

EUNOIA JUNIOR COLLEGE JC1 Promotional Examinations 2021 General Certificate of Education Advanced Level Higher 2



H2 Biology

Paper 2 Structured & Free Response Questions

9744

05 October 2021 2 hours

Additional Materials: 12-page Answer Booklet.

READ THESE INSTRUCTIONS FIRST

Write your name, civics group and registration number in the spaces at the top of this page.

Write in dark blue or black pen. You may use an HB pencil for any diagrams or graphs.

Do not use paper clips, highlighters, glue or correction fluid.

Section A

Answer **all** questions in the spaces provided on the Question Paper.

Section B

Answer all questions in the 12-page Answer Booklet.

The use of an approved scientific calculator is expected, where appropriate. You may lose marks if you do not show your working or if you do not use appropriate units.

The number of marks is given in brackets [] at the end of each question or part question.

For Examiner's Use		
Sec	tion A	
1	8	
2	8	
3	8	
4	8	
5	10	
6	10	
7	8	
Section B		
8	20	
Total	80	

READ THIS FIRST!

- 1. The biology teachers have spent time writing the examiners' comments in this answer key, as well as the individual comments on your scripts. Do **take time to read** through them <u>carefully</u>, and note the common mistakes, misconceptions and phrases to avoid. This is an **important part of learning**.
- 2. As you read the answer key, note about any questions that you may have, and **clarify** them with your respective tutor **as soon as possible**.
- 3. It is <u>not</u> about matching words in your answer to the mark scheme. Your answer should be <u>conceptually sound</u>; having all the words doesn't necessarily make your answer correct.
 E.g. enhancer is a gene, it codes for activators which binds to enhancers to increase rate of reaction by promoting formation of transcription initiation complex.
 The above statement "hits all the right words" but it is conceptually wrong, so no mark will be awarded.
- 4. Different questions have different requirements. You will need to apply your conceptual understanding and keywords based on the **context** of the question. It is not one size fits all.
- 5. You should <u>never</u> have the mindset that content / concepts tested will not come out again. You are preparing for the A-Level Examinations, all **learning** leading up to it should be **retained** (and not forgotten).
- 6. As part of learning, you should **<u>analyse your papers</u>** and find out what are your gaps:
 - i. Content
 - ii. Concepts
 - iii. Question analysis skills
 - iv. Answering techniques and writing skills
- 7. It is important to **reflect** on this learning experience, **consolidate** your learning and **build on** your learning resources (notes, tutorials, worksheets, tests, etc.) to better prepare for future examinations.
- 8. **Seek to understand, not just memorise.** Make sense of the terminology, which will help a lot as you try to understand the connections between different parts of the content necessary for conceptual understanding.

Section A

1 Fats are usually stored as oil in the seeds of the plants and occasionally in the fleshy part of the fruit, as in the olive and the oil palm. The oil is a reserve of high-energy food for use by the germinating seed for growth of the embryo. The sunflower seed is actually the fruit of the sunflower (Helianthus annuus) and may contain about 51% oil.





(a) On figure 1.1, circle an ester bond. [1]





Circle **must** include the two O atoms that are joined to a C atom (A: if all ester bonds) correctly circled); (R: circle(s) that do not meet stated criteria).

- Not always well answered, candidates are advised to use the above when • attempting Biology questions as this is the accepted version of an ester bond in A level Biology.
- Candidates are advised to put their circle carefully so as not to include the angular corners (red arrow) as these represent a C atom!

(b) Sunflower oil can be used to make biodiesel.

During the process, the fatty acids in the triglyceride react with methanol to form fatty acid methyl esters that are liquid products. The other component of the original triglyceride molecule forms another liquid product that is denser than the methyl esters.

Name the component of the molecule seen in Fig. 1.1 that forms this denser liquid.

......[1] 1. Glycerol

Examiner's Comments:

- Generally well-answered.
- A number of candidates did not appear to be familiar with the components that make up a triglyceride molecule.

1.1

(c) Table 1.1 shows the melting points of the three different fatty acid methyl esters.

Table

fatty acid methyl ester	formula	melting point / °C
methyl linoleate	$C_{19}H_{34}O_2$	
methyl linolenate	$C_{19}H_{32}O_2$	- 55.0
methyl oleate	$C_{19}H_{36}O_2$	- 20.0

(i) Complete Table 1.1 with one possible value for the melting point of methyl linoleate. [1]

Accept any value from <u>-20.1 °C to -54.9 °C;</u>

Note: The higher number of H atoms implies more saturation (less or no double bonds) of the fatty acid molecules and hence they have higher melting points (-20°C is a higher temperature than -55 °C). Since methyl linoleate has H atom numbers that is between that of the other two esters, its melting point is somewhere between the two values of -20 °C and -55 °C.

Examiner's Comments:

- Generally well-answered with most candidates able to give a value between -20°C and -55°C.
- (ii) Suggest one way the structure of fatty acids found in butter might differ from the fatty acid that forms methyl oleate.

.....

.....[1]

Compared to methyl oleate,

- the fatty acids in butter may have <u>no / less double bonds</u> (A: reference to '<u>more</u> <u>saturated</u>');
- 2. the fatty acids in butter may contain many more carbon atoms;

Note: Butter is a solid at room temperature compared to methyl esters, it is more saturated and hence has higher melting point. These saturated fatty acid molecules have straight fatty acid chains and thus pack more closely together and may be solids at room temperature.

Methyl esters that have more double bonds (less H atoms) may have more 'bent' or 'kinked' fatty acid tails thus they may pack less tightly, leading to lower melting points and they are liquids. In biodiesel production higher melting point methyl esters (more saturated) do not have good flow properties as they tend to become a gel that impedes flow of biodiesel in engines.

Examiner's Comments:

- Generally not well-answered.
- A number of students were not certain that the fatty acid molecules in butter were likely to have more saturated carbon chains that enable closer packing and hence a higher melting point and are solids at room temperature.
- Phrase to avoid: 'There are more kinks in this molecule' (Not precise: 'Kinks' are just bends in a molecule and these kinks could be due to a number of reasons). The specific reason for the presence of 'kinks' in methyl ester molecules is the presence of C=C double bonds, hence candidates must be specific in this!
- Phrase to avoid: 'The fatty acid chains in butter are saturated'. (Not precise: This phrase is misleading as the methyl esters may also have some fatty acid chains which are saturated. Hence the idea of 'more/less saturated fatty acid chains' would be better when making comparisons!)
- (d) Living organisms have many uses for triglycerides, one of which is the production of phospholipids.

