GENERAL BIOLOGY · STUDY GUIDE

Exam 3 Study Guide

This guide covers chapters 5 and 6. First you learn the rules of inheritance and how to turn any cross into a probability. Then you go inside the gene: how DNA is built and copied, how it is read into RNA and protein, and what a mutation does to that message. This exam has more math than the first two, so work every worked case with a pencil. Read the "what to know" paragraphs, copy the tables by hand, then try each case with the steps covered. Finish each chapter with its ten self-test items. The cram sheet puts every ratio and rule in one place.

WHAT THIS GUIDE COVERS

- Chapter 5 · segregation and independent assortment, Punnett squares, the multiplication and addition rules, the binomial expression, the two-thirds rule, pedigrees, chi square, incomplete dominance, codominance, ABO, epistasis, polygenic traits, X linkage, recombination maps, aneuploidy, imprinting and mitochondrial inheritance.
- Chapter 6 · the experiments that proved DNA is the gene, the double helix, chromatin, semiconservative replication, the replication fork, telomeres, DNA repair, transcription, RNA processing, the genetic code, translation and mutation types.

CHAPTER 5

Mendelian Genetics and the Chromosomal Basis of Inheritance

Read it in full: **Chapter 5 in the textbook**. See it in motion: **Mendelian Genetics Lab** and **Chromosomes and Inheritance Lab**.

What to know

Before Mendel, people thought traits blended like paint. Mendel showed inheritance is **particulate**: discrete factors, now called genes, pass on intact even when hidden. He used true breeding pea lines, characters with two clear forms, P, F_1 and F_2 generations and thousands of counts.

A gene comes in versions called **alleles** at a **locus**. Each organism carries two, one from each parent. Two identical alleles make a homozygote; two different alleles make a heterozygote. The **genotype** is the allele makeup; the **phenotype** is what shows. A dominant allele decides the phenotype in a heterozygote. Dominance has nothing to do with how common an allele is: polydactyly is dominant but rare.

The **law of segregation** says the two alleles separate into different gametes. A monohybrid F_2 gives 3:1 phenotypes and 1:2:1 genotypes. A **testcross** to a homozygous recessive reveals an unknown genotype: all dominant offspring suggest homozygous, and any recessive offspring prove heterozygous. The **law of independent assortment** says alleles of different genes sort independently, so a dihybrid F_2 gives 9:3:3:1, which is (3:1) × (3:1). Segregation comes from homologs separating in anaphase I; independent assortment from each pair lining up on its own in metaphase I.

Genetics is probability. Use the **multiplication rule** for "and" (independent events all happening) and the **addition rule** for "or" (mutually exclusive ways to the same result). For several genes, solve one gene at a time and multiply. For "exactly k of n," use the binomial: (number of orders) × $p^k \times q^{n-k}$. For "at least one," subtract "none" from 1. An unaffected sibling of a person with a recessive condition, whose parents are both unaffected, is a carrier with probability **2/3**.

In a **pedigree**, squares are males and circles females; filled means affected. Autosomal recessive traits skip generations and appear in children of unaffected parents. Autosomal dominant traits appear in every generation, and each affected person has an affected parent. X linked recessive traits mostly affect males and never pass father to son. Mitochondrial traits pass from a mother to all her children. The **chi square** test, $\chi^2 = \Sigma(O - E)^2/E$ with classes minus 1 degrees of freedom, asks whether a deviation could be chance. At $p = 0.05$ the critical values are 3.84 (1 df), 5.99 (2 df) and 7.81 (3 df).

Mendel's laws still hold when the genotype to phenotype link is richer. **Incomplete dominance** gives an intermediate heterozygote, so the F_2 is 1:2:1 in phenotype too (pink snapdragons). **Codominance** shows both alleles fully (MN and AB blood types). ABO has three alleles: I^A and

