

RAFFLES INSTITUTION
2025 Year 6 Preliminary Examination

Higher 2

CANDIDATE
NAME

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GROUP

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INDEX
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BIOLOGY

9744/03

Paper 3 Long Structured and Free-response Questions

17th Sept 2025

2 hours

Candidates answer on the Question Paper.
Additional Materials: Writing paper.

READ THESE INSTRUCTIONS FIRST

Write your index number, CT group & 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 in the writing paper 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.

At the end of the examination, **hand in your essay question SEPARATELY.**

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

For Examiner's Use	
Section A	
1	/ 31
2	/ 8
3	/ 11
Section B	
4 or 5	/ 25
Total	/ 75

This document consists of **15** printed pages.



Raffles Institution
Internal Examination

Section AAnswer **all** the questions in this section

- 1** Liver cells, or hepatocytes, are highly specialised and metabolically active. These cells are responsible for a vast array of vital functions, including the metabolism of carbohydrates, lipids, and proteins, detoxification of harmful substances, synthesis of plasma proteins, and bile production. Their constant activity makes them susceptible to damage from various factors such as viral infections (e.g., hepatitis), excessive alcohol consumption, and metabolic disorders like non-alcoholic fatty liver disease (NAFLD), which can impair their function and lead to serious health issues.

Fig. 1.1 is a transmission electron micrograph showing a section of a human liver cell.



Fig. 1.1

- (a) (i) Name organelles **A** and **B** shown in Fig. 1.1. [1]
A Mitochondrion (R: mitochondria)
B Rough endoplasmic reticulum (R: rER)
- (ii) In liver cells, enzymes are attached to the membrane of smooth endoplasmic reticulum (SER).
 With reference to the functions of smooth endoplasmic reticulum, suggest the advantages of having enzymes attached to the membrane rather than free in the lumen of the SER. [2]
- More efficient synthesis (R: metabolism) of lipid/ phospholipid/ steroid (R: other biomolecules);
 - Synthesis of products is in a pathway/ requires more than one enzyme thus allows enzymes to be close together/ products of one reaction are the substrates of next reaction/ in sequential arrangement of enzymes of a catalytic pathway R: compartmentalisation)
 - By embedding enzymes in membrane, enzymes are more stable/ greater protection from degradation;
 - AVP with sufficient elaboration;

e.g. idea of greater efficiency as enzyme held down so that active site faces substrates in lumen;
 allows reactions to occur on cytoplasmic side of membrane;
 allows reactions to continue in SER transport vesicles;

Neutral lipids or non-polar lipids such as triglycerides and cholesterol esters are synthesised in the membrane of SER of the liver cell. Fig. 1.2 shows the formation of a lipid droplet (LD) from the endoplasmic reticulum membrane.

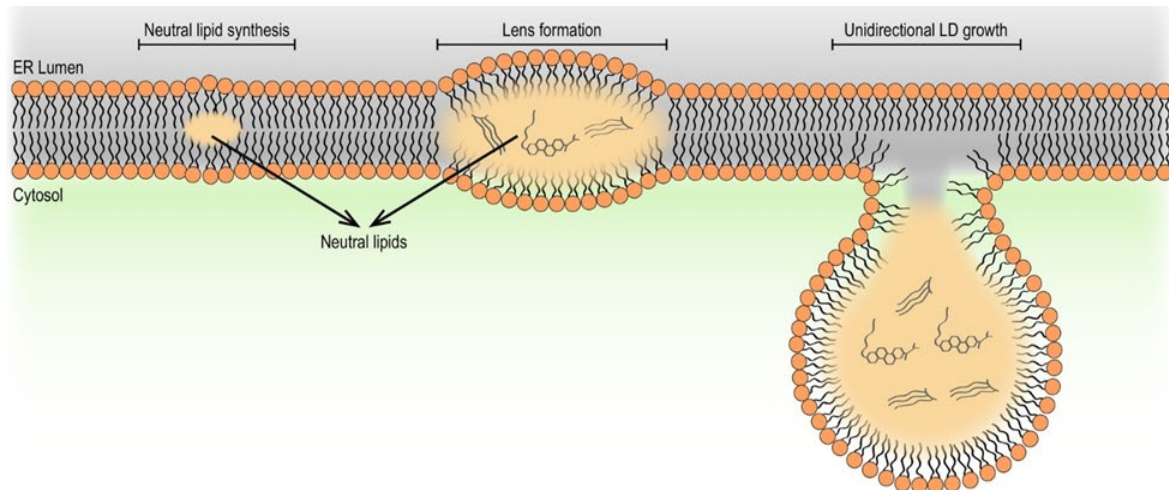


Fig. 1.2

- (b) (i) Describe the formation of the lipid droplet shown in Fig. 1.2. [3]
- Neutral lipids accumulate between the two layers of phospholipids (R: in the membrane of SER);
 A: lipids accumulate within phospholipid bilayer
 A: lipids accumulate within hydrophobic core of phospholipid bilayer
 - Continuous accumulation of neutral lipids leads to formation of a growing bulge
 A: Neutral lipids pushing two layers of phospholipids of SER membrane apart
 R: there is lens formation
 - Single phospholipid layer (R: single phospholipid bilayer, R: single membrane) pinches off from ER membrane into cytosol to form a lipid droplet;
- (ii) Lipid droplets serve as energy storage within the cell. Based on the properties of the biomolecules involved, explain how a lipid droplet maintains its stable structure in the cytoplasm. [2]
- Lipid droplet core is composed of hydrophobic* neutral lipids (R: lipids), which interact (R: face) with hydrophobic hydrocarbon tails (R: fatty acids) of a surrounding single phospholipid monolayer;
 - Amphipathic phospholipids in monolayer orient phosphate heads* outwards, which are charged and hydrophilic*, interacting with aqueous cytoplasm (R: cytoplasm alone/ outside cell);
 - while their hydrophobic* hydrocarbon tails* face inwards, effectively shielding neutral lipid (R: lipids) core from water and preventing its dispersion;

- (c) Prostaglandins are another type of lipid synthesised at the SER. Prostaglandins are small lipids produced in many tissues of the body. One role of prostaglandins is to cause inflammation at the site of an injury or infection. Inflammation is the normal first response of the immune system to injury or infection.

