H2

ANDERSON SERANGOON JUNIOR COLLEGE
HIGHER 2
ANSWERS

2022 JC2 PRELIMINARY EXAMINATIONS

CANDIDATE NAME

CLASS

INDEX NUMBER

BIOLOGY

9744/02

PAPER 2
SHORT STRUCTURED QUESTIONS

13 SEPTEMBER 2022
TUESDAY

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.

Answer **all** questions.

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.

For Examiner's Use	
1	/ 10
2	/ 11
3	/ 9
4	/ 10
5	/ 10
6	/ 10
7	/ 10
8	/ 10
9	/ 10
10	/ 5
11	/ 5
Total	/100

This document consists of **13** printed pages and **1** blank page

Answer **all** the questions.

1 Chitin, the second most abundant organic polymer after cellulose on Earth, is found in the cell walls of fungi and the exoskeleton of insects. Similar to cellulose, chitin is a structural polysaccharide. Chitin consists of N-acetylglucosamine.

Fig. 1.1 shows the structure of chitin.

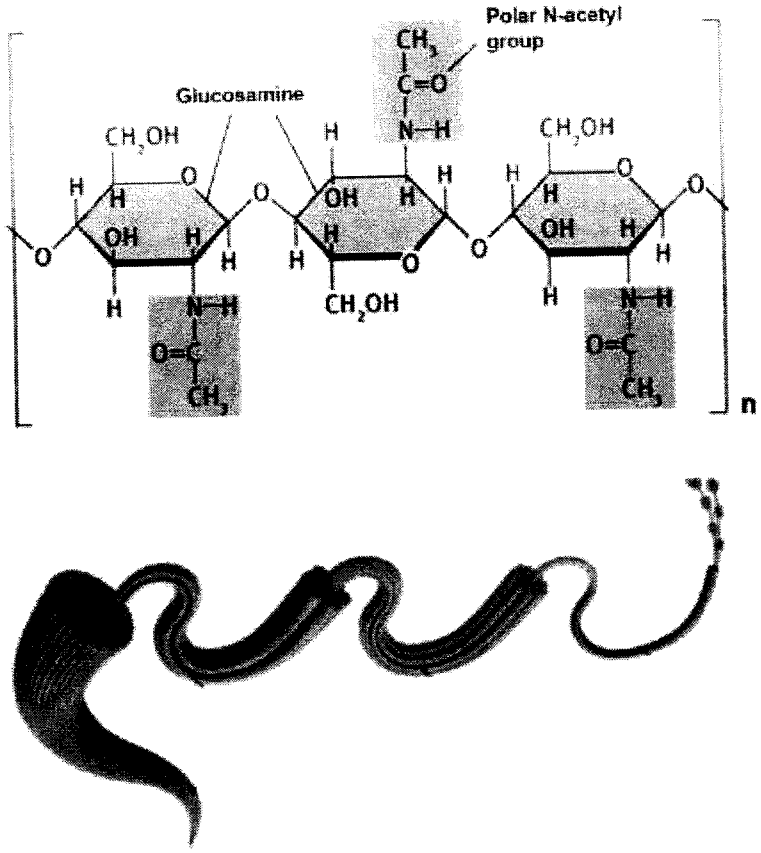


Fig. 1.1

(a) With reference to Fig. 1.1,

(i) name the covalent bond between two monomers in a chitin molecule and describe how this bond is formed.

1. Monomers are linked by **β (1,4) glycosidic bonds;**
2. Which is formed via a **condensation reaction**, with **loss of** 1 **water molecule per bond formed;**
3. between **a hydroxyl/OH group on carbon atom 1 of one N-acetylglucosamine and a hydroxyl/OH group on carbon atom 4 of another N-acetylglucosamine;**
4. **every alternate/successive N-acetylglucosamine monomer has to be rotated by 180°/inverted with respect to one another**, so that the –OH groups on carbon atoms 1 and 4 to line up alongside each other during condensation.

[3]

(ii) explain how the structure and property of chitin are related to its role as a structural polysaccharide in fungi and insects.

1. **Alternate N-acetylglucosamine** in chitin chain is **inverted/ rotated 180°** → results in a **straight / linear** chitin chain/molecule, allowing **bundling** of cellulose chains into microfibrils, macrofibrils and fibres;
2. **Polar/hydrophilic –OH groups and/or N-acetyl groups** in chitin chains are **projected outwards** in all directions;
3. This allows extensive **cross-linking** between chitin chains by formation of **interchain hydrogen bonds** between –OH groups and N-acetyl groups across parallel chains;
4. Parallel **straight / linear** chitin chains **bundle**/associate together to form **microfibrils**, which are arranged in larger bundles to form **fibres**/ Parallel chitin chains associate together to form a **fibrous structure;**
5. giving it **high tensile strength;**
6. each chitin chain/molecule is composed of **large number** of N-acetylglucosamine **monomers** → **large** molecule, thus is **insoluble** in water. [4]

(c) Chitinase is an enzyme found in plants. It degrades chitin in fungal cell walls and exoskeletons of insects, protecting the plants against a range of pathogens.

Describe the mode of action of chitinase.

1. Chitinase **lowers activation energy** + any one elaboration below
   1. **physically strains bonds within substrates** to be broken / distorts the substrate;
   2. **Altering charges** on substrates;
   3. active site provides a **favorable microenvironment** for catalysis to occur, eg **hydrophobic micro-environment** for hydrophobic substrates to react;

   max 2 from above
2. [**lock and key hypothesis**] The (**3D conformation** of) **substrate** (e.g., cellulose) is **complementary** to the **active site** of the **enzyme**, thus forming **enzyme-substrate complexes**;
3. [**induced fit** hypothesis] With ref. to **change in 3D conformation** of enzyme for a **better fit/ tighter binding** between substrate and enzyme;
4. Resulting in **hydrolysis of glycosidic bond** between N-acetylglucosamine residues

Note to answer in context, presence of 1 or more than 1 types of substrate

[3]

Easy: 3, Moderate: 5, Challenging: 2 [Total: 10]

2 Fig. 2.1 is an electronmicrograph of a human cell during mitosis.

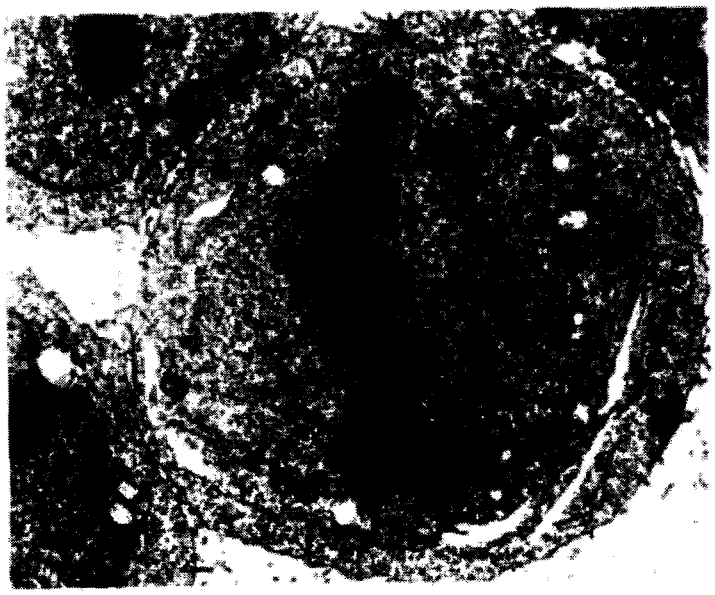


Fig. 2.1

(a) Describe the events that take place in the stage of mitosis **before** that seen in Fig. 2.1.

1. During **prophase**, **chromatin fibres condense** to become **discrete chromosomes;**
2. **nuclear envelope breaks down/disintegrates;**
3. **nucleolus** gradually **disappears** (reject disintegrate nucleolus);
4. **Centrioles** migrate to **opposite poles** of the cells;
5. The **mitotic spindle** begins to **form** and **spindle fibres** begin to **assemble/extend**.

Reject Interphase events
The majority of candidates were able to identify the correct stage of mitosis.

[3]

(b) The normal diploid number of chromosomes for a human cell, such as that shown Fig. 2.1, is 46.

The cell in Fig. 2.1 has 92 DNA molecules.

Explain the presence of 92 DNA molecules in this cell and why it is important to have this number.

