CHAPTER 1  Biology of Cancer

**Introduction**

Many in the lay public describe a diagnosis of “cancer” as if it is one disease. In reality, it encompasses more than 200 diseases that will occur at different ages with different rates of growth, differentiation, abilities to be detected, invasiveness, capacities to spread or metastasize, treatment responses, and prognoses. However, at the cellular and molecular levels, cancer is beginning to be viewed as a few diseases caused by genetic alterations and defective cell function that are actually very similar (Ward, Brueggemeier, Caligiuri, Gahbauer, & Kraut, 2005). These alterations can be associated with “nature,” such as inherited cancer syndromes like hereditary breast and ovarian syndrome (HBOS) or immune deficiencies. Or, the genetic alterations can be caused by “nurture,” which includes obesity, poor diet, and social habits, such as smoking. A malignant growth is the result of changes in DNA, gene transcription, or translation. The resultant defective protein or proteins lead to transformation of normal cell components into uncontrolled proliferation, spread, and/or metastasis (Ward et al.). This chapter focuses on a description of the malignant changes of a cell that will provide nurses new to oncology with a foundation for understanding the growth of cancer and its treatment, along with the basis to provide education to patients and their families.

**Models of Cancer Development**

Two models are commonly used to describe how a cancer develops. The first is the Stochastic Model, also known as Knudson’s random “two hit” model (see Figure 1-1). This model suggests that each cancer cell has the ability to multiply and form new tumors. Using one set of chromosomes within the nucleus of the cell as an example, the two chromosomes each have an identical allele, or gene, controlling the growth of a cell. When the initial gene on the first chromosome is damaged, the undamaged gene on the second chromosome is still able to take over the normal functions associated with that gene and its protein product. When the gene on the second chromosome becomes damaged, a normal gene message is no longer present, thus allowing malignant transformation. The malignant cells have a selective advantage over their normal neighbors and begin to proliferate rapidly, accumulating genetic damage with each generation. As the damage collects, the most aggressive characteristics promote immortalized growth and the formation of a tumor (Hanahan & Weinberg, 2000; Knudson, 1971).

The individual cancer stem cell (CSC) is the focus of the second model. This model states that many different types of cancer cells exist, demonstrating heterogeneity. With proliferation, cell division occurs, where all of these cells
have the ability to multiply, but only one cell type—the CSC—has the ability to become a new tumor (Reya, Morrison, Clarke, & Weissman, 2001). This is becoming the model most supported by cancer researchers. See Figure 1-2 for an illustration of CSC tumor development. Once the new tumor is established, the heterogeneous cells begin to proliferate, allowing the tumor to enlarge, and the CSC moves into the resting phase (G₀) of the cell cycle. It is well known that cells in this phase are resistant to treatment and would remain as one surviving cancer cell while treatment destroys the other rapidly dividing non-CSCs (Wicha, Liu, & Dontu, 2006). Thus, months or years later, the “resting” CSC could move into the active phases of the cell cycle, proliferate, and cause exacerbation of the once-dormant cancer that was thought to be destroyed (Reya et al.).
Structure and Function of DNA and Chromosomes

The human genome consists of 23 pairs of chromosomes. Each chromosome is a single double-helix DNA molecule with millions of base pairs connected in a long, unbroken string that intricately coils back upon itself and is scattered with proteins, called histones (see Figure 1-3).

Similar to sewing bobbins, histones control the long threads of DNA, wrapping them into a tight coil so that the DNA is able to fit inside the nucleus of a cell. The coiling is necessary because the strands of DNA in a person’s body would stretch almost six feet but would be only fifty-trillionths of an inch wide. This long but thin physical structure would be extremely fragile, hence the need for tight packaging to keep the DNA message intact. Once it is coiled...
Figure 1-3. DNA Packaging

around the histones, DNA continues twisting back upon itself (much like the continued twisting of a rope) until it is tightly wound, forming the chromatid seen in Figure 1-3. These chromatids enable the chromosomes to be visualized for karyotyping during the metaphase of cell division (Klug & Cummings, 2003).

Within the nucleus of each normal human cell, 23 pairs of chromosomes are present. These consist of 22 pairs of nonsex chromosomes (autosomes) and one pair of sex chromosomes (XX for female, XY for male). A person inherits one chromosome of the pair from the father and the other from the mother. A chromosome has a short arm ("p" for "petite") and a long arm ("q" because it follows "p" in the alphabet) with a unique banding pattern that identifies specific regions. These regions are numbered from the centromere to the end of each arm (Genetics Home Reference, 2009a). For example, the breast cancer gene (BRCA1) is found on chromosome 12q, with a band position of 12.1 on the long arm of chromosome 12 (see Figure 1-4). If the position of a gene is uncertain, a range might be noted, such as 17q21–24 (Genetics Home Reference, 2009b).

