**ST JOSEPH’S UNIVERSITY, BENGALURU -27**

**M.Sc. Biotechnology- II SEMESTER**

**SEMESTER EXAMINATION: APRIL 2024**

**(Examination conducted in May/June 2024)**

**BT 8122: ADVANCED CELL BIOLOGY**

**(For current batch students only)**

Registration Number:

Date & Session:



**Time: 2 hours Max Marks: 50**

**This paper contains TWO printed pages and THREE parts**

**PART-A**

**Answer any SEVEN of the following 2m x 7 = 14 marks**

1. How will you prove that a cell is apoptotic? Suggest any two diagnostic tests.
2. What post-translational modification do caspases undergo?
3. What is contact-inhibition and why do stem cells lack it?
4. In the Wnt-signaling pathway, which proteins should be present on the cell surface such that the cell can respond to Wnt ligand?
5. Other than metastasis, state two roles of epithelial to mesenchymal transition.
6. What is the ultimate outcome of non-apoptotic modes of cell death? Why are they immunologically active?
7. You have obtained a *Securin* TS mutant yeast strain. What would happen when you move it to the restrictive temperature?
8. Name any reader and writer of glycosylation.
9. What is the role of the stem cell niche in asymmetric division?

**PART B**

**Answer any FOUR of the following: 5m x 4 = 20 marks**

1. Cancer cells tend to have high levels of the BCL-2 protein. Illustrate the downstream signaling and how it will be affected in cancer cells. Why do you think they have evolved this feature?
2. Endo- and exocytosis are known to be important for cell movement. Discuss how it impacts integrin signaling. What would be the phenotype of a *Talin* KO cell in this context?
3. Compare basic negative- and positive-feedback systems making sure to cover

a) the action taken by a system after deviation from a setpoint is detected and

b) how the system returns to a setpoint.

1. Define “organoid” and discuss the relationship between organoids and induced pluripotent stem cells (iPSCs). Why do scientists see the production of organoids as the logical next step in understanding mechanisms of a) disease and b) therapy?
2. Draw the structure of a tight junction. How do some pathogens modulate this complex and what will be the consequences of an impaired tight junction?
3. Autophagy plays a crucial role in cellular responses to pathogens. Using the example of *Listeria*, a cytoplasmic bacterium, explain the process of macro-autophagy. What would be the main issue faced by an autophagy KO cell?

**PART C**

**Answer any TWO of the following: 8m x 2 = 16 marks**

1. Gullu is a cancerous cell line with numerous p53 mutations. What processes will this affect and how? Imagine you were unaware of the molecular nature of p53- design an assay to figure out if it is a oncogene or a tumor-suppressor.
2. Illustrate the primary structure of EGFR and c-Src kinase. Compare their modes of activation and deactivation. Why does EGFR exist as a dimer whereas Src exists as a monomer?
3. LDL is a cargo that is usually degraded inside the cell. Illustrate the life of an LDL molecule from outside the cell to degradation. Statins are a class of anti-obesity drugs that inhibit HMG CoA reductase. How will this influence the dynamics of LDL endocytosis? Can you design a better therapeutic strategy?