Accounting of 92 ds DNA molecules:

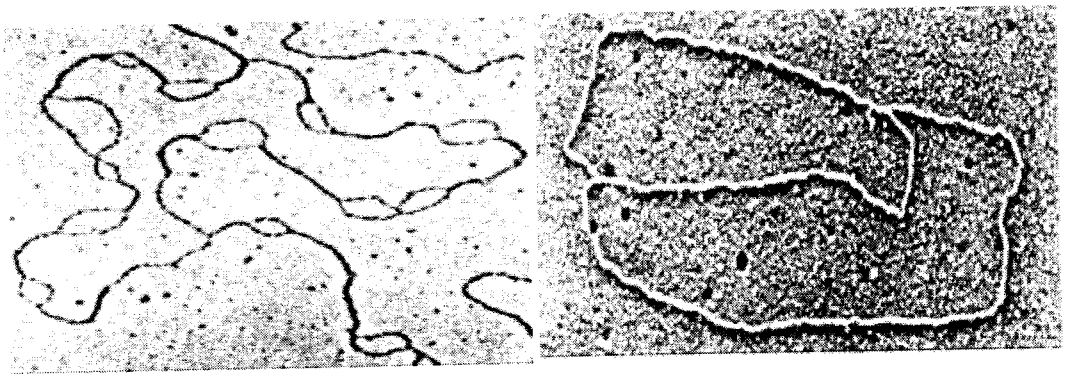
1. **DNA replication** occurs during **S phase of interphase** before mitosis occurs;
2. **each strand** of parental DNA is used as **template** to synthesize a **complementary** DNA strand → **doubling in number of DNA molecules from 46 to 92**;
3. After DNA replication, **each chromosome** consists of **two sister chromatids joined at the centromere;**

Importance of 92 ds DNA molecules:

1. ensures that after mitosis, **each daughter nucleus** is **genetically identical (same type and number of chromosomes)** to the parent nucleus;
2. for **growth in multicellular organisms(role)**, - increase in the **number of genetically identical cells**, carrying out the **same function;**
3. to **repair tissues by replacement of** (NOT REPAIR cells) **damaged or worn-out cells** with **genetically identical cells**

Most candidates demonstrated a good understanding of the underlying reason for DNA replication during mitosis, but few complete answers were seen. Details were often missing. Although the information in the question clearly stated ***that the context was mitosis, the most common error was for candidates to confuse mitosis with meiosis****. A number of candidates referred to the* ***formation of haploid gametes and the production of diploid zygotes following fertilisation. (WRONG!)*** [4]

(c) Fig. 2.2 shows DNA replication occurring in a human cell (**A**) and in an *Escherichia coli* (**B**). Diagrams are not shown to scale.



A **B**

Fig. 2.2

(i) State **one** visible difference in the structure of these two DNA molecules during DNA replication and account for this difference.

1. **Single** origin of replication in bacterium but **multiple** origins of replication in mammalian cell; Accept: multiple replication sites / bubbles;
2. **Larger / longer DNA** in **mammalian cell**, thus **multiple Ori R to increase rate of DNA replication**

[2]

(ii) Explain why there is both continuous and discontinuous synthesis of daughter strands during DNA replication.

1. **DNA polymerase** only adds **DNA nucleotides** to the **free 3' end** of the newly synthesized strand/ DNA polymerase synthesize DNA in the **5' to 3' direction**;
2. as the **active site of DNA polymerase** is **complementary** to the **shape of free 3'OH end**;
3. The two **parental/ template** DNA **strands** are **anti-parallel**

[2]

Easy: 3, Moderate: 5, Challenging: 3 [Total: 11]

3 Fig. 3.1 shows a diagram of protein synthesis.

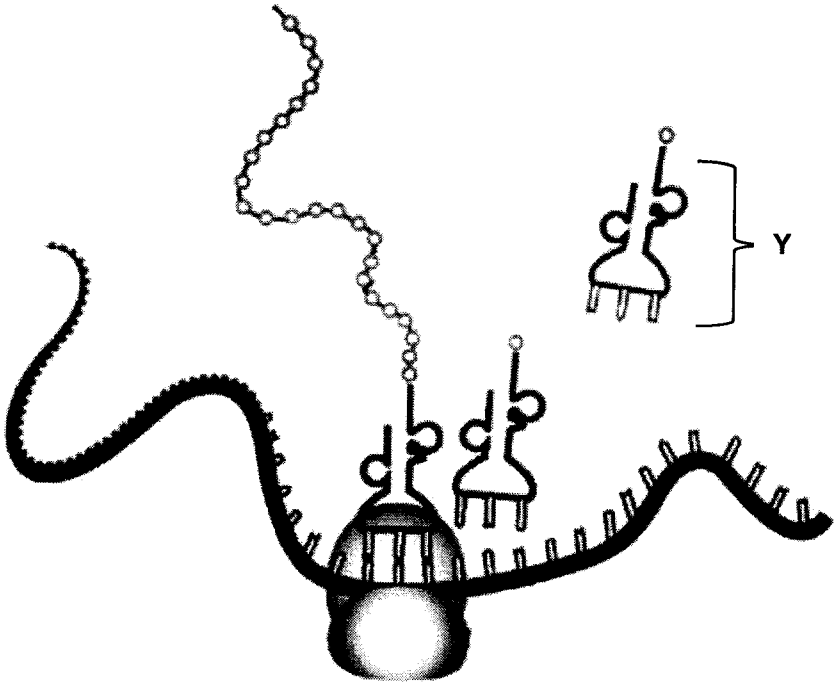


Fig. 3.1

(a) With reference to Fig. 3.1, outline the synthesis of the polypeptide chain from its mRNA.

1. **mRNA is used as a template for translation;**
2. **Anti-codon of aminoacyl tRNA complex** binds to **codons of mRNA** via **complementary base pairing,** at the A (aminoacyl tRNA binding) site;
3. the **growing polypeptide chain from its tRNA** in the **P (peptidyl tRNA binding) site is detached** and then attached to the **amino acid** carried by the **tRNA at the A site;**
4. **Peptidyl transferase** catalyses formation of **peptide bonds between the amino acids;**
5. Ribosome moves along mRNA in **5' to 3' direction until the stop codon (UGA, UAA, UAG)** is reached, releasing polypeptide;

[4]

(b) During protein synthesis in cells of an embryo, all molecules **Y** in Fig.3.1 are observed to be attached to the arginine amino acid instead of lysine.

(i) Suggest how the attachment of the wrong amino acid, arginine, to molecule **Y** may arise.

1. Errors during (amino activation stage) involving aminoacyl tRNA synthetases;
2. Ref. possible mutation in the **gene/DNA/base** sequence for the (lysine) aminoacyl tRNA synthetases,
3. resulting in **altered 3D conformation** of active site which is now **complementary** (in shape) to the amino acid arginine or the corresponding tRNA with anticodon for arginine

Reject mutation of tRNA gene

[2]

(ii) Suggest and explain the effect of attachment of the wrong amino acid, arginine, to molecule **Y** on the embryo.

1. ref. altered **primary sequence** of polypeptides (all lysine replaced by arginine) and folding of polypeptides to **tertiary structure** / 3D conformation is affected;
2. ref. **non-functional proteins** made in cells
3. [compulsory point] ref. possible disruption of metabolic processes in the **cell** / cells might die easily, **embryo cannot further develop** into a fetus [3]

Easy: 2, Moderate: 4, Challenging: 3 [Total: 9]

4 The building blocks of anterior (head) – posterior (tail) axis patterning in *Drosophila* embryo (fertilised egg) are laid out during egg cell formation. Four genes (*hunchback, caudal, bicoid, nanos*) are responsible for the polarity of the egg cell and then of the subsequent embryo. mRNA molecules of these four genes were found to be distributed along the anterior-posterior axis of the developing egg cell as shown in Fig. 4.1.

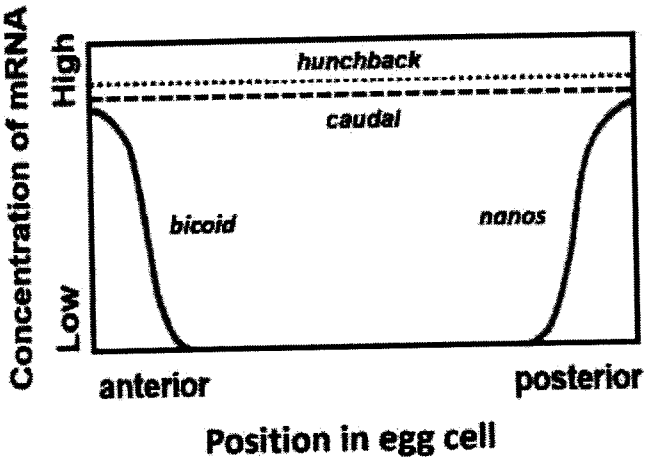


Fig. 4.1

(a) With reference to Fig 4.1,

(i) explain the types of chromatin modifications that may be carried out on the *hunchback* and *caudal* genes. [4]

1. **Histone acetylation/ Adding of acetyl groups to histones** catalysed by **histone acetyl transferase**;
2. **positive charges removed** on histones leads to less electrostatic attraction / reduced affinity between histones and negatively charged DNA;
3. **DNA demethylation / removal of methyl groups** from DNA catalysed by **DNA methyl transferase;**
4. Histone demethylation

5. idea of more DNA unwinding from histones / loosening of chromatin packaging/ to form **euchromatin** → Idea of **greater accessibility of RNA polymerase and transcription factors to the promoter** sequences/ Idea

of loosely condensed chromatin facilitate assembly of general transcription factors and RNA polymerase at the promoter (to form transcription initiation complex);

6. (compulsory point) Idea of upregulation of transcription / genes become transcriptionally active → **high** levels / concentrations of mRNA observed for both *hunchback* and *caudal* genes; [4]

(ii) The length of *hunchback* and *caudal* mRNA in the cytoplasm is shorter than the *hunchback* and *caudal* primary mRNA in the nucleus.

