H

RAFFLES INSTITUTION

2024 Year 6 Preliminary Examination
Higher 2

CANDIDATE NAME

CIVICS GROUP | 2 | 4 | S | 0 | 3 |

INDEX NUMBER

BIOLOGY

9744/02

Paper 2 Structured Questions

18th September 2024

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	/ 9
2	/ 10
3	/ 10
4	/ 10
5	/ 10
6	/ 11
7	/ 10
8	/ 10
9	/ 10
10	/ 5
11	/ 5
Total	/ 100

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

Raffles Institution
Internal Examination

[Turn over

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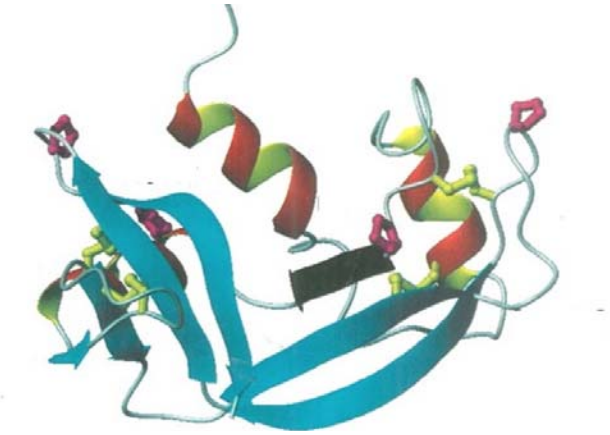
Section A

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

1 Fig. 1.1 shows the structure of pancreatic ribonuclease (RNase A) which hydrolyses RNA.

N-terminus



C-terminus

Fig. 1.1

(a) (i) Describe two features of RNase A that make it a globular protein. [2]

1. It has a spherical shape / description of secondary structures further fold to form a compact shape; (Reject: globular)
2. It has amino acid sequences that are irregular/unique/not repetitive;
3. It serves a metabolic role as an ***enzyme**** in hydrolysing RNA; (No need to mark for metabolic role)
4. It is soluble in water; (Marker to accept: hydrophilic R groups on the outside and hydrophobic R groups on the inside)

(ii) With reference to Fig. 1.1, describe two structural differences between RNase A and haemoglobin. [2]

feature	**RNAse A**	**haemoglobin**
1. Level of protein folding OR Number of polypeptide/ subunit	Tertiary structure One	Quaternary structure Four
2. Presence of ***haem**** group	No	Yes
3. Type of site for binding to specific biomolecule	***Active site**** for RNA (phosphodiester bonds)	Binding sites for oxygen (Marker to decide if he/she will accept no active site)
4. Beta-pleated sheet	Present	Absent

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(b) Christian Anfinsen performed pioneering experiments on the folding of RNase A.

He found that RNase A is most stable between pH 2.0 and pH 4.5 at 100^{0}C.

Fig. 1.2 shows the changes in the protein structure when he added urea and mercaptoethanol to RNase A. The numbers represent the various positions of a particular amino acid in the protein.

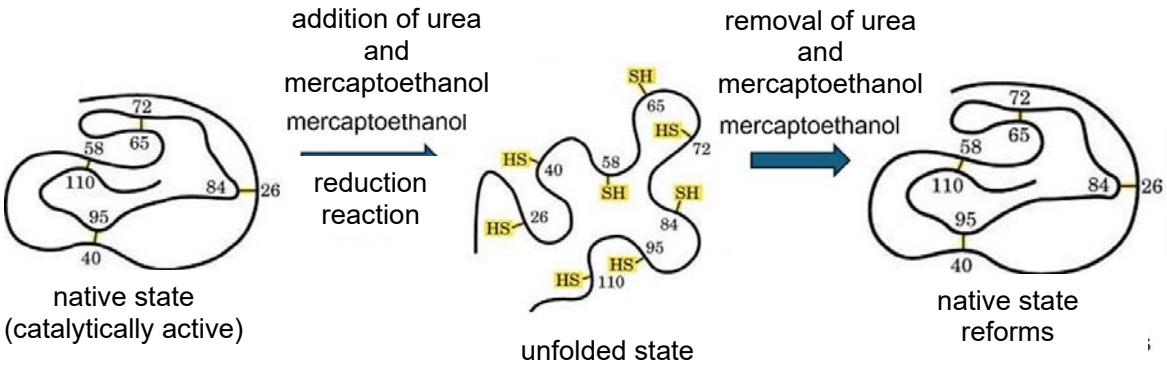

Fig. 1.2

(i) With reference to Fig. 1.2, explain why RNase A can withstand temperatures up to 100^{0}C, but is only stable between pH 2.0 and pH 4.5. [3]

1. RNase has four strong ***disulfide**** linkages which do not break easily at high temperature, allowing enzyme to maintain its 3D conformation;
2. Changes in pH beyond pH 2.0 and 4.5 causes changes in the ionization of the ***R groups**** that maintain its 3D conformation;
3. This will disrupt the weak ***ionic**** and ***hydrogen**** bonds, causing the enzyme to denature/unfold and lose the shape of ***active site****.

(ii) Renaturation does not commonly occur.

Suggest why RNase A is able to reform to its native state in this experiment. [2]

1. RNase is a smaller protein and hence more likely to fold back into original conformation;
2. The removal of urea and mercaptoethanol /denaturing agents allow for the bonds to oxidise again hence reforming;
3. Idea that disulfide bonds form between cysteine residues which are in close proximity at same positions;
4. The primary structure remains the same and the information for the folding into the tertiary structure is in the primary structure/ primary structure will fold to form tertiary structure to give the most stable protein which happens to be the native protein;

[Total:9]

2 Pancreatic cells were cultivated in a medium rich in radioactive amino acids and the locations of assimilated radioactive amino acids over time was ascertained using autoradiography.

When autoradiography was carried out, radioactive amino acids were detected as "autoradiographic grains".

Fig. 2.1 shows the changes in the percentage of autoradiographic grains in different regions of the cell as time elapsed.