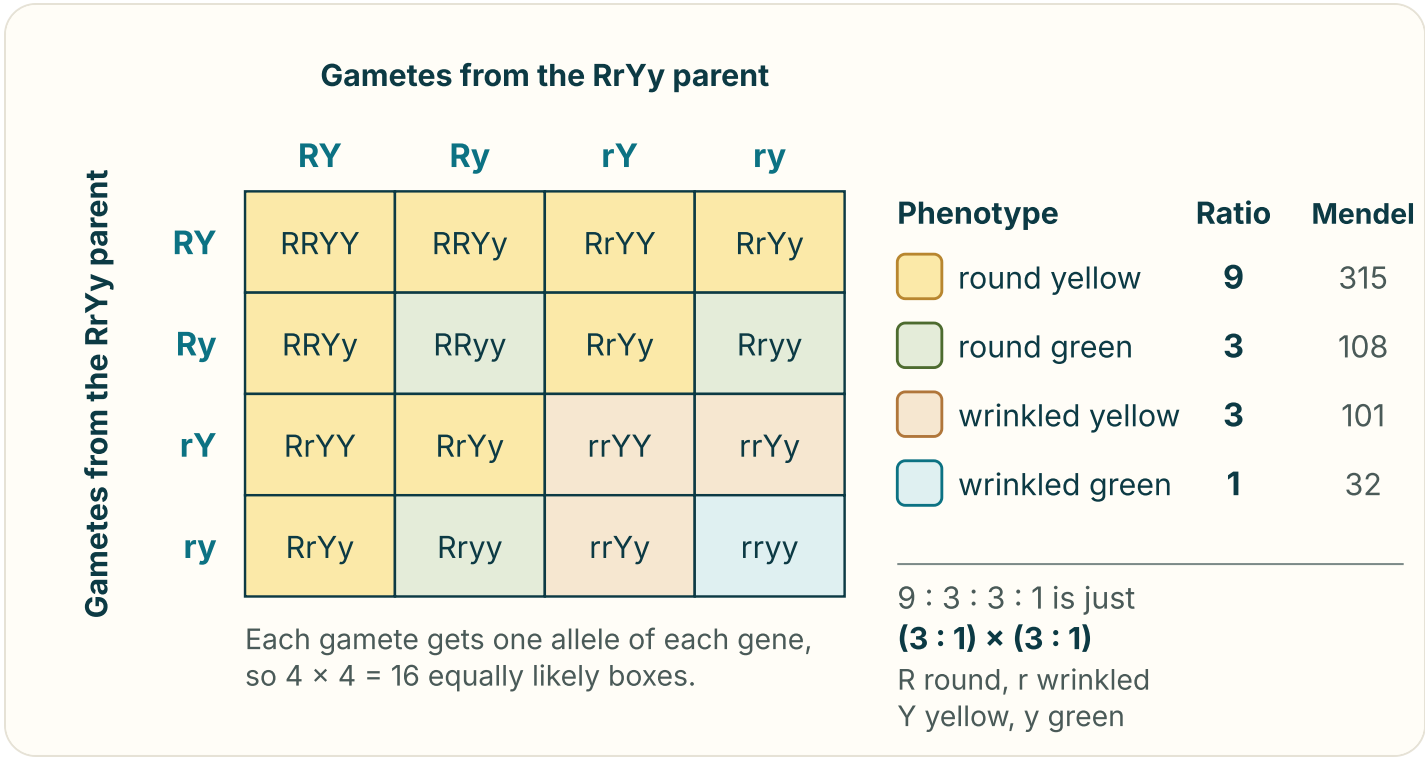
I^B are codominant and both dominant over i. **Pleiotropy** is one gene with many effects (sickle cell, Marfan syndrome). A lethal allele can turn 3:1 into 2:1, as with the yellow mouse allele.

Epistasis means one gene masks another. In Labradors, ee dogs are yellow whatever their B genotype, giving 9 black : 3 chocolate : 4 yellow. Other modified dihybrid ratios: 12:3:1 (dominant epistasis), 9:7 (complementary genes in one pathway) and 15:1 (duplicate genes). Any ratio over 16 that is not 9:3:3:1 points to two interacting genes. **Polygenic** traits such as height add small effects from many genes and form a bell curve. The environment shapes phenotype too (the norm of reaction). Penetrance is whether a genotype shows at all; expressivity is how strongly.

Morgan's white-eyed flies tied a gene to the X chromosome. In mammals, SRY on the Y starts male development; birds use Z-W, grasshoppers X-0 and bees haplo-diploidy. Males are **hemizygous** for X linked genes, so one recessive allele shows. A carrier mother passes the allele to half her sons (affected) and half her daughters (carriers). An affected father passes his X to every daughter and never to a son. Females inactivate one X at random in each cell, forming a **Barr body**, so they are mosaics, as calico cats show.

Genes on the same chromosome are **linked**. Crossing over in prophase I makes recombinants. **Recombination frequency** = recombinants ÷ total offspring. One percent equals one map unit (centimorgan). Farther apart means more recombination, but never more than 50 percent. Map distances add up to show gene order; double crossovers make long distances slightly short.

Nondisjunction gives aneuploidy: trisomy (2n + 1) or monosomy (2n − 1). Trisomy 21 (Down syndrome, about 1 in 700 births, risk rises with maternal age), trisomy 18, trisomy 13, 47,XXY, 45,X (the only viable human monosomy), 47,XXX and 47,XYY. Polyploidy is common in plants (bread wheat is 6n; triploid bananas are seedless). Structural changes are deletions (cri du chat), duplications, inversions and translocations (the Philadelphia chromosome). In **genomic imprinting**, the same 15q deletion causes Prader-Willi syndrome from the father and Angelman syndrome from the mother.



The dihybrid cross, box by box. Two genes on different chromosomes sort independently, so the 16 boxes give 9 : 3 : 3 : 1.