Describe what happens to the *hunchback* and *caudal* mRNA in the nucleus before they enter the cytoplasm.

- **Small nuclear Ribonuclear Proteins (snRNPs)** made up of **proteins** and **small nuclear RNA** associate with other proteins to bind at **splice sites at each end of an intron** (of a pre-mRNA), forming a **spliceosome**;
- A lariat structure (the intron looped structure) forms and spliceosome **cuts / cleaves / excise** at both ends of an intron
- to release the intron which is rapidly degraded by nucleases;
- exons flanking intron are **spliced** together via **phosphodiester bonds** formation to form **mature mRNA** with **continuous coding sequence**

[3]

(iii) mRNAs in cells are very unstable, having short half-lives of not more than 30 minutes. Explain how the *hunchback* and *caudal* mRNA levels are maintained within the cell.

- (post-transcriptional control) addition of **5' cap** and **3' polyA tail** and to prevent digestion of mRNA by exonucleases;
- (translational control) Idea of **increasing** the length of **3' polyA tail / long** 3'polyA tail of the mRNA to increase half life;
- (translational control) Binding of certain proteins/inhibitors/hormones which can slow down / block degradation of mRNA (by exonucleases); [2]

(b) The corresponding protein concentrations of the four genes were measured in the early stages of development of the *Drosophila* embryo as shown in Fig. 4.2.

It was found that bicoid and nanos proteins act as repressors to block the translation of *caudal* and *hunchback* mRNA respectively.

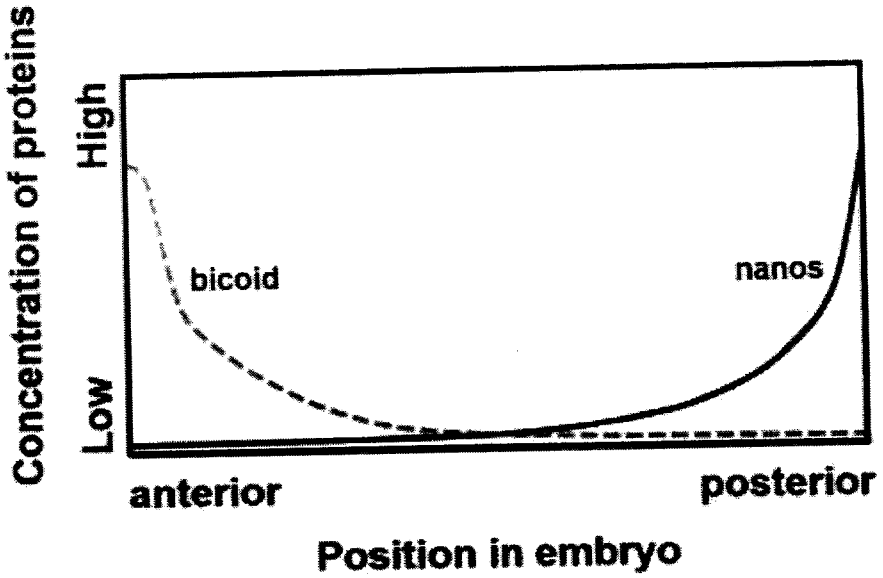
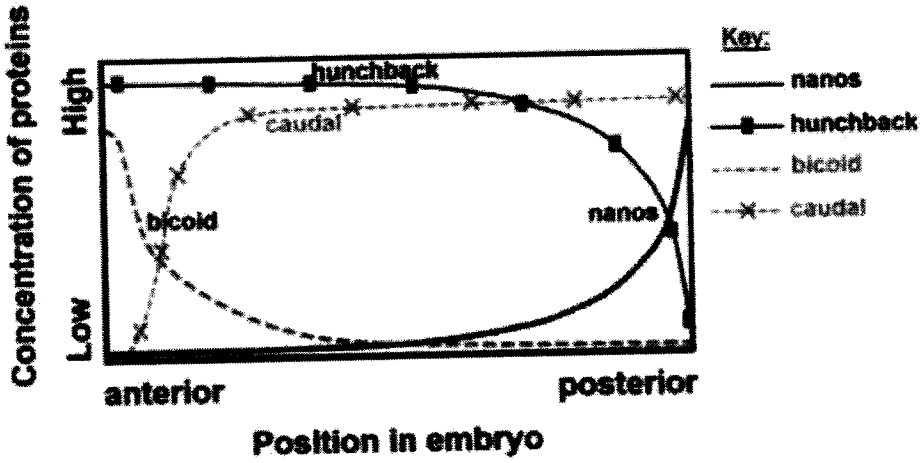


Fig. 4.2

Sketch **one** graph on Fig. 4.2, to represent the concentration of hunchback protein. [1]



Easy: 4, Moderate: 4, Challenging: 2 [Total: 10]

5 Fig. 5.1 shows the main structural features of the influenza virus.

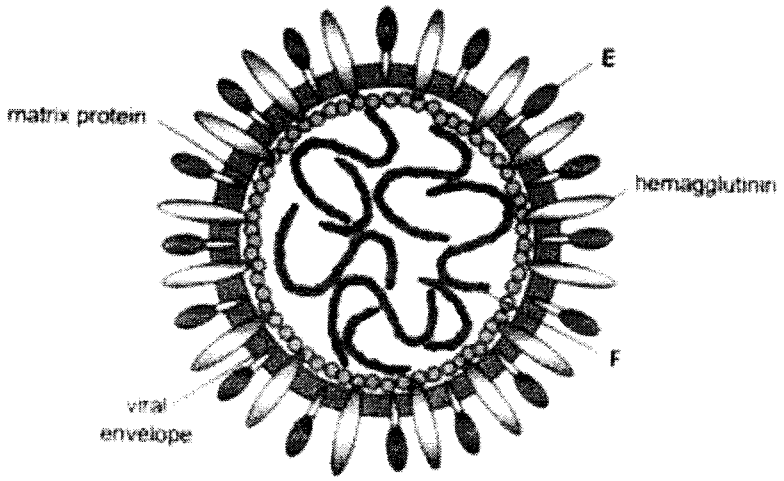


Fig. 5.1

(a) Explain the role of **E** and **F** in the influenza virus.

E .....

.....

.....

F .....

.....

.....

- **E** is neuraminidase
- Neuraminidase *facilitates the release* of newly formed virus from the infected host cell by cleaving the *sialic acid receptors*. (answers from 2013prelims AJ P2Q2a)

- **F** is ss (-) RNA

Max 1 from the following

- Comprises of genes for viral proteins (give at least one example such as RNA dependent RNA polymerase)
- Template for the synthesis of complementary (+) RNA which are used for translation by host cell ribosomes into viral proteins in cytoplasm/ templates for making new copies of the ssRNA (-) genome in the nucleus

[4]

The sub-types of the influenza **A** virus that infect birds, human and pigs in one area of the world in recent times are shown in the Table 5.1 below.

Table 5.1

time period	influenza A virus sub-types present		
	birds	humans	pigs
1918 – 1957	show any one of the H1 – H16 antigens combined with any one of the N1 – N9 antigens	H1N1	H1N1
1958 – 1970		H2N2	
1971 to present day		H3N2 H1N1	H3N2 H2N3

(b) Using the data in Fig. 5.1, two students, each has different claims that described how influenza **A** is a danger to human health in this area of the world.

(i) Student **X** claimed that "Antigenic drift of influenza human virus such as H3N2 would lead to vaccines that target hemagglutinin glycoprotein being less effective."

Put a tick (√) in one box to indicate whether or not this statement is true.

Give a reason for your answer.

true ☐ false ☐

Reason .....

..... [3]

- True
- (Antigenic drift) **Caused by accumulations of mutations** in the **haemagglutinin gene → Change shape/conformation/configuration** of H1 haemagglutinin glycoprotein
- mutated H1 protein is **more complementary in shape** to/ is **able to better fit or bind to** receptors containing sialic acid found on the surface of human host cells/ not complementary to antigen binding site of antibodies produced by memory plasma cells
- Gene evolves under the **selection pressure** of virus-specific antibody stimulated from the vaccine.

