

INNOVA JUNIOR COLLEGE
JC 2 PRELIMINARY EXAMINATION 2
in preparation for General Certificate of Education Advanced Level
Higher 1

CANDIDATE
NAME

MARK SCHEME

CLASS

INDEX NUMBER

BIOLOGY

8875/02

Paper 2 Core Paper

15 September 2015

2 hours

Additional Materials: Answer Paper
Cover Page

READ THESE INSTRUCTIONS FIRST

Write your name and class 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.

Section A

Answer **all** questions.

Section B

Answer **one** question.

At the end of the examination, fasten all your work securely together.

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

For Examiner's Use	
Section A	
1	9
2	10
3	11
4	10
Section B	
	20
Total	60

This document consists of **11** printed pages and **1** blank page.



Section A
Answer **all** questions.

- 1 Fig. 1.1 shows drawings of a stem cell at various stages in the mitotic cell cycle.



Fig. 1.1

- (a) (i) List the letters shown in Fig. 1.1 in the order in which these stages occur during a mitotic cell cycle. The first stage has been entered for you.

A C E D B [1]

- (ii) Describe the events occurring in Stage D.

1. ***(anaphase) where sister chromatids are separated to opposite poles of the cell due to shortening of kinetochore microtubules;***

2. ***centromere divides;***

sister chromatids led by centromere to opposite ends;

cohesion degraded by separase; (any 2 pts for 1m) [2]

- (iii) Outline the fates of the daughter cells following cell division.

1. ***one cell remains a stem cell, the other cell becomes a progenitor cell → further differentiate into specialised cell;***

2. ***as a result of asymmetric cell division;***

ER: Many candidates did not understand that the parent cell is a stem cell

[2]

- (iv) Mitosis gives rise to two genetically identical daughter cells. Suggest a reason for the different fates of the daughter cells in part (iii).

1. ***diff gene expression (resulting in diff prot produced) due to different internal & external signals;***

[1]

Stem cells have the potential to treat many diseases such as diabetes and Parkinson's. These diseases are caused by the death or malnutrition of particular cells.

- (b) Explain why stem cells have the potential to treat diseases.

1. ***unspecialised, with no tissue specific structures;***

2. ***display potency, able to divide and differentiate into different cell types***

to replace cells that died due to disease;

3. ***unlimited renewal capacity due to active telomerase activity***

to supply new cells throughout the lifetime of the patient; [3]

[Total:9]

