Suggested Answers

NAME: _____ CLASS: _____ INDEX: _____

CATHOLIC JUNIOR COLLEGE
JC2 PRELIMINARY EXAMINATION
Higher 2

BIOLOGY
Paper 3 Long Structured and Free-Response Questions

9744/03
13 September 2023
2 Hours

Candidates answer on the Question Paper.

Additional Material: Writing Booklet.

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 Endosymbiont is a cell which lives inside another cell with mutual benefit.

The organelle in Fig. 1.1 is believed to have evolved from the early prokaryote that was engulfed by phagocytosis.

The engulfed prokaryotic cell remained undigested as it contributed new functionality to the engulfing cell. Over generations, the engulfed prokaryotic cell lost some of its independent utility and became a supplemental organelle.

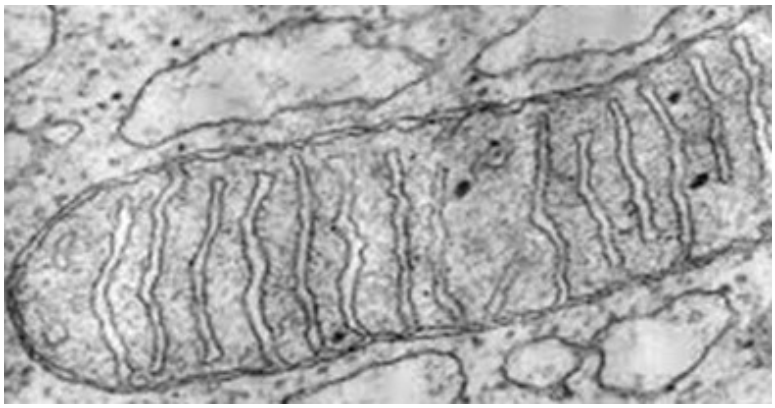

Fig 1.1

(a) Explain how the organelle shown above supports the endosymbiotic theory.

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…… [2]

1. **Circular DNA,** which is different from **nuclear linear DNA**.
2. **70s ribosome** as compared to **80s ribosome.**
3. **Double membrane structure**, the **outer membrane was part of the host cell**.

(b) Explain how the organelle is adapted to its function.

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…… [3]

1. The **inner membrane of mitochondria is highly folded**, forming structures called cristae. These cristae provide a significantly **increased surface area** for various enzymes involved in the **electron transport chain** and **ATP synthesis**.
2. Mitochondria have their own **DNA**, many of which are involved in oxidative phosphorylation and other mitochondrial functions.
3. The **double membrane** structure allows for compartmentalisation of the **proton gradient.**

Scientists use the DNA of the organelle shown in Fig. 1.1 for phylogenetic analysis.

Analysis of DNA has been used extensively to study the evolutionary relationships across many species.

(c) Explain the advantages of using the DNA of the organelle shown in Fig. 1.1 for phylogenetic analysis as compared to using nuclear DNA.

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1. Mitochondrial DNA tends to **mutate at a faster rate** compared to nuclear DNA. for **distinguishing between closely related species**.
2. Mitochondrial DNA does not undergo **recombination, avoids the complications of recombination and genetic shuffling** that can occur with nuclear DNA / Mitochondrial genome is **maternally inherited**, the **sequence ambiguities from heterozygous genotypes** are theoretically **avoided**.
3. Each cell contains **multiple copies of mitochondrial DNA**, making it relatively easy to obtain sufficient genetic material for analysis / **Higher copy number**, therefore, **greater abundance in sample extracts**.
4. Certain regions or genes within the mitochondrial genome are **relatively conserved** across species. For **ease of comparison**.

The DNA of the organelle shown in Fig. 1.1 is more susceptible to oxidation than nuclear DNA, possibly because of its proximity to the electron transport chain in its inner membrane. As a result, the rate of mutation becomes much higher.

(d) Suggest why the DNA of the organelle shown in Fig. 1.1 has a high mutation rate.

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…… [1]

1. **High concentration of oxyge**n in mitochondria induces the mutation.
2. **AVP**

Part of the sequence of the template DNA strand from 2 gene loci that code for different transport proteins in the organelle in Fig. 1.1 were shown in Fig. 1.2 below.

In addition, Fig. 1.2 shows the corresponding sequences containing the mutation, mutation **A** and mutation **B** respectively.

Wild-type sequence at gene locus 1	3' – CTT AGA CTT ACT – 5'
Sequence containing mutation **A**	3' – CTT AGT ACT TAC – 5'
Wild-type sequence at gene locus 2	3' – CTC CTA AAA CCT – 5'
Sequence containing mutation **B**	3' – CTC CCA AAA CCT – 5'

Fig. 1.2

(e) With reference to Fig. 1.2, state which mutation results in a frameshift.

..... [1]

1. Mutation **A**

Fig. 1.3 below shows the triplet codes that code for the different amino acids.

