JC2 PRELIMS 2024
H2 PAPER 2

1 (a) Fig. 1.1 is a diagram of a section through a **mitochondrion**.

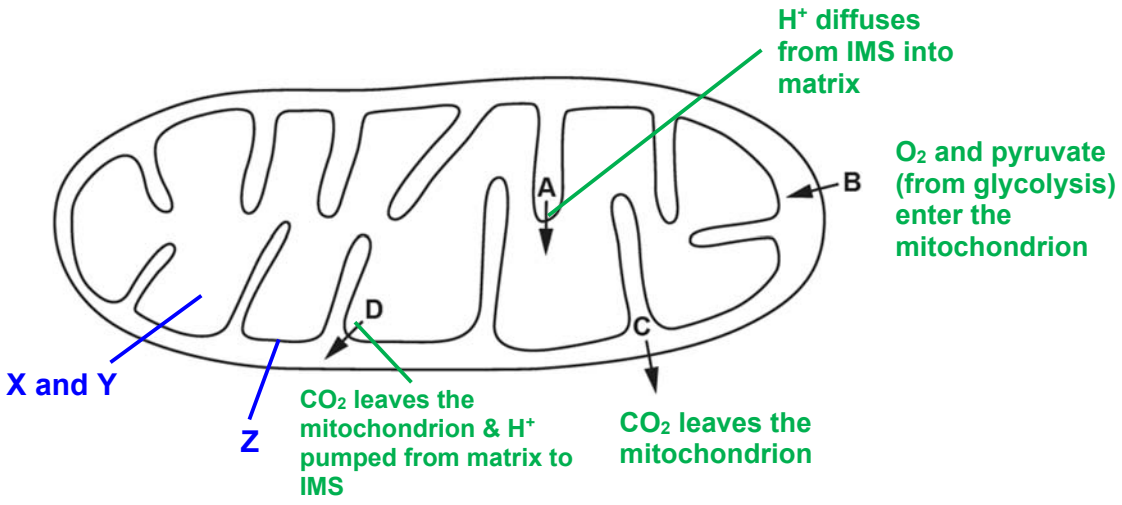


Fig. 1.1

(i) **On Fig 1.1**, **use label lines and letters** to **label the positions** where the following reactions take place: [3]

X - Link reaction
Y - Krebs cycle
} **[occurs in matrix of mitochondrion]**
Z - Oxidative phosphorylation **[occurs in inner mitochondrial membrane]**

1 mark each

(ii) The four arrows, **A**, **B**, **C** and **D**, show the **movement of molecules and ions**.

Use the letters to **identify all the arrows** (one or more) that show: [2]

Active transport of protons **D**

Diffusion of carbon dioxide **C and D**

(b) **Compare** the **process** of **oxidative phosphorylation** with **photophosphorylation**. [2]

SIMILARITIES (Any 1):

S1. Electrons passed down electron carriers of decreasing energy level in **electron transport chain** in **both processes**.

S2. **Pumping of H^+** across membrane to **create steep proton gradient** in **both processes**.

S3. **Diffusion** of H^+ via **hydrophilic channel** of **ATP synthase** **(stalked particle)** to **synthesize ATP** in **both processes**.

S4. Use of **energy released** **from electrons transported down energy level in electron transport chain to pump H^+ to create proton gradient** in **both processes**.

DIFFERENCES (Any 1):

Feature of comparison	Photophosphorylation	Oxidative phosphorylation
D1. Location	• **Thylakoid** of **chloroplasts**	• **Inner mitochondrial membrane**
D2. Source of electrons	• [Non-cyclic] **Water** • [Cyclic] **PS I**	• **NADH** • **$FADH_2$**
D3. Final electron acceptor	• [Non-cyclic] **$NADP^+$** • [Cyclic] **PS I**	• **O_2**
D4. Products formed	• **NADPH**	• **H_2O**
D5. Requirement of light energy	• **Yes for photolysis of water**	• **No**
D6. Source of energy	• **Light**	• **Oxidation of glucose**
D7. Direction of H^+ pumped to generate steep proton gradient	• **Pumped from stroma to thylakoid space**	• **Pumped from matrix to intermembrane space**
D8. Direction of H^+ diffusion to synthesize ATP	• **Diffusion from thylakoid space to stroma**	• **Diffusion from intermembrane space to matrix**

(c) **Apart from channel proteins** that **allow transport of ions**, plant and animals cells also have **channel proteins** such as **aquaporins** which permits the **movement of water across membranes**.

reasons

Explain why **aquaporins** are **necessary**. [3]

1. **Cell surface membrane** is made up of **phospholipid bilayer**
2. Has a **hydrophobic boundary/core** due to presence of non-polar fatty acid tails
3. Water molecules are **small and polar**
4. Only **small number of water molecules** can **move directly across** the cell surface **membrane** (i.e. rate of movement of water molecules is slow)
5. Aquaporins provide **hydrophilic channel** (due to polar amino acids line the interior part of aquaporins to interact with the water molecules)
6. Allowing **large number of water molecules** (i.e. rate of movement of water molecules is faster) to move **across membrane** via **osmosis**.

[Total: 10]

2 Fig. 2.1 shows the primary structure of a section of a polypeptide chain of collagen

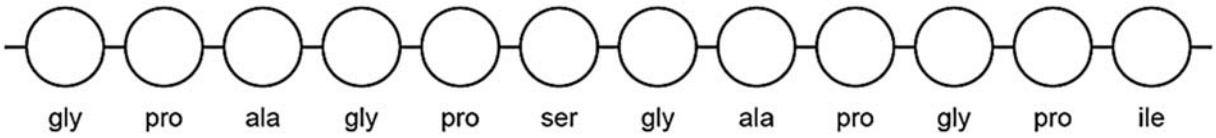


Fig. 2.1

reasons

(a) Explain how the primary structure shown in Fig. 2.1 indicates that the structure of the polypeptide is suited to be a component of a collagen molecule. [3]

Max marking (1/2m each point)

1. **Every third amino acid in the polypeptide is glycine.**
2. **The R-group of glycine is a H atom and is the only R-group that is small enough to fit into the centre of the triple helix (Note: not collagen).**
3. This **allows close association of the three polypeptide chains (note: not collagen).**
4. **Glycine (-NH) can form hydrogen bonds with C=O group in proline other polypeptides of triple helix. (Accept: *idea that* H bonds can form with other polypeptides in the triple helix)**
5. **resulting in a stable helical structure**
6. **Hydrophobic R-groups of proline residues will project on the exterior of the triple helix.**
7. **insoluble molecule.**
8. consists **mainly** of **repeated glycine X Y sequences.**
9. **repeating organisation,**
10. contributes to a **stable helical structure.**

Fibroblasts are cells that synthesize and secrete collagen, which forms the extracellular matrix.

Hydrolytic enzymes, known as collagenases, are secreted by some cells during wound healing.

