NANYANG JUNIOR COLLEGE
JC 2 Preliminary Examination
Higher 2

CANDIDATE NAME **ANSWERS**

CLASS

BIOLOGY

9744/02

Paper 2 Structured Questions

14 September 2023

Candidates answer on the Question Paper.

No Additional Materials are required.

2 hours

READ THESE INSTRUCTIONS FIRST

Write your name and CT on all the work you hand in.
Write in dark blue or black pen.
You may use an HB pencil for any diagrams or graphs.
Do not use staples, paper clips, highlighters, glue or correction fluid.
DO **NOT** WRITE IN ANY BARCODES.

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

The use of an approved scientific calculator is expected, where appropriate.
You may lose marks if you do not show your working or if you do no use appropriate units.

At the end of the examination, fasten all your work securely together.

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

For Examiner's Use	
1	
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8	
9	
10	
Total	

This document consists of **24** printed pages.

[Turn over

Answer **all** the questions.

1 Polysaccharides, such as glycogen, are composed of thousands of monomers.

Oligosaccharides are carbohydrates that contain three to ten monomers in their chain.

Nystose is one example of an oligosaccharide. The structure of nystose is shown in Fig. 1.1.

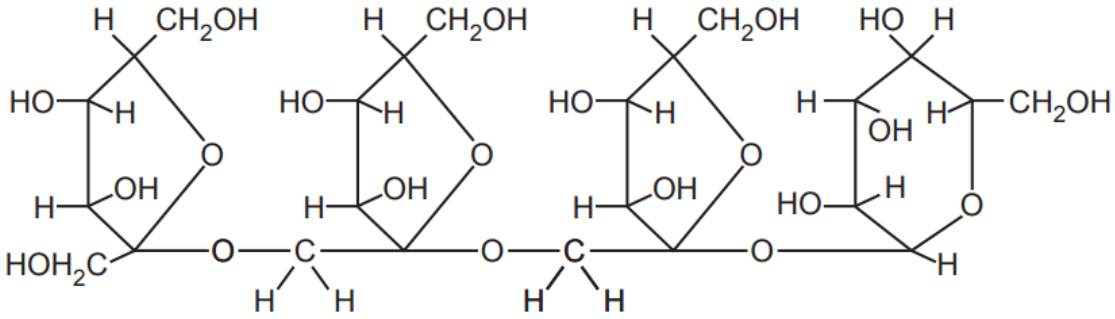

Fig 1.1

(a) State three differences between the structures of nystose and glycogen, **other** than the number of monomers in the molecules.

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[3]

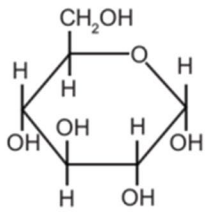
Question	Answer	Marks
6(a)	*any **three** from*: nystose is not branched / glycogen is branched ; different glycosidic bonds, qualified ; e.g. nystose has one type of glycosidic bond and glycogen has two types 1-4 and 1-6 (glycosidic) bonds in glycogen (v 1-2 glycosidic bonds in nystose) two types of monomer / AW, in nystose v only glucose in glycogen ; nystose contains one glucose (residue / monomer) ; *ref. to* ring shape, glycogen monomers have six-sided rings and nystose has (a) six-sided ring and five-sided rings ; AVP ; e.g. fructose in nystose and no fructose in glycogen **I** numbers of C,H and O	3

9700_s16_qp_22 Q: 6

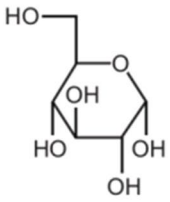
(b) One of the enzymes involved in glycogen synthesis is glycogen synthase. The monomer of the glycogen polymer is α-glucose.

(i) Draw the ring form of α-glucose in the space provided.

(i)



1



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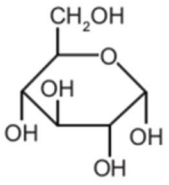


3

two marks for correct drawing of ring structure ;;
*all atoms shown **or** one of diagrams 1–3 above*

one mark if, inconsistent / incomplete, drawing:
diagram 1 – <u>one</u> missing H from any of carbons 2–6 (OH groups and rest of drawing must be correct)
diagrams 2 and 3 – adding the H to <u>one</u> of carbons 1–5 (OH groups and rest of drawing must be correct)

[2]

(ii) The gene coding for glycogen synthase is known as *GYS1*. Glycogen synthase catalyses the formation of a covalent bond between two α-glucose molecules during glycogen synthesis.

Name the type of bond formed.

glycosidic

[1]

(iii) Glycogen branching enzyme is another enzyme that is required for glycogen synthesis.

Suggest why glycogen branching enzyme is needed in addition to glycogen synthase.

To form/has (glycosidic α) 1-6 bonds/links (to make branches)

[1]

(c) Table 1.1 shows three functions of cell structures that are involved in the synthesis of glycogen synthase.

Complete Table 1.1 by naming the cell structure that carries out the function listed.

function	name of cell structure
assembles ribosomes for polypeptide synthesis	nucleolus
synthesizes ATP to provide a supply of energy for the transcription of *GYS1*	mitochondrion
folds and modifies synthesized polypeptide to produce functioning glycogen synthase	Rough endoplasmic reticulum or Golgi body/apparatus/complex

[3]

[Total: 10]

2 There are a number of mutations affecting the production of fetal haemoglobin, HbF, and normal adult haemoglobin, HbA.

- The Hb^A allele codes for the normal β-globin polypeptide of haemoglobin.
- The Hb^S allele, caused by a base substitution mutation, codes for an abnormal β-globin polypeptide.

The abnormal haemoglobin molecules (HbS) form fibres in low partial pressures of oxygen (pO_2). The fibres cause red blood cells to become sickle shaped and the cells can block blood capillaries.