2 Protein synthesis in a prokaryote is illustrated in Fig. 2.1.

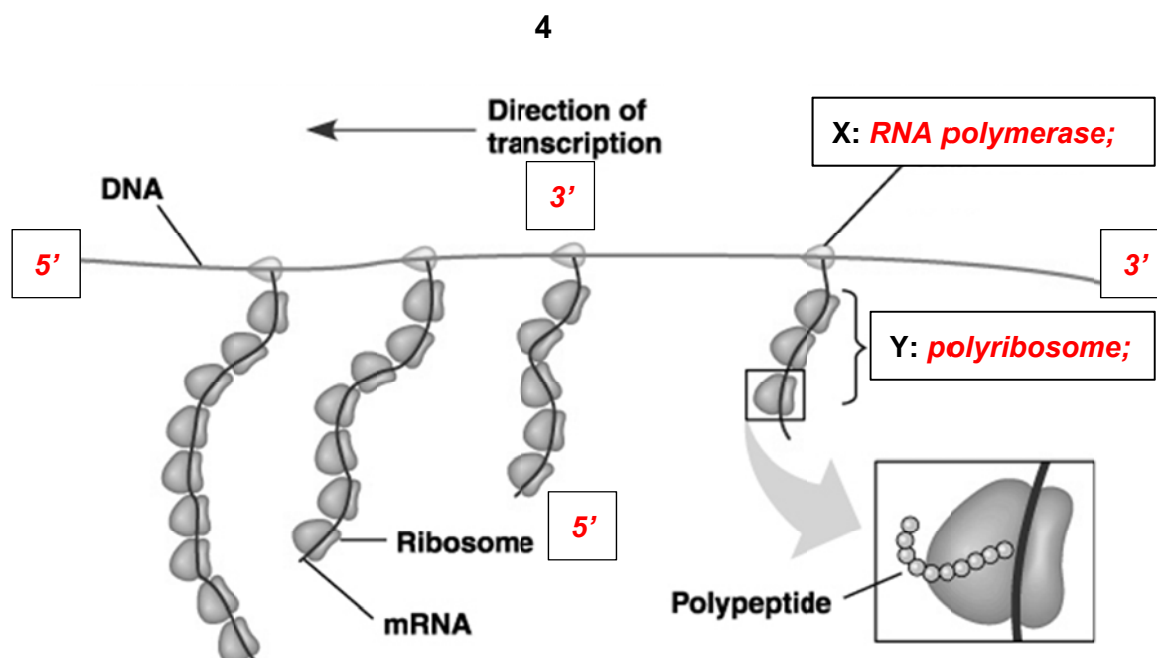


Fig. 2.1

(a) On Fig. 2.1,

(i) label the structures **X** and **Y**. [2]

(ii) label the 5' and 3' ends of the DNA template strand and the mRNA. [2]

(b) Explain the significance of structure **Y**.

1. cluster of ribosomes that simultaneously translate mRNA;

2. allow more proteins to be synthesized per unit time;

[2]

Chloramphenicol is an antibiotic that irreversibly binds to a receptor site on the large subunit of prokaryotic ribosomes, inhibiting the catalytic activity of the ribosome.

The mechanism of action of chloramphenicol is shown in Fig. 2.2.

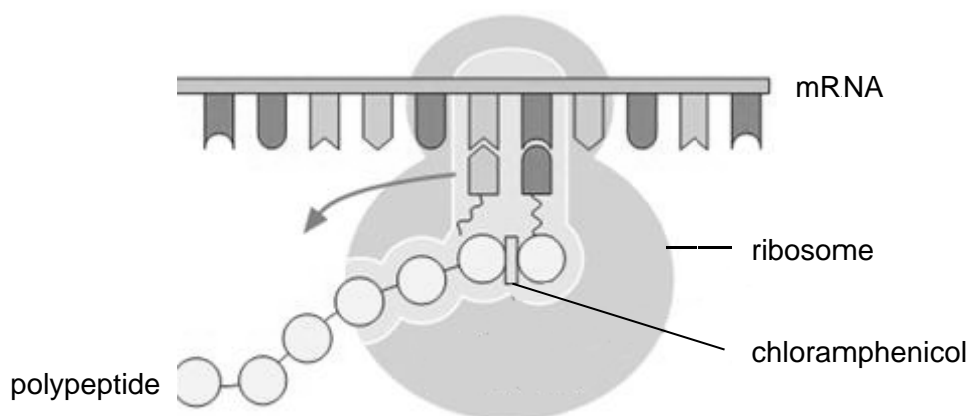


Fig. 2.2

- (c) (i) With reference to the information given and Fig. 2.2, explain how chloramphenicol kills bacteria.

1. ***inhibits peptidyl transferase, inhibiting formation of peptide bonds between aa on incoming tRNA and growing polypeptide chain;***
2. ***prevent formation of proteins which function as enz / used in metabolic pathways / form structures in the cell;***

[2]

- (ii) Suggest why chloramphenicol can be taken by humans.

1. ***chloramphenicol binds to 50S large subunit / 70S ribosome of prokaryotes;***
2. ***large subunit of eukaryotic ribosomes is 60S / eukaryotic ribosomes are 80S;***

[2]

[Total: 10]

- 3 A group of scientists measured the mean rate of respiration and mean rate of photosynthesis of a tree growing in sunlight, by measuring the concentration of carbon dioxide given out or taken in. Measurements were taken at different times of the day.

