

2023 JC2 PRELIMINARY EXAMINATION

CANDIDATE
NAME

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CLASS

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INDEX NUMBER

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BIOLOGY

9744/03

PAPER 3
LONG STRUCTURED AND FREE RESPONSE
QUESTIONS

15 SEPTEMBER 2023
FRIDAY

Candidates answer on the Question Paper.
No Additional Materials are required.

2 HOURS

READ THESE INSTRUCTIONS FIRST

Write your name and class on all the work you hand in.
Write in dark blue or black pen.
You may use an HB pencil for any diagrams or graph.
Do not use 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 the spaces provided on the Question Paper.

For Examiner's Use	
1	
2	
3	
4 or 5	
Total	/ 75

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, fasten all your work securely together.
The number of marks is given in brackets [] at the end of each question or part question.

This document consists of **25** printed pages and **3** blank pages.

Section A

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

- 1 Proteins must fold into defined three-dimensional structures to gain functional activity. In the cellular environment, newly synthesised polypeptides are at great risk of misfolding and aggregation. Cells hence engage proteins called chaperones to assist in protein folding.

These chaperones have two roles:

1. They bind to proteins to promote folding.
2. They direct misfolded polypeptides for degradation in the cytosol.

However, polypeptides that are in the midst of folding may be mistaken by chaperones as misfolded proteins and then directed for degradation. Therefore, protein folding needs to be completed quickly to prevent premature degradation.

A recently discovered endoplasmic reticulum (ER) protein complex called S-E complex was found to delay premature degradation of polypeptides that are in the midst of folding. In its absence, approximately 30% of newly synthesised proteins that could otherwise fold correctly are degraded.

Fig. 1.1 illustrates these processes.

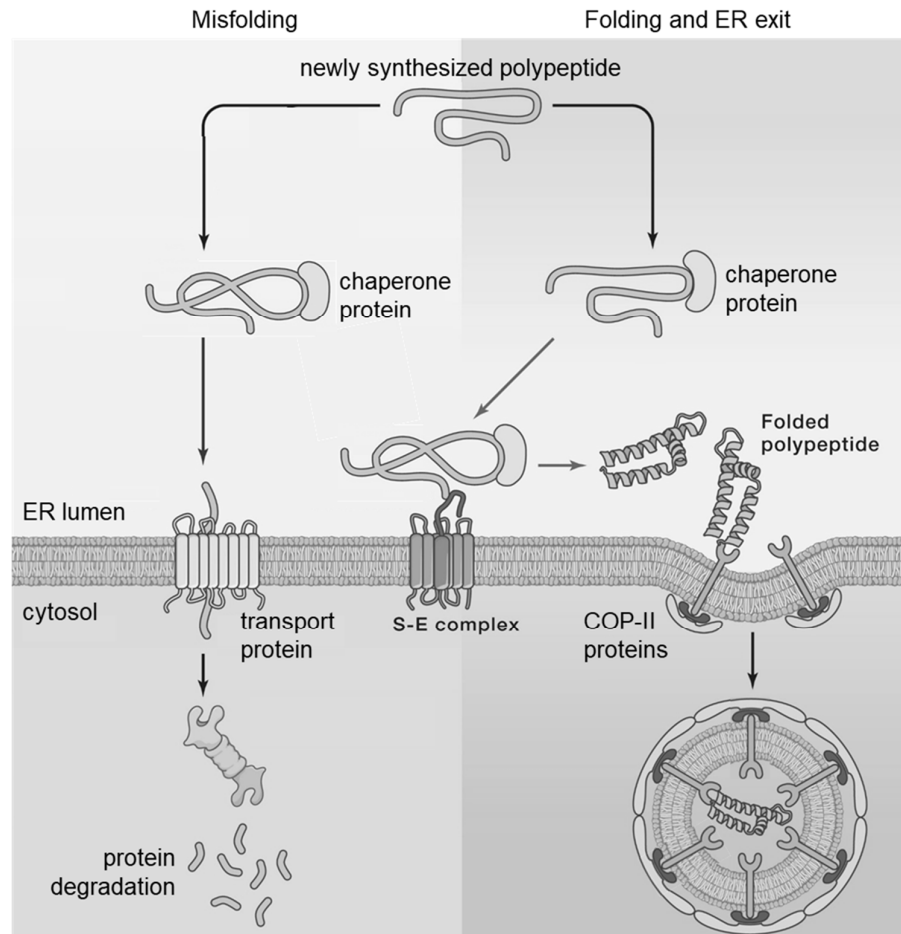


Fig. 1.1

With reference to Fig. 1.1,

- (a) (i) suggest how the S-E complex allows polypeptides to complete their folding.

