AP Bio Unit Reviews

Cell Communication & Cell Cycle

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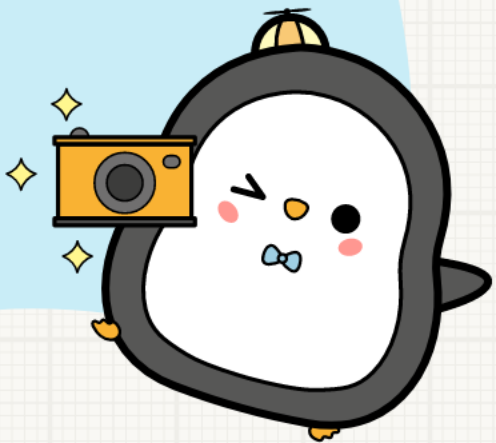


Today's Plan

Cellular Communication

Cell Cycle

Practice Questions

Unit 4 Q&A

Cellular Communication

Reception

Ligand (signaling molecule) binds to receptor

Causes confirmational shape change

Ex: G protein coupled receptor

Steroid Hormone

Release: Simple Diffusion

Receptor: Intracellular

Example: Testosterone, Estrogen

Protein Hormone

Release: Exocytosis

Receptor: Extracellular

Example: Insulin

Transduction

Signaling cascades relay signals from receptors to cell targets, often amplifying the incoming signals