The central dogma of molecular biology states that DNA (adenine [A], cytosine [C], guanine [G], and thymine [T]) is transcribed to RNA (adenine [A], cytosine [C], guanine [G], and uracil [U] instead of thymine) and then translated into proteins (Klug & Cummings, 2003) (see Figure 1-5). For example, the DNA (ACTGTC) would be transcribed as RNA (ACUGUC) and then to messenger RNA (mRNA), where it is divided into codons (three nucleotides used to specify an amino acid) (UGA CAG) for translation from amino acid to protein. This is important to remember because any changes in the codon (mRNA triplet) “spelling” could change the protein outcome. Some amino acids have multiple codon “spellings.” One example is leucine, which has six (Algorithmic Arts, 2007). These would allow several mistakes without creating a problem protein. However, tryptophan has one spelling (Algorithmic Arts, 2007). Any error in this codon spelling would cause the assembly of
a dysfunctional or nonfunctional protein (National Cancer Institute [NCI], 2006).

Changes in the DNA nucleotides can be either a mutation or a polymorphism. This depends on the frequency with which the change occurs in the general population. If it occurs in at least 1% of the population, it is called a polymorphism. If it occurs in less than 1%, it is labeled as a mutation (NCI, n.d.). To further explain, a normal length of DNA is similar to a recipe for the most common type of cake—for example, a chocolate cake. Although this is the most common, other types of cakes exist, including strawberry, white, spice, pineapple upside-down, and lemon. These would be polymorphisms. They are good-tasting cakes but are not the most common. Sometimes the recipe is misread, and the cake comes out of the oven as a pudding. This is uncommon and not the desired outcome. This is a mutation (NCI, n.d.) (see Figure 1-6).

Several types of mutations exist. The most common type is the point mutation, in which only one nucleotide base is altered. A nonsense mutation occurs when there is a premature termination of the protein. This happens when the stop codon, which signals termination of the length of amino acids, has been “spelled” incorrectly and gives an early or late signal to end the compilation of amino acids into a protein (NCI, 2006).

Mutations that occur within any of the cells of the body are labeled as somatic. They accumulate over a lifetime and are believed to cause sporadic cancers, which typically occur after an individual has reached 50 years of age. Mutations that are present in the ova or sperm are labeled as germ line and are associated with inherited cancers, which typically occur in people at ages younger than 50 (see Figure 1-7). The results of this genomic instability affect all future generations, depending on the pattern of inheritance (NCI, 2006).

Most patterns of inheritance follow the dominant or recessive model developed by Gregor Mendel (Klug & Cummings, 2003). Each individual normally has two sets of chromosomes. On each chromosome is a gene, or allele, for a particular characteristic. Although an allele may have a collection of many different traits (such as blue, green, hazel, or brown eyes), each chromosome can exhibit only one of these. So, one chromosome may have the blue-eyes gene (called an allele), and the second chromosome could have the brown-eyes gene (or allele). All of the other eye colors are still allelic options but are not displayed by this set of chromosomes. Each allele is either a dominant type or a recessive type. It is well known that the brown-eyes allele is dominant over the recessive blue-eyes allele. If the dominant allele is inactivated or lost, then the recessive allele will become active (Klug & Cummings).

Sometimes an individual will have the dominant allele without it being expressed. This is known as incomplete penetrance. The gene is there, but the phenotype (the observable physical trait) is not expressed. An example that illustrates this is a house in a fog; the house is there but is not visible because of the denseness of the low-lying cloud cover. Age, modifier genes, carcinogens, repair enzymes, and hormonal or reproductive factors affect penetrance (Klug & Cummings, 2003).