These cells also **secrete inhibitors** of **collagenases**. The activity of the enzymes and inhibitors is regulated so that the development and maintenance of the extracellular matrix is controlled.

(b) State and explain what the outcome will be for the composition of the extracellular matrix if collagenase inhibitor activity is **high**. [2]

1. **Higher collagen concentration / more collagen present. [must have, 1/2m]**
2. **Collagen not hydrolysed. Accept: less hydrolysis,**
3. **If competitive inhibitor,**
   - o **compete with collagen for the active site, block collagen from binding to active site**

   OR
3. **If non-competitive inhibitor,**
   - o **bind to a site other than active site and change the shape of the (active site) enzyme, collagen cannot bind to active site.**
4. **no / few, ESC / enzyme substrate complexes form.**

   Thus collagen not hydrolyze.

Collagenase has several important medical uses, such as in the treatment of burnt skin. Scientists investigated the effect of pH on the activity of collagenase at 37 °C.

The results of their investigation are shown in Fig. 2.2.

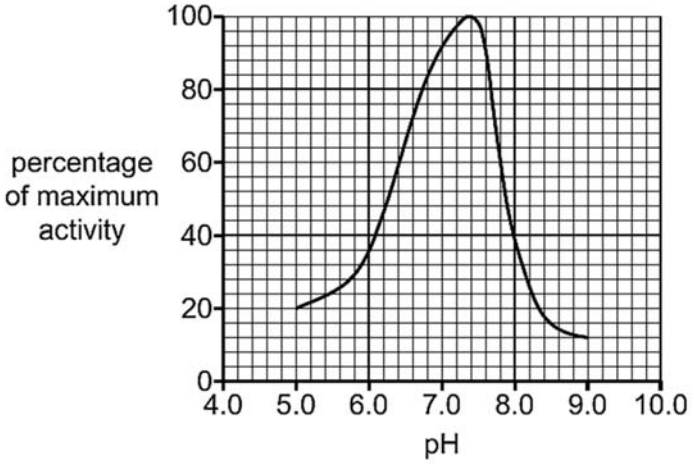


Fig. 2.2

(c) Explain why the activity of collagenase is lower at pH 8.0 than at the optimum pH. [3]

½ mark each

1. At pH lower than optimum pH, H^+ concentration is changed / decreased.
2. This alters ionic charges on the basic and acidic R-groups of amino acid residues on enzyme molecule.
3. Ionic bonds are disrupted, and substrate binding is affected.
4. Shape of active site is changed and is less complementary to shape of substrate.
5. Rate of effective collision decreases and less enzyme-substrate complex formed per unit time.
6. Less products formed.

Synthetic inhibitors have been trialed as potential treatment for diseases which are caused by a lack of regulation of collagenase activity.

Fig. 2.3 shows the rate of reaction of collagenase in the absence of the synthetic inhibitor.

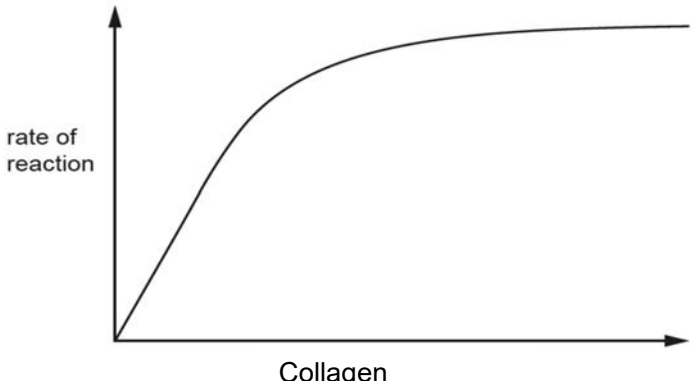
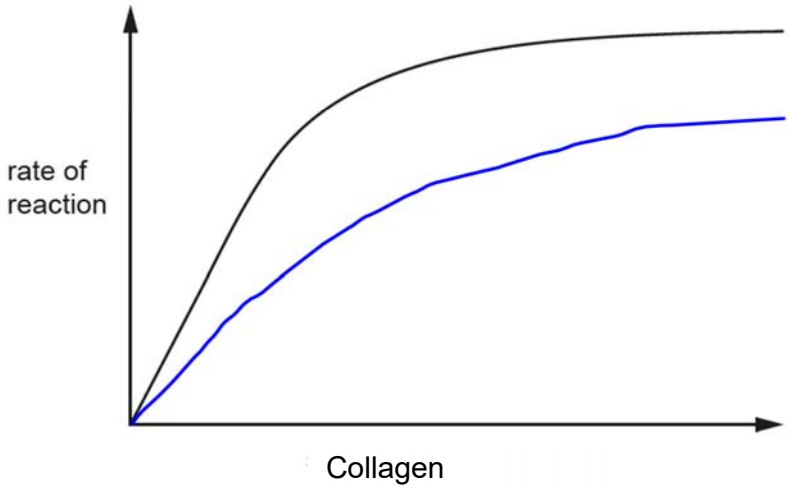


Fig. 2.3

(d) Sketch on Fig. 2.3 the curve that is expected if the synthetic inhibitor used in the trial is a non-competitive inhibitor. [1]

Note:
The 2 graphs cannot overlap at the initial rate of rxn. Separate right for the start.



[Total: 9]

multipotent, undergoes self-renewal via mitosis

3 Adult stem cells in a tissue are often at different stages of the cell cycle.

(a) Fig. 3.1 shows cells at different stages of the cell cycle.

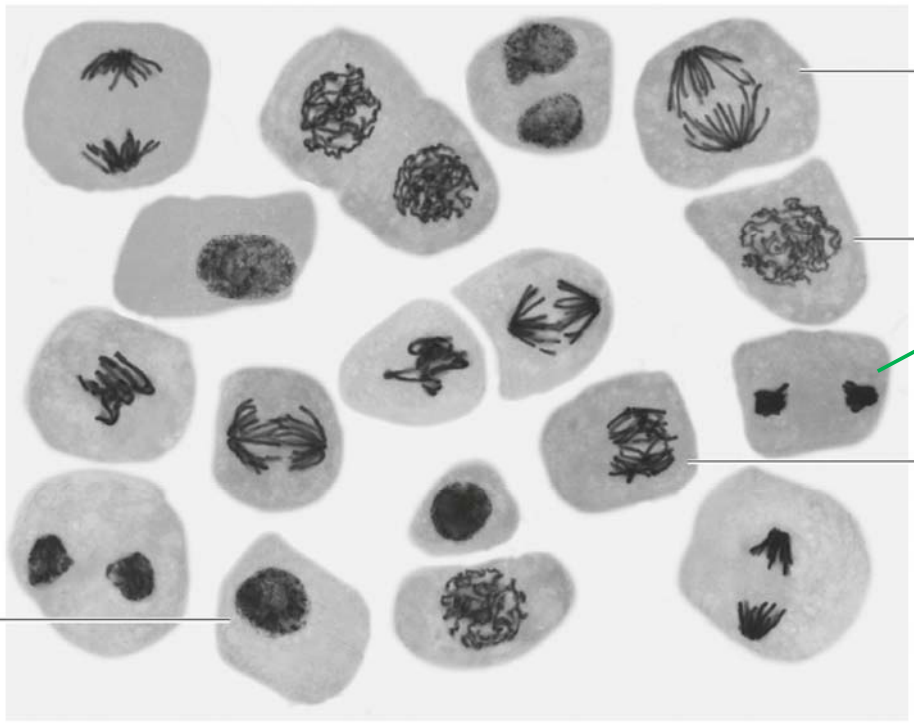


Fig. 3.1

P, M, A, T

(i) Identify the stages of mitosis occurring in the cells labelled B and C in Fig. 3.1. [2]

