

NAME: _____ CLASS: _____ INDEX: _____



CATHOLIC JUNIOR COLLEGE
JC2 PRELIMINARY EXAMINATION
Higher 2

Suggested Answers

BIOLOGY

Paper 3 Long Structured and Free-Response Questions

9744/03

16 September 2025
2 Hours

Candidates answer on the Question Paper.

No Additional Materials are required

READ THESE INSTRUCTIONS FIRST

Write your **name (as per NRIC)**, **class**, and **index number** on all the work you hand in.

Write in dark blue or black pen on both sides of the paper.

[PILOT FRIXION ERASABLE PENS ARE NOT ALLOWED]

You may use a soft pencil for any diagrams, graphs, or rough working.

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 in this section.

Write your answers in the writing 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	
Section A	50
1	
2	
3	
Section B	25
4 or 5	
Total	75

Section A

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

- 1** Based on the statistics by World Health Organisation (WHO), cancer is a leading cause of death worldwide, accounting for nearly 10 million deaths in 2020, or nearly one in six deaths.

Many causative factors, including chemical carcinogens may increase the chances of cancerous growth.

- (a)** Name one chemical carcinogen.

..... [1]

1. Benzo[a]pyrene / ethidium bromide / 5'-Bromouracil / AVP.

Studies showed that individuals inheriting *BRCA 1* gene that underwent loss-of-function mutation have an increased risk of developing breast and ovarian cancers.

- (b)** Based on the information above, identify the type of gene *BRCA 1* belongs to and justify your answer.

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..... [2]

1. Tumour suppressor genes
2. (The loss-of-function mutation of *BRCA 1* increases the risk of developing breast and ovarian cancers implies that) this gene codes for gene products that inhibit growth without the appropriate conditions (OWTTE).

Mutations in another gene, *BRCA 2*, was shown to increase the risk of developing breast and ovarian cancers. Fig. 1.1 shows the percentage risk of developing these cancers in general population, individuals with *BRCA 1* mutations, and individuals with *BRCA 2* mutations.

Percentage / %

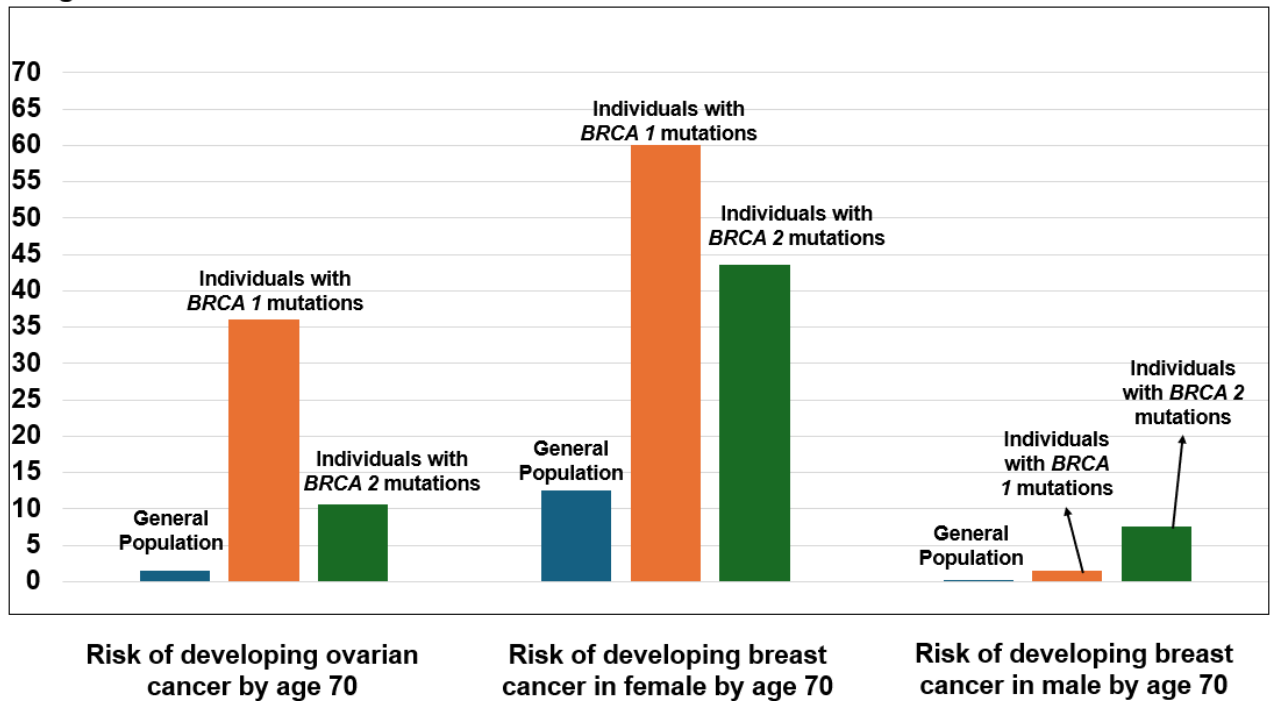


Fig. 1.1

(c) With reference to Fig. 1.1,

(i) describe the effect of *BRCA 1* mutations on the risk of developing ovarian cancer.

