Brain, Behavior, and Immunity 23 (2009) 159–166

Contents lists available at ScienceDirect

Brain, Behavior, and Immunity

journal homepage: www.elsevier.com/locate/ybrbi

Invited Review

Psychological intervention and health outcomes among women treated for breast cancer: A review of stress pathways and biological mediators ☆

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ARTICLE INFO

Article history:
Received 3 June 2008
Received in revised form 22 July 2008
Accepted 8 August 2008
Available online 22 August 2008

Keywords:
Psychosocial intervention
Breast cancer
Stress and psychological adaptation
Neuroendocrine
Immunologic
Health outcomes

ABSTRACT

Breast cancer is a common cancer among American women. The diagnosis, treatment, and the challenges of survivorship all have potential to increase women's levels of distress to levels that might influence their adaptation and possibly the course of their disease. Psychological distress can influence tumor progression via many different pathways (e.g., genetic changes, immune surveillance, pro-angiogenic processes). Psychological intervention has been shown to facilitate psychological adaptation to breast cancer. But can psychological intervention influence cancer relevant biological outcomes among breast cancer survivors? We review the literature on how psychological intervention can influence cancer relevant biological outcomes among breast cancer patients. We limited the present review to randomized controlled trials reported in the past 6 years that tested the effects of psychological intervention on biological dependent variables among patients with non-metastatic breast cancer. There are data to suggest that psychological intervention can influence neuroendocrine (e.g., cortisol) and immune function indicators, especially lymphocyte proliferation and TH1 cytokine production. Future psychological intervention studies should also focus on more newly discovered stress-tumor pathways (e.g., neuroendocrine processes promoting tumor growth and metastasis) and follow larger cohorts of the more vulnerable patients over longer periods to evaluate the biobehavioral mechanisms and lasting effects of these interventions on health and quality of life.

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1. Introduction

Breast cancer is the second leading cause of cancer death among women in the USA. In 2007 an estimated 178,480 women were diagnosed with invasive breast cancer and approximately 40,460 died of the disease (American Cancer Society, 2007). With advances in treatment and increased rates of early detection, however, the number of breast cancer survivors is increasing such that at the end of 2004 (the most recent date available) there were 2.4 million breast cancer survivors in the USA (Ries et al., 2007).

Despite the increasing survival rates, however, breast cancer continues to be a stressful experience for those affected (Carver et al., 1993). Breast cancer patients have significant psychosocial concerns and needs, which vary along the disease trajectory (diagnosis, active treatment, survivorship) (e.g., Spencer et al., 1999). Because of this, a recent Institute of Medicine (IOM) report recommended that psychosocial intervention (PI) be incorporated into standard medical care for breast cancer patients at all phases of treatment (Hewitt et al., 2004). A number of research studies have shown that PI can improve psychological functioning among breast cancer patients (e.g., Luebbert et al., 2001; Meyer and Mark, 1995; Trijsburg et al., 1992; Zimmermann et al., 2007). While there is a growing literature documenting the effects of stress on cancer relevant biological processes (Antoni et al., 2006b), less is known about how PI can influence these biological process in breast cancer patients. The purpose of the present manuscript is to review the literature demonstrating PI effects on biological outcomes among breast cancer patients. However, we will first briefly summarize several of the cancer relevant biological processes which may be influenced by stress and thus amenable to stress-modulating PI.

2. Stress influences on cancer relevant biological processes

As illustrated in Fig. 1, psychological distress can negatively influence multiple cancer relevant biological processes. Cancer initiation and progression is a complex process that relies on multiple steps including environmental exposures and behaviors, genetic changes, evasion of apoptosis, proliferation, escape from immune surveillance, vascularization, and metastases. There is emerging

☆ Funded by grants from the National Cancer Institute (KO7 CA107085-01; R01-CA-064710).

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0889-1591/$ - see front matter © 2008 Elsevier Inc. All rights reserved.
doi:10.1016/j.bbi.2008.08.002

