

RAFFLES INSTITUTION

2025 Year 6 Preliminary Examination
Higher 2

CANDIDATE
NAME

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CIVICS
GROUP

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INDEX
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BIOLOGY

Paper 2 Structured Questions

9744/02

24th September 2025

2 hours

Candidates answer on Question Paper.

No Additional Materials are required.

READ THESE INSTRUCTIONS FIRST

Write your index number, CT group & name in the spaces at the top of this page.
Write in a dark blue or black pen.
You may use a HB pencil for any diagrams or graphs.
Do not use staples, paper clips, glue or correction fluid.

Answer **all** questions 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

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	/ 8
3	/ 9
4	/ 10
6	/ 9
7	/ 11
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10	/ 8
11	/ 8
Total	/ 90

This document consists of **25** printed pages.



Section A

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

- 1 All cells are surrounded by a cell surface membrane.

All cell membranes have a similar basic structure. However, the proportions of the various molecules present vary between different membranes and even between the inner and outer layers of a membrane.

Fig. 1.1 shows a generalised cell surface membrane.

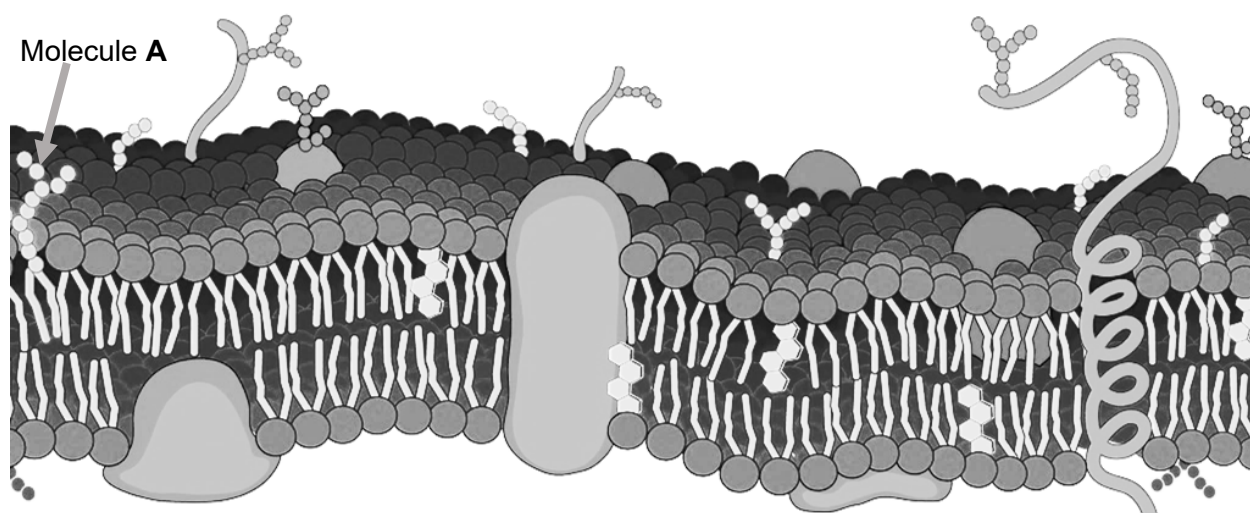


Fig. 1.1

- (a) Molecule **A** is a glycolipid. State a major role of glycolipids in the outer layer of the cell surface membrane. [1]
1. Act as markers/ antigens for cell-cell recognition* to distinguish cells as 'self'/'non-self' as basis of immune system;
 - or
 2. Act as markers for cell-cell recognition* resulting in cell adhesion allowing cells to be attached to one another to form tissues and organs;
 - or
 3. Act as receptor* for certain pathogens and toxins to bind to host cells and gain entry into cell (A: specific ligands for signal transduction);
- (b) In certain circumstances, additional phospholipid or glycolipid molecules are added to the cell surface membrane. These additional molecules are synthesised inside the cell.
- (i) State where lipids are synthesised inside the cell **and** where they are converted to glycolipids. [2]
 lipids synthesised smooth endoplasmic reticulum (R: SER);
 converted to glycolipids Golgi apparatus;
 - (ii) Suggest one reason why additional phospholipid or glycolipid molecules are added to the cell surface membrane. [1]
Actively dividing cells preparing to undergo cytokinesis may add additional phospholipids and/ or glycolipids in the cell surface membrane to replenish membrane/ ensure sufficient membrane available before it divides into two daughter cells;
 A: cell may lose membrane during processes such as endocytosis and phagocytosis;
 A: cell is growing;

In bacteria cells, it is important to maintain ion concentration gradients across the cell surface membrane. These gradients, particularly electrochemical gradients of ions like protons and sodium, are essential. They are needed in various cellular functions, including nutrient uptake, ATP synthesis, and maintaining proper cellular pH. The antibiotic Gramidicin A is used to treat bacterial infections.

Two molecules of the polypeptide Gramidicin A form the pore as shown in Fig. 1.2.

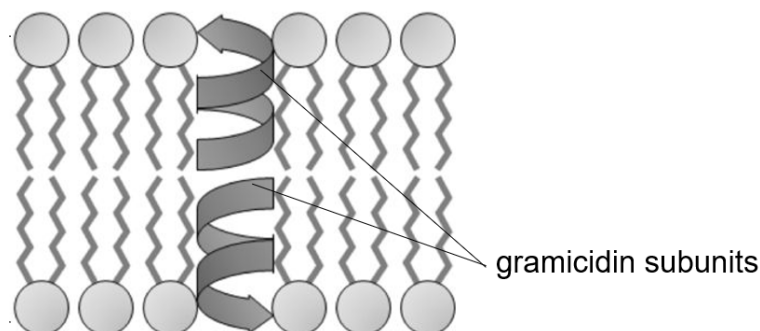


Fig. 1.2

- (c) Explain how Gramidicin A is used to treat bacterial infections. [3]
1. Gramidicin A has a hydrophilic pore, which allows ions to diffuse/ move down a concentration gradient;
 2. Resulting in equilibrium/ same concentration of ions on either side (A: cannot generate gradient) of the membrane being established);
 3. Loss of ion concentration gradient will disrupt important cell processes e.g. ATP synthesis and nutrient uptake/ protons which will lead to cell death;
- (d) Another antibiotic used to treat bacterial infections is penicillin. Describe the mode of action of penicillin. [3]
1. Penicillin acts as a competitive inhibitor* which binds to active site* of enzyme transpeptidase;
 2. This prevents transpeptidase from forming cross-links between peptidoglycan chains resulting in weakened bacterial cell walls in actively dividing cells;
 3. When bacteria take in water by osmosis, there is increased turgor pressure which causes bacteria to swell and lyse;

[Total: 10]

- 2 Glycolysis is the initial stage of respiration. It involves hydrolysis of glucose and the production of ATP. Fig. 2.1 shows part of glycolysis.

