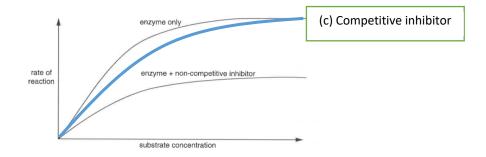
# 2016 H2 Biology P2 9744/02

- (a) Explain why, in the reaction with the enzyme only, as substrate concentration increases:
   (i) the rate of reaction increases at first [2]
  - 1. As <u>substrate concentration increases</u>, *frequency of effective collisions*\* between <u>enzyme</u> and <u>substrate</u> molecules <u>increases</u>;
  - Rate of <u>enzyme-substrate complex</u>\* formation <u>increases</u> and rate of reaction increases as <u>active sites</u>\* of enzymes are <u>readily available</u> and substrate concentration is limiting;
  - (ii) the rate of reaction becomes constant. [2]
    - <u>All active sites</u>\* of enzymes are <u>saturated</u> with substrate at any one point in time;
       <u>Concentration of substrate is no longer limiting</u> and <u>enzyme concentration is limiting</u>, rate of reaction will remain constant (graph plateaus).
  - (b) Explain why in Fig 1.1, the addition of a non-competitive inhibitor causes the reaction to become constant at a lower rate. [2]
    - 1. Inhibitor binds to site other than active site and changes conformation\* of active site\*;
    - 2. Hence inhibitor effectively <u>decreases availability of enzymes</u> as it forms an enzyme-inhibitor complex;
    - 3. Effects of inhibition cannot be overcome by increasing substrate concentration;
  - (c) Draw, on Fig. 1.1, the approximate shape of the curve if a competitive inhibitor were added to the enzyme instead of a non-competitive inhibitor. [2]



- 1. Correct shape and labelled;
- 2. approaches maximum rate of reaction;
- (d) Suggest why the penicillin molecule is an effective inhibitor of transpeptidase. [2]
  - 1. Cell wall peptide and penicillin are <u>similar in conformation</u> and hence <u>both are</u> <u>complementary in shape and charge to the active site</u>;
  - 2. Penicillin can act as a *competitive inhibitor*\*for the *active site* \*of transpeptidase;
  - 3. Hence, cell wall peptides cannot bind to active site and cross-links of cell wall peptides cannot form.
- **2** (a) Identify the molecules labelled **A** and **B** on Fig. 2.1. [2]
  - A: messenger RNA
  - B: RNA polymerase (R: general transcription factor as the molecule is unzipping and separating the double helix DNA)
  - (b) Explain how the control elements shown in Fig. 2.1 influence transcription. [4] *Promoter*

- serve as <u>recognition site</u> for the <u>binding of general transcription factors</u>\* and <u>RNA</u> <u>polymerase\*</u>;
- 2. to initiate transcription\*;
- 3. has critical elements, *TATA box*\* that determines the precise location of transcription start site;
- 4. has critical elements, <u>CAAT and GC boxes\*</u> to <u>improve efficiency of promoter</u> by <u>recruiting general transcription factors</u>\* and <u>RNA polymerase\*</u> to promoter.

#### Enhancer

- 5. when bound with <u>specific transcription factors\*</u> known as <u>activators</u>\*, promotes assembly of <u>transcription initiation complex</u>\*
- when bound with <u>specific transcription factors</u>\* known as <u>activators</u>\*, may <u>recruit</u> <u>histone acetyltransferase</u>\* and <u>chromatin remodeling complexes</u>\* to <u>decondense</u> <u>chromatin</u> (increase accessibility of promoter to general transcription factors and RNA polymerase)
- 7. increase frequency of transcription
- (c) Describe the role of a silencer control element in transcription. [2]
  - 1. allow <u>binding</u> of <u>specific transcription factors\*</u> called <u>repressors\*</u> by <u>preventing</u> <u>assembly</u> of <u>transcription initiation complex\*</u> at <u>promoter</u>
  - when <u>bound</u> with <u>specific transcription factors</u>\* known as <u>repressors</u>\*, may <u>recruit</u> <u>histone deacetylase</u>\* and <u>chromatin remodeling complexes</u>\* to <u>condense chromatin</u> (decrease accessibility of promoter to general transcription factors and RNA polymerase)
  - 3. decreases the frequency of transcription;
- (d) Describe a feature of the control of prokaryotic transcription that is not shown in Fig. 2.1. [2]
  - 1. <u>Genes coding for proteins</u> involved in <u>same biochemical pathway</u> usually clustered together on <u>one operon.</u>
  - 2. If the *repressor*\* is active, it <u>binds to the *operator*\*</u>
  - 3. and <u>prevent the **RNA polymerase**</u>\* from binding to the <u>promoter</u> thus preventing transcription

