Example Questions BIO 520 Master Cancer Biology

In most laboratories' cells are grown with 20% oxygen. How does this reflect real tissue oxygenation conditions? What might be the consequences?

What is the molecular basis of the limited proliferative potential of cultured primary cells?

What is the molecular basis of radiotherapy against cancer?

Describe how transgenic mice can provide models for cancer and the advantages and disadvantages of such models.

The retinoblastoma gene (RB1) was the first tumour suppressor gene cloned. Explain how a tumour suppressor functions, describe the function of the RB1 gene product and describe the disease RB1 is named after?

Discuss the potential problems of basing a treatment strategy on analysis of a small biopsy of a large malignant tumour.

Mutations in the p53 tumour suppressor gene belong to the most frequently found genetic alterations in many types of human cancers. However, there are still a lot of cancers that express wild type p53. These cancers have often lost both copies of the ARF gene indicating that p53 and ARF have similar roles in the process of malignant transformation. Please describe how p53 and ARF are interconnected on the molecular level.

Explain the difference between Tyrosine Kinase Receptors and Steroid Receptors and sketch at least a pathway through which the former transduces signals to the nucleus.

Describe at least four steps in the process leading to tumour extravasation from blood capillaries. Describe key tumour cell capabilities required in each step.

What makes Ras such a potent oncogene? (think of its downstream effects)

Name two well-established tumour suppressors and briefly describe their main cellular functions and how their deregulation can promote cancer development.

Describe the two major mechanisms that tumour cells employ to maintain their telomeres.

Mention different types of immunotherapy for cancer and describe their (dis)advantages.

Defects in several DNA repair processes associate with cancer. List at least three examples of DNA repair pathways, their physiological function, and the types of cancer that are linked to the defect in the particular repair pathway.

How do integrins tether to the ECM (extra-cellular matrix) and cytoskeleton?

Which technology is used for assessing the recruitment of proteins to specific chromatin loci? Explain this technology.

Tumours are classified into broad classes based on their cell of origin. Describe 3 of these broad classes of tumours and the classification of cell from which they arise and list two examples of specific tumour types that belong to each tumour class.