# TAMPINES MERIDIAN JUNIOR COLLEGE

## JC2 PRELIMINARY EXAMINATION

**H2 BIOLOGY** **9744/03**

Paper 3 Long Structured and Free-response Questions **13 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.<br>• Analyse the preamble and/or diagram carefully.<br>• 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?<br>• 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.<br>• Paraphrase the question in my own words.<br>• 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.<br>• Examine the trend of the graph. If appropriate, divide the graph into $\geq 2$ 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)<br>• 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.<br>• Be concise & succinct. Do not write excessively.<br>• 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.<br>• Examine any hint(s) / information in the preamble to suggest biologically sensible ideas. |

## Section A

Answer all questions in this section.

**1.** The movement of blood glucose across the cell surface membrane of mammalian cells involves a family of related proteins called glucose transporters (GLUTs). Different isoforms of GLUT are found in different tissues, as shown in Table 1.1.

Several studies have measured the changes in rates of glucose uptake by different forms of GLUT as the concentration of glucose is increased.

For each type of GLUT, the rate of transport levels off to a maximum value called $\mathbf{V_{max}}$.

The glucose concentration at which the rate of transport is half $\mathbf{V_{max}}$ is defined as the $\mathbf{K_M}$ of the transporter. A lower $\mathbf{K_M}$ is indicative of greater binding affinity of GLUT for glucose, which means that the GLUTs will be saturated at lower concentrations of glucose.

The $\mathbf{K_M}$ for the four forms of GLUT and the effect of increasing glucose concentration of the rate of glucose uptake are shown in Fig. 1.1.

**Table 1.1**

| isoform | function | location |
|---|---|---|
| GLUT1 | basal glucose uptake required to sustain respiration | all cells |
| GLUT2 | act as a glucose sensor | pancreatic β-cells, liver cells |
| GLUT3 | act as a scavenger for cells with high rate of glucose metabolism | neuronal cells |
| GLUT4 | insulin-responsive isoform that translocate to cell surface membrane upon insulin stimulation | skeletal muscle cells, adipose (fat) cells |

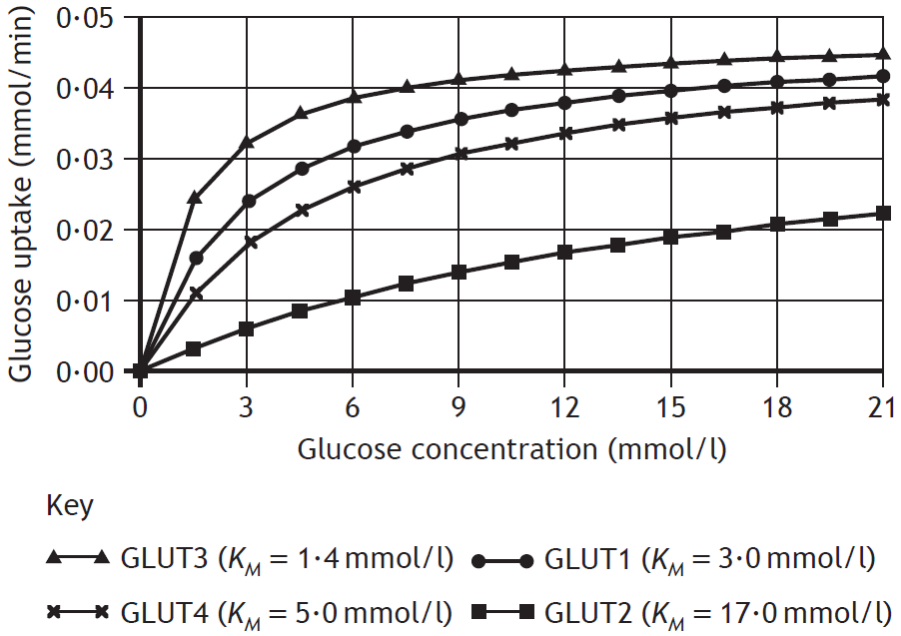



**Fig. 1.1**

**(a)** State the process of glucose uptake by GLUT into a cell. **[Transport, KU-1]** [1]

- facilitated diffusion

**(b)** The rate of glucose transport at a given glucose concentration can be calculated using the formula:

$$V = \frac{V_{max} \times [G]}{K_M + [G]}$$

V = rate of glucose transport
[G] = glucose concentration (mmol/l)

The physiological range of blood glucose concentration in a healthy individual after fasting ranges from approximately 3.9 to 5.5 mmol/l.

With reference to Fig. 1.1, calculate the rate of glucose transport by **GLUT2** when the blood glucose concentration is 5·5 mmol/l. Show your stepwise working and express your answer to 2 significant figures. **[Data, HI-3]** [2]

Rate of glucose uptake at $K_M$ of 17 mmol/l = 0.02 mmol/min (read off from graph)

Hence, $V_{max}$ = **0.02 x 2** = **0.04** mmol/min **[1]**

$V = \frac{0.04 \times 5.5}{17 + 5.5} =$ **0.0098** mmol/ min **[1]**

**[allow ecf for incorrect $V_{max}$ used]**

rate of glucose uptake = **0.0098** mmol/min

***ERRORS:***

✗ *"Vmax = 17 x 2 = 34"*

✓ **Please read the definition of Km and Vmax carefully:**
***The glucose concentration at which the rate of transport is half Vmax is defined as the Km of the transporter.***

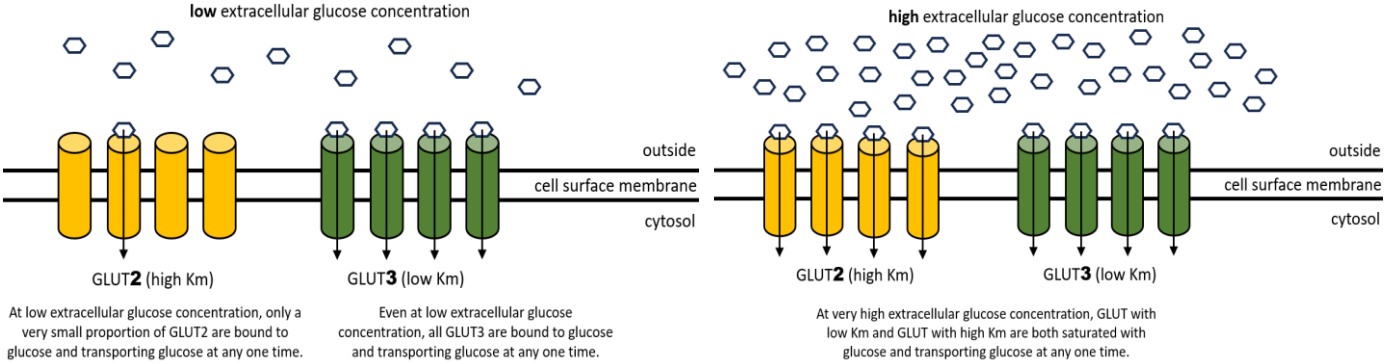
**(c)** In pancreatic β-cells, GLUT2 acts as a glucose sensor. When blood glucose increases, glucose enters the β-cells through GLUT-2, leading to an increase in intracellular glucose concentration. This increase in glucose concentration triggers a series of events leading to the release of insulin, which helps to lower blood glucose levels. The amount of insulin released is dependent on the amount of glucose taken into the β-cells.

