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Original article

Ameliorative effect of vitamin E and selenium against oxidative stress induced by sodium azide in liver, kidney, testis and heart of male mice

Reham Z. Hamza[a,b], Mohammad S. AL-Harbi[b], Nahla S. El-Shenawy[c,*]

[a] Zoology Department, Faculty of Science, Zagazig University, Zagazig 44519, Egypt
[b] Biology Department, Faculty of Science, Taif University, Taif 888, Saudi Arabia
[c] Zoology Department, Faculty of Science, Suez Canal University, Ismailia 41522, Egypt

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ABSTRACT

The study purported to define the effects of daily administration of vitamin E (Vit E) and selenium (Se) on antioxidant enzyme activity in mice treated with high doses of sodium azide (SA). Male mice were randomly split into nine groups. Groups 1, 2 and 3 were injected daily with saline, Vit E, and Se, respectively, while groups 4, 5 and 6 administrated with different doses of SA (low, medium and high, respectively). The mice in groups 7, 8 and 9 received 100 mg/kg Vit E, 17.5 mg/kg Se, and a combination of Vit E and Se, respectively before the SA-treatment. Hepatic, renal, testis and heart, antioxidant enzymes as well as levels of lipid peroxidation and total antioxidant capacity levels were determined. Vit E alone affected on the antioxidant parameters of the examined tissues. Se had a preventive effect on the decrease of antioxidant parameters caused by SA and improved the diminished activities of all of them. The study demonstrates that a high dose of SA may alter the effects of normal level antioxidant/oxidative status of male mice and that Se is effective in reducing the SA-damage. Se acts as a synergistic agent with the effect of Vit E in various damaged caused by SA.

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1. Introduction

Sodium azide (SA) has been utilized for a wide variety of military, laboratory, medical, and commercial uses. NaN_3 was also used as a preservative in aqueous laboratory reagents and biologic fluids and as an ingredient to inflate automobile airbag gas [1]. Attempts have been made to revitalize the use of NaN_3 as a potential alternative to methyl bromide applications as an insecticide, herbicide, nematicide, fungicide, and antibacterial. More recent evidence also indicates that the hypotensive side effects of NaN_3 poisoning could be due to the direct vasodilator properties of this compound [2,3].

Several neurotoxic agents as SA are known to cause degeneration and excitotoxicity by raising the permeability of mitochondrial membrane through peroxidation of lipids in mitochondrial membrane [4]. This effect leads to the release of intra-mitochondrial calcium and hydrogen peroxide which facilitates the increasing the levels of calcium from extracellular sources (reactive oxygen species (ROS)-dependent calcium release) [5]. Somade et al. [6] found that quercetin protects against the extrahepatic oxidative stress induced in rats by SA.

The antioxidant enzymes that provide the first line of defense are included superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and glutathione reductase (GRx). Glutathione (GSH) is the primary cellular non-enzymatic antioxidant and plays an important role in the antioxidation of ROS and other free radicals and, as a thiol- containing coenzyme, in the detoxification of xenobiotic compounds [52].

Selenium (Se) is a trace element that involved in many important enzymes, including the GPx and the iodothyronine deiodinases. These enzymes have roles in protecting against oxidative damage [7] by decreasing lipid peroxidation (LPO) and resynthesized the glutathione [8]. The biological function of GPx is to reduce lipid hydroperoxides conversion to their corresponding alcohols and to reduce free H_2O_2 reaction [9].

Vitamin E (Vit E) protects cellular structures against damage from oxygen free radicals and LPO [10,11]. El-Shenawy et al. [12] observed that Vit E separately and in combination with Se has a protective effect on nephropathy and hepatic changes induced by SA. It has likewise been proven to play a role in immune function, other metabolic processes [13] and prevention of losing sperm [14].

* Corresponding author.
E-mail address: elshenawy_nahla@hotmail.com (N.S. El-Shenawy).

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El-Shenawy et al. [12] suggested that many biological processes including immunity, biochemical and histological changes effected by Vit E and Se. They have beneficial effects and could be able to antagonize aluminum toxicity [15]. Therefore, the present study was undertaken to evaluate the ameliorator property of Vit E or/ and Se on toxic status during SA-treatment and studying their possible improving effect on the antioxidant status of male mice.

2. Materials and methods

2.1. Chemicals

Vitamin E (Vit E-50), sodium azide (NaN_3) and selenium (sodium selenite; Na_2SeO_3 purity 98%) were purchased from Roche (Germany), KKW Riedel (Germany) and Applichem GmbH (Germany), respectively. Other chemicals were bought from standard commercial suppliers.

2.2. Experimental animals design

ICR male mice, weighing 35–40 g were provided by King Fahd Medical Research Center in Jeddah, Saudi Arabia. The animals were kept in polycarbonate cages with stainless steel wire bar lids and wooden dust as a bedding material. Mice had free access to diet and water for a week before starting the experiment. The European Community Directive (86/609/EEC) and National regulations on animal care have been habituated. After a week of acclimation, animals were randomly divided into nine groups of 10 animals in each one as following:

Groups 1, 2 and 3 were served as untreated control and received 1.0 mL/kg of distilled water, 100 mg/kg Vit E [16] and 0.5 mg/kg Se [17], respectively. Mice in groups 4, 5 and 6 were treated with different doses of SA (1.0, 5.0 and 17.5 mg/kg/day; [18], respectively. SA at a dose of 17.5 mg/kg body weights (B.W) followed by Vit E or/and Se were given to groups 7, 8 and 9. All the groups were treated intraperitoneally (i.p.) for 30 consecutive days.