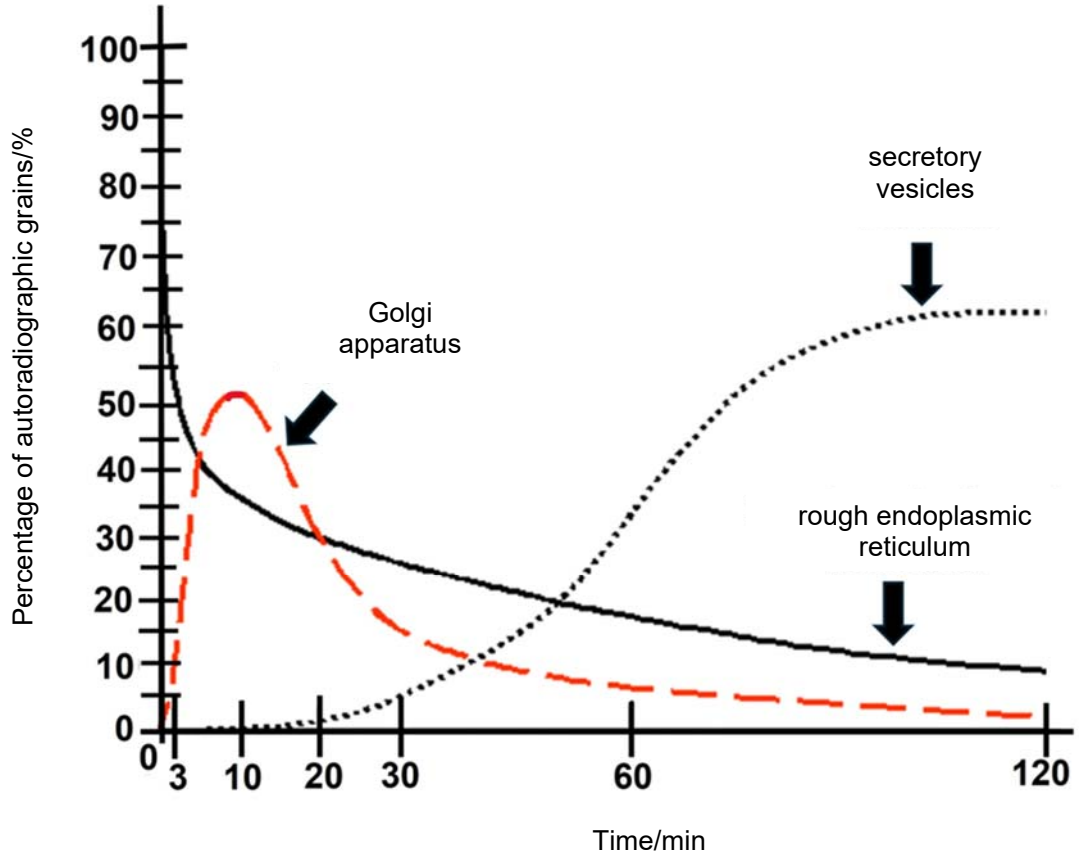

Fig. 2.1

(a) **(i)** With reference to Fig. 2.1, explain the changes in the first 10 min, [3]

1. Percentage of autoradiographic grains in the rough endoplasmic reticulum (RER) decreases from (65% at least) to 36% (A: 35% and 37%), while that in Golgi apparatus increases from 0% to 52% (A: 51% and 53%) from 0 to 10 minutes respectively;
2. The radioactive amino acids are used for protein synthesis at the bound ribosomes;
3. The radioactive protein in the RER lumen is packaged into transport vesicles which bud/pinch off from the RER cisternae;
4. Transport vesicle fuses with the *cis* face of Golgi apparatus, and the radioactive protein is chemically modified/(e.g. glycosylated) within the Golgi apparatus cisternae.

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(ii) With reference to Fig. 2.1, explain the changes in the Golgi apparatus and secretory vesicles from the 10th to 60th min. [2]

1. Percentage of autoradiographic grains in Golgi apparatus decreases from 52% (A: 51% and 53%) to 6% (A: 5%), while that in secretory vesicles increases from 0% to 34% (A: 33% and 35%);
2. The modified radioactive protein in the Golgi apparatus cisternae is packaged into secretory vesicles which bud/pinch off from the *trans* face of the GA;

(b) Not all the proteins synthesised at the rough endoplasmic reticulum are secreted out of the cell.

Suggest two potential fates of these proteins. [2]

The proteins could end up as

1. hydrolytic enzymes packages in lysosomes; [R: lysozyme]
2. receptors/transport proteins that are embedded on the plasma membrane/RER membrane/ Golgi apparatus membrane;
3. enzymes in the lumen of the RER/Golgi apparatus;

(c) Explain why pancreatic cells often have a lot of mitochondria. [3]

1. Many mitochondria are needed to synthesise ***ATP**** by aerobic respiration;
2. needed for the synthesis of a named protein (e.g. insulin/ glucagon/ pancreatic amylase/ pancreatic lipase – R: hormone / enzymes in general);
3. needed for amino acid activation / transcription / AVP;
4. needed for rearrangement of microtubules during ***exocytosis****;

[Total: 10]

3 Cystic fibrosis (CF) is a disorder caused by a mutation in the cystic fibrosis transmembrane conductance regulator (*CFTR*) gene. The mutation affects the production of the CFTR protein responsible for regulating the flow of chloride ions (Cl^-) and bicarbonate out of cells, as shown in Fig. 3.1. Individuals with CF experience thick, sticky mucus buildup in various organs, leading to respiratory, digestive, and other health issues.

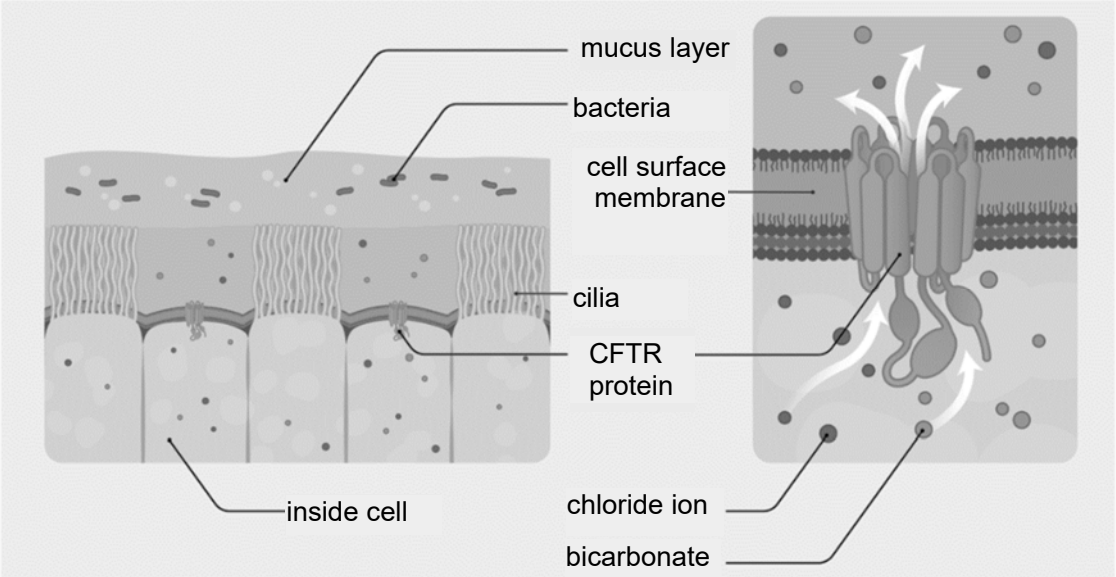

Fig 3.1

(a) CFTR protein regulates the transport of Cl^- across the cell surface membrane. The transport of Cl^- by epithelial cells expressing the normal *CFTR* allele was compared with that by epithelial cells expressing two different types of mutant *CFTR* alleles.

The results are shown in Table 3.1 where normal digestive function of the pancreas associated with a particular allele is indicated with a tick (✓) and the absence of normal functioning is indicated with a cross (✗).

Table 3.1

CFTR allele	percentage Cl^- transported in comparison with normal allele /%	normal digestive function in pancreas
normal	100	✓
mutation 1	6	✗**
mutation 2	50	✓

**Pancreatic insufficiency - pancreas no longer functions at a level needed to digest food.

(i) With reference to Fig. 3.1 and Table 3.1, explain which mutation has a more deleterious effect, causing greater harm to the individual. [3]

1. Mutation 1 has a more deleterious effect;
2. Because mutation 1 cause a greater reduction of the Cl- transported by 94% compare to 50% in mutant 2;
3. It also causes pancreatic insufficiency leading to less digestive enzyme secreted/ less food digested hence less nutrients absorbed
4. reduced Cl- transportation leads thicker and more viscous mucus which makes it difficult for cilia to sweep mucus hence trap more pathogens/ reduction in oxygen absorption in the lungs;

(ii) With reference to the information given in Fig. 3.1 and Table 3.1, suggest and explain the type of gene mutations that may have occurred in the two mutant alleles. [4]

1. Mutation 1 – insertion or deletion of a nucleotide causing ***frameshift**** mutation;
2. This leads to a complete change in 3D conformation results in production of non-functional CFTR protein;
3. Mutation 2 – substitution of a nucleotide causing a change in codon/ amino acid;
4. This leads to a slight change in 3D conformation results in the production of a functional CFTR but at a lower capacity;

(b) Some observations of the geographical distribution of CF showed that CF is more prevalent in populations with a history of exposure to infectious diseases like cholera or tuberculosis.

