

NAME : _____

CLASS : _____



JURONG PIONEER JUNIOR COLLEGE

JC2 Preliminary Examination 2025

BIOLOGY

Higher 2

9744/03
15 September 2025

Paper 3 Long Structured and Free-response Questions

2 hours

Additional Materials: Answer Booklet

READ THESE INSTRUCTIONS FIRST

Write your class and name in the spaces at the top of this page.

Write in dark blue or black pen.

You may use an HB pencil for any diagrams or graphs.

Do not use staples, paper clips, 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 Booklet provided.

The use of an approved scientific calculator is expected, where appropriate.

You may lose marks if you do not show your working or if you do not use appropriate units.

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

For Examiner's Use	
1	
2	
3	
Section B	
Total	

This document consists of **19** printed pages and **1** blank page.

Section A

Answer **all** questions.

- 1 Following a body injury, bone marrow stem cells migrate to the site of damage, where they undergo cell differentiation to replace damaged cells. In eukaryotes, cell growth and division are tightly regulated processes to ensure proper repair. Cells will only pass specific checkpoints in the cell cycle if certain conditions are met, one of which is the presence of growth factors.

Fig. 1.1 shows how this differentiation occurs.

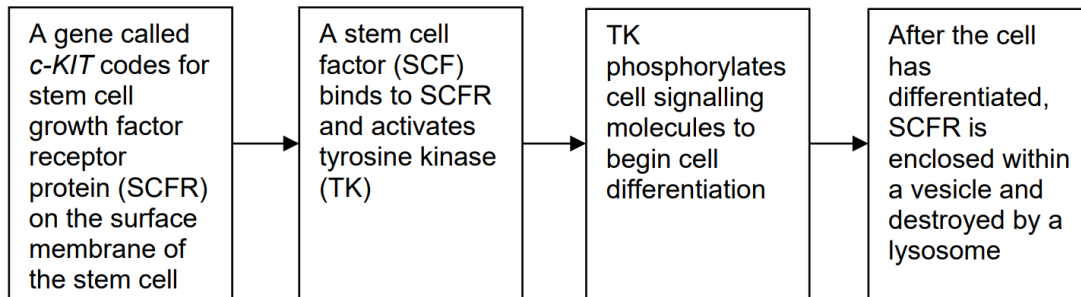


Fig. 1.1

- (a) (i) Outline how SCFR is produced and transported to the cell surface.

..... [4]

1. gene/*c-KIT* undergoes transcription in nucleus (to form pre-mRNA) ;
2. post-transcriptional modifications take place [A! RNA splicing, 5' capping, 3' polyadenylation (any 1)] to form mature mRNA ;
3. mRNA is translated at ribosomes on rough endoplasmic reticulum (rER) ;
4. biochemical modifications take place in rER ;
5. budding occurs with SCFR/receptor embedded in vesicle membrane / in vesicle ;
6. SCFR/receptor enter Golgi body to be further modified, sorted and packaged ;
7. budding occurs with SCFR/receptor embedded in vesicle membrane ;
8. (compulsory) vesicles with SCFR embedded in vesicle membrane move to and fuse with cell surface membrane (of stem cells) ;

MP5&7, award once

- (ii) Suggest how SCFR is destroyed by a lysosome.

..... [1]

1. The vesicle fuses/binds with a lysosome, the hydrolytic enzymes break down/hydrolyse/digest SCFR ;
A! Accept protease

Fig. 1.2 shows an example of how signals from growth factors are transduced to result in cell growth and division. The KRAS protein is a G-protein encoded by *KRAS* proto-oncogene.

