

The Nutritional Relationships of the Thyroid

David L. Watts, D.C., Ph.D., F.A.C.E.P.¹

Barnes, et al, estimate that 40% of the American population suffer from thyroid dysfunction and that hypothyroidism is the most common complaint seen by doctors in this country.¹ Several factors contribute to clinical thyroid dysfunction such as diseases affecting the gland itself or a disturbance in the hypothalamic — pituitary — thyroid axis. Subclinical thyroid dysfunction particularly, hypothyroidism, is more common than clinical pathology of the thyroid gland. Subclinical hypothyroidism can produce or contribute to an array of metabolic dysfunctions and symptoms that may respond readily to a conservative nutritional approach.

Conditions Associated with Hypothyroidism

Severe clinical thyroid insufficiency is associated with many conditions including myxedema, cretinism in children, goiter, arteriosclerosis, and hyperlipidemia.² Subclinical hypothyroidism is more common and is not always easily detectable through normal testing procedures. Subclinical hypothyroidism could be described as a syndrome rather than a disease and is characterized by fatigue, depression, cold sensitivity, and changes in the skin and hair texture. The development of fatigue is usually insidious, resulting in the requirement of extra sleep. Depression may develop often and become more prolonged with each episode. Cold sensitivity first develops in the extremities. Cold hands and feet may become present even in the summer. The skin becomes dryer, and the hair may become coarse or thin, with accompanying hair loss.

Nutritional Deficiency and Hypothyroidism

A number of nutritional deficiencies are known to develop in subclinical hypothyroidism. The most recognized is iron deficiency. ¹ Trace Elements, Inc., P.O. Box 514, Addison, Texas 75001.

The resulting anemia can be in the form of normochromic, normocytic, hypochromic, microcytic, macrocytic, or megaloblastic.³ The incidence of anemia is estimated to affect up to 60% of those patients with hypothyroidism and is not related to severity or duration of thyroid insufficiency. Other related deficiencies are protein deficiency, perhaps due to accompanying hypochlorhydria; deficiency in vitamins A, C, B₆, B₅, B₁; and mineral deficiency: phosphorus (P), manganese (Mn), magnesium (Mg), potassium (K), sodium (Na), and chromium (Cr). Keyvani, et al, found that low vitamin A levels are associated with an increase in the prevalence of goiter in subjects under 18. After the age of 18, females were more affected by goiter than males, who were found to have higher vitamin A levels.⁴ An increase in tissue estrogen sensitivity in the presence of vitamin A deficiency may explain the increased prevalence in women.⁵ Zinc deficiency has also been found in children with grade I goiters in conjunction with a deficiency of vitamin A.⁶

Thyroid Inhibitors

First we should review some of the naturally occurring thyroid inhibitors. These factors are not usually considered in the course of thyroid therapy; this may explain the often slow response of patients given synthetic thyroid preparations. Frequently patients prescribed synthetic thyroid or thyroxine do not respond to therapy for six months. Consideration of these known thyroid inhibitors may produce quicker response to medications, but they may also respond to a more conservative nutritional approach.

Figure 1 shows the many nutritional factors that are known to antagonize thyroid activity or expression.

B₁₇ — Laetrile — Amygdalin — Aromatic Isothiocyanates

It has long been known that amygdalan

affects thyroid function. Evidence that thiocyanates exhibited an anti-thyroid effect was noted with its use as an anti-hypertensive.⁷ Thiocyanates are still being used as an anti-hypertensive. Sodium nitroprusside (trade Name Niprid (R) Roche) was characterized as being "the drug of choice" in the management of hypertensive crises by the AMA Department of Drugs in 1977. It is interesting to note that cruciferae plants that are known to inhibit thyroid activity are also high in amygdalin. This plant group includes cabbage, broccoli, brussels sprouts, cauliflower,⁸ as well as almonds, cassava, yam, maize, apricots, prunes, and bamboo shoots.⁹

Vitamin B₁₂, Cobalt (Co)

Cobalt and vitamin B₁₂ are closely related; therefore, either can have adverse effects upon the thyroid and contribute to hypothyroidism.¹⁰⁻¹¹ Cobalt's effect on the thyroid was seen in patients being treated with cobalt for anemia.¹²

Vitamin D, Calcium (Ca)