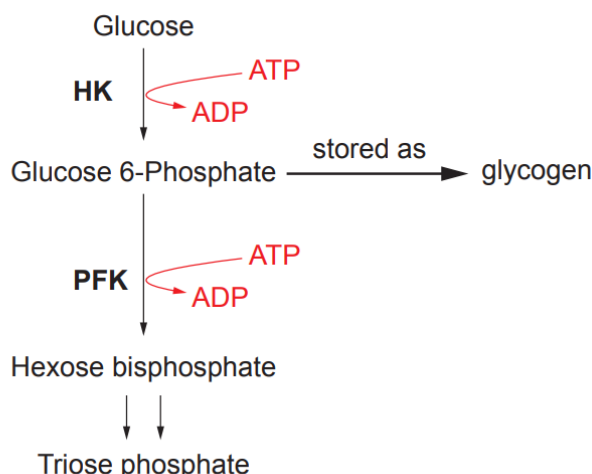


Fig. 2.1

Control of glycolysis is by end-product inhibition:

- High levels of ATP inhibit the enzyme PFK in the liver thus lowering its affinity for its substrate.
- PFK is the main regulatory enzyme in glycolysis, but it is not the only one.
- HK, the enzyme catalysing the first step of glycolysis, is inhibited by its product, glucose-6-phosphate.

- (a) Fig. 2.2 shows the effect of the concentration of substrate on the activity of the enzyme PFK.

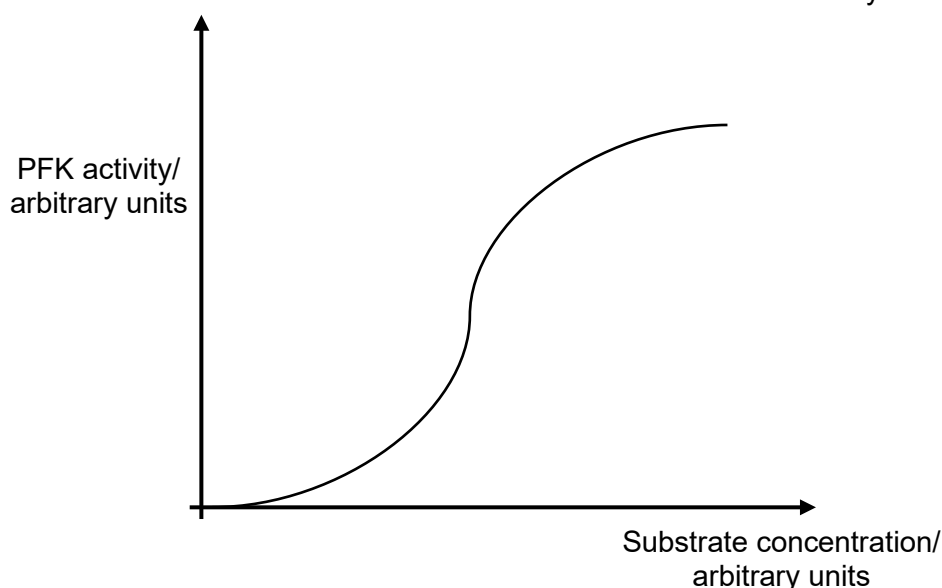
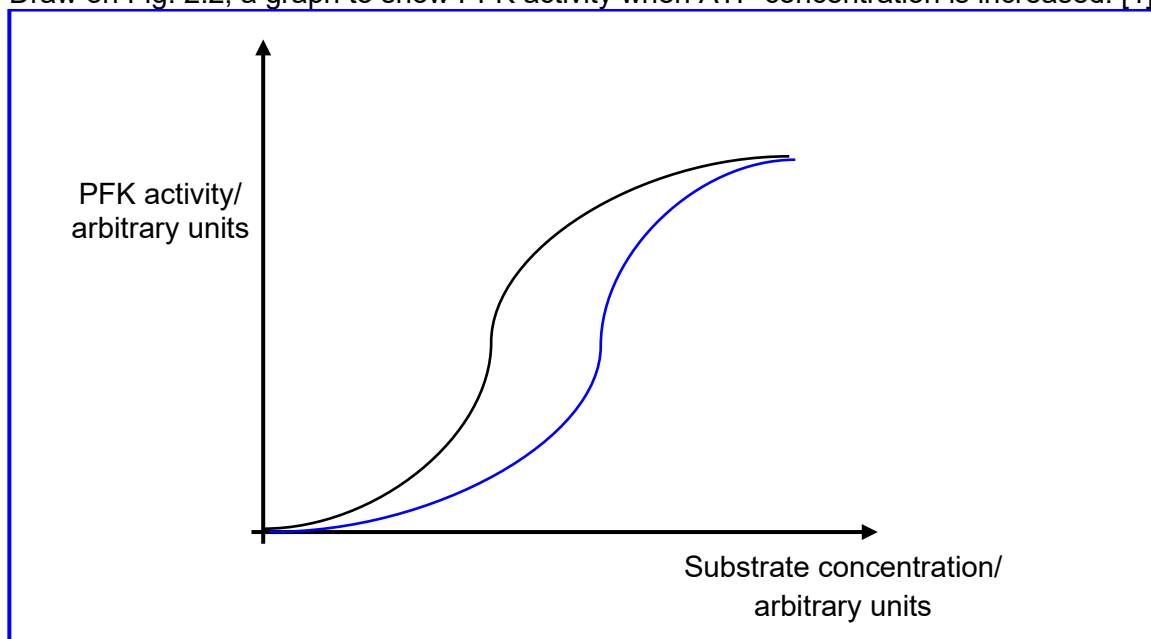


Fig. 2.2

- (i) Explain the shape of the graph shown in Fig. 2.2. [2]
1. PFK is an allosteric enzyme* with 2 or more subunits held together by weak intermolecular bonds, allowing movement that influences affinity for substrate,
 2. Allowing cooperative binding* of substrate hence sigmoidal/ S shape;
 3. The enzyme exists in two conformation states and binding of substrate at active site* stabilises active conformation;
 4. Binding of a substrate molecule to first subunit induces a conformational change in other subunits such that their affinity for substrate increases;

- (ii) Draw on Fig. 2.2, a graph to show PFK activity when ATP concentration is increased. [1]



- (iii) Explain the graph you have drawn. [2]

1. ATP is an allosteric inhibitor which binds to allosteric site*, stabilising PFK in its inactive conformation which slows down/ inhibits PFK activity;
2. The active site* of PFK has a lower affinity for its substrate;
3. Same maximum rate of reaction can be reached when substrate concentration is high;

- (b) With reference to Fig. 2.1, suggest why the cell uses inhibition of PFK as the main method of slowing glycolysis, rather than using the inhibition of HK. [1]

Product of HK, glucose 6-phosphate, can also be stored as glycogen (A: glycogenesis), hence inhibiting PFK is the most effective at inhibiting only glycolysis;

AVP: PFK catalyses the first committed step in glycolysis;

AVP: PFK catalyses the reaction where the glucose is activated so that glycolysis can take place;

- (c) Genetic mutation can result in PFK deficiency in humans. As a result, affected individuals display muscle weakness and fatigue. Explain why these individuals will display such symptoms. [2]

1. In the absence or decrease of functional PFK enzyme, glycolysis does not occur and cells are unable to produce pyruvate*;
2. Link reaction* and Krebs cycle* are unable to proceed due to lack of pyruvate;
3. Since no reduced NAD/ NADH* is formed, oxidative phosphorylation does not occur and;
4. ATP* is not formed, therefore there is insufficient energy for muscles;

[Total: 8]

- 3 Osteogenesis imperfecta (OI) is a genetic disorder of connective tissues caused by an abnormality in the synthesis or processing of type I collagen, a protein crucial for bone strength. It is also called brittle bone disease.

