Nov 2017 H2 Bio Paper 2

N17P2Q1

(a) (i) State the number of glycogen molecules shown in Fig. 1.1. [1]

2

(ii) Name the type of molecule forming the central core of the glycogen granule, in Fig. 1.1. [1]

Protein / polypeptide

(iii) Suggest the role of the molecule forming the central core of the glycogen granule. [1]

1. binds / holds 2 glycogen molecules together;
2. enzymes that catalyse the synthesis of glycogen

(b) Explain how the structure of glycogen is related to its role in living organisms. [4]

1. Large molecule/long chain so insoluble in water ;
2. Hence does not affect water potential of cells / no osmotic effect ;
3. Helical molecule so more ***glucose**** units per unit volume, thus compact structure for storage ;
4. Extensively branched so provides many ends for hydrolysis of glycosidic bonds ;
5. which releases more ***glucose**** molecules per unit time ;

(c) Describe how the structure of cellulose is different from the structure of glycogen. [3]

	Cellulose	**Glycogen**
monomer	***β-glucose****	***α-glucose****
bonds	***β-1,4 glycosidic bonds****	***α-1,4 and α-1,6 glycosidic bonds****
Orientation of monomer	***Alternate glucose*** units are rotated 180^0/***inverted*** with respect to each other	All glucose units in the chain have same orientation
Structure of each molecule	forming ***straight / linear unbranched chains ;***	Forms helical coil which is extensively ***branched ;***
Bonds between molecules	***Hydroxyl groups*** projecting outwards in both directions allow ***intermolecular/ interchain hydrogen bonding*** → leading to microfibril formation for cellulose	No interchain hydrogen bonding in glycogen

Note: must be written in full sentences

N17P2Q2

(a) (i) Explain why proteins are required for the transport of glucose across the cell surface membrane. [3]

1. Glucose is ***polar**** and thus, ***hydrophilic**;
2. Glucose is too large to pass through the transient pores of the phospholipid bilayer ;
3. The ***hydrophobic core**** of the phospholipid bilayer would repel glucose ;
4. Transport proteins / protein channel provides a ***hydrophilic**** channel/pore through the membrane for the passage of the solutes;

(ii) Describe the structural features of a protein that enable it to transport glucose into a cell. [2]

1. The protein is a transmembrane protein that;
2. has ***amino acid**** residues with R groups that is non-polar / uncharged, allowing it to form ***hydrophobic interactions**** with the ***hydrophobic core**** of the ***phospholipid bilayer****;
3. The protein has a ***hydrophilic**** pore/channel that is lined with ***amino acids**** with polar / charged R groups that allows hydrophilic glucose molecules to pass through the membrane;

(iii) State the letter of the protein in Fig 2.1 that could transport glucose into a cell. [1]

Q

(b) Using the information shown in Fig 2.2, describe and explain the effect of increasing the external glucose concentration on the rate of glucose uptake into a cell. [4]

Describe:

1. When external concentration of glucose increase from 0 - 1 mmol dm^{-3}, rate of glucose uptake increases sharply from 0 – 180 arbitrary units;
2. When external concentration of glucose increases from 1 – 11 mmol dm^{-3}, rate of glucose uptake increases from 180 – 395 arbitrary units, but rate of glucose uptake increase less with each unit increase in external glucose concentration;
3. Rate of glucose uptake plateaus at 395 arbitrary units when external glucose concentration increase from 11 - 12 mmol dm^{-3};

[A if no other pts awarded: Increase in external concentration of glucose from 0 - 11 mmol dm^{-3}, increases the rate of glucose uptake from 0 to 395 arbitrary units.]

Explain:

4. Increase in external glucose concentration increases concentration gradient of glucose across the membrane;
5. Maximum rate of glucose uptake is reached when all glucose transporters/transport proteins are saturated

N17P2Q3

(a) Describe the main features of the molecular structures of haemoglobin and collagen, visible in Fig. 3.1. [5]

Haemoglobin:

1. Haemoglobin is a globular protein;
2. It has a quaternary structure and comprises ***2 α-globin**** subunits and ***2 β-globin**** subunits;
3. Each subunit is associated with a ***haem**** prosthetic group;

Collagen:

4. Collagen is a fibrous protein;
5. It has both a secondary struc and a quaternary structure and comprises 3 polypeptide chains coiled around each other;
6. Each polypeptide is a loose helix;

(b) Explain how the molecular structures of haemoglobin and collagen are related to their functions. [5]