The inhibitory action of calcium on the thyroid has been suspected since the last century, but more recent studies have confirmed its effects.¹³⁻¹⁴ It is known that calcium decreases thyroid activity and that calcium absorption is increased in thyroid insufficiency.¹⁵ Vitamin D would also be considered to contribute to lowered thyroid function due to its close, synergistic relationship to calcium. Studies have shown that serum Vitamin D₃ metabolites are increased in hypothyroid patients¹⁶ and reduced in hyperthyroid patients.¹⁷

Para-aminobenzoic Acid (PABA)

PABA has been used in the treatment of thyrotoxicosis. PABA compounds used in the treatment of tuberculosis have been known to produce goiters.¹⁸

Lithium (Li)

Hypothyroidism is a well-known side effect associated with lithium therapy. Lithium apparently affects calcium as well. Studies have shown that serum calcium increases and serum phosphorus becomes depressed in patients treated with lithium,¹⁹ which is probably due to lithium induced hyperparathyroidism reported by

Shen, et al.²⁰

Molybdenum (Mo), Bromine (Br)

Experiments have shown that molybdenum suppresses thyroid activity in animals. Bromine, another anion, inhibits the active transport of iodide.²¹

Iodine

Iodine is required for normal thyroid function, but with excessive intake it becomes a thyroid suppressant. The occurrence of endemic goiter in China was found to be due to the high concentration of iodine in drinking water.²² Iodine has long been used therapeutically in the treatment of thyrotoxicosis.²³

Copper (Cu)

Copper can be considered a thyroid antagonistic agent due to its close relationship with estrogen (to be discussed later) as well as its antagonistic relationship to iron and zinc. Cecil²⁴ has described the relationship of iron deficiency and hypothyroidism. More recent studies have shown that iron deficiency can impair thyroid function and that iron status can reflect thyroid activity.²⁵ It has also been found that during iron deficiency, the conversion of L-phenylalanine to L-tyrosine is reduced by as much as 50%.²⁶

Thyroid Endocrine Inhibitors

Overactivity of other endocrine glands can contribute to hypothyroidism. An increase in circulating hormones, even while in normal serological limits, can suppress or decrease the response of thyroid therapy. The endocrine glands have opposing activity similar to vitamins and minerals. Overactivity can suppress an opposing gland, and underactivity can allow increased expression.²⁷

Estrogen

Estrogen is closely associated with the mineral copper. In the serum, they tend to fluctuate simultaneously. It is highly probable that estrogen as well as copper inhibits thyroid activity. Estrogen is known to decrease thyroid stimulating hormone (TSH) production by the pituitary.²⁸ During pregnancy, when estrogen and copper levels normally rise, enlargement of the

Figure 1.

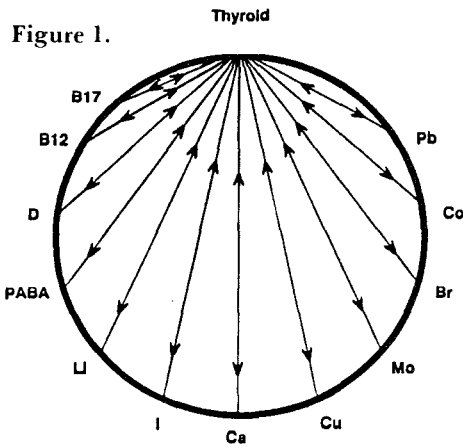
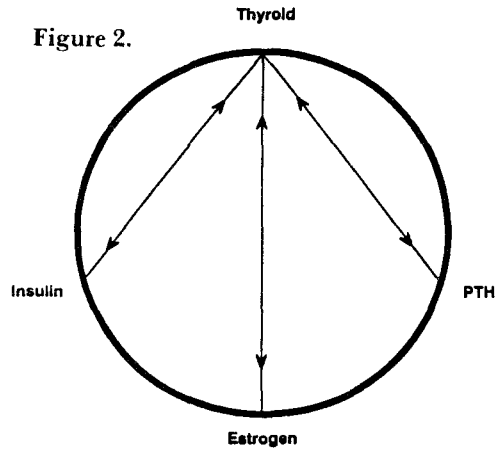


Figure 2.



thyroid is a common occurrence.²⁹ It is also well known that pregnancy relieves thyrotoxicosis.³⁰

