H

NATIONAL JUNIOR COLLEGE, SINGAPORE
Senior High 2
Preliminary Examination
Higher 2

CANDIDATE NAME			
BIOLOGY CLASS	2bi2___	REGISTRATION NUMBER	

BIOLOGY

9744/03

Paper 3: Long Structured and Free-response Questions **25 August 2023**

Candidates answer on the Question Paper and Answer Booklet **2 hours**

No Additional Materials are required.

READ THESE INSTRUCTIONS FIRST

Write your name and Biology class 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 Answer Booklet.

The use of an approved scientific calculator is expected, where appropriate.
You may lose marks if you do not show your workings or if you do not use appropriate units.

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

For Examiner's Use	
Section A	
1	**/30**
2	**/10**
3	**/10**
Section B	
4/5	**/25**
Total	**/75**

This document consists of **14** printed pages and **2** blank pages.

Section A

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

1 FoxO1 is a protein found in pancreatic β cells.

Fig.1.1 shows a model of FoxO1.

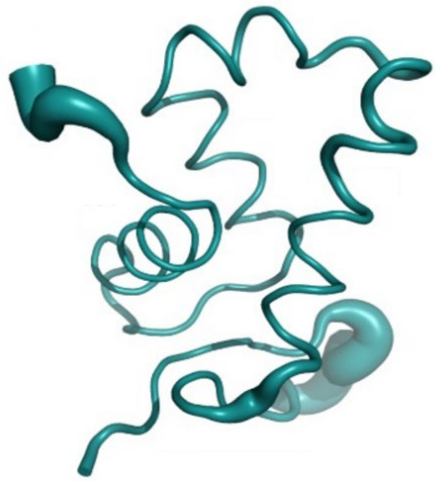

Fig. 1.1

(a) Describe how monomers may be linked together to form the FoxO1 polypeptide chain.

Peptide bonds formed between amino and carboxyl groups;
Condensation reaction;
Removal of water molecules;

[2]

(b) Explain how amino acids that are spaced far apart in the FoxO1 polypeptide chain may be brought together in its 3-dimensional structure.

Stretches of amino acids in the polypeptide chain first adopt/fold/coil into a regular, recurring shape/repeating pattern [OR ref to localized folding of polypeptide backbone into secondary structures];
such as α-helix; [Beta sheet not seen in Figure]
These secondary structures are stabilized by hydrogen bonds formed between the –C=O and –NH groups of the peptide bonds/backbone;
The secondary structures extensively/further folds to form a specific three-dimensional globular structure, bringing amino acids that were spaced far apart in the linear chain to be close together in the tertiary structure;
This tertiary structure is maintained by interactions between the R-groups of amino acids;
Ref to hydrogen bonds, ionic bonds, disulphide bonds and hydrophobic interactions (At least 2);

[4]

Diabetes is often associated with the failure of the β (beta) cells in the pancreas, but it is unclear what actually causes this failure. Some studies have suggested that this failure may be related to FoxO1.

A study was conducted using mice lacking the gene for FoxO1 in β cells (IKO) as well as normal (control) mice. Blood glucose levels after fasting were compared for four groups of mice: young (3 months old) male mice, young (3 months old) female mice, older females (who have had several pregnancies) and aging males (16–20 months). The results are shows in Fig. 1.2.

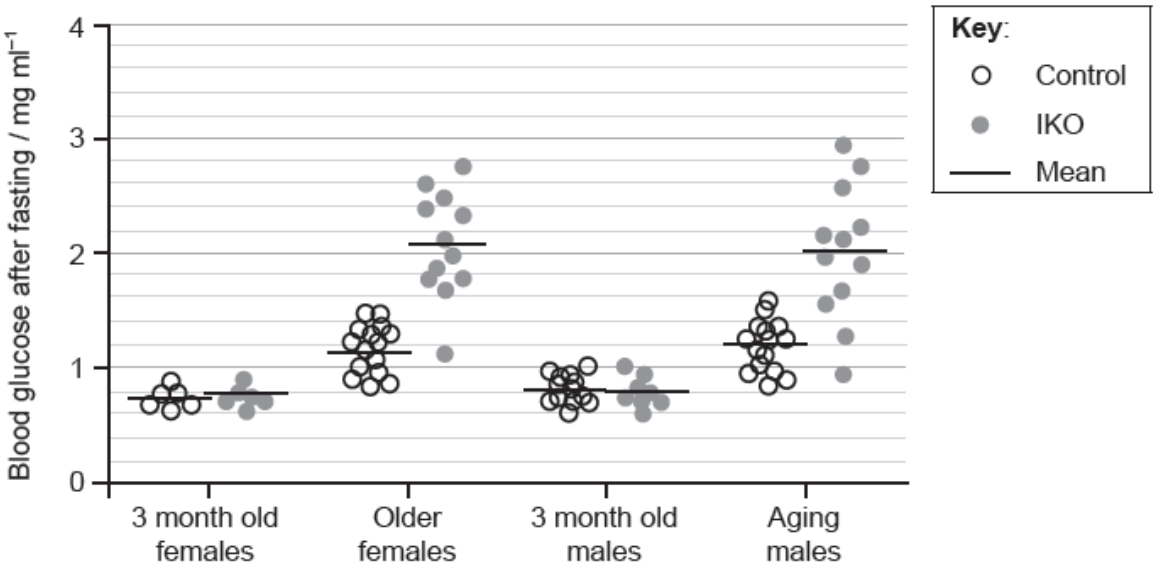

Fig. 1.2

(c) Describe and explain the blood glucose levels after fasting between young control mice and young IKO mice without FoxO1.