Describe how the roles of triglycerides and phospholipids differ in cells.

......[2]

- 1. Triglycerides can be used as <u>respiratory substrate</u> / serve as a good energy source / a good energy storage molecule; (R: thermal insulation / buoyancy)
- Phospholipids are the main structural component of cell membranes; (accept only if previous reference made to structural component idea - responsible for compartmentalisation of the cell / for cell membranes to act as selective barriers) (I: specific type of cell membranes)

- Not always well-answered. A number of candidates did not read and understand the question clearly. They were required to describe a difference in the respective roles of triglycerides and phospholipids in cells – meaning how their roles differ inside a cell. Thus functions like thermal insulation and buoyancy are not relevant because these can only function when there are aggregates of fat tissues (many cells).
- Phrases to avoid: 'Phospholipids act as bilayers' / 'Phospholipids serve to compartmentalise the cell' (Inaccurate: They cannot do so unless organised into membranes!);
- Phrase to avoid: 'Phospholipids are part of or form cell membranes' (Not precise: Not explicit that phospholipids are the <u>main structural component</u> of cell membranes);
- Phrase to avoid: 'partially permeable' (Inaccurate: With respect to living biological membranes, we should use 'selectively permeable')

(e) Explain why phospholipids are able to form bilayers in a cell.

.....[2]

- 1. <u>Hydrophilic</u> / <u>charged</u> <u>phosphate heads</u> (A: phosphate groups) and <u>hydrophobic</u> / <u>non-polar</u> <u>fatty acid tails</u> (A: <u>hydrocarbon tails</u> / <u>chains</u>) (I: amphipathic);
- Phosphate heads point outwards and face aqueous intracellular and extracellular environment while fatty acid tails form the hydrophobic core resulting in <u>fatty acid tails</u> <u>facing</u> / <u>interacting with each other</u> forming a bilayer; (R: if no reference to formation of two layers of phospholipids) OR
- 3. <u>Hydrophilic</u> / <u>charged</u> <u>phosphate heads</u> (A: phosphate groups) point outwards and face **aqueous** intracellular and extracellular environment;
- 4. **Hydrophobic** / <u>non-polar</u> <u>fatty acid tails</u> (A: hydrocarbon tails /chains) form the **hydrophobic core** resulting in <u>fatty acid tails facing</u> / <u>interacting with each other</u> forming a bilayer; (R: if no reference to formation of two layers of phospholipids)

[Total: 8]

- Not always well-answered by some candidates.
- A number of candidates did not make reference to the arrangement of the phospholipids resulting in two layers forming a bilayer. As the question asked for an explanation why phospholipids can form bilayers, answers must end with reference to the bilayer formation.
- Some candidates proceeded to describe the phosphate heads as polar, without mentioning and emphasising that they are negatively charged!
- Phrase to avoid: 'Hydrophilic, charged and polar phosphate heads' (Emphasis should be on the negative charge of the phosphate group, rather than its polarity)
- Phrase to avoid: 'Hydrophobic tails' (Not precise: There is no mention of what is hydrophobic. Writing the hydrophobic property but with no context of what molecule that is hydrophobic. Proper phrase should be 'hydrophobic hydrocarbon/fatty acid tails')
- Phrase to avoid: 'Two phospholipids with the fatty acid tails interacting with each other' (Not precise and inaccurate: The cell membrane is made up of many phospholipids, not just two!)

2 Phosphatases are enzymes that catalyse the removal of phosphate groups from organic compounds. A student investigated the effect of substrate concentration on the rate of the reaction catalysed by an acid phosphatase (enzyme **A**). The results are shown in Fig. 2.1.



Fig. 2.1

(a) (i) On Fig. 2.1, show how K_m for enzyme A is derived.

[1]

1. Concentration at which $\frac{1}{2}$ V_{max} = 7 µmol dm⁻³ min⁻¹ is attained

Examiner's Comments:

- A number of candidates did not appear to be familiar with deriving K_m , and thus wrongly drew tangent lines or a line at $V_{max} = 14 \ \mu mol \ dm^{-3} \ min^{-1}$
- (ii) State the K_m value.

.....[1]

1. 0.3mmoldm⁻³

- As this question is a follow-up from (a)(i), students who answered (a)(i) wrongly are likely to quote the wrong K_m value.
- **Mistakes** made by some students who answered (a)(i) correctly but answered this question wrongly:
 - 1. No units or wrong units (moldm⁻³)
 - 2. Read off the substrate concentration wrongly
 - 3. Indicated the value of $\frac{1}{2}$ V_{max} (7 µmol dm⁻³ min⁻¹)

(b) The student investigated a different phosphatase enzyme (enzyme B) and found the value of K_m to be higher than that of enzyme A.

Explain the difference between the values of K_m for these two phosphatase enzymes.

.....[2]

1. Enzyme **B** has a **lower affinity of enzyme for its substrate** (than enzyme **A**) OR the higher the K_m, the lower the affinity of the enzyme for its substrate ; (**R**: affinity of substrate for the enzyme)

(A: *if affinity not used, accept idea about* **extent** of *ability to form enzyme substrate complex*) (A: enzyme B's active site is a less precise fit for substrate / has a less complementary conformation and charge to that of substrate)

I: ref. to rate of reaction, turnover number(s)I: ref. to competitive inhibition

Enzyme B needs a higher concentration of substrate to reach ½V_{max} (than enzyme A);

A: vice versa (for enzyme A)

Examiner's Comments:

- Candidates should note that information, such as rate of reaction, V_{max}, enzyme concentration and presence of inhibitor, are not given. Do not assume that V_{max} is the same for both enzymes, nor suggest situations like inhibitor binding to enzyme B.
- K_m is **independent** of enzyme and substrate concentrations. It also does **not** apply for allosteric enzymes with multiple subunits and multiple active sites.
- An enzyme can have lower V_{max} but higher K_m than another enzyme (or vice versa).