..... [1]

1. The risk of developing ovarian cancer in female by age 70 increases from 1.5% (accept: 1% - 1.5%) in general population to 36% (accept: 36% - 36.5%) in individuals with *BRCA 1* mutations

(ii) describe the effects of *BRCA 2* mutations on the risk of developing breast cancer.

..... [2]

1. The risk of developing breast cancer in female by age 70 increases from 12.5% (accept: 12% - 13%) in general population to 43.5% (accept: 43.5% - 44%) in individuals with *BRCA 2* mutations.

2. The risk of developing breast cancer in male by age 70 increases from 0.1% (accept 0.1% - 0.3%) in general population to 7.6% (accept: 7.5% - 8%) in individuals with *BRCA 2* mutations.

- (iii) compare the effects of *BRCA 1* mutations and *BRCA 2* mutations on the risk of developing breast cancer.

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..... [3]

1. The risk of developing breast cancer in female and male by age 70 increases in individuals with either *BRCA 1* or *BRCA 2* mutations as compared to the general population.
2. The risk of developing breast cancer in female by age 70 increases more in individuals with *BRCA 1* mutations than in individuals with *BRCA 2* mutations as compared to the general population, where the increase is 47.5% (accept: 47% - 48%) and 31% (accept: 30% - 32%) respectively.
3. The risk of developing breast cancer in male by age 70 increases more in individuals with *BRCA 2* mutations than in individuals with *BRCA 1* mutations than as compared to the general population, where the increase is 7.5% (accept 7.2% - 7.9%) and 1.4% (accept: 0.1% - 1.4%) respectively.

- (iv) suggest an explanation for the differences in the effects of *BRCA 1* mutations and *BRCA 2* mutations on the risk of developing breast cancer.

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..... [1]

Any of the following:

1. Differences in gene functions and protein activities.
2. *BRCA 1* may be more highly expressed in female or more critical for maintaining the genomic stability of female breast cells.
3. *BRCA 1* may influence hormone regulation differently than *BRCA 2*, affecting the breast environment and contributing to cancer risk.
4. AVPs

DNA replication is an important process prior to cell division.

Base-pairs mismatch may sometimes occur during DNA replication. This base-pair mismatch can often be rectified during the elongation stage by the proofreading activity of DNA polymerase.

Fig. 1.2 briefly illustrates the mechanism of proofreading activity in eukaryotic DNA polymerase δ .

