2022 H2 Paper 2

Section A

Answer all questions

1(a) Outline the main features of the cell theory. [3]

1. The cell is the smallest unit of life ;
2. All known living organisms are made up of cells ;
3. All cells come from pre-existing cells ;

(b) **(i)** Complete Table 1.1 to show the name and function of the structures labelled **A, B**, **C** and **D** in Fig. 1.1. [4]

label	name	function
A	mitochondrion	Site of ***aerobic respiration**** where ***ATP**** synthesis occurs;
B	nucleus	Contains DNA where ***transcription**** occurs to produce ***mRNA****;
C	***Golgi**** body/apparatus	Modify lipids and proteins by ***glycosylation**** to form glycolipids and glycolipids respectively; To sort and package proteins into different vesicles and target the proteins to different parts of the cell or for secretion out of cell;
D	Rough endoplamic reticulum	***Translation**** of mRNA to protein/ protein synthesis by bound ***ribosomes****; ***Glycosylation**** of proteins; Transportation of proteins in transport vesicles to the Golgi apparatus;

(ii) Compare the process of asexual reproduction in yeast with binary fission in bacteria. [2]

1. Similarity – both result in 2 ***genetically identical**** daughter cells;
2. Difference – budding involves the ***evagination**** of the cell surface membrane to form a daughter cell but binary fission involves the ***invagination**** of the cell surface membrane to separate the 2 daughter cells;

[Total: 9]

2 (a) With reference to Fig. 2.1, describe the effect of increasing lopinavir concentration on the inhibition of HIV protease. [3]

1. As the concentration of lopinvar increased, the percentage inhibition increased;
2. From 0.0 to 0.4 $\mu moldm^{-3}$ lopinavir concentration, there is a gradual increase in percentage inhibition, from 0.0 to 8.0%;
3. From 0.4 to 1.6 $\mu moldm^{-3}$ lopinavir concentration,there is a steep increase in percentage inhibition from 8.0 to 97.0%;
4. From 1.6 to 2.4 $\mu moldm^{-3}$ lopinavir concentration, the pecentage inhibition starts to plateau from about 97.0 to 99.0%;

(b) Explain how lopinavir inhibits HIV protease. [4]

1. Lopinavir has a 3D conformation that is complementary in shape and charge to the ***active site**** of HIV protease;
2. and lopinavir has a similar shape as the substrate/polyproteins;
3. Lopinavir competes with the substrate/polyproteins for the active site of HIV protease and prevents the substrate from binding to active site and hence reduces the rate of reaction;
4. The binding of the inhibitor to the active site is not permanent/reversible;
5. However, at high substrate concentration, the effect of the inhibitor can be overcome and Vmax can be reached;

(c) Translation of viral RNA in cells infected with the human immunodeficiency virus (HIV) results in large polypeptides known as polyproteins. HIV protease cuts each of these viral polyproteins into smaller molecules.

Suggest how inhibition of HIV protease may limit the ability of HIV to reproduce. [3]

1. If HIV protease activity is inhibited, then viral polyproteins cannot be cleaved;
2. Hence proteins such as reverse transcriptase, integrase, protease and capsid proteins cannot be synthesised;
3. Fully functional virions cannot be produced and the ability for HIV to reproduce will be limited;

[Total: 10]

3 (a) Name the structures labelled **P, Q** and **R** in Fig. 3.1. [3]

P enhancer
Q promoter
R structural gene/ coding region

(b) Compare transcription in prokaryotes and eukaryotes. [4]

Feature	Prokaryotes	Eukaryotes
Binding of RNA polymerase	RNA polymerase binds directly to the ***promoter**** via the sigma factor;	RNA polymerase is recruited by ***general transcription factors**** to bind to the ***promoter**** forming the ***transcription initiation complex****;
Genes transcribed	Transcription of several genes in an operon controlled by one promoter;	Transcription of one gene controlled by one promoter;
mRNA formed	***Polycistronic**** mRNA is formed;	***Monocistronic**** mRNA is formed;
Upregulation of transcription	In lac operon, cAMP binds to ***catabolic activator protein**** (CAP). Active CAP binds to ***CAP binding site**** in the promoter region and increase affinity of ***RNA polymerase**** binding to the ***promoter****, thus increasing the frequency of transcription;	***Activator**** binds to ***enhancer**** causing bending of spacer DNA and promoting the assembly of the transcription initiation complex which will increase frequency of transcription;
Downregulation of transcription	Binding of ***repressor**** to ***operator**** prevents the RNA polymerase binding to the promoter and hence preventing transcription. E.g. lac repressor bind to operator in lac operon/ trp repressor bind to operator in trp operon;	Binding of ***repressor**** to ***silencer**** will prevent the assembly of the transcription initiation complex which will decrease frequency of transcription;

