GENETICS · STUDY GUIDE

Exam 4 Study Guide

This guide covers chapters 7 and 8, the molecular heart of the course. Chapter 7 follows DNA as it is copied, recombined, damaged and repaired. Chapter 8 follows a gene into RNA and then into protein. Most of this material is about machines and rules, so the tables matter. Work one chapter at a time, copy the tables by hand, then try each worked example with the steps covered. Finish each chapter with its ten item self-test. The cram sheet at the end puts every enzyme, rule and number in one place.

WHAT THIS GUIDE COVERS

- Chapter 7 · semiconservative replication, the fork, eukaryotic origins and telomerase, homologous recombination and gene conversion, mutation types and mutagens, repair pathways and transposable elements.
- Chapter 8 · bacterial and eukaryotic transcription, capping, splicing and polyadenylation, editing and NMD, cracking the code, wobble, the ribosome and protein targeting.

CHAPTER 7

DNA Replication, Recombination, Mutation and Repair

What to know

Replication is **semiconservative**: each daughter keeps one old strand. Meselson and Stahl grew *E. coli* in ^{15}N, moved them to ^{14}N and spun the DNA in a CsCl gradient. One generation gave a single hybrid band, which ruled out conservative copying. Two generations gave hybrid and light bands, which ruled out dispersive copying. After n generations the hybrid fraction is $2/2^n$.

Every DNA polymerase adds only to a free 3′-OH and needs a primer. Primase lays down short RNA primers. Because the strands are antiparallel, the **leading strand** grows continuously toward the fork and the **lagging strand** is made away from it as **Okazaki fragments**: 1,000 to 2,000 nucleotides in bacteria and 100 to 200 in eukaryotes.

At the *E. coli* fork, DnaA melts *oriC*, DnaB helicase unwinds, SSB coats single strands and gyrase relieves supercoils. Pol III does the main synthesis, with a beta clamp for processivity and an epsilon subunit for 3′ to 5′ proofreading. Pol I removes primers and fills gaps, and ligase seals the nicks. Base selection, proofreading and mismatch repair bring errors to about one in 10^9 to 10^{10}.

Eukaryotes use thousands of origins. In G1, ORC, Cdc6 and Cdt1 load MCM helicases to **license** origins. In S phase, CDK and DDK fire them, and high CDK blocks relicensing, so each origin fires once per cycle. Pol α-primase starts chains, pol ε makes the leading strand and pol δ the lagging strand, with PCNA as the clamp. FEN1 and RNase H remove primers.

Linear ends cannot be fully copied: this is the **end replication problem**. Telomeres of TTAGGG repeats buffer the loss, and shelterin and the t-loop hide the end. **Telomerase** is a reverse transcriptase (TERT) carrying its own RNA template (TERC). It extends only the G-rich 3′ strand. Most somatic cells lack it and senesce after 40 to 60 divisions, while most cancers turn it back on.

Homologous recombination repairs double strand breaks and makes meiotic crossovers. The steps are: break (Spo11 in meiosis), resection to 3′ tails, Rad51 strand invasion (loaded by BRCA2), synthesis, then either SDSA (always a noncrossover) or a double Holliday junction, which can be cut as a crossover or a noncrossover. Mismatches in heteroduplex DNA cause **gene conversion**, seen as 6:2 or 5:3 octads in fungi.

