2017 'A' Level
H2 Biology
Mark Scheme

PAPER 1 (MCQ)

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

PAPER 2 (CORE)

QUESTION 1

Fig. 1.1 shows a representation of a glycogen molecule. Glycogen molecules are made up of glycogen molecules surrounding a central core.

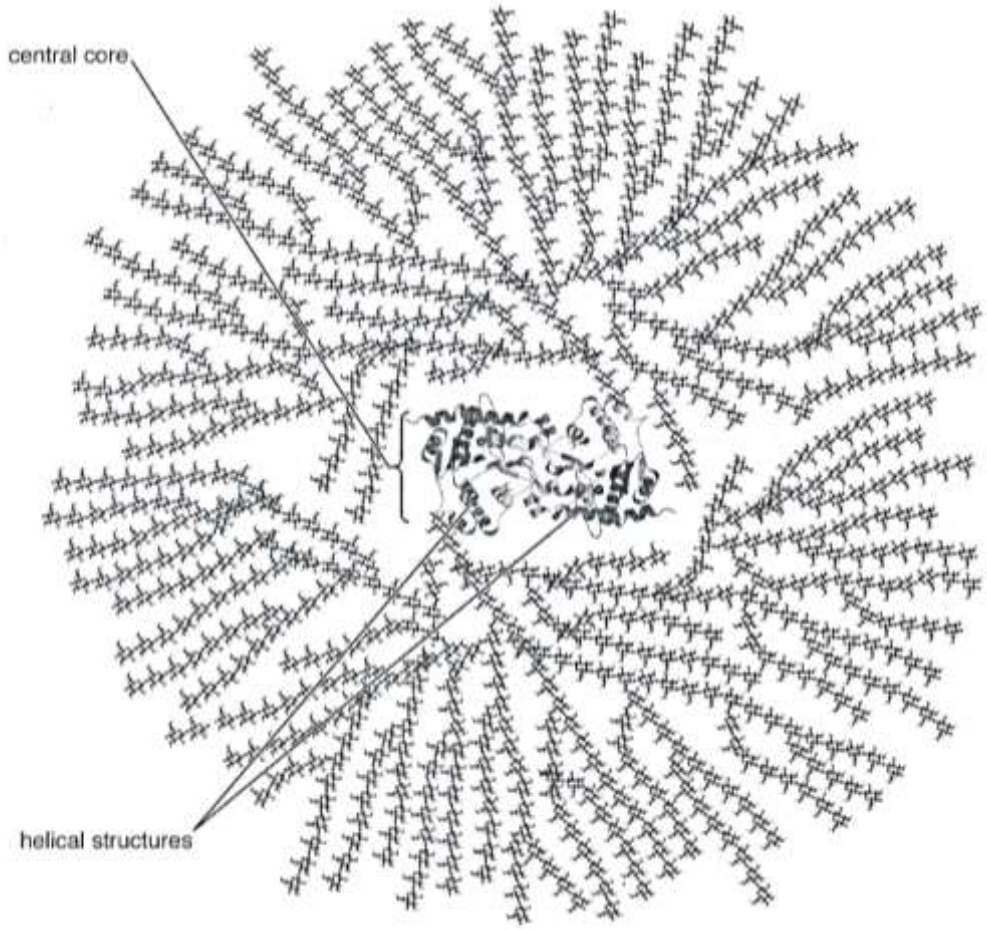


Fig. 1.1

(a) (i) State the number of glycogen molecules shown in Fig. 1.1.

…….. [1]

1 Two / 2

Examiner's comments:
Most candidates were able to interpret the diagram correctly.

(ii) Name the type of molecule forming the central core of the glycogen granule, in Fig. 1.1.

…….. [1]

1 Protein / Polypeptide

Examiner's comments:
The majority of candidates identified the type of molecule forming the central core.

(iii) Suggest the role of the molecule forming the central core of the glycogen granule.

…….. [1]

1 It allows glycogen molecules to attach / bind to it, so as to form the glycogen granule.

Examiner's comments:
Most candidates correctly deduced the role of this molecule.

(b) Explain how the structure of glycogen is related to its role in living organisms.

…….. [4]

1 Glycogen molecule has a very compact (helical) shape; this enables it to be stored in large quantities within a limited space.
2 Glycogen molecule is large in size and majority of its hydroxyl groups of glucose monomers project into the interior of helix; this makes it insoluble in water.
3 Glycogen molecule being insoluble in water; allows it to be stored in large quantities without greatly affecting the osmotic potential of the cell.
4 Glycogen molecule is highly branched (due to α (1,6) glycosidic linkages) and do not form inter-molecular cross-linkages; this enables it to be easily accessed by enzymes to be broken down into glucose.

Examiner's comments:
All candidates were familiar with both the structure and role of glycogen in living organisms, but not all considered how the structure was related to its role.

(c) Describe how the structure of cellulose is different from the structure of glycogen.

…….. [3]

[Any three]

	Cellulose	**Glycogen**
1	Linear/straight chains ;	Helical molecule ;
2	Unbranched molecule ;	Highly branched molecule;
3	Comprised of β-glucose monomers;	Comprised of α -glucose monomers;
4	Adjacent monomers are **orientated** 180º to each other;	Adjacent monomers are of **same orientation** / no 180º rotation;
5	Presence of β (1,4) glyosidic linkages;	Presence of α (1,4) glyosidic linkages and α (1,6) glyosidic linkages;
6	Presence of cross-linking / hydrogen-bonding between chains;	Absence of cross-linking / hydrogen-bonding between chains;

Examiner's comments:
Candidates were familiar with the structural differences between cellulose and glycogen, with many good answers being given.

[Total: 10]

QUESTION 2

Fig. 2.1 is a diagram representing a cell surface membrane. Five proteins are labelled **Q**, **R**, **S**, **T** and **V**.

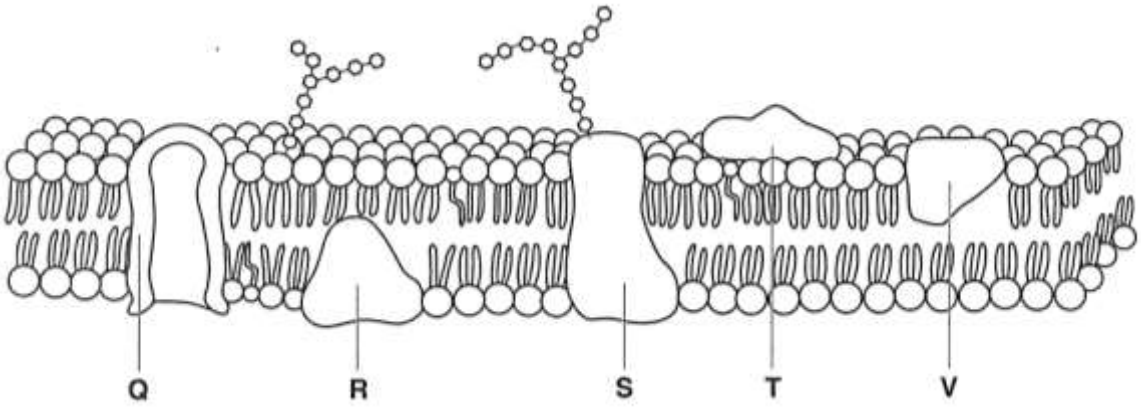


Fig. 2.1

(a) (i) Explain why proteins are required for the transport of glucose across the cell surface membrane.

……..…….. [3]

1 Glucose is too large in size, so it is unable to fit through the phospholipids in the cell surface membrane / diffuse through the phospholipid bilayer.

2 Glucose is a polar / hydrophilic / water-soluble molecule, which prevents it from passing through the hydrophobic core of the phospholipid bilayer.
[**Reject**: "Not lipid soluble", too vague]

3 Protein transporters, such as protein channels, help to provide a passageway for glucose to enter the cell via facilitated diffusion.

Examiner's comments:
Candidates were familiar with properties of the cell surface membrane and were able to give full accounts of why proteins are required to transport glucose across the membrane.

(ii) Describe the structural features of a protein that enable it to transport glucose into a cell.

……..…….. [2]

1 It is a transmembrane protein, to connect both the extracellular and intracellular region across the cell surface membrane.

2 The exterior of the protein contain mostly amino acids with non-polar R-groups, for hydrophobic interactions with the non-polar hydrocarbon chains of phospholipids of the cell surface membrane.

3 The interior of the protein contain mostly amino acids with charge / polar R-groups, so it can serve as a hydrophilic passageway for glucose transport.

4 The channel of the protein is complementary in conformation for glucose, to selectively allow for only glucose to move through it.

Examiner's comments:
Many candidates considered the significance of having both hydrophobic and hydrophilic regions for proteins that transport glucose into cells.

(iii) State the letter of the protein in Fig. 2.1 that could transport glucose into a cell.

…….. [1]

1 Q

Examiner's comments:
Nearly all candidates correctly identified the channel protein shown in Fig. 2.1.

(b) Fig 2.2 shows the effect of increasing the external concentration of glucose on the rate of glucose uptake into a cell.

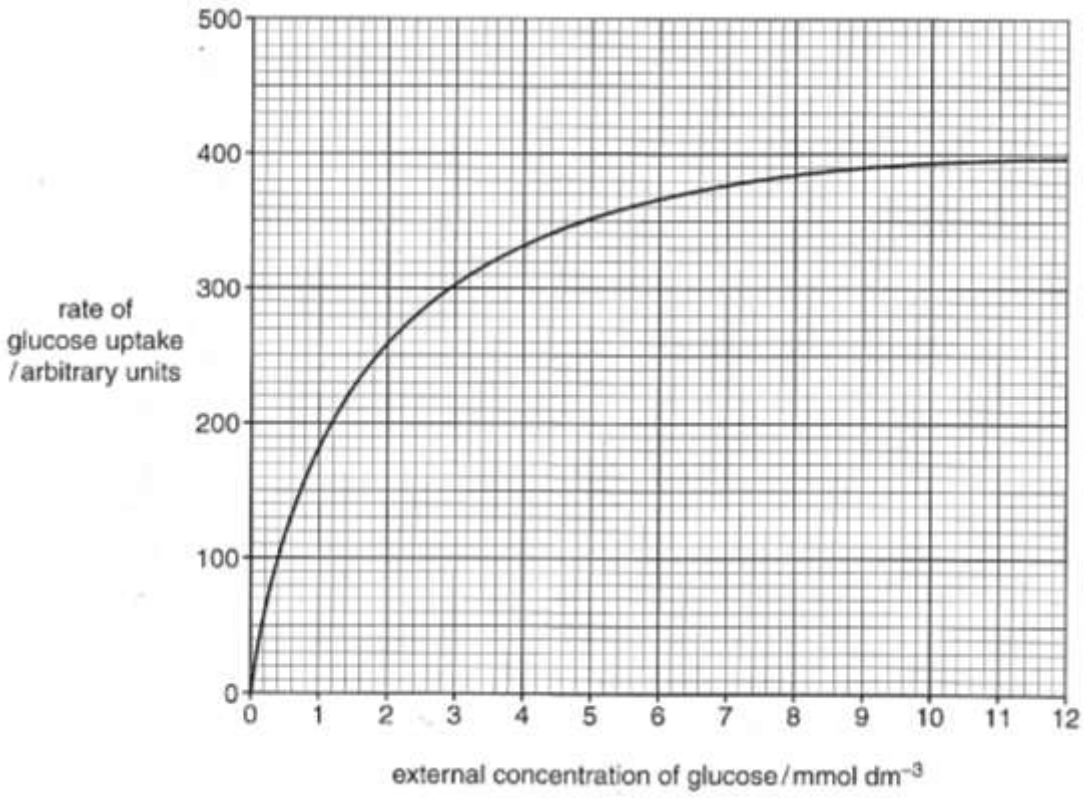


Fig. 2.2

Using the information shown in Fig. 2.2, describe and explained the effect of increasing the external glucose concentration on the rate of glucose uptake into a cell.

…….. [4]

1 Low level description: The rate of glucose uptake increased when external glucose concentration increased.

2 Higher level description: The rate of glucose uptake increased rapidly / sharply at the start, but eventually levels off to reach a plateau.
[**Accept:** "Increases at a decreasing rate" as alternative description]

3 Data Quoting: Specific and accurate reference to any pair of data (x-axis & y-axis), with correct units stated.

E.g.:

external concentration of glucose increased from 0 to 1 mmol dm^{-3}, rate of glucose uptake increased from 0 to 180 arbitrary units (AU) ;

rate of glucose uptake plateau off at 395 AU after external concentration of glucose increases beyond 10 mmol dm^{-3} [**ACCEPT**: 11 mmol dm^{-3}] ;

4 **Explanation for 1st half of curve:** The sharp increase in glucose occurred due to the increase in concentration gradient.
5 **Explanation for 2nd half of curve:** The 'levelling off' effect occurred as the transport proteins for glucose became saturated / fully occupied / AW.

