



AP BIOLOGY

UNIT 4: CELL COMMUNICATION AND CELL CYCLE



COGNITOEDU.LINK/AP-BIO-U4

Scan for Unit 4 notes

20 AP-style MCQs across topics 4.1 to 4.6. Practice, review, revisit.

WHAT'S INSIDE

- ✓ 20 MCQs, four options each, in the style of the real AP exam
- ✓ Every question tagged to a Unit 4 topic so you know what to revise
- ✓ Full answer key with a short explanation for every question
- ✓ Mix of standalone and stimulus-style stems, matching real AP question formats

HOW TO USE IT

- 1. Untimed pass.** Work through slowly, aiming for around 90 seconds per question. Read every option before you commit.
- 2. Timed pass.** Redo the pack in 30 minutes. That matches AP exam pace (1.5 minutes per MCQ) and builds stamina.
- 3. Review.** Check the answer key and mark every miss. Revise those topics on Cognito, then retry after a week.

TOPICS COVERED

- **4.1** Cell Communication
- **4.2** Introduction to Signal Transduction
- **4.3** Signal Transduction Pathways
- **4.4** Feedback
- **4.5** Cell Cycle
- **4.6** Regulation of Cell Cycle

STRUGGLING WITH A TOPIC?



Any topic tricky? Scan the QR to jump straight to the Unit 4 notes on Cognito. For the full AP Biology course (all 8 units, lessons, quizzes, flashcards and past-paper FRQs), head to **cognitoedu.link/ap-bio-notes**. Rework the questions you got wrong a week later to lock the concepts in.

AP EXAM CONTEXT

10-15% 60

UNIT WEIGHT (MCQ SECTION)

MCQS ON THE REAL EXAM

1.5m

AVERAGE TIME PER MCQ

AP BIOLOGY

Unit 4: Cell Communication and Cell Cycle

SECTION I · MULTIPLE CHOICE

Time—30 minutes

20 Questions

Directions: Each of the questions or incomplete statements below is followed by four suggested answers or completions. Select the one that is best in each case and mark it clearly on your answer sheet or circle your choice below.

- In mammalian long-distance signaling, endocrine hormones travel through the bloodstream to reach their target cells. Which feature of a target cell most directly determines whether it responds to a given hormone?
 - The overall size and shape of the target cell as it sits in its resident tissue.
 - The physical distance between the target cell and the endocrine gland that produced the hormone.
 - The rate at which the target cell divides.
 - Whether the cell has receptors that specifically bind that hormone.
- A neuron releases a neurotransmitter into a narrow synapse where it acts only on the next cell. Which type of cell signaling does this best represent?
 - Endocrine signaling to distant target cells.
 - Local signaling across a very short distance.
 - Direct contact between cell surfaces without any signaling molecule.
 - Autocrine signaling in which the neuron signals to itself.

Questions 3, 4 refer to the following information.

Cultured cells were treated with increasing concentrations of a peptide hormone. The response magnitude per cell was measured and expressed as a percentage of the maximum response to a reference agonist.

Table 1. Cellular response magnitude vs. hormone concentration

Hormone (nM)	Response magnitude (% of maximum)
0	2
0.1	8
1	36
10	78
100	95
1,000	96

- Based on Table 1, what happens to the cellular response as hormone concentration rises?
 - The response rises linearly with hormone concentration without limit.
 - The response drops sharply above 10 nM.
 - The response rises steeply, then approaches a plateau near 96%.
 - The response is unrelated to hormone concentration.
- Which claim is best supported by the plateau seen in Table 1?
 - Each cell has an essentially unlimited number of receptors for this hormone.
 - The hormone can only bind cells that lack receptors.
 - At high concentrations the receptors become saturated, so extra hormone adds little response.
 - The response requires no receptor at all above 100 nM.

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Questions 5, 6, 7 refer to the following information.

Four cell lines (W, X, Y, Z) were exposed to the same peptide hormone. Each line was tested for the presence of the hormone's receptor on the cell surface, the level of second messenger produced after treatment, and any downstream response.

