NANYANG JUNIOR COLLEGE
JC 2 PRELIMINARY EXAMINATIONS
Higher 2

CANDIDATE NAME

CLASS

BIOLOGY

9744/03

Paper 3 Long Structured and Free-response Questions

20 Sept 2023

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.

Section A

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

Section B

Answer any **one** question on the separate Answer 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	
Section A	
1	**29**
2	**12**
3	**9**
Section B	**(a) 13** **(b) 12**
Total	**75**

This document consists of **14** printed pages.

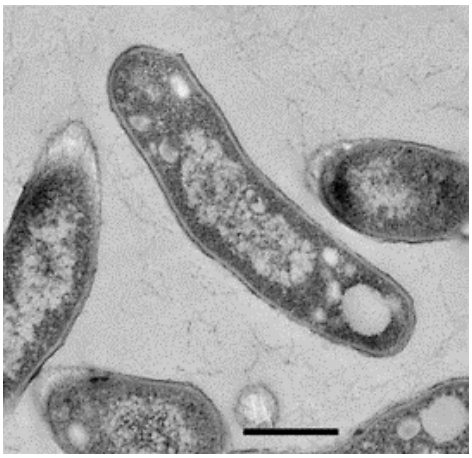
[Turn over

Section A

Answer **all** the questions in this section.

1 **(a)** Tuberculosis (TB) is a major cause of ill health worldwide. Prokaryotic *Mycobacterium tuberculosis* is the causative agent of TB.

Fig. 1.1 shows the longitudinal section of *M. tuberculosis*, viewed under the transmission electron microscope, with a scale bar representing 500nm.



Source: Muhsin Özel, Gudrun Holland/RKI

Fig. 1.1

(i) List **two** features visible in Fig. 1.1 that identify these cells as prokaryotes.

Presence of (peptidoglycan) cell wall;

Unicellular;

[2]

(ii) Outline the process by which *M. tuberculosis* reproduce.

Binary fission;

semi-conservative replication of parental DNA begins at the origin of replication

each origin moves rapidly toward the opposite end of the cell and adhere to the cell surface membrane.

cell elongates. Elongation of the cell also separates the two identical copies of the chromosomes.

cell surface membrane invaginates, and deposits new cell wall materials. Two daughter cells are formed which are genetically identical to the parent cell.

.....

.....

[4]

(iii) Identify and explain **one** problem of trying to classify prokaryotes such as *M. tuberculosis*.

P1 hard to classify prok on, phenotype / morphology / appearance ;

R1 as, small / simple / single-celled / share similar cell structure / have limited range of cell shapes OR

.....

P2 idea that (biological) species concept does not apply to bacteria ;

R2 as they reproduce asexually ;

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.....

.....

[2]

(b) The World Health Organization (WHO) introduced a strategy in 2015 to end the global TB epidemic.

An important part of the strategy is to:

- identify people at risk of becoming infected with TB
- use methods to prevent transmission of TB.

(i) State the mode of transmission of *M. tuberculosis* to humans.

Ref to airborne droplets ;

.....

[1]

(ii) The BCG vaccination is one method of prevention recommended for use in countries where TB is common. The BCG vaccine contains a non-pathogenic, living form of the microorganism that causes TB.

Complete Table 1.1 by using a tick (✓) to identify the type of immunity that develops in a person who has been given the BCG vaccination

Table 1.1

artificial active immunity	✓
artificial passive immunity	
natural active immunity	
natural passive immunity	

[1]

(c) The treatment for people with active tuberculosis (TB) lasts six months and involves a combination of antibiotics. This is usually very effective if the person has a susceptible (non-resistant) strain of *M. tuberculosis*.

Table 1.2 summarises one recommended treatment strategy that involves a combination of antibiotics.

Table 1.2

antibiotic	**length of treatment**	**mode of action of antibiotic**
rifampicin (R)	6 months	enters bacterial cells and inhibits protein synthesis
isoniazid (H)	6 months	prevents the synthesis of cell wall components known as mycolic acids
ethambutol (E)	first two months	prevents mycolic acids from being added to the cell wall
pyrazinamide (Z)	first two months	prevents the synthesis of fatty acids

Susceptible strains of *M. tuberculosis* will be killed using any one of the antibiotics listed in Table 1.2. However, combination treatment is preferred as it is one method that can be used to reduce the impact to society of antibiotic resistance.

(i) Explain how antibiotic resistance in *M. tuberculosis* develops.

four from
overuse of, antibiotics / rifampicin
or
example of over use ; e.g. taking for viral infection, over prescribing for bacterial infection

people not completing course of antibiotics ;
reservoir of bacteria remains ;
ref. to mutation ;
any detail of the mutation ; e.g. protein produced has a changed binding site

bacteria with resistance, survive / selected for
or
only bacteria sensitive to antibiotic are, killed / selected against ; **A** antibiotic acts as a selection pressure

bacteria reproduce and pass on, gene / allele, for resistance to offspring
or
vertical (gene) transmission ;

(alleles for resistance to antibiotics transferred by) horizontal (gene) transmission / described ;
frequency of resistance, gene / allele, increases in the bacterial population ;

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[4]

(ii) With reference to Table 1.2, explain how combination treatment for TB can help to reduce the impact of antibiotic resistance compared to single antibiotic treatment.

any ***four*** *from:*

1 (in combination treatment) antibiotics (in Table 2.1), act at different targets / have different modes of action / AW ;
A comparison of any two antibiotics from Table 2.1
A suggestion of how two antibiotics have different ways of killing

