HWA CHONG INSTITUTION (COLLEGE SECTION)
2017 JC2 9744 H2 BIOLOGY
PRELIMINARY EXAMINATIONS PAPER 2 MARK SCHEME

STRUCTURED QUESTIONS

Question 1

(a)(i) Outline the cell theory. [max 2]

any two:
1. living organisms composed of cells
2. cells form most basic unit of life
3. cells arise from other cells

(a)(ii) With reference to Figure 1.1, explain how Pasteur's experiment supports the cell theory. [2]

1. description of what happens to the nutrient broth (ref to stimulus)
2. shows that cells must come from pre-existing cells

(a)(iii) Suggest a reason for the universal acceptance of the cell theory in our world today. [1]

any one:
1. tested / scrutinized by other scientists
2. reproducible
3. overwhelmingly supported by scientific community

(b)(i) State the name and chemical composition of the stuctures labelled **A** to **C**. [3]

	name	chemical composition
A	1a. cell wall	1b. peptidoglycan
B	2a. cell membrane	2b. phospholipids and proteins
C	3a. nucleoid	3b. DNA

(c) *P. syringae* can cause disease in the leaves of its host plant by secreting toxins and cell wall degrading enzymes, without causing harm to itself. Explain why this is so. [2]

1. ref to toxins affect the functioning of membranous organelles
2. ref to different composition of cell wall
3. ref to specific 3D conformation of enzymes

(d) State and explain which of the three types of interactions best describes the relationship involved in the endosymbiotic theory. [2]

1. mutualism
2. prokaryote produces ATP, host cell provides shelter / nutrients

[Total: 12]

Question 2

(a) With reference to Fig. 3.1,

(i) name the stage of mitosis shown. [1]

(late) anaphase

(ii) describe what is happening during this stage of mitosis. [2]

1. centromere divide, forming daughter chromosomes
2. migrate to poles, centromere leading
3. pulled by kinetochore microtubules

(b) Distinguish between multipotent cells and totipotent cells. [max 2]

1. multipotent more specialized
2. multipotent cells cannot give rise to organs but totipotent cells can
3. correct example of multipotent cell and totipotent cell

(c) Suggest why there might be a connection between the use of stem cells in treatment and cancer. [max 3]

1. stem cells may undergo uncontrolled cell division
2. telomerase active
3. risk of developing tumour where stem cells implanted
4. ref to exposure to carcinogens
5. appropriate ref in context to cancer critical genes

(d) State whether you agree **or** disagree that this is unethical **and** explain why you reached this decision. [3]

A1 agree
A2 human animal hybrid would not happen in nature
A3 ref to disrespect for human life
A4 few examples of success in medical applications
A5 may lead to abuse in future
A6 possibility of unforeseen consequences
A7 unnecessary / there are alternative techniques

D1 disagree
D2 the amount of non-human DNA is negligible
D3 protocol limits keeping 'embryo' to 14 days
D4 more ethical alternative to ESC
D5 provides more stem cells than possible from ESC
D6 can relieve human suffering
D7 idea of rejection at implantation

[Total: 11]

Question 3

(a)(i) State the identity of ligand **X**. [1]

glucagon

(a)(ii) Explain why ligand **X** cannot diffuse directly into the liver cell to trigger a cellular response. [2]

1. Ref to: glucagon as a large and hydrophilic molecule
2. Ref to: hydrophobic core of cell membrane

(b) With reference to Fig. 3.1, describe how the structure of GPLR enables it to function as a membrane-bound receptor. [3]

1. Hydrophilic amino acid residues on inter-helical loops are soluble in aqueous medium
2. Hydrophobic amino acid residues on transmembrane helices enable embedding of GPLR within cell membrane
3. Ref. to specific 3D conformation of binding sites

(c) Explain how the structure of glycogen is adapted to its function as an efficient storage biomolecule. [3]

Any three:
1. Ref. to α(1,4) glycosidic bonds that result in coiling
2. Ref. to α(1,6) glycosidic bonds that result in branching
3. Ref. to glycogen having several hundreds to thousands of glucose monomers
4. Ref. to anomeric carbon being involved in glycosidic bond formation
5. Ref. to glycosidic bonds being easily hydrolysed to release glucose monomers

[Total: 9]

Question 4

(a) Describe two structural differences between helices **A** and **B**. [2]

1. Ref to: double-stranded vs single-stranded
2. Ref to: deoxyribonucleotides vs made of amino acids
3. Ref to: phosphodiester bonds vs peptide bonds
4. Ref to: Hydrogen bonds formed between complementary bases vs hydrogen bonds formed between NH and CO groups

(b)(i) Describe how a primer strand is synthesised. [3]

1. Ref to: DNA template
2. Ribonucleotides form complementary base pairs with the DNA template
3. Ref to: formation of phosphodiester bonds.
4. Ref to: synthesis in the 5' to 3' direction

