Nutrition News and Views
July/August 1999 Vol.3, No.4 For health professionals only

Underactive and Overactive Thyroid

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After diabetes, thyroid disease is the most common glandular disorder affecting about 20 million Americans, usually as **hypo**thyroidism (underactive thyroid) or **hyper**thyroidism (overactive thyroid). However, because symptoms are often mistaken for excess stress, depression, signs of aging, or are simply ignored, many cases of thyroid imbalance – up to 8 million — are not identified. By age 60, 17 to 20% of women and 9 to 10% of men have signs of low thyroid function.

Nestled just below the Adam's apple in the base of the neck, the thyroid gland is a butterfly-shaped (two-lobed) structure that weighs less than an ounce and produces less than a teaspoonful of hormone each year. Yet these hormones have a huge impact as the body's "accelerator," controlling the tempo or pace of all internal processes. They regulate how much energy the body uses, body weight, how the body uses nutrients, the rate of operation of virtually every organ and system in the body, pacing the heart, lungs, digestive tract, brain, and metabolic action of each cell. In addition to facilitating normal cell reproduction and growth, the thyroid regulates the rate of oxidation (use of oxygen) in all tissues, repair of damaged or diseased tissues, glucose liberation from the liver to the bloodstream, sense activity, water balance, function of circulating systems, muscles, nerves, sex organs, fat metabolism; and more. Since the thyroid controls the metabolic rate of every cell, it affects pathological conditions as they develop.

Thyroid disorders are more common in women, but men "are not immune." Often the tendency for thyroid imbalance runs in families. A malfunctioning thyroid can adversely affect – damage – other organs. Though the thyroid can be the site of benign or malignant growths, thyroid cancer "is relatively rare."

The three "active" hormones produced by the thyroid are: **triiodothyrine (T_3)**, **thyroxine (T_4)**, and **calcitonin** (used in calcium metabolism). Normal secretion of thyroid hormone depends upon the "intact" feedback loop involving the hypothalamus, pituitary, and thyroid glands. The adrenal glands and other glands and organs (including the liver) can also be involved. Thyrotropin-releasing hormone (**TRH**), produced by the hypothalamus, stimulates the anterior pituitary gland to secrete thyrotropin – thyroid stimulating hormone (**TSH**) – which, in turn, stimulates the thyroid gland to release thyroxine (T_4) and triiodothyronine (T_3). Release of TRH and TSH is suppressed (balanced) by the action of T_4 and T_3 to complete the feedback loop.

Laboratory tests include measuring levels of T_3, T_4, and TSH. TSH is usually the first, and sometimes the only, indication of thyroid imbalance as it is sensitive to mild imbalances. If the TSH level is high (above normal range), this indicates **hypo**thyroidism. The pituitary gland is "nudging" or instructing the thyroid to secrete more hormone when it is "not quite up to the task." The T_3 and T_4 may be normal if the thyroid is attempting to keep up with the demand; this is subclinical hypothyroidism. If the TSH is low (decreased), **hyper**thyroidism is indicated. The pituitary gland is "telling" the thyroid to slow down, not to produce so much hormone. Again, the thyroid is under stress, but if it is keeping the T_3 and T_4 levels normal, subclinical hyperthyroidism exists. T_3 and T_4 tests are not as sensitive as TSH, so even with some hormonal imbalance, may not appear outside normal limits until severe (clinical) disruptions exist. For example, a 15 to 20% decline in the secretion of thyroid hormones is not enough to register below normal in T_3 and T_4 blood tests. But it can cause a 50 to 100% increase in the TSH level – more than enough to raise it above normal range.[1]

SIGNS AND SYMPTOMS

Hypothyroidism (failure to produce sufficient hormones) is the most common thyroid dysfunction. The majority of cases are diagnosed as Hashimoto's thyroiditis. Symptoms may include any of the following, though certainly all will not occur simultaneously and some will not occur at all:

Fatigue, low body temperature, dry skin/hair. inappropriate weight gain, brittle nails, insomnia

and/or narcolepsy, poor short-term memory and concentration, headaches, migraines, menstrual or menopausal problems or irregularities, depression, hair loss (including outer third of eyebrows), low motivation and ambition, cold hands and feet, fluid retention, dizziness or lightheadedness, irritability, food intolerances, hoarseness, eye conditions such as myxedema (bulging, drooping eyes), skin problems/acne, infertility, miscarriage, dry eyes, blurred vision, puffiness around eyes, heat and/or cold intolerance, low blood pressure, elevated cholesterol, digestive problems (irritable bowel syndrome, acid indigestion, constipation, etc.), poor coordination, diminished sex drive, reduced or excessive sweating, frequent colds/sore throats, slow healing, exaggerated post-prandial response, itchiness, food cravings, decreased appetite, recurrent inflammations, angina, changes in kidney function, carpal-tunnel syndrome, slow speech.

Low thyroid activity may be likened to an engine that idles too slowly and, when required to move, cannot burn fuel properly so runs sluggishly. The horsepower (functioning ability) is depleted. Decreased thyroid hormone "can reduce the strength and resistance of every cell, including the billions involved in the immune system..." A common complaint is fatigue as thyroid hormones are vital to energy production. The mitochondria in each cell are the principal sites for the generation of energy. T_3 and T_4 "fire up" mitochondria to burn oxygen. With decreased hormone available, cells burn less oxygen, less energy is produced, and the body and brain become sluggish. T_3 interacts with neurotransmitters in the brain, so emotion and mood are effected. Sadly, many symptoms are frequently dismissed as a "normal part of aging," a psychological problem, overwork, or some other condition. Up to 20% of all chronic depression cases stem from low production of thyroid hormone. However, medical treatment for depression is usually prescribed and thyroid tests are never performed.

Some overweight people are convinced their "metabolism is slow" because of a thyroid problem. Yet the vast majority do not have underactive thyroid. Actually, conspicuous weight gain may not occur in many people with hypothyroidism. A sufferer is likely to get a little heavier because of the slower metabolism, but the average gain is only five to ten pounds.

Individuals with hypothyroidism have significantly higher intraocular pressure in both eyes than do controls. Thus many diagnoses of glaucoma (or intraocular pressure thought to precede glaucoma) may need therapy for the thyroid rather than the eyes. Enlarged salivary glands are common – parotids, submandibular, and especially sublingual glands. Women over age 65 with high blood levels of LDL (so-called "bad") cholesterol are likely to have an "undetected" underactive thyroid condition.

Hyperthyroidism (excessive level of thyroid hormone) is less common than underactive thyroid and usually produces different symptoms. Generally, too little thyroid hormone tends to slow the body's functions, and too much hormone tends to speed them up. The most diagnosed form of hyperthyroidism is Graves' disease, followed by toxic multinodular goiter and toxic adenoma. Iodine status affects incidence of all forms. Symptoms of hyperthyroidism may include:

Nervousness, anxiety, jitteriness, irritability, feeling "wired," muscle weakness, tremors of the hands, soft nails, rapid heartbeat, heart palpitations, weight loss, eyes appear wide open with little blinking, eyes may bulge outward (inflammation and buildup of tissue behind eyes), eye irritation, increased pressure on optic nerve, churning in stomach, frequent bowel movements, difficulty concentrating, memory problems, weakness, general fatigue, difficulty sleeping, heat intolerance, sweating, scanty menstrual periods, infertility, warm moist palms, hair loss, low blood cholesterol levels, accelerated bone loss especially in elderly women, enlarged thyroid (goiter), atrial fibrillation (heart rhythm disturbance), thyroid crisis or "thyroid storm" (drowsiness leading to stupor, fever, cardiac irregularities, dehydration, congestive heart failure, diarrhea, jaundice).

People with hyperthyroidism feel supercharged like an engine idling too fast. Metabolism speeds up. The body seems to function quicker than usual. Thyroid storm may be triggered or precipitated by serious acute inflammatory or infectious conditions, diabetic ketoacidosis, or surgery in an individual with hyperthyroidism.