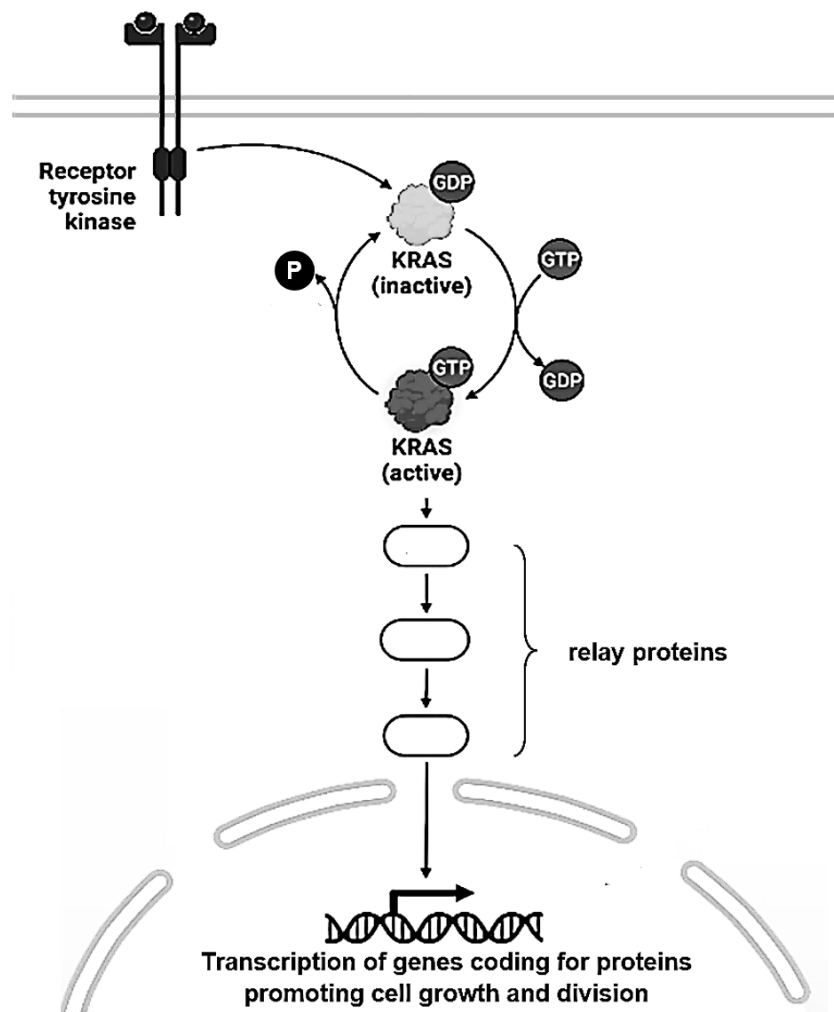


Fig. 1.2

(b) Describe the role of the KRAS protein in the transduction of signal from growth factors.

..... [3]

1. Growth factors recognise and bind to the specific binding site of tyrosine kinase receptors (RTK) on the target cell surface membrane, activating the RTK ;
2. The activated RTK binds to KRAS protein and activates it ;
3. A molecule of GTP replaces the GDP on the KRAS protein ;
4. The activated KRAS protein activates (downstream) relay proteins ;
5. to result in the transcription of genes coding for proteins promoting cell growth and division.

- (c) Mutations in *KRAS* proto-oncogene are common in colorectal (colon and rectum) cancer. The oncogene arises from the proto-oncogene by a mutation called G12D.

Fig. 1.3 shows part of the base sequence of the template strand of the *KRAS* proto-oncogene and the corresponding part of the oncogene. The corresponding parts of the primary structures of the two encoded *KRAS* proteins involved in GTPase activity are also shown.

amino acid position		6	7	8	9	10	11	12	
<i>KRAS</i> proto-oncogene	3' ...	GAA	CAC	CAT	CAA	CCT	CGA	CCA	... 5'
normal <i>KRAS</i> protein		leu	val	val	val	gly	ala	gly	
<i>KRAS</i> oncogene	3' ...	GAA	CAC	CAT	CAA	CCT	CGA	CTA	... 5'
mutant <i>KRAS</i> protein		leu	val	val	val	gly	ala	asp	

Fig. 1.3

- (i) State **one** possible environmental causative factor that increases the risk of G12D mutation shown in Fig. 1.3.

..... [1]

1. Ionising radiation e.g. X-ray, UV ray ;
 2. Chemical carcinogen e.g. tar in cigarette (smoke) ;
- (Any 1)

- (ii) With reference to Fig. 1.3, explain how the G12D mutation leads to colorectal cancer.

..... [5]

1. Single **Base (pair) substitution** (in *KRAS* proto-oncogene / idea of DNA) ;
2. Thymine replaces cytosine / CTA replaces CCA in the 12th triplet in the DNA ;
3. Instead of GGU, GAU is coded for in the mRNA / codon, resulting in a **missense mutation** ;
4. **Gain of function mutation** leading to **hyperactive** *KRAS* protein ;
5. **Gly replaced by asp, change in amino acid sequence in polypeptide/ primary structure** ;
6. Result in **change in 3D conformation** of (GTPase active site) in *KRAS* protein ;
7. Can no longer **hydrolyse GTP to GDP**, thus *KRAS* protein remains active ;
8. Results in continuous transcription of genes / excessive production of proteins, **promoting cell growth and division** ;
9. leading to **excessive cell cycle progression** and **uncontrolled cell division**, resulting in tumour formation and cancer ;

MP1-7 max. 4m

R! MP1 & 2 if just state at 12th amino acid position without idea that it occurred in DNA / gene ;

- (d) In a genetic study for colorectal cancer, two populations were screened for the G12D mutation. Table 1.1 shows the results of the study.

Table 1.1

group	total number of individuals	number of individuals with G12D mutation
without colorectal cancer (control group)	500	2
with colorectal cancer	400	120

- (i) Calculate the relative risk (RR) of G12D mutation in the cancer group compared to the control group using the formula provided.

$$RR = \frac{\text{risk of G12D in cancer group}}{\text{risk of G12D in control group}}$$

1. **Relative risk = (120/400) / (2/500) = 75 ;**

relative risk = [1]

- (ii) Discuss whether conducting screening tests to detect the G12D mutation is an effective way to screen for colorectal cancer.

..... [2]

1. **Effective because there is a strong association/link between G12D mutation and colorectal cancer ;**
2. **Ineffective because there are people with colorectal cancer who did not have G12D mutation (and their cancer diagnosis will be missed) ;**
3. **AVP ;**

The *KRAS* oncogene, with G12D mutation, can be detected in stool samples using a technique called droplet digital Polymerase Chain Reaction (ddPCR).

ddPCR partitions DNA samples into thousands of individual droplets within which DNA samples are amplified by PCR. Fluorescent-labelled probes specific to the *KRAS* oncogene are added after which the fluorescence is measured in each droplet.

The number of droplets containing the fluorescent signal indicates the amount of *KRAS* oncogene present in the sample. The method is highly sensitive, detecting even low levels of the *KRAS* oncogene.

- (e) (i) Suggest why stool samples are appropriate for the detection of *KRAS* oncogene.

..... [1]

1. ***KRAS* oncogene is present in cancer cells in colon/rectum cells which would be found/shed in stool when waste is passed out / egestion ;**

- (ii) Describe how DNA samples in each droplet are amplified by PCR.