Table 2. Signal transduction components in four cell lines

Cell line	Receptor present	Hormone binding to cells	Second messenger produced	Downstream response
W	Yes	Yes	Normal	Normal
X	No	No	None	None
Y	Yes	Yes	None	None
Z	Yes	Yes	Normal	None

- Which cell line in Table 2 is most consistent with a defect in the receptor itself, rather than a downstream component?
 - Cell line W
 - Cell line X
 - Cell line Y
 - Cell line Z
- Which conclusion about cell line Y is best supported by Table 2?
 - The receptor cannot bind the hormone at all, so no downstream signaling begins.
 - Its downstream transcription factor is mutated.
 - It contains no proteins involved in signal transduction.
 - The receptor binds hormone, but the step that produces the second messenger is disrupted.
- Cell line Z has a normal receptor and normal second messenger production, but no downstream response. Which additional experiment would best identify where the pathway is broken in this cell line?
 - Measure the amount of receptor on the cell surface using a fluorescently labeled antibody probe.
 - Add more hormone and measure the amount of second messenger produced.
 - Repeat the experiment using a different unrelated hormone.
 - Measure the activity of the protein kinase activated by the second messenger in Z cells.
- A drug binds to a G protein-coupled receptor and holds it in the active conformation even when no ligand is present. Which cellular effect would you most likely observe?
 - The receptor loses the ability to respond to any of the ligands that normally activate it.
 - Downstream signaling is turned off, because the receptor is occupied.
 - The pathway is activated constantly, even without the natural ligand.
 - The cell stops producing any receptors on its surface.

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Questions 9, 10 refer to the following information.

Insulin lowers blood glucose after a meal. Blood glucose and blood insulin were measured in a healthy participant every 30 minutes for 3 hours after eating a mixed meal at time 0.

Table 3. Blood glucose and insulin after a mixed meal

Time (min)	Blood glucose (mg/dL)	Blood insulin (μU/mL)
0	88	10
30	140	45
60	125	78
90	105	62
120	92	35
180	86	12

9. Which pattern in Table 3 best illustrates negative feedback?
- (A) Rising glucose triggers insulin, which lowers glucose back toward baseline.
 - (B) Rising blood glucose triggers a further rise in blood glucose that keeps accelerating over the 3-hour period tested.
 - (C) Insulin and glucose rise and fall together at exactly the same time.
 - (D) Blood glucose remains constant throughout the 3-hour period.
10. A patient with untreated type 1 diabetes cannot produce insulin. Based on Table 3, which prediction about their response to the same mixed meal is best supported?
- (A) Blood glucose would drop below the fasting baseline within the first 30 min after the meal is consumed.
 - (B) Blood glucose rises and stays elevated for hours, because the feedback loop is broken.
 - (C) Blood glucose would peak at the same value and return to baseline at the same rate.
 - (D) Blood insulin would rise even higher than in the healthy participant.
11. Which of the following is an example of positive feedback?
- (A) Rising body temperature triggers sweating, which cools the body back to its normal set point.
 - (B) Rising CO₂ in the blood increases breathing rate, lowering CO₂.
 - (C) Oxytocin causes uterine contractions in labor, which trigger more oxytocin release.
 - (D) Falling blood calcium triggers parathyroid hormone release that raises blood calcium back to its normal set point.

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Questions 12, 13, 14 refer to the following information.

A signaling pathway begins with a ligand binding a receptor tyrosine kinase (RTK), then activates a series of intracellular kinases, and finally activates a transcription factor. Different mutations were tested in a cell line for their effect on transcription of the target gene, with or without ligand added to the medium.

Table 4. Effect of pathway mutations on target gene transcription

Mutation	Description	Ligand added	Transcription of target gene (% of wild type)
None (wild type)	Reference	Yes	100
None (wild type)	Baseline	No	8
M1	RTK cannot bind ligand	Yes	9
M2	RTK is constitutively active	No	104
M3	Downstream kinase is constitutively active	No	108
M4	Transcription factor cannot bind DNA	Yes	6

12. Which mutation in Table 4 mimics the ligand-bound state of the RTK receptor itself, even though no ligand was added?
 - (A) M1
 - (B) M2
 - (C) M3
 - (D) M4
13. In a cell line, the target gene is transcribed at wild-type levels without ligand. If a drug that inhibits the RTK's kinase activity is added and transcription remains high, which mutation is the most likely cause?
 - (A) M1
 - (B) M2
 - (C) M3
 - (D) M4
14. Which conclusion about the pathway is best supported by comparing M1 and M4 in Table 4?
 - (A) The receptor and the transcription factor act on the same step of the pathway.
 - (B) The transcription factor works only when the receptor is inactive.
 - (C) Ligand binding is not required for pathway activation.
 - (D) Different mutations at opposite ends of the pathway can produce a similar loss-of-function phenotype.

