MERIDIAN JUNIOR COLLEGE
JC2 Preliminary Examinations 2016
Higher 1

CANDIDATE NAME

CIVICS GROUP | 1 | 5 | | | | |

INDEX NUMBER | | |

H1 BIOLOGY

8875/02

Paper 2 Core Paper

16 September 2016
2 hours

Additional Materials: Answer papers

READ THESE INSTRUCTIONS FIRST

Do not open this booklet until you are told to do so.
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 diagrams, graphs or rough working.
Do not use staples, paper clips, highlighters, glue or correction fluid/tape.

Section A
Answer **all** questions in the spaces provided on the question paper.

Section B
Answer **one** question on the answer paper provided.

At the end of the examination,
1. Fasten your answer papers to section B securely together.
2. Hand in the following separately:
   - Section A
   - Section B

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

For examiner's Use	
Section A	
1	/ 11
2	/ 9
3	/ 9
4	/11
Section B	
5 / 6	/ 20
Total	/ 100

ANSWER SCHEME

This paper consists of __ printed pages.

[Turn over]

Section A

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

QUESTION 1

The structure of the tubulin dimer, the protein that forms microtubules by polymerisation, is shown in **Fig. 1.1**.

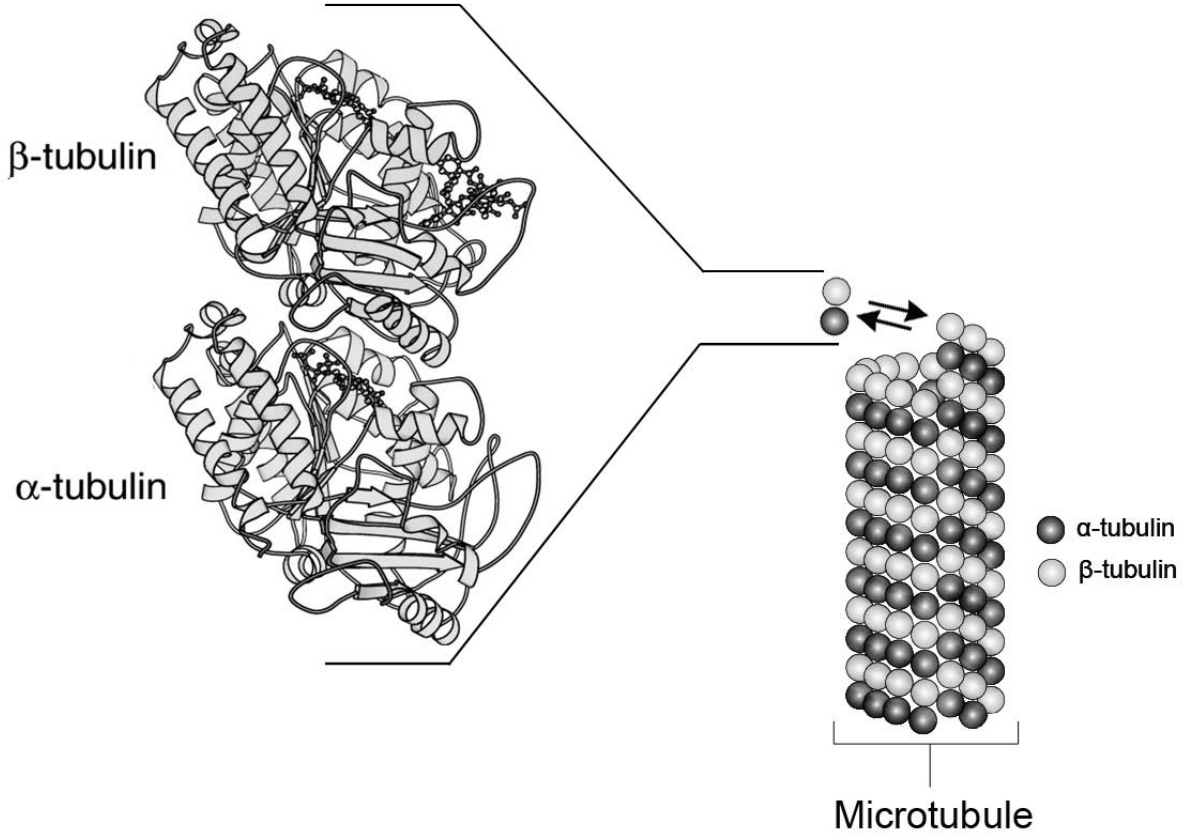

Fig. 1.1

a) With reference to **Fig. 1.1**, describe how tubulin attains its three-dimensional structure. [4]

- Tubulin has a **primary structure** comprised a **specific sequence of amino acids** joined by **peptide bonds**.
- The primary sequence of amino acids determines the **folding** of the polypeptide chain into the **secondary structure** which comprises **α-helices and β-pleated sheets** which is held by **hydrogen bonds** between the C=O and N-H groups of the peptide bond region