LOCK THESE IN

- Monohybrid F_2: 3:1 phenotypes, 1:2:1 genotypes. Dihybrid F_2: 9:3:3:1.
- "And" means multiply. "Or" means add. Solve one gene at a time.
- Unaffected sibling of an affected child of two unaffected parents: carrier with probability 2/3.
- Chi square critical values at p = 0.05: 3.84, 5.99, 7.81 for 1, 2, 3 df.
- Modified ratios: 9:3:4, 12:3:1, 9:7, 15:1 all mean two interacting genes.
- X linked recessive: more males, never father to son, carrier mothers.
- 1% recombination = 1 map unit; maximum 50%.
- Nondisjunction in meiosis I: all four gametes abnormal. In meiosis II: two of four.

Ratios and formulas

Situation	Rule or ratio	Example
Monohybrid F_2	3:1 (1:2:1 genotypes)	Pp × Pp
Testcross of a heterozygote	1:1	Pp × pp
Dihybrid F_2	9:3:3:1	RrYy × RrYy
Gamete types, n heterozygous genes	2^n	AaBbCc: 8
Offspring genotypes	3^n	AaBbCc × AaBbCc: 27
All recessive from two n-gene heterozygotes	$(1/4)^n$	aabbcc: 1/64
Exactly k of n	$[n! \div (k!(n - k)!)]\ p^k\ q^{n-k}$	2 of 3 affected: 9/64
Chi square	$\Sigma(O - E)^2/E$; df = classes − 1	Mendel 9:3:3:1 data: 0.47
Recombination frequency	Recombinants ÷ total × 100	(206 + 185) ÷ 2,300 = 17%
Incomplete dominance F_2	1:2:1 phenotypes	Red, pink, white
Recessive lethal in heterozygote cross	2:1	Yellow mice
Three additive genes, AaBbCc × AaBbCc	1:6:15:20:15:6:1 out of 64	Skin color model

Key terms and comparisons: inheritance patterns

Pattern	Clues	Examples
Autosomal recessive	Skips generations; affected children of unaffected parents	Cystic fibrosis, sickle cell disease, PKU, Tay-Sachs
Autosomal dominant	Every generation; affected person has an affected parent	Huntington disease, achondroplasia, Marfan syndrome
X linked recessive	Mostly males; never father to son	Hemophilia A, Duchenne muscular dystrophy, color blindness
X linked dominant	Affected father to all daughters, no sons	Hypophosphatemic rickets
Mitochondrial	Mother to all children	Leber hereditary optic neuropathy
Imprinting	Parent of origin decides	Prader-Willi and Angelman syndromes

Worked cases

WORKED CASE 1 · A MULTIGENE PROBABILITY

In the cross AaBbCc × aaBbCC, what fraction of offspring are aaBbCc? What fraction show all three dominant phenotypes?

STEP 1 · GENE A

Aa × aa gives aa with probability 1/2.

STEP 2 · GENE B

Bb × Bb gives Bb with probability 1/2.

STEP 3 · GENE C

Cc × CC gives Cc with probability 1/2.

STEP 4 · MULTIPLY

1/2 × 1/2 × 1/2 = **1/8**.

STEP 5 · ALL DOMINANT

A_ is 1/2, B_ is 3/4, C_ is 1 (every child gets a C). 1/2 × 3/4 × 1 = **3/8**.

WORKED CASE 2 · A COUNSELING PROBLEM

Tomas is unaffected, but his sister has cystic fibrosis and his parents are unaffected. His partner is a known carrier. What is the chance their first child is affected? If they have three children, what is the chance that exactly one is affected, assuming Tomas is a carrier?

STEP 1 · TOMAS

His parents must both be carriers. He is not aa, so he is a carrier with probability **2/3**.

STEP 2 · PARTNER

Known carrier: probability 1.

STEP 3 · CHILD

Two carriers have an aa child with probability 1/4. Multiply: 2/3 × 1 × 1/4 = **1/6**.

STEP 4 · EXACTLY ONE OF THREE

Three orders, each (1/4)(3/4)(3/4) = 9/64. So 3 × 9/64 = **27/64**.

WATCH THE UNCERTAIN STEP

Forgetting the 2/3 is the most common error. Whether to test, and what to do with the result, is the family's choice.

WORKED CASE 3 · CHI SQUARE ON A COAT COLOR CROSS

Two BbEe Labradors produce 160 puppies: 85 black, 35 chocolate and 40 yellow. Do the counts fit 9:3:4?

STEP 1 · EXPECTED COUNTS

160 × 9/16 = 90; 160 × 3/16 = 30; 160 × 4/16 = 40.

STEP 2 · EACH TERM

$(85 - 90)^2/90 = 0.28$; $(35 - 30)^2/30 = 0.83$; $(40 - 40)^2/40 = 0$.

STEP 3 · ADD

χ^2 = **1.11**.

STEP 4 · COMPARE

Three classes give 2 df. 1.11 is below 5.99, so we **fail to reject** the 9:3:4 model.

WORKED CASE 4 · MAPPING THREE GENES

A testcross of a dihybrid gives 412 AB, 398 ab, 96 Ab and 94 aB. Separate crosses give A to C = 12 map units and C to B = 7. What is the A to B distance and the gene order?