(ii) Student **Y** claimed that "Antigenic shift of influenza virus is happening within humans, by combining H2N2 from older people with H1N1 or H3N2."

Put a tick (√) in one box to indicate whether or not this statement is true.

Give a reason for your answer.

true ☐ false ☐

Reasoning .....

..... [3]

- *False*
- *For antigenic shift to happen, different strain of virus need to infect the same host cell in the same organism (not just same species).*
- *Influenza pandemic wave caused by H2N2 did not happen in the same time period as H1N1 or H3N2.*
- *Influenza does not integrate its genome and stay dormant in the host cell (so the H2N2 virus did not stay in the same host when H1N1 or H3N2 infect the human host).*

Easy: 3 , Moderate: 5, Challenging: 2 [Total: 10]

6 Wing pattern in the butterfly species *Heliconius melpomene* is controlled by genes on autosomal chromosomes.

The gene for banding pattern on the upper wing has two alleles:

- a dominant allele coding for a full band
- a recessive allele coding for a broken band.

The gene for ray pattern on the lower wing has two alleles:

- a dominant allele coding for rays
- a recessive allele coding for no rays.

Scientists crossed a butterfly that was homozygous dominant for both genes with a butterfly that was homozygous recessive for both genes. The scientists wanted to check whether the phenotypic ratio for offspring in the F2 generation agreed with the expected phenotypic ratio of 9 : 3 : 3 : 1.

The results of these genetic crosses are shown in Fig. 6.1.

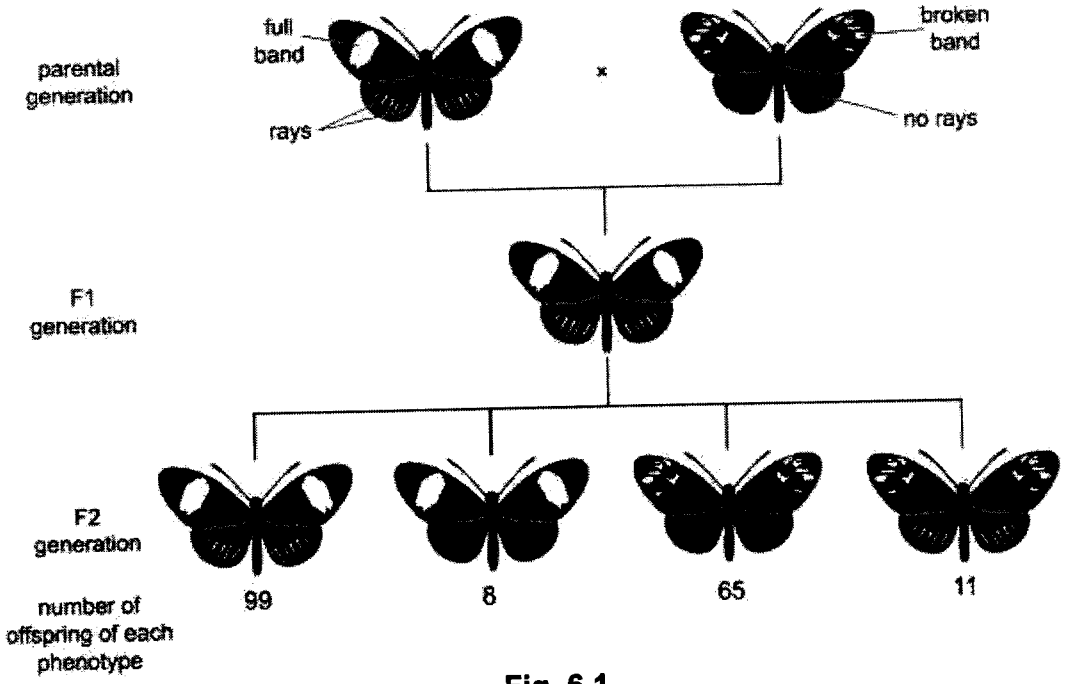


Fig. 6.1

(a) Draw a genetic diagram to explain these results.

5 Marking points (any 4), parental, F1 and F2 must be drawn
- Parental & F1 and F2 phenotype/genotype (drawn with linked genes) +legend
- All Gametes + circles (can ecf if not drawn with linked genes).
- Identified recombinant gametes and parental gametes in gametes produced in F1
- Cross / Punnett Square with correct gametes (gametes circled)
- Expected/observed offspring genotype and observed phenotypic numbers

[5]

(b) Two varieties of *Heliconius* butterflies, both pure-breeding for white wings, were crossed.

All the F1 generation progeny produced orange wings.
The F1 butterflies were then crossed.

In the F2 generation, 145 butterflies had orange wings and 111 butterflies had white wings. The control of wing colour is an example of epistasis resulting in a ratio that is close to 9:7.

Explain the term epistasis in this context.

- (define) Epistasis is a form of gene interaction in which a gene at one locus alters the phenotypic expression of a gene at a second locus;
- 2 copies of recessive alleles at either gene locus A and/or B is sufficient to prevent phenotypic expression of the dominant allele of the other (hypostatic) gene/ aa is epistatic to BB and Bb and bb is epistatic to AA and Aa
- e.g. allele A of one gene codes for a functional enzyme A, which converts the yellow precursor into a yellow intermediate. Allele B of another gene codes for enzyme B, which converts the yellow intermediate into the orange pigment;
- Genotype aa masks genotype BB and Bb. Thus lack of functional enzyme A in pea plant will result in white flowers despite presence of functional enzyme B/ When the organism is homozygous recessive (aa) at the gene locus A/a and/or homozygous recessive (bb) at the gene locus B/b, the phenotype of yellow wingis observed regardless of whether there is a dominant allele present at the other gene locus A/a and/ or B/b respectively.
- Accept other labels other than A and B

[3]

(c) The genus *Heliconius* contains more than 40 species of brightly patterned butterflies.

Researchers have investigated in the laboratory how one species, *Heliconius heurippa*, could have developed as a separate species. The phenotype of *H. heurippa* is intermediate between that of two other species, *H. cydno* and *H. melpomene* as it contains DNA from the two parent species as a result of hybridisation.

Laboratory breeding experiments showed that:

- matings between *H. cydno* and *H. melpomene* (parent species) produce fertile hybrid offspring
- controlled matings of the hybrids produces individuals identical in appearance to *H. heurippa* within three generations
- hybrid butterflies prefer to mate with each other, rather than with individuals of either of the parent species.

In the wild,

- the genus *Heliconius* butterflies taste unpleasant to predators such as birds.
- the bright colours on the wings of the butterflies act as warnings so that birds avoid eating them. Therefore, this pattern provides a selective advantage.
- *Heliconius* hybrids occur in small numbers and have patterns that do not resemble the established warning pattern of either parent species. These hybrids have a selective disadvantage.

The researchers thought that, because the hybrid butterflies preferred to mate with each other, this could make speciation more likely to occur.

Suggest why *H. heurippa* are still not regarded as a separate species in the wild.
1 hybrids, are eaten/ die thus fail to survive and reproduce (accept reproduce/ reject survive only) ; reject just lifting from the passage that they have selective disadvantage
2 hybrid gene pool not maintained because not enough individual to interbreed; [2]

Easy: 3 , Moderate: 4, Challenging: 3 [Total: 10]

7 Pancreatic cancer is an almost universally lethal disease.

Many genes are involved in the development of pancreatic cancer. Table 7.1 shows four of these genes.

Table 7.1

genes	genetic changes observed
P	homozygous deletion
Q	hypermethylation of the gene promoter
R	substitution in codon 56
S	amplification of gene

(a) Using the data in Table 7.1, identify an oncogene and a mutated tumour suppressor gene. Explain your answer.

(i) oncogene
- Gene R;
- (mutation → effect on protein) relates substitution to a gain of function mutation as one copy is sufficient to produce a <u>hyperactive/ constitutively active protein</u> that sends signals to the nucleus to stimulate cell division;

Or
- Gene S;
- relates amplification of gene to a gain of function mutation as multiple copies of the gene / <u>greater number of protein products</u> present that will drive the cell towards cell division/ cause dysregulation in cell cycle control/ uncontrolled cell division;

[2]

(ii) mutated tumour suppressor gene
- Gene P
- relates homozygous deletion to a loss of function mutation. Both copies of the alleles for the gene has to be lost for the proteins that regulate the cell cycle control not to be produced;

Or
- Gene Q;
- relates hypermethylation of the gene promoter to a loss of function mutation as it will lead to the gene being silenced hence no protein products that control cell division are produced;

[2]

(b) Fig. 7.1 shows where the restriction enzyme *EcoRI* cuts within the two different alleles of gene **R**, the sizes of the fragments produced and the regions that bind to two probes, **Y** and **Z**.