Examiner's comments:
Most candidates had little difficulty in describing the effect of increasing external glucose concentrations on the rate of glucose uptake. Better performing candidates provided sound explanations on why the effect was observed. Weaker responses stated that the rate of glucose uptake became constant when the concentrations of glucose on each side of the membrane were equal. In the absence of other mechanisms, this would have resulted in the net rate of glucose uptake falling to zero.

[Total: 10]

QUESTION 3

Fig. 3.1 shows model of a haemoglobin molecule and part of a collagen molecule, drawn to the same scale.

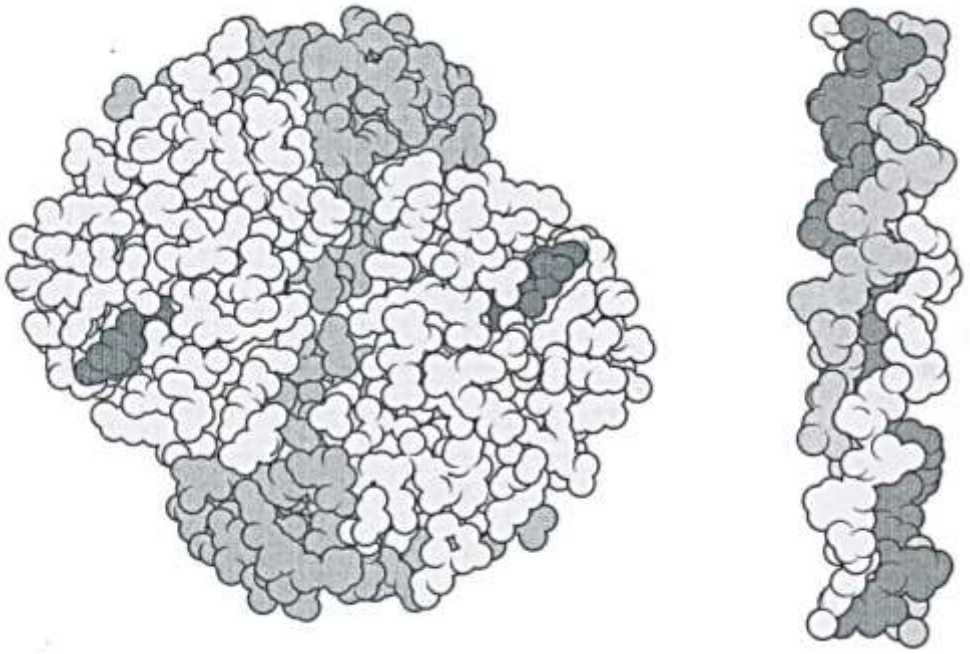


Fig. 3.1

(a) Describe the main features of the molecular structures of haemoglobin and collagen, visible in Fig. 3.1.

…….. [5]

Haemoglobin:

1 It has a globular structure.
2 It contains prosthetic heme groups.
3 It has a quaternary structure / comprises of four subunits.

Collagen:

4 It has a fibrous structure.
5 It has a helical structure.
6 It has a quaternary structure / comprises of three polypeptide chains.

Examiner's comments:
Candidates were familiar with the structure and function of haemoglobin and collagen. However, responses often included all of the candidates' knowledge about these molecules, rather than addressing the specific points required. Here, many full and detailed answers were given.

(b) Haemoglobin is a transport protein. Collagen is a structural protein.

Explain how the molecular structures of haemoglobin and collagen are related to their functions.

…….. [5]

Haemoglobin:

1 Amino acids with charged/polar R-groups are found on the exterior of haemoglobin; enabling the globular protein to be soluble in aqueous medium / blood.
2 Heme group is present in each subunit; enabling oxygen molecules to be carried by the haemoglobin protein.
3 The four subunits in haemoglobin associate together via non-covalent interactions; slight changes in oxygen concentration causes a conformational change in one subunit which is relayed to the other subunits, enabling cooperative binding of oxygen.

Collagen:
4 Every 3rd residue in each collagen polypeptide is glycine; allowing for stable helical structure to be formed.
5 It is a long molecule; thus allowing long collagen fibers to be formed.
6 The three polypeptides are held together by cross-linkages / inter-chain hydrogen bonds; contributing to its great tensile strength.
[**Note**: No mark was given for staggered arrangement answer, as noted in CIE workshop.]

Examiner's comments:
Candidates' knowledge of the structures and functions of haemoglobin and collagen were well demonstrated. Not all candidates linked specific aspects of structure to the relevant function.

[Total: 10]

QUESTION 4

Fig. 4.1 shows the replication of part of a DNA molecule.

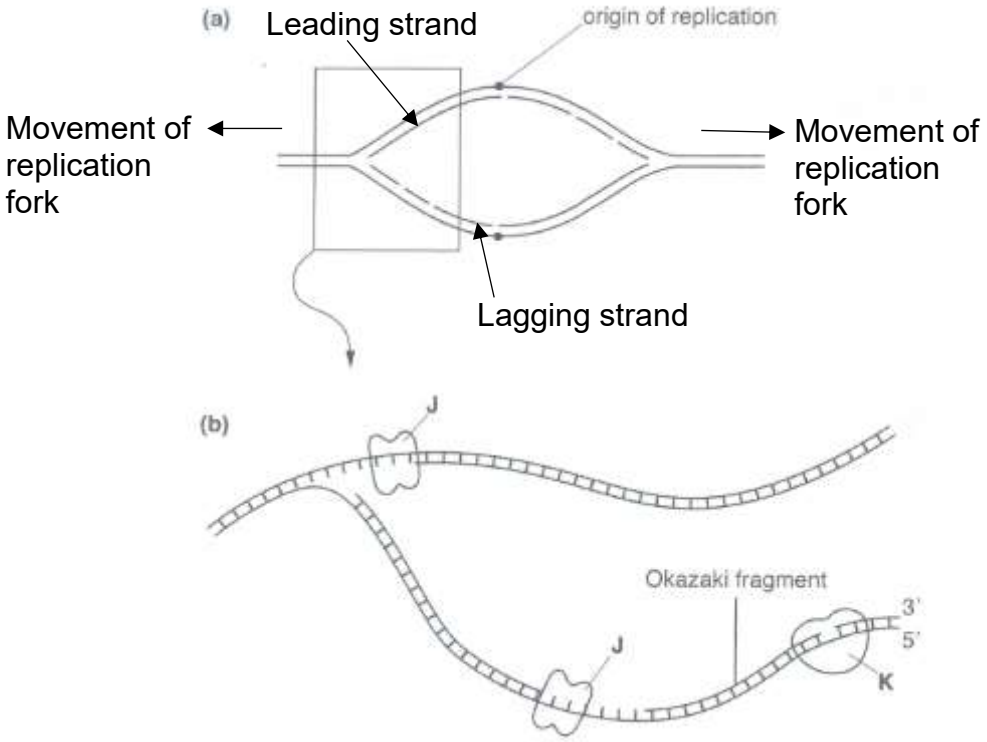


Fig. 4.1

(a) On Fig. 4.1(a),

- label a leading strand and a lagging strand
- draw arrows to show the direction of movement of both replication forks. [3]

(b) Name the enzymes **J** and **K** shown in Fig. 4.1(b).

……..…….. [2]

J: DNA polymerase III

K: DNA ligase

(c) Explain the reason for the production of Okazaki fragments.

……..…….. [3]

1 Two (parental) DNA strands are <u>antiparallel</u> oriented in <u>opposite</u> directions to each other

2 <u>DNA polymerase</u> can only add free deoxyribonucleoside triphosphates to a <u>free 3'OH group</u> of an existing polynucleotide, catalysing the formation of phosphodiester bond in one direction from <u>5' to 3'</u>

3 Specific **3D conformation** of the <u>active site</u> of DNA polymerase is **complementary** to that of the **conformation** of the substrates (3' OH group of existing chain and 5' phosphate group of incoming deoxyribonucleoside triphosphate)

(Leading strand is synthesized in the same direction as the opening of the replication fork hence dNTPs are added continuously. Lagging strand is synthesized in the opposite direction as the opening of the replication form hence dNTPs are added discontinuously.)

[Q4 Total: 8]

QUESTION 5

Fig. 5.1 shows the main stages in the development of cervical cancer over a number of years, following infection with the human papillomavirus. During this period of time, normal cells change into precancerous cells and then these precancerous cells develop into an invasive cancer.

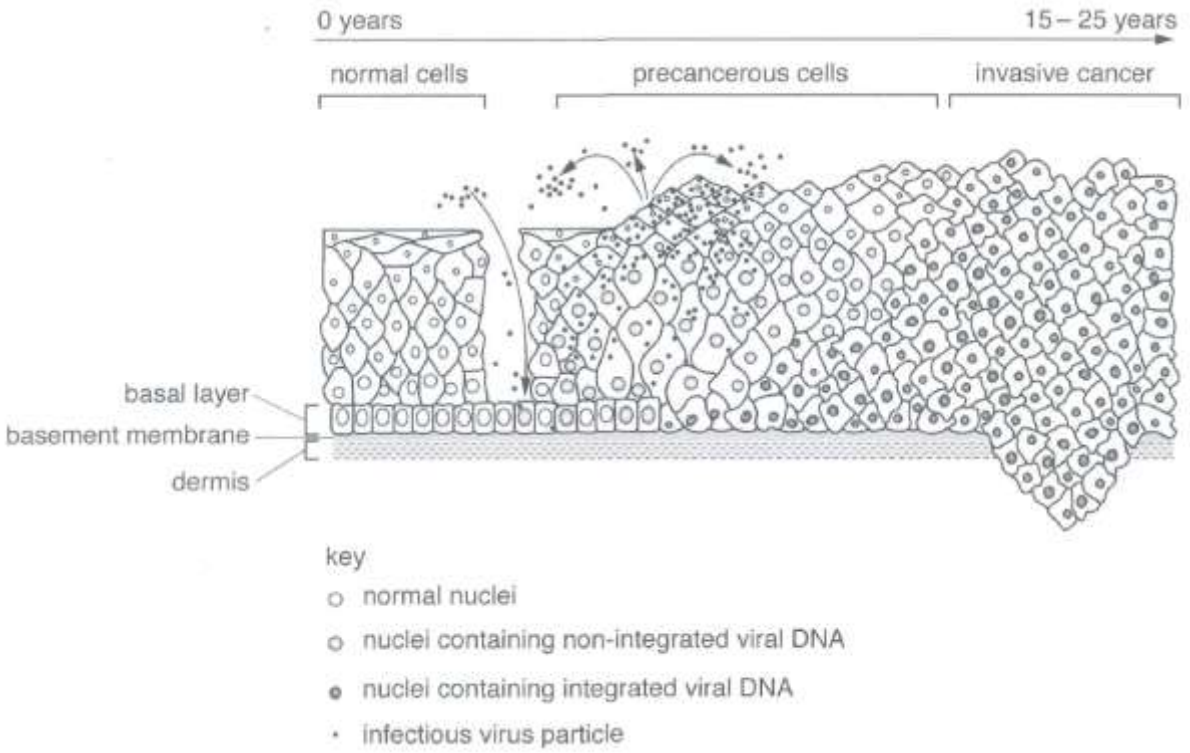


Fig. 5.1

(a) Describe the main changes visible in Fig. 5.1 in the development of cervical cancer, following the initial infection of cells in the basal layer by human papillomavirus.

…….. [4]

1 Upon initial infection of cells in the basal layer by human papillomavirus (HPV), the viral DNA does not integrate itself into the host cell genome in the nuclei and remain in the cytoplasm. **HPV uses the host cell machinery to replicate itself.**

2 Cells with low copy number of HPV are precancerous cells which <u>do not show anchorage dependence and density-dependent inhibition</u>. These cells divide beyond a single layer and pile up in chaotic fashion to give a clump of overlapping cells.

3 Overtime, the number of HPV increases in the cells and **some are eventually released** from the precancerous cells.

4 As time progresses, cancer becomes invasive as the viral DNA integrates into the host cell genome. These cells with nuclei containing integrated viral DNA **divide uncontrollably**, breaking through the basement membrane and dermis and to reach other parts of the body via metastasis.

The human papillomavirus genome contains a viral oncogene, *E6*. Oncogene *E6* codes for a viral protein that binds to, and destroys, the p53 protein in infected cells.

(b) Explain why the viral oncogene *E6* increases the chances of cancer developing.

…….. [3]

1 Viral protein encoded for by *E6* binds to and destroys p53 protein which is a tumour suppressor protein that helps to regulate the cell cycle by **inducing cell cycle arrest, promoting DNA repair or apoptosis** of cells with DNA damage.

2 In the absence of p53, there will be no cell cycle arrest, resulting in **the lack of time for the cell to repair its DNA.** Cells are **able to bypass the cell cycle checkpoints and evade apoptosis.**

3 This **increases the chance for the accumulation of mutations** that activates other oncogenes or inactivate tumour suppressor genes, hence increasing the chances of cancer developing.