similar/same/nearly same (means)/very small difference/both at a low level;
means/averages (all) close to 0.8 mg ml_{-1};
differences not (statistically) significant;
similar/same/nearly same range/spread of data;
lack of FoxO1/IKO/fewer beta cells does not affect/has little effect on blood glucose/sugar;

All marking points are comparisons between control and IKO mice. Do not award marks for comparisons between male and female mice.

[3]

(d) Aging and having pregnancies are considered to be physiological stresses.

Describe and explain the effect of stress on blood glucose levels in control mice.

stress causes marginal increase (in mean blood glucose/sugar);
older mice/males/females / aging mice show slight increase compared to IKO [Ref to appropriate data]
Stress reduces FoxO1 expression only slightly compared to IKO;
Fox01 regulates/maintains sensitivity to changes in glucose levels;

OR

stress causes increase (in mean blood glucose/sugar);
older mice/males/females / aging mice show increase [Ref to appropriate data]
Stress reduces expression of FoxO1;
Fox01 regulates/maintains sensitivity to changes in glucose levels;

[3]

(e) Insulin and glucagon are hormones that are synthesised by pancreatic cells and secreted into the bloodstream in response to changes in blood glucose concentration to maintain homeostasis.

Explain how such newly synthesised hormones are processed and released into the bloodstream by pancreatic cells.

Hormone polypeptide chains begin folding after being inserted into the ER lumen;
Polypeptides undergo initial post-translational modification in the RER;
Transported from RER to cis face of Golgi apparatus via transport vesicles;
Undergo post-translational modification in Golgi apparatus;
Bud out from trans face of Golgi apparatus in secretory vesicles;
[Max 1m for reference to PTM] [Max 3m]

AND
Secretory vesicles fuse with cell surface membrane to release polypeptide chains by exocytosis;

[Max 1m for reference to PTM]

[4]

The levels of pancreatic hormones and β cell mass in older female control mice and older female IKO mice lacking FoxO1 were then investigated. The results are shown in Fig. 1.3.

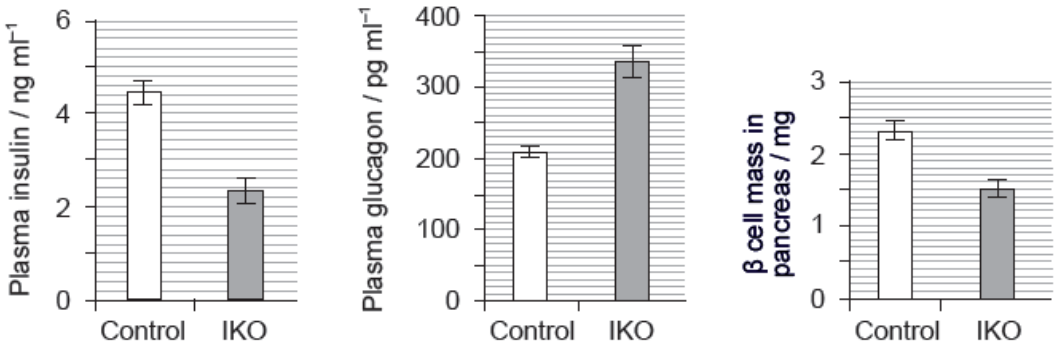

Fig. 1.3

(f) Calculate the percentage difference in β cell mass of the IKO mice compared to the control mice.

Show your workings and give your answer in **three** significant figures.

2.3-1.5;
0.8/2.3 X 100 = 34.78%;

..... % [2]

(g) Outline the relationship between lack of FoxO1 and levels of pancreatic hormones in mice.

lack of FoxO1 (correlates) with low/decreased insulin;
lack of FoxO1 (correlates) with high/increased glucagon levels;

[2]

(h) Using information from Fig. 1.2 and Fig. 1.3, deduce the relative levels of pancreatic hormones in young female control and IKO mice lacking FoxO1
Similar levels of insulin and glucagon/ pancreatic hormones;
[OR]
Slightly lower levels of insulin and slightly lower levels of glucagon compared to control mice;

[1]

To examine whether the changes observed were due to lack of β cell function or change in β cell number, investigators studied the following types of cells:

- still producing insulin
- newly formed β cells
- no longer producing insulin.

The results are shown in Fig. 1.4.