..... [3]

1. Name of stages - denaturation, annealing (of DNA primer), (primer) extension ;
2. At **95°C**, the heat **breaks hydrogen bonds** between complementary bases of the double-stranded DNA, separating double-stranded DNA into **single-stranded DNA** ;
3. At **65°C**, (DNA) **primers anneal/complementary base pair** with complementary sequences at the **3' end of the target sequence** in the single-stranded DNA ;
R! 3' OH end of target sequence
4. At **72°C**, **Taq polymerase** synthesises the rest of the new strand of DNA by **adding dNTPs** to the **3' -OH ends** of (both) **primers** by catalysing the formation of phosphodiester bonds between adjacent dNTPs ;

- (iii) Explain why gel electrophoresis after PCR would not allow detection of *KRAS* oncogene.

..... [1]

1. The *KRAS* proto-oncogene and oncogene (differ in base sequence / difference in 1 nucleotide) and are of **same length** / number of base pairs / molecular weight and will migrate the same distance in gel electrophoresis ;

- (iv) Suggest an advantage of using ddPCR in cancer screening programmes.

..... [1]

1. It is sensitive, can detect small amounts of *KRAS* oncogene, allowing detection of early stages of colorectal cancer (when there are not many cancer cells / tumour is small) ;
R! Just stating procedure is sensitive without context
R! Procedure is fast thus can test many patients

- (f) Scientists investigated a drug called MiTMAB as a treatment for cancer. MiTMAB inhibits cytokinesis.

Fig. 1.4 shows drawings of cancer cells seen with an optical microscope from a:

- sample treated with MiTMAB
- control sample.

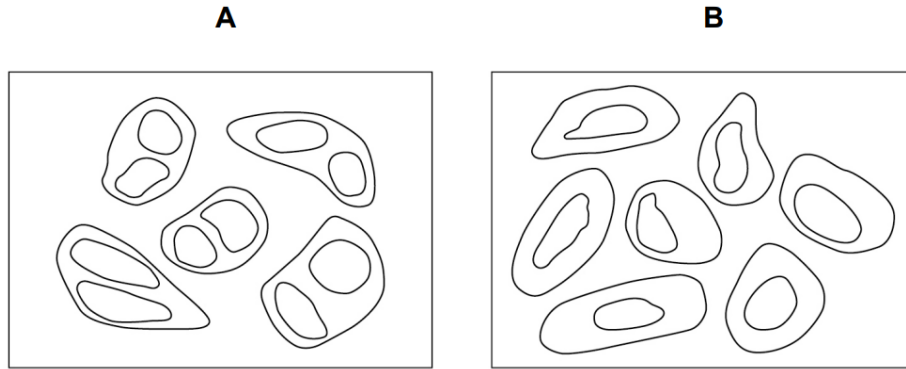


Fig. 1.4

The cells in drawing A can be identified as those treated with MiTMAB.

Explain why.

..... [2]

- 1. 2 nuclei/nuclear envelopes in 1 cell indicating that cytokinesis is inhibited / prevented;
A! 2 nucleus**
- 2. Cells stopped/arrested at telophase or stopped (new) cell membrane forming or stopped cytoplasm from dividing/splitting or prevents formation of cleavage furrow/invagination of cell surface membrane ;**

(g) MiTMAB acts as a non-competitive inhibitor of an enzyme called dynamin.

When active, dynamin has two functions:

- it stimulates cytokinesis
- it inhibits cell death.

The scientists treated actively growing cultures of cancer cells with MiTMAB.

They incubated:

- one sample of 2500 cells without MiTMAB as a control
- eight samples, each with 2500 cells and a different concentration of MiTMAB.

After 72 hours, the scientists measured the number of cells in each sample.

Fig. 1.5 shows the scientists' results.

A negative value for proportion of control growth means that fewer than 2500 cells were counted after 72 hours.

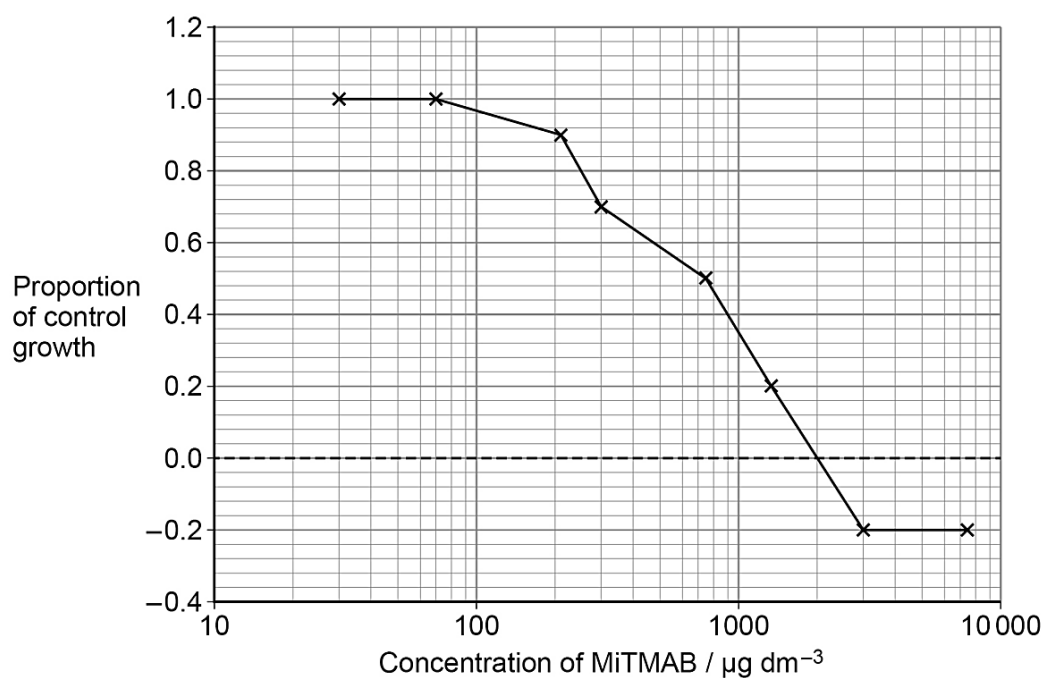


Fig. 1.5

(i) Use all the information given to explain the results shown in Fig. 1.5.