Subclinical or mild thyroid imbalances are very common, particularly among people over 50 years of age. Some of the same symptoms or problems may be experienced as with clinical forms, but usually to a lesser degree. Sometimes there are no symptoms. Very few people with subclinical imbalances have the full constellation of symptoms. In subclinical **hypo**thyroidism, there is often an elevated level of TSH and normal levels of T_3 and T_4. Achilles reflex time and other reflexes may be slowed. In subclinical **hyper**thyroidism, TSH is low, T_3 and T_4 are normal. Notably, "transient thyroid failure is increasingly recognized" – which may

resolve "spontaneously" as the body regains physiological and biochemical balance.

Medical treatment for **clinical** hypothyroidism is virtually always thyroid hormone medication. Treatment for **subclinical hypo**thyroidism is controversial. Some doctors advocate hormone therapy for symptom improvement or to supposedly forestall clinical hypothyroidism. But not all people with subclinical conditions will develop primary conditions. Clinical trials show that patients with subclinical hypothyroidism given hormone improved little more than those given placebos. Similarly, "risks" of subclinical **hyper**thyroidism "are not well defined," not known. So far there are no clinical trials that have evaluated whether medical treatment is of any benefit. Nevertheless, some doctors "treat" it with antithyroid drugs or radioactive iodine, a questionable approach.

Though a subclinical condition "sometimes goes away on its own," there is "a disturbing tendency to treat the results" of blood tests "and not the patient." Plus "no one has yet proven that there are benefits to putting a person on lifelong therapy because of a test abnormality alone."

In some cases, especially subclinical, symptoms of **both** hypo- and hyperthyroidism appear. These fluctuations may reflect the thyroid's attempts, (along with input from the adrenals, pituitary, hypothalamus, liver, nervous system, etc.), to balance and improve its function. Researchers admit it is "intellectually arrogant" to assume everything is known about thyroid hormone metabolism and the effects of exogenous medical treatment. Thus, "patients would benefit considerably, in this instance, if physicians were to live up to their reputation for knowing everything but doing nothing."

Small bumps that may appear on the thyroid gland – nodules or "incidentalomas" – are usually benign (not recurrent or progressive), and may shrink or disappear on their own. Due t o "medical uncertainties" there is a question whether benign nodules should be treated. "Ask three thyroid experts, and you'll get four opinions." Unnecessary biopsies and treatment are common. Thyroid hormone drugs are often prescribed, but these shrink only a minority of nodules and can cause bone loss, abnormal heart rhythms, increased pulse, and other side effects. The prevalence of nodules without **any** thyroid condition is 30 to 60%. The risk of malignancy in symptomless nodules is about 5%. Since nodules tend to grow slowly, some researchers suggest simply monitoring them. After a year, if the nodule is the same size or smaller, no therapy is needed. If there is high risk, enlargement, pain, interference with swallowing or speech, or the appearance of malignancy during ultrasound examination, biopsy and treatment may be considered. ||

CAUSES

The most common forms of thyroid dysfunction are Hashimoto's disease (hypothyroid) and Graves' disease (hyperthyroid). The cause is attributed – theoretically – to "autoimmune disease," wherein the body "mistakenly attacks healthy thyroid tissue." This does not make biochemical sense because the body is always striving for equilibrium, for survival, not self-destruction. If the immune system's white blood cells engulf and eliminate tissues, it is only due to cellular damage and death. Thus, some researchers recognize many thyroid conditions as **inflammation**. Tissue insult or injury initiates the processes for repair by the process of inflammation. Many factors can potentially injure or poison the thyroid gland, resulting in depletion of needed "fuel" – nutrition — for proper function, maintenance, and resistance. Whether harmful substances or malnutrition or both, the affect on the thyroid depends on the specific causes and the unique biochemistry of the individual.