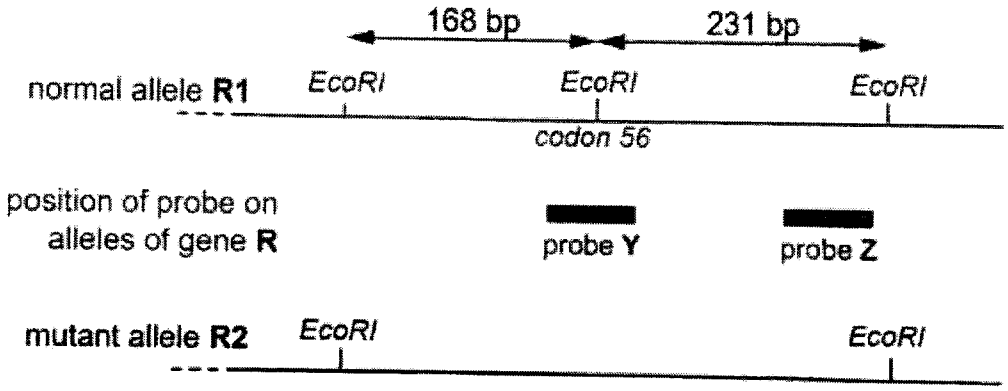


Fig. 7.1

(i) With reference to Fig. 7.1, explain how the two alleles of gene **R** can be distinguished using gel electrophoresis and detected by probe **Y**.

- ***Eco*RI** is used to **cut DNA** to generate **restriction fragments**
- DNA fragments were **separated** by **size** via **gel electrophoresis** under a influence of a **direct current**
- Substitution at codon 56 results in a loss of restriction site in mutant allele R2
- (allele) R1: has 3 *EcoRI* restriction sites, hence will generate two fragments of 168 kb and 231b([4]
- allele) R2: has 2 restriction sites/ hence will result in one fragment of 399kb

(ii) On the Fig. 7.2 below, draw the positions and label the sizes of the DNA fragments of an heterozygous individual if probe **Z** would to be used instead.

- Two fragments, One at 399bp close to the well, and another at 231 closer to 200bp band.
- Correct size label [2]

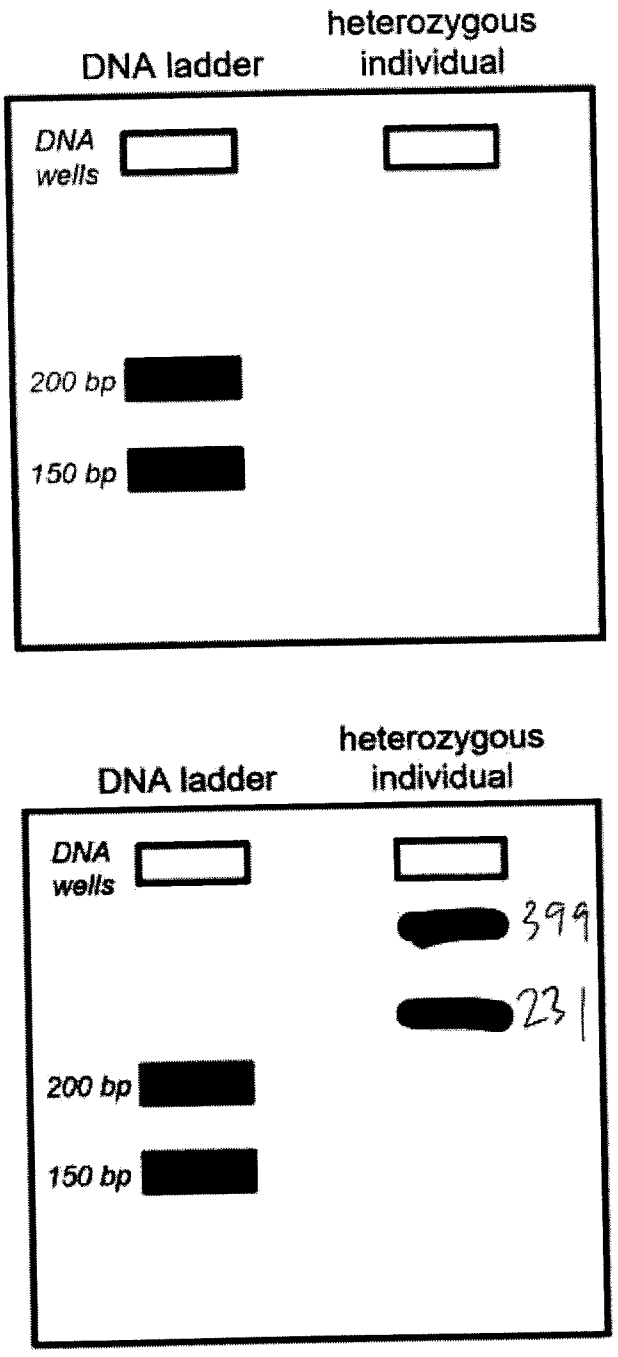


Fig. 7.2

Easy: 2, Moderate: 5, Challenging: 3 [Total: 10]

8 Red algae are multicellular photosynthetic eukaryotes that contain phycoerythrin. Phycoerythrin is a photosynthetic pigment.

Fig. 8.1 shows:

- the absorption spectrum of phycoerythrin
- the action spectrum of red algae.

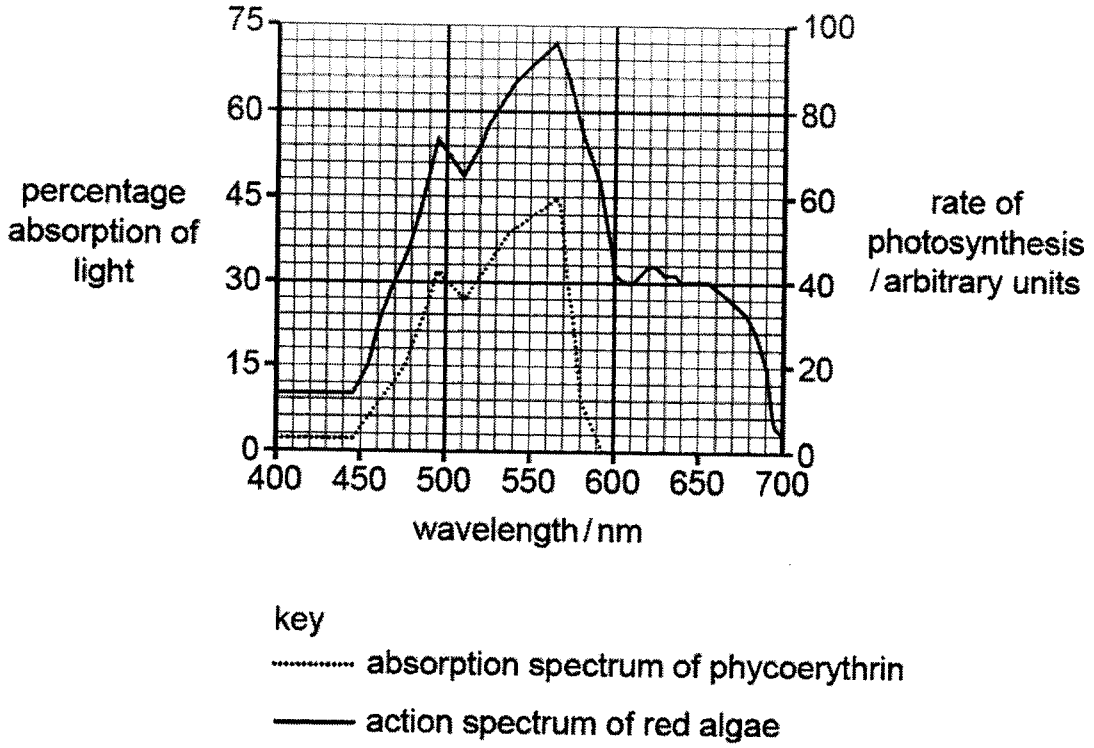


Fig. 8.1

(a) **(i)** With reference to Fig. 8.1, state the wavelength of peak absorption by phycoerythrin.
565nm

[1]

(ii) Explain how the data in Fig. 8.1 show that phycoerythrin is **not** the only photosynthetic pigment in red algae.

1. phycoerythrin does not absorb light between 590–700 (nm) ;
2. (but) photosynthesis still takes place (between 590–700 nm) with rate of photosynthesis in the range of 4-44 arbitrary units

[2]

(iii) Phycoerythrin is **not** the primary pigment (pigment in reaction centre) for photosynthesis in red algae.

Suggest the role of phycoerythrin in photosynthesis in red algae.

1. accessory pigment ;
2. absorbs light (energy) / photons of light and) and passes it to, primary pigment / reaction centre / chlorophyll *a* ;
3. **via transfer light <u>energy</u>** from one pigment molecule to another (in the light harvesting complex) through **excitation of electrons**

[2]

(b) The rate of photosynthesis is affected by factors other than wavelength of light. These factors may act as limiting factors. A student investigated the effect of limiting factors on rate of photosynthesis by measuring the volume of oxygen released from a plant.

