H2

ANDERSON SERANGOON JUNIOR COLLEGE
HIGHER 2
ANSWERS

2022 JC2 PRELIMINARY EXAMINATION

CANDIDATE NAME							
CLASS					INDEX NUMBER		

BIOLOGY 9744/03

PAPER 3
LONG STRUCTURED AND FREE RESPONSE QUESTIONS

15 SEPTEMBER 2022
THURSDAY

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.

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.

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

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 **13** printed pages and **1** blank page

Section A

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

1 Lactose is a disaccharide found in milk. Lactase, an enzyme found in mammals and some fungi, catalyse the breakdown of lactose.

The lactase enzyme is made up of 4 identical polypeptide chains. In humans, molecules of lactase are embedded in the cell surface membrane of epithelial cells lining the small intestine. As the lactose molecules float by in the lumen, they are broken down.

(a) Explain how the polypeptide chains in lactase are held together and how they interact with the cell surface membrane.

How polypeptide chains are held together

1. by **R group interactions** such as 2 named bonds: **ionic bonds**, **hydrogen bonds**, **hydrophobic interactions** (accept **disulfide bonds)**

how they interact with the cell surface membrane

2. Part of polypeptide chain with **polar/charged (acidic and basic) / hydrophilic** amino acids are able to associate with **polar/ charged/ hydrophilic** phosphate head of phospholipids via **hydrogen/ ionic/ bonds.**
Reject polar/ charged protein

3. Parts of polypeptide chain with **non-polar/ hydrophobic** amino acids associate with **non-polar/ hydrophobic** fatty acid tails of phospholipids via **hydrophobic interactions** [3]

(b) The products of lactose digestion, glucose and galactose are actively absorbed by intestinal epithelial cells. This absorption is carried out by the sodium-glucose linked transporter (SGLT).

SGLT is a secondary active transporter that works together with sodium-potassium (Na^+-K^+) pump. SGLT transports glucose and galactose concurrently with sodium ions (Na^+) into the intestinal epithelial cells. This transport of glucose and galactose uses the driving force generated by the sodium ion gradient created by the Na^+-K^+ pump.

Fig. 1.1 illustrates the transport process.

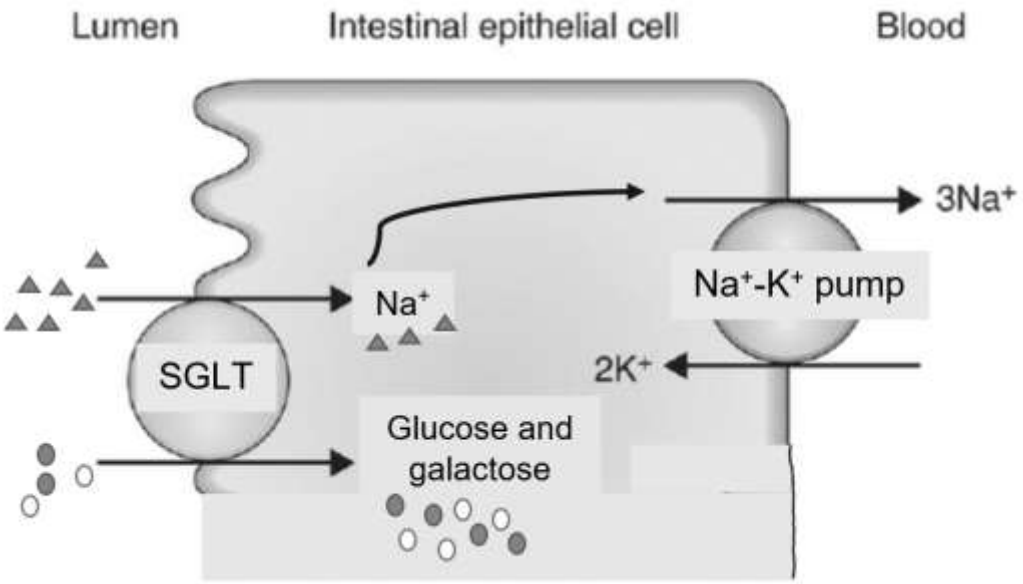

Fig. 1.1

Cyanide is a poison that binds with cytochrome oxidase, one of the electron carriers in the mitochondrial membrane.

It has been observed that the absorption of glucose and galactose from the lumen of the small intestine is reduced if the intestinal epithelial cells are treated with cyanide.

Using Fig. 1.1 and all the information provided, explain why absorption of glucose and galactose from the lumen of the small intestine is reduced when the intestinal epithelial cells are treated with cyanide.

1. Less **ATP** produced via **oxidative phosphorylation**
2. **less** sodium ions **actively transported** out of intestinal cells via **sodium potassium pump**
3. high sodium concentration inside intestinal cells means **less steep sodium concentration gradient** across the intestinal membrane
4. less **diffusion** of sodium into cell via SGLT means less driving force generated for the
5. less **active transport** of products of lactose digestion into cells, against their concentration gradients

[4]

(c) Many human adults do not produce lactase and are lactose intolerant. This means they cannot digest lactose. Lactose intolerance leads to side-effects such as abdominal pain after eating food containing lactose

Scientists have investigated ways to produce low-lactose cow's milk from normal cow's milk for people who are lactose intolerant. One method involved extracting lactase from fungi and mixing the extracted lactase with normal cow's milk. This method is, however, ineffective because one of the products of lactose digestion, galactose, is an inhibitor of lactase.

(i) Explain the effect of galactose on lactase activity.