Various steroid hormone drugs can induce thyroid imbalance; glucocorticoids such as cortisol inhibit thyroid function. Medication for depression such as sertraline (Zoloft) may deepen depression by altering levels T_4 and TSH in the blood. Tricyclic antidepressants and antipsychotic phernothiazines (e.g., Flavil, Anafranil, Adapin, Sinequan, Tofrinil, Surmontil, Vivactil, Asendin, Norpramin, Pertofrane, Ludiomil, Pamelor) have a deterimental impact on thyroid function. Other drugs including lithium, amiodarone (antiarrhythmic drug), and cytokines (interferon, alpha, interleukin-2, macrophage colony stimulating factor) can cause thyroid malfunction. Radiation treatments to the neck increase risk of thyroid problems. Persons with diabetes, Lyme's disease, or pernicious anemia are at higher risk.

Estrogen-containing medications such as oral contraceptives and hormone-replacement therapy may affect thyroxine-binding globulin, or may enhance thyroxine (T_4) transport and lead to falsely normal levels of thyroid hormone in blood tests. Dopamine (used for Parkinson's disease) can cause a decrease in TSH production with resultant drops in serum T_3 and T_4 levels. Cigarette smoking can produce goiter, increase the severity and metabolic effects of hypothyroidism, and raise the risk for thyroid-associated eye disorders. Iodine-

containing drugs (such as amiodarone, an antiarrhythmic drug) and iodinated radiographic contrast agents can depress thyroid function. Excessive iodine supplementation, particularly from non-food sources can cause thyrotoxicosis (thyroid poisoning). Over 54% of all salt purchased in the U.S. is fortified with between 45 and 76 parts per million of non-food iodine. Differences and variations are common.

Chlorine and sodium fluoride added to municipal water supplies; chlorine-based chemicals found in bleaches, pesticides, and other products; and fluoride found in toothpaste and supplements, all promote thyroid disruption. Chlorine and fluoride may block iodine receptor sites on the thyroid, making it difficult or impossible for the gland to receive and utilize iodine. Compounds produced by chlorination of water are inhibitors of thyroid peroxidase and thyroid iodine organification. Immunization (especially DPT shots) and mercury amalgam toxicity have been implicated in thyroid imbalances.

"Evidence for environmental chemical triggers" for thyroid pathologies has been accumulating. The list includes polybrominated biphenyls, polyhalogenated aromatic hydrocarbons, DDT, DDE, dieldrin and other organo-chlorines, pheolic and phenolic-carboxylic derivatives, PCBs, PBBs, and many more. Petroleum and coal derivatives are "antithyroid and goitrogenic compounds." The prototype of coal derivatives, resorcinol, has been known to damage the thyroid since the 1950s. The above chemicals may appear in polluted water, processed foods, pharmaceuticals, textiles, home furnishings, resins, adhesives, rubber products, plastics of all types, dyes, pesticides, cars, building and construction materials, capacitors, electric or heat transformers, and many other items. Red dye no.3, popularly used in foods, cosmetics, and medications, was shown to consistently decrease T_3 levels, and increase T_4 and TSH.

The thyroid gland obviously absorbs a "great deal of punishment" in the course of typical modern conditions. The typical American diet adds to the trouble since many people do not obtain sufficient amounts of nutrients needed for healthy thyroid function, let alone some degree of resistance to the onslaught of poisons to which they may be subjected. Support to biochemical and physiological balance – and to the thyroid specifically – is needed. [iii]

TEMPERATURE

Basal (resting) body temperature and axillary (armpit) temperature – the Barnes Thyroid Test — are often used by alternative therapists to determine thyroid imbalance. If the temperature is below "normal" (98.6°F) the inference is underactive thyroid; if above "normal," overactive thyroid. Can these measurements alone be used to diagnose thyroid dysfunction?

Studies have demonstrated that 98.6°F (37° C) "should be abandoned as a concept having any particular significance for the normal body temperature." It may reflect an average, but not necessarily a "normal" temperature. Many healthy people have an average oral temperature of 97.6°F. Virtually everyone has a temperature lower than 98.6°F sometime during the day. Lower temperatures are common in early morning, higher in late afternoon (up to an average of 99.9°F). Some people seldom reach 98.6°F at all. In fact, 98.6°F "had no apparent relevance at all" and made up only about 8% of readings. Overall average body temperature for the majority of people is 98.2°F.