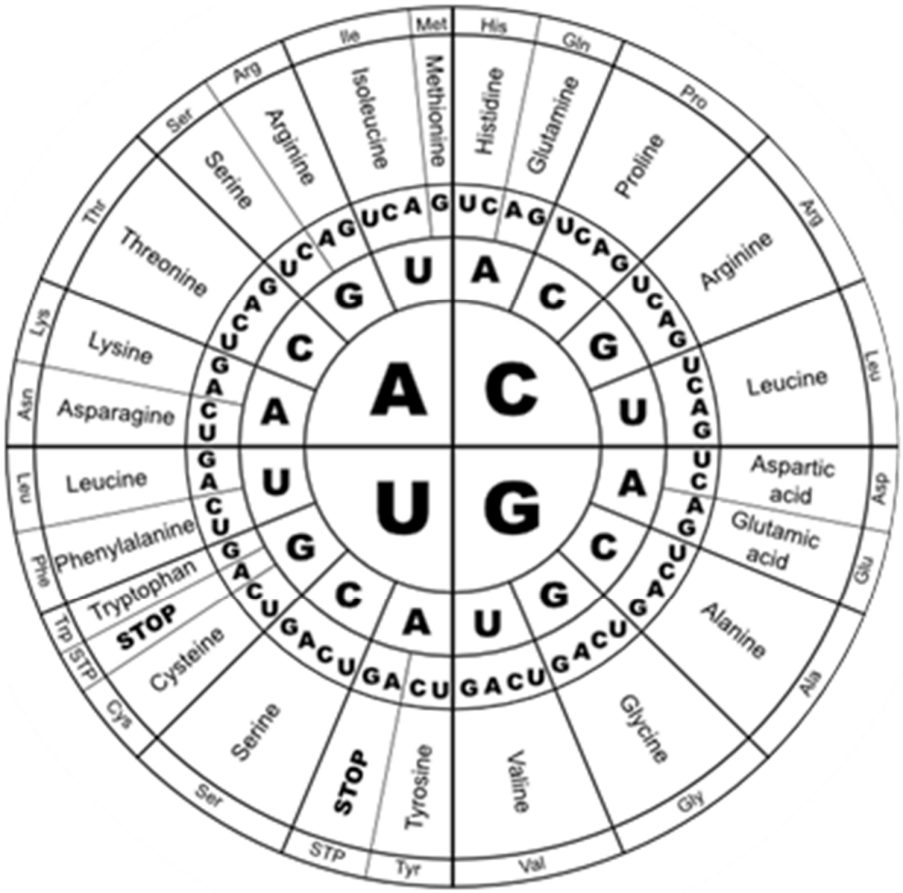

Fig. 1.3

It was found that mutation **A** produced a fully functional transport protein, while mutation **B** led to the production of a non-functional transport protein.

(f) With reference to Fig. 1.2 and Fig. 1.3,

(i) suggest how mutation **A** could still lead to the production of a fully functional transport protein. Explain your answer.

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1. Ref. to only a **loss of one amino acid** – **glutamic acid** (the 3rd codon is changed from glutamic acid to **STOP** codon)
2. Frameshift mutation (single base insertion) occurred near **the end** of the coding sequence. The change **not affecting the different / crucial** amino acid residue (i.e. binding)
3. This led to only a **small / insignificant change** in primary structure of protein at the C-terminus.

(ii) suggest how mutation **B** led to the production of a non-functional transport protein. Explain your answer.

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1. **Single base substitution mutation** led to change in the amino acid **aspartic acid to glycine**.
2. The **R group** changes from **negatively charged to non-polar**, the **initial bonds / interaction is disrupted**.
3. This amino acid position is **crucial** in **maintaining the 3-D conformation** of the transport protein.

Different respiratory substrates are available to muscles to maintain the ATP levels required for muscle contraction. These include glucose and fatty acids from the blood as well as glycogen in the muscle.

Table 1.1 shows the percentage contribution of each of these respiratory substrates to muscle respiration in an athlete, during a 40 km long-distance run. Results are shown at four different times from the start of the run.

Table 1.1

Time / Minutes	Percentage Contribution to Muscle Respiration		
	Glucose from blood	Fatty acids from blood	Glycogen in muscle
60	67	17	26
120	46	22	32
180	16	30	54
240	10	62	28

(g) With reference to Table 1.1., explain the change in percentage contribution of the different respiratory substrates to muscle respiration.

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…… [3]

1. **Glucose utilised readily at the start** as they were **readily available in the blood**.
2. **Depletion of glucose** in the blood **by 180 mins**, leading to **higher utilisation of glycogen** in the **muscle** to release more glucose.
3. **By 240 minutes**, **most glucose in the blood and glycogen are depleted / exhausted**, leading to **high contribution from fatty acid**.

(h) Suggest why fatty acids can also be utiilised for cellular respiration.

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…… [1]

1. Idea of: Fatty acids are oxidised to form acety-coA via beta-oxidation to be used for Krebs Cycle.

An in-vitro study showed that a complete oxidation of 1 molecule of glucose produces 2880 units of energy.

It was found that the hydrolysis of 1 molecule of ATP generates 31 units of energy.

(i) Based on the information above and your background knowledge,

(i) calculate the percentage efficiency of energy production of anaerobic respiration in the cell as compared to the complete oxidation of glucose in-vitro. Show your working clearly. [2]

Per glucose molecule

ATP for anaerobic = 2 ATP – [1]

[(2 X 31) / 2880] X 100% = 3% – [1]

(ii) calculate the percentage efficiency of energy production of aerobic respiration in the cell as compared to the complete oxidation of glucose in-vitro. Show your working clearly. [2]

Per glucose molecule

10 NADH X 3 + 2 $FADH_2$ X 2 + 2 ATP
Calculation of ATP production for aerobic = 38ATP – [1]

[(38 X 31) / 2880] X 100% = 57% – [1]

Note: accept any calculations using 1 NADH = 2.5 ATP and 1 $FADH_2$ = 1.5 ATP.