B prophase

C metaphase

(ii) Describe the behaviour of the chromosomes in the stage of mitosis shown in cell A.[2]

1. The centromere of each chromosome divides (reject split),
2. causing the sister chromatids of each chromosome to separate.
3. The sister chromatids move to opposite poles (reject ends/ respective ends) of the cell, centromeres first/ led by centromeres first.
4. This is due to the shortening of the spindle fibres.

(b) Distinguish between adult stem cells and zygotic stem cells. [2]

Contrasting Features	Zygotic stem cells	Adult stem cells
1. Potency	Totipotent	Multipotent

2. Cell specialisation ability	Have the **ability** to divide and **differentiate** into **ANY CELL TYPE** to form whole organisms.	Have the **ability** to divide and **differentiate** into **a LIMITED RANGE of cell type**
3. Self-renewal capability	**Limited** self-**renewal** capability, because they **no longer exist after a certain stage in the development of the embryo**	Undergo **continuous self-renewal throughout the lifetime of the organism**
4. Function	Can **differentiate** to **form all cells in** the **body** of the organism, **as well as the placenta**.	Cells **differentiate to form new cells to replace dead and worn-out cells of the same cell type** in the tissue that they are found.

(c) **Other than stem cells**, some human cells show a **higher than normal activity of telomerase**.

State the type of cells with higher than normal activity of telomerase and **explain** the **role of telomerase** in these cells. [3]

Type of cell **Cancer cell [1]**

reasons **coded by telomerase gene**

Explain role of **telomerase**.

1. **Telomerase will lengthen/extend the telomeres.**
2. **To prevent the shortening of the telomeres to critical length before affecting the genes**
3. **So that cells will not undergo apoptosis.**
4. **Cancer cell divides uncontrollably/replicate indefinitely.**

Note: ECF for points 1 and 2 if cell identified in earlier part is incorrect.

[Total: 9]

4 Table. 4.1 shows the probabilities of being diagnosis with cancer in the various age groups. Each year, more than 1 million cases of cancer are diagnosed in the United States and more than 500 000 people die from the disease.

Table 4.1

Cancer site	Gender	Age		
		Birth to 39	**40-59**	**60-79**
Breast	Female	1 in 235	1in 25	1 in 15
Prostate	Male	<1 in 10 000	1 in 53	1 in 7
Lung	Male	1 in 3300	1 in 92	1 in 17
	Female	1 in 3180	1 in 120	1in 25
Colon	Male	1 in 1500	1 in 124	1 in 29
	Female	1 in 1900	1in 149	1 in 33

(a) Using the information in Table 4.1,

(i) state the relationship between the age of a person and the likelihood of being diagnosed with cancer [1]

Likelihood of being diagnosed with cancer increases with age.

R: it has a direct / positive relationship

(ii) suggest a reason for your answer in **(a) (i)**. [1]

Time is needed to accumulate mutations / changes in genes related to tumours;

OR

Cancer / tumour takes time to develop;

Idea marking : Time + accumulation of mutation (1/2 m only if idea of time is not mentioned in answer.)

It is observed that *Ras* gene is mutated in 30% of the cancer cells.

Ras proto-oncogene codes for the Ras protein, a G protein that relays a signal from a growth factor receptor on the cell surface membrane.

Fig. 4.1 shows the cell signaling pathway involving the Ras protein. Accumulation of cyclin **D**, cyclin **E** and **E2F** proteins results in cell division.

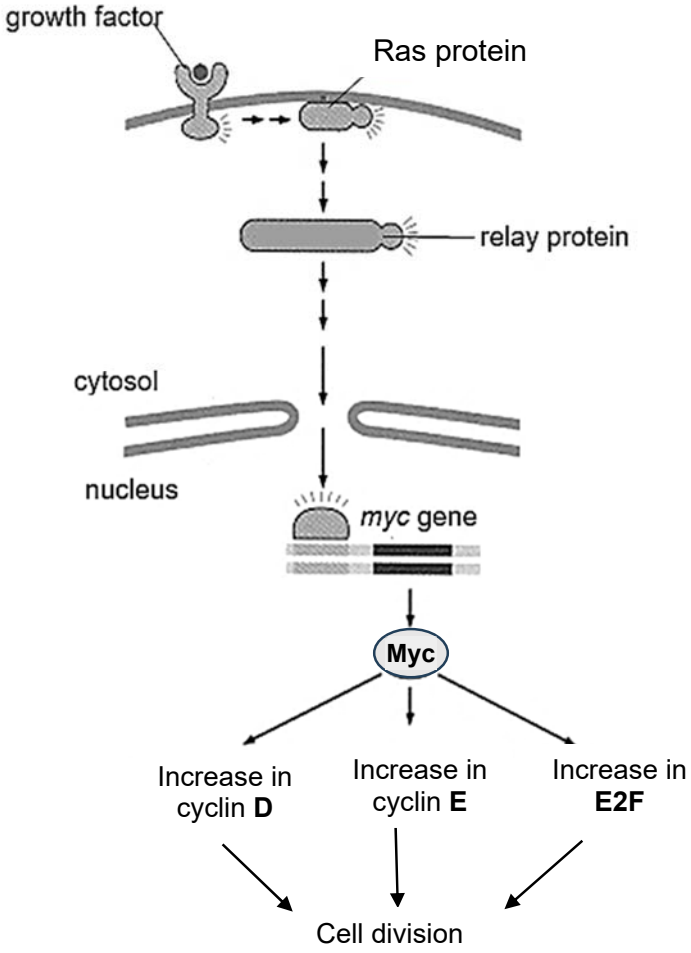


Fig. 4.1

(b) Using your knowledge and Fig. 4.1, explain how a mutation in the *Ras* gene can result in the development of cancer. [3]

1. **A gain in function mutation of the *Ras* gene**
2. **GTPase unable to hydrolyse GTP to GDP.**
3. **Ras protein is still associated with GTP leading to continuous activation of the Ras protein (OR hyperactivity of the Ras protein).**
4. **The hyperactive Ras protein stimulate expression of myc protein continuously.**
5. **Leading to increase in production of cyclin E, cyclin D and E2F proteins resulting in continuous cell division.**
6. **even in the absence of the growth factor.**

Note to marker: the idea of continuous can be marked either in point 4 or 5, only marked once.