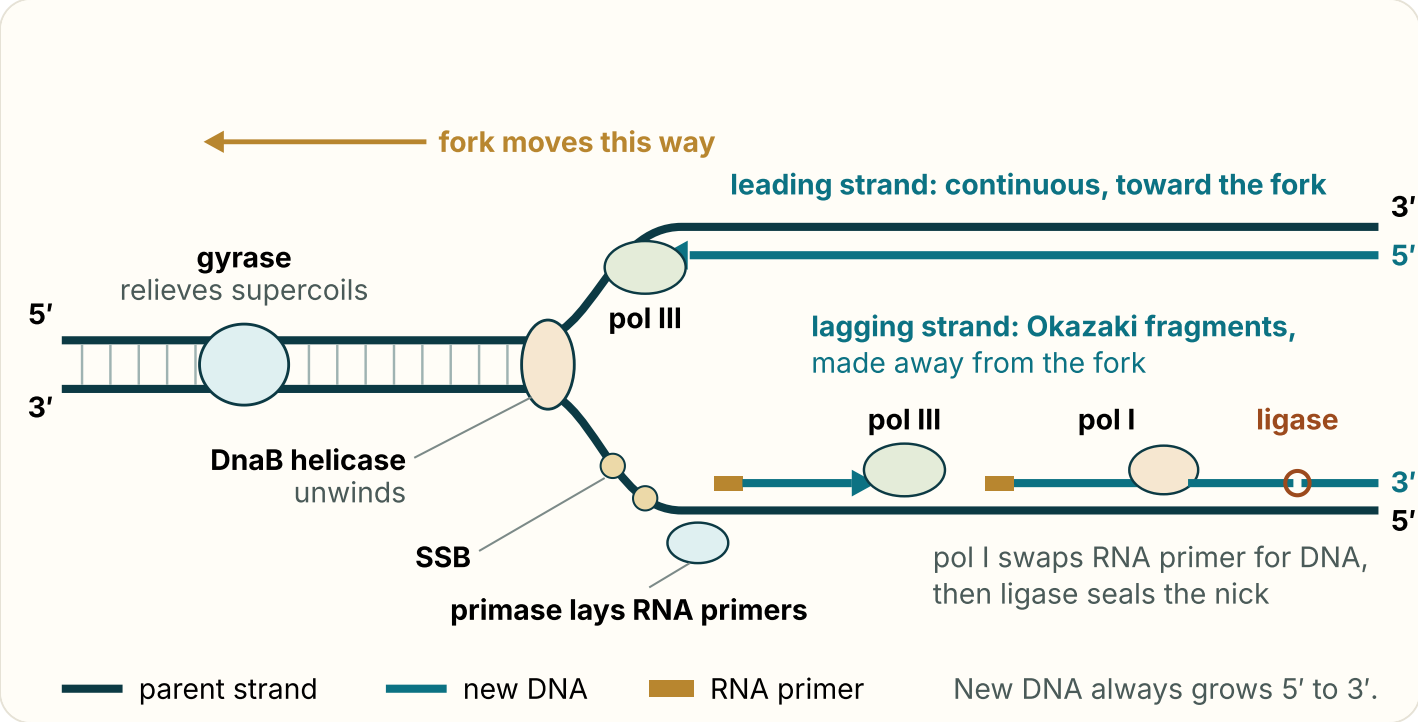
A **transition** swaps purine for purine or pyrimidine for pyrimidine. A **transversion** swaps a purine for a pyrimidine. By effect, mutations are silent, missense, nonsense or frameshift. Spontaneous sources include replication errors, slippage, depurination, deamination and oxidation. Deamination of 5-methylcytosine makes thymine, so CpG sites are hot spots. Mutagens leave fingerprints: EMS gives G:C to A:T, acridines give frameshifts, UV makes pyrimidine dimers and ionizing radiation breaks DNA. Repeat expansions show anticipation. The human germ line rate is about 1.2×10^{-8} per bp per generation, which is about 74 new mutations per child.

Each repair pathway has its damage. Direct reversal: photolyase and MGMT. Base excision: a glycosylase removes one base. Nucleotide excision: bulky lesions such as UV dimers (defective in xeroderma pigmentosum). Mismatch repair: replication errors on the new strand (defective in Lynch syndrome). Double strand breaks: homologous recombination or error prone NHEJ.

Transposable elements move as DNA (cut and paste) or through RNA (copy and paste, such as L1 and Alu), and leave a short target site duplication.

LOCK THESE IN

- Hybrid fraction after n generations = $2/2^n$.
- Polymerases add to 3′ ends and need a primer. That forces Okazaki fragments.
- Pol III builds; pol I removes primers; ligase seals.
- Origins are licensed in G1 and fired in S, once per cycle.
- Telomerase extends only the G-rich 3′ strand, using its own RNA template.
- SDSA gives noncrossovers. A double Holliday junction can go either way.
- Transition: A:T to G:C or back. Transversion: purine to pyrimidine.
- XP = nucleotide excision. Lynch = mismatch repair. BRCA = homologous recombination.



One fork, two ways of copying. Polymerase only extends a 3′ end, so one new strand runs toward the fork and the other is stitched together from Okazaki fragments.

