TAMPINES MERIDIAN JUNIOR COLLEGE

JC2 PRELIMINARY EXAMINATION

H2 BIOLOGY **9744/02**

Paper 2 Structured Questions

11 September 2024
2 hours

SUGGESTED ANSWERS

No	Oh dear, where did I go wrong? ☹	Affected Questions	I can improve by doing the following! ☺
1	I **don't understand** what the question wants from me.		• Identify topic(s) related to the question. • Analyse the preamble and/or diagram carefully. • Unpack the command term (e.g. explain, describe)
2	I don't know / can't remember the **conceptual facts**.		• Review my study techniques – what is effective and what is not? • Approach my tutor / peers for advice.
3	I did not give the **essential keywords** / wrong keyword.		• Reflect on why the missing words / phrases were essential in addressing the question.
4	My answers are **incomplete** / not of enough depth.		
5	I **misinterpreted** the questions / data, hence wrote the wrong answer / out-of-point answer		• Read the preamble carefully. • Paraphrase the question in my own words. • Unpack the command term (e.g. explain, describe)
6	I did not **contextualize my answers** to the question. That is, I did not make use of the information in the preamble / stimulus / figure.		• When the question revolves around a specific example, use the contextual information to craft the answers.
7	I did not **cite data** / I did not include the units for data / did not cite meaningful data for both axes.		• Cite complete data: both x & y axes, with units. • Examine the trend of the graph. If appropriate, divide the graph into ≥ 2 parts for meaningful citation of data.
8	I did not **organize my answers** properly, especially for comparison questions / essay questions.		• For comparison questions, ensure each sentence focuses on one feature. Use comparative words (e.g. but, whereas, while) • For essay questions, organise each major idea in a separate paragraph.
9	I did not manage to attempt the question due to **insufficient time.**		• Look through the whole paper and first attempt questions I am more confident in. • Be concise & succinct. Do not write excessively. • When I am stuck at a question, move on.
10	I was not able to **apply the conceptual facts** to this kind of 'suggest' questions.		• Identify topic(s) that the question is related to and draw links to the concept. • Examine any hint(s) / information in the preamble to suggest biologically sensible ideas.

Answer **all** questions.

1. Cholesterol is synthesized in the smooth endoplasmic reticulum (SER) in liver cells by a series of enzyme-catalyzed reactions.

Within the SER, molecules of cholesterol and triglycerides are surrounded by proteins and phospholipids to form lipoproteins. These lipoprotein particles enter the Golgi apparatus where they are packaged into vesicles and released into the bloodstream.

Fig. 1.1 is an electron micrograph of parts of two liver cells showing lipoprotein particles within the Golgi apparatus.

Fig. 1.2 shows the structure of a lipoprotein particle.

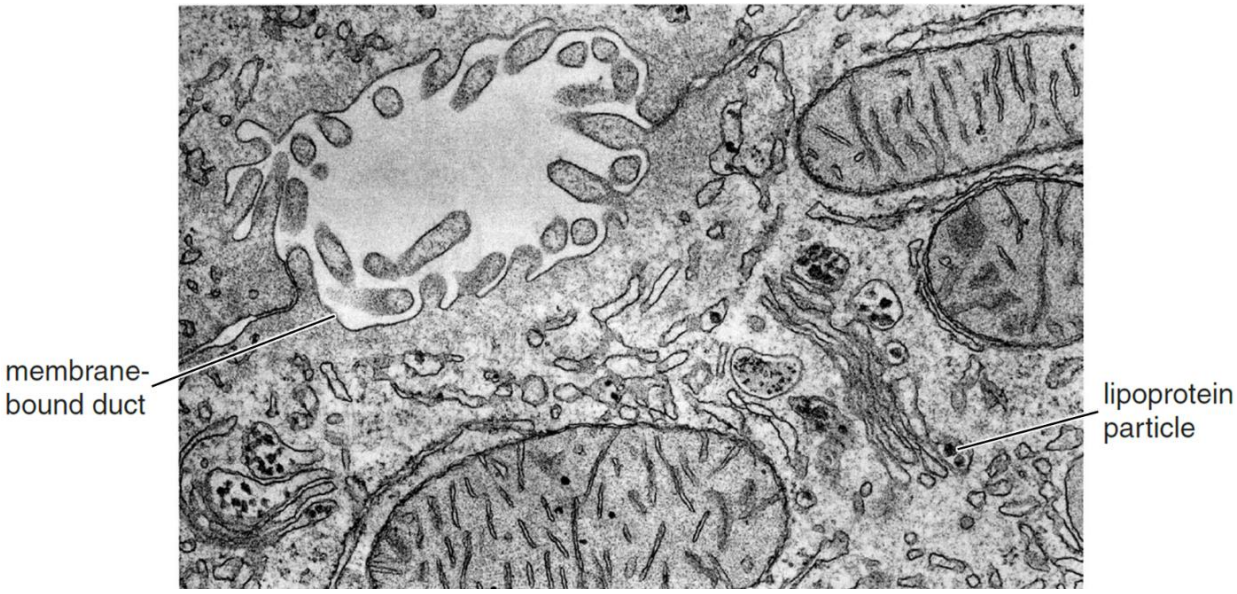


Fig. 1.1

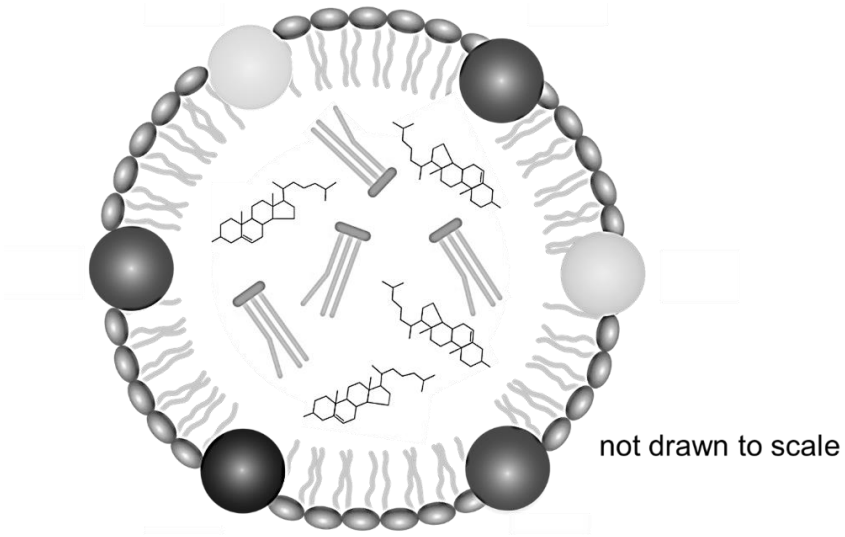


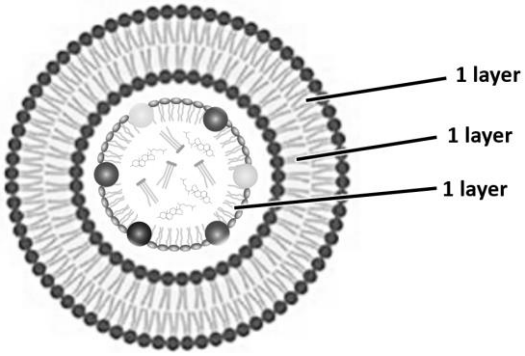
Fig. 1.2

(a) State the number of phospholipid layers formed when a **lipoprotein** particle is packaged into a **vesicle** by the Golgi apparatus. **[Cell, HI-2]** [1]

- 3

GENERAL COMMENTS:

- You need to learn to READ preambles and figures.



(b) (i) Explain why cholesterol and triglycerides need to be packaged into lipoproteins before they are released from liver cells into the bloodstream. **[Cell, KU-2]** [3]

1. Cholesterol/triglycerides are **hydrophobic** / **lipid-soluble** / **not water-soluble**.
2. Lipoprotein is a **phospholipid monolayer**
3. Its **hydrophobic fatty acid tails** **interact** with **cholesterol/triglycerides**.
4. Its **hydrophilic phosphate heads** allow cholesterol/triglycerides to be transported in **aqueous blood**.