2 *idea that* if resistance / mutation, occurs / exists, unlikely to be against all antibiotics / other antibiotics should still be effective ;

3 (in combination treatment) if resistance / mutation, occurs / exists, all bacteria will (still) be, killed / destroyed / AW ;
A no bacteria remain to develop resistance / no reservoir of resistant bacteria

4 antibiotic resistance, not / less likely to be, spread to affect people because no bacteria surviving (with combination treatment) ; AW

5 long treatment time / 6 months, with, combination treatment / AW, increases chance of killing all bacteria
or
long treatment time with a single antibiotic not effective in killing all bacteria if, resistance develops / a mutation occurs ;

6 AVP ; e.g.
combination treatment (is likely to) eliminate bacteria more quickly (so less chance of resistance occurring)

resistance to different antibiotics involves more genes so less chance of resistance occurring

gene for antibiotic resistance has more chance of being passed on if using single antibiotic (and not all killed) ora

if using single antibiotic (and not all killed) more chance of being passed on (to other bacteria) by horizontal / vertical / AW, transmission ora

[3]

Rifampicin binds tightly to an RNA polymerase molecule close to its active site. This affects the activity of the enzyme.

(d) (i) Outline briefly the main role of RNA polymerase in *M. tuberculosis*.

Transcription + forming mRNA using one of the DNA strand as template;

Catalyse phosphodiester bonds between ribonucleotides;

[2]

(ii) Suggest the effect of rifampicin on RNA polymerase.

*any **three** from:*
I stops transcription
1 alters shape of / blocks, active site ;
R enters / fits into, active site *as it is not competitive*
R *ref. to* choice of competitive or non-competitive
A alters shape of enzyme *only if mp2 gained*

2 substrate / nucleotides, cannot, bind to / enter, active site ;
A fewer / no, enzyme-substrate complexes / ESCs, form
allow ecf from mp1 if rifampicin described as, competitive / enters active site

3 complementary (base) pairs / (complementary) base pairs, cannot form / form less easily (between DNA and RNA nucleotides) ;

4, 5, 6 *(in context of rifampicin binding to RNA polymerase)*
prevents / AW, unwinding of DNA (double helix) ;
A uncoiling
I unzipping prevents / AW, attachment to DNA (strand)
A prevents / AW, attachment to promoter
prevents / AW, movement along DNA (strand) ;
prevents / AW, elongation (of polynucleotide) / formation of polynucleotide /nucleotides being joined ;
prevents / AW, phosphodiester bond formation ;
prevents / AW, proof reading ;

7 AVP ; e.g. prevents / AW, tRNA / rRNA, formation

[2]

(e) RNA polymerase is composed of five different polypeptides. Gene *rpoB* codes for one of these polypeptides known as the β-subunit.

One or more mutations in a specific region of *rpoB* result in strains of *M. tuberculosis* that are resistant to rifampicin. In these strains, mutations often occur in two DNA triplets in this region, in positions 526 and 531.

Table 1.3 summarises the results of an investigation into seven rifampicin-resistant strains, **A** to **G**, that have amino acid changes for positions 526 and 531.

Table 1.3 includes:

- the change in the **mRNA codon** for position 526 or position 531
- the amino acid change that has occurred as a result of the mutation
- the minimum concentration of rifampicin required to inhibit growth of the bacterial strain (MIC)
- the number of **other** mutations occurring within the specific region of *rpoB*.

Table 1.3

Key
≈ approximately ⩾ greater than or equal to ⩽ less than or equal to

strain	codon involved	mRNA codon change	amino acid change	MIC / $\mu g\,cm^{-3}$	number of other mutations in the specific region
A	526	CAC → UAC	His → Tyr	⩽50	0
B	526	CAC → AAC	His → Asn	⩾100	1
C	526	CAC → CGC	His → Arg	≈50–75	2
D	526	CAC → CGC	His → Arg	⩾100	3
E	526	CAC → CGC	His → Arg	≈50	3
F	526	CAC → UUC	His → Phe	⩾100	3
	531	UCG → UUG	Ser → Leu		
G	526	CAC → UAC	His → Tyr	⩾100	3
	531	UCG → UUC	Ser → Phe		

(i) Complete Table 1.3 to show the amino acid changes that have occurred in strains **F** and **G**. [1]

(ii) With reference to Table 1.3, list the strains of *M. tuberculosis* that show the greatest resistance to rifampicin.

B, D, F, G

.....

[1]

(iii) Suggest reasons to explain why strains **C**, **D** and **E** show different levels of resistances to rifampicin.

resistance

1 shape / tertiary structure, of, β-subunit / enzyme, altered / AW ;
I active site changes shape
A quaternary structure of enzyme altered

2 rifampicin / antibiotic, cannot / does not, bind (as well) ; AW
R *if context is binding to active site*

different levels of resistances
*any **two** from:*

3 (because) other mutations are involved ; AW
A described from Table 2.1
e.g. **C** has 2 (other) mutations and **D** and **E** have 3

4 mutations may result in different changes to, structure / shape of, β-subunit / enzyme, and result in different, effects / levels of resistance ;

5 data from Table 2.2 to support mp 4 ; *must be linked to concept of mp4*

6 AVP ; e.g. *ref. to* different binding abilities (of rifampicin to enzyme)
some (of the other) mutations may cause more of a change to binding site for rifampicin
mutation(s) may make it harder to bind
lower resistance = binds, more strongly / for longer time
lower resistance = higher proportion of transcription events hindered
e.g. idea that mutations still, produce functioning enzyme / allow catalysis to occur

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[3]

(f) WHO Global Tuberculosis Report for 2019 published data on the estimated number of deaths from TB and HIV/AIDS in 2018. All deaths of people from TB who were infected with HIV were also counted as deaths of people with HIV/AIDS.