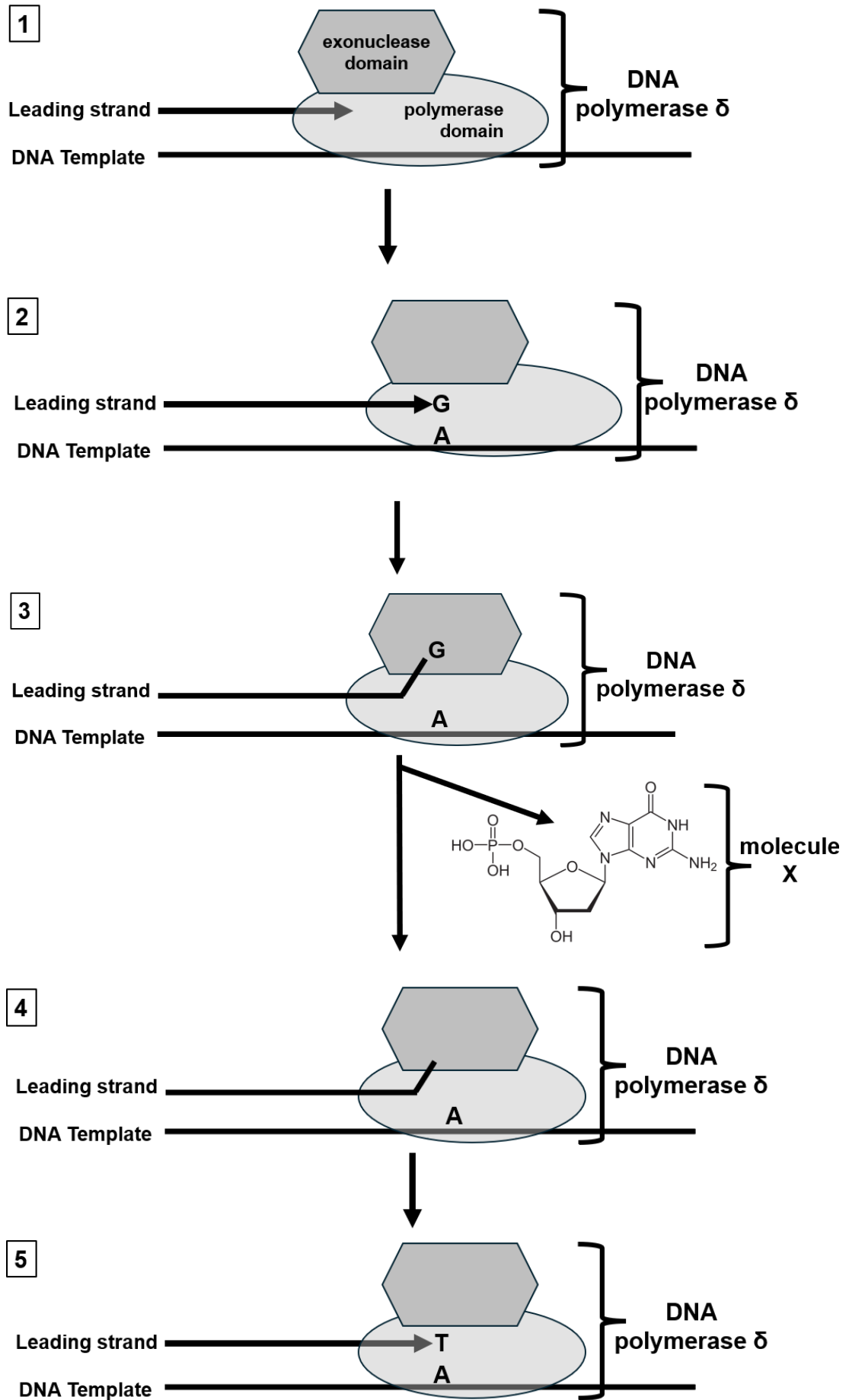


Fig. 1.2

(d) With reference to Fig. 1.2,

(i) identify molecule X.

..... [1]

1. dGMP / deoxyguanosine monophosphate / deoxyribonucleoside monophosphate with guanine base

(ii) describe the events that occurred to enable the correct base to be incorporated by DNA polymerase δ when there was a base-pair mismatch.

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 [5]

1. The (free 3') end of the growing daughter DNA strand / leading strand was moved **from polymerase domain into the exonuclease domain**.
2. At the **exonuclease domain**, the enzyme catalysed the **hydrolysis of phosphodiester bond** between **the leading strand** and the last deoxyguanosine monophosphate / deoxynucleoside monophosphate / molecule X.
3. **Molecule X / deoxyguanosine monophosphate** is released from the complex.
4. The (free 3') end of the growing daughter DNA strand / leading strand was moved back **from the exonuclease domain into polymerase domain**.
5. The **polymerase** catalyses the formation of **phosphodiester bond** between the correct **deoxythymidine triphosphate / nucleotide** with a **thymine** base and the **growing daughter DNA strand / leading strand** and through **complementary base pairing**.

(iii) suggest the directionality of the activity the exonuclease domain on the leading strand. Justify your answer.

.....

 [2]

1. **3' \rightarrow 5' direction**
2. The **polymerase domain** synthesises the DNA in **5' \rightarrow 3' direction**. Therefore, the end of the leading strand with G nucleotide must be 3' end.

If the proofreading activity of DNA polymerase and other DNA repair mechanisms fail to rectify errors during DNA replication, mutations occur.

Mutation in the Kirsten rat sarcoma (*K-Ras*) proto-oncogene is commonly found in patients with colorectal cancer.

(e) Mutation in *K-Ras* gene is not passed down to the offsprings. Name this type of mutation.

..... [1]

1. Somatic mutation.

In one of the studies, the resulting mutant K-Ras protein was purified from colorectal cancer cells and crystallography was performed to determine the structure of the protein.

Fig. 1.3 shows the structure of the mutant K-Ras protein in the presence and absence of growth factors.