- The secondary structure then folds into the **tertiary structure** which comprises a **three-dimensional globular** tubulin subunit that is held by **hydrogen bonds, ionic bonds, disulphide bonds and hydrophobic interactions**.
- The **quaternary structure** is the tubulin dimer, which comprises **two** tubulin subunits held together by **hydrogen bonds, ionic bonds, disulphide bonds and hydrophobic interactions**.

Tubulin inhibitors like paclitaxel and vinblastine have been utilised in chemotherapy drug trials to treat cancers. All tubulin inhibitors are known to bind to the β-tubulin subunit.

b) Explain how tubulin inhibitors reduce tumour formation. [3]

- Tubulin inhibitors **interfere / prevent polymerisation** of tubulin dimers to form microtubules / **spindle fibres** that make up the **mitotic spindle**.
- Without spindle fibres, **chromosomes cannot divide** / mitosis **stops at prophase** / mitosis **cannot take place**.
- Hence affected cells **exit the cell cycle** / **go into G_0** and **uncontrolled cell division is prevented**.

c) Suggest and explain why tumour cells may exhibit resistance to the chemotherapy drugs. [2]

- Different tumour cells exhibit **variation due to random mutation** to certain **genes**.
- Leading to formation of new alleles coding for
  - **transporter proteins** that **pump out** drugs.
  - **Tubulin** with a non-complementary **binding site** where the drug usually binds to.
  - **enzymes** that **hydrolyses** the drug.
  - **inhibitors** that bind to the drugs and render them ineffective.

Fig. 1.2 shows the changes in the number of small vesicles in an animal cell that is undergoing mitosis.

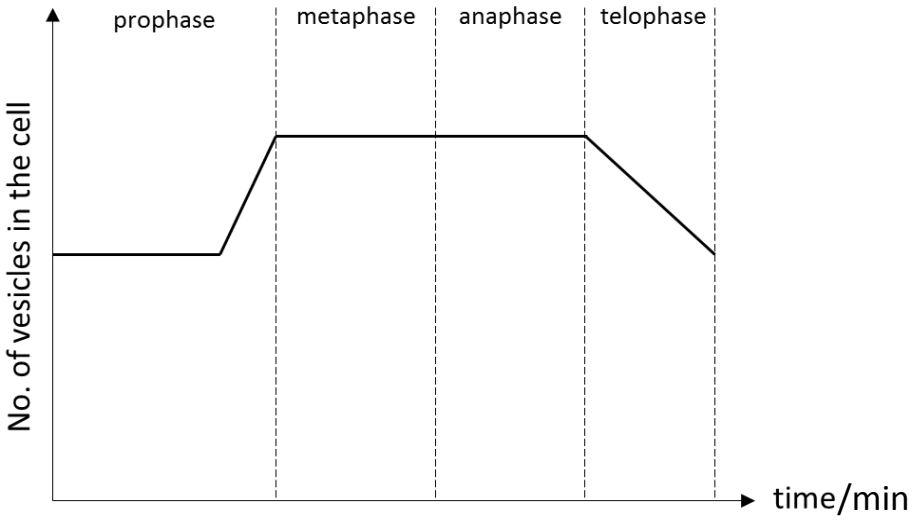

Fig. 1.2

d) Suggest reasons for the changes in the number of vesicles during mitosis. [2]

