2018 'A' Level
H2 Biology
Mark Scheme

PAPER 1 (MCQ)

Question Number	*Key*	*Question Number*	*Key*
1	**B**	16	**B**
2	**A**	17	**B**
3	**C**	18	**D**
4	**A**	19	**C**
5	**C**	20	**A**
6	**B**	21	**C**
7	**C**	22	**D**
8	**D**	23	**B**
9	**C**	24	**B**
10	**D**	25	**A**
11	**A**	26	**B**
12	**A**	27	**A**
13	**C**	28	**A**
14	**D**	29	**C**
15	**D**	30	**A**

PAPER 2 (CORE)

QUESTION 1

Fig. 1.1 represents the molecular structure of a G-protein linked receptor in the cell surface membrane.

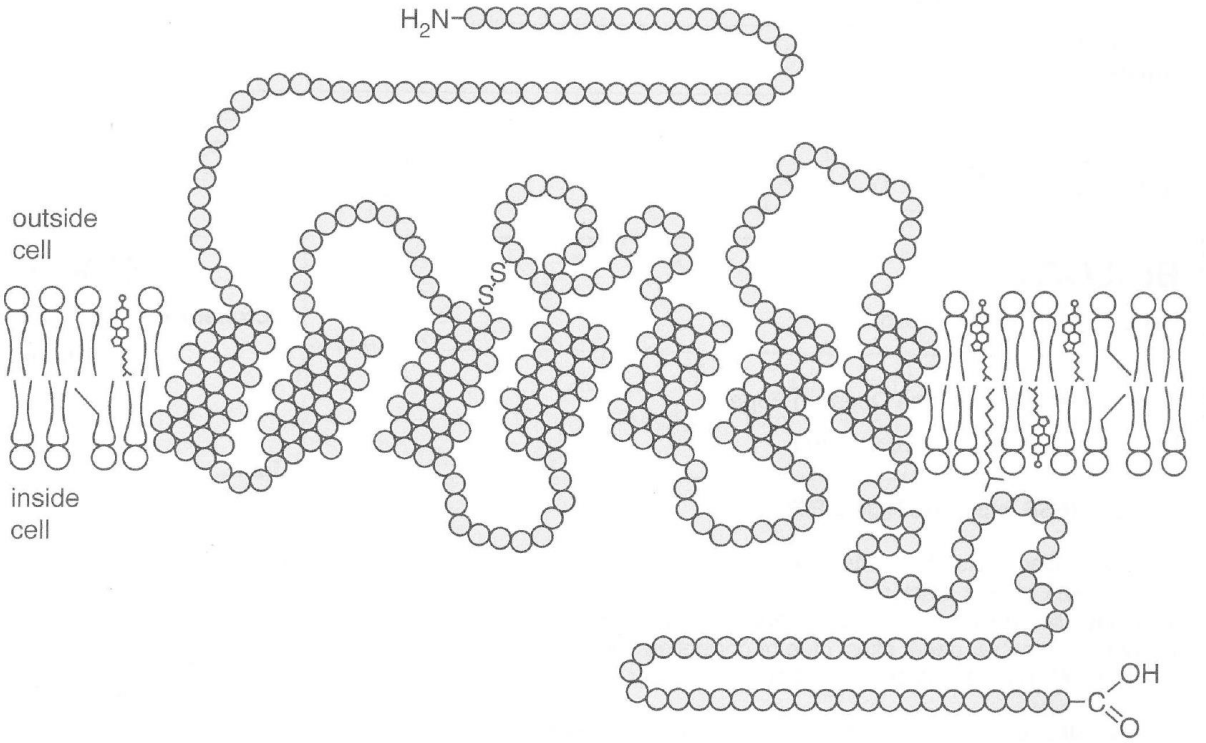


Fig. 1.1

(a) Discuss how the arrangement of molecules in Fig. 1.1 supports the fluid mosaic model of the cell membrane.

…….. [3]

1 'Fluid' refers to the <u>phospholipids</u> and <u>proteins</u> **(such as G-protein linked receptors)** being free to move within the membrane **laterally**

2 and the phospholipids also can move **transversely**

3 'Mosaic' refers to proteins **like G-protein linked receptors** being embedded in the phospholipid bilayer in a scattered or random arrangement.

Examiner's comments:
This question was well answered by the majority of candidates. Some responses were too vague and lacked specific details. For example, a number of candidates referred to 'membrane molecules moving' or 'being randomly scattered' **without identifying these as proteins or phospholipids**. Others
provided a general description of the phospholipid bilayer, including details of hydrophobic and hydrophilic interactions, **without considering Fig. 1.1.**

(b) Explain how the molecular structure of the protein shown in Fig. 1.1 enables it to function as a G-protein linked receptor.

…… [3]

1 The protein consists of an **extracellular domain** that binds to signal molecules/ligand on the extracellular side. [Reject: N and C terminus]

2 It also consists of a **cytoplasmic domain** that binds to G-proteins (on the cytoplasmic side). [Reject: N and C terminus]

3 It contains seven **transmembrane** segments which fold such that the hydrophobic amino acid residues are on the outside, to interact with the hydrophobic hydrocarbon chains of the phospholipids of the membrane.

4 Ref. hydrophilic amino acid residues to interact with the polar phosphate head of phospholipids

5 This anchors the G-protein linked receptor in the membrane and allows it to **function on both sides of the membrane**.
[Max 2 for points 3-5]

Examiner's comments:
Most candidates provided good responses. Some went into excessive detail about protein structure in general, rather than focusing on how the molecular structure of this protein was related to its function.

(c) A large range of stimuli can trigger the activation of G-proteins through G-protein linked receptors. These stimuli include light, calcium ions and hormones such as glucagon.

State the common effect that these diverse stimuli have on G-protein linked receptors.

…… [1]

1 They cause a **change in 3D conformation** of the (G-protein linked) receptors, **exposing its cytoplasmic domain** for the binding of a G-protein.

Examiner's comments:
The majority of candidates recognised the importance of conformational changes in G-protein linked receptors.

(d) Explain how activation of G-protein by the binding of glucagon to the G-protein linked receptor triggers downstream signalling pathways that result in a cascade of enzyme-catalysed reactions.

…… [3]

1 Binding of glucagon to ligand-binding site of G-protein linked receptor results in **conformational changes in the receptor, exposing the cytosolic G-protein binding domain**

2 A G-protein binds, **exchange of GDP for GTP** occurs, GTP-bound G-protein is activated.

3 G-protein dissociates from receptor and moves along the plasma membrane to **activate an enzyme** (embedded in the plasma membrane).

4 Activated enzyme can trigger the next step in the pathway, leading to a cellular response.

Examiner's comments:
Most candidates developed detailed responses that showed good understanding of the glucagon signalling pathway. Some referred to the subsequent effects within the cell rather than the effect of the stimulus on the receptor protein itself.

[Total: 10]

QUESTION 2

Fig. 2.1 shows the effect of increasing temperature on the activity of three protein-digesting enzymes:

- Thermitase from thermophilic *Thermoactinomyces vulgaris*
- Subtilisin from *Bacillus subtilis*
- Modified subtilisin

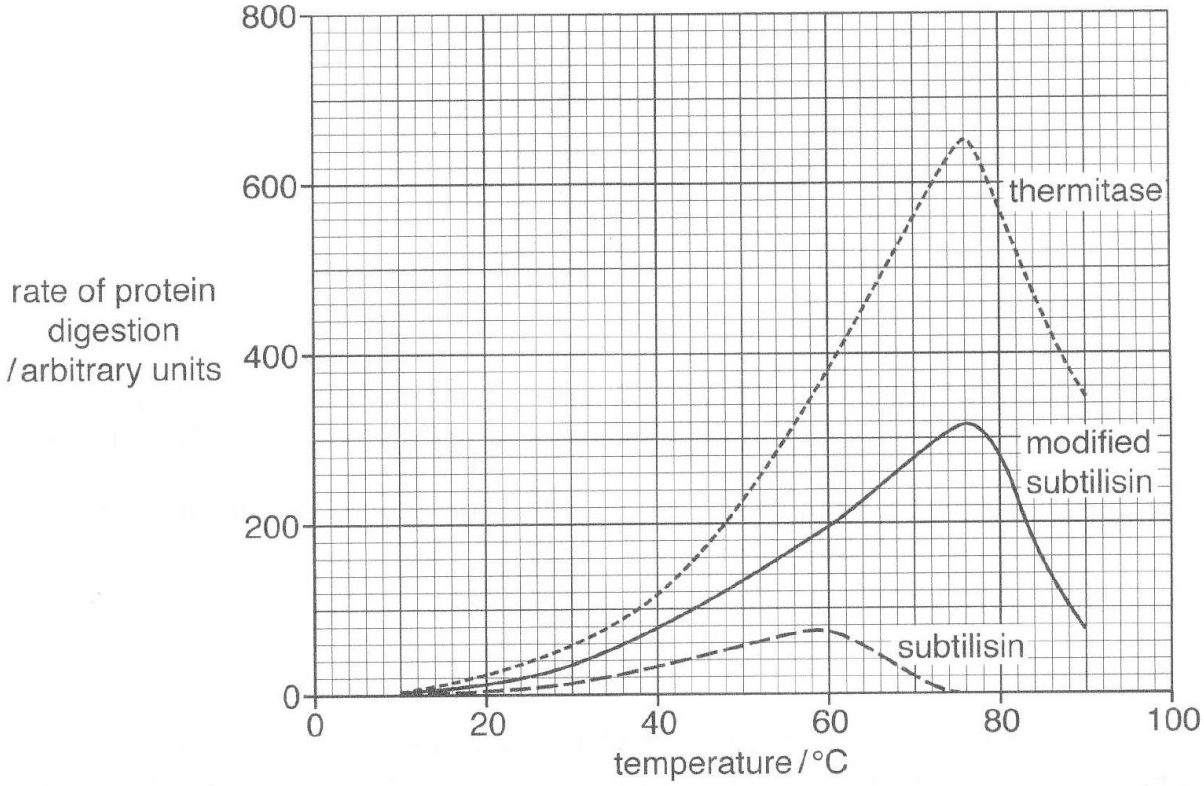


Fig. 2.1

(a) Describe, with reference to Fig. 2.1, the effect of temperature on the rate of protein digestion by thermitase.

…….. [3]

1 When temperature increases from 10°C to 76°C, the rate of protein digestion by thermitase increases from 0 to 650 arbitrary units.

2 The optimum temperature for thermitase is at 76°C, where maximum rate of protein digestion is 650 arbitrary units.

3 When temperature increases from 76°C to 90°C, the rate of protein digestion decreases sharply from 650 to 350 arbitrary units.

Examiner's Comments:

Most candidates gave sufficiently detailed responses. Some did not adhere to the single command word in the question, 'describe', and provided elaborate explanations about why the described changes occurred.

Errors included responses that mistakenly referred to all three enzymes and responses that quoted incorrect readings from the graph.

(b) Explain the effect on thermitase of increasing the temperature above 80°C.

…….. [3]

1 Increasing temperature above 80°C increases the kinetic energy of enzyme molecules, resulting in **thermal agitation**.
2 Intramolecular bonds such as **hydrogen bonds, ionic interactions and hydrophobic interactions** between R groups are disrupted.
3 Thermitase is denatured, loses the **3D conformation** of its active sites.
4 Substrate molecules can **no longer bind** effectively to the active sites, and hence decreases rate of protein digestion.

Examiner's Comments:
Almost all candidates demonstrated a sound understanding of the relevant principles and many good answers were seen. Some responses commented on conformational changes to the active site without indicating the consequences of this, such as loss of active site or poorer 'fit' of substrate.

(c) Modified subtilisin is similar to subtilisin, but has had eight of its amino acids replaced with different amino acids.

Describe **and** explain the effect of this modification on the activity of subtilisin.

…….. [4]

Describe [max 2 marks]

1 The modified subtilisin has a greater rate of protein digestion from temperatures of 10 - 90°C.
2 Modified subtilisin has a greater optimum temperature of 76°C than that of unmodified subtilisin, which is at 60°C.
3 Modified subtilisin is more thermostable as it is fully denatured at higher temperature of 90°C whereas unmodified subtilisin fully denatures at a lower temperature of 76°C.

Explain

4 The eight substituted amino acids are cysteine residues which can form disulfide bonds between their R groups.
5 Disulfide bonds are **strong covalent bonds** which are not disrupted at high temperatures, they **maintain the 3D conformation** of the modified subtilisin at temperatures beyond 76°C.