It is characterised by susceptibility to bone fractures and decreased bone density. While primarily affecting bones, OI can also impact other connective tissues like skin, ligaments, and teeth.

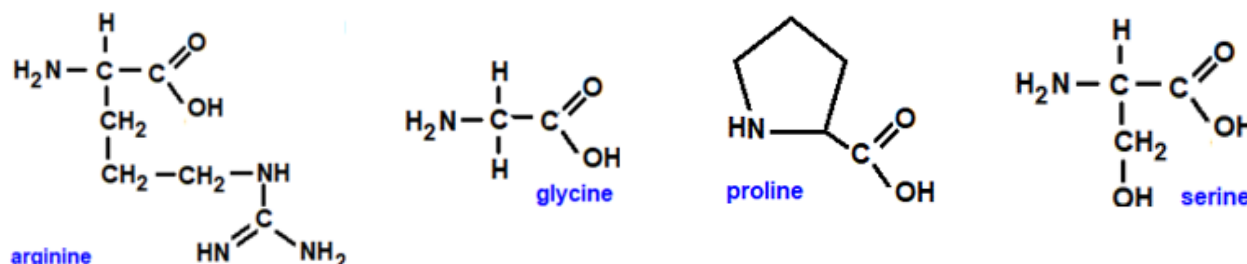


Fig. 3.1.

- (a) From Fig. 3.1, select the 2 amino acids that are most commonly present in collagen and show how they join in the formation of collagen. [3]
1. Labelled and correctly draw glycine + proline;
 2. Show condensation reaction/ lose of water molecule from OH and NH;
 3. Labelled peptide bond formed in the dipeptide; (to box the entire bond, R: pointing to a line)
- (b) Name the location where the process (a) occurs. [1]
Ribosome; R: rough endoplasmic reticulum only
- (c) With reference to the significance of R groups in the structure and function of collagen, explain the following:
- (i) mutations of Gly to Arg results in more severe cases of brittle bones than mutations to Ser and; [3]
1. Glycine* is smallest amino acid so that can fit in tight restricted space in center/ interior between 3 chains to form tight tropocollagen / triple helix; (R: helix)
 2. Arg has bulkier / larger side chain than Ser (A: reverse);
 3. Prevents tight packing to greater extent of tropocollagen* thus cannot form tropocollagen (A: reverse);
- (ii) mutations in an enzyme responsible for hydroxylating amino acid results in low tensile strength of collagen. [2]
1. Proline* and lysine in collagen chains not hydroxylated;
 2. Hydrogen bond* (between 3 chains in a tropocollagen) cannot be formed between hydroxy-proline and hydroxy-lysine between chains (R: tropocollagen molec);

[Total: 9]

- 4 The early development of an animal involves divisions of the zygote and daughter cells by mitosis. This forms an embryo consisting of genetically identical cells.

Fig. 4.1 shows several cells at various stages of the cell cycle in an embryo of whitefish, *Coregonus artedii*.