(c) Suggest why mutations in the *p53* gene are common in most other human cancers, but rare in cervical cancer.

…….. [3]

1 In cervical cancer, the viral protein coded by *E6* binds to and destroys p53 protein. This produces the **same effect as a loss of function mutation in the *p53 gene.***

2 Even in the absence of *p53 gene* mutation, the HPV infected cells are unable to induce cell cycle arrest, promote DNA repair or apoptosis.

3 This is unlike most other human cancers as they are **not caused by a virus infection.** Instead, it is the accumulation of gene mutations (eg *p53 gene* mutation) that results in the uncontrolled proliferation of cells.

[Q5 Total: 10]

QUESTION 6

In mice, fur colour is controlled by several genes.

Allele **A**, which allows the expression of other fur colour genes, is dominant to **a**. Mice with the genotype **aa** are albino (white), as any other fur colour gene present cannot be expressed.

Allele **B**, which causes agouti (brown) fur, is dominant to the allele **b**. Mice that express the genotype **bb** have black fur.

(a) State the name of this type of interaction between two genes

…… [1]

1 Recessive epistasis

(b) State **all** the possible genotypes for:

(i) a mouse with fur colour that is albino (white)

…… [1]

1 aaBB, aaBb, aabb

(ii) a mouse with fur colour that is black

…… [1]

1 AAbb, Aabb

(iii) a mouse with fur colour that is agouti (brown).

…… [1]

1 AABB, AABb, AaBB, AaBb

(c) For each of the following genetic crosses, use a genetic diagram to explain the results. Show the parental and offspring genotypes and the expected ratio of offspring phenotypes.

(i) black mouse x albino mouse produces 27 agouti and 24 albino offspring

…… [3]

Parental phenotypes:	Black mouse		x	Albino mouse
Parental genotype:	Aabb		x	aaBB
Parental gametes:	(Ab)	(ab)		(aB)

Offspring genotypes:

Punnett square:

	(Ab)	(ab)

(aB)	AaBb (agouti)	aaBb (albino)

Offspring phenotypes: Agouti Albino
Offspring phenotypic ratio: 1 : 1

1 parental genotypes with circled gametes
2 offspring genotypes
3 expected ratio of offspring phenotypes

(ii) black mouse x black mouse produces 28 black and 10 albino offspring
……..…….. [3]

Parental phenotypes:	Black mouse		x	Black mouse	
Parental genotype:	Aabb			Aabb	
Parental gametes:	(Ab)	(ab)		(Ab)	(ab)

Offspring genotypes:

Punnett square:

	(Ab)	(ab)
(Ab)	AAbb (black)	Aabb (black)
(ab)	Aabb (black)	aabb (albino)

Offspring phenotypes: Black Albino
Offspring phenotypic ratio: 3 : 1

1 parental genotypes with circled gametes
2 offspring genotypes
3 expected ratio of offspring phenotypes

[Q6 Total: 10]

QUESTION 7

1 Fig. 7.1 shows the change in the energy of electrons as they pass along the electron transport chain (ETC) during oxidative phosphorylation.

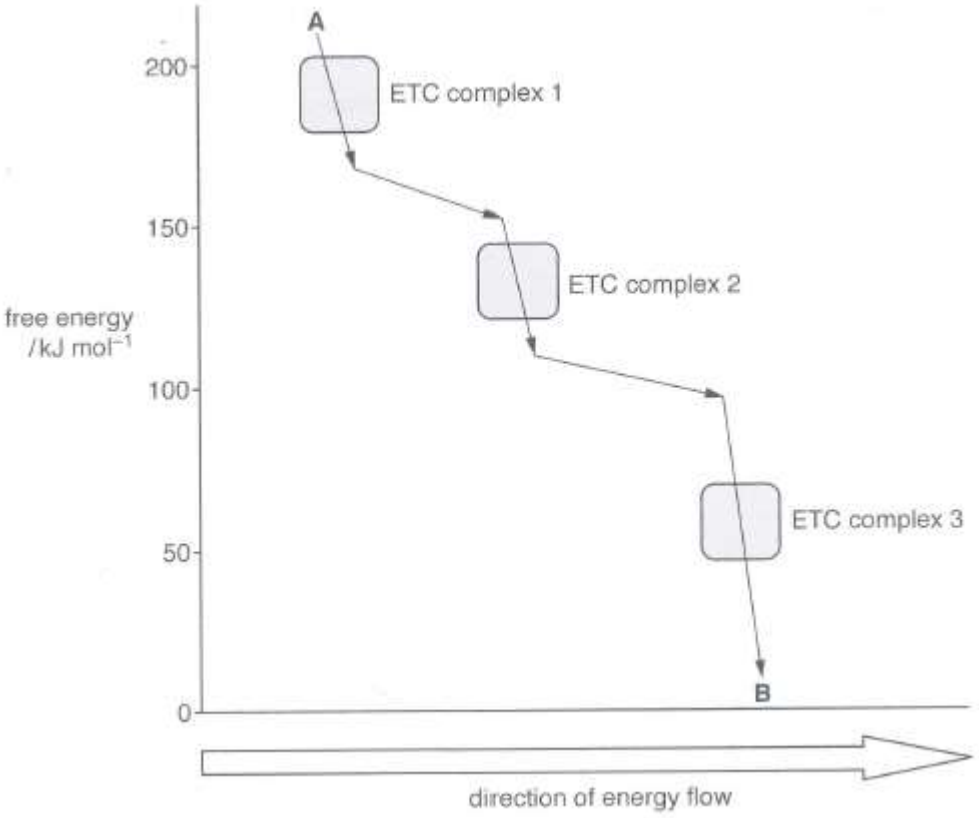


Fig. 7.1

(a) Carrier molecule A is the source of electrons for the electron transport chain shown in Fig. 7.1.

(i) Name carrier molecule **A**.

……[1]

1 NADH / Reduced NAD (nicotinamide adenine dinucleotide)

Examiner's report: Nearly all candidates correctly deduced the identity of **A** from Fig. 7.1.

(ii) Name the stages in respiration where carrier molecule **A** is formed.

……[1]

1 glycolysis, link reaction and Krebs cycle (for NADH)

Examiner's report: Better performing candidates named all three stages in which molecule **A** is formed. Some candidates omitted the Krebs cycle.

(iii) Explain why there is only a small amount of carrier molecule **A** in the cell at any one time.

……[1]

1 Once formed, NADH being of higher energy level immediately donates its electron, to the ETC Complex 1 which is of lower energy level. NAD is regenerated ;

Inter-conversion of NADH to NAD and transient existence of NADH (explained there is only a small amount of NADH in the cell at any one time).

[For understanding: NADH's electron come from the splitting of hydrogen atom into its constitutent hydrogen ion (H^+) and electron.]

Examiner's report: The majority of candidates had a sound conceptual understanding of the principles involved and were able to address this question.

(b) Describe how the energy released from the flow of electrons in Fig. 7.1 results in the formation of ATP.

……[3]

1 As electrons are transferred from ETC complex 1 (average about 190 kJ mol^{-1}) to ETC complex 2 (average about 140 kJ mol^{-1}) and from ETC complex 2 to ETC complex 3 with ETC complexes in the order of decreasing energy but increasing electronegativity, energy is released ;

2 Energy released is used to pump H^+ ions from the mitochondrial matrix into the intermembrane space, generating a proton gradient across the inner mitochondrial membrane ;

3 H^+ ions diffuse from the intermembrane space into the mitochondrial matrix down a concentration gradient through ATP synthase which catalysed the synthesis of ATP from ADP and P_i (inorganic phosphate).

Examiner's report: This was well answered by the majority of candidates, with detailed recall of the full sequence of events. Some candidates confused oxidative phosphorylation with photophosphorylation.

(c) Identify molecule **B** and explains what happens at the end of the electron transport chain in order to form molecule **B**.

……[3]

1 (Molecule **B** is) <u>water</u> ;

2 The hydrogen atoms from NADH are split into its constituent hydrogen ions (H^+) and electrons ;

3 While H^+ remains in solution in the mitochondrial matrix, electrons are transferred through the electron carriers in the ETC complexes and pass from ETC complex 3 to the <u>final electron acceptor</u>, oxygen ;

4 The electrons recombined with O_2 and H^+ ions to form water, catalysed by cytochrome oxidase.

Examiner's report: Most candidates correctly identified molecule **B** and considered the final electron acceptor in their answers.

(d) Suggest how the flow of electrons in photophosphorylation is different from the flow of electrons shown in Fig. 7.1.

……[3]

	Flow of electrons in Photophosphorylation	Flow of electrons in Oxidative phosphorylation (in Fig. 7.1)
1	Electron donor in non-cyclic pathway = water. Electron donor in cyclic pathway = PS I	Electron donors = NADH and $FADH_2$.
2	For cyclic pathway only: Electrons are passed on to electron carriers within the same , protein complex ie photosystem I	Electrons are passed on to electron carriers in different protein complexes, ETC complex 1, ETC complex 2 and ETC complex 3.
3	Final electron acceptor in **non-cyclic pathway** = $NADP^+$ Final electron acceptor in **cyclic pathway** = PS I	Final electron acceptor = oxygen.

Examiner's report: Candidates provided a wide range of valid responses to this question.

[Q7 Total: 12]

QUESTION 8

Fig. 8.1 shows a signalling cascade inside a cell.

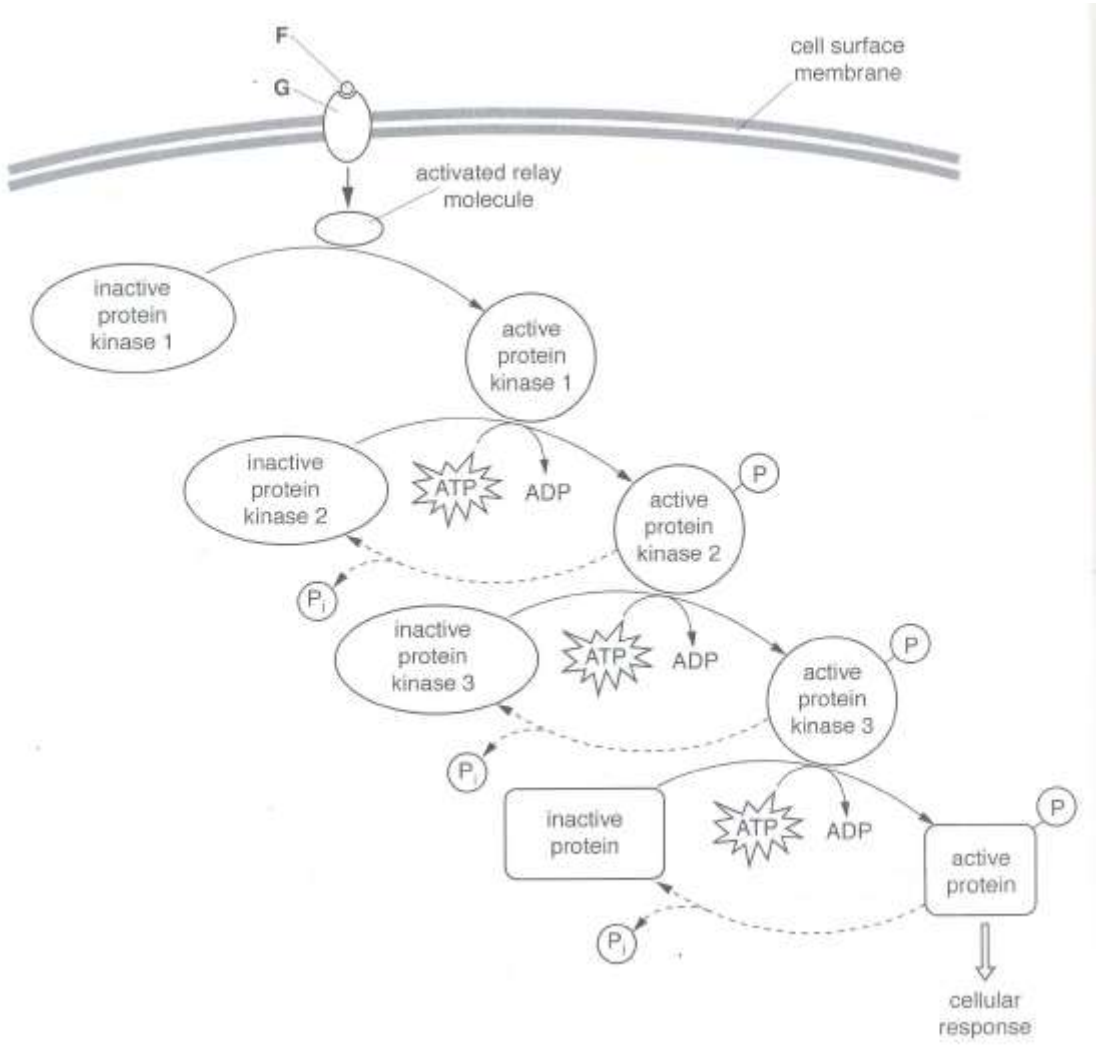


Fig. 8.1

(a) Name the structures labelled **F** and **G** in Fig. 8.1.

