The MOSS NUTRITION REPORT

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Product Review

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SELENIUM 200 – MORE THAN JUST A SUPPLEMENT TO NEGATE THE IMPACT OF METHYLMERCURY EXPOSURE

INTRODUCTION

Since we began manufacturing the Moss Nutrition product line in 2009, it has been my goal to focus on supplemental nutrients that, despite large volumes of published literature that demonstrated their clinical efficacy for a broad range of patient scenarios, have, traditionally, fallen through the cracks, so to speak. In 2009, at least for me, the most glaring example of this disparity between large bodies of supportive research and utilization in the typical clinical/nutritional practice was potassium. Therefore, our very first product was **K Alkaline**, a potassium bicarbonate product. As I am assuming many of you know who are long time customers, I have continued this focus on supplemental potassium over the years, hopefully to the benefit of your patients and your practices.

Of course, these days, with the knowledge/literature explosion on clinical nutrition and functional medicine over the years, you might feel there is not a single supplemental nutrient that has not been written about and discussed ad nauseum in functional medicine symposia and periodicals. I certainly was of that opinion, at least until recently. If you have been following my latest installments of the **Moss Nutrition Report** over the last few months, you have seen my claim that there is another supplemental nutrient that, while it has been deemed an essential nutrient for human health, has, based on its lack of mention in functional medicine symposia and publications over the last few, years, "fallen through the cracks."

This supplemental nutrient is selenium. In particular, as I have tried to demonstrate in my latest **Moss Nutrition Report** newsletters by virtue of quotes from several publications in highly regarded medical journals, selenium has been grossly underappreciated as a potent negating factor for mercury toxicity. This is especially true for ocean fish that have been getting a "bum rap" for years due to their mercury content. Why do I say, "bum rap?" Due to the significant content of selenium in these same mercury-bearing ocean fish, the highly prevalent and highly publicized dogma that ocean fish intake needs to be either reduced for the general population or avoided completely for pregnant women has been almost conclusively disproven by several "fell through the cracks" published studies that continuously affirm the health and neurologic benefits of ocean fish ingestion despite the mercury content. Why does this seemingly logical conclusion that, due to their mercury content, ocean fish need to be avoided, not even begin to reflect research and clinical reality? As I have pointed out in the latest **Moss Nutrition Report** newsletters, the high content of selenium in most ocean fish more than negates

any potential adverse effect of the mercury also present in these fish.

In addition, in these newsletters I have also featured published research that indicates that selenium supplementation is an excellent adjunct to the selenium in ocean fish in terms of greatly reducing or eliminating any adverse effect of ocean fish-based mercury.

Moss Nutrition has been providing to you for years the product **Selenium 200**, which is a selenomethionine form of supplemental selenium containing 200 mcg of selenium per capsule. Interestingly, it has never been a very popular product in terms of sales, probably, to a great extent, due to the "fallen through the cracks" phenomenon I have described above.

In this product newsletter, I would like to examine still another "fallen through the cracks" aspect of selenium supplementation – the impact of selenium supplementation on thyroid health. I will do this by reviewing the paper "Supplementation with selenium and coenzyme Q_{10} in an elderly Swedish population low in selenium – positive effects on thyroid hormones, cardiovascular mortality, and quality of life" by Alehagen et al (Alehagen U et al. *BMC Medicine*, Vol. 22, No. 191, 2024).

A DISCUSSION OF THE STUDY IN DETAIL

The first quotes I would like to feature from this paper discuss the intimate participation of selenium in thyroid health:

"In the human body, the thyroid gland has the highest content of selenium in proportion to weight. Correspondingly, selenium is an essential trace element for the function of the thyroidal gland, especially for the metabolism of thyroid hormones.

The effects of selenium on the thyroid gland and thyroid hormones are mediated via glutathione peroxidases (GPXs) and thioredoxin reductases (TXNRDs) that protect against oxidative cellular injury, iodothyronine deoidinases I, II and III (DIOI, DIOII, and DIOIII) that activate and inactivate T4 and T3, and selenoprotein P that provides selenium to the thyroid gland and other extrahepatic tissues."

With the above in mind, the authors conclude the following:

"A sufficient intake of selenium is mandatory in order to avoid disturbance of the thyroid hormone balance, as an insufficient intake could affect both the central and the peripheral capacity to convert T4 to T3, causing an increased level of T4, and a reduced level of T3."

Next, Alehagen et al discuss the impact of selenium on autoimmune thyroid disease:

"The mechanism behind the positive effects of selenium on autoimmune thyroid disease progression is probably due to the significant effect of selenium adequacy on inflammatory mechanisms."

Of course, as noted in the title of the paper, the paper is not just about selenium but the relationship between selenium and coenzyme Q_{10}, which is also important for thyroid health:

"Xia et al. reported an important interrelationship between selenium and coenzyme Q_{10} (ubiquinone) as TXNRD is required to obtain ubiquinol, the active form of the coenzyme."

Therefore, if selenium is deficient and the enzyme TXNRD is compromised, there will be less than optimal amounts of the active form of coenzyme Q_{10} in thyroid cells.

With this relationship in mind, Alehagen et al conducted the following study:

"This sub-study was conducted on a population recruited from a municipality in the south-east of Sweden were all inhabitant aged 70-88 were invited to participate in an epidemiological project in 1996. Out of the 1130 individuals in the appropriate age stratum, 875 agreed to participate in the epidemiological project. In 2003, a new invitation was sent out to the participants and of the 675 still living in the municipality, 443 agreed to participate in a dietary supplementation project that required taking selenium and coenzyme Q_{10} combined for

4 years. The inclusion started in January 2003 and finished in February 2010."

