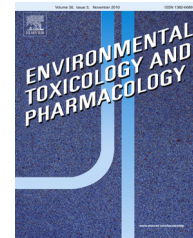




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# Interaction between blood selenium concentration and a levels of oxidative stress and antioxidative capacity in healthy children

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## ABSTRACT

The study aimed at defining the relationship between blood selenium concentration (Se-B) and levels of oxidative stress and antioxidative capacity in healthy children. The studies were conducted on 337 children (mean age:  $8.53 \pm 1.92$  years). The groups of individuals with Se-B <1st quartile (group I, Se-B <  $70 \mu\text{g/L}$ ), with Se-B fitting the range of 1st quartile and median (group II, Se-B:  $70\text{--}76.9 \mu\text{g/L}$ ), with Se-B between the median and 3rd quartile (group III, Se-B:  $77\text{--}83.9 \mu\text{g/L}$ ) and those with Se-B above the 3rd quartile (group IV, Se-B  $\geq 84 \mu\text{g/L}$ ) were distinguished. Level of oxidative stress was defined using determination of urine malonyldialdehyde concentration (MDA) and urine 8-hydroxy-2-deoxyguanosine concentration (8-OHdG). Urine total antioxidant status (TAS) was determined. In group IV TAS was significantly higher than in groups I–III. A positive correlation was detected between Se-B and TAS. In healthy children an appropriately high Se-B seems to ensure higher total antioxidative status.

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## 1. Introduction

In the second half of 20th century selenium was included to the group of trace elements. Since that time, numerous scientific investigations allowed to detect a wide

spectrum of properties manifested by the microelement (Rayman, 2012). Selenium represents a component of numerous enzymatic systems, mainly of glutathione peroxidases (selenium-containing proteins) (Burk and Hill, 2009). Selenium ions are indispensable for maintenance of appropriate function in immune system (Stone et al., 2010), procreative

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system (Boitani and Puglisi, 2008) as well as for physiological development of human foetus and child (Black, 2001). Nowadays, numerous scientific studies are being conducted aiming at determining significance of selenium in processes linked to inhibition of carcinogenesis (Rayman, 2012), an appropriate function of cardiovascular system (Lubos et al., 2010) and endocrine system (thyroid gland in particular) (Köhrle, 2013).

Currently, oxidative stress is thought to represent one of basic mechanisms leading to abnormal function of various body systems and to development of several diseases, including neoplastic diseases, neurological and psychiatric diseases (Parkinson's disease, Alzheimer's disease, autism, chronic fatigue syndrome), diseases of cardiovascular system (arteriosclerosis, heart failure, ischaemic heart disease) and dermatological diseases (lichen planus, albinism) (Otani, 2011). The body houses a delicate balance between intensity of oxidative stress, or generation and action of reactive oxygen species (ROS), and antioxidative capacity to detoxify reactive intermediate products or to eliminate biological effects of ROS (Augustin, 2010). Thus, the balance involves on one hand generation of ROS, first of all the superoxide anion radical ( $O_2^{\bullet-}$ ), hydrogen superoxide ( $H_2O_2$ ), hydroxy radical ( $OH^{\bullet}$ ), alkoxy radical ( $RO^{\bullet}$ ) and peroxyalkoxy radical ( $ROO^{\bullet}$ ), and products resulting from interaction of ROS with macromolecules (proteins, lipids and DNA) and, on the other existence of mechanism warranting antioxidative protection, involving mainly an adequate level of antioxidants. The oxidative damage to DNA may be reflected by concentration of DNA-8-hydroxy-2-deoxyguanosine (8-OHdg), concentration of malonyldialdehyde (MDA) represents a marker of lipid peroxidation while levels of advanced glycation end products (AGEs) provide an index of glycol oxidation of proteins (Hwang and Kim, 2007). The group of antioxidants is thought to include enzymes which catalyse the reactions eliminating ROS, including superoxide dismutases (SODs: zinc-cupric, manganese and superoxide dismutase), catalase (CAT), glutathione peroxidases (GSH-Px: cytosolic, gastroenteric, nuclear and lipid hydroperoxide GSH-Px), glutathione reductase (GR) and ceruloplasmins (CP), as well as nonenzymatic anti-oxidants:  $\beta$ -carotene (and other carotenoids:  $\alpha$ -carotene, lycopene, lutein and zeaxanthin,  $\beta$ -cryptoxanthin),  $\alpha$ -tocopherol (vitamin E), ascorbic acid (vitamin C), and selenium (Mayne, 2003).