[Points 4 and 5 are no longer in the syllabus]

- Formation of <u>RNA polymerase holoenzyme\*</u> which is made up of <u>core RNA polymerase</u> and a <u>sigma factor\*</u>; which <u>recognizes and binds</u> to <u>both</u> the <u>-10 sequence/Pribnow box\*</u> and <u>-35 sequences</u>
- 5. The <u>more similar</u> the <u>-10 and -35 sequences</u> are to the <u>consensus sequences</u>, the stronger the promoter and therefore the <u>higher frequency of transcription</u>.
- **3** (a) Describe the changes shown in Fig. 3.1. [3]
  - 1. Upon infection, the number of HIV viruses increase to a peak/maximum at week 3 and decline steeply to near 0 at week 8.5;
  - 2. From the week 9 to the year 6 (since primary infection), viruses remain relatively constant near to 0 with 2 small peaks at year 2 and year 4;
  - 3. From 6 to 8 years after infection, viruses increase steeply to a large number and plateau from year 8 to 10;
  - 4. The T helper cells decrease steeply from initial infection to week 3 and rises and remain relatively constant till year 3;
  - 5. From 4 to 9 years, the T helper cells decrease gradually to near to 0;
  - 6. From 9 to 10 year, the T helper cells remain constant near to 0;
  - (b) Suggest how the changes in the number of T helper cells shown in Fig. 3.1 would affect the health of an untreated HIV-infected individual over the course of the infection. [3]
    - 1. T helper cells help to <u>activate</u> specific <u>naïve B cells into plasma B cells</u> to <u>make antibodies</u> for antibody-mediated response;

- 2. the HIV infects more and more T helper cells, the levels of <u>T helper cells lower</u> from 4 to 9 years to near to <u>0</u>, as the infected <u>T cells are destroyed</u>;
- 3. The increasing loss of T helper cells leads to impaired immune responses in the affected individual who then becomes increasingly <u>susceptible to opportunistic diseases;</u>
- (c) Why is HIV described as a retrovirus? [2]
   1. A retrovirus is a <u>RNA virus</u> that duplicate via <u>reverse transcription</u> in the host cell;
   2. using the <u>RNA</u> genome as a <u>template</u>, <u>reverse transcriptase</u>\* produce DNA from its <u>RNA</u> genome by complementary base pairing;
- (d) Explain why viruses are described as obligate parasites. [2]
  1. An obligate parasite is an organism that cannot live independently of its host;
  2. They require the host cell to complete their life cycle and reproduce;
- 4 (a) Identify structures A, B, C and D, as shown on Fig. 4.1. [4] A : mRNA
  - B : subunits of ribosomes
  - C : transport vesicle
  - D : membrane of the rough endoplasmic reticulum (RER)
  - (b) Use Fig. 4.1 to suggest how the structures labelled **B** attach to the endoplasmic reticulum through the growing polypeptide. [2]
    - 1. <u>Molecules</u> called signal recognition particle (SRP) bind to one <u>end of growing polypeptide</u> <u>at ribosome;</u>
    - 2. The molecules bind to receptor protein on membrane of B, to bring ribosomes to it;
  - (c) Suggest why the newly synthesised polypeptides shown in Fig. 4.1 cannot pass directly into the cytosol. [1]
    - 1. Ribosome is attached to the membrane of RER where there is a <u>membrane channel</u> for the synthesized polypeptide to enter;
    - 2. Polypeptide folds into its 3D <u>conformation</u> in the lumen and is <u>too large</u> to <u>pass through</u> the <u>transient pores</u> of the <u>phospholipid bilayer of the membrane/ through the membrane</u> <u>channel;</u>
  - (d) Describe what happens to the newly synthesised polypeptides released from the rough endoplasmic reticulum. [3]
    - 1. Polypeptides are enclosed in a transport vesicle;