**(i)** Explain why it is important that pancreatic β-cells contain the isoform (GLUT2) that has the **highest $K_M$**. **[Cell Comm, KU-3]** [2]

1. ***Idea that*** Highest $K_M$ would mean that GLUT2 requires a high concentration of glucose before becoming saturated / ***reverse argument***

2. ***Idea that*** Allows β-cells to sense changes in blood glucose concentrations over a wide range, from low to high. **[reject: allow high glucose conc. to enter]**

3. ***Idea that*** Enables β-cells to release the right amount of insulin in response to a range of elevated blood glucose levels.




***ERRORS:***

✗ *"Ensure that GLUT2 only transport glucose at high blood glucose concentrations."*

✗ *"Ensures that insulin is released only when blood glucose is very high."*

✓ **GLUT2 can transport glucose <u>across a wide range</u> of blood glucose concentrations, hence releasing appropriate amount of insulin proportional to the blood glucose concentration.**

- **At lower elevated blood glucose, lesser glucose is transported into the β-cell, which leads to the secretion of little insulin.**
- **At higher elevated blood glucose, more glucose is transported into the β-cell, which leads to the secretion of more insulin.**

**A low Km GLUT would allow a lot of glucose to be transported into the β-cell even at lower elevated blood glucose. This will lead to an excessive insulin being secreted that leads to hypoglycemia (overly low blood glucose levels) that can be fatal.**

**(ii)** Fig. 1.2 shows the series of events that happen after glucose molecules enter the β-cells through GLUT2.

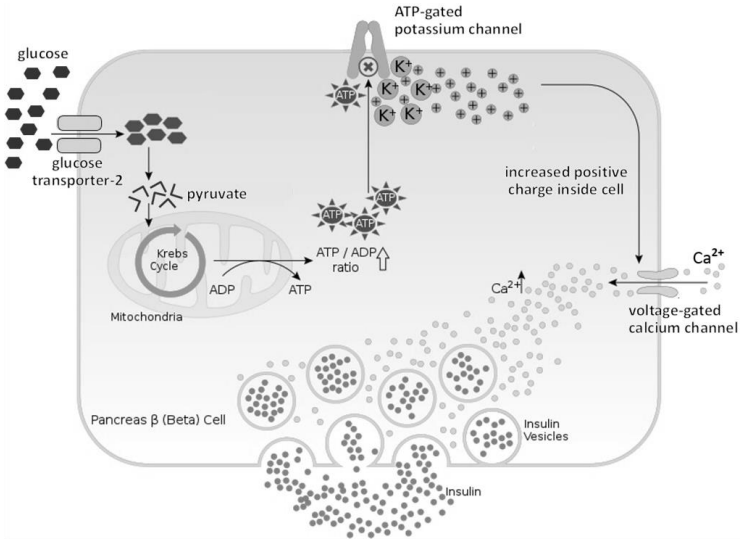



**Fig. 1.2**

Outline the roles of the cell surface membrane in the release of insulin after the entry of glucose into the β-cell. **[Transport, HI-2]** [3]

1. **ATP–gated $K^+$ channel** (on the cell surface membrane) **closes** when **ATP increases**, **preventing $K^+$ from diffusing out**.

2. **Voltage–gated $Ca^{2+}$ channel** (on the cell surface membrane) **opens** when **positive charge ($K^+$) increases in the cell**, allowing **$Ca^{2+}$ to enter the cell**.

3. The **cell surface membrane fuses** with **insulin vesicles** in response to the **increase in cytosolic $Ca^{2+}$** to release insulin via exocytosis.

4. ***Idea that*** hydrophobic nature of phospholipid bilayer prevents ions from diffusing freely across the cell surface membrane

***GENERAL COMMENTS:***

- This question is in your Cell Signaling assignment (paraphrased). Yet, many of you still could not do.
- Have you really learnt from the past tutorial and assignments? There is no point doing new questions if you don't learn anything 😊

***ERRORS:***

✗ *"The cell surface membrane compartmentalizes the cell."*

✓ **Compartmentalization is only for INTERNAL membranes.**

✗ *"Ions are polar.", "Glucose is charged."*

✓ **It's worrisome how, at this point in time, knowing the difference between "polar" and "charged" is still a struggle.**

**(d)** After a meal, blood glucose concentration rises above the normal concentration range (3.9–5.5 mmol/l), which is normally maintained by homeostasis.

With reference to **Table 1.1**, explain how the blood glucose concentration is quickly reduced to normal levels in a healthy person. **[Cell Comm, HI-2]** [3]

1. **All cells** have **GLUT1** to take in **basal amount of glucose** to sustain **respiration**.
2. **Liver cells** have **GLUT2** to take in **excess blood glucose** that are converted to **glycogen** for storage.
3. **Neuronal cells** have **GLUT3** to take in **more blood glucose** due to their **high respiration** rate.
4. **Muscle and adipose cells** have (cytosolic) **GLUT4** that **translocate to the cell surface membrane** upon insulin stimulation…
5. …increases the number of **surface GLUT4** to **increase rate of uptake** of glucose.
6. Excess glucose converted to **glycogen in muscle cells** / **fats in adipose cells**.

***ERRORS:***

✗ *"GLUT3 scavenges for cells with high metabolism"*

✗ *"GLUT3 looks for cells with high metabolism"*

✓ **Can you PLEASE read Table 1.1 properly??**
**"***GLUT 3 act as a scavenger for cells with high rate of glucose metabolism***."**
**It is present on cells that have high metabolism, and due to its high affinity for glucose, it binds to glucose and takes in glucose even at lower blood glucose concentrations. Hence, it scavenges the remaining glucose in the bloodstream.**

✗ *"produce energy", "synthesize energy"*

✓ **RELEASE energy. Energy can only be transformed from one form to another.**
**e.g. *energy in glucose is released via the process of respiration*.**

**GENERAL COMMENTS:**

- A significant number of you repeated the answers for part (c) in part (d). Didn't you find it strange that your answers to (c) and (d) are the same??
- Furthermore, answers to (c) explains how insulin is released. It does not explain how blood glucose conc. is quickly reduced to normal levels, which is required by (d).

**(e)** Mature red blood cells do not contain mitochondria and have relatively low energy requirements. Despite this, the cell surface membrane of red blood cells contains more GLUT molecules than other cells with similar energy requirements.

Explain why the cell surface membrane of red blood cells requires large number of GLUT molecules to supply their energy requirement. **[Resp, KU-2]** [3]

1. Only **glycolysis** takes place in RBC, which produces only **net 2 ATP per glucose** molecule.

2. Mitochondria produce the remaining (36) ATP via **Krebs cycle** and **oxidative phosphorylation**, which cannot occur in RBC (no mitochondria).

3. Compared with a cell with similar energy requirement, RBC would hence require (38÷2 = 19 times) **more glucose to produce the same amount of ATP**

4. ***Idea that*** RBC need more GLUT to take in more glucose to compensate for incomplete glucose oxidation.