Phosphorylation Cascade

Protein Kinase

Phosphorylate relay molecules

Secondary Messengers

Ca^{2+}

cAMP

Response

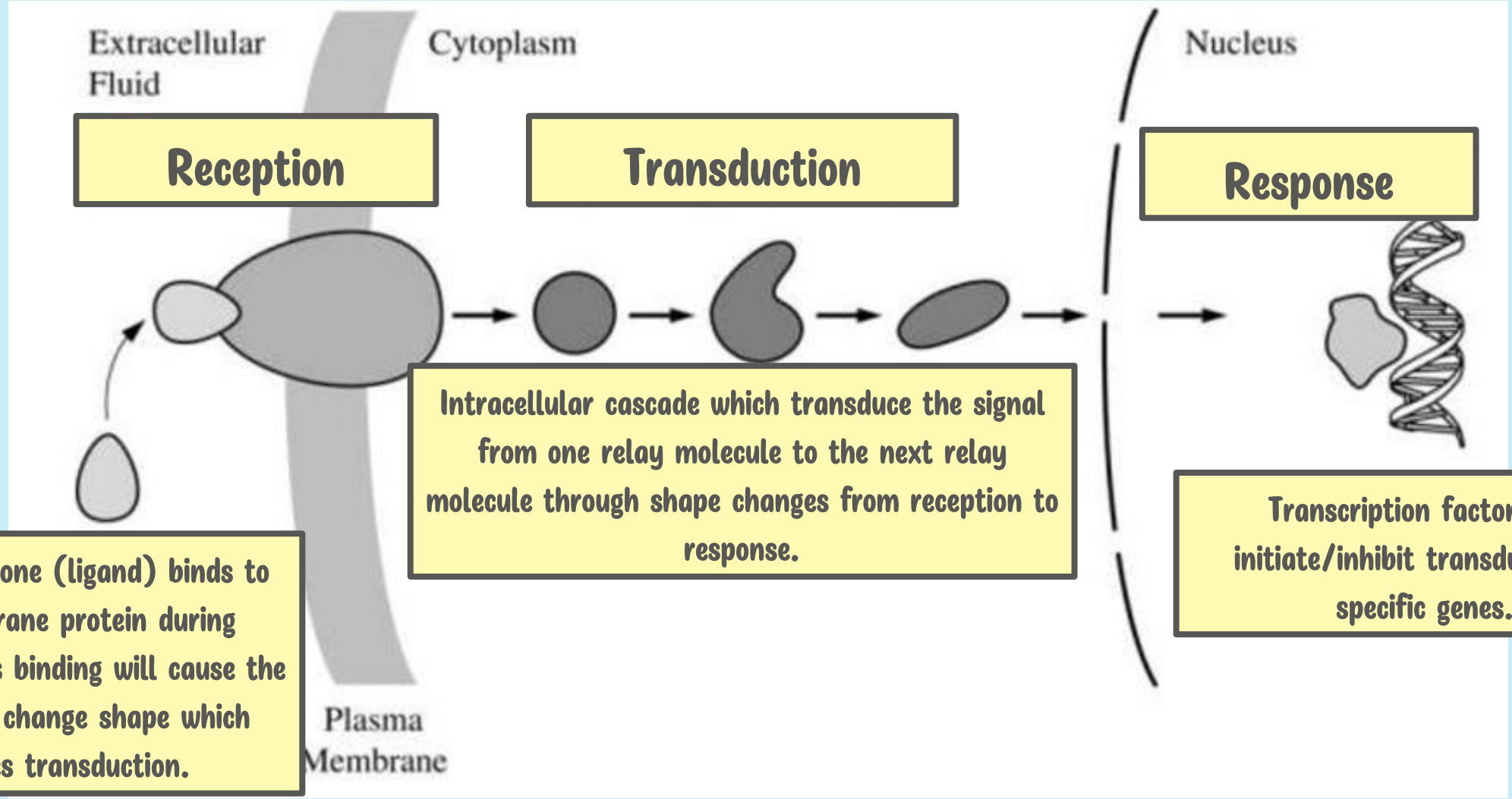
cell growth

secretion of molecules

gene expression

apoptosis

Cellular Communication



Protein hormone (ligand) binds to the membrane protein during reception. This binding will cause the receptor to change shape which initiates transduction.

Intracellular cascade which transduce the signal from one relay molecule to the next relay molecule through shape changes from reception to response.

Transcription factors will initiate/inhibit transduction of specific genes.

Example: 2021 #1

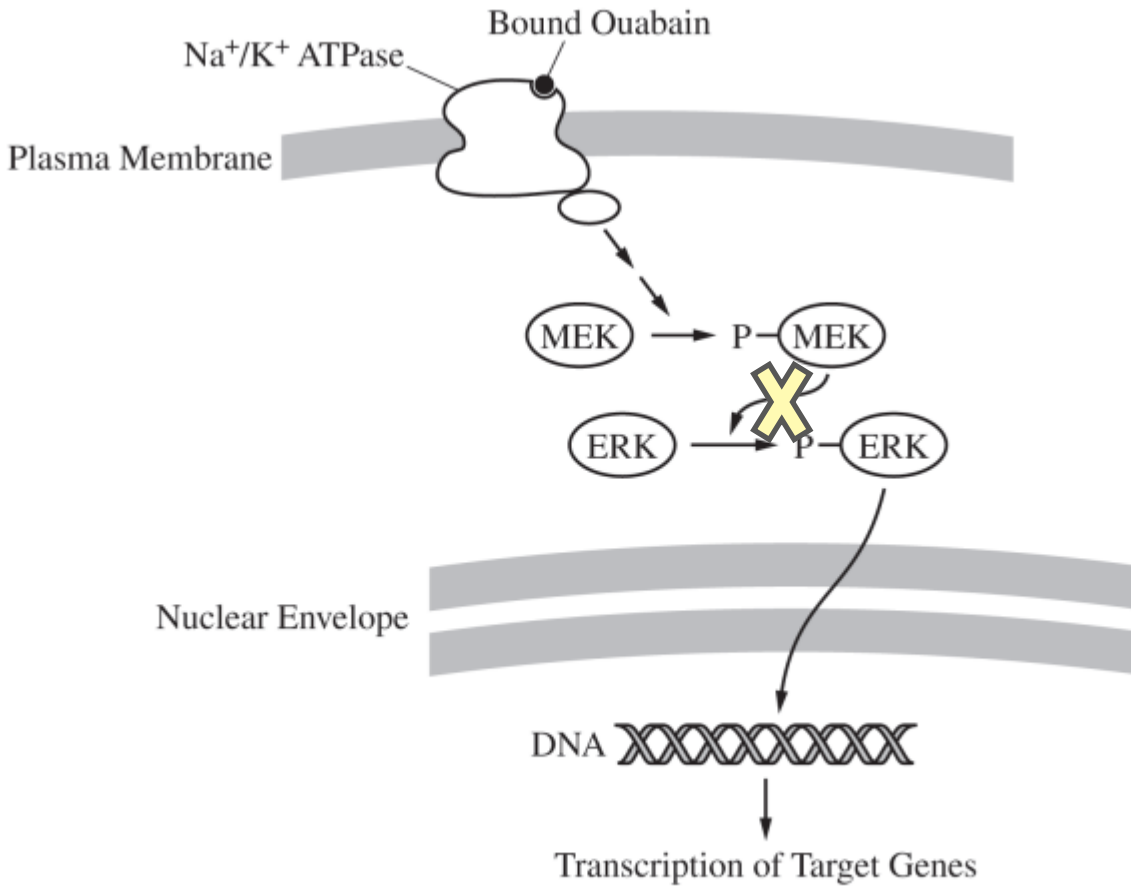


Figure 3. Signal transduction pathway hypothesized to play a role in the increased number o

