Thyroid Hypofunction I - Part Seven

What about the risk of Hashimoto's with increased iodine intake? It's true that studies have shown that increased iodine intake, especially in supplement form, can increase the autoimmune attack on the thyroid. Iodine reduces the activity of thyroid peroxidase, and TPO is required for proper thyroid hormone production.

On the other hand, restricting intake of iodine can reverse hypothyroidism. In one study, 78 percent of patients with Hashimoto's regained normal thyroid function with iodine restriction alone. In Germany, a low dose of potassium iodine, 250 mcg, was given to 40 people who tested positive for TPO antibodies and/or had a thyroid ultrasound showing a hypoechogenic pattern that is consistent with Hashimoto's. Nine patients from the iodine group developed thyroid abnormalities compared with only one person from the control group. Of the nine patients in the iodine arm, seven developed subclinical hypothyroidism, one became hypothyroid frankly, and another became hyperthyroid. Changes were also seen in levels of TPO antibodies as well as on ultrasound of the thyroid. Three of the seven subclinical hypothyroid patients and the hyperthyroid patient regained normal thyroid function after the withdrawal of iodine.

Selenium may protect against the harmful effects of iodine.

However, and this is a big however, it appears that iodine may only pose a problem for people with Hashimoto's and other autoimmune thyroid diseases in the presence of concurrent selenium deficiency. In studies where rats developed goiter while receiving excess iodine, when they were given adequate selenium, they did not develop the goiter. Other studies have shown that selenium protects against the effects of iodine toxicity and prevents the triggering and flaring of autoimmune disease that excess iodine without selenium can cause.

My recommendation is to test iodine levels using urine and hair analysis. If normal, and the patient is not in any of the groups that are at high risk for iodine deficiency, do not supplement with iodine. If iodine is low and/or if they are in any of the high-risk groups for deficiency, and there is no evidence of Hashimoto's, supplementing with doses up to 1,100 mcg per day appears to be safe, but I would still monitor thyroid antibodies and other thyroid markers closely. If iodine is low and/or they are in a high-risk group for deficiency, and the patient does have Hashimoto's or you suspect they do, you can try supplementing, but I would start at a very low dose such as maybe 100 mcg or just increase intake of foods with iodine but still at a low dose. I have a chart that shows the iodine content of food, and you can just make sure that they are not exceeding 100 mcg per day and monitor closely.

The next nutrient to consider is selenium. Adequate selenium nutrition supports efficient thyroid hormone synthesis and metabolism and protects the thyroid gland from damage from excessive iodine exposure. Glutathione peroxidase is selenium dependent, so if selenium intake is inadequate, accumulated peroxidases damage the thyroid, provoking autoimmunity. Additionally, selenium is also essential for the conversion of T4 to T3, as the deiodinase enzymes, the enzymes that remove iodine atoms from T4 during conversion, are selenium dependent. Several research studies have demonstrated the benefits of selenium supplementation in treating autoimmune thyroid conditions. One study found that selenium supplementation had a significant impact on inflammatory activity in thyroid-specific autoimmune disease, and reducing inflammation may limit damage to thyroid tissue. This may be due to the increase in glutathione peroxidase and thioredoxin reductase activity as well as the decrease in toxic concentrations of hydrogen peroxide and lipid hydroperoxides, which result from thyroid hormone synthesis.

Another study followed patients for nine months and found that selenium supplementation reduced thyroid peroxidase antibody levels in the blood, even in selenium-sufficient patients. All of this suggests that selenium supplementation in patients with hypothyroidism may be a very good idea, and I used to do it myself. However, selenium is another nutrient with a U-shaped curve, and long-term consumption of high doses can lead to complications such as gastrointestinal upset, hair loss, white blotchy nails, garlic breath odor, fatigue, irritability, and mild nerve damage. In addition, some studies have shown that supplementing with selenium in the context of low iodine status may actually aggravate hypothyroidism. Finally, a large clinical trial with 35,000 men found that selenium supplementation when selenium levels were adequate at baseline significantly increased the risk of prostate cancer, so this is yet another example of no-one-size-fits-all approach.

At this time, I think the best strategy is to simply ensure adequate selenium intake through the diet and possibly to use testing for selenium levels at baseline to determine whether selenium supplementation is warranted.

Iron: not enough and too much are both a problem.

Iron is the next nutrient to consider. Iron deficiency reduces heme-dependent thyroid peroxidase activity in the thyroid, resulting in impaired production of thyroid hormone. Iron-deficiency anemia during pregnancy can result in both higher TSH and lower T4 concentrations, so you definitely want to treat iron deficiency. However, as you know, you need to be careful with iron overload. Excess iron damages the hypothalamus, pituitary, and thyroid, among other organs. One study showed that people with hemochromatosis are 80 times more likely to have hypothyroidism. Yes,

80 times. Some thyroid bloggers online have suggested that women with hypothyroidism push their ferritin up to 75 to 100, but given that the Iron Disorders Institute recommends an optimal range of 25 to 75 for ferritin for women, I'm concerned about that recommendation. There is no evidence that higher iron levels improve thyroid function, and on the contrary, I just mentioned that people with very high iron levels are 80 times more likely to have hypothyroidism, so just as we should avoid iron deficiency for optimal thyroid function, we should be careful to avoid iron overload as well.

Nutrient	Function
Magnesium, B12 and zinc	Required for synthesis of TSH
Riboflavin and vitamin C	Required by iodine symporter
Vitamins A & D	Required to activate the nuclear thyroid receptor

Other nutrients required for proper thyroid function are zinc, B12, B2, vitamin C, vitamin A, vitamin D, and magnesium. Magnesium, B12, and zinc are required for the synthesis of TSH. Riboflavin and vitamin C are required by the iodine symporter to bring iodine into the thyroid gland. Vitamins A and D are required to activate the nuclear thyroid receptor on cell surfaces. There is not a lot of research on deficiencies of these nutrients and thyroid dysfunction, but understanding of thyroid physiology suggests that they should be present in adequate amounts to ensure optimal thyroid function. As you know from the exposome unit and other blood chemistry sections, many people are deficient in these nutrients.

Okay, that's it for now. We'll be back with Part Two next week.