(c) Explain how the introduction of mRNA into the cytoplasm of adult cells can result in the production of iPSCs **and** why these iPSCs are footprint free. [4]

How introduction of synthetic mRNAs can produce iPSCs:

1. ***Translation**** of the synthetic mRNAs will produce proteins which are needed to
2. switch off certain genes in specialised adult cells allowing them to de-differentiate;
3. and switch on expression of certain specific stem cell genes allowing the adult somatic cells to achieve pluripotency;

Why are these iPSCs footprint-free:

4. The synthetic mRNAs do not enter the nucleus and will not intergrate with the genomic DNA – they do not modify the genome of the adult cells;
5. The synthetic mRNAs are degraded after a while and will not have a trace inside the cells;

[Total: 11]

4(a) Explain how the end replication problem arises during DNA synthesis **and** describe the consequences of this problem. [5]

1. Occurs during replication of **linear** DNA.
2. Each round of DNA replication will result in the shortening of daughter molecules at the **5' ends of the telomeres**.
3. because ***RNA primers* at the 5'end*** is removed, but **DNA polymerase** is unable to replace with DNA; (idea of end replication problem)
4. result in overhangs at the **3' end of the telomeres**.
5. However, since telomeres are non-coding, this ensures that vital genetic information/genes are not lost / eroded with each round of replication;
6. If the telomeres shorten to critical length, cell will be signalled to stop dividing;

(b) **(i)** With reference to Fig. 4.1, describe the role of the short piece of RNA that forms part of the telomerase complex. [3]

1. 5 nucleotides of the telomerase RNA anneals and forms ***complementary base pairs**** with the single-stranded overhang at 3' end of the telomere;
2. which aligns the telomerase reverse transcriptase with respect to the DNA;
3. The telomerase RNA then serves as the ***template**** for formation of a complementary DNA sequence;
4. whereby ***adenine**** pairs with ***uracil****, ***thymine**** with ***adenine****, ***cytosine**** with ***guanine****, and ***guanine**** with ***cytosine****;
5. Resulting in tandem repeat sequences;

(ii) With reference to Fig. 4.1, describe the activity of the reverse transcriptase part of the telomerase complex. [3]

1. Telomerase has an ***active site**** that is ***complementary in conformation and charge**** to a ***specific**** telomeric DNA sequence;
2. Using telomerase RNA as a ***template****, telomerase reverse transcriptase forms a complementary ***DNA**** sequence through ***complementary base pairing****; (whereby adenine base pair with uracil, thymine with adenine, cytosine with guanine, and guanine with cytosine)
3. Catalyzes the formation of ***phosphodiester bonds**** between deoxyribonuceotides;
4. translocation in the 5' → 3' direction to produce a series of tandem repeats of GGTTAG, thus elongating the telomere/DNA at the 3' overhang;

[Total: 11]

5(a) **(i)** a loss of function mutation of the tumour suppressor gene *p53.* [2]

1. loss-of-function mutation in both alleles/two copies of p53 results in a non-functional gene product/ gene product/p53 protein not produced;
2. this results in the inability to inhibit cell cycle or inability to repair damaged DNA or inability to promote ***apoptosis****/***programmed cell death****;
   therefore cells with damaged DNA can continue to divide

(ii) a gain in function mutation of the proto-oncogene *ras*. [2]

1. ***Gain-of-function mutation**** such as ***proto-oncogenes**** to be converted to ***oncogenes****;
2. will result in overexpression of proteins/growth factors or production of hyperactive/degradation resistant proteins/growth factors or promote progression of cell cycle;
   therefore cells will proliferate

(b) Describe the process of angiogenesis and explain its importance in the development of cancer. [4]