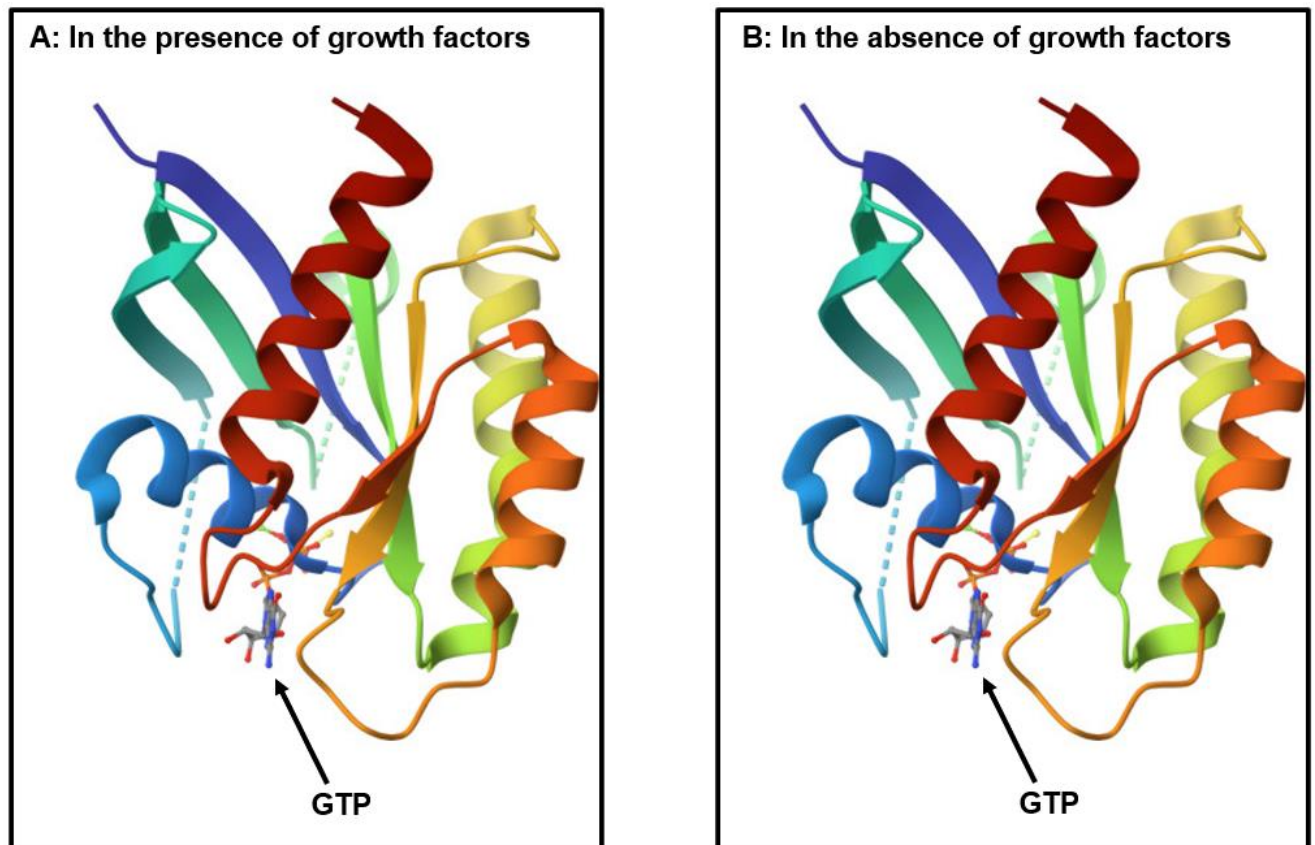


Fig. 1.3

(f) With reference Fig. 1.3, identify the level of protein structure of K-Ras protein.

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 [3]

1. Tertiary structure / level.
2. It comprises α -helices and β -pleated sheets and is further coiled and folded extensively forming a compact globular structure / a specific 3-D conformation.
3. It is composed of a single polypeptide chain which can be inferred from the presence of only 2 ends which will be the N-terminal and C-terminal.

(g) Based on the information provided above and Fig. 1.3, suggest how mutation in *K-Ras* gene contributes to the development of colorectal cancer.

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..... [4]

1. *K-Ras* gene undergoes **gain-in-function mutation** which results in a hyperactive mutant *K-Ras* protein.
2. Mutant *K-Ras* protein binds to **GTP** constitutively / continuously **even in the absence of growth factor.**
3. due to the **non-functional GTPase.**
4. Mutant *K-Ras* protein activates cell signalling pathway that promotes uncontrolled cell division / proliferation.

Cancer cells and stem cells are similar in many ways. One of the similarities is their abilities to maintain the length of telomere despite multiple rounds of successive DNA replication.

(h) Apart from their ability to maintain the length of telomere, compare the features of cancer cells and embryonic stem cells.

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..... [2]

1. Similarity: both types of cells are able proliferate indefinitely.
2. Difference: cancer cells do not have the ability to differentiate into almost all the different cell types whereas embryonic stem cells are pluripotent cells that have the ability to differentiate into almost all types of cells except cells of the extra-embryonic membranes.

- (i) “Telomerase prevents end-of-replication problem in both cancer and stem cells”.

Comment on the accuracy of the statement.

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..... [3]

1. The statement is **inaccurate**, because the **end-of-replication problem** still occurs in cancer and stem cells.
2. **Telomerase** catalyses the **lengthening of telomeres** at the **ends of linear DNA**, thus restoring the original lengths and compensating
3. Telomerase does not overcome the fact that DNA polymerase requiring 3'-OH end for DNA replication

[Total: 31]

- 2 Polymerase Chain Reaction (PCR) possesses the potential to generate billions of copies of target DNA from a single copy.

One of the requirements of this method is having information on at least partial sequences of the target DNA, which is needed to design primers that hybridise specifically to the target sequences.

DNA primers are often 18-30 bases in length.

- (a) Suggest why the length of the primers cannot be too short or too long.

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..... [2]

1. **Primers** that are **too short** may lack **specificity**, leading to non-specific binding and amplification of wrong sequences.
2. **Primers** that are **too long** may reduce efficiency by slowing the annealing step.

COVID-19 is caused by a positive-sense, single-stranded RNA virus called SARS-CoV-2.

During the COVID-19 pandemic, a modified PCR technique called Reverse Transcription quantitative Polymerase Chain Reaction (RT-qPCR) became a cornerstone for diagnosing the infection.

In RT-qPCR, the genetic material of SARS-CoV-2 RNA is first converted into double stranded DNA.

(b) Describe how double stranded DNA is obtained from SARS-CoV-2 RNA.