***ERRORS:***

✗ *"RBC transports glucose"*

✓ **An undiscovered role of RBC currently discovered by you?** 😊

✗ *"RBC requires energy to move through the bloodstream"*

✓ **The heart will be very sad to know that its nonstop pumping of blood is totally ignored by you.**

Diabetes mellitus currently affects at least 2.5 million people in the UK and is a condition in which the body is unable to maintain a normal blood glucose concentration. There are two forms of diabetes.

Type 1 diabetes:
- an autoimmune condition where the immune system destroys pancreatic β-cells.

Type 2 diabetes:
- develops later in life when the body does not respond to insulin.
- represents approximately 85–90% of cases.

**(f)** Pancreatic maltase is an enzyme that completes the digestion of starch in humans. Molecules of pancreatic maltase are bound to the microvilli of epithelial cells in the small intestine.

Acarbose is a drug used in the treatment of type 2 diabetes. Molecules of acarbose have a very similar shape to that of the substrate for maltase.

Suggest how acarbose can be used to treat people with type 2 diabetes. **[Enz, KU-2]** [3]

1. Acarbose is a **competitive inhibitor** of maltase.

2. **Competes** with **maltose** [**reject:** starch] for the **active site** of maltase.

3. Reduces the rate of enzyme-substrate complex (maltase-maltose) formation.

4. **Slows down** [**reject:** stops] the **rate** at which **maltose** [**reject:** starch] is **hydrolyzed** to **glucose**.

5. **Less glucose absorbed** (per unit time) through the intestine **into the bloodstream** (hence minimizing rise in blood level).

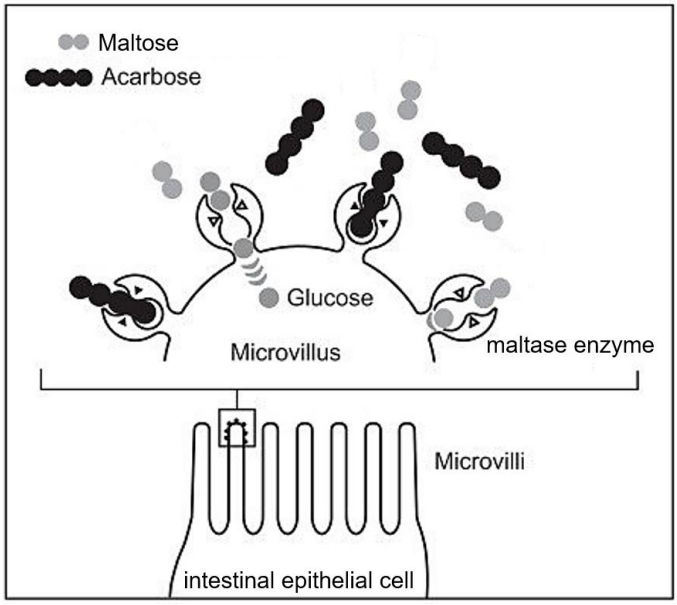
***ERRORS:***

✗ *"Acarbose has similar shape to the active site of maltase"*

✓ **Similar shape ≠ complementary shape**

**GENERAL COMMENTS:**

- **Starch** —?→ **?** —?→ **glucose**



**(g)** Table 1.2 presents the results of an experiment comparing rates of glucose production by a group of people with type 2 diabetes and a control group without the condition, during 23 hours of fasting.

**Table 1.2**

| | rate of glucose production per unit body mass / μmol $kg^{-1}$ $min^{-1}$ | | significance level |
|---|---|---|---|
| | patients with type 2 diabetes | control group | |
| total glucose production | 11.1 ± 0.6 | 8.9 ± 0.5 | $p < 0.05$ |
| glucose from hydrolysis of glycogen in the liver | 1.3 ± 0.2 | 2.8 ± 0.7 | $p < 0.05$ |
| glucose from gluconeogenesis | 9.8 ± 0.7 | 6.1 ± 0.5 | $p < 0.01$ |

Discuss the conclusions that can be drawn from the data in Table 1.2. **[Data, HI-3]** [5]

1. **Overall more glucose** is produced by a diabetic person at **11.1 ± 0.6** than by a healthy individual at **8.9 ± 0.5 μmol $kg^{-1}$ $min^{-1}$**.

2. **Lesser glucose** produced **from glycogen** by diabetic person at **1.3 ± 0.2** than by healthy person ay **2.8 ± 0.7 μmol $kg^{-1}$ $min^{-1}$**.

3. Likely due to less glycogen formed/stored in liver cells with type 2 diabetes.

4. **More glucose** is produced **from gluconeogenesis** by diabetic person at **9.8 ± 0.7** compared to in healthy individual at **6.1 ± 0.5 μmol $kg^{-1}$ $min^{-1}$**.

5. Gluconeogenesis is the production of glucose **from amino acids / proteins**.

6. Likely a mitigating response to the lack of glucose uptake in people with type 2 diabetes.

7. **$p < 0.05$** for all 3 comparisons, hence the **differences** are **significant** and not due to chance.

8. **Small standard deviation**/error/variation (±) indicate the data points in each set are close to the mean value, suggesting **high accuracy** / reliability.

   **[ACCEPT: data cited without standard deviation]**

***ERRORS:***

✗ *"Since $p < 0.05$, the results/values/data are significant."*

✓ **What EXACTLY is significant?**

**(h)** In 2010, scientists investigated the use of induced pluripotent stem cells (iPS cells) to treat type 1 diabetes in mice. The scientists used four transcription factors to reprogramme skin cells into iPS cells. The scientists then stimulated the *in vitro* differentiation of iPS cells into pancreatic β-cells.

The scientists set up three experimental groups:

- Group **A**: 30 mice with type 1 diabetes received pancreatic cell transplants derived from iPS cells.
- Group **B**: 30 mice with type 1 diabetes were left untreated.
- Group **C**: 30 mice without diabetes were left untreated.

The scientists measured the blood glucose concentration of all the mice on a weekly basis for 12 weeks.

The results the scientists obtained are shown in Figure 1.3.

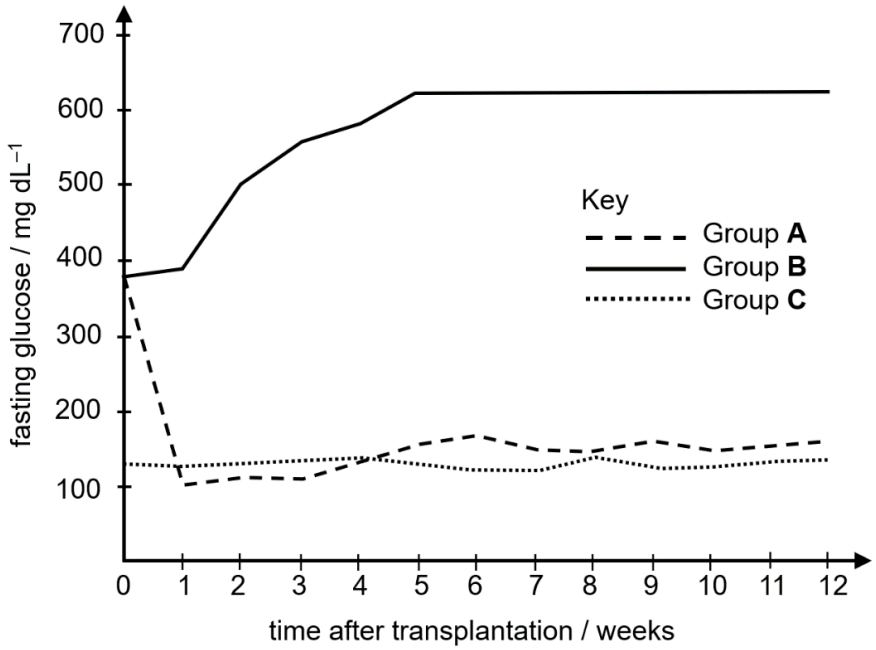



**Fig. 1.3**

With reference to Fig. 1.3, evaluate the use of iPS cells to treat type 1 diabetes in **humans**. **[Data, HI-2]** [3]

**[Argument for effective]** **– either point 1 or 2 must be mentioned**

1. **Effective**, since glucose level of **group A** mice is **comparable** (between 100 to 160 mg/dL) to that of **group C** (~120 mg/dL) **one week after transplantation**.