Individuals with adult haemoglobin molecules that are all abnormal (HbS) have sickle cell anaemia. This is a painful chronic condition that can be life-threatening.

(a) Explain why this mutation causes the HbS to form fibres.

change in amino acid from hydrophilic glutamate rej glutamine to hydrophobic valine

HbS molecules stick together /polymerise / hydrophobic interactions formed between HbS @ mutated haemoglobin

Rej valine / RBC / polypeptide chains alone polymerise

[2]

(b) Fetal haemoglobin, HbF, is produced by the fetus until just before birth, when adult haemoglobin begins to be made.

By the age of six months, adult haemoglobin has replaced most of the HbF. This change occurs when the genes coding for HbF are switched off and the genes coding for adult haemoglobin are switched on.

- A base substitution, British-198, causes fetal haemoglobin to continue to be produced.
- Normally by the age of six months, the concentration of HbF reduces to less than 1% of total haemoglobin.
- With the British-198 mutation, the concentration of HbF may be as high as 20% of total haemoglobin in an adult.
- HbF has a higher affinity for oxygen at low pO_2 than adult haemoglobin. Individuals who have both sickle cell anaemia and British-198 mutation have reduced symptoms of sickle cell anaemia.

Suggest why having the British-198 mutation reduces the symptoms of sickle cell anaemia.

any two from:

1 idea of both HbF and HbS rej HbA present (in the same cell) ;

2 reduces / stops fibre formation / polymerization between HbS ;

3 fewer red blood cells rej Hb, change shape / sickle / block capillaries ;

[2]

(c) Gel electrophoresis can be carried out to test individuals for the different versions of haemoglobin: HbA, HbS and HbF.

- A buffer with alkaline pH is used to make all haemoglobin molecules negatively charged.
- HbS molecules have an additional positive charge compared to HbA.

(i) Describe and explain how gel electrophoresis is used to diagnose sickle cell anaemia.

any four from:

1 current / potential difference / electric field (across gel) ;

2 (protein / Hb) moves / attracted, to, anode / positive electrode ;

3 HbS (more positive so) moves, more slowly

4 HbS moves, shorter distance / less (far) from negative end ;

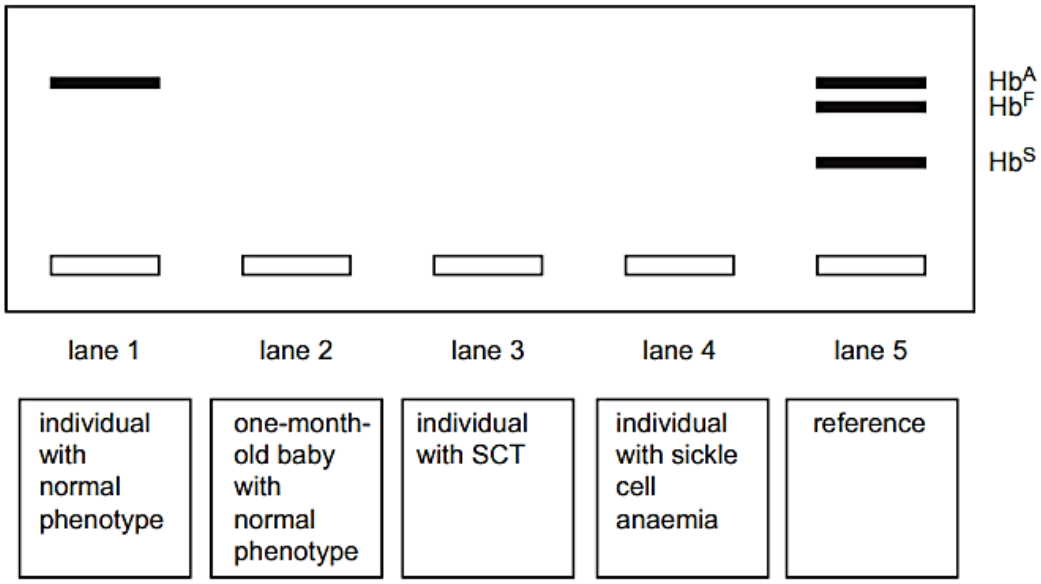
5 compare band positions to, known haemoglobins / reference bands ;

6 if single band seen at HbS position person has sickle cell anaemia ;

[4]

(ii) Four individuals had their haemoglobin analysed by gel electrophoresis. One of the individuals was heterozygous for the Hb^A and Hb^S alleles and had a condition known as sickle cell trait (SCT).
Some of the results are shown in Fig. 2.1. In Fig. 2.1, lane 1 and lane 5 are complete.

Fig. 2.1



Predict the results for the individuals analysed, by adding bands to lanes 2, 3 and 4 on Fig. 4.1. [2]

lanes 2, 3 and 4 correct = 2 marks ;;

two correct = 1 mark

one or none correct = 0 marks

[Total: 10]

3 **(a)** The house mouse, *Mus musculus*, has a diploid number of 40 chromosomes. Fig. 3.1 shows 6 of these chromosomes.

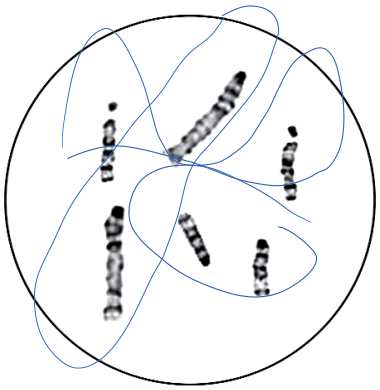


Fig. 3.1

Identify **one** pair of homologous chromosomes on Fig. 3.1 by drawing circles around two chromosomes. [1]

two homologous chromosomes circled ;

(b) Fig. 3.2 shows the banding pattern of chromosome pair 11 of *M. musculus*. The banding pattern is obtained by staining.