Haemoglobin

Molecular structure	Function
1. Each subunit is bound to a ***haem**** prosthetic group;	Allows for each subunit to bind to an oxygen molecule;
2. Hemoglobin has a quaternary structure as it is made up of ***2 α-globin**** subunits and ***2 β-globin**** subunits;	4 subunits held together by weak intermolecular interactions formed between R groups (hydrogen bonds, ionic bonds and hydrophobic interactions), allows movement that influences affinity for oxygen allowing for ***cooperative binding**** of oxygen; Or As a result binding of one oxygen molecule to one haemoglobin subunit induces a ***conformational**** change in remaining 3 subunits so that their affinity for oxygen increases; To highlight Idea: having multiple subunits, intermolecular bonds allow movement to influences affinity for oxygen → so when 1 subunit binds to oxygen, conformation changes in other subunits (cooperativity)
3. Amino acids with hydrophilic R groups are found on the exterior of the protein and amino acids with hydrophobic R groups are buried in interior of protein;	Allows molecule to be soluble in water so that it can be easily transported in blood;

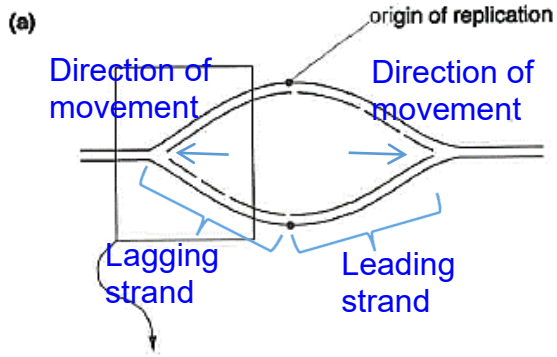
Collagen:

Molecular structure	Function
1. Numerous ***hydrogen bonds**** between on different polypeptides	Gives rise to high ***tensile strength**** which means can stretch without breaking;
2. Staggered arrangement of collagen molecules within the fibrils	Minimises point of weakness which means can stretch without breaking;
3. *3* ***polypeptide**** chains wound together to form 1 ***tropocollagen**** molecule;	Gives rise to high ***tensile strength**** which means can stretch without breaking;;
4. small ***glycine**** residues allow formation of a very tight triple helical structure (so that hydrogen hydrogen bonds can then form OH groups on different polypeptides) Point 4 and 5 will be awarded if students mention point 3. Reason being this point is relevant only if structure is part of a tropocollagen.	Gives rise to high ***tensile strength**** which means can stretch without breaking;
5. bulky, inflexible proline and hydroxyproline confer rigidity on the molecule Point 4 and 5 will be awarded if students mention point 3. Reason being this point is relevant only if structure is part of a tropocollagen.	Gives rise to rigidity;
6. ***covalent cross-links**** form between ***lysine**** residues at C and N ends of adjacent/parallel tropocollagen molecules; 7. collagen fibrils lie in parallel bundle to form collagen fibres; 8. staggered arrangement to minimises points of weaknesses along length of the fibrils; AVP	Gives rise to high ***tensile strength****;

For function of collagen: where appropriate + elaborated correctly, accept “insoluble in water” so that collagen can function as structural protein.

N17P2Q4

(a) On Fig. 4.1(a),
- label a leading strand and a lagging strand
- draw arrows to show the direction of movement of both replication forks. [3]



(b) Name the enzymes **J** and **K** shown in Fig. 4.1(b).
J **K** [2]

J DNA polymerase
K DNA ligase

(c) Explain the reason for the production of Okazaki fragments. [3]
1. This is because ***DNA polymerase**** works only in 5' to 3' direction;
2. Hence, strand extension occurs in direction away from replication fork / strand extends in opposite direction with respect to replication fork;
3. Synthesis of new strand can only occur in fragments as DNA double helix unzips;

N17P2Q5

(a) Describe the main changes visible in Fig. 5.1 in the development of cervical cancer, following the initial infection of cells in the basal layer by human papillomavirus. [4]
1. The virus infected cells reproduced to produce precancerous cells that possess nuclei with non-integrated viral DNA;
2. The virus infected cells become larger and go through more rounds of cell division, such that the layer of cells no long have a distinct boundary;
3. As the virus reproduces in the host cell, more infectious virus particles are released to infect even more cells;
4. Over time, the viral DNA can be intergrated into the host cell genome such that that cells are permanently altered genetically;
5. The cells in the basal layer are no longer cuboidal, but are irregular shaped;
6. They also continue to show uncontrolled cell proliferation and penetrate the basal membrane and the dermis to result in invasive cancer;