- During (late) prophase, the **nuclear envelope disintegrates into small vesicles** to allow the access of the microtubules to the kinetochores on the centromeres of chromosomes. This accounts for the increase in vesicles during prophase.
- During telophase, these **small vesicles fuse together** around the chromosomes to **reform the nuclear envelope**. This accounts for the decrease in vesicles during telophase.

[Total: 11]

QUESTION 2

Fig. 2.1 shows the process of translation.

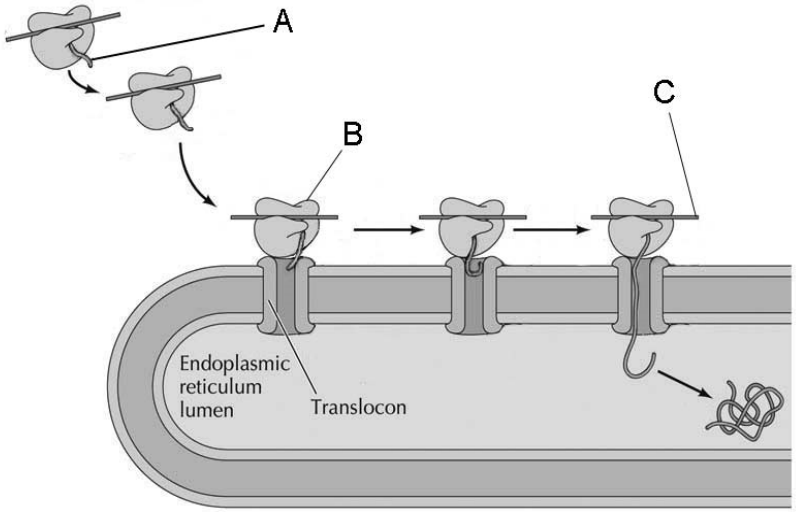

Fig. 2.1

a) (i) Label structures **A**, **B** and **C** [3]

- A – polypeptide chain
- B – small ribosomal subunit (accept ribosome)
- C – mRNA

(ii) Suggest the role of the translocon in protein synthesis. [1]

- The translocon serves as a **hydrophilic channel** to allow the passage of the polypeptide chain into the **lumen** of the endoplasmic reticulum.

b) List two ways in which transcription differs from DNA replication. [2]

	DNA Replication	**Transcription**
Template	Both strands of DNA	Template strand / one of two DNA strands
Raw materials	Deoxyribonucleotides	ribonucleotides
Final product	DNA	**mRNA, rRNA, tRNA**
Enzymes that catalyse the formation of the bonds between monomers	**DNA Polymerase** catalyses the formation of phosphodiester bonds between deoxyribonucleotides	**RNA Polymerase** catalyses the formation of phosphodiester bonds between ribonucleotides
Primers	**RNA** primers required	Primers not involved

c) Explain how complementary base pairing facilitates the storage and transmission of genetic information. [3]

- **[Compulsory for Storage]** Complementary base pairing between bases of the two template strands / parental strands is important as it **stabilises** the **double-stranded helix structure** of DNA for storage of genetic information.

[Transmission] Complementary base pairing enables . . .

- **[Replication]** the **formation of daughter strands during DNA replication** before mitosis in order to transmit genetic information to daughter cells.
- **[Replication]** the **proofreading function of DNA polymerase to find and repair mutations** to maintain genetic fidelity.
- **[Transcription]** the **formation of mRNA during transcription** in order to **transmit information** for the synthesis of **the primary sequence of polypeptides** in protein synthesis.
- **[Translation]** Complementary base pairing **between mRNA codons and tRNA anticodons during translation** transmits information for the primary sequence of polypeptides in protein synthesis.