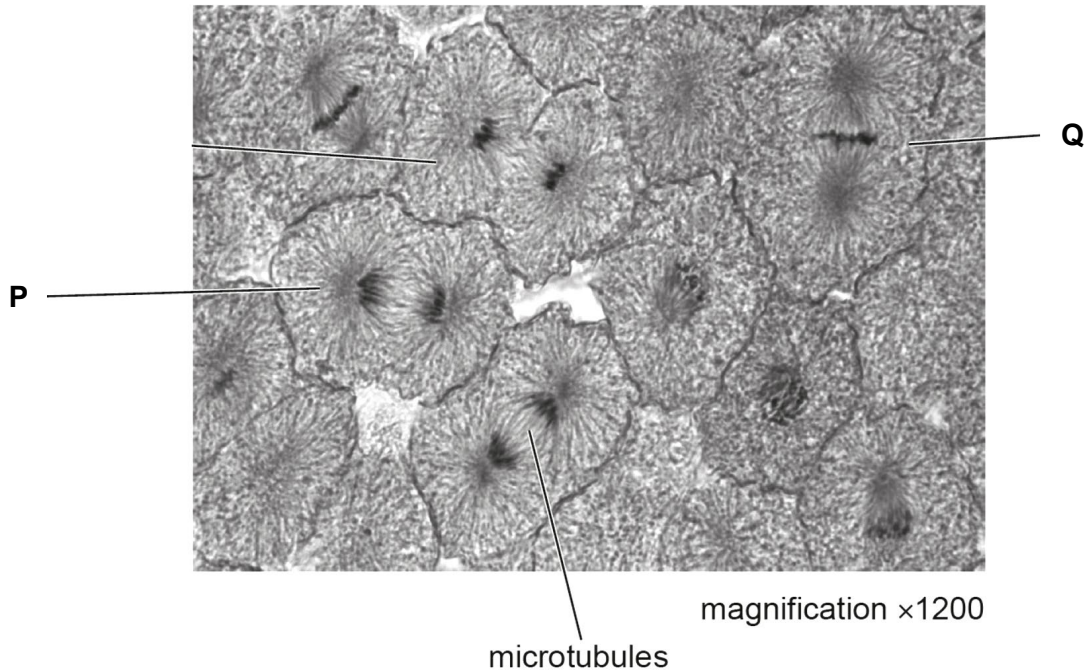


Fig. 4.1

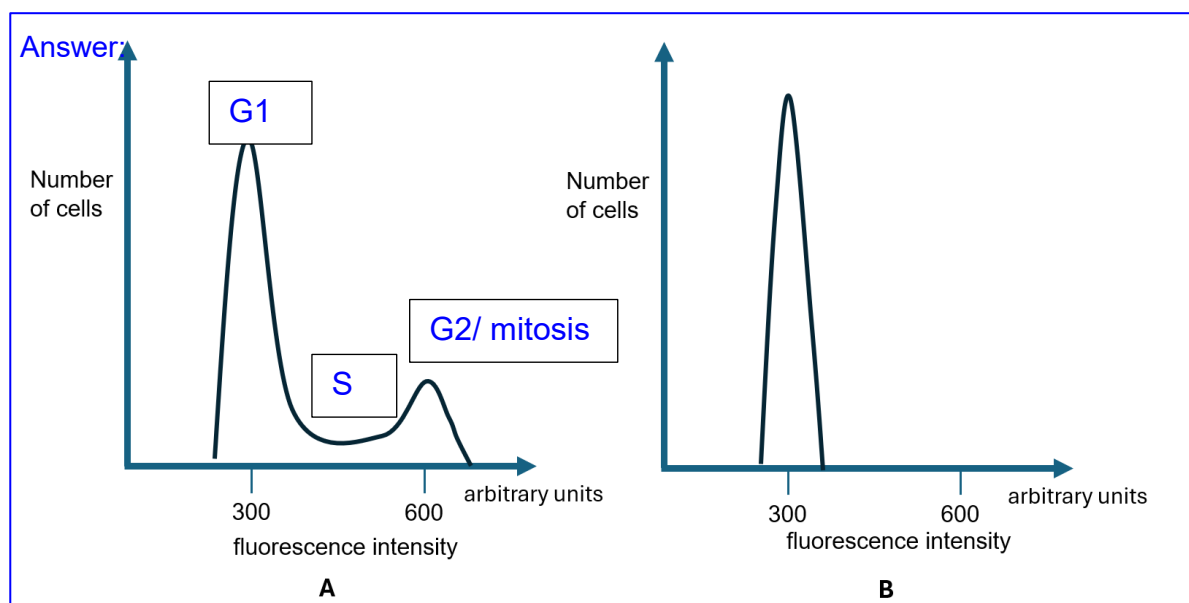
- (a) Explain how stages **P** and **Q** during the cell cycle will lead to genetically identical daughter cells. [2]
1. Stage **Q** which is metaphase* where chromosomes align singly on equator / metaphase plate* (so that the chromosomes can be equi-distant from the opposite poles);
 2. So that at Stage **P** which is anaphase*, centromere* divides and the genetically identical sister chromatids can separate and move to opposite poles* so each daughter cell receives a (sister / identical) chromatid of each chromosome;

- (b) Flow cytometry is used to analyse cell cycle phases by measuring DNA content. The DNA is stained with fluorescent dyes. This allows researchers to determine the percentage of cells in different phases of the cell cycle

Fig. 4.2 shows two cell cycle profiles, **A** and **B**, determined by Fluorescence-Activated Cell Sorting (FACS) analysis. The amount of fluorescent dye that binds to a cell is directly proportional to its DNA content:

- Graph **A** shows the profile of a population normal, actively dividing cells.
- Graph **B** shows the profile of an identical cell population after being treated with a chemical substance, Compound **X**.

- (i) Label on Graph **A** the different phases of the cell cycle. [1]



- (ii) Based on the information given, explain why there is a difference in cell numbers of the two distinct peaks in Graph **A**. [2]

- A higher peak at G1 with greater number of cells at 300 arbitrary units (au) represents the largest population of cells and a lower peak at G2/M with fewer number of cells at 600 au;
- At G1/ cells at 300 arbitrary units spend the longest time thus at a given moment, a greater proportion of cell population will be found in this extended period;

Or

At G2/ M phases are relatively much shorter in duration than G1 that at any single point in time, fewer cells will be captured;

Please note references should be made to longer time spent in G1.

- (iii) Compound **X** is being investigated as a potential anti-cancer drug. Based on Graph **B**, evaluate the suitability of Compound **X** as the drug. [3]

- Compound X is effective as a chemotherapy drug;
 - All cells are at 300 a.u. indicating that X arrests cell cycle at G1 checkpoint/ prevents entry into S phase/ prevent DNA replication;
 - Thus prevents cell proliferation and prevents uncontrolled cell division;
 - Not suitable because compound X may interfere with cell division of rapidly dividing normal cells thus preventing growth/replacement of worn out cells/ repair of tissues ;
- AVP e.g.: inducing cell cycle arrest can trigger apoptosis; R: prevent mitosis

- (c) A different drug, Taxol, is known to stabilise microtubules, preventing them from shortening.

Predict how a FACS analysis graph of cells treated with Taxol would appear. Justify your prediction by explaining its effect on the mitotic process. [2]

1. (Prediction): Graph for cells treated with Taxol would show a significant increase in G2/M phase/ higher peak at 600 arbitrary unit and the G1 peak, and S phase population might also be reduced over time;
2. Taxol prevents shortening of spindle microtubules, arresting cells in metaphase and thus prevent separation of sister chromatids during anaphase*.

[Total: 10]

- 5 In dogs like Dalmatians, the genes for piebald spotting (a type of white spotting pattern) and deafness are found on chromosome 20. These two gene loci are shown in Fig. 5.1 below.

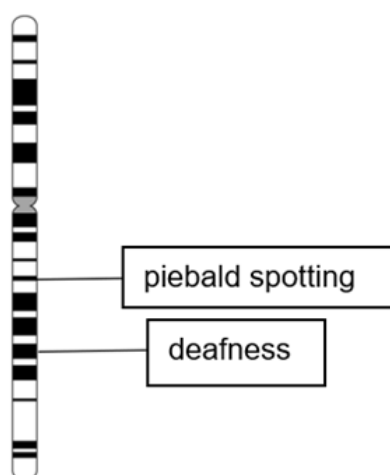


Fig. 5.1

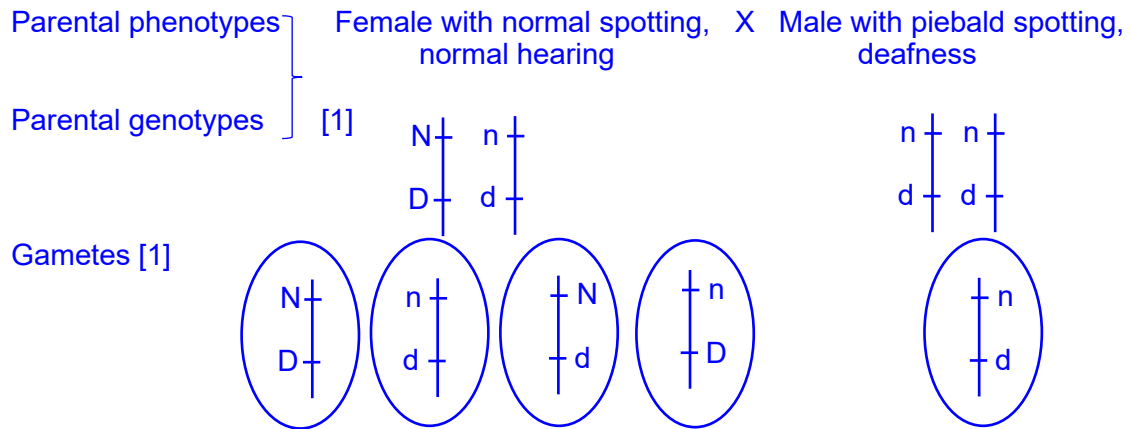
A pair of purebred Dalmatians mated. The female had normal spotting and hearing while the male had piebald spotting and was deaf. They had offspring who are all with normal spotting and hearing. One of these offspring mated with a deaf male with piebald spotting. They had 4 puppies, I – IV, with the phenotypes as shown in Table 5.1.

