

Selenium an Essential Micronutrient for Human Health

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Selenium is essential for human health [1-4]. It occurs naturally in metalloid form and is needed in traces for human health [5]. Selenium plays important role in body anti-oxidation system [6,7] in the process protect the body cells from free radicals. It is therefore protective against cancer, and cardiovascular diseases. It is also important in thyroid hormone metabolism, inhibits HIV virulence and reduces development of AIDS [6].

Selenium plays important biological role through incorporation into a group of proteins, seleno-proteins [8]. The many biological forms of selenium is in form of seleno-cystein, a cystein analogue that is synthesized from serine bound to tRNA. Seleno-cystein contains ionized selenium at physiological pHs [8]. All seleno-proteins has one or more seleno-cystein residues in their active sites. All seleno-proteins has enzymatic functions in which one or more seleno-cystein located at the catalytic sites, participates in redox reactions. The enzymes are involved in different metabolic reactions at cellular level. The functions vary from anti-oxidant defense, muscle development and function, thyroid hormone metabolism, and immune function [7]. The deficiencies therefore lead to several pathological conditions. Several studies has shown inverse relationship between selenium intake and incidence of different cancers.

Selenoproteins are also involved in thyroid hormone biosynthesis pathway [9]. The mechanism is through iodothyronine reductase (DIOs) enzymes. The DIOs 1 and DIOs3 are located on plasma membranes, while DIOs 2 is located on Endoplasmic Reticulum [8]. The DIOs is considered responsible for the activation and de-activation of proT4 converting it to T3 the active thyroid hormone, which regulates the growth in a gene mediated process.

Infection by HIV into the cells increases activation of mitochondria enzymes, this leads to damage and dysfunction of mitochondria [10]. The accumulation of mitochondria DNA (*mitDNA*) in the cytosol which follows the dysfunction, and over activation of mitochondria leads to increased production and accumulation of Reactive Oxygen Species (ROS) causing oxidative stress [10,11]. The accumulation of Reactive Oxygen Species (ROS) leads to activation of cytokine NF- $\kappa\beta$ which stimulates the expression of receptors of Tissue Necrosis Factor (TNF α) [8,12,13]. ROS further oxidizes catalytic sites of seleno-protein Txr causing it to detach from N-Terminal of the Mitogen Active Protein (MAP), Apoptosis Signaling-regulating Kinase1 (ASK1). The TNF α stimulates apoptosis of cells through activation of the Apoptosis Signaling-regulating Kinase ASK 1 [12,13]. The action of ASK 1 in turn is mediated through activation of the p38 Kinase pathway and cJun N-terminal Kinase (JNK) both leads to a gene mediated apoptosis of the CD4 T cells in HIV infected patients leading a compromised immunity.

The seleno-protein GSH-px is present in blood cells and blood platelets [6]. It has been noted that the activity of GSH-px enzymes decrease rapidly at early stage of selenium deficiency [12]. GSH-px is encoded by the genes GPX1 to GPX6. HIV encodes a seleno-protein with homology to GSH-px hence depriving the host of selenium and other components needed for endogenous synthesis of the selenoprotein GSH-px. Other studies shows that homozygote polymorphism in GSH encoding genes leads to a decreased GSH-px activity and leads to increased accumulation of ROS in cells [10].

Presence of GSH-px leads to scavenging of ROS, which reduces oxidative stress in the cytosol. This causes a reduction of Trx active terminal, which leads to its attachment to the N-Terminal of ASK 1. This process inhibits the gene-mediated apoptosis in a kind of negative feedback mechanism [12,13].

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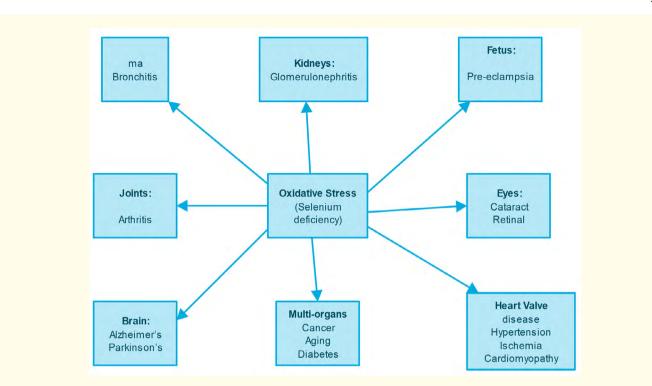


Figure 1: Effects of Oxidative Stress due to selenium deficiency (Lien., et al. 2008).

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