[Total: 9]

QUESTION 3

Sickle cell anaemia is a genetic disorder caused by the presence of two abnormal copies of the gene which codes for the β-globin chain in haemoglobin. Individuals heterozygous for sickle cell anaemia are phenotypically normal, but exhibit symptoms like sickle-shaped red blood cells when oxygen levels are low.

(a) Explain why individuals heterozygous for sickle cell anaemia are said to exhibit codominance. [3]

- Individuals heterozygous for sickle cell anaemia have **one copy** of the **normal allele** that codes for the β-globin protein and **one copy** of the **abnormal allele** that codes for abnormal β-globin
- Hence heterozygous individuals are said to exhibit codominance as both **alleles** are **equally expressed**.
- **Both normal and abnormal** β-globin **proteins** are produced.

Fragile X Syndrome (FXS) is a sex-linked recessive genetic disease in human that causes intellectual disabilities ranging from mild to severe.

A woman who is a carrier of both FXS and sickle cell anaemia married a man who is suffering from FXS with no family history of sickle cell anaemia. Their first son was born after 2 years of marriage.

(b) (i) Using a genetic diagram, show the possible offspring produced by the couple. [4]

Let X^F be the X chromosome with the allele coding for normal condition
X^f be the X chromosome with the allele coding for FXS condition
Y be the Y chromosome.
Hb^A be the allele coding for the normal β-globin allele
Hb^S be the allele coding for the abnormal β-globin allele

Parental phenotypes: **Woman FXS carrier, Sickle cell anaemia carrier** **x** **male FXS, normal for CF**

Parental genotypes (2n) $X^FX^f\ Hb^AHb^S$ **x** $X^f\ Y\ Hb^A\ Hb^A$ **[1]**

Gametes (n): X^FHb^A X^FHb^S X^fHb^A X^fHb^S **x** $X^f\ Hb^A$ YHb^A **[1]**

Offspring genotype and phenotype:

♀ \ ♂	X^FHb^A	X^FHb^S	X^fHb^A	X^fHb^S
$X^f\ Hb^A$	$X^FX^f\ Hb^AHb^A$ Female Normal for sickle cell	$X^FX^f\ Hb^AHb^S$ Female Normal for sickle cell	$X^fX^f\ Hb^AHb^A$ Female FXS Normal for sickle cell	$X^fX^f\ Hb^AHb^S$ Female FXS Normal for sickle cell
YHb^A	$X^FY\ Hb^AHb^A$ Male Normal for sickle cell	$X^FYHb^AHb^S$ Male Normal for sickle cell	$X^fY\ Hb^AHb^A$ Male, FXS Normal for sickle cell	$X^fY\ Hb^AHb^S$ Male, FXS Normal for sickle cell

[1]

Offspring phenotype:	Female, Normal for sickle cell	Female, FXS, Normal for Sickle cell anaemia	Male, Normal for sickle cell	Male, FXS Normal for sickle cell
Offspring ratio	1 :	1 :	1 :	1

[1]

(ii) The couple decided to have a second child. Determine the probability that their first son is phenotypically normal for both sickle cell anaemia and FXS and their second child has FXS and is a carrier of sickle cell anaemia. Show your working in the space below. [2]

Probability that their first son is phenotypically normal for both sickle cell anaemia = 0.5

Probability that their second child has FXS and is a carrier of sickle cell anaemia. = 0.25

Probability that their first son is phenotypically normal for both sickle cell anaemia and FXS and their second child has FXS and is a carrier of sickle cell anaemia = 0.5 X 0.25 = **0.125**

1 mark for working; 1 mark for solution **[Total: 9]**

QUESTION 4

The quantity of product formed from the Polymerase Chain Reaction (PCR) catalysed by *Taq* Polymerase was measured across a range of temperatures and three different durations. The results were plotted on the graph shown in **Fig. 4.1**.