1. angiogenesis is the process of forming new blood vessels in response to chemical signals given off by the tumour;
2. blood vessels are essential to transport blood with oxygen and nutrients to the tumour;
3. which are necessary for cell growth and cell division and hence the potential unlimited growth of the tumour;
4. blood vessels also provide a route for tumour cells to metastasise to other parts of the body to form secondary tumours;
   resulting in a malignant cancer

(c) Suggest **and** explain **one** reason why the recorded incidence of this cancer is rising. [2]

1. increased awareness and better diagnostic technology for breast cancer;
2. could have led to more cases being detected and therefore a steady increase in incidence detected from 25 cases per 100 000 women 1976 to 65 cases 2015;

OR

3. changes in lifestyle;
4. like poor diet and high stress levels working women (due to more women entering the workforce) led to higher incidence in breast cancer from 1976 to 2015;

OR

5. better healthcare and treatments for cancer patients;
6. allowed more women with breast cancer to have children and pass breast cancer causing alleles to their children;

OR

7. women bearing children at when they are older;
8. increases likelihood of accumulation of more mutations in cancer causing alleles within eggs;

Must quote data

[Total: 10]

6(a) Draw a genetic diagram to show the expected phenotypic ratio of the F2 offspring, assuming normal dihybrid inheritance.

Use the symbols **A** and **a** to represent the alleles for grain texture and **B** and **b** to represent the alleles for grain colour. [5]

Let **A** be the allele to code for smooth grain texture and **a** the allele to code for wrinkled grain texture while **B** is the allele that codes for purple grains and **b** is the allele that codes for yellow grains.

Parental phenotype	Smooth, purple grains	X	Wrinkled, yellow grains
Parental genotype	AABB	X	aabb
Gametes	(AB)		(ab)
F_1 phenotype	All smooth, purple grains		
F_1 genotype	AaBb		
Self-pollination of F_1 generation	AaBb x AaBb		

F_2 generation:

Gametes	(AB)	(Ab)	(aB)	(ab)
(AB)	AABB smooth purple grain	AABb smooth purple grain	AaBB smooth purple grain	AaBb smooth purple grain
(Ab)	AABb smooth purple grain	AAbb smooth yellow grain	AaBb smooth purple grain	Aabb smooth yellow grain
(aB)	AaBB smooth purple grain	AaBb smooth purple grain	aaBB wrinkled purple grain	aaBb wrinkled purple grain
(ab)	AaBb smooth purple grain	Aabb smooth yellow grain	aaBb wrinkled purple grain	aabb wrinkled yellow grain

F_2 genotypes:	**9 A_B_ :**	**3 A_bb :**	**3 aaB_ :**	**1 aabb**
F_2 phenotype:	9 smooth purple grain	3 smooth yellow grain	3 wrinkled purple grain	1 wrinkled yellow grain

1. Corresponding parental phenotypes and genotypes
2. All gametes drawn circled
3. Corresponding F_1 phenotypes and genotypes
4. Corresponding F_2 phenotypes and genotypes (in Punnett Square)
5. Correct phenotypic ratio

(b) One of the ears of maize resulting from self-pollination of the F1 plants had 216 smooth purple grains, 65 smooth yellow grains, 79 wrinkled purple grains and 21 wrinkled yellow grains.

A chi-squared test was carried out on these results to compare the expected number of each phenotypes with the observed number.

The calculated chi-squared value was 1.73.

The critical chi-squared value for these results at a probability of $p = 0.05$ is 7.81.

State **and** explain what can be concluded about inheritance of grain colour in maize from the chi-squared value of 1.73. [3]

1. The inheritance of grain colour in maize follows normal Mendelian monohybrid inheritance;
2. Since the $\chi^2 = 1.73$, it is less than the critical χ^2 value of 7.81,
3. and we do not reject the null hypothesis;
4. There is no significant difference between the observed and expected phenotypes in the offspring generation and any difference is due to chance only;

(c) Suggest how pure-breeding maize with smooth yellow grains can be obtained from the F2 grains by selection and breeding. [3]

1. Perform a cross between the smooth, yellow grain from the F_2 generation with ***homozygous recessive**** wrinkled yellow grains;
2. If all the offspring from the cross are smooth, yellow grains, then the original parent grain was a pure-breeding smooth, yellow grain;
3. If the offspring from the cross produced a ratio of 1 smooth yellow grain : 1 wrinkled yellow grain, then the original parent grain was not pure-breeding (i.e. Aabb);