Much of the scientific evidence about the development of cancer and its progression suggests that genomic instability is a precursor to changes associated with transformation of a cell into malignancy. Of question in this hypothesis is how the instability circumvents the careful security provided within the cell to monitor and guarantee genomic stability and purity for continued survival of the human cell. These protective teams include DNA monitoring and repair enzymes. Checkpoint gatekeepers function at significant points in the active phases of the cell cycle prior to DNA synthesis (S phase) and mitosis (M phase) to guarantee the accuracy of the genome and cell cycle
processes. If an error is present, the P53 tumor suppressor protein causes cell cycle arrest for repair or apoptosis (programmed cell death) if too much damage has occurred (Hanahan & Weinberg, 2000; Kastan & Bartek, 2004; Zhi-votovsky & Orrenius, 2006). Much research has confirmed that most human cancers have loss of function in the P53 tumor suppressor pathway. Other genes involved in targeting and repairing DNA damage also have been found to have loss of function in multiple cancers (American Cancer Society, 2005; Narter et al., 2009). Acquiring genomic damage permits evolving populations of precancerous cells to gain functional capabilities associated with malignant transformation. These include (a) self-sufficiency in growth signals, (b) insensitivity to antigrowth signals, (c) evasion of apoptosis, (d) sustained angiogenesis, (e) tissue invasion and metastasis, and (f) limitless replicative potential (Hanahan & Weinberg).

**Self-Sufficiency in Growth Signals**

**Growth Factors**

The behavior of cells is controlled by circulating growth factors (ligands) that have
the ability to act as chemical signals. They direct cell growth, differentiation, and survival in addition to determining tissue architecture and morphology. Growth factors must interact with their particular receptor to accomplish signaling (Bafico, Grumolato, & Aaronson, 2008; Pawson & Jorgensen, 2008).

Growth factors associated with the development of cancer include epidermal growth factor, transforming growth factor (TGF), and colony-stimulating factor (CSF). Other growth factors exist that are overproduced and are associated with different types of cancer. For example, platelet-derived growth factor is associated with sarcomas and glioblastomas (Pawson & Jorgensen, 2008).

**Growth Factor Receptors**

As the first component in signaling pathways, growth factors bind to receptors to initiate signal transduction across the cell membrane. Once a growth factor is bound to a receptor, a signal activates other markers in the cytoplasm, causing transmission of a message to the cell nucleus. The message causes a change in the expression of certain genes that help to usher the cell through its growth cycle (Zhivotovsky & Orrenius, 2006). Overpro-
duction of some growth factors causes altered cellular communication and is associated with cancers. One of these, vascular endothelial growth factor (VEGF), has an important role in tumor neoangiogenesis—that is, the new growth of vessels on a tumor.

Tumor cells induce hypoxia. This lack of oxygen leads to transcription of the VEGF-alpha protein by binding to its designated cell surface receptors. The binding trips a signal indicating the need for increased blood vessel permeability, resulting in angiogenesis with even more proliferation of cells (Nguyen, Tran, Lipkin, & Fruehauf, 2006). VEGF is overexpressed in metastases of breast and colorectal cancers.

**Growth Factor Receptors and Tyrosine Kinase Activity**

Many cancer-related growth factor receptors are stationed on the surface of the cell. Once they are bound by a ligand that causes activation, proliferative signals are sent into the cytoplasm. Most growth factor receptors possess tyrosine kinase (TK) activity, which leads to reactions that stimulate mitotic cell division, thereby allowing rapid growth of the malignant cell (Chan & Feng, 2007; Zhivotovsky & Orrenius, 2006). Examples of growth factor receptors that are cancer-causing (oncogenic) when overexpressed are epidermal growth factor receptor (EGFR), human epidermal growth factor receptor 2 (HER2/neu), and transforming growth factor-beta (TGF-β). A variety of cancers express EGFR, including non-small cell lung cancer and breast, ovarian, and colorectal cancers. Approximately 80%–100% of head and neck cancers overexpress EGFR, which is also associated with lower survival. Increased HER2/neu expression corresponds with more aggressive cancers, including ovarian and breast cancers. When EGFR and TGF-β are both expressed, it is a prognostic marker for tumor relapse and decreased survival (Bafico et al., 2008; Pawson & Jorgensen, 2008).

**Nonreceptor Tyrosine Kinases**

Some oncogenes do not require a receptor to initiate TK activity at the cell membrane. One example is the SRC gene family. The protein from this gene initiates TK activity at the C-terminus of the DNA where biosynthesis is supposed to end. Because there is no endpoint, the protein function persists, allowing continued signaling to the cell nucleus and causing persistent cell growth. Such SRC-initiated activity is increased in colon cancer and other malignancies such as neuroblastoma, small cell lung cancer, breast adenocarcinomas, and rhabdomyosarcoma (GeneCards, 2009c; Okutani, Lodyga, Han, & Liu, 2006).