Other data have suggested that populations with a higher prevalence of CF also experienced lower mortality rates during epidemics of infectious diseases mentioned above.

Experimental studies on mice showed that mice heterozygous for CFTR allele have enhanced resistance to certain pathogens.

Suggest an explanation for the higher prevalence of CF in some areas. [3]

1. ***Heterozygote advantage**** where carriers of a single copy of the mutated CFTR gene have advantage over individuals who have 2 copies of normal gene (homozygous dominant) or 2 copies of the mutated CFTR gene (homozygous recessive);
2. Those who are homozygous recessive are ***selected against**** will not have functional CFTR protein hence will suffer from CF;
3. Those who are homozygous dominant are ***selected against**** and could not to survive when exposed to the cholera/ tuberculosis;
4. Heterozygotes are ***selected for****, as they do not suffer from CF and have enhanced resistance to cholera/ tuberculosis;

[Total: 10]

4 Operons are a well-known feature of prokaryotic genomes.

(a) Fig. 4.1 shows the structure of the tryptophan (*trp*) operon of *Escherichia coli*.

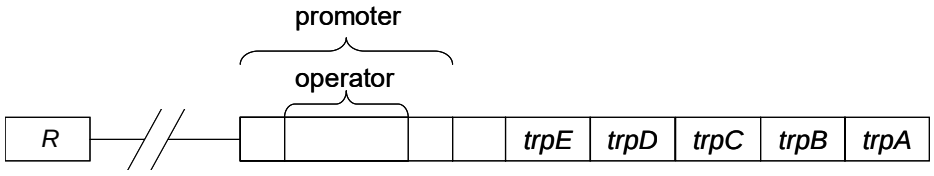

Fig. 4.1

(i) With reference to Fig. 4.1, briefly describe the function of *R*. [2]

1. *R* is a regulatory gene which codes for a ***trp repressor**** protein;
2. which prevents transcription/ RNA polymerase access to promoter by binding to the operator in the presence of tryptophan.
   which is expressed in an inactive form and becomes activated by the binding of a co-repressor, tryptophan.

(ii) There are 2 strains of bacteria. In strain 1 bacteria, an insertion of a single nucleotide occurred in *R*, while in strain 2 bacteria, the same mutation occurred at *trp C*.

Predict and explain the effects of the mutation in the two strains of bacteria when they are not supplied with tryptophan. [5]

Strain 1

1. The mutation in *R* leads to a production of a non-functional trp repressor/ no trp repressor produced;
2. Hence, no repressor to bind to the ***operator****, which allows binding of ***RNA polymerase**** to ***promoter*** *
3. hence constitutive expression of operon (R: upregulate) leading to bacteria continually producing tryptophan;

Strain 2

4. The mutation at *trp C* leads to no/ non-functional trp C protein production;
5. Trp C is an enzyme involved in tryptophan synthesis in a pathway;

Hence, no production of tryptophan and bacteria dies.

(b) Distinguish between repressible and inducible operons. [3]

(Any 3)

1. Repressible operon occurs in an ***anabolic**** pathway whereas inducible operon occurs in a ***catabolic**** pathway;
2. The effector molecule in repressible operon is the end product but in inducible operon, the substrate is the effector molecule;
3. In repressible operon, the repressor synthesized by regulatory gene is in an inactive form but in inducible operon, the repressor synthesized by regulatory gene is in active form;
4. In repressible operon, the repressor only binds to operator after a corepressor is bound to it. But in inducible operon, the repressor binds directly to the operator;
   OR
   In repressible operon, the repressor is synthesized in inactive state, but in inducible operon, the repressor is inactivated by binding to an inducer.
5. The default operon expression of repressible operon is on but that of inducible operon is off.
   OR
   In repressible operon, the operon is off when effector molecule is present but in inducible operon, the operon is on when effector molecule is present;

[Total: 10]

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5 Hormone-sensitive breast cancers are fueled by the natural hormones such as estrogen. A type of breast cancer that is sensitive to estrogen is called estrogen receptor positive (ER-positive) breast cancer.

ER-positive breast cancer cells have receptors for the hormone estrogen. These cancers develop because of increased estrogen concentrations in the blood. Effective treatment of ER-positive breast cancers often involves the use of drugs such as Tamoxifen which has a similar structure to estrogen.

Fig. 5.1 shows the effect of estrogen and Tamoxifen on cell proliferation.

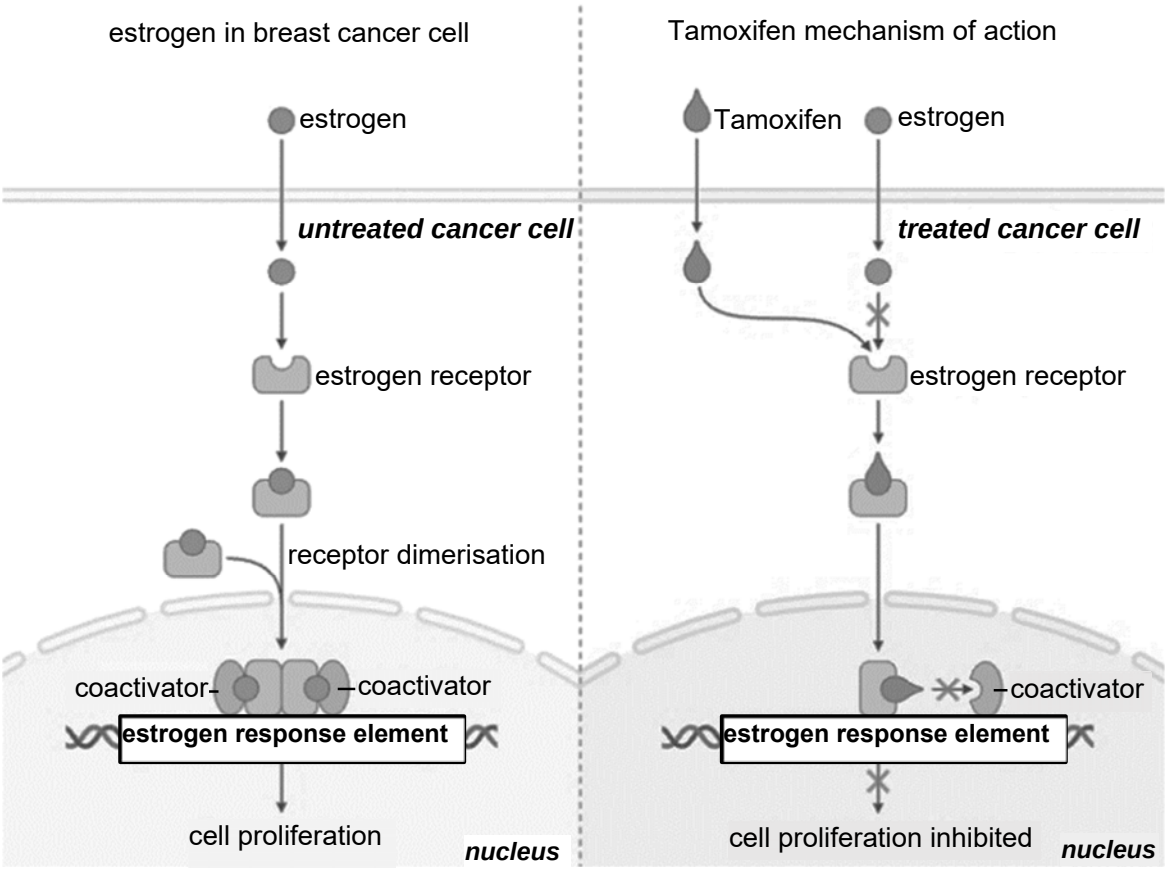

Fig. 5.1

(a) **(i)** With reference to Fig. 5.1, explain why estrogen is able to enter the cell. [2]

1. Estrogen is a small, non-polar and therefore hydrophobic molecule;
2. hence can diffuse through the ***hydrophobic core**** of the plasma membrane of the cell via transient pores;

(ii) Tamoxifen is considered an effective treatment of ER-positive breast cancers.