(ii) State **two** roles of cholesterol in cells. **[Cell, KU-1]** [2]

1. A component of cell membranes.
2. Regulate membrane fluidity.
3. The starting molecule to synthesize bile salts / steroid hormones / vitamins / ***named molecules***

ERRORS:

✗ *"Provide insulation for the cell", "source of energy", "converted into glucose"*

✓ **Undiscovered roles of cholesterol currently discovered by you?** 😊

(c) Cholesterol is also **packaged into vesicles** by the SER and then secreted from the cell into small fluid-filled spaces between the liver cells. These spaces form ducts that drain into the gallbladder to form bile.

Describe how cholesterol is secreted into ducts, such as the duct shown in Fig. 1.1.

[Cell, KU-1] [2]

1. SER vesicles move to the **cell surface membrane** / **space between liver cells**.
2. Vesicles **fuse** with the cell surface membrane.
3. Cholesterol released/secreted into the duct via **exocytosis**.

ERRORS:

✗ *"budding", "endocytosis", "pseudopodia"*

✓ **Seems like you didn't really learn anything from the Afternoon Practice.** 😊

(d) Other than the packaging of substances into vesicles for transport, the Golgi apparatus also modifies proteins into their active form.

State **two** ways in which a protein can be modified into its active form. **[Cell, KU-1]** [2]

1. Glycosylation / adding oligosaccharides to protein
2. Phosphorylation / adding phosphate group to protein
3. Hydroxylation / adding hydroxyl group to protein
4. Cleavage of protein into active form
5. Assembly of protein subunits into its quaternary structure

ERRORS:

✗ *"removal of 1st amino acid methionine"*

✓ **The removal of 1st amino acid methionine determines the half-life of the protein. Even with methionine, the protein is already active.**

✗ *"folding of the polypeptide into its 3D conformation"*

✓ **Folding is not considered as protein modification. Furthermore, the polypeptide has already folded in the rough ER upon translation.**

✗ *"hydrolysis of the protein"*

✓ **This sounds like the protein is degraded into individual amino acids or smaller peptides.**

[Total: 10]

2. **(a)** The major constituent of plant cell walls is cellulose. Selected parts of the cell wall contain another type of polysaccharide called callose.

Fig. 2.1 is a simplified diagram showing the structure of callose. Not all hydrogen atoms are shown.

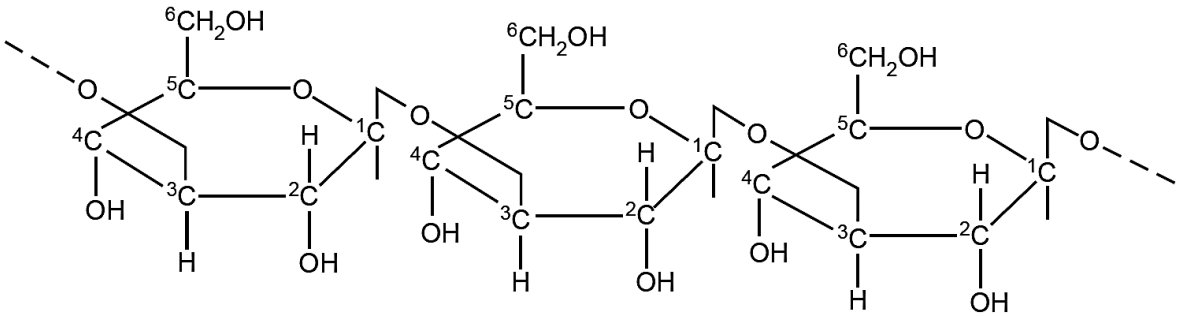


Fig. 2.1

Compare the structure of a callose molecule with a cellulose molecule. **[Biomol, HI-2]** [3]

feature	callose	cellulose
monomer	β-glucose	
branching	unbranched	
shape	straight / linear	
type of glycosidic linkage	1,3-glycosidic bond	1,4-glycosidic bond
orientation of monomer	adjacent glucose molecules same way up / **AW**	alternate glucose molecules rotated by 180°

ERRORS:

✗ *"Cellulose is formed by 1,6-glycosidic bond."*

✓ **Confusion with starch/glycogen?**

(b) Amino acids are the building blocks of proteins. Fig. 2.2 shows the R-groups of two amino acids, phenylalanine and serine.

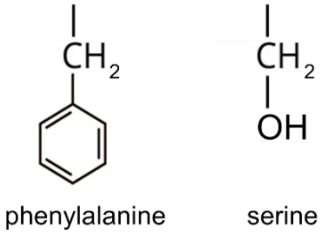
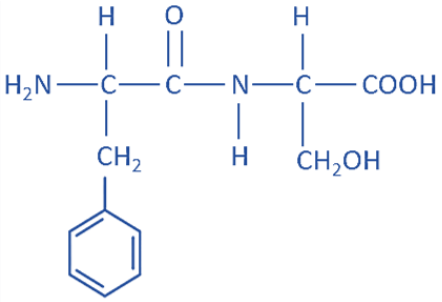


Fig. 2.2

(i) In the space below, draw a dipeptide formed from the condensation reaction between phenylalanine and serine. **[Biomol, KU-1]** [3]



1. correct peptide bond region (NHCO)
2. correct amino end (either NH_2 or NH_3^+) **and** carboxylic acid end (either COOH or COO^-)
3. correct structural formula of the two given R-groups [**Accept:** Phe-Ser or Ser-Phe]
4. a hydrogen connected to the central carbon

GENERAL COMMENTS:

- If you drew some weird diagrams, I suggest you revisit ALL the tutorials to reestablish your foundation all over again (albeit a race against time – about 5 weeks? 😊)
- Also, dipeptide = two amino acids (**not** two peptide bonds).

(ii) Collagen and haemoglobin are two examples of proteins.

Outline the structural differences between collagen and haemoglobin. **[Biomol, HI-1]** [4]

feature	collagen	haemoglobin
1. overall structure	fibrous	globular
2. secondary structure	left-handed helix	α-helix [**accept:** right-handed helix]
3. presence of tertiary structure	absent	present
4. no. of polypeptides	tropocollagen comprises three polypeptides	four polypeptides
5. no. of types of polypeptides	one type	two types [**accept:** α and β chains]
6. hierarchy	organized into fibrils & fibers	no hierarchical organization
7. nature of amino acid sequences	repeating	non-repeating
8. amino acid diversity	less varied / predominantly glycine, proline and hydroxyproline	wide variety
9. presence of other molecules	No additional groups	haem group

[Any 4]

[Total: 10]

3. Fig. 3.1 shows an overview of the main stages in the breakdown of a glucose molecule in a mammalian cell when oxygen is freely available.

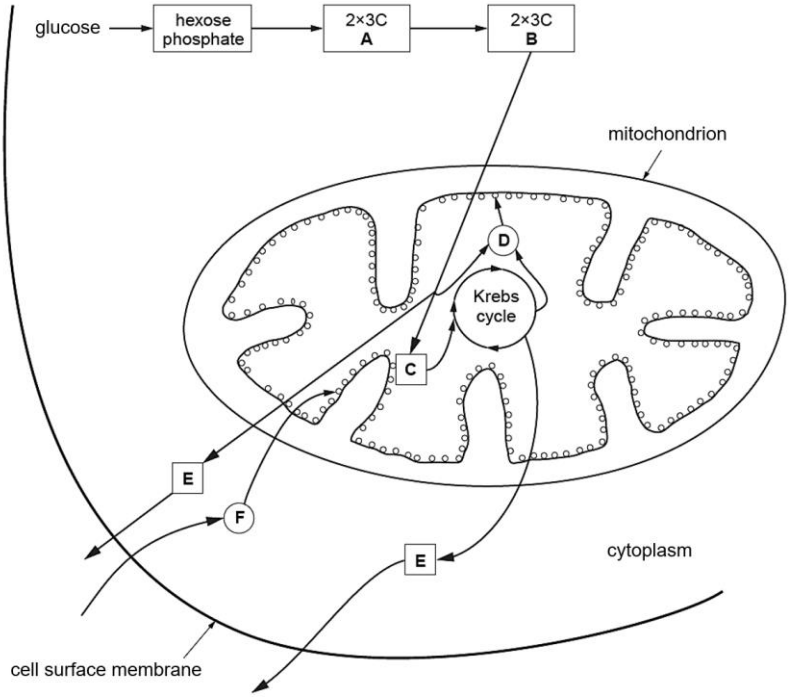


Fig. 3.1

(a) Name the molecules labeled **A** to **F**. **[Resp, KU-2]** [3]

