



Victoria Junior College
Biology Department
2021 Prelims H2 Paper 2 – Proposed Answers

1 (a) Outline how the enzymes are packaged into vesicles and release to the outside of the cell. [4]

1. Enzymes synthesised in rough endoplasmic reticulum (rER) are packaged into transport vesicles pinched off from rER and fuse with cis/forming face of Golgi apparatus;
 2. At Golgi apparatus, modification, sorting and packaging of enzymes occur;
 3. Secretory vesicles (A: Golgi vesicles) containing enzymes buds off trans/maturing face of Golgi apparatus;
 4. Secretory vesicles move to and fuse with cell surface membrane, releasing the enzymes via exocytosis;
- [Points 5 and 6 – 1m max]
5. Vesicles move towards cell surface membrane with the help of microtubules;
 6. ATP is required for exocytosis;

(b) Explain how the structure of the vesicle allows it to serve its function shown in Fig. 1. 1. [3]

1. Structure of vesicle: made up of a phospholipid bilayer;
2. Phospholipids (PL) provides fluidity, which allows for fusion of vesicle membrane with cell surface membrane for release of enzymes to the outside of the cell;
3. The hydrophilic phosphate heads of the PL interact with the aqueous environment/cytoplasm and within the vesicle for stability;
Or hydrophilic phosphate heads of the PL interact with the aqueous environment of the cytoplasm to allow for transport of vesicles within the cell;
4. Hydrocarbon tails of the phospholipids forms the hydrophobic core of the membrane prevents the enzymes from moving out of the vesicles while they are being transported;
5. AVP;

(c) Suggest and explain one modification to the vesicles to allow them to deliver the drugs to specific cells. [2]

- Modification to vesicle [Any one below]
 - Add glycoproteins/ glycolipids add to the phospholipids on the outer surface of the vesicle;
 - Attach carbohydrate specific epitopes/ antigen to the phospholipids on the outer surface of the vesicle;

which will recognise and bind to specific receptors found only on the specific cells by complementary shape;
- How uptake can take place:
(binding of glycoproteins on vesicle to specific receptors on specific cell results in)
receptor-mediated endocytosis/ endocytosis/ fusion, allowing uptake by cells;

- (d) Describe two differences between the release of enzymes shown in Fig 1.1 and the process in which flu virus leave the host cells. [2]

Release of enzymes	Process which flu virus leave the host cells
<p>[Any 1 of the following]</p> <ul style="list-style-type: none"> • <u>Exocytosis</u> • involves fusion of vesicle membrane with cell surface membrane • <u>Addition</u> of cell membrane 	<ul style="list-style-type: none"> • <u>Budding</u> • Involves evagination of host cell membrane; • Involves <u>removal</u>/loss of cell membrane;
<p>R: Budding vs no budding, fusion vs no fusion, loss vs no loss of membrane etc – when there is an equivalent process that occurs, students should describe the process.</p>	
<p>[Any 1 of the following]</p> <ul style="list-style-type: none"> • Vesicle can exit / leave / fuse at any part of the cell membrane <p>Any idea of non-specific sites vs specific sites/ do not require specific glycoproteins for exit vs require specific glycoproteins for exit.</p>	<ul style="list-style-type: none"> • Viruses leave the host cells a specific exit points that have haemagglutinin and neuraminidase / glycoproteins embedded;
<p>R: release of viruses results in lysis of host cells whereas release of enzyme does not as there is no lysis. Host cells die when substantial amount of cell membrane is lost via budding.</p> <p>R: release of viruses kills the host cell as it depends on the extent of budding. Not all host cells will die.</p>	

