Name:____KEY_____ Dr. Reichler's Bio 325 TTh 7:30-9pm Fall 2007 Quiz 11/08

1) What is different in a cell in G1 and G2 phases of the cell cycle?
The DNA is replicated in G2.

2) Can DNA replication initiate anywhere on the DNA?
No, there are specific origins of replication. But the primase puts primers randomly along the DNA.

3) What is different between DNA replication on the leading and lagging strand?
The mechanism is the same, but the frequency is different. The lagging strand replicated discontinuously while the leading strand is replicated continuously.

4) Helicase unwinds the DNA, and then DNA polymerase copies the unwound DNA, so these two proteins both act on the same region of DNA. Of the other proteins involved in DNA replication, which ones act on the same regions of the DNA?
There are several answers: Helicase and primase, ligase, or gyrase all eventually act on the same region of DNA. DNA polymerase and primase, ligase, or gyrase all eventually act on the same region of DNA. Primase and ligase all eventually act on the same region of DNA.

5) Why is it important for a cell to be able to identify recently copied DNA? How does *E. coli* do this?
If there are mismatches, the cell needs to know which is the correct sequence so it can replace the mismatched nucleotides. *E. coli* methylate their DNA about 10 minutes after DNA replication so the unmethylated DNA is recognized as the newly replicated strand.

6) What is the problem at the ends of DNA replication, and how is this problem resolved?
The last primer on the lagging strand cannot be replaced by DNA. The problem is not really solved, there is always a gap at the end of a DNA strand. However, telomerase can elongate the DNA.

7) What does the problem in #6 protect you from?
Shortening telomeres are a measure of DNA replication and replication errors.

8) In what human cells would you expect to find the shortest telomeres?
Any cell that divides regularly or is exposed to DNA damage such as the lining of the stomach, blood cells, skin, liver, kidneys, etc.

9) What can be learned by looking at the length of someone's telomeres?
How damaged their DNA is. Longevity may be reduced in people with short telomeres.

10) During mitosis, why does the DNA line up in the middle of the cell?
So that is can be evenly divided into the two progeny cells.

11) Would you expect many genes to be expressed during mitosis?
No, the DNA is tightly packaged.

12) Why are multiple mutations required for a cell to become cancerous?
There are several checkpoints and positive and negative signals for each checkpoint.

13) Are short telomeres a positive or negative signal for mitosis?
Short telomeres inhibit cell division. They are a negative signal.

14) How could looking at someone's genes help determine their risk of developing cancer? Could data about someone's environment help determine their risk of developing cancer? Explain.
Mutations in genes that code for products that regulate the cell cycle can lead to cancer. Many of these mutations are induced by toxins from the environment.

15) Why might lung cancer be such a deadly form of cancer?
There is a continuous and concentrated exposure to the toxins.

16) Is p53 gene of a cancerous cell likely to be absent or over-expressed?
Absent, p53 should induce apoptosis in cells with mutations.

17) What is measured by microarray analysis, and what is one weakness of the data obtained?
Differences in mRNA expression. It does not tell us about protein levels.

18) What are two changes that you could detect by microarray analysis of a cancer cell that would lead to using or not using a particular cancer treatment?
Reduced expression of cell adhesion genes could indicate malignant cancer. Increased expression of MDR could indicate that chemotherapy will not be effective.