E.g. Enzyme B with lower V_{max} but higher K_m than enzyme A

• Common mistakes:

- "Affinity of substrate for enzyme" × − K_m of enzyme is an inverse measurement of the affinity of enzyme for its substrate (i.e. tendency of enzyme to bind to substrate). "Affinity between enzyme and substrate" was accepted this time but note that this phrasing is not accurate and will be marked down in the future.
- "Takes longer time to reach ½ V_{max}" × mistake likely to stem from poor understanding of the definition of K_m and/or misconception that the graph shown is a graph of rate of reaction against time. Note that the x-axis is substrate concentration, not time.

(c) The student repeated the investigation on enzyme **A** with a competitive inhibitor.

The same concentrations of substrate were used as before, but a competitive inhibitor was added to each reaction mixture.

The same concentration of the inhibitor was used in each reaction mixture.

The student found that V_{max} was the same as before, but K_m was higher.

Explain how the addition of the competitive inhibitor results in the same value for V_{max} but a higher value for K_m .

.....[4]

- 1. The competitive inhibitor has <u>similar</u> (R: same) <u>conformation and charge to</u> <u>substrate molecule</u>;
- Competitive inhibitor binds to the active site thus blocking substrate from binding (A: competes with substrate for active site);
- This reduces availability of enzyme active sites for substrate binding (A: ref. to lower rate of formation of enzyme-substrate complexes) (R: no ESCs formed), and <u>rate of reaction decreases</u> at low substrate concentrations, so ¹/₂ V_{max} is reached at a higher substrate concentration (higher K_m)
- At high substrate concentration, competitive inhibition will be overcome / the degree of inhibition will be reduced (OR the substrate outcompetes the inhibitor);
- 5. Hence, V_{max} is the same as all active sites are saturated / fully occupied



[Total: 8]

- This question is fairly well done by many candidates.
- Some students appear confused about terms like "similar", "same", "complementary" and their differences. Similar ≠ same, similar ≠ complementary
- For point 4, alternative phrasing is accepted if the idea of **substrate** outcompeting **inhibitor** is suggested. Refer to the answer key for the recommended phrasing. Answers on higher frequency of substrate binding to enzyme active site without comparison to frequency of inhibitor binding to active site are rejected.

- 1. "Take longer time to reach V_{max} " × V_{max} is **not** the maximum product yield after all substrates have been converted to products over time, but rather the maximum rate of reaction (rate of product formation = how many products produced per unit time / at any given time interval, e.g. per second)
- "Lower affinity of enzyme for substrate → higher K_m" × Presence of inhibitor reduces apparent affinity of enzyme for substrate, i.e. enzyme affinity appears to be lower. K_m is not measured for inactive enzyme. Apparent K_m is observed instead of the "true" K_m, i.e. K_m appears to be higher.
- "prevent formation of enzyme-substrate complexes … lower rate of reaction"
 ★ It is correct to say that an enzyme-substrate complex cannot be formed when a substrate cannot bind to active site of enzyme that is bound by inhibitor, but not all enzyme active sites are bound by (reversible) inhibitor at any given time interval. If no enzyme-substrate complexes are formed, rate of reaction (which is dependent on the rate of formation of enzyme-substrate complexes) would have been zero, not lower.

• Phrases to avoid:

- "Enzyme has complementary conformation and charge to substrate" × (not precise) should be "enzyme active site has complementary conformation and charge to substrate"
- "Overcome inhibitor" × should be "overcome inhibition" (effect of inhibitor on enzyme activity)

3 Fig. 3.1 shows a transmission electron micrograph of a replication bubble.



Fig. 3.1

- (a) In eukaryotic cells, there are many sites of origin of replication for each DNA molecule.
 - (i) Explain an advantage of having many sites of origin of replication, rather than only one site.

```
.....[1]
```

 It speeds up (A: rate) replication of the extremely long (A: large genome) DNA molecule; (R: explanation on "only one site") (R: if no reference to length of DNA or size of genome – 'explaining an advantage' is not the same as 'stating an advantage')

Examiner's Comments:

- Poorly answered.
- Majority of the candidates did not seem to know the difference between the command phrases 'State an advantage of....' vs 'Explain an advantage of....'. Thus most answers wrote about the increased speed of replication but did not make explain that this increased speed of replication was an advantage because of the large genome of eukaryotic cells.
- A small number of candidates wrote about transcription instead of DNA replication.
- Phrase to avoid: 'It speeds up the replication of <u>a</u> long <u>DNA strand</u>' (Imprecise and Inaccurate: There are many DNA molecules in the cell and because of semiconservative DNA replication, both strands of a parental DNA molecule are used as templates, not just one!)
- (ii) The structure of origins of replication has a higher proportion of A-T base pairs than C-G base pairs. Suggest why this is so.
-[2]
- 1. There are 2 hydrogen bonds between A-T versus 3 hydrogen bonds between C-G;
- 2. Overall, there are **fewer hydrogen bonds to break** to **separate / unzip complementary strands** for replication; (Note: need to mark for both break *and* separate / unzip)

- Not always well-answered.
- Some candidates were not aware of the difference in the number of hydrogen bonds between Adenine and Thymine vs Guanine and Cytosine.
- Many answers did not refer to breaking AND separation of the DNA strands.

• Phrase to avoid: 'Adenine and Thymine are base-paired with double bonds between them' (Not precise: 'double bonds' could also mean C=C double bonds! Better phrase would be 'Adenine and thymine are base-paired with two hydrogen bonds between them').

(iii) Briefly explain the importance of DNA replication as part of the cell cycle.

It is to form genetically identical sister chromatids (A: genetically identical or sister) for mitosis / nuclear division to form two new nuclei; (A: two cells) OR
 It is to ensure each new daughter cell has genetically identical chromosomes as original cell; (A: same type and number of chromosomes) (R: 'Amount of DNA' – vague)

 AVP

Examiner's Comments:

- Poorly answered.
- Most answers were not precise with very vague reference to 'same amount of DNA', 'cells that are genetically identical' but not clearly making reference to what the genetic material was.
- (b) The amino acid sequence of haemoglobin in **person A** has been determined. The first five amino acids of this sequence are shown in Table 3.1.

Table 3.2 shows the genetic code (mRNA codons).