2 Fig 2.1 shows the structure of a natural triglyceride.

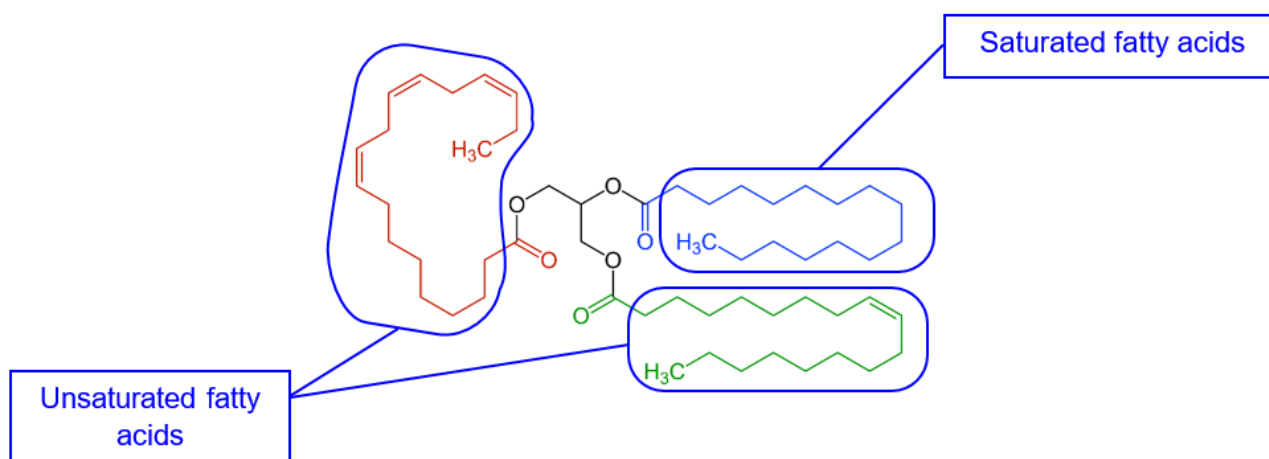


Fig. 2.1

- (a) (i) On Fig. 2.1, circle and label all the saturated and unsaturated fatty acid chains. [2]

1. Correct circle and label of saturated fatty acid chain;
2. Correct circle and label of unsaturated fatty acid chains;

Or

3. Correct circle for saturated and unsaturated fatty acid chains
4. Correct label of saturated and unsaturated fatty acid chains

(ii) Explain how the structure and properties of triglyceride is related to its role in living organisms. [3]

1. 3 long hydrocarbon chains/ large number of carbon-hydrogen bonds for energy store/ which can be oxidised to provide energy/ form ATP;
2. The hydrocarbon chains are also hydrophobic / non-polar which enables the molecule to be insoluble in water such that it will not affect the water potential of the cells;
3. Being non-polar / dehydrated means triglyceride can be packed compactly within cells as energy store;
4. AVP – maximum two
Large number of hydrogen atoms which yields metabolic water upon oxidation;
Less dense than water which provides buoyancy to aquatic mammals;
Good heat/ thermal insulator which prevents excessive heat loss in animals/ keep aquatic mammals warm;

(b) Triglycerides, such as the one shown in **Fig. 2.1**, can be catalysed by lipase. **Fig. 2.2** shows the reaction.

With reference to Fig. 2.2, explain the mode of action of lipase. [4]

1. Lipase catalyses the hydrolysis of triglyceride to form fatty acids and glycerol;
2. Binding of triglyceride to the active site of lipase causes lipase to have a slight conformation change, resulting in the triglyceride fitting more snugly in the active site;
Ref. to the induced-fit hypothesis;
3. Formation of the enzyme-substrate complex lowers the activation energy of triglyceride hydrolysis;
4. By placing the ester bond between glycerol and a fatty acid chain is placed under physical stress;
5. And catalytic residues in the active site alter the distribution of electrons within the ester bond;

3(a) With reference to Fig. 3.1, describe one difference and two similarities between the organization of polypeptide chains in keratin and collagen. [3]

- (i)
- difference – 3 polypeptide chains wound around each other to form a tropocollagen vs 2 polypeptide chains assemble to form each dimer;
 - similarities – aggregation of fibrils to form fibres in both keratin and collagen;
 - staggered arrangement of tropocollagen (collagen) and the keratin dimer (keratin) to form fibril;

(ii) Suggest how the structural organisation of keratin enable it to fulfil its function. [2]

