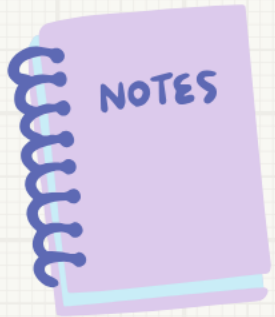
AP Bio FRQ Fridays

2022 #1
Cell Communication, Experimental Design, Tonicity, & Membrane Structure



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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).

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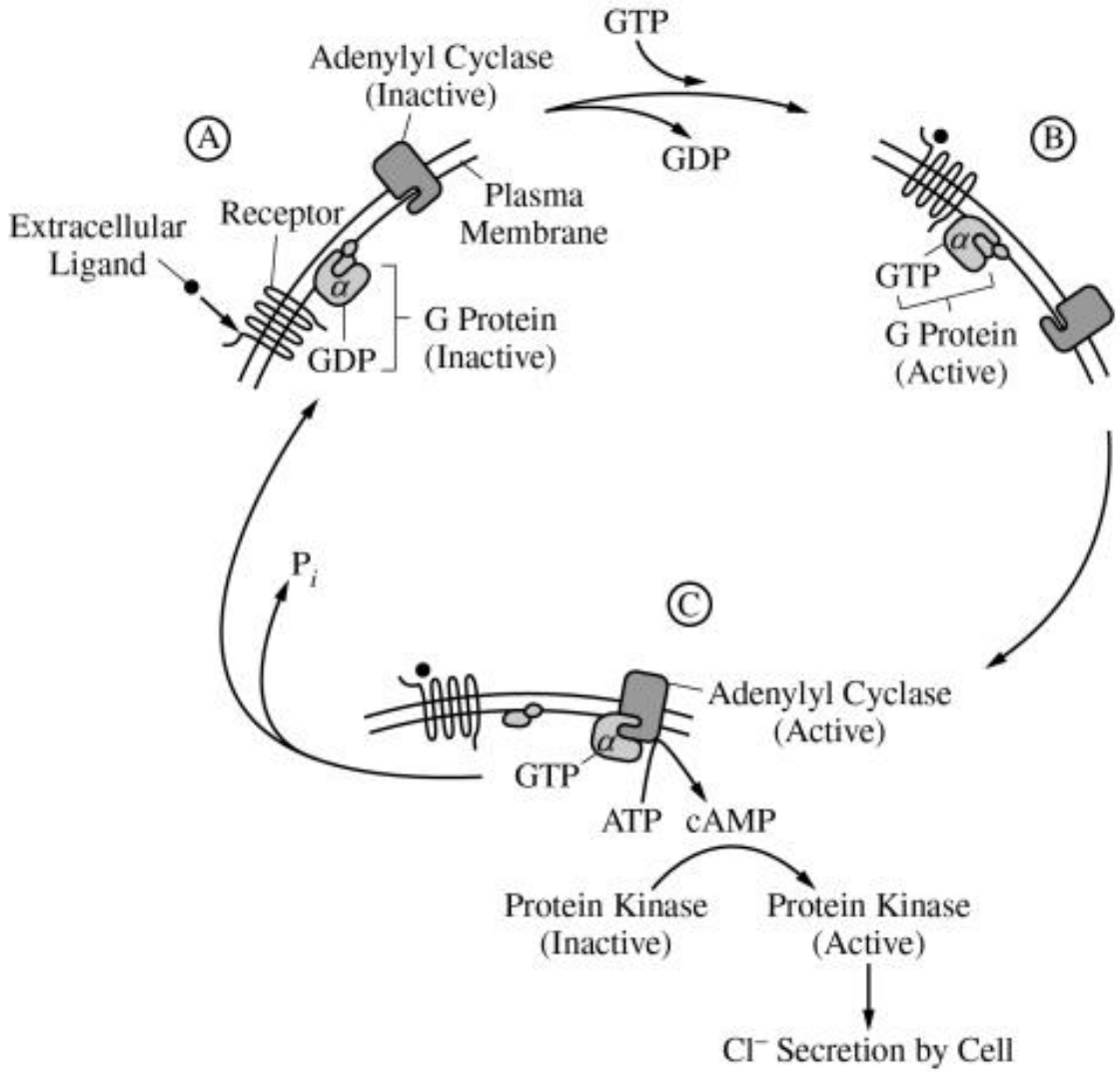

Figure 1. Under normal conditions, ligand binding to a G protein-coupled receptor results in chloride ion transport from an intestinal cell.

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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, −

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(a) **Describe** one characteristic of a membrane that requires a channel be present for chloride ions to passively cross the membrane. **Explain** why the movement of chloride ions out of intestinal cells leads to water loss.

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.

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.

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a. A membrane's hydrophobic interior would inhibit charged ions like chloride ions from passing through it. Therefore, a channel is needed to allow chloride ions to pass through. The movement of chloride ions

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to allow chloride ions to pass through. The movement of chloride ions out of intestinal cells lowers the water potential of the cells' external environment (more solute now) ; increases the water potential inside the cells (less solute now). Since water moves from areas of high water potential to areas of low water potential, water would move out of the intestinal cells, leading to water loss.

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Accept one of the following:

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

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

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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.

