

Civics Group	Index Number	Name (use BLOCK LETTERS)
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# H2



## ST. ANDREW'S JUNIOR COLLEGE 2025 JC2 PRELIMINARY EXAMINATIONS

**H2 BIOLOGY**

**9744/2**

**Paper 2**

**Wednesday**

**3<sup>rd</sup> September 2025**

**2 hours**

Materials:

Question Paper

### READ THESE INSTRUCTIONS FIRST

Write your name, civics group and index number on all the work you hand in.

Write in dark blue or black pen on both sides of the paper.

You may use a soft pencil for any diagram, graph or rough working.

Do not use staples, paper clips, highlighters, glue or correction fluid.

Answer **all** questions.

Write your answers in the spaces provided on the question paper.

The number of marks is given in brackets [ ] at the end of each question or part question.

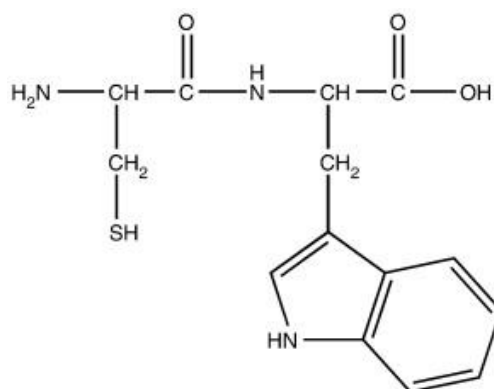
For Examiners' Use	
<b>1</b>	/10
<b>2</b>	/12
<b>3</b>	/10
<b>4</b>	/10
<b>5</b>	/10
<b>6</b>	/6
<b>7</b>	/9
<b>8</b>	/10
<b>9</b>	/8
<b>10</b>	/10
<b>11</b>	/5
<b>Total</b>	<b>/100</b>

This document consists of **x** printed pages and **0** blank page.

**[Turn over**

**QUESTION 1**

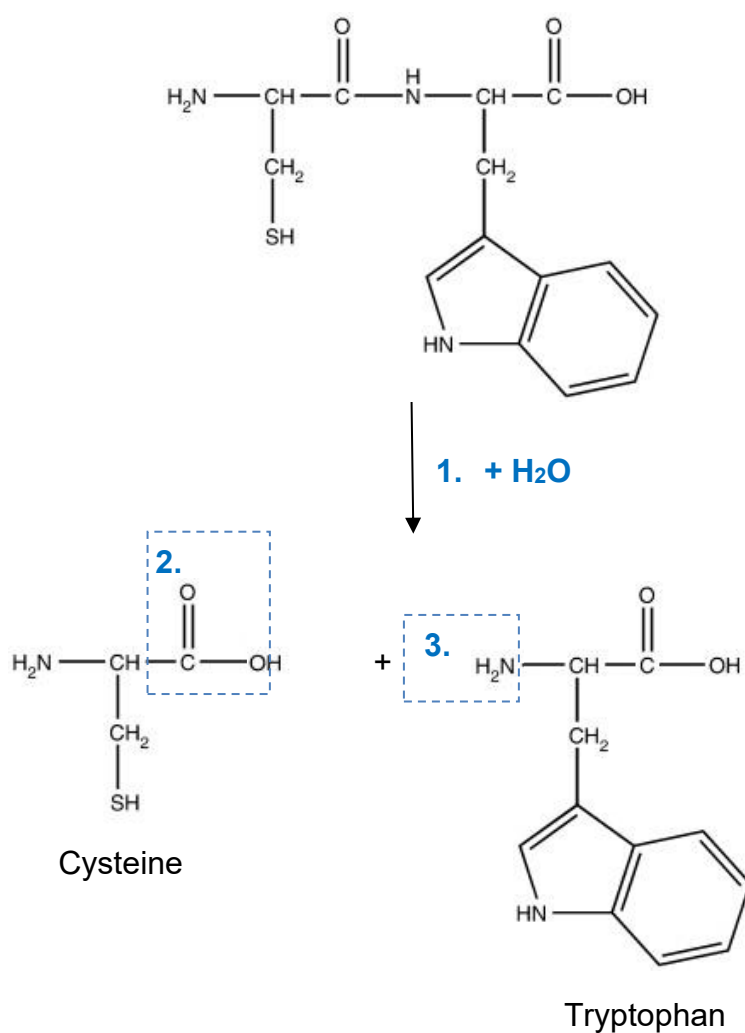
Fig. 1.1 shows the structure of the dipeptide consisting of cysteine and tryptophan.

**Fig.1.1**

Picture credits : <https://linkinghub.elsevier.com/retrieve/pii/S0039602810003869>

**(a)** Complete the diagram to show the hydrolysis of the dipeptide.

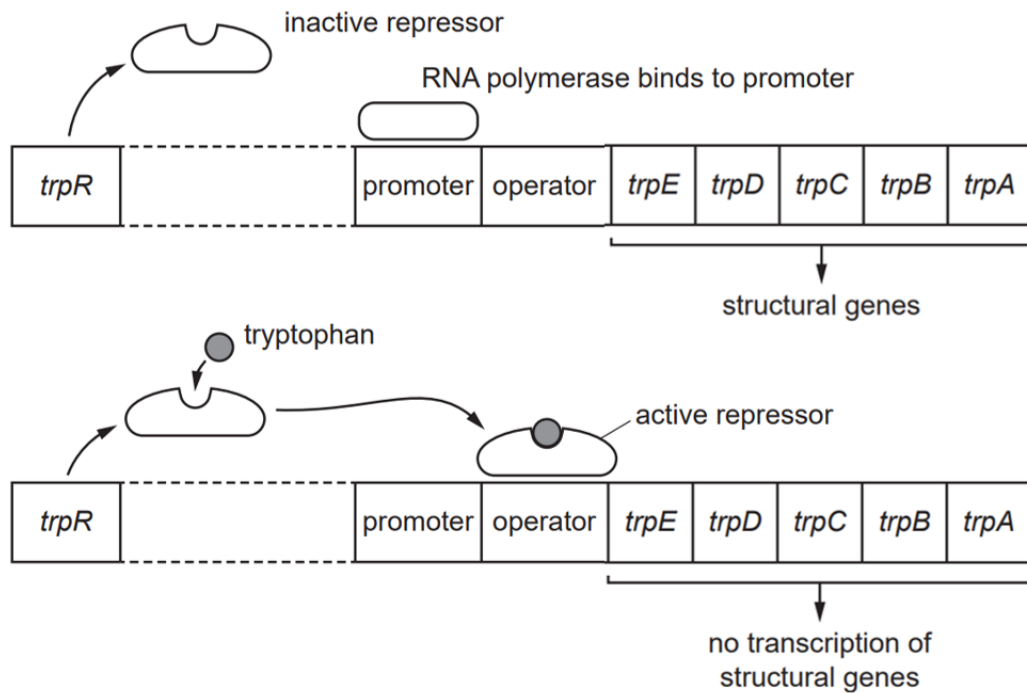
.....[2]



- 1 **Water** used in the hydrolysis reaction ;  
 2 Cysteine amino acid with a - **COOH group** ;  
 Tryptophan amino acid with a - **NH<sub>2</sub> group** ;  
 (No labels are needed)

In bacteria like *Escherichia coli*, tryptophan is incorporated into polypeptides during protein synthesis. The availability of tryptophan within the cell is tightly regulated by the *trp* operon which is known as a repressible operon.

Fig. 1.2 summarises the structure and control of the *trp* operon.



**Fig. 1.2**

Picture modified from 9700/W22/42

(b) Suggest and explain the advantage of having repressible operons in prokaryotes.

.....[2]

1 [Suggest]

Essential enzymes / proteins are continuously produced ;

2 [Explain]

End product inhibition occurs when enzymes / proteins are synthesized until product / tryptophan concentrations are high ; to prevent wastage of resources;

A number of mutations have been found in the *trp* operon. One of these mutations results in a mutant operator ( $O^C$ )

(c) Predict and explain the likely effect of the  $O^C$  mutation on the synthesis of tryptophan in *E. coli*.

.....[3]

[Predict]

1 no repression of tryptophan synthesis

/ tryptophan synthesized continuously (even when in abundance);

[Explain]

2 *trp* repressor (which is synthesized in inactive form) is no longer able to (recognise and) bind to  $O^C$  / mutated operator ;

3 RNA polymerase can bind to the promoter, transcription of the 5 *trp* structural genes, *Trp E*, *Trp D*, *Trp C*, *Trp B*, *Trp A*, occurs ;

4 Proteins / enzymes encoded by structural genes catalyse steps in tryptophan biosynthetic pathway;

(d) Describe **three** differences in the structure and organisation of prokaryotic and eukaryotic genomes.