(c) Suggest **one** reason why a mutated Ras protein in an eukaryotic cell will not always cause cancer directly. [1]

Any one

1. **Idea of cancer as a multi-step process – require accumulating at least half a dozen of mutations within one single cell to drive the cell towards cancer / uncontrolled cell division.**
2. **Tumour suppressor genes code for proteins that can inhibit cell cycle which will limit uncontrolled cell division.**
3. **Due to shortening telomeres after numerous rounds cell division that reduce telomeres to a critical length, triggering apoptosis.**
4. **Cells that present abnormal proteins / antigens, will be killed by natural killer cells or cytotoxic T-cells with perforins and granzymes.**
5. **AVP**

Ras protein signaling pathway also leads to activation of glycogen phosphorylase, which catalyzes the breakdown of glycogen. Glycogen as a polysaccharide is composed of thousands of monomers.

Oligosaccharides are carbohydrates that contain three to ten monomers in their chains.

Nystose is one example of an oligosaccharide. The structure of nystose is shown in Fig. 4.2.

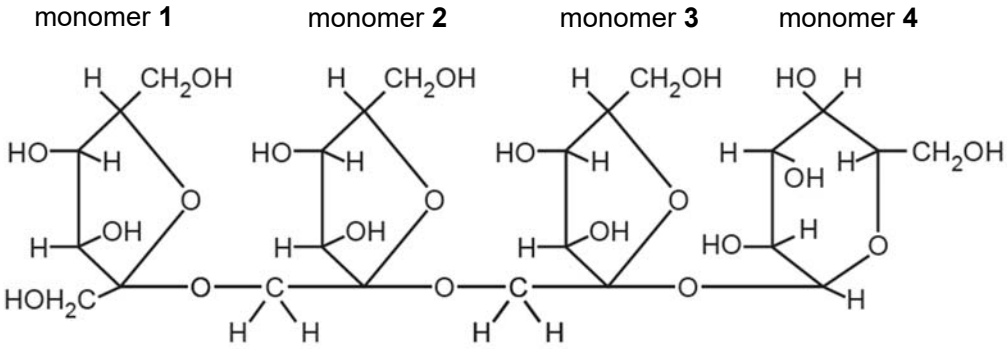


Fig. 4.2

(d) **(i)** Name the bond that is formed between monomer **3** and monomer **4**. [1]

Glycosidic bond

(ii) Other than the number of monomers in the molecules, describe **one** difference between the structures of nystose and glycogen.

***any one from*:**

1. **two types of monomer in nystose while only type of monomer in glycogen (which is glucose)**
2. **nystose is not branched while glycogen is branched**
3. **nystose has linear (non - helical) chains while glycogen has helical chains**

Reject:

1. **Nystose is made up of pentose / 5-carbon sugar and while glycogen is made up of hexose / 6-carbon sugars. Note that nystose also has 6-carbon sugars, does not have pentose.**
2. **Linear vs branched -> not PTPC.**
3. **Correct comparison =**

a. **non-helical vs helical**

b. **branched vs unbranched,**

c. **straight chain vs circular chain.**

(iii) Cells use oligosaccharides to synthesise glycoproteins, which are transported to cell surface membranes as receptors.

Describe the roles of the rough endoplasmic reticulum and the Golgi body in synthesizing glycoproteins. [5]

1a. Ribosome bound to the rough endoplasmic reticulum (rER) synthesise the polypeptide chain

1b. into the rough endoplasmic reticulum lumen

2a. [Note this is a receptor protein] Polypeptide chain is inserted into the membrane of the transport vesicle,

2b. which buds off from the ER.

3a. travels along microtubules of the cytoskeleton and

3b. fuses with the cis-face of the Golgi apparatus (GA)

4. [chemically modify] glycosylation of protein in GA / addition of the oligosaccharides to the polypeptide chains. [1mark]

5a. The vesicle containing the glycoprotein buds off from the trans-face of the GA, travels along microtubules of the cytoskeleton

5b. and fuse with the cell surface membrane, inserting the glycoprotein in the cell surface membrane.

Total: 14]

5 Fig 5.1 shows the mTOR intracellular signalling pathway that is involved in the control of blood glucose level.

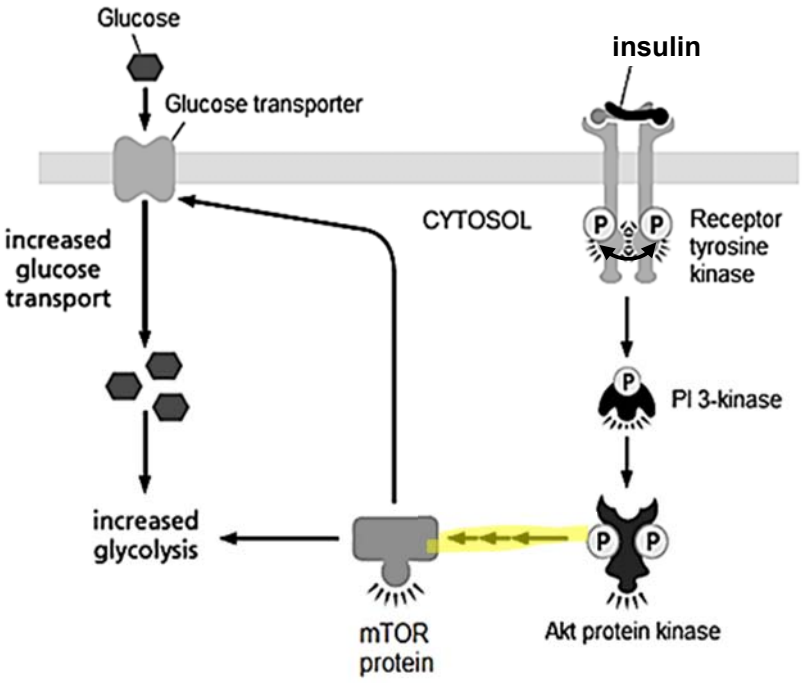


Fig. 5.1

(a) Describe how insulin leads to the activation of mTOR protein in Fig. 5.1. [3]

1. **Insulin binds to the extracellular side / ligand binding site of the receptor tyrosine kinase (RTK).**
2. **RTK undergoes a conformational change and becomes activated.**
3. **Cross-phosphorylation occurs, where tyrosine kinase of each subunit phosphorylates tyrosine residues on the intracellular tail of the other subunit**
4. **Both subunits are phosphorylated and is fully activated**
5. **Activated tyrosine kinase receptor then phosphorylates and activate PI 3-kinase**
6. **which phosphorylates and activate Akt protein kinase;**
7. **Akt protein kinase phosphorylates other kinases / in a phosphorylation cascade (reject signal transduction pathway)**
8. **and activates the mTOR protein. (NOTE: Atk is NOT the protein that activates mTOR protein. Multiple arrows refer to many steps in between)**

