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Editorial

Cancer and Inflammation: Suppress Inflammation, Suppress Cancer?

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INTRODUCTION

In the nineteenth century, Virchow noted the presence of leukocytes in neoplastic tissue, and made a connection between cancer and inflammation. His hypothesis was based on the observation that tumors often arose at sites of chronic inflammation [1,2].

In the last decade, accumulating evidence has supported Virchow's hypothesis that cancer and inflammation are linked. Several possible mechanisms have been proposed for the relationship between cancer and the systemic inflammatory response. First, tumor growth or invasion could induce tissue inflammation. Second, tumor necrosis and hypoxia or local tissue damage might activate an inflammatory response. Third, cancer cells themselves could increase the production of inflammatory cytokines, such as IL-6 and IL-8. These inflammatory cytokines and chemokines interact with the immunovascular system and facilitate cancer growth, invasion, and metastasis [1,3,4].

In fact, many studies have demonstrated that the presence of a systemic inflammatory response, as evidenced by an elevated C-reactive protein (CRP) level, and other markers of the systemic inflammatory response, such as the Glasgow Prognostic Score (based on the CRP and albumin levels; the GPS) or neutrophil to lymphocyte ratio (NLR) is associated with a poor outcome in a variety of cancers [3-8].

In addition to the prognostic role of the systemic inflammatory response, recent studies have suggested that the systemic inflammatory response can be applicable as a marker of the response to cancer treatment and for predicting treatment-related toxicity [9-11].

More recently, in vol. 8, issue 7 of PLoS ONE, Zhang et al. have demonstrated that a Chinese herbal medicine could inhibit pancreatic cancer cell invasion and metastasis via the suppression of cancer- related inflammation [12]. They examined the effects of the Chinese herbal medicine Qingyihuaji Formula (QYHJ), a seven-herb Chinese medicinal formula, on pancreatic cancer cell invasion and metastasis in nude mice. The authors found that the treatment with QYHJ inhibited cancer- related inflammation in tumors by decreasing the infiltration of tumor- associated macrophages (TAM) and by decreasing the IL-6 production, resulting in a prevention of cancer cell invasion and metastasis. Moreover, based on *in vitro* functional studies, they confirmed that IL-6 could induce the pancreatic cancer cell epithelial- to-mesenchymal transition (EMT), which plays an important role in cancer metastasis and/or drug resistance, and promotes cell invasion. Therefore, they concluded that the inhibition of the IL-6 production by QYHJ might result in reduced EMT and invasion in pancreatic cancer.

Drugs that target cancer- related inflammation have the potential to re- educate a tumor- promoting inflammatory infiltrate, to prevent such cells from migrating to the tumor site, or to induce a tumor- inhibiting microenvironment, thus leading to the suppression of cancer [2,12]. Although COX2 inhibitors, anti-TNF, and anti- IL-6 therapies have been successfully introduced for the treatment of inflammatory conditions, there has still been little experience in cancer patients [13]. In this context, the study by Zhang et al. is exciting and provides a ray of hope for patients with cancer.

CONFLICT OF INTEREST

The authors declared that they do not have anything to disclose regarding funding or conflict of interest with respect to this manuscript.

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