(b)(ii) With reference to Fig. 4.1, explain if the primer is priming the synthesis of the leading strand or lagging strand. [2]

Leading strand is synthesised towards the replication fork

(c) Explain the role of splicing in the structure and function of the two forms of IR. [3]

1. Ref to. different combinations of exons,
2. Ref to. different amino acid sequences and different specific 3D conformation of binding sites
3. Ref to. specific ligands IR-A and IR-B can bind, to different degrees / with different efficacy

[Total: 10]

Question 5

(a) With reference to Fig. 5.1, predict and explain the most likely mode of inheritance of G6PD deficiency. [3]

1. sex-linked recessive
2. all affected individuals in the family are males, indicating that males display disease phenotype more often than females as males are hemizygous
   OR
   approximately half of the sons of carrier females are affected, as every son has a 50% chance of receiving the X chromosome with recessive allele
3. unaffected parents can produce affected offspring such as II-1 / III-4 / IV-3 / IV-6, indicating that the mothers must have a dominant allele to mask the effect of the recessive allele
   OR
   if fathers are not affected, daughters will not be affected but may be carrier, as they will receive the X chromosome with dominant allele from their father

(b) Using suitable symbols, draw a genetic diagram to show the expected phenotypic ratio of the ABO blood group and G6PD production in offspring of II-3 and II-4. [6]

Let **D** be the dominant allele for production of G6PD
d be recessive allele for no production of G6PD
$\mathbf{I^A}$ be the (codominant) allele for production of A antigen
$\mathbf{I^B}$ be the (codominant) allele for production of B antigen
$\mathbf{I^O}$ be the recessive allele for no production of antigen

Parental phenotypes:	II-4 No G6PD deficiency, Blood group AB	x	II-3 No G6PD deficiency, Blood Group O	
Parental genotypes :	$X^DYI^AI^B$	x	$X^DX^dI^OI^O$	
Parental gametes :	X^DI^A X^DI^B YI^A YI^B		X^DI^O X^dI^O	

Random fertilization (as shown in the Punnett Square)

		male gametes			
		X^DI^A	X^DI^B	YI^A	YI^B
female gametes	X^DI^O	$X^DX^DI^AI^O$	$X^DX^DI^BI^O$	$X^DYI^AI^O$	$X^DYI^BI^O$
	X^dI^O	$X^DX^dI^AI^O$	$X^DX^dI^BI^O$	$X^dYI^AI^O$	$X^dYI^BI^O$

expected genotypic ratio	2 ($1X^DX^DI^AI^O$ + $1X^DX^dI^AI^O$) :	2 ($1X^DX^dI^BI^O$ + $1X^DX^DI^BI^O$) :	$1X^dYI^AI^O$:	$1X^DYI^BI^O$:	1 $X^dYI^AI^O$:	$1X^dYI^BI^O$
expected phenotypic ratio	2 normal, blood group A female :	2 normal, blood group B female :	1 normal, blood group A male :	1 normal, blood group B male :	1 G6PD deficient, blood group A male :	1 G6PD deficient, blood group B male

(c)(i) Using the information provided, calculate the χ^2 value for the observed results. Show your working clearly. [2]

$$\chi^2_{cal} = \frac{(99-90)^2}{90} + \frac{(155-180)^2}{180} + \frac{(106-90)^2}{90}$$

$$= 7.22$$

(c)(ii) Deduce if the observed results follow the expected phenotypic ratio of 1 blood group A: 2 blood group AB: 1 blood group B.

Explain your answer. [3]

1. No
2. Since $\chi^2_{calculated}$ (= 7.22) > $\chi^2_{critical}$ (= 5.99)
3. there is less than 5% probability that there is difference between observed and expected results is due to chance alone, indicating that the deviation is significant

[Total: 14]

Question 6

(a) Describe how the outer layers of bacterium **Y** differs from those of bacterium **X**. [2]

1. Y has an outer membrane / channel proteins which is / are absent in X
2. Y has a thinner peptidoglycan wall compared to X
3. X has the peptidoglycan wall exposed on the surface while Y has an outer membrane exposed on the surface / AW describing location of peptidoglycan wall

(b)(i) Identify the bacterium which turns purple when stained with the Gram stain. [1]

X

(b)(ii) Explain your answer to **(b)(i)**. [2]

1. Bacterium X is Gram positive as it has a thicker peptidoglycan wall
2. which helps to retain / trap crystal violet dye / prevent crystal violet dye from being washed away when alcohol is added, thus staining peptidoglycan wall blue / purple

(c) Explain the different effects of penicillin on bacteria **X** and **Y**. [3]

1. Penicillin is able to reach the peptidoglycan wall of X
2. where it binds and blocks transpeptidases, preventing formation of the cross-links between NAM residues in the transpeptidation step in cell wall synthesis in X
3. while the outer membrane of Y stops penicillin getting through to reach the peptidoglycan wall, thus penicillin exerts no effect on Y

