The Ultimate AP® Biology Unit 6 Cheat Sheet

6.1 DNA & RNA Structure

Building Blocks

Nucleotide = 5-C sugar + phosphate + nitrogenous base. Linked by covalent **phosphodiester** bonds (3' –OH to 5' phosphate) → sugar-phosphate backbone.
Purines (2 rings): A, G. **Pyrimidines** (1 ring): C, T, U. Purine always pairs w/ pyrimidine → even width.
Base pairing: A–T (2 H-bonds), G–C (3 H-bonds). RNA: A–U. GC-rich DNA → harder to separate (higher melting temp).

Feature	DNA	RNA
Sugar	Deoxyribose	Ribose
Bases	A, T, G, C	A, U, G, C
Strands	2 (helix)	1
Types	1	mRNA, tRNA, rRNA
Stability	High	Short-lived

Double Helix Rules

Antiparallel: one strand runs 5'→3', its partner runs 3'→5'. 5' end = free phosphate; 3' end = free –OH group.
Chargaff's rule: %A = %T, %G = %C. e.g., if G = 22% → C = 22%, so A + T = 56% → A = T = 28%.

Genomes

Prokaryotes: 1 circular chromosome in nucleoid + **plasmids** (small extra circles; e.g., antibiotic resistance).
Eukaryotes: multiple linear chromosomes in nucleus; DNA wrapped around **histones** → nucleosomes → chromatin.
Heredity molecule = DNA (Griffith, Avery, Hershey–Chase experiments). Some viruses use RNA instead.
Mito & chloroplasts: own small circular DNA, like bacteria → key evidence for endosymbiosis (Unit 2).

⚠ Chargaff's rule works only for double-stranded DNA, not for RNA or for a single strand of DNA on its own.

✓ Write complements 5'→3' to match AP answers: 5'-ATGC-3' pairs with 5'-GCAT-3' (= 3'-TACG-5' flipped).

6.2 DNA Replication

Semiconservative

Semiconservative: each new DNA = 1 old strand + 1 new strand (Meselson–Stahl, heavy ^{15}N → light ^{14}N).
Origins of replication: bubbles open, forks move both ways; prokaryotes 1 origin, eukaryotes many → faster.

Enzymes

Enzyme	Job
Helicase	Unzips (breaks H-bonds)
Topoisomerase	Relieves twisting ahead
Primase	Makes RNA primer
DNA pol III	Adds nts to 3' end
DNA pol I	Swaps primer for DNA
Ligase	Seals gaps (backbone)

Single-strand binding proteins keep strands apart.

Leading vs Lagging

DNA pol only adds to 3' end → new strand grows 5'→3'.
Leading strand: made continuously toward the replication fork; needs just one RNA primer to start.
Lagging strand: made away from the fork in short **Okazaki fragments**; many primers; DNA ligase joins them.
Why primers: DNA pol can't start a strand from scratch; it needs an existing 3' –OH to add onto.

Accuracy & Ends

Proofreading: DNA pol checks & fixes its own errors → only ~1 error per 10^9 nts left after mismatch repair.
Telomeres: repeat sequences at linear chromosome ends; shorten each round b/c lagging strand can't finish.
Telomerase extends them (stem cells, cancer cells).
Timing: S phase of interphase (Unit 4).

⚠ Polymerase reads the template 3'→5' but builds the new strand 5'→3'. Never the reverse direction.

✓ Lagging strand Q: fragments, many primers, ligase. Leading strand: continuous, just one primer.

6.3 Transcription & RNA Processing

Transcription (Nucleus)

Template strand (antisense) read 3'→5'; mRNA built 5'→3'.
Coding strand = same seq. as mRNA (T not U).
Initiation: transcription factors bind the **promoter** (TATA box) → RNA polymerase II binds & opens DNA.
Elongation: RNA pol unwinds DNA & adds complementary RNA nts to the 3' end. No primer needed.
Termination: terminator seq. (prokaryotes) or poly-A signal (eukaryotes) → RNA & RNA pol released from DNA.

Eukaryotic Processing

Change	Purpose
5' GTP cap	Ribosome binding; protects
3' poly-A tail	Stability; export
Splicing	Remove introns

Introns = intervening seqs, removed by the **spliceosome**.
Exons = expressed seqs, joined together.
Alternative splicing: diff. exon combos → many proteins from 1 gene (~20,000 genes → 100,000+ proteins).

