Solutions

Marking: **M1** a correct method, **A1** the correct answer, **B1** an independent mark.

Each answer shows the steps, then the **result**.

2.1

glucagon binds the receptor → G protein on

→ adenylyl cyclase on → cAMP made

→ protein kinase A active → glycogen broken down

2.2

$20 \times 1000 \times 2$

$= \mathbf{40\,000}$

2.3

$10^{-8} \times 2 \times 10^{-12} = 2 \times 10^{-20}$ mol

$\times 6.02 \times 10^{23}$

$\approx \mathbf{12\,000}$ molecules

2.4

glucagon is a water-soluble protein and cannot cross the lipid bilayer, so it acts at a surface receptor

cortisol is fat-soluble, crosses the membrane, and binds a receptor inside the cell that acts on DNA

3.1

(a) Gαs cannot switch itself off, so adenylyl cyclase stays on and cAMP stays high; protein kinase A keeps the CFTR chloride channels open; Cl^- flows into the gut and water follows by osmosis

(b) the changed G proteins stay on until the cells are replaced, in a few days; keeping up with the losses keeps the patient alive until then

(c) $0.75 \times 8 = \mathbf{6}$ L: more than the whole blood volume, so the losses must be replaced as they happen

3.2

(a) the GTPase of Gα turns the G protein off; phosphodiesterase breaks down cAMP; phosphatases remove phosphates from proteins

(b) the resting cAMP pool is replaced in about a second, so without fast off switches the cell could not follow changes in the signal, and the response would stay on

3.3

(a) mutant RAS cannot break down its GTP, so it stays on and keeps sending the division signal, like the cholera G protein

exercises.lol exercises.com.cn

(b) the drug blocks a step above RAS, but mutant RAS is already on and does not need the receptor

(c) $0.4 \times 60 = \mathbf{24}$ patients; the other 76 get side effects with no benefit, including all 40 with RAS mutations

(d) test every tumour for RAS first; give the drug only to patients without the mutation, and other treatment to the rest