Max 3 marks

(b) State **two** differences between **signal reception** in the pathway in Fig. 5.1 and glucagon signaling pathway. [2]

Any 2

1. **The insulin receptor is a receptor tyrosine kinase while the glucagon receptor is a G-protein coupled receptor.**
2. **Upon ligand binding, cross-phosphorylation of tyrosine residues on the intracellular tail of the other subunit while cross-phosphorylation does not occur in glucagon signaling pathway.**

3. **Activation of the RTK requires conformation change and phosphorylation of tyrosine residues while activation of the GPCR only involved conformational change after ligand binding.**
4. **Activated RTK does not bind to G-protein while activated GPCR binds to inactive G-protein.**

Accept AVP

(c) Describe how the receptor tyrosine kinase and glucose transporter is **held** in the membrane. [2]

1. **Both proteins have both hydrophilic and hydrophobic regions.**
2. **The non-polar amino acid residues interact with hydrophobic fatty acid chains of phospholipids via**
3. **hydrophobic interactions.**
4. **The hydrophilic (polar or charged) amino acid residues interact with hydrophilic phosphate heads of phospholipids via**
5. **hydrophilic interactions such as hydrogen bonds, ionic bonds.**

Max marking

(d) Suggest how the activated mTOR protein bring about the desired cellular responses to control blood glucose level.

1. **mTOR protein activate glucose transporter to increase uptake of glucose molecules into the cell.**
2. **mTOR protein increase the uptake of glucose molecules into the cell via increasing number of glucose transporters on cell surface membrane. [scientifically correct but not seen in picture, command word is"suggest"]**
3. **mTOR protein triggers increase in glycolysis, thus increase import of glucose into the cell.**
4. **mTOR increases production of enzymes that carry out glycogenesis, thus increase import of glucose into the cell.**

idea, 1 mark marking (note must have "into the cell")

Total: 8]

6 The unicellular green alga, *Chlorella*, a photosynthetic organism is studied for its many health benefits. It is produced and harvested for use as a health food supplement.

(a) To analyse the productivity of *Chlorella*, carbon dioxide concentration was altered to investigate its effects on the light-independent stage of photosynthesis.

- A cell suspension of *Chlorella* was illuminated using a bench lamp.
- The suspension was supplied with carbon dioxide at a concentration of 1% for 200 seconds.
- The concentration of carbon dioxide was then reduced to 0.03% for a further 200 seconds.
- The concentrations of RuBP and GP (PGA) were measured at regular intervals.
- Throughout the investigation the temperature of the suspension was maintained at 25 °C.

The results are shown in Fig. 6.1.

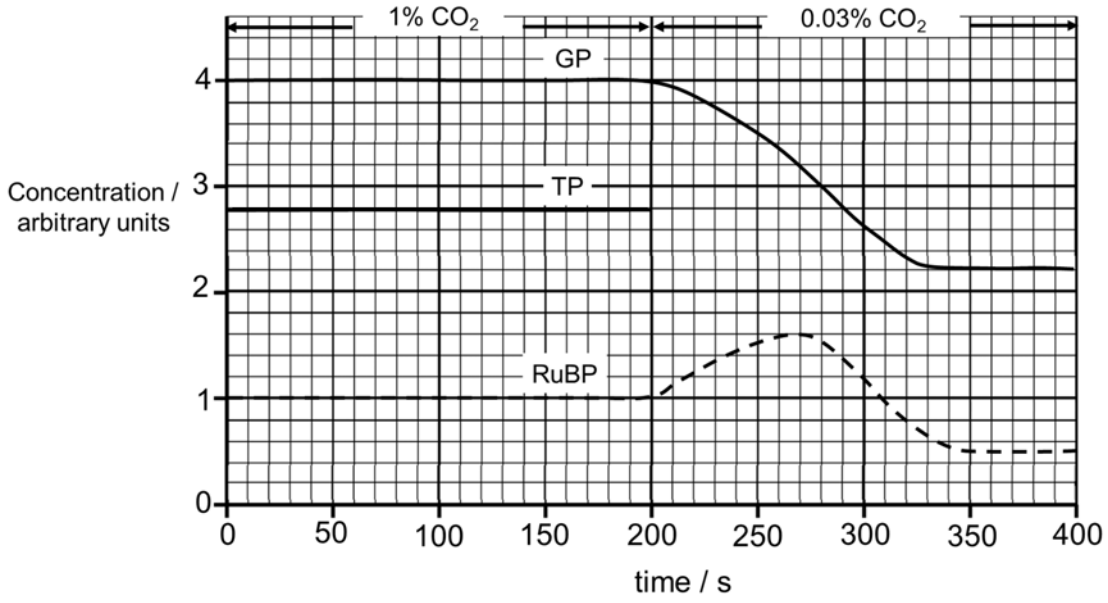


Fig. 6.1

(i) State **precisely** where RuBP and GP are located in the chloroplast. [1]

- **Stroma**

(ii) **Explain** the change in the concentration of **RuBP** between 200 and 275 seconds. [2]

1. **As time increased from 200 and 275 seconds. there is an increase in concentration of RuBP from 1 to 1.6 a.u. (MUST have units!!)**
   Note: Must give trend & QF because the question stem did not provide the trend
2. **This is due to lower CO_2 concentration of 0.03%;**
3. **less carbon fixation OR less CO_2 combine with RuBP**
4. **At the same time, RuBP is still regenerated from triose phosphate / glyceraldehyde-3-phosphate**
   NOTE:
   - **MUST use the term "regenerate" [R.G.]**
   - **Do NOT use "break down"**
   - **RuBP is regenerated from TP / GALP, NOT GP.**

(iii) **Calculate** the rate of decrease per second in the concentration of GP between 200 and 350 seconds.

Show your working and present your answer to **two decimal places**

Working:
Decrease in concentration of GP = 4 − 2.2 = 1.8 [1/2]
Time period = 350 – 200 = 150 [1/2]

rate of decrease = 1.8 ÷ 150 [1/2]

OR rate of decrease= (4 − 0.2) divided by 350 – 200

0.01 arbitrary units per second [2]

(b) **Suggest** how the decrease in the concentration of GP leads to a decreased harvest for commercial suppliers of *Chlorella*. [2]

1. **Less TP / GALP will be formed from GP.**
2. **Less conversion of TP to glucose / starch / lipids / amino acids / proteins / cellulose.**
3. **Less proteins for growth / cell division**
4. **Less carbohydrate / lipids for cellular respiration to produce ATP for cell division.**
5. **Decrease growth / cell division**
   Reject: Decrease in dry mass
   Reason: Dry mass refers to mass without water.

(c) In the absence of light, rubisco changes shape from an active form to an inactive form.