[Total: 11]

7(a) Outline the role of each of the structures **K, L, M** and **N** in Fig. 7.1 in the conversion of light energy to chemical energy during the light-dependent reactions of photosynthesis. [6]

K – Photosystem II (PSII) harvests light energy from photons and emit electrons which is passed to L;
Photolysis of water* by water-splitting enzyme so that the electron emitted can fill the electron hole in PSII;

L – Electron carrier and protein pump which uses energy loss from electron flow to pump protons from ***stroma**** into the ***thylakoid space****;
Protons generate the proton gradient for chemiosmosis to occur;

M – Photosystem I harvest light energy from photons and emit electrons;
Electrons are passed to ***NADP reductase**** and it catalyses the formation of ***NADPH**** from NADP;

N – ***ATP synthase**** allows protons to pass through it from the ***thylakoid space*** into the ***stroma**** down a concentration gradient via ***chemiosmosis****;
The ***electron motive force**** generated by the flow of protons is used to catalyse the ***formation of ATP**** from ADP and Pi;

(b) Describe the role of reduced NADP in linking the light-dependent reactions to the Calvin cycle. [2]

1. NADPH synthesized in the light dependent reaction is needed for the Calvin cycle to occur, hence they link the 2 reactions;
2. In the Calvin cycle, ***glycerate phosphate**** is reduced to ***glyceraldehyde-3-phosphate**** *(3C). NADPH provides the reducing power for the reaction as high energy electrons are stored in it;

(c) Explain why oxygen is not produced during cyclic photophosphorylation. [2]

1. In cyclic photophosphorylation, energy from light will be absorbed by PSI and be channeled to P700 of PSI which will be excited and displace an electron that will be accepted by the primary electron acceptor Y and subsequently transferred to the middle of the first electron transport chain;
2. The electron is then transported down the ***electron transport chain**** (ETC) and is finally recycled back to PSI filling the electron hole hence there is no photolysis needed to emit electrons to fill the electron hole and no oxygen produced;

[Total: 10]

8(a) Name the structures labelled **V, W, X, Y** and **Z** in Fig. 8.1. [5]

V – ligand
W – G-protein coupled receptor
X – adenylyl cyclase
Y – cAMP
Z – relay protein/ kinase protein (A: protein kinase A)

(b) Outline the stages in cell signalling that occur **after** the initial stages shown in Fig. 8.1. [5]

1. Signal transduction is initiated when activation of the intracellular domain of the GPCR, upon ligand binding, will enable the inactive G protein nearby to bind to it;
2. G-protein is activated when is displaces its attached GDP for GTP;
3. The activated (α subunit of the) G-protein will translocate along membrane and bind to enzyme ***adenylyl cyclase**** and phosphorylate it, thus activating it;
4. Adenylyl cyclase will catalyze conversion of ***ATP**** to ***cAMP****;
5. Signal amplification begins from ***cAMP**** where the number of activated product is always greater than those in the preceding step as one moves down the pathway;
6. 4 cAMP bind to and activates the relay protein/kinase protein/ protein kinase A (PKA) causing it to detach into subunits;
7. The two activated subunits of the relay protein/kinase protein/ protein kinase A (PKA) will initiate a sequential activation of kinases resulting in a phosphorylation cascade during which further amplification of the signal will occur;
8. Which leads to the activation of glycogen phosphorylase which catalyses the breakdown of glycogen to glucose (i.e. glycogenolysis in liver & muscle cells)
9. During the cellular response, there will also be a decrease in the rate of glycolysis, and an increase in the rate lipid and protein breakdown;

[Total: 10]

9(a) With reference to Fig. 9.1, explain why the Sumatran, Tapanuli and Bornean orangutans are considered to be separate species. [2]

1. The Sumatran and Bornean orangutans have a larger difference in DNA characteristics for variable 2 compared to variable 1 while the Sumatran and Tapanuli orangutans have a larger difference in DNA characteristics for variable 1 compared to variable 2 and the Bornean and Tapanuli have a larger difference in DNA characteristics for both variables 1 and 2;
2. Due to the large differences in DNA characteristics, between all 3 species they are likely to have evolved from a common ancestor a very long time ago and accumulated many mutations over time and eventually formed different ***reproductively isolated**** *species* that are unable to interbreed to form ***fertile****, ***viable**** offspring;

