EUNOIA JUNIOR COLLEGE
JC2 Preliminary Examinations 2019
General Certificate of Education Advanced Level
Higher 2

CANDIDATE NAME								
CIVICS GROUP	**1**	**8**	**-**			REGISTRATION NUMBER		

H2 Biology

9744/02

Paper 2 Structured Questions

16 September 2019

2 hours

Candidates are to **answer all questions** in this question booklet.

READ THESE INSTRUCTIONS FIRST

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

Write in dark blue or black pen on both sides of the paper.
You may use an HB pencil for any diagrams or graphs.
Do not use paper clips, highlighters, glue or correction fluid.

The use of an approved scientific calculator is expected, where appropriate.

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

For Examiner's Use	
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2	
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10	
Total	**100**

This document consists of **25** printed pages and **3** blank page.

Section A

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

1 Fig. 1.1 shows the molecular structure of α-glucose.

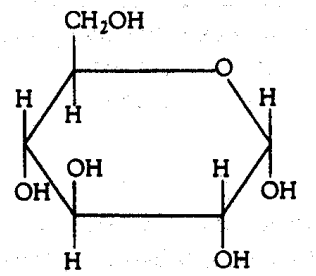
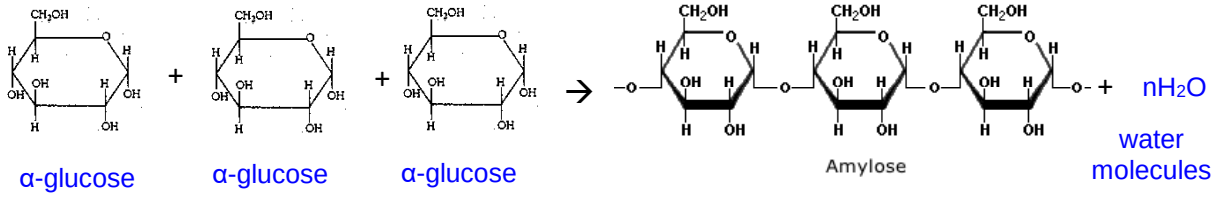


Fig. 1.1

(a) **(i)** Show in the space below, the reaction in which α-glucose molecules are joined to form the long chains found in starch. Label your diagram to show clearly all the components and resultant products of this reaction.



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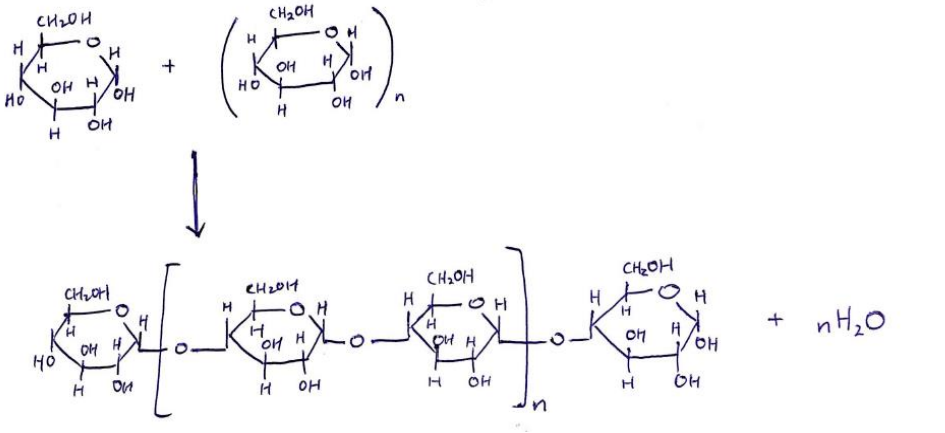


[2]

1. **Label diagram with reactants and products to show a chain e.g. as above (R: maltose structure);**
2. **Water molecules shown;**

Comments:

- Generally not well-answered.
- A large number of answers drew the structural formula of maltose. Candidates should be aware that for a molecule like starch, there should be more than 2 monomers of glucose.
- For candidates using the notation for repeating units, do bear in mind that the position of the brackets is important as one bracket should enclose the O atom of one glycosidic bond and the other bracket should not enclose the other O atom of the other glycosidic bond.
- If candidates are in doubt as to what they should do with repeating unit notation, they should just keep it simple and draw three or more units of glucose with dotted lines at either ends to show the continuation of the molecule as shown above.

(ii) State the precise name of the bond formed in (a)(i). [1]

α-1,4 glycosidic bond;

Comments:

- This part was generally well-answered.
- A small number gave the branch point α-1,6 glycosidic bond which is not so appropriate as the α-1,4 glycosidic bond is the main bond present.

(b) (i) Starch is made up of amylose and amylopectin. Amylopectin has a similar structure to glycogen.

Using your biological knowledge, state a structural similarity between amylopectin and glycogen. [1]

1. **Same monomer, α-glucose;**
2. **Same bonds - α-1,4 glycosidic bond, α-1,6 glycosidic bond;**

Comments:

- This question was generally well-answered.
- Many answers gave the branched pattern as a response but simple features like the type of glucose monomer or bond type would suffice as well.

(ii) Briefly explain how the structure of glycogen is related to its role in living organisms. [2]

1. **Many branch endpoints which are accessible to hydrolytic enzymes (e.g. amylase) → can be quickly hydrolysed to release many glucose molecules;**
2. **Extensive branching results in more glucose molecules present per unit volume → compact structure for storage;**
3. **Branched chains result in a macromolecular (large size) structure → insoluble in water → no osmotic effect on cell (suitable as a storage substance);**
4. **AVP; Any 2**

Comments:

- This part was not always well-done.
- A number of answers referred to the branch points being accessible to hydrolytic enzymes which can then break the molecule down to release energy. This is not exactly accurate as they should refer to the release many molecules of glucose which can be oxidized for energy.
- There were reference to a 'compact size' due to branching but many responses failed to unpack this, failing to refer to the ability to contain 'more glucose molecules per unit volume'.
- Some answers made reference to lack of OH groups in glycogen to form hydrogen bonds with water, hence glycogen was insoluble. This is not accurate as there are OH groups present but the macromolecular size prevented solubility.

(iii) Distinguish two structural differences between glycogen and cellulose. [2]

Feature	Glycogen	Cellulose
Monomer	**α-glucose**	**β-glucose;**
Glycosidic bonds	**α-1,4 glycosidic bonds (&α-1,6 glycosidic bond)**	**β-1,4 glycosidic bonds;**
Orientation of monomer	**All glucose units in the chain have the same orientation**	**Adjacent glucose molecules are rotated 180° with respect to each other;**
Structure of molecule	**Glycogen is a coiled branched molecule**	**Cellulose is a long, straight chain;**

Branching	Branching occurs at intervals of about 10-20 glucose units for amylopectin	No branching in cellulose;
AVP;		

Any 2

Comments:

- This was generally quite well-answerered.
- Some answers incorporated more than one feature of comparison in the same sentence.

Microorganisms present in a rabbit's gut are able to digest the carbohydrates in the plant materials that form their diet. **Fig. 1.3** shows the biochemical pathways for the digestion of cellulose and starch in the gut of a rabbit.

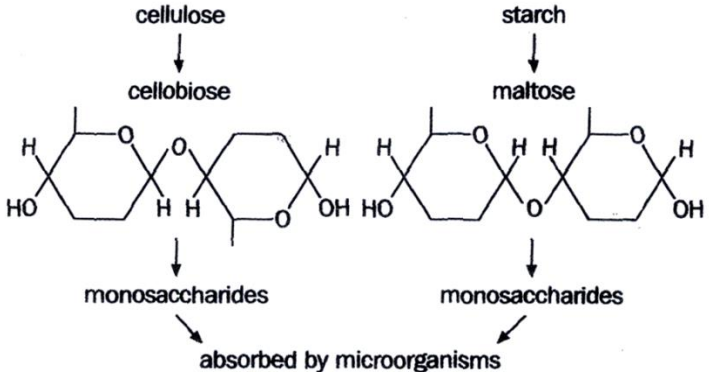


Fig. 1.3

(c) Cellobiose and maltose are both disaccharides. Explain why amylase enzymes produced by rabbits are unable to digest cellobiose. [2]

1. **β-1,4 glycosidic bond not complementary to active site of amylase**
2. **Amylase digest α-1,4 glycosidic bond since maltose is made up of α-glucose monomers but cannot digest cellubiose which has β-1,4 glycosidic bond since cellubiose is made up of β-glucose monomers;**
3. **Difference in the position of hydroxyl group / hydroxyl group is above the plane of the ring leading to different 3D conformations;**
4. **β-1,4 glycosidic bond has different 3D conformation compared to α-1,4 glycosidic bond;**

Any 2 but must include point 1.

Comments:

- This part was not always well-answered.
- Most answers were very vague about what exactly the active site of amylase was complementary or not complementary to.
- There were too few answers that made specific reference to the bond types although answers made reference to the different type of glucose monomers but failed to develop the answer further towards the different conformations of the α-1,4 glycosidic and β-1,4 glycosidic bond bonds.

[Total: 10]

2 Dehydrogenases are a metabolically important group of enzymes. They are involved in catalyzing reduction and oxidation (redox) reactions in many processes, including respiration.

(a) Dehydrogenases are specific in their action. The specificity of enzyme action can be explained by the 'lock and key' hypothesis or the 'induced fit' hypothesis.

Explain the 'induced fit' hypothesis for enzyme specificity. [3]

1. **As the substrate enters the enzyme active site, it induces a change in the structure of the active site of the enzyme;**
2. **This change causes the active site to enfold the substrate and hold it in place via the formation of weak bonds;**
3. **The change in conformation/structure allows the active site to be moulded into a more precise fit for the substrate, enabling the enzyme to perform its catalytic function most effectively;**
4. **The 3D conformation/structure of the enzymes reverts to its original state upon completion of the reaction and release of the product molecules;**

Comments:
- This question was not always well-answered.
- A number of answers were not explicit about point 1 with some answers being almost similar to 'lock and key' hypothesis.
- Many of the answers were not clear about the idea of the active site being moulded into a more precise fit, with some writing about the active site being 'more complementary'.
- Some candidates thought that enzymes with induced fit mechanism would have active sites that accept any substrate! This is incorrect! The active site is still complementary to the substrate concerned, just that the conformation of the active site is not a precise fit.
- Points 2 and 4 were not always appreciated by candidates.

(b) Dehydrogenases may require the presence of coenzymes to function properly.