(iii) Suggest why the amount of energy generated by the aerobic respiration in the cell is different from the complete oxidation of 1 molecule of glucose in-vitro.

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…….. [2]

1. Glucose may not be fully oxidized to carbon dioxide and water. Instead, various metabolic intermediates are formed.
2. Transporting of ATP to the site of metabolism, which utilises energy.
3. Energy lost as heat during the different stages of respiration.

Photosynthesis and respiration are cellular processes that are commonly compared.

(j) Contrast between Krebs cycle and Calvin cycle.

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…… [4]

	feature	Krebs cycle	Calvin cycle
1.	role of carbon dioxide	released by oxidative decarboxylation; (4 CO_2 released per glucose molecule)	used in carbon fixation; (CO_2 fixed by combining with RuBP)
2.	type of reaction involved	involves oxidation by dehydrogenation; (e.g. citrate undergoes oxidative decarboxylation to form α-ketoglutarate)	involves reduction; (e.g. GBP is reduced to triose phosphate;
3.	fate of coenzyme / hydrogen atom carrier	NADH / $FADH_2$ is formed; (NAD is reduced to NADH / FAD is reduced to $FADH_2$)	NADPH is used; (NADPH is oxidised to NADP)
4.	ATP	ATP is synthesised (via substrate level phosphorylation);	ATP is used; (in the phosphorylation of GP to GBP and in regeneration of RuBP)
5.	starting point	oxaloacetate is regenerated;	ribulose bisphosphate is regenerated;
6.	nature of reaction	catabolic reaction;	anabolic reaction; (formation of triose phosphate)

[Total: 30]

2 Tuberculosis (TB) is an infectious disease caused by *Mycobacterium tuberculosis*. This pathogen primarily infects the lungs, leading to pulmonary TB.

Fig. 2.1 below briefly illustrates the life cycle of *M. tuberculosis* in infected individuals.