Fig. 1.2 shows these data. The dark grey boxes show the estimated number of deaths of people from TB who were also counted as deaths of people with HIV/AIDS.

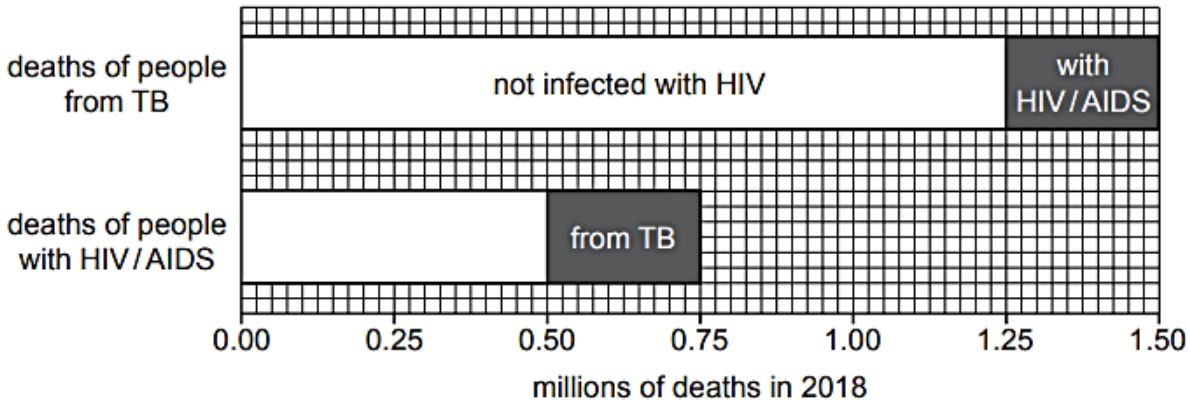

Fig. 1.2

A student used the data in Fig. 1.2 to predict that measures to control the spread of HIV will decrease the number of deaths from TB.

Discuss whether the data in Fig. 1.2 support this prediction.

*any **three** from:*

does not support:
1 much larger proportion do not have HIV / AIDS / AW ;
2 data to support ;
e.g. deaths of people from TB with HIV / AIDS is 0.25 million out of 1.5 million
3 other factors may cause more deaths from TB ;

supports:
4 large proportion of deaths of people with HIV / AIDS are caused by TB ;
5 data to support ;
e.g. deaths of people with HIV/AIDS caused by TB is a minority / 0.25 million out of 0.75 million
6 fewer people will be immune compromised ;
7 AVP ;
e.g. ref. to different modes of transmission so, cases of / deaths from, TB may not change
not enough information, qualified

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[3]

[Total: 29]

2 **(a)** All organisms respire. The ATP produced as a result of respiration is used as the energy currency of the cell.

(i) Outline **two** examples of movement in cells that use ATP.

*any **two** from:*

1 *ref. to* muscle fibre / sarcomere, contraction ;

2 active transport of (named), ion / molecule
or
active transport (of substance) against concentration gradient ;

3 movement of, vesicles / organelles, through cytoplasm / described ;

4 exocytosis of named substance ;

5 endocytosis / phagocytosis ;

6 spindle fibre / chromosome / chromatid, (movement) during, mitosis / meiosis ;

7 cilia / flagella, wafting / beating / AW ; **I** movement

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[2]

(ii) ATP cannot be stored in cells so it has to be continually re-synthesised to meet the demands of an organism.

A person with a total quantity of 0.2 moles of ATP needs to hydrolyse 150 moles of ATP per day.

Calculate how many times the total quantity of 0.2 moles of ATP has to be re-synthesised per hour to meet the demand of 150 moles per day.

Show your working and give your answer to the nearest whole number.

correct working ; e.g. $\frac{150}{0.2} = 750$ then $\frac{750}{24}$

or

$\frac{150}{24} = 6.25$ then $\frac{6.25}{0.2}$

or

$\frac{149.8}{0.2} = 749$ then $\frac{749}{24}$

31 ;

answer = ... [2]

(iii) Name the stages in which chemiosmosis occurs in respiration and in photosynthesis.

respiration oxidative phosphorylation

photosynthesis photophosphorylation / light dependent stage

[2]

(b) Fur seals are mammals that are adapted to live in cold temperatures. Fur seals have large quantities of a type of fat tissue known as brown adipose tissue. Brown adipose cells contain many mitochondria. These mitochondria contain a transport protein called thermogenin.

Fig. 2.1 shows the role of thermogenin in a mitochondrion of a brown adipose cell when external temperatures are cold.