F .....
G .....
[2]

1 **F** : ligand / signal
2 **G** : Receptor

Examiner's report: Most candidates correctly named both structures.

(b) Explain how the specificity of the response is determined in different cells.

.....[2]

1 Receptor is a transmembrane protein that has extracellular domain with 3D conformation of ligand-binding site complementary in shape to that of the ligand ;

2 (Receptor has) intracellular domain with 3D conformation (which is exposed upon receptor activation) complementary in shape for binding of specific relay proteins on the cytoplasmic side to activate the relay protein ;
[Different relay molecules activated based on specificity of receptor and ligand interaction, resulting in different cellular responses.]

Examiner's report: Candidates were able to address this question comprehensively.

(c) Describe how each protein kinase in the cascade is activated.

……[1]

1 Active protein kinase 1 adds phosphate group from ATP to inactive protein kinase 2, phosphorylating it to activate it into active protein kinase 2. Active protein kinase 2 then phosphorylates and activates protein kinase 3 in the same manner. Thus, each protein kinase is activated by phosphorylation by an active protein kinase.

Examiner's report: Most candidates realised that phosphorylation was the key concept required here.

(d) Explain how the signalling cascade shown in Fig. 8.1 amplifies the signal during transduction.

……[2]

1 One active protein kinase 1 can activate many protein kinase 2, each of which can activate many protein kinase 3. Each active protein kinase 3 can activate many active proteins.

2 The number of activated products of each step in the cascade is greater than the previous step, so a small number of signal molecules can rapidly produce a much greater number of final products (active proteins).

Examiner's report: Some candidates simply described the events shown in Fig. 8.1, rather than considering how these events result in signal amplification.

(e) Suggest how mutations in genes coding for the proteins shown in Fig. 8.1 could lead to cancer by altering signalling pathways.

……[3]

Gain of function mutation of proto-oncogene to oncogene:

1 Gene mutations could result in receptor being constitutively relaying message even without ligand binding to it ;

2 This will result in continuous signal transduction even in the absence of a ligand where relay proteins are continuously being activated causing protein kinases to be continuously activated and signal amplification to occur ;

3 This in turn will result in continuous cellular response. When cellular response is cell division, cells are triggered to divide continuously leading to uncontrolled cell division and hence cancer eventually.

OR

1 Gene mutations could result more of the protein kinase to be in permanently in active form ;
2 This will result in protein kinases in each step to be continuously activated and hence signal amplification to occur ;
3 This in turn will result in continuous cellular response. When cellular response is cell division, cells are triggered to divide continuously leading to uncontrolled cell division and hence cancer eventually.

Loss of function mutation of tumour suppressor gene

1 Gene mutations could result in non-functional proteins such as non-functional receptor ;
2 There will not be any receptor-ligand interaction, hence no signal transduction and eventually no cellular responses ;
3 Where cellular response is to synthesise proteins such as tumour suppressor protein e.g p53 which is required to limit cell divisions or to initiate programmed cell death (apoptosis), the lack of cellular response will result in uncontrolled cell divisions and hence cancer eventually.

OR

1 Gene mutations could result in non-functional proteins such as non-functional protein kinases ;
2 Even though there is receptor-ligand interaction, there will not be any signal transduction as protein kinases at each step are not activated and hence no cellular responses ;
3 Where cellular response is to synthesise proteins such as tumour suppressor protein e.g p53 which is required to limit cell divisions or to initiate programmed cell death (apoptosis), the lack of cellular response will result in uncontrolled cell divisions and hence cancer eventually.

Examiner's report: Many candidates provided coherent explanations in which mutations were linked to constant activation of signalling pathways with roles in stimulating cell division. Fewer considered the possibility that signalling pathways may also limit cell division, in which case relevant mutations would need to result in deactivation of signalling pathways.

[Q8 Total : 10]

QUESTION 9

Fig. 9.1 shows the islands between mainland Asia and Australia.

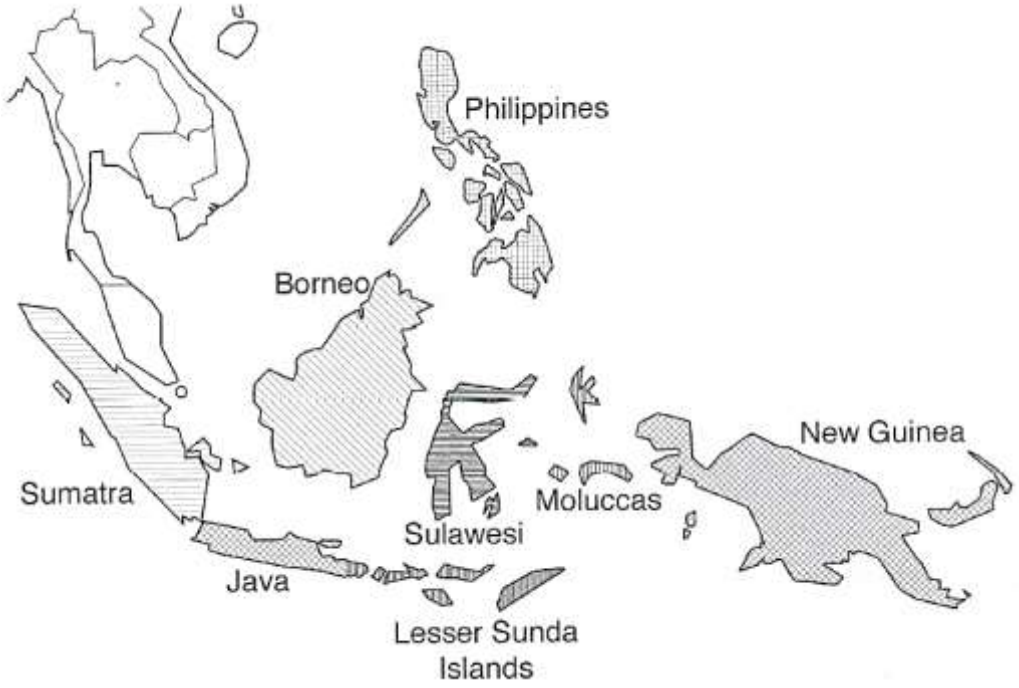


Fig. 9.1

Fig. 9.2 shows the sizes of the different islands and the total number of plant species on each island.

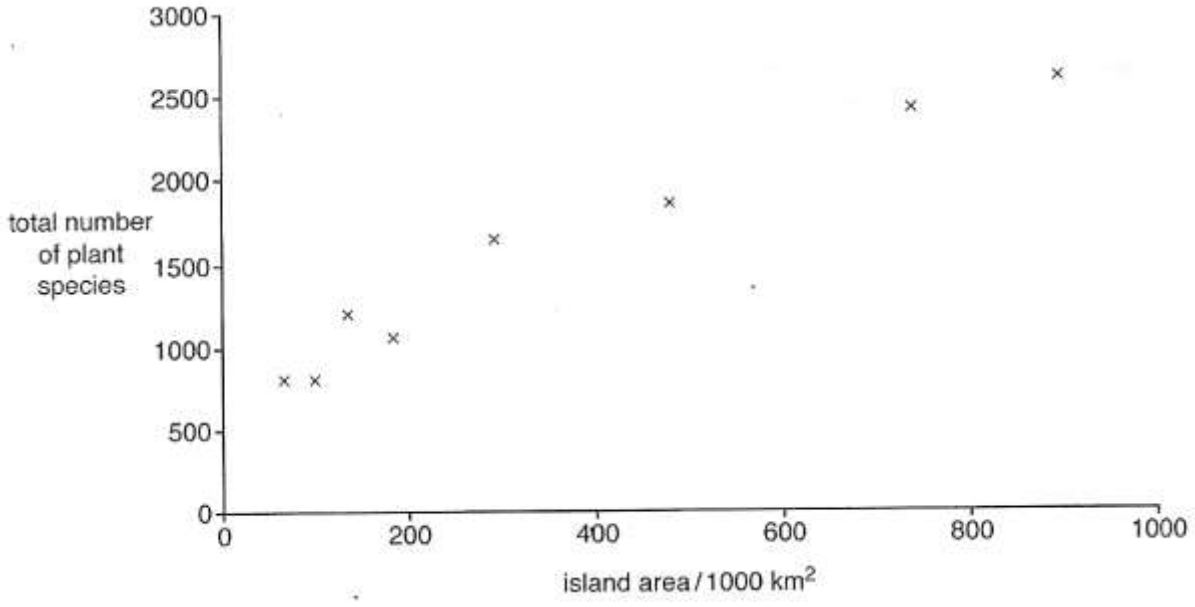


Fig. 9.2

(d) (i) Describe how the total number of plant species varies with island size, as shown in Fig. 9.2.

…… [3]

1 The larger the island area, the higher the total number of plant species present.
2 As island area increased from 50 000 km^2 to 900 000 km^2,
3 the total number of plant species increased proportionately from 750 to 2600 species.

Examiner's comments:
Most candidates provided descriptions to an appropriate level of detail. Some misinterpreted the units for area.

(ii) Suggest an explanation for the relationship that you have described in **(a)(i)**.

…….. [2]

1 Islands with larger area have **more complex microhabitats** than islands with smaller area (i.e. smaller area tend to be more homogenous).
2 Different microhabitats exert **different selection pressures** on the plant organisms living in them,
3 Causing more **adaptive radiation** to produce more species of plants in larger islands (that specialise in the various microhabitats present).

Examiner's comments:
Many explanations were vague and considered factors that would increase the number of individuals of each species, rather than increasing the number of species present. Some candidates included descriptions of speciation that were not relevant to the question.

(e) Islands often have many unique species of plants and animals that are not found anywhere else.

Explain why this is so.

…….. [5]

1 When animals or plants colonise a new island, many unoccupied niches are available for individuals to occupy.
2 As islands are **geographically isolated** from other land masses by bodies of ocean,
3 <u>gene flow</u> between animals and plants on the island and those on neighbouring islands is **disrupted**.
4 Different islands exert **different selection pressures** on the organisms living in them, leading to <u>natural selection</u>.
5 Over time, <u>mutations</u> and <u>genetic drift</u> can arise and cause **changes in allele frequencies** over time.
6 <u>Allopatric speciation</u> can occur to produce unique species on islands that are not found anywhere else.

Examiner's comments:
Speciation was well known by candidates and there were many detailed answers giving full accounts.

[Q9 Total: 10]

QUESTION 10 TNG

Fig. 10.1 shows infection of a cell by a virus.

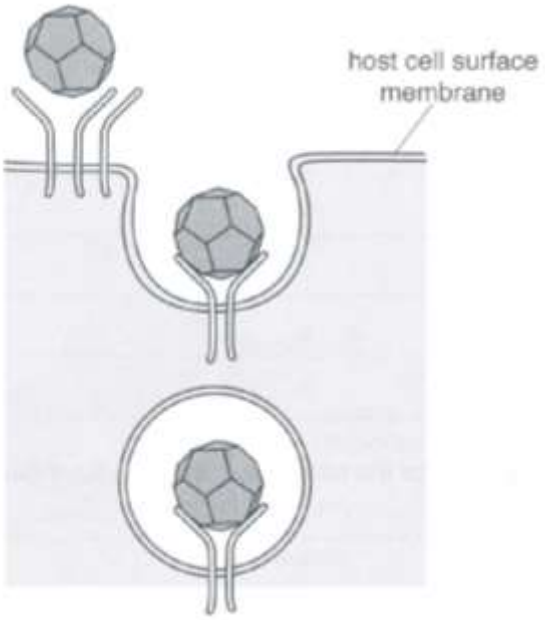


Fig. 10.1

Fig. 10.2 shows molecules of immunoglobulin G (IgG) attaching to viruses.

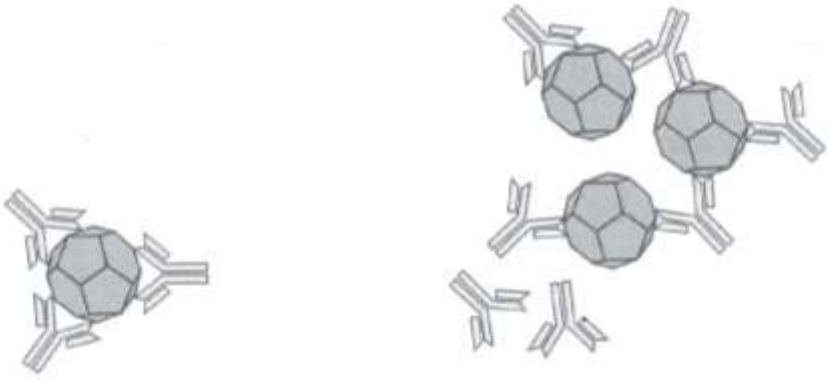


Fig. 10.2

(a) Describe how the molecules of IgG attach to the viral capsid.