1. Galactose has a **shape complementary** to **allosteric site/ site away from active site** on lactase
2. Binds (to allosteric site)and **change** the **shape of the active site** → no longer **complementary to substrate lactose**
3. Less **successful / effective collisions** between lactase and lactose → less **ES complexes** formed per unit time (reduces lactase activity)

Reject competitive inhibition because questions hinted "production inhibition"

Accept allosteric inhibition [2]

(ii) Explain why product inhibition is useful when lactase is acting as an intracellular enzyme in fungi cells but can be a disadvantage when extracted lactase is used free in solution for the production of low-lactose cow's milk.

(any 2)
1. *idea of* control / maintaining balance / efficient metabolism: e.g. if, (enough) glucose / galactose / monosaccharides, present then no need for, uptake / breakdown, of lactose
2. avoids osmotic problems as there is no build-up of monosaccharides

accept converse for extracted lactase

[2]

Another method of producing low-lactose cow's milk involved immobilising extracted lactase within alginate beads and putting in high-lactose cow's milk. As the high-lactose cow's milk comes into contact with the alginate beads, the immobilised lactase hydrolyses the lactose. Fig. 1.2 shows the set-up.

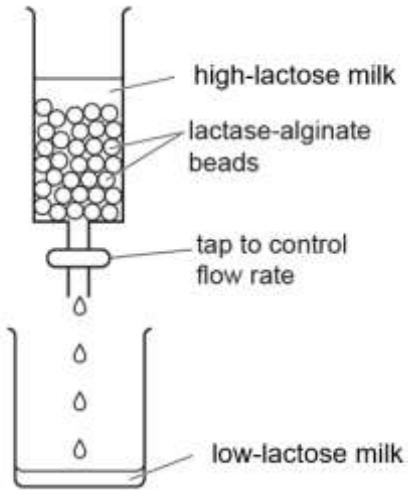

Fig. 1.

(iii) Suggest how using immobilised lactase for the production of low-lactose cow's milk helps to reduce the problem of product inhibition.

1. Galactose / end-product inhibitor and lactase kept separated

2. Galactose/ end-product inhibitor removed immediately

Accept idea of enzyme are kept inside the beads because opening of the tape ensures that only product will leave / Keeping the enzymes on the beads ensure it will not flow out together with the product when the tap is opened

But reject if answer talks about objective is to ensure enzyme can be re-used. [1]

(d) A company producing low-lactose cow's milk carried out an investigation to study the effect of drinking normal high-lactose cow's milk and the company's own processed low-lactose cow's milk.

Fig. 1.3 shows the results of the investigation which compares:

- the effects on 50 lactose-intolerant volunteers of drinking normal cow's milk
- the effects on the same 50 lactose-intolerant volunteers of drinking low-lactose cow's milk
- the effects on a control group of 15 volunteers, who were not lactose intolerant, of drinking normal cow's milk.

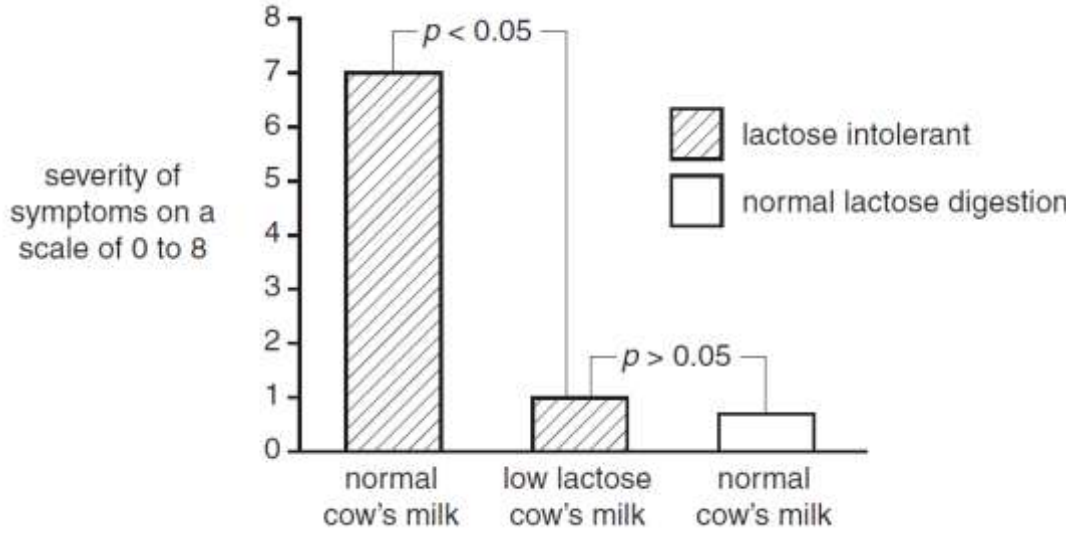

Fig. 1.3

The company claimed that their processed low-lactose cow's milk is suitable for consumption by lactose-intolerant individuals.

With reference to the probability (*p*) values shown in Fig. 1.3, comment on the validity of the claim.

Valid because (max 3)

1. statistical test **, t-test**, done

2. The severity of symptoms caused by (lactose-intolerant volunteers) drinking low lactose cow's milk, level 1, is **significantly lower** than that caused by drinking normal cow's milk, level 7
   Accept converse

3. (This is based on t-test) where the **probability** that **the differences** in severity of symptoms between drinking normal cow's milk and low lactose is **due to chance**, is low, less than 5% *reject 0.05%.*

4. There is **no significant difference** in severity of symptoms between lactose-intolerant people drinking low lactose milk, level 1 and non-lactose-intolerant people drinking normal milk, level 0.6-0.7

5. (This is based on t-test) where the **probability that chance caused the differences** in severity of symptoms is high, more than 5% *reject 0.05%.*

Invalid because (max 3)

6. self-reporting symptoms / subjectivity ; A qualitative / semi-quantitative/ different tolerance to pain *(reject merely saying severity of symptoms varies from person to person)*
7. small sample size / only 15 in the control group ;
8. variation between individuals e.g. age, gender, dietary and medical history, etc.
9. Lack of data e.g. did not have information on whether volunteers consumed other food during investigation
   Note: refrain from suggesting ideas on how to improve the experiment: e.g. having a diet log --- question asked for limitations
   Reject vague/ limitations with no basis e.g. experiment cannot be easily replicated ,

Accept lack of other controls e.g. no data on lactose tolerant peoples consuming low lactose cow's milk

[5]

(e) Scientists have found evidence of natural selection in humans.