STEP 1 · FIND RECOMBINANTS

The rare classes Ab and aB are recombinant: 96 + 94 = 190.

STEP 2 · DIVIDE

190 ÷ 1,000 = 0.19, or **19 map units**.

STEP 3 · ORDER

12 + 7 = 19, so C sits between: **A, C, B**.

CHECK

19% is below 50%, so the genes are linked, and the parental classes (412 and 398) far outnumber recombinants.

TRAPS PEOPLE FALL INTO

- **Equating dominant with common.** Dominance describes one heterozygote; frequency describes a population.
- **Dropping the 2/3 in sibling problems.** An unaffected sibling cannot be aa, so only AA and Aa remain, 1:2.
- **Adding when you should multiply.** "And" for independent events multiplies; "or" for exclusive outcomes adds.
- **Reading pink snapdragons as blending.** The F_2 brings back red and white, so alleles stayed intact.
- **Mixing linked and sex linked.** Linked genes share any chromosome; sex linked genes sit on a sex chromosome.
- **Using the wrong denominator in X linked questions.** "Fraction of sons" counts only sons.
- **Reading a big χ^2 as proof.** It signals something besides chance, such as linkage; it does not prove a model wrong.
- **Expecting recombination above 50%.** Far-apart genes act unlinked at 50%; it never goes higher.

Chapter 5 self-test · ten items

1. A tall pea plant crossed with a dwarf plant gives 51 tall and 49 dwarf offspring. What is the tall parent?

A. Homozygous recessive (tt)

B. A haploid (T only) plant

C. Heterozygous (Tt) plant

D. Homozygous dominant (TT)

C. Heterozygous (Tt) plant The dwarf parent gives only t, so a 1:1 ratio shows the tall parent made T and t gametes equally.

2. How many kinds of gametes does an AaBbCcDd individual make if all genes assort independently?

A. 8
B. 4
C. 32
D. 16

D. 16 Each heterozygous gene doubles the gamete types: $2^4 = 16$.

3. Two carriers of a recessive condition have three children. What is the chance at least one is affected?

A. 37/64
B. 1/64
C. 27/64
D. 3/4

A. 37/64 The chance none is affected is $(3/4)^3 = 27/64$, so at least one is $1 - 27/64 = 37/64$.

4. A type A mother and a type B father have a type O child. What are the parents' genotypes?

A. I^AI^B and ii
B. I^Ai and I^Bi
C. I^AI^A and I^BI^B
D. ii and I^Bi

B. I^Ai and I^Bi A type O child is ii, so each parent passed on an i allele.

5. Two white sweet pea strains cross to give purple F_1, and the F_2 is 9 purple : 7 white. What explains this?

A. Codominance of two alleles
B. Duplicate genes with one dominant
C. Incomplete dominance at one gene
D. Complementary genes in one pathway

D. Complementary genes in one pathway Purple needs a working allele at both genes (C_P_, 9/16). The white parents were blocked at different steps.

6. A pedigree shows an affected father whose daughters are all affected and whose sons are all unaffected. Which pattern fits best?

A. X linked dominant
B. X linked recessive
C. Autosomal recessive
D. Mitochondrial

A. X linked dominant A father gives his X to every daughter and his Y to every son.

7. A woman whose father had hemophilia has children with an unaffected man. What fraction of their sons will have hemophilia?

A. All of them
B. None of them
C. Half of them
D. A quarter of them

C. Half of them She must be a carrier. Sons get their X only from her, so half get the allele.

8. A testcross gives 450 parental and 50 recombinant offspring. How far apart are the genes?

A. 50 map units
B. 10 map units
C. 5 map units
D. 9 map units

B. 10 map units 50 ÷ 500 = 0.10, or 10 percent recombination.

9. A chi square test on a 3:1 cross gives 2.1. What do you conclude?

A. The genes must be linked together
B. Reject the model at p = 0.05
C. The data fit, so keep the model
D. The model is proven to be correct

C. The data fit, so keep the model With 1 df the critical value is 3.84, and 2.1 is below it.

10. Why does nondisjunction in meiosis I harm more gametes than in meiosis II?

A. Both meiosis I daughter cells go wrong
B. Meiosis II has no spindle to fail
C. Meiosis I gametes are always lost early
D. Crossing over is blocked in meiosis II

A. Both meiosis I daughter cells go wrong In meiosis I one cell gets both homologs and the other none, so all four gametes are abnormal.

CHAPTER 6

DNA Structure, Replication and Gene Expression

Read it in full: **Chapter 6 in the textbook**. See it in motion: **DNA Structure and Replication Lab** and **Gene Expression Lab**.

What to know

Three experiments made the case for DNA. **Griffith** (1928) found that heat-killed S pneumococcus could transform harmless R cells into lethal S cells. **Avery, MacLeod and McCarty** (1944) showed that only DNase stopped transformation. **Hershey and Chase** (1952) labeled phage protein with ^{35}S and DNA with ^{32}P; only the ^{32}P went into the bacteria. Chargaff found that base makeup varies by species but A = T and G = C within one (human DNA is about 30% A and 20% G).

