BIRLA INSTITUTE OF TECHNOLOGY AND SCIENCE, PILANI
FIRST SEMESTER 2023-24
BIO G526 CANCER BIOLOGY
MID SEM EXAMINATION-Closed Book

Max. Marks:20   **Time: 90 Min**   **Date:09.10.2023**

Q1. EGFR over-expression is a common factor in lung cancer. Tom was suffering from lung cancer and hence treated with Cetuximab. The doctors were very hopeful of the clinical outcome but unfortunately the treatment failed and cancer progressed. Explain the rationale behind the failure? [3M]

Q2. Julia received 2 types of cell A and B and was trying to transform/immortalize those cells by infecting them with transforming retroviruses. She observed that after subsequent transfection with retroviruses and passaging the cell type A showed transformation as revealed by formation of focuses and growth in soft agar assays but the cell type B is not showing any of the above characters. What do you think is the problem/ she has done some mistake and needs to repeat the experiment? Justify. [3M]

Q3. As a cancer biologist student you are working in the lab with 2 cell types. Cell 1 is NIH3T3 and Cell 2 is MDA-MB231 cells. Both the cells were growing fine in 10% fetal calf serum. But during a serum starvation experiment she was surprised to observe that Cell 1 has stopped growing in absence of serum while Cell 2 was growing fine. Give your comments for this observation? [2M]

Q4. Many patients show signs of severe anemia as a side effect of chemotherapy and are prescribed erythropoietin (EPO). EPO is a secreted protein, produced by the kidney, which binds to its receptor on erythroid precursor cells and stimulates the formation of red blood cells (RBCs). Four different mutations are described below. For each mutation, list whether the RBC production would **increase, decrease or not change** in an individual that was homozygous for this mutation as compared to the wild-type situation. Briefly explain your reasoning for each mutation. [4M]

i. A mutation in the EPO gene, which results in deletion of the signal sequence of the EPO protein.
ii. A mutation in the EPO receptor, which results in the deletion of its transmembrane domain.
iii. A mutation in the EPO receptor that results in the deletion of its cytoplasmic domain.
iv. A mutation that results in a truncated ectodomain of the EPO protein.

Q5. The responsiveness of a cell to exposure to a growth factor are usually attenuated after a period of time (e.g., *half* an hour), after which it loses this responsiveness. Given what you have already learned about growth factor receptors what mechanisms might be employed by a cell to reduce its responsiveness to a growth factor? [2M]

Q6. Nisha infected mice with mouse liver tumor virus (a retrovirus). After a period of time, most infected mice had developed liver tumors, whereas uninfected mice did not. You isolated cell lines from over 50 independent tumors. You demonstrated that all of these lines had virus integrations in the same chromosomal location. Can one conclude that the virus integrates into cellular DNA at only one site? Explain. [2M]

Q7. A Why is autocrine signaling an intrinsically destabilizing force for a normal tissue? B. Each growth factor elicits its own, quite characteristic set of biological responses in cells. How might you alter a cell so that its biological responses to one growth factor (e.g., EGF) are characteristic instead of responses that it usually makes after being exposed to another growth factor (e.g., PDGF)? C. What molecular mechanisms have been evolved to ensure that the signals coursing down a signaling cascade reach the proper end-point targets rather than being broadcast non-specifically to "unintended" targets in the cytoplasm? [1+1+2=4M]

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