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- (ii) ER vesicle formation is not a random event but is carefully co-ordinated. Describe how ER vesicle formation is triggered.

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- (iii) explain how unfolded polypeptides in the ER are degraded.

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.....[3]

Misfolded proteins in the ER tend to spontaneously associate with one another to form an aggregate, causing cellular toxicity. Hence it is important that any misfolded protein is immediately degraded and does not remain in the ER for too long.

The presence of the amino acids in Fig. 1.2 in the primary sequence contributes to the misfolded proteins forming an aggregate with one another.

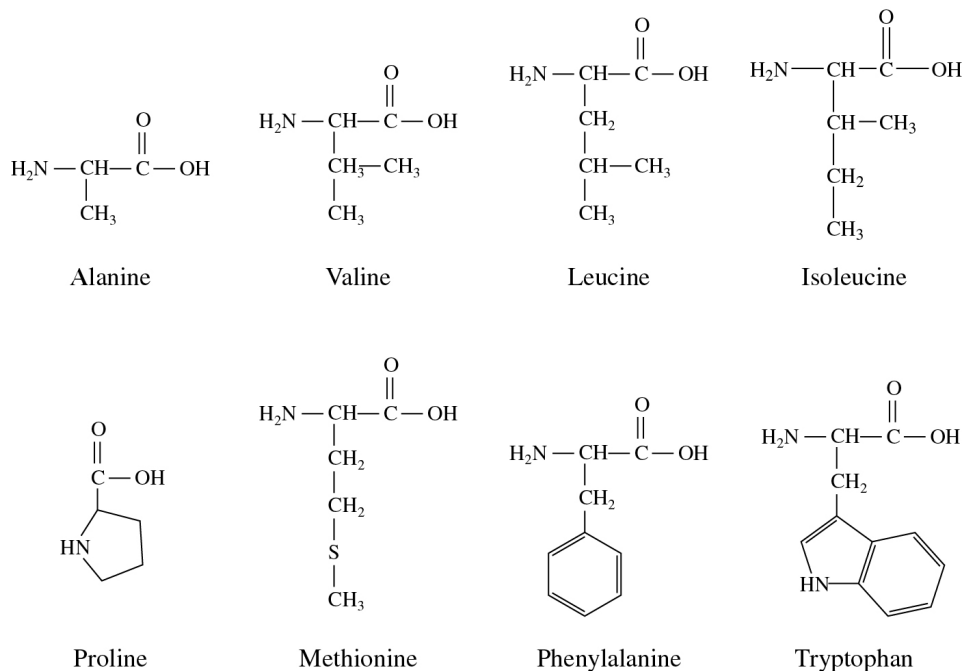


Fig. 1.2

- (b) (i) Explain how the amino acids in Fig. 1.2 contribute to the misfolded proteins forming an aggregate with one another.

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[2]

- (ii) Suggest how accumulation of protein aggregates in the ER can affect the function of the ER.

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[1]

Accumulation of protein aggregates can also cause mitochondrial diseases (MD).

Mitochondria are found in all nucleated eukaryotic cells and are the principal generators of cellular ATP. The mitochondrial circular DNA genome comprises 37 genes, which code for 13 polypeptides for oxidative phosphorylation and the necessary RNA machinery for their translation within the mitochondria.

- (c) (i) It was hypothesised that mitochondria arose when an early ancestor of the eukaryotic cell engulfed an oxygen-using, non-photosynthetic prokaryotic cell.

Explain two pieces of evidence for this hypothesis.

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.....[2]

One form of MD is caused by a mutation of a mitochondrial gene that codes for a tRNA.

- The mutation involves substitution of guanine for adenine in the DNA base sequence.
- This changes the anticodon on the aminoacyl-tRNA carrying leucine (tRNA^{leu}).
- This mutant tRNA^{leu} also recognises the phenylalanine codon, resulting in the formation of a non-functional protein in the mitochondrion.

- (ii) Suggest how the change in the anticodon of a tRNA leads to mitochondrial diseases.

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.....[3]

While some MDs are caused by mutations of mitochondrial genes inside the mitochondria, most MDs are caused by mutations of genes in the cell nucleus that are involved in the functioning of mitochondria. MDs caused by nuclear DNA mutations are autosomal recessive.

All of a person's mitochondria are inherited from their mother via the egg cell.