Examiner's Comments:
Most candidates were able to describe effects of the described modification on the activity of subtilisin. Fewer were able to explain how changes in the amino acids could result in a more thermostable enzyme. Of those who did, many candidates correctly referred to stronger bonds without providing more specific details.

Some candidates did not focus on the modified enzyme shown in the graph but discussed, instead, changes in amino acids that could result in a reduction, or no change, in enzyme activity.

[Total: 10]

QUESTION 3

(a) Define each of the following types of stem cell and name a naturally occurring example of each.

(i) Totipotent

…… [2]

1 [Define] (stem cells which are) able to differentiate into <u>any</u> cell type to form <u>whole</u> organisms, **<u>including</u> the extra-embryonic tissues**.

2 [Example] Cells of the morula.

Examiner's comments:
Some candidates showed confusion in their understanding between totipotent stem cells and pluripotent stem cells.

(ii) Pluripotent

…… [2]

1 [Define] (stem cells which are) able to differentiate into <u>almost</u> any cell type to form any organ or type of cells, **<u>except</u> those of the extra-embryonic tissues**

2 [Example] cells from the <u>inner cell mass of the blastocyst</u>.

Examiner's comments:
The majority of responses were sufficiently detailed and conveyed the understanding that pluripotent stem cells can give rise to 'almost all cell types'. Most candidates were able to give a suitable example.

(iii) Multipotent

…… [2]

1 [Define] (stem cells which are) able to differentiate into a **limited range** of cell type of a **specific lineage**.

2 [Example] Ref. Any cell derived from specialized tissues e.g. <u>Blood stem cells</u> are derived from the bone marrow

(b) Induced pluripotent stem cells (iPSCs) are a type of pluripotent stem cell that are produced directly from adult cells. To produce iPSCs, four genes need to be introduced into adult cells. These genes code for transcription factors.

Explain why these transcription factors are necessary for the production of iPSCs from adult cells.

…… [2]

1 These transcription factors **stimulate the expression and silencing of other genes** (by binding to their respective DNA elements)

2 The **gene products** of these genes enable iPSCs to exhibit characteristics of embryonic stem cells, such as unlimited replicative potential / produce active telomerase / being undifferentiated / AVP.

Examiner's comments:
Good responses clearly expressed the principle of unlocking potency by initiating transcription of inactive genes.

(c) Explain how the use of iPSCs may overcome some of the ethical concerns of using other types of stem cells in medical research and treatment.

…… [2]

1 [Ethical concern] Some believe that life begins at conception and the removal of the inner cell mass of a blastocyst is **equivalent to destroying a human life**.

2 [Explain] IPSCs alleviates the controversial use of embryos or oocytes as no embryos were destroyed in the process as IPSCs are derived directly from adult tissues (no embryos formed in the process).

3 [Ethical concern] Ref. process of oocyte extraction to obtain embryonic stem cells subsequently, is medical risks / is invasive

4 [Explain] Ref. IPSCs are obtained from somatic cells, method of extraction can be non-invasive (e.g. doing skin biopsy).

5 [Ethical concern] Ref. risk of tissue rejection by patient's immune system if the embryonic stem cells are derived from donated oocytes / oocytes used are not from the patient

6 [Explain] Ref. IPSCs are obtained from patient's **own** somatic cells, lower risk of tissue rejection

[Any 2 *sets* of points]

Examiner's comments:
Most candidates were only able to identify one ethical concern that can be overcome by using iPSCs, which was that of avoiding destruction of embryos. Few candidates recognised the benefit of using a patient's own cells to generate iPSCs.

Marker's comments:
Candidates are expected to pick relevant ethical concerns which IPSC can address, and not blindly provide ethical implications from the notes e.g. points about privacy and confidentiality, knowledge of healthcare professionals etc are not relevant.

[Total: 10]

QUESTION 4

Fig. 4.1 shows the main structural features of the influenza virus.

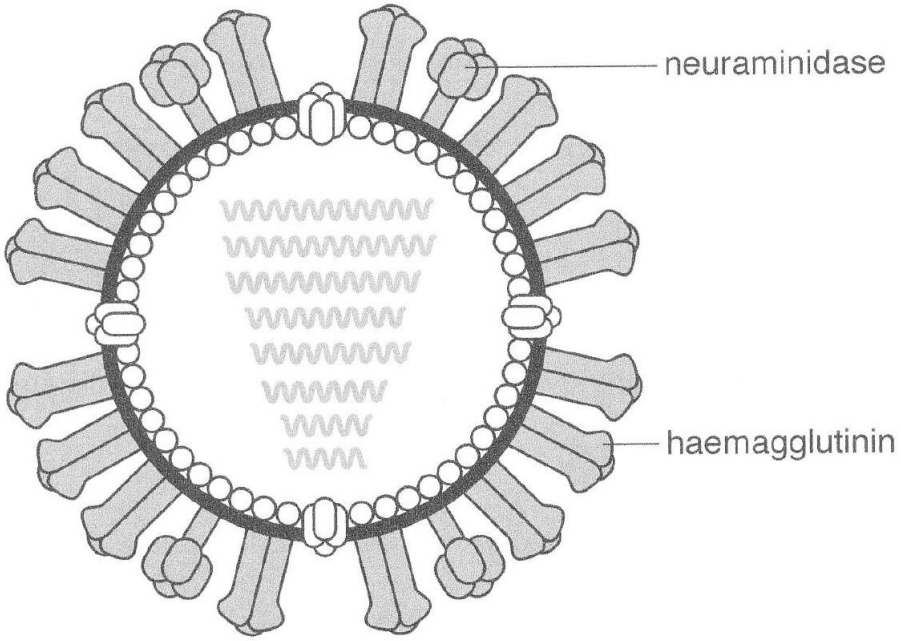


Fig. 4.1

(a) With reference to structures shown in Fig. 4.1, explain how an enveloped virus such as influenza:

(i) enters a host cell

,…..…….. [4]

1 Haemagglutinin on the viral envelope **recognises and binds** to sialic acid receptors on host **plasma membrane**
2 The virus enters host cell by receptor-mediated endocytosis, where host plasma membrane invaginates and pinches off
3 placing the virus in an endocytic vesicle / endosome.
4 **Acidic conditions** in the endosome causes the **viral envelope to fuse with the membrane of the endocytic vesicle** releasing viral nucleocapsid into host cell's cytoplasm.
5 Viral capsid is then enzymatically removed and the viral genome is released and transported into the nucleus of the host cell.

(ii) exits a host cell.

…..…….. [2]

1 The host plasma membrane pinches off to form the viral envelope via **budding**, enclosing the viral nucleocapsid and enzymes.
2 The **sialic acid is cleaved** and removed from the envelope of the new viral particle by neuraminidase, which prevents agglutination of viruses.

Fig. 4.2 shows two examples of antigenic shift in the influenza virus. The resulting human viruses were responsible for the 1957 and 1968 influenza epidemics.

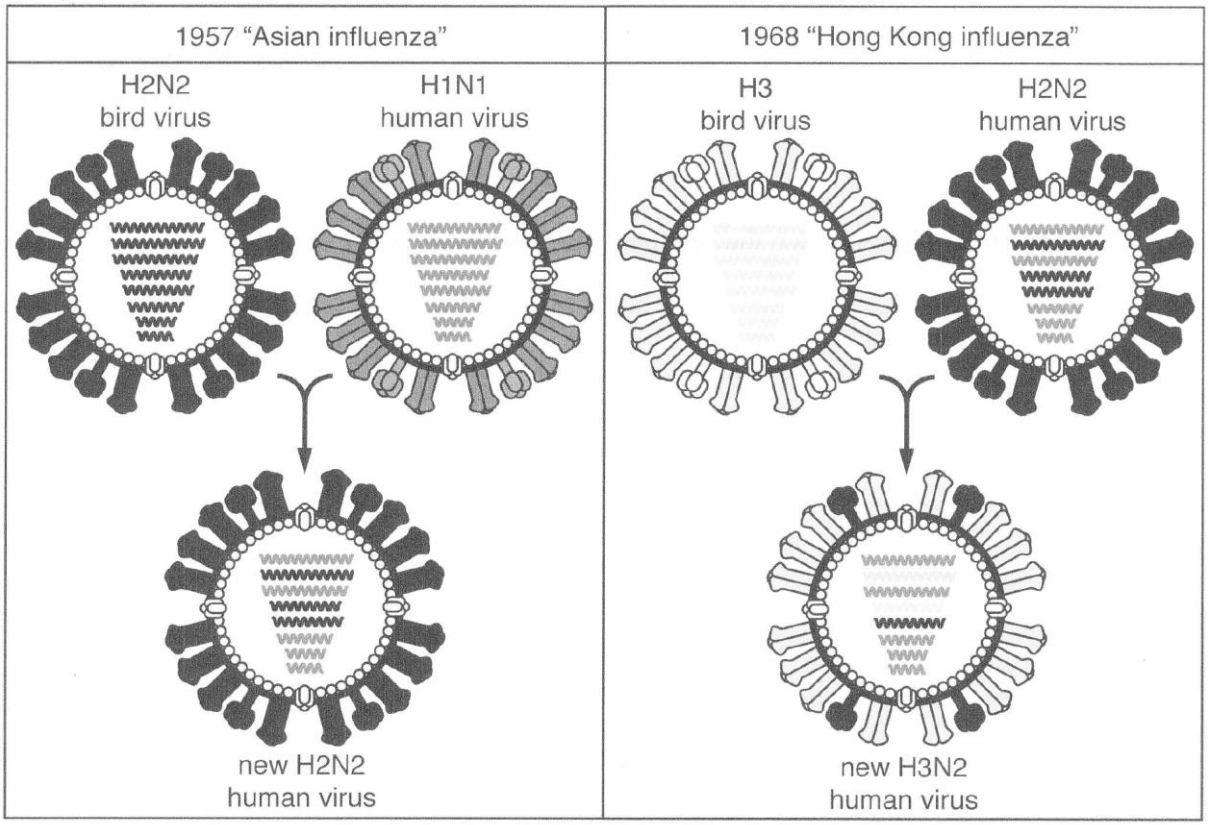


Fig. 4.2

(b) With reference to Fig. 4.2, explain how antigenic shift works.

……..…….. [2]

1 When H2N2 bird virus and H1N1 human virus **infects the same host cell**,
2 There is **reassortment of the viral RNA segments**, giving rise to a new combinations of RNA segments in new H2N2 human viral particles.

OR

3 When H3 bird virus and H2N2 human virus **infects the same host cell**,
4 There is **reassortment of the viral RNA segments**, giving rise to a new combinations of RNA segments and resulting in new combinations of surface antigens haemagglutinin and neuraminidase to produce a new H3N2 human virus strain.

(c) Viruses such as influenza rapidly accumulate gene mutations.

Explain the advantage of this to the virus.

……..…….. [2]

1 Rapidly mutating viruses are not detected by **existing** antibodies present in the immune system, hence antibodies do not bind to the viruses to stop its replication / unable to recruit macrophages to destroy the virus.
2 It is difficult to provide one vaccination that will protect an individual for life because of the genetic variability of rapidly mutating viruses.

3 The virus can acquire the **ability to infect other organisms** which it previously is not able to infect.

[Q4 Total: 10]

QUESTION 5

(a) Describe **two** ways in which the *lac* operon is **similar** to the *trp* operon.

…….. [2]

1 They are both repressed by a repressor molecule / they are both negatively controlled.

2 The structural genes are under the control of a single promoter and operator.

(b) Describe **four** ways in which *lac* operon is **different** to the *trp* operon.

…….. [4]

1 Lac operon is an inducible operon while trp operon is a repressible operon

/ The substrate molecule lactose serves as an inducer that signals the induction of the lac operon while the end-product tryptophan serves as a co-repressor that signals the repression of the trp operon.

2 Lac operon codes for enzymes involved in a catabolic pathway while trp operon codes for enzymes in an anabolic pathway.

3 Lac operon is under dual control or a repressor and activator while trp operon is only negatively controlled by a repressor.

4 Transcription of structural genes in lac operon is normally switched off while transcription of structural genes in trp operon is normally switched on.

(c) Suggest the advantages to bacteria of arranging some genes in operons.

…….. [4]

1 A single signal is able to switch on or off the expression of several genes.

2 As several genes are controlled by the same promoter and operator, it allows **bacterial genomes to be smaller / more compact**, to fit inside the small bacterial cell.

3 Grouping a few functionally-related genes under same regulatory control allows bacteria to **rapidly adapt to changes in the environment**.