2. It <u>fuses</u> with cis/ convex face of <u>**Golgi apparatus**</u>\* and undergoes <u>chemical modification</u>/ <u>eg: glycosylation;</u>

3. Golgi apparatus <u>targets and sorts</u> the polypeptides and <u>secretory vesicles</u> are released from trans face;

4. The secretory vesicles *fuse*\* with cell membrane and release polypeptides via exocytosis;

**5** (a) Draw a genetic diagram to explain the results of the first cross in which all offspring had purple stems and green leaves. [3]

Parental phenotyp Parental genotype		X X	White stems, green leaves nnGG
Correct parental pl	nenotype + genotype [1]		
Correct gametes [1	1]		
Meiosis Gametes	Ng	X	nG

Random fertilization

F1 genotype F1 phenotype **NnGg** Purple stems, green leaves

Correct offspring phenotype + genotype [1]

- (b) Explain the significance of the chi-squared value for these results. [4]
  - 1. P > 0.90
  - Since p > 0.05, at a level of significance of 5% we do not reject the null hypothesis that expected results are similar to the observed results;
  - 3. The difference between the observed and expected phenotypic ratios is <u>not significant</u> <u>difference</u> and is <u>due to chance</u>;
  - 4. Inheritance of these genes follows Mendel's laws of independent assortment, random segregation and dominance.
- (c) Suggest the advantages of using Fast Plants, instead of conventional crop varieties, as sources of useful mutations that can then be introduced into broccoli and cabbage through cross-breeding. [3]
  - 1. <u>Larger number of random new mutations</u> that can result from fast plants (while in cross-breeding, there is typically only reshuffling of combinations of alleles)
  - 2. Fast plants have <u>increased cycles of cell division</u>, which could be due to <u>mutations</u> in <u>tumour suppressor genes</u>
  - 3. (Mutations in tumour suppressor genes means that) when there are further random mutations that arise, <u>cell cycle will not be halted for DNA repair</u> resulting in <u>accumulation of further mutations</u>

## 6 Out of syllabus, from 2017 onwar

- 7 (a) Suggest how *N. diardi,* of Borneo and Sumatra, evolved as a separate species from *N. nebulosa,* found in the rest of Asia. [3]
  - When <u>ancestors of N. nebulosa</u>, occupied <u>Sumatra and Borneo</u>, they were <u>geographically</u> <u>isolated</u>\* due to <u>presence of sea</u> from other sub-populations of N. nebulosa and <u>could no</u> <u>longer interbreed</u> with them and hence <u>gene flow was disrupted</u>\*;
  - <u>Different niches</u> in Sumatra and Borneo presented <u>different selection pressures</u>\* and so individuals with <u>favourable traits</u> and hence a <u>selective advantage</u> were <u>selected for</u>, survived and reproduced, <u>increasing frequency of favourable alleles</u>;
  - Over time the different sub populations *N. nebulosa* <u>evolved independently of each other</u>, their <u>allele frequencies changed</u> as they <u>accumulated different genetic *mutations\**</u>, and were subjected to <u>genetic drift\*</u> and <u>natural selection</u>\*.
  - Over a long period of time, this led to <u>reproductive isolation</u>\* where each population could no longer interbreed to produce viable, fertile\* offspring.and formation of *N. diardi*, a new species (i.e. macroevolution) occurred;
  - (b) Suggest why they are not regarded as separate species. [2]
    - They are not regarded as separate species as they are capable of <u>interbreeding</u>\* and producing <u>fertile, viable offspring\*</u>;
    - 2. and they are *reproductively isolated*\* from other species;
    - 3. usually have similar morphological, physiological and behavioural features;
  - (c) Explain whether the data in Fig. 7.2 provide sufficient evidence on their own for the existence of two separate species of *Neofelis*. [2]
     From the data, it can be seen that *Neofelis diardi* and *Neofelis diardi* 1. speciated about 1.4 million years ago from a common ancestor;

