Thyroid: Misconceptions

by Ray Peat, Ph.D.
Ray Peat's Newsletter

When I describe some of the simple ways the thyroid gland works, I know that I'm not answering the questions most people have. In the last 50 years, a culture, a mythology, related to thyroid hormone has been manufactured in this country. Now and then, people send me *JAMA's* latest contribution to that culture. The articles and advertisements in medical journals, and the more direct contacts between drug companies and physicians, have covered most aspects of thyroid physiology with a thick layer of misinformation. A very different story is found in journals of endocrinology, biochemistry, nutrition and metabolism, and in foreign books and journals.

Most of the misinformation is based on something true. If a similar preoccupation with possible harmful side-effects surrounded the rest of medicine as it does thyroid supplementation, medicine would disappear; if it surrounded dietetics, people would starve.

It is sometimes true that "if you take thyroid it will suppress your own gland." By overdosing people until the output of their own glands had gone to zero, it was possible to see what happened when the overdose stopped. Everyone's gland was functioning perfectly again within two or three days. Occasionally, a person whose pituitary isn't producing TSH will respond to thyroid supplementation with normalization of TSH and restoration of their own thyroid gland's function.

It is sometimes (but rarely) true that a person will seem healthy when his serum thyroxine level is near one of the extremes of the "normal" range (which is often given as between 4 or 4.5 micrograms per 100 ml. and 12 or 14 mcg. per 100 ml). The story behind that extreme range (imagine a similar "normal" variation for cholesterol, or sodium, or potassium, or anything biochemically important) has to do with a revision in the 1940s of the definition of "normal" to include 95% of the population, based on the PBI measurement, which was later found to be irrelevant to thyroid functioning.

Thyroxine is often called "the thyroid hormone," in the same way that estrogen has been called "the female hormone." Progesterone is quantitatively and functionally the most important female hormone, and triiodothyronine, or liothyronine, is functionally the most important thyroid hormone. It is the metabolically active derivative of thyroxine, somewhat as insulin is the activated form of proinsulin. When thyroxine is added to brain tissue in vitro, it suppresses respiration; but when triiodothyronine is added to any tissue, respiration is increased. Triiodothyronine (T3) is mostly produced in the liver, depending on the availability of glucose to the liver, allowing a sensitive adjustment of metabolism to nutrition.

Armour thyroid, USP, is often said to be of imprecise dosage, but in fact every batch is biologically standardized, and studies have shown it to be reliably within 1% of the labeled potency. The best known brand of the supposedly chemically precise levothyroxine, however, was for a long time 30% below the labeled potency.

If the liver is the main source of the thyroid problem, then thyroxine pills can make the problem worse, by suppressing the portion of T3 still coming from the thyroid gland. Armour thyroid, USP, Thyrolar, Euthroid, Proloid, and a few other products contain both thyroxine and T3, in approximately the proportion secreted by a normally functioning gland.

Unsaturated oils interfere with thyroid function in several ways, including blocking the "digestive" proteolytic enzymes involved in the release of hormone from the globulin. Unsaturated fatty acids interfere with binding of the hormone to a transport protein, and with the conversion of T4 to T3 in the liver and in the pituitary. Linoleic and linolenic acids are roughly twice as toxic to these systems as oleic acid is. The tissue response to the hormone is inhibited by unsaturated fats in proportion to the number of double bonds in the fat. A deficiency of polyunsaturated fatty acids is anti-estrogenic, partly by allowing the thyroid-stimulated liver to excrete estrogen, and partly by failing to activate the pituitary secretion of gonadotrophins.

G.W. Crile found that the basal metabolism of the people in Yucatan, where coconuts are a staple food, is 125% of that of people in the United States. Animal experiments show that coconut oil added to a normal diet can lower serum cholesterol levels. There is a very reliable inverse relationship between the level of serum cholesterol and thyroid hormone action. A major effect of thyroid is to control the conversion of cholesterol into steroid hormones and bile acids.