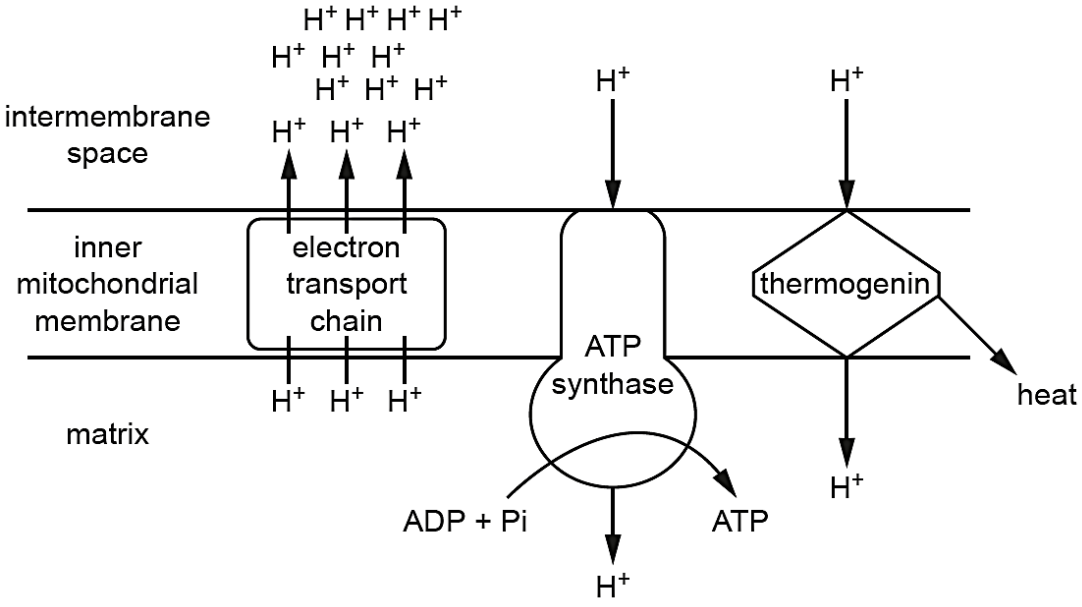

Fig. 2.1

(i) With reference to Fig. 2.1, describe **and** explain the effect of thermogenin on ATP synthesis.

1 reduces ATP synthesis / less ADP reacts with Pi ;

*any **two** from:*

2 protons diffuse through thermogenin ;

3 proton gradient reduced / reduces concentration of H+ in intermembrane space ;

4 fewer protons pass through ATP synthase ;

.....

[3]

(ii) When the external temperature is warm, thermogenin cannot function.

When the external temperature becomes cold, thermogenin is able to function as a result of cell signalling:

- adrenaline is released
- adrenaline acts on G protein-coupled receptors found on brown adipose cells
- a sequence of events is triggered that results in the activation of the enzyme lipase
- lipase hydrolyses triglycerides in the cells into fatty acids
- fatty acids enter the mitochondrion
- thermogenin starts to function.

Outline the stages of cell signalling that trigger the functioning of thermogenin.

1 G-protein / adenyl(yl) cyclase, activated ;

2 *ref. to* cAMP / second messenger, (is formed) ;

3 *ref. to* enzyme cascade / signal amplification / activation of kinase / signalling cascade ;

AVP: activation of lipase by phosphorylation ;

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[3]

[Total: 12]

3 **(a)** Scientists have produced structures known as virosomes, which are used in certain vaccines. Virosomes do not cause disease.

Fig. 3.1 is a diagram of a section through a virosome used in some vaccination to protect against the virus which causes influenza.

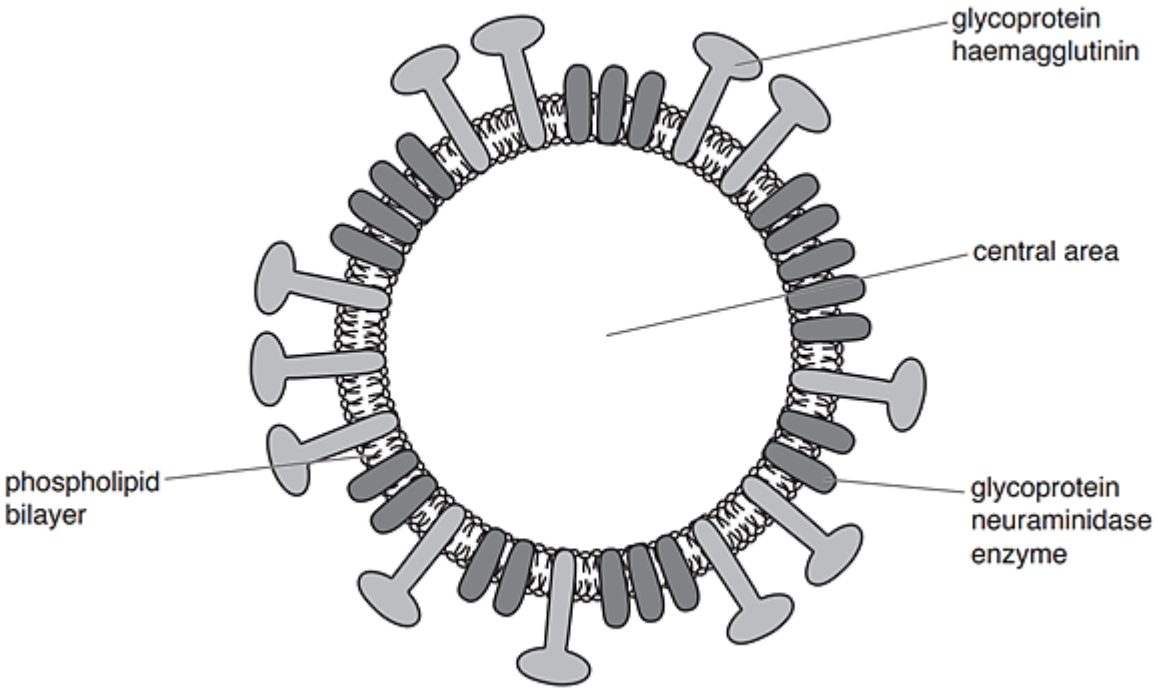

Fig. 3.1

(i) State the differences between the structure of a virosome and an influenza virus. [2]

no, nucleic acid / genetic material / DNA / RNA ;

no, capsid proteins ; A no capsomeres

[2]

(ii) Explain how the structure of the virosome shown in Fig. 3.1 suggests that the central area of the virosome is aqueous.

heads / phosphate, (groups) of phospholipids are hydrophilic

or

fatty acid tails of phospholipids are hydrophobic ;

A heads are hydrophilic and tails are hydrophobic

heads / phosphates, point towards the centre (of the virosome so, must be aqueous / water present) ; ora for tails

.....