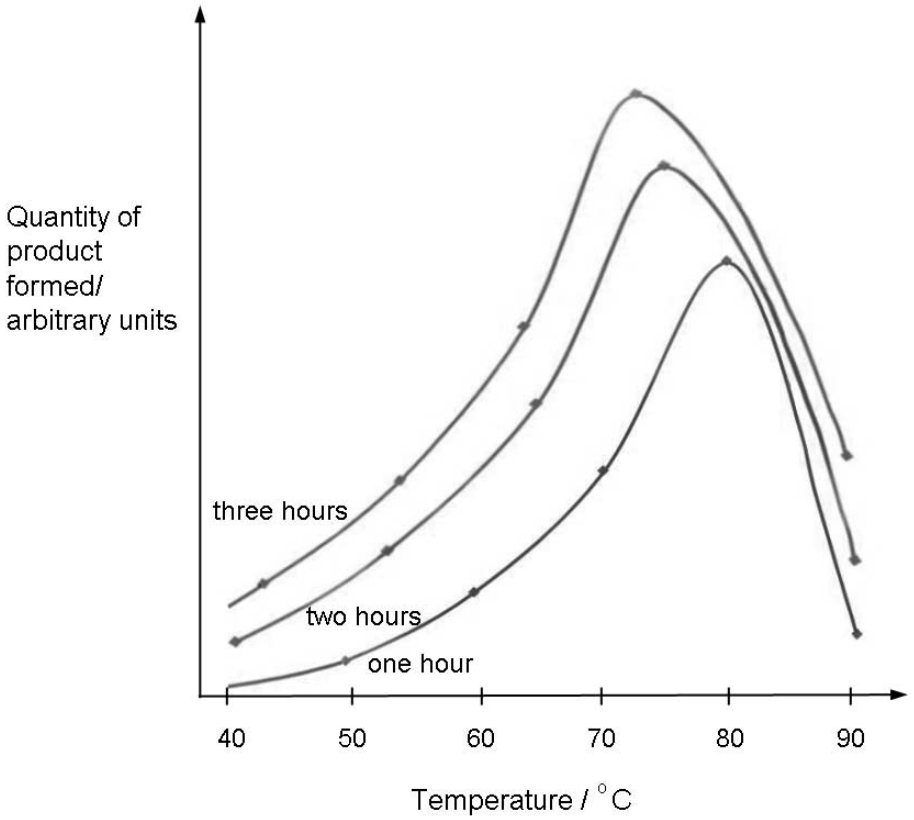

Fig. 4.1

a) Explain the effect of increasing temperature on the quantity of product formed for the sample kept at different temperatures for one hour. [4]

- Increasing the temperature would **increase the kinetic energy** of the molecules, which in turn **increases the rate of effective collisions** and **increases the formation of the enzyme-substrate complexes**.

- This results in an **increase in the quantity of products formed** until the **optimum temperature of 80°C** is reached.

- **Beyond** the optimum temperature, **denaturation** occurs as higher temperatures causes the **hydrogen bonds and weak ionic bonds to break**, resulting in the *Taq* polymerase **losing its tertiary / three dimensional structure**.

- The *Taq* polymerase enzyme **active site** is **no longer complementary** to the substrate and hence the **quantity of product formed decreases**.

b) Explain why the optimum temperature is lower if the quantity of product formed is measured after three hours rather than one hour. [2]

- The *Taq* Polymerase enzymes would be exposed to a high temperature for **a longer time** if the quantity of product formed is recorded after three hours, hence **more enzyme molecules will be denatured** due to the breaking of hydrogen bonds / weak ionic bonds and the subsequent loss of the tertiary structure/ shape of the active site.

- **More products would be formed at lower optimum temperature**

c) The products of PCR can then be analysed using gel electrophoresis.

Fig. 4.2 shows some of the steps involved in gel electrophoresis.