Insulin

Insulin is known to be competitive with thyroxine production.³¹ Plasma insulin levels, which elevate during the course of pregnancy concomitantly with estrogen,³² also rise during estrogen therapy.³³ Insulin is known to enhance the synthesis of vitamin D, and conversely, vitamin D enhances the synthesis of insulin.³⁴ The prevalence of thyroid failure in insulin-treated diabetics is becoming more recognized. A study of this relationship has been reported by Gray, et al.³⁵ Hypothyroidism is the most common endocrine disorder seen in children.³⁶ It is possible that excessive, prolonged milk intake could be responsible since milk is a significant source of insulin.³⁷

Calcium is known to mediate the release of insulin.³⁸⁻³⁹ The synergism between calcium and vitamin D can contribute to decreased thyroid activity, either through insulin stimulation or due to their antagonism of other nutrients that are required for normal thyroid function.

Parathyroid (PTH)

Hormonal PTH activity increases the absorption and retention of calcium and thus increases insulin production. PTH activity is also enhanced by vitamin D, and vice versa. The thyroid and parathyroid

glands appear to oppose each other's action. Patients with underactive thyroids frequently are found to have subclinically elevated PTH hormone levels⁴⁰⁻⁴¹ in the presence of normal serum calcium levels. PTH hormone secretion is increased by estrogen.⁴²

Insulin, estrogen, and PTH can be considered to be synergistic to each other. They may singularly or collectively produce a competitive or antagonistic effect upon thyroid expression.

We should also be aware that increased thyroid activity can decrease the expression of these opposing glands.

Determining Thyroid Function

The most common tests used in assessing thyroid function are radioimmune assays of thyroxine (T_4), Triiodothyronine (T_3), and thyroid stimulating hormone (TSH). Often the clinical manifestations conflict with these laboratory tests, making a clear-cut diagnosis difficult.⁴³ A patient may present many signs of hypo- or hyperthyroidism without a corresponding laboratory confirmation.

Tissue Mineral Analysis (TMA) Patterns and Thyroid Dysfunction

The endocrine glands (hormones) influence trace element metabolism, and the trace elements are known to influence endocrine function.⁴⁴ Therefore, TMA patterns may prove useful in evaluating endocrine activity.

The TMA pattern of hypothyroidism is associated with a disturbance in the following mineral ratios: calcium/phosphorus (Ca/P), calcium/potassium (Ca/K), calcium/magnesium (Ca/Mg), and sodium/magnesium (Na/Mg). The Ca/P ratio is elevated above normal (Trace Elements, Inc. (TEI) ideal = 2.63). This is to be expected. As discussed previously, calcium absorption is increased while renal phosphorus reabsorption is decreased in hypothyroidism. Increased PTH activity also contributes to the TMA pattern. PTH increases calcium and magnesium absorption while decreasing the renal reabsorption of phosphorus, as well as sodium and potassium. The Ca/Mg ratio may become elevated (TEI ideal = 7.1) depending upon the extent of PTH involvement.

Reduced adrenal activity is indicated by an elevated magnesium level relative to sodium, i.e., decreased Na/Mg (TEI ideal = 4).^{45 46 47} Copper is frequently elevated above normal (T.E.I. ideal 2.5 mg%) levels, also a consequence of adrenal insufficiency.

An increase in insulin secretion may be reflected by an increase in the Ca/Mg ratio, which also corresponds to increased PTH activity.

The above ratios are reversed in hyperthyroid TMA profiles. An increase in thyroid activity promotes calcium and magnesium excretion and increases phosphorus retention. This is a result of an apparent thyroid-parathyroid antagonism,^{48 49 50} which produces a reduction in the TMA Ca/P ratio.

An increase in adrenal activity is suggested by an elevated Na/Mg and reduced Ca/K ratio. Epinephrine increases tissue potassium retention,^{51 52} as does increased thyroid function, and is mediated by Na-K ATPase.⁵³

Conclusion

It should be noted that the TMA observations described are not from controlled studies. Continuing research and utilization of TMA testing as a routine part of examinations, along with other clinical data will improve its usefulness as a screening tool in evaluating thyroid function and will provide a guided nutritional approach to therapy. Further controlled studies in this area would be most wel-

comed.