4 It saves energy and resources as all proteins / enzymes encoded by operons are only synthesized when required.

[Q5 Total: 10]

QUESTION 6

Fig. 6.1 shows an example of the sequence of temperature changes required for one cycle of a polymerase chain reaction (PCR).

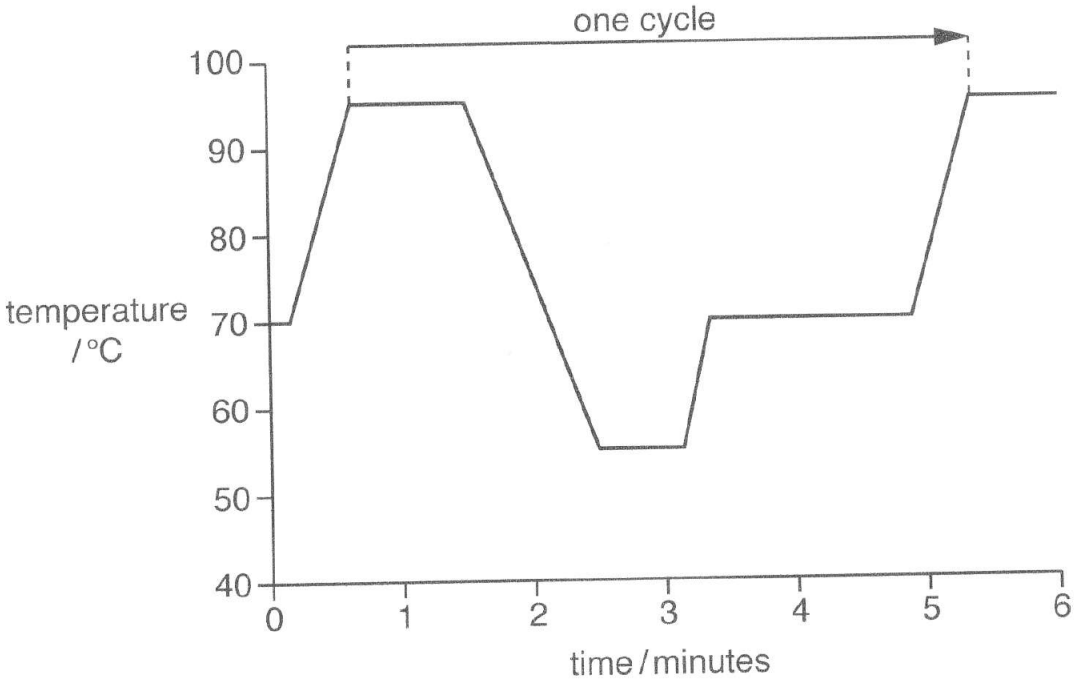


Fig. 6.1

(a) During the PCR cycle shown in Fig. 6.1, three different temperatures are required.

Explain why each of these **three** different temperatures is required during one PCR cycle.

…….. [3]

1 Heating the mixture to 95°C supplies **enough kinetic energy** to **denature** DNA molecules into **single strands** by **breaking the hydrogen bonds** between complementary base-pairs;

2 Cooling the mixture to 55°C to enable forward and reverse DNA primers to **anneal** to **complementary** single-stranded DNA templates at sequences flanking the target sequence at 3' ends

3 Heating to 70°C allows the thermostable DNA polymerase to **optimally** catalyse the **synthesis of a complementary DNA** strand for each template DNA from the 3'OH end.

Examiner's comments:
Most candidates provided detailed responses and many good explanations were seen. Some candidates did not understand that after setting up the reaction, the sequence of different temperatures continually cycles through without any further intervention. These candidates often linked stages with particular actions, such as adding primers, **without considering the significance of the particular temperature, e.g. to allow the primers to anneal or bind.**

(b) Explain the role of primers in the polymerase chain reaction.

…… [2]

1 DNA primers (anneal to 3' ends of the single-stranded DNA templates to) provide a free 3' OH group

2 for Taq DNA polymerase to add **free deoxyribonucleotide complementary** to the template for the synthesis of a complementary DNA strand.

(c) PCR products are usually separated by gel electrophoresis.

Outline the main principles that allow gel electrophoresis to separate DNA fragments.

…… [3]

1 DNA is negatively-charged due to negatively-charged sugar-phosphate backbone.

2 Hence, DNA **moves towards the positively-charged electrode** through an agarose matrix which acts as a molecular sieve.

3 DNA fragments separated by size, where **shorter DNA fragments move faster [Reject: further] than longer ones**.

(d) Marker DNA and three DNA samples were separated by gel electrophoresis.

Fig. 6.2 shows the DNA banding patterns after visualisation.

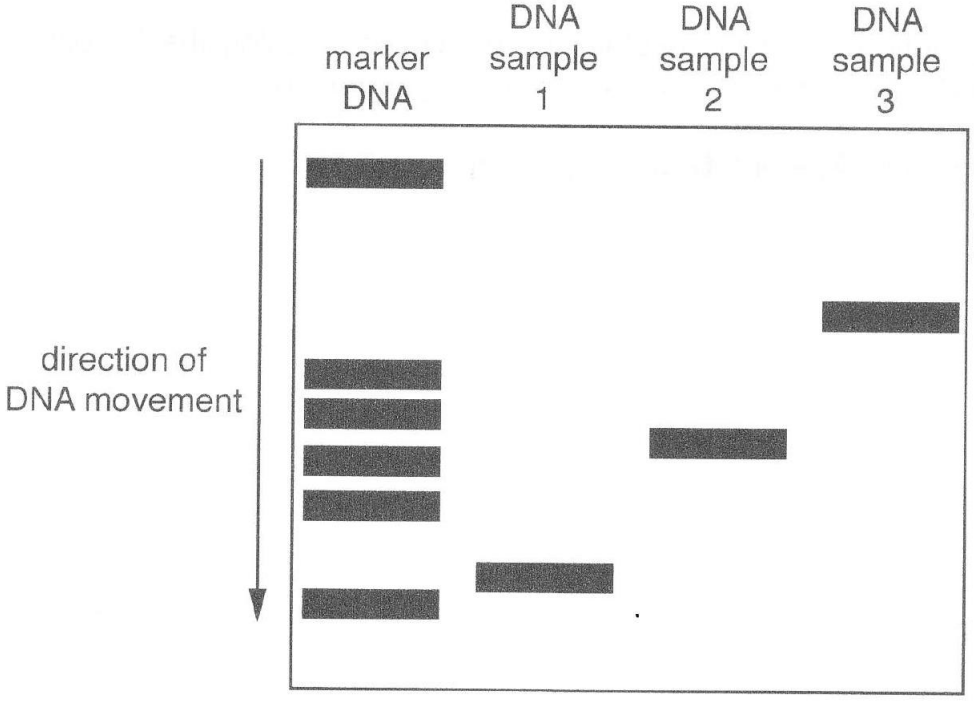


Fig. 6.2

Explain why marker DNA was included in this experiment

…… [2]

1 Marker DNA contains a set of DNA fragment of **known molecular sizes**

2 Which are used to **estimate** the sizes of unknown fragments.

Examiner's comments:
Many candidates made vague references to the use of marker DNA to identify DNA fragments, **rather than to allow an estimate of the actual size of individual fragments.**

[Q6 Total: 10]

QUESTION 7

Two pure-breeding varieties of summer squash, one producing white fruit, and one producing green fruit, were crossed. All the F1 generation progeny produced white fruit.

The F1 plants were then self-pollinated. In the F2 generation, 236 plants produced white fruit, 56 plants produced yellow fruit and 12 plants produced green fruit.

The control of fruit colour in the summer squash is an example of dominant epistasis. Dominant epistasis is expected to result in a 12 : 3 : 1 ratio of offspring phenotypes in the F2 generation.

(a) Explain the term epistasis in this context.

…… [3]

Scenario (I)

1 [General definition] Ref. (dominant) **epistasis** / the phenotypic expression of a gene at one locus **alters** the phenotypic expression of a gene at a second locus / **Allele A** is epistatic to gene locus **B/b** ; [**REJECT**: Gene A/a]

2 In this context, the presence of **a dominant** allele at the first gene locus, allele **B**, codes for **enzyme** that converts green pigment to yellow pigment;

3 while presence of **two copies / homozygous recessive bb** results in green pigment (coupled with aa). [**REJECT**: allele a, i.e. to write genotype]

4 The presence of a dominant allele at a second gene locus, allele **A**, codes for an **inhibitor** that prevents the deposition of either yellow or green pigments, resulting in white fruits.

Scenario (II)

1 [General definition] Ref. (dominant) **epistasis** / the phenotypic expression of a gene at one locus **alters** the phenotypic expression of a gene at a second locus / **Allele B** is epistatic to gene locus **A/a** ; [**Note for markers** : allow ecf if stated epistatic to]

2 In this context, the presence of **a dominant** allele at the first gene locus, allele **A**, codes for **enzyme/protein** that converts green pigment to yellow pigment;

3 while presence of **two copies / homozygous recessive aa** results in green pigment (coupled with bb). [**REJECT**: allele b, i.e. to write genotype]

4 The presence of a dominant allele at a second gene locus, allele **B**, codes for an **inhibitor** that prevents the deposition of either yellow or green pigments, resulting in white fruits.

[**Scenario (I)**
ACCEPT: allele B codes for enzyme that result in **conversion** of precursor (green) to yellow pigment and genotype bb codes for non-functional enzyme and green pigment remains. BUT **REJECT**: Allele A codes for inhibitor that inhibit the enzyme codes by gene B/b.

REJECT: allele B codes for enzyme that results in **(de novo) synthesis** of yellow pigment and genotype bb codes for enzyme that results in the **(de novo) synthesis** of green pigment. Allele A codes for inhibitor that inhibit enzymes coded by gene B/b.

Scenario (II)
ACCEPT: allele A codes for enzyme that result in **conversion** of precursor (green) to yellow pigment and genotype bb codes for non-functional enzyme and green pigment remains. BUT **REJECT**: Allele B codes for inhibitor that inhibit the enzyme codes by gene A/a.]

REJECT:
1) Allele that codes for protein that allow deposition of pigment.
2) Biochemical pathway that converts precursor (white) to coloured pigment.

Examiner's comments:

References to alleles were often not sufficiently clear and few candidates were able to explain the difference between green fruit and yellow fruit.

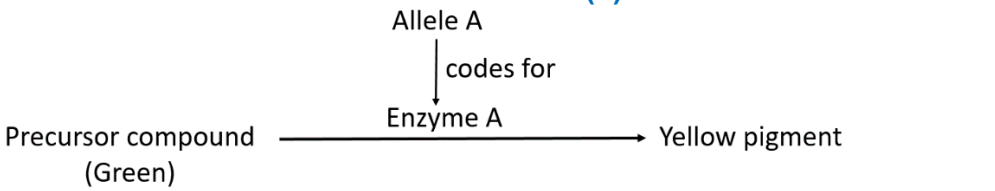
Scenario (I) Allele A codes for a inhibitor

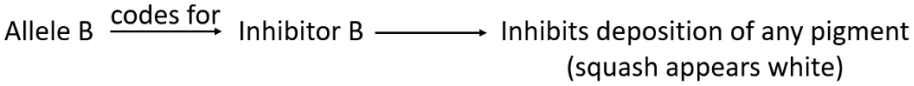
9 A_B_ white
3 A_bb white } 12
3 aaB_ yellow
1 aabb green

Scenario (II) – Allele B codes for a inhibitor

9 A_B_ white
3 aaB_ white
3 A_bb yellow } 12
1 aabb green

Possible biochemical basis: Scenario (II)





(b) Draw a genetic diagram to explain the results of crossing the F1 offspring to produce the expected 12 : 3 : 1 phenotypic ratio in the F2 generation.

Use the symbols **A**, **a**, and **B**, **b** to represent the alleles.