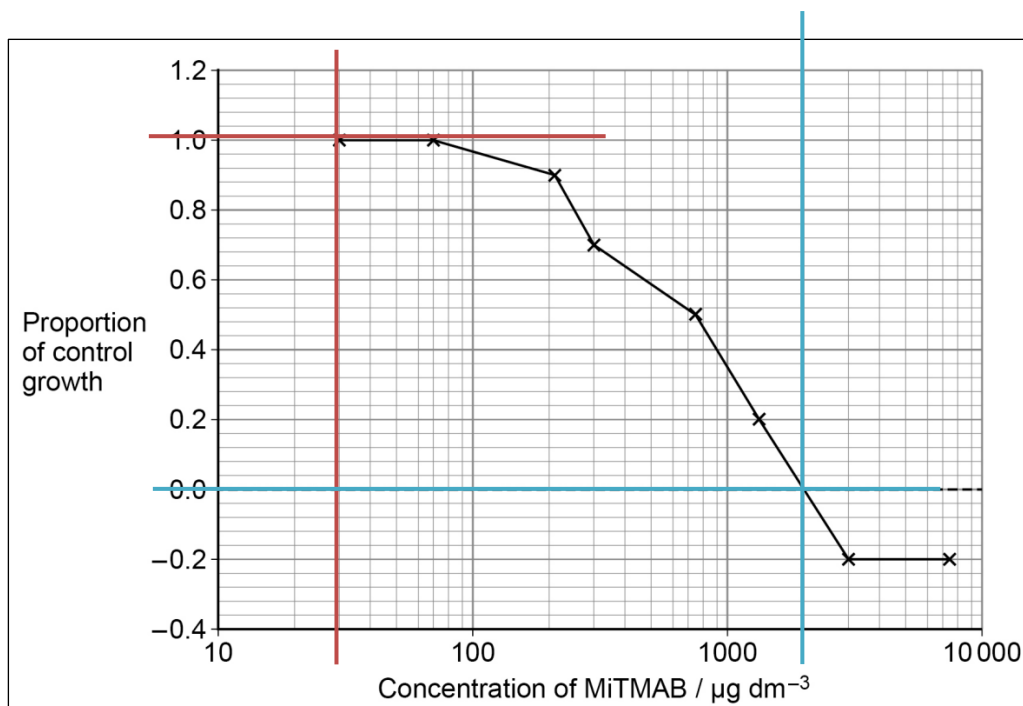
..... [3]

1. As concentration of MiTMAB increases from $30 \mu\text{g dm}^{-3}$ to $70 \mu\text{g dm}^{-3}$, proportion of control growth remain constant at 1.0, as insufficient dynamin is inhibited / is not inhibited thus MiTMAB does not cause cell death/does not inhibit cytokinesis ;
A! At lowest MiTMAB concentrations
A! has no effect on
2. As concentration of MiTMAB increases from $70 \mu\text{g dm}^{-3}$ to $2000 / 3000 \mu\text{g dm}^{-3}$, proportion of control growth decrease from 1.0 to 0.0 / -0.2, as more dynamin is inhibited thus fewer cells undergo cytokinesis and more cells undergo cell death ;
3. From 2000 to $7500 \mu\text{g dm}^{-3}$ of MiTMAB, there is less than 2500 cells after 72 hrs, as all dynamin are inhibited / MiTMAB does cause cell death and inhibit cytokinesis ;
A! At highest MiTMAB concentrations
4. [general trend] As concentration of MiTMAB increases from about 10 to about $10\,000 \mu\text{g dm}^{-3}$, proportion of control growth decrease from 1.0 to -0.2 ;
!! incorrect values
5. MiTMAB acts as non-competitive inhibitor, binding to allosteric site / site other than active site of dynamin, causing change in dynamin's 3D conformation causing it to become inactive thus cannot stimulate cytokinesis and inhibit cell death ;

- (ii) 0.01 dm^3 of MiTMAB solution was added to the treated cells.

Calculate the increase in mass of MiTMAB added to the cells to reduce the cell growth from equal to the control to 0.0 of the control.

Show your working and give your answer to **two** significant figures.



1. Concentration of MiTMAB at equal to the control (i.e. proportion of control growth = 1.0) = 30 OR 70 $\mu\text{g dm}^{-3}$

2. Increase in concentration of MiTMAB = $2000 - 30$ OR $2000 - 70$
= 1970 OR 1930

3. Increase in mass of MiTMAB = increase in conc. of MiTMAB $\times 0.01$

4. Correct answer – 19 OR 20 ;

MP1-3, max. 1m

mass = μg
[2]

[Total: 30]

- 2 *Mycobacterium tuberculosis* causes potentially fatal disease tuberculosis. With early diagnosis and the correct drug treatment, the pathogen can be eliminated from the body, especially if the infection has not progressed to active disease.