Cyclooxygenase (COX) is an enzyme that catalyses one of the steps in the reaction pathway for the formation of prostaglandins from phospholipids. The reaction pathway occurs in the SER of cells. Part of the reaction pathway is shown in Fig. 1.3.

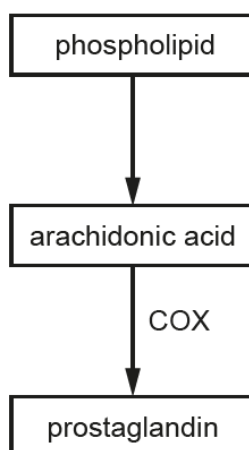


Fig. 1.3

- (i) Suggest an advantage for this reaction pathway occurring in the smooth endoplasmic reticulum of a cell rather than in the cytoplasm. [1]
1. SER is membrane-bound organelle so it can provide phospholipid/ synthesised phospholipid/ arachidonic acid which is a substrate in the reaction;
 2. SER is involved in lipid transport thus prostaglandins can be transported (from SER) to Golgi Apparatus since that is the next immediate member of the endomembrane system or stored (in SER);
 3. Compartmentalisation/ separated from other reactions in cytoplasm/ provides optimum conditions for enzyme reactions/ higher concentration of enzymes;
- (ii) Inflammation may have side-effects, such as pain. Aspirin is a drug that can be used to reduce these side-effects by reducing the catalytic activity of the COX enzyme by modifying the R-group of one of the amino acids.

Explain how modifying the R-group of an amino acid in the COX enzyme can reduce the catalytic activity of the enzyme. [3]

1. A change in R group of a structural amino acid may result in different interactions between R groups such as ionic bond, hydrogen bond or hydrophobic interactions;
2. A change in R group can result in a change in conformation of enzyme active site*;
3. Active site is not completely complementary in shape /conformation* to the arachidonic acid or substrate ;
4. Enzyme-substrate* complex, not formed / formed at reduced rate;
5. Catalytic R group is absent that cannot catalyse the reaction/ contact R group is absent and cannot hold the substrate well at active site;
6. Ref. to effect of activation energy not being reduced ; e.g. ref to changed charges (so no electron transfer) no longer provides hydrophobic regions for reaction to occur;

A number of chemicals can disrupt mitochondrial respiration of the liver cell. Some of these are listed in Table 1.1.

Table 1.1

chemical	effect on mitochondrion
oligomycin	blocks proton flow through ATP synthase
FCCP	causes protons to leak out of the inter-membrane space back into the matrix without going through ATP synthase
antimycin-A + rotenone	stops electrons from being passed down the electron transport chain (rotenone and antimycin-A affects proteins along the electron transport chain)

Scientists measured the oxygen consumption rate (OCR) of some cells. They then investigated how the OCR changed when oligomycin was added at 23 minutes, followed by FCCP at 56 minutes, followed by antimycin-A + rotenone at 93 minutes.

Fig. 1.4 shows the results obtained.

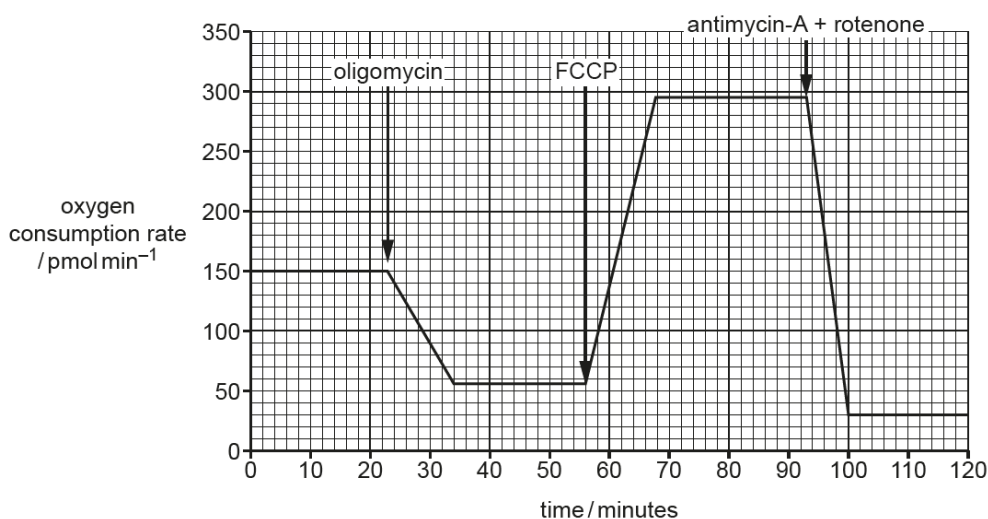


Fig. 1.4

- (d) (i) With reference to events at the inner mitochondrial membrane, describe and explain each of these **two** statements:

oligomycin causes a drop in OCR [3]

(blocks ATP synthase)