Prokaryotes Differ

No nucleus → transcription & translation happen at the same time in cytoplasm. No introns, cap or tail.
Operons: several related genes under one promoter → transcribed together as 1 mRNA (see 6.5 for control).
Other RNAs: tRNA & rRNA are also transcribed from genes; some small RNAs (miRNA) regulate other genes.
Rate: many RNA pol molecules can transcribe 1 gene at the same time → lots of mRNA made very quickly.
Example: template 3'-TACGGA-5' → mRNA 5'-AUGCCU-3' → codons AUG, CCU → amino acids Met-Pro.

⚠ mRNA matches the CODING strand (U for T) and is complementary to the TEMPLATE strand.

✓ Mutation in an intron usually has no effect on the protein; in an exon or at a splice site, it can change it.

6.4 Translation

The Players

mRNA: read in **codons** (3 nts each) 5'→3', starting at AUG. 64 codons → 20 amino acids + 3 stop codons.
tRNA: **anticodon** pairs w/ codon; carries matching amino acid (loaded by aminoacyl-tRNA synthetase, uses ATP).
Ribosome: rRNA + protein; small & large subunits; A, P, E sites. rRNA catalyzes peptide bond (ribozyme).

Steps

Initiation: small subunit binds mRNA (5' cap) → finds **AUG** (Met) → initiator tRNA in P site → large subunit joins.
Elongation: tRNA enters A site → peptide bond forms → ribosome shifts 1 codon → empty tRNA exits E site.
Termination: stop codon (UAA, UAG, UGA) reaches the A site → release factor binds → polypeptide freed.
Energy: GTP powers tRNA entry & ribosome movement.

Site	Holds
A (arrival)	New aminoacyl-tRNA
P (peptide)	tRNA w/ growing chain
E (exit)	Empty tRNA leaving

Code Facts

Redundant: several codons → same AA (3rd base "wobble").
Unambiguous: each codon → only 1 AA.
Nearly universal code across life → evidence of common ancestry; lets bacteria make human insulin.
Polyribosomes: many ribosomes on 1 mRNA → many copies fast. **Retroviruses**: RNA → DNA via reverse transcriptase.
Targeting: signal peptide → ribosome docks on rough ER → protein secreted or put in membrane (Unit 2).

⚠ Use the codon chart with mRNA codons only, never with DNA triplets or with tRNA anticodons.

✓ DNA template → mRNA (complement, U for T) → codon chart → AA sequence. Write each step out.

6.5 Regulation of Gene Expression

Prokaryotes: Operons

Operon = promoter + operator + genes. **Repressor** binds the operator → physically blocks RNA polymerase.

Operon	Type	Default	Switch
lac	Inducible	Off	Lactose on
trp	Repressible	On	Trp off

lac: no lactose → repressor on operator → OFF. Lactose (allolactose) binds repressor → it falls off → genes ON.
trp: repressor inactive alone → ON. High tryptophan binds repressor (corepressor) → OFF. Feedback.
Positive control: low glucose → cAMP-CAP binds lac promoter → transcription ↑ (needs lactose too).

Eukaryotes: Many Levels

Chromatin: histone acetylation → loose → genes ON. DNA **methylation** → tight → genes OFF. Both are **epigenetic** (can be inherited, no change in DNA sequence).
Transcription factors: **activators** bind **enhancers** (can be far away) → ↑ transcription; **repressors** bind silencers.
After transcription: alt. splicing; mRNA lifespan; **miRNA/siRNA** bind mRNA → degrade or block translation.
After translation: protein folding, cleavage, phosphorylation; a ubiquitin tag → the proteasome destroys it.

Coordinated Control

Shared regulatory sequences: genes w/ same enhancer elements switch on together (e.g., stress response).
Why regulate: save energy & materials; make proteins only when & where needed; respond to the environment.
Small RNAs: RNA interference (RNAi) is also used as a lab tool in research to silence specific genes on purpose.

⚠ Methylated DNA → genes OFF. Acetylated histones → genes ON. Don't swap them on the exam.

✓ Operon Q: ask "Is the repressor bound to the operator right now?" Bound → genes OFF; free → genes ON.

6.6 Gene Expression & Cell Specialization

Same DNA, Different Cells

All body cells carry the same genome (same DNA); they differ only in WHICH genes are expressed, and how much.
Differential gene expression → diff. proteins → diff. structure & function (neuron vs muscle vs liver).
Combinatorial control: each cell's unique mix of transcription factors decides which genes turn on.