Footnote

The TMA results (mineral levels and ratios) discussed in this article are based upon atomic absorption spectrophotometric analysis and laboratory procedures utilized by Trace Elements, Inc., and may not apply as well to different laboratory techniques and preparatory procedures.

References

1. Barnes BO, Galton L: *Hypothyroidism: The Unsuspected Illness*. Harper and Row Pub., N.Y., 1976.
2. Guyton AC: *Medical Physiology*. W.B. Saunders, Phil., 1971.
3. Fein HG, Rivlin RS: Anemia in thyroid disease. *The Medical Clinics of North America* Vol. 59. W.B. Saunders Co., Phil., 1975.
4. Keyvani F, Yassai M, Kimiagar M: International symposium on clinical nutrition and post graduate course. Apr., 1987. *Am. J. Cl. Nutr.*, 46,3, 1987.
5. White A, Handler P, Smith EL: *Principles of Biochemistry*. McGraw-Hill, 1964.
6. Yasaai M, et al: Correlations of serum zinc concentrations with goiter, physical development and serum vitamin A levels. International symposium on clinical nutrition and post graduate course. Apr., 1987, San Diego. *Am. J. Cl. Nutr.*, 46,3, 1987.
7. Barker MH: The blood cyanates in the treatment of hypertension. *J.A.M.A.* 106, 1936.
8. National Research Council. *Diet, Nutrition and Cancer*. Wash. D.C. Nat. Academy Press, 1982.
9. Eyjolfsson R: Recent advances in the chemistry of cyanogenic glycosides. *Zechmeister. Progress in the Chemistry of Organic Natural Products*, Vol. 28. Springer Pub. N.Y., 1970.
10. Sederholm T, et al: Cobalt-induced hypothyroidism and polycythemia in lipid nephrosis. *Acta Med.*, Scand., 184, 1944.
11. Washburn TC, Kaplan E: Cobalt therapy and goiter. *Clin. Ped.*, 3, 1964.
12. Kriss P, Carnes WH, Gross RT: Hypothyroidism and thyroid hyperplasia in patients treated with cobalt. *J.A.M.A.* 157, 1955.
13. Greer MA: Nutrition and Goiter. *Physiol. Rev.* 30, 1950.
14. Greer MA, Kendall JW, Smith M: Antithyroid compounds. *The Thyroid Gland*, Vol. I. Pitt, Rivers, Trotter, Eds. Butterworths, Lond. 1964.
15. Lowe CE, Bird ED, Thomas WC: Hypercalcemia in myxedema. *J. Clin. Endocrinol. Metab.*, 22, 1962.

