AP Biology Unit 4

Cell Communication & Cycle

One-page sprint review
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EXAM WEIGHT	MCQs	FRQ APPEARANCE	SPRINT TIME
10–15%	6–9	Very Frequent	~2 hours

Inside this pack

- **Quick Glance at every Unit topic with priority + format**
- **Topic-by-topic key traps, must-know rules, and exam frames**
- **Worked example questions on the highest-yield topics**

AP Biology Unit 4 — Cell Communication & Cell Cycle

Sprint Review • Exam in 3 Days

Sprint Review · Free at Sophriva

controlled cycle governs when they divide. The most exam-dense question type here is mutation tracing — identify what step is altered, then follow the consequence through the pathway.

Exam Weight	~MCQs	FRQ Appearance	Sprint Time
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Quick Glance — All Topics at a Glance

Topic	Priority	Exam Format	Key Trap / Must-Know
4.1 Cell Communication	★★★	MCQ, FRQ	Protein hormones NEVER enter the cell; steroid hormones cross freely → intracellular receptor
4.2 Signal Transduction Intro	★★★	MCQ, FRQ	Reception → Transduction → Response; amplification via phosphorylation cascade
4.3 Signal Pathways & cAMP	★★★	MCQ, FRQ	cAMP is made by adenylyl cyclase (NOT by receptor); GPCR → G protein → adenylyl cyclase → cAMP
4.4 Feedback Mechanisms	★★★	MCQ, FRQ	Positive feedback does NOT maintain homeostasis — it amplifies toward an endpoint
4.5 Cell Cycle & Mitosis	★★★	MCQ, Data	DNA replication = S phase, NOT mitosis; sister chromatids separate in anaphase
4.6 Cycle Regulation & Cancer	★★★	MCQ, FRQ	Oncogenes = dominant; tumor suppressors = recessive, need both copies lost

Best last-3-day focus: hormone receptor logic, GPCR/cAMP

4.1 Cell Communication

How cells talk: direct contact, local signaling, synapses, and endocrine hormones

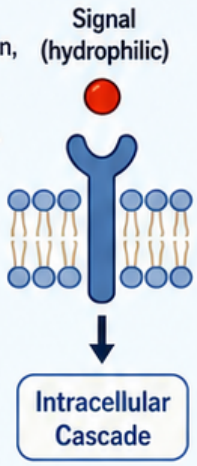
Four Modes of Cell-to-Cell Signaling

Mode	Distance	Mechanism	AP Examples
Direct Contact	Zero	Surface proteins bind adjacent cell; gap junctions move ions/small molecules	Immune T-cell recognition; cardiac muscle gap junctions; embryonic induction
Paracrine	Local (nearby)	Signal diffuses through extracellular fluid to nearby cells only	Growth factors; histamine; morphogens; neurotrophins
Synaptic	Synapse (~20 nm)	Neurotransmitter released into synaptic cleft → binds postsynaptic receptor	Acetylcholine; dopamine; serotonin
Endocrine	Long (bloodstream)	Hormone secreted into blood → travels to distant target cells with receptors	Insulin; testosterone; epinephrine

Hydrophilic Signals — CANNOT Cross Membrane

Cell Surface Receptors

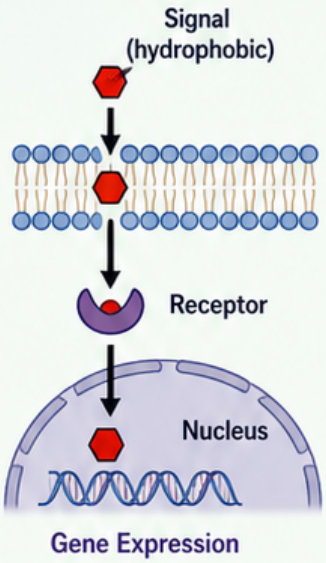
- Protein/peptide hormones: insulin, glucagon, growth hormone, oxytocin
- Neurotransmitters and most growth factors
- Signal binds surface receptor → conformational change → intracellular cascade begins
- Signal molecule never enters the cell
- Possible responses: enzyme activation, gene expression change, secretion, cell division



