

Please complete this quiz by recording your answers on the answer sheet provided.

Contact Theresa.kenney2@cchmc.org with any questions.

Questions for Module 6 part 2: Fagin Perspectives on Thyroid Cancer Pathogenesis

- 1) The following oncogenes code for tyrosine kinases mutated early in cancer development:
 - a. PLK in colon cancer.
 - b. BCR/ABL in chronic myelogenous leukemia.
 - c. VEGFR in prostate cancer
 - d. Estrogen receptor in breast cancer.

- 2) Cancer-inducing rearrangements of genes that are far from each other in the primary DNA sequence may be favored by spatial contiguity of the participating genes. A technique that could be used to demonstrate that they are contiguous includes:
 - a. Fluorescence in situ hybridization of interphase cells using genomic probes for the participating genes in the rearrangement
 - b. Coimmunoprecipitation of chromatin with specific antibodies.
 - c. RT-PCR with primers bracketing the recombined sequence.
 - d. Microscopy of lamin-stained nuclear membranes.

- 3) Mutations of BRAF, RAS and RET/PTC are mutually exclusive in well differentiated papillary thyroid cancers. This demonstrates that:
 - a. These mutations are likely to be pathophysiologically important
 - b. The signaling pathway in which they reside is likely required for thyroid cell malignant transformation.
 - c. Both A and B are correct.
 - d. Neither is correct.

- 4) You are the director of drug discovery for Fantastix Signal Transduction Pharmaceuticals. You have enough funds to develop compounds against two target oncoproteins. Which of the following arguments is logical in selecting a target for a selective anti-cancer kinase inhibitor?
 - a. Select a kinase oncoprotein acting distally in the pathway to block signals from activated mutants at all steps in the pathway.
 - b. Select an upstream kinase to block signals from activated mutants at all steps in the pathway.
 - c. Select a kinase receptor in endothelial cells to inhibit tumor angiogenesis because that cell population is less susceptible to developing a drug resistance mutation
 - d. Block the action of DNA polymerase to interfere with cell division in all tumor cells.
 - e. A and C
 - f. B and D