(i) Explain the role of coenzymes in the proper function of enzymes. [2]

1. **Coenzymes are organic molecules;**
2. **The attachment of the coenzyme to main enzyme (apoenzyme) changes the shape of the enzyme active site;**
3. **so as to allow the enzyme-substrate complex to form more easily;**

Comments:
- This part was not well-answered.
- Some answers just rehashed the answer from part (ii).
- Many candidates wrote that coenzymes were ions, which is not correct, inorganic ions are cofactors to enzymes.

(ii) Nicotinamide adenine dinucleotide (NAD^+) is a common coenzyme of dehydrogenases. In glycolysis, the molecule glyceraldehyde-3-phosphate (G3P) is oxidized by dehydrogenation to 1,3-bisphosphoglycerate. This reaction is catalyzed by glyceraldehyde-3-phosphate dehydrogenase. In the process, NAD^+ is reduced to NADH.

The reaction is shown in **Fig. 2.1.**

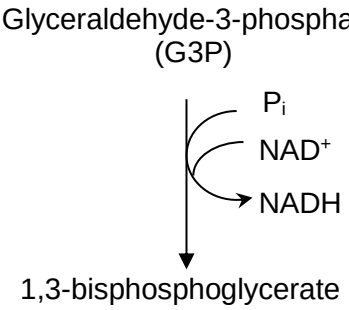


Fig. 2.1

Using information from your biological knowledge, suggest how NAD is converted to NADH during the oxidation of G3P to 1,3 bisphosphoglycerate. [2]

1. Removal of H oxidises G3P / oxidation of G3P via dehydrogenation
2. H is transferred to NAD (which is bound to G3P dehydrogenase), which became reduced to NADH;

Comments:
- A large number of answers were not able to link the removal of H from the oxidation of G3P (oxidation by dehydrogenation) and the subsequent transfer of this H to NAD.
- Some answers just revolved around the question stem with little clarity.

In a separate experiment, studies were carried out on soil-dwelling aerobic bacteria. Soil samples were taken at two depths, **A** and **B**. The samples were taken at intervals over six years to determine the activity of dehydrogenases, involved in the Krebs cycle.

Fig. 2.2 shows the mean dehydrogenase activity of the bacteria in these samples.

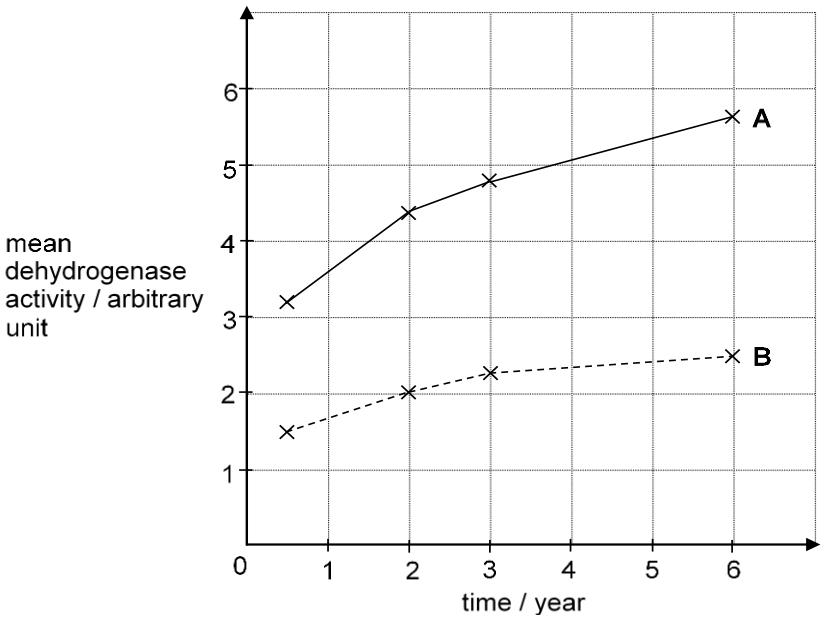


Fig. 2.2

(c) With reference to **Fig. 2.2** and your biological knowledge, explain which of the two samples, **A** or **B**, were taken from a greater depth. [3]

1. **Sample from depth B;**
2. **At a greater depths, lower oxygen concentration → Rate of oxidative phosphorylation / aerobic respiration was lower;**
3. **Mean dehydrogenase activity at greater depths was lower, ranging from 1.5 - 2.5 arbitrary units, while activity of sample at depth A was higher, ranging from approximately 3.2 - 5.6 arbitrary units; (comparison needed between A and B)**
4. **Regeneration of NAD^+ coenzyme at greater depth was slower hence lower rate of formation of enzyme-substrate complexes, lower rate of dehydrogenase activity;**

Comments:

- This part was generally well done.
- Candidates should be mindful that they need to quote supporting data
- However, some candidates thought that dehydrogenase activity was higher if there was less oxygen.
- Some candidates confused the Calvin cycle with Krebs cycle and stated that CO_2 needed for Krebs cycle.

3 Protein synthesis in a prokaryote is shown in **Fig. 3.1**.

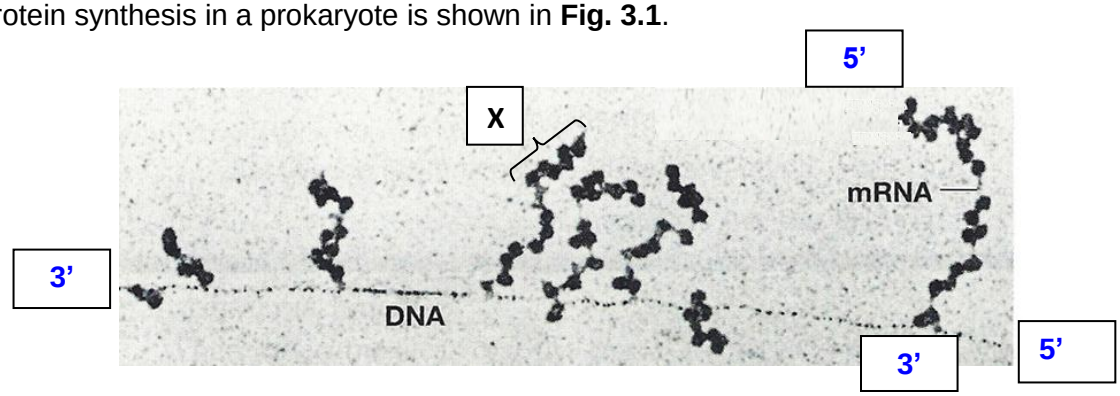


Fig. 3.1

(a) **(i)** Using the boxes provided, label the 5' and 3' ends of the mRNA and DNA template strand on **Fig. 3.1**. [1]

(ii) Identify **X** on **Fig 3.1**. [1]

X: polyribosomes / polysomes

(b) **(i)** State the main difference between the processes shown in **Fig. 3.1** and that occurring in eukaryotes.

……...

……. [1]

Translation of one mRNA strand can **occur simultaneously** with **transcription** of the same strand in prokaryotes but **not** in eukaryotes.

Also accept:

Location (nucleus vs nucleoid) and post-transcriptional modification (euk and not in the prokaryote)

(ii) Explain your answer.

For eukaryotes:

1. **Eukaryotic DNA** contains **introns** which need to be removed before translation (but prokaryotes do not) / **exons** need to be **spliced before translation**. (state post-transcriptional modification)

2. **Transcription** in eukaryotes takes place in the **nucleus** while **translation** occurs in the **cytoplasm** / **rough endoplasmic reticulum**

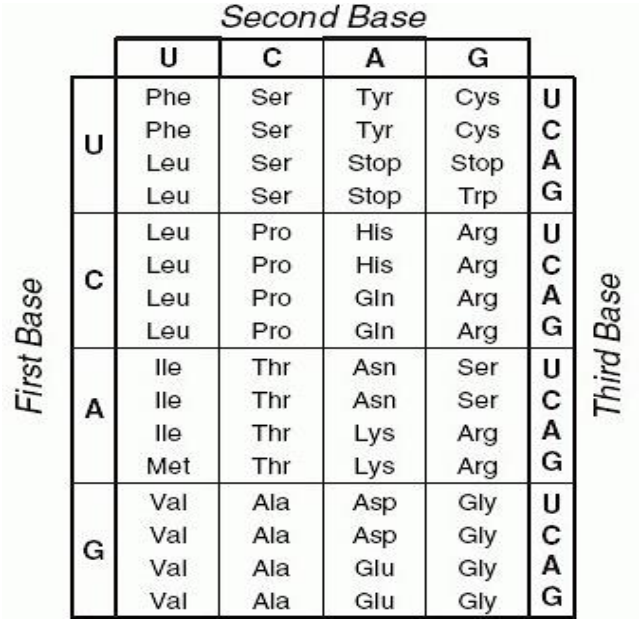
   or
   mRNA has to travel from the **nucleus** to the **ribosomes** in the **cytoplasm** / **rough endoplasmic reticulum** for **translation**.

3. State location of transcription (**Nucleus in Eukaryotes**)

Note: Accept reverse argument from the prokaryotic perspective

(c) A prokaryotic gene was expressed to produce a polypeptide chain. A section of the coding region of the gene, its transcribed mRNA and translated polypeptide are shown in **Table 3.1**.

Complete **Table 3.1** using the information provided in **Fig. 3.2**.



First Base	Second Base U	Second Base C	Second Base A	Second Base G	Third Base
U	Phe	Ser	Tyr	Cys	U
	Phe	Ser	Tyr	Cys	C
	Leu	Ser	Stop	Stop	A
	Leu	Ser	Stop	Trp	G
C	Leu	Pro	His	Arg	U
	Leu	Pro	His	Arg	C
	Leu	Pro	Gln	Arg	A
	Leu	Pro	Gln	Arg	G
A	Ile	Thr	Asn	Ser	U
	Ile	Thr	Asn	Ser	C
	Ile	Thr	Lys	Arg	A
	Met	Thr	Lys	Arg	G
G	Val	Ala	Asp	Gly	U
	Val	Ala	Asp	Gly	C
	Val	Ala	Glu	Gly	A
	Val	Ala	Glu	Gly	G

Fig. 3.2

Table 3.1

[1]

DNA double helix	Template strand (Non-coding strand)	AAG	TTC
	Non-template strand (Coding strand)	TTC	AAG
Transcribed mRNA		UUC	AAG [1]
tRNA anti-codon involved		AAG [1]	UUC
Amino acid incorporated into polypeptide		Phe [1]	Lys

[4]

Feedback:
Most students were able to get full credit. However, there were some students who mixed up the template and non-template strands, or/and, used anti-codon to derive the amino acid.