Hydrophobic Signals — CROSS Membrane Freely

Intracellular Receptors

- Streeid hormones: testosterone, estrogen, cortisol, aldosterone
- Thyroid hormones (T3, T4): lipid-soluble, intracellular receptor
- Diffuse directly through phospholipid bilayer
- Bind receptor in cytoplasm or nucleus
- Hormone–receptor complex acts as transcription factor → directly alters gene expression
- Longer-lasting gene-expression response vs. faster surface receptor responses

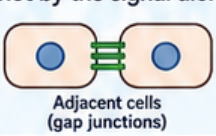


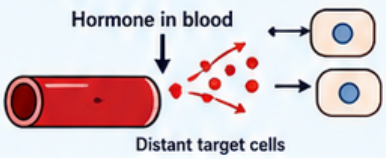
Exam Sniper

- Insulin is a protein hormone → binds a surface receptor, usually an RTK, and triggers a cascade; insulin does NOT enter the cell
- Testosterone is a steroid → crosses membrane → binds intracellular receptor → directly affects transcription
- Same signal can cause different responses in different cell types because receptor type and downstream pathway differ

Trap Alert

- Protein hormones NEVER cross the membrane
- Gap junctions are for adjacent-cell communication, not long-distance endocrine signaling
- Signal specificity is determined by the receptor, not by the signal alone





4.2 Signal Transduction Pathways

Reception → Transduction → Response

The Universal Three-Stage Framework

1 Reception

signal molecule binds receptor.

2 Transduction

intracellular relay / phosphorylation cascade / second messengers carry and amplify signal.

3 Response

gene expression change, enzyme activation, secretion, apoptosis, or cell division.

A pathway converts an outside signal into a specific cellular response.

Three Major Receptor Types

Receptor Type	How It Works	2nd Messenger?	Key Examples
G Protein-Coupled Receptor (GPCR)	Ligand binds → activates G protein (GDP→GTP) → G protein activates or inhibits adenylyl cyclase → ATP becomes cAMP → PKA → phosphorylation cascade	Yes: cAMP	Epinephrine, glucagon, olfactory receptors
Receptor Tyrosine Kinase (RTK)	Ligand binding → receptor dimerizes → autophosphorylation of tyrosines → docking sites recruit relay proteins → multiple pathways activated	No; direct phosphorylation	Insulin receptor, EGF, PDGF
Ligand-Gated Ion Channel	Ligand binds → channel opens → ions move down gradient → rapid electrical change	No; ion flux	Acetylcholine receptor, GABA receptor

Signal Amplification

- Each activated step can activate many molecules of the next step
- One receptor can activate many G proteins
- One hormone molecule can trigger a huge response
- Kinase cascades amplify at every level
- Hormones work at very low concentrations because of amplification

What Cellular Responsee Can Be Triggered?

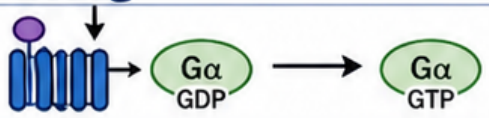
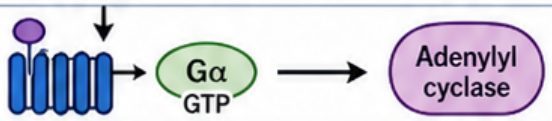
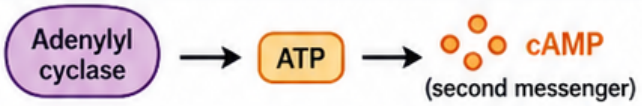
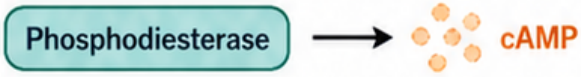
- Gene expression
- Enzyme activation or inhibition
- Secretion / exocytosis
- Cell division
- Apoptosis

Key idea: receptor type + downstream pathway determine the response.

