

Cancer Biology and Implications for Practice

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The media seem to announce a new scientific discovery related to cancer daily. Oncology nurses are challenged to keep up with the explosion of new knowledge and to understand how it ultimately relates to the care of patients with cancer. A framework for classifying new knowledge can be useful as nurses seek to understand the biology of cancer and its related implications for practice. To understand the molecular roots of cancer, healthcare practitioners specializing in cancer care require insight into genes, their messages, and the proteins produced from those messages, as well as the new tools of molecular biology.

Cancer as a Genetic Disease

Cancer is a genetic disease. In other words, cancer occurs because of changes in the genes of a cell or in the expression of those genes. Each gene consists of short stretches of DNA that specify instructions for making a particular protein. The coding region of a gene specifies those instructions by the order in which the chemical bases are arranged. Some proteins serve a structural function, whereas others have a role in telling cells how to behave. Mutations generally result in changes in the molecular instructions for the building of a protein and, in many cases, disrupt key regulatory pathways in the cancer cell.

Mutations and Cancer

Mutations in cancer cells frequently result from errors during DNA replication but also can occur from exposure to complex chemical mixtures encountered in the environment or through lifestyle and dietary factors. Researchers now know that the expression of a gene also can be altered through methylation of a regula-

ry region of the gene, and the end result is that the function of the gene is inhibited just as if a mutation had occurred. This new area of study is known as epigenetics and is defined as regulation in the expression of gene activity without alteration of gene structure (Das & Singal, 2004). See the glossary on p. 460 for definitions of different types of mutations.

The Cell Cycle and Cancer

One unique characteristic of a cancer cell, as compared to its normal counterpart, is that the cancer cell proliferates. It does not obey the normal regulatory mechanisms and restrictions present in normal tissues. A key to understanding tumor cell proliferation is to characterize how the mechanisms used to restrain the proliferation of a normal cell fail in a cancer cell. The cell cycle represents a series of integrated events that control how the

cell grows, proliferates, and ultimately dies. Critical parts of the cell cycle machinery are the cyclin-dependent kinases (CDKs), which, when activated, provide a means for the cell to move from one phase of the cell cycle to the next (see Figure 1). CDKs are regulated positively by cyclins and negatively by naturally occurring CDK inhibitors (CDKIs). Cancer represents a dysregulation of the cell cycle where cells that overexpress cyclins or do not express CDKIs continue to undergo unregulated cell growth (Schwartz & Shah, 2005).

The cell cycle also protects the cell from DNA damage. Cell cycle arrest serves as a survival mechanism that allows the cell to repair damage to its DNA. In contrast to healthy cells, tumor cells are unable to stop at predetermined points of the cell cycle because of loss of checkpoint integrity. This can result from inactivation of critical CDKIs or from overexpression of cyclins. One of the most common mutations in cancer

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