This question paper contains 5 printed pages

6859 *Your Roll No*

M.Sc. – Ph.D. Bio-Medical Sciences / III Sem. J

Paper— Bio-1004 Special Topics in Cell Biology and Molecular Oncology

Time : 3 hours *Maximum Marks* : 75

(Write your Roll No on the top immediately on receipt of this question paper)

There are two Sections, A and B Attempt Section A and Section B on separate answer sheets.

SECTION A

Attempt three questions in all. Question No. 1 is compulsory.

1. Attempt any *three* questions:

   (*a*) Enumerate the role of pRb in controlling transcription of genes including viral oncogenes during cell cycle progression and uncontrolled cell proliferation

   (*b*) How do telomere dynamics play role in cancer development?

   (*c*) How is Fluorescence Activated Cell Shorter (FACS) employed to study cell cycle?

   (*d*) What are cadherins?

P. T. O.

(*e*) How can miRNAs act as both oncogenes as well as tumor suppressor genes? 3×5=15

2. What are stem cells? Differentiate between a totipotent, pleuripotent, multipotent and unipotent cell. How can reprogramming of differentiated human somatic cells be made pleuripotent stem cells leading to development of patient and disease-specific stem cells including germ cells? 2+4+6=12

3. Define cell cycle With suitable illustrations describe cell cycle regulation in mammals and enumerate the differences with that of yeast. 2+7+3=12

4. Attempt any *three* of the following:

   (*a*) Explain mechanism of inhibition of topoisomerase inhibitors. Give an example of such a drug.

   (*b*) What is LOH? What is its role in cancer development?

   (*c*) What are cell cycle check-points?

   (*d*) What changes are required for an immortalized cell to turn metastatic? 3×4=12

5. Answer any *four* questions:

   (*a*) Why is DNA methylation used as a marker of cancer?

(*b*) Cancer cells, immortalized cells and stem cells—all have unlimited growth property and high telomerase activity but why are all not cancer cells? Justify your answer

(*c*) What is founder mutation? Why is it detected in familial cancer?

(*d*) How does cell differentiation occur in bacteria?

(*e*) What are VEGFs? How do they interact with receptors on the endothelial cell membrane?

(*f*) How can gene expression profiling help in discovery of molecular marker in cancer? 4×3=12

Section B

*Attempt **three** questions in all*
Question No. 6 is compulsory

6 (*a*) What are tumor suppressor proteins? Give any *four* examples.

(*b*) How does phosphorylation regulate the activity of p53?

(*c*) Give example of any *two* tubulin inhibitors. How do these bring about arrest of cell division?

(*d*) What are transgenics? What are advantages and disadvantages of transgenic animals?

P. T O.

(*e*) What are integrins?

(*f*) What are non-receptor tyrosine kinases? Give *two* examples 3×4=12

7 (*a*) A protooncogene CXN has functions as transactivator What experimental strategy will you use to check all the genes whose expression is modulated when CXN is overexpressed? Give the principle of the technique to be used to explain your answer

(*b*) We know that Ras is a GTP/GDP binding protein. A mutation resulted in constitutively active form of Ras How will you check whether this form binds to GTP/GDP or both or none? 6+6=12

8 What do you understand by Programmed cell death? Discuss critically the role of proapoptotic and antiapoptotic proteins as drug targets. 3+9=12

9. (*a*) p53 functions as a transrepressor or transactivator for gene expression regulation. How will you check (*i*) whether p53 binds to cyclin A promoter, (*ii*) whether p53 represses or activates cyclin A expression? Explain the principle of the technique used

(*b*) Cancer tissues generally show high gene expression of Receptor Tyrosine Kinases (RTKs).

How do RTKs function and what role do they play in cancer progression? Explain your answer taking a suitable example 6+6=12

10. (*a*) What is the role of ADF/cofilin and profilin in remodelling the cytoskeleton architecture?

(*b*) Explain the role of cdc42, Rac and Rho proteins during cellular migration

(*c*) What are MAPs and how are they regulated?

(*d*) Name various major classes of intermediate filaments along with their tissue-specific distribution and proposed functions. 4+4+2+2=12