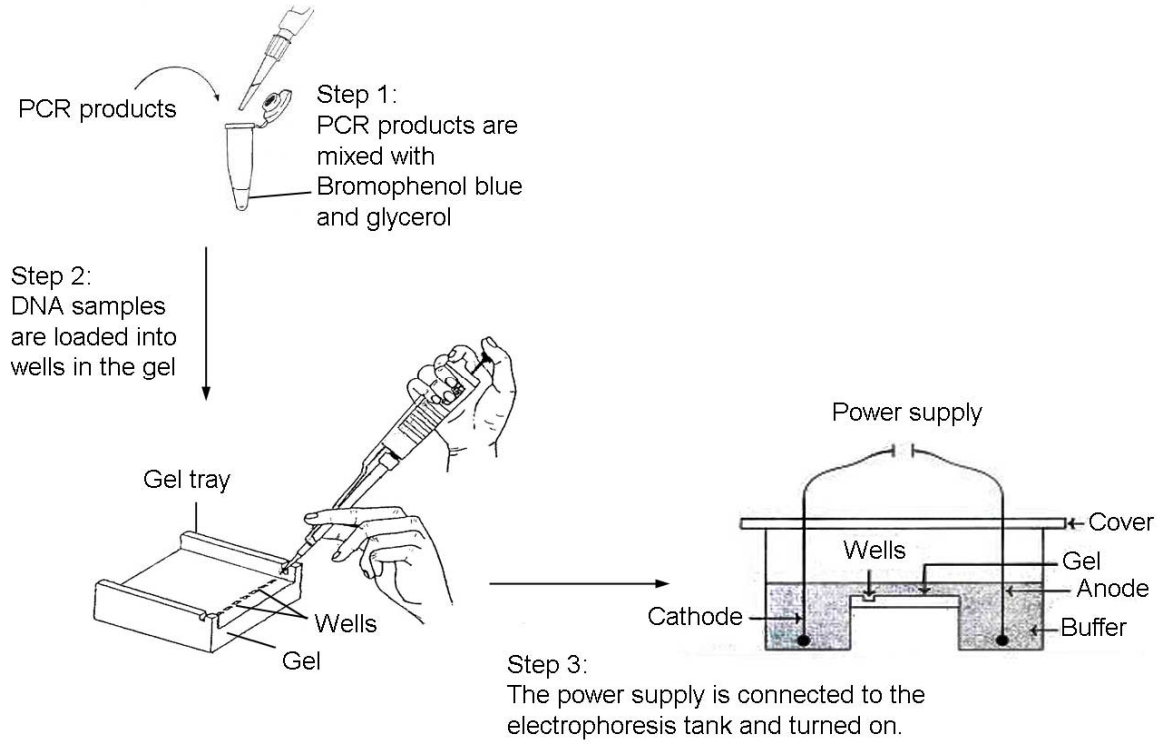

Fig. 4.2

(i) Explain the rationale behind Step 1. [2]

- The glycerol **increases the density of DNA** and allows it to sink to the bottom of the well so that it **does not float away into the buffer solution**

- The Bromophenol blue is the loading dye which allows **DNA migration** to be **monitored** / ensures that the samples are **loaded correctly** into the **wells**

(ii) Suggest the purpose of the buffer solution. [1]

- The buffer **maintains the neutral pH** of the solution so that the phosphate groups on **DNA retain their negative charge**, which allows DNA fragments to **migrate to the positive electrode.**

- The buffer contains **ions** which **allow electricity to pass through** the solution, so that positive and negative electrodes are present for **DNA migration**.

Huntington's disease (HD) is an autosomal-dominant disorder that results from the presence of 36 or more *CAG* repeats in the HD gene. **Fig. 4.3** shows the normal allele and the disease-causing allele.