Table 5.1

puppy	phenotype
I	normal spotting and hearing
II	piebald spotting and deaf
III	normal spotting and deaf
IV	piebald spotting and normal hearing

- (a) Using the symbols provided, draw a genetic diagram in the space below to show the cross that will produce these 4 puppies. [5]

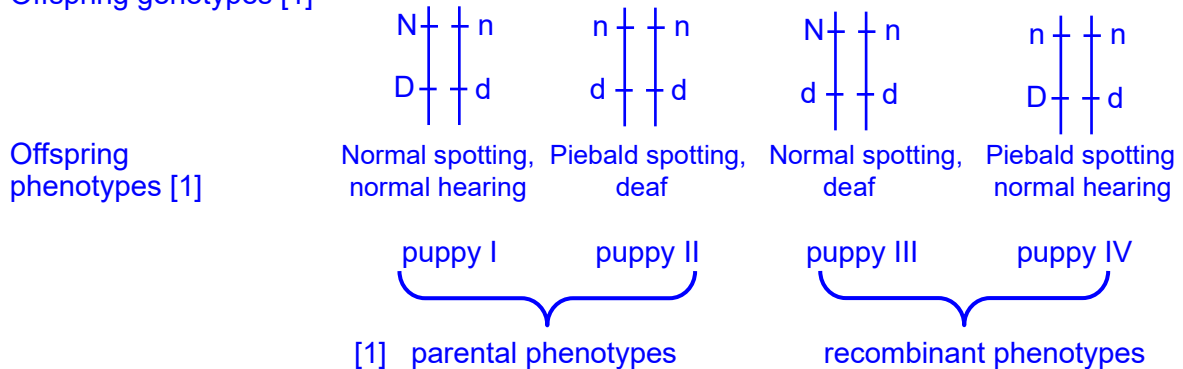
Let **N** represent the dominant allele for normal spotting
n represent the recessive allele for piebald spotting
D represent the dominant allele for normal hearing
d represent the recessive allele for deafness



Offspring genotypes and phenotypes:

Gametes	$\begin{array}{c} N \\ + \\ D \end{array}$	$\begin{array}{c} n \\ + \\ d \end{array}$	$\begin{array}{c} + \\ N \\ + \\ d \end{array}$	$\begin{array}{c} + \\ n \\ + \\ D \end{array}$
$\begin{array}{c} + \\ n \\ + \\ d \end{array}$	$\begin{array}{c} N \\ + \\ + \\ D \end{array}$ normal spotting, normal hearing	$\begin{array}{c} n \\ + \\ + \\ d \end{array}$ piebald spotting, deaf	$\begin{array}{c} N \\ + \\ + \\ d \end{array}$ normal spotting, deaf	$\begin{array}{c} n \\ + \\ + \\ D \end{array}$ piebald spotting, normal hearing

Offspring genotypes [1]



Minus 1 mark if did not use stick diagram

Minus 1 mark if did not use allele letters given

Minus 1 mark if genotypes are circled

Minus 1 mark if answer ended with Punnett square (ie. no offspring genotype / phenotypes)

Penalise once for illegible phenotypes (parental phenotypes and offspring phenotypes)
No penalty if "puppy I" etc not mentioned.
Parental, recombinant phenotypes must be clearly identified somewhere in diagram.

- (b) Suggest how the genotype of puppy III could have arisen. [2]
1. In the mother's cell, crossing over* during prophase I* of meiosis between non-sister chromatids of homologous chromosomes/ chromosome 20;
 2. can result in new combination of alleles (R: genetic material) resulting in production of a gamete with **N** and **d** alleles;
 3. The maternal gamete containing Nd alleles when fused with paternal gamete containing nd alleles results in puppy III with genotype **Ndd** (normal spotting and deafness);
- (c) Considering only the deafness gene, calculate the probability of a 5th puppy being male and being of normal hearing. Explain your reasoning. [2]
- Genotype of father who is deaf: **nn**
Genotype of mother with normal hearing: **Nn**
Cross will give ratio of 1 **Nn**:1 **nn**
1. (reasoning) For puppy whose father is nn and mother is Nn, the probability of having normal hearing, Nn, is 0.5. Probability of puppy inheriting the Y chromosome and being male is 0.5;
 2. (working) $0.5 \times 0.5 = 0.25$;
- Probability of their 5th puppy being male and of normal hearing is 0.25.

[Total: 9]

- 6 The gene *BRCA1* is expressed in breast tissue and in several other tissues of the body. *BRCA1* codes for a tumour suppressor protein. Some mutations in *BRCA1* are associated with an increased risk of developing breast cancer.

- (a) State one reason why a doctor would recommend that a person has genetic testing for a mutation in the gene *BRCA1* and explain why this mutation would put the person at a greater risk of breast cancer. [3]

1. family history of breast (A: ovarian cancer);

Plus any two from:

2. (mutation causes) change of primary structure of tumour suppressor protein to non-functional;
(A: changes conformation/ 3D shape/ tertiary structure of tumour suppressor protein)
3. DNA is not repaired/ no apoptosis / cells with damaged DNA do not die hence mutations accumulate;
4. (some mutations) cause uncontrolled cell division* of breast cells with mutation;
5. AVP;

- (b) Short tandem repeats (STRs) are short sequences of DNA, usually 2 to 7 base pairs. They are repeated a number of times, one after another. Mutations in STRs near the *BRCA1* gene could increase risks of developing cancers.

DNA sequence analysis on STRs requires amplification of the STRs using polymerase chain reaction (PCR).

- (i) The nucleotide sequences on either side of a specific STR are known. Explain the importance of knowing these sequences in PCR. [2]
1. These sequences are used to design primers* which are complementary* to these nucleotide sequences (A: complementary base pairs);
 2. So that the primers provide free 3'OH end to initiate DNA synthesis;
 3. Sequences are used to determine the specific STR regions to be amplified;

- (ii) After PCR, the different STRs are separated by gel electrophoresis. Give one feature of STRs which enable them to be separated by gel electrophoresis. [1]
1. They have different number of nucleotides/ repeats/ nucleotide bases (A: length/ mass);
 2. Negative* charge;
- (iii) A single STR molecule consisting of a 12 base pair sequence of AGAT was amplified 50 times using PCR.