With reference to Fig. 5.1, explain how Tamoxifen works. [4]

1. Tamoxifen is specific and ***complementary**** in shape and is able to bind to the (ligand binding site of the) estrogen receptor;
2. Thus preventing the binding of estrogen to the (ligand binding site of the) receptor.
   OR
   Tamoxifen blocks the ligand-binding site of the receptor.
   OR
   Tamoxifen competes for the same binding site of receptor; (R: competitive inhibitor)
3. Coactivator is not complementary and hence cannot bind to the Tamoxifen-receptor complex;
4. Hence upregulation of the transcription of genes that cause cell division is prevented so cell proliferation is inhibited/reduced and Tamoxifen is effective for cancer treatment.

(b) Recent research has indicated that several cancers result from epigenetic changes that do not alter the base sequence but influence chromatin structure instead.

Treatment with certain drugs have been found to reverse the epigenetic changes that cause cancers.

Suggest how these drugs can reverse epigenetic changes that have occurred in tumour suppressor genes. [4]

1. Drugs could remove methyl groups from selected ***cytosine**** nucleotides in on DNA (e.g. a CG sequence);
2. Drugs could recruit ***histone acetylase**** and add ***acetyl groups**** to lysine residues on histones thus reducing electrostatic interaction between negatively charged DNA and histones;
3. Drugs could recruit chromatin remodelling complexes that alter the structure of nucleosomes which;
4. results in DNA being less tightly coiled around histones (link to points 2 and 3)
   OR
   resulting in the decondensation of chromatin (link to point 1);
5. which will increase the accessiblity of ***promoter**** to ***general transcription factors**** and ***RNA polymerase**** and promotes formation of ***transcription initiation complex**** ;
6. Promotes ***transcription**** of the tumour suppressor genes and prevent cancer;

[Total: 10]

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6 The body's immune system normally functions to protect itself by attacking pathogens like bacteria and viruses.

In autoimmune diseases, the immune system mistakenly attacks cells in the body instead. Rheumatoid arthritis is an autoimmune disease in which the immune system attacks the synovial membrane which is a layer of connective tissue that lines the cavities of joints, causing inflammation.

Fig. 6.1 shows both a healthy joint and an arthritic joint.

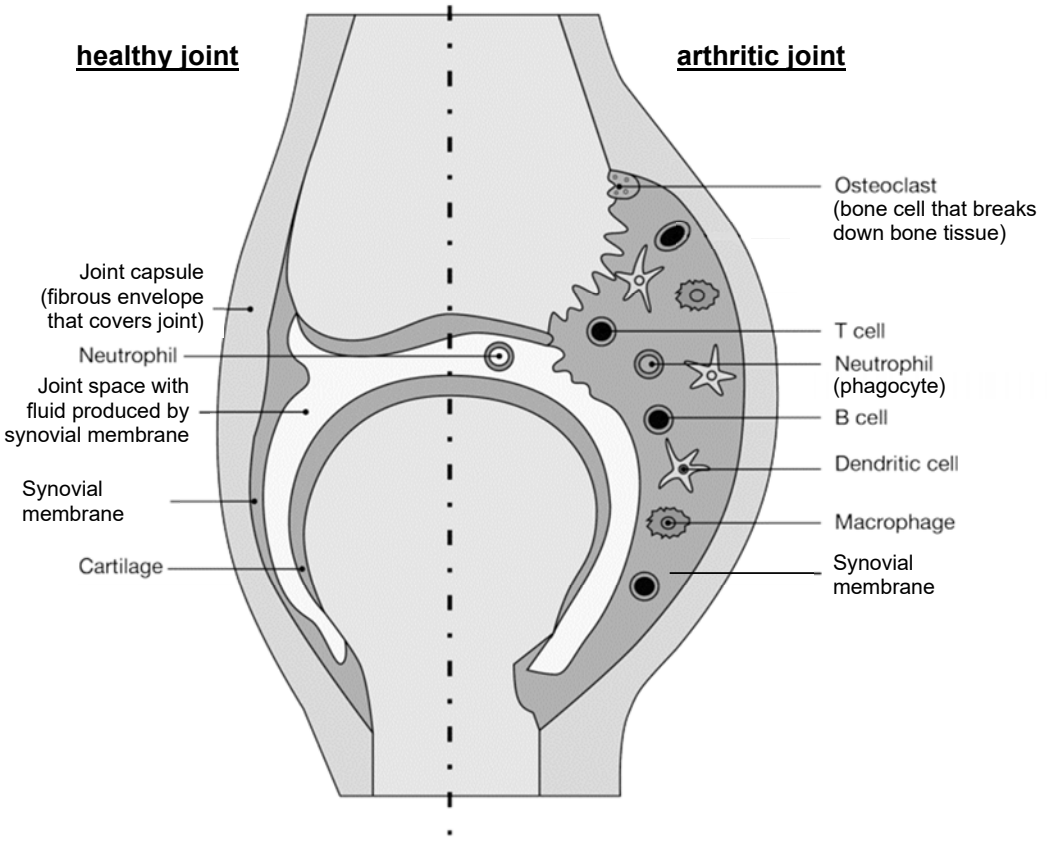


Fig. 6.1

(a) With reference to Fig. 6.1, outline the inflammatory response in the arthritic joint and its resultant effects. [3]

1. Macrophages and dendritic cells secrete signalling proteins/chemokines/cytokines;
2. Signalling proteins/chemokines/cytokines result in increased blood flow/dilation of blood vessels to joint resulting in redness and warmth in the joint;
3. Signalling proteins/chemokines/cytokines result in increased permeability of blood vessels to recruit neutrophils (A: white blood cells) and other plasma proteins/complements to joint, resulting in fluid accumulating/swelling in the joint
4. [From Fig.] Eventually leading thickening of the synovial membrane/bulging of the joint/bone erosion by osteoclast

[any 3]

(b) The dysregulation of the Janus Kinase-Signal Transducer and Activator of Transcription (JAK-STAT) pathway is associated with autoimmune diseases, including rheumatoid arthritis. JAKs are non-covalently associated intracellular non-receptor tyrosine kinases that transfer a phosphate group from ATP to other proteins.