2. **Effective**, since glucose level **remains lower in group A** (~160 mg/dL) **than group B** (~630 mg/dL) **throughout the 12 weeks**.

3. Since mice and humans are **both mammals**, treatment could be effective on human.

**[Argument for ineffective]**

4. Experiment done on mice, hence whether the results can be duplicated in humans in inconclusive.

5. Only shows results for 12 weeks, hence **long-term effects** (beyond 12 weeks) **not known**.

**(i)** Prolonged exposure to elevated blood glucose can cause glucose to be bound to haemoglobin to form glycated haemoglobin, in a process called glycation.

- The binding between glucose and haemoglobin is irreversible.
- The formation of glycated haemoglobin occurs continuously throughout the lifespan of the red blood cell (approximately 120 days).
- The proportion of haemoglobin that is glycated depends upon the age of the red blood cells and the blood glucose concentration.

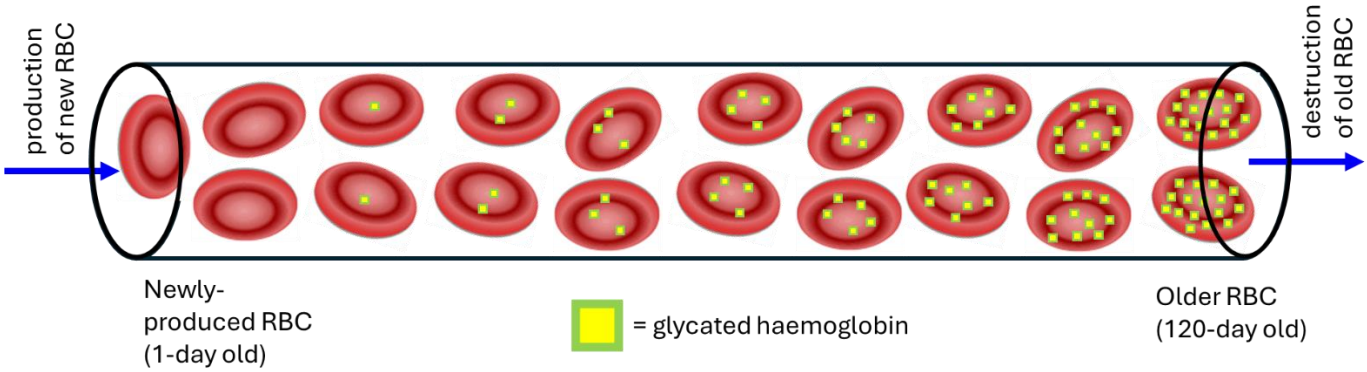
The proportion of glycated haemoglobin is expressed as a percentage of total haemoglobin and can be determined from a blood test. The results of the test can be used to monitor how well blood glucose concentration is being controlled in people with diabetes.

People with diabetes are only tested three times each year for glycated haemoglobin.

Using the information given, suggest why it is unnecessary to carry out this test more than three times each year to assess how well blood glucose concentrations are being controlled. **[Data, HI-3]** [3]

1. Test is done **every 4 months** (120 days), which is the **lifespan of RBC**.
2. ***Idea that*** Since lifespan of RBC is 120 days, the subsequent test would reflect **new glycation** events in **new RBCs not measured in the previous test**.
3. ***Idea that*** Since glycation is **irreversible** and **continuous**, the proportion of glycated Hb in a blood sample **reflects the average blood glucose concentration over the past 4 months**.
4. ***Idea that*** Testing more frequently would not provide significantly different results, as it takes time for glucose to bind to Hb (glycated only upon prolonged exposure to elevated glucose).

**[Total: 28]**




***ERRORS:***

✗ *"New RBC are produced three times in a year."*

✓ **New RBC are produced every millisecond and old RBC are also destroyed every millisecond. At any one time in the blood, there are RBC with age ranging from 1-day old to 120-day old.**

**2.** Keratin is the structural protein in feathers of birds. Keratin polypeptides are composed of a high proportion of cysteine amino acids, which have sulfur-containing R groups.

Keratin polypeptides are tightly bundled to form filaments. The two main types of keratin in feathers are α-keratin, which consists of many α-helices, and β-keratin, consisting of many β-pleated sheets.

**(a)** Keratin can be classified as α-keratin or β-keratin based on a study of protein structure.

State the level of protein structure used to **classify** the protein as α-keratin or β-keratin. **[Biomol, KU-1]** [1]

- Secondary structure

**(b)** Proteases are a group of enzymes that hydrolyse proteins into smaller peptides by breaking peptide bonds between amino acids. **Specific proteases recognize specific amino acids** and cleave the bond between them.

Suggest **two** reasons why it is possible for a specific protease to hydrolyse different types of protein. **[Enz, KU-2]** [2]

1. ***Ref to*** Induced fit hypothesis, where active site is flexible to bind to amino acids of similar shapes.

2. ***Idea that*** The protease hydrolyses peptide bond between two specific amino acids, which can be present in different types of protein.

3. The protease could be a quaternary protein comprising different subunits with different active sites.

***ERRORS:***

✗ *"Different proteins have the same amino acid sequence but fold differently into different 3D conformations."*

✓ **That is just not possible. Folding is determined by the R groups!**
**The same primary structure would result in the same secondary, tertiary and quaternary (if applicable) structures.**

✗ *"All amino acids are joined by the same peptide bond."*

✓ **Yes, but how would that help distinguish the hydrolysis sites of different proteases? Read the preamble again.**

**(c)** Proteases known as keratinases vary in the extent to which they can hydrolyse keratin. Feathers are not easily degraded (broken down) because keratin is a very stable protein.

Suggest features of keratin structure that contribute to its stability. **[Biomol, HI-2]** [2]

1. Insoluble in water (*Reactions require water. Reacting molecules need to be water-soluble*).

2. High proportion of covalent disulfide bonds between polypeptides due to many cysteine residues

3. ***Idea that*** Tight packing of keratin polypeptides (fibrous) such that majority of peptide bonds are not accessible to keratinases.

4. **AVP:** Tight bundling of keratin into filaments gives rise to high tensile strength

***ERRORS:***

✗ *"Keratin contains very strong hydrogen bonds, ionic bonds and hydrophobic interactions."*

✓ **These are all weak bonds / interactions.**

**Also, aren't they all present in all other proteins? What makes keratin so stable then?**

Keratinases are used to degrade the large quantities of waste feathers from chickens and turkeys that are processed in the food industry. The products of feather degradation can be used in animal feed.