Calculate the total number of base pairs produced after 50 cycles. Show your working. [2]

1. Correct working shown;
Total number of base pairs
= Number of base pairs x number of molecules produced at the end of 50 cycles
= 12×2^{50}
2. 1.35×10^{16} (A: 1.36×10^{16} / 1.4×10^{16});

- (c) One advantage of genetic screening is to determine which drugs should be selected to treat cancer.

A study was carried out on women with breast cancer who had been treated with a DNA-damaging drug that kills tumour cells. The women had all been tested for *BRCA1* mutations. Group **A** had a *BRCA1* mutation and Group **B** did not have a *BRCA1* mutation. The researchers used data to assess the probability of survival for the women in each group of the study after treatment with the drug.

Fig. 6.1 shows the probability of survival for a time period of up to 180 months.

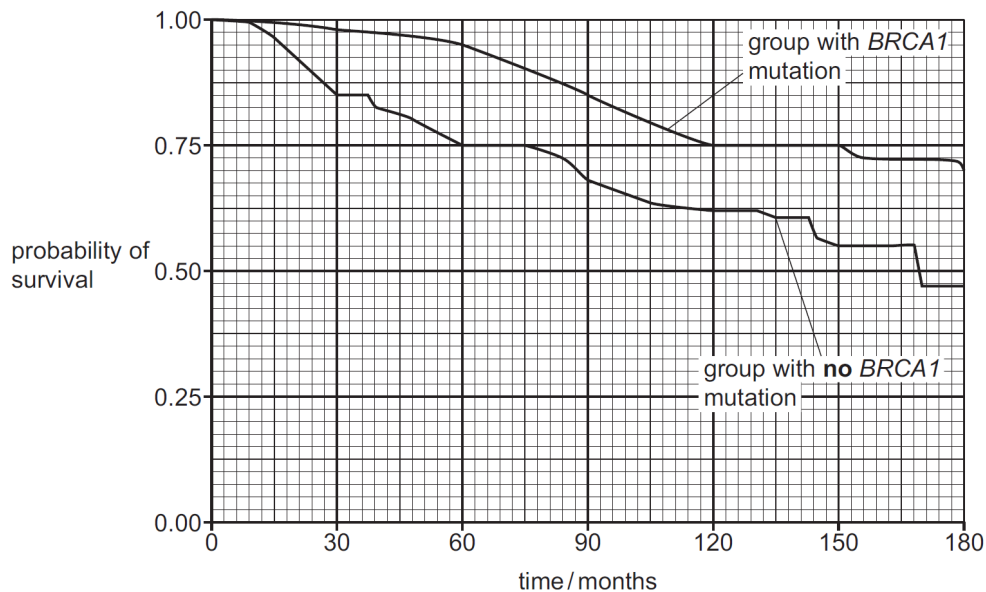


Fig. 6.1

The researchers suggested the hypothesis that the DNA-damaging drug is more effective in women with a *BRCA1* mutation (Group **A**) than those with no *BRCA1* mutation (Group **B**).

Discuss whether the results in Fig. 6.1 support the hypothesis. [3]

[supports]

- Women with *BRCA1*/ Group A have a higher probability of survival;
A: Women with *BRCA1*/ Group A's probability of survival decreases less/ there is a smaller rate of decrease of probability of survival over time;
A: bigger difference between the two groups over time for the probability of survival;
- Data quoted to show differences between the survival of the two groups at a particular time;

Time/ months	probability of survival	
	with mutation	without mutation
30	0.975	0.85
60	0.95	0.75
90	0.85	0.675
120	0.75	0.625 (A:0.63)
150	0.75	0.55
180	0.70	0.475 (A:0.47)

- study carried out for a relatively long time/ 15 years hence data is valid;

[does not support]

- no statistical test carried out/ ref. to sample size hence data is incomplete;
Note: no full marks if not explicitly written "support" or "does not support"

[Total: 11]

7 Fig. 7.1 below shows part of a signal transduction pathway.

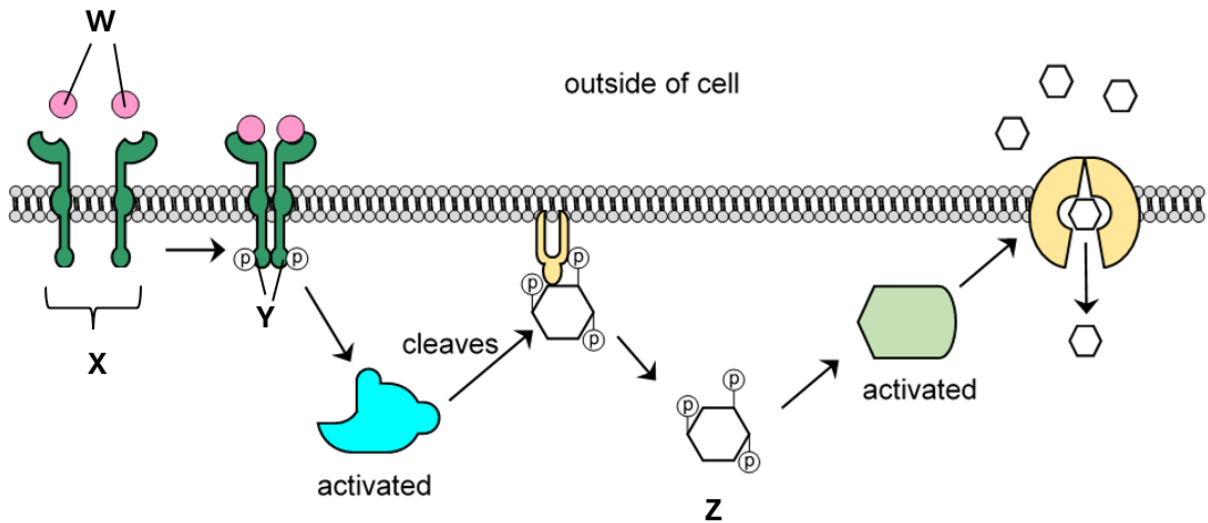


Fig. 7.1

- (a) Describe the events that happens when **W** binds to **X** until the point where **Y** causes an activation of an intracellular pathway. [3]
1. Binding of ligand W, which is complementary in shape to ligand binding site of receptor tyrosine kinase (X);
 2. The two receptor subunits to dimerise* causing conformational change in intracellular domain of receptor;
 3. Activates intrinsic tyrosine kinase activity of intracellular domain which cross-phosphorylates* the tyrosine residues at Y;
 4. Phosphorylated Y will provide a docking site for relay proteins to transduce signals;
- (b) Describe the role of non-protein molecule **Z**. [2]
1. Z is a second messenger*; (A: secondary messenger)
 2. Role is to diffuse throughout cytoplasm;
 3. To activate downstream proteins/ enzymes/ relay molecules;

Among the many homeostatic and metabolic reactions in the human body, signal transduction pathways such as that shown in Fig. 7.1 play a vital role in maintaining internal balance.