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Questions 15, 16 refer to the following information.

Flow cytometry measures DNA content per cell. A synchronous population of eukaryotic cells was sampled at three points in the cell cycle, and the DNA content per cell was measured relative to a G1 cell (set at 1).

Table 5. DNA content of cells at three cell cycle stages

Sample	Relative DNA content per cell	Number of centrosomes
1	1.0	1
2	2.0	2
3	1.0 (per daughter cell)	1 (per daughter cell)

15. Which sample in Table 5 most likely represents a cell in G2 (after DNA replication but before division)?
 (A) Sample 1
 (B) Sample 2
 (C) Sample 3
 (D) None of the samples
16. Which claim is best supported by comparing samples 2 and 3 in Table 5?
 (A) Mitosis then cytokinesis distributes the replicated DNA and centrosomes equally between the two daughters.
 (B) Sample 3 has undergone another round of DNA replication after sample 2, doubling its DNA content once more.
 (C) The two daughter cells contain twice as much DNA as the original G1 cell.
 (D) Mitosis and cytokinesis leave the DNA content per cell unchanged from Sample 2 to Sample 3.

Questions 17, 18 refer to the following information.

Human cells were treated with different agents that affect the cell cycle. The percentage of cells in each phase was measured after 24 hours by flow cytometry combining DNA content with a mitotic marker (phospho-histone H3, used to separate G2 from M).

Table 6. Cell cycle distribution under different treatments (% of cells)

Treatment	% in G1	% in S	% in G2	% in M
Control (no treatment)	55	25	15	5
UV radiation (DNA damage)	78	12	7	3
Nocodazole (blocks spindle formation)	10	8	6	76
Hydroxyurea (arrests cells at the G1/S boundary)	62	34	2	2

17. Which conclusion about the G1 checkpoint is best supported by Table 6?
 (A) The G1 checkpoint responds to DNA damage by halting cells before they enter S phase.
 (B) The G1 checkpoint responds only to problems with spindle microtubule attachment to kinetochores during mitosis.
 (C) UV radiation triggers a specific arrest at the M checkpoint.
 (D) The G1 checkpoint cannot detect any type of cellular stress.

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18. Which claim about the nocodazole treatment in Table 6 is best supported by the data?
- (A) Nocodazole causes cells to arrest before entering S phase.
 - (B) Nocodazole causes M-phase arrest via the spindle checkpoint detecting unattached kinetochores.
 - (C) Nocodazole prevents any cellular arrest and speeds the cell cycle.
 - (D) Nocodazole triggers arrest at the G2 checkpoint by inducing double-strand breaks in the DNA of the cell.

Questions 19, 20 refer to the following information.

Cyclin B and CDK1 (cyclin-dependent kinase 1) drive the G2/M transition. Their levels in a synchronized cell population were tracked across one cell cycle.

Table 7. Relative protein levels across the cell cycle

Phase	Cyclin B	CDK1	Active cyclin B-CDK1 complex
G1	5	100	3
S	20	100	12
G2 late	95	100	30
M early	90	100	88
M late	8	100	5

19. Which claim is best supported by the pattern of active cyclin B-CDK1 complex in Table 7?
- (A) CDK1 is constant, so activity depends on cyclin B, which must accumulate before CDK1 is fully activated at G2/M.
 - (B) CDK1 activity depends only on the level of CDK1 protein present in the cell at each phase, with cyclins playing no role.
 - (C) Cyclin B levels stay constant across the cell cycle.
 - (D) Active cyclin B-CDK1 peaks in G1.
20. Cyclin B is degraded by a protein complex activated in late mitosis. Which claim is best supported by comparing M early and M late in Table 7?
- (A) Cyclin B degradation lowers the active CDK1 complex, letting cells exit M phase into G1.
 - (B) Cyclin B degradation in late mitosis raises CDK1 protein levels sharply above their baseline value.
 - (C) Cyclin B is synthesized rapidly in late mitosis to drive the next cell cycle.
 - (D) CDK1 is destroyed at the end of mitosis, halting the cell cycle.