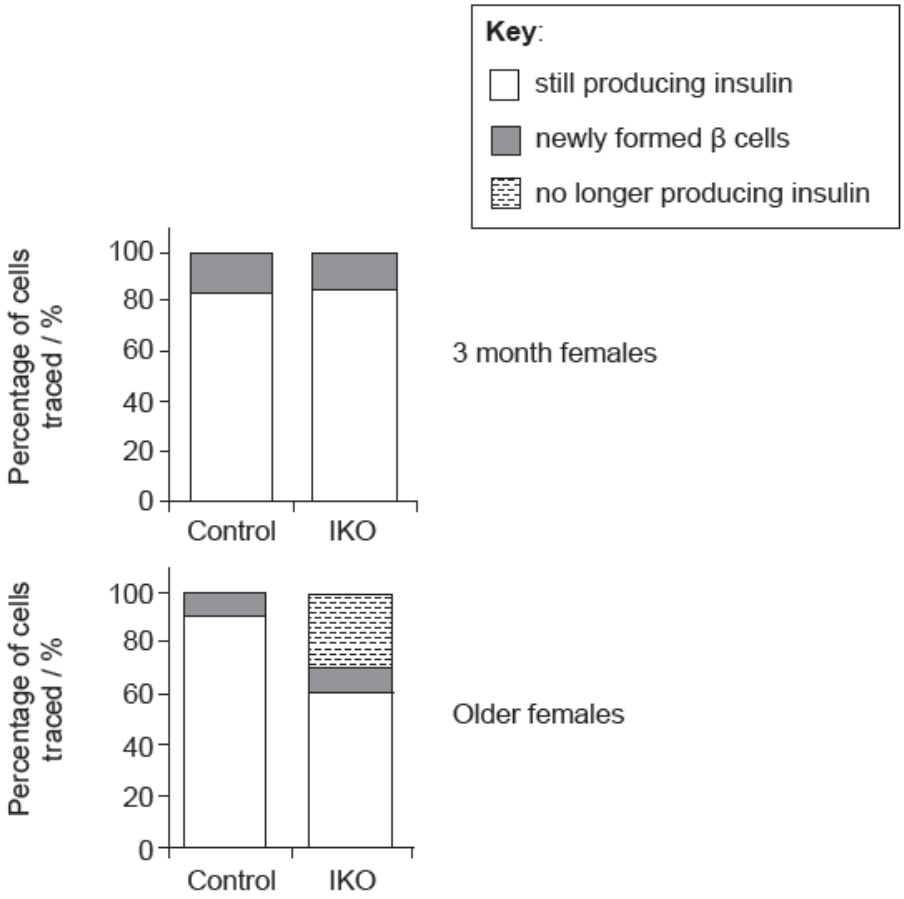

Fig. 1.4

(i) State which group of cells showed the least change in the mice studied.

newly formed β cells;

Accept if newly formed beta cells in IKO mice but not in control mice only.
Reject all answers apart from the first given and any comparisons between IKO and control mice, rather than between younger and older mice.

[1]

(i) Describe the effects of aging on the distribution of cell types in mice.

newly formed β cells fewer/reduced/smaller % (in control/IKO mice);
cells still producing insulin (slightly) more/increased/higher % in controls;
cells still producing insulin fewer/reduced/smaller % in IKO mice;
cells no longer producing insulin only in older IKO mice;

Accept answers where IKO mice are referred to as mice without FoxO1 and control mice are referred to as mice with FoxO1.

All marking points are deductions based on comparing older females with 3-month females and on the assumption that any changes in % are due to aging.

[3]

(j) A hypothesis has been suggested that diabetes is caused by β cells losing their function, not by their death.

Discuss the extent to which the data in Fig. 1.3 and Fig 1.4 support this hypothesis.

supported in older IKO mice/older mice lacking FoxO1 by:
cells no longer producing insulin present (only) in older IKO mice/mice lacking FoxO1;
(Type 2) diabetes/high blood glucose/lower insulin in older IKO mice/mice lacking FoxO1;

not supported by
lower mass of β cells in older IKO mice/mice lacking FoxO1;
no drop/small rise/small change in cells producing insulin in older control mice;

Candidates must make it clear in their answer to (h) whether the data is in support of the hypothesis or against. Evidence can be included for and against. Answers should specify whether the data is from older IKO mice or from older control mice. If the age is not specified in the answer, penalise for one of the marking points but not any others.

[3]

(k) When there are high blood glucose levels, more FoxO1 is found in the nucleus of the cell than in the cytoplasm.

Based on all the information provided, suggest a role for FoxO1.
promotes transcription of/expression of genes;
for differentiation /growth/mitosis/cell division in β cells / for making insulin;
[OR]
represses transcription of/expression of genes;
for making glucagon;

.....

.....

.....

..... [2]

[Total: 30]

2 The HIV/AIDS epidemic in the 2000s has had a very large impact on life expectancy in many African countries.

Table 2.1 shows estimated data for four African countries for:

- the average life expectancy of an individual born in 2002
- the average life expectancy of an individual born in 2002 if there was no HIV/AIDS pandemic
- the percentage of the population testing positive for HIV in 2002.