- (c) A person has non-functional insulin receptors. Explain the effect on his blood glucose concentration after he has taken a meal. [4]
1. There is no signal transduction;
 2. If there was reduced insertion of transporter, no increase in number of glucose transporters (GLUT4) on plasma membrane, causing no increase in glucose uptake into cell;
 3. Glycogen synthase is not activated, and there is no synthesis of glycogen* from glucose;
 4. Rate of glycolysis will not increase, and there will be a lower rate of aerobic respiration;
 5. Hence, blood glucose concentration remains high;

[Total: 9]

- 8 Corals grow in shallow seawater and consist of colonies of small animals called polyps. These polyps have photosynthetic protists called algae inside their cells, which is advantageous both to the coral polyps and to the algae.

The algae that live within the cells of the polyps can also live independently as free-living algae.

- (a) The rate of photosynthesis of algae that live within the cells of coral polyps is higher than that of free-living algae.
- (i) Explain how living inside the cells of coral polyps increases the rate of photosynthesis in these algae compared to free-living algae. [2]
1. Increase in carbon dioxide concentration from coral respiration so that it can be used for carbon fixation in Calvin cycle and hence photosynthesis ;
 2. Nitrogenous wastes of coral/ polyps (A: nitrates/ ions) provide nutrients for algae as there is generally low concentrations to synthesise enzymes/ proteins involved in photosynthesis ;
 3. Wastes of coral/ polyps provide phosphates to synthesise ATP for photosynthesis;
 4. Coral/ polyps are in relatively fixed positions/ shallow waters hence can get constant exposure to light for photosynthesis ;
- (ii) The relative abundance of five different chloroplast pigments in the algae of corals was determined. The results are shown in Table 8.1.

Table 8.1

chloroplast pigment	percentage of total
chlorophyll a	39
peridinin	39
chlorophyll c2	13
dinoxanthin	7
β -carotene	2

Table 9.2 shows the light wavelengths at which each algal chloroplast pigment shows its two largest peaks of light absorption.

Table 8.2

chloroplast pigment	peak 1 wavelength/ nm	peak 2 wavelength/ nm
chlorophyll a	430	662
peridinin	456	485
chlorophyll c2	450	396
dinoxanthin	442	471
β -carotene	454	480

Corals kept in tanks are often illuminated by lamps radiating mostly violet and blue light with wavelengths in the range of 400–490 nm.

With reference to Table 8.1 and Table 8.2, suggest why lamps radiating mostly violet and blue light are expected to increase coral growth. [3]

1. Table 9.2 shows that algae pigments absorb violet-blue/ 400–490 nm light best at 8 out of 10 peaks/ all pigments have at least one peak / peak 1 within 400 – 490nm ;
2. Table 9.1 shows that chlorophyll a and peridinin are most abundant pigments, both at 39% each / 78% in total and both these pigments absorb mostly at 400-490 nm;
3. Hence more light is absorbed leading to higher rate of photosynthesis of algae;
4. This increases sugars/ food passes from algae to coral and hence increase coral growth;

- (b) Acidification of the world's oceans is an increasing threat to the coral reefs. Corals perform calcification to create their calcium carbonate exteriors.

An experiment was conducted on Heron Island, Southern Great Barrier Reef, Australia. For the experiment, the pH was altered by dissolving carbon dioxide in the water. Two different coral species were used, with each test group at two different temperature ranges and three different pH values.

Fig. 8.1 shows the data gathered. The white line in each photograph represents 5 cm.

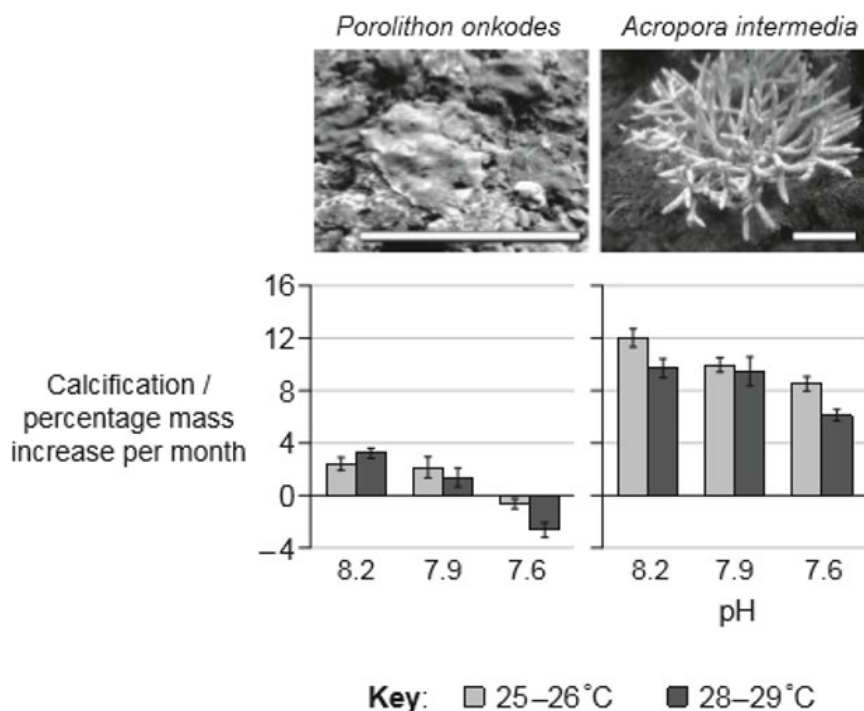


Fig. 8.1

- (i) In environmental studies, a critical value is the level at which a population declines or shows signs of poor health.

Suggest a critical pH for *P. onkodes*. [1]

7.6/ 7.7/ 7.8;

A: any pH that is 7.6 or higher, but lower than 7.9

- (ii) With reference to Fig. 8.1, comment on the hypothesis:
"Ocean acidification in warming seas will have the same effect on these two species of coral". [2]

1. Not supported

at lowest pH of 7.6 at highest temperature of 28-29°C, there is a greater impact on *P. onkodes* as there was a loss of calcification of 3% mass per month (A: -3% calcification, R: -2%) whereas there is still 6% increase in mass per month of calcification in A. intermedia;

A: at pH 7.6 *A. intermedia* still experienced growth but *P. onkodes* did not

OR

2. Supported

At highest temperature of 28-29°C, decrease in pH from 8.2 to 7.6 resulted in decrease in calcification rate for both *P. onkodes* (3% to -3%) and *A. intermedia* (10% to 6%);

3. Quote full data any point above (must include temperature 28-29°C, pH 7.6 and calcification with units)

- 9 The European hedgehog, *Erinaceus europaeus*, is a small omnivorous mammal. Its body is covered in spines, which are usually brown.