4.3 GPCR, cAMP & Signal Amplification

The most-tested signaling cascade

The cAMP Cascade (Step-by-Step)

1		Ligand (example: epinephrine) binds GPCR
2		G protein activated (GDP replaced by GTP)
3		Active G protein activates adenylyl cyclase
4		Adenylyl cyclase converts ATP → cAMP (second messenger)
5		cAMP activates Protein Kinase A (PKA)
6		PKA phosphorylates target proteins → cellular response
STOP		Termination: phosphodiesterase degrades cAMP → signal off

cAMP is made by adenylyl cyclase, NOT by the receptor.

Why One Signal = Big Response

- One activated receptor can activate many G proteins
- Many cAMP molecules are made from ATP
- Many PKA molecules are activated
- Thousands of downstream proteins may be phosphorylated
- This is **signal amplification**

Common Responses

- Glycogen breakdown in liver cells
- Gene expression changes
- Enzyme activation / inhibition
- Secretion
- Growth or division

Exam Sniper

Trace the pathway:

Epinephrine in liver cells: GPCR binding → G protein → adenylyl cyclase → cAMP → PKA → glycogen phosphorylase → glycogen breakdown → glucose released.

If phosphodiesterase is inhibited, cAMP levels rise and the signal lasts longer.

Mutation Scenario MCQ

Q: Adenylyl cyclase is constitutively active. What happens?

A: cAMP is continuously produced, PKA stays active, and downstream responses continue even without ligand → potentially contributing to cancer.

Ligand = first messenger; cAMP = second messenger. RTKs do **NOT** use cAMP.

4.4 Feedback Mechanisms

Negative feedback stabilizes; positive feedback amplifies

Negative vs. Positive Feedback

Feature	Negative Feedback	Positive Feedback
Basic effect	Output opposes original stimulus	Output amplifies original stimulus
Relationship to set point	Returns variable toward set point	Moves system away from set point
Homeostasis?	Yes, maintains homeostasis	No, does not maintain homeostasis
Typical endpoint	Ongoing regulation	Definitive self-limiting endpoint
Classic examples	Blood glucose, body temperature, thyroid axis, enzyme feedback inhibition	Childbirth, blood clotting, action potential, LH surge / ovulation

Negative Feedback Examples

- Blood glucose rises → insulin released → glucose uptake rises → blood glucose returns to normal
- Body temperature rises → sweating / vasodilation → heat loss → temperature drops
- TRH → TSH → T3/T4, and T3/T4 inhibit hypothalamus and pituitary
- Enzyme feedback inhibition: end product inhibits an early enzyme

Stimulus (change) → Response → Opposes change

Stabilizes • Returns to set point

Positive Feedback Examples

- Childbirth: oxytocin → contractions → more oxytocin → more contractions → birth
- Blood clotting: activated clotting factors activate more factors → clot forms
- Action potential: Na^+ channels open → depolarization → more Na^+ channels open
- LH surge: estrogen promotes LH release → ovulation

Stimulus (change) → Response → Amplifies change

Amplifies • Moves away from set point

Exam Sniper

- Stable blood glucose = negative feedback
- Childbirth with oxytocin = positive feedback
- High cortisol suppresses CRH and ACTH by negative feedback
- Positive feedback always ends when the endpoint is reached