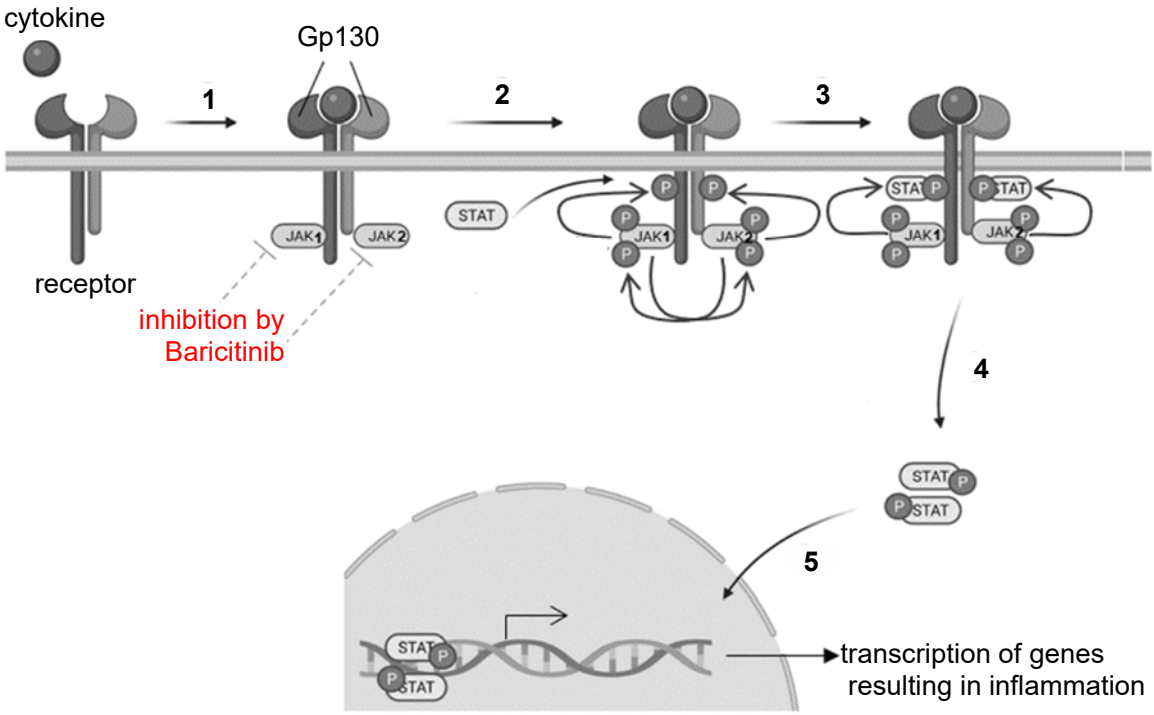


Fig. 6.2

With reference to Fig. 6.2,

(i) describe how the receptor is activated from stage **1** until the end of stage **2**, [3]

1. The ligand, ***cytokine****, is (specific and) complementary in shape to the extracellular ligand binding site of the ***receptor****;
2. ***GP 130**** receptor is a linked dimer/has two subunits;
3. ***JAK 1**** and ***JAK 2**** are recruited/can bind to the intracellular domains of each monomer of the GP130 receptor and is activated due to conformational change (to JAK 1 and 2);
4. Activated JAK 1 and JAK 2 cross-phosphorylates the ***tyrosine**** residues on each other
5. Activated JAK 1 and JAK 2 also phosphorylates the ***tyrosine**** residues on the intracellular domain of the GP130 receptor subunit that they (JAK1 and 2) are attached to;

(ii) explain how the phosphorylation of STAT allows for its dimerisation. [2]

1. Phosphorylation of STAT changes its conformation ;
2. so that it is ***complementary in shape**** and charge to another phosphorylated STAT;

(c) There are four different tyrosine kinases, namely JAK 1, JAK 2, JAK 3 and TYK2.

All four kinases are associated in different combinations with multiple receptors that are involved in numerous cytokine signaling pathways.

Baricitinib is a drug that inhibits these four kinases and is used in the treatment of rheumatoid arthritis, atopic dermatitis, alopecia and even COVID.

Suggest and explain how Baricitinib inhibits JAK 1, JAK 2, JAK 3 and TYK2. [2]

1. Baricitinib is a ***competitive inhibitor**** that is able to bind to ***active site**** of Jak as it is complementary in shape and charge to the active site;

OR

Baricitinib is a ***non-competitive inhibitor**** that is able to bind to a site other than the ***active site**** of Jak changing the conformation of the active site;

2. Blocks the binding of ***ATP/tyrosine**** at the active site, preventing the phosphorylation of tyrosine

(d) Identify the type of enzymes that can inactivate the receptor. [1]

1. Phosphatases;

[Total: 11]

7 Fig. 7.1 shows some structural features of a chloroplast and some processes that occur within it.

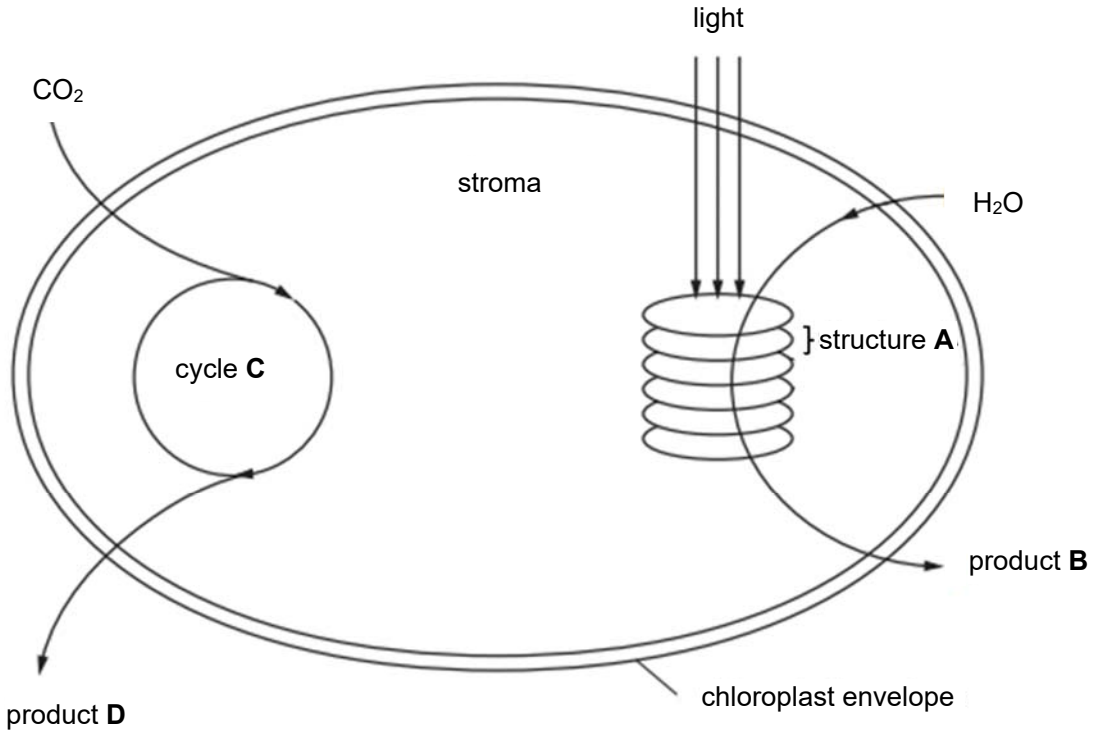


Fig. 7.1

(a) **(i)** With reference to Fig. 7.1, explain how structure **A** is related to its function. [3]

(Components **A** are the thylakoid sacs)

1. many ***thylakoid**** sacs are stacked up to form a granum to increase surface area for:
2. the thylakoid membranes have photosystems containing photosynthetic pigments embedded on the surface for light absorption;
3. the thylakoid membranes have electron carriers/electron transport chain embedded on the surface to pump protons/create a proton gradient between the thylakoid space and the stroma;
4. thylakoid membranes have ***ATP synthase**** embedded on the surface for chemiosmosis and ATP synthesis;
5. thylakoid membranes have ***NADP reductase**** to reduce NADP;
6. Hydrophobic core of phospholipid bilayer is impermeable to protons thus allowing a high concentration of protons inside the thylakoid lumen/space;

(ii) With reference to Fig. 7.1, explain why pathway **C** is described as a cycle. [2]

1. a cycle has no obvious start and end point; (A: intermediates formed are then used in the next step as a substrate/ every compound in the cycle is both a substrate and a product/ start and end with RuBP)
2. intermediates such as ***ribulose bisphosphate****, ***glycerate phosphate****, ***glyceraldehyde-3-phosphate*** (name any 1) are present all the time;
3. there is ***regeneration**** of intermediates such as ***ribulose bisphosphate****;

(iii) Identify the products of photosynthesis labelled **B** and **D** in Fig. 7.1. [2]
B: oxygen;
D: triose phosphate/ glyceraldehyde-3-phosphate /glucose;

(b) Compare the chloroplast envelope and a mitochondrial envelope. [3]
Similarities:

1. both consist of two ***phospholipid bilayers****
2. both a result of endosymbiosis (endocytosis of a prokaryote);
3. both contain membrane proteins embedded within their envelope; (R: If students wrote proteins that are not there, e.g. ATP synthase, ETC, etc)

Differences:

	chloroplast	mitochondria
inner membrane structure	not infolded;	highly infolded to form ***cristae*;***
Inner membrane function	No electron transport chain/ No ATP synthase/ no important biochemical processes take place there;	site of ***electron transport chain**** and ***chemiosmosis**** / ***oxidative phosphorylation*;***
Intermembrane space	No proton reservoir/ does not have any important functions;	contains high concentration of protons / functions as a proton reservoir;

Must have 1 similarity and 1 difference.