- Oxygen consumption decreases sharply from 150 to 55 pmol min⁻¹ in 11 min/from 23 to 34 min;
- No/less proton flow through ATP synthase so maintains high proton gradient / stays steep;
- steep gradient makes it inhibits e⁻ flow down electron transport/ no flow of e⁻ down **electron transport chain*** / harder to pump more protons from matrix to intermembrane space;
- as O₂ is the **final electron acceptor*** of **ETC***, fewer e⁻ moves down ETC so no/less e⁻ to combine with H⁺ and O₂ to form water;

FCCP causes a rise in OCR [3]

1. Oxygen consumption increases sharply from 55 to 295 pmol min⁻¹ in 12 min/from 56 to 68 min;
2. Diffusion of protons out of inter-membrane space decreases the proton gradient;
3. ETC attempts to maintain proton gradient increased the flow of e⁻ down ETC;
4. O₂ is **final electron acceptor of ETC*** thus increased O₂ combines e⁻ with H⁺ combines to form water;

(ii) Suggest why the oxygen consumption rate of the cell never falls to zero in these cells. [1]

1. Other uses of oxygen in cell / reference to other oxidation reactions that uses oxygen;
2. Inhibitor concentrations are not high enough to inhibit all ATP synthase and electron carrier of ETC

A: oxidation of lactic acid to pyruvate

During respiration, protons 'leak' through the inner mitochondrial membrane into the matrix. The OCR as a result of this leakage can be calculated by determining the difference between the OCR 20 minutes after the addition of oligomycin and the OCR 20 minutes after the addition of antimycin-A + rotenone.

(iii) Use Fig. 1.2 to calculate the OCR caused by proton leakage.

$$56 - 30 = 26 \pm 1 \text{ unit} \quad \dots\dots\dots \text{pmol min}^{-1} \text{ [1]}$$

(e) Rotenone has been proposed as a possible treatment for cancer.

Explain why cancer cells are more likely to be affected by rotenone than non-cancer cells. [2]

1. Cancer cells divide faster ;
2. Therefore need a lot more ATP / cancer cells more metabolically active / higher rate of aerobic respiration ;
3. More vulnerable to reduced aerobic respiration than non-cancer cells (WTTE);

(f) Alanine transaminase (ALT) is an enzyme that is normally found only in the cytoplasm of liver cells.

Scientists can use the concentration of ALT in a person's blood as a measure of damage to liver cells.

Suggest why blood ALT concentration provides a measure of damage to liver cells. [2]

1. ALT is an enzyme found in the cytoplasm and its release from liver cells is due to damage to/ leakage from cell surface membranes (A: cell lysis);
2. Provide a quantitative measurement as more damage of liver cells lead to more ALT in the blood;
3. AVP;

Another transaminase, aspartate transaminase (AST), is also found in the mitochondria of a variety of cells, including liver cells.

Table 1.2 shows the distribution of ALT and AST in the body, as well as the time taken for the quantity of each enzyme if released in the blood to halve.

Table 1.2

enzyme	location where found	percentage distribution in cell		time taken for quantity of enzyme if released in blood to halve/ hours
ALT	liver	cytoplasm	100	47
		mitochondria	0	
AST	heart, liver, skeletal muscle, pancreas and kidney	cytoplasm	20	17
		mitochondria	80	

- (g) The concentration of AST in the blood is not, by itself, a reliable indicator of liver damage.

Use Table 1.2 to explain why blood AST concentration by itself is **not** a reliable indicator of liver damage. [2]

1. AST could be coming from other tissues / organs (quote);
2. Its short half-life / volatile of 17 hr in the blood thus more difficult to detect liver cells damaged has occurred/ could lead to a false negative or underestimate the extent of past injury;
3. (a) It is less likely to be released because it requires damage to both membranes of mitochondria and cell; OR
(b) It is less likely to be detected in the blood as a high percentage of 80 percent is found in the mitochondria (A: 20% cytoplasm)

- (h) In clinical practice, diagnosis of liver damage is based on both:

- the concentration of ALT in the blood; and
- the ratio of concentration of AST:ALT in the blood.

Table 1.3 shows the liver conditions associated with different blood ALT concentrations and AST:ALT ratios.

Table 1.3

liver condition	blood ALT concentration / arbitrary units	AST:ALT
normal	1 to 40	0.8:1
acute (short-term) viral liver damage	100 to 1100	<0.8:1
acute non-alcoholic liver damage	30 to 100	<0.8:1
chronic (long-term) alcoholic liver damage	30 to 300	>2:1

- (i) Explain why using only blood ALT concentration, by itself is also **not** a good diagnostic tool for all types of liver damage. [2]
1. There is an overlap of ALT levels in different types of liver damage;
 2. e.g. non-alcoholic liver damage of 30 to 100 arbitrary units within chronic alcoholic liver damage range of 30 to 300 au/ acute viral liver damage and chronic alcoholic liver damage overlap in the 100 to 300 au range;
 3. hence cannot differentiate between these specific conditions;

The blood concentrations of AST and ALT were measured in five people who had recently been admitted to hospital.

Table 1.4 shows the data collected.

Table 1.4

person	blood AST concentration / arbitrary units	blood ALT concentration / arbitrary units	AST:ALT
A	37	62	0.6:1
B	27	38	0.7:1
C	26	33	0.8:1
D	620	1034	0.6:1
E	140	54	2.6:1

- (ii) Use the information in Table 1.3 to identify the people in Table 1.4 who could be diagnosed with acute non-alcoholic liver damage. [1]
A & B
- (iii) Alcohol causes damage to mitochondria.

