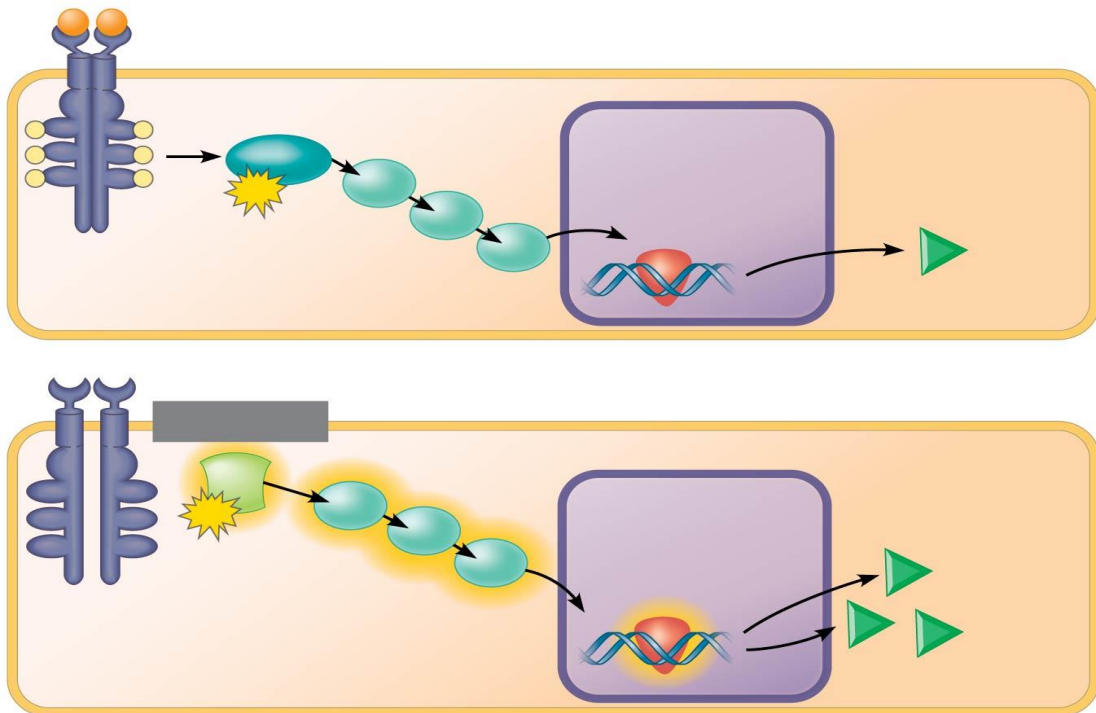


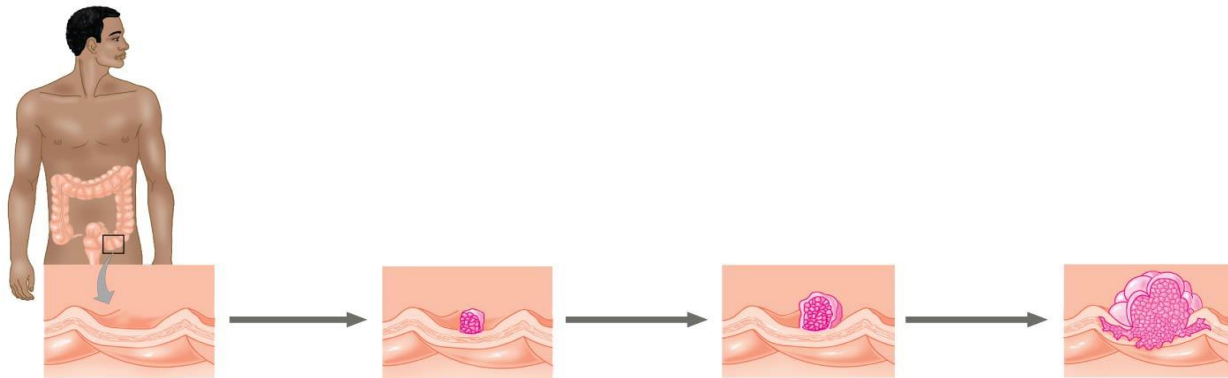
## Chapter 18.5 Guided Reading: Regulation of Gene Expression 10ed.

1. Mutations that alter growth factors, their receptors, or intracellular signaling pathway molecules, or affect regulation of the cell-cycle, can lead to cancer in somatic cells. Therefore, genetic mutation is the mechanism involved in the beginning of tumor growth. What can lead to these cancer-causing mutations?
2. Compare *oncogenes* and *proto-oncogenes*.
3. What are three mechanisms for converting a proto-oncogene to an oncogene?
4. There seem to be two categories of genes involved in cancer: *oncogenes*, which code for proteins to regulate cell growth, and should not be stuck “on,” much like the accelerator in a car; and *tumor-suppressor genes*, which work like the brakes on a car and must function. Let’s begin with the look of the *ras* gene, which codes for a G protein and is an *oncogene*. Label the following sketch to explain how a *ras* mutation leads to cancer.



5. *Tumor-suppressor genes* help prevent uncontrolled cell growth. One that is found mutated (and therefore nonfunctional) in more than 50% of a human cancer is *p53*. So important is the *p53* gene that it is sometimes called the “guardian angel of the genome.” Describe the double whammy that results from mutations of *p53*.

6. Explain the *misstep model of cancer development* by using the specific example of colorectal cancer. Use the following figure to label the five mutation levels leading to this form of cancer.



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7. You probably know someone who has been treated for breast cancer. Did you realize there were genetically distinct types? Why is it not surprising that signal receptors are over-expressed in most types of cancer?

8. Why do Tamoxifen and Herceptin not work against Basal-like breast cancer?

9. Why is said that people inherit predispositions to cancer not cancer itself?

10. Describe the role of viruses in cancer.

11. Give 2 specific viruses and their associated cancers.