

Past-paper walk-through

Tonicity and Osmoregulation ■ 2.7

eXercise

Questions in this set

- 2022 Q1
- 2019 Q2
- 2019 Q8
- 2018 Q2
- 2016 Q1

Reading the mark scheme

- **B1** a mark that stands on its own – the point is either made or not.
- **M1** a *method* mark: the right approach, even if the number is wrong.
- **A1** an *answer* mark, and it usually needs its M mark first.
- **C1** a working mark on the way to the answer.
- **//** separates answers the examiner accepts equally.
- **ecf** = error carried forward: a later part is marked on your own earlier answer.
- **owtte** = “or words to that effect” – the wording need not match.

Question 1

2022 ■ Q1

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 $G_s\alpha$, a subunit of the G protein (Figure 1, B). This causes $G_s\alpha$ 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, $G_s\alpha$ hydrolyzes GTP to GDP, thus inactivating adenylyl cyclase and stopping the signal (Figure 1, A).

[Figure 1: A cyclic diagram showing G protein-coupled receptor signaling in an intestinal cell membrane, with three stages labeled A, B, C connected in a cycle. Stage A: an "Extracellular Ligand" binds to a "Receptor" in the "Plasma Membrane"; the

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receptor is coupled to an inactive "G Protein" (with GDP bound) next to inactive "Adenylyl Cyclase"; GTP is shown converting the complex forward, releasing GDP, moving to stage B. Stage B: the "G Protein (Active)" is shown bound to GTP, moving to stage C. Stage C: "Adenylyl Cyclase (Active)" converts ATP to cAMP; GTP is bound to the G protein alpha subunit; cAMP activates "Protein Kinase (Inactive)" to become "Protein Kinase (Active)", which leads to " Cl^- Secretion by Cell"; a P_i arrow returns from stage C back to stage A, representing hydrolysis of GTP to GDP that inactivates adenylyl cyclase and restarts the cycle at A.]

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

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

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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).

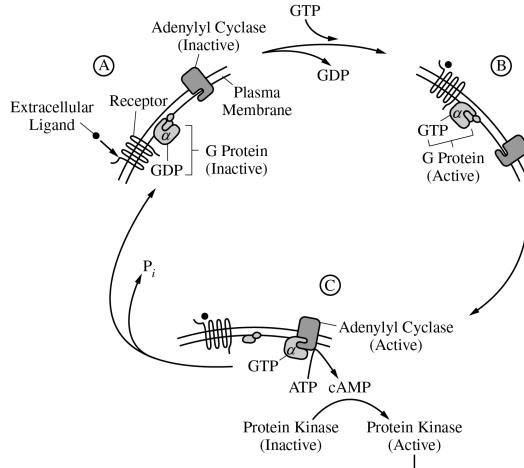
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I	—	—	0.5
II	+	—	10.0
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IV	+	+	127.0

present, +; absent, —

(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.

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Cl^- Secretion by Cell

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(b) Identify an independent variable in the experiment. Identify a negative control in the experiment. Justify why the scientists included Sample III as a control treatment in the experiment.

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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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2022 ■ Q1

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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 $G_s\alpha$. 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.

Solution 1

(a)

Mark scheme

1 pt A channel is required because the interior of the membrane (phospholipid tail) is nonpolar / not charged / hydrophobic, so charged chloride ions cannot passively cross the lipid bilayer without a channel. [1 pt] Movement of Cl^- out of intestinal cells makes the extracellular space hypertonic/hyperosmotic (lower water potential) compared with the cytoplasm, so water moves out of the cells by osmosis, causing water loss (dehydration).

Solution 1

(b) Mark scheme

- 1 pt** Independent variable: the presence or absence of cholera toxin (or presence/absence of GTP). [1 pt] Negative control: the sample lacking both cholera toxin and GTP (Sample I) - or equivalently, the samples lacking cholera toxin (I and II), the sample lacking cholera toxin but containing GTP (Sample II), or the samples lacking GTP (I and III). [1 pt] Justification for including Sample III as a control: Sample III (no GTP, + toxin) is compared with Sample IV (+GTP, + toxin) to evaluate whether cholera toxin's effect on cAMP production requires GTP, i.e. acts via the G-protein pathway.