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..... [4]

1. **DNA Primers** bind **SARS-CoV-2 RNA** through **complementary base pairings**.
2. **Reverse transcriptase** attaches to the DNA primers and begins adding DNA nucleotides / deoxyribonucleoside triphosphates that are **complementary** to the **RNA template** to the **free 3'- OH of the DNA primers** by catalysing **phosphodiester bonds**.
3. **RNase (H)** digests the RNA sequence.
4. **DNA polymerase** attaches to the DNA primers and begins adding DNA nucleotides / deoxyribonucleoside triphosphates that are **complementary** to the **DNA template** to the **free 3'- OH of the DNA primers** by catalysing **phosphodiester bonds**.

After conversion into double stranded DNA, the amplification process is monitored with the aid of using a DNA probe attached to a fluorescent dye.

Figure 2.1. illustrates how fluorescence is released during the amplification in RT-qPCR.

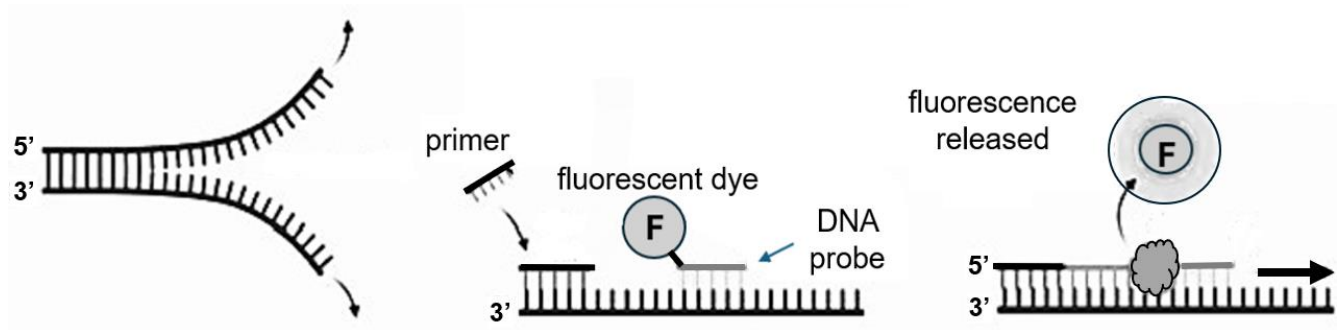


Fig. 2.1

(c) With reference to Fig. 2.1, explain how fluorescence is released during RT-qPCR.

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..... [3]

1. **Denaturation:** The double-stranded DNA (dsDNA) template is heated to **break hydrogen bonds** between complementary strands. This separates the DNA into **two single strands**, creating templates for replication.
2. **Annealing** of DNA **primers** and **probes** bind to **complementary sequences** on the single-stranded DNA templates.
3. Extension by **DNA polymerase** leads to **cleaving/hydrolysis/OWTTE** of DNA probes, **releasing** the fluorescent signal.

During the amplification process, the fluorescence intensity is quantified real time using a spectrophotometer.

Fig. 2.2 shows the graph of fluorescence against the number of cycles of amplification.

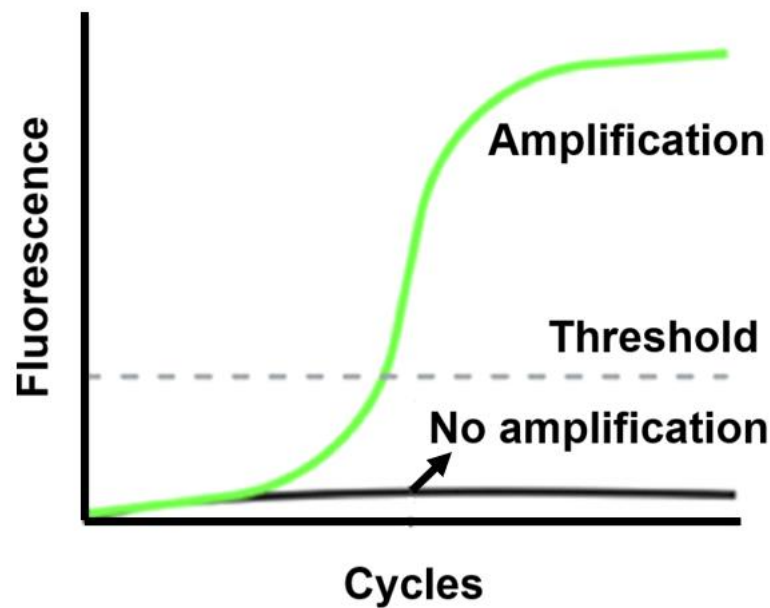


Fig. 2.2

The number of PCR cycles for fluorescence intensity to reach or exceed threshold depends on the concentration of SARS-CoV-2 in the sample.

(d) With reference to Fig. 2.2,

- (i) Suggest how RT-qPCR can distinguish patients with high SARS-CoV-2 viral load from patients with low SARS-CoV-2 viral load.

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 [1]

1. The higher the viral load, the smaller the number of cycles required to reach threshold, or vice versa.

- (ii) Suggest why there is low fluorescence intensity despite no amplification.

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 [1]

1. The fluorescent dye is cleaved spontaneously.

[Total: 11]

- 3 North America is home to diverse migratory bird species that travel long distances between their breeding grounds in the north and wintering grounds in the south. One such migratory bird species is the American robin. During the winter season, the robins migrate from the northern region of Canada to the southern region to seek for their food source – caterpillars.