Table 2.1

Country	Life expectancy / years			Percentage of population testing positive for HIV
	Without HIV/AIDS	With HIV/AIDS	% decrease	
Kenya	65.6	45.5	31	14.0
Malawi	56.3	38.5	32	16.0
South Africa	66.3	48.8	26	19.9
Zambia	55.4	35.3	36	20.0

(a) Using the data shown in Table 2.1, calculate the percentage decrease in life expectancy for Zambia due to HIV/AIDS.

Show your workings and give your answer to the nearest **whole** number.
55.4.-35.3 = 20.1;
(20.1 / 55.4) X 100% = 36.28% = 36% (WHOLE NUMBER);

..... % [2]

(b) Evaluate if there is a correlation between the percentage of the population testing positive for HIV and percentage decrease in estimated life expectancy with HIV/AIDS.

Percentage decrease in life expectancy with HIV/AIDS positively correlates with percentage of population testing positive for HIV;
As percentage decrease in life expectancy increases from 31% in Kenya to 32% in Malawi, percentage of population testing for HIV increases from 14.0% to 16.0% OR
As percentage decrease in life expectancy increases from 31% in Kenya to 36% in Zambia, percentage of population testing for HIV increases from 14.0% to 20.0% OR
As percentage decrease in life expectancy increases from 32% in Malawi to 36% in Zambia, percentage of population testing for HIV increases from 16.0% to 20.0%;
Ref to South Africa as outlier;

OR
No correlation;
As percentage decrease in life expectancy increases from 31% in Kenya to 32% in Malawi, percentage of population testing for HIV increases from 14.0% to 16.0% OR
As percentage decrease in life expectancy increases from 31% in Kenya to 36% in Zambia, percentage of population testing for HIV increases from 14.0% to 20.0% OR
As percentage decrease in life expectancy increases from 32% in Malawi to 36% in Zambia, percentage of population testing for HIV increases from 16.0% to 20.0%;
Ref to South Africa not following trend;

[3]

(c) Explain the role gp120 and gp41 in the reproductive cycle of HIV.
Gp120 Binds to CD receptor on CD4+ T helper cells;
GP41 Mediate virus entry via fusion of the viral envelope with the host cell surface membrane;

[2]

(d) Following infection, $CD4^+$ T cells will synthesise new copies of gp120 and gp41.

Explain how cellular synthesis of gp120 and gp41 can lead to the destruction of infected $CD4^+$ T cells by the immune system.

APCs can digest HIV and present fragments corresponding to GP120 and GP41;
Antigen presentation primes/ Activates T cells with complementary T cell receptor to recognize GP120 and GP41;
Incorporation of viral glycoproteins gp120 and gp41 in host cell surface membrane during viral synthesis changes antigenic surface of cell surface membrane;
Antibodies/ Cytotoxic T cells lead to destruction of infected T cells after binding to GP120 and GP41 on cell surface membranes;
Via Apoptosis/ ADCC;

[3]

[Total: 10]

Question 3 continues on page 11

3 Fig. 3.1 shows how sea surface temperatures have varied over the last century or so at the Great Barrier Reef. 0°C is considered as no change in the sea surface temperature. Rising sea surface temperatures have been suggested as a reason for more frequent coral bleaching events.