- Originally, in human populations it was only babies and children that needed to digest lactose. The gene coding for the enzyme lactase (*LCT* gene) was switched off before adulthood.

- Today, in many populations, only some adult individuals have lactose intolerance.

- A mutation has been identified that keeps the *LCT* gene switched on. An adult who has this mutation is able to digest lactose. This is called lactase persistence (which means lactose tolerance) .

- Lactase persistence increased in populations in Europe several thousand years ago. The increase in lactase persistence in Europe coincided with an increase in farming of cows for milk.

(i) Natural selection has caused an increase in lactase persistence in human populations.

State the type of selection that has caused this increase.
Directional selection [1]

(ii) Explain why there was selection for lactase persistence in humans several thousand years ago.

1. (within the population) **genetic variation/ DNA mutation** (in the LCT gene/ control of LCT gene expression) causing phenotypic variations where some individuals have the ability to digest lactose in adulthood

2. Presence of (only) lactose / milk (products) as food / limited supply of food with only lactose/ milk product as main source of food, acts as a **selection pressure**
   Reject increase farming for milk act as selection pressure

3. Adult individuals who can digest lactose/ milk (products) have **selective advantage** because they can acquire adequate nutrients, hence **selected for**

4. These individuals more likely to, **survive & reproduce** and **passed on** the (mutated) advantageous allele (or control element) *(reject lactase persistence)* to their **offspring**

5. over, time / many generations, the **allele frequency increased**

[4]

The conclusion that lactase persistence is evidence of recent human evolution is further supported by a study. This correlates lactase persistence allele frequency with fresh milk consumption and reliance on livestock (pastoral and non-pastoral populations) in Europe. The result of this correlation study is represented in Fig. 1.4.

- Squares (■)and triangles (▲) represent pastoral populations with high (> 0.6) and low (< 0.4) lactase persistence frequency respectively.
- Non-pastoral populations are represented by diamonds (◆) .
- Pastoral populations raised livestock such as cattle and goats for food while non-pastoral populations grow crops for food.
- Allele frequency is calculated by dividing the number of times the allele of interest is observed in a population by the total number of copies of all the alleles at that particular genetic locus in the population.

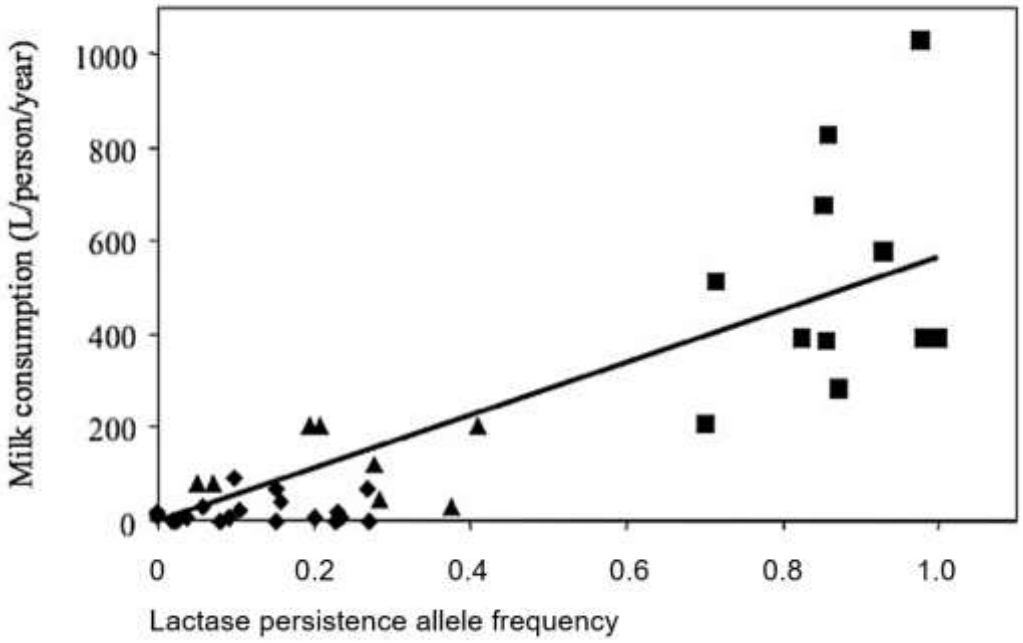

Fig. 1.4

(iii) The study concluded that direct fresh milk consumption has a stronger correlation with lactase persistence than reliance on livestock.

Use information from Fig. 1.4 to justify this conclusion.

1. idea of line graph showing lactase persistence is **positively correlated**/ increases with milk consumption (quote relevant data)

   as x-axis allele frequency increase, milk consumption y-axis increase (reject or accept?)

   Why reliance on livestock is weaker
2. idea of even though same reliance in livestock (pastoral) but lactase persistence varies with milk consumption: pastoral populations with high milk consumption of more than 200L/per person/per year had high lactase persistence frequency (> 0.6) while the pastoral populations with [2]

low milk consumption had low lactase persistence frequency (< 0.4) also at the same level as non-pastrol

(iv) The mutation causing lactase persistence does **not** occur in the *LCT* gene.