Numbers and formulas for this chapter

Quantity	Formula or value	Use
Hybrid fraction (Meselson and Stahl)	$2 \div 2^n$; light = $1 - 2/2^n$	n ≥ 1 generations in ^{14}N
Replication time, one origin	genome ÷ (2 × fork speed)	two forks share the work
Minimum origins	chromosome length ÷ (2 × speed × S phase seconds)	round up
Divisions before senescence	(start length − critical length) ÷ loss per division	telomere problems
Error layers	base selection 10^{-4} to 10^{-5}; proofreading about 10^{-7}; with mismatch repair 10^{-9} to 10^{-10}	fidelity
Gene conversion frequency	non 4:4 octads ÷ total octads	fungal octads
Postmeiotic segregation share	(5:3 + 3:5) ÷ all conversions	unrepaired heteroduplex
Mutation rate, P_0 method	$m = -\ln P_0$; rate = m ÷ final cells	zeros avoid jackpots
New mutations per child	$1.2 \times 10^{-8} \times 6.2 \times 10^9 \approx 74$	mostly paternal
Frameshift test	indel length not a multiple of 3	shifts every downstream codon

Worked examples

WORKED EXAMPLE 1 · FOUR GENERATIONS OF DENSITY LABELING

E. coli fully labeled with ^{15}N grow for four generations in ^{14}N. What fraction of DNA molecules is hybrid, and what fraction is light?

STEP 1 · COUNT MOLECULES

One molecule doubles four times: $2^4 = 16$ molecules.

STEP 2 · FOLLOW THE OLD STRANDS

The two heavy strands are never lost. They sit in exactly 2 molecules, each paired with a light strand.

STEP 3 · THE REST

16 − 2 = 14 molecules are all light.

STEP 4 · ANSWER

Hybrid = 2/16 = **1/8**. Light = 14/16 = **7/8**. No heavy band.

WORKED EXAMPLE 2 · HOW MANY ORIGINS

A chromosome is 150 Mb. Forks move at 50 nucleotides per second and S phase lasts 6 hours. What is the minimum number of origins?

STEP 1 · ONE ORIGIN PER SECOND

Two forks at 50 nt/s each cover 100 nt per second.

STEP 2 · ONE ORIGIN IN S PHASE

6 hours = 21,600 s. 100 × 21,600 = 2.16×10^6 nt, or 2.16 Mb.

STEP 3 · DIVIDE

150 ÷ 2.16 = 69.4, so at least **70 origins**.

REALITY CHECK

Real cells use more, because origins fire at staggered times and forks stall.

WORKED EXAMPLE 3 · A TELOMERE BUDGET

A cell line starts with telomeres of 12,000 bp and loses 125 bp per division. Cells senesce at 5,000 bp. How many divisions remain?

STEP 1 · ALLOWED LOSS

12,000 − 5,000 = 7,000 bp.

STEP 2 · DIVIDE

7,000 ÷ 125 = **56 divisions**, inside the Hayflick range of 40 to 60.

TRAP

Telomerase would add repeats only to the G-rich 3′ strand. The C-rich strand is filled in by ordinary lagging strand synthesis.

WORKED EXAMPLE 4 · GENE CONVERSION IN OCTADS

A cross of black (g^+) and gray (g) *Sordaria* gives 1,000 octads: 960 at 4:4, 14 at 6:2, 12 at 2:6, 9 at 5:3 and 5 at 3:5. Find the conversion frequency and the share of conversions from unrepaired heteroduplex.

STEP 1 · CHECK THE TOTAL

960 + 14 + 12 + 9 + 5 = 1,000.

STEP 2 · ALL CONVERSIONS

14 + 12 + 9 + 5 = 40. 40 ÷ 1,000 = **4.0 percent**.

STEP 3 · POSTMEIOTIC SEGREGATION

5:3 and 3:5 = 9 + 5 = 14. 14 ÷ 40 = **35 percent**.

STEP 4 · READ IT

The other 65 percent were mismatches repaired toward one allele, which gives 6:2 or 2:6.

TRAPS PEOPLE FALL INTO

- **Thinking DNA polymerase can start a chain.** It needs a primer. RNA polymerase and primase do not.
- **Saying the lagging strand runs 3′ to 5′.** Every new strand grows 5′ to 3′. The lagging strand just grows away from the fork in pieces.
- **Mixing up pol I and pol III jobs.** Pol III makes most DNA. Pol I removes primers and fills gaps.
- **Saying telomerase lengthens both strands.** Only the G-rich 3′ strand.
- **Calling a damaged base a mutation.** It becomes a mutation only after replication fixes the wrong pair.
- **Calling every substitution a transition.** Purine to pyrimidine is a transversion.
- **Mixing repair pathways and diseases.** XP is NER, Lynch is mismatch repair, BRCA is homologous recombination.
- **Confusing the target site duplication with the element's inverted repeats.** The duplication is host DNA, in direct orientation.

Chapter 7 self-test · ten items

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

- A Rolling circle replication
- B Dispersive replication
- C Semiconservative replication
- D Conservative replication

D. Conservative replication. Conservative copying would have given one heavy and one light band. Dispersive was ruled out only by the second generation.