A student was asked to use Table 3.2 to work out an mRNA nucleotide sequence that would correspond to the first five amino acids of haemoglobin. The student's sequence is shown in Table 3.1.

amino acid sequence	met	tyr	glu	pro	lys
student's nucleotide sequence	AUG	UAU	GAC	CCU	UGU
correct = ✓ incorrect = ×					

Table 3.1

Table 3.2

	d)	U	С	Α	G]
u	U	Phe Phe Leu Leu	Ser Ser Ser Ser	Tyr Tyr STOP STOP	Cys Cys STOP Trp	UCAG	
e in codo	С	Leu Leu Leu Leu	Pro Pro Pro Pro	His His Gln Gln	Arg Arg Arg Arg	UCAG	e in codo
1st base	A	Ile Ile Ile Met	Thr Thr Thr Thr Thr	Asn Asn Lys Lys	Ser Ser Arg Arg	U C A G	3rd bas
	G	Val Val Val Val	Ala Ala Ala Ala	Asp Asp Glu Glu	Gly Gly Gly Gly	UCAG	

2nd base in codon

(i) Complete Table 3.1 using a ✓ or a × to indicate whether the student has used Table 3.2 correctly to identify the codons for each amino acid in the nucleotide sequence. [1]

× √ × √ ×					
	\checkmark	✓	*	✓	*

Examiner's Comments:

- Generally well-answered with only a small number of candidates getting this part incorrect.
- (ii) Discuss, with reasons, how an mRNA sequence of **person A** may not be the same as the mRNA sequence for those five amino acids present in another person?

.....[3]

- 1. The genetic code is **degenerate**;
- Several amino acids can be coded for by more than one codon; (R: 'nucleotide sequences' / mRNA sequences – too vague) OR
 - There are 64 codons coding for 20 amino acids;
- 3. Quote a valid example from Table 3.2;

[Total: 8]

- Not always well-answered.
- A number of candidates thought that the degeneracy of the genetic code was always linked with mutation. This is not true as degeneracy of the genetic code is inherent in the code! Thus it may be useful and beneficial if the degeneracy of the code allowed for point mutations (substitution) in the third base of the codon to not change the amino acid at all (silent mutation) but it is <u>not absolute</u> for mutations to occur first for degeneracy of the code to have an impact!
- References to RNA splicing are not acceptable as the exons (consisting of many codons, for more than five amino acids) are spliced together!
- Phrase to avoid: 'The amino acids are degenerate" (Inaccurate: Degeneracy refers to the genetic code (codons), not the amino acids!
- Phrase to avoid: 'Difference mRNA sequences code for the same amino acid' (Not precise and potentially inaccurate: The highlighted phrase does not give any indication of the length but a codon is actually made up of a set of three nucleotides)

4 Fig. 4.1 shows an electron micrograph of actively dividing onion cells undergoing various stages in the cell cycle.





- (a) With reference to Fig. 4.1,
 - (i) state the order in which these stages (W, X, Y, Z) occur during the cell cycle.
 -[1]
 - 1. X, Z, Y, W (R: if don't start from X)

The actively dividing onion cells are undergoing **mitotic** cell cycle. **X** – interphase, **Z** – prophase, **Y** – metaphase, **W** – anaphase

- Generally well done ©
- The first phase of a cell cycle is interphase (stage X).
- Common mistakes:
 - 1. "Z, X, Y, W" ★ likely to have mistaken Z as interphase and X as prophase. Chromosomes can be distinctly seen in stage Z but not in stage X. Condensed chromosomes are found in prophase while chromatin are found in interphase.
 - "Z, Y, W, X" × likely to have mistaken X as telophase. There is only 1 nucleus in stage X, instead of 2 nuclei.

(ii) outline the events occurring in stage Z.

......[3]

- 1. Shortening and thickening / condensation of chromatin (R: DNA) to form chromosomes;
- 2. Nucleolus disappears (R: disintegrates) and nuclear envelope (R: membrane) disintegrates;
- 3. Formation of spindle fibres;
- 4. Kinetochore microtubules bind to kinetochores attached to centromeres of chromosomes;

[Any three]

(**R**: centrioles moving to opposite poles – **no** centrioles in onion cells, which are higher plant cells) (centrosomes and asters are also absent)

- Generally well done ©
- Stage Z is prophase, NOT interphase or telophase.
- Be specific and identify kinetochore microtubules as the spindle fibres that bind to kinetochores attached to centromeres of chromosomes
- Misconceptions:
 - 1. "Nucleolus disintegrates and nuclear envelope disappears" ×
 - Nucleolus is a structure consisting of ribosomal DNA, rRNA, and ribosomal proteins. As chromatin condenses to form chromosomes, nucleolus **disappears** (i.e. no longer see the structure) but not **disintegrates** (i.e. breaks down into smaller parts). The components of nucleolus are not hydrolysed.
 - Nuclear envelope disintegrates (i.e. breaks down into smaller parts), allowing the binding of kinetochore microtubules to centromeres of chromosomes, and also the movement of chromosomes.
 - 2. Formation of spindle fibres in interphase ×
 - 3. Condensation of chromatin in interphase ×
 - Formation of centromere in prophase ➤ it is a DNA sequence that is already (and always) present in the eukaryotic DNA, regardless of stage of cell cycle. Centromere replicates during DNA replication in S phase of interphase.
- Phrase to avoid:
 - 1. "Nuclear membrane disintegrates" ★ should be "nuclear envelope disintegrates", which refers to both the inner nuclear membrane and outer nuclear membrane.

(b) An experiment was carried out to investigate the effect of p53 on cell cycle in human liver cells. Two types of cell populations were used in the investigation, one with intact p53 gene and the other with mutated p53 gene. Both cell populations were subjected to gamma radiation which is a DNA damaging agent. Mitotic index of the cells were then measured and the results are shown in the Figure 4.2.



Fig. 4.2

Mitotic index reflects the number of cells in a population that are dividing. It is calculated by counting the number of cells with condensed chromosomes and divided it by the total number of cells observed.

(i) Suggest why the condensed chromosomes are used to calculate mitotic index.

