State of the Science: Stress, Inflammation, and Cancer

Judith K. Payne, PhD, RN, AOCN®, FAAN
2013 State-of-the-Science Lectureship

Diagnosis with a life-threatening illness such as cancer is almost universally experienced as stressful. The construct of stress has received substantial consideration as a correlate or predictor of psychological and health outcomes (Andersen et al., 2004) and has often been conceptualized within a stress and coping framework (Lazarus & Folkman, 1984). Biobehavioral factors have long been thought to affect many health processes. The relationship between inflammation of stress and cancer originated centuries ago and is now recognized as a facilitating characteristic of cancer (Mantovani, Allavena, Sica, & Balkwill, 2008). In addition, stress and the stress response are probable mediators of the effects of psychological factors on cancer, and specifically on progression of cancer (Powell, Tarr, & Sheridan, 2013). A substantial amount of new research activity has enlightened scientists and clinicians on the neuroendocrine regulatory function of physiologic pathways in cancer growth and progression (Lutgendorf, Sood, & Antoni, 2010; Thaker & Sood, 2008). It has been well documented that stress increases inflammation at the cellular level, which can directly influence responses from the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS), as well as contribute to changes in health-related outcomes (Antoni, 2013). Although some stress can be beneficial, excessive stress throughout a long period of time can result in inflammation. Although stress has been described by many terms, “at the cellular level it has been called inflammation” (Xing, 2012, p. C7).

Inflammation is a physiologic reaction generated by the body in response to injury, infection, or irritation (Reuter, Gupta, Chaturvedi, & Aggarwal, 2010). The links between inflammation and cancer can be viewed as two pathways: “An extrinsic pathway, driven by inflammatory conditions that increase cancer risk, and an intrinsic pathway, driven by genetic alterations that cause inflammation and neoplasia” (Mantovani et al., 2008, p. 436). Inflammation can be either acute or chronic. Acute inflammation is an initial stage of inflammation, called innate immunity, which is mediated through activation of the immune system and can help ward off infections (Reuter et al., 2010). If acute inflammation persists for a short time, it can be beneficial. If inflammation lingers over time, chronic inflammation sets in and may predispose the individual to various illnesses, including cancer (Lin & Karin, 2007).