A triose phosphate / glyceraldehye-3-phosphate / **any of the 3-carbon intermediate**
B pyruvate / pyruvic acid
C acetyl-coA / acetyl coenzyme A
D NADH / reduced NAD
E carbon dioxide / CO_2
F oxygen / O_2

[3m: 6, 2m: 4-5, 1m: 2-3]

(b) Explain how mammalian cells can continue to function in the absence of oxygen. **[Resp, KU-1]** [3]

1. **Reduction** of pyruvate to **lactate** / lactic acid
2. **Regenerates NAD**
3. NAD allows **glycolysis** to continue.
4. **ATP** produced by **substrate-level phosphorylation**

ERRORS:

✗ *"Pyruvate is converted/oxidised to lactate"*

✓ **Specifically, the reaction is a REDUCTION reaction. That's why NADH is oxidised back to NAD.**

✗ *"Ethanal, ethanol"*

✓ **The question asked for mammalian cells, NOT yeasts or plants.**

(c) Brown adipose tissue (BAT) is involved in the maintenance of a constant body temperature when the external environment is cold, which is especially important in infants. BAT is also found in adults but in relatively smaller quantities.

Mitochondria in BAT cells function differently from those in other cells during periods of cold environmental conditions.

Fig. 3.2 shows part of a mitochondrion in a BAT cell.

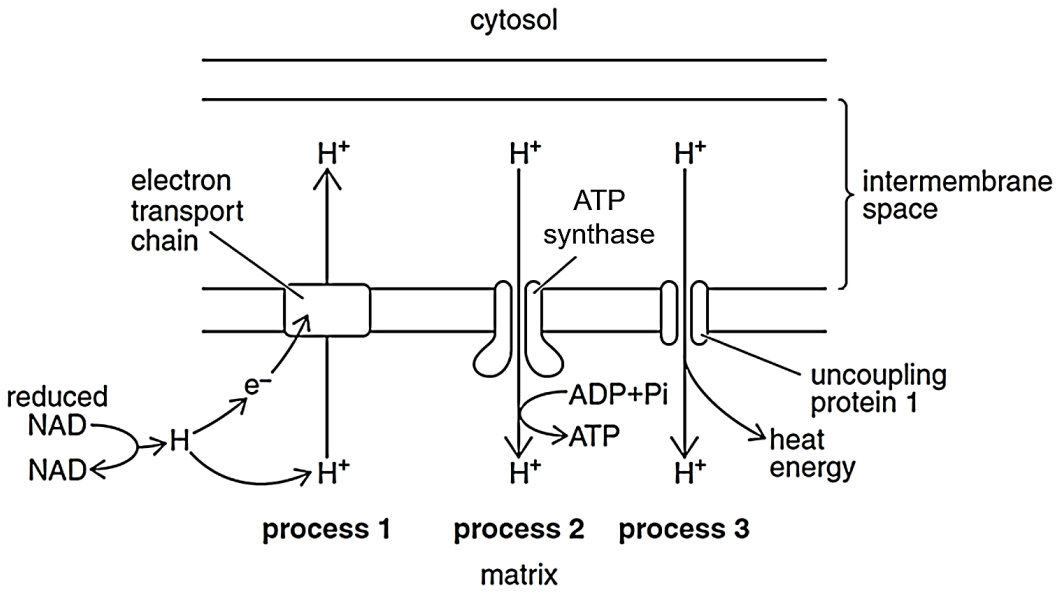


Fig. 3.2

It was found that during periods of cold external environmental conditions, the amount of ATP produced by BAT cells did not change.

State **and** explain which **two** of the three processes shown in Fig. 3.2 will be more active during periods of cold external environmental conditions. **[Resp, HI-2]** [4]

1. Processes 1 and 3

2. **[Process 1]** An increase in electron transport down electron carriers of progressively lower energy levels in the electron transport chain…

3. **[Process 1]**…releases more energy to pump H^+ from the matrix to the intermembrane space.

4. This gives a **steeper proton gradient** across the inner mitochondrial membrane / increases concentration of H^+ in the intermembrane space.

5. **[Process 3]** **More H^+** diffuse through **uncoupling protein 1** to **generate more heat energy**.

6. Amount of H^+ diffusing through ATP synthase remains unchanged, hence amount of ATP produced did not change.

[Total: 10]

4. Fig. 4.1 shows the transcription of part of the exon of the *β-globin* gene.

Structure **A** represents an enzyme involved in transcription.

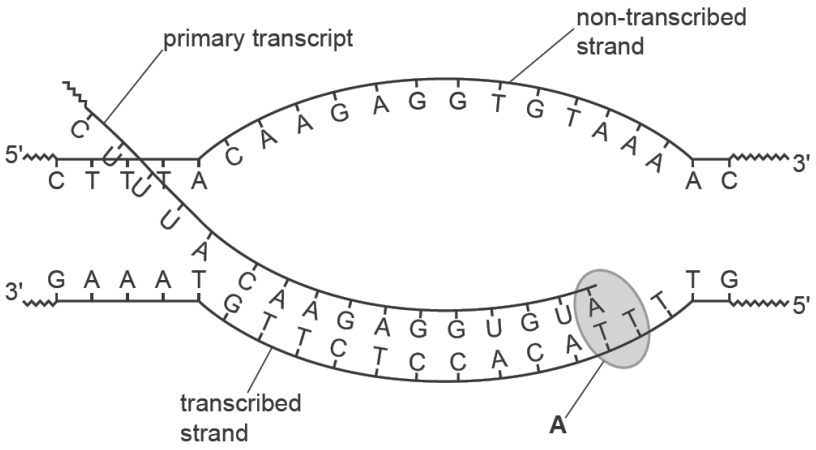


Fig. 4.1

(a) (i) Describe the next step carried out by the enzyme labelled **A** after the time point shown in Fig. 4.1. **[Gene Exp, KU-1]** [3]

1. RNA polymerase **reads the template strand** from **3' to 5' direction**
2. **Adds a ribonucleotide** carrying an **adenine** base…
3. …to the free 3'OH group of the growing primary transcript / mRNA
4. by **complementary base pairing** with **thymine**
5. RNA polymerase catalyses the formation of **phosphodiester bond** between the adjacent nucleotides.

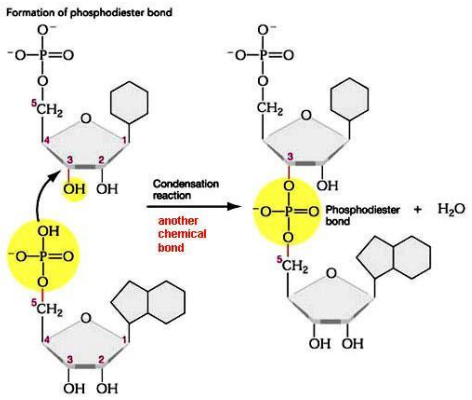
ERRORS:

✗ *"RNA Polymerase carries out proofreading"*

✓ **Only DNA polymerases do this when carrying out DNA replication.**

✗ *"Phosphodiester bonds are formed between adenine and adenine."*

✓ **They are formed between the 3'OH group of the last nucleotide and the 5' phosphate group of the incoming nucleotide. Adenine is the base.**



✗ *"RNA Pol transcribes the STOP codon, which terminates transcription."*

✓ **It may not be the stop codon, and it definitely does not have a role in transcription!**

(ii) Explain why one of the strands of DNA is **not** transcribed. **[Gene Exp, KU-2]** [2]

1. (a) The non-template strand has a **different nucleotide sequence**, which, if expressed, would **code for a different sequence of amino acids**.

   (b) This would result in a **different 3D conformation** of the protein, which would result in a **non-functional protein**.

2. mRNA is single stranded, so only one template strand is needed.

3. (a) The promoter is at the 5' end of the non-template strand…

   (b) hence impossible to read the non-template strand from 5' to 3'.

Actinomycin D and cycloheximide are drugs used in the treatment of chronic leukemia and are involved in the inhibition of β-globin synthesis.