Normal temperatures can vary anywhere from 96°F (35.6°C) to 99.9°F (37.7°C). There is "considerable individual variability." Many s cientists would prefer to see "normal" body temperature listed in medical references as a **range** of values rather than as a mean. Dr. Roger Williams showed that various organs and tissues have distinctive temperature patterns. That is, many different temperatures occur in different areas of the same body, depending on "biochemical activity" which affects heat production. For example, higher or lower temperatures may be found in the distal forearm or hand compared to the proximal arm. "It is evident that temperature measurements reveal inescapably the existence of biochemical individuality. Even overall temperatures, though fundamentally summations, often reveal substantial differences."

Variations exist between men and women due to amounts of fat deposits, body size, skin surface in relation to height, amount of muscle mass, and for women, cyclic hormonal changes. Nevertheless, "the average difference between the sexes is usually smaller than the differences between one person and another." Basal temperature can be lower than "normal" when various metabolic diseases or imbalances exist.

No wonder "the Barnes Thyroid Test (axillary temperature) alone is not always accurate." If an individual's normal average temperature is known (which may be changeable) **prior** to the onset of suspected thyroid imbalance, then a comparison of before and after may be helpful. But it probably should not be used as the only determinant. [iv]

MEDICAL TREATMENTS

Hypothyroidism is invariably treated with thyroid hormone (thyroxine) medication. The current medical view that "there is no way to cure underactive thyroid" leads to the conclusion that "most people require lifetime hormone replacement." If the thyroid is totally nonfunctional – incapable of producing hormone – or is surgically removed, hormone drugs are probably inevitable. Yet, if the gland is underfunctioning – operating albeit below normal – the gland may simply not be receiving sufficient nutritional, nerve, or blood supply to assure optimal performance, or that something (e.g., toxic substance) is interfering with its effectiveness.

The most prescribed thyroid hormone drug is L-thyroxine (Synthroid, Levothroid, Levoxine, Levo-T) — synthetic hormone. "Even a small mistake in dosage can have consequences..." Periodic blood tests are needed to determine if the "right amount" is given. Symptoms of overdose are similar to hyperthyroidism and may include heart irregularities and bone loss. The combination with certain other medications (anticoagulants, antidiabetic drugs, digitalis, beta blockers, estrogen replacement and oral contraceptive hormones) can cause problems. Hormone dosages should be reduced as a person ages; a 70-year old woman requires only 2/3 of the dosage for a 25-year old. Thyroid medication can cause toxicosis (poisoning).

Two points are clear: (1) A thyroid hormone drug cannot truly replace a person's own thyroid gland activities. The natural fluctuations in hormone secretion – with the complex and delicately balanced feedback system according to the body's constantly changing needs — are not duplicated. When taking a drug, the body "reads" the presence of hormone in the blood so does not send the normal "distress signals" of underactive thyroid. Without the systemic demands for activity (due to presence of hormone in the blood), the gland eventually slows and ceases function. Thus hormone therapy can lead to effective "death" of the thyroid gland, usually within two years. Then the drug is needed for life.

(2) It is known that "despite much attention to the long-term safety and possible side-effects of thyroxine therapy, no consensus has yet been reached and adequate long-term prospective studies are lacking." Using hormones as drugs is playing with fire; in minute amounts they are extremely powerful chemical compounds. Synthetic hormones have more potential for harm and are poor imitations of the real item.

There is always a "question of the wisdom of giving medication – in particular, naturally occurring hormones – without considering its total effect on the organism and person..." For example, a diagnosis of hypothyroidism in an elderly person might prompt the immediate administration of thyroid hormone. This can "tip the patient into coronary insufficiency or heart failure." Such people "might be better off left alone." In fact, some physicians, like Richard Asher, have wondered whether the diminution of thyroid function sometimes "might not be a physiological or adaptive measure," as in the case above, designed to reduce the load on an ailing heart or compensate for something else.