…… [4]

(I) Scenario 1:

F1 phenotypes:	White fruit			x	White fruit
F1 genotype:	AaBb			x	AaBb
F1 gametes:	AB	Ab	aB	ab	x	AB	Ab	aB	ab

F2 genotypes:

Scenario 1 : ['A' codes for inhibitor]

Punnett square:

	AB	Ab	aB	ab
AB	AABB (white)	AABb (white)	AaBB (white)	AaBb (white)
Ab	AABb (white)	AAbb (white)	AaBb (white)	Aabb (white)
aB	AaBB (white)	AaBb (white)	aaBB (yellow)	aaBb (yellow)
ab	AaBb (white)	Aabb (white)	aaBb (yellow)	aabb (green)

F2 phenotypes: white : yellow : green

F2 phenotypic ratio: 12 : 3 : 1

(II) Scenario 2 : ['B' codes for inhibitor]

Punnett square:

	AB	Ab	aB	ab
AB	AABB (white)	AABb (white)	AaBB (white)	AaBb (white)
Ab	AABb (white)	AAbb (yellow)	AaBb (white)	Aabb (yellow)
aB	AaBB (white)	AaBb (white)	aaBB (white)	aaBb (white)
ab	AaBb (white)	Aabb (yellow)	aaBb (white)	aabb (green)

1 F1 genotypes
2 F1 gametes (circled)
3 F2 genotypes / punnett square
4 F2 corresponding offspring phenotypes with genotypes
5 F2 phenotypic ratio of offspring phenotypes [no ecf from (a) ; allow ecf if mark point 4 is not awarded but provided mark point 3 and ratio number are correct]

Guidance for markers:
(1) ignore label e.g Parental or F1 genotype ;
(2) No marks if genetic cross is presented as linkage case or sex-linkage etc ;

Examiner's comments:
Most candidates were able to explain the results using genetic diagrams. Some did not link the F2 genotypes with their correct phenotypes.

(c) A chi-squared test was carried out to compare the expected number of each phenotype in the F2 generation with the observed number. This is shown in Table 7.1.

Table 7.1

	observed (O)	expected (E)	$(O - E)^2/E$
white	236	228	0.28
yellow	56	57	0.02
green	12	19	2.58
		χ^2 =	2.88

Critical values of this chi-squared distribution are shown in Table 7.2.

Table 7.2

degrees of freedom	probability								
	0.99	0.95	0.90	0.75	0.50	0.25	0.10	0.05	0.01
2	0.02	0.10	0.21	0.58	1.39	2.77	4.61	5.99	9.21

Explain how the chi-squared test value of 2.88 supports dominant epistasis as the correct explanation of these results.
…….. [3]

1 0.10 < p < 0.25. Since p-value is larger than 0.05,
2 there is **no significant difference** between observed and expected results. Any difference is due to chance.
3 The predicted phenotypic ratio of **12 white : 3 yellow : 1 green is correct**, (hence epistasis is supported as the correct explanation of these results.)

OR

4 As chi-squared test value of 2.88 is smaller than the critical chi-squared value of 5.99, at p=0.05,
5 there is **no significant difference** between observed and expected results. Any difference is due to chance.
6 The predicted phenotypic ratio of **12 white : 3 yellow : 1 green is correct**, (hence epistasis is supported as the correct explanation of these results.)

Examiner's comments:
Many candidates demonstrated a good understanding of how to interpret the results of a chi-squared test. A number of candidates used the chi-squared data to give the opposite interpretation to that expected, e.g. deviation is significant so the hypothesis that dominant epistasis is the correct explanation should be rejected. Some of these appeared to have incorrectly equated the *p* value at 0.5 to a five per cent probability.

[Q7 Total: 10]

QUESTION 8

Different respiratory substrates are available to working muscles to maintain ATP levels required for muscle contraction. These include glucose and fatty acids from the blood and glycogen in the muscle.

Table 8.1 shows the percentage contribution to muscle contraction of each of these respiratory substrates for an athlete, during a long-distance run of 40km. Results are shown at four different times from the start of the run.

Table 8.1

time / minutes	percentage contribution to muscle respiration		
	glucose from blood	fatty acids from blood	glycogen in muscle
40	27	37	36
90	41	37	22
180	36	50	14
240	30	62	8

(a) Describe the changes in the percentage contributions to muscle respiration of fatty acids from blood and glycogen in muscle during this long-distance run, as shown in Table 8.1.

…… [3]

1 The percentage contribution to muscle respiration of fatty acids from blood remained constant at 37% from 40 to 90 min

2 And **increased from 37% to 62%** from 90 to 240 min.

3 The percentage contribution to muscle respiration of glycogen in muscle decreased from 36% to 8% from 40 to 240 min.

(b) During the long-distance run, ATP may be generated at times using the type of respiration that normally occurs under anaerobic conditions. However, the athlete cannot use this type of respiration continuously throughout the whole period of the long-distance run.

Suggest why this type of respiration **cannot** be used continuously by the athlete to generate ATP during the long-distance run.

……[2]

1 Anaerobic respiration produces only 2 net ATP per glucose molecule and would deplete the glucose levels too rapidly.

2 Anaerobic respiration in mammalian muscle cells produces lactic acid, which if accumulated, would **decrease pH** in muscle cells and result in muscle lethargy or damage.

AVP: Conversion of lactic acid back to glucose/glycogen (oxidation of lactic acid) results in **oxygen debt**

(c) Explain the different patterns of change in the percentage contributions to muscle respiration of glucose from blood and glycogen in muscle during the long-distance run, as shown in Table 8.1.

……[4]

1 The percentage contributions to muscle respiration of glucose from blood **increases from 27% to 41% between 40-90 mins and decrease back to 30% at 240 mins** of the long-distance run while percentage contribution to muscle respiration of glycogen in muscle decreased from 36% to 8% during 40-240mins of the run.

2 Increase in percentage contribution of glucose results in decrease in glucose levels in the blood, it is detected by α–cells in the pancreatic islets of Langerhans, which are stimulated to secrete glucagon.

3 Glucagon stimulates a series of signaling events which eventually leads to the activation of glycogen phosphorylase.

4 Glycogen phosphorylase catalyses the **conversion of glycogen in muscles into glucose-6-phosphate**, restoring the blood glucose levels to normal and depleting the glycogen stores.

5 Glucagon also cause the **increase mobilization of fatty acids** (increase percentage contribution of fatty acids from 37% to 62%) for respiration therefore decreasing the percentage contribution of glucose back to 30%

(d) Suggest why fatty acids are not used more in the earlier stages of the long-distance run.

……[1]

1 Fatty acids need to undergo additional reactions to be broken down into smaller molecules which can enter the Krebs cycle/fatty acid oxidation occurs at a slower rate compared to glycogen oxidation

R: More energy required to break down fatty acid

R: Ester bond more difficult to break down

[Q8 Total : 10]

QUESTION 9

Fourteen different species of Darwin's finches are found in the Galapagos Islands. These finches have evolved from a common ancestor during the past 1.5 million years.

As the finches have adapted to different food resources on the different islands, their beak lengths, beak shapes, feeding habits and diets have become more diverse.

Fig. 9.1 shows beak lengths and body masses of three species of Darwin's finches.

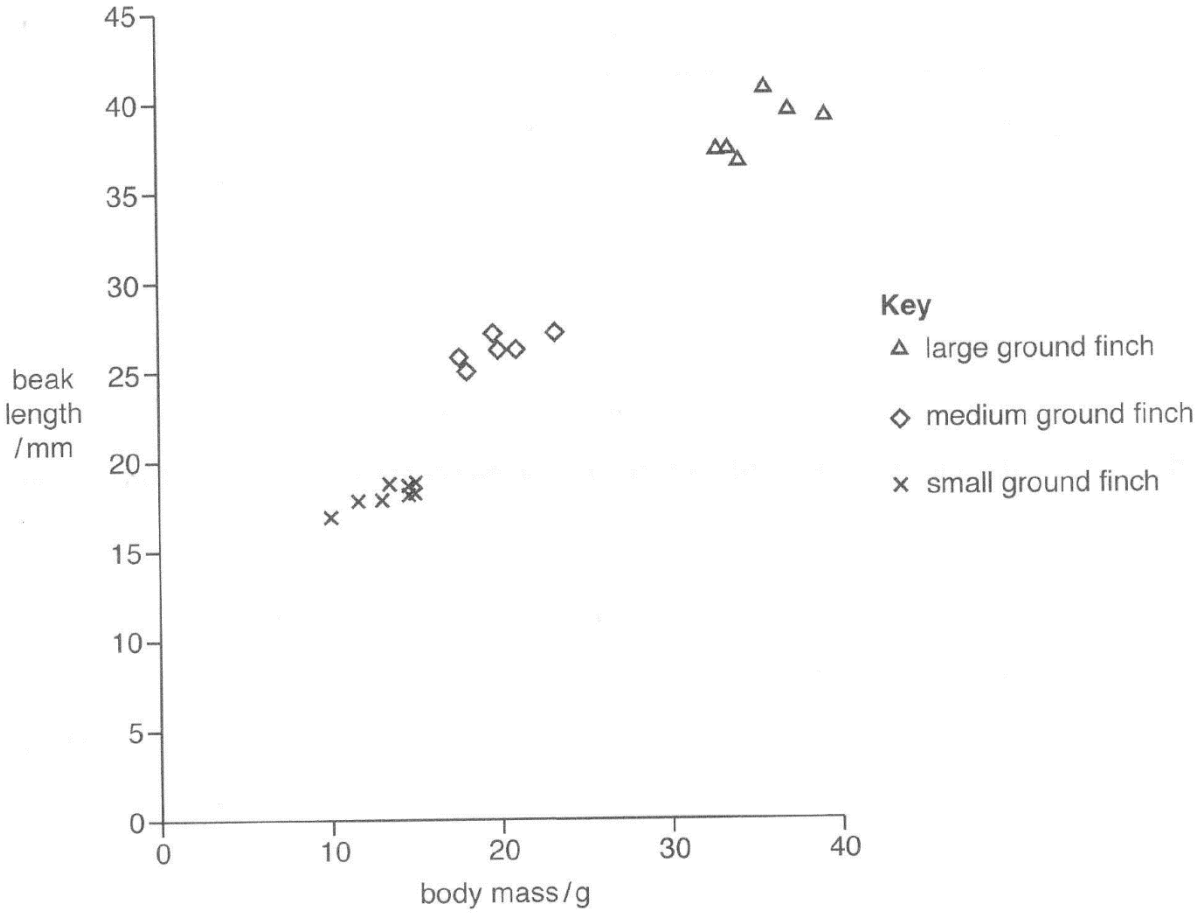


Fig. 9.1

(a) With reference to Fig. 9.1, explain why the large ground finch, medium ground finch and small ground finch are considered to be three separate species.

……[3]

1 There is genetic variation within the population of finches. The large, medium and small ground finches have distinctively different beak lengths (36-42mm, 18-23mm and 16-19mm respectively) and body masses (32-39g, 18-23g and 10-15g respectively);

2 The different island with different food resources selects for finches with different beak length. Different beak length are at selective advantage at different islands resulting in <u>geographical isolation</u> and hence no interbreeding between the 3 types of finches.

3 As Fig. 9.1 show that they have distinctively different beak lengths and body masses, it suggests that three species **do not interbreed** with each other/ there

is some form of **reproductive isolation** between the 3 species, hence they are considered to be separate species under the **biological species concept**.

(b) Fig. 9.2 shows the Galapagos Islands today and 630 000 years ago, when the sea level was much lower.

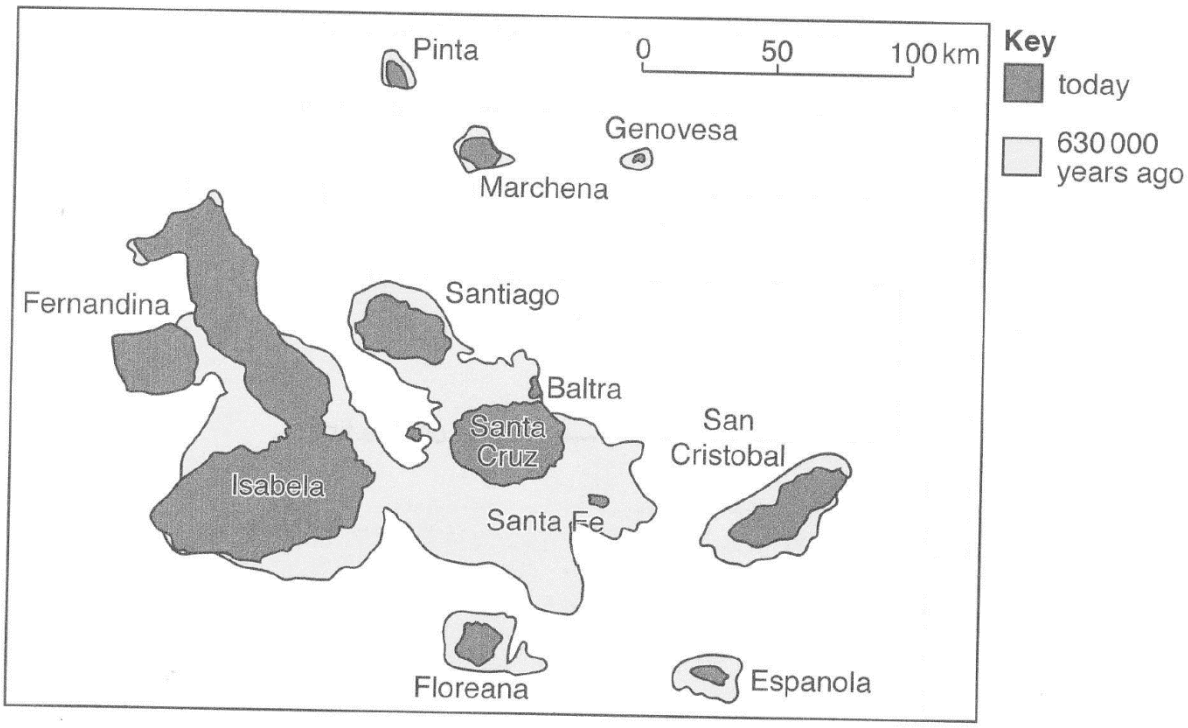


Fig. 9.2

The evolution of Darwin's finches provides examples of both micro-evolution and macro-evolution.