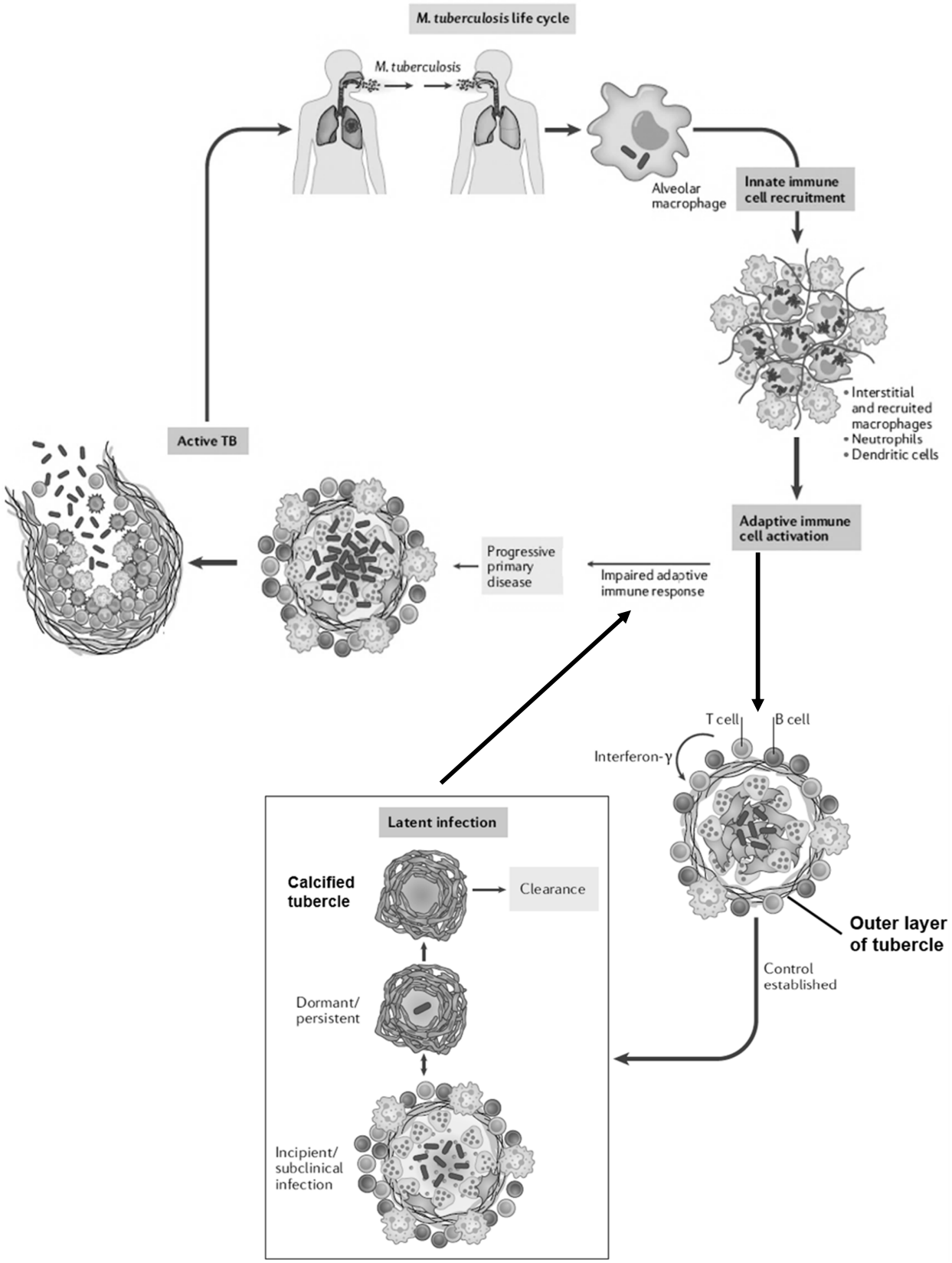

Fig. 2.1

With reference to Fig. 2.1 and your background knowledge,

(a) describe how *M. tuberculosis* could be transmitted from person to person.

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…… [2]

1. Via fine, **aerosol droplets**
2. When an infected person with the **active** (pulmonary) **TB** disease sneezes or coughs and an uninfected person inhales the droplet.

(b) explain how a tubercle is formed upon entry of *M. tuberculosis* into the lung.

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1. Once inside the lungs, **alveolar macrophages** phagocytose *M. tuberculosis* and form **phagosome** containing the bacteria.
2. Inside the phagosome, *M. tuberculosis* **inhibits the fusion of phagosome** with lysosomes. **No phagolysosome** is formed and no lysosomal enzymes are available to kill the bacteria
3. *M. tuberculosis* survives and continue to **multiply inside the macrophages**.
4. **More and more innate immune cells** such as neutrophils and dendritic cells are recruited to destroy *M. tuberculosis*
5. leading to the formation of a **tight ball-like structure** by **clustering of cells** (tubercle) where the **necrosis** occur at the **centre** of the structure.

(c) explain why individuals suffering Acquired Immunodeficiency Syndrome (AIDS) are more likely to develop active pulmonary TB.

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1. Individuals suffering from AIDS, have **low $CD4^+$ T cell count / impaired function of Helper T cells.**
2. $CD8^+$ T cells and B cells **cannot be activated** into **cytotoxic T cells** and **plasma cells**.
3. The **tubercle cavity enlarges** to form an air-filled cavity to multiply outside the macrophages.
4. **Tubercle ruptures**, allowing *M. tuberculosis* to spill into a bronchiole and **spread throughout the lungs**.

The treatment of active TB involves daily treatment with a combination of at least two antibiotics for about 6 months.

(d) Suggest why a combination of antibiotics is used for the treatment of active TB.

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…… [1]

1. **minimise the risk of developing resistance to the antibiotics**, and also to achieve an **additive effect** against the bacteria.

Isoniazid and rifampin are two antibiotics commonly used to treat active TB.
Isoniazid inhibits synthesis of cell wall while rifampin inhibits RNA synthesis during transcription.

(e) Suggest why isoniazid and rifampin are lethal to *M. tuberculosis* but not mammalian cells.

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…… [2]

(b) Mammalian cells are **eukaryotic cells** without a **cell wall**;
(c) **RNA polymerase** in mammalian cells is **different** from the prokaryotic RNA polymerase.

[Total: 13]

3 **(a)** Explain what you understand by 'coral bleaching'.

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1. Coral bleaching is the process when corals become white and this occurs when coral polyps expel the zooxanthellae that live inside their tissues.

2. Increased sea water temperature / thermal stress disrupts enzyme activities and metabolism of zooxanthellae and the heat-stressed zooxanthellae releases toxic compounds to corals, leading to their expulsion from the corals.
3. The lack of organic nutrient sources from the zooxanthellae eventually leads to death of coral tissues.

Rising seawater temperatures are contributing to coral bleaching, with mass coral bleaching events projected to increase in both frequency and severity.

Fig. 3.1 shows the impact of thermal bleaching stress on stored protein and carbohydrate reserves and reproductive output of two categories of corals five months after the bleaching event – corals that were resistant to bleaching events and corals that were bleached but later recovered.

For both types of corals, zooxanthellae was able to recolonise them and both types of corals also looked visually healthy.