- linear structure increase surface area for intra-chain bonds to form between chains / aggregation of fibrils into fibres → confer high tensile strength and ability to withstand stress;
- staggered arrangement – remove presence of weak areas in the fibril;
- no available OH-groups for interaction with H₂O → insoluble in water;

(b)(i) Explain the significance of the amino acid sequence to the function of collagen. [4]

- rich in amino acids **glycine and proline** / Every 3rd amino acid in the chain is a glycine (smallest amino acid) / repeating sequence of gly-x-y → forming a kinked helix;

- idea of each polypeptide chain able to wind more tightly around each other to form tropocollagen;
- contains **modified** amino acids hydroxyproline and/or hydroxylysine;
- idea that the OH groups (of modified amino acids) allow for formation of H bonds between polypeptide chains → high tensile strength;
- presence of lysine residues allowing for covalent cross-links to be formed between tropocollagen;

Max 4

(ii) Suggest why assembly of collagen cannot take place in the cytoplasm. [1]

- after assembly, it will be too large to pass through cell membrane (phospholipid bilayer) directly;

4(a) With reference to Fig 4.1 and your knowledge, suggest how phosphorylation of condensins and lamins prepares the cell for mitosis. [2]

- Phosphorylation changes conformation of condensins/ activates condensins, allowing for chromatin to pack more tightly/ condense into distinct chromosomes;
- Phosphorylation changes conformation/stability of lamins / deactivates/inactivates lamins, resulting in the breaks down / disintegration of the nuclear membrane / envelope into small membrane vesicles;

(b) Outline briefly the role of the microtubules in the formation of clones in mitosis. [3]

1. Microtubule based centrioles/ centrosomes are microtubule organizing centre for the assembly/ formation of spindle fibres;
2. Kinetochore microtubules / spindle fibres are attached to the kinetochore complex on the centromeres of chromosomes;
3. (depolymerisation of the microtubules) Allow the equal separation of sister chromatids to opposite poles/ prevent non-disjunction of chromosomes, leading to formation of genetically identical daughter cells/ nuclei that contain the same number and type of chromosomes as the parent nucleus/cell;
4. Nonkinetochore microtubules / spindle fibres that extend from one pole to the opposite pole are responsible for elongating the parent cell leading to cell division after telophase to form genetically identical daughter cells

For mp3 & 4, must mention forming clones, i.e., genetically identical cells at least once.

(c) Using the data in Table 4.1, identify with reason, an oncogene and a mutated tumor suppressor gene.

(i) Oncogene [2]

- Gene R;
- relates substitution to a gain of function mutation;
- as one copy is sufficient to produce a hyperactive/ constitutively active protein that sends signals to the nucleus to stimulate cell division;

Or

- Gene S;
- relates amplification of gene to a gain of function mutation;
- as multiple copies of the gene / greater number of protein products present

that will drive the cell towards cell division/ cause dysregulation in cell cycle control/ uncontrolled cell division;

(ii) Mutated tumor suppressor gene [2]

- Gene P
- relates homozygous deletion to a loss of function mutation;
- Both copies of the alleles for the gene has to be lost for the proteins that regulate the cell cycle control not to be produced;

Or

- Gene Q;
 - relates hypermethylation of the gene promoter to a loss of function mutation;
 - as it will lead to the gene being silenced;
- hence no protein products that control cell division are produced;

5(a) Explain the effect of the abnormal increase in CAG repeats on HTT protein structure and function. [3]

- [Structure] Production of an longer than the normal polypeptide due to an increase in glutamine residues;
- Alters primary structure of polypeptide and disrupts the R group interactions between amino acids that are far away/ name at least two different interactions such as hydrogen bonding, ionic, hydrophobic interactions and disulfide bridges;
- specific 3D shape/conformation/ tertiary structure of protein is changed and this leads to a non-functional protein;

R: answer that are vague eg. protein structure is affected/ protein function is affected as question asks for the effect of the abnormal increase on structure and function.