The relationship between native blood selenium concentration and redox balance in the body seems interesting. In the available literature of the subject we have not identified sufficient references to unequivocally define the relationship between blood selenium concentration and levels of oxidative stress and antioxidant capacity in the population of healthy children. According to the current knowledge the data obtained in adult population, additionally population suffering from different diseases, cannot be extrapolated to the population of healthy children. Up-to-date studies connected with the problem of selenium and redox balance was based on the studies carried out in adult patients with numerous diseases. From our point of view it was the main goal was the investigation in the area of redox balance in healthy children, which is a novel idea in the aspect of evidence-based medicine.

**Table 1 – Clinical characteristics in the whole study group.**

	$X_G$	X	SD	Min	Max
Age (years)	8.35	8.53	1.92	5.00	14.00
Height (m)	1.35	1.36	0.14	1.05	1.84
Body mass (kg)	31.41	33.45	13.31	14.00	100.80
BMI ( $kg/m^2$ )	17.25	17.58	3.58	8.54	35.71
Se-B ( $\mu g/L$ )	76.54	77.47	12.19	51.00	130.00
MDA ( $\mu g/g$ creatinine)	4.68	6.59	6.63	0.13	54.37
8-OHdg ( $\mu g/g$ creatinine)	3.96	4.75	2.45	0.17	14.33
TAS ( $\mu g/g$ creatinine)	2.82	3.89	5.16	0.06	70.70

8-OHdg – urine 8-hydroxy-2-deoxyguanosine concentration; BMI – body mass index; Max – maximal value; MDA – urine malonyldialdehyde concentration; Min – minimal value; SD – standard deviation; Se-B – blood selenium concentration; TAS – urine total antioxidative status; X – arithmetic mean;  $X_G$  – geometric mean.

This study aimed at determining interaction between blood selenium concentration and intensity of oxidative stress, expressed by urine malonyldialdehyde concentration and urine 8-hydroxy-2-deoxyguanosine concentration, and level of antioxidative capacity in a selected population of healthy children.

## 2. Materials and methods

### 2.1. Study population

The study was conducted in a group of consecutive, selected at random 337 children of less than 15 years of age, inhabiting Silesian voivodeship (Poland). The study was conducted in the years of 2007–2010. Among the participants boys comprised 56.7% (191 children), and girls 43.3% (146 children). The average age in the group amounted to 8.53 years, the average height to 1.36 m, body weight to 33.45 kg, and body mass index (BMI) to  $18.28 kg/m^2$ . General characteristics of the entire group are presented in Table 1.

At the principal stage of the study, accepting cut-off points of median value, 1st and 3rd quartiles of blood selenium concentration (Se-B), four groups were distinguished among the participants: those with Se-B < 1st quartile (group I, Se-B <  $70 \mu g/L$ ), those with Se-B between the first quartile and median value (group II, Se-B within the range of  $70$ – $76.99 \mu g/L$ ), those with Se-B between median value and 3rd quartile (group III, Se-B within the range of  $77$ – $83.99 \mu g/L$ ) and those with Se-B above the 3rd quartile (group IV, Se-B  $\geq 84 \mu g/L$ ). General characteristics of the distinguished groups are presented in Table 2.

At the subsequent stage of the study the criteria of gender and median BMI permitted to distinguish groups of boys (group A) and girls (group B), children with BMI below median value (group C) and those with BMI above median value (group D).

### 2.2. Laboratory measurements

In the participants selenium concentration was estimated in blood (Se-B). Level of oxidative stress and level of antioxidant capacity were determined in urine.

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