Briefly explain why rubisco **does not need** to be in an active form in the absence of light. [2]

1. **Rubisco catalyse CO_2 fixation.**
2. **Absence of light means there is no light dependent reaction / no photophosphorylation.**
3. **No ATP and reduced NADP / NADPH synthesized.**
4. **Calvin cycle / light independent reaction also stops if there is insufficient ATP & reduced NADP.**
5. **As no RuBP regenerated for carbon dioxide fixation, so no CO_2 fixation.**

(d) *Chlorella* can respire aerobically and anaerobically. When *Chlorella* cells switch from aerobic to anaerobic respiration, there is in a significant increase in the rate of glucose uptake and glycolysis in the *Chlorella* cells.

Suggest why the rate of glycolysis increases significantly when *Chlorella* cells switch from aerobic to anaerobic respiration. [3]

1. **ABSENCE of oxygen (i.e. anaerobic condition), no oxygen as final electron acceptor,**
2. **Electron transport chain CANNOT function OR no regeneration of NAD and FAD.**
3. **So oxidative phosphorylation CANNOT occur to produce 34 ATP per glucose.**
4. **Krebs Cycle and link reaction CANNOT occur.**
5. **only small amount of NAD^+ regenerated only sufficient for glycolysis.**
6. **Only glycolysis can occur to produce net 2 ATP per glucose via substrate level phosphorylation.**
7. **Rate of glycolysis increases because anaerobic respiration only produce net 2 ATP per glucose to match 38 ATP per glucose in aerobic respiration.**

[Total: 12]

7 (a) During DNA replication, two new daughter strands are synthesised using the original strands as templates.

(i) **State why** the antiparallel nature of the DNA molecule results in one of the strands being synthesised in short fragments. [1]

- **DNA Polymerase can only add to existing 3′ OH OR 3' end of an existing strand.**
  OR
- **Shape of DNA polymerase active site is complementary to shape of 5' phosphate group of in-coming nucleotide and 3'-OH of the last nucleotide of growing daughter strand.**
  This is the primary focus:
  It is because of how DNA polymerase catalyse joining of phosphodiester bond that give rise to Okazaki fragments, when the strands are antiparallel - rather than just stating lagging strand synthesized away from replication fork.
  OR
- **DNA Polymerase can only read the template from 3' to 5' direction.**
  OR
- **The new strand / daughter strand is only synthesized 5′ to 3′ direction.**

(ii) Template DNA, enzymes and ATP are necessary for DNA replication.

State **one** other component required for the process. [1]

- **Primer OR deoxyribonucleotides OR single-strand binding protein.**

Scientists investigated the cell cycle in heart cells taken from mice 6 days before their birth and then at 4, 14 and 21 days after their birth.

The results are shown in Table 7.1.

Table 7.1

Age / days	Percentage of heart cells undergoing mitosis	Percentage of heart cells undergoing DNA replication
−6	13.9	8.5
4	8.5	2.6
14	1.6	0.2
21	0.6	0.0

Age 0 days = day of birth

(b) With reference to Table 7.1, **explain** the decrease in DNA replication **(trend given so no need to state this trend and QF)** in the heart cells **after** the **birth** of the baby. [2]

1. **From day 0 to day 21, there is a decrease in the number of cells dividing by mitosis**
2. **after birth from 8.5% to 0.6%**

3. **DNA replication which takes place before mitosis decreases / takes place during S phase of interphase decreases**
4. **The rate of heart growth slows as many of the heart cells lost the ability to divide**
   OR
   Fewer new heart cells needed after birth.

(c) The scientists determined the percentage of heart cells undergoing DNA replication by using a chemical called BrdU. These cells use BrdU instead of nucleotides containing thymine during DNA replication.

Describe how BrdU would be **incorporated** into new DNA during semi-conservative replication.[2]

1. **Helicase breaks hydrogen bonds between 2 DNA strands.**
   OR
   DNA strands separate.

2. **BrdU form complementary base pair with adenine on template strand.**
   OR
   BrdU forms hydrogen bonds with adenine on template strand.

3. **DNA polymerase catalyse formation of phosphodiester bonds**

4. **between BrdU and adjacent nucleotides.**

5. **New DNA molecule consists of one parental strand (with thymine) and one daughter strand (with BrdU).**
   Note: Do not mix up molecule and strand.

The scientists also investigated the function of a protein called cyclin **A**, which binds to and activates one of the enzymes required at the start of DNA replication.

The percentage of cells with replicating DNA in different cell cultures was recorded as shown in Table 7.2.

Table 7.2

Cell Culture	Treatment given	Percentage of cells where DNA was replicating
C	Control cells, untreated	91
D	Antibody added that binds specifically to cyclin **A**	11
E	RNA added that prevents translation of cyclin **A**	10
F	Both RNA that prevents translation of cyclin **A** and cyclin **A** protein were added	92

(d) With reference to Table 7.2, **identify** and **explain** the treatment(s) that are suitable for targeting cancer. [3]

Note: To target cancer is to reduce cell division – can also have same effect by stopping DNA replication.

Treatment **D [1/2] and E [1/2]**

Explanation

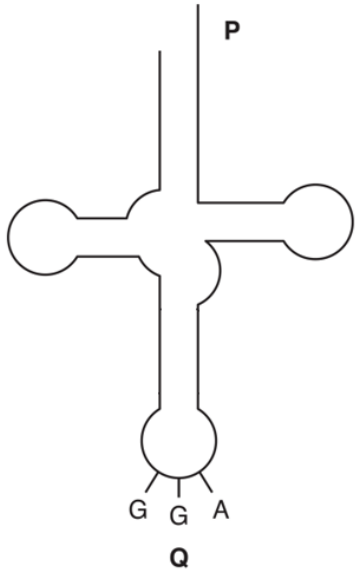
1. **QF: Low percentage of cells with replicating DNA for treatment D (11%) & treatment E (10%)**

2. **Treatment D, the antibody binds to cyclin A so that cyclin A cannot bind and cannot activate enzyme required to start DNA replication.**

   Treatment E, RNA interferes with translation so cyclin A is not synthesized.

3. **DNA replication cannot be initiated / no DNA replication, cancer cells cannot divide uncontrollably.**

Fig. 7.1 shows a molecule of tRNA involved in the process of translation.



(e) With reference to Fig. 7.1, **state** the name of region **Q** and **explain** the role of **Q** in translation. [3]

Name **anticodon [1]**

Explanation

1. **Anticodon on tRNA binds to codon on mRNA**
2. **via complementary bases pairing (c.b.p)**
3. **carries a specific amino acid to the ribosome during translation.**
4. **Results in correct amino acid sequence of polypeptide chain.**

[Total: 12]

8 (a) Scientists have produced structures known as virosomes, which are used in certain vaccines.

Virosomes do not cause disease.

Fig. 8.1 is a diagram of a section through a virosome used in some vaccinations to protect against the virus which causes influenza.