(b) Explain why the viral oncogene *E6* increases the chances of cancer developing. [3]
1. *p53* gene is a ***tumour suppressor gene**** coding for the p53 protein;
2. E6 protein destroys the p53 protein and hence;
3. Prevents apoptosis/ DNA repair;
4. This allows for uncontrolled cell division;
5. and accumulation of mutations;

(c) Suggest why mutations in the *p53* gene are common in most other human cancers, but rare in cervical cancer. [3]

1. In most other cancer, p53 gene would go through ***loss-of-function mutation**** to produce non-functional p53 protein;
2. In cervical cancer, p53 gene is not mutated and functional p53 protein can be produced;
3. The infection with human papillomavirus introduces the E6 gene into the cells allowing for the production of the E6 protein which destroys the p53 protein, hence the function of the p53 protein is disrupted, even though the protein is functional;

N17P2Q6

(a) State the name of this type of interaction between two genes. [1]

(recessive) epistasis;

(b) State all the possible genotypes for:

(i) A mouse with fur colour that is albino (white) [1]

aaBB, **aaBb** and **aabb** (must state all. R: **aa_ _**)

(ii) A mouse with fur colour that is black [1]

AAbb and **Aabb** (must state all)

(iii) A mouse with fur colour that is agouti (brown). [1]

AABB, AaBB, AABb and **AaBb** (must state all. R: **A_B_**)

(c) For each of the following genetic crosses, use a genetic diagram to explain the results. Show the parental and offspring genotypes and the expected ratio of offspring phenotypes.

(i) Black mouse x albino mouse produces 27 agouti and 24 albino offspring [3]

Parental phenotype: Black x Agouti
Parental genotype: Aabb x aaBB
Gametes: Ab ab aB

Offspring genotype: AaBb aaBb;
Offspring phenotype: 1 agouti: 1 albino;

(ii) Black mouse x black mouse produces 28 black and 10 albino offspring [3]

Parental phenotype: Black x Black
Parental genotype: AAbb x Aabb;
Gametes: Ab ab Ab ab

Offspring genotype: AAbb Aabb Aabb aabb

Offspring phenotype: 3 black : 1 albino

N17P2Q7

(a) Carrier molecule **A** is the source of electrons for the electron transport chain shown in Fig. 7.1.

(i) Name carrier molecule **A**. [1]

NADH

(ii) Name the stages in respiration where carrier molecule **A** is formed. [1]

Glycolysis, Link reaction, Kreb Cycle

(iii) Explain why there is only a small amount of carrier molecule **A** in the cell at any one time. [1]

NADH donates electrons to the electron transport chain and is re-oxidised to form NAD;

(b) Describe how the energy released from the flow of electrons in Fig. 7.1 results in the formation of ATP. [3]

1. Electrons passed down electron carriers in order of electronegativity releases energy which is used to pump H^+ from the matrix to the intermembrane space;
2. This produces a proton motive force / H+ concentration gradient, which allows H^+ to diffuse through ***ATP synthase**** back to the matrix;
3. This diffusion of H^+ through ATP synthase is coupled to the phosphorylation of ADP to form ATP by ***ATP synthase****;

(c) Identify molecule **B** and explain what happens at the end of the electron transport chain in order to form molecule **B**. [3]

1. Molecule B is water;
2. Oxygen serves as the ***final electon acceptor**** of the electron transport chain;
3. And accepts electrons and protons to form water;

(d) Suggest how the flow of electrons in photophosphorylation is different from the flow of electrons shown in Fig. 7.1. [3]

	photophosphorylation	oxidative phosphorylation
Electron donors	Water is the electron donor in the non-cyclic pathway while Photosystem I is the electron donor in the cyclic pathway;	NADH and $FADH_2$ are the electron donors to the first electron carrier of ETC;
Electron acceptor	$NADP^+$ is the final electron acceptor in the non-cyclic pathway while Photosystem I is the final electron acceptor in the cyclic pathway;	Oxygen is the final electron acceptor (and it combines with H^+) and is reduced to water;
Electron transport chain	2 electon transport chains involved;	1 electron transport chain;
Pathway of electrons	Linear for non-cyclic and circular for cyclic;	Linear;

N17P2Q8

(a) Name the structures labelled **F** and **G** in Fig. 8.1. [2]

F Ligand
G Receptor

(b) Explain how the specificity of the response is determined in different cells. [2]

1. Different cells have different intracellular signaling proteins;
2. Hence different signaling pathways are activated to produce different responses;

(c) Describe how each protein kinase in the cascade is activated. [1]