2. Why is the lagging strand made in pieces?

- A Polymerase adds only to 3′ ends, and strands are antiparallel
- B Primase can bind only to the lagging strand template DNA
- C Helicase unwinds that template much more slowly than the other
- D Ligase must cut the strand before it can be copied

A. Polymerase adds only to 3′ ends, and strands are antiparallel. On one template the polymerase has to move away from the fork. It can only restart from new primers, so it makes Okazaki fragments.

3. Which *E. coli* enzyme removes RNA primers and fills the gaps?

- A DNA gyrase
- B DNA polymerase III
- C DNA polymerase I
- D DnaB helicase

C. DNA polymerase I. Pol I uses its 5′ to 3′ exonuclease to remove primers and its polymerase to fill in. Ligase then seals the nick.

4. What stops a eukaryotic origin from firing twice in one cell cycle?

A Each origin is cut out after it fires once

B High CDK in S phase blocks new MCM loading

C Telomerase caps each origin after use

D Helicase destroys the origin DNA it opens

B. High CDK in S phase blocks new MCM loading. Licensing needs low CDK in G1. Once CDK rises, Cdc6 and Cdt1 are blocked, so no origin can be loaded again until the next G1.

5. Which pair of base changes are both transitions?

A A:T to G:C, and C:G to T:A

B G:C to T:A, and A:T to C:G

C A:T to T:A, and G:C to C:G

D A:T to C:G, and C:G to G:C

A. A:T to G:C, and C:G to T:A. A transition keeps the purine and pyrimidine positions: a purine is replaced by the other purine. The other options swap purines for pyrimidines.

6. Why are methylated CpG sites mutation hot spots?

A CpG sites are copied twice during each S phase

B Methylation draws UV damage to those cytosines alone

C Deaminated 5-methylcytosine becomes thymine, a normal base

D Methyl groups block DNA polymerase from proofreading there

C. Deaminated 5-methylcytosine becomes thymine, a normal base. Plain cytosine deaminates to uracil, which cells remove. Methylcytosine becomes thymine, and the G:T mismatch is fixed less reliably.

7. A chemical gives only single base insertions and deletions. What kind of mutagen is it most likely to be?

A. An alkylating agent such as EMS or nitrosoguanidine

B. A deaminating agent such as nitrous acid

C. A base analog such as 5-bromouracil

D. An intercalating agent such as an acridine

D. An intercalating agent such as an acridine. Intercalators slip between stacked bases and cause frameshifts. The other three cause substitutions, mainly transitions.

8. A person has severe sun sensitivity and many skin cancers from childhood. Which repair pathway is most likely defective?

A. Nonhomologous DNA end joining

B. Nucleotide excision repair

C. Base excision repair

D. Mismatch repair

B. Nucleotide excision repair. This is xeroderma pigmentosum. NER removes UV dimers, and without it UV damage piles up.

9. In an octad, a 5:3 ratio of spore colors shows what?

A. A crossover between the gene and its own centromere only

B. A normal meiosis with no recombination at all near the gene

C. A mismatch that was repaired toward one parental allele

D. A heteroduplex mismatch left unrepaired until the next mitosis

D. A heteroduplex mismatch left unrepaired until the next mitosis. An unrepaired mismatch separates at the postmeiotic mitosis, so one spore pair is non identical. A repaired one gives 6:2 or 2:6.

10. An Alu element is flanked by the direct repeat AGTCCA, which appears only once in the normal allele. What is AGTCCA?

A. The binding site for the L1 reverse transcriptase

B. A 6 bp target site duplication of host DNA

C. A promoter carried inside the Alu sequence

D. The inverted terminal repeat of the element

B. A 6 bp target site duplication of host DNA. Staggered cuts and gap filling copy the short host sequence on both sides of the insertion, in the same orientation.

Read the full chapter: Chapter 7 · DNA Replication, Recombination, Mutation and Repair

CHAPTER 8

Transcription, RNA Processing and Translation

What to know

RNA polymerase makes RNA 5′ to 3′ from the **template strand** and needs no primer. The RNA matches the **coding strand**, with U for T. Positions count from +1, the first transcribed base, and there is no position 0.

In bacteria, a **sigma factor** joins the core polymerase ($\alpha_2\beta\beta'\omega$) to make the holoenzyme. σ^{70} reads promoters with a −35 element (TTGACA) and a −10 element (TATAAT), spaced about 17 bp apart. The enzyme forms a closed complex, melts DNA into an open complex, makes some abortive short RNAs and then escapes. Bacteria stop at **intrinsic terminators** (a GC rich hairpin then a run of U) or with the **Rho** helicase. Transcription and translation are coupled.