Two couples, couple **A** and couple **B**, had one or more children affected by a MD. The type of MD was different for each couple.

None of the parents showed signs or symptoms of MD.

- Couple **A** had four children who were all affected by a MD.
- Couple **B** had four children and only one was affected by a MD.

- (iii) Using the information provided, suggest why all of couple **A**'s children had a MD and only one of couple **B**'s children had a MD.

couple **A**

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.....[2]

couple **B**

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.....[2]

Question 1 continues on page 8

It has been shown that curcumin has considerable mitochondria-protective properties that could prevent MDs.

Curcumin is a yellow pigment found in the spice turmeric, which is used in curry powder.

Eight plant species are known to have roots that contain curcumin. All eight species are found in Asia and several of these belong to the genus *Curcuma* in the ginger family (Zingiberaceae).

Fig. 1.3 shows the evolutionary relationships for five of the eight species with roots that contain curcumin. Lines that are not labelled represent other species that do not make curcumin.

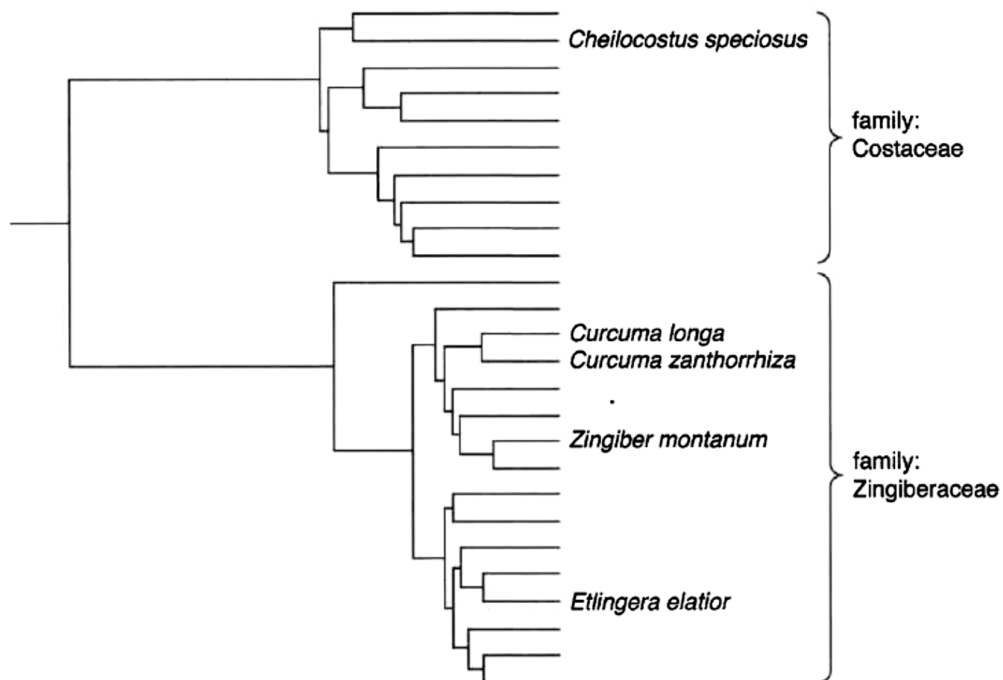


Fig. 1.3

- (d) (i) Name the term used to describe the organisation of species according to their evolutionary relationships, as shown in Fig. 1.3.

.....[1]

- (ii) The five species named in Fig. 1.3 occur in just two of the 400 or more families of flowering plants that exist.

Suggest why the ability to make curcumin is limited to these two families of plants.

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One of curcumin's medicinal properties is also as a potent antivenom against snake bites. However, its efficacy varies across species of snake bites.

The venom of a snake contains many protein toxins which can damage the tissues of a victim who has been bitten. The snake bite can lead to significant disability or death within hours, and a specific antivenom would be necessary for treatment.

The following steps describe how a specific antivenom is traditionally produced.

- The venom of a snake is collected.
- An animal, often a horse, is injected with a controlled quantity of the venom.
- The horse's blood is withdrawn and the antibodies produced in response to the protein toxins are isolated.
- The isolated antibodies are purified and formulated as an injection.

The antivenom produced is effective only against the species of snake from which the venom was obtained.

- (e) (i)** State the type of immunity that is conferred by the antivenom when administered to the snake-bite victim.

.....[1]

- (ii)** Describe how antibodies in the antivenom may reduce the harmful effects of toxins in the snake venom.