1. Condensed chromosomes are only found in stages of mitosis (R: prophase, meiosis, nuclear division, cell division), thus indicating that the cell is undergoing mitosis;

- Not well-answered by many candidates, due to these 3 common mistakes:
 - 1. Poor understanding of mitotic index $\left(\frac{number of dividing cells}{total number of cells}\right)$, i.e. proportion of
 - cells that are dividing) or did not note its definition given after Fig 4.2
 - 2. Reference to wrong processes, for e.g.
 - Prophase × (see below) + "chromosome condensation occurs in prophase" is factually correct but the question is on condensed chromosomes, not the process of condensing chromosomes. Condensed chromosomes do not just indicate the start of mitosis.
 - Meiosis × "condensed chromosomes are found in meiosis" is factually correct, but it is not relevant to the context of this question as question is on 1) mitotic index, and 2) human liver cells (no meiosis)
 - Nuclear division * not specific, as nuclear division can refer to mitosis or meiosis

- Cell division × wrong as cell division = nuclear division + cytokinesis (chromosomes have already decondensed into chromatin in cytokinesis)
- 3. Poor phrasing
- Common misconception:
 - 1. Condensed chromosomes are only found in prophase ★ chromosome condensation occurs in prophase but chromosomes remained condensed in metaphase, anaphase, and early telophase before they decondense.

(ii) With reference to Fig. 4.2, explain the difference between the two sets of results obtained

.....[3]

- Cells with <u>intact p53</u> have a <u>lower mitotic index</u> at <u>about 4%</u> (A: 3%) while cells with <u>mutated p53</u> have a <u>higher mitotic index</u>, peaking to around <u>32%</u> (A: 33%) at 48h; (A: 4% increasing to 32%, greater range from 4% to 32%) (R: 4% to 14% *not across time*)
- When p53 is <u>intact</u>, it will result in <u>cell cycle arrest</u> (A: halt cell cycle) (R: inhibit cell cycle) when exposed to gamma radiation / there is DNA damage, which results in most of the cells not undergoing mitosis, resulting in lower mitotic index;
- When p53 is <u>mutated</u>, it will result in cells unable to halt cell cycle arrest and repair damaged DNA, thus cells progress on with <u>mitosis/cell cycle</u> even in the presence of DNA damage, resulting in higher mitotic index.

[Total: 8]

- Poorly answered by most candidates.
- Question analysis:
 - "With reference to Fig. 4.2" + "two sets of results" data to be quoted from the two graphs of mitotic index against time, for cells with intact p53 and cells with mutated p53
 - 2. "**Difference**" do not just quote and describe data, need to compare and state the difference
 - 3. "Explain" (command word) elaborate why there is such a difference
- Common mistakes:
 - 1. Not understanding the question's context \times (see diagrams on next page)
 - 2. No data or non-specific inaccurate data **×** advice: use ruler to help determine the specific value to quote
 - Relatively constant mitotic index of cells with intact p53 vs mitotic index of cells with mutated p53 increasing then decreasing × p53 resulting in cell cycle arrest in presence of DNA damage does not explain this trend observed across time. Instead, it explains the magnitude of the mitotic index.
 - Low mitotic index of cells with intact p53 <u>vs</u> mitotic index of cells with mutated p53 increasing then decreasing × – not the same factor of comparison (magnitude vs trend)
 - Describing function of p53 gene without application to the context of the question ×
 - No mention of DNA damage (caused by exposure to gamma radiation)
 - No link or unclear link between DNA damage and cell cycle arrest
 - 6. Poor understanding of mitotic index $\left(\frac{number of dividing cells}{total number of cells}\right)$, i.e. proportion of cells that are dividing) or did not note its definition given after Fig 4.2

7. Misconceptions and/or poor phrasing – (see below)

Common misconceptions:

- 1. "p53 codes for proteins that halt cell cycle" ★ p53 gene only codes for p53 protein. p53 protein (an activator protein) activates other genes which code for proteins that halt cell cycle.
- 2. "p53 halts cell cycle" × (see explanation for the above misconception)
- 3. p53 mutated → excessive cell proliferation / uncontrolled cell division × (see diagrams below)

• Wrong phrasing:

- 1. "Inhibit / suppress cell cycle" × halt / arrest cell cycle ✓
- 2. "In intact/mutated p53, mitotic index …" ★ should be "for cells with intact/mutated p53, mitotic index…" Gene does not have mitotic index.



Question: What else is necessary to result in uncontrolled cell division? (Hint: What is another group of cancer-critical genes that regulate cell cycle checkpoints?)

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5 T cells (a type of white blood cell) synthesize a specific type of cell surface proteins known as CD proteins, which are inserted into the cell surface membrane.

Fig. 5.1 shows the stages involved in the synthesis of a CD protein in a T cell.



Fig. 5.1

(a) Explain the function of the promoter region of the DNA.

.....[3]

- 1. The promoter is the site where RNA polymerase and general transcription factors (R: transcription factors, specific transcription factors) bind to,
- (I: RNA polymerase recruit GTFs should be GTFs recruit RNA polymerase)
- 2. facilitating formation of transcription initiation complex, essential for initiating transcription of a gene at the basal rate.
- 3. It contains certain critical elements (e.g. TATA box, GC box, CAAT box) within the promoter, which determine the strength of the promoter, hence determine the rate of transcription. OR
- 4. It contains TATA box which determines location of transcription start site.

Examiner's Comments:

- Fairly well-answered by most students
- No mark for describing structure of promoter (e.g. upstream of transcription start site, critical elements without linking to function of determining rate of transcription)
- Some students wrongly associated function of promoter to DNA replication or translation
- **Promoter ≠ transcription start site** / site of transcription initiation (*see diagram*)
- **Promoter ≠ TATA box** (see diagram)
- Inaccurate to say that RNA polymerase and general transcription factors (GTFs) bind to TATA box. They bind to the promoter. TATA-binding protein (TBP), which is a subunit of TFIID (a GTF), binds to TATA box. (*see diagram*)



- **General transcription factors recruit RNA polymerase** to bind to promoter (*see diagram*), NOT RNA polymerase recruit GTFs.
- For point 3, good to note that the more the critical elements resemble the consensus sequence, the 'stronger' the promoter, hence higher rate of transcription. Many students who got this mark did not mention the sequence of critical elements resembling the consensus sequence to account for increase in rate of transcription.
- (b) Explain why the cuts made in pre-mRNA are necessary for the T cell to produce a functional CD protein.