[Total: 10]

8 Nail patella syndrome is a rare genetic syndrome characterised by unusual changes in one's nails, kneecaps and bones. It is caused by mutations in the *LMX1B* gene that encodes for a transcription factor that plays a vital role in early embryonic development.

Fig. 8.1 shows the transmission of the nail patella syndrome along with ABO blood type. Individuals suffering from the nail patella syndrome are shaded, and the blood type of each individual is also stated.

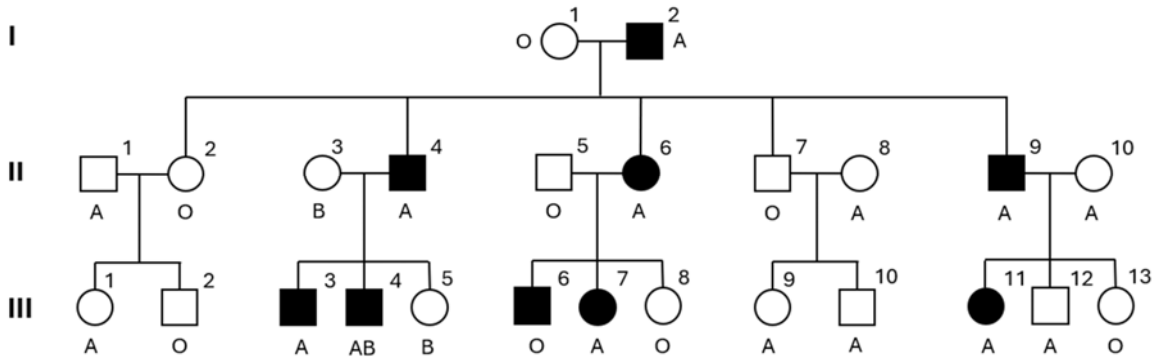


Fig. 8.1

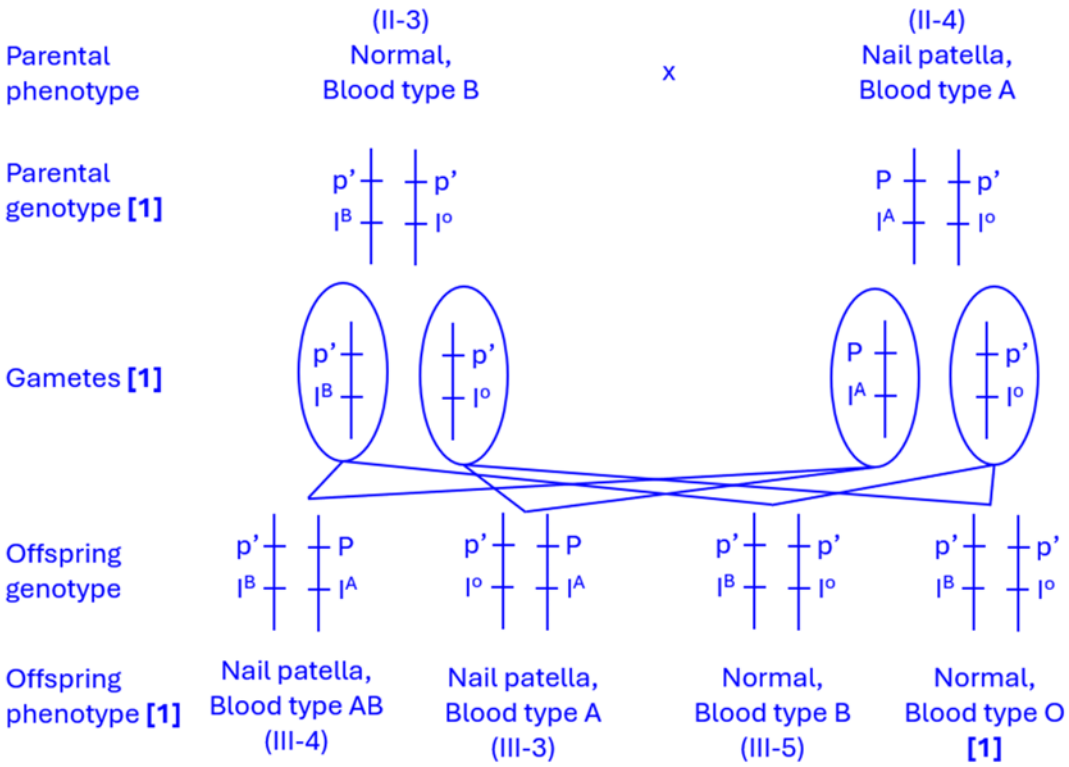
(a) With reference to Fig. 8.1, justify why nail patella syndrome is an autosomal dominant disease. [4]

1. Genetic disorder is rare, so individuals from outside the family eg II-3 and II-5 are unlikely to carry the allele;
2. Offspring III-3, III-4, III-6, III-7 would have inherited a mutant allele from affect parent, and a normal allele from other parent; (identify corresponding parents based on offspring raised as example)
3. Heterozygous III-3, III-4, III-6, III-7 are affected by nail patella syndrome, so the mutant allele is dominant;
4. Affected father (II-4) produced an unaffected daughter (III-5) which is not possible if the mutant allele is sex-linked dominant; A: if other individuals quoted

(b) The gene coding for the ABO blood type is located on the same chromosome as the *LMX1B* gene.

(i) Assuming crossing over does not occur, show the cross between **II-3** and **II-4** to produce offspring **III-3**, **III-4** and **III-5**, using a genetic diagram. [5]

Let:
P represent the dominant allele for nail patella
p' represent the recessive allele for normal phenotype
I^A represent the allele for blood type A / antigen A
I^B represent the allele for blood type B / antigen B
I^o represent the allele for blood type O / absence of antigen



1. Define alleles using key;
2. Parental phenotype and genotype (must show linkage);
3. Gametes circled and show linkage;
4. Link correct genotype to correct phenotype;
5. Correctly identify III-3, III-4 and III5 to the genotype and phenotype;

(ii) Identify an individual in generation **III** that is formed as a result of crossing over. [1]

III-6

[Total: 10]

For Examiner's Use

9 The family of Camelidae comprises two major subfamilies, namely *Camelinae* (Old World Camelids) and *Laminae* (New World Camelids).

The old world camelids include two domesticated species, the dromedary (*C. dromedarius*) and the bactrian camel (*C. bactrianus*). Both species are referred to as large camelids and distributed into different regions of the world. *C. dromedarius* is located mainly in the hot areas of Middle East and Africa whereas *C. bactrianus* inhabits the cold zones of Central Asia and China.

The new world camelids comprise four main species located in South America and are commonly known as small camelids. Two species, the llama (*Lama glama*) and the alpaca (*Vicugna pacos*), have been domesticated whereas the other two species, namely the guanaco (*Lama guanicoe*) and the vicuna (*Vicugna vicugna*), are wild species.

A classification and map distribution of members of the Camelidae family is shown in Fig. 9.1.