Each protein kinase is activated by phosphorylation;

(d) Explain how the signalling cascade shown in Fig. 8.1 amplifies the signal during transduction. [2]

1. Each step results in an increase in the number of activated molecules;
2. For example active protein kinase 1 is able to phosphorylate and activate many protein kinase 2, which can go on to activate even more protein kinase 3;

(e) Suggest how mutations in genes coding for the proteins shown in Fig. 8.1 could lead to cancer by altering signalling pathways. [3]

1. Mutations in genes could be ***gain-in-function mutations**** which would code for proteins that have an increased function;
2. For example, gene coding for protein kinase 1 could be mutated such that protein kinase 1 is always active, without requiring the activated relay molecule;
3. This could result in cells dividing excessively, without presence of appropriate signals, causing uncontrolled cell division;
4. Alternatively, mutations could also be ***loss-of-function mutations****, coding for proteins which are non-functional;
5. For example, gene coding for protein kinase 1 could be mutated such that protein kinase 1 cannot activate protein kinase 2;
6. This could result in cells being unable to trigger apoptosis or DNA repair, resulting in uncontrolled cell division;

N17P2Q9

(a) (i) Describe how the total number of plant species varies with island size, as shown in Fig. 9.2. [3]

1. As island size increases, the total number of plant species also increases;
2. Eg. 1000 species on island 200 000 km^2 to 2500 species on islands 800 000 km^2
3. Some exceptions/anomalies exist: island of 75 000 km^2 has same numebr of species (8000) as island of 100 000 km^2 or smaller island 150 000km^2 has 1200 species, which is more than a larger island of 200 000 km^2 with 1000 species;

(ii) Suggest an explanation for the relationship that you have described in (a)(i). [2]

Larger/Bigger islands have (any 2)

1. greater no. of habitats;
2. Greater variety of habitats/environments/named example;
3. More available niches;

More unique environments for selection to occur and hence more species;

(b) Islands often have many unique species of plants and animals that are not found anywhere else. Explain why this is so. [5]

1. Islands are ***geographically isolated**** as they are surrounded by water that acts as a physical barrier preventing interbreeding. This results in the ***disruption of gene flow****;
2. The islands due to their differing habitats / environments, present many niches for species to fill – idea of adaptive radiation;
3. e.g. of differences. Soil type – sandy or clayey, availability of water, availability of shade, plant types;
4. Thus (common) ancestral species on different islands were exposed to ***different selection pressures**** and natural selection act on them;
5. There exist variation in population and those with favourable traits are better adapted and have a selective advantage to the local conditions and will be selected for, increasing frequency of favourable alleles / and will survive, reproduce and pass on their alleles to the next generation;
6. As different sub populations evolved independently of each other, their allele frequencies changed as they accumulated different genetic mutations, and were subjected to ***genetic drift**** and ***natural selection****.
7. Over a hundreds and thousands of generations, each population on the different islands became ***reproductively isolated****;

N17P2Q10

(a) Describe how the molecules of IgG attach to the viral capsid. [2]

1. Ig G has an antigen binding site/variable region that is;
2. Complementary in shape and charge to the epitope on the antigen of the virus, allowing it to recognise and bind to the antigen;

(b) Using information shown in Fig. 10.1 and Fig. 10.2, explain how the attachment of IgG prevents the virus from infecting the cell. [3]

1. Binding of IgG to the antigen causes the neutralization of the antigen and
2. prevents the antigen/virus from binding to the host cell surface receptor on the host cell membrane and
3. prevents receptor mediated endocytosis from occurring;
4. Binding of IgG to the antigen also allows for ***opsonisation****, recruiting macrophages / phagocytes via the Fc region;
5. increasing the frequency of ***phagocytosis**** by macrophages/phagocytes

N17P2Q11

(a) Describe the change in the percentage cover of live corals shown in Fig. **11.1**. [3]

1. From mid1994 to the end 1997, there was a gradual increase in the percentage of coral cover from about 42 to 50%;
2. From the end 1997 to mid 1998, there was a sharp decrease in the percentage of coral cover from 50% to about 10%;
3. From mid 1998 to mid 2010, there was a gradual increase in coral cover from about 10% to 45%;

(b) Suggest why the density of herbivorous fish increases after the death of many of the corals in 1998. [1]

This is because there were more algae growing on the dead coral → more food for herbivorous fish.

(c) Suggest how the increase in herbivorous fish helps to restore the percentage cover of live corals over several years. [1]

Fish eat the algae on the corals and allow corals to recover