Fig. 3.1 shows the scientists' results.

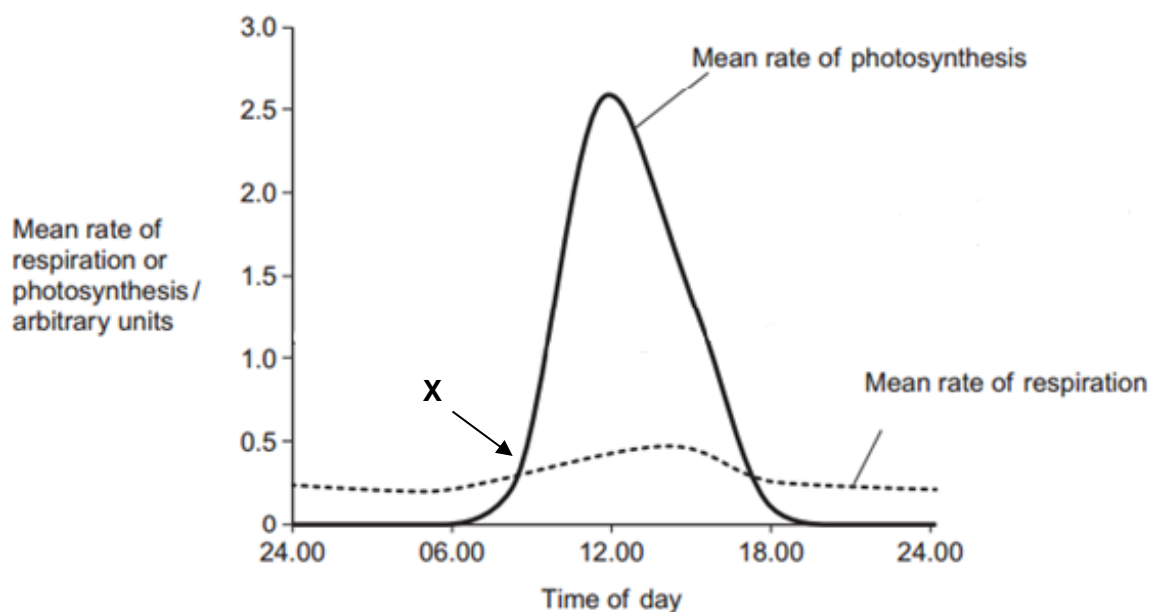


Fig. 3.1

- (a) With reference to Fig. 3.1,

- (i) Explain the peak in the mean rate of photosynthesis at 12.00.

1. **highest light intensity \rightarrow highest rate of photosynthesis (2.5au);**
2. **inc rate of photoactivation \rightarrow inc e^- transfer down ETC \rightarrow inc pdtn of ATP and red NADP;**
3. **ATP and red NADP used in Calvin cycle \rightarrow inc rate of Calvin cycle \rightarrow inc rate of carbon fixation;**

[3]

- (ii) With reference to the biochemical processes occurring in photosynthesis and respiration, explain the significance of the point labelled X.

1. **light compensation point, at light intensity where rate of photosynthesis = rate of respiration;**
2. **CO_2 given out during link rxn and Krebs cycle of respiration taken in during carbon fixation / light indep rxns of photosynthesis;**

[2]

- (b) The scientists suggested that the rise in the mean rate of photosynthesis was the cause of the rise in the mean rate of respiration.

Suggest how the mean rate of photosynthesis could lead to the rise in the mean rate of respiration.

1. **production of glyceraldehyde-3-phosphate during Calvin cycle of photosynthesis \rightarrow used to make glucose;**
2. **glucose used as respiratory substrate during glycolysis of respiration; OR**
3. **production of oxygen during photolysis of water of photosynthesis;**
4. **used as final electron acceptor of ETC in aerobic respiration;**

[2]

Glyceraldehyde-3-phosphate produced during photosynthesis in plants may be converted to other forms of carbohydrates, such as starch and cellulose.

