CLINICAL THYROIDOLOGY **FOR PATIENTS**

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THYROID AND THE ENVIRONMENT

High environmental exposure to perchlorate and thiocyanate, in combination with low urinary iodine, is associated with decreased thyroid hormone levels

BACKGROUND

Iodine, an essential component of thyroid hormone, is transported into thyroid cells through a specialized channel called the sodium-iodine symporter (NIS). When iodine levels in the body are low (i.e. from reduced dietary intake), transport of iodine across NIS may fall. Similarly, compounds such as perchlorate (an industrial chemical) and thiocyanate (a byproduct of cyanide found in food and tobacco smoke) inhibit NIS potentially reducing iodine transport and blocking the production of thyroid hormone. Human exposure to these compounds in the environment has been well documented and can be detected by high levels of perchlorate or thiocyanate in the urine. Previous research suggests that either of these compounds alone may reduce thyroid hormone levels although the effect is quite small. In this study, the authors examined whether exposure to all three of these factors – low iodine, increased perchlorate and increased thiocyanate – will have a substantially larger effect in reducing thyroid hormone levels.

THE FULL ARTICLE TITLE

Steinmaus C et al. Combined effects of perchlorate, thiocyanate, and iodine on thyroid function in the National Health and Nutrition Examination Survey 2007-08. Environ Res 2013;123:17-24. Epub March 7, 2013.

SUMMARY OF THE STUDY

The study used data from the National Health and Nutrition Examination Survey (NHANES 2007-2008). The NHANES 2007-2008 is large population-based survey designed to collect information on the health and nutrition of adults and children in the United States. Participants were categorized into three groups: Group A (the reference group) had the lowest levels of perchlorate and thiocyanate in the urine and high levels of urine iodine (>100mcg/L). Group B had the middle levels of perchlorate and thiocyanate in the urine and high levels of urine iodine (>100 mcg/L). Group C had the highest levels of perchlorate and thiocyanate in the urine and low levels of urinary iodine (<100mcg/l). There were 390 participants in group A, 533 in group B and 64 in group C. Serum total thyroxine (T_4) levels were 2.5% lower in individuals with high urine thiocyanate concentration and 5% lower in those with high urine perchlorate concentration. However, when the three groups (Group A-C) were analyzed T_4 was 5.1% lower in group B and 12.9% lower in group C as compared to the reference group A. In other words those with low iodine intake who also had the highest exposure to both perchlorate and thyocyanate had markedly reduced T_4 production, more so than exposure to any one single factor.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The findings from this study provide evidence that exposure to perchorate and thiocyanate act in combination to decrease thyroid hormone production in those patients with low iodine in their diet. These results will be important in developing public health policy since thyroid hormone is critical to neurodevelopment during infancy and childhood.

— Philips Segal, MD

ATA THYROID BROCHURE LINKS

Iodine Deficiency: http://www.thyroid.org/iodine-deficiency

ABBREVIATIONS & DEFINITIONS

NIS: Sodium-iodine symporter – a specialized channel that transports iodine into thyroid cells.

Thyroxine (T_4): the major hormone produced by the thyroid gland. T_4 gets converted to the active hormone T_3 in various tissues in the body.

Iodine: an element found naturally in various foods that is important for making thyroid hormones and for normal thyroid function. Common foods high in iodine include iodized salt, dairy products, seafood and some breads.

Perchlorate: an industrial chemical that is a component of rocket fuel. Perchlorate blocks the Sodium-iodine symporter and decreases iodine entry into thyroid cells.

Thiocyanate: a byproduct of cyanide found in food and tobacco smoke. Thiocyanate blocks the Sodium-iodine symporter and decreases iodine entry into thyroid cells