In a third experiment, the scientists added an inhibitor of phosphorylated MEK (pMEK) to the PKD cells exposed to 10^4 ouabain. Based on Figure 3, predict the change in relative ratio of ERK to pERK in ouabain-treated PKD cells with the inhibitor compared with ouabain-treated PKD cells without the inhibitor. Provide reasoning to justify your prediction.

Accept one of the following:

- Option 1: The ratio of ERK to pERK will increase in the cells with the inhibitor.
- Option 2: The ratio of ERK to pERK will stay the same in the cells with the inhibitor.
- The justification must indicate that the pMEK inhibitor blocks further phosphorylation of ERK AND one of the following:

Option 1:

- The amount of pERK will not increase as it does in cells without the inhibitor.
- The amount of ERK will not decrease as it does in cells without the inhibitor.
- The cell continues to synthesize ERK.
- Phosphorylated ERK is being dephosphorylated to ERK.

Option 2:

- No additional ERK is synthesized/pERK is not being dephosphorylated.

Example: 2018 #2

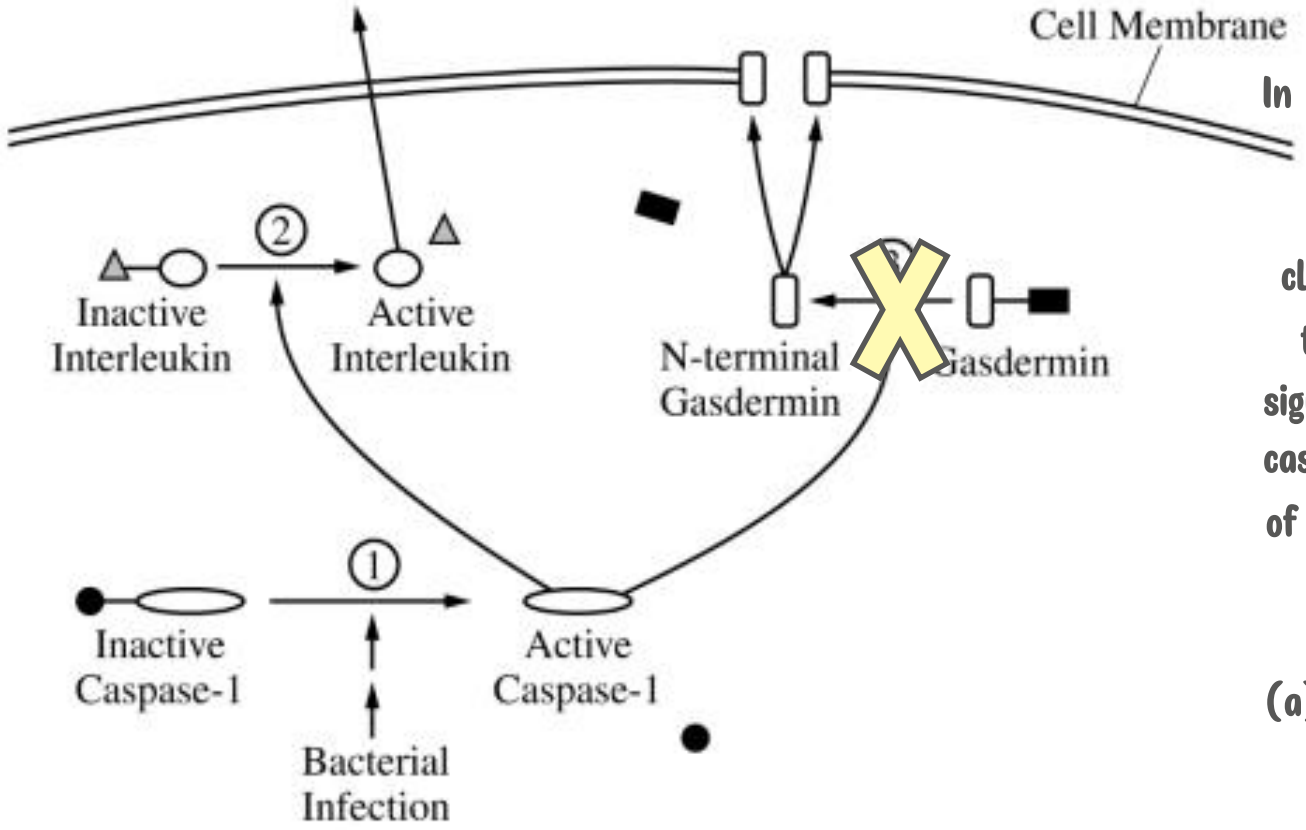


Figure 1. Cellular response to infection by pathogenic bacteria

In response to intracellular pathogens, the inactive caspase-1 is cleaved and forms an active caspase-1 (step 1). Active caspase-1 can cleave two other proteins. When caspase-1 cleaves an inactive interleukin (step 2), the active portion of the interleukin is released from the cell. An interleukin is a signaling molecule that can activate an immune response. When caspase-1 cleaves gasdermin (step 3), the N-terminal portions of several gasdermin proteins associate in the cell membrane to form large, nonspecific pores.

(a) Describe the effect of inhibiting step 3 on the formation of pores AND on the release of interleukin from the cell.

Description (2 points)

- Pores will not form.
- Interleukin release will not be affected/interleukin release continues.