Scientists investigated whether three different keratinases, K12, A22 and P3, were suitable as industrial enzymes. These enzymes were extracted from three different soil bacteria.

The effects of temperature and pH on the activity of each keratinase were investigated. The results are shown in Fig. 2.1.

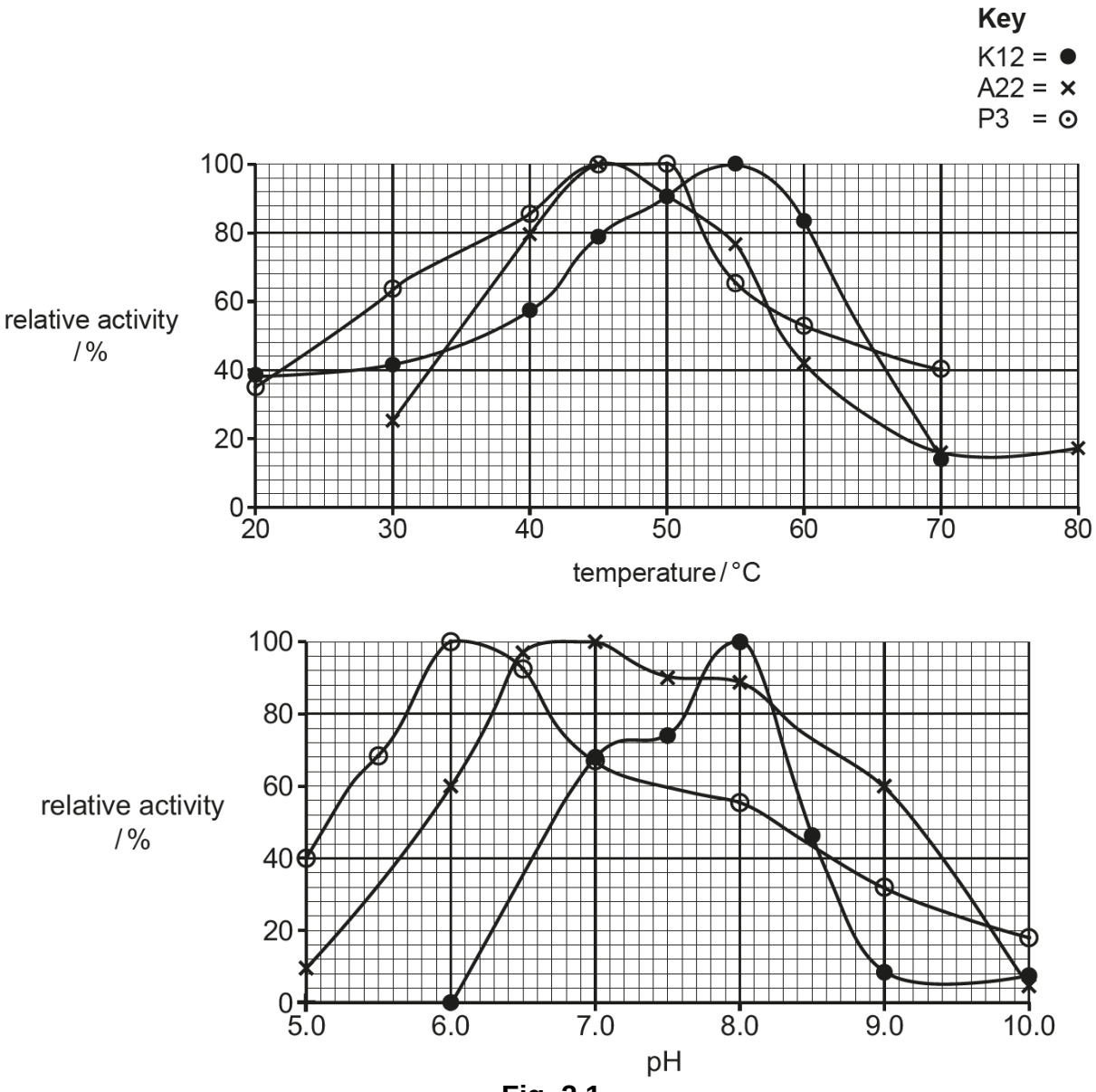



**Fig. 2.1**

**(d)** A food industry decided to use these three keratinases together to degrade large quantities of waste feathers.

Deduce a combination of temperature **and** pH that will produce the highest relative activity. **[Data, HI-1]** [1]

temperature **45–51°C** pH **8.0**

***COMMENTS:***
**While the activity of P3 is lower than at pH 7.0, the average relative activity of all 3 enzymes mixed together is highest at pH 8.0**

**(e)** To degrade feather waste, it is advantageous to use keratinases that show **at least 60%** relative activity in conditions where **temperature and pH can vary widely**.

With reference to Fig. 2.1, name the keratinase with a relative activity of at least 60% that has: **[Data, HI-1]** [2]

- the widest working range of temperature **P3 (27.5°C, from 29°C to 56.5°C)**
- the widest working range of pH **A22 (3.0 units, from pH6 to pH9)**

**(f)** Temperature and pH considerations have a significant impact on the detergent industry. Detergents that are used for washing clothes have an alkaline pH. These detergents contain proteases to remove stains from clothes.

Scientists reported that K12, A22 and P3 could be suitable for use in the detergent industry.

With reference to Fig. 2.1, evaluate the suitability of the three keratinases to be used in the detergent industry. **[Data, HI-2]** [3]

1. **All 3 keratinase** have a **wide range of working temperatures** between **20.5°C to 27°C** where its **activity is at least 60%**.
2. **A22** is the **most suitable** since it has the **widest working range of alkaline pH** from **7.0–9.0** where its **activity is at least 60%**.
3. **K12** is **not as suitable**, as it has a **narrower alkaline pH range of pH 7.0 to 8.4** despite reaching 100% activity at pH 8.
4. **P3** is the **least suitable** since its working alkaline pH range is only from **7.0–7.5**.

| keratinase | temperature range with at least 60% relative activity / °C | **alkaline pH** range with at least 60% relative activity |
|---|---|---|
| K12 | 41–63 (22) | 7.0 – 8.4 (1.4) |
| A22 | 36.5 – 57 (20.5) | 7.0 – 9.0 (2.0) |
| P3 | 29–56 (27) | 7.0 – 7.5 (0.5) |

**[Total: 11]**

**3.** Phytoplankton are microscopic plant-like photosynthetic organisms of the oceans. Fig. 3.1 and Fig. 3.2, show the results of a 50-year study into the annual and monthly mean phytoplankton colour for the North Sea.

The colour intensity of phytoplankton is measured using a colour index. A high colour index value indicates a high chlorophyll concentration.

Phytoplankton are the primary source of food for all oceanic food chains. The colour intensity of the phytoplankton is directly proportional to the water temperature. Phytoplankton is, therefore, a very sensitive indicator of sea temperature change.