(i) Explain what is meant by the term limiting factor and state an example of a limiting factor in photosynthesis.

<u>Explain</u>
(process / photosynthesis, affected by more than one factor)
1. rate is limited by the factor nearest its minimum value/ **in short supply** reaction.
2. It is the factor which **directly affects** a process (rate of a reaction) if its **quantity is changed**.

<u>State</u>
Light intensity/ temperature/ carbon dioxide concentration

Reject wavelength of light/ merely saying light [3]

(ii) Explain why the volume of oxygen released from a plant does not give a true rate of photosynthesis.
1. Idea of Volume is a **net** volume of oxygen / amount released during photosynthesis minus amount taken in during respiration / volume measured is lower than the actual released during photosynthesis
2. Because oxygen is used as a **final electron acceptor** (at the end of the electron transport)
3. during **oxidative phosphorylation** in respiration

[2]

Easy: 5, Moderate: 5, Challenging: 0 [Total: 10]

9 **(a)** Fig 9.1 shows the arrangement of bones in the pentadactyl forelimbs of four vertebrates. This is used by many people to provide evidence for evolution.

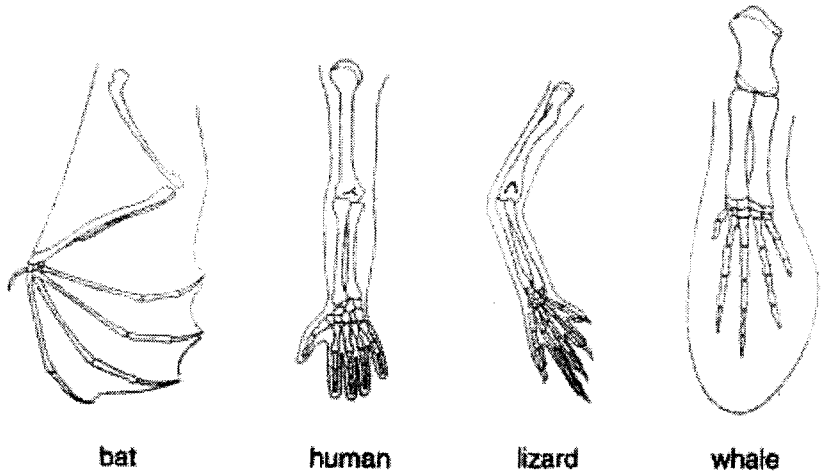


Fig. 9.1

(i) State the term used to describe the relationship between structures such as those in Fig. 9.1.

1. **Homology**

(ii) Explain how the relationship between the structures in Fig. 9.1 provides evidence to support the theory of evolution.

1. Although the forelimbs had **different functions (for instance grasping for humans and swimming for whales),** they have the **same arrangement of bones;**
2. they were likely to have **originated from a common ancestor;**
3. and had been **modified over time in descendent species** through **natural selection,** suggesting **descent with modification.**

*Candidates were expected to **relate their answer to the structures shown in Fig. 9.1**. Many candidates were able to say that **although the forelimbs had different functions** (often stated) they basically **have the same arrangement** (frequently described). Therefore they were likely to have **originated from a common ancestor and had been modified over time**.*

[3]

(b) There are many different species of lizards. Three of these species, *Liolaemus fabiani, L. molinai* and *L. multicolor*, are thought to be closely related.

Samples of these three species were collected from the Andes range in Western South America. The base sequences of four regions of DNA of each species were sequenced.

The percentage difference in the base sequences in *L. molinai and L. multicolor,* compared to the sequences in *L. fabiani,* was calculated. The results are shown in Table 9.1.

Table 9.1

DNA region	Lizard species	Percentage difference in base sequence from that of *L. fabiani* / %
Non-coding region 1	*L. molinai*	4.8
	L. multicolor	4.4
Non-coding region 2	*L. molinai*	8.1
	L. multicolor	7.3
Coding region 1	*L. molinai*	2.1
	L. multicolor	2.0
Coding region 2	*L. molinai*	1.9
	L. multicolor	1.7

(i) Using the evidence from the non-coding regions in Table 9.1, explain why *L. fabiani* may be more closely related to *L. multicolor* than to *L. molinai.*

1. The percentage of divergence of base sequence of *L. multicolor* from *L. fabiani* is **less compared** to divergence of *L. molinai* from *L. fabiani* for **both non-coding region 1 & 2**;
   Quote data : For region 1, the difference between *L. multicolor from L. fabiani* is **4.4%** while the difference from *L. molinai* is **4.8%.** Or region 2, the difference between *L. multicolor from L. fabiani* is **7.3%** while the difference from *L. molinai* is **8.1%.**
2. This means that *L. multicolor from L. fabiani* **share a more recent common ancestor** / **diverge from a common ancestor more recently;**
3. As a result, there was less time to **accumulate mutations/ fewer mutations** observed in the DNA sequences. [2]

(ii) The coding region 1 and 2 in Table 9.1 were measured by analysing *cytochrome c* gene.

Suggest why the *cytochrome c* gene is used to measure changes in DNA sequences in closely related species.

1. *cytochrome c* gene, is **essential** for aerobic respiration/ essential cellular functions;
2. therefore, **present in all / expressed in all cells of** the three species → serves as a good **basis for comparison;**
3. *cytochrome* c gene sequences **changes very slowly** → useful for studying divergence that occurred a long time ago + quote data;
4. cytochrome c gene is found on **mitochondria DNA**, so changes in DNA sequences are passed down the maternal line → does not undergo [2]

recombination (crossing-over and independent assortment). Any differences between mtDNA sequences show only the mutations accumulated since divergence from a common ancestor.

(c) State the importance of variation in the coding regions for evolution to occur.

1. It may result in formation of new alleles which codes for new phenotype / gene product (May confer selective advantage to the organism OR May allow better adaptation to the environment) in the event of sudden/ drastic change;
2. It increases variation in the population allowing natural selection to operate

[2]

Easy: 3 , Moderate: 5, Challenging: 2 [Total: 10]

10 Fig 10.1 shows an antigen presenting cell (APC) presenting an antigen from a pathogen such as a virus, to a cytotoxic T cell.

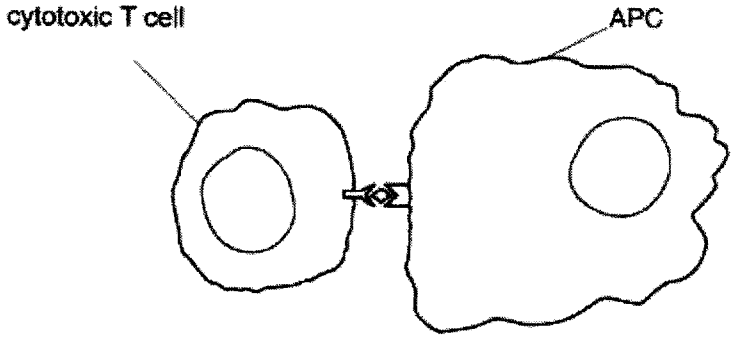


Fig. 10.1

(a) Using Fig. 10.1, describe how presentation of an antigens by APC will lead to the elimination of the pathogen.

Activation of cytotoxic T cells (max 2)

1. (Epitope of) antigen (peptide) on the **cell surface** of APC bind to the **T cell receptor** of a (naive) cytotoxic T cell
2. via **complementary shape**
3. Naïve T cells divide via **mitosis**/ undergo **clonal expansion** and **differentiate** into effector/ activated cytotoxic T cells

Action of cytotoxic T cells (at least 1)

4. T cell receptor on cytotoxic T cells binds to infected cells displaying corresponding antigen via complementary shape
   (no need to look out for complementary shape if already mentioned in MP2)
5. Cytotoxic T cells kill infected cells via **apoptosis** through release of **perforin** and/or **granzymes**

[3]

(b) State **two** differences between artificial active immunity and natural passive immunity.

Difference between artificial and natural immunity.....

artificial active	natural passive
deliberate / AW **A** from medical staff	**or** not deliberate / from mother / in breast milk / across placenta ;
vaccine / (foreign) antigens in injection	**or** antibodies passed on ;

Difference between active and passive immunity.....

immune response	**or** no immune response ;
antibodies / memory cells produced	**or** no. antibodies / memory cells produced ;
longer lasting	**or** short-lived ;
protection not immediate	**or** immediate protection ;

[2]

Easy: 2, Moderate: 3, Challenging: 0 [Total: 5]

11 Plant biodiversity varies throughout the world and is dependent on many factors, particularly climate.

Fig. 11.1 shows the relationship between the number of plant genera and the mean annual rainfall in seven countries.