Cell Cycle

G_1

Duplication of cell organelles

Synthesis of proteins, RNA, and building blocks

S

Replication of genetic material and centrosomes

G_2

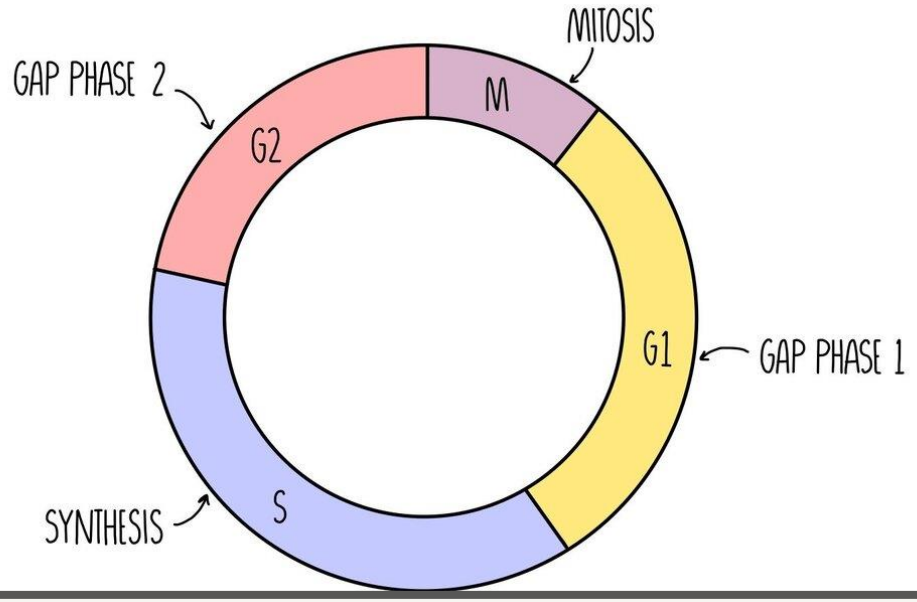
Synthesis of proteins and RNA

Makes organelles

Reorganizes cellular contents

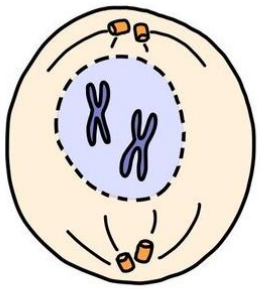
Interphase

The cell grows through all the different phases of interphase

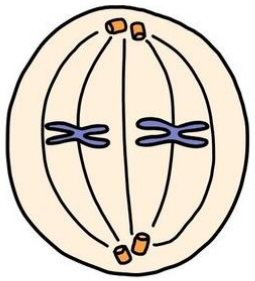


M

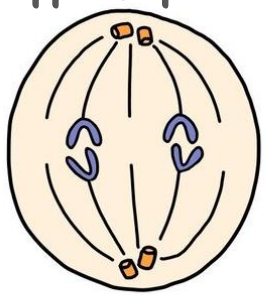
PREPARE to divide

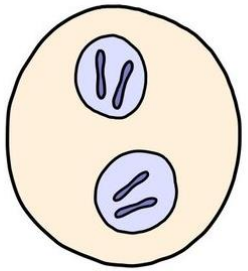