Over the years, climate change has impacted the population of American robins, their migration pattern, and possibly their survival.

Fig. 3.1 below shows the average global temperature over the years.

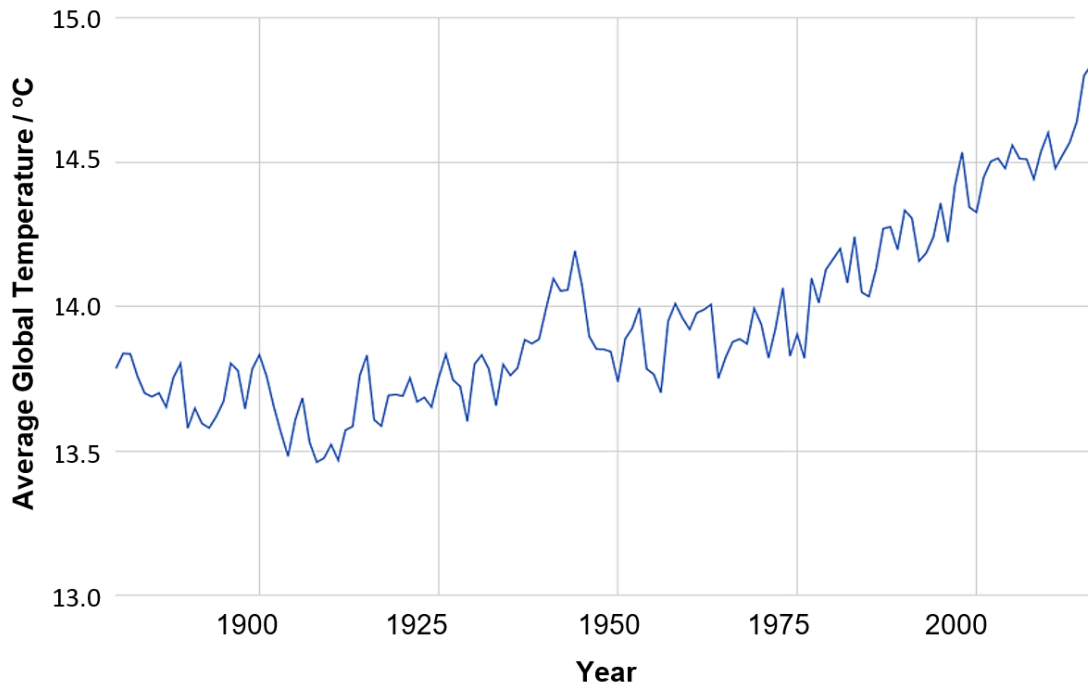


Fig. 3.1

Fig. 3.2 shows the migration period of the robins from north Canada to south Canada in 1950 and 2000.

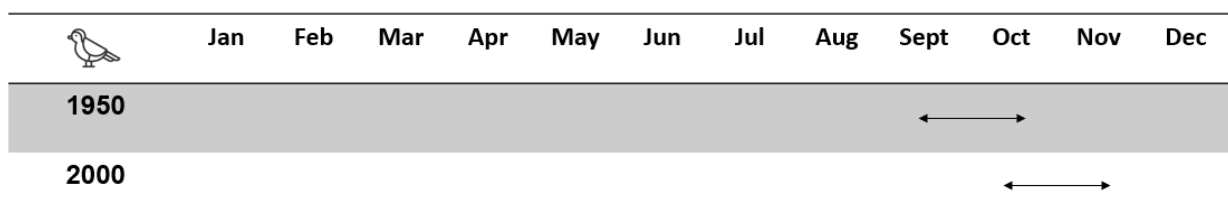


Fig. 3.2

Fig. 3.3 shows the period where there is great abundance of caterpillars in south Canada in 1950 and 2000.

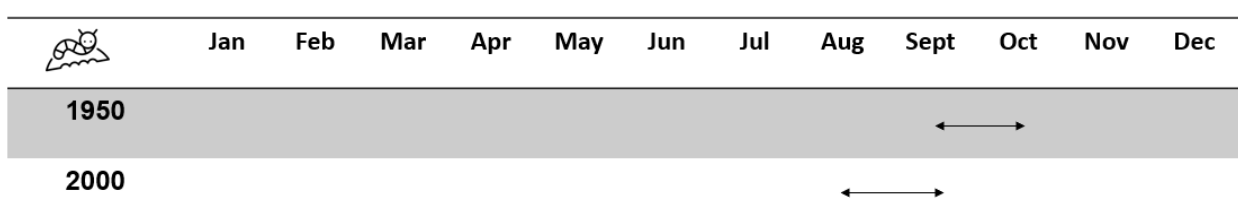


Fig 3.3

Climate change has been said to affect the migratory phenology of the American robins.

- (a) With reference to Fig. 3.1 and 3.2, describe and explain how climate change has caused the change in the migration pattern.

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..... [3]

1. From 1950-2000 there was an increase in the average global temperature, from 13.8 °C to 14.8°C due to global warming.
2. The migration of the robins was delayed by about 1 month from September to October in 1950 to October to November in 2000.
3. Global warming resulted in warmer winters or delayed winters, during which food resources remain available and this might have postponed the robin's migration / departure from southern Canada.