Of course, to truly appreciate the value of selenium supplementation, it is important to know the baseline status of selenium in the study population:

"Before starting the intervention, the selenium concentration in the population was found to be 67 µg/L (standard deviation (SD 16.8)), which approximated to a daily intake of about 35 µg/day, which is far below the level considered necessary for optimal physiological function (≥110 µg/L) and adequate production of selenoproteins."

Next, consider the supplemental protocol:

"The participants received 200 mg/day of coenzyme Q_{10} capsules…and 200 µg/day of organic selenium yeast tablets…or placebo (500 mg vegetable oil supplied with vitamin E and bakers' yeast, respectively) over 48 months."

How many participants were in the treatment and placebo groups?

"In this sub-analysis, 210 individuals were randomised to active intervention, while 204 individuals were randomised to placebo."

Study results

First consider the specific impact of selenium on thyroid hormones. As you will see, it is interesting to note that the impact was not the same on all thyroid hormones:

"In the present intervention study on elderly Swedes low in selenium, we found that thyroid hormone plasma concentrations, i.e., TSH and free T3, but free T4, reverse T3 or thyroglobulin were related to selenium plasma concentrations at baseline. Low selenium concentrations were associated with high TSH and low free T3 levels."

Concerning the impact of supplementation on specific thyroid hormones, Alehagen et al noted the following:

"The intervention lasting for 48 months resulted in an increase in free T3 and reverse T3 and a decrease in free T4, with no significant changes in the placebo group."

Of course, these findings would make sense since T4 is converted to T3. The authors go on to note that the impact on selenium on deiodinase activity is not limited to the thyroid:

"The changes in thyroid hormones accord well with the assumption that low selenium status is associated with a decrease in deiodinase activity, both in the thyroid gland and in the liver, and other peripheral tissues."

What was the impact of coenzyme Q10? The authors continue:

"In the present study, individuals with low selenium status presented with a significantly higher concentration of TSH and higher CV mortality, compared to those with higher selenium concentrations. It is tempting to suggest that the effect on CV mortality might, in part, have been mediated through an impact on thyroid hormones as well as a beneficial effect of selenium and coenzyme Q_{10} on inflammation and oxidative stress."

Next, Alehagen et al make a point relevant to what we have been suggesting as functional medicine practitioners for years – that nutrient deficiency is not an all or none proposition. Rather, there is a grey area between overt deficiency and optimal nutrient levels. In the case of selenium, the authors suggest that even a minor to moderate selenium deficiency in so-called "healthy populations" can have an adverse effect on thyroid function yet not be given the classic descriptor of "subclinical hypothyroidism":

"An important finding of this study is that less severe selenium deficiency may also impair the activity of selenium-dependent deiodinases, which is crucial in the metabolism and homeostasis of thyroid hormones. Hence, it might be suggested that a significant proportion of 'healthy' community-living elderly participants may suffer from impaired thyroid function beyond 'subclinical hypothyroidism'. Supplementation with selenium appears not only to restore deiodinase activity and thyroid hormone balance but also has clinical implications in terms of its association with reduced CV morbidity and inflammation."

Did the study population that demonstrated suboptimal thyroid function due to selenium deficiency also demonstrate any quality of life issues? As seen in the following quote, cognitive function issues seemed to be the most prevalent:

"We have also assessed different dimensions of health-related quality of life as obtained by the quality of life questionnaire SF-36, one of the most widely used generic health-related quality of life questionnaires in the world. From the evaluations, we were able to demonstrate symptoms, mainly concerning the mental dimension, in those with impaired thyroid function."

With the above findings in mind, Alehagen et al concluded the following:

"In this sub-analysis, 'healthy' elderly community-living persons low in selenium were evaluated regarding their thyroidal function in relation to selenium intake and CV morbidity. Elevated TSH and low free T3 levels were observed in those with the lowest selenium intake, and selenium/coenzyme Q_{10} supplementation resulted in significantly increased free T3 and decreased free T4 levels. Lower TSH levels were associated with reduced CV mortality and improved measures of health-related quality of life. The observed changes in thyroid hormones could be explained by an increase in selenium dependent diodinases. We conclude that a substantial part of the study population might suffer from suboptimal thyroid function."

SOME FINAL THOUGHTS

For most of us, we have been aware since our days in basic nutrition classes that selenium plays an important role in thyroid function by virtue of its importance in conversion of T4 to T3 via the deiodinase enzymes. What has been less certain is the importance of this fact in everyday clinical practice when making decisions on how to assist patients with suboptimal thyroid function. More precisely, how highly should we prioritize selenium supplementation when considering the myriad of potential supplemental options that can potentially assist in the optimization of thyroid function?

As suggested by Alehagen et al, the importance of selenium supplementation for those patients whose findings suggest suboptimal conversion of T4 to T3 is probably greater than what we have traditionally been thought to believe. Why? Suboptimal selenium status is probably more common than what we have long suspected. Furthermore, as noted in my **Moss Nutrition Report** series on the relationship between fish, mercury, and selenium, those individuals who demonstrate evidence of higher-than-average mercury exposures, the need for selenium supplementation to support optimal thyroid function will be even more profound.

Lastly, as also demonstrated by Alehagen et al, coenzyme Q10 supplementation can be an important and underappreciated adjunct to selenium supplementation.

The options for selenium and coenzyme Q10 supplementation from Moss Nutrition are the following:

- **Selenium 200** – 200 mcg selenomethionine per capsule
- **Coenzyme Q10 Select** – 100 mg of coenzyme Q10 as ubiquinone per capsule
- **CoQH Select** – 100 mg of coenzyme Q10 as ubiquinol per softgel.