Fig. 3.2 shows the part of the structures of starch and cellulose.

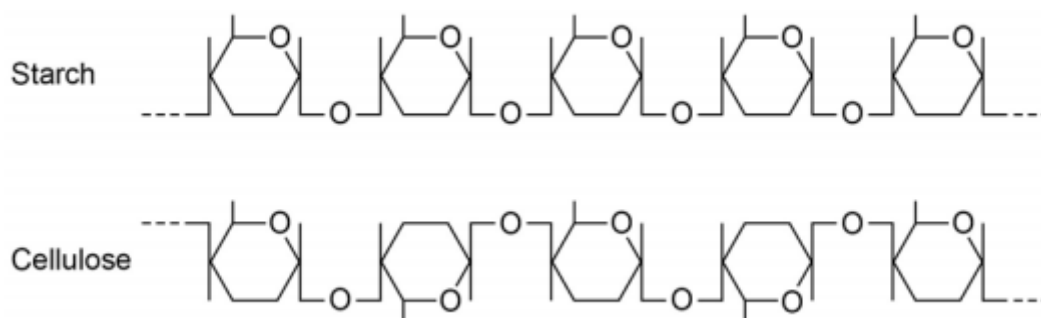


Fig. 3.2

- (c) Explain **two** differences in the structure of the starch molecule and the cellulose molecule as shown in Fig. 3.2.

1. α -1,4-glycosidic bond vs β -1,4-glycosidic bond;

2. adj glc monomers not inverted vs adj glc monomers inverted by 180°;

[2]

Starch molecules and cellulose molecules have different functions in plant cells. Each molecule is adapted for its function.

Explain **one** way in which starch molecules are adapted for their function in plant cells.

1. contain thousands of glc monomers; allow large amt of energy to be stored as glc is the main respiratory substrate;

2. helical str of amylose and branched str of amylopectin; compact str that allows for more efficient storage of glc molecules

3. projection of -OH groups into interior of helix; makes starch insoluble, allowing storage of starch w/o changing osmotic pot of cell;

[2]

(any 1 point, 1 max)

[Total: 11]

- 4 To reduce damage caused by insect pests, some farmers spray their fields of crop with pesticide. Many of these pesticides have been shown to cause environmental damage.

Bt plants have been genetically modified to produce a toxin that kills insect pests. The use of Bt crop plants has led to a reduction in the use of pesticides.

To produce Bt plants, the bacterium *Agrobacterium tumefaciens* was used to introduce the recombinant plasmid containing the gene for Bt toxin to plant cells.

The plasmid used had the following sequences:

- an origin of replication
- ampicillin resistance gene, amp^R
- tetracycline resistance gene, tet^R, containing the *Bam*HI restriction site

- (a) Explain how a recombinant plasmid containing the gene coding for Bt toxin is produced.

1. restriction digestion, where BamHI are used to cut gene coding for Bt toxin and plasmid vector;

2. annealing of plasmid and GOI via H bonding btwn comp bp

ligation using DNA ligase, catalysing formation of phosphodiester bond;

ER: Many candidates did not specify the RE to use despite BamHI being stated in the question

[2]

Replica plating was used to identify bacteria that successfully took up the recombinant plasmid, as shown in Fig. 4.1.