(a) Fig. 2.1 shows an electronmicrograph of *M. tuberculosis* cells.



Fig. 2.1

Identify **two** structural features that would distinguish *M. tuberculosis* from a typical eukaryotic cell.

..... [2]

1. Presence of peptidoglycan cell wall ;
2. Absence of membrane-bound organelles (e.g. mitochondria, ER, Golgi body) ;
3. Presence of 70S ribosomes ;
4. Absence of a nucleus / presence of nucleoid region ;
5. Presence of circular DNA ;

- (b) During tuberculosis infection, alveolar macrophages engulf the pathogen. Some researchers are investigating the use of stem cells in treating lung tissue damaged by tuberculosis.

Explain what is meant by a stem cell and suggest **one** reason why stem cells may be useful in treating tuberculosis.

..... [3]

Explain

1. A stem cell is an undifferentiated/unspecialised cell that is capable of, dividing indefinitely/self-renewal by mitosis ;
2. Has the ability to undergo differentiation, giving rise to specialised cell types ; (upon receiving appropriate molecular signals e.g. hormones, growth factors)

Suggest

3. Stem cells can regenerate / repair, lung tissues (A! replace lung cells), damaged by tuberculosis infection and potentially restoring normal lung function / restore respiratory function / promote healing of alveolar structures ; (idea marking)

To help prevent the development and spread of drug resistance in *M. tuberculosis*, the World Health Organization recommends using a treatment known as combination antibiotic therapy.

This therapy involves two different types of drugs:

- a fast-acting drug such as isoniazid, which rapidly kills actively dividing *M. tuberculosis*
 - one or more longer-acting drugs such as rifampicin or pyrazinamide that eliminate any remaining pathogens, including those in a dormant state.
- (c) Suggest why using combination antibiotic therapy with two different types of drugs is more effective in preventing the development of drug resistance in *M. tuberculosis* than treatment using only one type of drug.

..... [2]

1. Using drugs with different modes of action reduces the overall pathogen population more effectively ;
2. If a *M. tuberculosis* is resistant to one drug, the 2nd drug can still kill it, preventing the survival and multiplication of resistant strains ;
3. Combination antibiotic therapy reduces the chance that a *M. tuberculosis* will have mutations conferring resistance to both drugs ;
(A! bacterium)
4. Combination antibiotic therapy lowers the probability of *M. tuberculosis* that has developed resistance from being selected and becoming the dominant strain in the population ;

Beside tuberculosis, other infectious diseases caused by eukaryotic pathogens, such as malaria, is also a major public health concern. Like *M. tuberculosis*, *Plasmodium falciparum* is capable of evolving drug resistance, complicating treatment strategies and eradication efforts.

P. falciparum begins its life cycle in a mosquito vector and continues its life cycle within the red blood cells of its human host. The cells of *P. falciparum* in this stage are known as trophozoites.

Fig. 2.2 is a photomicrograph of a blood smear (thin layer of cells). Some of the red blood cells contain trophozoites.

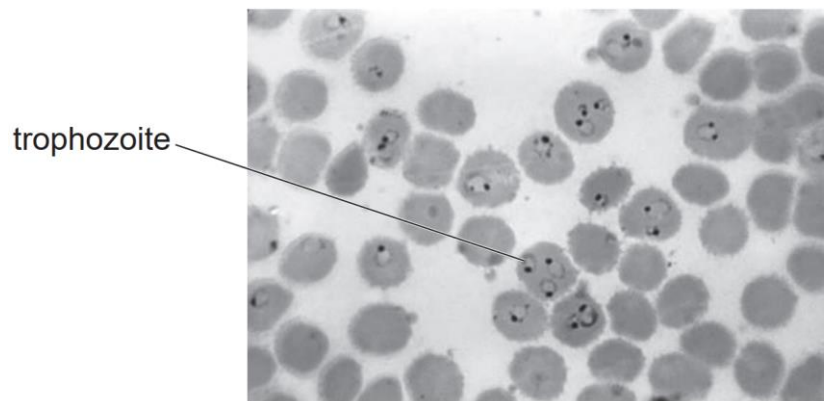


Fig. 2.2

Researchers have investigated how genetic mutations in *P. falciparum* affect the efficacy of artemisinin-based drugs. Artemisinin is a compound derived from the *Artemisia annua* plant and is commonly used to treat malaria. One such artemisinin-based drug is dihydroartemisinin (DHA).

In the following investigation, scientists measured the survival rate of trophozoites within red blood cells when exposed to two different concentrations of DHA.

Two different strains, **A** and **B** of *P. falciparum* were tested. For each strain, three different cultures were prepared:

- One with **no mutation in the *kelch13* gene** (serving as the control),
- One with the ***kelch13* F446I mutation**,
- One with the ***kelch13* C580Y mutation**.

These mutations in the *kelch13* gene have been linked to DHA resistance, making it important to study how they affect parasite survival and treatment outcomes.

Table 2.1 shows the six different cultures tested and the trophozoite survival rate for each culture.

Table 2.1

culture number	culture details	mean percentage survival rate of trophozoite	
		DHA concentration 20 nmol dm ⁻³	DHA concentration 700 nmol dm ⁻³
1	strain A no mutation	3.15	0.00
2	strain A, F446I mutation	26.00	0.73
3	strain A, C580Y mutation	33.08	0.91
4	strain B no mutation	2.86	0.00
5	strain B, F446I mutation	13.50	0.53
6	strain B, C580Y mutation	17.50	0.63

(d) State the main conclusions that can be drawn from the results shown in Table 2.1.