**Intercellular Signaling Enzymes**

Oncoproteins with certain enzyme activity are important for sending signals within cells and are called intracellular signaling enzymes. A common example is the enzymatic protein produced by the RAF1 gene (GeneCards, 2009a). In the cytoplasm, TK activates the RAF1 enzyme. Once activated, the enzyme acts as a mediator between the RAS (associated with the RAS oncogene) receptor on the cell membrane and processes occurring in the cell nucleus by activating a series of other kinases, including mitogen-activated protein (referred to as MAP) kinases. These kinases are critical for regulating the onset of cell division, apoptosis, cell differentiation, and cell migration (Bafico et al., 2008; Chan & Feng, 2007; GeneCards, 2009a; Zhivotovsky & Orrenius, 2006).

**Membrane-Associated G Proteins**

The guanine nucleotide-binding proteins (G proteins) are products of a family of genes, the ras proto-oncogenes, which normally act as “on-
off switches” for cell-surface growth factor receptors. Instead of being transmitted inside the cell membrane, they transform adjacent G protein subunits below the membrane surface, which then begin the signaling cascade inside the cell (Bafico et al., 2008; Bos, Rehmann, & Wittinghofer, 2007; Pawson & Jorgensen, 2008).

When the RAS gene mutates and becomes a cancer-causing gene (oncogene), the changes interrupt a cascade of normally occurring signals that take place in the cell cytoplasm. Normal RAS genes wait for prompting to send stimulatory signals from growth factor receptors to other proteins. Mutant RAS genes activate signaling pathways, even when unprompted. Mutant RAS is found in virtually all types of human cancer and occurs in approximately two-thirds of all malignant tumors (Bos et al., 2007). G proteins act at the cell membrane to cause malignant transformation (Bafico et al., 2008; Pawson & Jorgensen, 2008).

Transcription Factors

Proteins that bind to DNA and cause changes in gene expression are called transcription factors. These proteins have structures that can recognize specific DNA sequences (genes) involved in growth and survival. Mutation of the transcription factors that bind to genes involved in cell growth and survival allows for the malignant transformation found in many tumors. Examples of cancers caused by this mechanism include Ewing sarcoma, clear cell sarcoma, alveolar rhabdomyosarcoma, and many kinds of leukemia. Many of the transcription factor-induced cancers are characterized by translocation of chromosomes (Aplan, 2006) (see Chromosomal Abnormalities later in this chapter). One of the tumor suppressor genes, TP53, also acts as a transcription factor. In this role, TP53 “senses” DNA damage and halts cell division by controlling the expression of other genes that directly regulate the cell cycle (Ozanne, Spence, McGarry, & Hennigan, 2007).

Tumor Suppressor Genes

Tumor suppressor genes (also called anti-oncogenes) normally suppress or negatively regulate cell proliferation by encoding proteins that block the action of growth-promoting proteins. Using the example of a car, with cell growth caused by an accelerator, the tumor suppressor genes are the brakes, which can prevent cellular proliferation or suppress malignant transformation. At the cellular level, mutations in the cell cause tumor suppressor genes to lose function of both alleles. In other words, the loss of function or mutations of both copies of the gene are required for uncontrolled cell growth leading to tumorigenesis (National Center for Biotechnology Information, 2008).

Loss of Heterozygosity

Homozygosity refers to alleles that are identical. If there is an inherited mutation of a tumor suppressor gene, it is termed heterozygous because the alleles are different. Because there is a normal allele, the function of the gene and its protein product is maintained. Once the remaining allele is mutated, the gene and its product will lose normal functioning. The heterozygosity has been further altered and is now labeled as loss of heterozygosity (LOH). Cells can experience LOH with the loss of an entire chromosome, translocation of a piece of the chromosome, reduplication of a piece of chromosome that already has an abnormal gene, or the development of a point mutation in the second functioning allele. LOH is associated with cancer susceptibility genes such as oncogenes and tumor suppressor genes, such as TP53. Basic research is identifying an increasing number of tumor suppressor genes that, when mutated, are closely associated with the development and progressions of human cancers (American Cancer Society, 2005; Levine, Hu, & Feng, 2008; NCI, 2006; National Center for Biotechnology Information, 2008).
The tumor suppressor gene TP53 (located on 17p13) commonly has deletions and mutations associated with a wide variety of cancers, including lung, breast, esophageal, liver, bladder, and ovarian carcinomas; brain tumors; sarcomas; lymphomas; and leukemias. It is believed to contribute to half of all sporadic human cancers, making TP53 the most common genetic target for mutations leading to cancers (Levine et al., 2008). When TP53 is inherited in the germ line as a mutation, it is transmitted in an autosomal dominant fashion, a hallmark of Li-Fraumeni syndrome. This is a rare disorder causing multiple types of cancers, including soft tissue sarcomas, osteosarcomas, breast cancers, and different types of leukemias (Genetics Home Reference, 2007; Levine et al.).