Solution 1

(c)

Mark scheme

1 pt Cholera toxin increases cAMP production in the presence of GTP (Sample IV = 127.0 vs Sample II = 10.0); cholera toxin has no effect on cAMP production in the absence of GTP (Sample III = 0.5 vs Sample I = 0.5). [1 pt] Percent change = $[(127-10)/10] \times 100 = 11.7 \times 100 = 1,170\%$ increase in the rate of cAMP production due to cholera toxin (Sample IV compared with Sample II).

Solution 1

(d)

Mark scheme

- 1 pt If the drug binds all of the toxin, the toxin is neutralized, so the predicted rate of cAMP production equals the GTP-only rate (Sample II) = 10 pmol per mg adenylyl cyclase per min. [1 pt] Justification for the mutant-adenylyl-cyclase claim: even in the presence of cholera toxin, cAMP will not be produced via this pathway (because the mutant adenylyl cyclase cannot be activated by Gs-alpha), so the protein kinases will not be activated and Cl⁻ ions will not be secreted, so less water will leave the intestinal cells (no excessive water loss).

Question 2

2019 ■ Q2

A student studying two different aquatic, plant-eating, unicellular protist species (species A and B) designed an experiment to investigate the ecological relationship between the two species (Table 1).

TABLE 1. EXPERIMENTAL TREATMENT GROUPS

Group	Description
Group I.	Species A and B are each grown in separate containers.
Group II.	Species A and B are grown together in the same container.

Question 2

In treatment group I, the student placed 10 individuals of species A into a container with liquid growth medium and 10 individuals of species B into a separate container with an equal amount of the same liquid growth medium. In treatment group II, the student placed 5 individuals of each species into a single container with the liquid growth medium. The student then maintained the containers under the same environmental conditions and recorded the number of individuals in each population at various time points. The results are shown in Table 2.

TABLE 2. NUMBER OF INDIVIDUALS IN EACH PROTIST POPULATION IN BOTH TREATMENT GROUPS

Time (h)	Group I. Grown Separately — Species A	Group I. Grown Separately — Species B	Group II. Grown Together — Species A	Group II. Grown Together — Species B
0	10	10	5	5
10	100	50	45	20
20	400	200	100	50
30	1100	500	250	25
40	1400	650	525	20
50	1500	700	900	10
60	1500	700	1250	0
70	1500	700	1400	0

Question 2

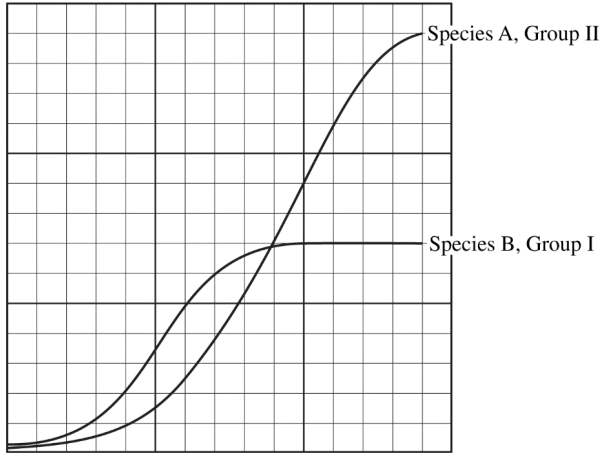
[Graph template: a blank grid (graph paper) with two curves already plotted (shaded columns of Table 2): “Species A, Group II” - an S-shaped curve rising steeply then leveling off at the top right; “Species B, Group I” - an S-shaped curve rising then leveling off at a lower plateau, labeled partway up the right side. Axes are unlabeled on the template, to be completed by the student with the unshaded columns (Species A, Group I and Species B, Group II).]

(a) The growth curves for species B in group I and for species A in group II (shaded columns) have been plotted on the template. Use the template to **complete** an appropriately labeled line graph to illustrate the growth of species A in treatment group I and species B in treatment group II (unshaded columns).

Question 2

	Group I. Grown Separately		Group II. Grown Together	
Time (h)	Species A	Species B	Species A	Species B
0	10	10	5	5
10	100	50	45	20
20	400	200	100	50
30	1100	500	250	25
40	1400	650	525	20
50	1500	700	900	10
60	1500	700	1250	0
70	1500	700	1400	0

Question 2



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