The Biology of Cancer: What Do Oncology Nurses Really Need to Know

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Objectives: *To describe the impact of genetics and genomics on the biology of cancer and the implications for patient care.*

Data Sources: *Pubmed; CINAHL.*

Conclusions: *Cancer research in genetics/genomics has identified new mechanisms influencing personalized risk assessment/management, early detection, cancer treatment, and long-term screening/surveillance.*

Implications for Nursing Practice: *Understanding the basics of genetics/genomics on the biology of cancer will facilitate patient education and care delivery, including the administration and monitoring of genetically targeted therapies whose toxicities may in part be mediated by the molecular pathways targeted by the specific agent.*

Key Words: *Cancer biology, cancer genetics, tumor biology, molecular biology cancer*

MOST CANCER is diagnosed as people grow older; currently at a median age of 66 years.[1] The DNA molecule was discovered in 1943 and the double helix first described in 1953.[2] Until the late 1960s, high school textbooks contained little explanation of DNA beyond the simple structure and partnering of the nucleotides. Since the human genome was sequenced when the oldest baby boomers were entering their 50s, most newly diagnosed patients with cancer have little knowledge of genetics and genomics or how these sciences have changed treatment for malignancies. In addition, until 2008, application of genetics and genomics was not included in the curricula of most nursing education programs.[3] This reveals a gap in knowledge for most individuals diagnosed with cancer and their professional caregivers.

The sciences of genetics and genomics (hereafter referred to as "genomics") are assisting with the interpretation of the working mechanisms of cancer. Today, patients with cancer may have heard about genomics and searched the Internet

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for information related to their diagnosis. However, Internet-accessed information may be too technical or easily misinterpreted. Oncology nurses at all levels of care need to be able to provide patients with accurate information about the impact of genomics on risk identification and management, diagnosis, treatment, and surveillance of cancer. In addition to foundational information about DNA, complex molecular aspects incorporate epigenetics, additional RNA types, newly identified cancer growth factors, and various kinases into signaling pathways cascading within the cell. These genomic-associated cell mechanisms are affecting personalized patient care; requiring an expanding knowledge base for nurses. This article describes how the genetics/genomics revolution has changed what oncology nurses really need to know to meet the needs of their patients.

Models of Cancer Development

All cancer is a result of accumulated inherited[4] and/or acquired genetic mutations causing oncogene activation, tumor suppressor gene inactivation, and production of telomerase (an enzyme enabling continued cancer growth[5] through lengthening of telomeres, and DNA sequences at the ends of chromosomes that normally shorten with aging).[6] One model of carcinogenesis that oncology nurses could use to explain how malignancies develop is Knudson's random two-hit model (the Stochastic model).[7] In this model, a single "hit" by a DNA damaging agent mutates the DNA in a single cell of a chromosome pair, a second hit in the undamaged chromosome is required for gene function to be lost and cancer to develop. This mechanism is especially important if the damage is in a tumor suppressor gene such as *p53*. Inherent within this model is the difference between cells that join to create an embryo (germline) and all other cells of the body (somatic).[8] For inherited cancers, the first hit occurs in a germline cell and, therefore, is in almost every cell of the body; the second hit occurs in single cells after birth, and cancer generally develops at a younger age. For sporadic cancers, both hits accumulate in one somatic cell, are acquired often years after birth, and typically cause cancer later in life.[4] In reality, however, an accumulation of mutations in at least four genes is required to result in cancer.[9]

Essential in Knudson's two-hit model is the concept of random expansion of cancer cells, in which all proliferating malignant cells have the capability to form new tumors. A newer hypothesis extending this concept, the cancer stem cell model, suggests that all cells can proliferate but only rare stem cells from the original tumor have the ability to form metastatic tumors in a new location.[10,11] These rare cancer stem cells seem to remain in a resting phase of cellular growth, resistant to treatment, which targets rapidly proliferating cells. This model explains how a patient could initially have tumor regression but still develop a resistant tumor years after any sign that cancer remained in the body.[12]

DNA, Chromosome Structure and Function

Each of the 23 human chromosome pairs consists of a single double-helix DNA molecule. The chromosomes are a fragile string of millions of base pairs coiled around proteins, called histones. Much like multiple, tiny spools of thread, the histones tightly wind and coil the DNA strands for each chromosome. Once coiled, the DNA continues to be twisted (akin to a continually twisted jump rope curling back on itself) until all 23 chromosomes are tightly wound and able to fit within the nucleus of every human cell.[13]

Each chromosome has a constricted area (centromere) with a short arm ("p" for "petite") and a long arm ("q"; the next letter in the alphabet). The arms are numbered, beginning from the centromere (eg, p1, p1.1, p2; and q1, q1.1, q2), to designate the location of a gene. A common example is the *BRCA1* gene which, when mutated, increases susceptibility to breast and ovarian cancer. The gene location is designated as 17q21, which identifies position 21 on the long arm of chromosome 17. An uncertain gene locus might be designated 5p7-10, designating a chromosomal region (p7-10) where a gene is believed to be located.[14]

The DNA double helix is composed of a phosphate molecule, a sugar, and pairs of four chemical bases: the purine bases guanine and adenine, and the pyrimidine bases cytosine and thymine (designated G, A, C and T, respectively). Bases T and A are consistently paired, as are G and C. Because each pair consists of a purine and pyrimidine, their molecular structure and hydrogen bonds