BIRLA INSTITUTE OF TECHNOLOGY & SCIENCE, PILANI

FIRST SEMESTER 2016-17

Advanced Cell and Molecular Biology (BIOG542)

Mid-Semester Examination (Closed book/ Open book)

Duration: 90 minutes **M. Marks: 60** **Date: 7. 10. 2016**

NOTE: Submit closed book part before beginning open book part. Make use of figures and diagrams wherever necessary.

PART A - Closed Book (45M)

(1) Describe the current model for the import of cytoplasmic cargo proteins. **[5]**

(2) What are the factors that maintain unidirectional diffusion of cargo across NPC? **[5]**

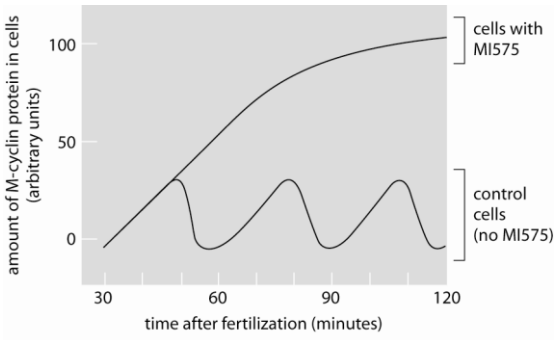
(3) Describe the similarities between Ran and Ras activities with reference to their regulation. **[5]**

(4) What are modular interaction domains? Describe three types of them involved in cell signaling. **[5]**

(5) What is 'lateral inhibition' in 'contact dependent' signaling? Describe a pathway mentioning the relevant components, which display the above behavior. **[5]**

(6) You want to study the cell cycle in fish embryo cells, in which the cell cycle length is about 30 min. You examine the amount of M-cyclin in these cells and see that it varies over the cell cycle. Your friend works in a pharmaceutical company where they are making drugs that block the cell cycle. She gives you some of their secret drug, called MI575, and you add it to cells, examine M-cyclin, and get the results shown in Figure. **[3+3]**

A. From these data, would you predict that cells treated with MI575 are arrested in the cell cycle at various stages (G_1, S, G_2, or M), or do you predict that cells will be blocked at a particular stage? If you think cells will be blocked at a particular stage, describe the stage at which you think the cells would be blocked.



B. From the data, propose a simple molecular explanation for how MI575 acts to block the cell cycle.

(7) Your friend works in a lab that studies origin licensing. He is particularly interested in the pre-replicative complex (pre-RC) and has isolated a temperature-sensitive yeast mutant that does not seem to assemble the pre-RC at the origins of replication. However, he has gotten into an

argument with a new student in the lab. The student thinks that this yeast mutant will arrest in late mitosis or early G_1, because that is when the pre-RC is normally assembled. Your friend disagrees. Who is right, and why? Comment on the fate of cells as per your friends logic. **[4]**

(8) Several mutational changes to proteins involved in apoptosis are listed below. For each, indicate whether you expect it to have a dominant-negative phenotype. In other words, do you expect it to cause a defect in apoptosis even in cells that also contain the normal protein? Attempt any five. **[2X5=10]**

A. Deletion of death domain of Fas death receptor.
B. Deletion of the death effector domain of procaspase-8.
C. Deletion of the CARD domain of Apaf-1.
D. Mutation of the catalytic cysteine of caspase-8 to serine.
E. Mutational change to Bax that prevents its oligomerization with itself and with Bak.
F. Mutational change to IAP that increases its affinity for caspases.

PART B - Open Book (15M)

(Attempt any 3 question)

1)
a. Which genes are under direct control or indirectly related to the activity of Bcl2? Justify. (3)
b. Draw a schematic representation to detail the hypothesis proposed by Cao et al. on Bcl2 silencing. (2)

2) How can the activated CDK1 act as biomarker in case of colorectal cancers? Mention the name of possible techniques that can be used to identify phosphorylation of proteins. (5)

3) Why did Pfister et al. include RRM2 in their study? How is it related to AZD1775 and SETD2? (5)

4) How is the Bax-α9 related to mitochondrial membrane shape? (5)