Eukaryotes use three nuclear polymerases. Pol I makes large rRNAs, pol II makes mRNAs and most snRNAs, and pol III makes tRNAs and 5S rRNA. Low α-amanitin blocks pol II. Pol II needs general factors: TFIID (TBP binds the TATA box), TFIIA, TFIIB, pol II with TFIIF, TFIIE and TFIIH. TFIIH opens DNA and phosphorylates the **CTD** on serine 5 for escape. Serine 2

phosphorylation follows for elongation. The CTD recruits processing factors, so processing happens as the RNA is made. Enhancers act from afar through Mediator.

Pre-mRNA gets a **5′ cap** (7-methylguanosine joined 5′ to 5′), loses its **introns** and gains a **poly(A) tail** after cleavage downstream of AAUAAA. Introns start with GU and end with AG, with a branch point A upstream. The **spliceosome** (U1, U2, U4, U5, U6) runs two transesterifications and frees the intron as a **lariat**. The spliceosome is a ribozyme. Alternative splicing touches about 95 percent of human multi exon genes.

RNA editing changes coded sequence: APOBEC1 turns one C to U in apoB mRNA in the intestine, making apoB-48, and ADAR turns A to inosine. An exon junction complex left downstream of a stop codon flags it as premature, and **nonsense mediated decay** destroys the mRNA.

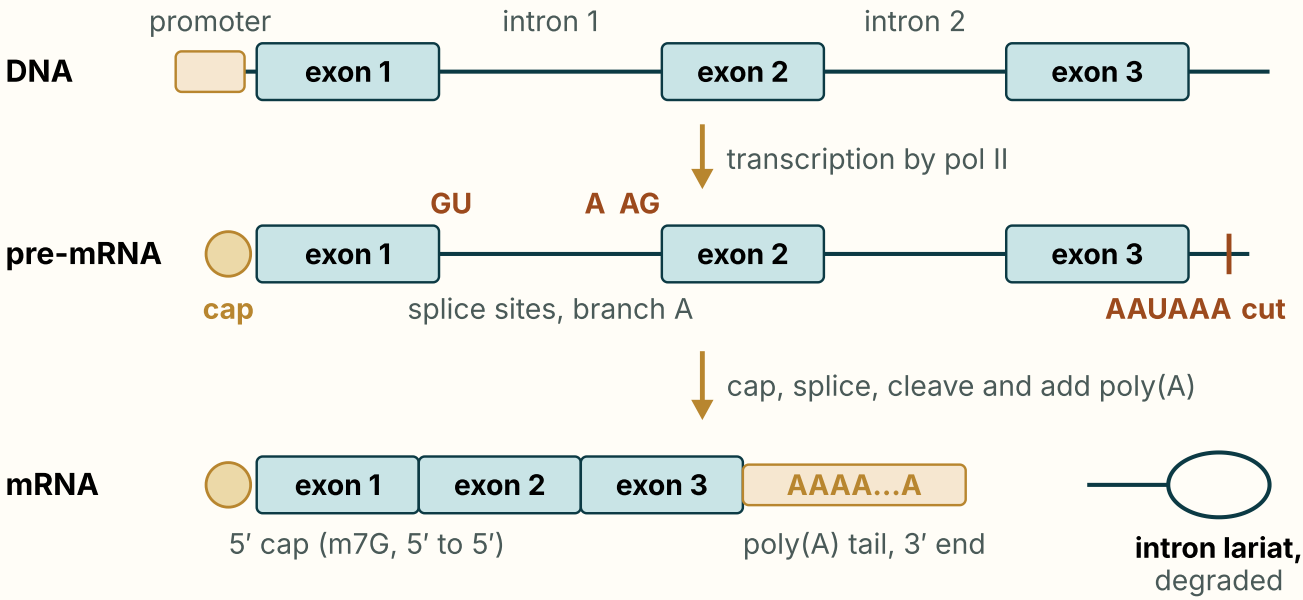
The code is a nonoverlapping **triplet** read from a fixed start. Crick and Brenner showed this with frameshifts: three + or three − mutations restored function. Poly(U) gave polyphenylalanine. Random copolymers, triplet binding and repeating copolymers assigned the rest. There are 61 sense codons and 3 stops (UAA, UAG, UGA), and AUG starts. The code is degenerate but unambiguous and nearly universal. Vertebrate mitochondria read UGA as tryptophan.

Codon and anticodon pair antiparallel. The anticodon's 5′ base pairs with the codon's 3′ base and can **wobble**: G reads C or U, U reads A or G, and inosine reads U, C or A. That is why fewer than 61 tRNAs are needed.

