Prostate cancer is the second most common type of cancer among men in the United States, after skin cancer. The American Cancer Society (ACS) estimated that more than 186,320 new cases of prostate cancer were diagnosed in the United States in 2008 (Jemal et al., 2008), representing 33% of all new cancer diagnoses among men in that year. Although the numbers of men affected—one in six—are almost overwhelming, mortality is relatively low: Only one in 35 men will die of the disease. Also, mortality figures for prostate cancer are decreasing (ACS, 2007b; Jemal et al.). The difference between incidence and mortality is large, ensuring that men with prostate cancer comprise a significant percentage of the cancer survivor population (Jemal et al.). Prostate cancer is a chronic illness that threatens the health and well-being of a substantial proportion of older men (Lepore, Helgeson, Eton, & Schulz, 2003).

### Background

The treatment options for prostate cancer (based on the stage or extent of the cancer, the age of the man, and his associated comorbidities) are surgery, radiation therapy, androgen-deprivation (hormonal) therapy (ADT), and watchful waiting (National Cancer Institute [NCI], 2005). More than 50 years ago, Huggins and colleagues documented the dependence of the prostate gland upon androgens. Androgens are important in growth regulation of the prostate gland and in the pathogenesis of prostate cancer (Denis & Griffiths, 2000; Rashid & Chaudhary, 2004). Hormonal therapy is designed to interrupt the supply of testosterone to prostate cancer cells, thus interfering with their growth. ADT (generally achieved with administration of a gonadotropin-releasing hormone agonist), remains a well-established treatment option for men with metastatic or locally advanced disease, as an adjuvant to local therapy, and in cases of prostate-specific antigen (PSA)-only occurrence (Sharifi, Gulley, & Dahut, 2005). However, inducing castration levels of testosterone with ADT is not without significant deleterious side effects. ADT contributes to osteoporosis, anemia, loss of muscle mass, weight gain, decrease in high-density lipid cholesterol, and subjective complaints of breast tenderness and enlargement, hot flashes, decreased cognitive function, fatigue, and depression (ACS, 2007a; Higano, 2003; O’Connor & Fitzpatrick, 2006). Additionally, patients experience significant sexual side effects from...