- 2. and have the <u>same genus name but different species name</u>/specific epithet, suggesting that they are <u>different species;</u>
- 3. However, only by <u>checking if they can interbreed and produce fertile viable offspring</u> will there be sufficient evidence that 2 separate species of *Neofelis* exist.
- (d) Describe the advantages of using DNA sequence data in constructing phylogenies such as that shown in Fig. 7.2. [3]
  - 1. The DNA sequence data is *objective*\*. <u>Molecular character states are *unambiguous*\* as A, C, G and T are easily recognisable and cannot be confused;</u>
  - The DNA sequence data is <u>quantitative</u>\* and is <u>easily converted to numerical form</u>\* and hence are amenable to mathematical and <u>statistical analysis</u> and hence computation. The <u>degree of</u> <u>relatedness can be inferred and quantified</u> by calculating the nucleotide differences between species;
  - 3. The DNA sequence data can be used to <u>compare species which are morphologically</u> <u>indistinguishable</u> due to <u>convergent evolution</u>\* or are because they are <u>very closely related</u>
- 8 (a) Describe what is happening in:
  - (i) phase 1 [2]
  - 1. During carbon fixation, *carbon dioxide*\* combines with *RuBP\*;*
  - to form an unstable 6C compound that will immediately <u>split</u> to form 2 molecules of (3C) <u>glycerate</u> <u>phosphate\* (GP)</u>;
  - 3. Carbon fixation is catalyzed by enzyme, *<u>RuBP carboxylase/oxygenase</u>\** (Rubisco);
  - (ii) phase 2 [2]
  - 1. NADPH is the reducing power used to reduce\* GP to glyceraldehyde-3-phosphate (G3P);
  - 2. <u>ATP</u> is the source of energy required;
  - 3. <u>Triose phosphate/G3P</u> is the <u>first sugar</u> formed in photosynthesis and the end product of Calvin cycle;
  - (iii) phase 3 [2]
  - 1. 5 molecules of <u>G3P are used to **regenerate**\*</u> 3 <u>RuBP</u> so that the cycle of carbon dioxide fixation can continue.
  - 2. This requires 3 <u>ATP;</u>
  - (b) Light is not a factor that directly limits the rate of the Calvin cycle but it is needed for the Calvin cycle to continue.

Outline why light is needed for the Calvin cycle to continue. [2]

The products from light reaction, <u>ATP</u> and <u>NADPH</u> are required for the <u>reduction\* of GP to G3P;</u>
 <u>ATP</u> is also used in the <u>regeneration of RuBP\*;</u>

(c) State two environmental factors that can directly limit the rate of the Calvin cycle and explain how they act. [2]

factor 1: <u>Low carbon dioxide concentration</u> (0.04%) in atmosphere explanation: low carbon dioxide concentration <u>decreases</u> frequency of <u>effective collisions</u>\* between <u>CO<sub>2</sub>, ribulose bisphosphate (RuBP) and rubisco</u> thus, <u>decrease</u> <u>enzyme-substrate</u> <u>complex</u>\* formed per unit time, lowering rate of carbon fixation, which limits rate of Calvin cycle.

factor 2: <u>Low temperatures</u> (winter in temperate countries) explanation: low temperature, leads to <u>lower kinetic energy</u> of <u>CO<sub>2</sub>, ribulose bisphosphate (RuBP)</u> and rubisco, decreases frequency of **effective collisions**\* between them thus, <u>decrease **enzyme**</u>-<u>substrate complex</u>\* formed per unit time, lowering rate of carbon fixation, which limits rate of Calvin cycle.

9 (a) Describe the different functions of proteins incell surface membranes. [7]
 1. Allow *facilitated diffusion*\* of *polar/charged*\* molecules or ions (solute) across the membrane;