- (b) Explain why the caterpillars are found in abundance earlier as shown in Fig. 3.3.

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..... [2]

1. The increase in global temperature can increase enzymatic activity / the metabolic rate of the insect that speeds up developmental time.
2. This can result in the shortening of life cycle of the insect, resulting in caterpillars emerging from the egg much sooner, from September to October in 1950 to August to September in 2000.
3. The rise in global temperatures may also cause plants to produce new leaves sooner. This provides food for caterpillars earlier in the season, leading to their earlier abundance.

- (c) Discuss how the changes shown in Fig. 3.2 and Fig. 3.3 can impact the American robins.

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..... [3]

1. Global warming causes caterpillars to become abundant earlier, while robins migrate later, leading to a mismatch in timing.
2. By the time the robins arrive in southern Canada, their main food source is already depleted as the caterpillars might have pupated and/or metamorphosed into adult butterflies, reducing the robins' chances of survival and successful breeding.
3. As a result, the robins may be forced to fly further south or rely on alternative food sources to survive.

[Total: 8]

Section B

Answer **one** question in this section.

Write your answers on the lined paper provided at the end of this Question Paper.

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

Your answer must be in continuous prose, where appropriate.

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

- 4 (a) Describe how transformation, transduction and conjugation give rise to variation in prokaryotic genomes and explain why genetic variation is important in bacteria. [15]

Transformation

1. A (competent) bacterium takes up foreign DNA ;
2. The foreign DNA is incorporated into bacterium's chromosome via **homologous recombination** ;
3. By crossing over where there is sufficient **homology** between the DNA fragments and bacterial chromosome ;
4. Segments of the bacterium's genome are replaced ;

Transduction

5. Bacterial DNA / genes are transferred from one bacterium to another via a bacteriophage ;
6. 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 ;
7. Defective phage infects another bacterial cell and injects the piece of host bacterial DNA into the newly infected bacterial cell cytoplasm ;
8. 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 improperly excised and the phage-host hybrid DNA is packaged within the capsid (of a defective phage);
9. The defective phage infects another bacterial cell and injects the phage-host hybrid DNA into the newly infected bacterial cell cytoplasm ;
10. The host bacterial DNA / phage-host hybrid DNA is incorporated into recipient bacterium's genome / DNA via **homologous recombination** ;

Conjugation

11. **F⁺ donor** cell produces **sex pilus** to attach to **F⁻ recipient** cell ;
12. Sex pilus retracts upon contact, pulling the two cells closer and forming a temporary cytoplasmic mating bridge between F⁺ donor cell and F⁻ recipient cell ;
13. A single strand of F plasmid breaks at a specific point (origin of transfer) and the F plasmid is transferred as a single strand ;
14. From F⁺ donor cell into the F⁻ recipient cell via the cytoplasmic mating bridge ;
15. Each single stranded DNA in each respective cell (donor and recipient cells) now acts as a **template** for the synthesis of a complementary daughter strand; ref. to rolling circle mechanism
16. F⁻ recipient cell (without F plasmid) is now a F⁺ cell ;

[Max: 13]

Why genetic variation is important

17. Genetic variation is important in bacteria so that bacteria may acquire new alleles / genes ;
18. That confer selective advantage under a particular selection pressure (e.g. antibiotic resistance is a selective advantage, in the presence of antibiotics) ;
19. Bacteria with selective advantage are selected for and reproduce (via **binary fission**) to pass on favourable alleles to subsequent generations ;

[Max 14]

QWC [1]

Clearly expressed and well structured, with all 4 parts of question addressed using correct terminology.

- (b) Microorganisms such as *Escherichia coli* (*E. coli*) colonise the intestine and obtain nutrients from their surroundings.

Describe how *E. coli* responds to the presence of lactose and absence of glucose in the intestine and explain how a mutation in the regulatory sequence of the *lac* operon may affect how *E. coli* responds to changes in lactose supply. [10]

How *E. coli* responds to changes [Max 6]

Presence of lactose

- 1 Lactose **isomerizes** to form allolactose; ref. allolactose as an inducer;
- 2 allolactose binds to the (allosteric site of) the lac repressor, and **alters its 3D conformation** (at the DNA-binding site) - lac repressor become **inactive** and is no longer able to bind to the operator (lac O);
- 3 RNA polymerase can **access and transcribe** the **structural genes**;
- 4 ref. lacZ , lacY and lacA genes being transcribed and translated into β -galactosidase, lac permease and lactose transacetylase enzymes respectively;

[Absence of glucose]

- 5 **cAMP concentration increases** and binds to (allosteric site of) catabolite activator protein (CAP) site - alters its **3D conformation** of the (DNA-binding site of) CAP;
- 6 CAP is **active** and binds to the CAP-binding site;
- 7 ref. facilitates more **efficient positioning of RNA polymerase** at the promoter, resulting in **high rate of transcription** of lac operon;

Effect of mutation affecting the regulatory sequences [Max 4]

Any one of the following:

Regulatory Sequence: lac promoter

- 8 Mutation of the promoter results in a change in the sequence / structure / shape of the promoter;