.....[3]

	Bacterial genome	Eukaryotic genome
<b>Points specified in syllabus</b>		
1. Genome size / Amount of DNA	<u>Smaller</u> genomes, 0.6 to 10Mb / less total DNA per cell <i>[for general info only: Mb – Megabase is the unit length for DNA fragments equal to 1 million nucleotides].</i>	<u>Larger</u> genomes, 10 Mb – 100,000 Mb / more total DNA per cell, about 1000 times more DNA.
2. Gene length	<u>Shorter</u> gene sequences / more compact genetic organisation	<u>Longer</u> gene sequences / presence of more intragenic ( <i>within genes</i> ) spaces (e.g. introns)
3. Chromosome structure	<u>Circular</u> DNA molecule which is closed covalently	<u>Linear</u> DNA molecule with 2 ends
4. Packing of DNA	Prokaryotic DNA is <b>not complexed with histones</b> ;  (DNA is not packaged into nucleosomes. DNA is supercoiled and later folded and condensed via <u>non-histone proteins</u> .)	Eukaryotic DNA is <b>complexed with histones</b> and other proteins to form chromatin;  (DNA is coiled around histone octamer core and subsequently further packed into higher order chromatin structure.)
5. Introns	No introns within genes. (Coding sequence proceeds from start to finish without interruption by introns)	Presence of <u>introns</u> within genes. (Introns account for the main difference in average length between human and prokaryotic genes)
<b>Points <u>not</u> specified in syllabus</b>		
6. Chromosome number	<u>Single</u> chromosome / Haploid	<u>Many</u> chromosomes / Diploid or polyploid
7. Presence and absence of operons	Presence of <u>operons</u> , where two or more genes may be expressed and regulated as a unit	Absence of operons.
8. Repetitive sequences	Few repetitive DNA sequences.	Many repetitive DNA sequences

9. Coding and non-coding DNA	Most of DNA are coding sequences (codes for protein, tRNA, or rRNA).	Most of DNA are non-coding.
10. Origins of replication	One origin of replication present	Many origins of replication present
11. Presence of extrachromosomal DNA	Independent small, double stranded, circular DNA called plasmids	Circular, double-stranded DNA in mitochondria / chloroplasts.
12. Telomeres	Absent	Present

**[Total: 10]**

## QUESTION 2

(a) In chickens, feather color is either white, black or speckled.

The alleles for black feathers and white feathers are denoted by “  $C^B$  ” and “  $C^W$  ” respectively.

A cross between a white chicken and a black chicken gave rise to a speckled chicken.

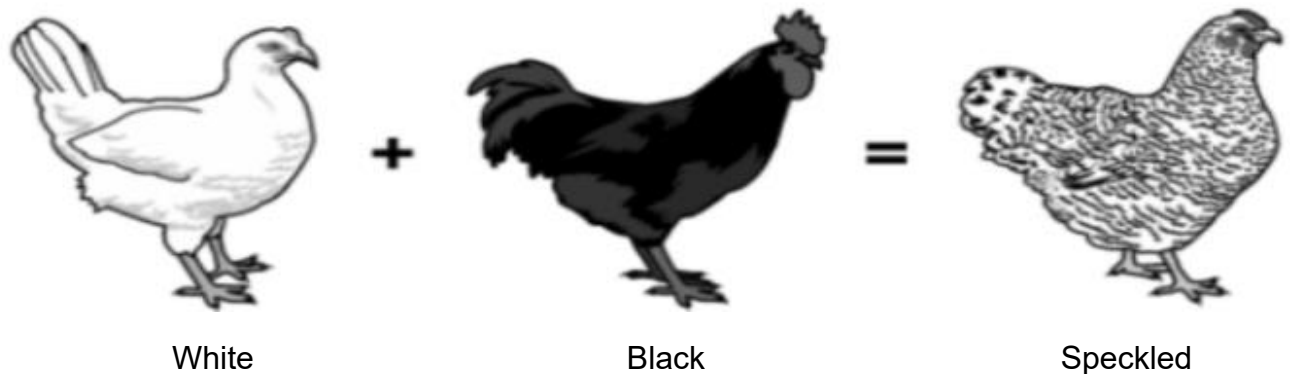


Fig. 2.1

(i) Using the information provided, explain the appearance of the speckled chicken.

.....[2]

1. The black /  $C^B$  and white /  $C^W$  alleles for feather color are codominant.
2. Both alleles of the gene for feather color,  $C^B$  and  $C^W$ , are **equally expressed** in the phenotype of the **heterozygote** (resulting in speckled phenotype).

[Reject: incomplete dominance]

(ii) Using the symbols provided, draw a genetic diagram to show the results of a sibling mating between two speckled chickens.

.....[3]

F1 phenotype: Speckled chicken X Speckled chicken

F1 genotype:  $C^B C^W$  X  $C^B C^W$

F1 gametes:  $\begin{pmatrix} C^B \\ C^W \end{pmatrix}$  X  $\begin{pmatrix} C^B \\ C^W \end{pmatrix}$  ;1m for

F2 genotype:  $C^B C^B$  ,  $C^B C^W$  ,  $C^W C^W$  ;1m for genotypes

F2 phenotype: black chicken, speckled chicken, white chicken

F2 phenotypic ratio: 1 : 2 : 1 ;1m for phenotype and phenotypic ratio

[No ecf for wrong F1 genotypes]

[Penalise 1m if correct symbols are not used]

(b) *Drosophila melanogaster*, commonly known as the fruit fly, has been a cornerstone of genetic research for over a century. The practical benefits of using *Drosophila* are numerous and contribute significantly to its widespread adoption in laboratories.

(i) Suggest why fruit flies are good experimental organisms for carrying out crosses in genetic research.

.....[1]

[Any 1]

1. Fruit flies have a short life cycle; allowing researchers to study multiple generations in a short period.
2. Fruit flies lay can produce a large number of offspring from a single cross; providing sufficient sample sizes for statistical analysis.
3. Fruit flies are easy to handle or maintain / are small and require minimal space and resources / can be cultured in large numbers in simple lab vials with standard media / are easily anesthetized (e.g., by chilling) for examination and manipulation under a microscope
4. Fruit flies show distinct phenotypes which make it straightforward to observe (and track the inheritance of specific genes through crosses)
5. Fruit flies have a relatively small number of chromosomes (only four pairs (three autosomes and one pair of sex chromosomes), simplifying analysis.

The *purple* gene is known for its impact on eye color in *Drosophila*. The recessive allele results in a characteristic "purple eye" phenotype, a clear deviation from the normal, wild-type red eyes typically observed in fruit flies

The vestigial gene controls wing development in *Drosophila*. The recessive allele leads to a distinctive "short, 'vestigial' wings" phenotype. Flies exhibiting this mutation are unable to fly, representing a significant morphological and functional alteration from the normal, long wings of wild-type flies.

Pure breeding flies with red eyes and long wings were crossed with pure breeding flies with purple eyes and vestigial wings. All the F1 flies show red eyes and long wings.

These F1 flies undergo a **test cross** and the following results were obtained in this F2 generation.



<b>Phenotype of F2 flies</b>	<b>Observed number</b>
red eyes and long wings	305
red eyes and vestigial wings	41
purple eyes and long wings	39
purple eyes and vestigial wings	315
<b>Total</b>	<b>700</b>

(ii) A chi-squared ( $\chi^2$ ) test was carried out. **Fill in the blanks in the table** below to reflect the expected ratio and expected numbers for each class of phenotype. Calculate the  $\chi^2$  value.

$$\chi^2 = \sum \frac{(O - E)^2}{E} \quad \nu = c - 1$$

where  $\Sigma$  = 'sum of...'

O = observed 'value'

$\nu$  = degrees of freedom

E = expected 'value'

c = number of classes

### A chi-squared table.

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

[2]

<i>Classes</i>	<i>Observed number (O)</i>	<i>Expected ratio</i>	<i>Expected number (E) 2d.p.</i>
red eyes and long wings	305		
red eyes and vestigial wings	41		
purple eyes and long wings	39		
purple eyes and vestigial wings	315		
<b>Total</b>	<b>700</b>		

Calculated  $\chi^2 = \dots\dots\dots$

Answers:

<i>Classes</i>	<i>Observed number (O)</i>	<i>Expected ratio</i>	<i>Expected number (E) 2d.p.</i>
red eyes and long wings	305	1	175.00
red eyes and vestigial wings	41	1	175.00
purple eyes and long wings	39	1	175.00
purple eyes and vestigial wings	315	1	175.00
Total	700		

[1m]

Calculated  $\chi^2$  value:

$$\chi^2 = \sum \frac{(O - E)^2}{E}$$

$$= \frac{(305-175)^2}{175} + \frac{(41-175)^2}{175} + \frac{(39-175)^2}{175} + \frac{(315-175)^2}{175}$$

$$= 416.87 \text{ (2 d.p.) (follow the pattern of representation in the chi-squared table)} \quad [1m]$$

(iii) Using the **probability that the difference between observed and expected results is due to chance** for the calculated  $\chi^2$ , state the conclusions that can be drawn.