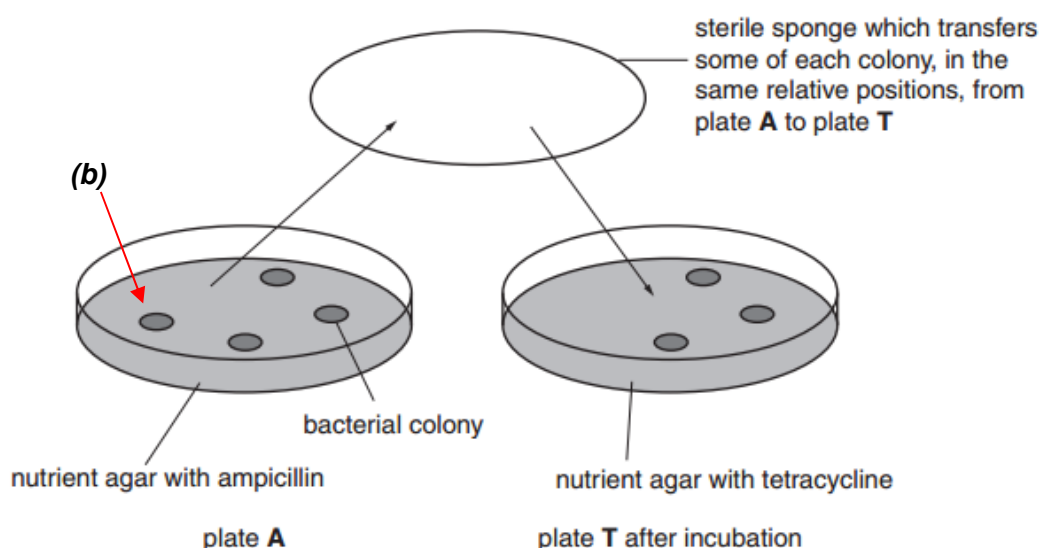


Fig. 4.1

- (b) On Fig. 4.1, use labelled arrow(s) to indicate the bacterial colony / colonies containing the recombinant plasmid. [1]
 ® **No labels**

- (c) Explain your answer in part (b).
 1. **transformed bacteria able to grow on medium containing amp due to presence of amp^R gene on plasmid;**
 2. **insertional inactivation of tet^R gene → bact containing recomb plasmids not able to grow on medium containing tet;**

ER: Few candidates did not realise it was replica plating and make reference to X-gal instead

[2]

Scientists have found that some species of insect pests have become resistant to the toxin produced by the Bt crop plants.

Fig. 4.2 shows information about the use of Bt crops and the number of species of insect pests resistant to the Bt toxin in one country.