..... [2]

1. Cultures with either kelch13 mutation show significantly higher trophozoite survival rates than the control at both DHA concentrations ;
(A! F446I or C580Y)
2. Increasing DHA concentration from 20 nmol dm⁻³ to 700 nmol dm⁻³ greatly decrease the survival rates in all cultures (with or without with kelch13 mutations) ;
3. Both controls are highly sensitive to DHA, with very low survival rates at both concentrations ;
4. Both strains (A and B) show similar, trends/ patterns of resistance in response to DHA and the kelch13 mutations, suggesting the effect of these mutations on resistance is not strain-specific ;
5. kelch13 mutations (F446I and C580Y) in *P. falciparum* increase resistance to DHA, as shown by higher trophozoite survival rates, but high drug concentrations can overcome this resistance ;
6. AVP ;

(A! strains without mutations / no mutation control / wild-type)

[Total: 9]

- 3 Corals are simple marine animals and usually exist in colonies of thousands of individuals. Corals absorb calcium carbonate from the sea to build their skeletons, which help to form large coral reefs. Coral reefs are some of the most diverse ecosystems in the world.

Coral reef ecosystems are severely threatened. One of the threats include global warming, which can result in ocean warming, stressing corals and leading to coral bleaching and possible death.

- (a) Fig. 3.1 shows the changes in average global temperature and the changes in CO₂ concentration from Year 1400 to Year 2000.

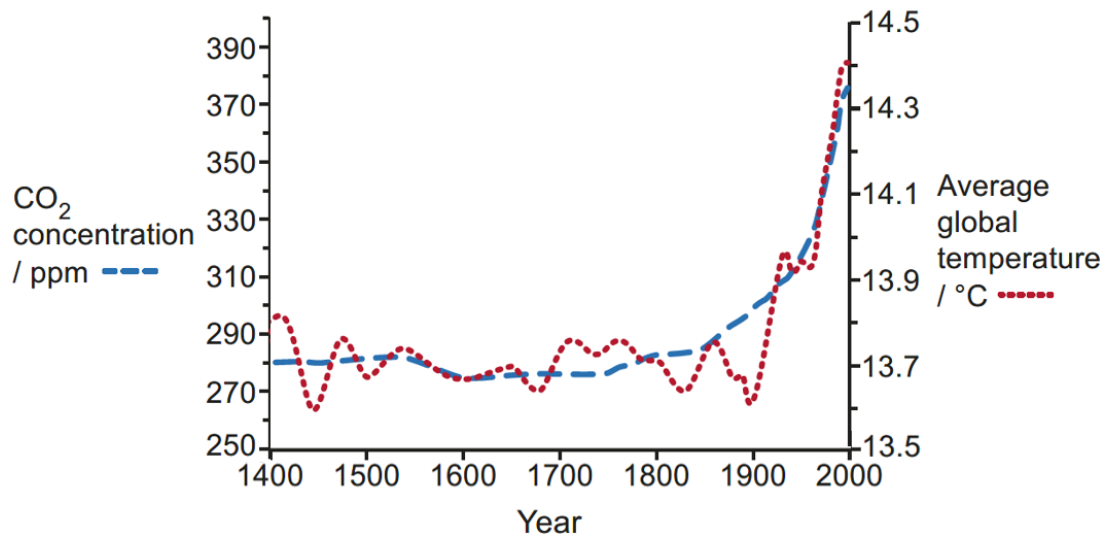


Fig. 3.1

- (i) Using Fig. 3.1 and your own knowledge, explain how global warming occurred.

..... [4]

1. From Year 1750 to Year 2000, CO₂ concentration increases from 280ppm to 370ppm ; A: +/- other values (1750, cos TYX says norm for 200 year)
2. (lead to) average global temperature increases from to 13.6 °C to 14.4 °C from Year 1900 to Year 2000 ;
A: increase from Year 1400 to Year 2000
3. Increased greenhouse gas (GHGs) emissions/the level of greenhouse gases in the atmosphere has increased, resulting in enhanced greenhouse effect ;
4. The enhanced greenhouse effect has increased the amount of heat trapped, resulting in global warming and increase in global average temperature ;

- (ii) Suggest **one** reason why some corals in natural reef systems might still survive despite ocean warming.

..... [1]

1. Some corals have more heat-tolerant strains ;
2. Corals in deeper/colder waters may be less exposed to thermal stress ;

- (b) The crown-of-thorns starfish (COTS) is also one of the main causes of the decline of the world's coral reefs.

Marine biologists used a choice chamber to investigate the effects of flashing and constant light on the behaviour of COTS. Table 3.1 shows their results as they presented them. The P values show results from a statistical test.

Table 3.1

behaviour of COTS	type of light used in choice chamber	
	flashing	constant
COTS moving towards the stimulus	22	12
COTS moving away from the stimulus	28	38
P value	0.69	0.02

- (i) The natural habitat of COTS is coral reefs of tropical oceans.

Suggest **two** factors that should be kept constant in the choice chambers so that COTS display normal behaviour.

..... [1]

1. Salinity / salt concentration of the water ;
2. Temperature (of the water) ;
3. Amount / distribution of food ;
4. pH (of the water) ;
5. Oxygen/carbon dioxide concentration Intensity/wavelength of (constant and flashing) light ;

Ignore humidity/type of coral/depth of water

- (ii) Evaluate the claim that either type of light could be used to cause COTS to move away from coral reefs.