Fig. 4.2 shows the results obtained when each drug is added to immature red blood cells in separate experiments. The thickness and intensity of the bands are an indication of the relative amount of β-globin mRNA or β-globin protein present.

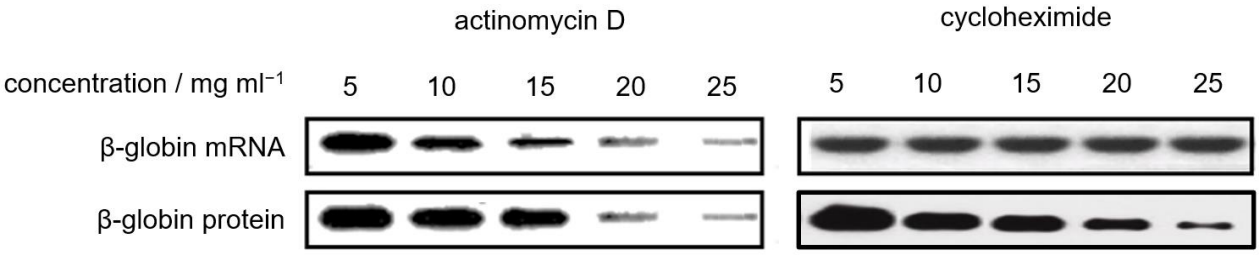


Fig. 4.2

(b) (i) State the process in β-globin synthesis that is inhibited by each drug.

[OCGE in Euk, HI-1] [2]

actinomycin D transcription

cycloheximide translation

(ii) Justify your answer to **(b)(i)**. **[OCGE in Euk, HI-2]** [4]

1. As concentration of actinomycin D **increases from 5 to 25 mg ml^{-1}**, **both** the amount of β-globin mRNA and β-globin protein **decreases**.
2. This shows that **less mRNA** templates were **produced for translation**.
3. As concentration of cycloheximide increases from **5 to 25 mg ml^{-1}**, amount of **β-globin mRNA remains the same** while amount of **β-globin protein decreases**.
4. ***Idea that*** This shows that **mRNA** that was produced was **not used for translation**.

[Total: 11]

5. Scientists have produced structures known as virosomes, which are used in certain vaccines.

Virosomes do **not** cause disease.

Fig. 5.1 is a diagram of a section through a virosome used in some vaccinations to protect against the virus which causes influenza.

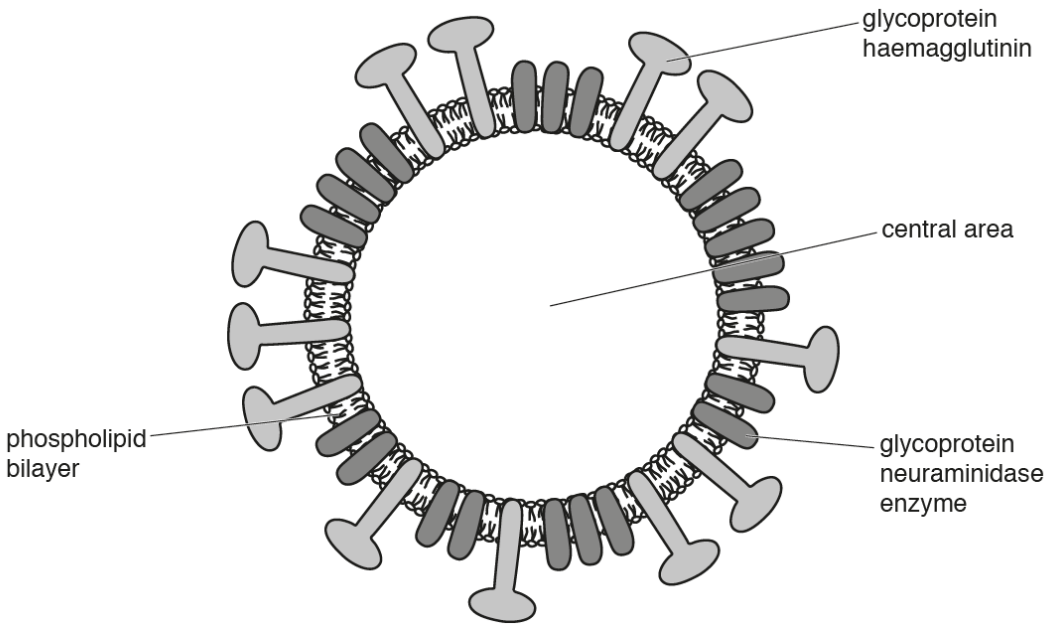


Fig. 5.1

(a) Explain why the virosome does **not** cause disease. **[Virus, HI-2]** [3]

1. It has **no RNA / DNA / genetic material** (NOTE: Influenza contains RNA, not DNA)
2. **No viral proteins** can be coded for / no **new viruses can be assembled.**
3. ***Idea that*** host cell / tissues not damaged, hence no disease.

ERRORS:

✗ *"The virosome cannot infect the host cell."*

✓ **Since it contains HA, receptor-mediated endocytosis can occur, but nothing will enter the cell.**

(b) New strains of the influenza virus render existing vaccines ineffective.

Fig. 5.2 shows the structure of haemagglutinin and the positions of amino acids where changes are frequently observed in antigenic variants of the virus.

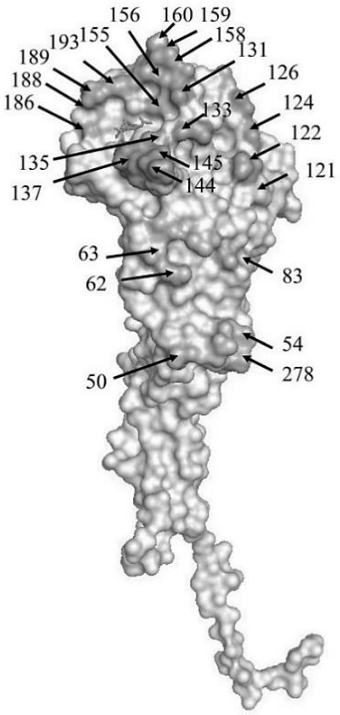


Fig. 5.2

(i) Identify the type of antigenic change that results in the different variants of the influenza virus. **[Virus, KU-1]** [1]

- **Antigenic drift**

(ii) Explain how features of the influenza virus result in antigenic variants of the virus. [3] **[Virus, KU-2]**

1. RNA-dependent **RNA polymerase lacks proofreading ability** so any errors are not corrected.
2. RNA-dependent **RNA polymerase** has a **high error rate**
3. **Single stranded RNA** genome, so there is no complementary/backup strand to act as template for repair.
4. **[compulsory]** Leads to changes in nucleotide sequence / **mutations** in the viral genome, thus changing the **amino acid sequence** of the HA molecule.

ERRORS:

✗ *"The changed HA can now bind to more host cells / more types of receptors."*

✓ **Changes in HA that result in antigenic variants cause them to be a different antigen to antibodies. They can still bind to the same receptors on the same epithelial host cells.**

(c) Distinguish between the reproductive cycles of HIV and the lambda phage.