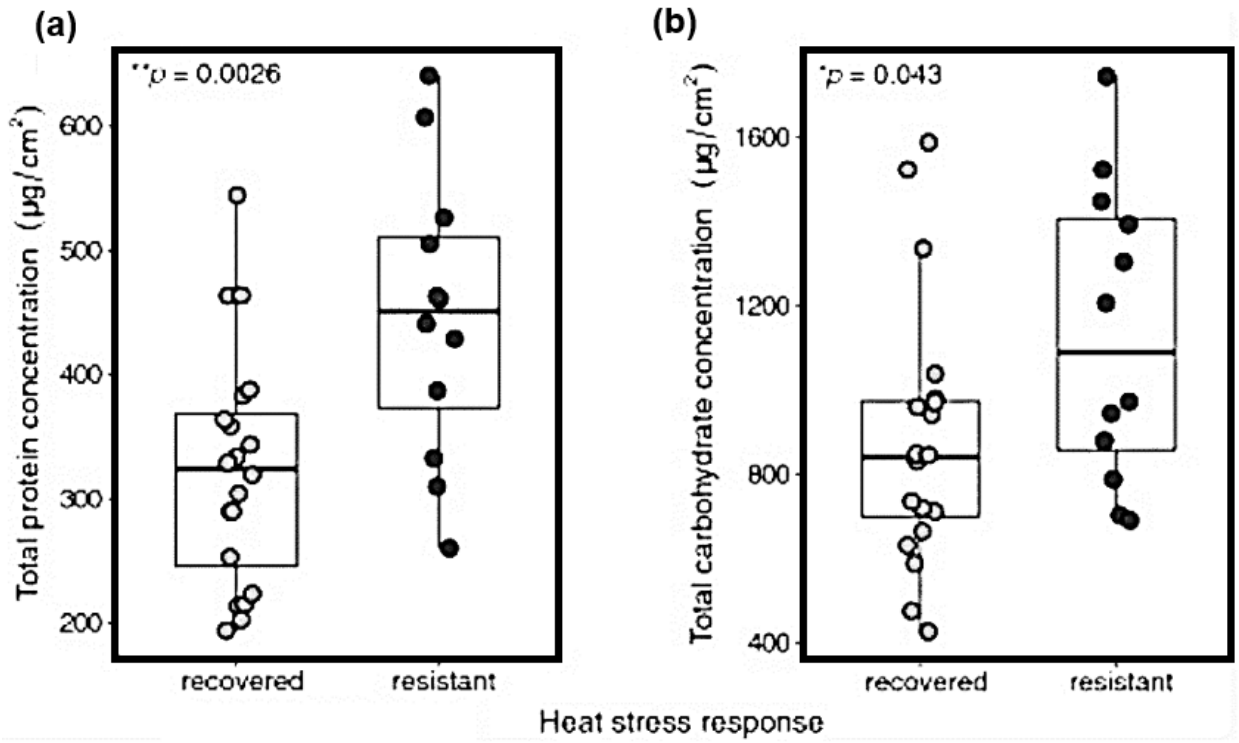

Fig. 3.1: Energetic condition of recovered and resistant coral colonies five months after mass bleaching event. **(a)** Total protein content normalised to host tissue surface area. **(b)** Total carbohydrate content normalised to host tissue surface area. The data point represents a single colony.

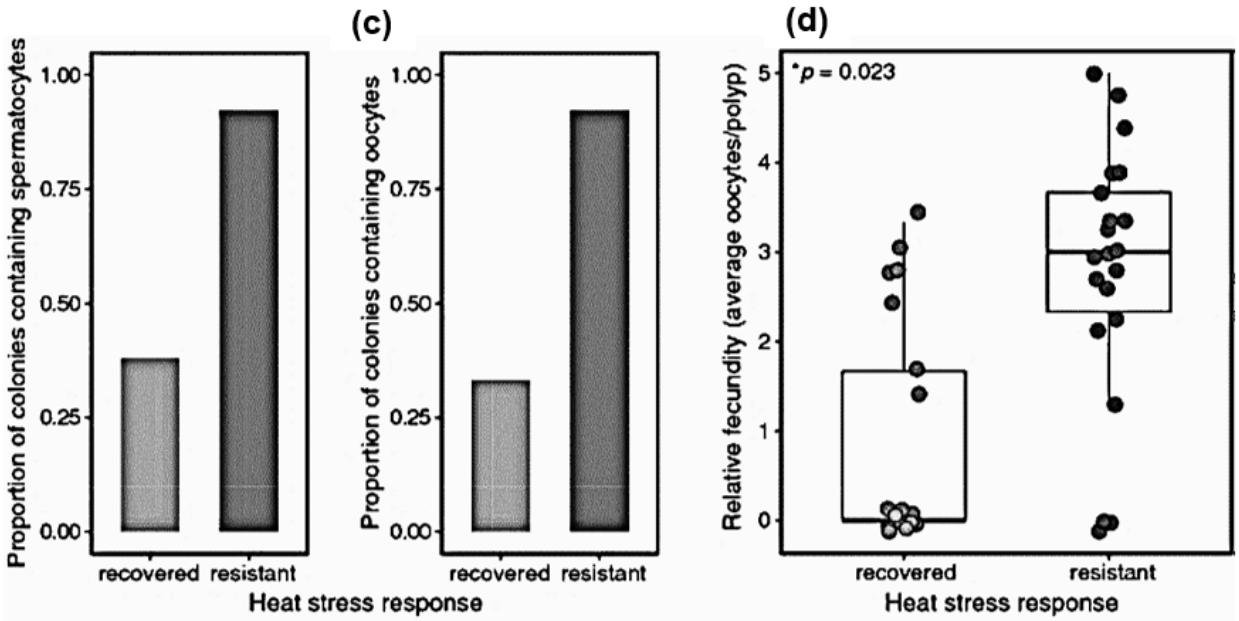

Fig. 3.1: (c) Proportion of recovered and resistant colonies containing male gametes (spermatocytes) & female gametes (oocytes). **(d)** Relative fecundity (fertility) in recovered and resistant colonies. Each data point represents one colony.