Q1. Answer: D

Topic 4.1 · Cell Communication

Even though every cell in the body is bathed in blood containing a circulating hormone, only cells that display a matching receptor can bind the hormone and respond. Cell size (A), distance (B), and division rate (C) do not select responsive cells; the presence of the correct receptor does.

Q2. Answer: B

Topic 4.1 · Cell Communication

Synaptic signaling is a form of local (paracrine-like) signaling; the neurotransmitter travels only across the narrow synaptic cleft. A describes hormonal (long-distance) signaling. C ignores that a chemical messenger is used. D describes autocrine signaling, which is not what happens between two different neurons.

Q3. Answer: C

Topic 4.2 · Intro to Signal Transduction

Response rises quickly across the 0.1 to 100 nM range, then levels off between 100 and 1,000 nM at about 95 to 96%. The plateau indicates receptor saturation: nearly all receptors per cell are bound at high concentration. A ignores saturation, B contradicts the plateau, and D is contradicted by the rise from 36% to 78% when concentration increases tenfold.

Q4. Answer: C

Topic 4.2 · Intro to Signal Transduction

The response reaches a maximum near 95 to 96%, so adding more hormone above 100 nM does not raise the response magnitude. This is the signature of receptor saturation: once every available receptor is bound, extra hormone adds little response. A contradicts the plateau (unlimited receptors would give a linear rise). B contradicts basic receptor biology. D invents receptor-free signaling that is not shown.

Q5. Answer: B

Topic 4.2 · Intro to Signal Transduction

Cell line X lacks the receptor and produces no second messenger and no response. This matches a receptor-level defect. W is normal. Y has the receptor but makes no second messenger, so the defect is between receptor and second messenger. Z has the receptor and normal second messenger but no downstream response, so the defect is further down the pathway.

Q6. Answer: D

Topic 4.3 · Signal Transduction Pathways

Y has the receptor AND shows hormone binding to cells but produces no second messenger and no response. That places the defect between hormone binding and second messenger production, for example in the G protein or the enzyme that makes cAMP. A is contradicted by the 'Hormone binding: Yes' column for Y. B would fit Z better (normal messenger, no response). C is refuted by the receptor being intact.

Q7. Answer: D

Topic 4.3 · Signal Transduction Pathways

The second messenger is being made in Z, but the downstream response fails, so the defect must lie between the second messenger and the final response. Assaying a kinase directly downstream of the second messenger in Z isolates whether that step is intact. A tests something already known to be normal (receptor). B tests messenger production, already normal. C tests an unrelated pathway.

Q8. Answer: C

Topic 4.3 · Signal Transduction Pathways

Locking the receptor in an active shape drives downstream signaling regardless of ligand binding. This is exactly the pattern seen with many pathogenic mutations and some drugs; the pathway is inappropriately switched on. A confuses activation with blocking. B reverses the effect. D describes receptor downregulation, which is a possible secondary response but not the direct result.

Q9. Answer: A

Topic 4.4 · Feedback

After the meal, glucose spikes to 140 mg/dL, which triggers insulin release (10 to 78 μ U/mL). Insulin drives glucose uptake, and by 120 to 180 min glucose has returned close to baseline. That is textbook negative feedback: the response counteracts the disturbance. B describes positive feedback. C contradicts the offset seen in the timing. D is directly refuted by the numbers.

Q10. Answer: B

Topic 4.4 · Feedback

Without insulin, the signal that lowers blood glucose is absent. Glucose still rises after the meal, but there is no negative feedback response, so glucose stays high (this is the clinical hyperglycemia of untreated type 1 diabetes). A reverses the direction. C ignores the missing regulator. D contradicts the assaying of insulin production.

Q11. Answer: C

Topic 4.4 · Feedback

In positive feedback, the response amplifies the initial change. Oxytocin driving contractions that release more oxytocin is a canonical example, and it accelerates until birth. A, B, and D all counteract the initial change and return the system to a set point; those are all negative feedback loops.