Thyroid promotes the formation of progesterone, which in turn promotes the secretion of thyroid hormones. Estrogen blocks their release from the thyroid gland, causing the gland to enlarge.

Iodine deficiency used to cause goiters in the United States, but now it is almost impossible to have an iodine deficiency in this country. Iodine supplements can suppress the formation of thyroid hormone, producing classical signs of hypothyroidism.

Hashimoto's thyroiditis is often diagnosed, without appropriate evidence. Even when correctly diagnosed, the knowledge isn't relevant to treatment. Thyroid cells contain estrogen "receptors," and estrogen inhibits thyroid secretion, and excess estrogen is involved in most "autoimmune" disease. Supplemental thyroid so easily normalizes estrogen and the immune system that it, with progesterone and pregnenolone and DHEA, should be considered as the cure for autoimmune diseases, including thyroiditis – at least until someone finds an "autoimmune" disease that doesn't respond to a balancing of these hormones.

Breast discomfort and even the secretion of milk not associated with pregnancy or parturition, produced by excess prolactin, can often be relieved by thyroid, especially when taken with vitamins A and E. The incidence of breast cancer is high in hypothyroid regions. Excess prolactin and excess estrogen are associated with each other, with breast cancer, and with hypothyroidism.

Most physicians know how to test the Achilles tendon reflex, but believe it is a poor indicator of thyroid function. Often, this is because they have observed the reflex contraction itself, rather than the relaxation rate, which gives a clear indication of the rate at which energy is regenerated in the muscle. The "T wave" or repolarization wave in the electrocardiogram gives a similar indication of lagging energy production in hypothyroidism. Magnesium retention by cells is dependent on thyroid hormone, and is essential for cell relaxation.

The rate of energy restoration is relevant to the tired brain, too. Insomnia is the most commonly troublesome symptom of menopause, and it usually responds immediately to a small bedtime dose of T3, just as T3 rapidly restores the normal quick relaxation rate to the calf muscles and heart muscle. (Two or three grams of sodium chloride taken at bed-time in a little broth, or juice or water, has a strong sleep inducing effect; it lowers adrenaline, promotes glucose absorption by the intestine, and has other antistress actions, including "sparing" magnesium.)

Thyroid permits energy to be produced efficiently from glucose, and hypothyroidism causes glucose waste leading to hypoglycemia, which in turn leads to excessive

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secretion of adrenaline. Adrenaline, like the synthetic drugs that mimic its actions, has been known to be toxic for many years. It mobilizes fatty acids from storage, and its most obvious toxicity is to the circulatory system.

The circulatory complications of diabetes were avoided in Broda Barnes' diabetic patients, who received thyroid supplements. In diabetes, glucose is unable to enter cells, and the body's response resembles that of hypoglycemia, secreting adrenaline and mobilizing fats from storage. Since a certain amount of intracellular glucose is needed before the liver can form T3, diabetics, like hypoglycemics, or like anyone during starvation, are unable to activate the thyroid hormone.

Adrenaline secretion leads to cortisol secretion. The "Cushingoid" fat distribution, sudden balding, and other signs of hormone imbalance secondary to hypothyroidism are statistically associated with heart disease. Broda Barnes clearly showed the protective action of thyroid against heart disease.