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Accept one of the following:

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

b. An independent variable in the experiment is the addition of cholera toxin. Sample I is a negative control. Scientists included Sample III as a control to observe the impact the addition of GTP has on cAMP production when the cholera toxin is added. Because GTP isn't added in Sample III, scientists can see the impact adding GTP had by comparing it to Sample IV.

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(c) Based on the data, **describe** the effect of cholera toxin on the synthesis of cAMP. **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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Percent change = $\frac{final - intital}{initial}$

- 1,170% [(127-10)/10 = 11.7 × 100]

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).

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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).

c. The cholera toxin dramatically increased the rate of cAMP production in the presence of GTP, with the rate jumping from 10.0 pmol per mg adenylyl cyclase per minute to 127 pmol per mg adenylyl cyclase per minute.

$$\frac{127.0-10.0}{10.0} \cdot 100 = \frac{117.0}{10.0} \cdot 100 = 11.7 \cdot 100 = \boxed{1,170\%}$$

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(d) 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. **Predict** the rate of cAMP production in pmol per mg adenylyl cyclase per min if the drug binds to all of the toxin. In a separate experiment, scientists engineer a mutant adenylyl cyclase that cannot be activated by Gsα. 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.

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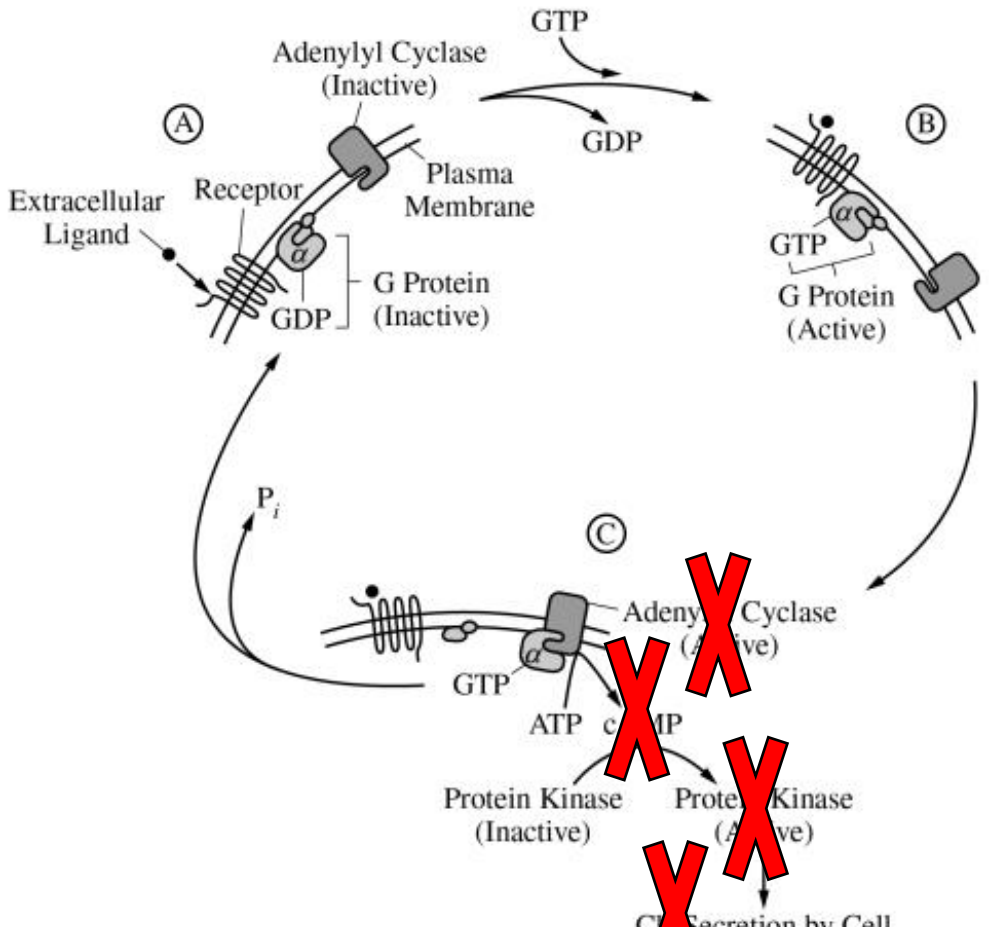
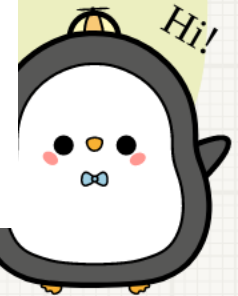


- (Even in the presence of the toxin) cAMP will not be produced (by this pathway), the protein kinases will not be activated, and Cl^- ions will not be secreted (and less water will leave the intestinal cells).



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d. The rate of cAMP production would be 10.0 pmol per mg adenylyl cyclase per min if the drug binds to all of the toxin. The intestinal cells won't experience excessive water loss because cAMP won't

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d. The rate of cAMP production would be 10.0 pmol per mg adenylyl cyclase per min if the drug binds to all of the toxin. The intestinal cells won't experience excessive water loss because cAMP won't be able to be produced as it's only transformed from ATP when Gsα binds to adenylyl cyclase, which the mutated version doesn't allow. With cAMP unable to be produced, protein kinases won't be activated, meaning Cl^- ions won't be secreted out of the cell. Since Cl^- ions aren't being pumped out, the intestinal cell's water potential will remain lower, meaning there's less water movement out of the ~~loss~~ cell.