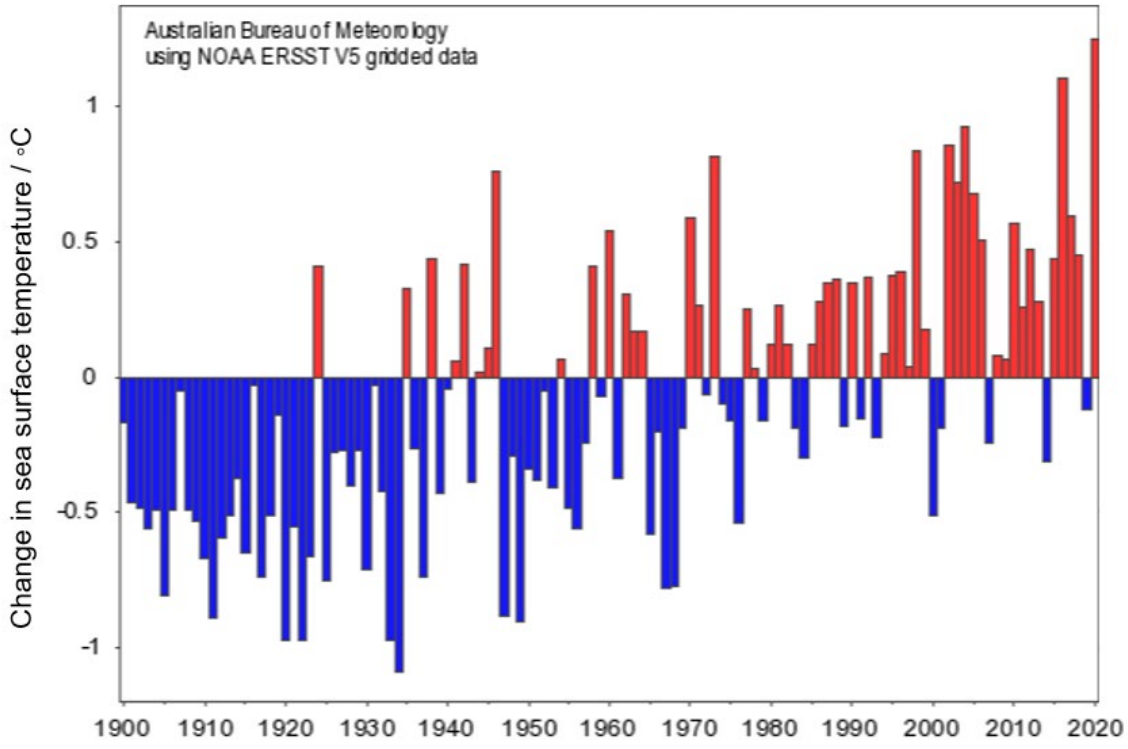

Fig. 3.1

(a) Explain how increase in sea surface temperature may lead to coral bleaching.

Photosynthetic **algae / zooxanthellae** are **sensitive to heat stress**, especially when the temperature rises above a critical threshold;

When photosynthetic **algae / zooxanthallae** **will be expelled by corals**, coral bleaching occurs as the **coral appears white** as it is the natural white colour of the calcium carbonate which forms the coral skeleton;

Examiner's comments: Many students think zooxanthellae will first die or have their enzymes denatured due to heat and thus be expelled out of corals. That is mainly not true as they are expelled when they are still alive. Some students stated that death of corals lead to bleaching while it should be prolonged coral bleaching can lead to corals dying. There are also students who did not refer to zooxanthellae and just highlighted how heat denatures enzymes in corals. Others provided points that are irrelevant, eg. migration of fish, activation of bleaching enzymes etc.

[2]

Fig. 3.2 shows the trend for coral bleaching events globally since 1980.

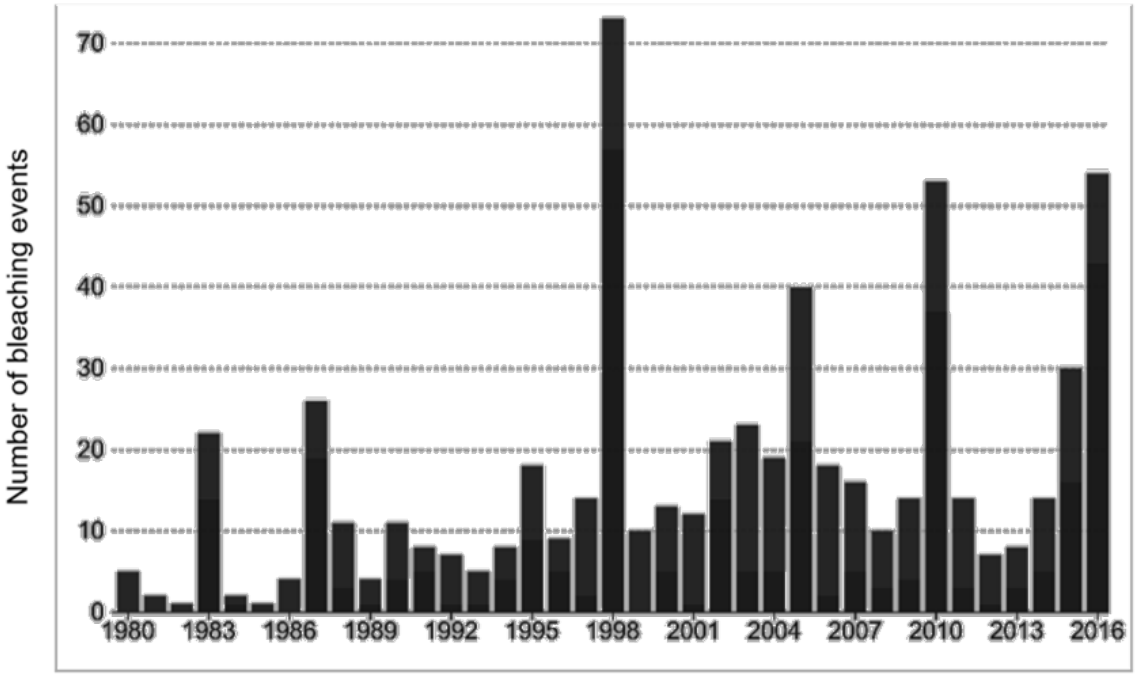

Fig. 3.2

(b) With reference to Fig. 3.1 and 3.2, evaluate if the data provides sufficient evidence that rising sea surface temperatures has led to an increase in frequency of coral bleaching events over the last 40 years.

