**These review questions are for Bio 1 Mitosis Checkpoints and Cancer topic. The questions were adapted from several sources, including the textbook’s review questions.**

1) Why do neurons and some other specialized cells divide infrequently?

A) They no longer have active nuclei.

B) They are cancer cells.

C) They are in G0 phase.

D) They can no longer bind to other cells of their type.

E) They can no longer carry out cellular aerobic respiration at levels required to sustain cell division.

2) Which of the following triggers the cell's passage past the G2 checkpoint into mitosis?

A) The chromosomes have been duplicated.

B) The spindle fibers are attached to the chromosomes.

C) The spindle fibers are attached to the centrosomes.

D) The chromosomes are aligned on the cell equator.

E) A growth factor binds to a receptor.

3) To pass the metaphase checkpoint, which of the following must occur?

A) The chromatids must lose their kinetochores.

B) The sister chromatids must attach to each other.

C) The spindle fibers must be attached to the kinetochores.

D) The kinetochores must attach to the centrosomes.

E) Spindle microtubules must begin to depolymerize.

4)The Ras protein is best described as an...

A) a relay protein activated by a growth factor receptor

B) an enzyme that carries out DNA replication

C) an enzyme that carries out DNA repair

D) a digestive protein that degrades damaged RNA

E) a cellular response protein that carries out attachment of the spindle fibers to duplicated chromosomes

5) Forms of the Ras protein found in tumors usually **directly** cause which of the following?

A) DNA replication to stop

B) DNA replication to have a high rate of gene mutation

C) cell-to-cell adhesion to be nonfunctional

D) cell division to cease

E) growth factor signal transduction to be constantly active

6) Which of the following statements describes normal (not mutated) proto-oncogenes?

A) Their normal function is to stop cell division.

B) They are introduced to a cell initially by retroviruses.

C) They are produced by mutations of oncogenes.

D) They can code for proteins that are activators of normal cell growth.

E) They can code for proteins that change normal cells into cancer cells.

7) Proto-oncogenes can change into oncogenes that cause cancer. Which of the following best explains the presence of these potential time bombs in eukaryotic cells?

A) Proto-oncogenes first arose from viral infections.

B) Proto-oncogenes normally help carry out cell division.

C) Proto-oncogenes are genetic "junk."

D) Proto-oncogenes are mutant versions of normal genes.

E) Cells produce proto-oncogenes as they age.

8) Proto-oncogenes can lead to cancer when...

A) the gene mutates to encode an overactive form of the normal protein.

B) the gene mutates to encode an underactive form of the normal protein.

9) One difference between cancer cells and normal cells is that cancer cells...

A) are unable to synthesize DNA.

B) are arrested at the S phase of the cell cycle.

C) continue to divide even when they are tightly packed together.

D) cannot function properly because they are tightly packed together.

E) are always in the M phase of the cell cycle.

10) For a chemotherapeutic drug to be useful for treating cancer cells, which of the following is most desirable?

A) It stops all cell growth

B) It stops all dividing cells.

C) It only attacks cells that stop growing when they are in contact with other cells.

D) It interferes with cells entering G0.

E) It interferes with rapidly dividing cells.

11) Which of the following is **not** true concerning cancer cells?

A) They do not exhibit contact inhibition when growing in culture.

B) They originate from foreign cells that enter the body from outside.

C) They are not subject to cell cycle controls.

D) They can be grown in cell culture (test tubes).

E) They do not require growth factor signal molecules to grow.

12) Cells from an advanced malignant tumor most often have very abnormal chromosomes, and often an abnormal total number of chromosomes. Why might this occur?

A) Cancer cells are no longer density dependent.

B) Cancer cells are no longer require growth factors to exit Go.

C) They have lost the metaphase checkpoint.

D) Chromosomally abnormal cells are enslaved and absorbed by cancer cells.

E) Cancer introduces new chromosomes into cells.

13) A research team began a study of a cultured cell line (cells grown in the laboratory). Their preliminary observations showed them that the cell line did not exhibit either contact inhibition or growth factor dependence. What could they conclude right away?

A) The cells originated in the nervous system.

B) The cells are unable to form spindle microtubules.

C) The cells have reversed their order of cell cycle phases.

D) The cells show characteristics of tumors.

E) They were originally derived from an elderly organism.

14) Tumor-suppressor genes...

A) are frequently over expressed in cancerous cells.

B) are cancer-causing genes introduced into cells by viruses.

C) can encode proteins that promote DNA repair.

D) often encode proteins that begin the cell cycle.

15) Tumor-suppressor genes can lead to cancer when...

A) the gene mutates to encode an overactive form of the normal protein.

B) the gene mutates to encode an underactive form of the normal protein.

16) Which of the following is related to the function of the *p53* gene?

A) It prevents cells with damaged DNA from dividing.

B) It speeds up the cell cycle.

C) It causes cell death whenever the DNA is replicated.

D) It allows cells to pass on beneficial mutations.

E) It slows down the rate cell reproduction by inhibiting growth factor receptors.

17) The *p53* gene is involved in which cell cycle checkpoint?

A) Go

B) G1

C) G2

D) Metaphase

18) In the 1920s researchers discovered that X-rays caused mutations in *Drosophila* (fruit fly). Later, it was discovered that certain chemicals can also cause mutations. Suppose a new chemical food additive is developed by a cereal manufacturer. Why do we test for its ability to cause mutations?

A) We worry that it might cause mutation in cereal grain plants such as wheat and barley.

B) We want to make sure that it does not emit radiation.

C) We want to be sure that it increases the rate of mutation sufficiently for human evolution to continue.

D) We want to prevent an increase cancer rates.

E) We worry about its ability to cause infection.

**Answers to review questions:**

1) C

2) A

3) C
4) A

5) E

6) D

7) B

8) A

9) C

10) E

11) B

12) C

13) D
14) C

15) B

16) A

17) C

18) D