Suggest and explain where the mutation that causes lactase persistence may occur.

Lactase persistence = LCT gene is being expressed instead of being switched off in adulthood

1. Mutation within the gene encoding for **repressors** controlling expression of **LCT**
2. → binding site no longer **complementary in shape** to silencer→ transcriptionally active

OR
1. Mutation within the **silencer** controlling expression of **LCT**
2. → DNA sequence no longer **complementary shape** to **binding site** of repressor → transcriptionally active

OR
1. Mutation within the gene encoding for **activators** controlling expression of **LCT**
2. → binds with higher affinity/ permanently to enhancer → transcriptionally active

OR
1. Mutation within the enhancer controlling expression of **LCT**
2. → binds with higher affinity/ permanently to activator → transcriptionally active

OR
3. Mutation within the **promoter** of **LCT**
4. → binds with higher affinity/ permanently to RNA polymerase → transcriptionally active

[2]

(f) In bacteria, the enzyme β-galactosidase breaks down lactose. β-galactosidase is an inducible enzyme but lactase is not.

(i) Explain what is meant by an inducible enzyme.

1. Gene expression is controlled by the **substrate** of the **catabolic** pathway which functions as an **inducer**.

2. Transcription of structural genes is usually turned off but can be stimulated by the presence of an / Production of enzyme occurs/ is increased when lactose is present.

[2]

(ii) Describe **two other** differences between the transcriptional control of β -galactosidase and lactase genes in bacteria and human cells.

	Eukaryotes(humans)	Bacteria
Effect of promoter	Each gene (involved in lactose metabolism) is controlled by a single promoter	Many / all 3 genes involved in the lactose metabolism are controlled by the same/single promoter
Product of transcription	Monocistronic mRNA	Polycistronic mRNA
Location of regulatory sequences involved	Regulatory sequences /e.g. enhancer and silencer /control elements involved in controlling rate of transcription may be distal from gene	Regulatory sequences / operator/ CAP binding site involved in control of transcription is proximal/ near to the genes under its control

[2]

[Total: 30]

2 **(a)** Name the pathogen that causes tuberculosis (TB).

Mycobacterium tuberculosis [1]

(b) Antibiotics are drugs which are very important in the treatment and cure of some diseases, including TB.

(i) Describe the modes of action of antibiotics.

1. Penicillin / competitive inhibitor binds to **transpeptidases/ enzymes** involved in **bacterial cell wall synthesis**
2. inhibit **transcription** by binding to **RNA polymerase**
3. block the access of **peptidyl-tRNAs to the ribosome** → subsequently blocking **translation elongation**
4. blocking access of **aminoacyl-tRNAs to the ribosome**.
5. induce an alteration in the conformation of the complex formed between an mRNA codon and its activated aminoacyl-tRNA at the ribosome, promoting tRNA mismatching which can result in protein mistranslation.

[2]

(ii) Antibiotic treatment of active TB is done with a combination of several antibiotics that are taken over a period of about nine months.

Suggest why the antibiotics used to treat TB are taken in combination over a long period of time.

Combination

1. (bacteria likely to be) resistant to (at least) one antibiotic (so useless) → less likely to be resistant to all

Long time (max 1)

2. Prevent development of antibiotic resistance by ensuring all bacteria are killed
3. Long time needed because bacteria are often found inside macrophages, difficult for antibiotics to access [2]

(c) Fig. 2.1 shows the number of deaths from TB and the number of new cases of TB from 1925 to 2000 in Canada.

Antibiotics, such as streptomycin, were introduced in Canada from 1940.

Vaccine for TB was introduced in Canada for use from 1948.

