

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. $2 \times 10 = 20$

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.

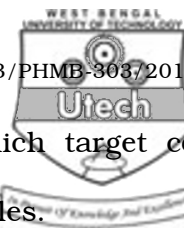
GROUP – B

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 activities 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)

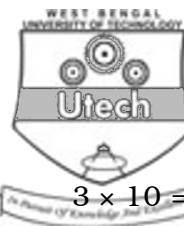
SECTION – I

Answer *all* questions.

5 × 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 ?

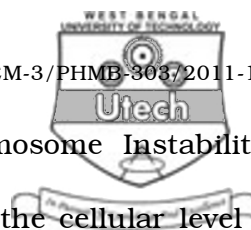
4 + 4 + 2

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

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