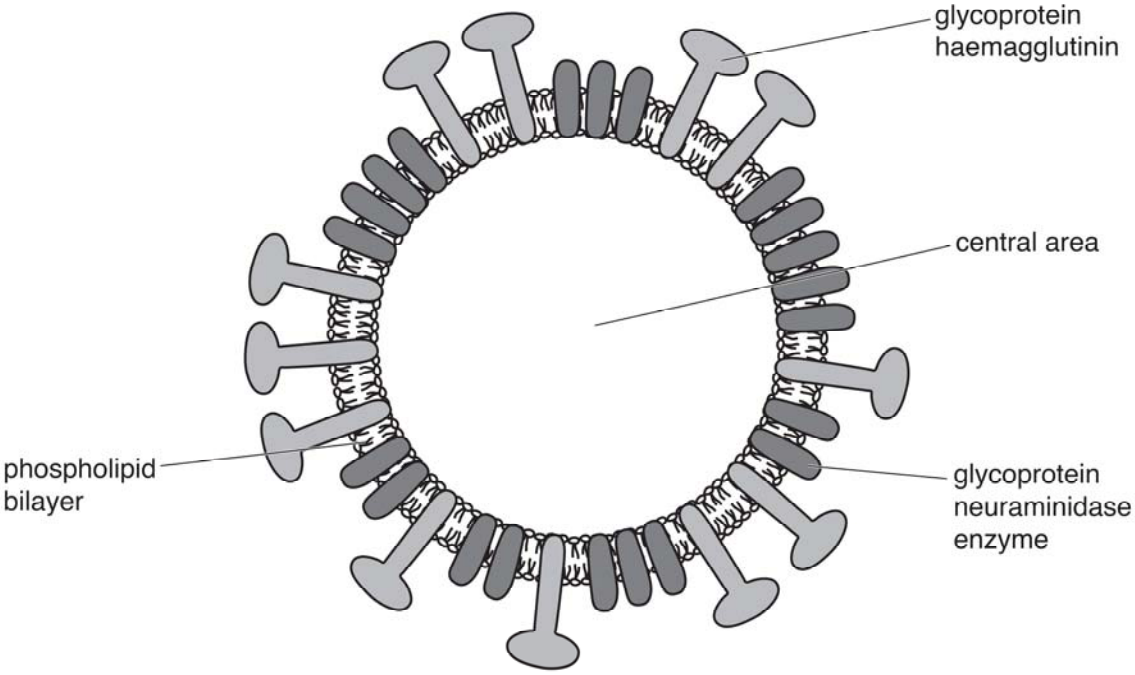


Fig. 8.1

(i) State **one** difference between the structure of a virosome and an influenza virus. [1]

1. **Virosome has no RNA / genetic material while influenza virus has 8 segments of RNA.**
2. **Virosome has no capsid / protein coat / while influenza virus has.**

(ii) Explain how the structure of the virosome shown in Fig. 8.1 suggests that the central area of the virosome is aqueous. [2]

1. Phosphate head of phospholipids are hydrophilic. [1 mark]

2a. point towards the centre of the virosome [1/2]

2b. interact with the central area via hydrophilic interaction (ionic bond / hydrogen bond). [1/2]

OR

2a. Hydrophobic fatty acid chains of **phospholipids face inwards** of phospholipid bilayer / **face away from central area** and **[1/2]**

2b. are **sandwiched** between **phosphate heads [1/2]**

(b) Haemagglutinin and neuraminidase are found in the virosomes which are used in a vaccine against the influenza virus.

Briefly explain why virosomes must contain haemagglutinin. [3]

1. Haemagglutinin acts as a non-self / foreign antigen.

2. Triggers / stimulates, primary immune response
or

provides artificial active immunity.

3. **Antigen phagocytosed by an antigen-presenting cell / macrophage will be presented to naive CD4 T cells.**

4. **This activates the naive CD4 T cell which then proliferate / divides by mitosis and differentiate to become helper T cells and memory T cells.**

5. **Naive B cell which recognises and binds to same antigen, with cytokines from helper T which completes the activation of B cell.**

6. **B cells proliferate and differentiate to form plasma cells which secrete antibodies and memory B cells.**

7. **Formation of memory B and T cells will allow the body to quickly mount a secondary immune response when infected by the influenza virus in future.**

Different strains of the influenza virus ~~have~~ **are** formed as a result of mutations. However, it was observed that the primary structure of the neuraminidase enzyme active site remains unchanged in each strain of the virus.

(c) **Suggest** why the primary structure of the active site of neuraminidase remains unchanged in each strain of the influenza virus. [2]

1. **If neuraminidase gene is mutated / change in 3-D conformation of active site.**
2. **neuraminidase cannot cleave the sialic acid receptors**
3. **unable to facilitate release of new viral particles**
4. **will not allow new viruses to infect other host cells so, the mutation is not passed on.**

For students who wrote about the role of neuraminidase:

5. **neuraminidase can cleave the sialic acid receptors**
6. **facilitate release of new viral particles**

(d) Occasionally antigenic shift occurs in the influenza virus, resulting in human viruses responsible for influenza pandemic.

State **two** differences between antigenic shift and antigenic drift. [2]

Type of genetic change:

1. **Antigenic shift involves genetic recombination of RNA segments while genetic drift involves changes to nucleotide sequences / gene mutations.**

Number of strains of viruses involved:

2. **Antigenic shift involves many influenza strains infecting the same host cell while antigenic drift can take place with one strain infecting the host cell.**

Types of glycoprotein spikes in new viral particles:

3. **Antigenic shift results new combinations of glycoprotein spikes while antigenic drift same type of glycoprotein spikes with increased infinity.**

Effects of each:

4. **Antigenic shift results in pandemic while antigenic drift results in seasonal epidemic.**

Ability to infect new species of animals:

5. **Antigenic shift results in a new strain that can infect / jump from one animal species to another while antigenic drift results in new strain that bind more effectively to same host cells.**
   OR
   Antigenic shift results in a new strain that can infect / jump from one animal species to another while antigenic drift does not.

[Total: 10]

9 The fruit fly, Drosophila melanogaster, has autosomal genes for body colour and wing shape. Pure bred wild type flies have dominant phenotypes.

Gene B/b is involved in the production of body colour:
- B = dominant allele for brown body colour
- b = recessive allele for black body colour.

Gene D/d is involved in wing shape:
- D = dominant allele for straight wing
- d = recessive allele for curved wing.

A dihybrid test cross was carried out between flies heterozygous for body colour and for wing shape and flies homozygous recessive for body colour and for wing shape.

Table 9.1 shows the number of offspring of each phenotype obtained in the test cross.