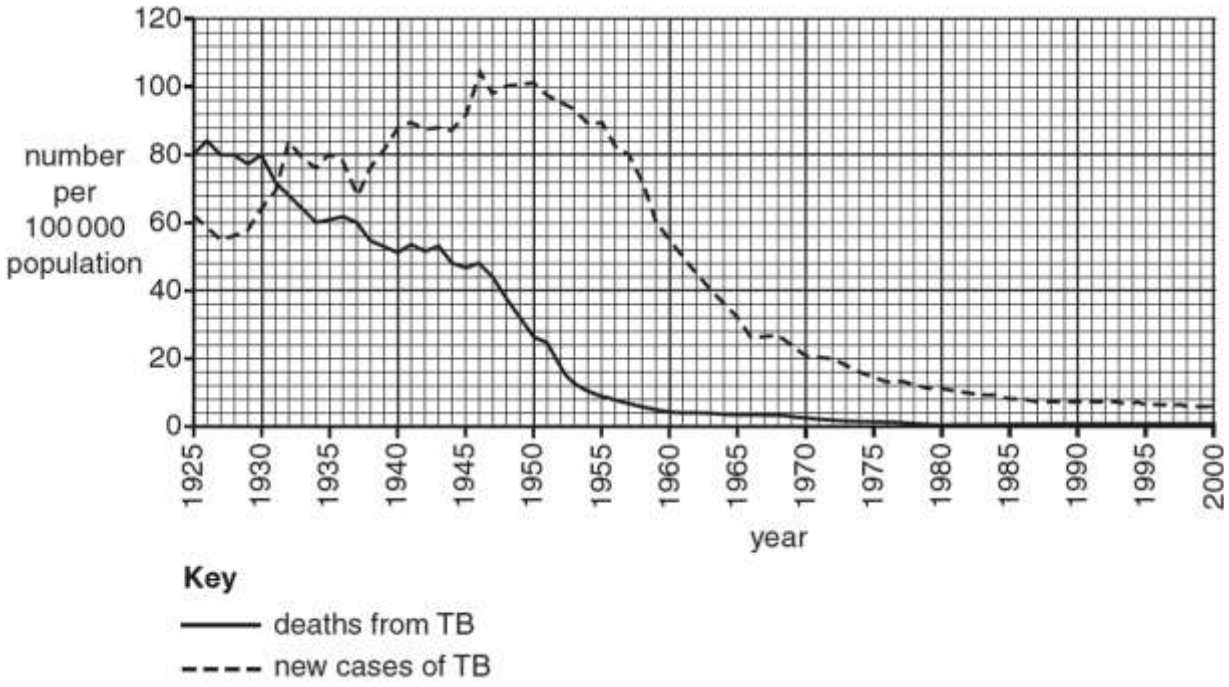

Fig. 2.1

(i) Use data from Fig. 2.1 to evaluate the effectiveness of the introduction of the vaccine and antibiotics on the number of new cases and deaths from TB.

(after introduction of antibiotics) Max 2

1 deaths from TB (generally) **decreased** from 52 per 1000 000 population in 1940 to 38 per 1000000 population in 1948

2 new cases of TB **increased** from 88 to 100 per 1000 000 population in the same time period

[4]

3 antibiotics is effective in preventing deaths from TB, but not preventing new cases from TB/ preventing transmission

(after introduction of vaccination) Max 2

4 new cases of TB **decreased** from 100 per 1000 000 population to 8 per 1000 000 population in 1980 or any relevant years which show decrease

5 deaths from TB **decreased drastically/ sharply** with the introduction of vaccination, from 38 per 1000 000 per population in 1948 to 10 per 100 000 in 1955 / decrease was more compared to antibiotics alone / quote relevant data

6 vaccination is effective in preventing/ decreasing both deaths from TB and new cases from TB / preventing transmission

7 Vaccination triggers the production of **memory (T and B) cells,** resulting in **faster** and **stronger** immune response

No marks for herd immunity- Bcos no data to show rate of uptake of vaccine

(effect of both antibiotics and vaccination)

8 the use of both antibiotics and vaccination for a long period of time is most effective because there is no death and low number of new TB cases

9 quote values (0 deaths per 1000 000 population, and) new cases of TB in the range of 4-8 per 1000 000 population from 1980 onwards

(ii) Suggest why the numbers of new cases or deaths **per 100 000 population** were calculated instead of stating the numbers of new cases or deaths alone.

1. (number of cases per 100 000) shows, **proportion** / AW, of population affected / **same** basis of comparison

2. idea of years with larger populations will usually have more cases / higher
number of cases/ deaths may just mean larger population in that year

[1]

[Total: 10]

3 Mangroves are plants that are able to live in harsh coastal conditions through various adaptations. One such adaption is the ability to grow in low oxygen concentrations in waterlogged mud. Mangroves have lateral roots known as pneumatophores that grow upward out of the mud and water to absorb gases directly from the atmosphere as shown in Fig. 3.1.

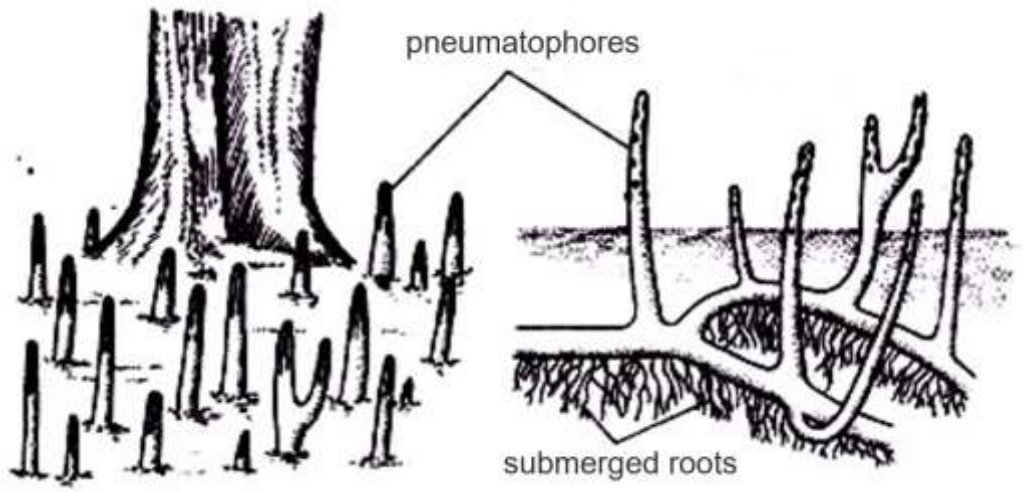

Fig. 3.1

(a) Explain how mangrove plants with more pneumatophores are able to yield more ATP.

1. Plants with more pneumatophores are exposed to more (atmospheric) oxygen and can undergo more aerobic respiration / less anaerobic respiration as compared to plants with fewer pneumatophores

OR complete/incomplete oxidation of glucose;

2. Cells of pneumatophores can synthesise 38 ATP which is more compared to cells of submerged roots which can synthesise 2 ATP from 1 molecule of glucose

***OR* idea of 19 times more ATP can be synthesised from 1 molecule of glucose;** [2]

One effect of climate change is rising sea levels, often resulting in severe storm surge and coastal flooding.

(b) Explain how climate change can lead to rising sea levels.

- (climate change) **Increase global temperature** cause **melting of ice shelves/ sheets in glaciers of Antartica and Arctic**
- And the **thermal expansion of seawater** as the oceans warm/ expansion of volume of seawater
- **Melting of polar ice caps** (Arctic) cause **reduce ice albedo effect** due to increase darker ocean surface exposed, less sun radiation reflected, more heat absorbed by land and sea leading to increased global temperature
- Reject heavy rain fall leading to rising sea level

[3]

(c) In many tropical and subtropical regions, mangroves as shown in Fig. 3.2 reduce waves and storm surges, and serve as a first line of defense against flooding and erosion.