There is "substantial evidence" that doses of thyroxine sufficient to suppress TSH secretion "are associated with changes in other organs and tissues" – such as the heart, liver, kidney, and muscle – "that resemble those recorded in overt (obvious) hyperthyroidism."

Bone density may be significantly reduced after six months of treatment with a thyroid hormone drug, which "could represent a physiological response to restoration of normal serum thyrotropin {TSH} concentrations after a lengthy period of thyroid failure." Suppression of blood levels of TSH by thyroid hormone medication in a postmenopausal woman who is already at risk of osteoporosis "may not be in the patient's best interests." Long-term hormone therapy above physiological dosages "may predispose patients to decreased bone density in the hip and may increase the risk of age-related bone loss." Significant bone loss still occurs without excessive dosages. So "even minimal suppression of thyrotropin is accompanied by biochemical evidence for increased bone turnover," and may lead to osteoposis.

Most doctors prescribe **synthetic** hormones thinking they are more pure, more predictable, more consistent, more effective than natural. This has not been proved and synthetics do not "work" for many people. The synthetic thyroxine (T_4) must be converted in the body to T_3 in order to be utilized. "Many people suffering from hypothyroidism cannot make this conversion."

Many clinicians claim the natural form of thyroid hormone (desiccated porcine or bovine gland such as Armour Thyroid) is more effective. It contains all the gland's hormones and other naturally-occurring components. For example, Dr. Alan Gaby states that 38% of the iodine present in the thyroid gland is in the form of diiodotyrosine, also found in the blood. It is not yet known what diiodotyrosine does, but it is certainly not inert. This compound, one of a

Dr. Smith does not recommend Armour

multiplicity of known and unknown components, is present in natural thyroid **and** in natural glandular supplements (hormones are removed from supplements). Substituting natural thyroid for synthetic thyroid leads to improvements in neuro-psychological functioning, cognition (language, learning, memory), and mood. However, hormone compounds, even those from natural sources, are very powerful; an excess can cause "thyroid storm." Glandular supplements could not have such effects; they simply provide food to support thyroid health.

Hyperthyroidism is medically treated by one of three methods: (1) radioactive iodine to disable the gland, (2) drugs to suppress hormone production, or (3) surgery to remove the thyroid followed by lifetime hormone replacement. Each of these methods can be "tricky."

Most doctors in the U.S. recommend radioactive iodine. The thyroid, with its special affinity for iodine, selectively absorbs this medication. Radioactive iodine necessitates radiation exposure, which has an "indisputable link" to cancer, "causes almost inevitable hypothyroidism," and may worsen eye problems or other symptoms. Although some studies report that thyroid cancer incidence was not increased after radioactive iodine therapy, other studies find "a small but statistically significant increase in thyroid cancer risk," an excess of thyroid cancer mortality after treatment, and a higher mortality rate from brain cancer and several other cancers. Also reported is increased risk for fracture of the femur, deaths due to thyroid disease, cardiovascular disease, and cerebrovascular disease.

The radiation dose to the thyroid is so high that in most cases the thyroid gland is "obliterated." The predominant effect "should be" cell killing, not mutilation. The outcome is hypothyroidism or athyroidism (no function). Thyroid hormone treatment is then recommended for 90% of patients.

Drugs such as propylthiouracil (PTU) or methiamazole (Tapazole) are sometimes used to inhibit synthesis of thyroid hormone with the hope that the hyperthyroid condition will go into remission. Such antithyroid drugs "usually do not cure the patient" and have "rare but potentially life-threatening" adverse effects including inhibition of bone marrow function (agranulocytosis, granulocytopenia, aplastic anemia, thrombocytopenia), drug fever, a lupus-like syndrome, insulin autoimmune syndrome (can result in hypoglycemia coma), hepatitis, inflammation of arteries, decreased level of prothrombin (for blood clotting), and more.

Surgery to remove the thyroid leaves a scar, causes permanent hypothyroidism (with lifetime hormone therapy needed), and "can be a source of significant morbidity [disease] from complications." There is some risk of injury to the parathyroid glands or vocal cords. Additionally, the operation "does not have a good success rate." About 19% of sugical cases are still hyperactive following surgery.