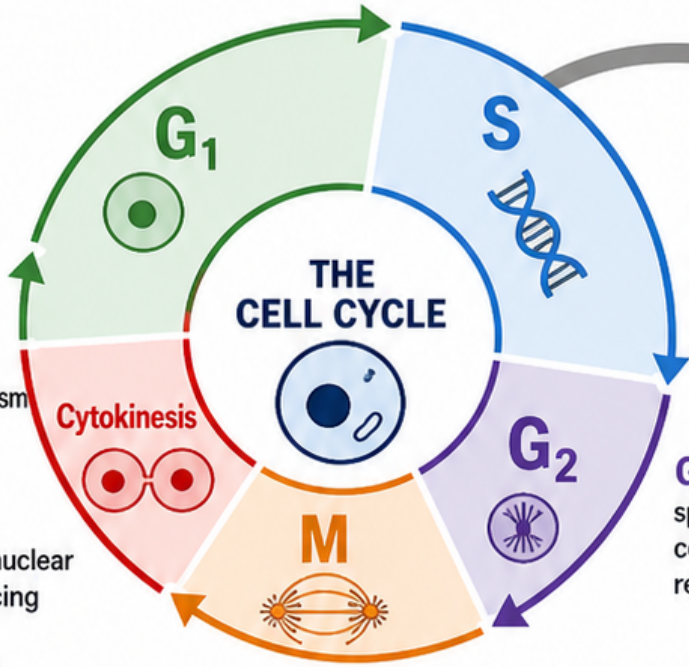
Trap Alert

- Positive feedback does NOT maintain homeostasis
- Only negative feedback stabilizes a set point
- Enzyme feedback inhibition is negative feedback, not positive feedback

4.5 The Cell Cycle

Interphase, mitosis, cytokinesis, and G_0

G_1: cell growth, proteins and organelles made, receives signals to divide or not; **restriction point** here.



G_0: non-dividing state; neurons often permanent, liver can re-enter cycle; not cell death.

S: DNA replication; chromosome copied into 2 sister chromatids; DNA content doubles from **2N** to **4N**.

Cytokinesis: cytoplasm divides to form 2 daughter cells.

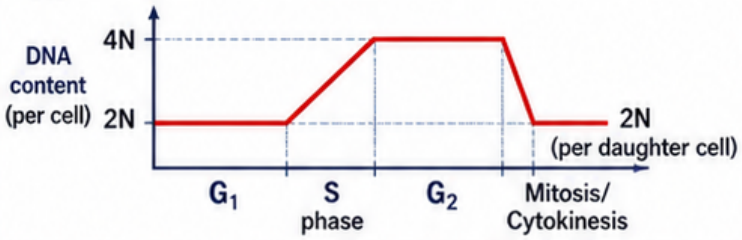
G_2: more growth, spindle proteins made, cell checks that DNA replication is complete.

M: mitosis = nuclear division producing 2 genetically identical nuclei.

Cell Cycle Phases — Know What Happens

Phase	Key Events	AP Exam Identifier
G_1	Cell growth, proteins and organelles made; receives signals to divide or not; restriction point here.	Largest phase; growth & decision point
S	DNA replication; chromosome copied into 2 sister chromatids; DNA content doubles from 2N to 4N.	DNA synthesis; 2N → 4N
G_2	More growth, spindle proteins made; cell checks that DNA replication is complete.	Preparation & quality control
Mitosis (M)	Nuclear division producing 2 genetically identical nuclei.	Four phases: PMAT
Cytokinesis	Cytoplasm divides to form 2 daughter cells.	Occurs after mitosis
G_0	Non-dividing state; neurons often permanent, liver can re-enter cycle; not cell death.	Quiescent but active; can re-enter

DNA Content Must-Know



DNA replication occurs in S phase, NOT during mitosis.

Exam Sniper

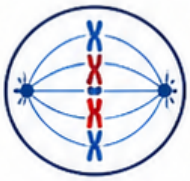
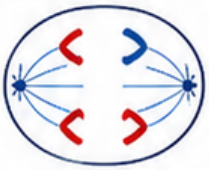
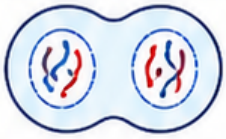
- G_1 usually has the largest percentage of cells.
- A drug that blocks spindle formation arrests cells before anaphase, usually metaphase.
- G_0 is metabolically active, not dead.
- By prophase, DNA is already fully replicated.

Don't confuse G_0 with death, and don't say DNA replicates during mitosis.