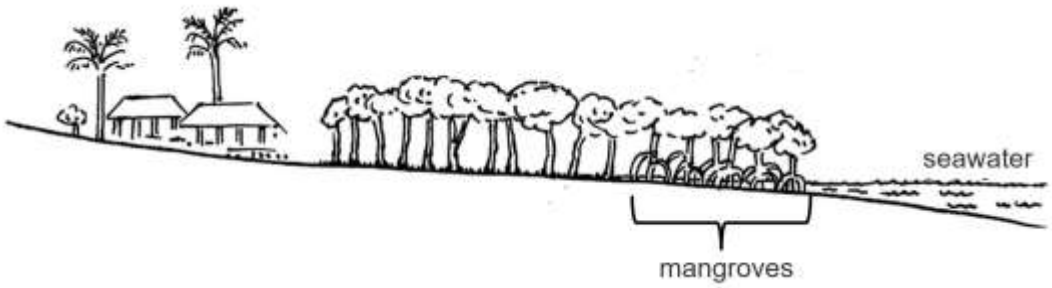

Fig. 3.2

One study quantifies global mangrove benefits by estimating the difference in flood damages between two scenarios: one "with mangroves" (current global extent of mangroves) and another "without mangroves".

Table 3.1 shows the land flooded, people and property damaged with and without mangroves across 700,000 km of mangrove coastlines globally. The difference between scenarios is the benefits provided by current mangroves.

Table 3.1

global benefit of mangroves in terms of	annual expected		
	with mangroves	without mangroves	benefit
land flooded (x1000 km^2)	122	157	35
people affected (million)	53	68	**15**
property loss ($US billion)	732	797	**65**

(i) Calculate the annual expected benefit of mangroves in terms of people affected and property loss and fill in your answers in Table 3.1. [1]

both to be correct for 1 m

(ii) With reference to Table 3.1, explain how **one** human activity could **directly** damage mangroves leading to greater climate change impact on humans.

- (state relevant human activity) accept industries release toxin into the sea that damage mangrove (Remove mangroves) for land reclamation/ the conversion of land occupied by mangroves for
- (state purpose) for aquaculture/ agriculture/ coastal development/ urban/ city development/ port/ residential areas
- Lesser mangroves for coastal protection from severe tsunami/ storms/ flood/ rising sea levels (severe weather event due to weather change)
- (max 1) (quote any one data with correct units from Table 2.1) without mangroves, 35000km^2 more of land flooded, 15 million more people affected/ $US 65 billion of property loss [4]

[Total: 10]

Section B

Answer **one** question in this section.

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

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)**, **(b)**, as indicated in the question.

4 **(a)** Describe how membrane fluidity is regulated in cells and explain the significance of membrane fluidity to the functions of vesicles. [13]

(b) Describe the structure of an antibody and explain how the vast diversity of antibodies is generated in B lymphocytes. [12]

[Total:25]

5 **(a)** There are many examples of concentration gradients in a cell, for example, the proton gradient in mitochondria plays an important role in aerobic respiration.

Describe how the proton gradient is established in mitochondria and explain the importance of concentration gradients in aerobic respiration. [13]

(b) Describe the life cycle of *Aedes aegypti* and discuss the possible impacts of global warming on geographical patterns of dengue. [12]

[Total:25]

4 **(a)** Describe how membrane fluidity is regulated in cells and explain the significance of membrane fluidity to the functions of vesicles. [13]

A. REGULATION OF MEMBRANE FLUIDITY

1. Membrane fluidity arises from the **movement** of ***both*** **phospholipids and proteins**.

2. Phospholipids are held by weak **hydrophobic interactions between the hydrocarbon tails**, and hence can move **laterally** (and sometimes flip-flopping)

3. Some **fatty acids** of phospholipids are **unsaturated/** with one or more **double bonds** introduce **kinks** in the fatty acid tails

4. At **low temperatures**, cholesterol acts as **spacers**

5. Kinks and/or cholesterol **prevents close packing** of phospholipid molecules

6. At **high temperatures**, the **bulky nature** of cholesterol thus **restricts phospholipid movement** (preventing it from becoming too fluid)

B. SIGNIFICANCE OF MEMBRANE FLUIDITY TO VESICLE FUNCTION

1. Structure of vesicles : **Spherical** compartments bounded by a **single membrane/ phospholipid bilayer**

2. Budding and fusion of vesicles from and to membranes involves **movement of phospholipids/ rearrangement** of phospholipids

3. Fluidity allows the **membrane** to **reseal itself** if it is disrupted by the budding and fusion of vesicles

C: Transport (Max 8)

Loading of substances through formation of vesicles

1. **Transport vesicles**: **Bud/ pinch** off from membrane of RER, carrying **proteins** to Golgi apparatus

   OR
   Secretory vesicles: **Bud/ pinch** off from the (*trans* face of the) membrane of Golgi body carrying **modified proteins** to the cell surface membrane

Releases of substances through fusion of vesicles

2. Membrane of **transport vesicles fuse** with the membrane of the (*cis* face of the) Golgi body to deposit their proteins within the lumen of the Golgi body.
   OR
   Secretory vesicle membrane **fuses** with the cell surface membrane to release the proteins out of the cell via **exocytosis**.