Too much thyroid does cause enlargement of the heart. Meerson, and others, have shown that growth of the heart muscle is a normal process of adapting to an increased workload. In a more extreme situation, too much thyroid can cause relative starvation, arresting growth, by causing the tissues to "work" too intensely. When mice are given food containing 1% thyroid by weight, they stop growing, but normal growth resumes if they are also given liver as a nutritional supplement. Rats that were given enough Armour thyroid to stop their growth (up to 0.7% of their food) had bones that were slightly heavier than the bones of the rats on the normal diet that had grown at the normal rate. That is, even when the diet isn't adequate to sustain normal growth, hyperthyroidism doesn't necessarily cause osteoporosis. Very large doses of thyroxine, though, are not the same as hyperthyroidism. An often cited report on osteoporosis that was observed in a few women who were taking thyroxine, actually provided no clear data to support its conclusions, but it did illustrate two important fallacies: Failing adequately to take into account that women who take thyroid medication have a history of hypothyroidism, and ignoring that thyroxine reliably suppresses the pituitary TSH and thus decreases the T3 secreted by the thyroid gland, but doesn't reliably increase the liver's formation of T3. Hypothyroidism, whether natural or promoted by administered thyroxine, retards bone remodeling and tissue repair in general. As hypothyroidism predisposes to excess cortisol effects, it must also be a common factor in "Cushingoid" osteoporosis.

When the thyroid is normal, vitamin A and cholesterol are used rapidly in forming progesterone. Carotene tends to accumulate in hypothyroidism, and it can bind to the cells of the corpus luteum, preventing the use of vitamin A to form progesterone. Adrenalin, cortisol, prolactin, and ACTH are likely to be elevated in hypothyroidism, and they have been proposed as inhibitors of ovulation or progesterone secretion. Estrogen is usually elevated in hypothyroidism, and can prevent implantation of the embryo, or cause miscarriage if implantation occurs. Adequate thyroid normalizes these anti-fertility factors, which is why it was considered the basic fertility hormone. It is also essential for the vitality of sperm cells.

Animals which are artificially made hypothyroid develop cystic ovaries, so it is reasonable to consider hypothyroidism as an important factor when women have cystic ovaries. Thyroid hormone's effects on all systems of the body – nervous, immune, digestive, excretory, respiratory, etc. – contribute to optimal fertility, and to the vitality of the offspring.

Exercise lowers the level of thyroid hormones, partly by accelerating their breakdown. The stress of winter appears to do the same thing, and most people (and animals) need much more thyroid in the winter than they do in the summer. Exercise lowers human (and some animals') fertility, and winter lowers animals' fertility. I think human fertility, as indicated by sperm count, for example, is likely to be lower in winter.

Because some estrogen is secreted in the bile, adequate fiber in the diet (oats, potato, or raw carrots, for example) and regular bowel function help to prevent the build-up of estrogen, which inhibits the thyroid. (Estrogen which has been excreted in the bile can be reabsorbed from the intestine if there is slow transit time and too little fiber.) A deficiency of B vitamins or protein is also known to prevent the liver from excreting estrogen. One of the ways in which starvation inhibits thyroid function is by damaging the liver function. Vegetarians are sometimes dangerously deficient in protein, and in that state the body is very resistant to thyroid hormone. Elevated serum calcium is probably one of the factors in creating a state of thyroid-resistance during stress.

Thyroxine can protect against lipid peroxidation, yet the hormone is essential for the normal oxidation of fat. One aspect of its "antioxidant" function is that it allows respiration in the mitochondria to consume the reduced (electron donor) substances, which otherwise can accumulate and be oxidized by stored iron, to start the free-radical attack on lipids.

There are many directly anti-thyroid substances, but the only directly thyroid-activating substances I know of are coconut oil, progesterone, and pregnenolone. The saturated fatty acids, especially the highly soluble smaller molecules found in coconut oil, probably tend to simply dilute and weaken the inhibition that is chronically exerted by the polyunsaturated fatty acids, but butyric acid seems to have some specific effects, such as facilitating the uptake of T3 by nerve cells and shifting cells away from the expression of the stress-related proteins. Progesterone promotes the release of hormones from the thyroid gland, and by promoting the formation of glycogen, it probably supports the formation of T3 by the liver. Pregnenolone, which is formed from cholesterol in the mitochondria, also normalizes thyroid function, probably partly by optimizing the balance of steroid hormones, and partly by stabilizing the enzyme systems of the mitochondria.

Correspondence and Subscriptions:
Ray Peat, PhD
Ray Peat's Newsletter
1585 Moss
Eugene, OR 97403

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