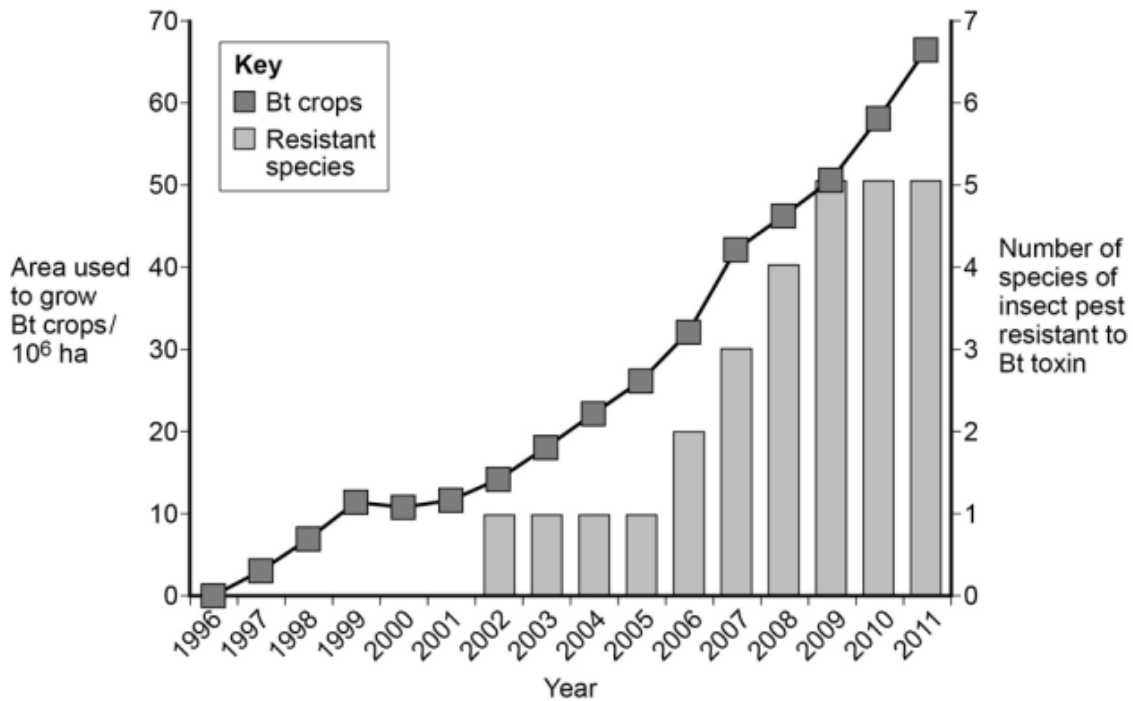


Fig. 4.2

- (c) Discuss whether the increase in number of insect pest resistant to Bt toxin was due to the increase in the area used to grow Bt crops.

1. **yes**

↑ in area of Bt crop grown correspond to ↑ in no. of resistant spp, QV;

2. *presence of Bt toxin acts as selection pressure, where Bt-resistant insects have selective advantage;*

3. *↑ in survival rate & repro success → pass fav alleles to offspring;*

4. **no**

↑ in area of Bt crop grown ⊗ always correspond to ↑ in no. of resistant spp, QV;

5. *resistant allele arise in gene pool due to spontaneous mutation;*

6. *↑ in no. of resistant spp due to genetic drift;*

[3]

- (e) Apart from the possibility of pests developing resistance to Bt toxin, describe two other ethical and social implications of genetically modified organisms.

1. *threat to human safety due to transfer of antibiotic resistance genes (present on selectable marker) to pathogenic bacteria;*

2. *threat to ecological balance, e.g. establishment of invasive superweeds / e.g. death of unintended organisms;*

3. *inadequate risk assessment and lack of studying long term effects of GMO / many countries without legislation on GM labelling*

[2]

[Total: 10]

Section B

Answer **one** question.

Write your answers on the separate answer paper provided.

Your answers should be illustrated by large, clearly labelled diagrams, where appropriate.

Your answers must be in continuous prose, where appropriate.

Your answers must be set out in section **(a)**, **(b)** etc., as indicated in the question.

- 5 (a) With reference to the levels of protein structure, explain how the specificity of an enzyme is determined by its structure. [6]

1. **primary structure of prot refers to unique, linear seq of aa linked by peptide bonds;**
2. **secondary structure of prot refers to regular folding of polypeptide to form α -helix or β -pleated sheet maintained by hydrogen bonding between $-\text{CO}$ and $-\text{NH}$ of peptide bonds;**
3. **tertiary structure of prot refers to further folding of secondary structures to form a globular structure maintained by R group interactions;**
4. **maintained by hydrophobic interactions between nonpolar R groups, hydrogen bonding between polar R groups, ionic bonds between electrically charged R groups, disulphide bonds between cysteine residues;**
5. **quaternary structure of prot involving 2 or more polypeptides, maintained by R group interactions;**
6. **folding of polypeptide resulting in specific 3D configuration \rightarrow specific 3D configuration of enzyme active site;**
7. **catalytic and contact amino acids of enzymes brought together via folding to form active site;**
8. **able to bind specific substrate due to complementary shape or charge;**

- (b) Describe the effect of inhibitors on the rate of enzyme activity. [7]