[Virus, HI-2] [4]

feature	HIV	lambda phage
1. host cell	$CD4^+$/ Helper T cells / macrophages	Bacterial cells / *E. coli*
2. protein involved in attachment to host cell	gp120	tail fibres
3. entry mode	**Fusion** of HIV envelope with cell surface membrane	**Injects DNA** into cytosol
4. structures that enter the host cell	Nucleocapsid + RNA enters host cell	Only DNA enters host cell
5. fate of capsid	Capsid is digested in the host cell	Capsid is left outside the host cell
6. reverse transcription before integration	Viral RNA is reverse transcribed before it is integrated as a provirus	Viral DNA is not reverse transcribed
7. replication of viral genome	Viral RNA needs to be reverse transcribed into DNA before it can be replicated	Viral DNA can be replicated directly
8. excision of genome from host	Provirus is not excised	Viral DNA is excised when induced
9. host genome degraded	Does not cause host genome to be degraded	In the lytic mode, the host genome is degraded
10. release of viruses	**host cell is lysed** to release viruses	HIV **buds off** the host cell

ERRORS:

✗ *"HIV uses reverse transcriptase while lambda phage uses host DNA polymerase to replicate its genome."*

✓ **Both viruses integrate their DNA (only after reverse transcription for HIV) into the host genome and replicate along with the host genome using DNA polymerase.**

✓ **This question requires only differences.**

✓ **Compare not the structure of the viruses, but their phases.**

[Total 11]

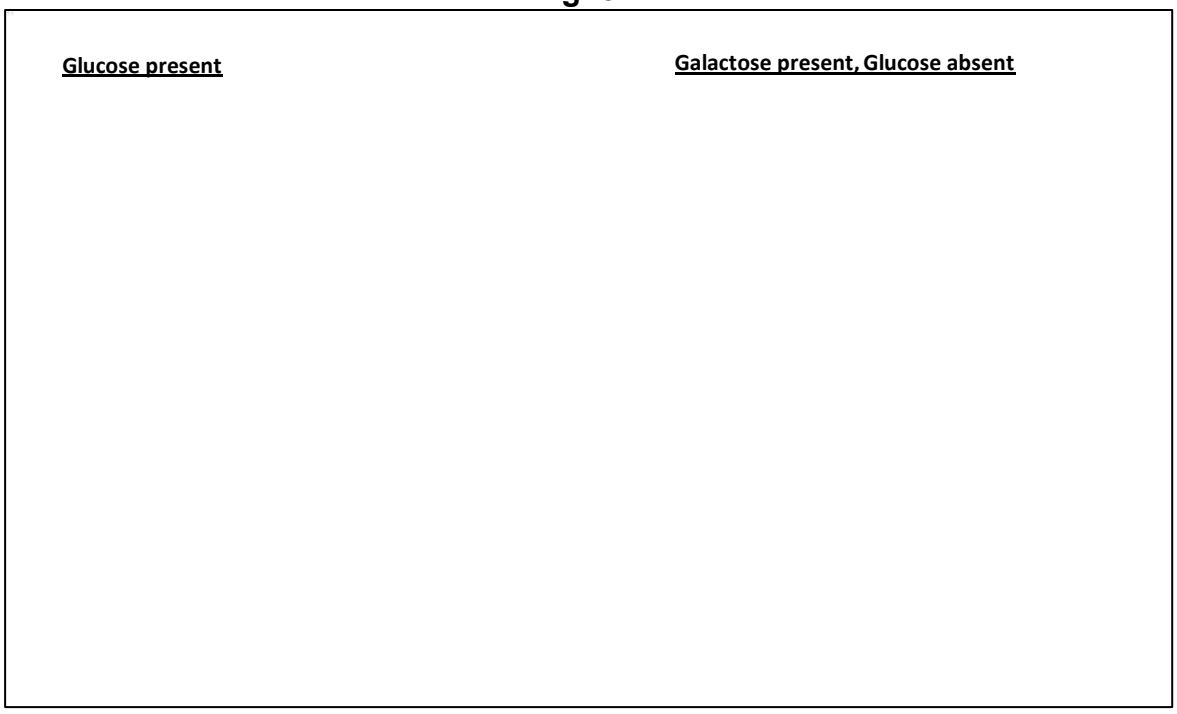
6. Yeast cells are unicellular eukaryotes that respond to the presence and absence of different sugars by switching genes on or off. One example of this is summarised in Fig. 6.1.

If glucose is **present**, a sequence of events occurs.
- Yeast cells metabolise glucose using constitutively expressed enzymes.
- Mig1 transcription factor (**A**) binds to promoter **B**.
- This stops transcription of gene **C**.
- Production of enzyme **D** stops.

If galactose is present and glucose is **absent**, a different sequence of events occurs.
- The Msn2 transcription factor (**E**) binds to promoter **B**.
- This activates transcription of gene **C**.
- Enzyme **D** is produced and helps convert galactose to glucose.

Gene **F** codes for the Mig1 transcription factor, **A**.
Gene **G** codes for the Msn2 transcription factor, **E**.

Fig. 6.1



(a) (i) With reference to Fig. 6.1, identify **one** letter corresponding to: **[OCGE Prok, HI-1]** [3]

a structural gene **C**

a control (regulatory) sequence **B**

a repressor molecule **A**

NOTE
✓ **Genes F and G are regulatory genes.**
Regatory sequences are not genes i.e. non-coding sequences.

(ii) Explain why enzyme **D** is described as inducible. **[OCGE Prok, HI-2]** [2]

1. Enzyme **D** is not made all the time / Gene **C** is switched on only when the enzyme is needed.

2. This is when **glucose is absent & galactose is present** / Msn2 binds to the promoter of Gene **C**.

ERRORS:

✗ *"Gene C can be switched on and off."*

✓ **Any gene can be switched on and off! To explain if a gene is inducible or repressible, start off with the default state first.**

(b) The repression of genes involved in galactose metabolism in yeast is similar to events at the *lac* operon in the bacterium *Escherichia coli*.

Explain when **and** how *E. coli* **represses** the production of proteins needed to metabolise lactose sugar. **[OCGE Prok, KU-2]** [4]

[When]

1. When **lactose is absent**…

2. …there is **no inducer to inactivate the repressor**.

[How]

3. ***Lac I* gene codes for an active repressor protein**

4. The **repressor protein binds to operator** and **prevents RNA polymerase** from binding to the **promoter of the *lac* operon**.

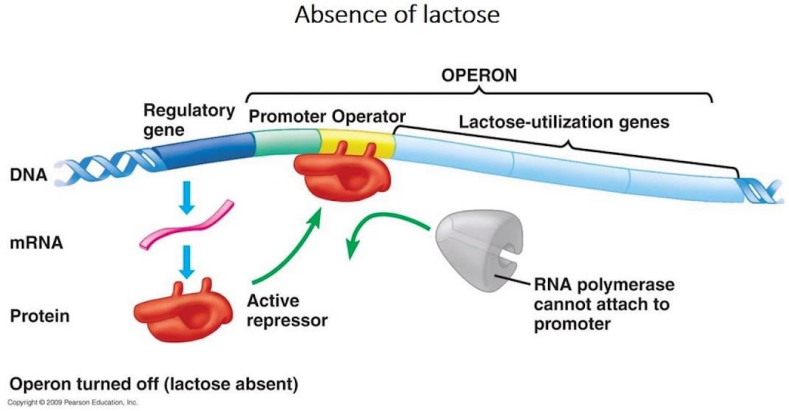
5. The **structural genes** / *lac Z, Y, A* genes are **not transcribed** to produce the proteins that metabolise lactose.

ERRORS:

✗ *"When glucose is present, lactose is not converted to allolactose."*

✓ **Why not? As long as lactose is present, the existing β-galactosidase will isomerise lactose to allolactose. It has nothing to do with glucose.**

✗ **About half of you still think that the repressor protein binds to the promoter.**



[Total: 9]

7. The tiger barb, *Puntigrus tetrazona*, is a South American fish that is popular worldwide as an aquarium fish.

Fig. 7.1 shows the appearance (phenotype) of a normal (wild-type) tiger barb.

Fig. 7.1

- Tiger barbs that show a wild-type phenotype are gold with black stripes.
- Tiger barbs that show an albino phenotype are gold with white stripes.
- In 2012, a fish breeder discovered a tiger barb with a new, transparent, phenotype. This fish had a transparent body and black stripes **(ggBB)**.

The fish breeder crossed the tiger barb showing the new transparent phenotype with a tiger bar showing the albino phenotype **(GGbb)**.

All the F1 offspring were wild-type **(GgBb)**. These F1 offspring were crossed with each other **(GgBb x GgBb)**.

Table 7.1 shows the phenotypes obtained in the F2 generation and the number of fish showing each phenotype.

Table 7.1

F2 phenotype	number of fish
wild-type (gold with black stripes) **G_ B_**	173
albino (gold with white stripes) **G_ bb**	57
transparent with black stripes **gg B_**	58
transparent with white stripes **gg bb**	19

(a) State the approximate whole-number ratio shown by the results in Table 7.1.