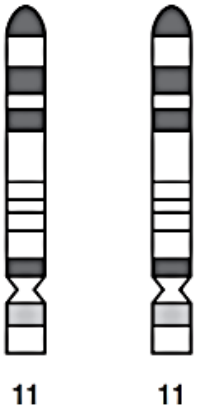


Fig. 3.2

(i) Explain why chromosomes, such as those in Fig. 3.2, are described as a homologous pair.

1 same, length ; reject similar

2 (same banding pattern which) shows same genes / gene loci ; reject if same alleles mentioned

3 same centromere position

[3]

(ii) State the number of chromosomes that are present in *M. musculus* spermatozoa.

20;

Note: if qn asks for chromosome number, it will have to be presented as **n = 20**

[1]

(c) *M. musculus* produces gametes by meiosis. These gametes are genetically different.

There is random fusion of gametes at fertilisation.

(i) Explain why meiosis is important in the life cycle of *M. musculus*, **apart from** producing genetically different gametes.

1 *ref. to* reduction division / halves the chromosome number / formation of haploid gametes

2 to give diploid zygote during fertilization / restore diploid condition in zygote

3 prevents chromosome number doubling / maintain diploid chromosome number with each successive generation

[2]

(ii) Explain how the random fusion of gametes leads to the expression of rare, recessive alleles.

1 (by chance) both gametes (may) have recessive allele **;**

2 zygote / offspring, has, pair / two / homozygous, recessive alleles **;**

[2]

[Total: 9]

4 (a) Cats with either black fur or white fur are common in Europe whereas cats with brown fur are less common.

A gene, coding for an enzyme involved in pigment production, has two alleles.

- The dominant allele, **B**, results in black fur
- The recessive allele, **b**, results in brown fur

A second gene can affect fur colour.

- The dominant allele, **A**, prevents pigment production, resulting in a cat with white fur.
- The recessive allele, **a**, has no effect on fur colour.

The two genes are on different pairs of autosomes.

(i) Use a genetic diagram to show how a cross between two cats, heterozygous at both loci, can produce offspring with three different colours: white, black and brown.

State the expected ratio of the different coloured offspring. [4/5]

PUBLISHED

Question	Answer	Marks
7(a)	*parental genotypes* **AaBb** x **AaBb** ; *gametes* **AB Ab aB ab x AB Ab aB ab** ; *offspring* **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** black; **aaBb** black **ab**: **AaBb** white; **Aabb** white; **aaBb** black; **aabb** brown ;; *max 2 for all offspring correct* *max 1 if one error* *max 0 if more than one error* offspring phenotype correctly linked to genotype ; *ratio* 12 white : 3 black : 1 brown ;	**6**

[5]

(ii) Suggest how the presence of allele **A** prevents pigment production.

Example of gene interaction / epistasis;
Ref to blocking (one step) pathway to pigment production;
(allele A) product / protein inhibits enzyme (producing pigment);
(allele A) product / protein is a repressor; **A** allele codes for repressor
(which) blocks transcription / RNA polymerase cannot bind / switches off allele (coding for pigment);
(by) binding to / blocking promoter;
(allele A) product / protein, prevents transcription factor complex formation / AW;

[3]

(b) Apart from having different fur colour, a variety of domestic cat does not have a tail. This condition is controlled by a single gene with two alleles. These alleles are

- 'with tail'
- 'without tail'

Table 4.1 shows the results of four crosses between cats with tails and cats without tails. Each male was crossed with several females.

parental phenotype			offspring phenotype			
			male		female	
cross	**male**	**females**	**with tail**	**without tail**	**with tail**	**without tail**
1	without tail	without tail	21	32	19	40
2	with tail	with tail	65	0	70	0
3	with tail	without tail	40	25	25	36
4	without tail	with tail	35	27	38	36

Table 4.1

(i) Explain how the results of **crosses 1 and 2** show that the allele 'without tail' is dominant.

cross 1. offspring of cats 'without tails' (dominant) crossed together have offspring with tails' (recessive) ;

cross 2. offspring of cats 'with tails' (recessive) crossed together are always 'with tails' (recessive) ;

[3]

(ii) Explain how the results of **crosses 3 and 4** show that the gene for this condition is not sex linked.

idea of 'with tails' (recessive) and 'without tail' (dominant) phenotypes occur in (approximately) equal numbers in each sex / 1:1 ratio in each sex / 1:1:1:1 / AW ;

cross 4: male 'without tail' (dominant) and female 'with tail' (recessive). If it were sex linked all males would be tailed. (This is not so), so not sex linked / AW ;

[3]

[Total: 11]

5 Different signalling pathways generate calcium (Ca^{2+}) signals which regulate many cellular functions such as smooth muscle contraction.

Fig. 5.1 shows the inositol trisphosphate/calcium (IP_3/Ca^{2+}) signalling pathway.

Phosphatidyl inositol-bisphosphate (PIP_2) is hydrolysed into IP_3 and diacylglycerol (DAG) by phospholipase C.