(b) Explain how these three species of orangutan may have evolved from a common ancestor. [3]

1. When sea levels rose at the end of the ice age when ice melted, the ancestors of the orangutans became ***geographically isolated**** (A:a reproductive barrier formed). Thus the 3 sub-populations of orangutans were prevented from interbreeding and ***gene flow was disrupted****;
2. Different environments/niches presented ***different selection pressures**** and so individuals with favourable traits and were best adapted had a selective advantage and were selected for;
3. and survived and reproduced and passed on their favourable alleles to their offspring which led to an increasing frequency of favourable alleles;
4. As the 3 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****. Over a long period of time this led to ***reproductive isolation**** and formation of 3 distinct but closely related orangutan species (i.e. macroevolution occurred);

(c) Describe the advantages of using molecular methods to classify organisms. [3]

1. Nucleotide data are objective. Molecular character states are unambiguous as A, C, G and T are easily recognisable and cannot be confused;
2. Nucleotide data are quantitative. Molecular data are easily converted to numerical form and hence are amenable to mathematical and statistical analysis and hence computation. Degree of relatedness can be inferred and quantified by calculating nucleotide differences between species;
3. Nucleotide data can be used to compare species which are morphologically indistinguishable especially if they are very closely related;
4. As changes in nucleotide sequences accumulate over time with clockwork regularity. We can estimate the time of speciation of modern to ancient species;

[Total: 8]

10 (a) Using the information provided, calculate the total number of different antigen-binding sites for immunoglobulin molecules that are possible as a result of somatic recombination. [2]

(Number of possible κ chain x number of possible heavy chains) + (Number of possible λ chain x number of possible heavy chains)
= [(40 x 5) x (40 x 25 x 6)] + [(30 x 4) x (40 x 25 x 6)]
= 1 920 000

(b) Following infection or vaccination, there is a progressive increase in the affinity (binding ability) of antibodies for the antigen that has been introduced.

This progressive increase is called affinity maturation.

Explain how this process occurs. [3]

Somatic hypermutation / aka affinity maturation

1. **Activated B** cells divide rapidly by **mitosis** during ***clonal expansion****
2. some of these B cells can undergo somatic hypermutation, ***mutation**** occurs on the Ig genes Ig / gene that code for antibodies.
3. each of these B cells will acquire slight amino acid difference in the **variable regions** of Ig chains / antigen-binding sites of BCR on cell surface membrane).
4. The B cells that contains BCR with higher affinity for the antigen will go be selected to go through further ***clonal expansion**** and ***differentiation**** to form ***plasma cells****.

[Total: 5]

11 (a) Explain how increases in temperature and rainfall could lead to an increase in the number of cases of viral dengue disease. [2]

Increase in temperature

1. would lead to increased rate of enzyme catalysed reactions in the mosquito vector *Aedes aegypti*;
2. leading to faster development rate and decreased length of reproductive cycles and mosquito population increase and hence greater spread of viral dengue disease;

Increase in rainfall

1. may lead to more pools of stagnant water ;
2. which female mosquitoes lay eggs in and for the larval and pupal stages of the mosquito life cycle hence greater spread of viral dengue disease;

(b) With reference to Fig. 11.1, evaluate how effective the scientists' model is in predicting the number of cases of viral dengue disease. [3]

Effective

1. the model accurately predicted a peak exceeding the epidemic threshold of 192 cases of viral dengue disease from week 26 to week 32 as both the predicted and reported data exceeded the threshold in the same weeks;
2. The model accurately predicted the overall trend of low number of cases of around 80 cases of viral dengue disease from week 0 to week 10 and sharp increase from week 10 to around week 31 (A: sharp decrease with relevant data quoted);

Not entirely effective

3. The actual peak exceeded the predicted peak of 216 cases by 50 cases (actual peak was 266 cases) indicating the spread was more severe than predicted;
4. There were periods of inaccurate predictions e.g. predicted a peak in week 3, instead there was a minimum number of cases in week 5;

[Total: 5]

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