2015

BIOCHEMISTRY

Paper — BCT – 201

(Cell Biology II)

Full Marks – 25

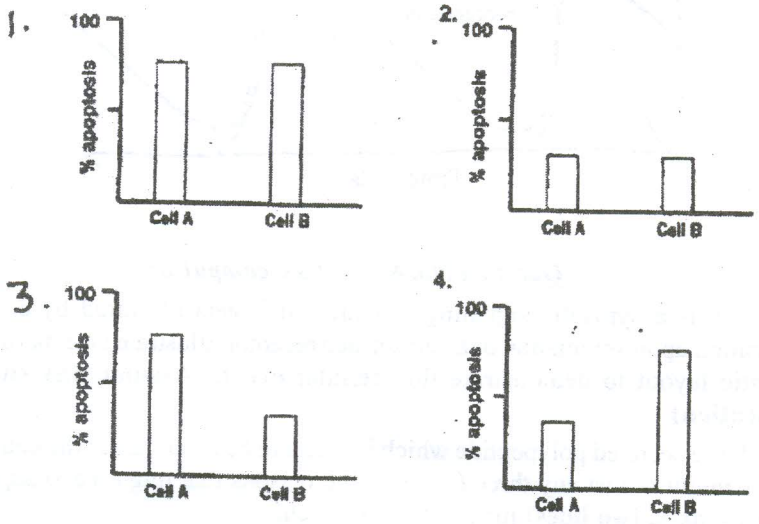
The figures in the margin indicate full marks

Candidates are required to give their answers in their own words as far as practicable

Question No. 1 is ***compulsory***

1. In an experiment, in cell A, cytochrome C is introduced by microinjection whereas in cell B, cytochrome C is introduced by microinjection, but Bax and Bak are inactivated. What will be the most appropriate apoptotic response in both cells ? Choose the correct answer from given figures with justification. 1+3

Figure of Q.1



2. Mice that lack a gene encoding MDM2 die at an early stage of development whereas mice lacking genes that encode both MDM2 and P53 (double knockouts) survive to adulthood but are highly prove to cancer. Explain. 2+2

Or

3. (a) Briefly explain the differences between autophagy and apoptosis. 2
   (b) What will be the fate of a cell population where *Rb* is absent ? 2
4. What do you suppose happens in mutant cells that
   (a) cannot degrade cyclins ?
   (b) always express high levels of p21 ?
   (c) cannot phosphorylate Rb ? 1+1+1

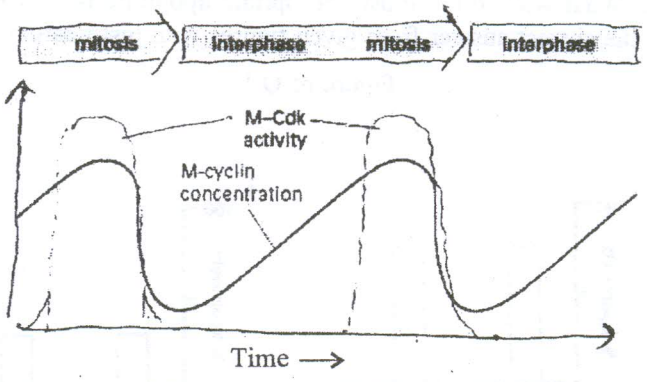
Or

5. When fused with an S-phase cell, cells in which of the following phases of the cell cycle will initiate DNA replication prematurely–G1? G2? M? Predict the effect of fusing a cell in G1 and a cell in G2 with respect to the timing of S phase in each cell. 1+2

6. Colchicine is a poison that binds to tubulin and prevents its assembly into microtubules. What effect do you think that Colchicine would have on cell division in animal cells ? 2

Or

7. The figure below shows the rise of cyclin concentration and the rise of M-Cdk activity in cells as they progress through the cell cycle. It is remarkable that the cyclin concentration rises slowly and steadily, whereas M-Cdk activity increases suddenly. How do you think this difference arises ? 2



Question No. 8 and ***9*** are ***compulsory***

8. X is a cytosolic signaling protein which gets activated by covalent modification upon stimulation of a cell surface receptor. Illustrate an experimental schematic layout to demonstrate this cellular event. **(Assume any suitable modification).** 3

9. S is a secreted polypeptide which is found to be stuck inside the cell under certain condition. List any three (3) plausible reasons that might have happened with brief **(max. two lines)** justification for each. 3

10. Illustrate schematically the attenuation of cytokine signaling that involve polyubiquitination. Name one biochemical tool which can be used to show polyubiquitination in a cell and is this an early or late event ? 2+½+½

Or

11. What is arrestin ? It is always present in a mammalian cell yet it does not show its activity always-briefly explains. 1+2

12. In the N-glycosylation, one step that occurs in the ER is reversible-why ? A treatment to a cell with a pharmacological agent rendered accumulation of anterograde but not retrograde vesicles-how would you prove this by an experiment ? **(Only layout, no detailing required)** 1+2

Or

13. Specific damage in the extracellular matrix stimulates fibroblasts-briefly justify. Cite an example where GPCR and cytosolic protein factors that belong to RTK pathway form a signaling cascade. 2+1