[Inherit, HI-1] [1]

- 9 : 3 : 3 : 1

(b) Explain how the results of a chi-squared (χ^2) test would be able to increase confidence in the conclusion in **(a)**. **[Inherit, KU-2]** [3]

1. (At df = 3) If the calculated χ^2 value is lower than the critical value at 5% significance level…
2. …the probability that the deviation of the observed numbers from the expected ratio of 9:3:3:1 being due to chance is **more than 5%** / **0.05**.
3. Hence, the deviation is **due to chance**, so the **observed numbers conform to the expected ratio**.

[**Accept:** reverse argument]

	Significance level (α)							
Degrees of freedom (*df*)	.99	.975	.95	.9	.1	.05	.025	.01
1	-------	0.001	0.004	0.016	2.706	3.841	5.024	6.635
2	0.020	0.051	0.103	0.211	4.605	5.991	7.378	9.210
3	0.115	0.216	0.352	0.584	6.251	7.815	9.348	11.345
4	0.297	0.484	0.711	1.064	7.779	9.488	11.143	13.277
5	0.554	0.831	1.145	1.610	9.236	11.070	12.833	15.086

(c) Explain what the results in Table 7.1 show about the nature of the genes that determine the phenotypes in tiger barbs. **[Inherit, KU-2]** [3]

1. There are **two genes** controlling the two phenotypes.
2. The genes are on **separate chromosomes** / **unlinked** / **assort independently**
3. The genes are **not sex-linked** / **autosomal**.
4. Each gene has **two alleles**. [must be explicitly stated]
5. **White stripes** and **transparent** body is caused by the **recessive allele** of both genes
6. **wild type** / **gold & black** stripes is **dominant** for both genes

(d) Variation in phenotypes can arise as a result of various factors, such as mutations.

The fish breeder concluded that the new transparent phenotype had occurred because of a mutation.

Explain how this conclusion is supported by the phenotypes observed across the three generations. **[Inherit, KU-3]** [2]

1. transparent phenotype reappears in F2…
2. …so it is inherited / passed down / genetic, not environmental.
3. The mutation resulted in the formation of a **recessive allele**…
4. …as shown by the transparent allele being masked by the dominant gold allele in the F1 generation.
5. A new allele was created as a result of the mutation.

[Total: 9]

8. Fig. 8.1 shows the initial stages of insulin signaling in response to elevated blood glucose concentrations.

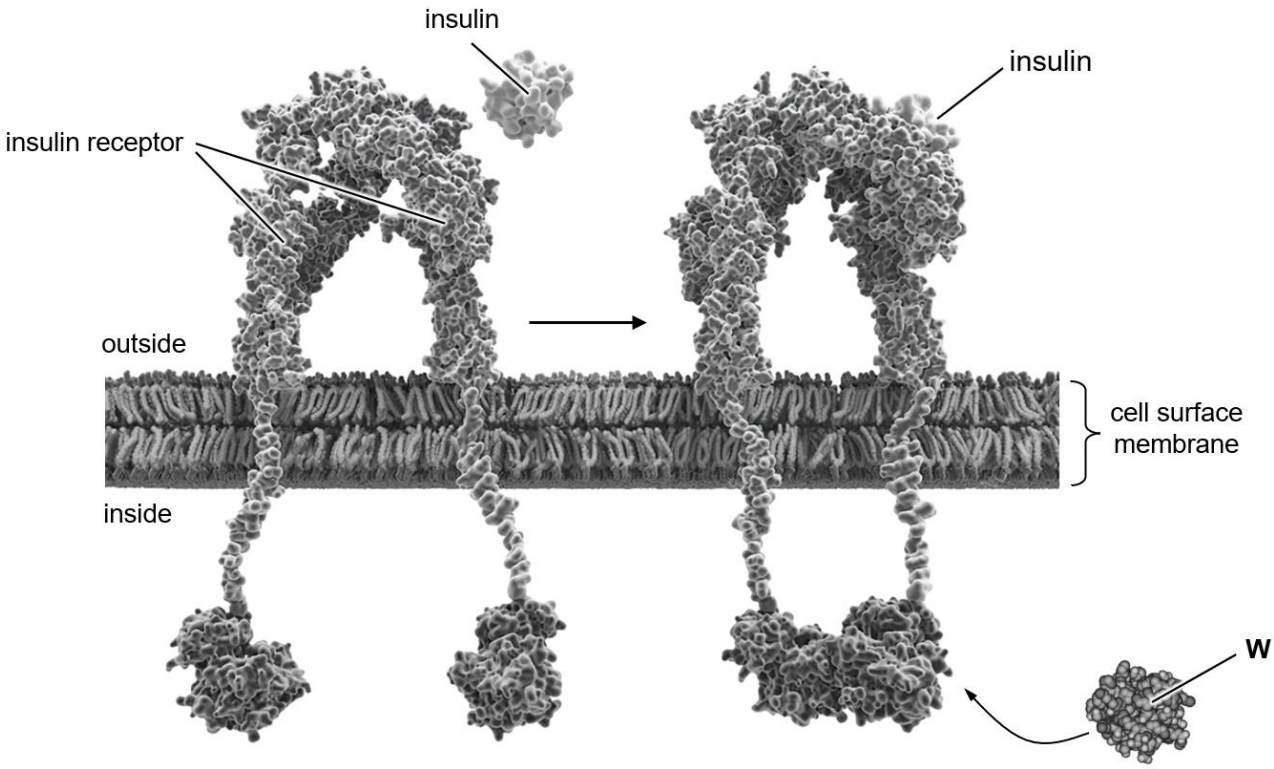


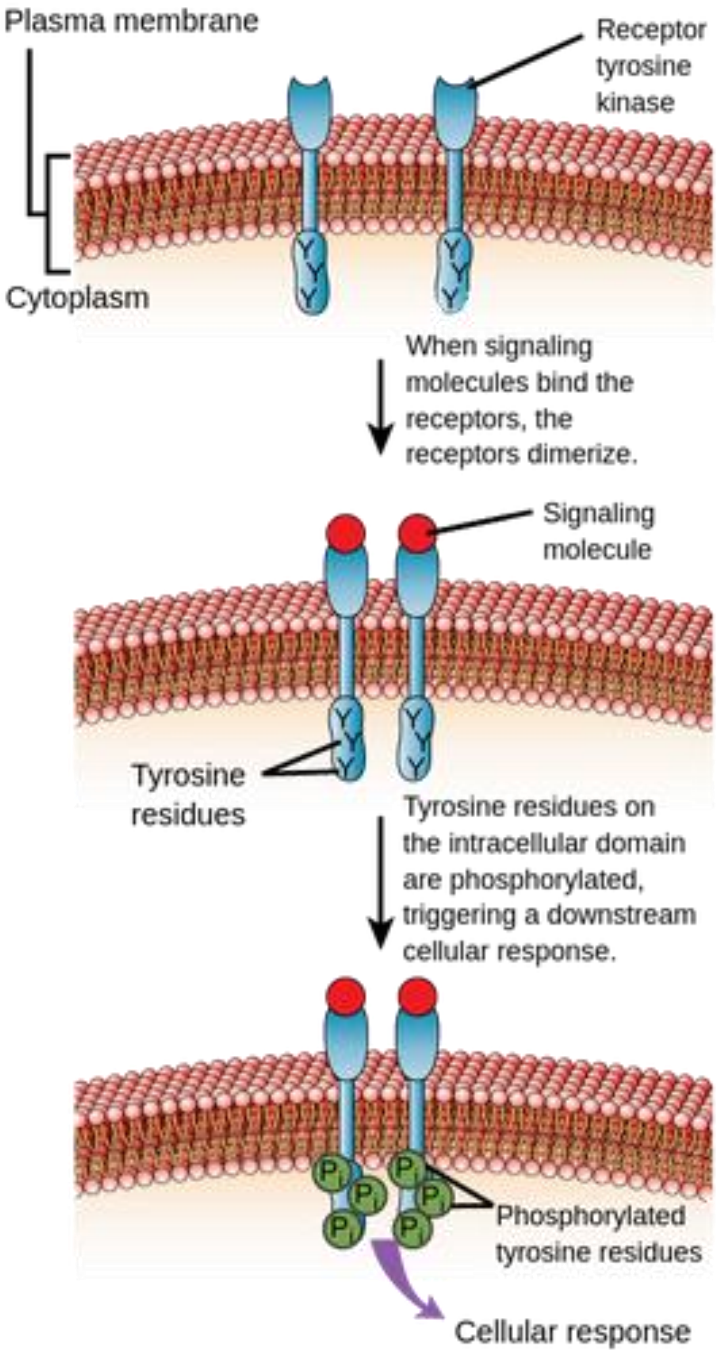
Fig. 8.1

(a) Name the molecule labelled **W** in Fig. 8.1. **[Cell Comm, HI-1]** [1]