3. For proteins that have to be **embedded** in the **cell surface membrane** they are transported as proteins embedded in the **membrane** of the (transport/ golgi/ secretory) **vesicles**

4. Movement of vesicles in cells: move along **cytoskeleton/ microtubules** of the cell using **energy/ hydrolysis ATP** *(mark once only)*

Entry of substances into cells

5. Entry of substances into cells is done via **endocytosis** : **Pinocytosis-** take in fluid / solutes/ molecules dissolved in fluids, **phagocytosis**- take in large particles, **receptor-mediated phagocytosis** – take in specific substances

Description of endocytosis / description of receptor mediated phagocytosis using context of antigen presentation in antigen-presenting cells /phagocyte/ macrophages

6. Specific substances to be taken into cells, bind to **complementary binding sites** of transmembrane **receptor proteins** on the plasma membrane/ When the pathogen bind to the complementary membrane receptors on the cell surface membrane of the phagocyte

7. the membrane of the phagocyte form **pseudopodia** to **engulf** the pathogen/

Or (a small portion of the) cell surface membrane first **invaginates** to form a cavity containing the substance (for endocytosis and pinocytosis)

8. The tips of the pseudopodia **fuse** and **pinch off** to form vesicle/ **phagocytic vacuole/ phagosome** inside the cell

   Or The membrane then **pinches off** to form an (endocytic) **vesicle** within the cell containing the substance (for endocytosis and pinocytosis)

9. After the ligand molecules are released from the vesicle, the **receptors are recycled back** to the cell surface where the same vesicle containing the receptors will fuse with the cell surface membrane.

10. The phagocytic vacuole then **fuses** with a **lysosome** (→ phagolysosome) **t**he hydrolytic enzymes in the lysosome **digest/ breakdown** the **pathogen.**

11. Debris from the pathogen is released by exocytosis

During antigen presentation

12. A **vesicle containing MHC molecules** fuse with the phagolysosome containing the antigen-peptides (and the antigen- peptides bind to MHC molecules) → **peptide-MHC complexes.**

13. The vesicle will then **fuse** with the cell surface membrane and the specific peptide-MHC complexes are **embedded** on the **cell surface membrane** of APC

D: Compartmentalisation

1. **Lysosomes**: vesicles that budding off from the **Golgi apparatus** containing **hydrolytic enzymes**

2. **Isolating/ compartmentalising** hydrolytic enzymes in lysosomes/ vesicles helps maintain/ create **low pH** for the hydrolytic enzymes to function

Functions of lysosomes

3. **Autophagy :** A **damaged / worn-out organelle** becomes surrounded and enclosed by a **membrane from the ER** → this forms an **autophagosome** that encloses the organelle.

   (The autophagosome fuses with a primary lysosome to form an autolysosome, in which the unwanted organelle is digested – marked in MP 16)

4. **Autolysis:** Self-digestion of a cell / autolysis by release of lysosome contents within the cell, resulting in cell death.

   (Phagocytosis: marked in MP 13-16)

QWC 1 mark: Writes in continuous prose with proper paragraphing. At least 1 correct point from each of section A, B, C,D

(b) Describe the structure of an antibody and explain how the vast diversity of antibodies is generated in B lymphocytes. [12]

1. **Structure of an antibody**
2. a **globular** protein with **quaternary structure/4 folded polypeptide chains;**
3. **Two identical heavy** chains and t**wo identical light** chains;
4. **Each light and heavy chain** is made up of 2 domains,
5. The **amino-terminal** end of each light/heavy polypeptide chain contains the **variable** ($\mathbf{V_L}$) & ($\mathbf{V_H}$) domain +
6. The **carboxyl-terminal** end of the polypeptide chain contains the **constant** ($\mathbf{C_L}$) **constant** ($\mathbf{C_H}$) domain
7. **Each light and heavy chain** is folded into its specific **tertiary structure**, maintained by **disulfide bonds** and non-covalent interactions between R groups. *(name at least two) (award once for bonds);*
8. In the quaternary structure, the **variable domains of the light and heavy chain** ($\mathbf{V_H}$ and $\mathbf{V_L}$) are brought together to form an **antigen-binding site;**
9. The antigen-binding site has a **3D conformation** which is **complementary in shape** to the **epitope** of an antigen, allowing the antibody to bind to the specific antigen;
10. The Fab and Fc regions are connected by the **hinge** region + The hinge region is **flexible** and allow independent movement of the two Fab arms.
11. Each antibody has 2 Fab region and 1 Fc region -
    Fab (**F**ragment **a**ntigen **b**inding) region: Contains antigen-binding site to bind to epitope of antigen +
    Fc (**F**ragment **c**rystallisable) region: The Fc fragment of the antibody determines the **effector function** and therefore **class** of the antibody.

These points, to award credit?

12. Each antibody has **two** identical antigen-binding sites;
13. Each **light chain pairs** with a **heavy chain** via **disulfide bonds** and non-covalent interactions such as hydrogen bonds, hydrophobic interactions and ionic bonds between R groups *(name at least two); (award once for bonds)*
14. The **two heavy chains** are also linked together via **disulfide bonds** and non-covalent interactions between R groups. *(name at least two); (award once for bonds)*

Large diversity of antibodies

1. There are multiple **gene segments** at heavy and light chain **gene loci**;

2. **Somatic recombination** occurs during B cell maturation and development, where there is **DNA rearrangement** to assemble gene segments:
3. At **heavy chain** gene locus, one **V** gene segment, one **D** gene segment and one **J** gene segment are rearranged together to form **VDJ** exon to code for **variable domain** of the heavy chain;
4. At **light chain** gene locus, one **V** gene segment and one **J** gene segment are rearranged together to form **VJ** exon to code for **variable domain** of the light chain;
5. different light chain and heavy chain variable domains combine form to **different antigen – binding sites which bind different antigens**, generating different antibodies of **different antigen specificities**;

6. **Somatic hypermutation** occur in **rearranged VJ** and **VDJ** exons of the light and heavy gene loci during clonal expansion of B cells,
7. This result in **changes in the 3D conformation of the antigen binding sites**
8. resulting in antibodies of **increased affinity** to **antigens** (affinity maturation);

9. **Class switching** can occur in activated B cells, leading to **different classes of antibodies** which bind to specific antigen being produced;
10. **Class switching occurs where DNA recombination/ rearrangement** occur at the **constant region** of the **heavy chain gene locus** containing the **C_H gene segments**.
11. On the antibody, only the **heavy chain constant domain change**.
12. There can result in **different classes of antibodies**, each with **different effector functions**, that all have the **same antigen specificity**.