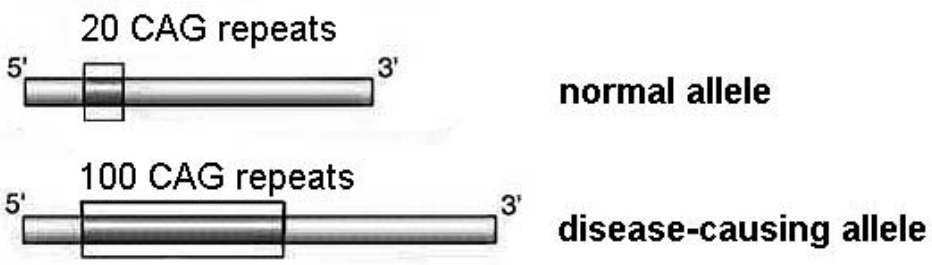

Fig. 4.3

d) Explain why the normal HD allele and the disease-causing HD allele can be differentiated by gel electrophoresis. [2]

- The two different alleles will have different lengths due to different number of CAG repeats
- Hence the two alleles can be differentiated by gel electrophoresis as the disease-causing allele is longer/larger in size, hence it will migrate slower through the gel than the normal allele (and vice versa).

[Total: 11]

QUESTION 5

a) Describe the structure of collagen and how it is related to its function. [6]

1. **Tropocollagen** is formed when **three** collagen **polypeptide chains** wound around each other to give a **triple helix**.

2. Each collagen polypeptide chain is in the shape of a **loosely wound left-handed helix** that wind around the other two.

3. The three strands are linked together by **hydrogen bonds** formed between peptide N-H group of glycine and peptide C=O group of other amino acids on the other strands.

4. The sequence of amino acids of each strand is usually a repeat of
   Glycine – Proline – X, or
   Glycine – X – Hydroxyproline

   where X is any other amino acids except glycine

5. The presence of **glycine** at every third amino acid within each polypeptide chain allows **close packing** of the triple helix to form a **tight coil**.

6. Each complete triple helix of tropocollagen interacts with other tropocollagen molecules running parallel to each other by forming **covalent bonds** between the **lysines** in chains lying next to each other.

7. These cross-links hold many tropocollagen molecules side by side, forming **fibrils**, giving rise to **high tensile strength** (mark once for high tensile strength).

8. In collagen **fibrils**, tropocollagens lie **parallel** with **staggered ends,** which would permit them to **overlap** with the tropocollagens in adjacent fibrils.

9. Aggregation of overlapping collagen **fibrils** form strong collagen **fibers.**

10. [Function] Hence collagen is able to **provide structural support** for skin, tendons, cartilage, bones, teeth and connective tissue of blood vessels.

b) Discuss the role of named proteins in photosynthesis. [8]

Photosystems:

1. **Photosystems I** and **II** are **embedded** in the **thylakoid membranes** and consist of various **photosynthetic pigments** such as **chlorophylls and carotenoids** . . .
2. . . . which **absorb red and blue-violet wavelengths of light for photoactivation**

Electron carriers:

3. Proteins also serve as **electron carriers** of **progressively lower energy levels** that form the electron transport chain (ETC) embedded in the thylakoid membrane.
4. The electron transport chain transports electrons from higher to lower energy levels **to release energy**.

Proton pumps:

5. **Proton pumps** in the ETC harness energy released from electron transport chain to **pump protons (H^+) from stroma into thylakoid space** via **active transport**.

6. This creates a **proton gradient across the thylakoid membrane**.

ATP synthase:

7. ATP synthase is required for the **facilitated diffusion of protons down its concentration gradient** from **thylakoid space to stroma** . . .
8. … which is coupled to the **synthesis of ATP** from **ADP and inorganic phosphate.**

Other enzymes:

9. **NADP reductase** is an enzyme that catalyses the **reduction of NADP** .
10. **Reduced NADP / NADPH** produced is used as a **reducing agent** in the **light-independent reactions** (**Calvin cycle**).

11. **RUBISCO / ribulose bisphosphate carboxylase oxygenase** catalyses the **fixation** of **carbon** in the Calvin Cycle . . .