Table 9.1

phenotype	observed number	expected number
brown body colour, straight wings	2843	**1827**
brown body colour, curved wings	855	**1827**
black body colour, straight wings	842	**1827**
black body colour, curved wings	2768	**1827**

(a) Use the information in Table 9.1 to **calculate** the **expected** number of each phenotype if the two genes are on different autosomes. Write your answers in Table 9.1. [1]

(b) A chi-squared (χ^2) test was carried out to compare the observed results with the results that would be expected from a dihybrid cross involving genes on different autosomes.

The value of $\chi^2 = 20.98$

Table 7.2 shows the critical values for the χ^2 distribution.

Table 9.2

degrees of freedom	probability, p				
	0.10	*0.05*	*0.02*	*0.01*	*0.001*
1	2.71	3.84	5.41	6.64	10.83
2	4.61	5.99	7.82	9.21	13.82
3	6.25	7.82	9.84	11.35	16.27

4	7.78	9.49	11.67	13.28	18.47

(i) **Explain how** the value of χ^2 and Table 9.2 can be used to assess the significance of the difference between the observed results and the expected numbers in Table 9.1. [2]

1. **For 3 degrees of freedom and p = 0.05, the calculated χ^2 value of 2098 is more than 7.82, [1/2]**
2. **therefore the p-value is less than 0.05, [1/2]**
3. **The deviation is therefore statistically significant and not due to chance.**
4. **Reject null hypothesis. [1/2]**
   Therefore the 2 genes are on the same chromosome

(ii) Provide **explanations** for the test cross **observed** numbers shown in Table 9.1. [3]

1. **The genes for body colour and wing shape are linked / on the same chromosome.**
2. **Hence, the genes are inherited together**
3. **Therefore, a large number of gametes are the parental types.**
4. **During Prophase I, crossing over between homologous chromosomes occurs,**
5. **thus resulting in two new combination of alleles.**
6. **As crossing over is a chance event / random, the probability of getting a recombinant gamete is always lower thus resulting in small number of recombinant phenotypes.**

(iii) Complete Table 9.3 by **stating** the genotypes of the parents involved in the test cross which gave rise to the results in Table 9.1.

⇒ **Dominant B allele is linked with Dominant D allele**
⇒ **recessive b allele is linked with recessive d allele**

Table 9.3

parent phenotypes	brown body and straight wing	X	black body and curved wing
parent genotypes	**B b / D d**		**b b / d d**

v[2]

In a separate genetic studies, it is observed that the inheritance of fruit colour in summer squash plants is controlled by two genes, **A** and **B**. Each gene has two alleles.

Fig. 9.1 shows the interaction of these two genes in controlling fruit colour in summer squash plants.

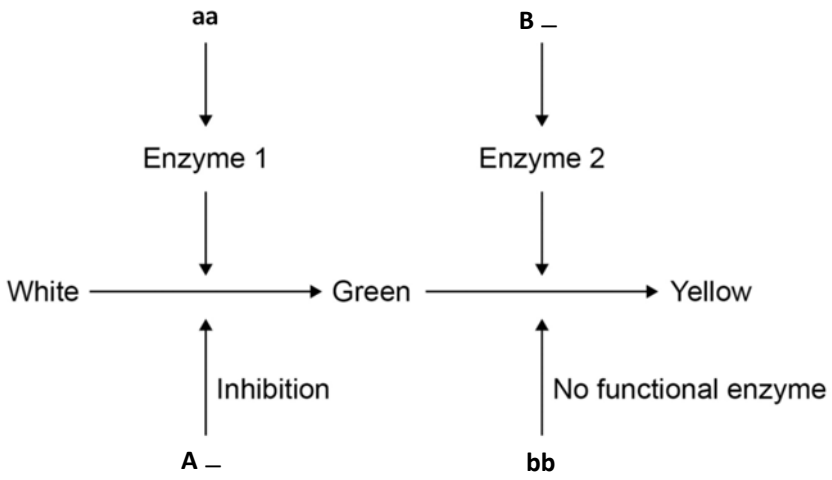


Fig. 9.1

(c) **(i)** Name the type of gene interaction shown in Fig. 9.1. [1]

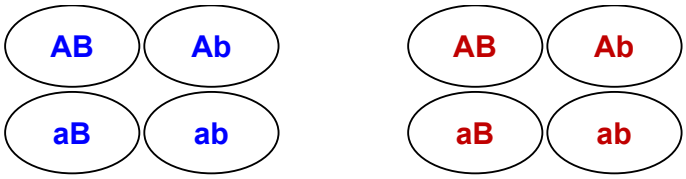
- **Dominant epistasis**

(ii) Genes **A** and **B** are not linked.

Complete the genetic diagram to show all the possible genotypes and the ratio of phenotypes expected in the offspring of this cross. [3]

Genotypes of parents **AaBb** × **AaBb**

Parental gametes:



	AB	Ab	aB	ab
AB	AABB white	AABb white	AaBB white	AaBb white
Ab	AABb white	AAbb white	AaBb white	Aabb white
aB	AaBB white	AaBb white	aaBB yellow	aaBb yellow
ab	AaBb white	Aabb white	aaBb yellow	aabb green

Offspring phenotypes:	**White squash**	**:**	**Yellow squash**	**:**	**Green squash**
Offspring phenotypic ratio:	**12**	**:**	**3**	**:**	**1**

[Total: 12]

10

Table 10.1 shows the numbers of dengue cases between 2007 and 2019 in Santa Catarina, a temperate climate state in Brazil.

Table 10.1

Year	Number of dengue cases
2007	7851
2010	9618
2013	11212
2016	12630
2019	14234

(a) **Calculate** the rate of increase in the number of dengue cases between 2007 and 2019.

Show your working and give your answer to the **nearest whole number**.

1. **Number of dengue cases increased from 2007 to 2019 = 14 234 – 7851 = 6383**
2. **Rate of increase over 12 years = 6383 / 12 = 531.9 = 532**

- **Working 1 m**
- **Answer 1m**

rate of increase = **532** per year [2]

(b) Using your knowledge of the effects of climate change, explain the rise in dengue cases between 2007 to 2019. [3]

1. **Idea marking – temperate region is now more favourable/suitable for the mosquitoes to survive (due to climate change)**

2. **Increased temperature (up to a threshold)**
3. **accelerates the emergence of *Aedes aegypti* mosquitoes / shortening mosquitoes life cycle,**
4. **[can only get this bonus mark if students write point 2 and 3] - thus increasing vector/mosquito population to spread dengue.**

5. **[must have] Increased temperature also reduces the extrinsic incubation period of the virus,**
6. **[must have] allowing the virus to replicate faster within the mosquito vector, increasing its spread.**

7. **Increased precipitation leads to increased rainfall,**
8. **resulting in more freshwater bodies as habitats for mosquito breeding.**

9. **Increased humidity leads to reduced desiccation of mosquito eggs,**
10. **allowing most of the eggs laid to hatch/develop into mosquitos, increasing vector population to spread dengue.**

Note to marker: max marks is 2 if points 5 and 6 are not included.

[Total: 5]