.....

[2]

(b) The glycoproteins haemagglutinin and neuraminidase are found in the influenza virus and in the virosomes used in a vaccine against the influenza virus.

Haemagglutinin binds to a receptor in the cell surface membrane of phagocytes.

Suggest why haemagglutinin is present in virosomes used in the vaccine for influenza.

any two from:

acts as a non-self / foreign, antigen ;

triggers / stimulates, primary immune response

or

provides (artificial) active immunity ;

(leads to) formation of antigen presenting cell ;

A endocytosis / phagocytosis, to present antigen (by, macrophage neutrophil)

activates, B lymphocytes / T lymphocytes ;

A clonal selection

formation of memory cells ;

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.....

[3]

(c) Different strains of the influenza virus have formed as a result of mutations. Each strain of the virus contains the enzyme neuraminidase. Neuraminidase helps the virus to leave host cells after the virus has replicated. In each strain of the influenza virus, the primary structure of the active site of the neuraminidase enzyme remains unchanged.

Suggest why the primary structure of the active site of neuraminidase remains unchanged in each strain of the influenza virus.

any two from:

no change in, shape / tertiary structure / conformation, of active site ; ora

(same) substrate able to bind to active site ; ora

consequence ; e.g. so viruses can leave host cell to infect other cells

the mutation is not occurring in the, gene / section of gene coding for amino acids in active site for neuraminidase ;

the gene for neuraminidase is essential for survival ;

AVP ;

.....

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.....

[2]

[Total: 9]

Section B

Answer **one** question in this section.

Write your answers on the separate answer paper provided.

Your answers should be illustrated by large, clearly labelled diagrams, where appropriate.

Your answers must be in continuous prose, where appropriate.

Your answers must be set out in parts **(a)** and **(b)**, as indicated in the question.

4(a) 'All mutations in the cells of a human body are harmful.' [13]

Discuss this statement.

(b) Cycles play important roles in both natural and man-made biological processes. [12]

Explain the significance of cycles in biology.

[Total: 25]

5(a) 'All cells of a human body are genetically identical'. [13]

Discuss this statement.

(b) 'Membranes of different types of cells are involved in many different functions.' [12]

Explain this statement.

[Total: 25]

4(a) 'All mutations in the cells of a human body are harmful.' [13]

Discuss this statement.

QwC – Argument presented for both sides (agree/disagree) with at least 2 examples to substantiate each.

Agree, mutations are harmful (max 8)

A. Multi-gene disorders – Cancer

1. Cancer cells have gain of function mutations in one allele of proto-oncogenes such as Ras gene and
2. loss of function mutations both alleles in tumour suppressor genes such as p53 gene;
3. [Ref to any 2] Accumulation of other mutations such as in genes involved in **density-dependent inhibition, anchorage-dependence, angiogenesis, activation of telomerase in cancer cells;**

[Negative Effects of cancer – 2 for 1 mark]

1. Unable to arrest cell division;
2. Unable to carry out DNA repair;
3. Unable to induce apoptosis in cancerous cells;
4. Leads to **uncontrolled cell division**
5. Forms benign tumours;
6. Can spread to other parts of the body and become malignant cancer;
7. Cancerous cells **compete** for nutrients with healthy cells, causing death of healthy cells;
8. **Reduced life span** of affected individual;

B. Point mutation disorder - Sickle cell anaemia

4. T is substituted for A at 17th nucleotide in gene encoding β chain of haemoglobin.
5. Glutamic acid (6th position) is being substituted by valine. Glutamic acid is hydrophilic whereas valine is hydrophobic.
6. As a result of the mutation (which changes the primary structure), **the tertiary structure of the molecule changes** as the R groups of valine form **different interactions** with other amino acids/ ionic or hydrogen bonds to hydrophobic interactions between R group
7. At **low oxygen concentrations**, HbS molecules stick to each other via their **hydrophobic regions.**
8. This results in the **polymerisation** of the molecules into **long fibres** inside red blood cells.
9. Fibres of abnormal haemoglobin deform red blood cells (RBCs) into a sickle shape.

[Negative Effects of SCD]

1. Sickle-shaped red blood cells are **less effective** at transporting oxygen around the body.

2. Due to their shape, sickled-shaped red blood cells **clump and clog** small capillaries, obstructing other cells from moving through the capillaries.
3. This may interfere with circulation, thus causing **oxygen deprivation** and leading to other symptoms such as [any 1] **breathlessness/ physical weakness/ pain / organ damage.**
4. Sickle-shaped RBCs have a shorter lifespan compared to normal RBCs and **haemolyse readily,** resulting in anaemia.

C. Chromosomal mutations – Down syndrome (accept other syndromes)

10. Failure of homologous chromosomes 21 to separate during anaphase I of meiosis / Failure of chromatids of chromosome 21 to separate during anaphase II of meiosis
11. This results in the formation of gametes carrying **(n + 1)/24** chromosomes
12. Subsequent **fusion with normal haploid gamete** would then lead to the **zygote carrying 2n+1/47 chromosomes**
13. Individuals with Down syndrome caused by trisomy 21 have **one extra copy of chromosome 21**

[Negative Effects of Downs' Syndrome – 2 for 1 mark]

1. Characteristic facial features, short stature,
2. Speech defects
3. Heart defects
4. Susceptibility to respiratory infection
5. Mental retardation
6. Greater risk of developing Alzheimer's disease
7. Greater risk of developing leukaemia.