[Total: 10]

4 X-linked severe combined immunodeficiency (SCID) is an inherited recessive disorder of the immune system that occurs almost exclusively in males. It is caused by mutations in the IL2RG gene. This gene, which resides in the X chromosome, codes for a protein that is important for the growth and maturation of developing immune system cells called lymphocytes. Lymphocytes defend the body against potentially harmful invaders, make antibodies, and help regulate the entire immune system. Mutations in the IL2RG gene prevent these cells from developing and functioning normally. Without functional lymphocytes, the body is unable to fight off infections.

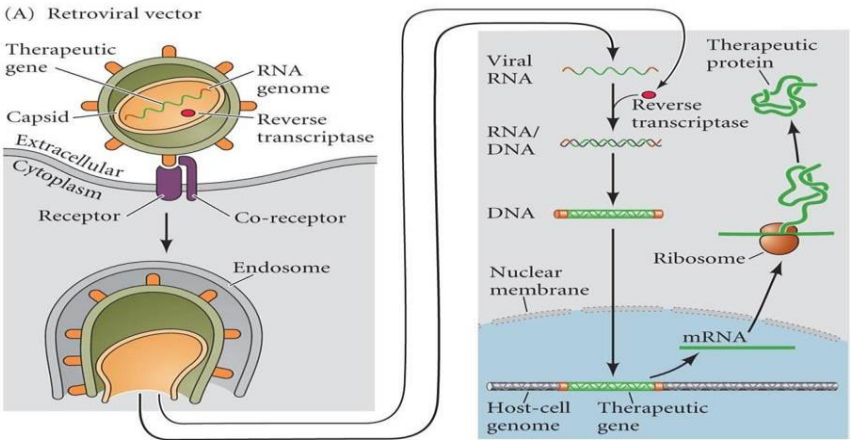
(a) **(i)** Gene therapy can be used to treat SCID. This approach makes use of a vector such as a retrovirus, containing the normal copy of the gene, to introduce the normal copy of the gene into ***blood stem cells*** that are derived from the patient. The cells are then re-introduced back into the patient.

Using your biological knowledge, explain how gene therapy using ***retroviruses*** may lead to long-term treatment of the disease. [3]

1. **Retrovirus** is able to **integrate the normal copy of the gene into the chromosome**.

2. As **stem cells** are able to undergo **long term self-renewal by mitotic cell divisions** to **maintain a constant pool of stem cells**, during which the **normal copy of the gene** can be replicated and passed to the **daughter cells**.

3. When the blood stem cells **differentiate into the lymphocytes**, the normal gene could be **expressed and result in the normal functional protein being produced**.



Feedback:

- A significant number of students did not realize that retrovirus is able to insert the gene of interest (normal copy of the gene) into chromosome of the patient's cell. Furthermore, the use of blood stem cells allow for the maintenance of a constant pool of cells even as some of the stem cells differentiate into the specialized lymphocytes.

  Instead, many students described the reproductive cycle of retrovirus which is not the focus of the question requirement.

- Students are also reminded that they should mention "normal copy" of the IL2RG gene or "normal allele" of the gene instead of just "normal gene".

(ii) Suggest why this approach is unlikely to work for diseases such as cancer. [2]

1. Cancer is caused by the accumulation of **mutations of multiple/different genes**. [1]
2. For this approach to work, **every normal copy of the different genes** must be introduced **successfully** into the target cells which is challenging. [1]
3. Furthermore, gain-of-function gene (e.g. ras gene) mutations are dominant. For this approach to work, the **mutant dominant allele / copy must be removed** from the target cells and replaced by the **2 copies of the (normal) recessive alleles.**

Note: Point 1 must have. Either Point 2 or 3.

Feedback:

- The question also proved to be challenging to most students as they failed to realize that unlike SCID (which is caused by a *single* gene mutation), cancer is caused by the *mutation of several / different genes*.

It is also important to note that the term "genes" should be mentioned in the answer. It should be *"mutations of different genes"* instead of just "mutations" as the latter may mean in the context of the single gene undergoing multiple mutations.

(b) Telomerase is present in the blood stem cells. This enzyme is responsible for extending the telomeres of the chromosomes. The extension of telomeres is in response to the end replication problem during DNA replication.

(i) Explain how the end replication problem arises during the DNA replication of blood stem cells. [2]

1. DNA polymerase requires a **free 3'OH of a RNA primer/pre-existing DNA strand** to initiate DNA replication
2. However, at the **5' end of the newly synthesized daughter strand / lagging strand**, there is **no existing 3'OH group after the last RNA primer is removed**.

This results in a small section of the template / parent DNA template which is **not replicated** (or **shortening of chromosome** after subsequent rounds of DNA replication)

(ii) **Fig. 4.1** shows a section of the telomeric DNA at one end of a chromosome in the blood stem cell.

Fig. 4.1

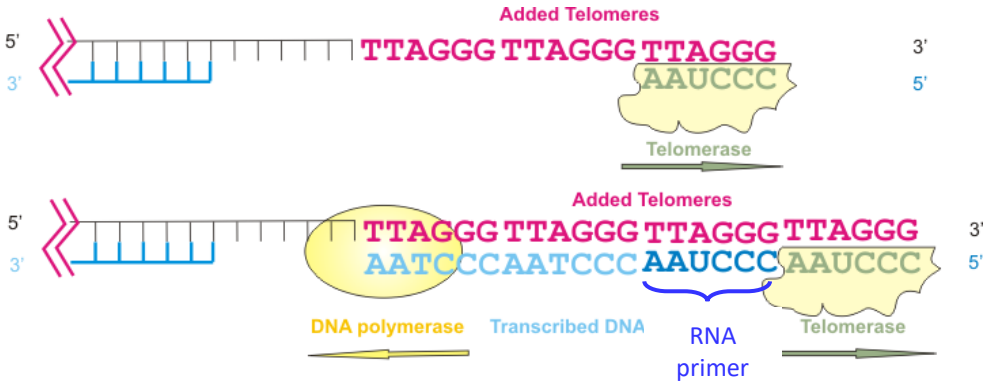
Legend: 5' → 3' DNA strand synthesized by telomerase

5' - - - → 3' DNA strand synthesized by DNA polymerase

Using the legend provided, draw the arrowed lines representing new DNA strands synthesized by telomerase and DNA polymerase onto **Fig. 4.1**. The directionality of the new DNA strands should be correctly shown. [2]

Answer

Additional info below:



(iii) Explain why telomerase is not present in bacterial cells.

……

…… [1]

Bacterial cells / prokaryotes have **circular chromosomes** / their chromosomes are circular. Unlike eukaryotic cells (e.g. blood stem cells), circular chromosomes do not have any **ends**.

(c) Outline one similarity in the characteristic of the DNA sequence that makes up the telomere and centromere.

……

…… [1]

Both are **non-coding DNA** consisting of **tandem repeats**

[Total: 11]

5 Wheat and maize are two of the most important cereal crops in the world. **Fig. 5.1** shows the effect of temperature on the rate of photosynthesis of wheat plants.

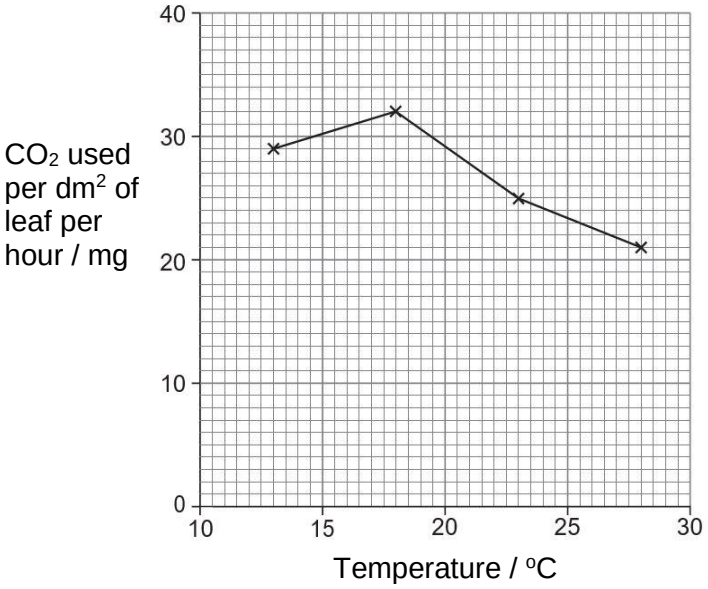


Fig. 5.1

(a) (i) With reference to **Fig. 5.1,** describe the effect of temperature on the rate of photosynthesis of wheat plants. [2]

1. **Quote paired data – As temperature increases from 13^{0}C to 18^{0}C, rate of CO_2 used increased from 29 mg per dm^2 of leaf per hour to 32 mg per dm^2 per leaf per hour; (accept reference to peaking at 18^{0}C with supporting data)**

2. **Quote paired data – As temperature increases from 18^{0}C to 28^{0}C, rate of CO_2 used decreased from 32 mg per dm^2 of leaf per hour to 21 mg per dm^2 per leaf per hour;**

FYI: 1 dm^2 is equivalent to 100cm^2

Comments:
- This part was well-answered.
- Candidates are reminded that for a graph with such clear grid squares, there is NO leeway for carelessness! Candidates should use a ruler to read off the values!
- Some candidates did not include the peak of the graph at 18^{0}C. This is a significant part of the graph. While candidates are not required to describe every point on the graph, significant points and sections need to be described.

(ii) Suggest why temperature affects the rate of photosynthesis in the way you have described in (a)(i). [3]

1. **Inceasing temperature (from 13 °C to 18°C optimum temperature) increased kinetic energy of enzyme and substrate molecules;**
2. **Resulting in an increased frequency of collisions between enzymes and substrates leading to an increase in rate of formation of enzyme-substrate complexes / increase in enzyme activity;**
3. **Beyond 18°C / 18°C - 28°C / optimum temperature, enzymes become (partly) denatured;**

Note:
e.g. Temperature effects on:

a. **rate of light independent reaction / Calvin cycle;**
b. **carbon dioxide availability – initial temperature increases, more carbon dioxide available, leading to faster diffusion rate and rates of photosynthesis;**
c. **stomata closure as temperature rises due to increased transpiration rate which decreases carbon dioxide availability;**

Comments:

- This part was generally well-answered.
- Some candidates referred to the plant's response like closure of stomata leading to less carbon dioxide as temperature increased. While this may be possible, candidates should still make reference to effects of temperature on enzymes.