Franklin's X-ray images showed a helix 2 nm wide with repeats at 0.34 and 3.4 nm. Watson and Crick built the model: a right-handed **double helix**, backbones outside, bases inside, strands **antiparallel**, A with T (two hydrogen bonds), G with C (three). Each pair is a purine plus a pyrimidine, which keeps the width even. Eukaryotic DNA wraps around eight histones (about 147 bp) to form **nucleosomes**, then the 30 nm fiber, loops and metaphase chromosomes. Euchromatin is open and active; heterochromatin is packed and mostly silent.

Replication is **semiconservative**: each new helix keeps one old strand. Meselson and Stahl grew E. coli in ^{15}N, then ^{14}N. After one round there was a single hybrid band (ruling out conservative); after two rounds, hybrid and light bands (ruling out dispersive). Bacteria use one origin; eukaryotes use thousands, because their forks are slower (about 50 nucleotides per second in humans).

At the fork, **helicase** unwinds, single-strand binding proteins hold the strands open, **topoisomerase** relieves strain ahead, **primase** lays an RNA primer, **DNA polymerase** adds to the 3′ end and proofreads, the sliding clamp holds it on, DNA polymerase I swaps the primers for DNA, and **ligase** seals nicks. Because polymerase only builds 5′ to 3′, the **leading strand** is continuous and the **lagging strand** is made as Okazaki fragments (1,000 to 2,000 nucleotides in bacteria, 100 to 200 in eukaryotes).

Proofreading and mismatch repair bring errors to about one in 10^9 to 10^{10} nucleotides. Linear chromosomes shorten each round because the last primer gap cannot be filled. **Telomeres** (TTAGGG repeats in humans) buffer the loss, and **telomerase**, a reverse transcriptase with its own RNA template, rebuilds them in germ and stem cells and in about 90 percent of cancers. Repair

pathways: mismatch repair (Lynch syndrome), base excision, nucleotide excision for thymine dimers (xeroderma pigmentosum), nonhomologous end joining and homologous recombination for double-strand breaks (BRCA1 and BRCA2).

Beadle and Tatum's one gene, one enzyme idea became one gene, one polypeptide, with exceptions for RNA genes and alternative splicing. The **central dogma** is DNA to RNA to protein. RNA polymerase reads the **template strand** 3′ to 5′ and builds RNA 5′ to 3′ with no primer. The mRNA matches the **coding strand** with U for T. Transcription has initiation at a promoter, elongation and termination. Bacteria use one polymerase and a sigma factor that reads the −35 and −10 boxes. Eukaryotes use three polymerases; transcription factors such as TFIID bind the TATA box before polymerase II joins.

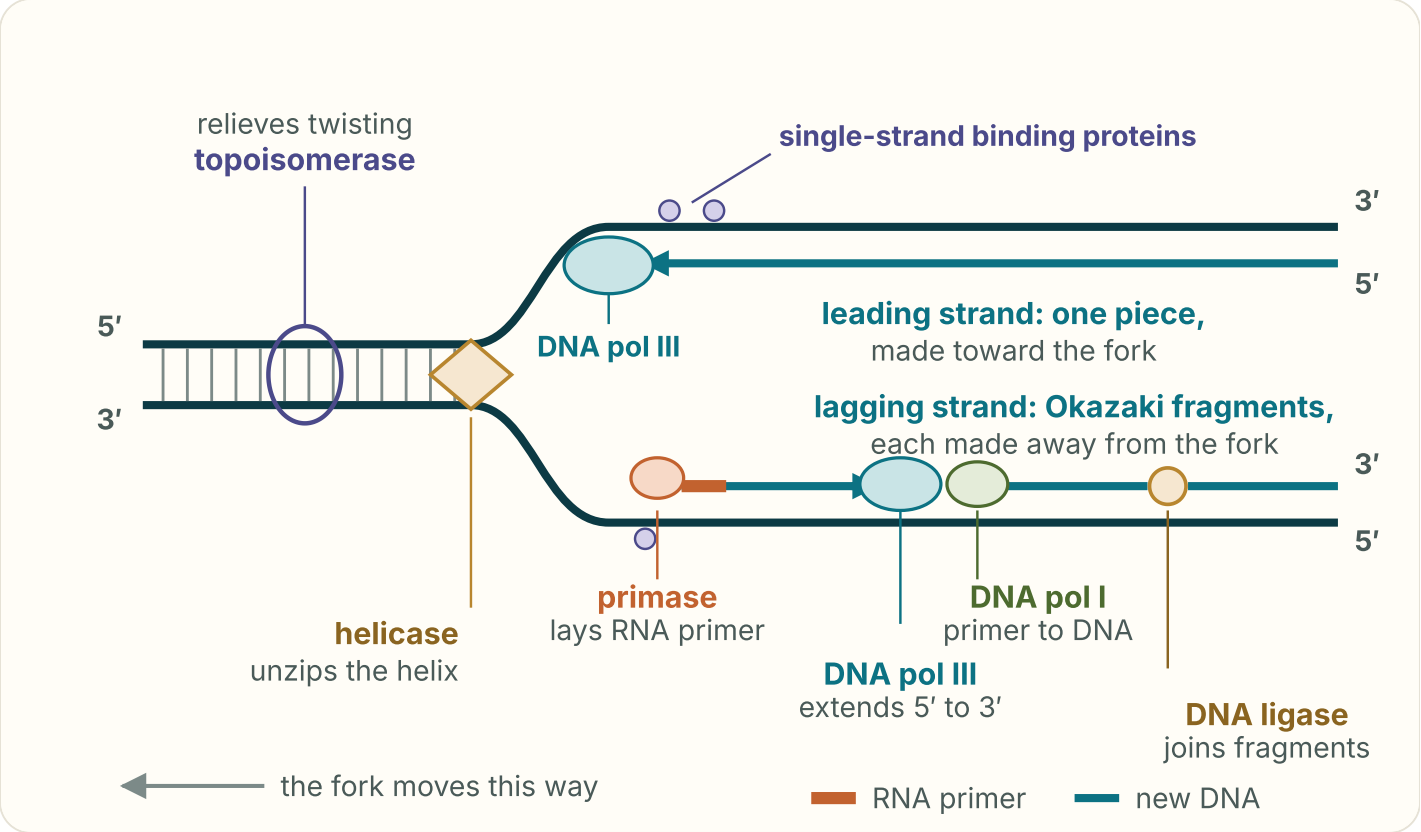
Eukaryotic pre-mRNA is processed: a **5′ cap** of modified guanine, a **poly-A tail** of 50 to 250 A's, and **splicing** by the spliceosome, which removes introns (GU at the 5′ end, AG at the 3′ end) as a lariat. The snRNAs do the catalysis, so the spliceosome acts as a ribozyme. **Alternative splicing** lets more than 90 percent of human multi-exon genes make several proteins from about 20,000 protein coding genes.