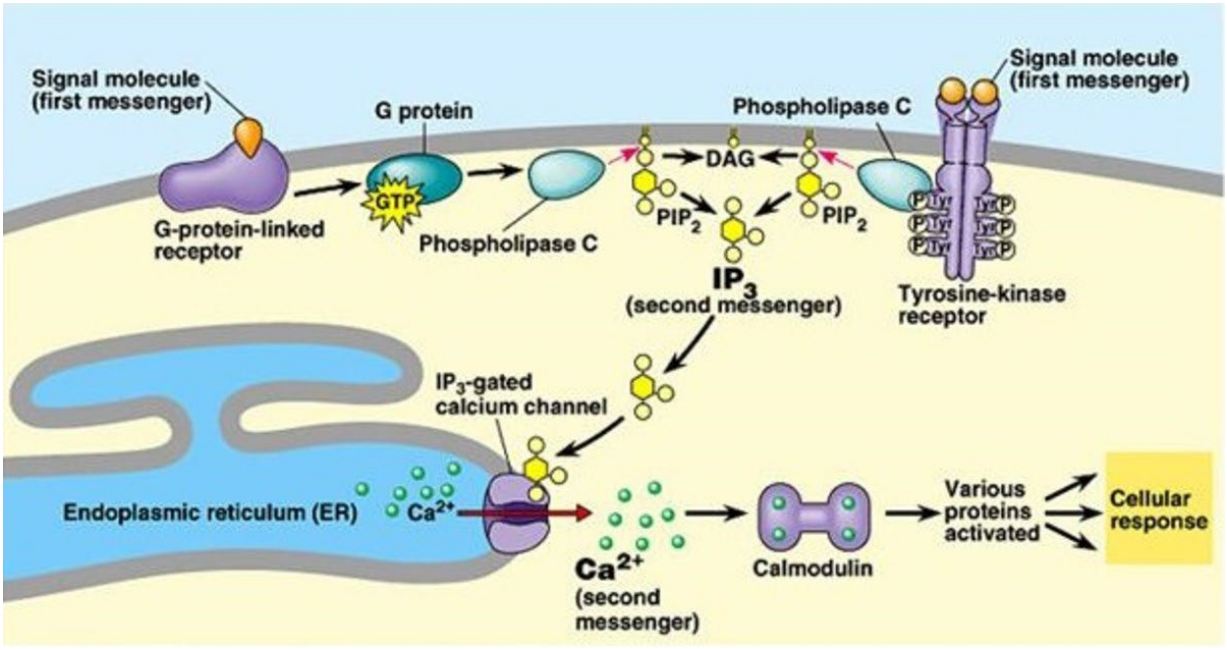


Fig 5.1

(a) With reference to Fig. 5.1,

(i) describe the structural differences of the two receptors.

1. G-protein linked receptor (GPLR) is **a protein monomer** (**one protein molecule**) (R: not dimer) while tyrosine-kinase receptor (TKR) is **a dimer with two protein subunits**;
2. GPLR has **one ligand-binding site** while TKR has **two ligand-binding sites;**
3. GPLR and TKR have **different ligand-binding sites complementary in conformation** (and charge) to **different ligands** (A: different conformation of ligand-binding sites);
4. GPLR and TKR have **different intracellular binding sites complementary in conformation** (and charge) to **G protein** and **phospholipase C** respectively;

(R: *yes/no comparison*, e.g. GPLR does not have phosphorylated tyrosine residues while TKR has phosphorylated tyrosine residues

.....

[2]

(ii) define second messengers and explain their role in signal transduction.

define: **Small**, **non-protein**, **water-soluble molecules/ions** that relay signal in signal transduction pathway

role: **Bind, induce change in conformation** and **activate relay protein** (e.g. **IP_3 binds and induces change in conformation** in **IP_3-gated calcium channel**, **opening the IP_3-gated calcium channel**

OR **Ca^{2+} binds and induces change in conformation** in **calmodulin**, **activating the calmodulin**)

[2]

(iii) outline two stages where signal amplification may occur.

1. **Each tyrosine kinase receptor** activating **many phospholipase C**, where each binds to a phosphorylated tyrosine residue (A: **each G-protein linked receptor** activating **many G protein**)
2. **Each phospholipase C** producing **many IP_3**
3. **Each IP_3** opening a **IP_3-gated calcium channel** and releasing **many Ca^{2+}**

[2]

(iv) Dysregulation of the IP_3/Ca^{2+} signalling pathway can lead to many different possible human diseases. Hypertension is caused by increased smooth muscle contraction due to enhanced IP_3/Ca^{2+} signalling.

Suggest the type of mutation in phospholipase C that could lead to hypertension.

1. **Gain-in-function mutation** in **gene coding for phospholipase C**, causing **phospholipase C** to be **constitutively active**
2. Resulting in **increased IP_3 produced**, thus **increased concentration of Ca^{2+} in cytosol**, leading to **increased smooth muscle contraction (hypertension)**

(*↑ IP_3/Ca^{2+} signal transduction → ↑ cellular response*)

[2]

(b) Calcium binding by calmodulin exhibits considerable cooperativity, making calmodulin an unusual example of a monomeric (single-chain) cooperative-binding protein with four calcium-binding sites.

Fig. 5.2 shows calmodulin without calcium (left), and calmodulin with calcium (right). Sites that bind target proteins are indicated by the stars (*).

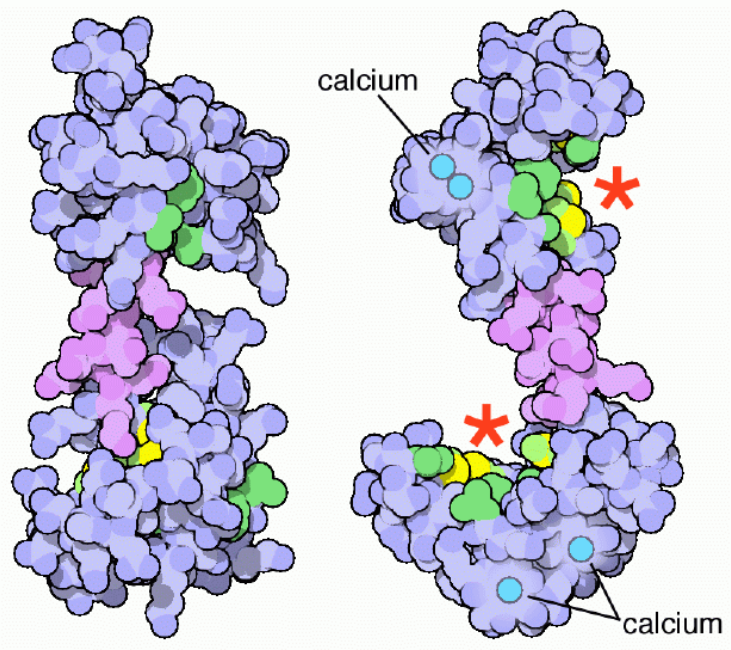


Fig. 5.2

(i) With reference to Fig. 5.2, suggest how cooperative binding of Ca^{2+} ions to calmodulin is necessary for calmodulin activity.