A rare variant, which lacks the brown pigment, has blonde (pale yellow) spines. This characteristic is coded for by a recessive allele. Fig. 9.1 shows a European hedgehog with blonde spines.



Fig. 9.1

- (a) Alderney is a small island between the UK and France. Hedgehogs were **not** found on Alderney until the 1960s, when three pairs of hedgehogs were introduced to the island. The hedgehogs started to breed and some of the offspring had blonde spines.

By 2017, the population of hedgehogs had increased to approximately 600. These included individuals with brown spines and individuals with blonde spines.

Suggest reasons why the population of hedgehogs increased to such a large number. [2]

1. There was little competition for resources as there was plenty of food / shelter available / niches not occupied when they were introduced;
2. Well adapted to environment e.g. spines for defence when introduced to the island/ small size can easily hide;
3. Short generation time/ high birth rate - evolve fast enough to be adapted to new environment;
4. Low selection pressures (A: no selection pressure) against them e.g. diseases/ predators;

- (b) A survey taken in 2017 showed that 60% of the hedgehogs on Alderney had blonde spines. Suggest and explain reasons why the proportion of hedgehogs with blonde spines was so high. [3]

Reason 1

1. Since only 3 pairs of hedgehogs were introduced into the island, genetic drift*/ Founder effect* occurred;
2. and alleles coding for blonde spines were over-represented in this founding population;
3. when breeding occurred within the small founding population, the frequency of alleles coding for blond spines increased;

Reason 2

4. Hedgehogs with blonde spines may have had a selective advantage / were selected for as they were camouflaged against the light coloured sand in the island and hence were not easily detected by predators;
5. Hence hedgehogs with blond spines (favourable trait) survived and reproduced and passed the favourable alleles coding for blonde spines to the offspring;
6. Thus, over time, the frequency of blonde spines alleles increased in the population;
7. Directional selection had occurred as blonde spines were favoured;

- (c) Explain the advantages of using molecular methods, such as multiple sequence alignment of nucleotides, over using anatomical homologies (such as spine coloration) when establishing the evolutionary relationships between these hedgehog populations.

1. Molecular methods are objective, allowing a less ambiguous determination of the evolutionary relationship between the species;
2. With quantitative data from molecular methods, statistical analysis may be performed, allowing the accurate estimation of evolutionary history;
3. Nucleotide sequencing can detect silent mutations or changes in non-coding regions that alter the genotype without changing the phenotype (spine color), allowing scientists to measure evolutionary change even if the organisms look identical;

[Total: 8]

- 10** Chickenpox is a highly contagious disease caused by the varicella zoster virus (VZV). It is usually diagnosed based on characteristic symptoms such as skin rash with small, itchy blisters.

People usually only get chickenpox once. Although reinfections by the virus can occur, these reinfections may not cause symptoms or the symptoms may be very mild.

- (a) Explain why the symptoms of chickenpox reinfection are very mild. [3]

1. During the first infection, antigen presenting cells (APCs) activates naïve T cell to form helper T cell and memory T cells, helper T cells will activate specific naïve **B cells*** to form memory B cells;
2. When exposed to virus a second time, **memory B and T cells***, will recognise it and mount a faster and stronger and longer lasting secondary immune response;
3. As more **plasma cells*** are produced, concentrations of **antibodies*** increased resulting in milder symptoms/ preventing a second infection;

- (b) The varicella vaccine was introduced in the United States in 1995.

Fig. 10.1 shows the results of a study conducted on children between the ages of 2 and 18 years from a population of approximately 350,000 persons in Antelope Valley, California. Vaccination coverage refers to the percentage of children between the ages of 2 and 18 who were vaccinated against the varicella virus.

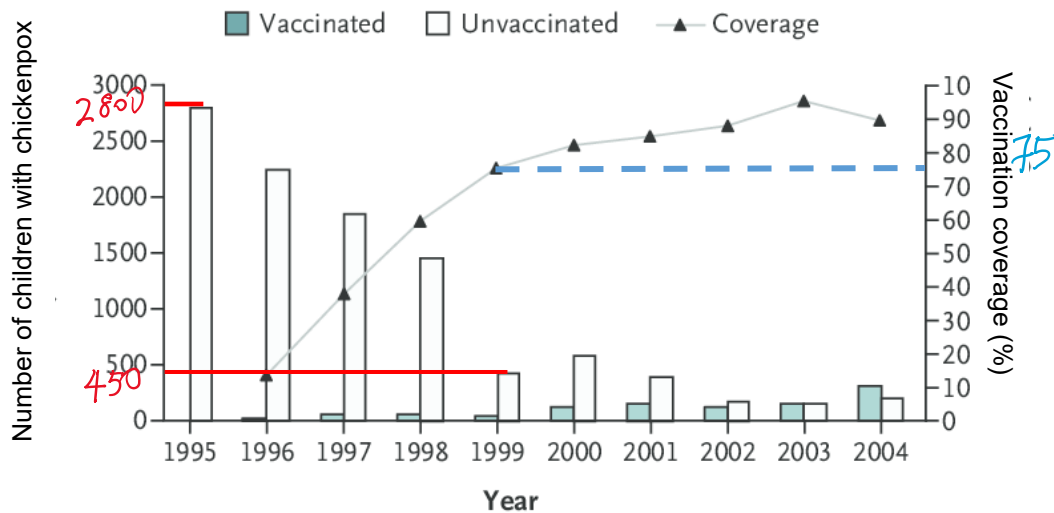


Fig. 10.1

- (i) Describe and explain the trend observed for number of unvaccinated children with chickenpox from 1995 to 1999. [2]
1. Steep decrease in children with chickenpox from 2800 children (A: 2750 to 2850) in 1995 to 450 children (A: 375-450) in 1999;
 2. As more children received vaccinations, from 0% and 75%, fewer children are able to spread the disease/ more children are protected;
I: herd immunity without elaboration
- (ii) Based on this study, a second dose of varicella vaccine was recommended for all children to improve their protection against the varicella virus.

Suggest how Fig. 10.1 provides the evidence in support of administering a booster dose of the varicella vaccine. [2]

1. Children who were vaccinated were becoming more susceptible /less protected;
(compulsory point)

(any 1 from below)

2. Number of vaccinated children with chickenpox increased despite total number of children with chickenpox remaining low from 1999 to 2004;
3. In 2003, equal number of vaccinated and unvaccinated children with chickenpox;
4. In 2004, there were more vaccinated children than unvaccinated with chickenpox;
AVP: if use data on vaccinated alone data, which unexpectedly have more chickenpox cases;

- (c) Complete Table 10.1 below with ticks (✓) to show the type of immunity described in (a) and (b) of this question. [1]

Table 10.1

type of immunity	(a)	(b)
active		
passive		
natural		
artificial		

type of immunity	(a)	(b)
active	✓	✓
passive		
natural	✓	
artificial		✓

All ticks must be correct to get 1m

[Total: 8]

- END OF PAPER -