4.5 Mitosis — PMAT Master Page

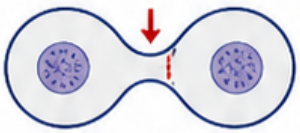
Know what chromosomes look like in each stage

Mitosis Phases — PMAT

Phase	Key Events	AP Exam Identifier
 **Prophase**	Chromatin condenses into visible chromosomes; nuclear envelope breaks down; spindle forms	Chromosomes visible but not aligned
 **Metaphase**	Chromosomes align at metaphase plate; spindle attaches to kinetochores on both sister chromatids	Chromosomes lined up in the middle
 **Anaphase**	Sister chromatids separate; centromeres split; chromatids pulled to opposite poles	V-shaped chromosomes moving apart; cell elongates
 **Telophase**	Nuclear envelopes reform; chromosomes decondense; spindle breaks down	Two nuclei forming

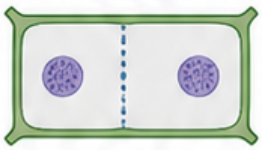
Cytokinesis

Animals



cleavage furrow; actin ring pinches cell

Plants



cell plate forms from vesicles

Sister chromatids separate in mitosis anaphase and meiosis II anaphase.

Homologous chromosomes separate in meiosis I.

Flow Cytometry / DNA Content MCQ

- 2N = G1 or G0
- Between 2N and 4N = S phase
- 4N = G2 + mitosis before cytokinesis
- Dropping from 4N to 2N = late mitosis / cytokinesis

Exam Sniper

- Metaphase is easiest for counting chromosomes
- Taxol / paclitaxel blocks spindle function and arrests cells before anaphase
- Plants do not need centrioles to form a spindle
- Sister chromatids separate in anaphase, not in metaphase

4.6 Cell Cycle Regulation

Cyclins, CDKs, checkpoints, and p53

MOLECULAR DRIVERS

Cyclins & CDKs

- **CDKs** are enzymes that drive cell-cycle transitions by phosphorylating target proteins
- **Cyclins** are regulatory proteins whose concentration rises and falls
- **Cyclin + CDK** = active complex that pushes the cycle forward
- Cyclin degradation inactivates CDKs and helps enforce checkpoints
- Different cyclin-CDK pairs control different transitions such as G1→S and G2→M

QUALITY CONTROL

The Three Checkpoints

G1	G1 checkpoint	size, DNA integrity, growth signals; pass → S phase, fail → G0 or apoptosis
G2	G2 checkpoint	DNA replicated completely and correctly?
	M checkpoint / spindle checkpoint	are ALL kinetochores attached before anaphase?

GUARDIAN OF THE GENOME

p53 Tumor Suppressor

- **p53** is a transcription factor activated by DNA damage
- Halts the cycle at G1 to allow DNA repair
- If damage cannot be repaired, p53 triggers apoptosis
- p53 is mutated in about 50% of human cancers
- It is called the guardian of the genome
- p53 is a tumor suppressor, so loss of function promotes cancer

EXAM SNIPER

- A mutation in checkpoint proteins can allow damaged DNA to pass on
- If spindle checkpoint fails, daughter cells may become aneuploid
- One working copy of a tumor suppressor can still provide partial control; both copies are often needed to fully lose function

TRAP ALERT

- Checkpoints do not "create" DNA damage; they detect and respond to it
- p53 is not an oncogene; it is a tumor suppressor
- The spindle checkpoint prevents chromosome mis-segregation, not DNA replication errors

4.6 Cancer, Oncogenes & Cell Death

How cancer develops when accelerators stick and brakes fail

Oncogenes vs. Tumor Suppressors — Most Tested Cancer Concept

Oncogenes		Tumor Suppressors	
Derived from:	proto-oncogenes	**Normal job:**	inhibit division or promote apoptosis
Mutation type:	Gain-of-function mutation	**Mutation type:**	Loss-of-function mutation
Genetic effect:	Dominant: one mutant copy is enough	**Genetic effect:**	Recessive: both copies must be lost (two-hit hypothesis)
Effect:	Growth signal always on	**Effect:**	Cell cannot stop dividing
Examples:	mutant Ras, mutant RTK / EGFR, amplified growth factor receptors	**Examples:**	p53, Rb
Analogy:	stuck accelerator	**Analogy:**	cut brake lines
Common mechanisms:	point mutation, gene amplification, chromosomal translocation	**Hereditary cancers often start with one inherited mutant copy plus one somatic mutation**	