1. [Cooperative binding of Ca^{2+}] Binding of **one <u>Ca^{2+}</u> ion** to **one <u>calcium-binding site</u>** of calmodulin induces **conformational change** in the **<u>other</u> three <u>calcium-binding sites</u>**, increasing **the affinity of calcium-binding sites for Ca^{2+}**; (R: subunits)
2. [Necessary for calmodulin activity] This causes **<u>change in 3D conformation</u>** of calmodulin, **exposing** the **two <u>binding sites for target protein</u>** for binding of target proteins and calmodulin activity; (R: active sites)

[2]

[Total: 10]

6 The diagram below shows the structure of Human Immunodeficiency Virus (HIV).

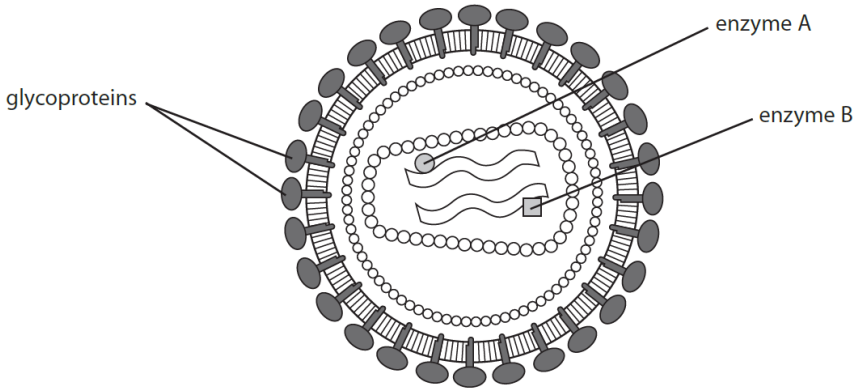


Fig. 6.1

(a) State how the genetic material in HIV differs from the genetic material in the bacterium *Mycobacterium tuberculosis* that causes TB.

Question Number	Answer	Mark
2(a)	1. RNA in HIV and DNA in {bacterium / eq} ; 2. comparative description of nucleic acid e.g. circular in bacterium and linear in HIV / eq ; 3. plasmids in {bacterium / eq} and no plasmids in HIV ;	**maximum (2)**

[2]

(b) Some anti-viral drugs prevent HIB entering the host cells.

Suggest how these anti-viral drugs could prevent HIV entering the host cells.

Question Number	Answer	Additional Guidance	Mark
3(b)	1. idea that the drugs could {bind to / alter shape of} {glycoproteins / gp120} ; 2. idea that drugs bind to {receptors / antigens} on membrane / eq ; 3. called CD4 (antigen / molecules) ; 4. preventing virus attaching to T (helper / $CD4^+$) cells / eq ;		**(3) XP**

[3]

(c) Describe how the enzymes shown in the diagram are involved in HIV infection.

Question Number	Answer	Additional Guidance	Mark
*3(c)	1. reference to reverse transcriptase ; 2. idea of formation of (viral) DNA ; 3. from (viral) RNA ; 4. reference to integrase ; 5. idea of integration of (viral) DNA into (host) DNA ; 6. idea that {T helper cells / eq} would be {destroyed / killed / burst / eq} (by virus particles leaving cell) ; 7. idea that more T (helper) cells would become infected ;	**QWC focussing on clarity of expression** 2. reject idea that RNA is {turned into / converted into} DNA 5. ACCEPT idea of {latency / formation of provirus / eq}	**(5) XP**

[5]
[10 marks]

7 The results of investigations carried out on mitochondria show how the structure of a mitochondrion is related to its role in aerobic respiration. **(9700 43 Oct/Nov 2022)**

- Intact mitochondria (not damaged) were removed from cells.
- A technique was used to remove the outer mitochondrial membrane, leaving the inner membrane intact.
- The inner mitochondrial membrane was separated from the contents of the matrix so that both could be analysed.

(a)

(i) The removal of the outer membranes of mitochondria involces placing the organelles in pure water. This results in the rupture (bursting) of the outer membrane. The inner mitochondrial membrane does not rupture and remains intact.

Suggest **and** explain why the inner membrane of a mitochondrion remains intact when the organelle is placed in pure water.

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[2]

(ii) Name **three** molecules, other than coenzymes, that are found in the mitochondrial matrix **and** explain their role in aerobic respiration.

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[3]

(iii) The inner mebrane contains a very high proportion of the molecule cardiolipin. Cardiolipin makes the membrane impermeable to some ions.

Suggest why the inner membrane contains a very high proportion of cardiolipin.

