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Antioxidant status and lipid peroxidation in small intestinal mucosa of children with celiac disease

Vesna Stojiljković^a  , Ana Todorović^a, Snežana Pejić^a, Jelena Kasapović^a, Zorica S. Saičić^b, Nedeljko Radlović^c, Snežana B. Pajović^a

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Abstract

Objective

To explain the role of oxidative stress in the pathology of celiac disease.

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The activities of analyzed enzymes varied significantly between the examined groups. An increase in the activities of superoxide dismutase was observed in patients with active and silent celiac disease, while the activities of glutathione peroxidase and glutathione reductase and the glutathione content were significantly reduced. The level of lipid hydroperoxides was significantly elevated in these groups.

Conclusions

Oxidative stress is an important factor in the pathogenesis of celiac disease. The antioxidant capacity of celiac patients is significantly reduced, mostly by a depletion of glutathione. Natural antioxidants and appropriate dietary supplements could be important complements to the classic therapy of celiac disease.

Introduction

Reactive oxygen species (ROS) are produced during metabolic processes in cells. Because of their high reactivity towards biomolecules, ROS are potentially very dangerous. In physiological conditions deleterious effects of ROS are counteracted by antioxidant defense system consisting of nonenzymatic antioxidants (glutathione (GSH), vitamins A, C, E, carotenoids) and antioxidant enzymes superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and glutathione reductase (GR). If production of ROS overwhelms antioxidant (AO) capacity of the cell, a condition known as oxidative stress occurs. Lipid peroxidation (LPO), a chain reaction in which membrane lipids are oxidized, is one of the first consequences of oxidative stress. LPO provokes changes in membrane lipid bilayer structure, causing loss of membrane elasticity and selective permeability and disruption of its other functions [1], [2]. The damage provoked by LPO can be prevented by chain-breaking antioxidants (β -carotene, lycopene, vitamins A, C, E). On the other hand, progressive LPO can reduce extremely AO capacity of the cell by depleting its antioxidants [3]. It is well known that ROS and oxidative stress are involved in the pathogenesis of many human

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patients gastrointestinal symptoms are absent or not remarkable [10], [11]. The only treatment for celiac disease is a lifelong gluten-free diet (GFD), which usually leads to a rapid symptomatic improvement. Mucosal healing, however, may take months or years, particularly in adults [12]. GFD also prevents complications that may occur if the disease remains untreated, including osteoporosis and gastrointestinal neoplasms [13].

It is generally accepted that the activation of immune system by gluten peptides is responsible for pathogenesis and progression of CD [14], [15]. In addition, many studies indicate that direct cytotoxic effect of gluten on enterocytes may be one of the mechanisms underlying pathogenesis of CD [16], [17], [18]. In the last decade the results of several investigations showed that gluten corrupts the pro-oxidant–antioxidant balance in intestinal mucosa, probably by an overproduction of free radicals [19], [20], [21]. Nevertheless, the data concerning AO status of celiac patients are scarce. The results of few investigations indicate that AO capacity of celiac patients is reduced [22], [23], [24]. In our previous paper [25] we showed that oxidative stress is strongly associated with CD and that the AO capacity in peripheral blood of celiac patients is weakened by a depletion of GSH and reduced activities of GSH-dependent AO enzymes GPx and GR. In order to further examine the role of oxidative stress and AO status in the pathogenesis of CD, in this study we investigated the activities of AO enzymes manganese SOD (MnSOD), copper–zinc SOD (CuZnSOD), CAT, GPx and GR in the samples of small intestinal biopsies of children with different forms of CD. We also measured the concentrations of GSH and lipid peroxides (LOOH) in the same samples.

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The activities of antioxidant enzymes are shown in Fig. 1. All parameters, except CAT activity, varied significantly between the analyzed groups: MnSOD: $H = 9.57$, $P < 0.05$; CuZnSOD: $H = 10.92$, $P < 0.05$; CAT: $H = 3.99$, $P > 0.05$; GPx: $H = 17.19$, $P < 0.001$; GR: $H = 12.51$, $P < 0.01$ (Kruskal–Wallis test). In comparison to the controls, MnSOD activity was significantly elevated in silent group ($t_d = 2.69$, $P < 0.05$), while CuZnSOD activity was increased in active group ($t_d = 3.48$, $P < 0.01$). GPx activity was significantly lower...

Discussion

Several investigations suggested that free radicals are implicated in the pathogenesis of CD. Activation of xantine-oxidase (XO) is one of the mechanisms of overproduction of free radicals and ROS in small intestinal mucosa of celiac patients. The results of Boda et al. [28] suggest that in patients with active CD gluten, along with mucosal lesions of immunological origin, causes activation of XO in enterocytes, which results in overproduction of ROS and further damage to mucosa. These...

Acknowledgment

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...Therefore, studies on the clinicopathological aspects of gluten-related diseases are required. In the previous reports, oxidative stress has been shown to be an important mechanism that plays a crucial role in gluten/gliadin toxicity [22–25]. Results from the present study support this idea by showing that ROS production is induced following gliadin intake in *C. elegans*....

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...A preliminary cytotoxicity assay presented a significant change in the proliferation of HCT116 cells as compared to VC in the presence of 20 μ g gliadin and was further chosen for all experiments in present study. The antioxidant capacity during wheat intolerance disease such as CD has been reported to be significantly reduced mainly due to depletion of glutathione (Stojiljkovic et al., 2009). In this context, an adjunct of natural antioxidants and appropriate dietary supplements is often recommended as a standard therapy for a CD patient...

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by the selenium deficiency associated with chronic inflammatory damage of the small intestine [268]...

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...In addition, LPO generate more ROS, causing exacerbation of oxidative stress. Combined effects of LPO deleterious mechanisms in the GIT results in intestinal inflammation (Stojiljkovic et al., 2009), and consequently diarrhoea unless efficient inhibition mechanisms of the processes are in place. The good LPO inhibitory activity of the phenolic-enriched extracts of the Ozoroa and Searsia species tested in this work indicate that the plant have potential as supportive therapy in diarrhoea episodes....

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