…….. [2]

1 IgG has an <u>antigen-binding site / variable region</u> (made by light and heavy chains) **recognise and bind** to capsid proteins / epitopes
2 Ref. 3D conformation complementary in shape to that of the viral capsid proteins

Examiner's comments:
Candidates were familiar with the steps in viral infection in cells and the role of immunoglobulins in preventing viral entry into cells.

Some candidates omitted correct reference to the complementary nature of the attachment.

(b) Using information shown in Fig.10.1 and Fig. 10.2, explain how the attachment of IgG prevents the virus from infecting the cell.

…….. [3]

1 Ref. neutralisation of capsid after IgG attachment to capsid epitope via antigen-binding site / IgG surrounds the capsid
2 Virus capsid **cannot** bind to receptors on host cell plasma membrane
3 Ref. receptor mediated endocytosis **cannot** occur, preventing infection of cell
4 Ref. agglutination of multiple viruses bound by variable region of IgG; preventing infection of cell

Examiners' comments:
Many candidates considered the effect of IgG binding to the virus, but some did not go on to explain how this could prevent the virus from infecting the cell.

QUESTION 11

Fig. 11.1 shows the percentage cover of live corals and the density of herbivorous (plant-feeding) fish on a coral reef over a number of years. Due to unusually warm water, many of the corals living on the reef died in 1998.

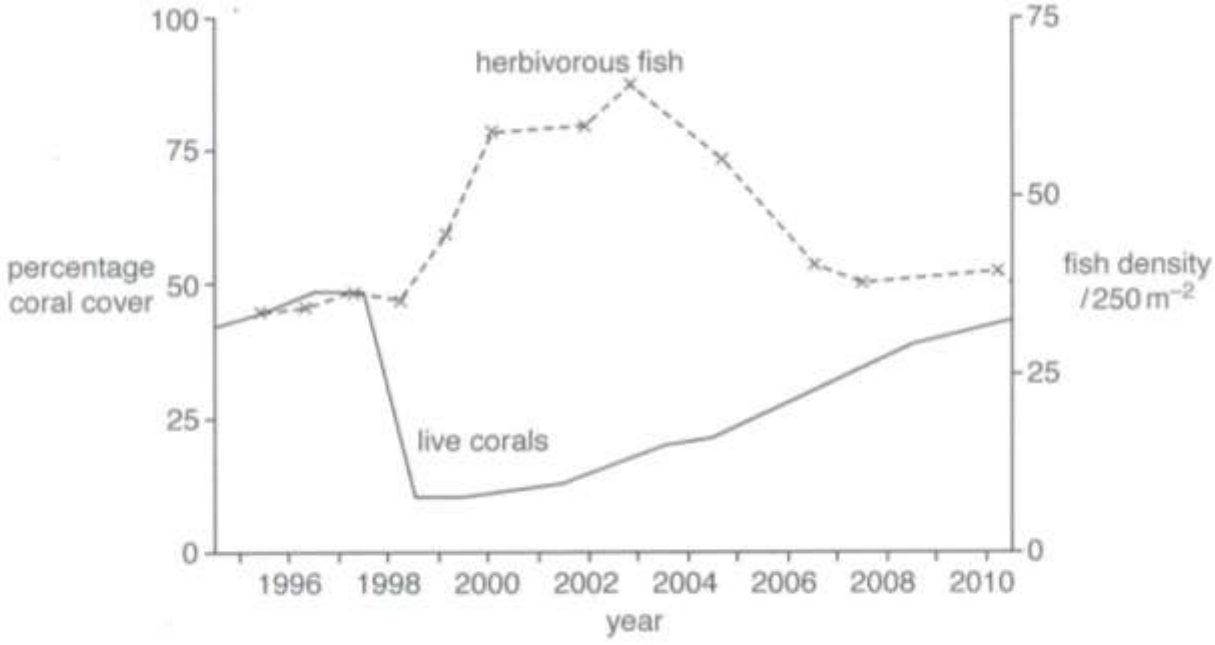


Fig. 11.1

(a) Describe the change in percentage cover of live corals shown in Fig. 11.1.

.....[3]

1 Slight **increase** in percentage coral cover from around **40% to 50%** observed from **mid 1994 till mid 1997**,
2 Followed by a sharp **decrease/dip** in percentage coral cover (from 50%) **to 10% till mid 1998,**
3 Percentage coral cover start to **increase** steadily (from 10%) **to around 50% after mid 1998** (to year 2011);

Examiners' Comments:
Many candidates described in detail the changes in percentage cover of live corals over the years shown in Fig. 11.1.

(b) Suggest why the density of herbivorous fish increases after the death of many of the corals in 1998.

.....[1]

1 ref. death of corals leading to expulsion of more zooxanthellae/algae for herbivorous fish to feed on

Examiners' Comments:
Many candidates did not consider the fact that herbivorous fish feed on plants. This information was provided in the question stem.

(c) Suggest how the increase in herbivorous fish helps to restore the percentage cover of live corals over several years.
.....[1]

1 Herbivorous fish feed on plants living in the same habitat that may compete with the corals for space/nutrients/resources etc;

Examiners' Comments:
Many candidates did not consider the interactions that would occur between the herbivorous fish and other organisms living on the coral reef.

PAPER 3

QUESTION 1

The rare genetic. disease phenylketonuria (PKU) is inherited in an autosomal recessive manner. The disease can be detected soon after birth and can then be treated by feeding the baby a modified diet, which is low in the amino acid phenylalanine and supplemented with the amino acid tyrosine. However, if the disease is undetected, the PKU phenotype will be expressed.

(a) (i) State what is meant by a genetic disease and explain how genetic diseases are caused. [4]

- A genetic disease is a **dysfunction / illness** caused by **abnormalities** in the **genome** that can be **inherited** or caused by **new mutations** or changes to the **DNA**
- Caused by the inheritance of mutations in genes due to **base substitutions, insertions and/or deletions** where the **gene product** is **unable to carry out its normal function** or that it carries out an **abnormal function**
- Can also be caused by **chromosomal aberration**, resulting in a change in the **number or structure** of chormosomes
- **Non-disjunction** in mitosis or meiosis leads to a change in the number or chromosomes while **chromosomal breaks** occurring during mitosis or meiosis results in a change in structure of chromosome

Examiner's comments:
Many candidates were able to state the meaning of the term genetic disease and explain its hereditary nature, including possible causes. Some candidates went on to explain in more detail the nature of genetic change. Some responses demonstrated logical development from DNA base change to primary polypeptide change to non-functional protein.

(ii) Justify the claim that the PKU phenotype is the result of genotype and the environment acting together. [2]

- A **homozygous** person with **2 copies** of the **recessive allele** responsible for the PKU phenotype will exhibit the phenotype if he/she **does not undergo treatment**
- However, if the individual is **treated**, in the form of receiving a modified diet **low in phenylalanine** and **tyrosine supplementation**, the **environment** of the individual is thus tweaked to prevent expression of the PKU phenotype
- If the individual does not have the recessive allele or if the person only has one copy of the recessive allele with the other allele allowing the individual to produce a functional enzyme, the person will not exhibit the PKU phenotype

Examiner's comments:
Many candidates added insight to the question stem material to explain how genotype and the environment act together on the phenotype of PKU. Some largely repeated the information in the question stem without pertinent comment or analysis. The dietary effect was explained more successfully than the influence of genotype.

(iii) Phenylalanine and tyrosine are both amino acids. Describe two ways in which these molecules are similar in structure and explain why these features are important for their function. [4]

- Both have an **amino group** linked to the **central α-carbon** that is able to accept a H^+ to become $NH3^+$ in aqueous medium
- Both have a **carboxyl group** linked to the **central α-carbon** that is able to lose a H^+ to become COO^- in aqueous medium
- The presence of amino and carboxyl groups in amino acids allows them to exist as **zwitterions** in an aqueous medium at their isoelectric point and thus, allowing them to be **amphoteric** to function as **buffers**
- The **carboxyl group** of **one amino acid** and the **amino group** of **another** amino acid can form a **peptide bond** by means of a **condensation** reaction with the **loss of a water molecule**. This allows the amino acids to act as **monomers** that can join together to form a polypeptide chain

Examiner's comments:
Many candidates correctly identified the chemical groups that are shared by amino acids and explained their importance. Some candidates did not note that the question was addressing similarities between phenylalanine and tyrosine. These candidates focused instead on differences between the amino acids and discussed the importance of R groups for tertiary level folding.

(b) Babies are usually tested for PKU within a few days of birth. The most common test is called the Guthrie assay, which involves the following steps.

- The baby's heel is pricked and a drop of blood transferred to a piece of filter paper.
- A small disc of the filter paper is placed on an agar gel containing a suspension of thebacterium Bacillus subtilis, together with β-2thienylalanine. β-2thienylalanine is a synthetic amino acid with a similar shape to phenylalanine. It inhibits bacterial growth.
- The agar gel is incubated for 24 hours.

The results are interpreted as follows.
- Normal blood containing a low concentration of phenylalanine has no effect on the inhibition in growth of the bacteria.
- Blood from a baby with PKU provides enough phenylalanine to overcome the inhibition by β-2thienylalanine and allow growth of the bacteria present under the disc. These grow into a visible colony within 24 hours.
- The diameter of the bacterial colony around the filter paper disc is compared to reference discs loaded with a known concentration of phenylalanine to estimate the concentration of phenylalanine in the baby's blood.

(i) In the case of a positive result, further tests are needed to determine whether the high phenylalanine concentration is due to the presence of PKU alleles.

Suggest, in outline, a procedure that could determine whether or not a PKU allele is present. [4]

- Baby's heel is pricked to obtain a sample of blood and genomic DNA is isolated from the sample

- Forward and Reverse primers designed for PKU allele and Polymerase Chain Reaction conducted using the designed primers
- Restriction enzyme that recognises a restriction site that is only present in the PKU alleles and not in other alleles at the gene locus is added before gel electrophoresis is conducted
- Blotting process: DNA fragments (in the gel) are transferred onto nitrocellulose paper/membrane
- ref. nucleic acid hybridization - treatment with a single-stranded radioactive DNA probe which binds/anneals to PKU allele via complementary base pairing + Bands are visualized on X-ray film / autoradiography
- Presence of band(s) that correspond to the banding pattern of a digested PKU allele (after probing) indicates the presence of PKU allele

Examiner's comments:
Answers generally showed familiarity with various genetic tests and procedures that could, in combination, identify whether a PKU allele was present. Many candidates described a coherent procedure that linked a number of relevant practical techniques.

Some candidates did not identify the source of DNA to be tested or simply added blood to the wells in a gel. Others only based their answers around details of the Guthrie assay already given in the question, rather than outlining a confirmatory procedure to follow on from a positive Guthrie test.

(ii) A baby is treated with antibiotics from birth for a particular problem. During this treatment, a blood sample is taken for the Guthrie assay.

Predict and explain the effect of this treatment on the result of the Guthrie assay for a baby that is homozygous for the PKU alleles. [3]

- **No bacterial growth** around the filter paper containing the baby's blood sample on the Guthrie assay is predicted after 24 hours, and hence a **false negative** result is predicted. The result is thus **not accurate**.
- **Antibiotics** given to the baby would be present in the **blood** sample in a concentration high enough to **inhibit the growth** of the bacteria in the Guthrie assay.
- Although inhibition by β-2-thienylalanine can be overcome by a high enough concentration of phenylalanine in the blood sample, the inhibition by antibiotics is not prevented or overcome by any factors present.

Examiner's comments:
Many candidates showed a thorough understanding of how the scenario described would lead to a false negative result from the Guthrie assay. Knowledge of how antibiotics inhibit bacterial cell growth was shown, as was evidence of familiarity with the appearance of colonies of bacteria cultured on agar.

A number of candidates did not understand the implications of antibiotic treatment in this context. Some stated that the antibiotic treatment would not 'work' in combating PKU or that the antibiotic would cause the level of phenylalanine to change. Others confused antibiotics with antibodies.

(iii) During the incubation step of the Guthrie assay, if phenylalanine is not present then bacteria use β-2-thienylalanine for protein synthesis. β-2-thienylalanine is added to the growing polypeptide chain instead of phenylalanine. This inhibits bacterial growth. Raising the concentration of phenylalanine overcomes this inhibition.

Suggest how β-2-thienylalanine inhibits bacterial growth. [3]

- β-2-thienylalanine has a **similar structure** to phenylalanine and thus, β-2-thienylalanine would have a **complementary 3D conformation** to the **active site** of the **amino-acyl tRNA synthetase** meant for phenylalanine. β-2-thienylalanine would hence be able **bind** to the active site of the enzyme, allowing β-2-thienylalanine to bind to phenylalanine's cognate tRNA.
- Therefore, β-2-thienylalanine would compete with phenylalanine for the active site of the enzyme, acting as a **competitive inhibitor**.
- β-2-thienylalanine would be added to the polypeptide chain instead of phenylalanine, leading to a change in **primary structure** of proteins in the bacteria. **3D conformation** of these proteins would also change as a consequence, leading to the protein being **non-functional or having a reduced function**. As proteins are important for bacterial growth, e.g. for ATP synthesis, growth will be inhibited as a result.