Disagree – [Max 8]
Mutations are beneficial:

D. Evolutionary advantage for natural selection

14. Spontaneous mutations in cells creates **new alleles**
15. Chromosomal mutations in gametic cells cause **new combinations of alleles/shuffling of alleles**
16. These new combination of alleles/new alleles may confer a selective advantage under a selection pressure
17. Individuals with mutations can **survive, reproduce and pass down these alleles** to their offsprings
18. Genetic variation is **necessary for natural selection** to act on

E. Heterozygote advantage

(same as B4-9)

4. T is substituted for A at 17th nucleotide in gene encoding β chain of haemoglobin.

5. Glutamic acid (6th position) is being substituted by valine. Glutamic acid is hydrophilic whereas valine is hydrophobic.
6. As a result of the mutation (which changes the primary structure), **the tertiary structure of the molecule changes** as the R groups of valine form **different interactions** with other amino acids/ ionic or hydrogen bonds to hydrophobic interactions between R group
7. At **low oxygen concentrations**, HbS molecules stick to each other via their **hydrophobic regions.**
8. This results in the **polymerisation** of the molecules into **long fibres** inside red blood cells.
9. Fibres of abnormal haemoglobin deform red blood cells (RBCs) into a sickle shape.

Note: Do not double credit if pts 4-9 has been mentioned for sickle cell anaemia

[Positive Effects]

1. Malaria infection leads to low oxygen concentration in the blood/hypoxia induces sickling of red blood cells. Sickled red blood cells and the parasites trapped inside are destroyed by white blood cells
2. Sickled cells also reduce the flow of blood that hampers the parasite's ability to travel and rapidly infect new cells.
3. Hb^AHb^S individuals are a selective advantage and are able to survive and reproduce in malaria-infected areas

F. Somatic hypermutation

19. Activated **B cells**/plasma cells undergo somatic hypermutation
20. This introduces frequent point mutations into the **variable (v) regions / V, D, or J segments** of the already rearranged **heavy and light chain** gene locii at a high rate

[Positive Effects]

1. Altered variable regions of the B cell receptors/antibodies have a higher affinity for the antigens/bind better to the antigens
2. Allows for only B cells with **higher affinity B cell receptors** to be selected to **differentiate** into **plasma** and **memory cells**.

G. Some mutations have no effect (neither harmful nor advantageous)

21. Ref to silent mutations that are base substitutions mutations in the third base of a codon that does not change the amino acid encoded for
22. due to degeneracy of the genetic code

23. Ref neutral mutations that are base substitution mutations in the codon that change the amino acid to another amino acid with a similar R group/chemical properties or;
24. mutations in the codon that change the amino acid in a non-essential part of the proteins

25. No effect on **3D conformation and function** of the protein
26. Point mutations in non-coding sequences such a centromeres, telomeres, inter-genic regions have no effect on phenotype of a cell

(b) Cycles play important roles in both natural and man-made biological processes. [12]

Explain the significance of cycles in biology.

A. Cell Cycle [max 5]

1. The cell cycle is the **sequence of events** which occurs **between the formation of a cell and its division into daughter cells,** with the cycle **repeating** for each daughter cell;
2. Three main stages: **interphase, nuclear division, cytokinesis**;
3. During interphase - Cell produces many materials and or organelles required for carrying out all its functions;
4. Cell **replicates its DNA** (during **S phase of interphase**) to prepare for nuclear division;
5. Nuclear division – **mitosis** – forming **identical** cells for **growth / repair / asexual reproduction** AND **meiosis** – forming **gametes**;** (significance)
6. Cytokinesis - Division of **cytoplasmic contents** into **2 daughter cells**;
7. **Fusion** of a **haploid** sperm and **haploid** egg during **fertilisation** results in the formation of a **diploid zygote**;** (significance)
8. After fertilisation, the zygote undergoes a process of nuclear division **(mitosis)**; **(**This generates cells that are **genetically identical** to the original zygote)

B. Krebs Cycle [max 5]

9. **Pyruvate** from glycolysis is converted to **acetyl-CoA** via **oxidative decarboxylation**;

Krebs cycle involves 3 main stages:

10. Acetyl CoA (2C) joins the cycle by combining with **oxaloacetate** (4C) to form **citrate** (6C);
10. Citrate is **decarboxylated** and **oxidised by dehydrogenation** to form **α-ketoglutarate** (5C) and **NADH;** (A: **oxidative decarboxylation**)
10. **Oxaloacetate** (4C) is **regenerated**; (idea of a cycle)
10. Krebs cycle involves multiple reactions, including **1 substrate-level phosphorylation**, **1 decarboxylation**, and **3 dehydrogenation reactions**;
10. As a result, **1 ATP, 1 CO_2, 2 NADH, and 1 $FADH_2$** are produced **per molecule of (-ketoglutarate**;

10. For **each** molecule of glucose, glycolysis yields **2 molecules of pyruvate**, and hence **2 molecules of acetyl CoA**. As such, **two rounds of Krebs cycle** are needed to **completely oxidise one molecule of glucose**;

10. All 6 carbon atoms in glucose are lost as 6 CO_2;
10. For each 6C glucose molecule, **2 CO_2** are released via **link reaction**, and **4 CO_2** are released via **Krebs cycle;**
10. The mobile electron carriers (**NADH and $FADH_2$**) with their reducing power will next be transported to the **electron transport chain**, where the bulk of **ATP** is generated;** (significance)