.....[3]

- 1. The cuts involves **excising** / **removing** (**R: splicing out)** the <u>introns</u>, which are <u>non-coding</u> regions / sequences, and
- 2. joining / splicing the exons together, which are coding regions / sequences.
- Splicing of pre-mRNA is necessary to form the <u>mature mRNA</u> that consists of only exons / no introns, so that the correct sequence of amino acids can be coded for to produce the functional CD protein. OR
- If not cut, the mRNA will have an incorrect sequence / length (I: consists of both coding and non-coding) (A: the introns would be translated, resulting in an incorrect amino acid sequence), resulting in non-functional protein being synthesized. (R: more functional or less functional)

(**R:** alternative splicing – results in production of different proteins, which is irrelevant to context of question on production of **a** functional **CD protein**)

- Fairly good attempt by most candidates
- The question requires students to:
 - 1. associate the cuts made in pre-mRNA to process of **splicing**, and
 - 2. **explain why** splicing is **necessary** for production of a <u>functional</u> CD protein (insufficient to just describe process of splicing)
- Excise (remove) ≠ splice (joined together), so reject "introns are spliced out"

- It is necessary to state that **exons** are **coding** sequences and **introns** are **noncoding** sequences as the sequence of mature mRNA that is translated into the amino acid sequence of the functional CD protein only contain exons, which are joined together.
- Introns are known to be non-coding sequences because they do not code for proteins as they are usually absent in mature mRNA. However, if introns are not removed from pre-mRNA,
 - 1. the mRNA cannot be termed as a mature mRNA, and
 - 2. the introns will code for amino acids as the codons (sequences of three nucleotides) in mRNA are read by a ribosome during translation.
- (c) Explain the functions for the poly(A) region attached to the mRNA.

......[2]

- 1. Enhances half-life / stability of mRNA transcript by slowing down (R: prevent / protect from) its degradation by ribonucleases in nucleus and cytoplasm.
- 2. Serves as a signal to direct the export of mature mRNA from nucleus to cytoplasm.
- 3. Together with 5' cap, recruit translation initiation factors to form translation initiation complex

Examiner's Comments:

- Fairly good attempt by most candidates.
- The question asked for function**S** (more than 1 function) of **poly-A tail**. 2 functions for 2 marks (1 function for 1 mark, so need not over-elaborate).
- 5' cap prevents mRNA degradation while 3' poly-A tail slows down mRNA degradation. Note the difference in effect on mRNA degradation. Slow down ≠ prevent, as degradation is still happening at the 3' end but at a slower rate.
- A few students mistook poly-A tail of mRNA for telomere (DNA sequence) and elaborated on preventing loss of genes, reaching critical length, etc. Genes are made up of DNA in eukaryotic cells and mRNA does not have a "critical length".
- (d) A misfolded CD protein can be tagged by a type of molecule and subsequently degraded.
 - (i) Identify the type of molecule that can be tagged on a misfolded CD protein.

.....[1]

1. Ubiquitin.

- Not well-answered; many candidates seem to be unfamiliar with this posttranslational modification
- Some candidates spelt ubiquitin wrongly (NOT ubiquitinin)

(ii) Describe how the tagged CD protein is degraded.

.....[1]

1. The tagged CD protein enters a **proteasome** which **hydrolyses** it into smaller peptides.

Examiner's Comments:

- Poorly answered; many candidates seem to be unfamiliar with this post-translational modification
- Degradation is already mentioned in the question. By asking "describe how", the question is testing candidates on the **mechanism** of degradation, which is **hydrolysis** of peptide bonds by **proteasome**.
- Some candidates spelt proteasome wrongly (NOT proteosome)
- Common wrong answers:
 - 1. Proteases ×
 - 2. Ribonucleases × degrade RNA, not proteins
 - 3. Hydrolytic enzymes ★ group of enzymes that hydrolyse covalent bonds such as peptide bonds, glycosidic bonds, etc.
 - 4. Lysosomes × organelles that contain hydrolytic enzymes
 - 5. Ubiquitinase ★ enzyme that catalyses the ubiquitination reaction (tagging of proteins with ubiquitin)

[Total: 10]

- 6 HIV is a retrovirus, which has RNA as its genetic material.
 - (a) Outline the role of gp120 in HIV.
 -[1]
 - 1. It interacts with / binds to the CD4 receptor on the T-helper cells (A: target / host cell).
 - (b) Outline the role of reverse transcriptase in HIV.
 -[2]
 - 1. **Reverse transcriptase** will first catalyse the **synthesis of a DNA strand** complementary to the viral RNA strand, forming a **RNA-DNA hybrid**. (R: convert RNA to DNA)
 - 2. RNA strand is degraded / hydrolysed and a second DNA strand complementary to the first is synthesised to form a double-stranded DNA molecule. (A: double stranded provirus)

Examiner's Comments:

• A number of students used the term "convert" / "conversion" instead of synthesis. The meanings of these two words are *not* the same. The word "convert" doesn't encompass the concept of using RNA as a template to make a new DNA strand.

HIV can remain in a dormant state within infected immune system cells for many years. A person diagnosed as HIV-positive (HIV+) has the virus but does not have symptoms of HIV/AIDS.

The chances of an HIV+ person developing HIV/AIDS can be greatly reduced with a drug treatment programme known as anti-retroviral therapy (ART).

(c) In 2010, the World Health Organization (WHO) published recommendations for the treatment of pregnant women living with HIV. This includes both HIV+ women and women who have developed HIV/AIDS.

The publication recommended that **all** pregnant and breastfeeding women living with HIV should be provided with ART.

Fig. 6.1 shows the number of pregnant women living with HIV, and the number of these receiving ART, between 2005 and 2013, in low and middle income countries.



Fig. 6.1

(i) From the data in Fig. 6.1, it can be calculated that 13% of pregnant women living with HIV received ART in 2005.

Calculate the percentage of pregnant women living with HIV that received ART in 2013.

960 000 / 1 460 000 x 100 = **65.8%**

- 1. Show correct working
- 2. Correct answer (R: any value not 960 000 for with ART & A: 1 440 000 to 1 480 000 for total) (Indicate range of percentage for ease of marking)

Examiner's Comments:

- No marks will be awarded if the wrong group(s) is/are identified. No ecf in such cases.
- Answers should preferably be given in 3 s.f.

answer = % [2]

(ii) Suggest and explain the importance of providing ART to all pregnant and breastfeeding women living with HIV.

.....[3]

<u>Suggest</u>

1. It is an effective treatment to reduce chance of developing HIV/AIDS. [Compulsory point]

<u>Explain</u>

- 2. [Mother-Child Transmission] **decrease HIV transmission** during pregnancy / labour / birth / breastfeeding (A: reduces, number / proportion, of babies born with HIV)
- 3. [Development of AIDS] it can **reduce the number of HIV+ women becoming ill** (with AIDS) / women with HIV/AIDS **dying from the disease**.