(b) The conditions in which young plants of wheat and maize are grown affects their ability to photosynthesise at high and low temperatures when they are mature.

Young maize and wheat plants were grown to maturity at high and low temperatures. When they were mature, their rate of photosynthesis was measured at different temperatures.

The results are shown in **Fig. 5.2**.

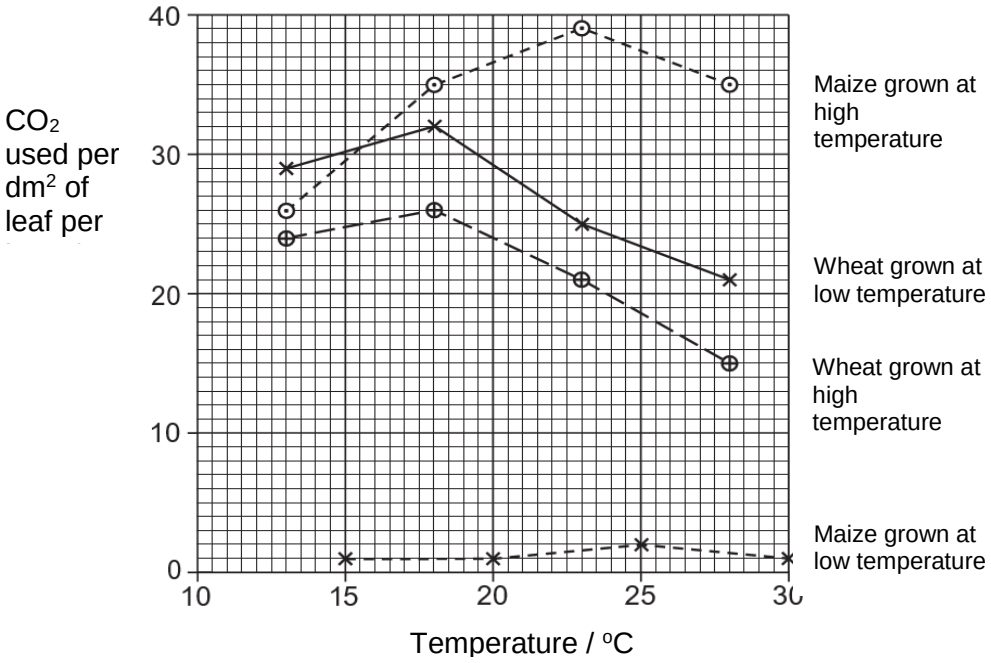


Fig. 5.2

(i) With reference to **Fig. 5.2**, compare one effect of temperature on the rate of photosynthesis of maize plants and wheat plants that were grown at a high temperature when they were young. [2]

Any 1 pair

1a) Maize has greater rate of photosynthesis compared to wheat at all temperatures;

1b) QV: 2 mg per dm^2 of leaf per hour higher (at 13°C) to 20 mg per dm^2 of leaf per hour higher (at 28°C)

2a) Higher optimum temperature for maize compared to that of wheat

2b) QV: Optimum temperature for maize is 23^0C (39 mg per dm^2 of leaf per hour) while optimum temperature for wheat is 18^0C (26 mg per dm^2 of leaf per hour)

3a) Steeper increase for maize as temperature increases to optimum

3b) QV: From 13^0C to 18^0C, CO_2 used for maize was up to 36 mg per dm^2 of leaf per hour compared to up to 45 mg per dm^2 of leaf per hour for wheat;

Comments:
- This part was not always well-answered.
- Candidates should be aware that when comparing photosynthetic rates between the two plants, they should compare the photosynthetic rates at the SAME temperature range for both maize and wheat.
- Candidates should write a summary statement of the trends they observed from the graphs, followed by numerical data to support their claim.
- Some candidates compared the photosynthetic rates of maize and wheat grown at both high and low temperature. This was not what was required by the question.

(ii) Low temperatures slow down the formation of the membranes inside chloroplasts in maize leaves, but not in wheat leaves.

Use this information to suggest a possible reason for the different results for maize and wheat grown at low temperatures as shown in **Fig. 5.2**. [2]

1. **Thylakoid membranes / chloroplast membranes, are needed for light dependent reactions;**

2. **Less membranes in maize resulted in less chlorophyll to absorb light / less surface area exposed to light;**

   Hence very little photosynthesis occurring in maize grown at low temperatures.

AVP

Comments:
- This questions was not always well-answered.
- Many answers did not elaborate further on the significance of the thylakoid / chloroplast membranes on photosynthesis like placement of photosystems with chlorophyll for absorption of light etc.

[Total: 9]

6 A group of Eunoians discovered a new species of forest cockroach (*Ectobius eunoiahall*) beside their campus. Female cockroaches of this species are homogametic and the species follows the XY sex determination system.

Upon analysis of the samples they had collected, it was observed that there are three different eye colours in the species. These eye colours are determined by two genes, Gene B and Gene R, which are found on different chromosomes. One of the genes is present on the X chromosome while the other is found on an autosome. **Fig 6.1** shows the formation of eye pigment in this forest cockroach species.

Blue pigment $\xrightarrow{\text{Enzyme B}}$ Purple pigment $\xrightarrow{\text{Enzyme R}}$ Red pigment

Fig 6.1

The dominant B allele converts a blue pigment into a purple pigment while the dominant R allele converts the purple pigment into a red pigment.

(a) **(i)** State the pattern of inheritance of the eye colour in *Ectobius eunoiahall*.

…….. [1]

(Recessive) **Epistasis**

(ii) A true-breeding blue-eyed male was crossed to a true-breeding purple-eye female. All of the F1 offspring from this cross had red eyes.

Deduce the gene that is located on the X chromosome.

…….. [1]

Gene B

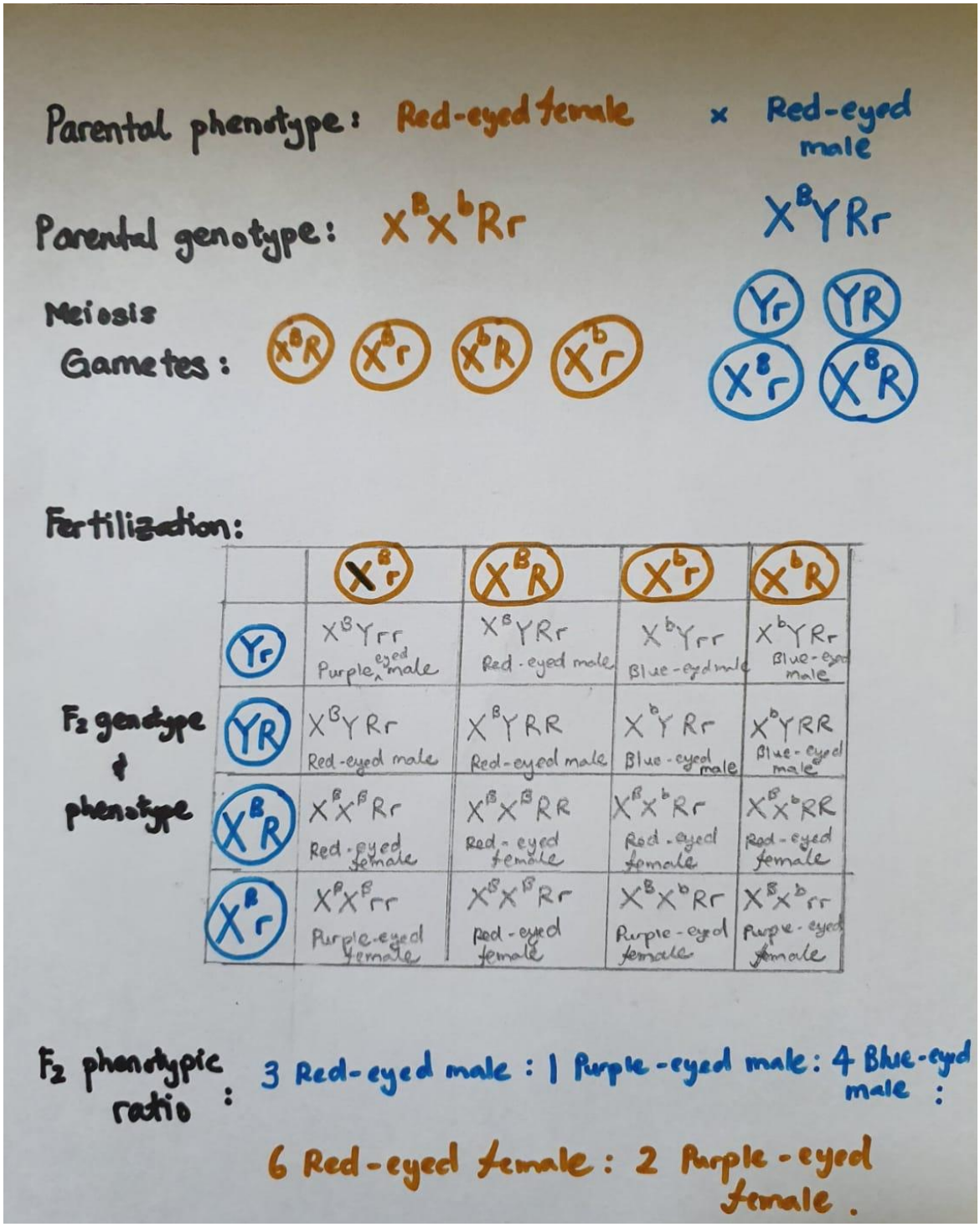
If Gene B is located on X chromosome,

	♀		♂
Parental phenotype	**Purple-eyed female**	x	**Blue-eyed male**
Parental genotype	$X^B X^B r r$		$X^b Y RR$
Meiosis			
Gametes	$X^B r$		$X^b R$ YR
Fertilization			
F₁ genotype	$X^B X^b Rr$		$X^B Y Rr$
F₁ phenotype	Red eyed female		Red eyed male

If Gene R is located on X chromosome,

	♀		♂
Parental phenotype	**Purple-eyed female**	x	**Blue-eyed male**
Parental genotype	$X^r X^r B B$		$X^R Y b b$
Meiosis			
Gametes	$X^r B$		$X^R b$ Yb
Fertilization			
F₁ genotype	$X^R X^r Bb$		$X^r Y Bb$
F₁ phenotype	Red eyed female		Purple-eyed male

(iii) Draw a genetic diagram showing the expected ratio of phenotypes when the F1 progeny are crossed. [4]



Correct parental phenotypes & genotypes [1]
Correct gametes [1]
Punnett square drawn correctly [1]
Correct F2 phenotypic ratio shown [1]

(b) Another distant species of *Ectobius* also has three eye colours but the expression of eye color in this species of *Ectobius* is only controlled by one gene C found on an autosome.