C. Electron Transport Chain and Cyclic Photophosphorylation [max 5]

19. The **photo-excited electron from P700** is captured by the **PS I primary electron acceptor;**

19. and then passed on to the middle part of the **first electron transport chain (ETC);**

19. As the photo-excited electron travels down the ETC, which consists of **electron carriers** of **progressively lower energy levels**, energy lost is **coupled to the formation of ATP via chemiosmosis;**

19. This electron eventually **fills the electron "hole" left in P700**, completing the cycle; (idea of cycle)

This way of synthesizing ATP using light energy is called **cyclic photophosphorylation.**

23. **No NADPH** is produced but passing the excited electrons to the second electr transport chain as in the non-cyclic light-dependent reaction, they are transferred the first electron transport chain;
23. **No O_2** is produced as there is **no photolysis of water;**
23. Hence, **only ATP** is produced by **cyclic light-dependent reaction;**** (significance)

D. Calvin Cycle [max 5]

Calvin cycle is a pathway that reduces carbon dioxide to produce carbohydrates. It comprises of 3 phases:

26. Carbon fixation - This step involves **carbon dioxide** combining with **RuBP** (ribulose bisphosphate, a 5C sugar);
26. Product is an unstable 6C intermediate that will immediately split to form 2 molecules of **glycerate phosphate (GP) / phosphoglycerate (PG)** (for every one molecule of CO_2);
26. PGA reduction - **GP is reduced (gains electrons)** to form a 3C compound, **glyceraldehyde-3-phosphate (G3P)**;
26. This reaction requires the reducing power of **NADPH** and energy of **ATP** (products of non-cyclic light-dependent reaction);
26. RuBP regeneration - G3P has to be used to regenerate **RuBP;** (idea of cycle)
26. **5** molecules of **G3P** (a 3C molecule; total 15C) used to regenerate **3 RuBP** (a molecule; total 15C);
26. In each round of Calvin cycle, **1 G3P channeled out** for eventual synthesis i **glucose**, and glucose can be polymerized into **starch** and **cellulose** AND regenera **$NADP^+$**;** (significance)

E. Polymerase Chain Reaction (PCR) [max 5]

33. A brief heat treatment (up to **95°C**) to **denature / unzip and separate** the two strands of DNA double helix into single-strands;
33. Cooling of the DNA (to **~60°C**) in presence of a large excess of **DNA primers** allows their specific **annealing** to complementary sequences at the 3' ends of each of the template DNA strand;

33. *Taq* polymerase **synthesises** the complementary DNA strand by catalysing the formation of **phosphodiester bonds** between dNTPs at an optimum **72°C**;
33. **Chain extension** occurs from **3' end of DNA primer** which provides free **3' OH group** required by *Taq* polymerase;
33. When the above elongation is completed, the primer has been lengthened into a **new complementary strand** of the single-stranded DNA fragment;
33. Because both separated DNA strands are used as templates, there are now **two copies of the original fragment** (each DNA molecule becomes two);
33. Each **repeated** cycle results in a **doubling** in number of DNA molecules being replicated;
33. The amount of desired sequence hence **increases exponentially** after **multiple cycles**;** (significance) (A: reference to 2^n cycles)

** Required for full credit of each section.

Possible AVPs:
Feedback cycle / Enzyme feedback inhibition / Life cycles (named organism)

QWC (1 m): Coherent, comprehensive and well-organised accounts of relevant cycles (minimum of three such accounts, each clearly indicating the cyclical nature, importance or significance of the cycle.

[Total: 25]

5(a) ‘All cells of a human body are genetically identical’. [13]

Discuss this statement.

QwC 1 mark – Discussed both sides of the argument (agree/disagree) + at least 2 examples of genetically different cells

Reasons for cells being genetically identical

1. Single-cell **zygote** divides and multiplies via **mitosis** to form a **genetically identical cells** of a multicellular human body
2. [ref to] **equal division of chromosomes** during mitosis
3. [ref to] semi conservative replication
4. [describe semi conservative replication] DNA unwinds and both strands of the double helix separate from each other through the breaking of hydrogen bonds between the complementary base pairs.
5. Each DNA parent strand (of a double helix) acts as a template to direct the synthesis of a complementary daughter strand.
6. Ref to **complementary base pairing** between parent and daughter strand

Reasons for cells being genetically different [max 8]

A. Gametes –

7. **Sperms and eggs** are not genetically identical to other gametes/rest of the somatic cells
8. As they are **haploid**/have half the number of chromosomes
9. Homologous chromosomes undergo **crossing over during prophase of meiosis I new combination of alleles** on chromosomes
10. Homologous chromosomes undergo **independent assortment and** segregation during **metaphase and anaphase of meiosis I different combinations of maternal and paternal chromosomes** in gametes
11. Ref. aneuploidy/polyploidy

B. B lymphocytes–

12. Immature B cells undergo somatic recombination
13. Specific V and J segments on light chain locus and V, D and J segments on the heavy chain locus are ligated together.

