Name : .....

Roll No. : .....

Invigilator's Signature : .....

CS/M.TECH/INT. PhD (Mol. Bio)/SEM-3/PHMB-303/2011-12

2011

SIGNAL TRANSDUCTION & ONCOLOGY

Time Allotted : 3 Hours *Full Marks* : 70

The figures in the margin indicate full marks.

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

GROUP – A

Answer any *two* of the following. 2 × 10 = 20

1. a) Discuss the components of cell cycle control system emphasising different check points and cycline-Cdk complexes. 6

   b) How does the inactivation of mitosis promoting factor (MPF) lead to anaphase and telophase ? 4

2. a) What are the collagens ? 2

   b) How are they characterised by their typical formation in extra-cellular matrix ? 4

   c) Show how from typical helix structure they assemble in regular staggered array to form fibrils. 4

3. a) What are the integrins ? 2

b) Draw their structure and discuss their function in relation to the attachment of cells to the extracellular matrix. 4

c) Fill in the blanks any *four* of the following : 4 × 1

i) The principal adhesion protein of connective tissue is ..... .

ii) The fibrous structural proteins of the extracellular matrix are embedded in gels formed from polysaccharides called ..... .

iii) A family of proteins known as ..... are typically activated in the early stages of apoptosis.

iv) Gap junctions are constructed of transmembrane proteins called ..... .

v) ..... is the only glycosaminoglycans that occurs as single long polypeptide chain.

4. a) What are cell adhesion molecules ? 1

b) Discuss with suitable sketches the leukocyte-endothelial cell interaction. 3

c) Write short notes on any *two* of the following : 2 × 3

i) Programmed cell death

ii) Fibronectin

iii) Gap junction.

GROUP – B

Answer any *three* of the following. 3 × 5 = 15

5. a) When an irreparable DNA damage is induced to cells by UV irradiation, how would p53, a transcription factor, suppress this damage ? Explain the consequences for the cells carrying a loss-of-function mutation for the p53 gene. 3

b) What are 'second messengers' ? Give suitable examples in a G-protein coupled receptor signaling. 2

6. a) Animal development is part depends on regulated proteolysis. Explain with a suitable example. $2\frac{1}{2}$

   b) Describe in brief how a signaling pathway would operate through the activation of cytokine receptors. $2\frac{1}{2}$

7. a) How would killer lymphocytes induce apoptosis of target cells ? $2\frac{1}{2}$

   b) Why would the loss of retinoblastoma (Rb) gene function lead to excessive cell proliferation and cause cancer ? $2\frac{1}{2}$

8. a) Name the gas that helps in signaling during smooth muscle contraction. Explain the mechanism in brief. $2\frac{1}{2}$

   b) Describe how bacterial chemotaxis is mediated by chemotaxis receptors. $2\frac{1}{2}$

9. The acivities of Cyclin-Cdk complexes that direct the cell-cycle progression are controlled by regulated proteolysis in a ubiquitin-dependent manner. Elucidate with suitable examples.

10. a) State briefly the different ways by which target cells become desensitized to signaling molecules. 3

    b) What is the mechanism of action of cholera toxin ? 2

11. a) How do prostaglandins participate in pain and inflammatory responses ? $2\frac{1}{2}$

    b) Differentiate between neurotransmitter and hormonal signaling. $2\frac{1}{2}$

GROUP – C

(ONCOLOGY)

SECTION – I

Answer *all* questions. $5 \times 1 = 5$

State *True* or *False* for the following :

12. 'Immortalized cells can grow indefinitely.'

13. 'During tumor development a single cell accumulates mutations sequentially.'

14. 'Invasive tumors are less aggressive than localized tumors.'

15. 'SV 40 large *T*-antigen inhibits p53 protein function.'

16. 'MDM2 stabilizes p53 in the cell.'

SECTION – II

Answer any *three* questions. 3 × 10 = 30

17. Elaborate the phrase 'Parallel Pathways of Cancer Development'. Justify the statement — 'Cancer cells show increased genomic instability'. Why a cancer cell does not require external growth signals ? 4 + 4 + 2

18. How was it known that early region of DNA tumor viruses is responsible for cellular transformation ? Why oncogenic retroviruses are replication defective ? Why an oncogenic receptor tyrosine kinase constitutively sends growth signals ?

3 + 3 + 4

19. Why tumor suppressor genes show recessive phenotype ? Flanking a putative tumor suppressor gene (TSG) are two microsatellite markers *A* and *B* with alleles *A*1, *A*2 and *B*1, *B*2. Assuming that a cancer patient is heterozygous at both the loci how a loss of heterozygosity analysis would predict it as a TSG locus ? Explain your interpretations with diagram.

2 + 8

20. Which experiment suggested that Chromosome Instability (CIN) phenotype may act dominantly at the cellular level ? What is Microsatellite Instability (MIN) phenotype ? Mutation in which genes are responsible for MIN phenotype ? What is Microsatellite Instability Assay ?

4 + 2 + 2 + 2

21. Suggest an experiment that will show that DNA from tumor cell can transform normal cell. How many different ways p53 is inactivated in a tumor cell ? 5 + 5