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[1]

Question	Answer	Marks
3(a)(i)	*any **pair** (max 2) from:* 1 cristae / folds, let inner membrane expand ; 2 when water enters (matrix), by osmosis / down water potential gradient ; 3 inner membrane (relatively) impermeable to water ; 4 (so) water does not enter (matrix), by osmosis / down water potential gradient ; 5 inner membrane moves (H^+) ions out of matrix ; 6 so less water enters (matrix), by osmosis / down water potential gradient ;	2
3(a)(ii)	*any **three** names plus explanations from:* 1 pyruvate, for link reaction / to bind to coenzyme A / to make acetyl (CoA) / to make reduced NAD / be dehydrogenated ; 2 oxaloacetate to, accept acetyl / make citrate ; 3 citrate to, make reduced NAD / be dehydrogenated ; 4 enzymes to catalyse, link reaction / Krebs cycle ; 5 oxygen to, accept electrons / accept protons / form water ; 6 water as a, solvent / medium for reactions ; 7 DNA / RNA, to make (named) respiratory, enzymes / proteins ;	3

(b) In further experiments it was found that, in an intact mitochondrion:

- There is a membrane potential across the inner mitochondrial membrane, with the matrix having a negative charge
- The transport of ATP, ADP and inorganic phosphate (P_i) is driven by the membrane potential across the inner membrane.

Fig. 7.1 shows the location of some inner membrane carrier proteins.

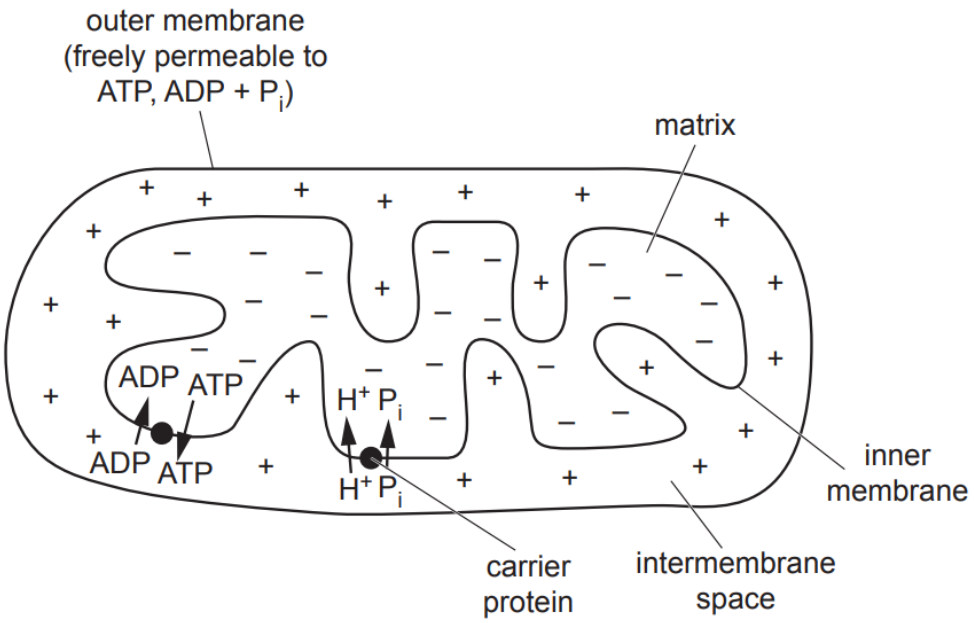

Fig. 7.1

(i) Suggest **and** explain how P_i is transported across the inner membrane of the mitochondrion into the matrix.

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[2]

(ii) Suggest the advantages of linking ATP transport to ADP transport across the inner membrane of the mitochondrion.

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[2]

[Total: 10]

Question	Answer	Marks
3(a)(iii)	*any **one** from:* 1 so H^+, cannot move through / must move through ATP synthase ; 2 to maintain proton gradient ;	1
3(b)(i)	*any **four** from:* 1 protons and 2 electro 3 (releas membrane space ; 4 H⁺ / positive brane space ; 5 (c / sets up) proton / electrochemical, ient ;	4
3(b)(ii)	*any **two** from:* 1 (P_i) by facilitated diffusion **or** through a protein, channel / carrier ; 2 P_i and H^+ move together ; 3 (as) H^+ ions diffuse (through ATP synth(et)ase / to matrix) ;	2
3(b)(iii)	1 constant / sufficient / correct, supply / amount of, ADP / reactant ; 2 (so) ATP can continue to be made / so enough ATP can be made ;	2

8 Arabinose is a monosaccharide containing five carbon atoms. The arabinose operon, also known as the *araBAD* operon, is an operon required for the breakdown of arabinose in the bacteria, *Escherichia coli*.

The *araBAD* operon contains three structural genes: *araB*, *araA*, *araD*, which code for three different enzymes that are required for the breakdown of arabinose. Other than the promoter of the structural genes, there are other regulatory regions of the operon including the CAP binding site and the operator, as shown in Fig. 8.1.