- 4. [Epidemiological Control] it can reduce the spread of HIV to other sexual partners (R: spread to child, mark under point 1).
- 5. [Social / Economical Impact] more healthy women can contribute to workforce / take care of children (A: any valid explanation).
- (R: prevent. It should be reduce not complete prevention / cure)

[Any two]

Examiner's Comments:

- Many candidates did not interpret the question correctly. The question is asking for *importance of treatment* not mechanism of ART.
- A significant group of candidates have the misconception that HIV can be inherited. This is <u>incorrect</u>. HIV is most likely transmitted through body fluids such as blood, it does not integrate their genome into germ cells (HIV infect T-helper cells!).
- Many candidates used the wrong term to describe latent stage (R: dormant stage).
- Many candidates also did not understand the concept of latency period. During latency period, the HIV patient <u>can still infect others</u>.
- (d) The existence of viruses, including HIV, continue to challenge the cell theory. Suggest reasons why this is so.

......[2]

- 1. They are <u>acellular</u> and <u>lack cellular organelles</u> (R: metabolic / cellular machineries) [Not exactly the same as cellular organelles, and not a technical term]
- 2. Viruses do not carry out metabolism (e.g. respiration)
- 3. They <u>lack the ability to reproduce on their own independently</u> and can only undergo replication in <u>living cell</u> / <u>host</u>.
- 4. They do not grow / do not undergo developmental changes
- They do not respond to stimuli when outside the host cell. A: They can only evolve by natural selection within a host cell.
 R: obligate parasites without explaining / stating one of the above reasons

R: Statements that indicate viruses behaving like cells (e.g. HIV can replicate in host cells) [Any 2]

Examiner's Comments:

- The concept of Cell Theory was tested in MYE but many candidates were still unable to score 2 marks for this question. Candidates should not assume that a concept will not be tested again (no one knows what is going to come out for A-Level Exams).
- A number of candidates have the misconception that viruses can metabolise inside host cells. Viruses <u>do not</u> metabolise at all (e.g. they do not respire, they do not break down glucose to give energy, etc).
- Similarly, a number of candidates have the misconception that viruses can grow in host cells. They <u>do not</u> grow at all. The concept of growth is different from the concept of replication / reproduction.
- Cell theory does not state that a cell has to be of a certain size, stating viruses are smaller than most cells are not answering the question.
- Not all organelles are membrane-bound. Candidates who state that viruses challenged the cell theory because they do not have membrane-bound organelles are incorrect because prokaryotic cells do not have membrane-bound organelles as well.

9744/J1H2/Promo/2021

7 The *lac* operon is a section of DNA present in the genome of the bacterium, *Escherichia coli* (*E. coli*). The structural genes of the *lac* operon are only fully expressed when the bacteria are exposed to high lactose concentrations.

Fig. 7.1 is a diagram showing the lac operon and a nearby region of the *E. coli* genome.

Fig. 7.1

- (a)
 - (i) Fig. 7.1 shows how the *lac* operon consists of structural genes and regulatory sequences.

Complete Table 7.1 to name each structural gene and its product. [2]

structural gene	name of gene product
lacZ	β – galactosidase (R: incorrect spelling)
lacY	Permease (R: incorrect spelling)
lacA	Transacetylase (R: incorrect spelling)

Table 7.1

1 - 2 rows correct = 1 mark, 3 rows correct = 2 marks.

Examiner's Comments:

- Not always well-answered.
- Common mistakes were wrong spelling due to lack of knowledge of the gene product's correct name.
- (ii) Gene *l* is transcribed all the time to produce its protein. This is constitutive expression.

Suggest why gene / shows constitutive expression.

.....[1]

- 1. No operator present in gene / for repressor to bind to, so transcription occurs all the time
- No repressor to bind to operator (regulatory sequence, I: promoter, R: silencer) in gene *I* to prevent transcription

- Very poorly answered.
- Most candidates did not read and understand the question and proceeded to write about the *lac I* gene product (lac repressor protein) causing the operon to be 'off'.
- The question required candidates to suggest a reason why the lac l gene was always expressed. Only a handful of candidates suggested a plausible explanation.

(iii) Describe the effect of the product of gene I on the lac operon.

......[2]

- 1. Repressor protein binds to operator and blocks promoter;
- 2. **RNA** polymerase unable to bind to promoter and <u>no</u> transcription / <u>expression</u> of <u>structural genes;</u> (A: Reference to *lac Z, lac* Y and *lac A* genes)

Examiner's Comments:

- Not always well-answered.
- Some candidates did not know where exactly the repressor protein would bind with references to promoter region.
- Some candidates were not specific in their answers regarding the binding and action of RNA polymerase.
- Phrase to avoid: 'RNA polymerase does not have access to promoter.....' (Imprecise: 'Access' does not equate to 'binding'! Candidates must clearly state if the RNA polymerase <u>can bind</u> or <u>not bind</u> to the promoter).
- Phrase to avoid: 'RNA polymerase does not bind to the promoter and no transcription of lac operon occurs' (Not precise: It is the *lac* structural genes that are not transcribed!)
- (b) If *E. coli* is placed into a nutrient medium containing lactose, *lac* operon is induced.

Explain why.

......[3]

- 1. <u>Lactose</u> (A: allolactose) binds to <u>allosteric site</u> on <u>repressor protein</u> (R: *lac I* gene) and <u>inactivates</u> the repressor (A: changes 3D conformation of repressor);
- 2. Repressor no longer complementary to operator binding site; (R: active site)
- 3. <u>RNA polymerase</u> can <u>bind</u> to the <u>promoter</u> to initiate the <u>transcription</u> of the <u>structural</u> <u>genes</u> coding for enzymes, on the *lac* operon;

[Total: 8]

- Generally well-answered.
- There were many overly detailed accounts that started with the entry of the lactose, via permease, conversion of lactose to allolactose by beta-galactosidase. This section is not necessary given the weightage and lines given (3 marks and 6 lines only). Candidates can proceed straight to the binding of allolactose to the allosteric site of the lac repressor protein.
- Phrase to avoid: 'The repressor changes conformation after binding of allolactose to repressor protein allosteric site and thus cannot bind to the operator sequence' (Not precise: The inability of the repressor to bind to the operator is because of the change in conformation of the DNA-binding site of the repressor. The changed binding-site conformation is no longer complementary to the operator sequence conformation).

