[This question paper contains 8 printed pages.]

Your Roll No.....

Sr. No. of Question Paper : 1052 **I**

Unique Paper Code : 2492013501

Name of the Paper : Molecular Cell Biology

Name of the Course : **B.Sc. (Hons.) Biochemistry**

Semester : V

Duration : 2 Hours Maximum Marks : 60

Instructions for Candidates

1. Write your Roll No. on the top immediately on receipt of this question paper.
2. There are **6** questions.
3. Attempt any **4** questions.
4. **All** questions carry equal marks.
5. Question no. **1** is compulsory.

P.T.O.

1. (a) Explain the following statements **(Any FOUR)** :

(i) Steroid hormones can act as transcription regulators.

(ii) Mutation in *KDEL* sequence of a resident ER protein may lead to its loss from the cell.

(iii) Cancer patients undergoing chemotherapy often need bone marrow transplantation.

(iv) DAG and IP_3 act as second messengers.

(v) Pre-sequences of mitochondrial proteins are positively charged whereas the transit peptides of chloroplast proteins are not.

(b) Discuss the contribution of following scientists :

(i) Tim Hunt

(ii) Gunter Blobel

(iii) Yoshio Masui and Clement Markert

(c) Write the biological functions of the following proteins :

(i) SNARE

(ii) PDE

(iii) STAT

(iv) Caspases (8,3,4)

2. (a) Explain the structure of G-protein coupled receptors and their mechanism of action with an example. How does intake of caffeine affect GPCR signaling?

(b) Comment on the following :

(i) Conventional chemotherapeutic drugs usually target all dividing cells, leading to common side effects like hair loss, nausea, and vomiting whereas oncogene- targeted drugs specifically act against cancer cells.

(ii) Treatment of cells with a drug that makes membranes permeable to protons, affect the function of lysosomes.

(c) Describe the events by which APC/c promotes the separation of sister chromatids at anaphase.

(6,4,5)

3. (a) Predict the effects of the following mutations on the ability of the cell to undergo apoptosis:

(i) Mutation in Bad such that it cannot phosphorylate protein kinase B.

(ii) Mutation in Bax such that it cannot form dimers.

(iii) Mutation in adaptor proteins such that it cannot form dimers.

(iv) Overexpression of Bcl-2.

(b) Explain the process of N-linked glycosylation of a secretory glycoprotein. What is the role of Dolichol phosphate in the synthesis of membrane glycoproteins?

(c) Explain the molecular mechanism that leads to cancer when Rb protein and p53 protein are inactivated by mutation. (4,6,5)

4. (a) Elaborate on the four major mechanisms of regulation of CDK activity during the cell cycle.

(b) Write the mechanism of action of the following drugs/inhibitors:

A. Chemotherapeutic drugs :

(i) *Herceptin* and

(ii) *Imatinib*

B. Inhibitors of intracellular signaling :

(i) *Sildenafil* and

(ii) *Phorbol esters*

(c) Explain the role of CDK2/cyclin-A complex in ensuring that the DNA is replicated only once per cell cycle in the S-phase. (5,4,6)

5. (a) With the help of diagram, explain the following :

(i) Role of BiP in post-translational translocation of protein into the ER lumen

(ii) Nitric oxide signaling cascade leading to vasodilation

(b) How do ATR and ATM proteins regulate the DNA damage checkpoint of the cell cycle?

(c) What is oncogene addiction? Why is this concept important for selecting molecular targets for cancer therapy? (6,5,4)

6. (a) Explain with a diagram, the steps involved in the progression of a genetically altered cell into a cancerous cell.

(b) Explain the differences between the process of autophagy and necrosis.

(c) Explain the Ras/MAPK signaling pathway and its activation. How does the dysregulation of this pathway lead to cancer? (5,4,6)