.....[2]

[Marking guidance: no error carried forward if calculated  $\chi^2$  in (ii) is wrong]

- 1 [How to arrive to conclusion] As p-value (for the calculated  $\chi^2$ ) is less than 0.001 / <0.001, which is **smaller than benchmark p=0.05**;

[Conclusions]

- 2 there is significant difference between the observed and the expected values at the 5% level (at least 1 mention of p=0.05 in either pt 1 or 2) in each phenotypic class. Any difference is due to chance.
- 3 The observed numbers (Reject: ratio) do not fit the expected phenotypic ratio of 1 red eyes, long wings : 1 red eyes and vestigial wings : 1 purple eyes and long wings : 1 purple eyes and vestigial wings.

[Any 2]

[Reject: if students uses critical  $\chi^2$  method to arrive at conclusion]

(iv) Explain the conclusion derived from (iii).

.....[2]

[No ecf if (ii) is wrong]

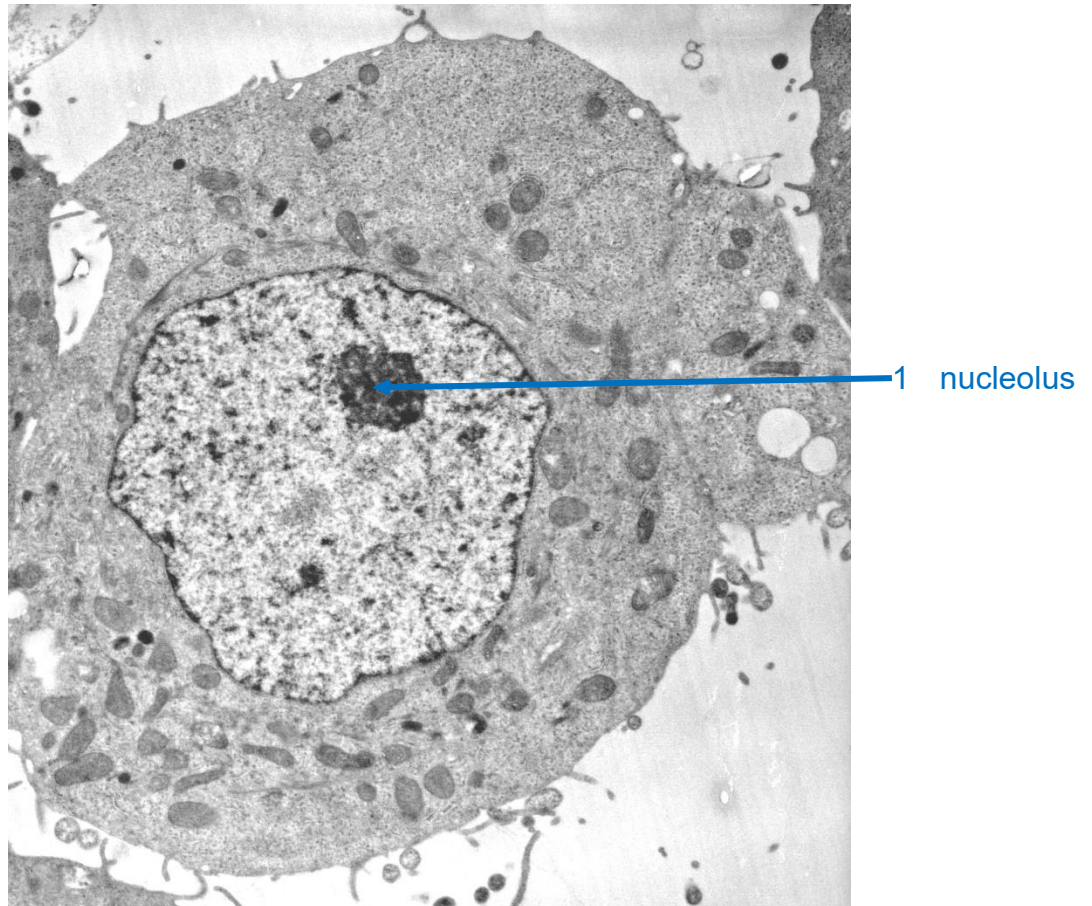
1. There is no independent assortment of genes ;  
as the **genes for eye color and wing length** are located on the same chromosome  
/ there is **partial linkage of genes** for eye color and wing length;
2. Recombinants are present ; due to **occasional crossing over** that **breaks the linkage** between the 2 genes on the same chromosome ; (resulting in the F2 generation results showing a larger number of non-recombinant and smaller number of recombinant phenotypes)

[Total: 12]

**QUESTION 3**

Translation is a fundamental biological process involving several key macromolecules. One such macromolecule is ribosomal RNA (rRNA) which is a structural component of ribosomal subunits.

- (a) Use an arrow on Fig. 3.1 to indicate the location where ribosomal RNA (rRNA) and proteins are assembled to form ribosomal subunits. Identify the organelle.  
.....[1]

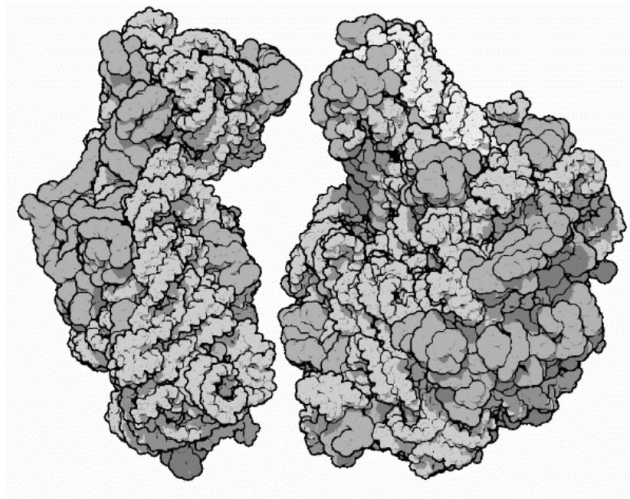


**Fig. 3.1**

Picture modified from <https://commons.wikimedia.org/wiki/>

1. Both correct location using an arrow, and name of organelle;

Fig. 3.2 shows the two eukaryotic ribosomal subunits formed.



**Fig. 3.2**

**(b)** Explain the functions of the two ribosomal subunits in translation.

.....[2]

[Small subunit] [Any 1]

- 1 Binds to the mRNA / **contains mRNA binding site**, ensures that each codon is correctly paired with the (complementary) anticodon of a tRNA ;
- 2 Ref. takes part in the scanning for start codon from the 5' end of mRNA;

[Large subunit] [Any 1]

- 3 Has rRNA / ribozyme / peptidyl transferase activity, to catalyse peptide bonds formation between amino acids ;
- 4 Ref. presence of A, P, E sites which are complementary in shape to incoming tRNA;

**(c)** Outline the key events that occur during the initiation of translation in a eukaryotic cell.

.....[4]

[Description of translation from lecture book minus GTP point]

- 1 **Initiation factors** and the **initiator tRNA** which carries the amino acid methionine associates with the **small ribosomal subunit** forming a pre-initiation complex ;
- 2 Pre-initiation complex then moves downstream along the mRNA (from 5' end) until it reaches the **start codon, AUG** which signals the start of translation ;
- 3 **Initiator tRNA** with **anti-codon UAC**, binds to the start codon, AUG on the mRNA via **complementary base-pairing / hydrogen bonds** ;
- 4 Large ribosomal subunit attaches to the small ribosomal subunit in the pre-initiation complex to form the translation initiation complex ;
- 5 Initiator tRNA is positioned at the **Peptidyl-tRNA binding site (P-site)** of the **large ribosomal subunit**.  
(The **Aminoacyl-tRNA binding site (A-site)** is exposed for **incoming aminoacyl-tRNA** in the elongation of polypeptide chain)

- (d) With reference to the reproductive cycle of the influenza virus and named cellular structures or molecules in its host, explain why the influenza virus is described as an obligate parasite.

