RESEARCH BRIEF

An Exploratory Study of Cognitive Function and Central Adiposity in Men Receiving Androgen Deprivation Therapy for Prostate Cancer

Jamie S. Myers, PhD, RN, AOCNS®, FAAN, Alana Manson, PhD, Sandra A. Billinger, PT, PhD, FAHA, William Parker, MD, Jill Hamilton-Reeves, PhD, RD, CSO, Francisco J. Diaz, PhD, Ron Krebill, MPH, and Sally Maliski, PhD, RN, FAAN

OBJECTIVES: To prospectively assess cognitive function, anthropomorphic measures, and bone mineral density in men receiving androgen deprivation therapy (ADT) for prostate cancer; explore relationships between cognitive function and central adiposity; and gather preliminary data from a personalized education, exercise, and nutrition intervention.

SAMPLE & SETTING: 33 participants consented from a randomized controlled intervention trial.

METHODS & VARIABLES: Neurocognitive performance and self-report of cognitive function were assessed at baseline and 6 and 12 months. Dual-energy x-ray absorptiometry (DEXA) scans were obtained at baseline and 6 months.

RESULTS: No between-group differences in cognitive function were demonstrated. Increased visceral adiposity was not associated with decrements in visuospatial abilities. Significant increases in fat mass without increases in body mass index or waist–hip ratio provided further evidence for DEXA as the preferred central adiposity measure.

IMPLICATIONS FOR NURSING: Well-powered prospective research is needed to fully characterize the effects of ADT on cognitive function and the potential benefits of exercise and nutrition-based interventions.

KEYWORDS prostate cancer; cognitive function; androgen deprivation; central adiposity

***ONF*, *49*(2), 142–150.**

DOI 10.1188/22.ONF.142-150

Significant metabolic risks are inherent during androgen deprivation therapy (ADT) for prostate cancer (Melloni & Roe, 2020). Testosterone suppression causes decreases in bone mineral density (BMD), central weight gain (subcutaneous and visceral fat), and sexual dysfunction (Nguyen et al., 2018) and may impair cognitive function, negatively affecting quality of life (Shahinian et al., 2006).

Cognitive decline represents an area of major clinical concern, particularly considering the potential irreversibility of the loss. The trajectory of cognitive function decline for men with prostate cancer has not been well characterized. Testosterone metabolites (dihydrotestosterone for spatial ability and estradiol for verbal memory) are important for cognitive function (Hampson et al., 2015) (see Figure 1). Central adiposity and secretory products related to visceral fat, including inflammatory cytokines such as tumor necrosis factor and interleukin-6 (Montague & O'Rahilly, 2000), are postulated to impair cognitive function (Dahl et al., 2010). Because ADT is a known risk factor for increased central adiposity, it may affect cognition directly and indirectly. However, study results have been mixed (Treanor et al., 2017).

The relationship between increased body mass index (BMI), increased visceral fat, and BMD is somewhat contradictory. Although increased BMI typically is associated with increased BMD, there is an inverse relationship between visceral fat and BMD (Eckstein et al., 2016). This inverse relationship likely is because of a chronic inflammatory state and cytokine-related stimulation of bone resorption. The association of visceral fat with increased production of adiponectin