Szafraniec-Porada Aneta, Porada Dominik, Konopelko Michał, Przybylska Daria, Pucułek Małgorzata. The influence of selenium on thyroid gland. Journal of Education, Health and Sport. 2018;8(9):1501-1508. eISNN 2391-8306. DOI http://dx.doi.org/10.5281/zenodo.1435937
http://ojs.ukw.edu.pl/index.php/johs/article/view/6111

The journal has had 7 points in Ministry of Science and Higher Education parametric evaluation. Part B item 1223 (26/01/2017).
1223 Journal of Education, Health and Sport eISSN 2391-8306 7

The authors declare that there is no conflict of interests regarding the publication of this paper.

Received: 22.08.2018. Revised: 28.08.2018. Accepted: 15.09.2018.

The influence of selenium on thyroid gland

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Abstract

The proper functioning of the thyroid gland depends on many factors, which include adequate supply of various trace elements, including iodine, but also selenium or iron. The purpose of this article was to attempt to summarize the current knowledge on the effects of selenium on the thyroid gland and the clinical implications associated with it.

Selenium affects the homeostasis of the entire human body, and its main function in the thyroid gland is to counteract damage of oxidative origin. This microelement is part of selenoproteins, which include glutathione peroxidase and thioredoxin reductase. Both selenium deficiency and excess can negatively affect the thyroid gland. The research results show that selenium supplementation may bring benefits in some morbidities, eg Graves-Basedow disease. Currently, I advise recommending semi-annual selenium supplementation in a mild form of thyroid orbitopathy. However, no clinical benefit has been demonstrated in increasing the supply of selenium in the diet of other patients with thyroid disease, including Hashimoto's disease. In conclusion, despite the numerous studies already conducted, further work is necessary to fully explain the mechanisms of action of this element on the thyroid

gland, but also to deepen knowledge about the benefits and risks of selenium supplementation in various types of thyroid structural disorders and, most importantly, to establish safe guidelines for possible treatment.

Key words: selenium, thyroid gland, selenoblasts, thyroid hormones

Introduction

Selenium (Se) is a trace element necessary to maintain homeostasis of the body. It is a component of many proteins, the so-called selenoproteins, including: glutathione peroxidase, iodothyronine 5 thiodiodiodate, trioredoxin reductase, selenoprotein P or selenioprotein W [1-3]. Demand for selenium differs depending on age and sex, and increases during pregnancy and lactation. The currently recommended daily dose for healthy adults should not exceed 70 μg. Increased Se consumption is allowed, however higher doses may be toxic. The range of normal selenium concentration in the blood serum ranges between 75-140 μg / l. [2-6]. In many areas of the world there is hyposelenic soil, including in Poland, which results in insufficient amount of this element in food products, both animal and vegetable [7].

Purpose of work: This article is an attempt to summarize current knowledge about the influence of selenium on the thyroid gland. The analysis of scientific literature concerned both the deficiency and excess selenium in the body and possible suplementation opportunities.

The role of selenoblasts in the thyroid gland

The highest concentration of selenium is observed in the thyroid gland, even when it is deficient in the body, which confirms its important role in the proper functioning of this gland [8-10]. Among the most important selenoid proteins found in the thyroid gland are: glutathione peroxidase, thioredoxin reductase and iodothyronine deiodinase. Thyroid peroxidase uses hydrogen peroxide to oxidize iodide ions during the production of thyroid hormones, decomposing it into water and oxygen, thus protecting thyrocytes against free radicals. Reactive oxygen species in the thyroid are also neutralized by thioredoxin reductase, which is an electron donor for other oxidoreductive enzymes. On the other hand,

iodothyronine deiodinase plays a role in the thyroid hormone metabolism by transforming thyroxine (T4) to triiodothyronine (T3) and reverse triiodothyronine (rT3) [2.3, 11-16].

Selenium deficiency

Selenium deficiency is widespread and affects different populations in many countries of the world [17]. The effects of deficiency of this element affect the entire human body, and result with disorders associated with proteins containing selenium [18]. Selenium deficiency may result in an increase in incidences of cancer (including colon, thyroid, prostate, lung and breast) or cardiovascular morbidities (heart failure, arrhythmia, dilated cardiomyopathy) [19, 20]. It can also lead to the progression of many inflammatory diseases (including Kashin-Beck's disease, systemic lupus erythematosus) and metabolic (diabetes), as well as worsening existing degenerative diseases of the central nervous system [21-25]. Additionally, in the case of selenium deficiency, the risk of strokes, male infertility, nephropathy increases and the symptoms of autoimmune diseases may increase [26-28]. It was found that low selenium concentration correlates with an increased risk of thyroid disease [29]. Inadequate supply of selenium may cause disorders of thyroid hormone synthesis, both as a consequence of abnormalities associated with the functioning of selenoproteins, as well as by disturbances of the metabolism of iodine in the body [27].