Sister Chromatids line up in the MIDDLE



Sister Chromatids pulled APART to opposite poles

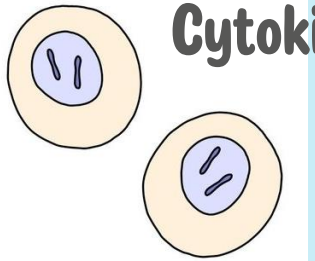




TWO new nuclei are formed

Division of the cytoplasm

Cytokinesis



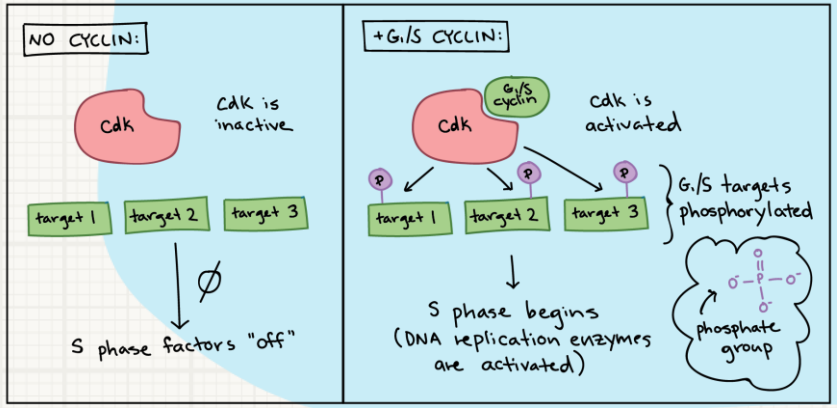
Checkpoints

G_1

During G_1, determines whether to complete the cell cycle

- Growth factor
- Adequate reserves
- Check for DNA damage

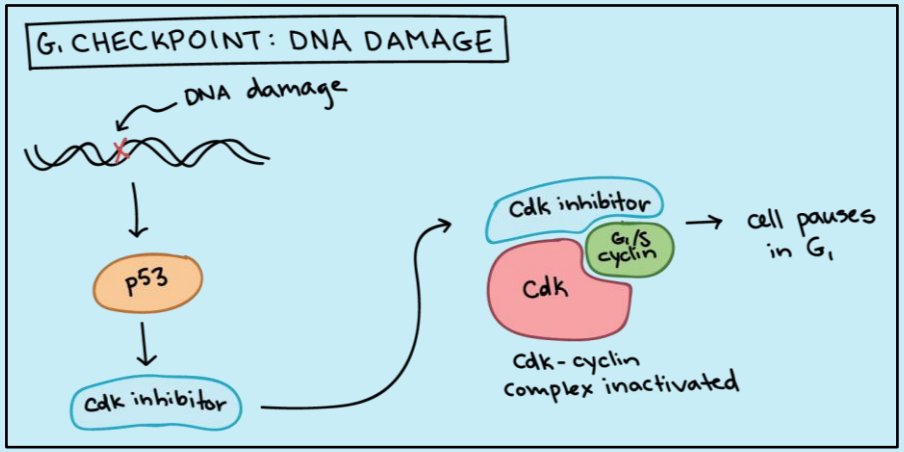
If do not pass, enter G_0 (nondividing state)



G_2

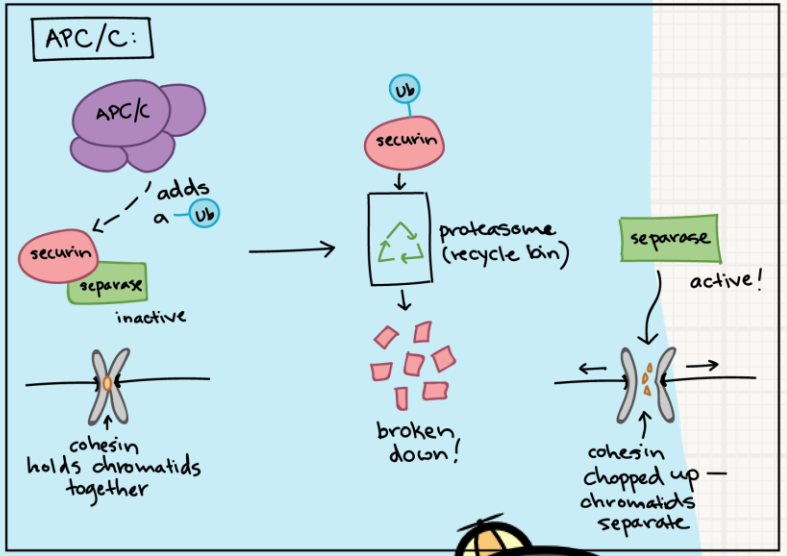
Check all DNA replicated and not damaged.