Aminoacyl tRNA synthetases charge tRNAs at a cost of two high energy bonds, and the ribosome trusts them. The ribosome has A, P and E sites, and its peptidyl transferase is 23S rRNA, another ribozyme. Bacteria start at a Shine Dalgarno sequence with fMet. Eukaryotes bind the cap and scan to a Kozak AUG. Each elongation cycle is EF-Tu decoding, peptide transfer and EF-G translocation. Release factors read stop codons. Signal sequences, SRP and the Sec61 translocon send proteins into the ER, and many antibiotics exploit 70S versus 80S differences.

LOCK THESE IN

- RNA = coding strand with U for T. Never the complement of the coding strand.
- Bacterial promoter: −35 TTGACA, −10 TATAAT, 17 bp spacer.
- Pol I rRNA, pol II mRNA, pol III tRNA and 5S. Low α-amanitin stops pol II.
- Introns: GU at the 5′ end, AG at the 3′ end, branch point A.
- Skipping an exon whose length is not a multiple of 3 shifts the frame.
- 61 sense codons, 3 stops. AUG starts.
- Wobble: G reads C or U; U reads A or G; I reads U, C or A.
- Bacterial protein cost is about 4 high energy bonds per peptide bond.



From pre-mRNA to mRNA. The cap goes on first, introns leave as lariats, and the tail is added after a cut downstream of AAUAAA. All of it happens while pol II is still working.

Code, wobble and counting rules

Item	Rule or formula	Note
Codon count	4^3 = 64; 61 sense, 3 stop	pairs give only 16
Reading frames	3 per strand; 6 per duplex	an ORF starts with AUG and has no in frame stop
Random copolymer	P(codon) = product of base fractions	sum codons for each amino acid
Repeating dinucleotide $(UC)_n$	one alternating polypeptide	same in every frame
Repeating trinucleotide $(UUC)_n$	three homopolymers	one per frame
Wobble G	reads codons ending C or U	anticodon 5′ base
Wobble U	reads codons ending A or G	
Wobble I	reads codons ending U, C or A	inosine from adenosine
Wobble C	reads G only	
Isoforms from exon clusters	product of choices in each cluster	product rule
Exon skip and frame	exon length ÷ 3 has a remainder: frameshift	remainder 0 keeps frame
Bacterial energy for n amino acids	2n (charging) + 1 (IF2) + 2(n − 1) (elongation) + 1 (RF3)	about 4n
Release factors	RF1: UAA, UAG. RF2: UAA, UGA. eRF1: all three	

Worked examples

WORKED EXAMPLE 1 · FROM CODING STRAND TO PROTEIN

A bacterial gene's coding strand reads 5′-ATGAAACCGTGGTAG-3′. Write the template strand, the mRNA and the peptide.

STEP 1 · TEMPLATE

Complementary and antiparallel: 3′-TACTTTGGCACCATC-5′.

STEP 2 · MRNA

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

STEP 3 · CODONS

AUG AAA CCG UGG UAG.

STEP 4 · TRANSLATE

Met-Lys-Pro-Trp, then UAG stops.

CHECK

If your mRNA is the complement of the coding strand, you copied the wrong strand.

WORKED EXAMPLE 2 · A RANDOM COPOLYMER

A random copolymer is made with A and C in a 2:1 ratio. Predict the share of each amino acid in the product.

STEP 1 · BASE ODDS

P(A) = 2/3 and P(C) = 1/3.

STEP 2 · CODON ODDS

AAA = 8/27. AAC, ACA and CAA = 4/27 each. ACC, CAC and CCA = 2/27 each. CCC = 1/27.

STEP 3 · GROUP BY AMINO ACID

Lys (AAA) 8/27. Asn (AAC) 4/27. Thr (ACA + ACC) 6/27. Gln (CAA) 4/27. His (CAC) 2/27. Pro (CCA + CCC) 3/27.

STEP 4 · PERCENTS

Lys **29.6%**, Thr **22.2%**, Asn and Gln **14.8%** each, Pro **11.1%**, His **7.4%**. Check: 8 + 6 + 4 + 4 + 3 + 2 = 27.

WORKED EXAMPLE 3 · READING CODONS WITH WOBBLE

Glycine uses GGU, GGC, GGA and GGG. What is the fewest tRNAs that can read all four? Which codons does an anticodon 5′-GCC-3′ read?

STEP 1 · LINE UP ANTIPARALLEL

The anticodon's 5′ base pairs with the codon's 3′ base.