Discuss how the information in Fig. 9.1 and Fig. 9.2 shows how these two processes may have contributed to the evolution of Darwin's finches.

……[4]

Micro-evolution:

1 Genetic drift and natural selection occurs to result in different **changes in allele frequencies** in the different populations of finches.

2 Different selection pressures on different islands favored different beak lengths and body mass; finches with advantageous beak lengths and body mass survive more and reproduce, **passing down their alleles** to offspring.

3 Different mutations accumulate over time in different finch populations on different islands.

[max 2 marks]

Macro-evolution:

4 As land mass separates into individual islands due to higher sea levels, geographical isolation occurs and **disrupted gene flow** between populations of finches on different islands.

5 Over time, changes in allele frequencies can lead to allopatric speciation where different finch populations can no longer interbred with each other.

(c) Fig. 9.3 shows the phylogeny based on whole genome sequencing of the six closely related species of Darwin's finches that have evolved most recently.

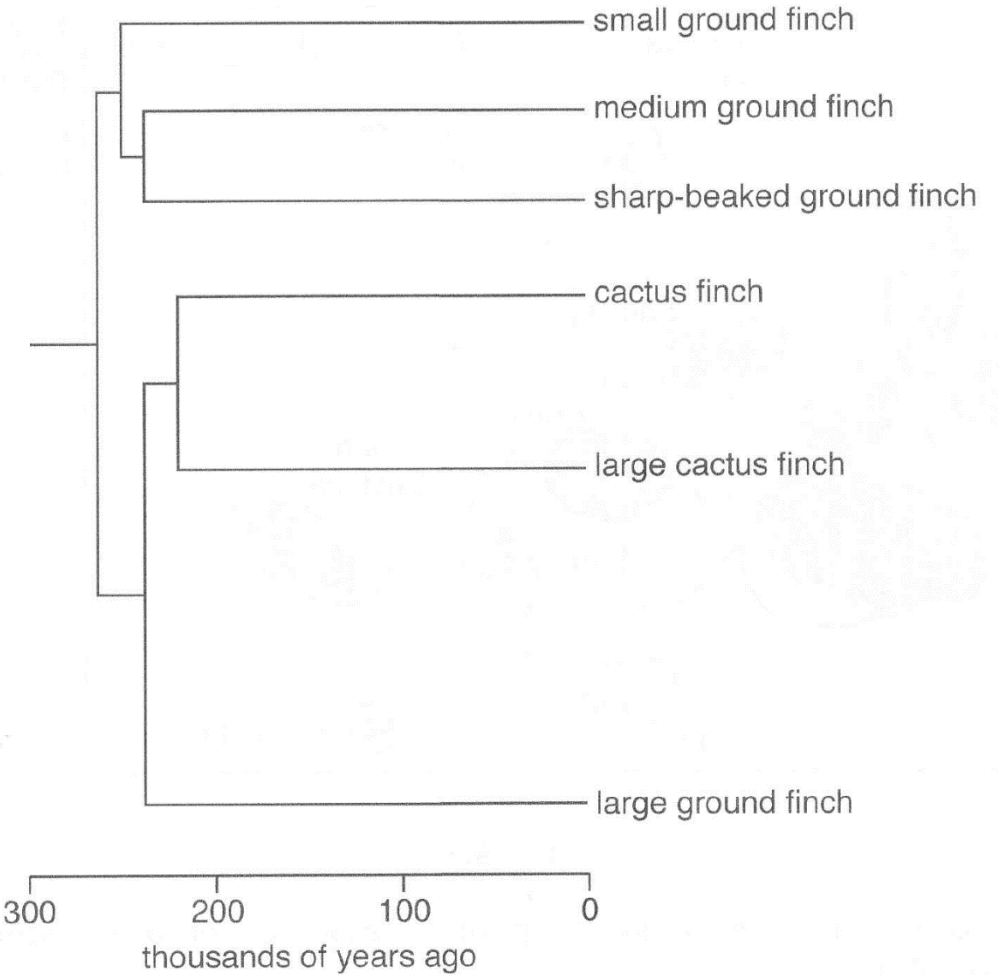


Fig. 9.3

Describe the advantages of using genome sequences in reconstructing phylogenetic relationships.

…… [3]

1 Molecular data such as genome sequences are **quantifiable**, in abundance and open to statistical analysis while there is little morphological data available.

2 Molecular data can be easily described in an unambiguous manner and facilitates the **objective assessment** of evolutionary relationships while morphological data may differ depending on the way in which it was classified.

3 Molecular data is based strictly on heritable material while morphological data is based on anatomical characters which may be influenced by environmental factors as well as variation due to genotype of the organism.

4 All organisms can be compared with the use of some molecular data as all living organisms have nucleotides, so molecular data can be collected from any organisms.

5 DNA information provides abundance of data for analysis and it allows easy homology assessment while morphological traits are few and it is often difficult to assess homology for less complex structures.

[Max 3 marks]

[Q9 Total: 10]

QUESTION 10

Fig. 10.1 shows the percentage of the population having the TB vaccination and the rate of TB transmission from 1980 to 2010 in country **P**.

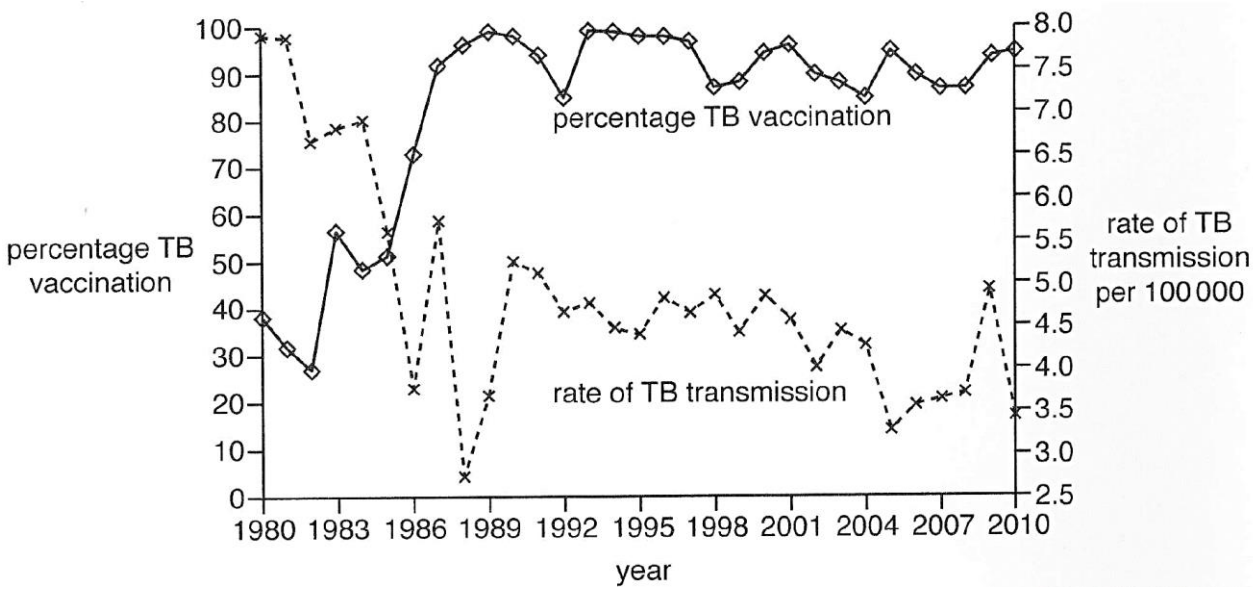


Fig. 10.1

(a) Describe **and** explain the changes in the rate of TB transmission that are shown in Fig. 10.1.

…….. [3]

1 **[Description]** There is an overall **decrease** in rate of TB transmission from 1980 to 2010, from 7.9 per 100 000 to 3.5 per 100 000.
2 **[Explanation]** This is due to the overall **increase** in percentage TB vaccination in the same period of time, from 38% to almost 95%.
3 **[Explanation]** TB vaccinations **stimulate the production of antibodies** against TB (virus) / ref to **active immunity**, so vaccinated individuals become protected from TB. Hence, the higher the percentage TB vaccinations, the lower the rate of TB transmission.

Examiner comments: Most candidates were able to describe and explain the changes in the rate of TB transmission.

(b) Fig. 10.2 shows the percentage of the population having the TB vaccination and the rate of TB transmission from 1980 to 2010 in country **Q**.

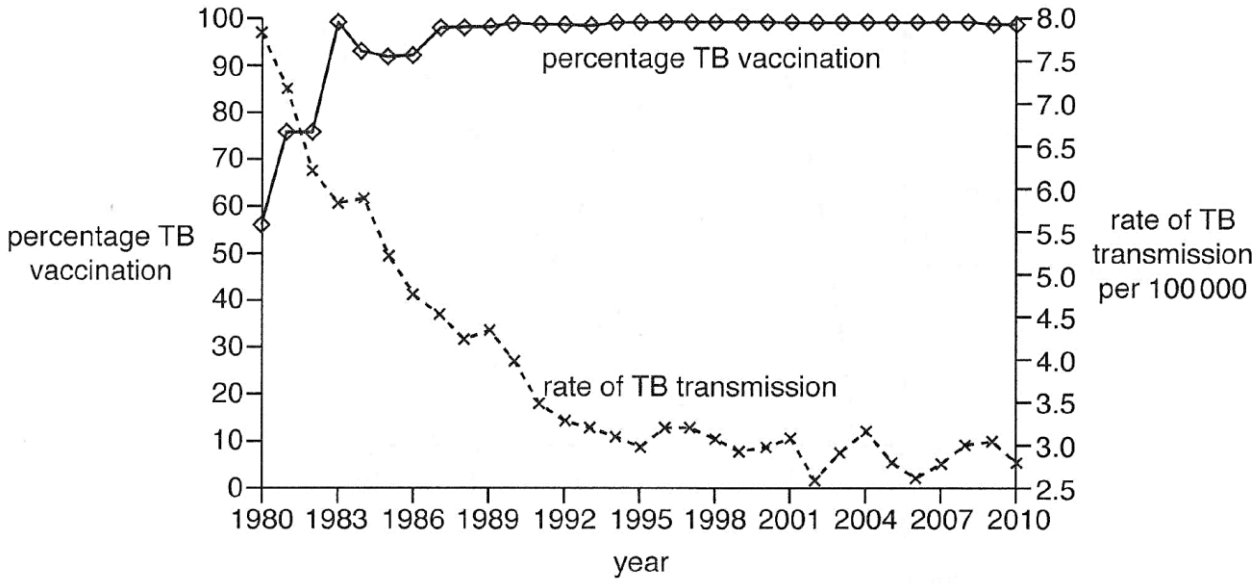


Fig. 10.2

Suggest reasons for the differences in the rates of TB transmission between country **P** and country **Q**, as shown in Fig. 10.1 and Fig. 10.2.

…….. [2]

1 **[Description]** From 1992 to 2010, the rate of TB transmission in country P ranged between 3.3 to 4.9 per 100 000, which is **higher than** that in country Q, which ranged between 2.6 to 3.3 per 100 000.
2 **[Reason]** This is because TB vaccination in country P **fluctuated** between 85% to 99% in that time period , while TB vaccination is **constantly** at 100% in country Q.
3 **[Reason]** A lower rate of TB vaccination results in poorer protection against TB transmission for the population.

Examiner comments: Many candidates recognised differences in the percentage TB vaccination in the two countries and developed their answers around this. Some candidates only gave a description of the graph.