Explain why alcohol damage to the liver can cause an increase in concentration of AST in the blood. [2]

1. Mitochondria contains large/ high quantities/ 80% of AST;
2. Liver cell contain many mitochondria needed for many metabolic processes;
3. Liver cells allow alcohol entry/more permeable to alcohol ;
4. Alcohol denatures membrane proteins/ dissolves membranes of mitochondria and liver cells thus allows AST to move out of liver cell into blood;
5. AVP ;

[Total: 31]

- 2 Kiribati is a low-lying island nation situated close to the equator, in the middle of the Pacific Ocean. It comprises isolated, narrow islets that are at a maximum elevation of 3 m above sea level. Climate change research studies have projected estimates that two-thirds of the islands may be submerged by 2100 and become uninhabitable due to the impacts of climate change.

Fig. 2.1 shows part of Tarawa, one of Kiribati's islands, that has experienced flooding and coastal erosion (charted in 2013).

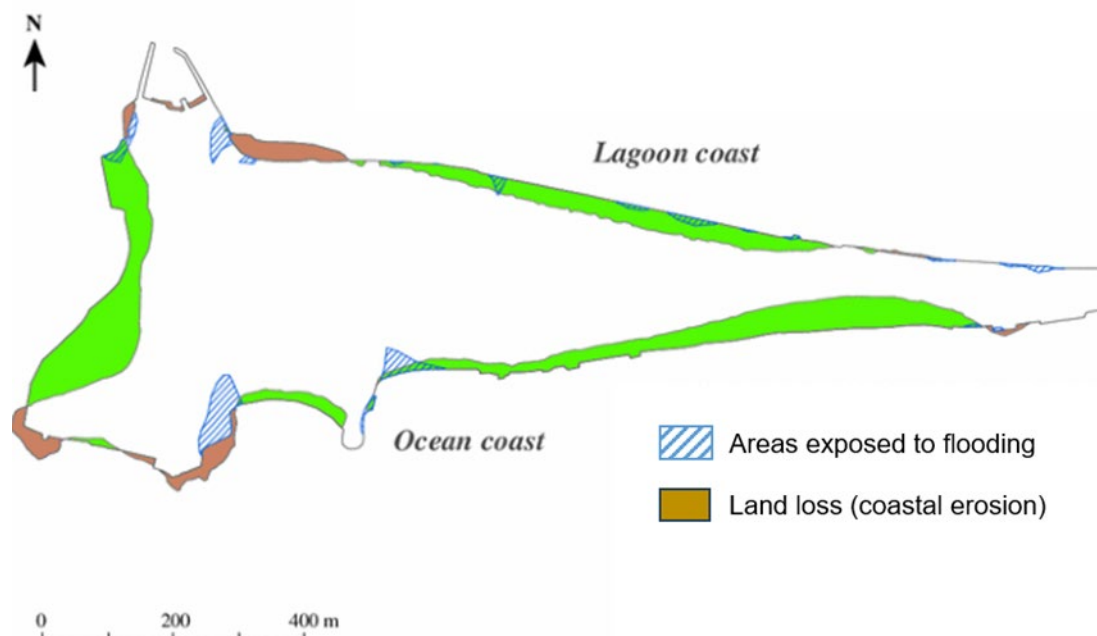


Fig. 2.1

- (a) (i) Describe two ways in which climate change may cause sea levels to rise. [2]
1. Melting of land ice/ice caps/ice sheets/glaciers/ice from land enters sea;
 2. Global warming will lead to thermal expansion of water;
- R: ice masses in the sea
- (ii) Explain the consequences of rising sea levels on Tarawa. [3]
1. Saltwater intrusion increases soil salinity/ changing soil may affect growth of crops;
 2. Flooding of freshwater with seawater/ reduce freshwater sources (wells, streams, lakes) may cause organisms to die/ face extinction due to change in habitat conditions;
 3. Colonisation of habitats by displaced populations of animals/isolation of animal populations (in high areas);
 4. Increased coastal erosion due to increased wave action;
 5. AVP (e.g. migration might become necessary: the erosion Kiribati is facing from the encroaching ocean, its increasing need for desalination equipment to provide drinking water, and the use of sandbag walls to try to keep tides at bay);

Fig. 2.2 below shows the projected sea level rise, in metres, from 2020 to 2127 for Kiribati.