It was found that a cross between true-breeding blue-eyed and red-eyed individuals produced all purple-eyed F1 progeny.

If the purple-eyed F1 progeny are mated with each other, deduce the expected F2 phenotypic ratio.

……..

…… [1]

1 blue : 2 purple : 1 red (~~multiple alleles &~~ Incomplete dominance)

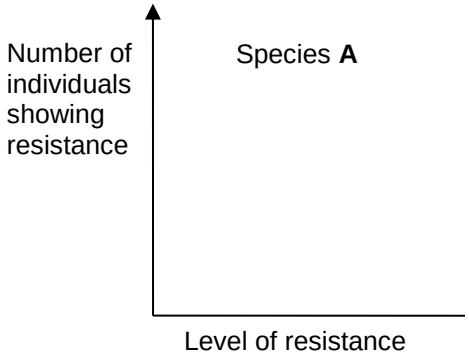
Ganison : Cannot be multiple alleles for this answer

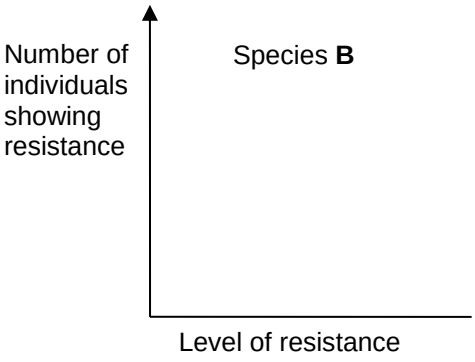
(c) Weeds and weed control cost farmers worldwide millions of dollars, each year. Herbicides have been used for a long time to control weed populations. Weeds have diverse genetic backgrounds that give them the ability to adapt to many different environments. The first reports of weeds resistant to herbicides came out in 1968. By 1991,120 weed biotypes that were resistant to triazine herbicides and 15 other herbicide families were documented across the globe. Herbicides work by interacting with the enzymes or proteins essential to the weeds' growth and development.

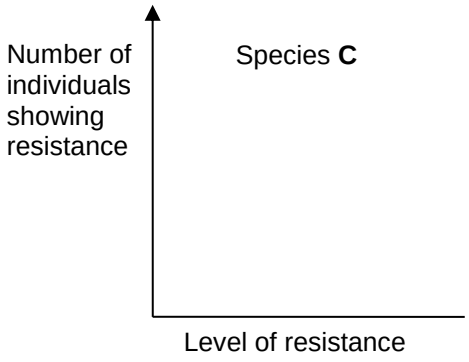
There are three different species, A, B and C, and their genetic basis for resistance are indicated in the table below.

Species **A**	Continuous pattern of inheritance controlled by multiple genes.
Species **B**	Discontinuous pattern of inheritance controlled by a single gene with alleles that have a dominant-recessive relationship.
Species **C**	Discontinuous pattern of inheritance controlled by a single gene with alleles that have a co-dominant relationship.

Complete the graphs below to show the number of individuals that exhibit various levels of resistance. [3]

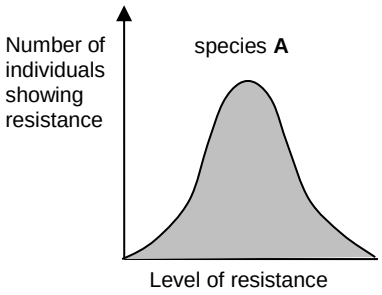


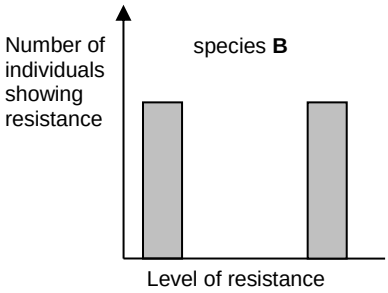


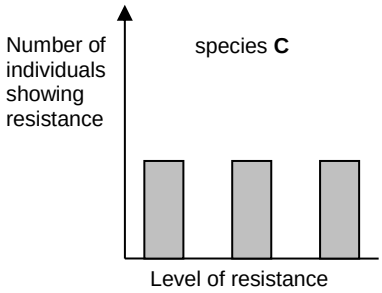


[Total: 10]

Answer:







For Examiner's Use

7 A study was carried out to examine the effectiveness of bacteriophages in treating Colibacillosis, a fatal condition caused by *E. coli.*

In the study, broiler chickens were first subjected to an aerosol spray containing bacteriophages on day 0. They were then separated into five treatment groups. Each treatment group was subsequently injected with *E. coli* on days 0, 1, 2, 3 and 4 respectively. The mortality rate for each treatment group was determined after 21 days.

The results of the study is represented in **Fig 7.1**.

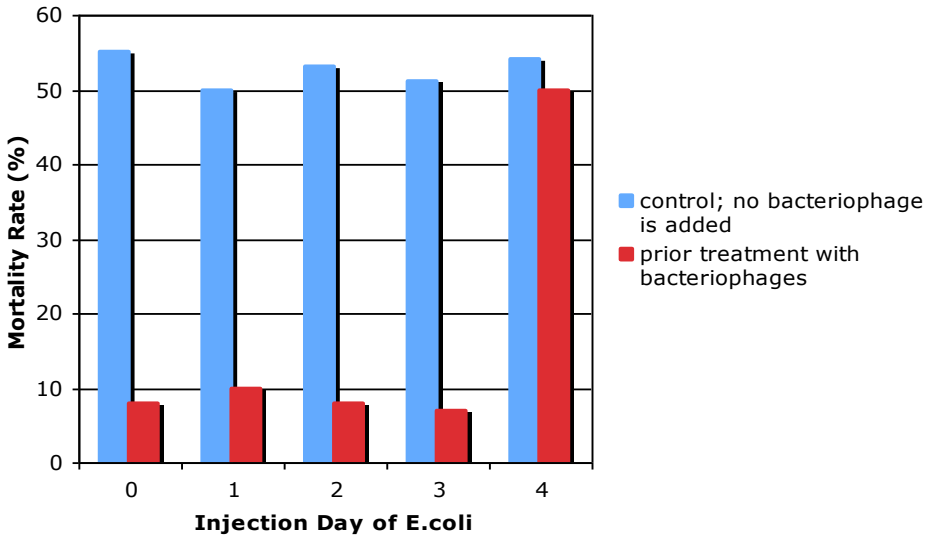


Fig 7.1

(a) **(i)** With reference to **Fig. 7.1**, compare the general trends observed in the control groups and the groups that have been treated with bacteriophages.

……

……

……

…… [2]

*Must quote data

1. For **days 0 – 3**, the mortality rate for the **control** was **consistently higher than the treatment groups**. Mortality rates for the **control groups** were at about **50-55% as compared to the treated groups (about 6-10%)**

2. However, for **Day 4**, there was a spike in mortality rate for the **treated chicken** to **50%**, which was comparable to that of **control** which was **53-54%**%.

(ii) Suggest how the results from this study would help the farmer effectively manage the disease. [1]

1. To effectively curb the spread of the disease, the farmer **must apply a new dosage every 3 days** as the treatment is **only effective for 3 days**. WTTE.

(iii) Suggest why the use of bacteriophages is a better alternative to antibiotic therapy for the chickens.

……..

……. [1]

Any 1

1. Bacteriophages do not infect eukaryotic cells as they are host-specific. Hence, lesser side-effects to humans.
2. Bacteriophages will mutate alongside with the bacteria for long-term effectiveness (co-evolution)
3. *E.coli* may evolve resistance to the antibiotics over time.

(b) A person is infected with the H1N1 influenza virus but does not develop the symptoms. Medical tests show that the person's immune system has produced antibodies that binds to the H1 haemagglutinin molecules.

(i) Explain why the person does not develop influenza-like symptoms.

……..

……..

……..

……. [2]

1. The antibodies bind to haemagglutinin preventing them from recognizing and binding to (complementary) antigens/cell surface receptors (sialic acid) on epithelial cells. Thus, the virus is neutralized and is unable to enter the cells to cause an infection.
2. Antibody-coated viruses are readily engulfed and destroyed by phagocytes through opsonisation before an infection can take place.

(ii) After several months, the person begins to develop influenza symptoms. Tests for the antibody showed that it is still present in the blood.

Account for the observation. [2]

1. After many viral generations, the virus would **accumulated mutations via antigenic drift;**
2. Reason: **Lack of proofreading by the viral RNA-dependent RNA polymerase**
3. Influenza virus now encodes a structurally different haemagglutinin / surface antigen that is not complementary to the antibody.

Note: Point 1 + point 2 or 3

(iii) Comment on the implication of the observation on the development of H1N1 influenza vaccines. [1]

Any vaccine produced may soon **lose its effectiveness** due to the rapidly changing phenotype of the virus. Hence, there is a need to design **new vaccines on a seasonal/regular basis.**

[Total: 9]

8 **Fig. 8.1** shows the *arg* operon in *E. coli*, which is a repressible operon with three genes, *arg B, arg C* and *arg H*, that code for enzymes responsible for the biosynthesis of the amino acid arginine.

Another gene upstream of the operon (not shown in **Fig. 8.1**) codes for a repressor protein R, which binds to the operator to regulate the operon. Arginine acts as a corepressor, which activates repressor *R*.