STEP 2 · USE INOSINE

5′-ICC-3′ reads GGU, GGC and GGA.

STEP 3 · THE LAST CODON

GGG ends in G, which inosine cannot read. Add 5′-CCC-3′ or 5′-UCC-3′. So the minimum is **two tRNAs**.

STEP 4 · ANTICODON GCC

Its 5′ G reads C or U, so it reads **GGC and GGU**.

WORKED EXAMPLE 4 · THE ENERGY BILL

How many high energy phosphate bonds does a bacterium spend to make a 150 amino acid protein?

STEP 1 · CHARGING

150 × 2 = 300 (ATP to AMP).

STEP 2 · INITIATION

IF2 uses 1 GTP.

STEP 3 · ELONGATION

149 peptide bonds × 2 GTP (EF-Tu and EF-G) = 298.

STEP 4 · TERMINATION

RF3 uses 1 GTP.

STEP 5 · TOTAL

300 + 1 + 298 + 1 = **600**, about 4 per peptide bond.

TRAPS PEOPLE FALL INTO

- **Copying the coding strand into RNA as its complement.** The RNA has the coding strand sequence.
- **Looking for the U run on the template.** The template has A, and the U run is in the RNA.
- **Thinking eukaryotic pol II finds promoters by itself.** It needs the general transcription factors.
- **Thinking UTRs are introns.** UTRs sit in exons. Not all exon sequence codes for protein.
- **Pairing the anticodon's first base with the codon's first base.** Pair them antiparallel.
- **Saying the ribosome checks the amino acid.** It checks only the codon and anticodon. Synthetases must be accurate.
- **Counting 300 EF-Tu deliveries for a 300 residue protein.** fMet goes to the P site with IF2, so only 299.
- **Calling a nonsense mutation silent because the mRNA is gone.** NMD destroys the mRNA, but the mutation is still nonsense.

Chapter 8 self-test · ten items

1. A template strand reads 3′-TACGGA-5′. What is the RNA?

A 5′-AUGCCU-3′

B 3′-AUGCCU-5′

C 5′-UACGGA-3′

D 5′-TACGGA-3′

A. 5′-AUGCCU-3′. The RNA is complementary and antiparallel to the template, written 5′ to 3′: AUG CCU.

2. Which part of the bacterial holoenzyme recognizes the −35 and −10 elements?

A The α subunits

B The β′ subunit

C The sigma factor

D The Rho helicase

C. The sigma factor. Region 4 of sigma reads the −35 element and region 2 reads the −10 element. The core enzyme alone cannot find promoters.

3. A low dose of α-amanitin stops new mRNA but not new tRNA. Why?

A Pol III makes mRNA and pol II makes all tRNA

B Pol II is very sensitive; pol III is only moderately so

C tRNA is made by pol I, which resists the toxin

D Pol I is the enzyme blocked at the lowest dose

B. Pol II is very sensitive; pol III is only moderately so. Pol II makes pre-mRNA and is blocked at low doses. Pol III, which makes tRNA, needs a much higher dose.

4. What does TFIIH do at a pol II promoter?

A. Opens the DNA and phosphorylates CTD serine 5

B. Cuts the RNA downstream of the AAUAAA signal site

C. Adds the 7-methylguanosine cap to the RNA

D. Binds the TATA box and bends the DNA there

A. Opens the DNA and phosphorylates CTD serine 5. TFIIH is the last general factor. Its XPB subunit opens DNA and its CDK7 kinase marks serine 5, helping the polymerase escape.

5. What attacks the 5′ splice site in the first step of splicing?

A. The 3′-OH at the end of exon 2

B. A free guanosine from the nucleus

C. The triphosphate of the 5′ cap

D. The 2′-OH of the branch point A

D. The 2′-OH of the branch point A. The branch A forms a 2′ to 5′ bond with the intron's first base, making the lariat. A free guanosine is used by group I introns.

6. A splice site mutation causes skipping of a 98 nucleotide exon. What is the most likely effect?

A. Loss of exactly 32 amino acids in frame

B. An extra 98 amino acids added at the C end

C. A frameshift downstream of the skipped exon

D. No change, because exons are not coding

C. A frameshift downstream of the skipped exon. 98 ÷ 3 = 32 with remainder 2, so the reading frame shifts. A 120 nucleotide exon would remove 40 amino acids in frame.

7. Why does the intestine make apoB-48 while the liver makes apoB-100 from one gene?

A. The intestine reads the gene from the other DNA strand

B. The liver adds a much longer poly(A) tail to the mRNA

C. The intestine splices out half of the exons of the gene

D. Intestinal APOBEC1 edits a C to U, making a stop codon

D. Intestinal APOBEC1 edits a C to U, making a stop codon. Editing turns CAA (Gln) into UAA (stop), so intestinal protein is truncated.