.....[3]

[Obligate parasite]

- 1 Influenza virus cannot replicate independently on its own (because it lacks proteins, enzymes and organelles involved in processes of ATP synthesis, replication, and protein synthesis);

[Quote 2 cellular structure or molecule]

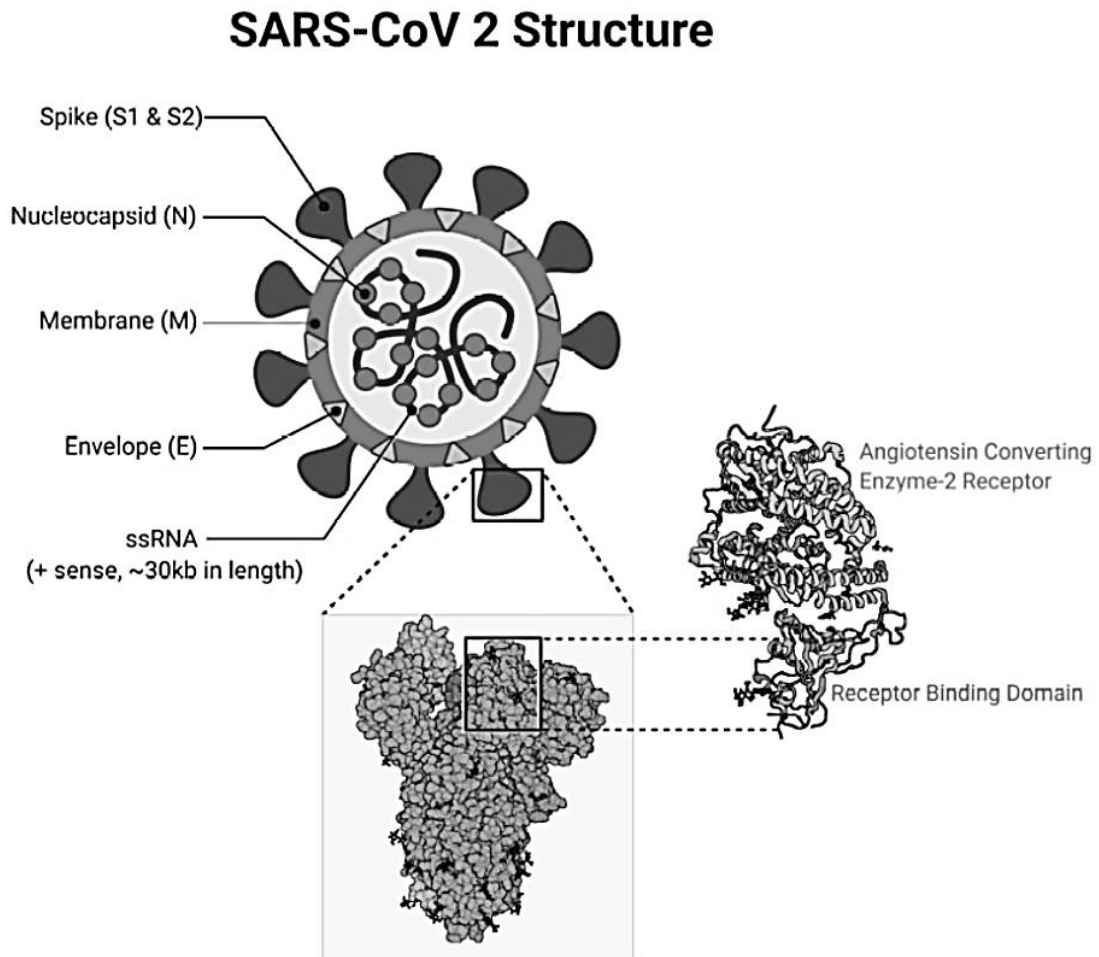
- 2 Use of host cell's ribosomes; for translation of viral mRNA into viral proteins ;
- 3 Use of host cell's amino acids; for incorporation into viral proteins ;
- 4 Use of host cell's tRNA ; for transporting specific amino acids to the ribosomes;
- 5 Use of host cell's ribonucleoside triphosphates [Reject: deoxyribonucleoside triphosphate]; for viral RNA replication ;
- 6 Use of host cell's ATP ; for energy-requiring processes such as viral replication and protein synthesis;
- 7 Use of host cell's endoplasmic reticulum and Golgi apparatus; for post-translational modifications / glycosylation and packaging of viral envelope proteins ;
- 8 Use of host cell's cytoskeleton; for intracellular transport of viral components to cell surface membrane for assembly of viruses ;
- 9 Use of cell surface membrane for viral envelope;

**[Total: 10]**

**QUESTION 4**

The Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infects human host cells of multiple organ systems, resulting in the disease COVID-19.

Fig. 4.1 shows the structure of the SARS-CoV-2, and its interaction with angiotensin-converting enzyme 2 (ACE2) receptor found on host cells.

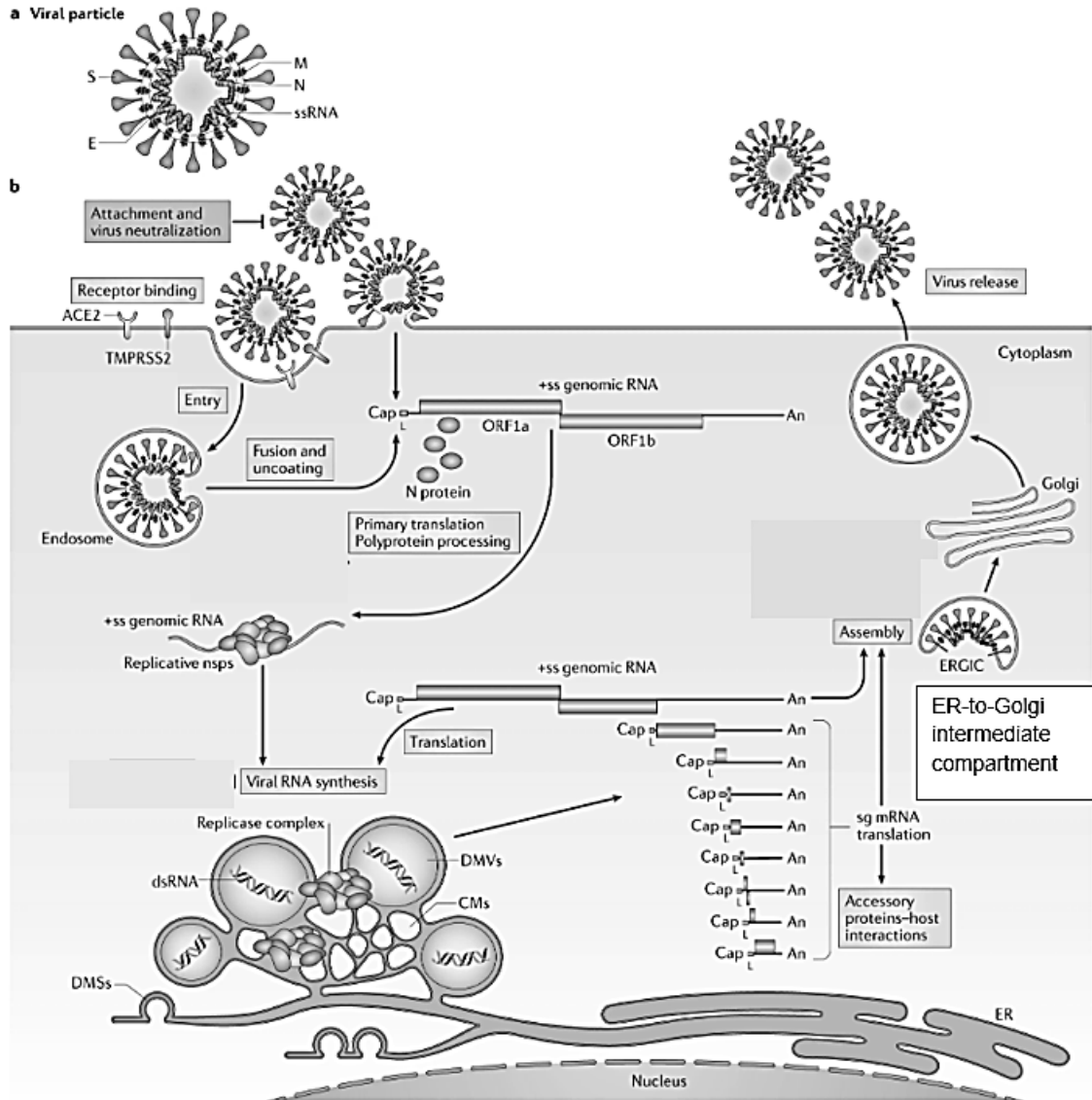


SARS- CoV 2 Structure Contributed by Rohan Bir Singh, MD; Made with Biorender.com

Fig. 4.1

Fig. 4.2 shows the reproductive cycle of SARS-CoV-2.