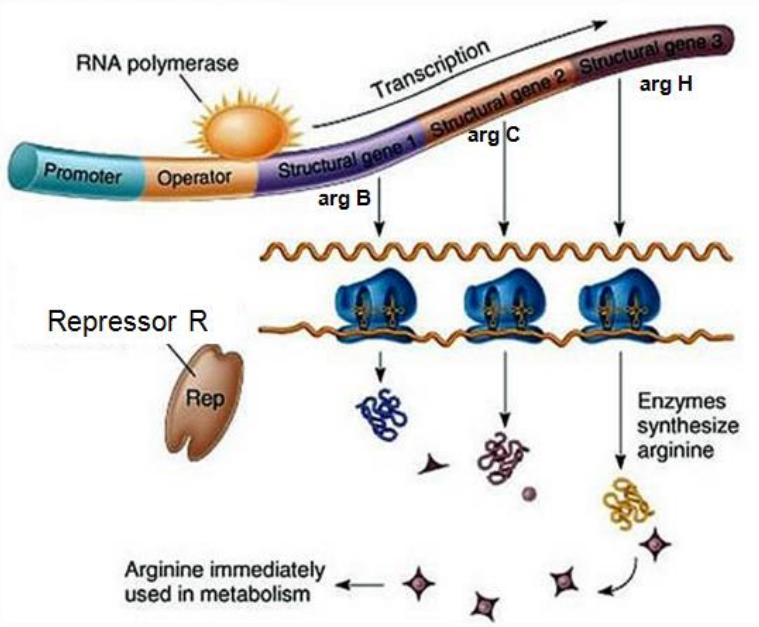


Fig 8.1

(a) Distinguish between a structural gene and a regulatory gene in the *arg* operon.

……

……

……

…… [2]

1. A structural gene (is a region of DNA that) **codes for a protein / enzyme** that **synthesises arginine, e.g. arg B / arg C / arg H**;

2. A regulatory gene codes for a specific protein product, repressor R, that **regulates the expression of the structural genes** of the **arg operon**

Feedback:

- Students should answer the question in the context of arg operon instead of providing a generic answer.
- Common error – regulatory genes refer to genes that code for regulatory proteins (e.g. repressor and activator) that bind to regulatory sequences (e.g. promoter, operator, etc). Many students mixed up the terms: regulatory genes vs regulatory sequences
- This mode of control is "end product repression" (refer to Bacteria lecture notes p32) and NOT "end product inhibition" or "feedback inhibition" of a metabolic pathway (refer to Enzyme lecture notes p31).

(b) Based on your understanding of a repressible system, explain how arginine is able to control the level of enzymes involved in arginine synthesis to ensure the conservation of resources by *E. coli*.

……

……

……

……

……

……

……

……

…… [4]

1. When **arginine is present**, it acts as an effector molecule/**co-repressor that binds to the allosteric site of repressor R**

2. Results in the **change** in 3D **conformation of repressor R** into its **active** state / change into the active conformation of repressor R

3. **Repressor R binds to the operator**, **RNA polymerase is unable to bind to the promoter**

4. As arg B, arg C and arg H genes are not transcribed / the operon is switched off, **enzymes responsible for the synthesis of arginine are not synthesized in the presence of excess of arginine**

Feedback:

- Students need to be more careful in reading the question. The three genes, *arg B, arg C* and *arg H* of the arg operon codes for enzymes involved in the synthesis of arginine, an amino acid. Many students erroneously stated that the genes code for arginine protein which is not the case.
- Many students also discussed the involvement of general transcription factors and the formation of transcription initiation complex in their answer. Do note that in prokaryotes, there are no general transcription factors involved.

These promoter regions the eukaryotes and prokaryotes are recognised and bound to by proteins which will recruit and facilitate the binding of RNA polymerase to the promoter. In prokaryotes, the Pribnow box is recognised by sigma factor while the TATA box in eukaryotes is recognised by the TATA binding Protein (TFIID, which stands for Transcription Factor II D). There other proteins which are involved in facilitating transcription.

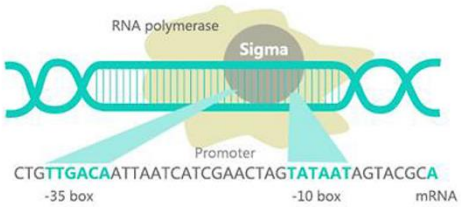


Figure 31. Prokaryotic Transcription Initiation.

Initiation of transcription in the *prokaryotic* gene involves RNA polymerase (and its bound sigma factor) binding to the promoter

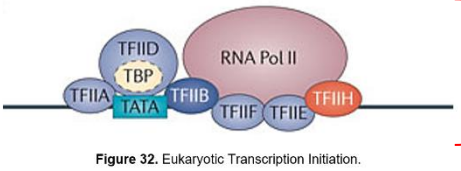


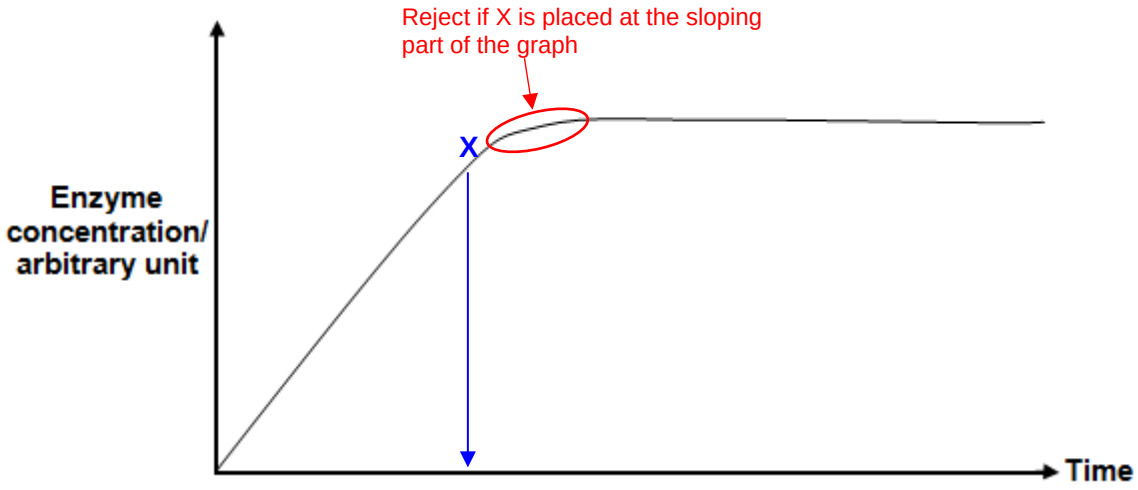
Figure 32. Eukaryotic Transcription Initiation.

Formation of transcription initiation complex is required for the initiation of transcription of *eukaryotic* gene

Source of image: DNA & Genomics lecture notes p28 + refer to Organization and Control lecture notes (Control notes p25)

(c) *E. coli* was cultured in a growth medium with all the essential nutrients needed except the amino acid arginine. **Fig. 8.2** shows a graph of the concentration of enzymes that are involved in the synthesis of arginine against time. At time X, arginine was added to the growth medium.

Using an arrow, indicate on **Fig. 8.2**, the time when arginine was added. [1]



Feedback:

- Since the question stated that arginine was added at time X, we assume that there was no arginine in bacterial culture. As such, the enzyme concentration should continue to increase linearly and not at the (circled) region where the increase in enzyme concentration is slowing down.
- As question stated "time" in which arginine was added, students are advised to be more specific in their answer by including the arrow pointing down to a spot on the x-axis (time) instead of placing at arrow pointing at the drawn graph.

(d) Besides having operons, prokaryotes have other means to adapt to changing environments through gene transfer.

Describe how viruses can facilitate the transfer of advantageous alleles in a bacterial population.

……

……

……

……

……

……

……

…… [4]

Either one:

Generalized transduction	Specialized transduction
1. Phages undergoing **lytic life cycle** can facilitate **generalised transduction**;	1. Phages undergoing **lysogenic life cycle** can facilitate **specialized transduction;**
2. During the lytic cycle, **bacteria DNA is hydrolysed into fragments**;	2. During the lysogenic cycle, viral DNA is **integrated into bacterial chromosome** forming a **prophage** which may be **improperly excised** to **include adjacent segment of bacterial DNA** during an **induction event**
3. Errors may occur in the **assembly stage** when **advantageous alleles of bacteria inside the fragments are randomly/mistakenly packaged into new capsid head/phage**	3. The excised **bacterial DNA including advantageous alleles may be packaged into a capsid head / phage**
4. Phage released can infect another bacterium, introducing the **advantageous allele into the bacterial chromosome via homologous recombination / integration**;	4. Phage released can infect another bacterium, introducing the **advantageous allele into the bacterial chromosome via homologous recombination / integration;**

Note: Point 3 and 4 can only be awarded once

Feedback:
Although the answer scheme allows for either approach (i.e. generalized transduction or specialized transduction), many students attempted to answer using both approach. Unfortunately, points from both approach were often lumped together and the answers were poorly organized or/and phrased. These made the answer hard to understand. Illegible writings of some students further aggravated the situation.

Students need to be aware that they are communicating to their answers in the written form, and hence they need to write clearly and to spell biological terms (e.g. homologous recombination, etc) properly despite time constraint. Coherence of answer trumps the amount of information presented.

[Total: 11]

9 Huntington's disease (HD) is an inherited disorder that results in the death of brain cells. The earliest symptoms are often subtle problems with mood or mental abilities. A general lack of coordination and an unsteady gait often follow. As the disease advances, uncoordinated, jerky body movements become more apparent. Physical abilities gradually worsen until coordinated movement becomes difficult and the person is unable to talk. Mental abilities generally decline into dementia. The symptoms usually begin between 30 and 50 years of age.

The disease is caused by an autosomal dominant mutation in a gene called *Huntingtin* which codes for the protein Huntingtin (HTT).

Part of this gene is a repeated section called a trinucleotide (CAG) repeat, which varies in length between individuals. When the length of this repeated section exceeds a normal range, it produces an altered form of the protein that results in the disease.

Table 9.1 shows the disease status of the individual based on the number of repeats.

Table 9.1

Repeat count	Disease status
Less than 36	Will not be affected
36 – 39	May or may not be affected
More than 39	Will be affected

Fig. 9.1 shows a family who is afflicted by this disease. The father (**B**) and his eldest son (**C**) suffer from this disease but not the mother (**A**). However, the disease status of the daughters (**D** and **E**), being younger than age 30, are unknown. To determine their disease status, the two copies of the gene were isolated and the relevant part of the gene was amplified by PCR.