Programmed Cell Death

Apoptosis vs. Necrosis

- **Apoptosis:** ordered, energy-requiring, cell shrinks, fragments are phagocytosed, no inflammation
- Roles: development, immune selection, cancer surveillance, removal of damaged cells

- **Necrosis:** accidental injury-related cell death, membrane ruptures, contents spill, inflammation occurs

Key distinction: apoptosis does NOT cause inflammation.

Cancer = Loss of Both Controls

How Cancer Develops

- Cancer usually requires multiple mutations over time
- Activation of oncogene(s) + loss of tumor suppressor(s)
- Cells divide uncontrollably, evade apoptosis, and ignore checkpoints
- **Metastasis:** invasion, bloodstream spread, colonization of distant organs
- Mutations may be inherited, spontaneous, or caused by carcinogens such as UV, chemicals, or viruses

FRQ-Style MCQ Cancer Genetics

Question:
EGFR is continuously active without ligand, and both copies of *Rb* are inactivated. How does each mutation contribute?

Answer summary:
EGFR is a dominant oncogene gain-of-function mutation that continuously stimulates growth signaling; *Rb* is a recessive tumor suppressor loss-of-function mutation, and losing both copies removes the G1→S brake. Together they remove both accelerators and brakes, promoting uncontrolled cell division.

EXAM SNIPER: A permanently active Ras or RTK pathway is a classic oncogene scenario.

Final Review — Exam Traps + Checklist

Use this page the night before and the morning of the exam

1. Unit 4 High-Frequency Exam Traps

- Protein hormones never enter the cell; only lipid-soluble signals such as steroids and thyroid hormones cross the membrane
- cAMP is produced by adenylyl cyclase, not by the receptor or G protein directly
- Positive feedback does NOT maintain homeostasis; only negative feedback stabilizes a set point
- DNA replication occurs in S phase, not during mitosis
- Sister chromatids separate in mitosis anaphase; homologs separate in meiosis I
- Oncogenes are dominant gain-of-function; tumor suppressors are recessive loss-of-function
- Apoptosis does not cause inflammation; necrosis does
- Signal specificity depends on receptor type and downstream pathway

2. Pre-Exam 10-Minute Checklist

A. Cell Communication (4.1)

- [ ] Hydrophilic signal → surface receptor
- [ ] Hydrophobic signal → intracellular receptor
- [ ] Can distinguish direct contact, paracrine, synaptic, endocrine
- [ ] Receptor type determines response

B. Signal Transduction (4.2–4.3)

- [ ] Reception → Transduction → Response
- [ ] GPCR → G protein → adenylyl cyclase → cAMP → PKA
- [ ] RTK = dimerization + autophosphorylation; no cAMP
- [ ] Signal amplification and phosphodiesterase termination

C. Feedback + Cell Cycle (4.4–4.5)

- [ ] Negative vs positive feedback examples
- [ ] G1, S, G2, M, cytokinesis, G0
- [ ] DNA replication = S phase
- [ ] PMAT + cytokinesis in animals vs plants

D. Regulation + Cancer (4.6)

- [ ] Cyclins activate CDKs
- [ ] G1, G2, and M checkpoints
- [ ] p53 = DNA damage response + apoptosis
- [ ] Oncogene vs tumor suppressor; apoptosis vs necrosis

3. Final Sprint Strategy for Unit 4

Top exam format: mutation-tracing through a pathway.

Must-master pathways: insulin receptor logic, GPCR/cAMP cascade, blood glucose negative feedback loop, cell cycle with checkpoints.

Cancer FRQ template: gene type → normal job → mutation effect → uncontrolled division.

Connections forward: signaling, meiosis comparison, immunity, ecology feedback loops.

If you can explain every checked item out loud, you're ready.

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