..... [3]

Yes (no mark)

1. COTS moves away from either type/both types of light ;
2. Significant movement away from constant light as $p=0.02/0.05=69\%/>5\%$ OR Movement away from constant light is not due to chance as $p=0.02/<0.05=2\%/<5\%$;

No (no mark)

3. Flashing light causes a large number of COTS to move towards it ;
4. Movement away from flashing light is not significant as $p=0.69/>0.05=69\%/>5\%$ OR Movement away from flashing light is due to chance as $p=0.69/>0.05=69\%/>5\%$;

- (iii) One of the reasons COTS can destroy coral reefs in a short time is because COTS move quickly, allowing them to move from one reef to another.

Table 3.2 shows the maximum speeds recorded of COTS in constant light.

Table 3.2

response to light	maximum speed / mm min ⁻¹
COTS moving towards constant light	259
COTS moving away from constant light	564

Calculate the shortest time one COTS would take to move up a coral reef from 66 m under water to 18 m under water in hours of daylight.

Show your working. Give your answer to the nearest hour.

- 1. Distance = 66-18 = 48 m = 48000 mm ;**
2. Time = 48000 / 259 = 185 min = 3 hours ;

Allow 1 mark for distance of 48 000 mm in working

1 max for answer of 185 minutes/3 hours and 5 minutes/3.09 hours

1 max for answer of 1 hour (ie answers that use 564 in their calculation)

time = hours
 [2]

[Total: 11]

Section B

Answer **one** question in this section.

Write your answers to this question on the separate Answer Booklet 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)** The accuracy of DNA replication is critical to maintaining genomic integrity across cell divisions. Multiple molecular mechanisms operate during replication to ensure fidelity, yet errors still occur and can contribute to variation, adaptation, or disease.

Describe the roles of the key proteins and enzymes involved in DNA replication and explain how errors during replication can give rise to different mutations. [13]

1. Helicase causes the DNA molecule to unwind and unzip (at the origin of replication) and the hydrogen bonds between complementary bases break, causing the 2 parental DNA strands to separate ;
2. Single-stranded DNA binding proteins bind to the 2 separated DNA strands to stabilise the single-stranded DNA formed ;
3. so each strand at the unwound region can serve as a template for the synthesis of a new complementary strand ;
4. (extra pt) Topoisomerase helps to relieve the supercoiling in the DNA by breaking a phosphodiester bond of the strained strand, swivelling the strand and then rejoining the cut strand ;
5. Primase catalyses the formation of a short RNA primer (the start of a new strand in the 5' to 3' direction) ;
6. DNA polymerase then binds to the RNA primer and adds free DNA nucleotides to the free 3' –OH end of the RNA primer ;
7. DNA polymerase can only work in one direction from 5' to 3', and can only add nucleotides to the free 3' –OH end of an existing strand ;
8. DNA polymerase catalyses the formation of phosphodiester bonds between adjacent DNA nucleotides ;
9. RNA nucleotides of all the RNA primers are replaced with DNA nucleotides by another DNA polymerase ;
10. DNA ligase seals the gaps between the DNA fragments by catalysing the formation of phosphodiester bonds between adjacent nucleotides to form a continuous strand;
11. DNA polymerase has a proofreading function to identify and excise mismatched bases before reinserting the correct bases / nuclease, recognises and binds to faulty or damaged sequences and cuts out the faulty sequences, (to minimise errors during DNA replication) ;
12. replication error by DNA polymerase / nuclease, during DNA replication can cause gene mutations ;

13. base pair substitution, replacement of one nucleotide base pair with another base pair in the gene might occur ;
14. can result in missense mutation / nonsense mutation and results in significant change in the encoded protein ;
15. can result in silent mutation with no / little effect on the encoded protein ;
16. base pair addition – gain of one or more nucleotide base pairs in a gene ;
17. base pair deletion – loss of one or more nucleotide base pairs in a gene ;
18. leads to frameshift mutation and results in significant change in the encoded protein ;
19. Gene mutation can result in change in 3D conformation of protein and the formation of a non-functional protein ;

QWC: Proper paragraphing and at least two enzymes linked to 2 functions and at least 1 type of gene mutation explained

- (b) Bacteria are prokaryotes that do not undergo sexual reproduction but undergo binary fission to give genetically identical daughter cells. However, genetic variation exists in bacterial populations.

Describe how genetic variation arise in bacterial populations.

[12]

1. Genetic variation arise in bacterial populations through transformation, conjugation and transduction ;

Transformation

2. Transformation where naked (double stranded) DNA from the surrounding environment binds to the competence factors on the cell surface membrane of a competent cell ;
3. One strand is degraded by exonucleases, while the other strand enters the bacterium ;
4. The DNA taken up by the bacterium can be incorporated into its chromosome via homologous recombination by crossing over ;

Conjugation

5. Conjugation where F⁺ (donor) cell produces sex pilus to attach to specific receptors on the F⁻ (recipient) cell ;
6. Upon making contact, the sex pilus retracts, pulling the two cells closer ;
7. A temporary cytoplasmic mating bridge is formed between the F⁺ donor cell and the F⁻ recipient cell ;
8. A single strand of F plasmid breaks at the origin of transfer and is transferred as a single strand (beginning at its 5' end) from the F⁺ (donor) cell into the F⁻ (recipient) cell via the cytoplasmic mating bridge ;
9. After transfer of F plasmid, the complementary strands of F plasmid are synthesised in F⁻ (recipient) cells.