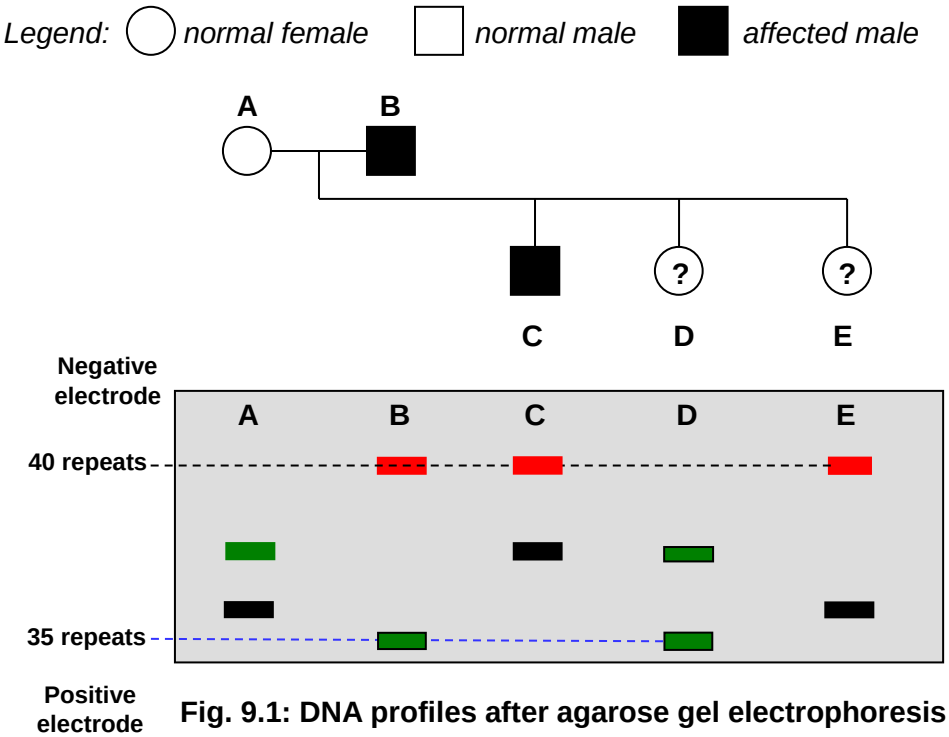


Fig. 9.1: DNA profiles after agarose gel electrophoresis

(a) **(i)** With reference to **Fig. 9.1**, state whether it is possible to *conclusively* determine the disease status of the daughters **D** and **E**. [1]

Daughter **D**: **Yes** (Will not be affected because mother carries normal allele and is inherited by D).
Daughter **E**: **Yes** (Will be affected)

(ii) Explain your answer.

Daughter **D**: [2]

1. Daughter D's **larger band** corresponds to the **larger / upper band** of the **unaffected mother** (**A**)
2. Which is the **normal allele** even though it may consist of **36-39 repeats**

Daughter **E**: [2]

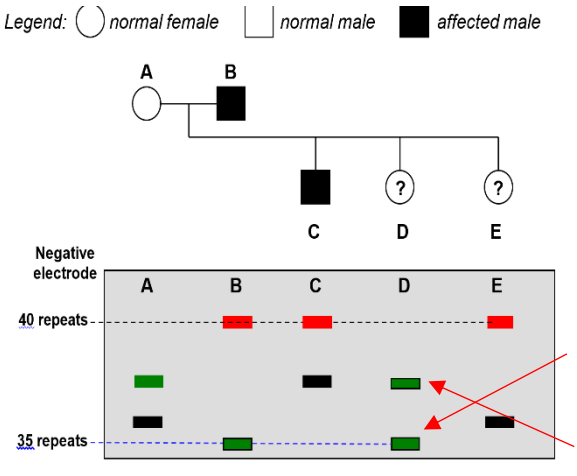
3. **Larger / upper band** corresponds to the **size of the larger band of the affected brother (C) / father (B) which consists of 40 repeats.**
4. As the disease is an **autosomal dominant condition**, **one mutated copy** of the gene is sufficient to give rise to the disease.

Feedback for (a)(i) and (ii):

2 copies of the gene are required to give rise to the normal phenotype

Normal allele (recessive allele)	No. of CAG repeats: 35 or less
Normal or mutant allele	No. of CAG repeats: 36 - 39
Mutant allele (dominant allele)	No. of CAG repeats: 40 or more

One copy of the gene is sufficient to give rise to the diseased phenotype



For daughter D, most students stated it was *not possible* to determine the disease state since the total no. of repeats in her upper band is between 35 and 40.

They failed to see that the band actually corresponded to her mother (A)'s upper band while the lower band corresponded to her father (B)'s lower band (35 repeats).

The band with *35 repeats was derived from a normal allele and must be inherited from the affected father*, while the band with *36-39 repeats must be inherited from the unaffected, normal mother.*

To describe the outcome of the electrophoresis gel (or X-ray film if it was southern blotting), students are advised to describe in terms of "bands" instead of "alleles".

(b) It was discovered that another RFLP marker exists near to the *Huntingtin* gene. **Fig 9.2** shows the position of the RFLP marker relative to the *Huntingtin* gene, and the location in which a radioactive probe binds to.

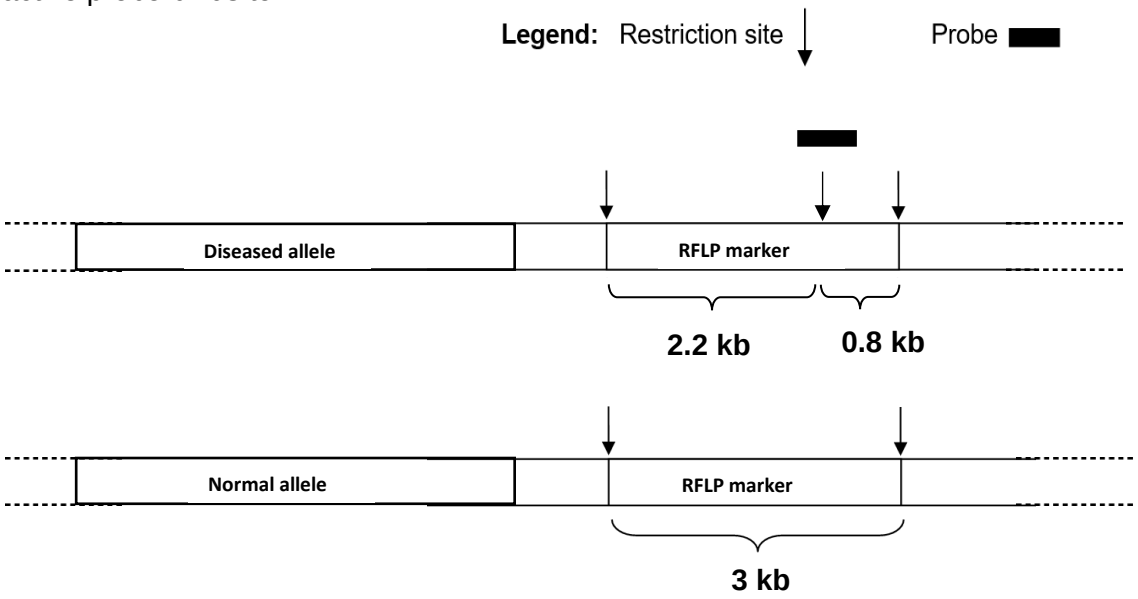


Fig 9.2

Note: 1kb refers to 1000 base pairs (bp)

An RFLP analysis of both parents were carried out and the results are shown in **Fig. 9.3** below.

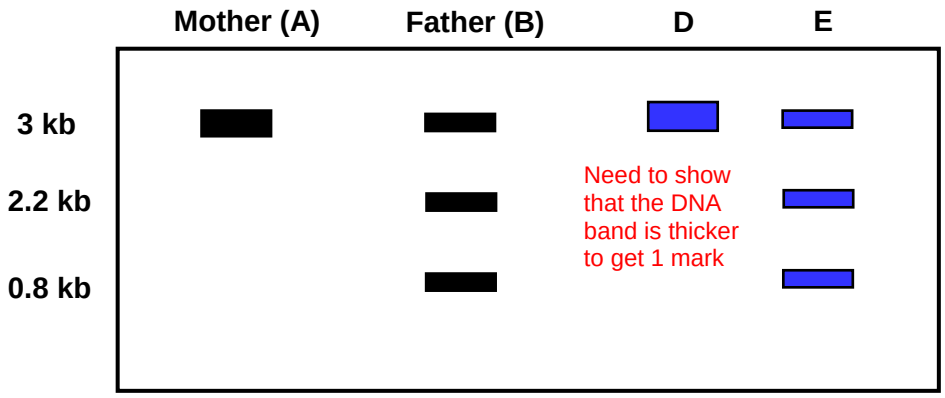


Fig 9.3

(i) With reference to the information provided in **Fig. 9.3**, as well as **Fig. 9.1** and **9.2,** draw the expected band pattern of daughter **D** and **E** in the space provided in **Fig. 9.3**. Assume same amount of DNA was extracted from each individual. [3]

1 mark for correct no. and relative position of bands in D and E [2]
1 mark for correct relative thickness of band [1] *(since same amount of DNA was extracted from each individual as mentioned by the question)*

(ii) If the location of this RFLP marker is relatively far away from the *Huntingtin* gene, predict if it is a reliable marker for Huntington's disease. Explain your answer. [2]