14. Activated B cells/plasma cells undergo somatic hypermutation at the **variable regions** of the already rearranged **heavy and light chain** gene loci;
15. ref. frequent point mutations at variable region;

16. Mature B cells undergo class switching at heavy chain locus;
17. **Different heavy chain** constant regions are linked to the **same variable region** on the heavy chain locus

C. Spontaneous mutations –

18. **Point/gene mutations + 2 examples** (deletion, substitution, addition, inversion)
19. [ref to] mutations during **DNA replication** during **mismatching of base pairs**
20. Ref. to error in proof reading
21. **Chromosomal mutations** can occur due to carcinogens/radiations that cause double strand breaks in the DNA

22. Example : Cancer cells have gain of function mutations in oncogenes and loss of function mutations in tumour suppressor genes;
23. Accumulation of other mutations such as in genes involved in **density-dependent inhibition, anchorage-dependence, angiogenesis, activation of telomerase in cancer cells;**

24. Example: Mutations in the **non-coding areas/silenced genes/neutral mutations/silent mutations** do not have any effect on the proteins.
25. Genetic variability arises due to **accumulation of mutations** degree of genetic difference

(b) 'Membranes of different types of cells are involved in many different functions.' [12]

Explain this statement.

NOTE:
1. Focus is on functions of membranes *not* functions of organelles.
2. The headings highlight the various functions that membranes (of different cell types) are involved in.
3. It is important to provide specific examples of cells and/or membranes so that you can meaningfully answer the question.

Transport Processes
1. **Channel protein / carrier proteins** embedded allow for **facilitated diffusion** of specific molecules across cell surface membrane into or out of cell down a concentration gradient;
2. Example:
   - **aquaporins** allow for **water** to move into or out of cells;
   - **glucose transporters** allow for uptake of glucose into cells for **respiration**;
3. **Pumps** embedded in cell surface membrane allow for **active transport** of molecules into or out of cells against a concentration gradient;
3. Example: **sodium-potassium pump** which pumps three sodium ions and two potassium ions out and into cells respectively to maintain membrane potential;
3. **Membrane pores** that allow specific molecules to be transported across the cell membrane
3. Example:
   - **Nuclear pores** allow nucleotides to enter nucleus for DNA replication / transcription

7. AVP (e.g. bacterial transformation / endocytosis)

[Max 4]

Protein Secretion

1. In the **β cells of the islets of Langerhans**, **A:** other examples such as α cells of the islets of Langerhans
2. **Ribosomes,** which are embedded into membranes such as cisternae of the **rough endoplasmic reticulum** ,which synthesise insulin polypeptides;
3. Insulin packaged into **transport vesicles** which bud off from membrane of rough ER and **move to Golgi apparatus**
   OR
   Insulin packaged into **secretory vesicles** which bud off from trans face of Golgi apparatus and **move towards cell surface membrane** for **secretion out** of the cell;
4. **Post-translational modification** occurs to insulin within the **cisternae** of **rough ER** / within **cisternae** of **Golgi apparatus**;
   A: any other relevant examples

Cell Signalling / Communication

1. **Insulin receptors** are **embedded** on cell surface membrane of **liver cells / muscle cells**; **A**: Any other relevant examples such as glucagon receptor
2. Insulin receptors **bind to insulin** + one of the following effects
   - **increased uptake of glucose** by liver/muscle cell from bloodstream to **decrease blood glucose concentration back to set point / 90mg/d**;
   - **increased rate of glycogen synthesis** via glycogenesis in liver cell / muscle cell;
   - AVP
3. **Glycoproteins** embedded on cell surface membranes of **epithelial cells** for **cell-to-cell adhesion**

Immune Response

1. **B cell receptor** (BCR) embedded on cell surface membrane of **naïve B cells**;
2. BCR allows for the **uptake of antigens** into naïve B cells to be **processed** for subsequent naïve **B cell activation**;
3. **T cell receptor** (TCR) embedded on cell surface membrane of **T helper cells**;
4. TCR binds to peptide-MHC complex on naïve B cells to activate naïve B cells into antibody-producing **plasma cells**; **A**: activation of naïve T cells / activation of memory B cells
5. Cell surface membrane of **phagocytes / macrophages** **form pseudopodia** extensions;
6. Ends of pseudopodia fuse and **pathogen** is **phagocytosed**, and **broken down** within phagocyte **A**: broken down by lysosomes
7. **Antigen** of pathogen can be **presented** on cell surface membrane of phagocyte. **A**: Any other APC

[Max 4]

Photosynthesis

1. **Thylakoid membrane** of **chloroplast** in plant cell;
2. **Photosystems, electron transport chains** and **ATP synthases embedded** on thylakoid membrane;
   (mention max 2 examples of proteins embedded on thylakoid membrane)
   3. Photosystems embedded allow for **absorption of light energy** during the **light dependent stage of photosynthesis / photophosphorylation**;
   3. **Energy released** during the movement of electrons down electron transport chain results in the **formation of a proton gradient** across the thylakoid membrane;

OR
Hydrophobic core of thylakoid membrane allows for proton gradient to be generated;
5. ATP synthases uses proton gradient to **synthesise ATP** via **chemiosmosis**;

[Max 4]

Respiration
1. **Inner mitochondrial membrane** is thrown into folds called **cristae**;
2. **Electron transport chains** and **ATP synthases** embedded on cristae;
3. **Energy released** during the movement of electrons down electron transport chain results in the **formation of a proton gradient** across the cristae;
   OR
   Hydrophobic core of cristae allows for proton gradient to be generated;
4. ATP synthases uses proton gradient to **synthesise ATP** via **chemiosmosis**

Structural
1. **Cellulose synthase** found on **plant cells**.
2. Catalyse the synthesis of **β-glucose** to form **cellulose cell wall.**
3. **Lysyl oxidase** of **animal cells**.
4. Catalyse **covalent bonds between tropocollagen.**
5. AVP

1. QWC: scope of answer to include functions of membranes from at least three different functions.

[Total: 25]