2.3. Tissue homogenates preparation

2.3.1. Preparation of liver and kidney homogenates

Antioxidant parameters were determined in tissues of liver and kidney. Tissues were perfused with a 50 mM of sodium phosphate buffer saline (100 mM Na_2HPO_4/NaH_2PO_4, pH 7.4), 0.25 M sucrose and 0.1 mM ethylene diamine tetra-acetic acid (EDTA). Then, tissues were homogenized in 5 mL cold buffer/g tissue using a Potter–Elvehjem homogenizer. The homogenate was centrifuged (Z 216 MK HERMLE refrigerated centrifuge) at 10,000 x *g* for 20 min at 4 °C for estimating enzymatic assays and was centrifuged at 2500 rpm for MDA level, and the resultant supernatant transferred into Eppendorf tubes that preserved in a deep freeze until being used [19].

2.3.2. Preparation of testis homogenates

Antioxidant parameters were determined in testis. Tissues were perfused with a 50 mM of sodium phosphate buffer saline (100 mM $Na_2HPO4/NaH2PO_4$, pH 7.4), 0.25 M sucrose and 0.1 mM ethylene diamine tetra-acetic acid (EDTA) and then we added a protease inhibitor solution composed of A sulfhydryl reagent, 2-mercaptoethanol or dithiothreitol (0.1–0.5 mM) was added in testis tissues to protect enzymes and integral membrane proteins with reactive sulfhydryl groups, which are susceptible to oxidation [20]. Then, tissues were homogenized in 5 mL cold buffer/g tissue using a Potter–Elvehjem homogenizer. The homogenate was centrifuged at 10,000 × *g* for 20 min at 4 °C for estimating enzymatic assays and was centrifuged at 2500 rpm for MDA level, and the resultant supernatant transferred into Eppendorf tubes that preserved in a deep freeze until being used.

2.3.3. Preparation of heart tissue homogenates

Heart tissue samples were collected, weighed and added to 9-timesvolume of lysis buffer. The lysis buffer is 50 mM Tris-HCL with 2 mM EDTA, pH 7.4. Then we added protease inhibitor: 1 mM phenylmethylsulfonyl fluoride (PMSF) and 2 g/mL each of

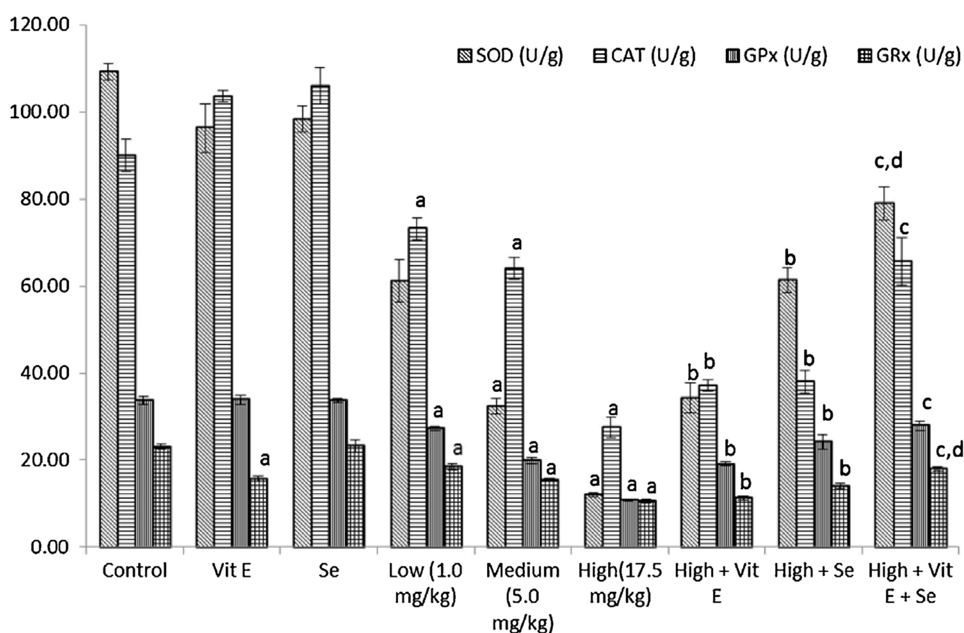

Fig. 1. Changes of antioxidant enzymes activities of liver tissue in male mice treated with vitamin E or selenium or both of them with different doses of sodium azide. Values are expressed as means ± SE; n = 10 for each treatment group. [a] Significant difference (P < 0.05) as compared to control group, [b] significant (P < 0.05) as compared to high dose of SA (17.5 mg/kg/day for 30 days), [c] significant difference (P < 0.05) as compared to the group of high dose with Vit E (100 mg/kg), [d] Significant difference (P < 0.05) as compared to the group of high dose with Se (0.5 mg/kg).

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