The **genetic code** uses 64 triplet codons: 61 sense codons and three stops (UAA, UAG, UGA). AUG means methionine and start. The code is redundant but never ambiguous, read in a fixed frame and nearly universal. Nirenberg and Matthaei cracked UUU = phenylalanine with poly-U. **Aminoacyl-tRNA synthetases** (20 of them) load each tRNA using ATP. Wobble at the third codon base lets about 45 tRNAs read 61 codons.

Ribosomes (70S in bacteria, 80S in eukaryotes) have A, P and E sites. In initiation, the small subunit finds the start (Shine-Dalgarno in bacteria; the cap and scanning in eukaryotes) and the initiator tRNA enters the P site. Elongation repeats codon recognition, peptide bond formation by rRNA (a ribozyme) and translocation, about four high-energy phosphate bonds per amino acid. A release factor reads the stop codon. Signal peptides send proteins to the rough ER.

A **mutation** is a permanent change in DNA. Germline mutations can be inherited; somatic ones cannot. Substitutions are **silent** (same amino acid), **missense** (a different one, as in sickle cell, GAG to GUG, Glu to Val) or **nonsense** (a stop). Insertions or deletions not in multiples of three

cause **frameshifts**. Cystic fibrosis ΔF508 is an in-frame loss of three bases. Repeat expansions cause Huntington disease (CAG) and fragile X syndrome (CGG). Mutagens include base analogs, alkylating agents, intercalators, UV light and X-rays; the Ames test screens chemicals using Salmonella.



The replication fork. Polymerase only adds to a 3′ end, so one new strand is made smoothly toward the fork and the other in pieces away from it.

LOCK THESE IN

- Hershey and Chase: ^{32}P (DNA) went in; ^{35}S (protein) stayed out.
- Chargaff: A = T and G = C. A to T 2 hydrogen bonds; G to C 3.
- Semiconservative: after one round in ^{14}N, one hybrid band.
- Polymerase needs a primer and adds only to a 3′ end. Lagging strand = Okazaki fragments.
- mRNA matches the coding strand (U for T) and complements the template.
- Cap, tail, splicing. GU to AG introns. Alternative splicing multiplies proteins.
- AUG start; UAA, UAG, UGA stop. 64 codons, 61 sense, about 45 tRNAs.
- Frame first, then codon: frameshift, silent, missense or nonsense.