If detect problems with DNA, the cell cycle is halted, to complete DNA replication or repair the damaged DNA.



M

Check sister chromatids attached to the spindle microtubules



Multiple Choice Practice:

Insulin is a protein hormone that is secreted in response to elevated blood glucose levels. When insulin binds to its receptors on liver cells, the activated receptors stimulate phosphorylation cascades that cause the translocation of glucose transporters to the plasma membrane.

Based on the information provided, which of the following best describes the role of insulin in this liver cell signal transduction pathway?

a. It acts as a ligand.

b. It acts as a receptor.

c. It acts as a secondary messenger.

d. It acts as a protein kinase.

Multiple Choice Practice:

The endocrine system incorporates feedback mechanisms that maintain homeostasis. Which of the following demonstrates negative feedback by the endocrine system?

a. During labor, the fetus exerts pressure on the uterine wall, inducing the production of oxytocin, which stimulates uterine wall contraction. The contractions cause the fetus to further push on the wall, increasing the production of oxytocin.

b. After a meal, blood glucose levels become elevated, stimulating beta cells of the pancreas to release insulin into the blood. Excess glucose is then converted to glycogen in the liver, reducing blood glucose levels.

c. At high elevation, atmospheric oxygen is more scarce. In response to signals that oxygen is low, the brain decreases an individual's rate of respiration to compensate for the difference.

d. A transcription factor binds to the regulatory region of a gene, blocking the binding of another transcription factor required for expression.

Free Response Practice (2022 #1):

The binding of an extracellular ligand to a G protein-coupled receptor in the plasma membrane of a cell triggers intracellular signaling (Figure 1, A). After ligand binding, GTP replaces the GDP that is bound to Gsα, a subunit of the G protein (Figure 1, B). This causes Gsα to activate other cellular proteins, including adenylyl cyclase that converts ATP to cyclic AMP (cAMP). The cAMP activates protein kinases (Figure 1, C). In cells that line the small intestine, a cAMP-activated protein kinase causes further signaling that ultimately results in the secretion of chloride ions (Cl^-) from the cells. Under normal conditions, Gsα hydrolyzes GTP to GDP, thus inactivating adenylyl cyclase and stopping the signal (Figure 1, A).

Free Response Practice (2022 #1):