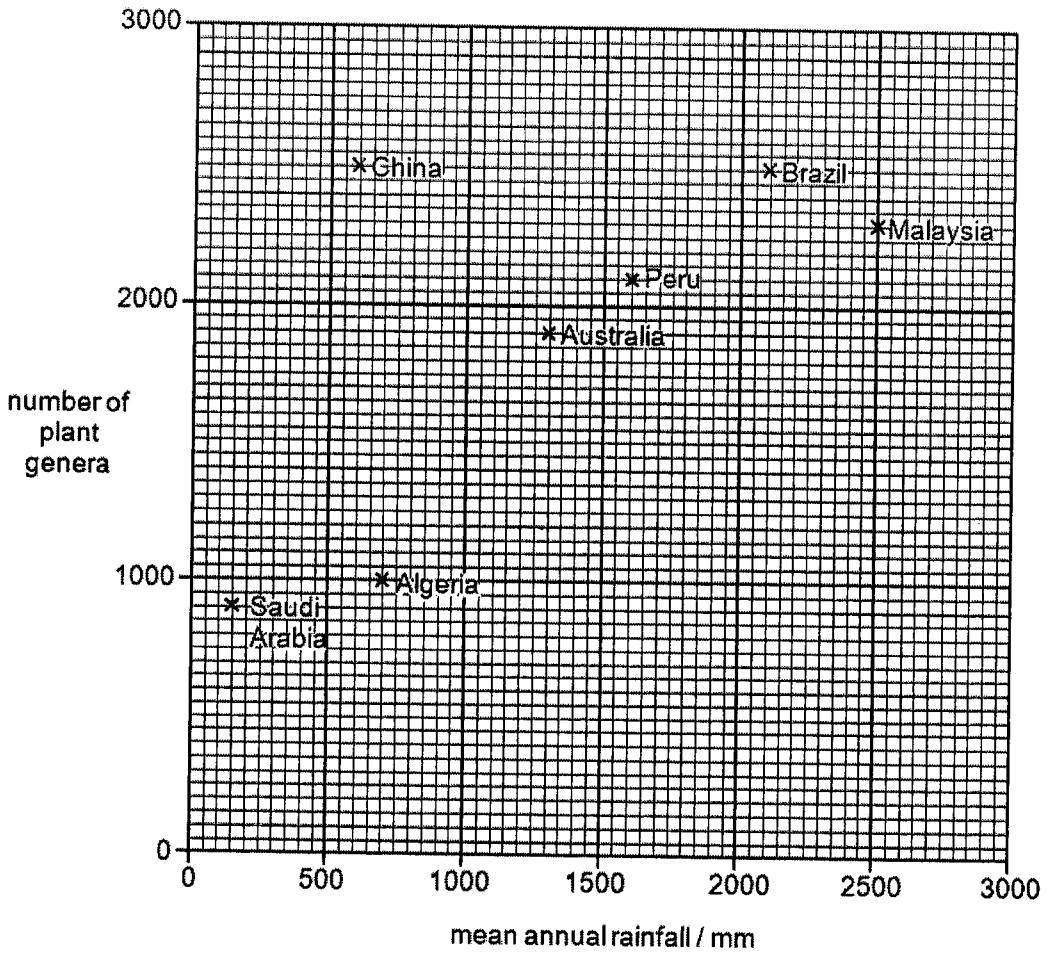


Fig. 11.1

(a) (i) Describe the relationship between the number of plant genera and the mean annual rainfall in these seven countries.

- *Ref to overall trend (i.e. positive correlation) / number of plant genera increases as mean annual rainfall increases*
- *Ref to paired figures (i.e. genera number and mean annual rainfall in 2 named countries showing the trend) correctly quoted with units*
  - *Saudi Arabia with lowest mean annual rainfall of with 150mm has lowest number of plant genera at 900*
  - *Algeria with higher mean annual rainfall of 700mm also has higher number of plant genera at 1000*
  - *Australia with higher mean annual rainfall of 1300mm also has higher number of plant genera at 1900*
  - *Peru with higher mean annual rainfall of 1600mm also has higher number of plant genera at 2100*
  - *Brazil with higher mean annual rainfall of 2100mm also has higher number of plant genera at 2500*

- *Malaysia with higher mean annual rainfall of 2500mm also has higher number of plant genera at 2300*

- *Ref to China not fitting the trend as she has low mean annual rainfall of 600mm but has high number of plant genera at 2500 (accept Malaysia as anomaly)* [2]

(ii) Global warming has led to changes in rainfall in many parts of the world.

Explain how changes in rainfall can decrease plant biodiversity.

- *Ref to increase / decrease in rainfall result in increased incidence of flooding / drought, shorter / longer rainy season*
- *Ref to relevant consequence on plants (e.g. plant wilting from loss of water /stomata closure/ loss in cell turgidity/ decrease in photosynthetic pigments/ decrease in Rubisco/ plant rotting from waterlogged roots / plants infected by pests and pathogens)*

[2]

(b) The Millennium Seed Bank is located in the United Kingdom. So far it has successfully stored seeds from 10% of the world's wild plant species.

Suggest **one** benefit to humans of conserving plant species.

may be of use in the future

- *(may produce) biomedicines / AW*
- *resources (for humans) e.g. wood for building / fibres for clothes / fuel / food / agriculture*
- *maintain, gene pool / genetic diversity*
- *to maintain stability in ecosystems*
- *aesthetic reasons*
- *(eco)tourism*

[1]

Easy: 2, Moderate: 2, Challenging: 1 [Total: 5]

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.

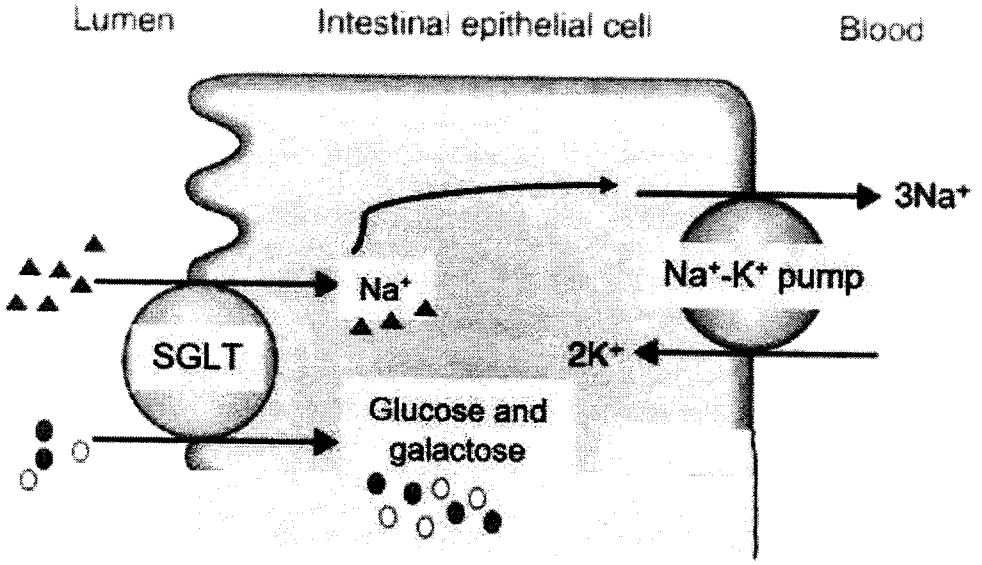


Fig. 1.1

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.

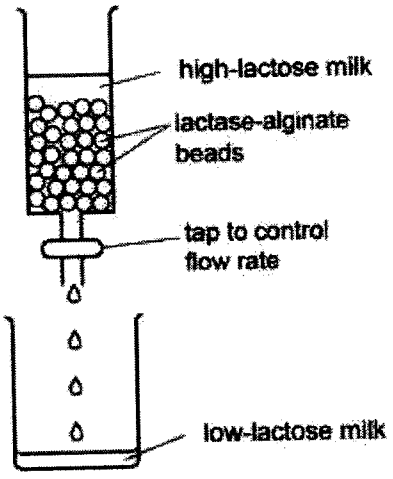


Fig. 1.

(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.