[Q10 Total: 5]

QUESTION 11

Fig. 11.1 shows the concentration of dengue virus antigen and antibody in the blood of a person following infection with the virus for the first time.

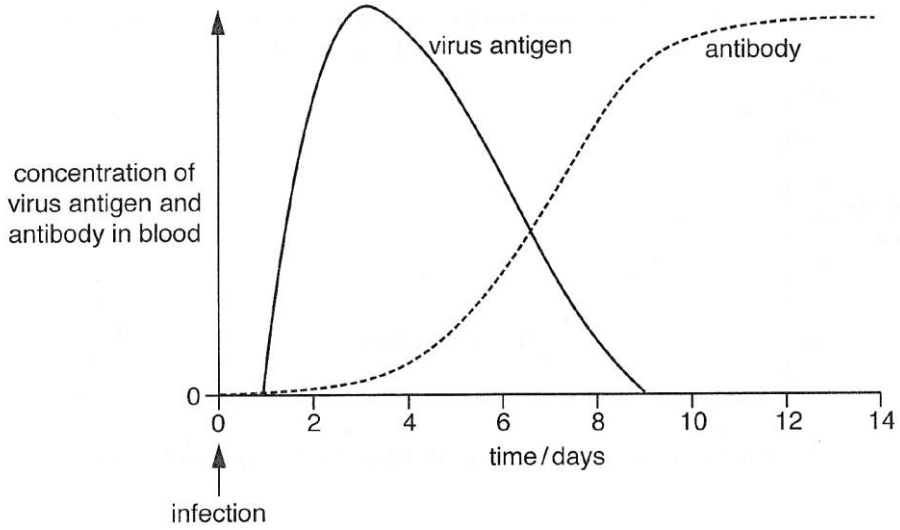


Fig. 11.1

(a) Describe how the concentrations of dengue virus antigen and antibody change during the first ten days following infection, as shown in Fig.11.1.

..... [2]

1 Concentration of virus antigen **increased exponentially** from 0 on day 1 to a **maximum** on day 3. Thereafter, it **decreased** back to 0 from day 3 to day 9.

2 Concentration of antibody **increased gradually** from day 1 to day 4, followed by a **sharp(er) increase** until day 9. Thereafter, it continues to increase slowly and **begins to plateau** on day 10.

Examiner comments: A number of candidates mistakenly referred to the peak of the virus at four days and some did not note the plateauing of antibody concentration towards the end of the period.

(b) Outline the role of antibodies in eliminating the dengue virus from the blood.

..... [3]

1 Antibodies recognize and <u>bind to specific surface proteins</u> on the dengue virus, <u>neutralizing</u> their function, thus **preventing** the virus from **entering** a host cell.

2 Promotes <u>opsonization</u>; binding of antibodies to antigens on the surface of the dengue virus promotes its **phagocytosis by phagocytes** (e.g. macrophages and neutrophils).

3 Antibodies can cause the bound dengue viruses to <u>agglutinate</u>, making it easier for immune cells to target and eliminate them from the blood.

Examiner comments: This question was well answered.

[Q11 Total: 5]

PAPER 3

QUESTION 1

Gene expression in eukaryotes can be regulated at different levels. Long-term changes in gene expression that are passed on to daughter cells are called epigenetic changes. An epigenetic change does not alter the DNA nucleotide sequence.

(a) Change in DNA methylation is one type of epigenetic change. DNA methylation patterns change within cell lineages as cells specialize. A cell lineage is the developmental history of any particular cell type traced back to the original zygote through repeated rounds of cell division. Closely related cell types will share the same cell lineage for much of their developmental history, with divergence only occurring after later cell divisions.

One example of a cell lineage where DNA methylation patterns change as cells specialize is shown in Fig. 1.1.

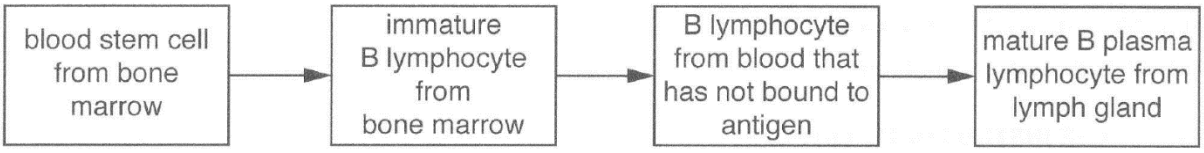


Fig. 1.1

Table 1.1 gives data for the DNA methylation and expression of **one** gene in cells of this lineage.

Table 1.1

cell type	DNA methylation / arbitrary units	gene expression / arbitrary units
blood stem cell from bone marrow	1.00	1.0
immature B lymphocyte from bone marrow	0.89	2.5
B lymphocyte from blood that has not bound to antigen	0.78	4.5
mature B plasma lymphocyte from lymph gland	0.61	5.5

(i) Describe the patterns shown in Table 1.1.

..... [2]

1 As the cells become more specialized, the DNA methylation decreased from 1.00 to 0.61 arbitrary units.

2 As DNA methylation decreases from 1.00 to 0.61 arbitrary units, gene expression increased from 1.0 to 5.5 arbitrary units.

(ii) Explain how DNA methylation changes gene expression.

..... [3]

1 Addition of methyl groups to CG pairs in DNA by DNA methyltransferases recruits histone deacetylases.
2 Histone deacetylases remove acetyl groups from histone tails restores the positive charges and promote tighter binding of negatively charged DNA to the histones.
3 Chromatin becomes more condensed,
4 **Transcription factors and RNA polymerases cannot access the promoters,** hence <u>decreasing</u> rate of transcription of the genes.

(iii) Table 1.2 shows similar data for cancerous B lymphocytes.

Table 1.2

cell type	DNA methylation / arbitrary units	gene expression / arbitrary units
cancerous B lymphocytes	0.95	1.5

Compare the data for the cancerous B lymphocytes in Table 1.2 with the data in Table 1.1 **and** discuss what this suggests about the nature of the cancerous B lymphocytes.

..... [4]

1 DNA methylation of cancerous B lymphocytes (0.95 arbitrary units) is closer to that of blood stem cells from bone marrow (1.00 arbitrary units) than to immature B lymphocytes from bone marrow (0.89 arbitrary units).
2 Gene expression of cancerous B lymphocytes (1.5 arbitrary units) is also closer to that of blood stem cells from bone marrow (1.0 arbitrary units) than to immature B lymphocytes from bone marrow (2.5 arbitrary units).
3 This suggests that cancerous B lymphocytes may also be <u>multipotent</u> like the blood stem cells
4 And may be able to **replicate indefinitely**, and remain <u>undifferentiated</u> and <u>unspecialized</u>.

(b) Epigenetic changes may be important in the development and treatment of cancer.

Fig. 1.2(a) represents the balance between two types of genes controlling cell division in a healthy cell. If genes of type X become overexpressed or genes of type Y become under expressed, the balance is tipped towards a cell becoming cancerous, as shown in Fig. 1.2(b).

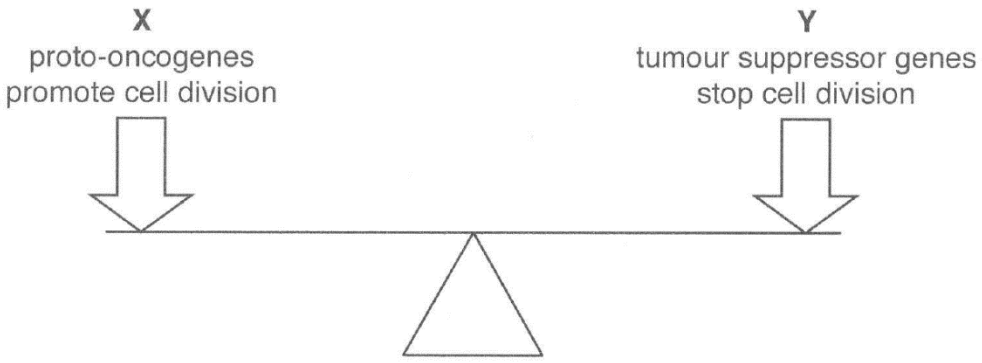


Fig. 1.2(a)

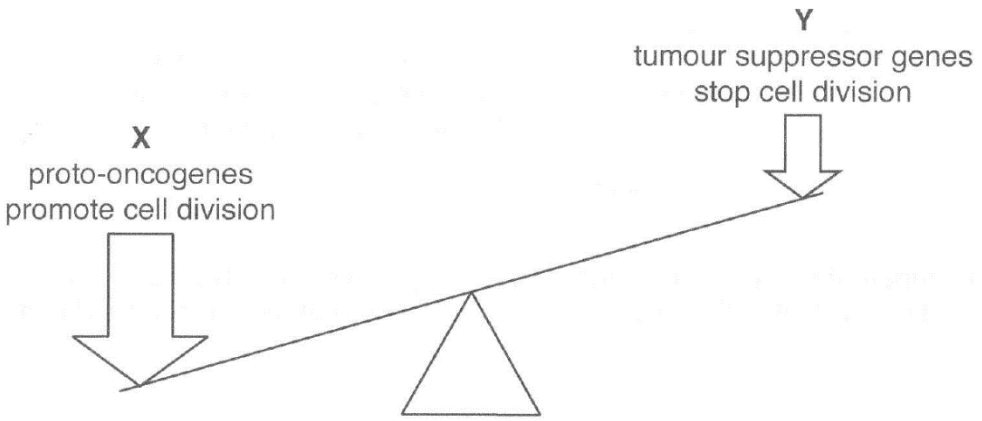


Fig. 1.2(b)

Epigenetic DNA methylation patterns change as cell lines age. For example, the promoter region of the *p53* tumour suppressor gene has a tendency to become methylated in the cells of older people.

(i) Describe **and** explain the potential consequences for the cell and individual person of methylation of the promoter region of the *p53* tumour suppressor gene.

..... [4]

1 Methylation of promoter region results in **silencing** of p53 gene

2 Ref. **lack of** p53 proteins, which act as <u>transcription factors</u> which stimulate expression of other genes; **lack of** gene products, leading to **no arrest cell division, repair damaged DNA and trigger apoptosis**.

3 [effect on cells] cells are more likely to pass through cell cycle checkpoints unchecked / resulting in uncontrolled cell division; and hence **cells** can accumulate further mutations to become cancerous.

4 [effect on person] As a result, the older an individual gets, higher the chances of getting cancer.

Examiner's comments:
Many candidates provided detailed answers. Less effective responses included **irrelevant description** of the mechanism through which methylation downregulates gene expression or listed the positive roles of p53, **rather than identifying what would happen in the absence of expression of the *p53* gene (use negative phrasing).**

Marker's comments:
Candidates are expected to address the consequence on **both** the cell and the body.

(ii) 5-azacytidine is a chemical that inhibits the enzyme DNA methyltransferase. This enzyme adds methyl groups to DNA.

Explain, with reference to Fig. 1.2, why 5-azacytidine may be useful in treating cancers in older people.

..... [3]

1 Addition of 5-azacytidine **decreases methylation** of promoter region of p53 gene.
2 Transcription factors and RNA polymerase can access the promoter and transcribe the p53 gene.
3 Thus increasing the expression of genes of Type Y, restoring the balance between the two types of genes controlling cell division in cancerous cells / stopping cell division in cancerous cells.

Examiner's comments:
Many candidates made use of the information provided to develop sound explanations. Some responses focused on details of how the inhibitor might alter the active site of the enzyme, **rather than considering why the inhibitor might be useful in treating cancers.**

(c) As cell lines get older, another type of change in DNA occurs in the telomeres of chromosomes, leading to a progressive decrease in telomere length.

Fig. 1.3 shows a small part of the DNA sequence at the end of a telomere. Human telomeres may consist of hundreds of repeats of the sequence TTAGGG.

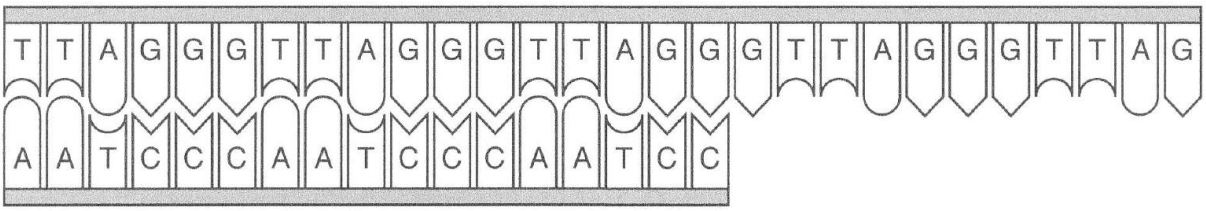


Fig. 1.3

(i) Telomere DNA is tightly condensed due to histone modification. DNA methylation would have a similar effect on the packaging of the DNA, but DNA methylation is not possible at telomeres.