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[2]

- (iii)** Explain why a particular antivenom is effective only against a specific species of snake.

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[2]

- (iv) Suggest why an injection containing inactivated protein toxins is not an effective treatment for a snake-bite victim.

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[Total: 28]

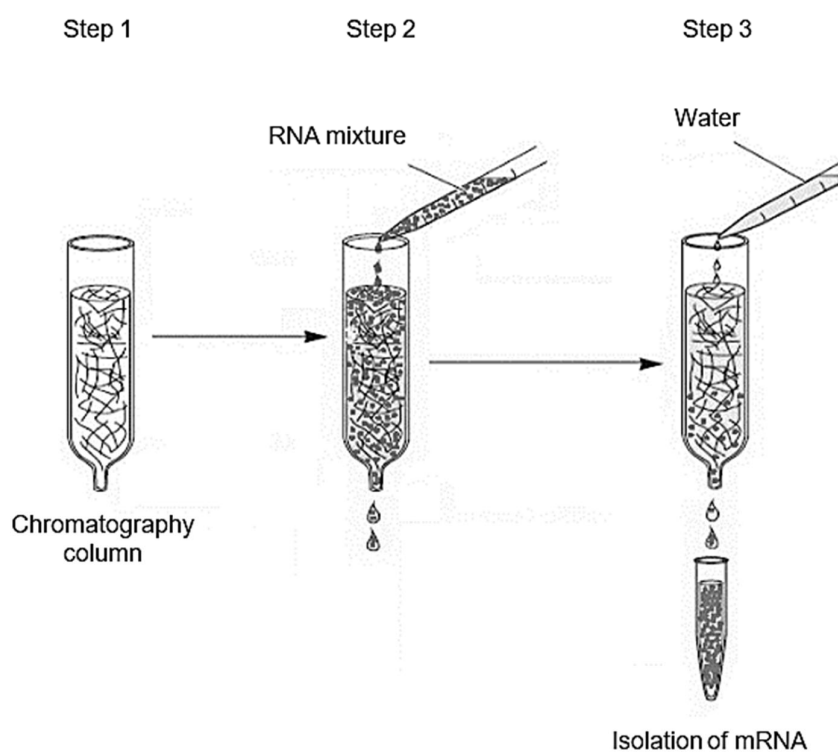
- 2 The sleep-wake pattern describes when, during a 24 hour day, a person is asleep and when they are awake. Sleep-wake patterns are in turn affected by a multitude of factors, including the levels of hormones such as melatonin.

Two examples of sleep wake-patterns are:

- pattern 1 – asleep during the night and awake during the day (normal)
- pattern 2 – asleep during the day and awake during the night.

To identify which genes have their expression changed by a person's sleep-wake pattern, researchers isolated mRNA from a group of volunteers with sleep-wake pattern 1, and the same group of volunteers whose sleep-wake pattern was changed to pattern 2.

mRNA from volunteer samples were isolated from total cytoplasmic RNA, using the following procedure in Fig. 2.1.



Step 1:	Set up a chromatography column which contains short lengths of uracil nucleotides attached to a solid support medium.
Step 2:	Add total RNA mixture to the chromatography column. RNA that do not hybridise with the uracil nucleotides will pass through and leave the column.
Step 3:	Add water to the chromatography column to remove and isolate the hybridised mRNA.

Fig. 2.1

- (a) Explain why the procedure in Fig. 2.1 is effective in isolating mRNA from total cytoplasmic RNA.

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With the isolated mRNA, these researchers then used microarray analysis to determine changes in gene expression due to altered sleep-wake pattern.

Microarrays are used to detect the expression of thousands of genes at the same time. A microarray slide is printed with thousands of spots in defined positions. Each spot contains single-stranded DNA of known sequence, which acts as a probe to detect gene expression.

Fig. 2.2 shows a typical microarray slide.