Transduction

10. Transduction where bacterial DNA is transferred from one bacterium to another via a bacteriophage ;
11. When a (virulent) bacteriophage undergoes lytic cycle, a small piece of the host bacterial cell's degraded DNA is (mistakenly) packaged within the capsid (of a defective phage) ;
12. When a (temperate) bacteriophage enters into lytic cycle from lysogenic cycle, a small region of the host bacterial DNA that was adjacent to the prophage is excised and the phage-host hybrid DNA is packaged within the capsid (of a defective phage) ;
13. The defective phage infects another bacterial cell and inject the piece of host bacterial DNA/ phage-host hybrid DNA into the newly infected bacterial cell cytoplasm ;
14. The host bacterial DNA/ phage-host hybrid DNA is incorporated into recipient bacterium's DNA via homologous recombination / crossing over with a homologous region ;

QWC – Proper paragraphing and answers include at least 2 types of genetic transfer ;

[Total: 25]

- 5 (a) The reproductive cycles of enveloped viruses involve intricate interactions with host membranes and often include mechanisms that promote rapid evolution.

Describe the roles of the key proteins and enzymes involved in the reproductive cycle of influenza virus and explain how variation in its viral genome arises. [13]

key proteins and enzymes

1. Haemagglutinin on the envelope of the influenza virus recognises and binds to the specific glycoprotein/ (sialic acid containing) receptors on the cell surface membrane of host cells/ cells of the respiratory tract.
2. Facilitates influenza virus entry into host cell by receptor-mediated endocytosis ;
3. Neuraminidase catalyses the cleavage of sialic acid residues from haemagglutinin;
4. Facilitates the exit and release of newly replicated viruses from the infected host cell by budding ;
5. Clathrin aids in the formation of clathrin-coated vesicles during receptor-mediated endocytosis, allowing influenza virus to enter the host cell ;
6. Cellular enzymes degraded the capsid during uncoating, releasing the viral nucleic acid / eight segments of single-stranded RNA, and RNA-dependent RNA polymerase into the cytoplasm ; .
7. RNA-dependent RNA polymerases catalyse the synthesis of a complementary RNA (cRNA) strand from ss RNA.
8. Capsid protein is assembled around the viral RNA and the RNA-dependent RNA polymerase during assembly on new virions ;
9. Variation in its viral genome arises via antigenic drift and antigenic shift ;

Antigenic drift

10. Point mutations in the genes that code for haemagglutinin in the influenza virus can occur during the replication of viral RNA ;
11. Accumulation of mutations results in slight changes to the shape of haemagglutinin ;
12. The change in shape in the new haemagglutinin may now enable it to be complementary in shape to other membrane receptors found on new types of host cells;
13. Resulting in the host immune system no longer recognising the antigenic sites on the haemagglutinin on the new strain of influenza virus / allows new strain of influenza virus to evade host immunity ;

Antigenic shift

14. Two or more different existing strains of influenza virus infect the same host cell simultaneously;
(A! Different strains)
15. Random assembly / genetic reassortment of RNA segments from these different strains of viruses leads to novel combinations of hemagglutinin and neuraminidase genes (to form a new strain);
16. (enabling the influenza virus to spread from one animal species to a species that it normally does not infect (e.g. avian flu infecting humans));

QWC: Proper paragraphing and at least two enzymes/proteins linked to 2 functions and at least 1 type of variation explained

- (b) Bacteria live in environments where the supply of nutrients may change rapidly. To survive, they regulate the expression of certain genes so that enzymes are produced only when required.

Two examples of gene regulation in bacteria are the *lac* operon and the *trp* operon in *E. coli*.

Discuss how these two operons regulate gene expression when there are high levels of lactose and tryptophan, and explain the advantages of such regulation to the bacteria. [12]

[Total: 25]

High levels of lactose

1. At high levels of lactose, some lactose enters the cell through the small amount of lac permease present in the plasma membrane ;
2. The lactose is converted to allolactose by β -galactosidase ;
3. Allolactose binds to the allosteric site of the lac repressor, the lac repressor changes its 3D conformation from active to inactive ;
4. The repressor protein is unable to bind to / dissociates from the operator, turning the operon ON ;
5. RNA polymerase is able to recognise and bind to the promoter and transcribe the structural genes of the *lac* operon. lac permease, β -galactosidase and lactose transacetylase are produced ;

High levels of tryptophan

6. At high levels of tryptophan, tryptophan (a co-repressor) binds to the *trp* repressor at its allosteric site ;
7. *trp* repressor changes from inactive to active conformation, and can recognise and bind to the operator. Thus, the *trp* operon is turned off ;
8. RNA polymerase is unable to bind to the promoter, and unable to transcribe the structural genes of the *trp* operon, mRNA is not synthesised ;
9. Enzymes that synthesise tryptophan are not produced, tryptophan no longer synthesised ;

Advantages

10. Operons allow for the simultaneous regulation of related genes with related functions / which are involved in the same metabolic activity ;
11. Genes coding for related proteins/enzymes in a single biochemical pathway / of related functions / in the same metabolic activity are grouped into a operon for easier control ;
12. Bacteria are able to utilise a variety of carbohydrates as respiratory substrate (for their energy requirement) ;
13. Operons allow the bacteria to adapt and respond to environmental changes ;
14. The genes on the operons are only expressed when required, thus preventing wastage of energy and resources / increasing efficiency in utilisation of energy and resources ;
15. Thus, the bacteria are at a selective advantage / This confers selective advantage to the bacteria ;

QWC: at least one point for lac operon, one point for trp operon, and one advantage of operon with proper paragraphing ;