**Specific Functions of Tumor Suppressor Genes**

Tumor suppressor gene products have specific functions in the cell nucleus and cytoplasm. If deregulation of the cell cycle occurs, which results in excess cell proliferation, the normal TP53 gene can halt cell division and induce programmed cell death, or apoptosis (Levine et al., 2008) (see Apoptosis later in this chapter).

Tumor suppressor genes also can encode for proteins in the cytoplasm. The NF1 (neurofibromatosis) gene encodes a protein that is similar to the proteins that modulate the ras oncogene function. Loss of NF1 may keep ras activated and prolong the signal for cell proliferation. Loss of other tumor suppressor genes, such as NF2 and APC (adenomatous polyposis coli), may cause cellular disorganization that leads to abnormal cell proliferation (Schindeler & Little, 2008).

**Insensitivity to Anti-Growth Signals**

In the normal cell, anti-growth signals move the cell out of the cell cycle growth phases into the resting from proliferation, or G0, phase. The TGF-β, referred to as both tumor or transforming growth factor, pathway is the best example of a signaling mechanism that causes the inhibition of cell growth and proliferation. This occurs in two ways in the normal pathway. First, it prevents inactivation of the retinoblastoma protein (pRb), a tumor suppressor protein and synthesis of the tumor suppressor genes p15INK4a and p21. Second, synthesis of these genes will block cyclin, a protein that allows cells to move into the cell cycle. In cancer, the TGF-α pathway causes the loss and or mutation of the TGF-α receptor function (Dhara, 2008). The tumor suppressor function of one protein, SMAD4, is eliminated because of mutation of its gene (GeneCards, 2009b). The p15 protein is not synthesized, so cyclin is not blocked, thereby allowing cells to continuously move into the active cell cycle with growth and proliferation. Finally, pRb tumor suppressor function is lost. Any of these interfering mechanisms, alone or in combination, allow the cell continued growth and proliferation (Dhara).

**Evasion of Apoptosis**

Apoptosis is “programmed” cell death. With this process, cell death is a controlled, deliberate, and distinct series of biochemical and cellular changes that allows an organism to remove old, dead, or unwanted cells. There is no inflammation, and some of the cellular materials are ingested by neighboring cells and reused. Apoptosis is a normal process that occurs when there is severe or irreparable damage to the DNA in order to prevent duplication of inaccurate messages (Zhivotovsky & Orrenius, 2006). The ABL and BCR genes are associated with this process (see Figure 1-8). A translocation of ABL on chromosome 9 to the BCR locus on chromosome 22 becomes the Philadelphia chromosome, prevents apoptosis, and is associated
with chronic myeloid leukemia (Zhivotovsky & Orrenius).

Cells that lose their ability to signal apoptosis contribute to early tumorigenesis because they are unable to repair problems in the DNA in order to eliminate genetically damaged cells. Without repair, survival of damaged cells ultimately leads to tumorigenesis (Zhivotovsky & Orrenius, 2006). Inactivation of the TP53 gene leads to decreased apoptosis and rapid tumor progression. The loss of TP53 function may indirectly contribute to tumor development by permitting the proliferation of mutated cells (Levine et al., 2008).

Follicular lymphoma, a type of indolent (slow-growing) non-Hodgkin lymphoma, is an example of the loss of apoptosis. This slow-growing lymphoma accounts for approximately 20% of all non-Hodgkin lymphomas and commonly has a rearrangement of the BCL2 gene. The overexpression of the bcl2 protein inhibits apoptosis, allowing continued cellular proliferation and making it difficult to destroy this lymphoma (NCI, 2009). It is hypothesized that restoration of apoptosis may provide an approach to cancer therapy.
**Sustained Angiogenesis**

Tumor cells have limitations in oxygen supply that cause areas of hypoxia. This boosts the need for glucose uptake and glycolysis to generate energy, resulting in lactate production. Although this supports the use of positron-emission tomography in nuclear medicine to identify increased metabolism, it also can result in decreased adenosine triphosphate production and ultimately contribute to the fatigue experienced by patients with a malignancy (Stasi, Abriani, Beccaglia, Terzoli, & Amadori, 2003). This lack of oxygen also curtails the proliferation of the malignant cells (Hanahan & Folkman, 1996). For tumors to grow to a larger size, they need to develop a microcirculatory system, through the process of angiogenesis (Hanahan & Weinberg, 2000).