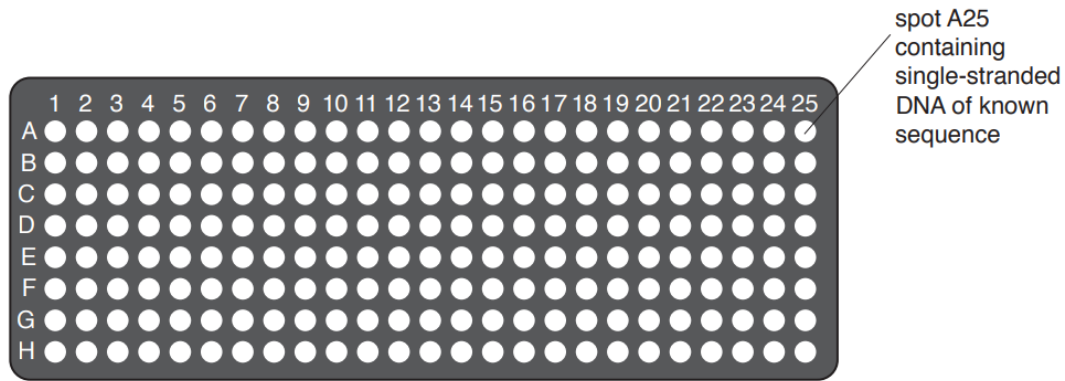


Fig. 2.2

The key steps in this microarray analysis are as follows:

- Each sample of mRNA is converted to complementary DNA (cDNA) and fluorescently labelled.
- Different colour fluorescent dyes are used for the samples taken from before and after the change in sleep-wake pattern.
- The samples are added to the slide and allowed to hybridise onto the DNA on a microarray slide.
- The microarray slide is then scanned to measure the expression of each gene.

- (b) Name the enzyme used to convert each sample of mRNA to cDNA.

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- (c) Suggest how the level of each gene expression is determined.

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[1]

- (d) A summary of the results is shown in Table 2.1.

Table 2.1

sleep-wake pattern	number of genes with increased expression		
	during the day	during the night	all the time
pattern 1	661	733	108
pattern 2	134	95	8

- (i) Suggest and explain how eukaryotic genes can be switched on and off at certain times of day, at the transcriptional level.

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[3]

- (ii) Comment on whether the information in Table 2.1 is useful in concluding how an individual's health is affected by sleep-wake pattern.

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[2]

[Total: 10]

- 3 Reef-building corals are marine invertebrates found in tropical seas and oceans. Each coral consists of a colony of animals called polyps which form by asexual budding.

Each coral polyp has the following structure:

- two layers (inner and outer) of epithelial cells that enclose a central gut cavity
- a non-cellular gelatinous layer
- a thin layer of symbiotic bacteria over the polyp's outer epithelial cells.

Zooxanthellae are a group of unicellular photosynthetic eukaryotes that live within the coral polyp's inner epithelial cells. They have chloroplasts and photosynthetic pigments with structures similar to those in plant cells. Without zooxanthellae, coral polyps are white in colour (bleached).

Fig. 3.1 shows a transverse section through a polyp and a close-up view of a zooxanthellae producing hydrogen peroxide (H_2O_2) and reactive oxygen species due to damages in its chloroplast when sea temperatures are high.

It was observed that proteins in the chloroplasts were not denatured due to the higher temperature, but membranes became more leaky. Although water was taken in by the chloroplasts at a much faster rate, the rate of oxygen production decreased drastically.