QWC: At least 1MP from A and at least 1 MP from (2 to 5) + (6 to 8) + (9 to 12)

5 **(a)** There are many examples of concentration gradients in a cell, for example, the proton gradient in mitochondria plays an important role in aerobic respiration.
Describe how the proton gradient is established in mitochondria and explain the importance of all concentration gradients in aerobic respiration. [12]

How is concentration gradient of hydrogen ions established:

1. **Transport of electrons** down electron transport chain (ETC) **provides energy** for
2. the **active transport/ pumping of H+ ions**/ against concentration gradient by **hydrogen pumps**
3. idea of **hydrophobic core** of inner mitochondrial membrane → prevents charged ions from moving across membrane freely into the matrix
4. to facilitate the **rapid build up of H+ ions/ H+ reservoirs** in **inter-membranal space** and thylakoid space → to establish the electrochemical/ proton gradients

Significance of concentration gradients

5. (gases) Movement of gases via diffusion thus gaseous exchange of CO_2 and O_2 in alveoli of lungs/ across cell surface membrane of cells/ mitochondrial membranes
6. to **remove metabolic waste** such as CO_2 produced from oxidative decarboxylation from link reaction and Krebs cycle
7. and provide O_2 **final electron acceptor** for oxidative phosphorylation → so that link reaction, Krebs cycle, OP can occur
8. without which only anaerobic respiration will occur, only substrate-level phosphorylation in glycolysis can only produce 2 ATP per glucose as compared to **38 ATP per glucose**, 19 times lesser
9. (H+ reservoirs in inter-membranal space) Facilitated diffusion of H+ ions down the hydrophilic channel associated with ATP synthase
10. **to generate proton-motive force** for ATP synthesis/ convert ADP and Pi to ATP
11. movement of substances down concentration gradient **without input of energy**
12. Facilitated diffusion of glucose across specific glucose channels into the CSM of cells
13. Example of reactions to maintain concentration gradient (Max 2)
    - Phosphorylation of glucose (1st step of glycolysis) keep concentration of glucose in cytoplasm low

- Reduction of NAD in cytoplasm for glycolysis and in matrix for link reaction and Krebs cycle by accepting electrons and hydrogen from the substrates
- Oxidation of NADH to inner mitochondrial membrane for oxidative phosphorylation to be regenerated back into NAD
- Diffusion of ADP into mitochondria for energy generating reaction and ATP out mitochondria (into cytoplasm) for energy requiring reactions

(b) Describe the life cycle of *Aedes aegypti* and discuss the possible impacts of global warming on geographical patterns of dengue.

1. Dengue can only occur in climates where **mosquitoes** as **vectors** are present to transmit the disease;

[Life cycle of *Aedes aegypti*]

2. **Female** mosquitoes **lay eggs** on surfaces of **stagnant water** bodies;
3. When the **eggs are submerged in water**, the **eggs hatch;**
4. **larvae emerge/hatch from eggs** + larvae develop into **pupae in water**;
5. mature/develop into an **adult mosquito**, which **emerge head-first** by **ingesting air** to **expand the abdomen** and thus splitting open the pupal case

[link global warming to dengue]

6. Global warming results in **temperate regions becoming warmer**, such that the **temperatures** and conditions in these regions become **more optimal** for both **mosquitoes and dengue virus**;

[Geographic distribution: Latitude]

7. Mosquitoes will thus move from **equator** to **higher latitudes** (**subtropical regions) (e.g. Europe),** so they spread to new areas **expanding their distribution**, thus spreading this diseases beyond the tropics;

[Geographic distribution: Altitude]

8. Global warming also results in **higher altitudes becoming warmer**, thus mosquitoes will be able to colonise **altitudes (elevation) higher up from plains to hills or mountains (e.g. Nepal)**

[Impact: Increased temperature on mosquitoes' survival]

9. **Increased temperature (up till 32°C) increases** the **survival** and results in **faster development rate** of the **mosquito;**
10. as an increase in temperature **increases their metabolic processes by increasing rates of enzyme-catalysed reactions;**
11. and the **female mosquitoes bite/feed more often** due to **increased rate of digestion, increasing the transmission of dengue virus**

[Impact: Increased temperature on viral replication]

12. The higher temperatures also allows for a **shorter virus replication cycle** in the mosquito vector → aiding spread of dengue

[Impact: Increased rainfall on breeding grounds]

13. **Increased rainfall may result in more stagnant pools of water and increases the number of breeding habitats for mosquitoes → lay more eggs**

[QWC]
Paragraphing + At least 2MP in life cycle + Explain geographical changes based on reasoning linked to 2 aspects (at least 1 MP for each) of climate change (temperature and rainfall) [1]

Accept Original mosquitoes population at tropics
At 40˚C which is beyond the thermal safety margin of the mosquito, metabolic **enzymes *denature****, (and lose their 3D conformation,). No mosquitoes to act as **vectors** for transmission of dengue.