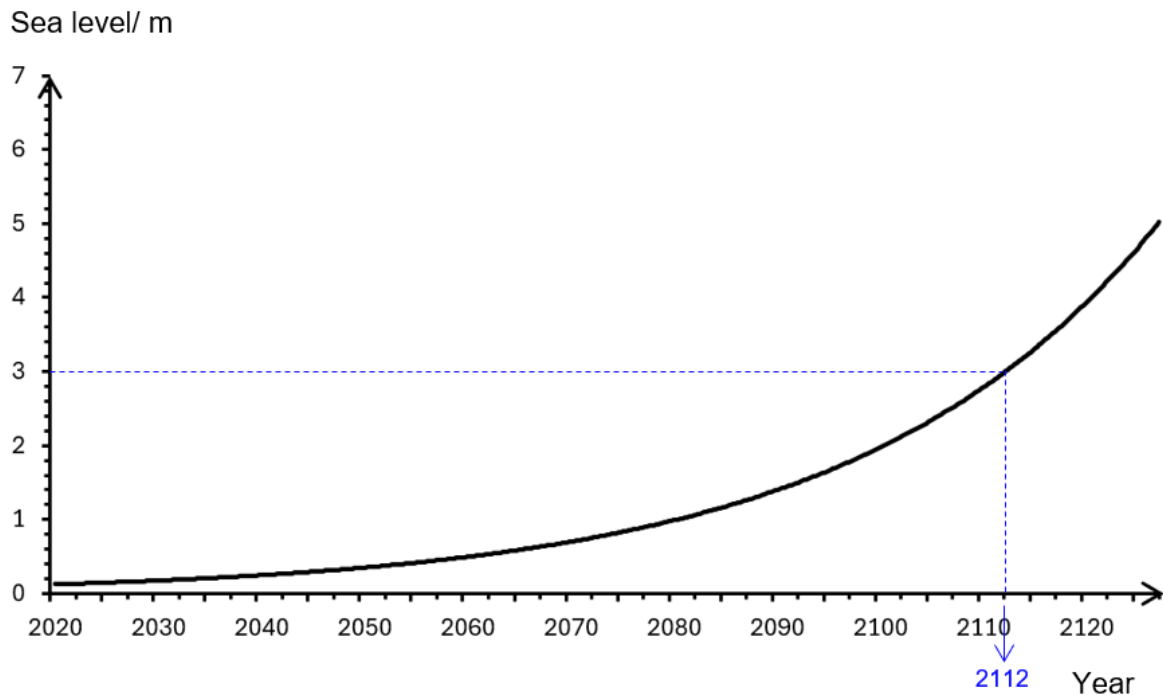


Fig. 2.2

- (b) (i) Based on Fig. 2.2, and the information given above, when is the estimated year that Kiribati will be fully submerged? Show on the graph how you arrived at your answer.

Estimated year:.....[2]

2112 or 2113

- (ii) Suggest a reason for the exponential increase in the graph after year 2110. [1]
- Positive feedback loop in climate change: Melting of glacier sheets releases water and exposes darker land surfaces, reducing reflectivity and accelerating warming/ melting permafrost releases methane which amplifies warming;
 - Non-linear melting of ice sheets: early melting may be slow but once a critical threshold is reached, ice sheet collapse can occur rapidly which leads to a sudden and large sea level rise;
 - Thermal expansion of oceans: warmer water expands, so as global warming intensifies, the rate of thermal expansion accelerates and deeper ocean layers warm over time, adding to the later rapid rise;
 - Climate system tipping point/ threshold reached, beyond which the sea level rises in an exponential, non-linear manner;
 - AVP

[Total: 8]

- 3 The Hawaiian archipelago is a group of volcanic islands in the Pacific Ocean.

Fig. 3.1 shows the relative locations of five of these islands.



Fig. 3.1

Table 3.1 shows the size and age of these five islands and the total number of *Mecyclothorax* ground beetle species and their species density, on each island.

Data for the island of Maui is shown as two distinct regions, West and Haleakala. This is because they formed at different times from two separate volcanoes.

Table 3.1

island	area / km ²	age of island / million years	total number of <i>Mecyclothorax</i> species	species density / number of species per km ²
Hawai'i	10 433	0.4	30	0.003
Lāna'i	364	1.3	3	0.008
Maui (West)	443	1.3	27	0.061
Maui (Haleakalā)	1440	1.1	116	0.081
Moloka'i	673	1.9 – 1.8	43	0.064
O'ahu	1545	3.7 – 2.6	20	

Fig. 3.2 shows a ground beetle of the genus *Mecyclothorax*. All the beetle species of this genus on the Hawaiian archipelago descended from one original colonising species that reached Maui from Australia.

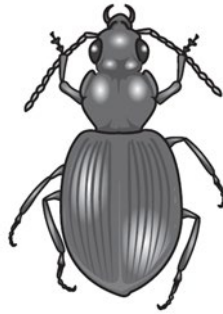


Fig. 3.2

- (a) (i) Complete Table 3.1 by calculating the density of *Mecyclothorax* beetle species on the island of O'ahu. [1]
0.013; (3 dp)
- (ii) Use Table 3.1 to explain why the island of Hawai'i has the lowest density of *Mecyclothorax* beetle species. [2]
1. Hawai'i is youngest island, formed only 0.4 million years ago (from Table 3.1)
2. less time passed for *Mecyclothorax* beetles to colonise and speciate/early stage of succession (A: less time for evolution / mutations to accumulate)
3. Hawai'i has the largest area so, density is low.
- (iii) Using information from Fig. 3.1 and Table 3.1, suggest why O'ahu has a lower number of *Mecyclothorax* beetle species than Moloka'i. [2]
1. Maui is where the beetles first arrived and O'ahu is further away from Maui than Moloka'i as seen in Fig 3.1;
2. so it took more time to reach O'ahu and colonise later;
3. hence has less time to speciate;
4. quote data: fewer *Mecyclothorax* beetles reached O'ahu hence it has fewer species (20) than Moloka'i (43);
A: answers about Moloka'i being nearer to Maui
- (b) The rate of speciation of *Mecyclothorax* beetles on the slopes of the volcano Haleakala, on Maui, is the highest recorded for any genus or location on earth.

The volcano last erupted 400 years ago. This produced lava flows which cut through ancient forest and vegetation, dividing it into many separate microhabitats.