From **1980 to 1998**, the number of **coral bleaching events are mainly less than 20 when the temperature increase is about 0.25°C**;

After 1998, there have been higher number of **coral bleaching events from about 40 to above 70** when the temperature increase ranges from 0.5 to 1°C;

Change in temperatures ranging from **0.5 to 1°C** are **higher from 1980 onwards** compared with past 80 years;

There is a general increase in temperature of **0.1 to 1.3°C (or 1.1°C)** from **1980 to 2020 (or 2016);**

While an increase in number of coral bleaching events of **5 to 53** took place from **1980 to 2016;**

However, there was an unusually high number of **more than 70 coral bleaching events** in **1998**, which may not be directly due to increase in **temperature of about 0.7°C**;

While there are more than **50 coral bleaching events** in **2010**, the **increasing in temperature** is only **about 0.5°C**;

The relationship drawn from the data compares global coral bleaching events with change in sea surface temperature from Great Barrier Reef. Temperature data from other reefs should also be represented to provide a more accurate evaluation.

AVP: Factors other than temperature can also affect coral bleaching, Bleaching event data before 1980 / from 2016-2020 is missing

Examiner's comments: A number of students either did not quote values from the two Figures or provided incomplete or inaccurate quoting of data. Some students only quoted from a narrow range of years (eg. 1990-2000) or identified specific years (eg. 1998, 2016) without considering the time scale of past 40 years.

[3]

(c) Other than rising sea surface temperature, climate change also leads to an increase in dissolved carbon dioxide in seawater.

Explain, with reference to biochemical details, the direct effects of increasing carbon dioxide on the rate of photosynthesis within corals.

Increasing CO_2 concentration allows more substrate to enter **Calvin cycle / light-independent reaction** by combining with **ribulose bisphosphate (RuBP)** to form two molecules of 3-phosphoglycerate (3-PG);

This process is catalysed by **RuBP carboxylase oxygenase / Rubisco**;

Each molecule of 3-PG becomes 1,3-bisphosphoglycerate, which is then reduced by NADPH to **glyceraldehyde-3-phosphate (G3P)**;

Increase in **G3P / Glucose** leads to **increasing rate of photosynthesis** and provide nutrients for corals;

Examiner's comments: Most students who recognise the requirement for biochemical details are able to score well while some do not.

[3]

(d) Suggest two other ways climate change can potentially affect growth of coral reefs.

Rising sea levels affecting the ability of zooxanthellae to photosynthesise due to lack of sunlight;

Increased carbon dioxide concentrations leading to ocean acidification. This leads to less formation of calcium carbonate, a key component of the shells and skeletons of marine animals;

Extreme weather events such as typhoons directly damaging corals;

AVP: (Requires logical description) Release of pollutants, increase in UV radiation, denaturation of enzymes, increase in algal bloom thus reducing sunlight for corals etc;

R: Corals migrate to cooler waters (like how marine fish does) as they are mainly sessile

Examiner's comments: Some students provide explanation that are inaccurate or are too brief and thus do not demonstrate links between climate change to growth of coral reefs.

.....

.....

..... [2]

[Total: 10]

Section B

Answer **one** question in this section.

Write your answers on the Answer Booklet provided.

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

Your answers must be in continuous prose, where appropriate.

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

4 **(a)** Describe how prokaryotes and mammals are able to detect and respond to changes in supply of sugars. [15]

(b) Discuss the advantages of the response of prokaryotes and mammals to changes in supply of sugars. [10]

[Total: 25]

5 **(a)** Describe how complementarity between biomolecules enable eukaryotic cells to carry out their diverse functions. [15]

(b) Discuss the advantages of having different degrees of complementarity between biomolecules in eukaryotic cellular processes. [10]

[Total: 25]

Answers:

4 (a) Describe how prokaryotes and mammals are able to detect and respond to changes in supply of sugars. [15]

1. Prokaryotes responds to changes in the nutrients available in the environment in order to optimise use of resources / produce enzymes only when necessary;
2. Specific example: ***lac* operon**: genes involved in lactose metabolism under control of a single ***promoter*** and ***operator***; (award once*)
3. *Lac I* codes for active lac repressor which binds to operator region to prevent expression of structural genes in *lac* operon;

Presence of lactose results in (accept absence of lactose)

4. Lac permease coded by *lacY* to transport lactose into bacteria cell;
5. β-galactosidase coded by lacZ to convert lactose to allolactose;
6. allolactose binds to lac repressor to inactivate it;
7. Allowing for structural genes to be transcribed and translated to produce β-galactosidse, lac permease and transacetylase;
8. β-galactosidase break down lactose into glucose and galactose;

Absence of glucose results in (accept presence of glucose)

9. high cAMP;
10. cAMP binds to catabolite activator protein (CAP), activating it;
11. Activated CAP is binds to CAP site;
12. And upregulate / increase the rate of transcription of the structural genes;

13. Eukaryotes eg humans have mechanisms to maintain a constant internal environment despite changes in the external environment;
14. Specific example: maintenance of blood glucose concentration;

Intake of food results in

15. an increase in blood glucose concentration ;
16. which stimulates production of ***insulin*** by ***β-cells*** in the ***islets of Langerhan***;
17. Insulin binds to insulin receptors on ***muscle cells*** and ***liver cells***;
18. Causes increase uptake of glucose into cells and conversion of glucose to glycogen;