- 2. Via transmembrane <u>channel proteins</u>\* that has a <u>hydrophilic pore</u> through which a <u>specific</u> <u>molecule</u> can diffuse
- 3. Or via *carrier proteins*\* where <u>changes in conformation</u> upon solute binding results in the solute being transported to the other side of the membrane, and released ;
- Assist in the <u>active transport</u>\* of polar/charged molecules or ions across the membrane via <u>pumps</u>\*/ carrier proteins, <u>against concentration gradient</u> using <u>ATP</u>\* to drive the active process; (pumps preferred)
- For transport of <u>molecules in large quantity</u> <u>against concentration gradient</u>, <u>receptor-mediated</u> <u>endocytosis</u>\* occurs where specific ligands bind to receptor proteins on the membrane causing invagination of membrane;
- 6. Membrane proteins interact with the <u>extracellular matrix</u> on the <u>exterior side</u> and the <u>cytoskeleton</u> on the <u>cytoplasmic side</u>, <u>maintaining the shape of the cell</u> and to <u>fix the location</u> of some <u>membrane proteins</u> such as receptors and transport proteins;
- 7. As a <u>receptor protein</u> which a specific ligand will bind to. The formation of the ligand-receptor complex will initiate an intracellular signaling cascade for <u>signal transduction [learn in cell signaling]</u>;
- Some proteins which are <u>enzymes</u> that carry out <u>catalytic/chemical reaction</u> at cell membrane e.g. adenylyl cyclase that catalyses conversion of ATP to CAMP/ acetylcholinesterase on postsynaptic membrane [learn in cell signaling];
- When combined with carbohydrates, glycoproteins are excellent markers for <u>cell-cell</u> <u>recognition</u>\* due to their diversity. This enables distinguishing <u>self from non-self</u> and is crucial for immune cell recognition and <u>immune response</u>;
- 10. For <u>cell-cell adhesion</u> where cells of the same type to form tissues/ membrane protein of adjacent cells may hook together to form various kinds of junctions such as tight junctions or gap junctions;

*R* : Act as electron carriers (e.g. cytochromes) which are part of the energy transfer systems that are utilised during photosynthesis and respiration (question asked for cell surface membrane)

- (b) Describe the process of endocytosis. [6]
  - 1. Endocytosis is a process of *bulk transport*\* which involves bringing in <u>macromolecules or</u> <u>molecules in large quantity</u> into the cell;
  - 2. <u>ATP</u>\* is needed for the <u>rearrangement of the cytoskeleton</u> that causes infolding or extension of the cell membrane;
  - 3. For macromolecules, there is *phagocytosis*\* where *pseudopodia*\* are formed and extended outwards to engulf the <u>large insoluble macromolecule/solids</u>;
  - 4. When the ends of the *pseudopodia* **fuse**\*, a <u>vesicle / vacuole</u> containing the solid matter is pinched off and enters into the cytoplasm;
  - 5. There is also *pinocytosis*\* which is to bring in <u>tiny *vesicle*\* of liquid</u> when a small area of the plasma membrane <u>invaginates</u>. Substances brought in are not specific;
  - For transport of molecules in large quantity <u>against concentration gradient</u>, <u>receptor-mediated</u>\* <u>endocytosis</u> occurs where <u>specific</u> ligands bind to receptor proteins on the membrane causing <u>invagination</u>\* of the membrane;
- (c) Outline the functions of membranes within cells. [7]
  - 1. Due to the <u>hydrophobic core</u>\* of phospholipid bilayer, membranes <u>serve as a selectively</u> <u>permeable barrier</u> to movement of <u>ions, polar, charged and large</u> molecules;
  - 2. Within a cell, membranes allow compartmentalization to occur;
  - 3. This allows the formation of <u>unique environments for specialized activities</u> such as <u>enzyme</u> <u>reactions</u> in lysosomes where an optimal pH is required;
  - 4. For the <u>establishment of proton gradients</u> within specialized organelles such as mitochondria and chloroplasts for <u>chemiosmosis</u> and formation of <u>ATP</u>;

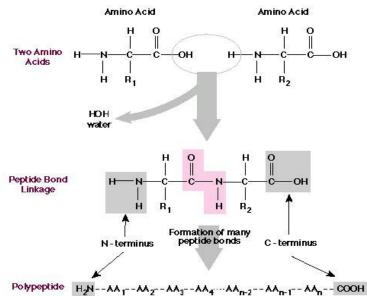
- 5. Membranes within cells allow <u>localization of proteins</u> of <u>related function</u> together so that sequential biochemical processes are facilitated;
- 6. E.g. Enzymes and proteins are grouped together into photosystems II and I on thylakoid membranes of chloroplasts facilitating electron energy transfer (A: electron carriers of electron transport chain)
- 7. <u>Increase surface area</u> for <u>chemical reactions</u> such as the <u>highly folded cristae</u> of <u>mitochondria</u> increase surface area for insertion of electron transport carriers and ATP synthase complexes for <u>oxidative phosphorylation</u> to take place;
- 8. Site for <u>protein synthesis</u> at *rough endoplasmic reticulum*<sup>\*</sup> where translation takes place at ribosomes at RER membrane;
- 9. Allows storage of food sources such as starch in amyloplasts;
- **10** (a) Explain how different types of bonding hold protein molecules in shape. [7]