Explain how the large number of *Mecyclothorax* beetle species on Haleakala developed. [4]

1. Flow of lava destroyed habitats and formed a physical barrier which separated the continuous forest and vegetation and led to **geographical isolation*** and **disruption of gene flow***;
2. As many different microhabitats/niches were available after the event, the beetles were subjected to **different selection pressures** in their different habitats;
3. As different sub populations evolved independently of each other, their allele frequencies changed as they accumulated different genetic mutations, and were subjected to **genetic drift*** and **natural selection***;
4. After 400 years, beetles in different microhabitats were **reproductively isolated*** and could no longer **interbreed*** to produce **viable fertile*** offspring;

5. This finally resulted in adaptive radiation / allopatric speciation;

AVP: Accept bottle neck event that changes allele frequency in the different populations and founder's effect where beetles colonise the place where the lava flowed

(c) Table 3.2 shows the DNA sequences from 6 *Mecyclothorax* beetles species on Haleakala.

Table 3.2

Species A	A	A	C	T	T	G	A	A	G	G	G	G	A	A	C	T	T	C	A	G	G
Species B	G	A	T	G	A	G	A	A	G	G	T	T	A	C	C	G	C	A	T	A	C
Species C	A	A	C	G	A	C	T	G	T	G	T	G	G	G	G	C	T	C	G	G	G
Species D	A	A	C	T	T	G	A	A	T	G	G	G	A	A	C	T	T	C	G	G	G
Species E	C	T	T	G	G	G	C	A	A	C	T	G	T	C	T	G	T	C	A	G	T
Species F	A	G	A	C	T	T	A	T	T	A	T	C	C	G	C	G	A	C	G	T	C

Explain which species is the most closely related to Species A. [2]

1. Species D;
2. They have smallest number of differences of 2 nucleotides whereas there were more than 2 for other species B, C, E and F. (A: reverse argument)
3. Closely related species have greater proportion of their DNA in common due to their descent from a common ancestor

In a follow-up study, scientists investigated a large population of a specific *Mecyclothorax* beetle species within an isolated forest patch on the slopes of Haleakala. They focused on a gene that controls exoskeleton coloration, which has two alleles: a dominant allele for dark coloration (**D**) and a recessive allele for light coloration (**d**).

- (d) (i) Explain what is meant by the Hardy–Weinberg model and state two conditions necessary for a population to maintain Hardy–Weinberg equilibrium. [2]
The Hardy–Weinberg model states that allele and genotype frequencies in a population will remain constant / in equilibrium from generation to generation in the absence of evolutionary influences. [1]

☐

Conditions (Any 2 to get 1 mark):

- No mutation.
- No natural selection (all genotypes have equal survival and reproductive success).
- The population size is infinitely large (no genetic drift).
- Mating is completely random.
- No gene flow / migration into or out of the population. [1]

- (ii) Out of 500 beetles sampled from this isolated population, 45 beetles were observed to have the light coloration phenotype.

Assuming the population is in Hardy-Weinberg equilibrium, calculate the frequency of the heterozygous genotype. Show your working clearly.

Step 1: Find the frequency of the homozygous recessive genotype (q^2)

Frequency of dd = $45 / 500 = 0.09$ [1]

Step 2: Find the allele frequencies (q and p) q (frequency of allele d) = square root of 0.09 = 0.30 p (frequency of allele D) = $1 - 0.30 = 0.70$ [1]

Step 3: Find the frequency of the heterozygous genotype ($2pq$) Frequency of Dd = $2 \times p \times q = 2 \times 0.70 \times 0.30 = 0.42$ [1]

[3]

[Total: 16]

Section B

Answer **one** question in this section.

Write your answers on the separate answer paper provided.

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

Your answers must be in continuous prose, where appropriate.

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

- 4 (a) Understanding what causes disease in humans is essential for improving human health outcomes and guiding medical research.

Explain the causes of diseases in humans.

[20]

Scientific Content (max 16 marks)

A. Gene mutations

1. a gene mutation arises from a change in the sequence of nucleotide bases in DNA of a gene;
2. e.g. substitution, addition (insertion), deletion, inversion (any 1);
3. e.g. sickle cell anemia → substitution mutation → HbS (A Huntington any gene mutation);

B. Chromosomal mutations

[Variation in chromosomal structure]

4. alterations in chromosomal structure brought about by e.g.: deletions, duplications, inversion, reciprocal translocation (any 1);

[Variation in chromosomal number]

5. aneuploidy - cell does not have a chromosome number that is a multiple of the haploid number/ chromosomes are present in either extra or fewer copies than wild type;
6. due to non-disjunction where homologous chromosomes do not move properly to opp. poles at meiosis I, OR when sister chromatids fail to separate properly to opp. poles at meiosis II;
7. e.g. Down syndrome → 24 chromosome in gamete due to extra chromosome 21 and hence aneuploidy;

AB. Effect of gene/ chromosomal mutations

8. change in mRNA codon/ amino acid/ protein conformation → non function protein;
9. gene expression altered by neighbouring genes or regulatory sequences in its new location;
A: changes in promoter → no transcription initiation complex/ enhancer → activator cannot bind → low gene expression/ silencer → repressor cannot bind → high gene expression;

C. Heredity causes

10. e.g. BRCA increases risks of breast cancer;
11. some elaboration e.g. 1 recessive copy from each parent may result in homozygote child with disease;

12. sex-linked predominantly affects males as he has only one X chromosome that expresses mutant allele/ females can be carriers of the allele but usually have milder or no symptoms;