(b) Describe how splicing occurs with a normal HTT gene. [3]

- Several snRNP (small nuclear ribonucleoproteins) bind to the splice sites/ intron-exon boundary/ 'GU-' and '-AG' and complex with one another to form the spliceosome
- This brings the exons upstream and downstream (or exons 1 and 2) of the intron close together/ join together (this is mark under exon splicing) folding the pre-mRNA into a specific 3D conformation for splicing;
- Intron loops out/ form a lariat and is excised (to form the mature mRNA);

(c) Based on the information given, suggest how the mutant HTT protein can bring about the death of nerve cells. [2]

- Any valid suggestion on how HTT protein slows down protein synthesis – answer must target at ribosome since ribosome movement is in the text eg. HTT protein binds to ribosome, inhibits the formation of translation initiation complex/ inhibit peptidyl transferase, thereby inhibiting peptide bond formation, preventing ribosome translocation etc;
- Decrease/ No synthesis of critical/essential enzymes eg. respiratory enzymes decrease, resulting in death of nerve cells;

R: no synthesis of proteins necessary for nerve cells to function

6 In bacteria, there are two main types of operons: inducible and repressible operons

(a) Using a specific example, explain what is meant by a repressible operon. [3]

1. Example : Trp operon
2. In the absence of tryptophan/ under normal conditions: Trp operon is switched on for the transcription of structural genes for synthesis of tryptophan;
3. Presence of tryptophan: tryptophan acts as a co-repressor, binds to and activates repressor which binds to operator of Trp operon, prevent binding of RNA polymerase to promoter, hence no transcription of structural genes;

With reference to Fig. 6.1,

(b) (i) identify the graph that shows the light-inducible system. Explain your answer. [2]

- Graph X;
- Transcription of gene is induced / turned on by the presence of blue light & QV
- as mRNA abundance increase between 120 – 240 min and 360 – 480 min;

(iii) suggest how it is possible for DNA-binding protein EL222 to regulate the gene expression of two different operons. [3]

1. Blue light causes DNA-binding protein EL222 to change its conformation shape;
2. such that it acts as a repressor protein and is able to bind to the operator for the light-repressible system/ operon shown by Graph Y, preventing RNA polymerase from transcribing the genes downstream;
3. while in the light-inducible system/ operon shown by Graph X, it is an inactive repressor protein and unable to bind to the operator to stop transcription of genes downstream/ it acts as an activator protein to enhance the binding of RNA polymerase to the promoter for transcription of genes downstream;

(c) Eukaryotes are structurally different from prokaryotes and can exhibit differences in their **control of gene expression**.

Explain the **significance** of two such differences that occurs **in the cytoplasm** of the **eukaryotic** cell. [4]

Any 2

- [Control] Translational control through long polyA tail/ presence of 5' cap/ long half-life of mRNA;
- [Significance] to maintain stability of mature mRNA in the cytoplasm as templates for translation of more protein;
- [Control] Translational control through translational repressors binding to the 5' UTR of mRNA/ complementary RNA strand annealing to critical regions of the mRNA;
- [Significance] To prevent initiation of translation of mRNA whose proteins are not required by the cell;
- [Control] Post-translational control by named examples of, / biochemical modification of protein;
- [Significance] to activate/ inactivate proteins in response to appropriate signals/ control activity of synthesised proteins;

- [Control] Post-translational control by proteolytic cleavage;
- [Significance] to allow maturation of protein to enable it to be functional;
- [Control] Post-translational control by protein degradation via ubiquitinylation;
- [Significance] to control the amount of protein required by the cell;

7(a) Suggest how phenotype for pointed coats arises in cats. [2]

- Arise in cats which are homozygous recessive / have genotype *rr* / have both copies of the recessive allele coding for pointed coats;
- **Substitution** mutation leading to the recessive allele;
- Leading to a **non-functional enzyme** that cannot deposit pigment properly / deposit pigments in pointed pattern etc on cats' skin (ref idea of protein expression based on altered nucleotide sequence)

Max 2

(b) Explain using evidence from Fig. 7.1,

(i) the mode of inheritance for coat pattern in cats. [3]