Almost one-third of cases of hyperthyroidism will resolve without any therapy. Whether **hypo**thyroid or **hyper**thyroid, the key is to "look for the underlying problems" – the cause – instead of treating the result, the symptoms. Various factors have been implicated in disrupting thyroid function including nutritional deficiencies or imbalances. [v]

NUTRITION

If not severe, the imbalance of an under- or overactive thyroid gland may possibly "be corrected through natural means." Deficits – or interference with availability of – iodine and other nutrients are usually involved. "Simply replacing these through supplementation can often improve function dramatically."

The thyroid gland contains a high concentration of iodine as well as ionized calcium, sodium, thiocyanate (CNS), bromine, fluorine, cloride, arsenic, and aluminum. No wonder the thyroid has an affinity for – and thus a predisposition for – toxic insult or injury by thiocyanate-type drugs (thiouracil, sulfa drugs, salicylates as aspirin), bromide drugs, industrially-produced sodium fluoride, chlorine chemicals, and aluminum (unnatural sources). Many of these toxic chemicals have a tendency to replace iodine which may be "a very mobile and vunerable substance in the thyroid..."

The bones have a thousand times more calcium than the thyroid, but among 34 other types of body structures, "calcium is found at its maximum in the thyroid and [is] activated or ionized there."

Conversion of T_4 into T_3 requires the minerals zinc, copper, and selenium as well as the essential fatty acids. Thus, even if sufficient hormone is produced by the thyroid, it may not be effectively converted into the most active or needed form if there are deficits of these and other nutrients. In one study, supplementation with zinc reestablished normal thyroid function in hypothyroid patients who had normal serum T_4 levels and zinc deficiency. About half of American diets are deficient in selenium. Persons living in areas of the world where the

soil is low in selenium have greater incidence of thyroid disease. Glutathione (glutamic acid, cysteine, glycine) is involved in conversion of T_4 to T_3; selenium increases the availability of glutathione. Deficits of potassium, phosphorus, boron, magnesium, manganese, chromium, and sodium are associated with hypothyroidism. "Nutritional" lithium compounds (inorganic isolates) can contribute to subclinical hypothyroidism.

A lack of vitamin A complex (or other fat-soluble vitamin) reserves can adversely affect thyroid function. An underfunctioning thyroid can decrease the conversion of carotenes into retinol (vitamin A). Vitamin A and zinc deficits have been reported in patients with increased incidence of goiter formation. Vitamins A, B complex (particularly B_2, B_3, B_6) and associated factors such as PABA (paraminobenzoic acid), E complex, and C complex are all nutrients required by the thyroid. Vitamin A deficiency reduces the thyroid's ability to take up iodine. Laboratory animals deficient in vitamin E complex have pituitary glands that do not synthesize adequate amounts of TSH. B vitamins are essential for efficient transport of oxygen to cells and thus to metabolism.

Calcium and vitamin D complex influence endocrine gland activity. In hypothyroidism, calcium absorption and retention is increased. Serum vitamin D_3 metabolites are elevated in hypothyroid patients and reduced in hyperthyroidism. Unsaturated and essential fatty acids play an essential role in thyroid function and its effect on metabolic rate. Essential fatty acids – particularly the omega-3 group and its derivatives – "are like a poker that stokes the fire," benefiting oxidation and metabolism. Natural, unaltered fats contain substances that balance metabolic rate, some slowing it down and others speeding it up.

Iodine is, of course, the primary nutrient related to thyroid function. It is a regulator of oxidation, metabolic rate, and energy production. A deficiency of iodine may result from the faulty metabolism of fats. Unsaturated fatty acids are actually rated by their iodine number. In hyperthyroidism, there is elevated **blood** iodine, but a deficit in the gland. In hypothyroidism, the gland has an overabundance of iodine, but low **blood** levels. Supplementation of iodine in hyperthyroidism slows or depresses excess thyroid function, allowing the gland to accept more needed iodine. Supplementation in hypothyroidism allows unsaturated fatty acid bonds to pick up iodine from the thyroid gland, raising blood iodine levels, and improving thyroid function. Further, many authorities believe that all thyroid enlargements – including exophthalmic (abnormal eye protrusion) goiter – are examples of "a compensatory work hypertropy" (attempts to rectify imbalance) due to iodine deficiency. The demands made upon the thyroid to produce its hormone without an adequate iodine – or other nutrient – supply results in hypertrophy (increase in size), hyperplasia (excessive proliferation of normal cells), adenomatous growths (multiple glandular tissue overgrowths), and in some instances, final exhaustion atrophy (wasting).