Lack of food results in

19. low blood glucose concentration as glucose;
20. which stimulates production of ***glucagon**** by ***α-cells**** in ***islets of Langerhan****;
21. Glucagon binds to glucagon receptors on ***liver cells***; (Reject if muscle cells included)
22. Causes breakdown of glycogen into glucose and release of glucose into blood;

(Signal transduction – award once)

23. Receptor allows transduction from extracellular to intracellular;
24. Multiple steps in pathway allowing for many pts of control / different pathways in different cells to produce a coordinated response;
25. signal amplification to produce a large response;

QWC: at least 1 ref to prokaryotes and 1 ref to eukaryotes;
Max prokaryotes 7m, eukaryotes 7m

(b) Discuss the advantages of the response of prokaryotes and mammals to changes in supply of sugars. [10]

Both prokaryotes and mammals:

1. Benefit from being able to **take advantage of increased sugar supply by increasing metabolic rate** when sugars supply is high;

Prokaryotes:

1. Able to **utilise the sugars that are more easily metabolised / can be directly used for respiration first**;
2. E.g. when both glucose and lactose are present, glucose will be used first;
3. Proteins required for metabolism of sugars are **only synthesised when required**;
4. Able to use sugars present to **multiply/reproduce more**;
5. When sugar supply is low, prokaryotes can regulate their metabolic activities by **entering a state of dormancy to reduce the need for ATP**;
6. AVP;

Mammals:

1. **Glucose transporter** are present in **vesicles** in cells, **ready to be transported to cell membrane to increase cell permeability to glucose** when supply of glucose supply increase;
2. This allows mammals to **respond fast to changes** in blood sugar levels (as they no need to synthesise the proteins);
3. Able to **store sugar** in the form of **glycogen** for **longer term usage**;
4. Can **use glycogen storage during fasting** / when **food is limited**;
5. Can **oxidise fats** for energy when sugar supply is low;
6. Allow **homeostasis** for blood sugar levels / **maintenance** of blood sugar levels within a **narrow range**;
7. AVP;

QWC: at least 1 adv for prokaryotes and 1 adv for eukaryotes;

5 (a) Describe how complementarity between biomolecules enable eukaryotic cells to carry out their diverse functions. [15]

Category	Description
Transport protein + molecule to be transported	A **transport protein** has a **binding site** complementary in shape and charge to **specific for molecule**, Examples: • Channel protein e.g. aquaporin specific for water • Carrier protein e.g. glucose transporter • Pump e.g. Na^+-K^+ pump • Protein complex e.g. nuclear pore specific for nucleotides, exit of mater mRNA, entry of ribosomal proteins, exit of ribosomal subunits • ATP synthase recognise H^+ can diffuse through its channel to form ATP from ADP and Pi; • Haemoglobin transport to oxygen in blood;
Enzyme + substrates	**Enzyme active site** is complementary in shape and charge to **substrate**; Examples: • DNA polymerase – active site can bind to deoxyribonucleotides, • catalyses formation of phosphodiester bonds between adjacent DNA nucleotides to synthesise new strand (DNA replication); • DNA ligase – active site can bind to deoxyribonucleotides, • forms a phosphodiester bond between two DNA fragments/ sealing nick; • RNA polymerase – active site can bind to ribonucleotides; • and catalyses formation of phosphodiester bonds between adjacent RNA nucleotides of newly synthesised strand; • Amino acyl tRNA synthetase – active site complementary to an amino acid and tRNA molecule; • Ribosome – Aminoacyl (A) and Peptidyl (P) sites are complementary to amino acyl tRNA • Peptidyl transferase – form peptide bonds between amino acids; • Cytochrome oxidase – reduces O_2 to water in the final step of electron transport chain in mitochondria; • $NADP^+$ reductase - reduces $NADP^+$ to NADPH in the final step of electron transport chain in chloroplast;
Protein + **D**NA	Proteins may have **DNA binding domains / sites** that are complementary in shape and charge to **specific DNA sequence**; Examples: • TATA binding protein – TATA box; • Recruiting general transcription factors and RNA polymerase; • RNA polymerase – promoter sequence; • Initiating RNA synthesis/transcription; • Activators – specific enhancer sequences; • to increase frequency of transcription; / promoting assembly of transcription initiation complex; • Repressors – specific silencers sequences • to decrease frequency of transcription/ inhibiting assembly of transcription initiation complex; • Histone deacetylases – recognize and bind to methylated DNA;