- relay protein / relay molecule / insulin receptor substrate

(b) With reference to Fig. 8.1, describe how insulin can trigger a response inside the target cell. **[Cell Comm, KU-1]** [4]

1. **Insulin** binds to the complementary **ligand-binding domain** of insulin receptors.
2. Induces a **conformational change** in the insulin receptors.
3. (Cytoplasmic domains of) the **receptors** **dimerize**.
4. Activates the catalytic **tyrosine kinase** tail of each receptor.
5. **Cross-phosphorylation** of multiple **tyrosine residues** on the tyrosine kinase tail.
6. **Relay proteins** **bind to** phosphorylated tyrosine residues and are **activated** (through phosphorylation)


Plasma membrane
Receptor tyrosine kinase
Cytoplasm
When signaling molecules bind the receptors, the receptors dimerize.
Signaling molecule
Tyrosine residues
Tyrosine residues on the intracellular domain are phosphorylated, triggering a downstream cellular response.
Phosphorylated tyrosine residues
Cellular response

(c) Insulin signaling is terminated through **several** mechanisms to ensure that the effects of insulin are appropriately regulated.

Suggest how insulin signaling can be terminated. **[Cell Comm, KU-2]** [3]

1. **Ubiquitination** of the receptors at their cytoplasmic tails / relay proteins
2. **[receptor]** Triggers **endocytosis of receptors** in endosomes.
3. **[receptor]** **Fusion of endosomes with lysosome** degrades the receptors.
4. **[relay proteins]** **Degradation of relay proteins** by **proteasome**.
5. **Removal of phosphate groups** (by phosphatases) from the tyrosine tails of the receptors / relay proteins to inactivate them.
6. Binding of a protein/molecule that changes conformation of receptor proteins.
7. **AVP**

Reject: (1) mutation, (2) insulin dissociates from receptor

ERRORS:

✗ *"Mutation of the gene coding for receptor / relay protein / IRS".*

✓ **Question is asking for the normal process to terminate insulin signalling.**

✗ *"Glucose level drops, so no insulin release. / Insulin dissociates from receptor"*

✓ **Question is asking for termination, means after the cell has responded to the signal (i.e. insulin), how to stop the transduction pathway from continuing.**

In glucagon signalling, an enzyme called phospholipase C is activated. This enzyme converts a membrane phospholipid called PIP_2 to two different second messengers, DAG and IP_3.

Fig. 8.2 shows the formation of DAG and IP_3 from PIP_2.

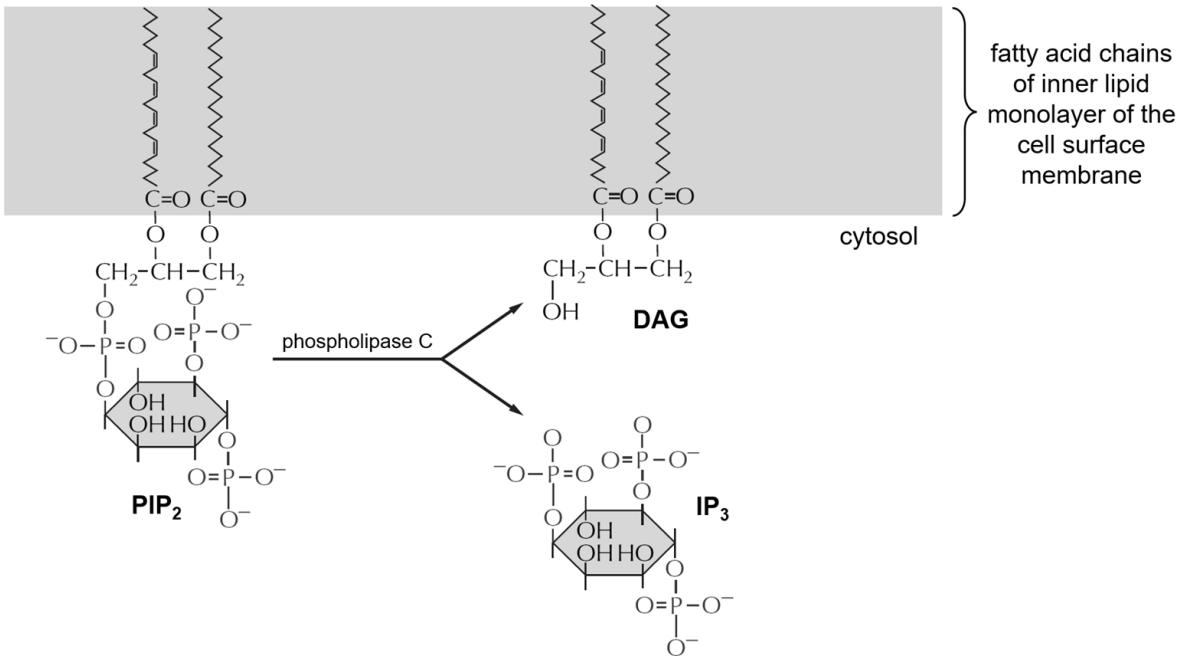


Fig. 8.2

(d) Apart from size, state one similarity and one difference between DAG and IP_3. [2]

[Cell Comm, HI-1]

similarity — **non-protein** in nature

difference — DAG is lipid-soluble / hydrophobic / amphipathic / membrane-bound, while IP_3 is water-soluble / hydrophilic / charged / cytosolic.

ERRORS:

✗ *"Both have hydroxyl group" "IP3 has phosphate group but not DAG" "DAG has fatty acid chain but not IP3"*

✓ **Trivial points.**

[Total: 10]

9. Blind mole rats, *Spalax ehrenbergi*, are mammals that live in groups in underground burrows. They are blind and communicate with each other through sound and scent. Males make a purring call when attempting to persuade females to mate with them.

In Israel, the mole rats found in different parts of the country **all look identical**. However, there are actually four different populations **with different chromosome numbers**, which **live in different climatic regions**. These are shown in Table 9.1.

The table also shows information about the purring calls used by the males in each population. The calls of the males were analyzed by measuring the number of sound pulses per second, and the frequencies of the sounds that they made.

Table 9.1

population		A	B	C	D
chromosome number		52	54	58	60
climatic region in which population lives		cool and humid	cool and dry	warm and humid	warm and dry
purring call made by males	mean number of pulses per second	21.0	25.3	23.9	23.2
	mean major frequency / kilohertz	595	555	583	562

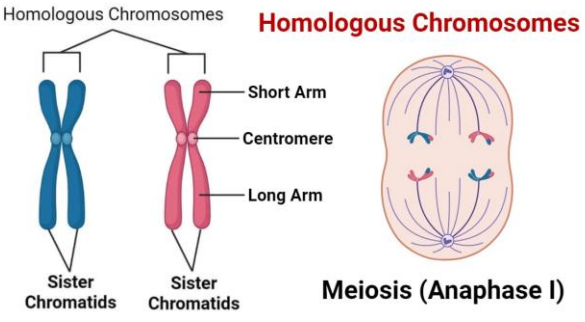
(a) Explain why the chromosome number of each of the four populations of mole rats is an even number. **[Cell cycle, KU-2]** [2]

1. They are **diploid** organisms / **two sets of chromosomes** / **2n**.
2. One chromosome of each pair from each parent / maternal and paternal.
3. **Meiosis** produces **haploid gametes**, which upon **fertilization restores diploid state**.
4. Allows pairing of homologous chromosomes during meiosis I / prophase I / metaphase I.

ERRORS:

✗ *"Chromosome has 2 sister chromatids, hence diploid"*

✗ *"Chromosome has 2 sister chromatids, one from father and one from mother"*



✓

(b) Researchers investigated how female mole rats from each of the four populations responded to purring calls made by males from the same population, and by males from different populations.

A female was placed midway between two loudspeakers, and recorded calls from two males were played to her simultaneously. The researchers noted which loudspeaker the female moved towards. This was repeated with many different females from each population. The results are shown in Table 9.2.