Pointers for Essay Writing

- Writing a Biology essay is **<u>not</u>** about "regurgitating" facts.
- Biology essays are generally synoptic and involves pulling together content and concepts to synthesise a cohesive piece of writing that is based on the context of the question.
- **Question analysis** is an important skill. It is important to identify the <u>requirements</u> of the question before attempting it.
- Once you have analysed the question, you would need to plan. **Planning** is critical part of essay writing, you should *never* start writing without a plan.
- It is an essay, your writing should be in continuous prose (as stated on the instructions). Most essays <u>should not</u> be in table form.
- <u>Check</u> your essay after you have finished writing, it's a good practice and can potentially eliminate careless mistakes.
- Last but not least, without a <u>good grasp of content and concepts</u>, the above pointers would be meaningless.
- 8 (a) Describe the functions of structural and regulatory genes in eukaryotic genome. [10]

Structural Gene

- 1. Codes for structural protein / enzyme <u>AND</u> rRNA / tRNA (I: any named protein other than a transcription factor (e.g. transporter / receptor / named hormone / immunoglobulin / haemoglobin / etc.) (R: if any of these are identified as product of regulatory gene)
- 2. <u>Named</u> structural protein / other protein / enzyme, or tRNA AND <u>function</u> (R: if named protein does not match its function)
- idea that needed for structure / function of cell OR have a phenotypic expression (OWTTE)

Regulatory Genes (Max: 7 Marks)

4. (product) controls, gene expression / transcription ; A promote / prevent / start / stop, gene expression or transcription

General Transcription Factors Genes

- 5. codes for transcription factor
- 6. binds to promoter / proximal control elements
- 7. **involves in initiation of transcription** / **formation of transcription initiation complex** (A: description on recruitment of RNA polymerase to promoter)

Activator Genes

- 8. <u>codes</u> for activator which binds to enhancer on DNA
- 9. Increase the rate of transcription by promoting the assembly of transcription initiation complex (TIC)

Repressor Genes

- 10. codes for repressor which binds to silencer on DNA
- 11. Decrease the rate of transcription by inhibiting the assembly of transcription initiation complex (TIC)

Genes involves in chromatin modifications

- 12. <u>codes</u> for <u>DNA methyltransferases</u> / <u>histone acetyltransferase</u> / <u>histone deacetylase</u> (Any 1)
- 13. decrease or increase the rate of transcription + correct mechanism

Genes involves in post-transcriptional modification

- 14. <u>codes</u> for capping enzyme for 5' Cap / <u>spliceosome</u> / enzyme for 3' polyadenylation (Any 1)
- 15. <u>correct function</u> + <u>correct mechanism</u>

Genes involves in translational control

- 16. codes for translation initiation factors / translational repressor (Any 1)
- 17. <u>correct function</u> + <u>correct mechanism</u>

Genes involves in post-translational modification

18. <u>codes</u> for protease / enzymes for phosphorylation or glycosylation / ubiquitin ligase / proteasomes (Any 1)

19. correct function + correct mechanism

- Most candidates did not do well for this question.
- The majority of the cohort did not have the correct understanding of a gene. The basic unit of a eukaryotic gene consists of a promoter, introns, exons and terminator. A more comprehensive definition of a eukaryotic gene would include the proximal and distal control elements as well. Refer to the diagram below to understand what is a gene.

- Majority of the candidates incorrectly identified control elements and/or non-coding sequences as genes. This is a serious misconception, do seek clarification from your tutor if you are still unsure what a gene is.
- To approach this question, candidate should first draw their understanding from prokaryotic genome (*lac* and *trp* operons), specifically regulatory gene and structural genes. From there, candidate could make an **inference** on what the functions of these genes are **and apply** it in the **eukaryotic genome context**. Next, candidate should draw on their knowledge from the topic on *Control of Eukaryotic Genome* and **synthesise** their answers.
- Majority of candidates incorrectly identified promoter, enhancers and silencers as genes. No
 marks will be awarded for point 5, 6, 8 and 10 because the context is wrong, and the mark
 scheme requires candidates to identify genes coding for transcription factors, activators and
 repressors.
- Another common misconception is that majority of the candidates think telomeres and centromeres are genes. This is incorrect, telomeres and centromeres are non-coding sequences. Even though they have unique functions, it does not mean they are genes.

(b) Discuss how microscopes are used in the study of biology. [10]

Microscope Type & Function

- 1. Use of **light microscope** allows for the **viewing of <u>cells</u>** (I: large organelles)
- 2. Use of electron microscope allows for the ability to view more <u>organelles</u> / <u>cellular</u> <u>structures</u>
- 3. <u>Staining</u> of cells facilitate the viewing of cellular structures under the microscope (OWTTE)

Uses of Microscopes

Measurement

4. Allows for measurement of cells / organelles / cellular structures.

Organelles

- 5. Study / identification of organelles in detail
- 6. Named organelle + one structural feature (e.g. Mitochondrion and cristae)

Microorganisms

- 7. Discovery / study / identification of bacteria / viruses
- 8. Named microorganism + one structural feature

Cell Division

- 9. Study of mitosis / meiosis
- 10. Description of one stage of mitosis / meiosis

Cell Types

11. **Study** / **identification** of **different cell types** (A: differentiation between eukaryotic and prokaryotic cells)

Cell Theory

12. It contributes to the development of cell theory (A: description of cell theory)

Application

13. Any relevant application (e.g. diagnosis) (max 1)

14. AVP ((max 1)

Examiner's Comments:

- This question was generally better answered than 8(a).
- Do note that this mark scheme is brief because there are many possible examples for each point. However, most points still require some form of elaboration.

For example, simply stating that it contributes in cell theory will not garner you a mark for point 12. Some elaboration of how it contributes is still required.

- Many candidates were unable to score marks due to lack of specific details in their writing.
- A number of candidates did not clearly identify the types of microscopes i.e. light and electron microscopes and their specific usage.
- A number of candidates had the misconception that *current* microscopes are powerful and sophisticated enough to observe processes at molecular level (e.g. DNA replication, bacterial transduction, phospholipid bilayer structure, etc). This perception is incorrect.

[Any 10]