- (Full colour coat is dominant / Pointed coat is recessive with explanation) Pointed is recessive because full colour parents (VIII) & (IX) gives rise to pointed offspring (XI) OR (II) & (III) cross give rise to heterozygous offspring who are all full colour;
- Identifying **autosomal** inheritance; (reject autosomal linkage)
- (Explanation for autosomal inheritance) Full colour male (III) gave rise to both full colour male (VIII) and female (VII) indicating that the *R* allele is passed down via non-sex chromosome / Full colour male (VIII) must have inherited the *R* allele from his full colored father (III) OR pointed male (XI) must have inherited the *r* allele from his full colored father (VIII);

(ii) that the "variable marker locus is thought to lie close to the *R/r* locus"? [2]

- **No crossing over** was observed at all throughout the pedigree tree;
- The same allele for coat pattern (*R/r*) OR *Hpa*II restriction site (+/-) was **always inherited together as one unit** with the same numerical value of marker allele;
- Reference fig: Marker allele 1, 2, 3, 4 always inherited along with *r* allele / Marker allele 6, 7 always inherited along with *R* allele;

Max 2

(c) Describe how one could determine the genotype of a cat showing the full colour phenotype. [2]

- Conduct a test cross with a pointed coat cat / Cross with a cat with genotype *rr* OR pure-breeding pointed coat cat;
- If $\frac{1}{2}$ offspring is pointed & $\frac{1}{2}$ offspring is full colour, the cat is heterozygous, but if all offspring is full colour, the cat is homozygous dominant for *R*;

8 (a) Using information from Fig 8.1, identify, with reasons, the factors that limit the rate of photosynthesis between X and Y. [3]

- Temperature and light intensity;

- Increase in temperature (2-10°C) causes increase in net rate of photosynthesis / uptake of carbon dioxide (QV: 0.9-1.5 mg g⁻¹h⁻¹);
- From X – Y, increase in light intensity from low to medium light causes an increase in net rate of photosynthesis (QV: from 0.9-1.5 mg g⁻¹h⁻¹ increase to 1.0-1.8 mg g⁻¹h⁻¹);

(b) Suggest an explanation for this difference. [3]

1. [Diff] For PS, the rate of reaction increases with increase in temp till a max and then decreases whereas for respiration, the rate increases with increase in temp;
A: QV
2. Enzymes involved in cellular respiration enzymes are maintained by more extensive bonds/ have more disulfide bonds;
3. hence they do not denature at higher temperatures/ are more resistant to high temperatures;

A: reverse explanation based on photosynthesis

(c) NAD and NADP are coenzymes that serve similar and yet different roles in respiration and photosynthesis respectively.

Compare the roles of NAD and NADP. [3]

Similarities:

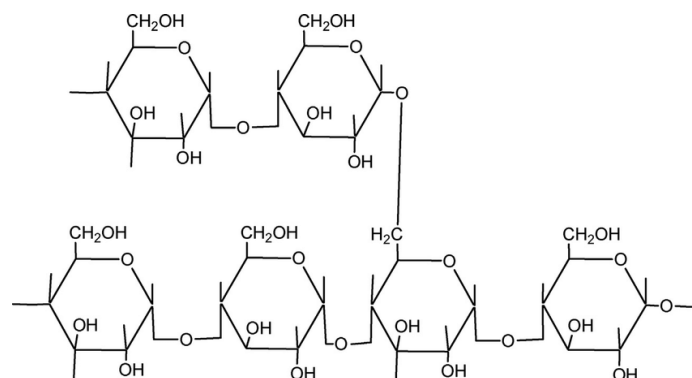
- Both can become reduced by accepting protons and electron;
- Both allow electron transfer to proceed in the electron transport chain;

Differences:

- [Role in accepting electrons & protons] NADP is the final acceptor of electrons and protons of non-cyclic photophosphorylation vs NAD accepts electrons and protons from the reduction/dehydrogenation of substrates (in glycolysis, link reaction, Krebs cycle etc);
- [Role in donating electrons & protons] Reduced NADP is used in Calvin cycle to reduce glycerate-3-phosphate to form triose phosphate vs reduced NAD carries protons and electrons to ETC on mitochondrial membrane to result in ATP synthesis;

(c) Starch synthesised in photosynthesis is composed of amylose and amylopectin.