1. **graph showing competitive inhibition;**
2. **competitive inhibitors are structurally similar to substrate, competing with substrate for active site of enzyme;**
3. **(formation of enz-inhibitor complex \rightarrow) dec in rate of effective collisions between enz and substrate \rightarrow dec in rate of formation of ES cplx \rightarrow dec in rate of reaction;**
4. **further inc in substrate conc able to overcome effect of inhibition due to inc in probability of forming ES cplx;**
5. **graph showing non-competitive inhibition;**
6. **non-competitive inhibitor binds to site other than active site \rightarrow change in 3D config of enz \rightarrow change in config of active site;**
7. **enz no longer able to bind to substrate \rightarrow dec in rate of formation of ES complex \rightarrow dec in rate of rxn;**
8. **further inc in substrate conc does not overcome effect of inhibition;**

- (c) The enzyme catalase is found in potatoes. This enzyme catalyses the breakdown of hydrogen peroxide to water and oxygen.

Describe an experiment investigating the effect of temperature on the activity of catalase in potatoes, by measuring the release of oxygen. [7]

1. *labelled diagram of set-up;*
2. *placing of potatoes in hydrogen peroxide solution and collecting oxygen released using a gas syringe;*
3. *control temp using a water bath, using 5 different temperatures (e.g. 10, 20, 30, 40, 50°C);*
4. *control pH (~ pH 7) using pH buffer to ensure that rate of enz rxn is not affected by changes in pH;*
5. *standardise concentration of H₂O₂ volume (e.g. 5 ml) and concentration (e.g. 5%);*
6. *standardise size of potato discs (e.g. 0.2 cm width, 1 cm diameter);*
7. *graph showing expected results;*
8. *hydrogen peroxide corrosive – require use of gloves;*

[Total: 20]

6 (a) Describe and explain the fluid mosaic model of cell membranes. [6]

1. *lateral movement within phospholipid monolayer due to hydrophobic interaction (with slight possibility of flip-flop movement of phospholipids between monolayers);*
2. *unsaturated fatty acid tails (with C=C) double bonds → formation of kinks → less extent of hydrophobic int between fatty acid tails of adj phospholipids → fluidity;*
3. *cholesterol regulate memb fluidity by inc fluidity at low temp and dec fluidity at high temp;*
4. *asymmetrical distribution of phospholipids between monolayers;*
5. *glycolipids comprising short carbohydrate chains attached to lipids and glycoproteins comprising short carbohydrate chains attached to proteins;*
6. *proteins of variable size and shape embedded and scattered throughout bilayer, e.g. peripheral and integral proteins;*

(b) Describe the structure of a phospholipid and its arrangement in cell membranes. [5]

1. *phospholipids comprise a phosphate head, glycerol backbone and two fatty acid tails;*
2. *phosphate head joined to glycerol backbone via phosphoester linkage and fatty acid tails joined to glycerol via ester bond;*
3. *phosphate head is negatively charged, interacting with aqueous medium via hydrogen / ionic bonds;*
4. *fatty acid tails (comprising hydrocarbon chains) are nonpolar / hydrophobic, interacting via hydrophobic interactions;*
5. *forming a phospholipid bilayer, with phosphate heads on exterior and fatty acid tails facing the interior (forming a hydrophobic core);*

(c) Discuss the role of membrane proteins in cell membranes. [9]

1. *transport of hydrophilic / polar substances in and out of the cell by providing a hydrophilic channel;*
2. *as they are repelled by the hydrophobic core of the phospholipid bilayer made up of fatty acid tails of phospholipids;*
3. *via active transport, requiring use of ATP to transport substances against concentration gradient;*
4. *via facilitated diffusion, transporting substances down concentration gradient;*
5. *proteins involved in enzymatic catalysis of reactions;*

6. *e.g. ATP synthase embedded in inner mito memb / thylakoid memb involved in phosphorylation of ADP to ATP;*
7. *receptor proteins involved in communication between cells;*
8. *where ligand binds to receptor to allow ext signals to be passed to influence cellular reactions;*
9. *glycoproteins involved in cell-cell adhesion and cell recognition;*
10. *structural support to maintain cell shape via interaction with the cytoskeleton;*

[Total: 20]