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Oxidative stress and antioxidant status in non-metastatic prostate cancer and benign prostatic hyperplasia

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Abstract

Objectives: We undertook the present study to investigate the possible alteration of oxidant/antioxidant status in the circulation of patients with prostate cancer and benign prostatic hyperplasia.

Design and methods: Thiobarbituric acid reactive substances (TBARS), the enzyme activities of superoxide dismutase (SOD), glutathione peroxidase (GPX), catalase (CAT) and copper (Cu) and zinc (Zn) levels were estimated in the erythrocytes of 25 non-metastatic prostate cancer patients, 36 benign prostatic hyperplasia (BPH) patients and 24 age- and sex-matched healthy subjects (controls).

Results: TBARS concentrations were significantly increased, while erythrocyte GPX and SOD activities were significantly decreased in the prostate cancer group versus controls ($P < 0.001$) and BPH group ($P < 0.05$). Zn levels were lowered in prostate cancer patients versus controls ($P < 0.01$) with no significant changes between BPH and cancer groups. Similarly, lipid peroxidation was increased ($P < 0.05$) with decreased SOD activity and Zn level ($P < 0.05$) in BPH versus controls.

Conclusion: These results reveal an alteration in the lipid peroxidation index, with concomitant changes in the antioxidant defense system in prostate cancer patients compared to BPH patients. We hypothesize that an altered prooxidant–antioxidant balance may lead to an increase in oxidative damage and consequently may play an important role in prostate carcinogenesis.

Keywords: Prostate cancer; Oxidative stress; Lipid peroxidation; Antioxidant enzymes

Introduction

Prostate cancer continues to be the most frequently diagnosed neoplasm and the second leading cause of cancer-related mortality in men [1–3]. Increasing evidence has indicated that oxidative stress is associated with aging and severe age-related degenerative diseases, including cancer [4–7]. A wide variety of reactive oxygen (ROS) and nitrogen species (RNS) can attack DNA directly and form mutagenic lesions. ROS may also cause the formation of DNA adducts indirectly by initiating autocatalytic lipid peroxidation, which generates a large variety of potential genotoxic breakdown products, including alkoxyl radicals ($LO^{\bullet}$), peroxyl radicals ($LOO^{\bullet}$), and aldehydes, such as malondialdehyde (MDA) [8,9]. Endogenous defenses against reactive oxygen species (ROS) include antioxidant enzymes such as glutathione peroxidase (GPX), catalase (CAT), and superoxide dismutase (SOD) [10]. Lower levels of essential antioxidants in the circulation have been found to be associated with an increased risk of cancer [11].

A few studies in the past have described the altered prooxidant–antioxidant status in the prostatic tissue of men, rats, or in permanent cell lines [12–16]. However, data concerning the antioxidant status and the degree of lipid peroxidation in the circulation of the prostate cancer patients are limited [17,18], and their results are conflicting. Therefore, we undertook the present study to evaluate the possible alteration of oxidant/

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