

Selenium, An Antioxidant Critical in HIV/AIDS Pathogenesis

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When cells use oxygen to generate energy, free radicals are created as a by-product of adenosine triphosphate production by mitochondria [1]. These by products are either Reactive Oxygen Species (ROS) or reactive nitrogen species (RNS) [1]. ROS and RNS are produced either by enzymatic or non-enzymatic process. The enzymatic process includes respiratory chain, phagocytosis, prostaglandin synthesis and cytochrome 450system. Non-enzymatic include ionizing radiation, and oxidative phosphorylation in mitochondria [2]. At high concentration, they generate oxidative stress which can damage all cell structures [1]. The human body has several mechanisms to counteract oxidative stress by producing antioxidants, which are either endogenous or exogenously supplied in foods. The anti-oxidants act as free radical scavengers by preventing and repairing damages caused by ROS and RNS hence enhance immune response [2].

Selenium, an anti-oxidant plays essential role in both innate and humoral immune responses [3,4]. The deficiency occurs due to low intake in food [5] and cultural practices which encourage intake of foods deficient in selenium, in adequate eating frequency. Further the deficiency also occurs due to diseases which lead to disturbed body metabolism.

The deficiency of selenium leads to oxidative stress, a condition in which cellular damage is caused by oxygen and oxygen derived oxidants [3]. Oxidative stress can be increased under certain conditions such as exposure to viral infections [6], this will lead to cells being damaged. Superoxide (O_2^-) and hydrogen peroxide (H_2O_2) are most common reactive oxygen species (ROS) and are assumed to contribute to the pathogenesis of several human diseases [1]. Anti-oxidants including selenium inhibit or delay oxidation of DNA, lipid membranes and proteins [7].

Cell entry of viruses disturbs the normal biochemistry of endoplasmic reticulum and mitochondria, causing ROS generation and depletion of anti-oxidants [8]. Early in the path of infection phagocytes release increased amounts ROS leading to increased lipid peroxidation of cell membranes and oxidative damage. Lipid peroxidation contributes to disease progression by further reduction of anti-oxidant activity, thereby supporting further viral replication [9]. In case of CD4 T cells this leads to rapid gene mediated programmed destruction (apoptosis) and several metabolic disturbances including deficiency of Tri-iodothyronine, malfunctioning intestinal epithelial cells and impairment of macrophage activity, increased DNA damage which leads to viral RNA mutation [6,4]. This further leads to immunity impairment in HIV infected patients and rapid deterioration to AIDS, a kind of positive feedback mechanism [4]. The deficiency of selenium also leads to disturbed thyroid gland metabolism leading to deficiency of Thyroxin (T3) [10]. T3 is needed to stimulate protein deposition through gene mediated process. Its' deficiency leads to loss of weight [11], thyroid tissue damage by ROS, which further complicates the disease outcomes.

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