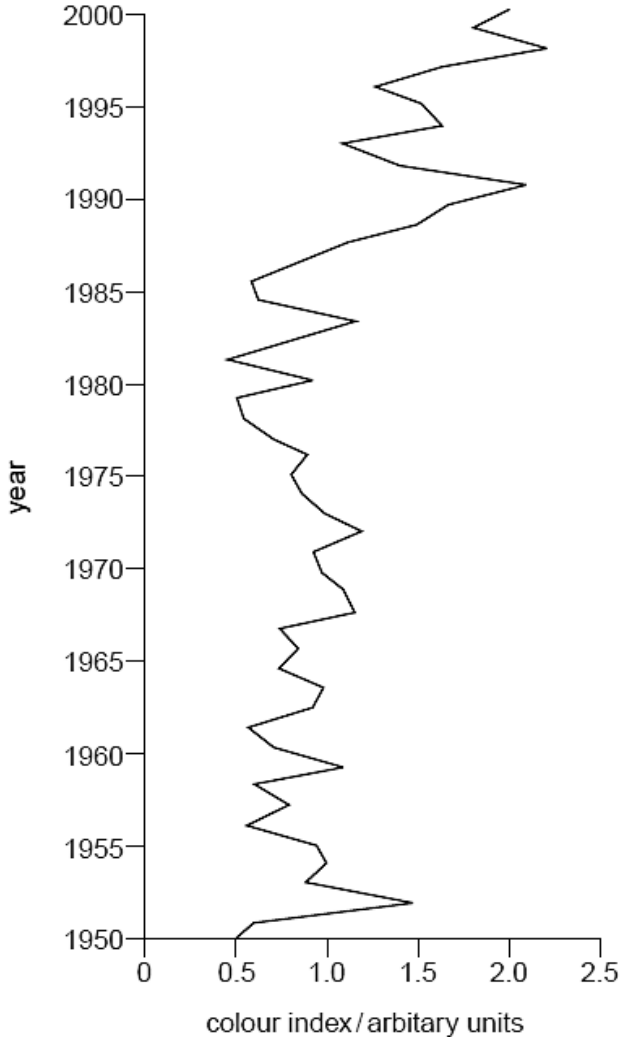



**Fig. 3.1**

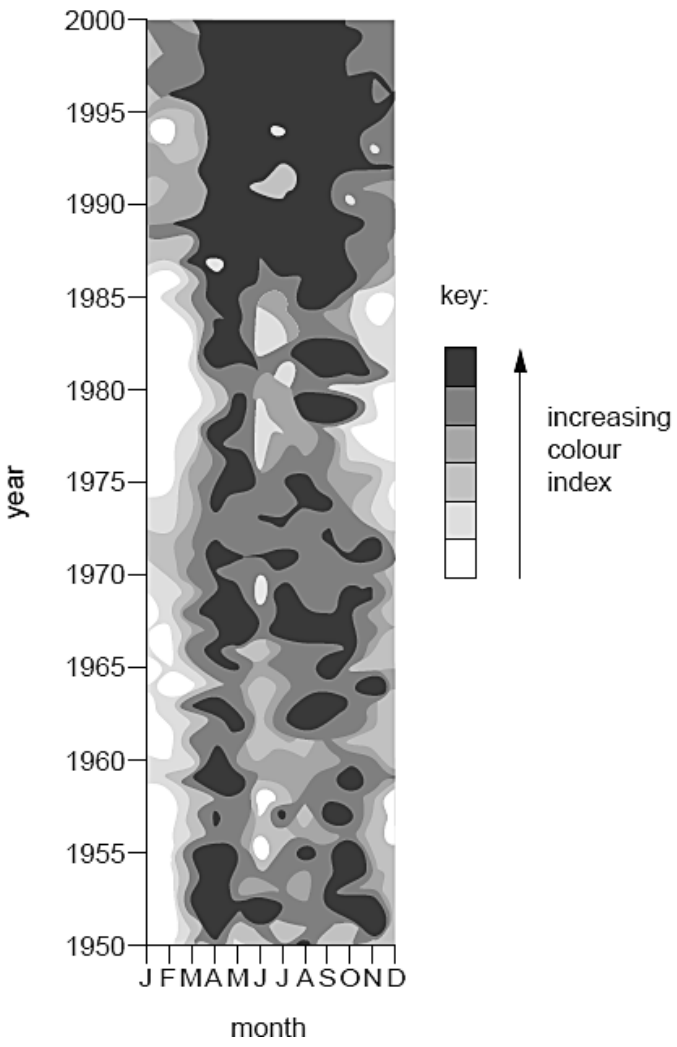



**Fig. 3.2**

**(a)** Suggest why the colour intensity of phytoplankton is directly proportional to the sea temperature. **[Photosyn, KU-2]** [3]

1. The increase in **kinetic energy** results in increased **effective collisions** between **enzymes** (e.g. rubisco) and **substrates**.

2. **Rate** of **fixation of carbon dioxide** / **Calvin cycle increases**.

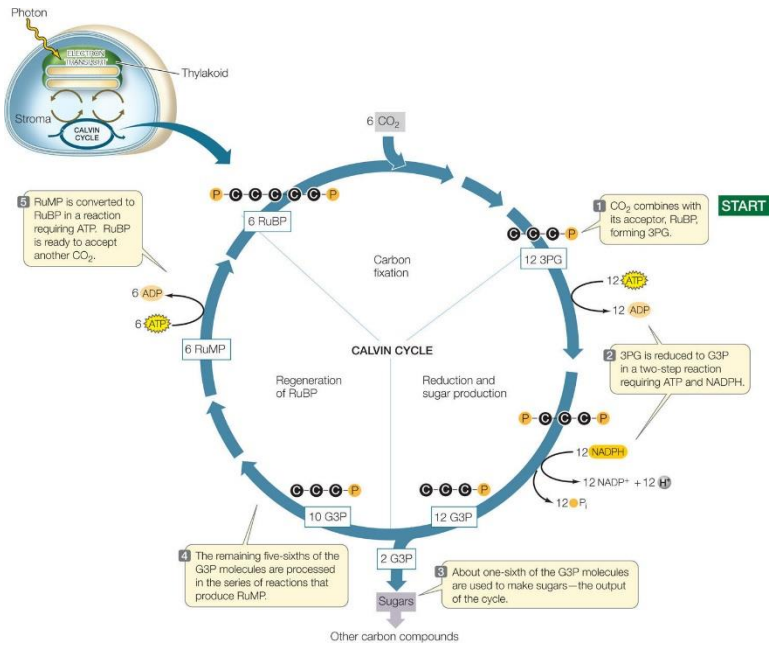
3. **Increased chlorophyll production** (which leads to higher colour intensity) to **increase rate of light-dependent reaction**…

4. …to produce **more ATP** and **NADPH** for Calvin cycle.

***COMMENTS:***
**The Calvin cycle consists of many enzymatic reactions (organized into 3 phases that you know of).**

✗ *"Increase in sea temperature suggests an increase in light intensity."*

✓ **These are two separate variables all together.**




**(b)** In 2024, scientists predicted that the population of phytoplankton in the North Sea will decline drastically in the **next 50 years**.