From: Coronavirus biology and replication: implications for SARS-CoV-2



(a) With reference to Fig. 4.1 and/or Fig. 4.2,

(i) Explain why the virus can affect multiple organ systems.

- .....[1]
1. Ref. presence of specific Angiotensin Converting Enzyme 2 / ACE2 receptors on the cell surface of cells of different organ systems which are **complementary in shape** to the viral glycoprotein for binding.

(ii) Both influenza virus and SARS-CoV-2 replicate their own genome. The influenza virus carries its own viral RNA polymerase in its virions but not SARS-CoV-2. Suggest why.

- .....[2]
1. Influenza virus genome is negative sense RNA there is **no available enzyme in host** cells to use negative sense RNA as a template to make positive sense RNA;



2. whereas SARS-CoV-2 has a positive sense RNA genome, which can be used as a template for translation using host ribosomes to make the viral RNA dependent RNA polymerase.

(iii) Contrast the mode of exit from host cells, between influenza virus and SARS-CoV-2.

- .....[2]
1. The influenza virus exits host cells by budding while SARS-CoV-2 exits host cells by exocytosis  
/  
[Process description] During budding, the **host cell surface membrane pinches off to form the viral envelope**, enclosing the viral nucleocapsid and enzymes ; during exocytosis, the virus is being **packaged into vesicles** which **bud off from the Golgi body** inside the cell and **transported** to the membrane for **release** after **fusion** of vesicle membrane with the cell surface membrane.
  2. [Effects on cell surface membrane] Budding pinches off the cell surface membrane (to form viral envelope), **removing** it in the process;  
Exocytosis **adds to** the cell surface membrane after fusion of vesicle membrane with the cell surface membrane
  3. [Source of viral envelope] During budding, the virus acquires its viral envelope (with embedded viral glycoproteins) **through the host cell surface membrane**;  
during exocytosis, the virus **obtains its viral envelope from intracellular membranes / ERGIC membrane**
  4. [time of acquiring viral envelope] During budding process itself, the virus acquires its viral envelope (with embedded viral glycoproteins) **through the host cell surface membrane**; during exocytosis, viral envelope is often **acquired earlier** (e.g., in the ERGIC) before exocytosis.  
[Any 2]

(iv) Explain how viral protease inhibitors work in the treatment of SARS-CoV-2 infection

- .....[2]
1. viral polyproteins [from Fig. 4.2] would not be cleaved.
  2. (newly formed) virus would not be functional, hence **unable to further infect cells**.

**(b)** The SARS-CoV-2 infections may induce the formation of syncytia, which are multinucleated cells, similar to those formed in HIV infections.

**(i)** Describe how HIV infections lead to syncytia formation

.....[2]

1. Presence of viral Gp120 on **infected host cells** **bind** to CD4 receptors **on other non infected cells**

2. Ref. **fusion** of cell surface membranes to form multinucleated cell / syncytium.

**(ii)** State how T cells of the adaptive immune system may react towards syncytia to result in its direct destruction.

.....[1]

1. Ref. cytotoxic T cells killing infected host cells by releasing perforin and granzymes.

**[Total: 10]**

### QUESTION 5

Fig. 5.1 shows a process occurring between a bacterium (*Escherichia coli*) and a phagocyte.

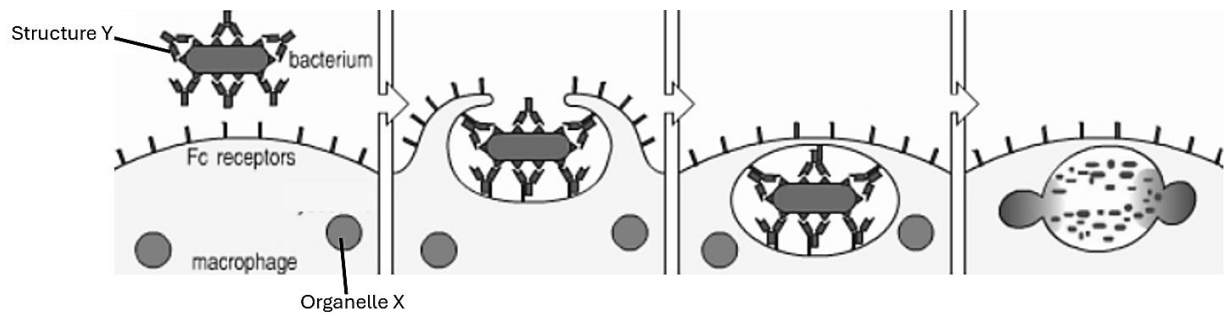


Fig. 5.1

(a) Identify organelle X and explain how its structure facilitates the process in Fig. 5.1 .

..... [3]

Organelle X : .....

1. Lysosome

AND

How structure facilitate process:

.....

2. Ref. **phospholipid bilayer** (structure) to allow fusion with phagosome / vesicle containing bacterium (function).

3. Ref. presence of **hydrolytic enzymes** (structure) to hydrolyse or break down bacterium during phagocytosis (function).

(b) Explain how the structure of Y facilitates the process in Fig. 5.1.

..... [2]

How structure of Y facilitate process:

.....

1. Ref. Variable region / antigen binding site of antibodies with **complementary shape** (structure) that allows binding of antigen on bacterium (function).

2. Ref. Constant region with **complementary shape** (structure) that allows binding to Fc receptors (from Fig. 5.1) on phagocyte (function)

[Reject: other structures on antibodies which do not help in opsonization]

(c) Fig. 5.2 shows a process occurring in *Escherichia coli* which facilitates gene transfer between bacteria cells. Beneficial genes such as genes that code for bacterial capsule found outside the cell wall.

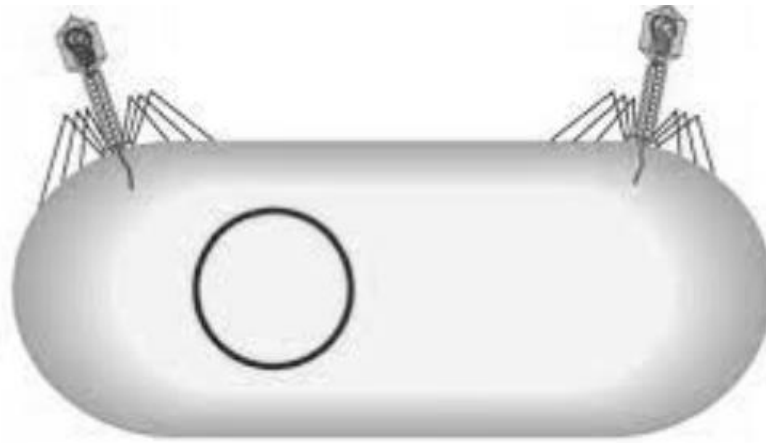


Fig. 5.2

With reference to the reproductive cycle of bacteriophages, describe this process of gene transfer and how it benefits the recipient bacteria.

- ..... [3]
1. During generalized transduction, host bacterial DNA can be accidentally packaged into capsid of newly assembled (lytic) phages
  2. The resulting transducing phages infect other bacteria, injecting donor DNA into new host; **homologous recombination** occurs in the new host cells;
  3. New host **expresses the genes for capsule synthesis** to make capsule which physically covers the antigens; so existing **antibodies are unable to recognise and bind to the antigen on bacteria**;

[Reject: specialised transduction due to showing of lytic phage which usually facilitates generalised transduction]

**(d)** Bacteriophages can also serve as antibacterial agents.

Comment on the potential effectiveness of bacteriophages and **antibodies** of the immune system in bacteria infections.

- ..... [2]
1. Both/either are/is highly **specific** due to **complementary shape recognition** between attachment sites on tail fibres of bacteriophages/variable region on antibodies.
  2. Both/either have/has a **chance of random mutations** and can evolve alongside with the bacteria for long-term effectiveness.
  3. Antibodies can bind better to the same antigen when B cells undergo somatic hypermutations in the segments coding for variable region;

[Any 2]

**[Total: 10]**

## QUESTION 6

Fig. 6.1 shows a pedigree tree tracing the inheritance of a genetic disorder in a family.