Table 9.2

population	chromosome number	percentage of females preferring the purring call of males from their own population
A	52	79
B	54	77
C	58	77
D	60	44

With reference to Table 9.2, describe the **extent** **to which female mole rats show a preference** for the purring calls of males from their own population. **[Data, HI-1]**
[2]

1. **77-79%** of females in **three** populations / **A-C** prefer calls from their own population.

2. Females in **population D** / population with **2n = 60** have a lower preference at **44%**.

ERRORS:

✗ *"As chromosome number increases from 52 to 60, percentage of females preferring purring calls of males from their own population decrease"*

✓ **The trend here based on chromosome number has no significance. Question asked for the EXTENT of preference**

✗ *"close to 80%" "above 75%"*

✓ **Vague. Cute ACCURATE data**

Discuss: you need to give both side of the argument. In this case…
Yes, they are different species because…..
No, they are the same species because…..

(c) With reference to the data in both Table 9.1 and Table 9.2, **discuss** whether these four populations of mole rats should be classified as different species. **[Evo, HI-2]** [5]

[Argument for classifying as different species] – at least 1

1. These four populations have **different chromosome numbers**.

2. Cannot interbreed to form fertile offspring / hybrids are infertile.
3. …since not all chromosomes will be able to pair in meiosis / hybrids may have odd number chromosomes.

4. Geographically isolated / lives in different habitats / climatic regions… **[Reject: ecologically isolated]**
5. …hence likely to be reproductively isolated / unlikely to interbreed.

6. Most females prefer males from their own population, hence tend to breed within their own population.

7. Different frequencies and pulses per second in the purring calls, …

8. …hence behaviourally isolated

[Argument for classifying as same species] – at least 1

9. Some females are willing to mate with males from other populations.

10. Morphologically identical.

[Total: 9]

10. Fig. 10.1 shows the number of people that have been newly infected with the Human Immunodeficiency Virus (HIV) in 2018 across the world and the percentage changes in the number of new infections since 2010.

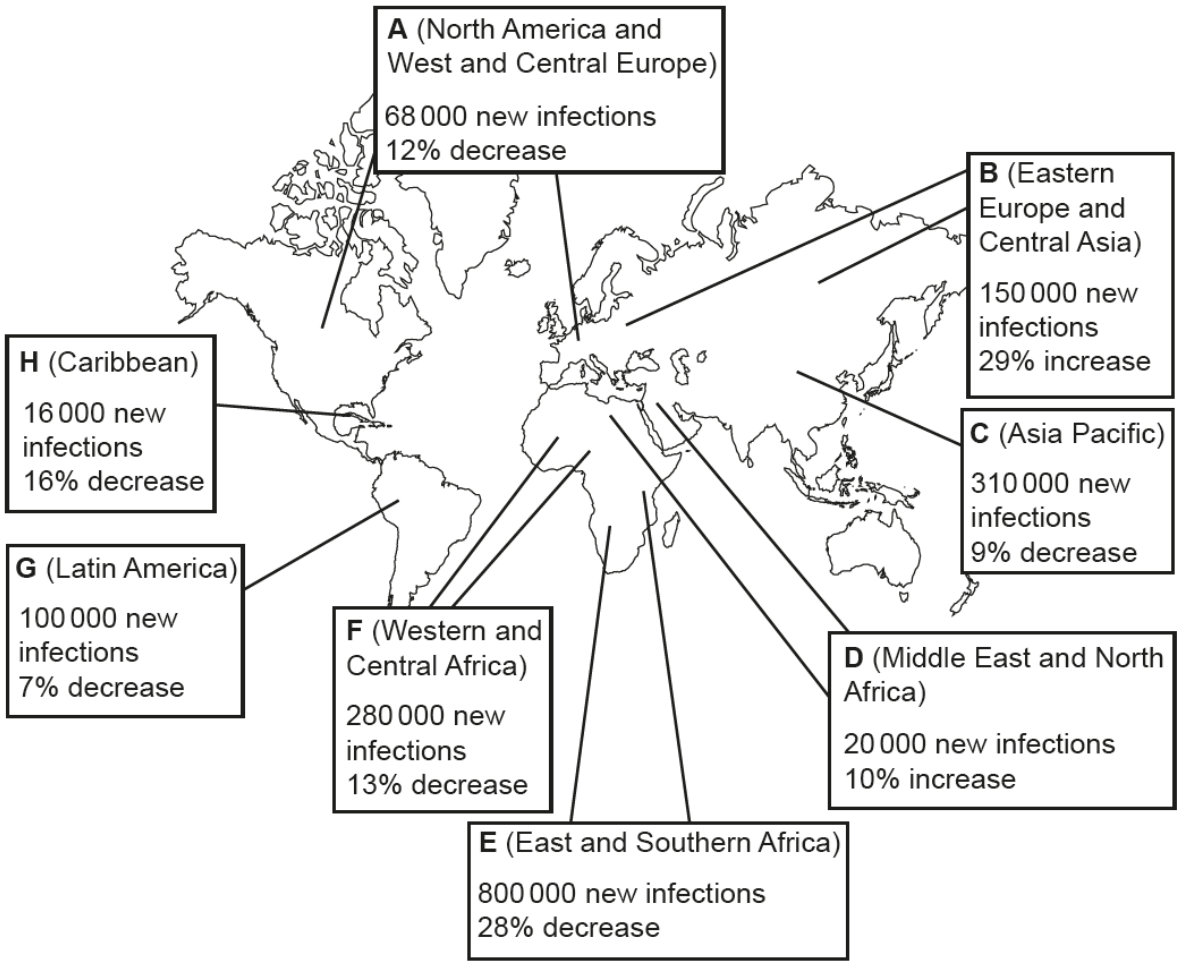


Fig. 10.1

Is the increase/decrease in percentage or the number of new infections more relevant here?

(a) With reference to Fig. 10.1, **describe the change** in new HIV infections across the world between 2010 and 2018.

You may **use the letters** in Fig. 10.1 to identify the regions of the world. [3]

[Data, HI-1]

1. There is a **decrease** in the percentage infections in **all regions except B and D**.
2. **B = 29% increase and D = 10% increase**. / **Biggest percentage increase** in **B** at **29%**.
3. **Biggest percentage decrease** in **E** (East and Southern Africa) at **28%**.
4. **Smallest percentage decrease** in **G** (Latin America) at **7%**.

(b) Using the data for East and Southern Africa (**E**), calculate the number of new infections that was recorded for the year 2010.

Show your working and give your answer to the **nearest whole number**. [2]
[Data, HI-1]

- 800 000 × 100 **[1]**
  100−28

- **1,111,111 [1]**

(c) Explain why people with HIV/AIDS are more likely to develop tuberculosis (TB). [4]
[Immunity, KU-1]

1. TB is an **opportunistic** infection.
2. Caused by the bacterium ***Mycobacterium tuberculosis***.
3. HIV infects and kills **helper T cells**.
4. Weakened immune system.
5. **Less cytokines** secreted by the **fewer helper T cells**.
6. **B cells not activated** to divide and differentiate to become **plasma cells**.
7. **Lesser antibodies secreted to bind to *M. tuberculosis* / TB bacteria**.
8. Hence, lesser phagocytosis of *M. tuberculosis* (due to lesser antibodies bound to the bacteria to label them for phagocytosis)

ERRORS:
✗ *"TB virus"*
✓ **TB is caused by bacteria**

(d) Suggest how climate change can worsen the health of existing HIV-infected individuals. [2]
[Climate change, KU-2]

1. Climate change can **alter the distribution** of **mosquito-borne diseases** / malaria / dengue / ***named disease***
2. Climate change result in increased in temperature, hence increase metabolic rate and reproduction rate of mosquitoes, which in turn increased its population, and the spread of mosquito-borne diseases.
3. **Melting of permafrost** can release **ancient viruses** that can cause diseases.
4. ***Idea that*** Such diseases can be more severe in HIV-infected individuals due to a compromised immune system.
5. ***Idea that*** Climate change-related disasters (e.g. floods) can disrupt the supply chain of HIV medications, leading to interruptions in treatment.
6. **AVP**

[Total: 11]

☺ END OF PAPER 2 ☺