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Opinion

Selenium and Autoimmune Thyroiditis

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The essential mineral, selenium, is of fundamental importance to human health. As a constituent of selenoproteins, selenium has structural and enzymic roles, in the latter context being best-known as an antioxidant and catalyst for the production of active thyroid hormone. Selenium is needed for the proper functioning of the immune system. An elevated selenium intake may be associated with reduced cancer risk [1,2].

Selenium is an trace element that is required for the correct functioning of the immune system with a recommended daily intake for adults of 55 µg [3]. Selenium-rich sources include nuts, meat, cereals, fish and shellfish [4]. Selenomethionine and sodium selenite are the two most common oral forms of selenium supplementation that are available in variable dosages (100 and 200 µg/day) [5]. Selenium plays a key role in thyroid cell physiology, it is incorporated in the molecular structure of several enzymes in the thyroid gland [6,7]. One of these enzymes, glutathione peroxidase, is critically involved in protecting the gland against oxidative damage. Thyroid peroxidase uses hydrogen peroxide (H_2O_2), a free radical capable of inflicting oxidative damage, as a substrate in catalyzing the iodination and coupling of tyrosyl residues in thyroglobulin to produce thyroid hormone. The tri-iodothyronine (T3), is produced by de-iodination of the hormone T4 by type I and type II iodothyronine de-iodinases in, a two-substrate, along with degradation of H_2O_2 to water by glutathione peroxidase. Iodothyronine de-iodinases are also selenoproteins, as is glutathione peroxidase. If there is selenium deficiency, these two enzymes cannot function properly, which results in both ineffective production of T3 and inefficient protection against free radicals, the latter facilitating cell damage and autoimmune destruction of the gland [6-8].

Autoimmune thyroiditis is one of the most prevalent autoimmune diseases and affects more than 10% of females and 2% of males. Cellular destruction by CD4 cell-mediated autoimmune attacks results in permanent hypothyroidism in more patients [9]. More than one-third of the patients have other autoimmune diseases. In areas with severe selenium deficiency there is a higher incidence of thyroiditis due to a decreased activity of selenium-dependent glutathione peroxidase activity within thyroid cells. Selenium is strongly involved, via the variable selenoproteins, in antioxidant, redox, and anti-inflammatory processes. Selenium enhances CD4+/CD25 FOXP3 and T regulatory cells activity while suppressing cytokine secretion, thus preventing apoptosis of the follicular cells and providing protection from thyroiditis. Environmental factors, such as iodine intake, immunotherapeutic agents or viral infections, can also trigger the disease. Selenium substitution may improve the inflammatory activity in patients with autoimmune thyroiditis, especially in those with high activity. Whether this effect is specific for autoimmune thyroiditis or may also be effective in other autoimmune diseases [9].

Low selenium status is associated with an increased risk of overall mortality, a reduced immune response and a decline of cognitive functions [10]. On the other hand, dietary selenium supplementation has shown to exert antiviral effects [11], improves male and female reproduction [12] and lower the risk of autoimmune diseases [13].

Furthermore, several clinical studies have highlighted a significant association between higher selenium status and reduced risk of prostate, lung, colorectal and bladder cancer. However, other clinical observations failed to demonstrate such correlation. In this scenario,

recent clinical observations have provided evidence that excess selenium consumption may be associated with increased risk for type 2 diabetes [14].

The Author's opinion is that, the plasma concentrations of selenium should be determined before to sort out diets *integrated* with *selenium.* This, in order to identify those subjects with an effective selenium deficiency and who may benefit from selenium dietary supplement in terms of health gain. Unfortunately, the determination of selenium levels in the plasma or urine is not a clinical test that is routinely performed. On the other hand, the knowledge of selenium concentrations in biological fluids of patients with autoimmune thyroiditis may be useful as diagnostic tool in order to detect possible deficiencies of this trace element that can eventually be *corrected* to reduce the rate of gland inflammation. Furthermore, the intake of alimentary selenium or diets *integrated* with *selenium plays a pivotal role in patients with a simultaneous presence thyroiditis and autoimmune gastrointestinal diseases* as these pathological conditions can lead to a reduction in the absorption of trace elements and vitamins with antioxidant activity.

In thyroiditis, as in other autoimmune diseases, nutrition is very important for controlling the evolution of the disease, it becomes important in these diseases to have any deficiencies of trace elements and vitamins under control, especially in autoimmune thyroiditis, it would be useful to study the link between any deficiencies of selenium, zinc and vitamin D.

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