Q12. Answer: B

Topic 4.3 · Signal Transduction Pathways

M2 is a constitutively active RTK: even without ligand added, it drives transcription at 104% of wild-type, meaning the receptor behaves as if the ligand is bound. M3 also drives transcription (108%) without ligand, but it does so downstream of the receptor, so it does not mimic the receptor's ligand-bound state. M1 blocks ligand binding, and M4 blocks the final step, so neither mimics receptor activation.

Q13. Answer: C

Topic 4.3 · Signal Transduction Pathways

Blocking the RTK's kinase activity would silence transcription driven by M2 (which acts at the receptor), but not transcription driven by M3, which acts on a downstream kinase and bypasses the receptor. So if RTK inhibition fails to lower transcription, M3 is the best candidate. M1 and M4 both suppress transcription rather than raising it, so they are inconsistent with the high baseline.

Q14. Answer: D

Topic 4.3 · Signal Transduction Pathways

M1 (top of pathway, no ligand binding) and M4 (bottom of pathway, no DNA binding) both give transcription of about 6 to 9% of wild type. Breaking a linear pathway at either end knocks out the response. A is wrong because the two mutations affect different steps. B ignores that a functional receptor is normally required. C is contradicted by the wild-type-no-ligand baseline.

Q15. Answer: B

Topic 4.5 · Cell Cycle

During S phase, DNA replicates, so a G2 cell has 2× the G1 amount of DNA (relative content 2.0) and two centrosomes ready to organize the future spindle. Sample 2 matches. Sample 1 (1.0, 1 centrosome) is a G1 cell. Sample 3 (1.0 per daughter) is after cytokinesis, showing that division has already restored the G1 DNA amount in each daughter.

Q16. Answer: A

Topic 4.5 · Cell Cycle

Sample 2 has 2.0× DNA and 2 centrosomes in one cell. Sample 3 has 1.0× DNA and 1 centrosome per daughter cell. Mitosis separates the sister chromatids into two nuclei, and cytokinesis then divides the cytoplasm, so the replicated DNA and the two centrosomes end up equally distributed between the daughter cells. B ignores that DNA content per daughter fell, not rose. C is wrong (each daughter is at 1.0×). D contradicts the halving of DNA per cell.

Q17. Answer: A

Topic 4.6 · Regulation of Cell Cycle

UV radiation causes DNA damage, and Table 6 shows a large shift into G1 (55% in the control to 78% under UV) with a drop in later phases. This matches the role of the G1 checkpoint: prevent entry into S phase (DNA replication) until damage is repaired. B describes the M-phase checkpoint. C is contradicted by the very low M count in the UV row (3%). D contradicts the specific G1 accumulation.

Q18. Answer: B

Topic 4.6 · Regulation of Cell Cycle

Nocodazole blocks spindle formation, so chromosomes cannot attach properly to spindle microtubules. The spindle assembly checkpoint detects unattached kinetochores and halts the cell cycle before anaphase, producing the 76% M accumulation seen in the table. A misidentifies the phase (G1 or S). C contradicts the high M number. D invents a DNA-damage mechanism that nocodazole does not use.

Q19. Answer: A

Topic 4.6 · Regulation of Cell Cycle

CDK1 is at 100 units in every phase, so it is not the level of CDK1 that limits activity. Cyclin B rises from 5 to 95 units, and while cyclin availability sets the ceiling, the active complex remains low in late G2 (30) even after cyclin B has peaked (95). A further activation step at the G2/M transition (inhibitory phosphates removed by Cdc25) then jumps the active complex from 30 to 88 in early M. B ignores that CDK1 is constant. C contradicts the data. D reverses the peak (M early, not G1).

Q20. Answer: A

Topic 4.6 · Regulation of Cell Cycle

Cyclin B collapses from 90 to 8 between M early and M late, and the active complex drops from 88 to 5. Losing cyclin B inactivates CDK1, which lets cells exit mitosis and enter G1. B is contradicted (CDK1 is constant at 100). C is contradicted by the drop in cyclin B. D is contradicted by the constant CDK1 level.