D. Viruses, bacteria, other pathogens

13. pathogens include bacteria, viruses and fungi - cause disease by damaging cells and producing toxins*;
14. e.g. Human Immunodeficiency Virus
targets T cells and macrophages, loss of T helper cells leads to impaired immune responses in affected individual who then becomes increasingly susceptible to opportunistic disease;
15. e.g. influenza virus
virus bind to epithelial cells of respiratory tract lead to inflammation and buildup of dead epithelial cells in airways cause symptoms/ weakening of epithelial layer caused by viral replication make respiratory passage susceptible to bacterial infections;
16. e.g. Mycobacterium tuberculosis bacteria cause tuberculosis;
- some elaboration on TB infection such as lungs will be progressively destroyed by the formation of cavities due to the rupture of tubercles;
17. fungi/ protozoa
e.g.: organisms that can cause skin infections (e.g., ringworm, athlete's foot)/
parasites: organisms that live on or in a host and derive nutrients from it, leading to diseases like malaria caused by protozoa;

A: generic point 1 mark in place of 14, 15 such as the following
effect of viruses in causing diseases

e.g.: death of host cell/ production of toxic substances/ cell transformation/ suppression of immune mechanism/ induction of non-normal host-specified products/ induction of structural alterations to host cell; (any 1)

E. Environmental factors

18. e.g. smoking – has tar in cigarette smoke/ carcinogenic tar including benzene, benzpyrene, formaldehyde and phenols, and causes e.g. lung cancer;
19. e.g. ionizing radiation – cause DNA damage/ultraviolet light may modify DNA structure, and causes skin cancer;

F. Mutations in cell cycle

Mutations in genes involved in controlling cell cycle resulting in cancer such as

20. protooncogenes – some elaboration such as gain in function mutation coding for excess gene products;
21. tumour suppressor gene – some elaboration such as loss in function mutation in genes coding for DNA repair during cell cycle;
22. mutations in these genes can lead to uncontrolled cell division leading to formation of tumour/ cancer;

G. Other factors

23. e.g. other lifestyle factors e.g. – diet (elaborate)
A: obesity - excess of calories/ processed food e.g. cardio vascular diseases, diabetes
A: alcohol – excess alcohol intake e.g.: liver damage
24. climate change e.g. clean water availability;
25. human factors e.g. overcrowding facilitates spread of pathogen, vaccination (A: any socio-economical factors);
26. vector presence e.g. mosquitoes help spread dengue fever;

27. environmental effects such e.g. air pollution from industries (e.g. particulate matter, industrial emissions), water contaminants (e.g., heavy metals, chemicals, pathogens);

A: allergens: substances in the environment (e.g., pollen, dust mites, pet dander) that trigger allergic reactions in susceptible individuals

Communication (max 2 marks)

Marks	Descriptors
2	presented information concisely, in a well-structured and organised way, and used correct terminologies in appropriate contexts.
1	attempted to organise information and use several correct terminologies, so that with re-reading the meaning becomes apparent.
0	presented an unstructured answer with poor use of terminology.

Breadth (max 2 marks)

Marks	Exemplification
2	refers to at least two points from any three sections each: mutations (A/B/AB/F) + heredity (C) + viruses/bacteria (D) + environmental factors (E) + other factors (G)
1	refers to at least two points from any two sections each: mutations (A/B/AB/F) + heredity (C) + viruses/bacteria (D) + environmental factors (E) + other factors (G)
0	refers to only one point any one of these sections: mutations (A/B/AB/F) + heredity (C) + viruses/bacteria (D) + environmental factors (E) + other factors (G)

[Total: 20]

- 5 (a) The structure of DNA confers stability but also allows for variation. The DNA structure will also allow for its interaction with other biomolecules.

Relate the structure and chemical properties of DNA to its role in evolution.

[15]

Scientific Content (max 16 marks)

DNA – stability - In semi conservation replication for inheritance of alleles to offspring – advantageous traits – affects the allele frequency on population.

A : Double Helix (max 7)

Structure of DNA in DNA Replication: 1. <u>Double stranded DNA molecule held by tgt by H-bonds - increases stability of the molecule – impt as genetic molecule;</u> 2. <u>Double stranded DNA molecule – each strand can be used as template for DNA repair of the other strand;</u> 3. <u>H-bonds between the 2 strands - easily broken –</u>	Role in evolution: (can award if these are anywhere in essay) 6. <u>inheritance / pass on favourable alleles to offspring, which codes of advantageous traits in natural selection;</u> 7. <u>results in an increase in the allele frequency in population (microevolution);</u> 8. <u>transmitting / passing on accurate genetic information from parents to offspring, maintaining the integrity of species over long periods;</u>
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allows <u>unzipping / separation</u> (R: unwinding alone) of <u>2 strands</u> each to act as <u>template</u> in semi-conservation <u>DNA replication</u> (R: mitosis); 4. <u>Nitrogenous bases</u> - allows for <u>complementary base-pairing*</u> – <u>genetically identical daughter molecules</u> / DNA is <u>accurately replicated</u>; 5. <u>Sugar-phosphate backbone</u> – covalent <u>phosphodiester bond*</u> – <u>stability of molecule</u>;	
DNA structure in Transcription: 9. <u>H-bonds between the 2 strands</u> - <u>easily broken</u> – allows <u>separation of 2 strands</u> each to act as <u>template in transcription</u>; 10. <u>Nitrogenous bases</u> - allows for <u>complementary base-pairing*</u> – <u>accurate transfer of codes to mRNA</u> – <u>produce functional protein</u>; 12. <u>Nitrogenous bases</u> – <u>variation in sequence in alleles</u> <u>determines seq of amino acids</u>;	Role in evolution: 11. expression of <u>functional proteins</u> – <u>expressed traits / phenotype</u> – confers <u>selective advantage/ disadvantage</u> for survival/ results in <u>directional selection</u>;

DNA's structure facilitates protein binding and modification through several key features. The double helix shape creates major and minor grooves, which provide binding sites for proteins. Perhaps also there are many parts of DNA which are complementary in shape and charge to DNA binding sites to specific proteins so that they can bind.