(d) With reference to Fig. 6.2, describe and explain mode of infection of *V. cholerae*. [4]

1. The B subunit of cholera toxin binds to ganglioside receptor on the surface of the epithelial cells of the small intestine, enabling entry of cholera toxin subunit A via receptor-mediated endocytosis into the cell
2. Cholera toxin subunit A binds to and activates the G protein, resulting in one of the G protein subunits to dissociate and bind to adenylate cyclase, activating it
3. Activated adenylate cyclase generates high levels of intracellular cAMP from ATP, which activates the protein kinase
4. stimulating secretion of chloride ions, with associated sodium ions and water secretion, resulting in acute diarrhoea

[Total = 12]

Question 7

(a) Explain why influenza viruses can only attack the cells on the inside of the nose. [2]

1. Specific glycoproteins haemagglutinin on the viral membrane
2. They recognise and bind to sialic acid containing receptors on the membrane of the nose

(b) Suggest why enzymes **S** and **T** are needed at Stage 4. [2]

1. To synthesise (-) viral RNA using (+) sense RNAs as templates to be packaged into new viral particles as their nucleic acid using replicase / RNA-dependent RNA polymerase
2. For amino acids to bind to tRNA using host aminoacyl tRNA synthetases
3. For peptide bond formation between amino acids using host peptidyl transferase

(c) Suggest how enzyme **U** might catalyse the breakdown of the host cell membrane at Stage 5. [2]

1. Neuraminidase is a hydrolytic enzyme that breaks glycosidic bonds
2. The active site binds to and cleaves sialic acid residues on the receptor

(d) Most people in 1957 were susceptible to influenza caused by the new virus. Explain why. [4]

1. Antigenic shift
2. A sudden change in the antigenicity of a virus due to reassortment combination of the segmented virus genome with another genome of a different antigenic type
3. New viral strain has RNA segments 2, 4 and 5 from H2N2 avian virus and segments 9, 11, 14, 15 and 16 from H1N1 human virus
4. New combination of RNA segments causes the virus to change its 3D conformation of HA and/or NA
5. New 3D conformation of the new virus binds more effectively to receptors on the cells of lungs and airways of humans
6. New 3D conformation of the new virus cannot be recognised by antibodies / memory cells / B cells

[Total: 10]

Question 8

(a) Define *biological classification*. [2]

1. Organizing / arranging into groups of organisms / species
2. Ref to shared / morphological, characteristics / similarities / traits

(b)(i) Explain how multiple sequence alignment can be used in biological classification of the five genera of organisms. [4]

1. The DNA / amino acid sequence of a sample from the tissue of an organism of each genus
2. The aligned sequences of all five genera are homologous
3. The percentage similarity of sequences used to, estimate / establish, evolutionary relationship
4. The greater the degree of homology in the sequences, the more closely related the species are

(b)(ii) Identify the longest amino acid sequence where there are no differences amongst the five genera. [1]

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(b)(iii) Suggest, with a reason, whether the DNA coding for the amino acid sequence identified in **(b)(ii)** must be identical for the five genera. [2]

1. No
2. Degeneracy of the genetic code

(c) Describe what a cladogram represents. [4]

1. A type of phylogenetic tree
2. Inferred by shared derived characters
3. Shows the presence of clades
4. Ref to Fig. 8.3 to illustrate examples of clades for example

(d) State a reason each for illegal sale of the respective meat samples in Japan:

(i) Sample 1 [1]

It is from a North Atlantic population of whales

(ii) Sample 4 [1]

It is from a species that is not in the same clade as the, Minke / Humpback / Fin, whales

[Total: 15]

Question 9

(a) Outline **one** possible mechanism by which urushiol could enter the keratinocytes and Langerhans cells. [2]

1. endocytosis
2. (further detail) e.g. membrane invaginates

OR

1. diffusion
2. (further detail) e.g. across phospholipid bilayer across hydrophobic core of the cell membrane

(b)(i) Describe and explain the events that are likely to occur during an immune response to bring about poison ivy rash. [max 6]

1. Langerhans cell / keratinocyte / macrophage, as antigen-presenting cell
2. Ref to T-cells recognition through binding with complementary receptors
3. Activation of T-cells
4. Proliferation / mitosis of activated T-cells
5. T memory cell formation
6. Description of T-cytotoxic cell action
7. Explanation of faster response for second and subsequent contacts
8. T-helper cells secrete cytokine to stimulate, T-cytotoxic cell response/macrophages

(b)(ii) Suggest **one** reason why some people are not sensitive to skin contact with urushiol. [1]

Any one:

1. immunocompromised / described
2. may not have, specific T-cells / T-cells with, quinone / hapten, receptors
3. may have T-cells but low in number and not come across APC
4. may need several doses to build up sufficient numbers of T-cells
5. inability of cells to convert urushiol

[Total: 9]