### 1. Hydrogen bond\*;

- 2. Oxygen (e.g. O of <u>C=O group</u>\*) and nitrogen (e.g. N of -<u>NH group</u>\*) are electronegative ( $\delta$ -). Hydrogen of -NH or -OH group is electropositive ( $\delta$ +).Electropositive and electronegative atoms form hydrogen bonds;
- 3. lonic bond\*;
- 4. Formed between **oppositely**-<u>*charged*</u>\* <u>*R* groups</u>\* of amino acids;
- 5. Hydrophobic interaction\*;
- 6. Formed between hydrophobic *<u>non-polar</u>\* <u><i>R* groups</u>\* of amino acids;
- 7. <u>Disulfide bond</u>\* / bridge
- 8. Formed only between two <u>cysteine</u> amino acids by oxidation of <u>sulfydryl (-SH)</u> groups, which contains <u>sulphur</u>\*;
- 9. In collagen, <u>covalent cross-links</u>\* form between <u>lysine</u>\* residues at C and N ends of <u>adjacent/parallel tropocollagen molecules;</u>

#### Examples of shapes:

- 10. <u>Secondary structure</u>\*: <u>α-helix or β-pleated sheet</u> held in place by <u>hydrogen bonds between</u> <u>C=O and N-H groups of polypeptide backbone;</u>
- 11. <u>Tertiary structure</u>\*: <u>single</u> polypeptide chain further <u>extensively folded</u> and bended into specific conformation held in place by <u>hydrophobic interactions</u>, <u>ionic bonds</u>, <u>disulfide bridges</u> and <u>hydrogen bonds</u> formed between R groups of amino acids;
- 12. <u>Quaternary structure</u>\*: association of two or more polypeptide chains held together by <u>same</u> four types of interactions involved in tertiary structure;
- (b) Describe how amino acids are joined together. [6] *Note: If diagram is drawn, it should be properly annotated.*



1. Amino acids undergo condensation\* to form polypeptides with removal of 1 water molecule;

- 2. <u>OH group from carboxyl group</u> and <u>H atom from amino group</u> contribute to formation of water molecule (showing OH and H in diagram coming together);
- 3. Box up and label *peptide bond*\*;
- 4. Correct structure and label of amino acids;
- 5. Box up and label amino terminus and carboxyl terminus;
- 6. <u>Polypeptide</u> formed from condensation;
- 7. This reaction is *catalysed*\* by enzyme, *peptidyl transferase*\*, at *ribosomes*\*;
- (c) Outline the structure of haemoglobin and relate this to its function. [7]
  - 1. Quaternary structure of 4 <u>polypeptide</u> subunits: <u>2  $\alpha$ -globin</u>\* subunits and <u>2  $\beta$ -globin</u>\* subunits;
  - 2. each subunit is arranged so that most of <u>hydrophilic</u> amino acid side chains are on external surface while <u>hydrophobic</u> amino acid side chains are buried in interior;
  - 3. makes it <u>soluble in water/aqueous environment</u>, can be transported and carry O<sub>2</sub> from lungs to tissues vice versa;
  - 4. each subunit is made of <u>globin polypeptide</u> and a prosthetic (non-protein) component called <u>haem group\*;</u>
  - 5. each haem group consists of a *porphyrin ring*\* and an *iron ion (Fe*<sup>2+</sup>)\*;
  - <u>Fe<sup>2+</sup></u> of <u>haem group binds temporarily to O<sub>2</sub></u>, so <u>1 Hb molecule can carry up to 4 O<sub>2</sub></u>, at a time forming oxyhaemoglobin; (ref. <u>transport oxygen</u> in blood)
  - <u>4 subunits</u> held together by <u>intermolecular interactions formed between R groups</u> (hydrogen bonds, ionic bonds and hydrophobic interactions A: no disulfide bonds), allows movement that influences <u>affinity for oxygen</u> allowing for <u>cooperative binding</u>\* of oxygen;
  - 8. As a result binding of one oxygen molecule to one haemoglobin subunit induces a <u>conformational change</u> in remaining 3 subunits so that <u>affinity for oxygen increases</u>;