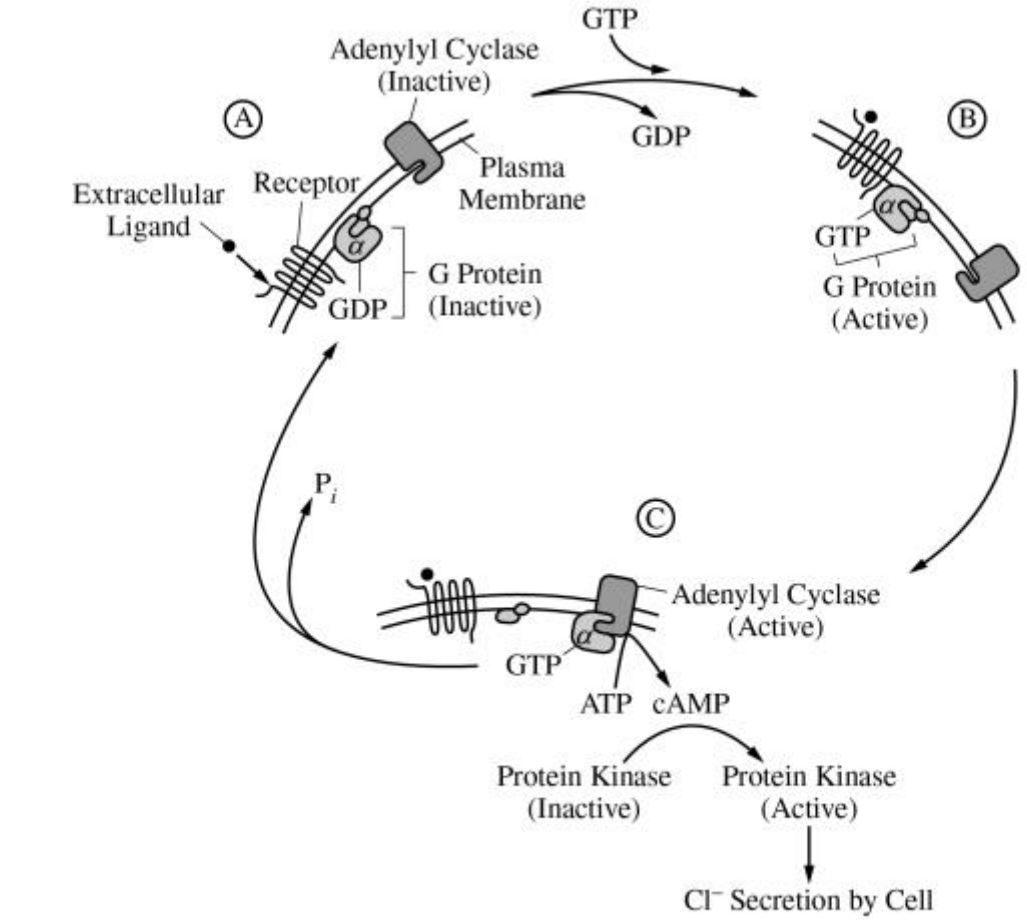


Figure 1. Under normal conditions, ligand binding to a G protein-coupled receptor results in chloride ion transport from an intestinal cell.

Free Response Practice (2022 #1):

Individuals infected with the bacterium *Vibrio cholerae* experience severe loss of water from the body (dehydration). This is due to the effects of the bacterial cholera toxin that enters intestinal cells. Scientists studied the effects of cholera toxin on four samples of isolated intestinal cell membranes containing the G protein-related signal transduction components shown in Figure 1. GTP was added to samples II and IV only; cholera toxin was added to samples III and IV only. The scientists then measured the amount of cAMP produced by the adenylyl cyclase in each sample (Table 1).

TABLE 1. AMOUNT OF cAMP PRODUCED FROM INTESTINAL CELL MEMBRANES IN THE ABSENCE OR PRESENCE OF CHOLERA TOXIN

Sample	GTP	Cholera Toxin	Rate of cAMP Production (pmol per mg adenylyl cyclase per min)
I	–	–	0.5
II	+	–	10.0
III	–	+	0.5
IV	+	+	127.0

present, +; absent, –

Free Response Practice (2022 #1a):

i. Describe one characteristic of a membrane that requires a channel be present for chloride ions to passively cross the membrane.

Accept one of the following:

- The interior of the membrane/phospholipid tail is nonpolar.
- The interior of the membrane/ phospholipid tail is not charged.
- The interior of the membrane/ phospholipid tail is hydrophobic.

ii. Explain why the movement of chloride ions out of intestinal cells leads to water loss.

Accept one of the following:

- The space outside of the cells becomes hypertonic/hyperosmotic compared with the cells, so water moves out of the cells.
- The space outside of the cells would have a lower water potential compared with the cells, so water will move out of the cells.

Free Response Practice (2022 #1b):

TABLE 1. AMOUNT OF cAMP PRODUCED FROM INTESTINAL CELL MEMBRANES IN THE ABSENCE OR PRESENCE OF CHOLERA TOXIN

Sample	GTP	Cholera Toxin	Rate of cAMP Production (pmol per mg adenylyl cyclase per min)
I	–	–	0.5
II	+	–	10.0
III	–	+	0.5
IV	+	+	127.0

present, +; absent, –

i. Identify an independent variable in the experiment.

Accept one of the following:

- The presence or absence of cholera toxin
- The presence or absence of GTP

ii. Identify a negative control in the experiment.

Accept one of the following:

- The sample lacking both cholera toxin and GTP/sample I
- The samples that lack cholera toxin/samples I and II
- The sample that lacks cholera toxin but contains GTP/sample II
- The samples that lack GTP/samples I and III

Free Response Practice (2022 #1b):

TABLE 1. AMOUNT OF cAMP PRODUCED FROM INTESTINAL CELL MEMBRANES IN THE ABSENCE OR PRESENCE OF CHOLERA TOXIN

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II	+	–	10.0
III	–	+	0.5
IV	+	+	127.0

present, +; absent, –

iii. Justify why the scientists included Sample III as a control treatment in the experiment.

Accept one of the following:

- (Sample III serves as a control) to compare cAMP production with that of the sample having cholera toxin and GTP/sample IV.
- Comparing sample III and sample IV enables the scientists to evaluate whether the activity of cholera toxin requires GTP/acts via the G protein pathway.