VEGF causes the growth of new vessels, forming a microcirculatory system in a tumor (i.e., angiogenesis). Tumor cells induce hypoxia, leading to the transcription of VEGF-alpha, which binds to cell surface receptors and ultimately causes increased blood vessel permeability, angiogenesis, and the proliferation of cells (Nguyen et al., 2006). Multiple malignancies, including metastatic breast and colorectal cancers, overexpress VEGF.

**Tissue Invasion and Metastasis**

**Altered Cytoskeletal Control**

Cells have a skeleton with interior and exterior functions. The shape and the cell’s ability to move are included in the external function of the cytoskeleton. The internal function of the cytoskeleton permits substances to move within the cell. On the exterior membrane, microtubules evoke a rigidity to add strength to the membrane surface. Internally, they promote movement of organelles within the cytoplasm. During mitosis, the microtubules are arranged in a centriole as nine bundles of three microtubules each. These form the spindle fibers, which are responsible for the separation of the chromosomes prior to the actual splitting of the cell (McCance, 2010). With a malignancy, the cell loses external cytoskeleton control. This enables the cell to lose rigidity and be more amenable to continued cellular division. In addition, the cytoskeleton is needed for spindle microtubule formation, mitosis, and cellular growth. Multiple protein types participate in these changes associated with malignant transformation (Liaw, Chang, & Kavallaris, 2007).

**Altered Mobility of Membrane Components**

Proteins, glycoproteins, and glycolipids are known to have altered mobility on the membrane of a malignant cell. One outcome of this change enables the cancer to avoid immune surveillance. Other outcomes could promote spread and metastases (Hanahan & Weinberg, 2000; Wallach, 1968).

**Modified Contact Adhesion and Inhibition of Movement**

After replication, normal cells contact the adjacent cell membrane and are inhibited to grow. Malignant cells lose this characteristic and continue to proliferate even though they are touching the cell next to them. This contributes to the lack of control in malignant cells (Hanahan & Weinberg, 2000; McCance, 2010).

**Altered Surface Charge Density**

The electrical potential of the malignant cell membrane has a lower level than that of a normal cell. This is because of the increased amounts of negatively charged phospholipids in the cell membrane (Dobrzynska, Szachowicz-Petelska, Sulkowski, & Figaszewski, 2005). Positively charged sodium and calcium channels
contribute to apoptosis (Williams & Djamgoz, 2004). Changes in the charge of the cell membrane inhibit apoptosis and contribute to the longevity of the malignant cell.

**Increased Lectin Agglutinability**

Alterations in lectin binding enable leukocytes to adhere to and cover malignant cells. This change allows the malignant cell to escape surveillance and travel to distant sites in the body as a bolus of normal and abnormal cells (Hanahan & Weinberg, 2000; McCance, 2006).

**Limitless Replication Potential**

One factor allowing the potential for limitless replication is the expression of telomerase. This enzyme prevents the destruction of the telomere. Because telomeres protect chromosomes, cells with increased telomerase are known to be associated with longer telomeres and longevity of cell life. Short telomeres are associated with a shorter life span. Cancer stem cells are known to have increased levels of telomerase and thus have an extended life enhanced by the protected telomeres at the ends of the chromosome. This also protects the cell from apoptosis (NCI, 2010).

**Conclusion**

The curricula in undergraduate nursing programs do not typically include molecular biology. With completion of the sequencing of the human genome in 2003, many diagnostics and treatments have been developed that require a level of understanding of certain characteristics of the cell, the central dogma, and how cell signaling and communication occur. For nurses in oncology, this knowledge is important to understand and helps them to anticipate the symptoms of cancer in their patients, to be aware of how the treatments work, and to have a basic foundation to use when developing teaching plans related to individualized treatment plans for their patients. This chapter has provided a description of how malignancies develop and some of the molecular biology of the cell that is used for diagnostics and treatment in oncology. These include self-sufficiency in growth signals; insensitivity to antigrowth signals; evasion of apoptosis; sustained angiogenesis; tissue invasion and metastasis; and limitless replicative potential (Hanahan & Weinberg, 2000). For people new to oncology or for those who simply want to review and close some gaps in their knowledge, an understanding of the biology of cancer will assist them as they care for patients and their families.

**References**