8. Three single base insertions near each other in rIIB restored function. What did that show?

A. Each codon is separated by one spacer nucleotide

B. The code is read in triplets from a fixed start

C. Insertions are always silent in any coding DNA

D. The code is read in overlapping pairs of bases

B. The code is read in triplets from a fixed start. Three insertions put the frame back in step after a short garbled stretch. Only a triplet code read without commas fits.

9. Which codons can an anticodon with U at its 5′ wobble position read?

A. Codons ending in A or G

B. Codons ending in U, C or A

C. Codons ending in C or U

D. Codons ending in G only

A. Codons ending in A or G. U at the wobble position pairs with A and, by wobble, with G. G reads C or U, and inosine reads U, C or A.

10. Where on the ribosome does a new charged tRNA first bind during elongation?

A The E site

B The P site

C The A site

D The SRP site

C. The A site. EF-Tu delivers each aminoacyl tRNA to the A site. The peptidyl tRNA sits in the P site, and the empty tRNA leaves from the E site.

Read the full chapter: Chapter 8 · Transcription, RNA Processing and Translation

ONE PAGE

Cram sheet

Everything from chapters 7 and 8 in one place. Cover the right column and see if you can rebuild each row.

Chapter 7 at a glance

Topic	Key facts
Meselson and Stahl	gen 1: one hybrid band; gen 2: hybrid + light; hybrid fraction $2/2^n$
Fork enzymes	DnaA, DnaB helicase, SSB, gyrase, primase, pol III (beta clamp, epsilon proofreading), pol I, ligase
Eukaryotic replication	ORC, Cdc6, Cdt1 license MCM in G1; CDK and DDK fire in S; pol α, ε, δ; PCNA; FEN1
Telomeres	TTAGGG; shelterin; t-loop; telomerase = TERT + TERC; Hayflick limit 40 to 60
Recombination	Spo11 break; resection; Rad51 (BRCA2); D-loop; SDSA noncrossover; double Holliday junction either way
Gene conversion	6:2 or 2:6 (repaired); 5:3 or 3:5 (unrepaired, postmeiotic segregation)
Mutation classes	transition, transversion; silent, missense, nonsense, frameshift; repeat expansions show anticipation
Spontaneous damage	depurination; C to U; 5mC to T (CpG hot spots); 8-oxoG gives G:C to T:A
Mutagens	base analogs and EMS: transitions; acridines: frameshifts; UV: dimers; X rays: breaks
Ames test	*his*$^-$ Salmonella; S9 liver extract; count revertants
Repair	photolyase, MGMT; BER; NER (XP); mismatch repair (Lynch); HR (BRCA); NHEJ (Ku, ligase IV); TLS (pol η)
Transposons	DNA: cut and paste, IS, Tn; RNA: LTR, L1 (17%), Alu (10%); target site duplication

Chapter 8 at a glance

Topic	Key facts
Bacterial transcription	sigma + core; −35 TTGACA, −10 TATAAT; open complex; intrinsic or Rho termination
Eukaryotic polymerases	I: rRNA; II: mRNA (α-amanitin sensitive); III: tRNA, 5S
Pol II assembly	TFIID (TBP), IIA, IIB, pol II + IIF, IIE, IIH; CTD Ser5 then Ser2
Processing	cap m^7G 5′-5′; GU…A…AG introns; lariat; AAUAAA then cleave and add about 200 A
Editing and NMD	apoB C to U (APOBEC1); A to I (ADAR); EJC downstream of a stop triggers NMD
Code	triplet, nonoverlapping, degenerate, unambiguous, nearly universal; UAA, UAG, UGA stop
Wobble	G: C or U; U: A or G; I: U, C or A; C: G
Ribosomes	70S (30S + 50S) and 80S (40S + 60S); A, P, E sites; 23S rRNA forms peptide bonds
Initiation	Shine Dalgarno + fMet (IF2) in bacteria; cap, scanning, Kozak AUG in eukaryotes
Elongation	EF-Tu decoding, peptidyl transfer, EF-G translocation; about 4 high energy bonds per residue
Targeting	signal sequence, SRP, Sec61; N-linked glycosylation at Asn-X-Ser/Thr; preproinsulin to insulin
Inhibitors	streptomycin, tetracycline (30S); chloramphenicol, macrolides (50S); diphtheria toxin (eEF2); ricin (28S)

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