**(i)** Discuss the extent to which the data in Fig. 3.1 and Fig. 3.2 supports the scientists' prediction. **[Data, HI-2]** [4]

1. **[Fig. 3.1]** From **1950 to 2000**, colour intensity increases from **0.5 arbitrary units** to **2.0** **[accept: 1.9 – 2.1]** **arbitrary units**

2. **[Fig. 3.2]** The **number of months** with highest colour intensity has increased from only **August in 1950**, to **March to October in 2000**

3. Both data reflects an **increase in sea temperature** from **1950 to 2000**.

4. ***Idea that*** If the trend continues, the increasing sea temperature will increase beyond their optimal temperature for survival (hence death).

5. **Data** for **2001-2023 not available** to show how the North Sea temperature has changed since 2000, thus **not conclusive**.

**(ii)** Suggest **and** explain **two** consequences of a declining phytoplankton in the North Sea. **[Climate Change, KU-2]** [4]

1. **[Suggest]** An increase in oceanic carbon dioxide levels leading to ocean acidification.

**OR**

**[Suggest]** An increase in atmospheric carbon dioxide levels that further warms the atmosphere.

2. **[Explain]** Due to less oceanic/atmospheric carbon dioxide used in photosynthesis by phytoplankton.

3. **[Suggest]** Decrease in marine populations.
4. **[Explain]** Less phytoplankton for marine organisms to feed on, leading to their decline.

5. **[Suggest]** Death of marine organisms that rely on well-oxygenated sea water.
6. **[Explain]** Less oxygen is produced through photosynthesis by phytoplankton, reducing oxygen levels in the ocean water.

***ERRORS:***

✗ *"Coral bleaching will occur if there is a decline in phytoplankton."*

✓ **These kinds of corals (hard corals) live in shallow, tropical seas.**

✗ *"$CO_2$ will not be converted to $O_2$."*

✓ **If you wrote this because of the general photosynthesis equation ($6CO_2 + 6H_2O \rightarrow C_6H_{12}O_6 + 6O_2$) you learned in the $iOS_{Olevels}$, it is time to update to $iOS_{Alevels}$.**

**[Total: 11]**

**Section B**
Answer **ONE** question.

Write your answers on the lined paper provided at the end of this Question Paper.
Your answers should be illustrated by large, clearly labelled diagrams, where appropriate.
Your answers must be in continuous prose, where appropriate.
Your answers must be set out in parts (a) and (b), as indicated in the question.

**4.** **(a)** Outline the processes of bacterial conjugation and transduction. **[OCGE Prok, KU-1]** [11]

**[Conjugation]**

1. Between F+ cell and F− cell
2. F+ cell contains the F plasmid
3. Carries genes that code for proteins that form the sex pilus
4. Sex pilus forms a cytoplasmic bridge between F+ cell and F− cell
5. **One strand** of F plasmid DNA is nicked, and the **5' end** enters F− cell
6. ***Ref to*** transferosome or relaxosome (spelling must be correct)
7. **Complementary DNA strand** synthesized to produce double-stranded plasmids in **both** cells.
8. **Leading strand** / **continuous** synthesis of daughter strand in the **F+ cell**
9. **Lagging strand** / **discontinuous** synthesis of daughter strand in the **F− cell**
10. Outcome: two F+ cells

**[Transduction]**

[generalized]

11. A **lytic phage / T4 phage / Virulent phage** attaches to and injects its DNA into a bacterial cell
12. Host DNA hydrolysed into smaller fragments
13. Viral DNA is replicated and viral proteins are synthesized
14. During assembly, a **fragment** of bacterial DNA is packaged into the capsid head
15. Forms a **generalized transducing phage** that infects another bacterial cell
16. DNA from the previous bacterial cell is injected and incorporated into the recipient cell

[specialized]

17. **Temperate / Lambda phage** attaches to and injects its DNA into the bacterial cells, the phage DNA in turns circularizes and integrates into the bacterial genome.
18. Lysogenic prophage enters lytic phase, where **prophage** is excised from bacterial DNA
19. Imprecise excision – some phage DNA is left behind while some bacterial DNA is excised together

20. This phage-bacterial hybrid DNA is packaged into the capsid head, to form specialized transducing phage.

**Accept: well-annotated drawings relevant to each point**

**(b)** The RNA World Hypothesis theorizes that RNA existed on Earth before modern cells arose. According to this hypothesis, RNA stored genetic information, catalyzed chemical reactions, and played structural roles in primitive cells. Only later in evolutionary time did DNA take over as the genetic material, while proteins became the major catalyst of reactions and structural component of cells.

To date, the RNA World Hypothesis remains controversial.

Discuss, with examples, how our current understanding of RNA
- supports the RNA World Hypothesis
- refutes the RNA World Hypothesis. **[Biomol, KU-3]** [14]

1. RNA world hypothesis states that **RNA can perform the functions of DNA and proteins**.

**SUPPORT [max 9]**

2. ***Ref to*** RNA can be an enzyme (ribozyme) **+**
    a. e.g. peptidyl transferase in ribosome, **OR**
    b. e.g. spliceosomal enzyme

3. ***Ref to*** RNA can play structural role **+**
    a. e.g. in the stability of ribosome (as rRNA) , **OR**
    b. e.g. in the stability of telomerase (as telomerase RNA)

4. ***Ref to*** RNA as the genetic storage molecule **+**
    a. in influenza virus (−)ssRNA, **OR**
    b. in HIV (+)ssRNA, **OR**
    c. **AVP:** e.g. in SARS-Cov2 (+)ssRNA

5. ***Ref to*** how RNA can self-replicate like how DNA does **+**
    a. influenza (−)ssRNA → (+)ssRNA → (−)ssRNA again

6. ***Ref to*** how RNA can be used directly as mRNA or be transcribed into mRNA **+**
    a. influenza (−)ssRNA to (+)ssRNA, **OR**
    b. HIV (+)ssRNA as mRNA

7. ***Ref to*** RNA as a transport molecule **+**
    a. tRNA carries amino acid to ribosome

8. ***Ref to*** RNA can achieve specific folding like proteins to assume specific function **+**
    a. clover leaf structure of tRNA, **OR**
    b. multiple hairpin loops of rRNA / snRNA

9. ***Ref to*** RNA can be the starting message to synthesize DNA **+**
    a. reverse transcription in telomerase, **OR**
    b. reverse transcription in retroviruses such as HIV

10. ***(cont'd from point 9)*** suggesting that DNA arose from reverse-transcribing RNA.

11. RNA can be translated to make proteins, but not vice-versa, suggesting RNA precedes proteins in existence.

12. ***Ref to*** how RNA can be complexed with proteins just like DNA with histones to ensure stability **+**
    a. e.g. nucleocapsids in HIV

**[Points 2−9 must be substantiated by an example]**

**REFUTE [at least 1]**

***Student should use your understanding of the structure / properties of RNA to refute the hypothesis. And not explain why DNA is the genetic material / proteins are enzyme / serve structural components.**

**[Intrinsically unstable nature of RNA]**

13. RNA is intrinsically unstable due to 2'OH that is more reactive than 2'H / being single stranded.

14. more prone to hydrolysis / degradation.

15. such instability raises concerns about the long-term viability of RNA.

**[Limited monomer diversity in RNA]**

16. RNA comprises only 4 types of monomers as opposed to 20 in proteins.

17. This limitation in monomer diversity could constrain the range of functional structures and functions that RNA can adopt.

18. hence, to achieve a diversity of structure and function, RNA needs to be extremely long, which increases instability.