In the space below, draw a representative section of the amylopectin. Label the bonds clearly. [3m]



1m – for correct structure of α-glucose;

1m – correct α-1,4 glycosidic bond

1m - correct α-1,6 glycosidic bond

9(a) Based on the data provided in Table 9.1, explain why skull size can be used to support the hypothesis that Population A and Population B belong to different species? [2]

- [Idea of] The average skull measurements of different species are different/ all smaller than both populations A and B Or each species of tortoise has a distinct average skull size / skull size that is different from other species;
- The skull of population B is consistently larger than that of population A – QV at least one data for position 1 – diff is 20 mm;

(b) (i) State how you would expect the data in Table 9.2 to differ if Population A and B belongs to the same species. Explain your answer. [2]

[Any 2 below]

- Sequence divergence should be close to or less than 1.4% or close to zero;
- Same species, greatest similarities in terms of sequences as they share the same recent common ancestor and would have accumulated similar mutations;

R: Population A and population B should show similar sequence divergence as all the 4 groups shown do show similarities in their sequences, it is the extent to this similarities that we should be looking at.

- Sequence divergence between population A and the other 2 species should be very similar /the same as the that between population B and the other 2 species;

(ii) Explain why information about the sequence divergence provides more information about evolutionary relationship than skull size. [3]

- Differences in skull size is only one characteristic compared to many different non-coding regions in sequence divergence;
- [Idea of] Skull size – may be affected by environment and environmental effects are not inheritable; Or
- Similarities in skull size may be a result of adaptations to similar niches/environments or convergent evolution;
- Sequence divergence shows comparison between species that allows for objective determination of differences between species as smaller the difference, the more related the 2 species are/ share a more common recent ancestor;
Or sequence divergence more accurately quantify the closeness between species to a common ancestor;
- Whereas for skull, measurements taken do not allow for determination of phylogenetic relationship between different species/ subjective in determining the extent of differences between skull size to all for classification into different species;
- Non-coding regions are not expressed and hence mutations are neutral/not selected for or against;
- Thus can remain and accumulate within the individuals/ populations from generations to generations;
- If mutation rate is constant, can use to determine the time of divergence;

(c) Suggest an explanation for the relationship shown. [2]

- As generation time increases, the rate of evolution decreases exponentially/ decreasing gradient;
- [Idea of] (longer generation time) so less reproduction/ less generations for a fix period of time;

- reference to fewer mutations, less chance of evolution due to lower genetic variation;

10(a) State precisely the type of cell and where it is located in the human body. [1]

- (developing/ immature) B cell in bone marrow;
Reject plasma cell

Examiner's Comments:

A number of students are not aware of the location of somatic recombination with answers ranging from thymus to lymphoid organs to blood. By the time naïve B cells circulate in the bloodstream, somatic recombination would have already taken place.

(b) Explain why Process Q is essential to maintaining the health of an individual. [3]

- Q is somatic recombination;
- Random assembly of different combinations of V, D, J gene segments (in immunoglobulin heavy chain) enables the diversifying of antigen-binding specificities of B cell receptors;
- Idea of creating repertoire of receptors that can recognise any potential pathogen → protection against external threats;

Examiner's Comments:

Most students focus on the diversity of antibodies produced by B cells which are able to recognise the antigens/ pathogens to eliminate them but this is the result of clonal expansion and selection which includes class switching and somatic hypermutation. The immediate outcome of somatic recombination is the production of naïve B cells with receptors that can potentially recognise any pathogens that a person may encounter in one's lifetime.

(c) Besides the location, describe two differences between process Q and class switching. [2]

Process Q	Class switching
Occurs during maturation process of B cell/ in developing B cells	Occurs after activation of B cell (by cytokines from helper T cell)/ in activated B cells
Involves changes to variable region/ antigen binding site	Involves changes to constant region
result in changes to antigen binding specificities	result in changes to effector function

Examiner's Comments:

Most students who did the question were aware of the differences between the 2 processes.