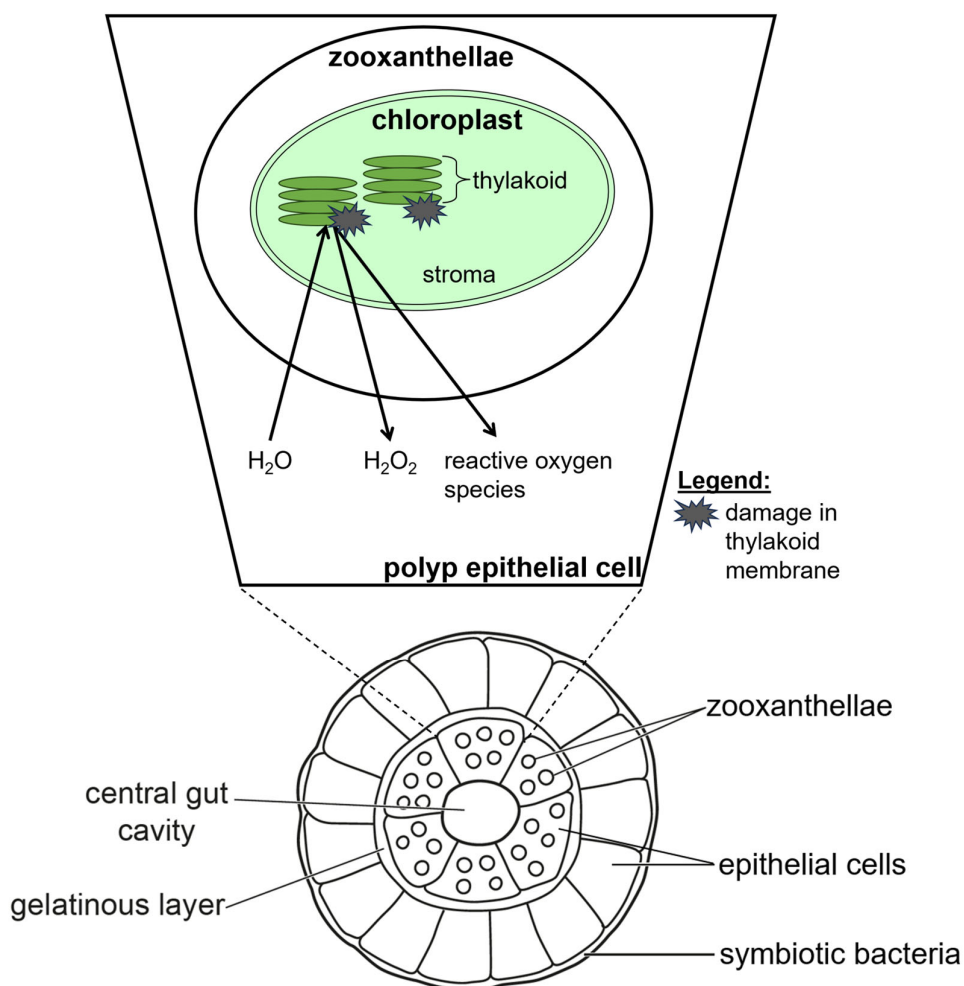


Fig. 3.1

In 2016, higher than usual sea temperatures due to the El Niño effect caused about 65% of Singapore's coral reefs along the fringes of the Southern Islands and in the north-east to be moderately or severely bleached.

(a) Using the information given, including Fig. 3.1,

(i) explain how rising sea temperatures affect photosynthesis in zooxanthellae.

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(ii) suggest why zooxanthellae are expelled by coral polyp epithelial cells when sea temperatures are high.

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Extensive land reclamation in Singapore over the last few decades has severely impacted the water turbidity (extent of water murkiness due to suspended particles) and hence the amount of light available to coral reefs in shallow waters.

Scientists measured the percentage of coral cover and water turbidity to the south of mainland Singapore over a period from 1988 to 2010. Some of the results are shown in Fig. 3.2.

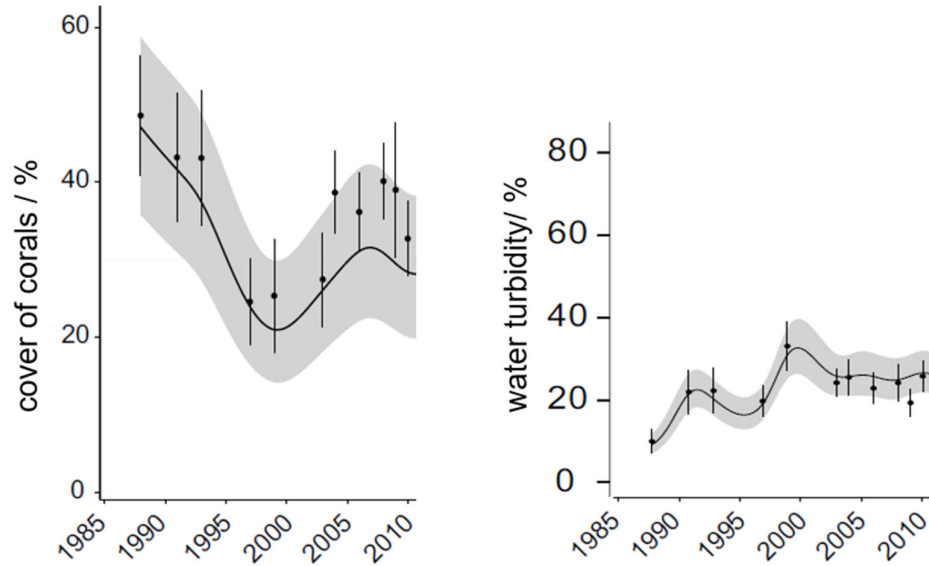


Fig. 3.2

- (b) (i)** Describe the relationship between percentage coral cover and water turbidity.

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- (ii)** Suggest why high percentage water turbidity may be protective against coral bleaching.

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- (c) While coral reefs in Singapore have been showing signs of recovery since 2010, the recovery rate has been slow.

Explain why individual coral species have low genetic diversity **and** how this affects their chances of survival in response to environmental change from global warming.

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[Total: 12]

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