Significant variability exists in normal tissue reactions in patients with cancer receiving radiotherapy, with a subpopulation exhibiting increased toxicity to ionizing radiation. Genomic studies have proposed that single nucleotide polymorphisms in DNA repair genes, cytokines, and reactive oxygen species may play a role in clinical radiosensitivity. Additional research examining the association between genetic variants and radiation-induced inflammation and fibrosis may spur the development of targeted therapy in radiation oncology, which could increase cure rates and limit toxicity. As more people become long-term cancer survivors, oncology nurses must aggressively assess and manage late treatment side effects to optimize patient functioning and quality of life. The purpose of the current article is to describe the effect of ionizing radiation on normal and irradiated tissue, discuss genetic mutations that are proposed to influence radiosensitivity, and identify future areas of research on the association between genetics and radiation toxicity.

Carol Proud, MSN, CRNP, ANP-BC, AOCNP®, is an oncology nurse practitioner in the Department of Radiation Oncology at the University of Pennsylvania Medical Center in Philadelphia. The author takes full responsibility for the content of the article. The author did not receive honoraria for this work. The content of this article has been reviewed by independent peer reviewers to ensure that it is balanced, objective, and free from commercial bias. No financial relationships relevant to the content of this article have been disclosed by the author, planners, independent peer reviewers, or editorial staff. Proud can be reached at carol.proud@uphs.upenn.edu, with copy to editor at CJONEditor@ons.org. (Submitted April 2013. Revision submitted July 2013. Accepted for publication July 15, 2013.)

Key words: genetics; genomics; late effects of cancer treatment; radiation therapy

Digital Object Identifier: 10.1188/14.CJON.185-189