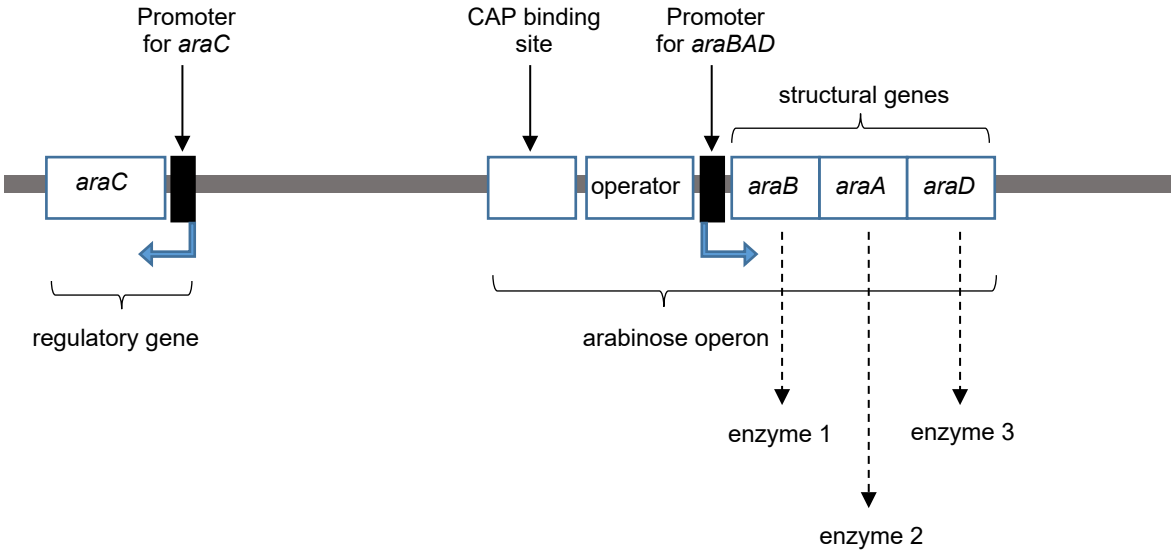

Fig. 8.1

When the cell is starved of glucose, the CAP-cAMP complex binds to the CAP binding site resulting in positive regulation of the *araBAD* operon.

The regulatory protein, araC protein, is coded for by the regulatory gene, *araC*. In the presence of arabinose, arabinose will bind to the araC protein forming the arabinose-araC protein complex. The arabinose-araC protein complex will then bind to the operator of *araBAD* operon, recruiting RNA polymerase to the *araBAD* promoter. When there is no arabinose, araC protein binds to the operator of *araBAD* operon in a manner that prevents RNA polymerase from binding to the *araBAD* promoter.

(a) With reference to Fig. 8.1,

(i) Explain the term 'operon.

1. *araBAD* operon contains a group of *ara* structural genes (A: *araB, araA* and *araD*) with related functions for the breakdown of arabinose;
2. group of genes under the control of the same promoter and operator;
3. to product a single polycistronic mRNA coding for enzymes 1, 2 and 3;

[2]

(ii) explain if the operon is an inducible or a repressible operon

1. inducible operon;
2. enzymes coded by the operon are involved in catabolic reaction/breakdown of arabinose;
3. operon is normally "off" and only switched "on" in the presence of arabinose;

[2]

(iii) describe the positive regulation of gene expression in the *araBAD* operon.

1. In the absence of glucose/when glucose levels are low, cAMP is high and binds to allosteric site of the CAP protein, activating CAP;
2. Active CAP/CAP-cAMP complex binds to CAP binding site, increasing the affinity of RNA polymerase for the araBAD promoter; (A: increase binding affinity of arabinose-araC complex to the operator);
3. In the presence of arabinose, this upregulates transcription/expression of *araB, araA,* and *araD*/structural genes;

[3]

(b) Fig. 8.2 shows the changes in the activity of enzymes in normal *E. coli* cells that synthesise tryptophan and break down arabinose after the addition of tryptophan and arabinose.

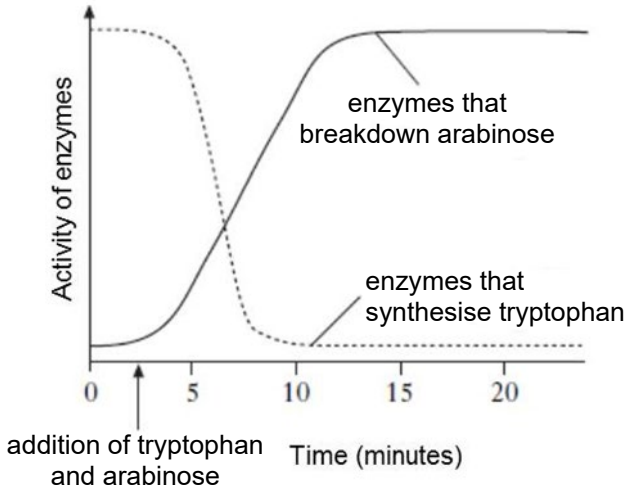

Fig. 8.2

In a scientific investigation, the *trp* operator sequence in the *E. coli* was altered such that it was no longer complementary to the DNA-binding site on the trp repressor proteins. The *araC* coding sequence was also altered such that the araC protein was no longer complementary to arabinose.

Describe and explain the effects of the mutations on the activity of enzymes that breakdown arabinose and enzymes that synthesise tryptophan in the altered *E. coli* upon the addition of

(i) arabinose,

1. Activity of enzymes that breakdown arabinose is low;
2. Arabinose was not able to bind to araC protein / cannot form the arabinose-araC protein complex

thus could not recruit RNA polymerase for the transcription/expression of *araB, araA,* and *araD*/structural genes (R: upregulate)

[2]

(ii) tryptophan,

1. Activity of enzymes that synthesise tryptophan is high;
2. *Trp* repressor cannot bind to operator even in the presence of tryptophan;
3. RNA polymerase is able to bind to promoter hence transcription/expression of *trp A-E* genes/structural genes occurs all the time;

[2]

[Total: 11]

9 The evolutionary origin of the four-legged amphibians (such as frogs and toads) from fish has been the subject of much debate for many years.

Among living fish, the rarely-caught coelacanth and the lungfish are thought to be most closely related to these amphibians.

Samples of blood were taken from two coelacanths that were captured recently near Comoros.

The amino acid sequences of the α and β chains of coelacanth and lungfish haemoglobin were compared with the known sequences of amphibian adults and their aquatic larvae (tadpoles). Organisms with more matches in the amino acid sequence of a polypeptide chain share a more recent common ancestor than those with fewer matches.

The comparisons with three species of amphibians, *Xenopus laevis* (Xl), *X. tropicana* (Xt) and *Rana catesbeiana* (Rc) are shown in Table 9.1.