	• to condense chromatin (A: converse) results in gene silencing/ no gene expression;
Protein + **p**rotein	Proteins may have protein binding domains / sites that are complementary in shape and charge to other protein molecules; Examples: • Mediator proteins allow specific transcription factors to interact with general transcription factors
Base **p**airing	**Adenine pairs with thymine in DNA / uracil in RNA** and **guanine pairs with cytosine** by complementary base pairing; Examples: • Semi-conservative DNA replication using one parental DNA strand to synthesise a complementary daughter DNA strand; • Transcription using DNA template strand to synthesise RNA; • Spliceosome has snRNA which recognises splice sites on RNA to carry out splicing; • Translation involves complementary base-pairing between codon of mRNA and anticodon on tRNA; • Telomerase has a template RNA which is complementary to the 3' overhang of telomeres, allowing the elongation of telomeres;
Ligand + receptor	Ligand has a complementary shape that allows it to fit precisely into (extracellular domains of) receptor molecules on target cells; in order to **transmit external signal into cell** when specific molecules (ligands) bind to them / convert extracellular signal to an intracellular signal; Examples: • Cell surface receptors: has an extracellular ligand-binding site binding to hydrophilic ligand, • Binding of ligand causes conformational change in intracellular domain of receptor; • GPCR: Ligand binding to GCPR causes conformational change in intracellular domain of receptor allowing it to bind to and activate G protein; • RTK: Ligand binding to GCPR causes dimerisation and conformational change in intracellular domain of receptor, leading to activation of tyrosine kinase for autophosphorylation;
Immune system	Immune cells have **specific receptors** that can **recognise pathogens** to **eliminate** them; Examples: • Pathogens has pathogen-associated molecular patterns (PAMPs) which can be recognised by pattern recognition receptors (PRRs) on innate immune cells; • E.g. Neutrophils recognise pathogens and carry out phagocytosis; • T cell has specific T cell receptor that recognises and binds to peptide-MHC complex presented on antigen presenting cells / naïve B cells / infected cells; • Helps to trigger cell proliferation/clonal expansion and differentiation of naïve T cells / B cells; • Antibodies have unique antigen binding site that can bind to epitope of antigens; • Antibodies functions to neutralise pathogens / toxins; • Fc region of antibodies can be recognised by Fc receptors on neutrophils / natural killer cells, facilitating opsonisation / antibody-dependent cell-mediated cytotoxicity;
QWC: 3 different categories + overarching principle for each category Max 2m for examples in each category;	

(b) Discuss the advantages of having perfect complementarity between biomolecules in some cellular processes and less perfect complementarity between biomolecules in other cellular processes in eukaryotes. [10]

Perfect / High complementarity

1. Perfect complementarity allows for **precise matching** of biomolecules enables **highly specific interactions** / **prevent unwanted interactions** between biomolecules that share similar structure;
2. **Lock-and-key hypothesis** where shape of **enzyme active site** is **exactly complementary to shape and charge of substrate / enzyme has absolute specificity to one substrate only**;
3. The absolute specificity of enzyme enables enzyme to **hold substrates in precise orientations**, increasing the catalytic efficiency;
4. **Precise matching of substances to transport proteins** ensures that **only the specific substance can be transported** into/out of cells/organelles;
5. The precise transport of substances allows **maintenance of relatively constant internal concentration of ions and molecules / osmotic pressure**;
6. Perfect complementarity facilitates precise **cell-cell signalling / communication / recognition / adhesion** in multicellular organisms, crucial for **proper tissue organisation / development / coordinated physiological responses**;
7. **Affinity maturation** of B cells resulting in **antibodies** with **increased affinity for specific antigen**;
8. High affinity between antibodies and antigen enables a **targeted and stronger immune response**;
9. Perfect complementary interactions between **proteins and specific nucleotide sequence** enables **tight regulation of gene expression which is needed for cell/tissue specialisation/differentiation**;
10. Perfect complementary **base-pairing** are **more stable**, maintaining the **structural integrity of DNA double helix**;
11. Perfect complementary **base-pairing** ensuring **high fidelity / accuracy in transmission of genetic information during replication and transcription**;

Imperfect / Lower complementarity

1. **Induced-fit hypothesis** where the **initial enzyme active site is not exactly complementary to substrate**, binding of substrate results in a slight **conformational change** of the enzyme which leads to a **precise fit** of the substrate to the active site;
2. Imperfectly complementarity enables enzyme to bind to a **range of substrates** with similar functional groups / bonds, increasing the catalytic efficiency;
3. **Innate immune cells can recognise general patterns found on a wide range of pathogens** and elicit an immune response against them;
4. Imperfectly complementarity allows **reversible binding**, ensuring that **signals are transient** / **preventing overstimulation** due to irreversible binding;
5. Imperfectly complementarity in **base-pairing** allows for **mutations** in **genetic information during replication**;
6. (Mutations) provides **variation** which allows eukaryotes to **adapt to changing environment** by **natural selection**, enabling **gradual evolution**;
7. **Wobble base** effect where the **pairing of the third base of the codon in mRNA with the anticodon of tRNA is not perfect**, resulting in redundancy / degeneracy of genetic code;
8. **Redundancy / degeneracy of genetic code** allows for **more than one codon to code for the same amino acid**;
9. (Redundancy / degeneracy of genetic code) provides a level of **error tolerance** which can help **mitigate the impact of mutations**, ensuring that **essential functions can still be carried out to some extent**;

QWC: at least 1 perfect/high complementarity and 1 imperfect/lower complementarity;

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