**[Dependency on protein complexes for function]**

19. RNA needs to be complexed with proteins to function as enzymes (e.g. ribosome, spliceosome)

20. challenges the notion of RNA by itself can be an enzyme.

**[Negative charge hindering membrane embedding]**

21. RNA is highly negatively charged due to phosphate groups.

22. Hydrophobic interactions are essential for molecules to embed into hydrophobic membranes.

23. Hence, RNA unlikely to be embedded into membranes to serve as a transport molecule.

***ERRORS:***

✗ *"RNA has catalytic property, for example RNA polymerase carries out transcription."*

✓ **RNA polymerase is a PROTEIN**

✗ *"tRNA provides catalytic function in translation" "tRNA function as peptidyl transferase"*

✓ **tRNA does not provide any catalytic activity during translation. tRNA functions to carry amino acid to the ribosome for translation.**

**5. (a)** Explain the processes in B lymphocytes that give rise to a diverse array of antibodies.[11]

**[Immunity, KU-1]**

**[Somatic recombination] – at least 1**

1. Somatic recombination / V(D)J recombination
2. Involves one heavy chain gene and two light chain genes (κ or λ)
3. Either κ L chain gene or λ L chain gene is expressed
4. Only one allele of the H chain gene and one allele of L chain gene are expressed
5. H chain gene: one segment each from V, D and J are randomly selected and joined
6. L chain gene: one segment each from V and J are randomly selected and joined
7. The rearranged VDJ and VJ segments are transcribed and translated into the variable region of the H and L chain proteins.
8. The variable region of H chain protein and variable region of L chain protein constitutes the antigen-binding site.
9. Multiple gene segments for each region (V, D, J) create numerous potential combinations.

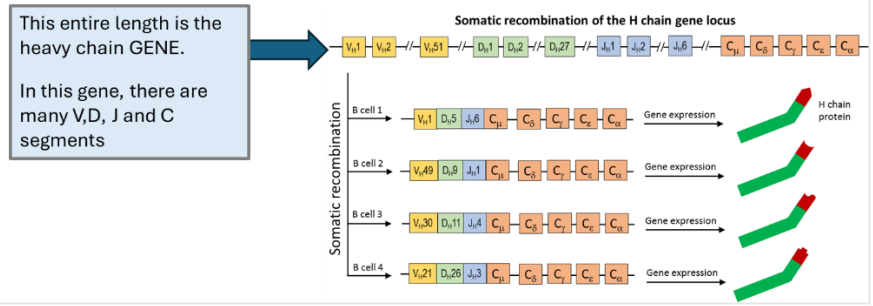
***ERRORS:***

✗ *"V gene, D gene and J gene codes for variable region on heavy chain"*

✗ *"Variable genes code for variable region on heavy chain"*

✓ **VDJ segments are found on the heavy chain gene.**

**Immunity notes page 24**




✗ *"Somatic recombination occurs on VDJ segments on heavy chain"*

✓ **Somatic recombination occurs on VDJ segments on heavy chain <u>GENE</u>"**

✗ *For pt 4: "*Only one H chain gene and one L chain gene is expressed."

✓ **There are 2 alleles for each gene. Need to specify that only one allele of each gene is expressed.**

**[Somatic hypermutation] – at least 1**

10. Somatic hypermutation

11. **Point mutations / single nucleotide substitutions** in the **rearranged V(D)J segments of the light and heavy chain loci / gene**

12. Generates antibodies of varying affinity to the antigen

13. B cells with deleterious/lethal/self-reactive mutation undergo apoptosis

14. B cells with increased affinity for the antigen are selected to undergo clonal expansion

**[Class switching] – at least 1**

15. Class switching

16. A new constant region is cut and joined to the rearranged V(D)J region of the antibody genes.

17. Generates all the five classes/isotypes of antibodies – IgM, IgG, IgA, IgD, IgE

18. The different classes of antibodies produced all bind to the same antigen.

**Accept: well-annotated drawings relevant to each point**

***ERRORS:***

✗ *"Antibodies with the increased affinity to the antigen undergoes clonal expansion"*

✓ **The B cells undergo clonal expansion, not the antibodies.**

**(b)** Chronological age is the number of years that has elapsed since birth, while biological age refers to how old the **cells** are based on physiological and molecular changes.

Suggest the physiological and molecular changes associated with biological ageing **and** suggest reasons why people with the same chronological age can have vastly different biological ages. **[Cell, OCGE in Euk, DNA Mutation, Inheritance KU-3]** [14]

**[Physiological changes]**

1. Cellular senescence – the cessation of cells' ability to divide due to Hayflick limit.
2. Decline in autophagy…
3. …leading to accumulation of damaged organelles / ***named organelle***
4. Changes to membrane integrity…
5. …leading to decline in cell-to-cell signaling / transport across membranes / **AVP**

**[Molecular changes]**

6. Shortening of telomere length as a result of end-replication problem.
7. Changes in DNA methylation pattern results in genes being switched off.
8. Changes in histone methylation / deacetylation results in genes being switched off.
9. A decline in the efficiency of DNA repair mechanisms…
10. …leading to accumulation of DNA mutations.
11. Protein aggregation from the misfolding and accumulation of proteins within cells (leading to age-related diseases).
12. Damaged proteins and DNA as a result of accumulation of free radicals.

**[Why same chronological age but different biological age]**

[Genetic Variability]

13. ***Idea that*** Genetic variation across individuals contributes to differences in efficiency of cellular functions / susceptibility to certain health conditions / **AVP**

[Environmental Exposures]

14. Exposure to radiation / ***named radiation*** e.g. UV light / X-rays / gamma rays
15. Exposure to carcinogens / mutagens / ***named mutagen*** e.g. ethidium bromide
16. Causes DNA lesions/damage that leads to replication error.
17. Exposure to pollutants from car exhaust / factories can cause cellular damage.
18. Infection by viruses/bacteria can cause inflammation and damage cells.

[Lifestyle Choices]

19. ***Idea that*** Diets rich in antioxidant / vitamins mitigate oxidative damage to DNA, slowing ageing.
20. ***Idea that*** Excessive intake of processed foods / sugars / unhealthy fats can contribute to inflammation, accelerating ageing.

21. ***Idea that*** Sedentary lifestyle associated with faster ageing / Regular physical activity associated with slower ageing.

22. ***Idea that*** Tobacco and carcinogens in cigarettes can damage respiratory cells.

23. ***Idea that*** Excessive alcohol consumption can damage liver cells (leads to liver cirrhosis).

[Healthcare Access]
24. ***Idea that*** Timely healthcare interventions can influence the trajectory of ageing.

[Individual Health History]
25. ***Idea that*** Variations in individual health histories (e.g. past illnesses, injuries, medical treatments) contribute to differences in the rate of ageing.

[Immune System Function]
26. ***Idea that*** Weaker immune system increases susceptibility to diseases and the ageing process.

[Stress Levels]
27. ***Idea that*** Chronic stress influences ageing by affecting hormone levels and cellular signaling.

[Sleep Patterns]
28. ***Idea that*** Variations in sleep patterns, including sleep duration and quality, can influence cellular repair processes and overall health.

29. **AVP**

**[Max 3 for point 19 to 28]**

😊 **END OF PAPER 3** 😊