Explain why telomere DNA cannot be methylated.

..... [1]

1 Ref. absence of methylation sites in telomere DNA.

~~**2** As telomere DNA is highly condensed, DNA methyltransferase cannot access and bind to methylation sites to add methyl groups~~

Examiner's comments:
Many responses provided a clear explanation of the **lack of methylation sites at the telomere.**
Some suggested that telomeres could not be methylated due to the absence of cytosine residues, despite the fact that Fig. 1.3 showed eight cytosine residues within the telomere section of the double-stranded DNA molecule.

(ii) Outline **two** functions of a telomere containing hundreds of repeat sequences.

..... [2]

1 Telomeres act as <u>disposable buffers</u> to protect the coding DNA from gene erosion during DNA replication as DNA shortens with each round of replication.
2 Proteins bind to telomeres to form nucleoprotein cap which protect the ends of chromosomes from degradation by nucleases / prevent end to end joining of chromosomes which can lead to chromosomal mutations / prevent unintentional cell death.

Examiner's comments:
The majority of candidates gave detailed responses. A number of candidates wrote about all aspects of telomeres rather than focusing on the question asked.

(iii) Most human cell lines stop dividing after 40 to 60 rounds of cell division. This is known as the 'Hayflick limit' and is due to changes in the DNA in telomeres.

Cancer cell lines and stem cell lines, in contrast, can divide indefinitely without limit.

Explain how changes in the DNA in telomeres prevent most human cell lines from dividing beyond the Hayflick limit **and** suggest how cancer cells and stem cells are able to overcome this limit.

..... [4]

1 During DNA replication, DNA polymerase I is unable to replace the RNA primer at the 5' end of the lagging strand with deoxyribonucleotides due to the lack of a free 3' OH end on the daughter strand.
2 This causes telomere DNA to shorten with every round of replication. Once it reaches the critical minimum length, it will signal the cell to enter into senescence / stop dividing / begin apoptosis.
3 Cancer cells and stem cells have active telomerase genes which are expressed to produce telomerase enzyme.
4 Telomerase enzyme lengthens the telomeres by adding new telomeric repeats to the 3' end of parental template strands, preventing them from shortening to the critical minimum length in cancer and stem cells.

Examiner's comments:
Most candidates provided a detailed response.

A number of candidates did not make it clear which type of cells were being discussed, confused the terms 'telomere' and 'telomerase' or described changes in telomerase genes, rather than changes in telomere DNA.

(d) People of the same chronological age may have different biological ages due to environmental factors, such as diet and exposure to pollution.

Since both telomere length and DNA methylation patterns change as individuals get older, both have been suggested as possible measures of a person's biological age.

Fig. 1.4 shows how telomere length varies in a sample of people of different chronological ages. Each data point represents an individual person.

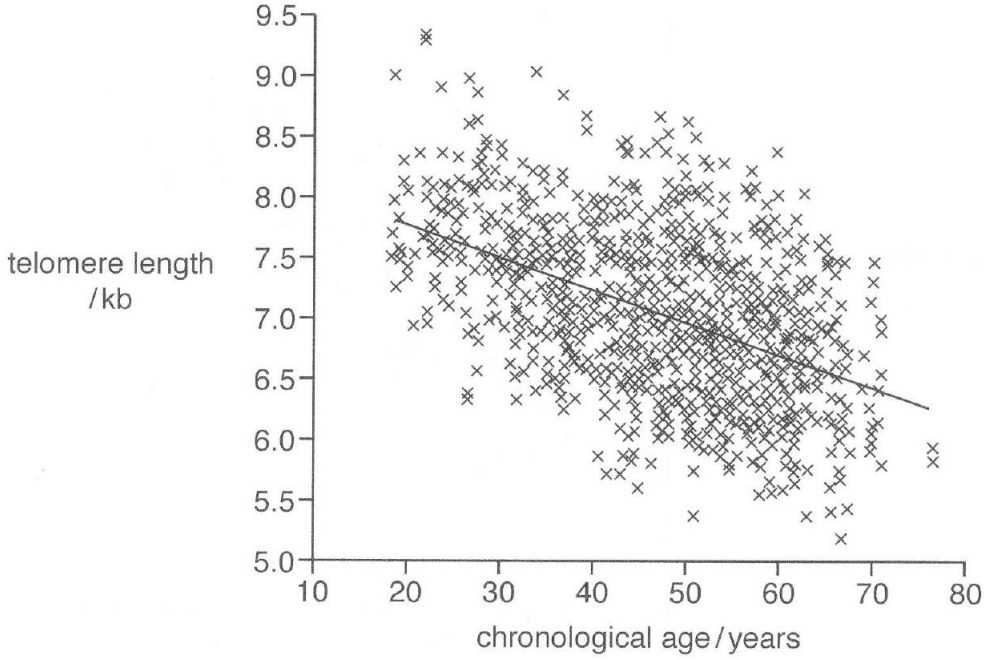


Fig. 1.4

(i) With reference to Fig. 1.4, evaluate the extent to which telomere length can predict a person's chronological age.

..... [3]

1 [Effective predictor] Ref. line of best fit, indicating a general linear trend that the **older a person's chronological age is, the shorter his telomere length** is, / Ref. negative correlation between telomere length and chronological age

2 [Evidence] For instance, a person who is 20 years old may have telomere lengths from 7.3 to 9.0 kb; while a person who is 70 years old may have telomere lengths from 5.9 to 7.5 kb.

3 [Ineffective predictor] same telomere length may lead to a big range of possible chronological ages e.g. telomere length of 7.5kb corresponds to age of 20 to 70 years old

Examiner's comments:

*Most answers correctly described the relationship shown by the **line of best fit** and a number used correct terminology such as **negative correlation or inversely proportional**. Good answers also **quoted selected data, such as examples of individuals that conflicted with the overall trend.***

*Some candidates **incorrectly** considered that the data was tightly clustered and therefore concluded that telomere length can be an effective predictor of an individual's chronological age.*

(ii) Scientists have analysed DNA methylation patterns in people of different ages and have established a method to obtain a DNA methylation age from these patterns. The DNA methylation age is the expected chronological age of a person predicted from their DNA methylation patterns. Fig. 1.5 shows how the DNA methylation age varies in a sample of people of different chronological ages. The line represents people whose DNA methylation age is the same as their chronological age.

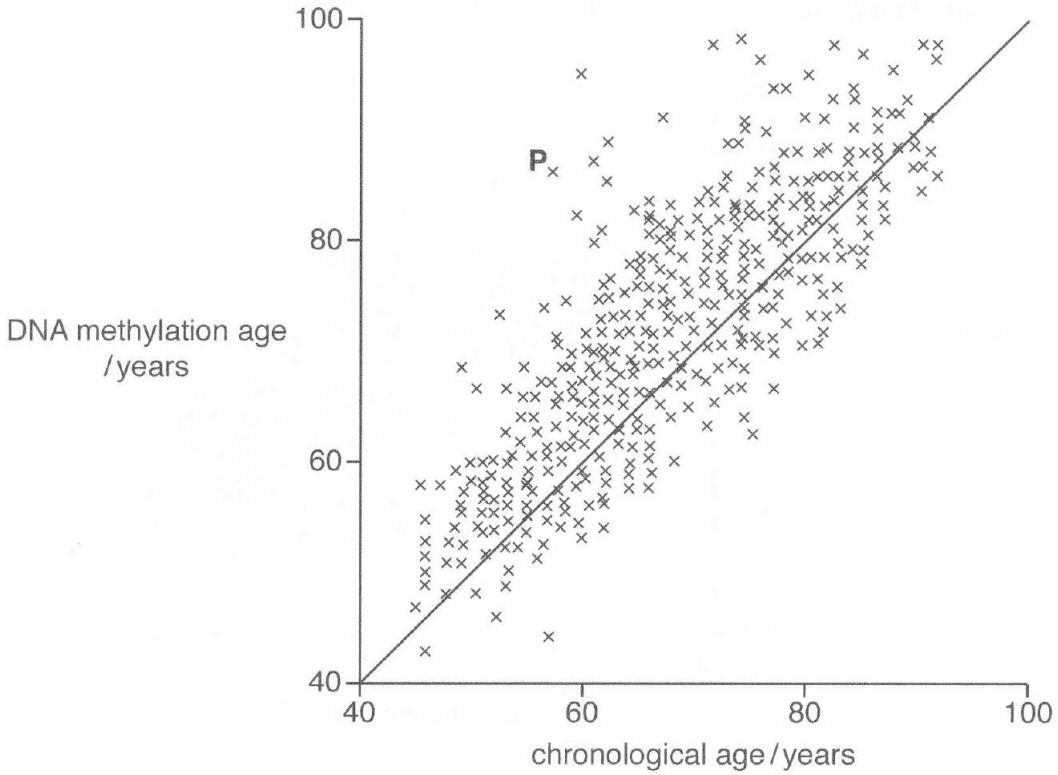


Fig. 1.5

On Fig. 1.5, the data point for person **P** lies above the line.

Suggest what this implies for the health and life expectancy of person **P**.

..... [4]

1 Person P has a much higher DNA methylation age of 84 than his chronological age of 58.
2 It implies that his health is comparable to an 84 year old man even though he is only 58
3 And that his life expectancy is lower than expected.
4 His high DNA methylation age may also suggest a higher likelihood of him contracting a cancer.

Examiner's comments:
*The majority of candidates clearly addressed the health and life expectancy of **P** in the light of a*

*statement that his or her DNA methylation age was higher than his or her chronological age. Many made sensible use of information derived from earlier questions, e.g. by stating that **P** might be more at risk of cancer.*
*Some candidates compared figures without drawing any inference from them. A small number based their reasoning on the misconception that a point above the line was advantageous to **P**.*

[Q1 Total: 30]

QUESTION 2

Mammals have both a non-specific (innate) and a specific (adaptive) immune system.

(a) Outline the components of the non-specific immune system in mammals.
…… [4]

1 **Physical barriers** such as skin, nose, hair, cilia, which **blocks the entry** of pathogens.
2 **Soluble factors** such as saliva, tears, mucous, provide a **washing action** that inhibits colonization by fungi and bacteria / contain lysozymes which **destroys bacterial cell walls**.
3 **Phagocytes** such as macrophages, neutrophils and dendritic cells, engulf pathogens and **digest** them with lysosomal enzymes.
4 **Natural killer cells** recognize the abnormal array of surface proteins characteristic of virus-infected and cancer cells and release chemicals that leads to cell death.

Examiner comments: There was variation in candidates' approaches to outlining the components of the non-specific immune system. Many responses included details of mechanical and chemical barrier defences and the cells and chemicals involved in the inflammatory response and in phagocytosis. A few responses showed knowledge of the complement system and the actions of natural killer cells.

Some candidates listed cells without specifying their roles and went beyond the brief of the question by discussing cells and events involved in adaptive immunity.

(b) To protect their offspring from specific pathogens in the environment, female mammals pass on antibodies through the placenta and in the milk.

In contrast, honey bees cannot make antibodies since they only have a non-specific immune system. However, queen honey bees can pass on fragments of pathogenic bacteria in their eggs to their offspring. These fragments stimulate an enhanced immune response in the offspring.

Complete Table 2.1 with **ticks** to show whether these two examples of enhanced immunity in offspring are active, natural, or passive or a combination of these.

Table 2.1

type of immunity	mammal	honey bee
active		✓

natural	✓	✓
passive	✓	

[2]

1M for correct ticks, per organism.

Examiner comments: Some candidates were uncertain about whether or not these examples of enhanced immunity were natural.

(c) Honey bees are important for pollinating fruit crops. Without pollination, fruits are not produced.

Suggest why it is economically important to investigate the mechanism of immunity in honey bees.

…… [3]

1 This allows scientists to create <u>vaccines</u> for honey bees, which will **protect honey bee populations** from infectious agents that can kill them.
2 This stabilizes and **increases the honey bee populations**, which will increase their ability to pollinate more fruit crops, leading to higher production of fruits.
3 Failure to protect honey bees from infectious disease may lead to **extinction** of honey bees as well as valuable fruit crops that require honey bees for pollination.
4 OVP

Examiner comments: Many candidates used the information provided in **2(b)** to make creative suggestions, such as vaccinating bees by manipulating the eggs in order to maintain the size of the honey bee population.