Development

Cytoplasmic determinants: mRNA & proteins unevenly spread in the egg → early cells get diff. signals.
Induction: signals from nearby cells change their neighbors' gene expression (via cell communication, Unit 4).
Homeotic (Hox) genes: master transcription factors → body plan (head-to-tail order). Conserved across animals.
Apoptosis (programmed cell death) shapes tissues, e.g., it removes the webbing between fingers.

Cell	Gene ON	Product
RBC precursor	Hemoglobin	O_2 carrier
Pancreas β	Insulin	Hormone
Muscle	Myosin, actin	Contraction
Lens cell	Crystallin	Clear lens

Evidence

Cloning (Dolly the sheep): nucleus from a differentiated cell → whole organism → shows the genome stays intact.
Stem cells: undifferentiated; can become many cell types. Embryonic stem cells > adult stem cells in range.
Environment can shift gene expression (e.g., temp-dependent sex, Unit 5) via TFs & epigenetic changes.

⚠ Specialized cells don't lose genes. They keep the full genome and silence the ones they don't use.

✓ FRQ chain: diff. transcription factors → diff. genes expressed → diff. proteins → diff. cell structure & function.

6.7 Mutations

Point Mutations

Type	Change	Effect
Silent	Same AA	None
Missense	Diff. AA	Varies
Nonsense	→ stop	Short protein
Frameshift	Ins./del.	All AAs after Δ

Frameshift: insertion/deletion not a multiple of 3 → reading frame shifts → usually a nonfunctional protein.
Sickle cell: GAG → GTG → Glu → Val (missense) → Hb clumps → sickled RBCs. Heterozygotes resist malaria.

Causes & Effects

Causes: replication errors, mutagens (UV → thymine dimers, X-rays, chemicals), viruses, transposons.
Effects can be harmful, neutral, or beneficial; the same mutation's effect depends on the environment.
Only germline (gamete) mutations pass to offspring. Somatic → can cause cancer in that individual.
DNA repair: mismatch & excision repair fix most damage; failed repair → mutations build up (e.g., UV → skin cancer).

Other Sources of Variation

Gene duplication → extra copy is free to mutate & evolve a new function (e.g., the globin gene family).
Bacteria: **transformation** (take up DNA), **transduction** (virus carries DNA), **conjugation** (pilus transfer), plasmids → antibiotic resistance spreads between cells.
Viruses: high mutation rate (RNA viruses), recombination of genomes → new strains (e.g., flu, HIV) each year.
Mutation = raw material for natural selection (Unit 7).

⚠ Inserting 3 nts adds one AA but keeps the frame. Inserting 1 or 2 shifts everything after.

✓ Mutation Q: translate the sequence before AND after the change; then compare the AA sequences.

6.8 Biotechnology & Exam Strategy

Core Tools

Tool	Does
PCR	Copies DNA region
Gel electrophoresis	Sorts DNA by size
Restriction enzyme	Cuts at site
Plasmid/ligase	Inserts gene
DNA sequencing	Reads base order
CRISPR-Cas9	Edits target seq.

PCR: denature (~95 °C) → anneal primers (~55 °C) → extend w/ Taq polymerase (~72 °C). Doubles each cycle: 2^n.
Gel: DNA neg. (phosphates) → moves to + end. Small fragments travel farthest. Ladder = size reference.
Transformation: gene + plasmid (cut w/ same restriction enzyme → matching sticky ends) → ligase seals → bacteria take up plasmid → select w/ antibiotic.

Uses

Medicine: insulin from bacteria, gene therapy, diagnosis.
Forensics: DNA fingerprinting (STRs). **Agri.**: GMO crops.
Gel reading: same bands = same DNA. Each of a child's bands must match mom or dad (paternity test).
Bioinformatics: compare DNA or protein sequences across species → find genes, build phylogenies (Unit 7).

Exam Moves

Central dogma chain: DNA → (transcription) mRNA → (translation) protein → phenotype (trait).
Predict: where is the change (DNA, mRNA, protein)? Which step is hit? What changes downstream?
Links: Unit 1 nucleic acids, Unit 4 signaling → TFs, Unit 5 alleles & traits, Unit 7 mutation → evolution.

⚠ On a gel, the band farthest from the wells is the SMALLEST fragment, not the biggest.

✓ PCR math: copies = starting copies × 2^n (n = # of cycles). e.g., 1 copy after 3 cycles → 2^3 = 8 copies.