Examiner's comments:
Many responses demonstrated excellent understanding of translation and the role of enzymes in loading tRNA molecules with the correct amino acids. Some responses logically developed the idea that an incorrect amino acid in the primary structure could produce a non-functional or truncated protein.

(c) Fig. 1.1 shows the biochemical pathways affected by PKU. The letters Q, R, S, T and V represent the enzymes that catalyse each reaction.

Individuals who are homozygous recessive for the PKU allele make a non-functional version of enzyme Q. The resulting high concentration of phenylalanine causes transporter proteins that move large amino acids into the brain to become blocked by phenylalanine, preventing the transport of other amino acids into the brain.

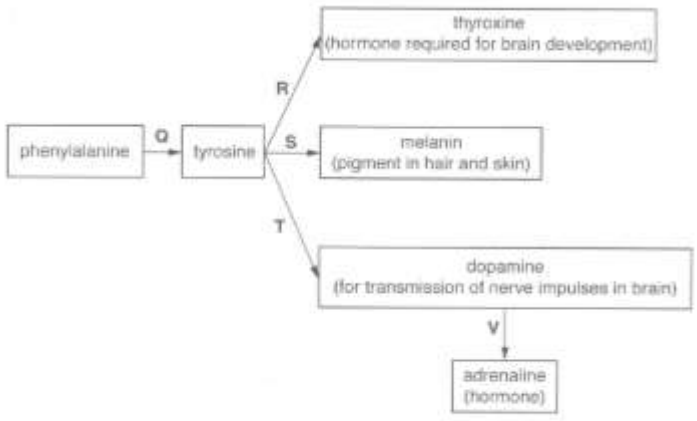


Fig. 1.1

With reference to **Fig. 1.1** and the **information given**, explain how loss of function of enzyme Q causes poor intellectual and behavioural development as well as very fair skin and hair. [4]

Loss of function of enzyme Q leads to:

- an **accumulation** of **phenylalanine** that blocks transporter proteins which move large amino acids into the brain, thereby preventing the transport of these amino acids into the brain, leading to **poor intellectual and behavioural development** due to a **deficiency of these amino acids in the brain for protein synthesis**
- **decreased conversion** of phenylalanine to tyrosine and thus **insufficient** tyrosine for conversion to thyroxine which is the hormone required for brain development. Thus, leading to **insufficient** levels of thyroxine and hence **poor intellectual and behavioural development**
- **decreased conversion** of phenylalanine to tyrosine and thus **insufficient** tyrosine for conversion to dopamine which is required for the transmission of nerve impulses in brain. Thus, leading to **insufficient** levels of dopamine and hence **poor intellectual and behavioural development**
- **decreased conversion** of phenylalanine to tyrosine and thus **insufficient** tyrosine for conversion to melanin which is the pigment in hair and skin. Thus, leading to **low levels** of pigments that result in **fair skin and hair**

Examiner's comments:
Many candidates demonstrated logical and consistent mental processing to interpret the diagram correctly.

(d)

Table 1.1 shows the frequency of newborn babies who test positive for PKU in different areas of the world.

Table 1.1

geographical area	climate	frequency of newborn babies who test positive for PKU
Northern Europe	cold, wet	1 in 10 000
South East Asia	hot, wet	1 in 60 000
Scandinavia	cold, dry	1 in .....
sub-Saharan Africa	hot, dry	1 in 100 000

(i) In Scandinavia, 1 in 140 people are carriers of the PKU allele.

Complete Table 1.1 by calculating the frequency of newborn babies positive for PKU that would be expected in Scandinavia.

You should show your working. [2]

Probability of two heterozygous individuals mating
= 1/140 x 1/140
= 1/19600

Probability of a child from 2 heterozygous individuals being positive for PKU
= ¼

Frequency of newborn babies positive for PKU
= 1/19600 x ¼
= 1/78400

Therefore, answer is 1 in **78400**

Note: probability of 2 homozygous individuals mating is insignificant, thus, there is no need to take that into consideration in the calculation

Examiner's comments:
Not all candidates could combine the probability of two carriers having a child with the expected offspring ratio from such a cross, to calculate the frequency of new-born babies testing positive for PKU. For many candidates, this two-step exercise in logical numerical thinking was straightforward.

(ii) The difference in frequency of PKU in different parts of the world may be due to environmental conditions. In wet climates, some species of fungi can grow on stored grain and produce a toxin, ochratoxin A.

- Eating ochratoxin A increases the chance of developing renal cancer.
- Heterozygote carriers of PKU have some resistance to ochratoxin A and, compared to individuals without the PKU allele, are less likely to develop renal cancer if they ingest the toxin.

Explain how evolution could have resulted in PKU being more common in the Northern European population compared to that of sub-Saharan Africa. [4]

- Sub-Saharan Africa has a **dry** climate while Northern Europe has a **wet** climate. Thus, Northern Europeans who are living in wet climates have a **higher probability of consuming ochratoxin A** due to some species of fungi being able to grow on stored grain in wet climates to produce the toxin
- Thus, Northern Europeans are exposed to a **greater risk of developing renal cancer** as a result of this higher probability of toxin consumption which acts as a **selection pressure**
- With the presence of such selection pressure, individuals who are **heterozygote carriers of PKU** are at a **selective advantage** as compared to homozygous individuals without the PKU allele as the heterozygotes have some **resistance** against the toxin as compared to the homozygous individuals
- This heterozygote advantage is **not present** in sub-Saharan Africa due to the absence of such **selection pressure**
- Thus, the **PKU allele** will **persist at a higher frequency** in the Northern European population as heterozygotes in this region can **survive and reproduce better** to **pass down the PKU allele** to their offspring, inevitably resulting in **PKU being more common**

Examiner's comments:
Few responses made explicit reference to natural selection, but many correctly identified the selection pressure, the genotype of the individuals selected for and the effect this had on the frequency of the PKU allele in Europe. Some candidates were not specific in distinguishing adequately between genotypes and did not use the appropriate term, *allele*, where it was relevant in their descriptions.

QUESTION 2

A new therapy to treat HIV infection is designed to work as follows.

- Blood stem cells from a patient infected with HIV are extracted.
- The gene for the receptor protein CCR5 is made inactive in the stem cells by a process called gene editing.
- The modified stem cells are put back in the patient's bone marrow.
- The patient makes new T lymphocytes that lack the receptor protein CCR5.
- HIV cannot infect the new T lymphocytes as it cannot bind to cells lacking CCR5.

(a) Explain the normal function of blood stem cells.

……[4]

[Function]

1. For replacement of worn out blood cells from wear and tear, fighting infections in diseases and from injury

[Explain]

2. Blood stem cells are **multipotent**
3. Able to **differentiate** into cells of **limited range of a specific lineage** through **asymmetric division**
4. E.g. red blood cells, white blood cells, and platelets

Examiner's comments:
Candidates were knowledgeable about the general functions and features of stem cells. Not all candidates related their answer specifically to blood stem cells.

(b) Outline the roles of named T lymphocytes in fighting infection.

……[4]

1. CD4 Helper T cells: T cell receptor recognizes and binds to B cells that presents antigens on MHC Class II, and releases **cytokines** to **activate B cells** into plasma cells
2. CD8 cytotoxic T cells: T cell receptor recognizes and binds to infected cells (Reject: pathogens) that presents antigens on MHC Class I,
3. leading to release of **perforins and granzymes** to lyse the infected cells
4. Memory T cells: takes part in secondary immune response; recognizes the same antigens and gives rise to **effector cells** in **short period of time** (helper T cells or cytotoxic T cells) upon reinfection by the same pathogen /antigens

Examiner's comments:
Candidates generally had some knowledge of the roles of cytotoxic, helper and memory T lymphocytes. Some candidates confused cell-mediated immunity with the humoral response, suggesting, for example, that cytotoxic T cells killed pathogens directly.

Other errors included describing the role of B memory cells at length, instead of T memory cells. Some candidates did not distinguish at all between different types of T lymphocyte.

(c) Comment on the ethical aspects of this new therapy.

……[2]

[Positive]

1. No involvement of human embryos / embryonic stem cells, which are potential source of life.
2. Ref. potential treatment in curing patient's ill health\

[Negative]

3. Ref. unknown safety or future side effects in a long run
4. Ref. need for patient to undergo invasive procedures to obtain blood stem cells from bone marrow
5. Ref. mutations that might arise from gene editing that will cause side-effects

Examiner's comments:

Most candidates considered the ethical implications of a therapy that does not use embryonic stem cells. Many made insightful, fact-based comments on possible problems that could arise from the therapy.

Economic arguments were sometimes developed, but few candidates commented on the ethical implications related to curing a person's ill health or requiring a patient to undergo an invasive procedure experimentally.

Some candidates relied on generic answers that were not applicable in this case.

QUESTION 3

Maize (corn) and soybean are important crop plants, which are grown for their edible seeds. In some areas of the world, such as North America and South East Asia, both crop plants are grown.

Table 3.1 lists key temperatures affecting leaf and stem growth and seed production in these two crop plants. For maize and soybean, the crop fails if few seeds are produced.

Table 3.1

crop plant	temperature range for optimum leaf and stem growth		temperature range for optimum seed production		temperature above which crop fails / ^{o}C
	minimum / ^{o}C	maximum / ^{o}C	minimum / ^{o}C	maximum / ^{o}C	
maize (corn), *Zea mays*	25	37	17	22	35
soybean, *Glycine max*	25	36	22	24	39

(a) Maize and soybean seeds contain nutrient molecules that derive from triose phosphate produced in the leaves.

Outline how photosynthesis produces triose phosphate.

……...[4]

1 (In stroma of chloroplast), CO_2 (1C molecule) enters the Calvin cycle and combines with a 5C compound called ribulose bisphosphate (RuBP) catalysed by RuBP carboxylase / Rubisco ;

2 An unstable intermediate 6C compound formed immediately splits into half to form 2 molecules of a 3C compound known as 3-phosphoglycerate (PGA) ;

3 PGA is reduced to 3-phosphoglyceraldehyde (PGAL) ie triose phosphate by the electrons from reduced NADP (NADPH) using ATP as an energy source. Both NADPH and ATP are produced from the light dependent reactions ;

4 For every 3 molecules of CO_2, there are 6 molecules of PGAL but only 1 molecule of PGAL exits the Calvin cycle to be used by the plant cell to synthesise carbohydrate like glucose which are used to synthesise nutrient molecule ;

Examiner's report: Most candidates outlined the main steps by which plants produce triose phosphate, but many went into much more detail than was required.

Errors included confusing the Calvin cycle with the Krebs cycle and confusing reduced NADP with reduced NAD. Carbon dioxide was not always identified as the molecule fixed.

(b) Explain the biochemical reasons why leaf and stem growth rates decrease at temperatures both above and below the optimum ranges, as shown in Table 3.1

……[3]

1 Leaf and stem growth rates are dependent on rate of photosynthesis which is dependent on factors affecting enzyme activities, one of which is temperature ;

2 At temperature below the optimum ranges, there is low frequency of effective collisions between enzymes (involved in photosynthesis) and substrates due to

low kinetic energy of both enzymes and substrate molecules. Hence low concentration of products formed per unit time. Less nutrients results for growth, thus growth rate decreases.

3 At temperature above the optimum ranges, enzyme may be denatured as the tertiary structure of enzymes is disrupted due to the increase in thermal agitation of enzyme molecules that causes hydrogen, ionic bonds and hydrophobic interactions maintaining the 3D conformation to be disrupted. Substrates unable to bind or bind ineffectively to enzyme's active sites to form enzyme-substrate complex.

4 At high temperature, the plant responds by the closure of stomata on the leave to minimise water loss. This results in less CO2 entering the leave for Calvin cycle of photosynthesis to occur and hence, less nutrients synthesised for growth to occur.

Examiner's report: Good responses linked leaf and stem growth to the activity of enzymes and summarised how enzyme activity is affected by temperature. Some candidates did not distinguish the effects of low and high temperatures.

Some candidates correctly considered less direct temperature-mediated effects on growth, such as stomatal closure.

(c) A country in South East Asia currently has the following temperature range:

- minimum temperature 21^{o}C
- mean temperature 27^{o}C
- maximum temperature 35^{o}C.

Some climate change models expect a 2^{o}C temperature rise over the next 100 years.

Suggest which one of the two crop plants should be grown in this country if the minimum, mean and maximum temperatures rise by 2^{o}C.

Use the data in Table 3.1 for leaf and stem growth, seed production and temperature above which crop fails to justify your answer.