Mutation of lac promoter - increases binding affinity)

- 9 ref. RNA polymerase able to **bind** to the (mutated) promoter with **greater affinity**; [Reject: permanently/ irreversibly]
- 10 ... hence **increase the rate of the transcription** of structural genes **in the presence of lactose**;

OR

Mutation of lac promoter - decrease binding affinity

- 11 ref. **3D shape/conformation** of the promoter is **no longer complementary** to the (active site) of RNA polymerase / RNA polymerase **no longer able to bind** to the (mutated) promoter;
- 12 ref. **No transcription** of structural genes **even in the presence of lactose**;

Regulatory Sequence: Operator

- 13 Mutation of the operator results in a change in the sequence/structure/shape of the operator;
- 14 ref. repressor binds to the operator **permanently/irreversibly**;

15 ref. **No transcription** of structural genes **even in the presence of lactose**;

OR

16 ref. **3D shape/conformation** of the operator is **no longer complementary** to the of the **repressor** / (active) repressor can **no longer bind** to the operator;

17 ref. **RNA polymerase** is now able to **access / bind** to **promoter**, **allowing transcription** of structural genes **even in the absence of lactose**.

QWC [1]

Accurate communication of the response to lactose and absence of glucose in *E.coli*, and to include at least one mutation in either promoter and operator

5 (a) Explain the role of the different coenzymes in respiration. [15]

1. **NAD⁺ (Nicotinamide Adenine Dinucleotide)**

2. Acts as an **electron carrier** by accepting **2 electrons + 1 proton (H⁺)**, reducing to **NADH**.

3. Delivers electrons to the **electron transport chain (ETC)** for oxidative phosphorylation.

Key Reactions:

4. **Glycolysis**: $\text{NAD}^+ \rightarrow \text{NADH}$ (oxidation of glucose).

5. **Pyruvate Oxidation**: $\text{NAD}^+ \rightarrow \text{NADH}$

6. **Krebs Cycle**: 3 steps generate NADH (isocitrate \rightarrow α -KG, α -KG \rightarrow succinyl-CoA, malate \rightarrow oxaloacetate), any one example.

ATP Yield:

7. **1 NADH \approx 2.5 ATP** (via ETC chemiosmosis).

FAD (Flavin Adenine Dinucleotide)

8. Accepts **2 electrons + 2 protons (H⁺)**, reducing to **FADH₂**.

9. Unlike NADH, FADH₂ enters ETC at **Complex II**, yielding less ATP.

Key Reactions:

10. **Krebs Cycle**: $\text{FAD} \rightarrow \text{FADH}_2$ (succinate \rightarrow fumarate, catalyzed by succinate dehydrogenase).

ATP Yield:

11. **1 FADH₂ \approx 1.5 ATP** (due to lower energy electrons).

12. **Acetyl-CoA**

13. Links glycolysis and the Krebs Cycle.

14. Carries **2-carbon acetyl groups** for oxidation in the Krebs Cycle.

15. **Pyruvate Oxidation**: Pyruvate \rightarrow Acetyl-CoA (+ CO₂ + NADH).

16. **Krebs Cycle**: Acetyl-CoA + oxaloacetate \rightarrow citrate.

17. **Succinyl-CoA**

18. **Substrate-level phosphorylation**: Converts GDP \rightarrow GTP (\approx ATP) in the Krebs Cycle.

19. Intermediate in heme synthesis and amino acid metabolism.

20. **Krebs Cycle**: α -KG \rightarrow succinyl-CoA (+ NADH + CO₂) \rightarrow succinate (+ GTP).

21. **1 mark for QWC (At least 3 types of coenzymes)** [1 mark Compulsory]

- (b) Cyanide is a rapidly acting and lethal toxin that disrupts cellular respiration, leading to severe oxygen deprivation at the cellular level. It irreversibly binds to the iron found in haemoglobin and the enzymes in the electron transport chain.

Explain the effect of cyanide on respiration and suggest possible treatments.

[10]

Effects of Cyanide [Max 8 marks]

1. **Prevents Chemosis:**
2. This halts ATP production by preventing the transfer of electrons in the ETC,
3. No proton gradient established;
4. Anaerobic respiration
5. Lactic Acidosis: Cells switch to anaerobic respiration, causing a dangerous buildup of lactic acid
6. **Prevents oxidative phosphorylation.**
7. **Deplete oxygen transport** ability of red blood cell
8. Binds to haem group of haemoglobin, resulting in oxygen not able to bind.
9. No final electron acceptor to remove electrons from the ETC
10. NADH and FADH cannot be reoxidised to continue with kreb cycle and link reaction.
11. Suffocating and killing cells despite adequate inhaling oxygen levels.

Proposed Treatment [Max 2 marks]

12. Binds cyanide to form complexes so as to lower cyanide concentration in body .
13. **100% Oxygen:** increases the chance of oxygen binding to haemoglobin.
14. **Fluids** For shock or hypotension.
15. **Alkaline** for example: Sodium Bicarbonate: Corrects lactic acid induced acidity.
16. **QWC effects and treatment** [1 mark Compulsory]

[Total: 25]

END OF PAPER

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