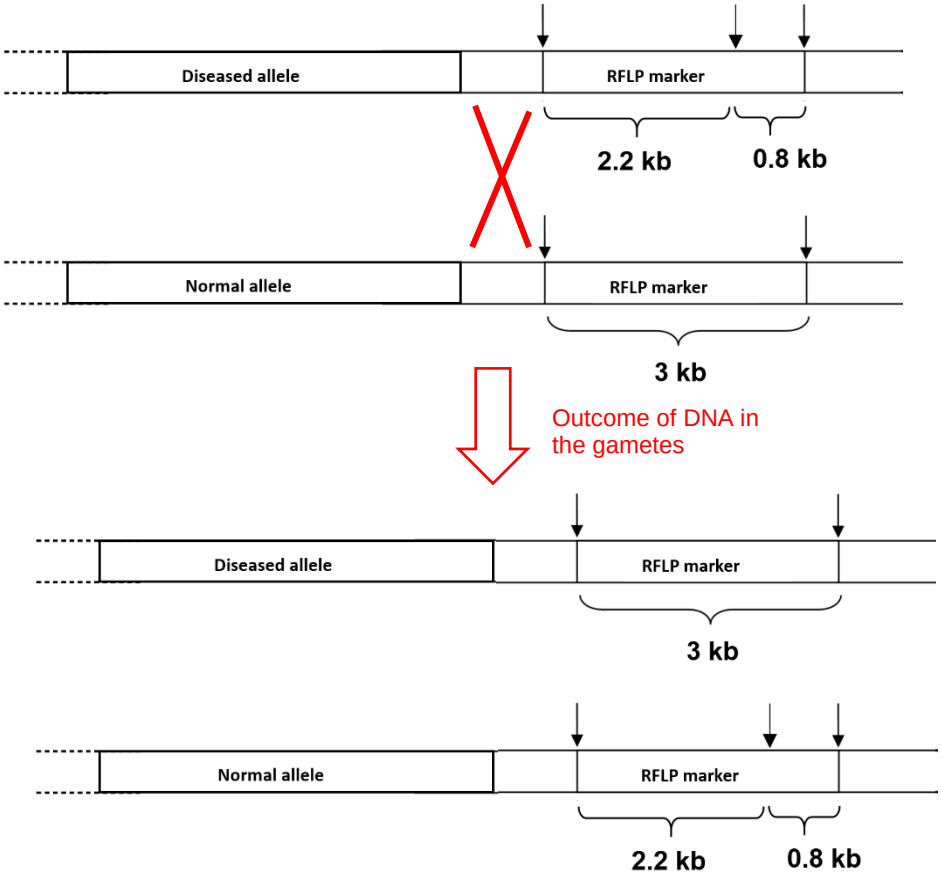
1. **Not reliable**. During the formation of gametes in the parents, **crossing over** between the **gene and RFLP marker** may occur in **prophase I of meiosis**. [1]

2. Result: The **disease allele is now linked to the RFLP marker** which gives rise to the **3kb band** **(instead of 2.2kb and 0.8kb**). Hence, the **result will be negative** even though the individual **actually suffers from the disease.** [1]

Accept reverse argument.

Feedback:

- Almost everyone could not answer this question correctly ☹
  The identification of the normal allele is based on a single 3kb band while the identification of the diseased/mutant allele is based on 2 bands (2.2kb and 0.8kb). However in the parent's sex cells, crossing over during prophase I could occur as seen below.



Assuming a child inherited the normal allele from the parent, the child will nevertheless shows 2 bands (2.2kb and 0.8kb) which will result in an inaccurate diagnosis! Hence, it is important for the RFLP marker to be closely/tightly linked to the gene of interest for it to be a reliable marker/indicator of the disease.

[Total: 10]

10 The *Archaeopteryx* lived in the late Jurassic Period around 150–145 million years ago. It could grow to about 0.5 meters in length.

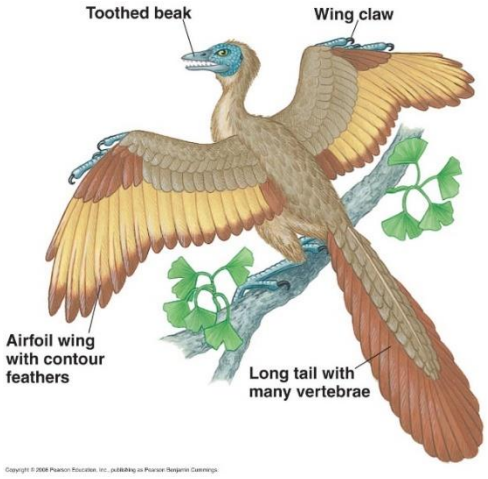


Fig. 10.1

Despite its small size, broad wings, and inferred ability to fly or glide, *Archaeopteryx* had more in common with small theropod dinosaurs than it did with modern birds. These theropod-like features included, jaws with sharp teeth, three fingers with claws, a long bony tail, hyperextensible second toes ("killing claw"), feathers and various skeletal features.

These make *Archaeopteryx* a clear candidate as a transitional fossil between dinosaurs and birds. Thus, *Archaeopteryx* plays an important role not only in the study of the origin of birds but also in the study of dinosaurs.

The first complete specimen of the *Archaeopteryx* was announced in 1861, only two years after Charles Darwin published his paper on the 'On the Origin of Species'. Thus, this became a key piece of evidence in support of the idea of descent with modifications from ancestral species. Over the years, nine more fossils of *Archaeopteryx* have surfaced. Despite variation among these fossils, most experts regard all the remains that have been discovered to be belonging to a single species.

(a) **(i)** Explain what is meant by 'transitional fossils'. [2]

Any 2

1. **Fossilized remains of intermediary forms of life; WTTE**
2. **that illustrate an evolutionary transition of descent of one (group of) species from an ancestral (group of) species; WTTE**

3. **Transitional fossils exhibit some traits like their ancestors and others like their descendants; Or**
   They can be identified by their retention of certain primitive/ancestral traits in comparison with their descendent relatives, who may have shared derived traits.

(ii) List two ancestral characters that are found in both theropods and modern birds. [2]

1. **Jaws/ rej. toothed jaws**
2. **three-toed foot, with claw**
3. **feathers**
4. **bony tail rej. Long bony tail**
5. **hollow bones**
6. **brooding of the eggs.**
7. **a furcula (wishbone),**

(iii) What does the presence of feathers suggest about theropods? [2]

1. **They may be warm blooded/homotherms/able to control their own body temperature**
2. **They <u>glide</u> short distances *(rej. fly)***

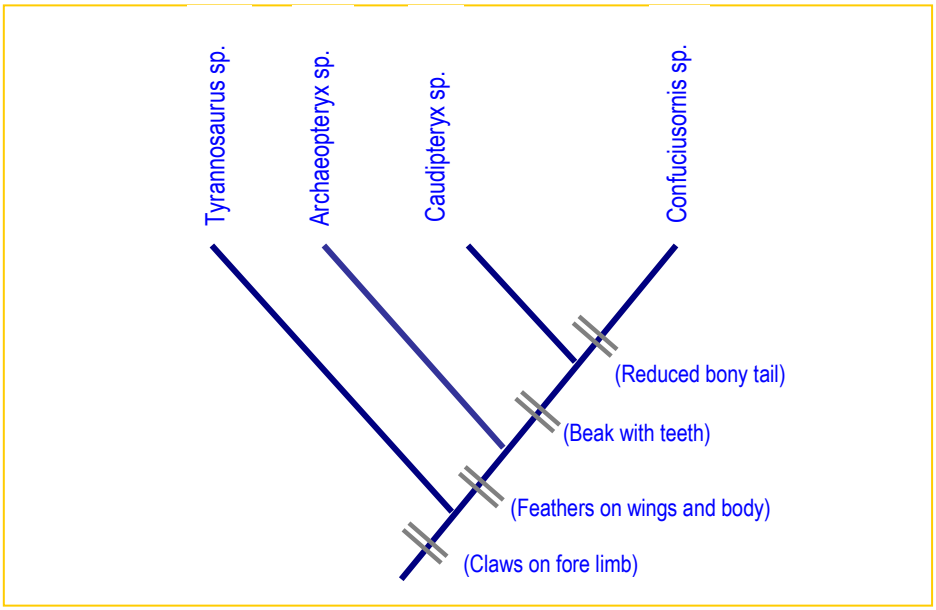
(b) *Archaeopteryx lithographica, Caudipteryx zoul* and *Confuciusornis sanctus* are thought to be missing links between dinosaurs and birds. They share many dinosaurian features and have wings and feathers. **Table 10.1** illustrates the characters observed in these species in a particular investigation by a group of paleontologists.

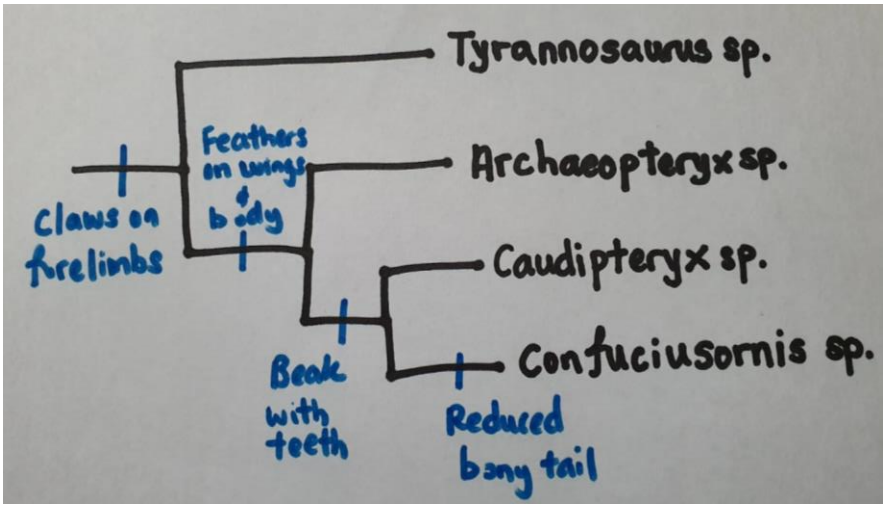
Character	*Tyrannosaurus sp.*	*Archaeopteryx sp.*	*Caudipteryx sp.*	*Confuciusornis sp.*
Claws on fore limb	1	1	1	1
Feathers on wings and body	0	1	1	1
Beak with well-developed teeth	0	0	1	1
reduced bony tail	0	0	0	1

<u>Character table</u>.
0 - Indicates that a character is absent
1 - Indicates that a character is present

Table 10.1

Construct a phylogenetic tree to illustrate their evolutionary relationships between these species in the space provided. On the tree drawn, indicate when the characters, shown in **Table 10.1**, arose. [2]





Relationship in correct sequence = 1
Correct indication of characters = 1

(c) A number of modern birds today are not capable of flight. The Emperor Penguin which lives in the Antarctic is an example of a flightless bird.

A fossil of an ancestral penguin species, *Kumimanu biceae*, was recently discovered from bones packed in a rock on a New Zealand beach. It is among the earliest known penguin species and it probably at stood 5 feet 7 inches tall and weighed about 220 pounds.

Despite having similar niches, modern-day Emperor Penguins are not found in the Arctic region. Suggest why this is so.

……

……

……

……

…… [2]

1. **Descent with modification from a common ancestor**;

2. Ancestral penguin species were found in the southern hemisphere/near to Antarctica/New Zealand. Hence, **speciation events** which lead to the arising of new modern penguin species must have taken place in **same biogeographic location**;

   AVP: Penguins are non-migratory birds and therefore they cannot fly from one pole to another (far apart). Penguins are not adapted to fly past the tropics.

[Total: 10]