MASTER OF SCIENCE EXAMINATION, 2025

(1st Year, 1st Semester)

BIOTECHNOLOGY

PAPER : SC/BT/PG/131T

(Cell Biology)

Time : 2 *Hours* *Full Marks* : 40

GROUP—A

Answer *any* **three** questions, each carries **5** marks : 5×3=15

1. *(a)* What kind of membrane lipid would a bacteria synthesize in order to survive after changing the growth temperature from 20 ºC to 37 ºC? 2

(b) What are the significances for the presence of ar/R selectivity filter and NPA motif in the protein structure of aquaporin? 3 **[CO1]**

2. *(a)* Arrange the following substances according to their rate of diffusion across the lipid bilayer and justify it. 3
Chloride ion, phenylalanine, toluene, galactose

(b) How does the protein get transported from the Endoplasmic Reticulum (ER) to the Golgi bodies? 2 **[CO1]**

3. *(a)* What would be the consequence of using uncouplers on the rate of Electron Transport Chain (ETC) reaction and on ATP synthesis? 3

(b) How does glutathione molecule is able to predict the oxidative status of a cell? 2 **[CO1]**

4. *(a)* Write any two signaling events that get activated upon the induction of ER stress. 3

(b) How would you experimentally prove that ATP hydrolysis is required to keep the protein in an unfolded state for the incorporation of the protein into the mitochondria? 2 **[CO1]**

5. *(a)* Explain the technique by which the lateral diffusion of proteins in membrane can be followed and the diffusion rate be calculated? 3

(b) What kind of molecules do we see in the formation of lipid rafts and why? 2 **[CO1]**

GROUP—B

Answer *any* **two** questions : 5×2=10

6. State the differences between Desmoglein and Desmoplakin in terms of position and function. Which proteins form occluding junctions? State two primary functions of occluding junctions. Name any two intracellular anchor proteins. 2+1+1+1=5 **[CO2]**

19. How can you distinguish between a tumor suppressor gene and a proto-oncogene? Describe briefly how suppression of cell survival is controlled by PTEN and also explain how the mutation of PTEN leads to carcinoma. 2+3=5

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7. What is Critical concentration (Cc) in the polymerization process? Why smooth muscle contraction is comparatively a slower process? State the function of Thymosin â4. How Formin protein is associated with Rho-dependent pathway in actin polymerization? 1+1+1+2=5 **[CO2]**

8. How Dsh protein regulates calcium release in non-canonical wnt pathway? State the regulatory function of â-catenin in wnt signaling. What is the role of Cul3 in Nrf2/Keap1 signaling pathway? 2+2+1=5 **[CO3]**

9. Define the roles of SMURFs and SARA in TGF-â/Smad signaling pathway. What are the functions of ADAM metalloprotease and ã-secretase in NOTCH signaling? 3+2=5 **[CO3]**

GROUP—C

Answer *any* **four** questions : 2.5×4=10

10. Describe the differential roles of Anaphase Promoting Complex (APC) in mitotic exit and G1/S transition. **[CO2, CO3]**

11. Mention the role of Licensing factors chromatin licensing and DNA replication factor 1 (cdt1) and cell division cycle 6 (CDC6) in the initiation of replication fork progression. **[CO2]**

12. How Tumor Necrosis Factor Alpha (TNFá) induces necroptosis in cells? **[CO3]**

13. How autoinhibited monomer of APAF1 converted to apoptosome during apoptosis? **[CO3]**

14. Other than cytochrome C, which factors are released during Mitochondrial Outer Membrane Permeabilization (MOMP), mention their functions also. **[CO1, CO2, CO3]**

15. What is hayflick limit? How telomere length varies with number of cell division in somatic cells, stem cells and cells transfected with Telomerase Reverse Transcriptase enzyme (TERT) (Draw graph only). **[CO2, CO3]**

16. Why senescence cells are arrested in Early G1 phase? **[CO2, CO3]**

17. How persistence DNA damage responses induce senescence? **[CO3]**

GROUP—D

Answer *any* **one** question : 5×1=5

18. "Cancer is a genetic disease or not" — put up your views with scientific reasoning. "pRb restricts the cell's ability to replicate DNA by preventing its progression from the G1 to S phase of the cell division cycle." Explain how pRb plays an efficient role as protooncogene. 2+3=5