(b) Suggest which type of corals would demonstrate greater potential and better ability in reef recovery after disturbance. Justify your choice with evidence from Fig. 3.1.

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1. Resistant corals
2. ref. **higher energy reserves** which can be allocated for **coral reproduction / production of gametes**, to **repopulate coral reef**;
3. Quote any one of the following data
   - **(a)** Higher total protein content (range of 250 – 650 ug/cm^3) for resistant corals compared to (200 – 550 ug/cm^3) for recovered corals
   - **(b)** Higher carbohydrate content (range of 700 – 1700ug/cm^3) for resistant corals compared to (400 – 1600ug/cm^3) for recovered corals

     *[**Accept:** using average / mean protein or carbohydrate content]*

   - **(c)** Higher proportion of resistant corals containing spermatocytes and oocytes (around 0.95) compared to recovered corals (of around 0.30-0.3)

   - **(d)** Higher relative fecundity (average 3 oocytes per polyp) in resistant corals compared to (ave. slightly more than 0 oocytes per polyp) in recovered corals

     *[**Accept**: using mean as comparison between resistant and recovered corals]*

Methods of coral reef restoration are evolving rapidly with investment in research and development. A number of emerging interventions are currently being tested experimentally across various scales, from individual corals (e.g., genetics, reproduction, physiology), to coral populations, reef communities, and reef ecosystems.

These include manual removal of macroalgae and coral predators (such as crown-of-thorns starfish), the direct transplantation of coral colonies or coral fragments or coral larvae at designated restoration sites and rubble stabilization (where the loose coral rubble beds of dead and broken down coral skeletons and rock fragments have been secured to serve as natural substrates for young corals to survive and grow to form stable new reefs). Artificial structures have also been deployed to mimic natural processes and integrated into reef landscapes to serve as substrates for coral recruitment, coral planting and for fish aggregation.

To improve coral reef resilience, scientists are now looking at the possibility of isolating genes of zooxanthellae found in corals that are more resistant to thermal stress and transferring them to the more heat-susceptible zooxanthellae and introducing these genetically modified zooxanthellae to corals before planting the corals back into reefs.

(c) Discuss the possible implications of using such an intervention for reef restoration.

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Pros

1. Such an intervention can complement existing methods of reef restoration to **hasten** the process of reef restoration, given that some of the methods (such as rubble stabilization and manual removal of crown-of-thorn starfish) will only reduce the death of existing coral or will a longer time to recover in numbers.
2. Restoration of reefs in this way will restore the ecosystem services of the coral reef, such as coastal protection and as a nursery for marine species, **despite a rising sea temperature,** since the reefs will now be more resilient with the more heat resistant corals.

Cons

3. The genetically modified corals might out-compete the native corals and drive some of them to extinction, leading to overall reduction in genetic diversity.
4. Breeding and the direct transplanting of the heat-resistant corals can achieve the same result but is less risky (to the environment), instead of using genetically engineered corals.
5. The use of genetically modified corals might not be effective in restoring the reef's ecosystem services (idea that the reef might be rebuilt, but the marine species do not return to the reef)
6. There might be unforeseen adverse effect on the species that feed on the genetically engineered coral, and hence affects the whole food chain/web.

[Reject: Generalised statements like playing God, GE corals are unnatural, resulting in negative impacts on marine life etc. Suitable elaboration or explanations are needed]

[Total: 7]

Section B

Answer **One** question in this section.

Write your answers on the writing booklet provided.

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)** Using **two** named examples, discuss the significance of operon in bacterial growth. [13]

1. Operons allow for the **coordinated control of genes** (structural genes) which are **involved in the same metabolic activity OR allows for regulation of gene expression of the group of structural genes at the same time**.

2. This is because **operons group structural genes that have a related function together**.

3. **Transcription of genes can be switched on or switched off as one transcriptional unit**, permitting the **rapid synthesis of related gene products (that are involved in the same metabolic pathway) as and when required**.

On *lac* operon

4. In the **presence of lactose**, lactose is isomerised into allolactose, **allolactose** acts as an **inducer molecule**.

5. Allolactose binds to *lac* repressor protein leading to conformation change, inactivating **the *lac* repressor protein**. This will cause the **transcription of structural genes** in ***lac* operon.**

6. In the **absence of lactose**, the **transcription of these structural genes can be stopped together** through the **binding of a repressor** on an **operator** found upstream of all the genes.

7. In the **presence of low concentration of glucose and high concentration of lactose**, **cAMP is high**, there will be high concentration of cAMP-CAP complex that binds to CAP binding site. This increases the affinity of RNA polymerase to the promoter, **increasing the rate of transcription of lac structural genes. Allowing the bacteria to utilise lactose.**

8. In the **presence of high concentration of glucose and lactose**, the **preferred source of respiratory substrate is glucose**. Hence, **the operon is switched off due to glucose repression.**

On *trp* operon

9. **In the presence of tryptophan, tryptophan acts as the co-repressor.**

10. **tryptophan** will **bind to the *trp* repressor protein** leading to a conformational change, and **activation of the *trp* repressor protein**. This will cause the **transcription of structural genes** in ***trp* operon** to be **repressed.**

11. **When tryptophan is absent in the environment, *trp* repressor protein** is **inactive**, this will allow for the **transcription of structural genes** in ***trp* operon** to synthesise enzymes required for tryptophan synthesis**.**

12. **Operons** are useful in helping to **direct the limited resources** and **energy** of the bacteria **toward important metabolic activities**.