……[3]

1 soybean, *Glycine max* (should be grown in this country) ;

2 With the current minimum temperature of 21^{o}C and maximum temperature 35^{o}C, both maize (corn), *Zea mays*, and soybean may have optimum leaf and stem growth but maize has a narrower range of 21 to 22^{o}C compared to soybean with a range of 22 to 24^{o}C for optimum seed production. Less seed produced would mean less crop plants can be grown. Without seed production, next generation crop plants cannot be grown ;

3 With expected 2^{o}C temperature rise, the country will have a new minimum temperature of 23^{o}C, maximum temperature 37^{o}C and mean temperature of 29^{o}C. At the maximum temperature 37^{o}C, crops fail for maize plant (35^{o}C) but not for soybean (39^{o}C). Soybean will have optimum leaf and stem growth and optimum seed production in the new temperature range and the maximum temperature (37^{o}C) is 2^{o}C lower than the temperature above which soybean fails (39^{o}C) ;

Examiner's report: Well-developed responses considered all three parameters affected by temperature in Table 3.1 and related these to the new minimum, mean and maximum temperatures in the South East Asian country.

Some candidates made incorrect deductions, for example by stating that the country's new temperature range fitted within the optimum range for leaf and stem growth of soybean.

QUESTION 4 Essay

(a) Describe the reproductive cycle of an enveloped virus such as influenza and explain why this is referred to as a reproductive cycle, not a life cycle. [15]

[Reproductive cycle of enveloped virus using influenza as example]

Attachment / Adsorption

1. Influenza virus infects the **epithelial cells** of the **respiratory tract**
2. **Haemagglutinin** on the **viral envelope recognises and bind** to **sialic acid receptors** on **host plasma membrane.**

Entry

3. The virus enters host cell by **receptor-mediated endocytosis,**
4. where **host** plasma membrane **invaginates** and pinches off
5. placing the virus in an **endocytic vesicle / endosome**

Uncoating

6. Acidic condition in the endosome activates the M2 ion/proton channel to allow **protons** into the **virus**, making the inside of the virus **acidic**.
7. This causes the **viral envelope** to **fuse** with the **membrane** of the **endocytic vesicle**
8. Viral **capsid** is then **enzymatically removed**
9. The viral genome is **released** and transported into the **nucleus** of the cell.

Replication

10. the **viral** RNA-dependent RNA polymerase uses **(-) sense RNA** as a template to synthesise a complementary **(+) sense RNA** strand,
11. which acts as a template to replicate more **(-) sense RNA viral genome**

12. the **viral** RNA-dependent RNA polymerase uses (-) sense RNA strand as a template to synthesise complementary **(+) sense RNA** strand which **migrates to the cytoplasm**,
13. where it serves as a template for **host** cell's ribosomes to synthesise viral proteins and viral enzymes during **translation**.

Maturation

14. Newly synthesised **viral envelope proteins**, haemagglutinin, neuraminidase, M2 ion channel, are transported through the rough endoplasmic reticulum and Golgi body, then **inserted** in **host plasma membrane.**
15. Newly synthesised **viral enzymes** (RNA-dependent RNA polymerase) and **viral nucleocapsid** are assembled.
16. The newly formed viral nucleocapsid and viral enzymes **migrate** into the cytoplasm to the region on the host plasma membrane where the glycoproteins and M2 channel have been inserted.

Release

17. The **host** plasma membrane pinches off to form the **viral** envelope via **budding,** enclosing the viral nucleocapsid and enzymes.
18. The sialic acid is cleaved and removed from the envelope of the new viral particle by neuraminidase;
19. This aids in the **release** of the new viral particles from each other, thus, **preventing agglutination** of viruses.
20. The new viral particles released are able to travel and **infect other cells**.

[Why reproductive cycle but not life cycle]

Viruses can have non-living characteristics and are thus, not considered alive.

Non-living characteristics of viruses:

1 unable to carry out any metabolic activity (such as synthesis of organic molecules such as sugars, fats and proteins) **outside of a host cell** due to no cytoplasm or organelles

2 unable to reproduce/multiply **unless inside living cells** as viral enzymes involved in reproduction only functions in the cytoplasm of a living host cell

However, viruses also show reproductive characteristics:

3 can **reproduce / carry out metabolic activities** (only when they are inside living host cells) because it contains enzymes which may be used in reproductive cycles

4 contains genetic material which can be **replicated** and passed on to progeny

5 contains genetic material which **carries coded information** that can result in the production of polypeptides

6 can result in viral progeny (through mutation) with phenotypes that are best suited to adapt and survive in a particular environment / undergo natural selection

[QwC] clearly expressed and well structured, using correct terms to address both parts of question

Examiner's comments:

Most candidates provided good descriptions of the reproductive cycle of an enveloped virus such as influenza. The level of detail was appropriate and did not obscure the main principles.

Some candidates omitted details or confused terms or ideas, particularly concerning replication of nucleic acids and assemblage of viruses.

Fewer candidates were able to explain why the reproductive cycle was not referred to as a life cycle.

Most answers were clearly expressed and addressed both parts of the question, but many lacked any form of structuring.

(b) Climatic factors affect the length of time that viruses remain in the environment, the survival and reproduction of their vectors and the susceptibility of humans to viral diseases. For example, when enveloped viruses, such as influenza, are exposed to the environment, their survival decreases as the temperature increases.

Discuss the possible impact of global warming on seasonal and geographical patterns of viral disease. [10]

[Geographical patterns of viral disease]
1. Ref. migration of **viral vectors** spreading diseases to **latitudes** further from the equator or to higher **altitudes**
2. Ref. how global warming might affect **insect** vectors e.g. named insect vectors like *Aedes aegypti* mosquito for dengue virus
3. Elaboration on **insect** vectors: e.g. increased metabolic and reproductive rate at higher temperatures; feeding behavior increases in mosquito vectors at increased temperatures; global warming reduces the risk of insects with narrow temperature tolerance from freezing to deaths at higher latitudes and altitudes; increased CO2 concentration in atmosphere reduces plant nitrogen concentration and increases secondary metabolites that lower the nutritional value of plants ➔ insect vectors that feed on such plants will have reduced growth and reproduction
4. Ref. effects of **increased rainfall** at some areas/ flooding / melting of ice ➔ increase standing water at **low-lying regions**, for breeding of more mosquito vectors
5. Ref. effects of **decreased rainfall** at some areas ➔ less insect vectors and less transmission
6. Ref. **permafrost** melting releases frozen organic matter and viruses ➔ higher viral infection at **Northern and Southern Hemispheres**
7. Increased temperatures at some **urban regions** led to human activities e.g. decrease in human infections as more people avoid staying outdoors etc

[Seasonal patterns of viral disease]
8. Ref. changes in the timing of periods when particular diseases would be at their most prevalent e.g. **longer and hotter summers** and **shorter and colder winters**
9. Elaboration: specific details such as named viral diseases or countries.
10. Ref. how global warming might affect **viruses** e.g. dengue and influenza viruses,
11. Elaboration on **viruses** impact: e.g. heat decreases virus infectivity due to enzymatic denaturation etc
12. Colder temperatures ➔ lowered immunity ➔ higher chance of viral borne disease transmission

Examiner's comments:
Candidates used a wide range of valid approaches to address this question. The commonest examples of viral diseases discussed were dengue and influenza, but some answers mentioned Ebola. A number of candidates incorrectly referred to malaria.

Many candidates provided well developed answers that considered both geographical and seasonal changes in patterns of viral disease as a consequence of global warming. Their responses often referred to migration of viral diseases to latitudes further from the equator or to higher altitudes and to changes in the timing of periods when particular diseases would be at their most prevalent. Such descriptions were linked to specific details such as named viral diseases or countries. Most candidates supported these conjectures with sound reasoning and included

discussion of effects on viral vectors.

Less well developed responses were often vague and lacked supporting explanations.

Common features of such responses included:
• detailed discussion of how global warming might affect viral vectors without referring back to the consequences for the viruses
• description of the current pattern of variation in incidence of disease through the year, rather than description of how these patterns might be affected by global warming
• discussion of the effects of global warming that were limited to temperature effects, without considering other relevant consequences such as changes in rainfall, flooding or melting of ice.

QUESTION 5 Essay

A 75 kg human consists of ten times more prokaryotic cells than eukaryotic cells, with a total prokaryote mass of at least 1kg. This assembly of prokaryotic cells is known as the human prokaryotic microbiome community.

(a) Outline the differences between typical prokaryotic and eukaryotic cells and state the methods by which these differences can be shown. [15]
(b) Explain the expected problems of trying to classify the prokaryotes present in the human prokaryotic microbiome community and describe advantages of using molecular methods for this process. [10]

(a) Outline the differences between typical prokaryotic and eukaryotic cells and state the methods by which these differences can be shown. [15]

	Prokaryotic cell	Eukaryotic cell
1	Cell has nucleoid not enclosed by any membrane ;	Cell has distinct nucleus bound by double-membrane ;
2	Cell has peptidoglycan cell wall ;	Animal cell does not have cell wall and plant cell has cellulose cell wall ;
3	Genome made up of circular DNA ; / Genome made up of one circular DNA ;	Genome contains linear DNA in nucleus ; / Genome contains multiple linear DNA in nucleus, the number of linear DNA molecules depends on the species of organism ;
4	Circular DNA is not complexed with histone and not packaged into nucleosomes ;	Linear DNA is complexed with histones and other proteins to form chromatin and DNA is packaged into nucleosomes ;
5	Cell has 70S ribosomes found free in cytoplasm ;	Cell has 80S ribosomes which may be attached to the endoplasmic reticulum or found free in the cytoplasm ;
6	Cell lacks membrane-bound organelles ;	Presence of many membrane-bound organelles such as mitochondria, chloroplasts, Golgi body ;
7	No introns within genes in prokaryotic genome ;	Presence of introns within genes in eukaryotic genome ;

8	Presence of operons, where two or more genes may be expressed and regulated as a unit ;	Absence of operons ;
9	Simple regulatory sequences such as promoters in prokaryotic genome ;	More complex regulatory sequences such as enhancers and silencers in eukaryotic genome ;
10	One origin of replication per molecule of DNA present in prokaryotic genome ;	Many origins of replication per molecule of DNA present in eukaryotic genome ;
11	Presence of independent small, double stranded, circular DNA called plasmids in cytoplasm ;	Circular, double-stranded DNA present in mitochondria / chloroplasts ;
12	Absence of telomeres in DNA ;	Presence of telomeres in DNA ;
13	Cell divide by binary fission ;	Cell divide via mitosis ;
14	Cells are smaller (about one-tenth the size of eukaryotic cell) [and measure about 0.5 – 5.0 μm in diameter] ;	Cells are larger ;

Methods by which these differences can be shown :

15 View the cells under transmission electron microscopy which allows internal structures of the cell to be seen ;

16 Gel electrophoresis allow DNA sizes and quantity (estimates) to be distinguished ;

17 Southern blot allows specific sequence such as telomeres to be picked up in eukaryotic cells but not in prokaryotic cells ;

Examiner's report:

Candidates were able to provide comprehensive descriptions of differences between typical prokaryotic and eukaryotic cells. A number of candidates were able to describe higher level differences related to features such as the degree of condensation of DNA, the structure of promoter sequences and the number of origins of replication per molecule of DNA.

Many candidates considered the use of microscopes to show these differences. Fewer candidates considered the application of techniques other than microscopy to reveal differences between prokaryotic and eukaryotic cells.

(b) Explain the expected problems of trying to classify the prokaryotes present in the human prokaryotic microbiome community and describe advantages of using molecular methods for this process. [10]

1 Classification is the organization of species according to particular characteristics based on **physical and assumed similarities.** e.g. distribution, morphology & molecular similarities, for meaningful grouping of organisms together ;

Distribution

2 Prokaryotes are present in all parts of the human body, such as on the skin, in the alimentary tract, nose etc. There may be locations of prokaryotes on the human body that cannot be reached easily to isolate the microbes. Thus in the process of isolating these prokaryotes, many prokaryotes may not be picked up and this will result in incomplete data collection, which will lead to under-representation of types of prokaryotes in the human prokaryotic microbiome community (Community) ;

3 As they are microscopic and not visible to the naked eyes, prokaryotes in the Community can be classified using microscopy to look at morphology of cells, biochemical tests to identify prokaryotes based on their differences in the biochemical activities of different bacteria and culturing technique based on their growth pattern or substrate utilization on growth media ;

Morphology

4 Morphological classification is subjective as researchers may not agree on which structural feature distinguishing the prokaryotes. Furthermore, different species of bacteria often have similar morphologies such as shape, size. Thus light microscopy alone cannot be used to identify different type of bacteria. Light microscopy usually requires the use of appropriate stains to distinguish morphological differences between species, for example, use of gram staining to pick up gram-positive or gram-negative bacteria ;

[For general info only: Gram positive bacteria stain violet due to the presence of a thick layer of peptidoglycan in their cell walls, which retains the crystal violet these cells are stained with. While Gram negative bacteria stain red, which is attributed to a thinner peptidoglycan wall, which does not retain the crystal violet during the decoloring process.]

