

Past-paper walk-through

Facilitated Diffusion ■ 2.6

eXercise

Questions in this set

- 2024 Q3
- 2022 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 3

2024 ■ Q3

To investigate whether red blood cells of animals lose the ability to take in glucose from their environment as they age, scientists collected red blood cells from guinea pigs that ranged in age from one day old to seven months old. Scientists incubated an equal number of red blood cells in separate culture dishes that contained a 300 nM solution of radioactively labeled glucose. The amount of radioactively labeled glucose present inside the red blood cells of each group was measured over time.

- (a) **Describe** a difference between passive transport and active transport.
- (b) **Justify** why the scientists used an equal number of red blood cells in each culture dish as a control.
- (c) Glucose transporters are required for the facilitated diffusion of glucose into red blood cells. The scientists claim that the expression of the gene encoding these transporters decreases as guinea pigs age. If the scientists' claim is supported by

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experimental data, **predict** the effect of increased age on the amount of radioactively labeled glucose present inside the cells of each group.

(d) Justify your prediction in part (c).

Solution 3

(a)

Mark scheme

- Active transport requires energy/ATP while passive transport does not (equivalently: in passive transport substances move from high to low concentration down their gradient, while in active transport substances move from low to high concentration against their gradient).

Solution 3

(b)

Mark scheme

- Using an equal number of red blood cells in each dish lets the scientists attribute any differences in results/glucose transport specifically to guinea pig age rather than to differing cell numbers — it eliminates cell number as a confounding variable that could otherwise affect the measured amount of glucose.

Solution 3

(c)

Mark scheme

- As guinea pig age increases, the amount of radioactively labeled glucose present inside the cells decreases.

Solution 3

(d)

Mark scheme

- If glucose-transporter gene expression decreases with age (as claimed), older guinea pigs' red blood cells will have fewer glucose transporters, so fewer glucose molecules will be moved into the cells by facilitated diffusion — consistent with the predicted decrease.

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).

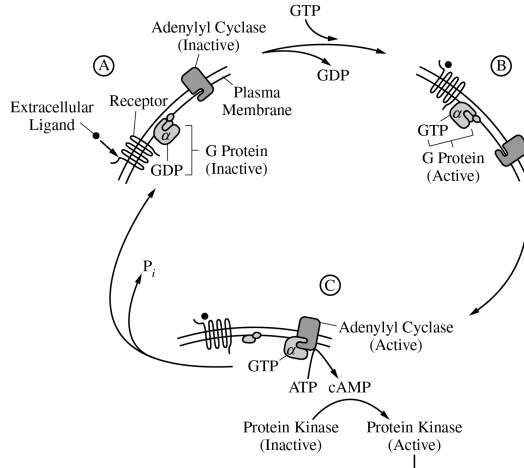
TABLE 1. AMOUNT OF cAMP PRODUCED FROM INTESTINAL CELL MEMBRANES IN THE ABSENCE OR PRESENCE OF CHOLERA TOXIN

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II	+	—	10.0
III	—	+	0.5
IV	+	+	127.0

present, +; absent, —

(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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2022 ■ Q1

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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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(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).