Key terms and comparisons: the replication fork

Protein	Job
Helicase	Unwinds the helix (uses ATP)
Single-strand binding proteins	Keep separated strands apart
Topoisomerase	Relieves overwinding ahead of the fork
Primase	Makes a 5 to 10 nucleotide RNA primer
DNA polymerase III (bacteria); ε and δ (eukaryotes)	Adds nucleotides 5′ to 3′; proofreads
Sliding clamp	Keeps polymerase on the template
DNA polymerase I	Replaces RNA primers with DNA
Ligase	Seals nicks with phosphodiester bonds
Telomerase	Adds TTAGGG repeats using its own RNA

Key terms and comparisons: expression and mutation

Item	Bacteria or rule	Eukaryotes or example
RNA polymerases	One; sigma factor reads −35 and −10	Three; TFIID binds TATA box
Processing	Little or none	5′ cap, poly-A tail, splicing
Translation start	Shine-Dalgarno; fMet	Cap and scanning; Met
Ribosome	70S (50S + 30S)	80S (60S + 40S)
Silent	Same amino acid	CTT to CTC (Leu)
Missense	Different amino acid	Sickle cell Glu6Val
Nonsense	Early stop	Some Duchenne alleles
Frameshift	Indel not a multiple of 3	Tay-Sachs four base insertion
In-frame indel	Multiple of 3	Cystic fibrosis ΔF508
Repeat expansion	Repeat grows over generations	Huntington (CAG), fragile X (CGG)

Worked cases

WORKED CASE 1 · FROM TEMPLATE STRAND TO PROTEIN

A template strand reads 3′-TACAAAGGCATT-5′. Write the coding strand, the mRNA and the amino acids.

STEP 1 · CODING STRAND

Complement the template: 5′-ATGTTTCCGTAA-3′.

STEP 2 · MRNA

Same as the coding strand with U for T: **5′-AUGUUUCCGUAA-3′**.

STEP 3 · READ CODONS

AUG Met, UUU Phe, CCG Pro, UAA stop.

STEP 4 · RESULT

Met-Phe-Pro, then translation stops.

ANTICODON CHECK

The tRNA for UUU has anticodon 3′-AAA-5′. Always write anticodons antiparallel.

WORKED CASE 2 · NAMING THE MUTATION

An mRNA reads 5′-AUG GCA UCA UGG UAA-3′ (Met-Ala-Ser-Trp-stop). Classify these changes: (a) GCA to GCC; (b) UGG to UGA; (c) the G at position 4 is deleted.

STEP 1 · FRAME FIRST

(a) and (b) are substitutions, so the frame is kept. (c) removes one base, so the frame shifts.

STEP 2 · (A)

GCC is still alanine: **silent**.

STEP 3 · (B)

UGA is a stop: **nonsense**; the protein ends after Ser.

STEP 4 · (C)

The new reading is AUG CAU CAU GGU AA...: Met-His-His-Gly and onward. Every codon after the deletion changes: **frameshift**.

WORKED CASE 3 · MESELSON AND STAHL, ONE MORE ROUND

Cells grown in ^{15}N are moved to ^{14}N. What share of DNA molecules is hybrid after three rounds?

STEP 1 · COUNT MOLECULES

One molecule becomes 2, then 4, then **8**.

STEP 2 · COUNT OLD STRANDS

The 2 original heavy strands never break up, so 2 molecules are hybrid.

STEP 3 · DIVIDE

2 ÷ 8 = **1/4 hybrid**; the other 3/4 are light.

WHY THE MODELS DIFFER

Conservative would keep one fully heavy molecule; dispersive would give one blended band. Semiconservative alone fits.

WORKED CASE 4 · CHARGAFF ON A NEW SAMPLE

Double stranded DNA from a bacterium is 21 percent guanine. Find A, T and C.

STEP 1 · PAIR G WITH C

C = G = **21%**, so G plus C = 42%.

STEP 2 · FIND A PLUS T

100 − 42 = 58%.

STEP 3 · SPLIT IT

A = T = **29%**.

MELTING

This sample has fewer G-C pairs than a 60% GC sample, so it melts at a lower temperature.

TRAPS PEOPLE FALL INTO

- **Saying the mRNA matches the template.** It matches the coding strand (U for T) and complements the template.
- **Letting polymerase start a strand alone.** DNA polymerase needs a primer; RNA polymerase does not.
- **Saying new DNA grows 3′ to 5′.** New strands always grow 5′ to 3′.
- **Forgetting to reverse the anticodon.** Codon 5′-GGA-3′ pairs with anticodon 3′-CCU-5′.
- **Calling every substitution harmful.** Silent changes leave the protein alone; many mutations are neutral.
- **Thinking any indel causes a frameshift.** Multiples of three keep the frame, as ΔF508 does.
- **Expecting bacteria to splice introns.** They lack spliceosomes, so scientists insert cDNA.
- **Treating ribosome S values as additive.** 50S plus 30S makes 70S because S measures sedimentation.

Chapter 6 self-test · ten items

1. In the Hershey and Chase experiment, which label ended up inside the bacteria?

A. ^{32}P in phage DNA

B. ^{14}C in phage sugars

C. ^{15}N in phage bases

D. ^{35}S in phage protein

A. ^{32}P in phage DNA Phosphorus marks DNA. It entered the cells and appeared in new phages, while sulfur-labeled coats stayed outside.