13. The operon system allows the bacterium to **express their genes to produce enzymes only when required**.
14. This **prevents** the **wastage of energy** and **resources** which provides a **selective advantage** for their **growth and survival**.

(b) The presence of the F factor in bacteria is the most important factor that contributes to the development of antibiotic resistance in bacteria. Do you agree? Justify. [12]

No, I do not agree to a large extent (Accept: Yes, I agree)

On F Factor

1. The **F factor** is made up of **25 genes,** can exists either as **a plasmid** or as **a segment of DNA within the bacterial chromosome**.
2. If the **F factor** exists in the **plasmid form**, it is called the **F plasmid**. (Just as any other plasmids, F plasmid can **integrate** itself into the bacterial chromosome by genetic recombination or replicates **autonomously**.)

On conjugation

3. The presence of the F factor which may **carry antibiotic resistance genes** in a bacterium will allow this bacterium to transfer to another bacteria via **conjugation**.
4. About the requirement of **contact**; reference to **pilus** joining the 2 cells OR formation of **cytoplasmic / mating bridge**.
5. Correct details on DNA transfer via conjugation: origin of transfer, rolling circle mechanism, etc.

On the other horizontal gene transfer

6. However, there are other horizontal gene transfer in bacteria that may contribute to the development of antibiotic resistance in bacteria: transformation and transduction.

On transformation

7. **Transformation** is the **uptake of foreign DNA** from **the surrounding environment** (or naked DNA) by bacterial cells. As a result, the bacterial cell's **genotype** and subsequent **phenotype** can be **altered.**
8. In transformation, the **require the living bacteria to be competent.**
9. Correct details on DNA transfer via transformation: A **short piece of DNA,** released to the **environment** (by the **donor** bacterial cell) is **actively taken up** (i.e. requires energy expenditure) by the **recipient** cell. This **foreign allele** then **replaces** the **native allele** in the bacterial chromosome by **homologous recombination**.
   When **a live bacterial strain,** with no antibiotic resistance takes up foreign DNA (containing the **allele for antibiotic resistance)** from the environment / medium containing **dead**, **broken-up cells** of the pathogenic strain and become transformed into bacterial cells with an **antibiotic resistance.**

On transduction

10. **Transduction** occurs when a **bacteriophage carries and transfers bacterial genes** from **one bacterial cell (= host cell of bacteriophage)** to **another bacterial cell** as a result of **aberrations in the phage reproductive cycle**. (reference to the error in the process)
11. **2** types of **transduction**: **generalised transduction and specialised transduction.**
12. Correct details on DNA transfer via transduction: When **bacteriophage** infects bacteria cells containing **antibiotic resistance gene**, the phage may **facilitate the transfer of this antibiotic resistance gene** to another bacteria via either generalised or specialised transduction.

13. **Mutation** may also lead to an allele for antibiotic resistance gene.
14. This mutated allele may be propagated by the various horizontal gene transfer as well as vertical gene transfer / binary fission.
15. Mismanagement of antibiotic treatment may also contribute to the development of antibiotic resistance in bacteria.

[Total: 25]

5 (a) Explain the importance of mitosis and describe how this process is important in the production of lymphocytes and adaptive immune response. [13]

Explaining the importance of mitosis [Max: 7]

1. Mitotic cell cycle ensures **GENETIC STABILITY**.
2. The daughter cells produced are **genetically IDENTICAL to the parent cell** due to SEMI-CONSERVATIVE replication of DNA at S phase.
3. The daughter cells produced has the **SAME number of chromosomes** (2n) as the parent cell (2n) due to EQUAL SEPARATION of chromosomes at anaphase.

4. **Significance #1 GROWTH:** one parent cell divides into two daughter cells, multicellular organisms increase in cell numbers, from embryo to adult, due to mitotic divisions.

5. **Significance #2 REGENERATION, REPLACEMENT AND REPAIR**

6. **Cells get damaged** by free radicals, chemicals, radiation, physical abrasion and lose their functions. They **die naturally by a process known** as **apoptosis**.

- In higher animals, **most organs** (e.g. liver) have **unspecialised cells** that undergo mitosis to produce **new cells to replace the dead ones**. Epithelial cells undergo mitosis to replace cells that are brushed off as food passed through the oesophagus.
- In lower animals, such as **invertebrates, mitosis helps to replace missing parts** to varying degrees. For example, the lost arm of a starfish can be regenerated.
- In higher animals, it is not possible to regenerate a lost limb but **mitosis is needed for healing a wound or injury**

7. **Significance #3 ASEXUAL REPRODUCTION**

- Unicellular organisms such as bacteria, yeasts, *Amoeba*, undergoes **binary fission** to propagate rapidly when the environment is favourable.

8. Asexual reproduction allows the rapid colonization of a habitat, provided that the parent has advantageous adaptations to the habitat.

