linkage) is acting.

Steps (Formula Sheet)

1) Expected = total × ratio fraction. e.g., 160 offspring, 3:1 ratio → expect 120 dominant : 40 recessive offspring.
2) $\chi^2 = \Sigma\ (o - e)^2 / e$ for each category, then add.
3) Degrees of freedom = # categories − 1.
4) Compare your χ^2 to the critical value at p = 0.05 (table on the formula sheet), in the row for your df.

df	1	2	3
p = 0.05	3.84	5.99	7.81

Reading the Result

χ^2 < critical → fail to reject H_o → the difference is likely due to chance alone; the data fit the model.
χ^2 > critical → reject H_o → something besides chance is going on (e.g., linked genes, lethal allele, selection).
Worked: obs. 130 tall : 30 short; exp. 120 : 40 → $(10^2/120) + (10^2/40) = 0.83 + 2.5 =$ **3.33**. df = 1; 3.33 < 3.84 → fits 3:1.
Wording: never "prove" or "accept" H_o; say "fail to reject".
Dihybrid example: 4 phenotype classes (9:3:3:1) → df = 4 − 1 = 3 → critical value 7.81 at p = 0.05 in the table.
Large χ^2 = big gap between observed & expected counts → likely real. Small χ^2 = close fit to the model.
Also used for behavior choice chambers, Hardy-Weinberg tests (Unit 7), any count vs a prediction.

⚠ Use raw COUNTS, never percentages or ratios, in χ^2. And df = categories − 1, not sample size − 1.

✓ Show work: expected values, each $(o - e)^2/e$ term, the sum, df, critical value, and a conclusion.

5.4 Non-Mendelian Genetics

Beyond Simple Dominance

Pattern	Example	F_2 ratio
Incomplete dom.	Red × white → pink	1:2:1
Codominance	AB blood, roan	1:2:1
Multiple alleles	I^A, I^B, i	Varies
Polygenic	Height, skin	Bell curve
Pleiotropy	Sickle cell	1 gene → many

Incomplete dominance: heterozygote = blend.
Codominance: both alleles fully shown (AB blood type).
Blood type (1 gene, 3 alleles): I^A, I^B codominant; both dominant to i. Type O = ii. $I^Ai \times I^Bi$ → A, B, AB, O each ¼.

Sex-Linked Traits

X-linked recessive: males (XY) have 1 X → 1 allele shows. Colorblindness, hemophilia, Duchenne MD.
Carrier cross: $X^AX^a \times X^AY$ → ½ of sons affected; no daughters affected, but ½ of daughters are carriers.
Father → son: a dad passes his Y, never his X, to his sons → X-linked traits are never passed dad → son.

Linked Genes

Linked genes: close on same chromosome → inherited together → don't assort independently.
Recombination frequency = recombinant offspring ÷ total × 100. 1% = 1 **map unit** (cM). Max ~50%.
Map: A–B 8%, B–C 3%, A–C 11% → gene order A–B–C (the 2 small distances add up to the big one).
Mito/chloroplast genes: usually inherited from mother (via egg cytoplasm) → no Mendelian ratios.

⚠ Linked genes give MOSTLY parental phenotypes + few recombinants, not the 9:3:3:1 or 1:1:1:1 ratio.

✓ Recombinants = the 2 smallest classes in a testcross. Add them, divide by total, × 100 = map units.

5.5 Environmental Effects on Phenotype

Genotype + Environment

Phenotypic plasticity: one genotype → diff. phenotypes in diff. environments (e.g., hydrangea color).
Norm of reaction: the full range of phenotypes one genotype can produce across different environments.
Epigenetics: environmental factors can alter gene expression w/o changing the DNA base sequence.

Example	Environment → effect
Hydrangea	Soil pH → blue or pink
Himalayan rabbit	Cold → dark fur tips
Arctic fox	Season → white/brown
Sea turtles	Nest temp → sex
Daphnia	Predators → helmet
PKU	Diet low Phe → normal

Temp-sensitive enzymes: Himalayan rabbits & Siamese cats make pigment only in cooler body parts (ears, paws).
PKU: same genotype, but a low-phenylalanine diet prevents damage → environment changes outcome.
Height, skin color: polygenic + nutrition, sun exposure → continuous variation (a bell-shaped curve).

Why It Matters

Identical twins can differ in traits → environmental & epigenetic effects (e.g., DNA methylation; see Unit 6).
Plasticity lets organisms survive changing conditions w/o waiting for new mutations or selection.
Experiment design: same genotype (clones, twins), vary environment → isolates environment's effect.
Plants: same seed genotype grown in sun vs shade → diff. leaf size & thickness, stem height, branching.

⚠ Environment changes PHENOTYPE, not genotype. The alleles stay the same; only expression changes.

✓ Data Q: same genotype + diff. phenotype → environment; same environment + diff. phenotype → genes.

5.6 Chromosomal Inheritance & Strategy

Chromosome Theory

Genes sit on chromosomes; their behavior in meiosis explains Mendel's laws (Morgan: fruit fly white eyes = X-linked, the first gene placed on a specific chromosome).
Karyotype: picture of chromosomes sorted by size & banding → spots extra or missing chromosomes.
Sex chromosomes: XX female, XY male (humans). The SRY gene on the Y triggers male development.

Chromosome Disorders

Condition	Karyotype	Cause
Down	Trisomy 21	Nondisjunction
Turner	XO (45)	Missing X
Klinefelter	XXY (47)	Extra X
Cri du chat	Del. chrom. 5	Deletion

Structural changes: deletion, duplication, inversion, translocation (e.g., chronic myeloid leukemia).
Risk ↑ w/ maternal age for nondisjunction (Down syndrome).
Single-gene disorders: sickle cell (1 base change → Hb shape), cystic fibrosis, Huntington's disease.

Exam Moves

Genetics math: write parent genotypes → list gametes → product/sum rule → reduce the fraction.
Links: Unit 4 mitosis → meiosis; Unit 6 DNA, mutation & expression; Unit 7 Hardy-Weinberg & selection.
Pedigree + Punnett: get the parents' genotypes from the pedigree first, then do the cross from them.
χ^2 check: use it whenever data "don't quite" match a predicted ratio; decide chance vs a real effect.
X inactivation: one X in each female cell condenses (Barr body) → mosaic traits like orange/black calico cats.

⚠ Nondisjunction in meiosis I affects all 4 gametes; in meiosis II, only 2 of the 4 are abnormal.

✓ Every genetics FRQ: state genotypes, show the cross, give the ratio or probability, then explain why.