Unsaturated fatty acids, vitamin C complex, and all the specific amino acids peculiar to the thyroid gland are essential to the thyroid follicles' ability to properly utilize and metabolize iodine. High T_3 and free T_4 levels in the blood suggest thyroid difficulty in metabolizing iodine and T_4. The body contains only about 14 milligrams (mg) of iodine. In order to form normal quantities of thyroid hormone, approximately 35 to 50 mg of iodine must be ingested each year – about 1 mg each week – between 100 and 200 micrograms (mcg) per day. However, the thyroid can adapt to wide fluctuations of iodine intake and individual needs may vary. Unfortunately, modern farming methods have depleted much of the iodine in soils, resulting in low iodine levels in foods. Organically-produced foods are likely to have higher amounts of iodine and other trace minerals. Still, the amount of iodine in foods in different regions will vary widely.

Non-food inorganic iodine has been added to salt for many years. This is not ideal since it is not in a natural complex or protein-bound form. Many researchers believe the form of iodine in iodized salt may contribute to thyroid problems. Food sources such as deep-ocean seafood, kelp and other seaweed, are preferable. Excess iodine intake, especially from nonfood sources, can result in hypothyroidism and, in the presence of low selenium nutriture, may promote thyroid tissue damage.

Whole, natural foods and real food-concentrate supplements can do much for "restoration of relatively normal function" of the thyroid. Clinical reports indicate that, especially in subclinical imbalances, nutritional support alone is all that is required. Fish and other seafood, kelp, dark green and yellow vegetables, root vegetables (including potatoes), whole grains, raw nuts and seeds, for example, may help.

Some foods contain naturally-occurring **goitrogens** (thyroid depressants) and/or progoitrogens (thyroid stimulants). The list includes cabbage, kale, kohrabi, rutabega,

cauliflower, mustard greens, radishes, broccoli, brussels sprouts, maise (corn), peas, soy, lima beans, sweet potatoes, cassava, sorghum, apricots, prunes, walnuts, cherries, almonds, bamboo shoots. Although it is admitted that these foods are unlikely to adversely affect persons with normal thyroid function, persons with hypo- or hyperthyroidism are cautioned about frequent consumption.

However, the "negative influence" of these foods is based on tests using isolated chemicals – such as cyanogenic glucosides, thiocyanate, pressor amines – found in the foods OR tests with processed portions of the foods rather than the **whole**, natural foods. For example, isolated soy protein, as found in baby foods and f ormulas, is reported to produce goiters in i nfants. When whole foods are ingested, all the phytochemicals and nutrients are intact, so they will have a **balancing**, healthful effect on the thyroid. That is, whole foods may contain compounds that **both** slow and speed thyroid function. The body's **selective absorption** allows a choice of what and how much of these components are used. In traditional "medicines" around the world, many of the foods listed above have been used to treat both hypo- and hyperthyroidism. Nature balances. Human alterations or synthetic chemicals distort. [vi]

CONCLUSION

Although thyroid health may seem complex, the essence of thyroid function encompasses the a simple principle that permeates biochemistry: The healthy body is far more than just the operation of its individual parts. Each gland, organ, tissue, even each cell, cooperates and strives for dynamic balance. Toxic substances, imbalances, deficiencies, or excessive stresses may interfere with the operation of a susceptible area such as the thyroid gland. Often there is a cascade of responses involving other body parts. Avoiding poisons and providing natural, functional nutrients may accomplish much in aiding the body to achieve the equilibrium for which it persistently toils.

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