12. . . .where one molecule of **carbon dioxide** combines with a **5C ribulose bisphosphate (RuBP)** molecule to give a **6-carbon intermediate**.

c) Describe the roles of vesicles in a cell. [6]

1. For transport of proteins from **Endoplasmic Reticulum** to **Golgi Apparatus**
2. For transport of proteins from **Golgi Apparatus** to **Cell Surface Membrane**
3. For transport of proteins from **Golgi Apparatus** to other cellular organelles or destinations
4. For **fusion** of vesicle with **cell surface membrane** which results in **replenishment of cell surface membrane**
5. For **fusion** of transport vesicle with GA which results in **replenishment of Golgi Apparatus membrane**
6. For **enclosing** foreign particles by the process of **endocytosis**
7. Functions as **lysosomes that store hydrolytic enzymes + 2 functions** of lysosome
8. **Cellulose**-containing vesicles fuse to form the **cell plate** during cytokinesis in plant cells
9. Named examples, e.g. insulin (secreted), G-protein coupled receptor (embedded in CSM), proton pumps (embedded in lysosomal membrane), neurotransmitters (exocytosis).

QUESTION 6

a) Describe how ATP is synthesised by anaerobic respiration in yeast. [6]

Alcoholic fermentation:

1. Pyruvate under goes **alcoholic fermentation** in **yeast** when **oxygen concentrations are low**.
2. Pyruvate undergoes **decarboxylation** to give **ethanal** and **carbon dioxide**.
3. **NADH reduces** ethanal to **ethanol** in a reaction catalysed by **alcohol dehydrogenase**.
4. . . . and NADH is **oxidised to NAD**.
5. Purpose is to **regenerate NAD** so that **ATP** can be continuously synthesised by **substrate level phosphorylation** during **glycolysis**.
6. Anaerobic respiration produces **2 molecules of ATP per molecule of glucose**.

b) Explain why variation is important for natural selection and how variation arises in a population. [6]

Why variation is important (max 2):

1. Environmental change acts as **selection pressure** to select those individuals with advantageous traits or alleles.
2. The more variation a species has, the higher the chances of the species surviving different types of environmental change.
3. Variation is important as it **decreases the chances of extinction**.

How variation arises (max 4):

4. **New genes/alleles** arise from gene and chromosomal **mutations**

5. Organisms may only pass mutations gained in their lifetime to their offspring when the mutation arises during **gamete formation**.

6. **Meiosis** can contribute to **genetic variation** due to **recombination** of **alleles** via
   - o Crossing over during prophase I
   - o Random assortment and segregation of homologous chromosomes at metaphase I and anaphase I
   - o Random assortment and segregation of non-identical sister chromatids at metaphase II and anaphase II.

7. **Random fusion of gametes** results in genetic variation during **fertilisation.**

8. Phenotypic variation can also arise as a result of **environmental influence**. Genes can **interact with the environment** to give rise to variation.

9. **Migration** into a population of individuals from another population, together with **random mating**, promotes **gene flow**.

c) Discuss the detrimental environmental and economic effects of growing genetically modified herbicide resistant crops. [8]

Environmental effects:

1. **Genetically-modified crop plants** may be hardier and become **agricultural weeds** that invade natural habitats
2. The **introduced transgene(s) to wild species** may result in **more invasive hybrid offspring** when pollen transfer to wild relatives.
3. **Intensive** use of herbicide selects for **herbicide-resistant weeds**
4. Intensive use of herbicide reduces biodiversity, **upsetting the natural balance of the ecological system**.

Economic effects (4 max):
5. High cost of GM seeds/plants, farmers cannot afford/ erode farmers' income
6. Heavy use of herbicide, thus, cost more
7. contamination of organic farming due to accidental mixing of GM crops with non-GM crops
8. cleaning pollution associated with heavy use of herbicide
9. human health problems associated with the use of herbicide

[Total: 20]

• END OF PAPER 2 •