16. Mosekilde L, Lund B, Sorensen OH, Christensen MS, Melsen F: Serum 25-hydroxycholecalciferol in hypothyroidism. *Lancet*, I, 1977.
17. Valentzas C, Oreopoulos DG, From G, Porret B, Rapoport A: Vitamin D levels in thyrotoxicosis. *Lancet*, 1977.
18. Danowski TS: *Clinical Endocrinology* Vol II. Williams and Wilkins, Baltimore, 1962.
19. Cox M, Singer I: Lithium. *Disorders of Mineral Metabolism*, Vol. I. Bronner, F, Cobkurn, J.W., Eds. Academic Press, N.Y., 1981.
20. Shem F-H, Sherrard DJ: Lithium-induced hyperparathyroidism: An alteration of "set-point". *Ann. of Intern. Med.*, 96, 1982.
21. Langer P, Greer MA: *Antithyroid Substances and Naturally Occurring Goitrogens*. S. Keager, Basel, Switz., 1977.
22. Mu L: Endemic goiter in central China caused by excessive iodine intake. *Lancet* II, 1987.
23. Wolf J, Chaikoff IL: Plasma inorganic iodide as a homeostatic regulator of thyroid function. *J. Biol. Chem.*, 174, 1948.
24. Cecil RL: *Textbook of Medicine*. Saunders Co., Phil. 1938.
25. Dillman E, et al: Hypothermia in iron deficiency due to altered triiodothyronine metabolism. *Am. J. Physiol.*, 239, 1980.
26. Lehmann WO, Henrich HC: Impaired phenylalanine-tyrosine conversion in patients with iron-deficiency anemia studied by a L-(2H5)phenylalanine loading test. *Am. J. Clin. Nutr.*, 44, 1986.
27. Watts DL: Nutritional interrelationships, minerals, vitamins, endocrines. (Unpub.) Trace Elements, Inc., Dallas, 1988.
28. Brown JHU: *Integration and Coordination of Metabolic Processes. A Systems Approach to Endocrinology*. Van Nostrand Reinhold Co., N.Y., 1978.
29. Innerfield R, Hollander CS: Thyroidal complications of pregnancy. *The Medical Clinics of North America*, Vol. 61. W. B. Saunders Co., Phil., 1977.
30. *Ibid.*
31. Selenkow HA, Birnbaum MO, Hollander CS: Thyroid function and dysfunction during pregnancy. *Clin. Ob. Gyn.* 16, 66, 1973.
32. Spellacy WH, Goetz FC: Plasma insulin in normal and late pregnancy. *N.E.J.M.*, 268, 1963.
33. Gershberg H, et al: Glucose tolerance in women receiving an ovulatory suppressant. *Diabet.*, 13, 1964.
34. Cross HS, Peterlik M: Hormonal and ionic control of phosphate in differentiating enterocyte. *Progress in Clinical and Biological Research*, Vol. 168. *Epithelial Calcium and Phosphate Transport, Molecular and Cellular Aspect*. Bonner F, Peterlik M, Eds. Alan R. Liss, Inc., N.Y., 1984.
35. Gray RS, Smith AF, Clark BF: Hypercholesterolemia in diabetes with clinically unrecognized primary thyroid failure. *Horm. Metab. Res.*, 13, 1981.
36. Hughes JF: *Pediatrics*. Mosby Co., St. Louis, 1971.
37. Sheard NF, Walker WA: The role of breast milk in the development of the gastrointestinal tract. *Nutr. Rev.*, 46, 1, 1988.
38. Leclercq-Meyer V, et al: Effect of calcium and magnesium on glucagon secretion. *Endocrinol.*, 93, 1977.
39. Malaisse WJ, et al: The stimulus-secretion coupling of glucose-induced insulin release. *J. Lab. Clin. Med.*
40. Adams P, et al: Parathyroid function in spontaneous primary hypothyroidism. *J. Endocrinol.*, 40, 1968.
41. Bouillon R, DeMoor P: Para-thyroid function in patients with hyper- or hypothyroidism. *J. Clin. Endocrinol.* 38, 1974.
42. Greenburg C, et al: Parathyroid hormone secretion effect of estradiol and progesterone. *Metabol.*, 36, 2, 1987.
43. Larsen PR: Thyroid-pituitary interaction. *N.E.J.M.*, 306, 1, 1982.
44. Henkin RI: Trace metals in endocrinology. *The Medical Clinics of North America*, Vol. 60. W.B. Saunders Co., Phil. 1976.
45. Douglas WW, et al: Effects of alkaline earths and other divalent cations on adrenal medullary secretion. *J. Physiol.*, 175, 1964.
46. Harrop GA, et al: Studies on the suprarenal cortex. *J. Exp. Med.*, 58, 1933.
47. Wacker WE, et al: Magnesium Metabolism. *N.E.J.M.*, 259, 1958.
48. Harden R, et al: Phosphate excretion and parathyroid function in thyrotoxicosis. *J. Endocrinol.*, 28, 1964.
49. Bortz W, et al: Differentiation between thyroid and parathyroid causes of hypercalcemia. *Ann. Int. Med.*, 54, 1961.
50. Malamos B, et al: The renal handling of phosphate in thyroid disease. *J. Endocrinol.* 45, 1969.
51. Rosa RM, et al: Adrenic modulation of extrarenal potassium disposal. *N.E.J.M.*, 302, 1980.
52. Silva P, et al: Sympathetic systems in potassium homeostasis. *Am. J. Physiol.*, 241, 1981.
53. Clausen T, et al: The effect of catecholamines on Na-K transport and membrane potential in the rat soleus muscle. *J. Physiol.*, 270, 1977.

Reprinted from **The Journal of Orthomolecular Medicine**
Third Quarter 1989 – Volume 4 Number 3
Publication Office: 16 Florence Ave., Toronto, ON, Canada M2N 1E9
Reproduction without permission is prohibited