Some discussed dangers to honey bees that were unrelated to the question material about infectious diseases, e.g. use of insecticides and genetically modifying bees for insecticide resistance. Other <u>incorrect responses included suggestions that bees could pass pathogens to fruit and then onto humans, or that understanding the bees' mechanism of immunity could lead to better human vaccinations</u>.

[Q2 Total: 9]

QUESTION 3

Coal-fired power stations release large quantities of carbon dioxide into the atmosphere. Two approaches to reduce the quantity of carbon dioxide released are:

- Carbon capture and storage (CCS)
- Carbon capture and utilisation (CCU).

CCS involves the permanent storage of carbon dioxide underground. CCU involves collecting the carbon dioxide and using it in other industrial processes.

(a) Explain why environmentalists would like all new coal-fired power stations to include CCS or CCU facilities.

……..…... [4]

1 Carbon dioxide is a **greenhouse gas**.
2 Including CCS or CCU facilities in coal-fired power stations will **reduce carbon dioxide emissions**, and slow down / reverse the <u>global warming</u> problem.
3 This will in turn reduce the melting of polar ice caps and the resultant rise in sea levels,
4 which would lead to lower stress on fresh water supply and less submerging of low-lying coastal areas.
5 It will also reduce extreme weather conditions like heat waves and heavy storms
6 and reduce death of coral reefs.

(b) In one new type of coal-fired power station, both CCS and CCU approaches are used. This is shown in Fig. 3.1.

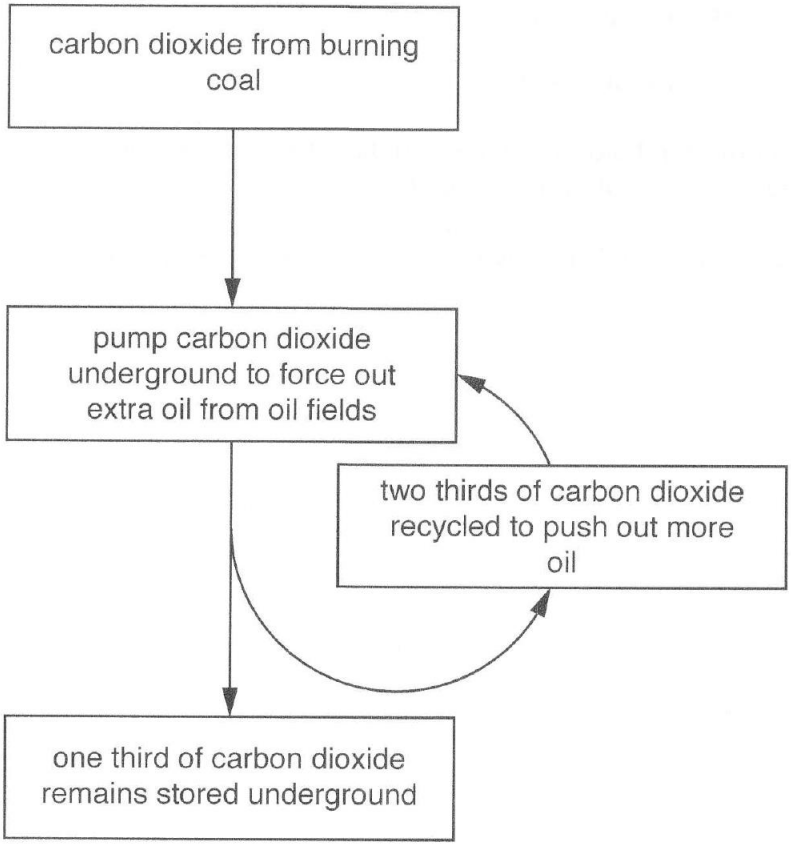


Fig. 3.1

Evaluate the effectiveness of the process shown in Fig. 3.1 as a solution to the problem of carbon dioxide emissions from burning fossil fuels.

.....[3]

1 [Take a stand]: The process will not effectively solve the problem of carbon dioxide emissions from burning fossil fuels.
2 [Explain]: The carbon dioxide stored underground will be slowly released back into the atmosphere as underground soil is not a natural carbon sink.
3 The process in Fig. 3.1 also pumps out more oil, which will release more carbon dioxide when it is burnt as a fossil fuel.

(c) The release of carbon dioxide into the atmosphere from coal-fired power stations must be considered in relation to other processes that affect the concentration of carbon dioxide in the atmosphere.

For examples, respiration also adds carbon dioxide to the atmosphere.

Outline how carbon dioxide is produced in respiration.

.....[4]

1 Carbon dioxide is produced in the <u>mitochondrial matrix</u>
2 via <u>oxidative decarboxylation</u> reactions.
3 <u>Linked reaction</u> **converts pyruvate to acetyl CoA** with the release of one carbon dioxide and one NADH per pyruvate molecule.
4 <u>Krebs cycle</u> releases **two carbon dioxide molecules per cycle**

[Q3 Total: 11]

QUESTION 4 Essay

(a) Describe the process by which a C3 plant makes sugars in its leaves using sunlight, water and carbon dioxide. [15]

1 Photon of light strikes an accessory pigment molecule in the light harvesting complex of photosystem II (PS II) located in the thylakoid membrane.
2 Pigment molecule absorbs light energy, and its electron is excited to a higher energy level.
3 When the "excited" electron returns to the "ground" state (unexcited state), energy released is passed on to the electron in a neighboring pigment molecule.
4 Energy is transferred from one electron in a pigment molecule to another.
5 Finally channeled to P680 special chlorophyll a molecules in the reaction centre of PS II located in the thylakoid membrane.
6 An electron of special chlorophyll a molecule is boosted to a very high energy level and captured by PS II's primary electron acceptor.
7 Electron displaced from the PS II reaction centre is replaced via the Hill reaction where a water molecule is split by an enzyme in the photolysis of water using energy from the sunlight to produce H^+ ions and oxygen molecule and electrons.
8 Photo-excited electrons are passed from PS II's primary electron acceptor to photosystem I (PS I), via a series of electron carriers in an electron transport chain.
9 Electrons are transported along the series of electron carriers of progressively lower energy levels.
10 Energy released from the electron transport is used to pump H^+ from the stroma, across the thylakoid membrane and into the thylakoid space.
11 A proton gradient is generated.
12 H^+ ions diffuse from the thylakoid space into the stroma of the chloroplast, through the ATP synthase, down a concentration gradient.
13 ATP synthase enzyme of the complex catalyses the synthesis of ATP from ADP and Pi.
14 Ref to chemiosmosis
15 Meanwhile, light is harvested by the accessory pigments in the light harvesting system of PS I and the energy is passed along to the electrons in the neighbouring pigments and eventually channeled to P700 special chlorophyll a molecules in the reaction centre of PSI located in the thylakoid membrane.
16 An electron in the special chlorophyll a molecule is boosted to a very high energy level and captured by PS I's primary electron acceptor.
17 Photo-excited electrons are passed from PS I's primary electron acceptor down a second electron transport chain to $NADP^+$.
18 H^+ ions from the stroma and the electrons from PS I reduce $NADP^+$ to NADPH. The enzyme that catalyses the reduction is called $NADP^+$ reductase.
19 ATP (from light dependent reactions) and NADPH from non-cyclic light dependent reactions) function as chemical energy and reducing power, respectively, for the subsequent manufacturing of carbohydrates using organic compounds formed from the fixation of carbon dioxide in Calvin cycle.

(b) Germination is the process by which a seed with access to water, oxygen and a suitable temperature begins to grow into a young plant. A seed consists of an embryo and a food store.

The mass of a soaked seed undergoing germination in the light was recorded. In the first five days, the mass of the seed decreased. In the next five days as the young plant grew, this mass was regained.

Explain the decrease and gain in mass as the seed germinated and grew into a young plant over this period of ten days. [10]

1 In the first five days, the mass of the seed decreased because carbohydrates and fats stored in the seed were broken down by enzymes into glucose.
2 Glucose is converted by a series of reactions into pyruvate in the cytoplasm of the cells, with the release of 2 NADH and 2 net ATP per glucose molecule
3 via dehydrogenation and substrate level phosphorylation.
4 Pyruvate is transported into mitochondria matrix via specific protein channels and converted into acetyl CoA and carbon dioxide
5 via oxidative decarboxylation, releasing one NADH.
6 Acetyl CoA enters the Krebs cycle and combines with oxaloacetate to form citrate, which undergoes several reactions to produce 3 NADH, 1 $FADH_2$, 2 CO_2, 1 ATP per cycle.
7 NADH and $FADH_2$ (produced from glycolysis, linked reaction and Krebs cycle) donate their electrons to electron carriers in the electron transport chain embedded in the inner mitochondria membrane.
8 Which carriers out oxidative phosphorylation to produce ATP from ADP and Pi.
9 ATP generated is used to drive cellular activities such as growth and cell differentiation as the seedling grows.
10 Hence the seedling grows in mass after five days due to rapid mitosis, resulting in more cells being produced.
11 When the seedling grows its new leaves, it begins to be able to carry out photosynthesis to produce its own supply and store of carbohydrates.

[Q4 Total: 25]

QUESTION 5 Essay

(a) Birds are thought to have descended from dinosaurs.

Explain how a population of a species of dinosaur could change over time into a new species with bird-like features. [15]

……

Micro-evolution:

1 Genetic variations exist in a population of dinosaurs.
2 Mutations occurred, giving rise to new alleles for the development of bird-like features.
3 Individuals with bird-like features are at a **selective advantage**
4 because e.g. their wing-like structures allow them to be able to evade predators by being air-borne for short periods of time
/ AVP describing a selective advantage relevant to a specific bird-like feature
5 They survive better, reproduce more, and pass down alleles coding for bird-like features to their offspring.
6 The proportion of individuals with bird-like features increases with each successive generation.
7 This leads to changes in allele frequencies in the population over time.

Macro-evolution / Speciation:

8 Geological processes such as rise in sea levels, or islands breaking off from a mainland can cause geographic isolation of these dinosaurs from the rest of the population.
9 This barrier disrupts gene flow between these dinosaurs and the original population.

10 Genetic drift / ref to genetic drift
11 Ref to bottleneck effect or ref to founder effect
12 and natural selection occur.

13 Different populations accumulate different mutations.
14 Over time, populations of dinosaurs become so different that they can no longer interbreed with each other.
15 Allopatric speciation is said to have occurred if **geographical isolation** produced a new species with bird-like features.
16 Alternatively, sympatric speciation may have occurred, if a **behavioural isolation / morphological isolation** prevented a population of dinosaurs with bird-like features from interbreeding with other dinosaurs.
17 QwC

Examiner comments: Many responses that drew on knowledge of micro-evolution and macro-evolution were seen, often including examples relevant to the context, such as a bird-like feature and its selective advantage.

Some candidates focused on one or two aspects only and re-iterated the same reasoning many times, using different examples of bird-like features and their advantages. Others considered micro-evolution only.

(b) Dinosaurs are now extinct but there are approximately 10000 species of living birds. It is thought that birds evolved from small dinosaurs and that being small helped their survival during a time of rapid environmental change when species of large dinosaur went extinct.

Suggest **and** explain why, in a rapidly changing environment, small animals may be at an evolutionary advantage compared to large animals. [10]

……..….

Small animals have a higher chance of survival than large animals because they can adapt better to changes in environment faster.

1 Small animals are **more mobile** than large animals and can move to habitats more conducive to survival more easily.
2 For instance, small animals can move to higher altitudes more easily than large animals.
3 Small animals can **find / build shelter** more easily than large animals,
4 as they need less space / building materials.
5 Small animals require **less food to thrive** than large animals,
6 as they have a smaller biomass and thus a smaller energy requirement.
7 Small animals are **less conspicuous to predators** than large animals,
8 as they can hide / camouflage more easily among vegetation or rock formations.

9 Small animals tend to have **shorter life spans and more reproductive cycles** than large animals.
10 They also tend to have **more offspring per brood** than large animals.
11 Hence, they can generate a lot more offspring with more genetic diversity within the same period of time, as compared to large animals.
12 This ensures that at least some of its offspring will be able to survive and reproduce in a rapidly changing environment, passing down advantageous alleles to the next generation.
13 QwC

Examiner comments: Many candidates looked beyond the individual animal to the life-cycle and population, and identified reasons why small animal populations will adapt and evolve more quickly based on their high reproductive output and short generation time.

Some candidates provided more limited responses, considering only the survival of individual small or large animals in terms of risk of predation and requirement for food.

[Q5 Total: 25]