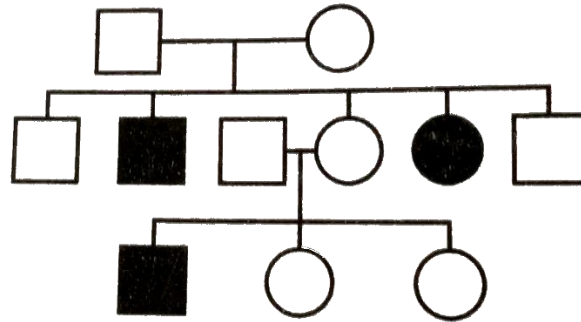


Fig. 6.1

(a) With reference to Fig. 6.1, state the mode of inheritance of this genetic disorder.

.....[1]  
 1 Autosomal recessive ;

Fig. 6.2 shows the two alleles of a gene involved in the same genetic disorder.

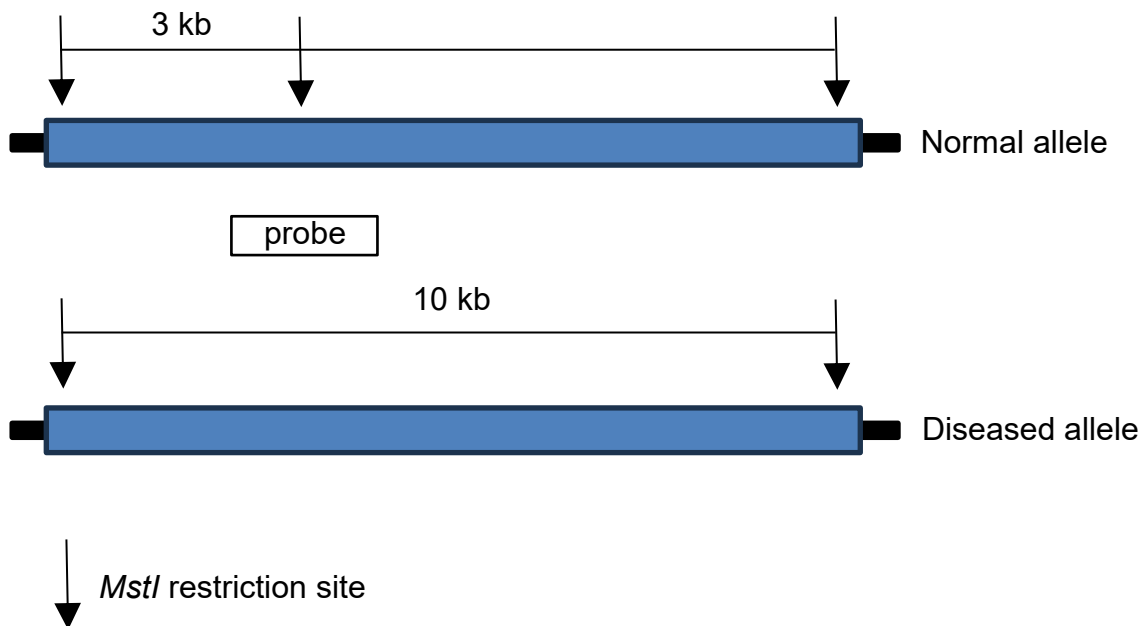
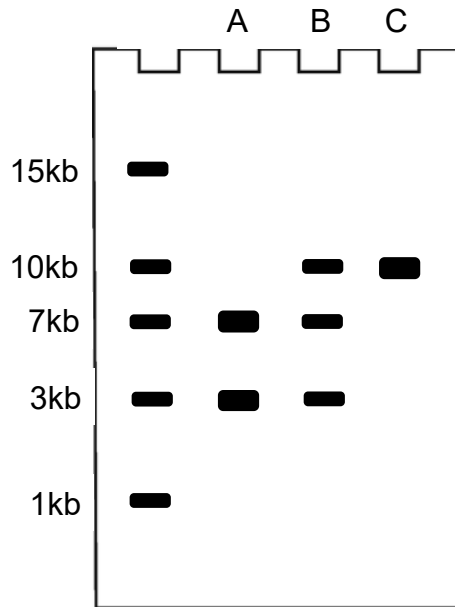


Fig. 6.2

A family was screened for this genetic condition. Genomic DNA from cheek cells were isolated and treated with restriction enzyme *MstI* which cut DNA at specific sites indicated (↓). The gel was loaded with a DNA molecular weight ladder and samples from one healthy individual (A), one carrier (B), and one affected individual (C).

(b) Draw the expected banding pattern on the **autoradiogram** for individuals A – C.

.....[3]



Mark pt	Genotypes	Fragment sizes
1	A (homozygous for normal allele), thickness double of B's	3kb, 7kb
2	B (heterozygous, carrier for diseased allele) thickness half of A or C's	3kb, 7kb, 10kb
3	C (homozygous for diseased allele), thickness double of B's	10kb

(c) Explain how harmful recessive alleles may be preserved in a natural population.

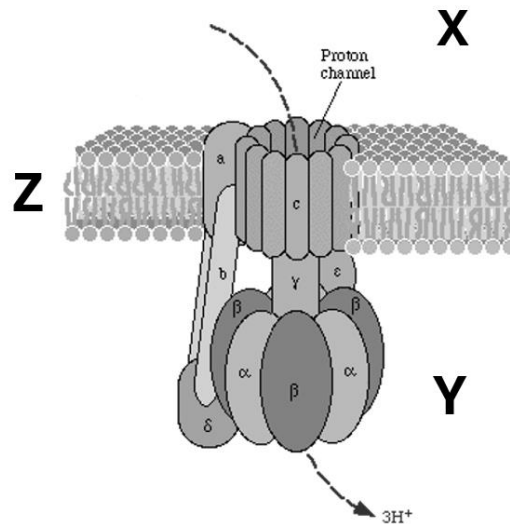
.....[2]

- 1 [Diploidy] Recessive allele in heterozygote not expressed, genetic variation is maintained in the form of recessive alleles ;  
(Only alleles present in the homozygous recessive organisms will be expressed and be exposed to natural selection)
- 2 [heterozygote advantage] heterozygotes tend to have a greater fitness compared to homozygotes;
- 3 [frequency dependent selection], the phenotype that is rare tends to be at an advantage ; this preserves rare alleles in the population ; [e.g. cichlids]
- 4 When an organism containing the recessive allele in its genotype is able to **survive and reproduce, the recessive allele is passed down** to the offspring (and is preserved in the population) ;

[Total: 6]

## QUESTION 7

ATP synthase is an enzyme that synthesizes ATP from ADP and inorganic phosphate (Pi). Fig.7.1 shows the structure of ATP synthase. It is found in both chloroplasts and mitochondria.



**Fig. 7.1**

- (a) Identify the regions X, Y and structure Z as found in mitochondria and chloroplast respectively.

	X	Y	Z
Mitochondria			
Chloroplast			

[2]

	X	Y	Z
Mitochondria	intermembrane space	Mitochondrial <u>matrix</u>	Inner mitochondrial membrane
Chloroplast	thylakoid lumen	stroma	Thylakoid membrane

1. 1m for each correct row.

In both organelles, a proton gradient exists across the membrane where ATP synthase is located. The synthesis of ATP via ATP synthase is coupled with the diffusion of  $H^+$  across the proton channel down its concentration gradient. Describe how this proton gradient is generated in the **chloroplast**.

..... [3]

1. **Excited** special chlorophyll a electrons are accepted by primary electron acceptor in the reaction centre
2. Electrons are passed down the electron transport chain; and energy released is used to **pump**  $H^+$
3. From stroma into thylakoid space against concentration gradient
4. Ref. to thylakoid membrane being impermeable to  $H^+$

[Any 3]

- (c) Inhibitors can block electron flow at specific points in the electron transport chain, preventing further electron transfer downstream of the block. By observing which carriers become reduced and which become oxidized when an inhibitor is introduced, we can deduce their positions relative to the block.

An investigation to study the effect of three inhibitors A, B and C, on the electron transport chain in the mitochondria was carried out. In each of the three experiments, a different inhibitor was added. Table 7.2. shows the state of the electron carriers, W to Z, after the addition of inhibitor.

**Table 7.2**

Inhibitor added	Electron carrier			
	W	X	Y	Z
A	oxidised	reduced	reduced	oxidised
B	oxidised	oxidised	reduced	oxidised
C	reduced	reduced	reduced	oxidised

- (i) Give the order of the electron carriers in this electron transport chain.