Table 9.1

		percentage of matches of amino acid sequence					
		species of amphibian adults			**species of amphibian larvae (tadpoles)**		
	fish species	*Xl*	*Xt*	*Rc*	*Xl*	*Xt*	*Rc*
α chains	coelacanth	42.0	47.5	no data	45.4	42.6	48.2
	lungfish	40.4	42.1	no data	40.7	39.0	37.9
β chains	coelacanth	42.1	43.2	40.7	52.1	52.1	58.2
	lungfish	44.1	45.9	41.4	47.3	45.9	48.6

(a) (i) Explain whether or not the information in Table 9.1 supports the suggestion that coelacanths and amphibians share a more recent common ancestor than do lungfish and amphibians.

Supports:

1. coelacanth α chain has higher percentage of matches with both adult and larval amphibians ;

2. coelacanth β chain has higher percentage of matches with larval amphibians ;

Does not support:

3. (but) lungfish β chain has higher percentage of matches with adult amphibian (than coelacanths) ;

4. figures to support mp1 or mp2 or mp3

[4]

(ii) Suggest why adults and tadpoles of the same species of amphibian have different amino acid sequences in their haemoglobin.

Any two:

1. larvae aquatic and adults (partly) terrestrial / AW ; @ idea of different habitat / environment, hence different selection pressure
2. different oxygen concentration available ;
3. need haemoglobins with different oxygen affinities ;

[2]

(b) Coelacanth haemoglobin has a very high affinity for oxygen, suggesting that coelacanths, which have been captured at depths of between 200 m and 400 m, live in water that has a low concentration of oxygen.

Explain how an environmental factor, such as the low concentration of oxygen in deep water, can act:

(i) as a stabilising force in natural selection

1. idea of, unchanging / constant, environment / selection pressure ;
2. extreme (phenotypes) selected against / intermediate phenotypes selected for ;

[2]

(ii) as an evolutionary force in natural selection.

1. ref. change in oxygen concentration ;
2. (low) oxygen concentration acts as selective agent ;
3. some individuals (in population) are better adapted ;
4. these are more likely to survive ; ora
5. directional selection ;
6. sketch graph ;
7. populations develop in different concentrations of oxygen ;
8. disruptive selection ;
9. sketch graph ;

allow either mp6 or mp9 but not both

[2]
[Total: 10]

10 Table 10.1 shows different stages in the life cycle of a female *Aedes aegypti* mosquito, which is responsible for the spread of dengue.

Table 10.1

Stage	Aquatic	Terrestrial	Able to transmit dengue virus
Eggs	✓		
Larva	✓		
Pupa	✓		
Adult		✓	✓ ;

(a) (i) Place a tick (✓) in appropriate boxes that applies to each stage. [2]

(ii) Despite the protection offered by the antibodies in the primary infection, the recurrent exposure to DENV, particularly of a different serotype, can result in the manifestation of severe dengue fever.

Explain why the infection by a different serotype can result in severe dengue fever.

a. due to antibody-dependent enhancement;

b. where the antibodies produced from the activation of memory B cells from the primary infection binds less effectively to the infective DENV virus during a subsequent infection / secondary response; (These antibodies from the primary response cannot neutralise the virus)

c. the antibody-bound DENV is then recognised by (circulating) monocytes / macrophages, resulting in entry of virus into the cell (monocytes/macrophages) via endocytosis

d. virus escapes the endocytic vesicle → replicate to large numbers → (viremia) → severe dengue fever;

@ description of original antigenic sin (ref to memory T cells)

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[3]

To teach using mark scheme:

- *H1 2022 A level FRQ: Following an infection with a dengue virus, outline the development of viral dengue disease in an individual human. (6m)*

- Patients with dengue may progress through three clinical phases known as febrile phase, the critical phase and the recovery phase

- Once infected with DENV, after an initial incubation period of typically 3–7 days, the infection manifests with a sudden onset of high fever, accompanied by high viraemia, which is known as the febrile phase.
- Some individuals proceed to the critical phase, which lasts for 24–48 hours and is associated with plasma leakage; whereas others directly proceed to the recovery phase without developing plasma leakage.
- Severe dengue is associated with a transient increase in vascular permeability due to endothelial dysfunction in the critical phase.
- Virus infects keratinocytes / skin cells and specialised dendritic cells in the skin called Langerhans cells
- Langerhans cells display dengue viral antigen on their surface and travel to lymph nodes while dengue virus is replicating in the cells
- In the lymph node, dengue virus is released and virus infects other cells such as monocytes and macrophages
- Infected cells travel through lymphatic system and circulatory system, spreading the virus as the virus infects more cells
- Spread and increase of dengue virus results in viremia where there is high level of dengue virus in the bloodstream
- Virus stimulates / activates innate / non-specific immune response / immune cells (e.g. dendritic cells) in the first four days → fever
- Cytokines and chemokines released by infected cells increased permeability of blood vessels
- Resulting in inflammation and also pain
- Pyrogen released by activated macrophages lead to rise in systemic body temperature, resulting in dengue fever.
- Dengue hemorrhagic fever results from fever and increased vascular permeability leading to damage of lymph and blood vessels
- Dengue shock syndrome is the severe form of dengue which can lead to failure of circulatory system and send body into bleeding and shock.

(b) Insects are the most dominant group of organisms on the planet in terms of species richness, abundance, and biomass. Global warming has a marked influence on the physiology of insects, including *A. aegypti*.

(i) Outline how increases in temperature as a result of global warming can impact insects such as *A. aegypti*.

Insects take on ambient temperatures;

Ref to higher reproduction / egg laying rate due to increased rainfall / humid conditions ;

Ref to poleward migration of insects beyond the tropics ;

[3]

(ii) The male *A. aegypti* mosquitoes feed on nectar instead of blood and is considered an insect pollinator.

Using this information and your answers in **(b)(i)**, explain how global warming may affect global food security.

1. Results in loss of biodiversity
2. Reduces crop yield
3. Disrupt food chain

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[2]

[Total: 10]