Biochemical method

5 Biochemical tests identify prokaryotes based on the products of metabolic reactions produced or the type of compounds the organism can utilize for growth. Different species of prokaryotes can give the same results as they are able to utilize various substrates for growth or produce multiple metabolite products. Hence there may be ambiguous results for such test. Furthermore, many biochemical tests often need to be performed in order to positively identify a prokaryote.

Culturing method

6 Culturing prokaryotes on different types of nutrient media can reveal the different types of prokaryotes present in a sample. However, it is time-consuming as some slow-growing prokaryotes will take weeks to grow and sufficient number in term of inoculation size on the nutrient media before the prokaryotes will grow. Hence, such prokaryotes may be missed and incomplete data collection will result. Culturing of a large number of sample will require much space and expensive equipment such as incubators, thus making implementation difficult for large number of sample ;

Advantages of using molecular methods to classify prokaryotes in the human prokaryotic microbiome community

7 Quantifiable and open to statistical analysis

Molecular data such as nucleotide and amino acid sequences are quantifiable, in abundance and open to statistical analysis.

8 Unambiguous and objective

Molecular data can be easily described in an unambiguous manner. Protein and nucleic acid sequence data are precise and accurate. This facilitates the objective assessment of evolutionary relationships.

9 Not affected by convergent evolution

Molecular data provides a clear model of evolution by comparing the nucleotide and amino acid sequence as the rate of molecular change in genes and proteins is regular like a molecular clock.

10 Based strictly on heritable material

Molecular data is based strictly on heritable material.

Morphological data is based on anatomical characters which may be influenced by environmental factors as well as variation due to genotype of the organism.

11 Greater number of organisms can be compared

All organisms can be compared with the use of some molecular data. All living organisms have nucleotides and amino acids, so molecular data can be collected from any organisms.

Examiner's report:
Candidates applied their knowledge and understanding to consider a range of difficulties in classifying prokaryotes, including the extent to which the biological species concept can be applied to prokaryotes and difficulties in recognising morphological differences. Fewer candidates attempted to identify problems that could be specific to the microbiome community of humans.

Most candidates were able to explain a range of advantages of using molecular methods to classify prokaryotes.

The responses of some candidates were expressed only in vague or general terms that did not always match to the context of the question.

PAPER 4

2017 P4 Planning Mark Scheme

A student repeated this experiment with a nutrient medium containing 10% glucose and 10% melibiose and found that no α-galactosidase was produced.

The student hypothesised that α-galactosidase would not be produced in the presence of 10% melibiose, if concentration of glucose was higher than a specific concentration.

Design an experiment to estimate the lowest concentration of glucose needed to prevent α-galactosidase synthesis by yeast in a medium containing 10% melibiose.

You do not need to include details of how to test samples with X- α-gal or how the yeast cells are removed from the sample before testing.

In your plan, you must use:

- A growing culture of yeast in a water-bath at 30ºC ($0.5cm^3$ of this culture will be required for inoculation of each additional culture)
- A sterile solution of 20% glucose in nutrient medium
- A sterile solution of 20% melibiose in nutrient medium
- A sterile solution of nutrient medium
- A water-bath at 30ºC

You may select from the following sterilised apparatus and plan to use appropriate additional apparatus:

- Normal laboratory glassware, e.g. test-tubes, boiling tubes, beakers, measuring cylinders, graduated pipettes and pipette fillers, glass rods, etc.
- Syringes
- Timer, e.g. stopwatch
- Sterile cotton wool plugs

Mark Scheme

Introduction

- **Background knowledge:**
  - Enzyme α-galactosidase is produced in yeast when grown in a nutrient medium containing melibiose.
  - However, there would be no production of the enzyme if glucose is also present in the medium at a concentration equal to or higher than a particular minimum.
  - The enzyme hydrolyses melibiose into glucose and galactose.
  - The enzyme also hydrolyses a compound structurally similar to melibiose, called X-α-gal, which is colourless, into a blue product. The intensity of the blue colour is proportional to the enzyme concentration.
- **Rationale of setup:**
  - The presence of this blue product is hence indicative of the presence and production of the enzyme by yeast.
  - Glucose solution of known concentrations (less than 10%) in the presence of 10% melibiose is used as the nutrient medium for the growth of yeast.
  - The lowest concentration of glucose that results in no production of the blue product and hence, a colourless solution would be observed (after removal of yeast cells and testing using X-α-gal), would be an estimate of the lowest concentration of glucose needed to prevent α-galactosidase synthesis by yeast in a medium containing 10% melibiose.
  - A spectrophotometer could be used for more accurate quantification of the blue colour intensity. The results that give a colourless solution would have the lowest absorbance and would be used as a reference to determine the lowest concentration of glucose that provides a colourless solution result.

Variables

1. **Independent variable**: concentration of glucose / % (0%, 1%, 2%, 3%, 4%, 5%, 6%, 7%, 8%, 9%, 10%) **[at least 5 values; regular intervals; Correct unit]**
2. **Dependent variable**: Colour intensity of X-α-gal and sample media / A.U., measured by the absorbance using a spectrophotometer
3. Other variables to be **kept constant**: **[Apparatus & quantity** to be indicated**]**
    - Fixed **concentration of yeast**
        - **0.5cm^3** is taken from the growing culture of yeast for each concentration of glucose tested.
        - The growing culture of yeast is stirred and mixed thoroughly before drawing the 0.5cm^3 using a **syringe**.
        - Final volume of each sample (glucose + melibiose + nutrient medium + yeast) is fixed at 5.5 cm^3
    - Fixed **concentration of melibiose**
        - Concentration is fixed at 10% by adhering to the dilution table shown in Step 1 of procedures. Different clean **syringes** are used to draw the various volumes of solutions.
    - The **temperature** (**30.0°C**) for incubation of the yeast is maintained throughout by a **water bath**.
    - The **duration** for incubation of the yeast is fixed at **48 hours**, which is monitored using a **stopwatch**.

Controls (control 1 is more important than control 2)

Control 1:

✓ **Purpose of control:** The control is set up to show that no α-galactosidase is produced in the presence of glucose at a high enough concentration even though 10% melibiose is also present. This could also be used as the colourless reference to determine the lowest concentration of glucose that gives the same colourless results.

✓ **Control set-up: Steps are performed** with **all other experimental conditions remaining the same** except that **glucose is present at 10% concentration (achieved by adding 10.0cm^3 of 20% glucose as in Step 1 of procedures)**.

✓ **Expected results:** Colourless mixture expected after removal of yeast and testing using X-α-gal.

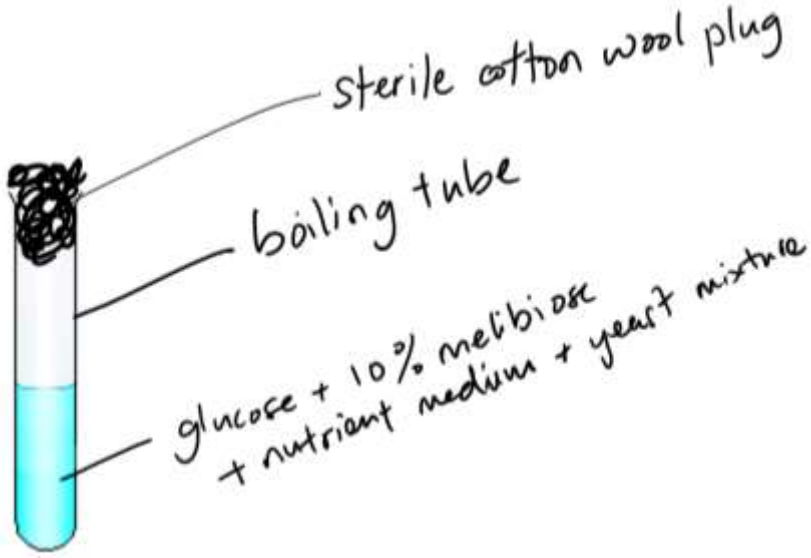
Control 2:

✓ **Purpose of control:** The control is set up to show that α-galactosidase is produced in the absence of glucose and presence of 10% melibiose. This could also be used as the reference for the most intense blue colour observed.

✓ **Control set-up: Steps are performed** with **all other experimental conditions remaining the same** except that **glucose is replaced with 10.0cm^3 of nutrient medium as in Step 1 of procedures**.

✓ **Expected results:** Most intense blue colour mixture expected after removal of yeast and testing using X-α-gal.

Labelled Diagram



Procedure [Apparatus and quantity stated]

1. Using **simple dilution**, obtain the various glucose concentrations in 10% melibiose as below:

Concentration of glucose / %	Volume of 20% glucose to be added / cm^3	Volume of 20% melibiose to be added / cm^3	Volume of nutrient medium to be added / cm^3	Total volume / cm^3
0	0.0	10.0	10.0	20.0
1	1.0	10.0	9.0	20.0
2	2.0	10.0	8.0	20.0
3	3.0	10.0	7.0	20.0
4	4.0	10.0	6.0	20.0
5	5.0	10.0	5.0	20.0
6	6.0	10.0	4.0	20.0
7	7.0	10.0	3.0	20.0
8	8.0	10.0	2.0	20.0
9	9.0	10.0	1.0	20.0
10	10.0	10.0	0.0	20.0

2. Use **syringe** to add **5.0cm^3** of 10% glucose (with 10% melibiose) into a boiling tube.
3. Stir and mix the growing culture of yeast thoroughly using a glass rod before using a syringe to draw **0.5cm^3** of the culture.
4. Add the **0.5cm^3** yeast culture to the **5.0cm^3** 10% glucose (with 10% melibiose) solution and mix using a glass rod.
5. Place a **sterile cotton wool plug** into the mouth of the boiling tube. This is to **minimise contamination** while still **allowing gaseous exchange** for yeast growth.
6. Place the boiling tube into a **water bath at 30.0°C** and start the **stopwatch**. Incubate for 48 hours.

7. After 48 hours, remove the boiling from the water bath.
8. Remove and discard the yeast cells in the boiling tube.
9. Test the sample with X-α-gal.
10. Transfer the sample + X-α-gal mixture into a cuvette and measure the absorbance using a spectrophotometer.
11. To ensure **reliability of results**, **perform 2 more replicates** by repeating steps 2 – 10, using fresh 10% glucose (with 10% melibiose) prepared in step 1 and fresh **0.5cm^3** yeast from the same culture in step 3.
12. **Repeat** steps 2 – 11 for **0, 1, 2, 3, 4, 5, 6, 7, 8 and 9% glucose solutions** (with 10% melibiose).
13. To ensure **reproducibility of data**, **repeat the entire experiment** (steps 1 – 12) **twice using freshly prepared glucose + melibiose + nutrient medium mixture and fresh growing yeast culture**.

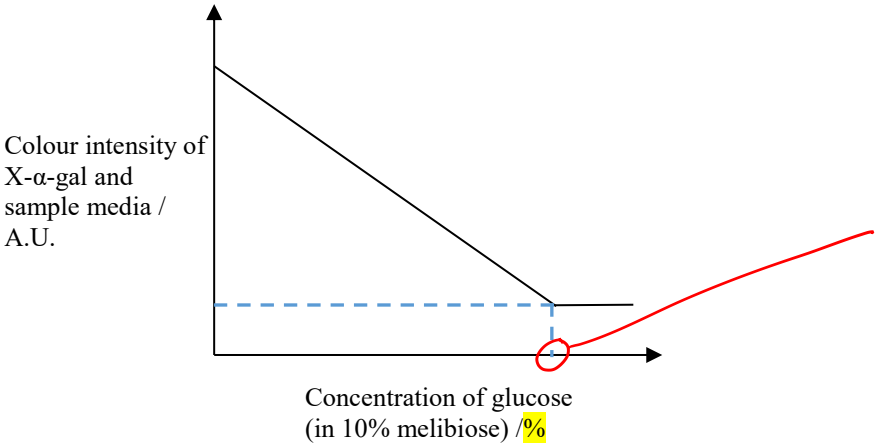
Table and Data

Table showing the colour intensity of X-α-gal and sample media / A.U. with different concentrations of glucose (in 10% melibiose)

Concentration of glucose (in 10% melibiose) / %	Colour intensity of X-α-gal and sample media / A.U.			
	Replicate 1	Replicate 2	Replicate 3	Average
0				
1				
2				
3				
4				
5				
6				
7				
8				
9				
10				

Graph

Graph of colour intensity of X-α-gal and sample media against concentration of glucose (in 10% melibiose) /%



lowest concentration of glucose needed to prevent α-galactosidase synthesis by yeast

Risks & Precautions

Risk	Precaution
X-α-gal solution is flammable	Ensure that there is no naked/open flame nearby.
X-α-gal solution is harmful	Wear safety goggles and gloves when handling X-α-gal solution. Wash off any splashes on skin immediately.