Excess of selenium

Increased selenium intake may also have negative health effects. Toxic action of high doses of selenium consists of inactivation of proteins responsible for the repair of damaged DNA and may lead to increased production of free radicals [30]. Mild symptoms of selenium intoxication are often non-specific, and the most common include malaise, gastrointestinal complaints (such as nausea, vomiting or diarrhea), as well as neurological disorders [31, 32]. In the case of chronic consumption of high doses of selenium, both through diet and dietary supplements / drugs may lead to selenosis, which results in liver damage and damage to the hematopoietic system, as well as skin lesions and serious dysfunctions of the nervous system [31, 33]. Excess selenium may also result in disruption of thyroid hormone synthesis and increase the risk of developing type 2 diabetes [31, 34].

Effect of selenium on thyroid disease

Disorders of the structure and / or function of the thyroid gland are one of the most common endocrine diseases. Adequate supply of selenium affects the proper functioning of the thyroid by optimizing the synthesis and metabolism of thyroid hormones, but also reducing the production of pro-inflammatory factors, including proinflammatory cytokines, leukotrienes or prostaglandins. As previously stated, it protects the thyroid against oxidative stress. In addition, due to its regulatory effect on the immune system, both T and B lymphocytes cause a decrease in thyroid antibodies [35]. It was found that selenium deficiency promotes thyroid volume increase (in women) [36], as well as the development of focal lesions in the thyroid gland [37]. However, the importance of the supply of selenium in the context of thyroid cancer is mixed [38, 39]. In the literature on the subject, attention is drawn to the significant effect of selenium on thyroid diseases of autoimmune etiology. It has been demonstrated that the supply of selenium suppresses the immune cellular response, which is important for the occurrence and severity of chronic lymphocytic thyroiditis [40]. In patients with Hashimoto's disease, there is a significant reduction in selenium concentration compared to a healthy population [41]. In numerous studies on the effect of selenium supplementation in people with autoimmune thyroiditis, it has been demonstrated that the use of selenium preparations results in the lowering of anti-TPO antibody concentration and often anti-TG antibodies and improvement of thyroid gland echogenicity. The best effects were observed with a dose of 200 μg / day [42-46]. However, a meta-analysis carried out by van Zuuren et al. showed that despite current research, there is currently no clear scientific evidence for the effectiveness of selenium supplementation in Hashimoto's disease, therefore there is no indication for the general use of selenium preparations in this group of patients, and any possible treatment with selenium preparations should be approached with caution [47].

In patients with Graves' disease, lower blood selenium levels were also observed in comparison with the control group. In addition, in a state of remission the level of this microelement is significantly higher than in patients with overt hyperactivity [48, 49]. The use of selenium supplements in this group of patients tended to normalize the concentration of free thyroid hormones and TSH and lower the concentration of antibodies against the receptors for TSH, and results in a higher remission rate [50]. The best documented and currently recommended medical indication for selenium supplementation is mild thyroid orbitopathy. It was found that the use of increased doses of selenium causes regression of ailments and prevents the progression of eye changes in Graves' disease, which was reflected

in the recommendations of the European Thyroid Society, which recommends 6-month selenium therapy (200 μg in a divided dose) [51, 52]. Selenium therapy cannot replace thyrostatic treatment, but selenium supplementation during treatment may shorten the time needed for clinical and biochemical euthyroidism [53-55].

Summary

Selenium is a trace element, for which both excess and deficiency result in disorders of the body's homeostasis, in particular thyroid function. Selenium supplementation in areas with deficiency of this element brings benefits to patients with mild orbitopathy in the course of Graves-Basedow disease. Currently, there is no reliable scientific data on the legitimacy of selenium supplementation in other thyroid diseases. The use of this element cannot replace the previously used pharmacological treatment, but the possibility of adding selenium preparations is still an interesting therapeutic option in thyroid diseases. One should remember about the negative impact of large quantities of selenium on the human body. Further research is necessary to obtain reliable data that will allow a full explanation of the mechanisms of action of this element on the thyroid gland in pathological conditions, but also to establish safe and effective guidelines for possible treatment.

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