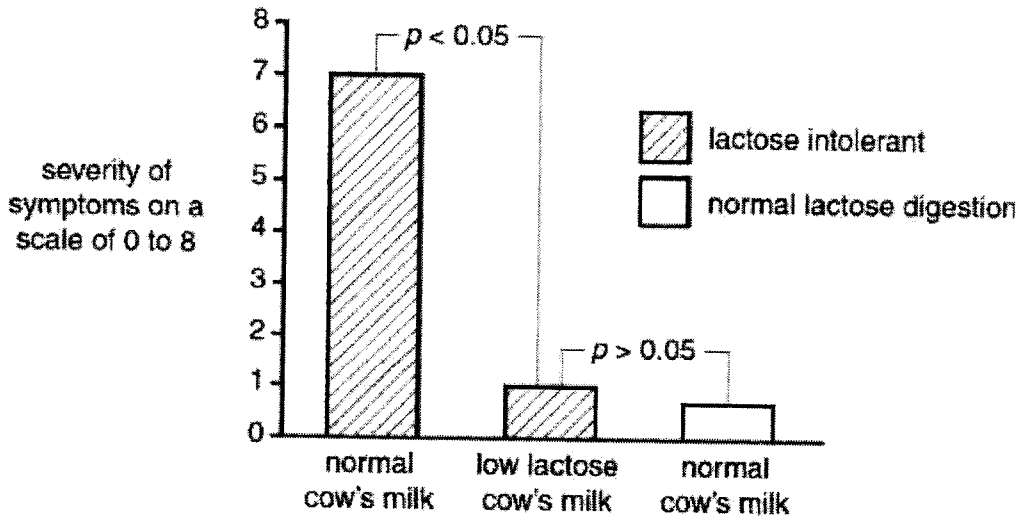


Fig. 1.3

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.

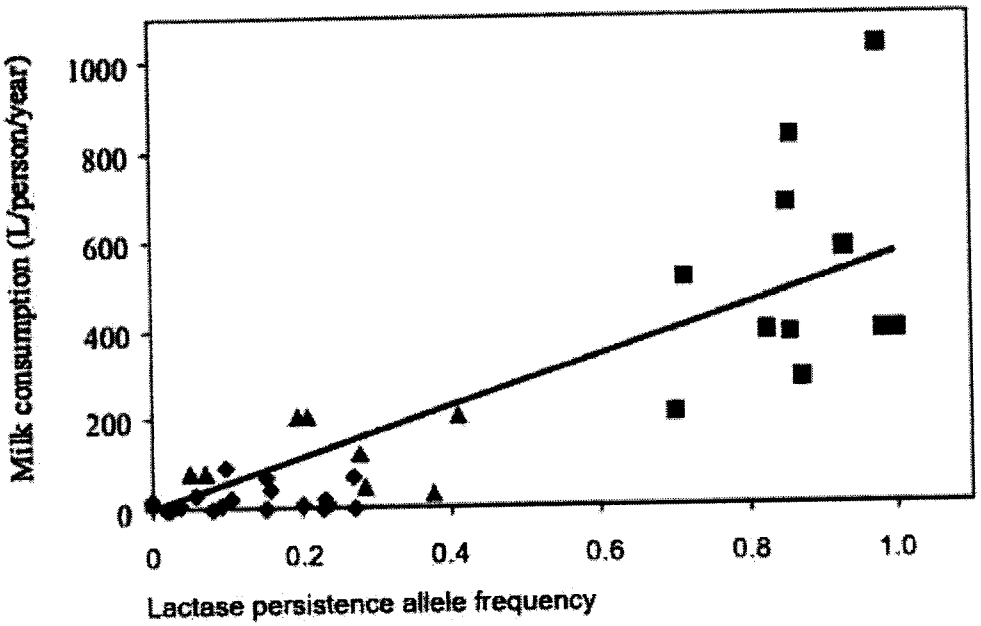


Fig. 1.4

(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.

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

[2]

	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.

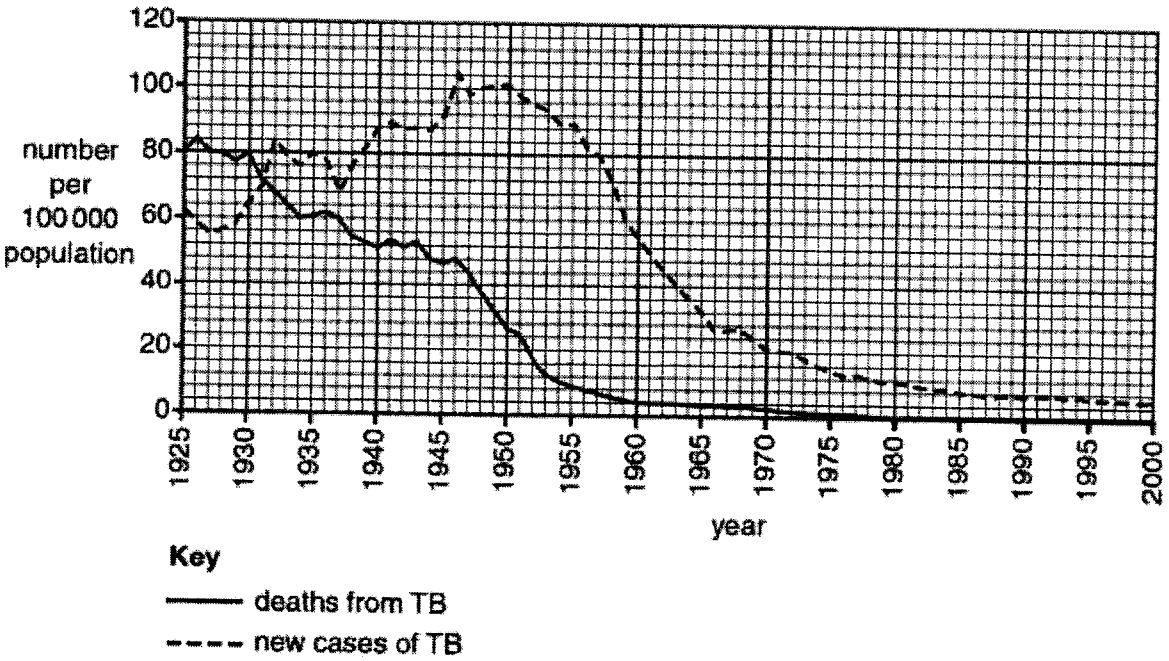


Fig. 2.1

(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, <u>but not</u> 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 <u>drastically/ sharply</u>** 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 <u>both</u> 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 / **<u>same</u>** 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.

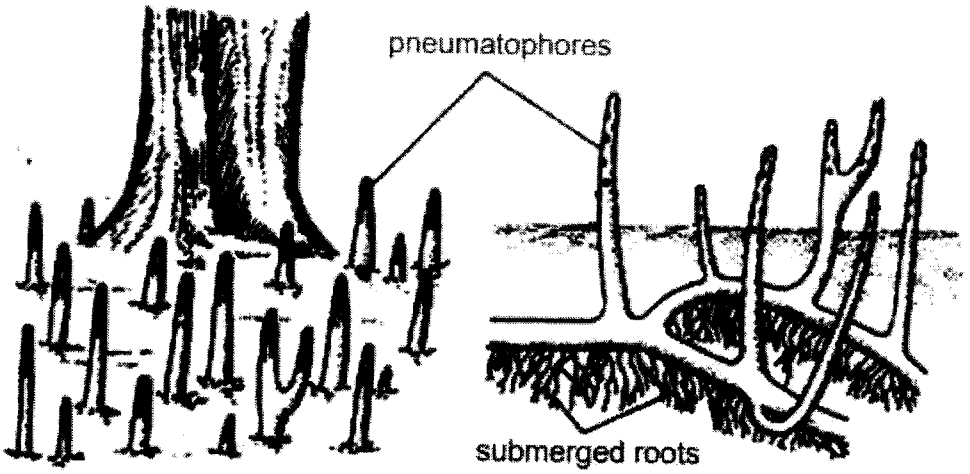


Fig. 3.1

(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 <u>aerobic</u> respiration / less <u>anaerobic</u> respiration as compared to plants with fewer pneumatophores**

OR complete/incomplete oxidation of glucose;

2. **Cells of pneumatophores can synthesise <u>38 ATP</u> which is more compared to cells of submerged roots which can synthesise <u>2 ATP</u> 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.

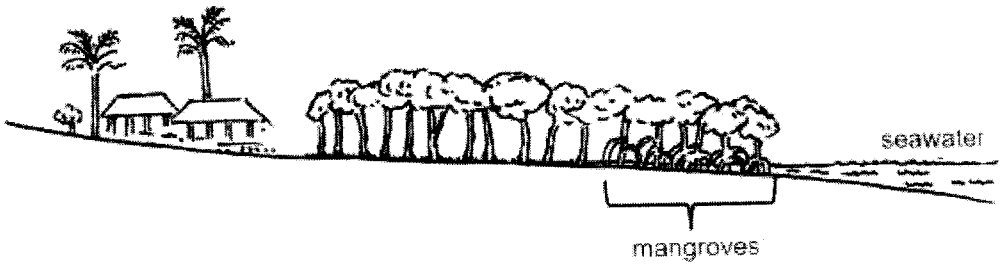


Fig. 3.2

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) the 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 **two 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** (V_L) & (V_H) domain +
6. The **carboxyl-terminal** end of the polypeptide chain contains the **constant** (C_L) **constant** (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** (V_H and 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.