				[1]
--	--	--	--	-----

**Y X W Z**

- (ii) Explain your answer

..... [3]

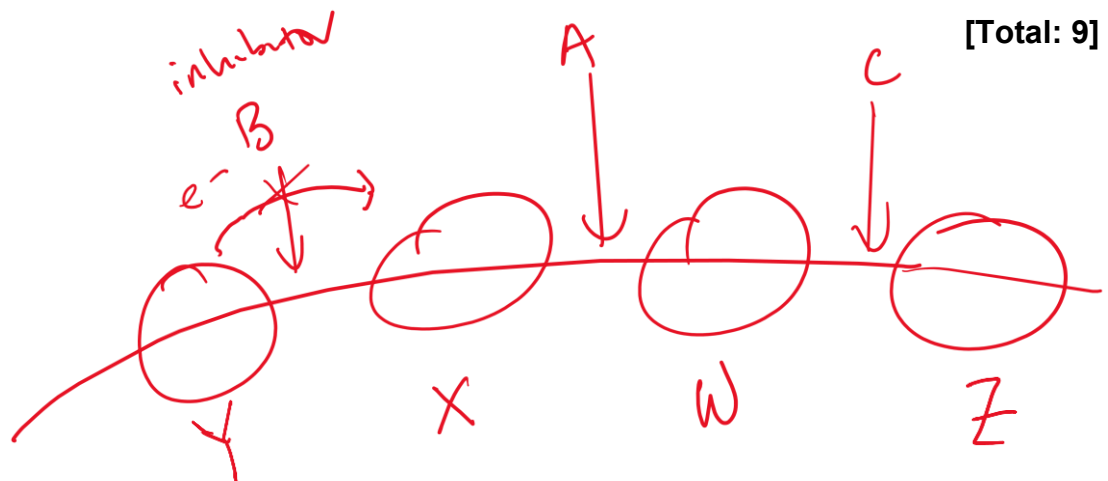
(Inhibiting a carrier will cause electrons to accumulate upstream of the inhibition site, leading to a reduced state of the carriers in that region.



Also downstream of the inhibition site, the carriers will become more oxidized as they cannot donate their electrons.)

1. Inhibitor B leads to only electron carrier Y being reduced; electron carrier Y **carries the electron** and is unable to transfer it to the next electron carrier; (thus, is the first electron carrier of the block)
2. Inhibitor C leads to only electron carrier Z being oxidised; electron carrier Z **does not receive electron** from previous electron carrier; (thus, is the last electron carrier of the block)
3. Inhibitor A leads to electron carriers Y and X being reduced, and electron carriers W and Z oxidised, thus electron carrier X is unable to transfer the electron to electron carrier W;

[Max 3]



[Total: 9]

### QUESTION 8

Retinoblastoma is a malignant childhood tumour of the eye, originating from neuronal cells of the developing retina and typically diagnosed by clinical symptoms in children under 3 years of age. At the cellular level, retinoblastoma develops when both alleles of the *RB1* gene on chromosome 13 are mutated.

If an early mutation of one *RB1* allele occurs and subsequently, non-disjunction occurs in the same cell lineage, a subset of cells will be mutated (mosaicism) while others remain normal. Such individuals with the mutated cells have an increased risk for the development of retinoblastoma.

(a) State if the *RB1* gene is a tumour suppressor gene or an oncogene. Explain your answer.

.....[2]

- 1 [State] Tumour suppressor gene ;
- 2 (mutated) tumour suppressor alleles behave like recessive alleles, **two mutated alleles** are required **to produce non-functional protein products** that cannot inhibit cell cycle arrest / DNA repair / apoptosis ;  
(Oncogenes behave like dominant alleles, presence of **one mutated allele** is needed to produce abnormal protein which overstimulates cell divisions)

(b) Explain how non-disjunction during mitosis can contribute to the development of retinoblastoma in a patient with a mutation in one *RB1* allele.

.....[2]

- 1 Failure of chromatids [~~reject: homologous chromosomes~~] for chromosome 13 carrying **normal allele** to separate in anaphase ;
- 2 Results in formation of some cells with only a **single mutated allele** (and no normal allele),  
lack of functional Rb protein products in these cells increase risk of cancer development ;

The *RB1* gene encodes a protein that is involved in cell cycle progression through the recruitment of histone deacetylase, which controls the expression of genes required for S phase entry.

(c) Explain the effect of non-functional RB proteins on S phase entry.

.....[4]

- 1 Histone deacetylase **not** recruited,  
acetyl groups remain bound to lysine residues on histone tails ;
- 2 **Reduced** affinity of histone tails for binding to DNA /  
Nucleosomes do not bind to neighbouring nucleosomes ;
- 3 Chromatin remains in a relaxed (open) state  
/ Histone complex loosens to form 10nm chromatin fibre or “beads on a string” structure ;
- 4 RNA polymerase and transcription factors have **greater** access and transcribe genes required for S phase entry / **uncontrolled** cell division ;

In recent studies, scientists have identified “kill switches” among small ribonucleic acids called microRNAs (miRNAs), as well as in larger protein-coding RNAs, all of which are encoded by the genome of eukaryotic cells. These microRNAs function by binding to specific messenger RNA (mRNA) targets in cancer cells, resulting in the degradation of the mRNA or inhibition of its translation into protein. This regulatory mechanism make them promising candidates for cancer treatment over traditional chemotherapy.

(d) Explain the advantages of using microRNA-based therapies compared to traditional chemotherapy for the treatment of cancer.

.....[2]

- 1 Targeted action as miRNAs target **specific** mRNAs involved in cancer pathways, allows for precise regulation of gene expression in cancer cells / restores normal cell cycle control and promote apoptosis in cancer cells (**compared to non specific nature of chemotherapy inside cancer cells**);
- 2 Reduced side effects, less likely to affect healthy cells; (compared to chemotherapy affecting healthy cells)
- 3 Will not develop drug resistance; (compared to drug resistance from chemotherapy),

Reject:

- 4 Personalized treatment options possible based on the expression profile of individual tumours, e.g. targeting specific types of mRNA, allows for more effective treatment ; compared to chemotherapy (still allows personalisation to some extent)

[Any 2]

**[Total: 10]**

### QUESTION 9

In 2017, researchers have successfully cloned **2 macaque monkeys (*Macaca fascicularis*)** for the first time, by using the somatic cell nuclear transfer (SCNT) **method**.

Fetal fibroblasts derived from an aborted monkey fetus were used for nuclear transfer into oocytes obtained from female monkeys of the same species. Artificial activation of oocytes is carried out before being transferred to healthy surrogates for implantation in the womb. Pregnancy was subsequently confirmed in surrogates and yielded 2 live births. This is shown in Fig. 9.1 below.

Such cloning via SCNT allows the production of genetically uniform monkeys as animal models for basic research in primate biology, for studying human disease mechanisms and therapeutic treatments.

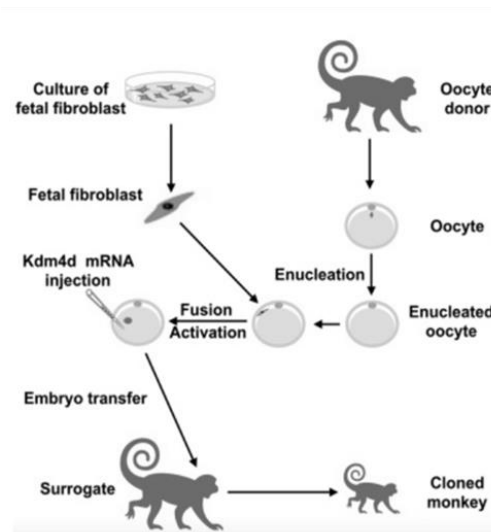


Fig. 9.1

(a) SCNT involves removing the nucleus of an unfertilized egg cell, replacing it with the nucleus of a somatic cell (fetal fibroblast). Explain why this step is necessary.

.....[2]

1. Fetal fibroblast nucleus is **diploid**; while the **replaced** unfertilized egg nucleus is **haploid** ;
2. So that upon mitotic division during embryo development will maintain a state of diploidy ; (since no fusion of gametes in SCNT)

- (b) Upon artificial activation of the oocytes, they can either be directly transferred to a surrogate female to produce a cloned animal or such oocytes are allowed to form blastocysts *in-vitro* and embryonic stem cells can be extracted for therapeutic treatments.

What is the potency level and function of such cells used for therapeutic treatments?

.....[3]

1 [potency level] Pluripotent ;

[Function]

2 Able to differentiate into **almost any** cell type or **organ except extra embryonic tissues**

3 Normal function is to divide and differentiate into multiple specialized cell types to give rise to specific **organs** such as the heart, lung, skin

OR

give rise to cells that form three primary layers of cells known as embryonic germ layers – endoderm, mesoderm and ectoderm.

Scientists have an alternative method of generating embryonic stem cells, which is to genetically reprogramme adult cells to an embryonic stem cell-like state by introducing transcription factors into adult cells such as skin cells. Such induced pluripotent stem cells (iPSCs) are generally preferred over SCNT as they overcome moral questions about the status of the embryo and its potential destruction.