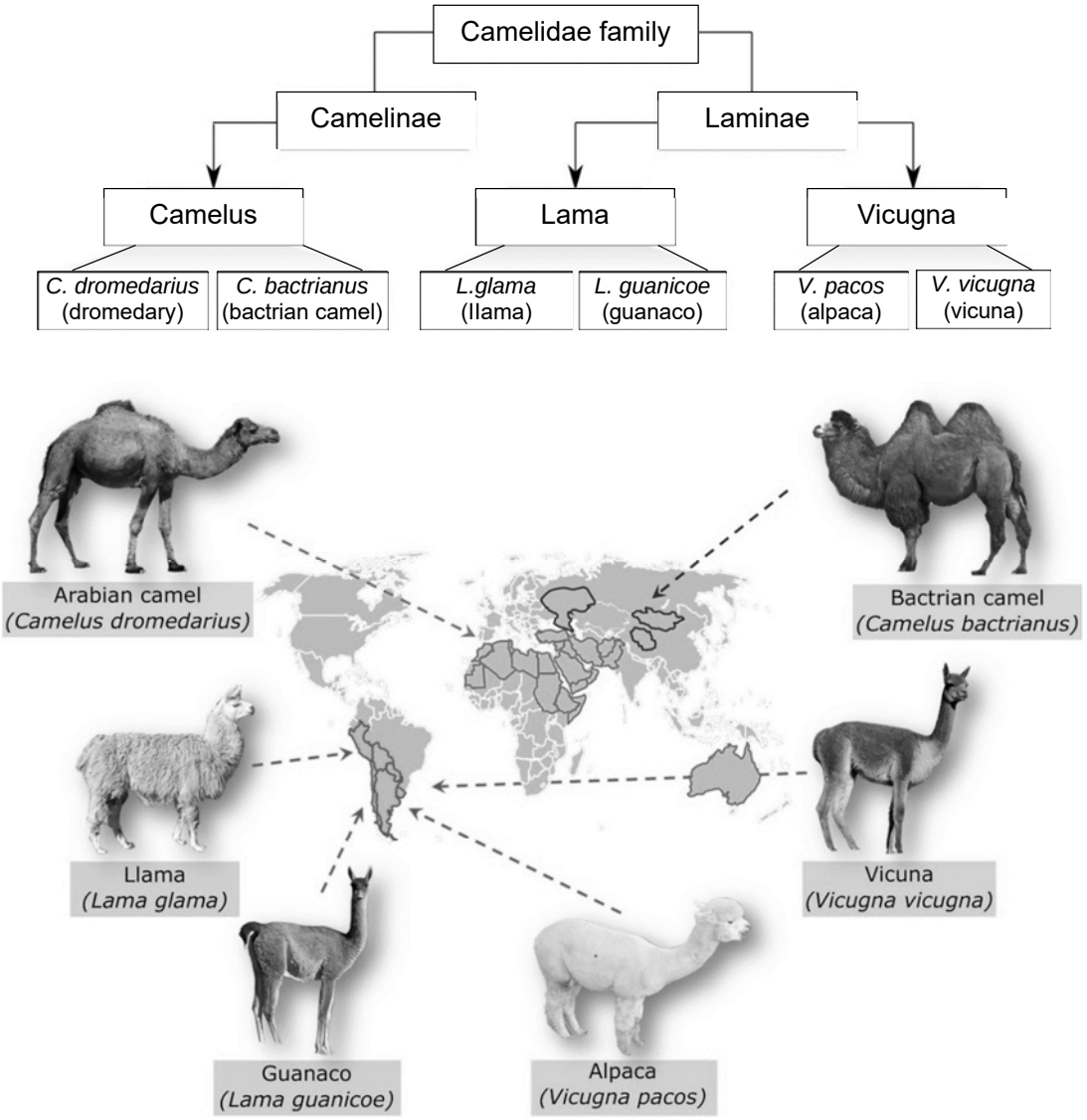


Fig. 9.1

(a) Explain how all the camel species today arose from one common ancestor. [4]

1. The common ancestor existed when the landmasses were connected in the past, as a supercontinent;
2. As a result of continental drift / the landmass started to split to give rise to smaller continents, resulting in ***geographical isolation**** between the subpopulations;
3. The sea/ocean/water formed a physical barrier which prevented interbreeding and resulted in the disruption of ***gene flow****;
4. Within each continent and on different continents, there were different environments and hence ***different selection pressures**** acting on each subpopulation;
5. Individuals with favourable traits which were better adapted and had a selective advantage to the local conditions and were selected for, survived, reproduced and passed on their alleles to the next generation increasing frequency of favourable alleles;
6. As different sub populations evolved independently of each other, their allele frequencies changed as they accumulated different genetic ***mutations*,*** and were subjected to ***genetic drift**** and ***natural selection****.
7. Over a hundreds and thousands of generations, each population on the different continents became ***reproductively isolated**** species which are unable to interbreed to produce fertile, viable offspring;

Fig. 9.2 shows the characteristics of the ancestors of the *Camelidae* family that were constructed based on fossils that were found.

Age	Paleocene 65 million years ago	Eocene 54 million years ago	Oligocene 33 million years ago	Miocene 23 million years ago	Present
Organism					
Skull and teeth					
Limb bones					

Fig. 9.2

(b) With reference to Fig. 9.2, describe how fossils can serve as evidence of evolution. [2]

1. Fossils show preserved(or any reference to ancestral species) anatomical structures such as limb bones/skull/teeth ;
2. Radioactive dating techniques allow us date fossils from (quote one time period from Fig. 9.2) and arrange them in a chronological sequence (or ref to order of existence);
3. Allows us to see how ***homologous structures**** have been modified through time, to demonstrate descent with modification;

(c) Recently, a giant camel fossil has been found in the Arctic. Fig. 9.3 shows the shards of a camel tibia (one of the bones in the lower leg) that was unearthed.

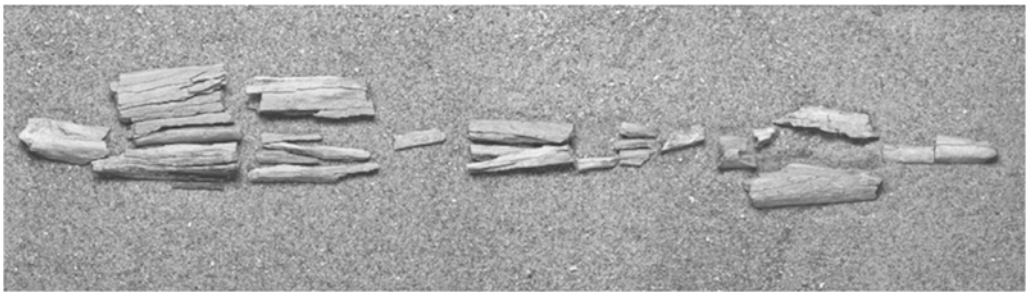


Fig. 9.3

To investigate further, scientists took collagen, a dominant protein found in bone, from the fossils, and compared this with collagen found in other fossils and modern animals.

One of the research scientists said, "These biomolecules tell us that it is an ancestor of modern camels."

(i) Suggest why it was necessary to investigate further despite having fossil evidence of the camel ancestor. [1]

1. Fossil is incomplete / damaged;
2. Fossil does not have sufficient characteristics for comparison to show that it was obtained from an ancestor of camels;

(ii) Explain why collagen was a good choice for the study described above and how the findings could help to conclude that the fossil found is from an ancestor of camels. [3]

1. Collagen is a protein common to animals that have bones, and hence is a good basis for comparison;
2. Collagen is found in large amounts in the bone so there is sufficient amount for analysis;
3. The amino acid sequence of collagen found in the fossils must have a high percentage / degree of similarity with the amino acid sequence of collagen from present day species from the subfamilies *Camelinae* (Old World Camelids) and *Laminae* (New World Camelids);
4. The amino acid sequence of collagen found in the fossils has a lower percentage similarity with the collagen amino acid sequence obtain from other unrelated animals;

[Total: 10]

10 When a person first becomes infected with the Human Immunodeficiency Virus (HIV), both the innate and adaptive immune responses are triggered.

