

Correspondence

Selenium Deficiency and Hypothyroidism

I should like to comment further on the relationship between selenium deficiency and hypothyroidism, a topic recently raised by Dr. Hayashida (1994). T_4 deficiency can occur in several ways. It is often associated with dietary iodine inadequacy, commonly exacerbated by goitrogens in water or food supplies (Matovinovic, 1983). Goitre also may occur in individuals consuming excess iodine. To illustrate, in China, it is associated with drinking water containing >300 micrograms/litre of iodine or the consumption of large quantities of seaweed (Tan et al, 1990). Depressed serum T_4 levels, however, are not necessarily accompanied by below normal serum T_3 . When severe iodine inadequacy is present, serum T_3 tends to remain stable, or may even rise as T_4 levels drop (Pharoah et al, 1976). This relationship occurs because T_3 contains less iodine, weight for weight, than does T_4 . It is, however, more metabolically active and hence is produced by the thyroid when iodine is scarce (Hatzel, 1989). Only in extreme iodine deficiency, when there is inadequate iodine even to produce T_3 , does its level decline. However, the T_4 to T_3 conversion requires the catalytic selenoenzyme iodothyronine deiodinase. As a consequence, T_4 and T_3 deficiencies together are commonest in individuals living in environments depleted in both iodine and selenium. In contrast, depressed T_3 , without unusually low serum T_4 , is a characteristic of the populations of regions where diets contain adequate iodine, but lack selenium. Furthermore, animal studies suggest that just as excess iodine consumption results in lowered serum T_4 levels, an elevated intake of selenium may depress serum T_3 (Behne et al, 1992).

These diverse relationships have major implications for the treatment of hypothyroidism. It has been demonstrated, for example, that in areas of endemic goitre, selenium supplementation by itself induces a dramatic fall in the already impaired thyroid function in clinically hypothyroid subjects (Contempre et al, 1991). This is prob-

ably because selenium supplementation accelerates the conversion of T_4 to T_3 , which, in individuals who are already iodine deficient, quickly causes T_4 reserves to drop.

Such a decline in serum T_4 levels is likely to be most significant during pregnancy, since this hormone is of major importance to the developing fetal brain. Shortages are known to result in mental retardation and even neurological cretinism (Hetzel, 1989). Interestingly, depressed intelligence also is a feature of populations in regions of selenium deficiency (Svistonova, 1988), suggesting that T_3 inadequacy may result in abnormal brain development. Furthermore, it has been argued that T_4 and possibly T_3 deficiencies play significant roles in both respiratory distress (Schonberger et al. 1981; Lucas et al, 1988; Marsh et al, 1988) and in sudden infant death syndromes (Foster, 1992; 1993). Since there appears to be some maternal transfer of thyroid hormones to the developing fetus, the T_4 and T_3 status of the mother during pregnancy is also probably very significant (DeLong, 1993).

It follows from this discussion, therefore, that selenium supplements should not be given to pregnant women, or those that are likely to become so, unless iodine intake is known to be very adequate. For this reason, it seems preferable to treat pre-menopausal hypothyroid patients with desiccated thyroid tablets, unless of course their hypothyroidism is due to excess dietary iodine and/or selenium.

References

1. Behne D, Kyriakopoulos A, Gessner H, Walzog B and Meinhold H, 1992. Type 1 iodothyronine deiodinase activity after high selenium intake, and relations between selenium metabolism in rats. *J. Nutr.* 122:6. 1542-46.
2. Contempre B, Dumont JE, Ngo B, Thilly CH, Diplock AT, and Vanderpas J, 1991. Effects of selenium supplementation in hypothyroid subjects of an iodine and selenium deficient area: The possible danger of indiscriminate supplementation of iodine-deficient subjects with selenium. *J. Clin. Endocrinol. Metab.* 73(1): 213-215.

3. DeLong GR, 1993. Effect of nutrition on brain development in humans. *Am. J. Clin. Nutr. Suppl.* 57: 186S-290S.
4. Foster Hd, 1992. *Health, Disease and the Environment*. London: Belhaven Press, 24-69.
5. Foster HD, 1993. Sudden infant death syndrome: The Bradford Hill criteria and the evaluation of the thyroxine deficiency hypothesis. *J. of Orthomolecular Medicine* 8(4): 201-225.
6. Hayashida T, 1994. Selenium deficiency and hypothyroidism. *J. of Orthomolecular Medicine* 9(3): 186.
7. Hetzel BS, 1989. *The Story of Iodine Deficiency*. Oxford: Oxford University Press.
8. Lucas A, Rennie J, Baker BA, and Morley R, 1988. Low plasma triiodothyronine concentrations and outcome in preterm infants. *Arch. Dis. Child.* 63: 1201-1206.
9. Marsh TD, Freeman D, McKeown RE, Bowyer FP, 1993. Increased mortality in neonates with low thyroxine values. *J. Perinatol.* xiii(3): 201-204.
10. Matovinovic J, 1983. Endemic goitre and cretinism at the dawn of the third millennium. *Ann. Review Nutr.* 3: 341-412.
11. Pharaoh POD, Ellis SM, Ekins RP and Williams ES, 1976. Maternal thyroid function, iodine deficiency and fetal development. *Clin. Endocrinol.* 5: 159-166.
12. Schonberger W, Grimm W, Emmrich P and Gempp W, 1981. Reduction of mortality rate in premature infants by substitution of thyroid hormones. *Eur. J. Pediatr.* 135: 245-253.
13. Svistonova TP, 1988. Biogeochemical influences on the psychological development of school children (in Kaschin-Beck areas). *Abstracts, International Symposium on Environmental Life Elements and Health*. Beijing: Academy of Sciences, 283.
14. Tan J, Li R and Zhu W, 1990. Medical geography, in Geographical Society of China (ed.). *Recent Development of Geographical Science in China*. Beijing: Science Press, 259-279.

Harold D. Foster, Ph.D.
University of Victoria
P.O. Box 8050
Victoria, B.C. V8W 3P5

An Alternative Treatment for Tic Douloureux

Trigeminal neuralgia (tic douloureux) is a disease which causes severe lancinating pain lasting several seconds to several minutes, which may be repeated many times for many months. It is often set off by touching a trigger point, or by an activity such as chewing or brushing one's teeth. The usual treatment consists of drugs such as Tegretol, Baclofen, Phenytoin, and antidepressants, and has included surgery to sever the fifth nerve. But there is an alternative which has worked very well for four of my patients who followed it.

September 15, 1992, a woman born in 1915 told me that she had been awakened one night in 1978, screaming from pain on the right side of her face. She suffered over six episodes of the severe pain. She was diagnosed as having tic douloureux. Since then she had not been free of pain. In addition, over the previous year she had also developed severe pain in her jaw diagnosed as arthritis.

I advised her to take niacin 500 mg after each meal, ascorbic acid 1000 mg after each meal, B-complex 50s once a day, vitamin E 800 IU daily, vitamin B₁₂ sublingually 2 mg per day, and folic acid 5 mg twice a day.

One week later she was free of pain. September 20, 1994, she called me to discuss something not related to this problem. I asked her about the pain. She replied it was a miracle, and she had not suffered any further pain..

Tic douloureux should be added to the list of diseases which respond to Orthomolecular therapy. The three main elements should be vitamin B₁₂, ascorbic acid and 1-lysine up to 3 g per day. I did not give this patient 1-lysine and she responded well, but with other patients this amino acid has been very helpful.

A Hoffer, M.D., Ph.D.
3A - 2727 Quadra Street
Victoria, B.C. V8T 4E5