Describing how mitosis is important in the production of lymphocytes

9. **Haematopoietic stem cells** can **divide and renew themselves for long periods** via **mitosis**, while still **maintaining the undifferentiated state**;
10. **Haematopoietic stem cells** can also undergo **asymmetric division** by **mitosis** to form **one daughter stem cell** and **one lymphoid progenitor stem cell**;
11. Where the lymphoid **progenitor stem cells** can **differentiate** into **Natural Killer (NK) cells, T lymphocytes and B lymphocytes** under the **presence of appropriate chemical signals.**

Describing how mitosis is important in the adaptive immune response [Max: 5]

12. Mitosis is required for **cells proliferation** involved in adaptive immune response; e.g. **B cells** OR **T cells proliferation**.
13. Mitosis is required for **differentiation** involved in adaptive immune response; e.g. **B cells** into **plasma cell** OR **$CD4^+$ T cells** into **Helper T cells** OR **$CD8^+$ T cells** into **cytotoxic T cells**.
14. Mitosis is required for **immunological memories** which is one of the features of adaptive immune response; e.g. **B cells divide and differentiate into memory B cells** OR T cells **divide and differentiate into memory T cells**.
15. Mitosis is required for **generation of different classes of antibody as** B cells need to divide during **class switching** so that different B cells can differentiate into plasma cells secreting different classes of antibody.
16. Mitosis is required during the process of **affinity maturation** during **somatic hypermutation** of **B cells**.
17. Mitosis is required in the **replacement of adaptive immune cells** that die to maintain a healthy concentration of these immune systems in the body.

(b) Discuss what genetic variation is and how variation, including harmful recessive alleles, may be preserved in a natural population. [12]

Discussion on genetic variation [max: 8]

1. Genetic variation refers to the **variation / differences of alleles / genes / DNA sequences in the genome** (of individuals within a population / species or across different species)
2. Genetic variation within a population can arises due to **mutation**;
3. which can be a result from **spontaneous mutation** (randomly occurring), **DNA replication errors, Mutagens** (e.g. UV light, chemicals, carcinogens).
4. The mutation could be **gene mutation** which is a **change** in the **sequence of DNA nucleotides** of a **gene**.
5. This could be due to: **nucleotide-pair / base-pair substitution** OR **nucleotide-pair / base-pair addition** (**insertion**) OR **nucleotide-pair / base-pair deletion.**
6. The mutation could be **chromosomal aberration** which is a form of mutation where there is a **change** in: the **number** of chromosomes (**Numerical aberration**);
7. or the **structure** of chromosomes (**Structural aberration**)
8. Genetic variation may also arise due to **synapsis and crossing-over during Prophase I**;
9. **independent Assortment of Homologues during Metaphase I;**
10. **random Fusion of Gametes during fertilisation**

Discussion on how variation (including harmful recessive alleles) may be preserved in a natural population [max: 8]

[Diploidy]

11. As most eukaryotes are **diploid organisms,** a considerable amount of **genetic variation can be hidden** from **selection pressure** in the form of **recessive alleles in heterozygotes**.
12. **Dominant alleles mask the expression of recessive alleles**.
13. **Selection pressure** can only act on **expressed phenotypes**, i.e. they cannot act upon recessive alleles in heterozygotes. (**Heterozygous protection)**
14. Thus, **even if recessive alleles might be harmful** in an environment, they can **persist** by **propagation in heterozygous individuals** without being eliminated from the population.
15. Heterozygotes provide protection for recessive alleles and **maintain a larger gene pool** than possible if organisms are not diploid.

[Heterozygote advantage]

16. Under certain circumstances, some individuals who are heterozygous at a particular locus may have **greater fitness than both kinds of homozygotes**;
17. these individuals exhibit **Heterozygote advantage**.
18. People with the sickle cell trait, **Hb^A Hb^S**; are **normally healthy** unless **oxygen concentration is low**, then their **red blood cells may turn sickle-shaped**.
19. Despite the survival disadvantage of the allele **Hb^S**, in West Africa, the **Hb^S allele persists** in the population because there is a high incidence rate of **malaria** in **West Africa**.
20. The malarial parasite spends part of its life cycle in human red blood cells. **Normal individuals** (**Hb^A Hb^A**) would be **susceptible to malaria** as the parasite will infect their red blood cells.
21. Red blood cells that contain **haemoglobin S die** when they are **infected with the malarial parasite**, thus **terminating their life cycle** and stopping the infection of the disease.
22. The **heterozygous individuals** (**Hb^A Hb^S**) are at **a selective advantage** in West Africa as they are more likely to be able to survive a malarial infection and reproduce, **passing on the otherwise disadvantageous Hb^S allele to their offspring**.

[Frequency-dependent selection]

23. **Frequency-dependent selection** occurs when the fitness of a genotype or phenotype in a population is related to its frequency in the population.
24. **Positive frequency-dependent selection** occurs when the more common a variant is in a population, the higher its fitness,
25. while **negative frequency dependent selection** occurs when a variant has higher fitness the less common it is.
26. **Positive frequency dependent selection** therefore tends to **eliminate variation from populations**, while **negative frequency dependent** selection acts to **retain polymorphism**.

[**Genetic drift**]
27. **Genetic drift** is a process that happens due to **random chance events** that leads to **change** (both increase and decrease) in the **frequencies of alleles within a population over time**.

[Total: 25]

END OF PAPER