(a) Describe how the innate immune response differs from the adaptive immune response. [2]
(any 2)

	Innate immunity	Adaptive immunity
1. Response Speed	Rapid (within hours)	Slower (days to weeks)
2. Specificity to pathogens	Not specific and can respond to any pathogen;	Specific to specific antigens of pathogens;
3. Antibodies production	No antibodies production;	Antibodies are produced;
4. Memory Formation/ Immunological memory	No memory B (and T) cells/ No immunological memory	Memory B (and T) cells/ Has immunological memory
5. Immune cell types Involved	***Phagocytes**** Or neutrophil, macrophage and dentritic cell	Lymphocytes/ B cells and T cells

HIV can infect both non-dividing and dividing T cells, including helper T cells and memory T cells.

The onset of disease, which can occur many years later, coincides with a severely lowered primary and secondary immune response, owing to greatly reduced numbers of T cells in the body.

(b) Suggest and explain how the destruction of memory T cells will contribute to a lowered secondary immune response. [3]

1. No/fewer memory T cells remain in circulation for second encounter with ***antigen****;
2. no/less ***cytokine**** secreted by T helper cells to stimulate naïve B lymphocyte to undergo clonal expansion and differentiation to ***plasma cell****:
3. so ***no/less antibodies**** specific to the antigens of HIV are produced;

[Total: 5]

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11 Climate change has a negative impact on coral reef ecosystems. Coral bleaching is a major threat to reefs.

Fig. 11.1 shows coral bleaching and recovery.

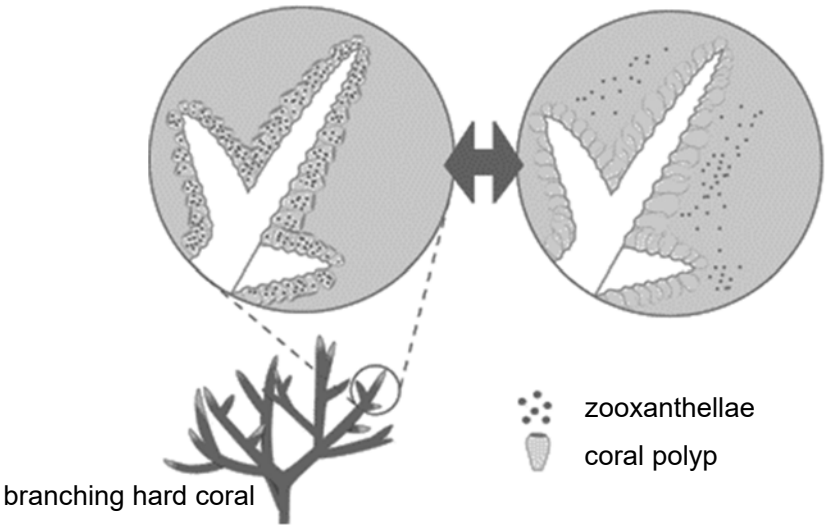

Fig. 11.1

(a) With reference to Fig. 11.1, explain how a rise in ocean temperature leads to bleaching and eventual death of corals. [2]

1. Stressed by excessive heat, corals expel zooxanthellae thus losing their color, leading to bleaching.
2. Death occurs due to prolonged loss of nutrients/ organic compounds made via ***photosynthesis**** carried out by zooxanthellae.

Artificial reefs are widely used to regenerate coral reef ecosystems by providing a surface for the mobile larvae of the corals to settle on and subsequently develop into coral polyps.

Scientists investigated the settlement of coral larvae and growth of coral polyps on four different types of artificial coral skeletons.

Equal numbers of coral larvae were introduced into four separate tanks, each containing one type of artificial coral skeleton.

The percentage of larvae attached to each type of coral skeleton was recorded over a 14-day period, and the growth rate of those that attached was determined.

Fig. 11.2 shows the percentage of larvae attached to each type of coral skeleton material.

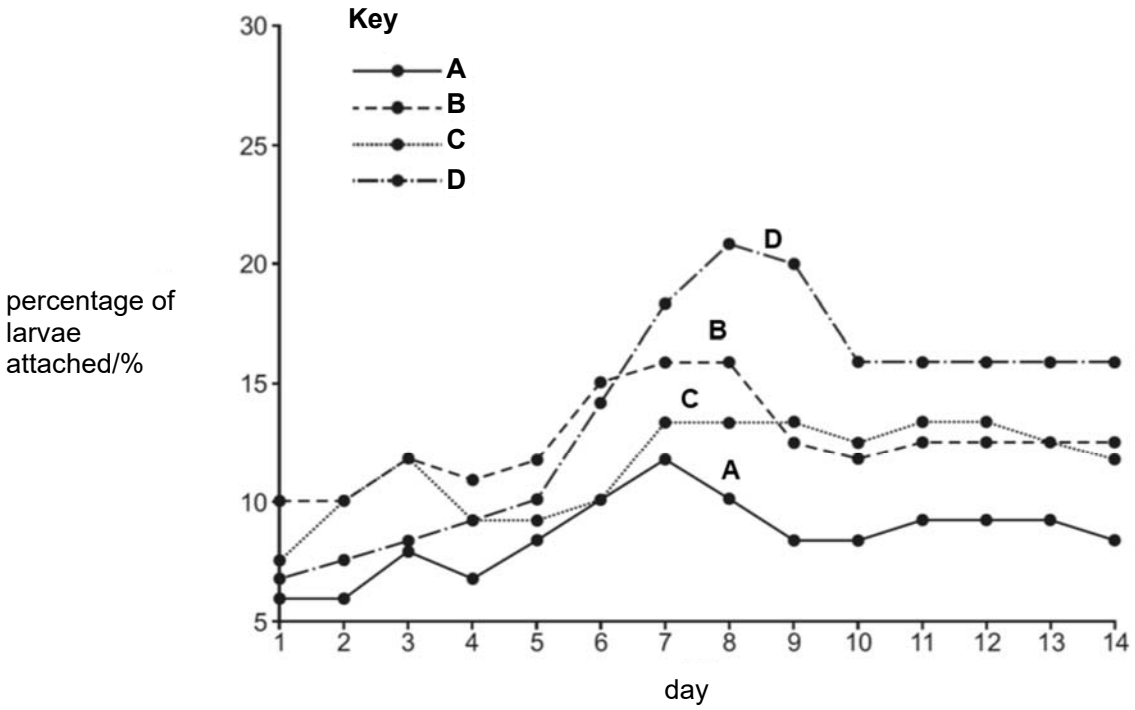

Fig. 11.2

Table 11.1 shows the mean growth rate of attached coral larvae.

Table 11.1

coral skeleton material	mean growth rate of coral polyps / mm^2 per week
A	0.078
B	0.230
C	0.211
D	0.162

(b) Using the results shown in Fig. 11.2 and Table 11.1, state and explain which of the materials **A-D** is best to use for coral regeneration. [3]

1. ***B****;
2. B has the highest growth rate of 0.230 mm^2 per week, and second highest percentage larvae attachment 12.5% at day 14;
3. To regenerate corals, the larvae need to first attach and then grow.
4. Despite D having the highest percentage larvae attachment 16% at day 14, its growth rate of 0.162 mm^2 per week is the much lower;

OR

1. ***D****;
2. D has the highest percentage larvae attachment 16% at day 14;
3. To regenerate corals, the larvae need to first attach and then grow.
4. Even though growth rate is the slowest at 0.162 mm^2 per week, the total biomass of coral may eventually be more.

[Total: 5]

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