- (c) Suggest one **other reason** why the use of iPSCs evades the ethical concerns over SCNT derived **embryonic stem cells**

.....[1]

1 **Patient-Specific:**

iPSCs can be generated from a patient's own cells, reducing the risk of immune rejection in transplantation.

2 **Accessibility:**

iPSCs can be derived from readily available somatic cells (e.g., skin cells), making them more accessible than oocytes needed for SCNT.

3 **Non invasive:**

The use of somatic cells is a relatively non invasive procedure. Hence, this poses fewer risks to donors, compared to oocyte retrieval which is an invasive procedure.

[Any 1]

- (d) Suggest two applications of how embryonic stem cells can be used in regenerative medicine and disease research

Application


1 .....

Application

2 .....

[2]

[Any 2 from 3<sup>rd</sup> column]



1	Regenerative medicine	Repair organs & tissue	ESCs can be differentiated into specific cell types and used to repair or replace damaged tissues and organs.  [Extra info] cardiomyocytes - heart repair neurons - spinal cord injuries retinal cells - vision restoration
		Organ transplantation	ESC-derived cells could potentially reduce the need for organ donors  Decrease the risk of organ rejection in transplants.
2	Drug development & Toxicology testing	Disease Modelling	By differentiating ESCs into cells affected by specific diseases, researchers can gain a better understanding of disease progression
		Drug screening	Allowing researchers to test potential drugs for safety and efficacy on cells, including assessing cardiotoxicity and other potential side effects
3	Others	Personalized Medicine	Patient-specific ESC lines could be created to test the effectiveness of different treatments for an individual.
	Reject (focus is on ESC)	Animal model	Creating genetically uniform monkeys as animal models for basic research in primate biology, for studying human disease mechanisms and therapeutic treatments.

[Total: 8]

### QUESTION 10

Fig. 10.1 shows the Kakapo, New Zealand's unique, critically endangered herbivorous parrot. It is the world's only flightless parrot with green plumage, strong legs and large feet. Formerly widespread, Kakapo are now found exclusively on predator-free offshore islands, consisting of dense forest undergrowth and trees.



Fig. 10.1

(a) Explain how natural selection resulted in the evolution of kakapos with strong legs and large feet.

- ..... [4]
1. Variation in flight ability / size of feet / strength of legs exists due to mutation.
  2. Ref. **Selection pressure**; is the presence of food in the forests / on trees  
[Reject: presence of predators as context stated so]
  3. Kakapos with strong legs and large feet are **better able to navigate the dense forests and climb trees to access food**;
  4. Hence they have a selective advantage over those with weaker legs and smaller feet , and are able to survive better, and **reproduce**, and **pass down the advantageous alleles** to their offspring.
  5. [talk abt definition of evolution] Over time, allele frequencies will change, causing kakapos with strong legs and large feet to become more and more common.

(b) Suggest a reason why kakapos have lost the ability to fly.

- ..... [1]
1. Ref. food sources can be found on low ground (without the kakapos having to fly)
  2. Ref. absence of predators, thus kakapos fly less and conserve energy for other survival traits
  3. Due to absence of predators, kakapos may build nests on low ground  
[Any 1]

(c) Table 10.1 shows the taxonomic classification information of the kakapos.

Fill in the table to depict accurately, the taxonomic ranks.

..... [1]

<b>Taxonomic rank</b>	<b>Information</b>
Kingdom	Animalia
	Chordata
Class	Aves
	Psittaciformes
Family	Strigopidae
Genus	
Species	<i>Strigops habroptilus</i>

Answers

<b>Taxonomic rank</b>	<b>Information</b>
Kingdom	Animalia
<b>Phylum</b>	Chordata
Class	Aves
<b>Order</b>	Psittaciformes
Family	Strigopidae
Genus	<b>Strigops</b>
Species	<i>Strigops habroptilus</i>

[All for 1m]



(d) Describe the principles which scientists use to classify organisms into taxonomic groups.

..... [2]

1. Scientists nest/place smaller groups within bigger groups in a **hierarchical manner** (compulsory marking point)
2. Based on (morphological, anatomical, physiological) similarities.

(e) There is another parrot native to New Zealand. Similar to the kakapos, this parrot also has green plumage, and also has strong legs.

Discuss why the information provided is insufficient to conclude that this parrot and the kakapos are the same species.

..... [2]

The information provided (morphology similarity) is **not sufficient** to conclude if the parrot and the kakapos are the same species.

1. There is subjectivity in determining the structural features to be similar or different / it is difficult to determine the degree of difference that is required to indicate separate species
2. Some organisms of different species **may look alike due to convergent evolution.**
3. More tests need to be done , allowing the parrots to interbreed, and if viable and fertile offspring (biological species concept), then they are the same species / study the degree of similarity in DNA sequence of the same gene;

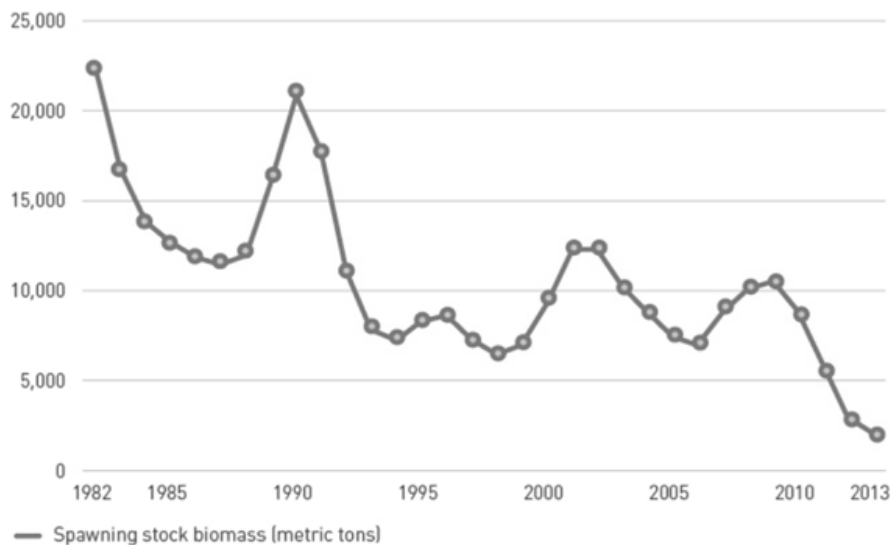
[Any 2]

**[Total: 10]**

## QUESTION 11

In the Gulf of Maine, Atlantic cod was once so abundant that Cape Cod was named in its honour. Fig. 11.1 shows data of how spawning stock biomass have plummeted drastically, leaving barely enough stocks to sustain a healthy population.

Yet despite increasingly strict quotas on commercial fishing, fish populations have not rebounded as expected. A recent study suggests that rapidly-warming waters in the Gulf is holding back the recovery.



spawning stock biomass = the total weight of all sexually mature fish within a population that are capable of reproduction.

Population of reproductive-age Atlantic cod in Gulf of Maine. Source: Pershing et al. (2015)

- (a) With reference to Fig. 11.1, calculate the percentage decline in spawning stock mass of cod from 1982 to 2013. Show your working and give your answer in **whole number**.

$$\frac{22\,500 - 2\,000}{22\,500} \times 100\% = 91.11 \approx 91\%$$

**Marking Reference:**

1982 - 22500

2013 - between 2000 - 2500

Percentage decline = accept between 89% to 91%

percentage decline = 91% [2]

1. correct calculation and working;
2. correct presentation to whole number

Global warming is significantly impacting fish migration patterns as rising ocean temperatures force fish to move towards cooler waters, often shifting their ranges northward or into deeper, colder areas.

Another extensive study was done on 2,572 fish populations belonging to 146 species in the Atlantic and Pacific Oceans. They found that the faster the fish migrates toward the poles, the faster their abundance declines. According to data collected, a poleward shift of 17km per year may result in a decline of 50% in the abundance of populations.

(b) Suggest and explain 2 factors that are contributing to these declines as fish populations are forced to migrate?

.....[3]

1. Stress of adapting to new environments / adjust to new water temp, salinity
2. Disruptions in food web/ Lack of suitable food source
3. Lack of suitable breeding grounds
4. Increased Predation from a lack of shelter / reduced defences against unfamiliar predators
5. Increased susceptibility to diseases

[Any 2]

AND

6. (Any reason from first 3 points) → Resulting in reduced fertility rates
7. (Any reason from pts 1,2,4,5) → Resulting in increased mortality

[Any 1] [only award pt 6/7 if paired with a main factor from pt 1-5]

**[Total: 5]**

End of paper