Part B : Major/ minor grooves – binding of proteins

1. Helical structure of DNA creates two grooves: a major groove and a minor groove - allows proteins / other molecules to bind and interact;
2. Specific DNA nucleotide sequences determine their conformation and charges in the grooves - making them recognizable complementary DNA binding sites of specific proteins / many proteins bind to DNA in a sequence-specific manner / recognize and bind to specific nucleotide sequences;

Specific processes 3. Allows for <u>specific processes to occur with precision</u> e.g. <u>ref to DNA replication / transcription / gene regulation</u>; Including centromere sequence / enhancer / silencer having specific sequences – allowing respective proteins to bind for various processes	Role in evolution: 4. transmitting / passing on <u>accurate genetic information</u> from parents <u>to offspring</u>, <u>maintaining the integrity of species</u> over long periods; 5. expression of <u>functional proteins</u> – <u>expressed traits / phenotype</u> – confers <u>selective advantage / disadvantage</u> for survival / results in <u>directional selection</u>;
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Recombination Need to have ref to proteins that lead to breaking to phosphodiester bonds - Allows proteins to bind to specific sequence and <u>results in a recombination / restructuring of part of a genome</u>; <u>During meiosis - crossing over</u> occurs between chromatids of homologous chrom - <u>new combinations of alleles</u>; 7b. similar ref to recombination in bacteria; - Transposition of a mobile element from one position to another within a chromosome or between chromosomes;	Role in evolution: <u>increase genetic variation in offspring – prevent species from becoming genetically somatic recombination / prevent species wipe out / extinction</u>;
Somatic recombination <u>Somatic recombination of immunoglobulin genes – diversity of lymphocytes / antibodies</u>;	Role in evolution: <u>selective advantage – survival of fittest – when exposed to different pathogens / prevent species wipe out / extinction</u>;
Need to reference to how <u>DNA structure</u> allows for mutation. Do not just describe mutations. Mutation <u>Grooves allows mutagen to bind / intercalate – causing the mutation / changes in seq of DNA.</u> (<u>Intercalating agents</u> often results in insertion mutations. e.g. <u>ethidium bromide</u> can intercalate between base pairs in the double helix, slightly unwinding the helix and hence increasing the distance between adjacent base pairs);	Role in evolution: Genetic Variation: <u>only process that adds new alleles / introduce new variation within population – variation is raw material for evolution – new traits / new phenotype</u>; <u>mutations can be beneficial, harmful, or neutral in their effect on an organism's traits</u>; Natural Selection: If a mutation provides a <u>selective advantage</u> (e.g., better adaptation to an environment), individuals with that <u>mutation</u> are more likely to <u>survive and reproduce, passing on the advantageous alleles</u> hence trait to their offspring – <u>allele freq increases / survival of the fittest</u>; - over time, this process can lead to accumulation of mutations in DNA and lead to <u>significant changes in populations</u> and even the <u>emergence of new species</u>;
AVP : Associate with histone / scaffold proteins to Form chromosomes - that allows equal separation of sister chromatids / homologous to opposite poles of cells	Same as point 4 & 9.

Form chromosomes – prevent damage;	
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Part C : Chemical properties

allows for interactions / precise binding of proteins and chemical modifications of DNA (phosphate backbone and base composition).

1. Negatively charged phosphate backbone allows proteins e.g. histones to bind through electrostatic interactions with the proteins;
2. DNA can be chemically modified, including methylation of cytosine bases, acetylation and phosphorylation of the bases;

Epigenetics / Gene regulation

3. These modifications can alter chromatin / DNA structure and influence protein binding e.g. methylation of cytosine bases - regulation of gene expression;

Role in evolution:

4. expression of functional proteins – affect expressed traits / phenotype – confers selective advantage / disadvantage for survival / results in directional selection;
5. these epigenetic modifications can be passed on during cell division - subsequent generations, a process known as epigenetic inheritance;

Mutation

6. Methylations, for example, often result in modified nucleotides with altered base-pairing properties and so lead to point mutations;

Role in evolution:

Refer to B

Genetic Variation:

13. only process that adds new alleles / introduce new variation within population – variation is raw material for evolution – new traits / new phenotype;
14. mutations can be beneficial, harmful, or neutral in their effect on an organism's traits;
15. **Natural Selection:** If a mutation provides a selective advantage (e.g., better adaptation to an environment), individuals with that mutation are more likely to survive and reproduce, passing on the advantageous alleles hence trait to their offspring – allele freq increases / survival of the fittest;
16. over time, this process can lead to significant changes in populations and even the emergence of new species;

In summary, the structure of DNA, including its grooves, specific sequences, and chemical properties, allows for the precise binding of proteins. These proteins play crucial roles in DNA replication, transcription, repair, and other cellular processes, making DNA structure fundamental to life.

Communication (max 2 marks)

Marks	Descriptors
2	presented information concisely, in a well-structured and organised way, and used correct terminologies in appropriate contexts.

1	attempted to organise information and use several correct terminologies, so that with re-reading the meaning becomes apparent.
0	presented an unstructured answer with poor use of terminology.

Breadth (max 2 marks)

Marks	Exemplification
2	refers to at least three points from any two sections each: double helix (A) + binding of proteins (B) + chemical properties (C)
1	refers to at least two points from any two sections each: double helix (A) + binding of proteins (B) + chemical properties (C)
0	refers to only one point from any of these sections: double helix (A) + binding of proteins (B) + chemical properties (C)