2. A DNA sample is 22 percent thymine. What percent is cytosine?

A. 56 percent
B. 28 percent
C. 22 percent
D. 44 percent

B. 28 percent A = T = 22%, so A plus T = 44%. That leaves 56% for G plus C, so C = 28%.

3. After one round of replication in ^{14}N, Meselson and Stahl saw a single hybrid band. Which model did that rule out?

A. The dispersive model
B. The semiconservative model
C. The conservative model
D. No model at all

C. The conservative model The conservative model predicts one heavy and one light band. A single hybrid band rules it out.

4. Why is the lagging strand made in short pieces?

A. Primase cannot read that template
B. Ligase works only on short pieces
C. Helicase opens only short stretches
D. Polymerase can only build 5′ to 3′

D. Polymerase can only build 5′ to 3′ On the lagging template, 5′ to 3′ synthesis points away from the fork, so new primers are needed as the fork opens.

5. Which enzyme carries its own RNA template to extend chromosome ends?

A. Topoisomerase
B. Primase
C. Telomerase
D. DNA ligase

C. Telomerase Telomerase is a reverse transcriptase that adds TTAGGG repeats to the 3′ overhang.

6. A coding strand reads 5′-ATGGCC-3′. What is the mRNA?

A. 3′-AUGGCC-5′
B. 5′-AUGGCC-3′
C. 5′-GGCCAU-3′
D. 5′-UACCGG-3′

B. 5′-AUGGCC-3′ The mRNA has the coding strand sequence with U in place of T.

7. A human gene placed in bacteria makes a long, nonworking protein. What is the likely cause?

A. Its introns were never spliced out
B. Bacteria read a different genetic code
C. The poly-A tail blocked the ribosome
D. Bacterial ribosomes cannot make peptides

A. Its introns were never spliced out Bacteria lack spliceosomes, so introns are translated. Using cDNA from mature mRNA avoids this.

8. A codon changes from UAC (Tyr) to UAA. What kind of mutation is this?

A. Missense
B. Frameshift
C. Silent
D. Nonsense

D. Nonsense UAA is a stop codon, so translation ends early.

9. Which site on the ribosome holds the tRNA carrying the growing chain?

A. The P site
B. The A site
C. The E site
D. The cap site

A. The P site The P (peptidyl) site holds the chain; the A site takes the next charged tRNA; the E site lets empty tRNAs exit.

10. Which repair system fails in xeroderma pigmentosum?

A. DNA mismatch repair pathway
B. Homologous recombination
C. Nucleotide excision repair
D. Base excision repair

C. Nucleotide excision repair Nucleotide excision repair removes bulky thymine dimers made by UV light.

Cram sheet

Everything from chapters 5 and 6 in one place. Cover the right column and see if you can rebuild each row.

Numbers and ratios

Item	Value	Note
Monohybrid F_2	3:1; 1:2:1	Testcross 1:1
Dihybrid F_2	9:3:3:1	Testcross 1:1:1:1
Epistasis ratios	9:3:4, 12:3:1, 9:7, 15:1	Two interacting genes
Chi square critical	3.84, 5.99, 7.81	1, 2, 3 df at p = 0.05
Sibling carrier	2/3	Parents both carriers
Map unit	1% recombination	Maximum 50%
Down syndrome	About 1 in 700 births	About 95% from nondisjunction
Helix	2 nm; 0.34 nm per bp; about 10 bp per turn	A-T 2 bonds, G-C 3
Nucleosome	About 147 bp on 8 histones	H1 on linker DNA
Okazaki fragments	1,000 to 2,000 nt (bacteria); 100 to 200 (eukaryotes)	Lagging strand
Error rate	About 1 in 10^9 to 10^{10}	After proofreading and mismatch repair
Codons	64 total, 61 sense, 3 stop	About 45 tRNAs
Elongation cost	About 4 high-energy bonds per amino acid	2 to charge tRNA, 2 GTP
Huntington repeat	CAG; under about 27 normal, 36 or more disease	Fragile X: CGG over 200

Key terms and comparisons

Pair	How to tell them apart
Incomplete vs codominance	Intermediate blend in the heterozygote; both alleles fully shown.
Pleiotropy vs epistasis	One gene, many traits; one gene masks another.
Penetrance vs expressivity	Shows at all; how strongly it shows.
Linked vs sex linked	Same chromosome; on a sex chromosome.
Aneuploidy vs polyploidy	One chromosome extra or missing; whole extra sets.
Leading vs lagging strand	Continuous; Okazaki fragments.
Template vs coding strand	Read by polymerase; same sequence as mRNA.
Intron vs exon	Spliced out; kept in mature mRNA.
Germline vs somatic mutation	Can be inherited; affects only that cell line.

Rules that win points

Topic	The rule
Probability	Multiply within a combination, add across combinations.
At least one	1 minus the chance of none.
X linked	Write alleles on the X; answer by sex.
Gene order	Distances add; the middle gene splits the longest distance.
Reading the code	Write mRNA 5′ to 3′, then read triplets from AUG.
Mutation effect	Check the frame first, then the codon.

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