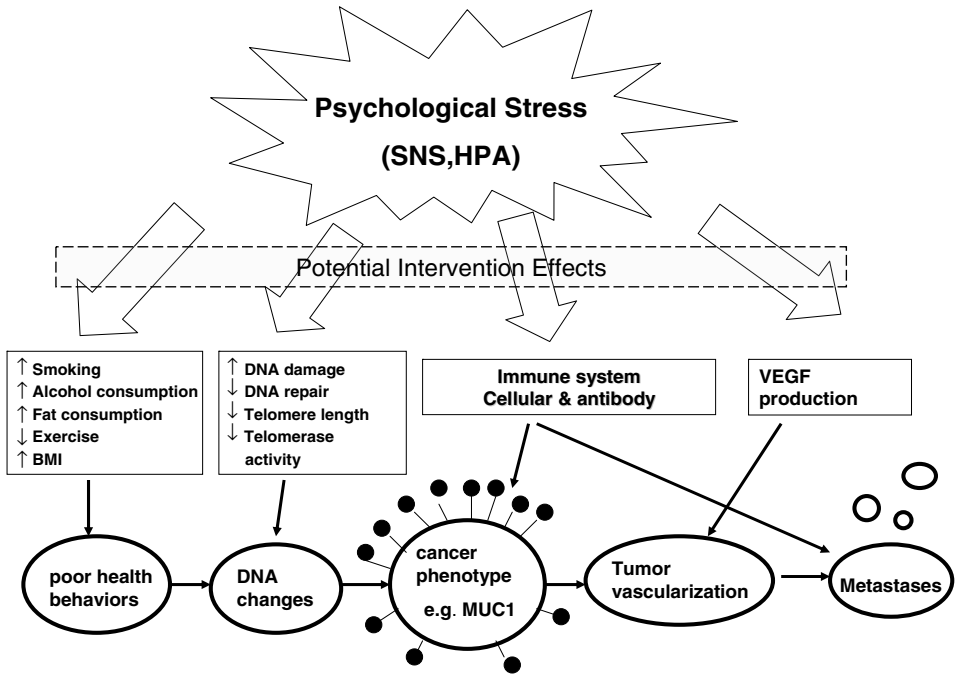

Fig. 1. The development and progression of cancer and how psychological stress and psychological interventions might influence the process. SNS, sympathetic nervous system; HPA, hypothalamus pituitary adrenal axis; BMI, body mass index; VEGF, vascular endothelial growth factor.

evidence that psychosocial stress can influence the course of disease at many points during this process (Antoni et al., 2006b).

Psychological distress is associated with health behaviors that may promote tumor growth and development. Distress states are associated with increased body mass index (BMI) and greater waist circumference (Wing et al., 1991). Increased body weight, especially central adiposity, is associated with increased risk for breast cancer (Connolly et al., 2002). Increased BMI is thought to be due to stress-related increases in consumption of sweet foods and high fat foods (O'Connor et al., 2008), and stress-related decrements in physical activity (e.g., Scully et al., 1998). Stress-related increases in waist circumference are due to the fact that visceral fat is highly vascular so is more accessible to factors in the blood, such as cortisol. Visceral fat also has a high concentration of glucocorticoid receptors. When cortisol binds to these receptors, the lipoprotein lipase (a fat-storing enzyme) gene in adipose tissue is activated and increased visceral fat storage results (Bjorntorp and Rosmond, 1999).

Psychological distress states are also associated with changes in gene function. Psychological stress is associated with increased DNA damage and poorer DNA repair (Flint et al., 2007; Gidron et al., 2006), and DNA repair pathways are important in the etiology of breast cancer. The two known genes associated with increased breast cancer risk, BRCA1 and BRCA2, both code for proteins that are involved in DNA repair pathways (Venkitaraman, 2002). Chronic stress is also associated with shortened telomeres and decreased telomerase activity (Epel et al., 2004), and genetic instability associated with telomere dysfunction (i.e., short telomeres) is an early event in tumorigenesis (Wu et al., 2003).

There is a large literature documenting the effects of psychological distress on immune function (Segerstrom and Miller, 2004). In the past 10 years, it has become clear that human breast cancer is immunogenic (Disis and Lyerly, 2005). The immune system can recognize breast cancer antigens (Disis and Lyerly, 2005) such as HER-2 Neu (Disis et al., 1999) and MUC-1 (Finn et al., 1995), and patients who have immune responses to tumor antigens have better outcomes (von Mensdorff-Pouilly et al., 2000). Tumor eradication may be influenced by many different immune pathways. Antigen presenting cells, such as dendritic cells, natural killer (NK) cells, cytotoxic-T-cells, T regulatory (T regs) cells, and B cells, are all believed to play an important role in the host response against spontaneous tumors (Disis and Lyerly, 2005) and in dealing with metastatic shed of tumor cells (Melief and Kast, 1991). Tumor cells evade detection by the immune system not only because they present antigens that are recognized as "self" by the host, but also by actively secreting immunosuppressive factors (e.g., Transforming Growth factor-beta (TGF-β), interleukin (IL)-10, prostaglandin E2 (PGE2) (Wojtowicz-Praga, 1997).

Elevated levels of psychological distress have been associated with suppressed cellular immune function among breast and ovarian cancer patients. In a study with 116 stage I–III breast cancer patients in the weeks after surgery, increased psychological distress (intrusive thoughts about cancer) were associated with decrements in lymphocyte proliferative response (LPR) to anti-CD3, natural killer cell cytotoxicity (NKCC), and NK cell response to interferon-gamma (IFNγ) (Andersen et al., 1998). In a study of ovarian cancer patients, greater levels of distress were associated with reduced NKCC in peripheral blood and also NKCC in the tumor microenvironment (Lutgendorf et al., 2005). Breast cancer patients have significantly lower NKCC compared to healthy controls, even among stage I–III patients, and NKCC appears to be even lower in stage IV patients (Baxevanis et al., 1993; Konjevic and Spuzic, 1993) and in those with liver metastases (Yamasaki et al., 1993). Among patients with solid tumors, higher NKCC predicted longer survival time without metastases over a 13-year period (Pross and Baines, 1988). *Lower* NKCC also predicted development of distant metastases in patients with head and neck tumors (Schantz and Goepfert, 1987). Thus, psychosocial distress has been associated with decrements in LPR and lower NKCC in cancer patients, including those with breast cancer, and this may in turn predict poorer clinical outcomes.

Psychological stress is thought to exert its influence on the immune system via both behavioral and neuroendocrine pathways. During periods of chronic stress, people are more likely to experi-

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