Free Response Practice (2022 #1c):

i. Based on the data, describe the effect of cholera toxin on the synthesis of cAMP.

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II	+	–	10.0
III	–	+	0.5
IV	+	+	127.0

present, +; absent, –

Accept one of the following:

- Cholera toxin increases the production of cAMP in the presence of GTP (IV vs II).
- Cholera toxin has no effect on the production of cAMP in the absence of GTP (III vs I).

Free Response Practice (2022 #1c):

ii. Calculate the percent change in the rate of cAMP production due to the presence of cholera toxin in Sample IV compared with sample II.

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II	+	–	10.0
III	–	+	0.5
IV	+	+	127.0

present, +; absent, –

$$\text{Percent change} = \frac{final - intital}{initial}$$

- 1,170% [(127-10)/10 = 11.7 × 100]

Free Response Practice (2022 #1d):

A drug is designed to bind to cholera toxin before it crosses the intestinal cell membrane. Scientists mix the drug with cholera toxin and then add this mixture and GTP to a sample of intestinal cell membranes.

i. Predict the rate of cAMP production in pmol per mg adenylyl cyclase per min if the drug binds to all of the toxin

- The rate will be 10 (pmol per mg adenyl cyclase per min).

TABLE 1. AMOUNT OF cAMP PRODUCED FROM INTESTINAL CELL MEMBRANES IN THE ABSENCE OR PRESENCE OF CHOLERA TOXIN

Sample	GTP	Cholera Toxin	Rate of cAMP Production (pmol per mg adenylyl cyclase per min)
I	–	–	0.5
II	+	–	10.0
III	–	+	0.5
IV	+	+	127.0

present, +; absent, –

Free Response Practice (2022 #1d):

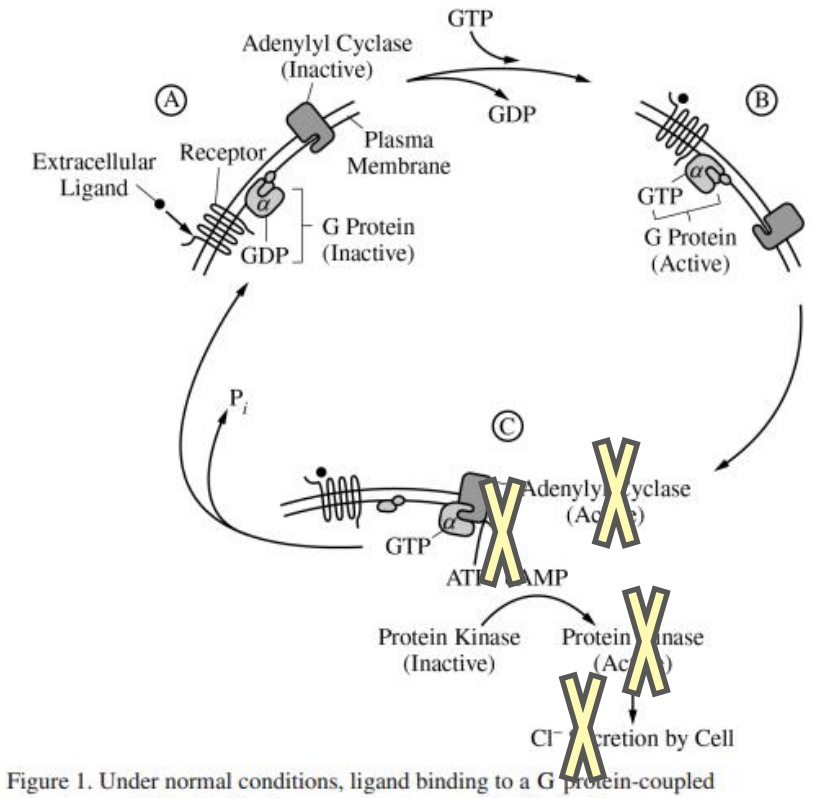

Figure 1. Under normal conditions, ligand binding to a G protein-coupled receptor results in chloride ion transport from an intestinal cell.

ii. In a separate experiment, scientists engineer a mutant adenylyl cyclase that cannot be activated by Gsa. The scientists claim that cholera toxin will not cause excessive water loss from whole intestinal cells that contain the mutant adenylyl cyclase. Justify this claim.

- (Even in the presence of the toxin) cAMP will not be produced (by this pathway), the protein kinases will not be activated, and Cl^- ions will not be secreted (and less water will leave the intestinal cells).

Q & A

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