Cell Growth Control

Readiness Assessment Questions

1. Many colorectal cancers have mutations which inactivate the signal transduction pathway that is stimulated by bone morphogenetic proteins (BMPs). Which of the following proteins would most likely be expressed by the BMP pathway?

- A. Cdc25
- B. E2F1
- C. Ink4
- D. Cyclin D

2. Tyrosine-kinase inhibitors are used to treat many types of cancer. These inhibitors would most likely arrest cells in which stage of the cell cycle?

A. G1

B. S

C. G2

D. Mitosis

3. Paclitaxel is a drug used to treat a variety of cancers. Paclitaxel binds tubulin and prevents disassembly and rearrangement of microtubules. Which stage of the cell cycle is most strongly affected by paclitaxel?

A. G1

- B. S
- C. Mitosis
- D. Cytokinesis

4. Human papillomavirus (HPV) encodes a protein, E7, that can bind and inactivate retinoblastoma proteins (pRb). Which of the following would most likely be associated with HPV infection?

- A. Decreased expression of cyclin E
- B. Increased expression of cyclin E
- C. Decreased expression of Cdk2
- D. Increased expression of Cdk2

5. Radiation therapy is a common treatment for some cancers as it causes DNA damage. An inhibitor of which protein would increase the effectiveness of radiation therapy on tumor cells?

A. Ras

- B. Cyclin D
- C. p53
- D. Myc

6. Ibrance (palbociclib) has proven effective in treating women with advanced breast cancer. Ibrance inhibits CDK4 and CDK6. In which stage of the cell cycle would you expect to find cells treated with Ibrance?

A. G1

- B. S
- C. G2
- D. Mitosis

7. PTEN is protein that associates with the cell membrane and converts PIP3 to PIP2. Which of the following best reflects the role of PTEN?

- A. It creates a positive feedback loop
- B. It creates a negative feedback loop
- C. It is an oncogene
- D. It is a tumor suppressor

8. DNA sequencing from your patient reveals a deletion in one copy of P53 and an activating mutation in one Ras gene. Which of the following future changes would be of greatest concern?

- A. Activating mutation in second Ras gene
- B. Overexpression of Myc
- C. Inactivating mutation in second P53 gene
- D. Overexpression of cyclin D

9. Mutations that generate a missense mutation in codon 12 of RAS are associated with tumor development. Codon 12 likely plays a role in which activity?

- A. Interaction with guanine nucleotide exchange factor
- B. GTPase rate
- C. Protein folding
- D. Interaction with MAP Kinases

- 10. Positive feedback loops plays important role in which of the following steps?
 - A. Transition from G1 to S phase
 - B. Transition from metaphase to anaphase
 - C. Activation of Ras
 - D. Expression of Ink4

Application Questions

- 1. Cells that over-express a mitogen-binding, tyrosine-kinase receptor can develop into tumors. To treat these types of tumors, you develop an antibody to an epitope in the receptor. You find that in an in vitro assay the antibody slows the growth of cells that over-express the receptor. In a mouse model of colon cancer, in which you engineered colonic epithelial cells to over-express the receptor, you find that the antibody shrinks the tumors in most mice. In a clinical trial of the antibody on patients with colon cancer, you find that the antibody on tumor growth declines. In your next clinical trial, you decide to combine the antibody with another drug. Which drug would you choose?
 - A. A second antibody against a different epitope on the receptor
 - B. An inhibitor of the kinase in the cytosolic domain of the receptor
 - C. An inhibitor of PI3-kinase
 - D. Paclitaxel, an inhibitor of microtubule depolymerization

- 2. You are treating a patient with breast cancer and consider treating her with an inhibitor to CDK4 because you have read about positive results in other patients who have taken the inhibitor. Unfortunately, her tumor does not seem to respond to the drug. To gain more information about your patient's tumor, you obtain a biopsy and perform genome sequencing. Which of the following mutations would you most likely find?
 - A. Activating mutations in codon 12 of KRAS
 - B. Partial deletions of Rb genes
 - C. Amplification of Cyclin E gene
 - D. Mutations in INK4