NP Thyroid™ tablets (USP) for oral use is a natural preparation derived from porcine thyroid glands. They contain both tetraiodothyronine (T4) and triiodothyronine (T3) and are marketed in 15 mg (1/4 gr) tablets containing 5 mcg of T3 and 60 mcg of T4 per grain of thyroid (or per 60 mg of the labeled amount of thyroid). The inactive ingredients are calcium stearate, dextrose (agglomerated), and vegetable oil.

**Clinical Pharmacology**

The drugs in the synthetic thyroid hormone preparations are controlled by Thyroid Thyroid Stimulating Hormone (TSH) secreted by the pituitary. The hormone secretion is in turn controlled by a feedback mechanism effectuated by the thyroid gland themselves and by thyrotropin releasing hormone (TRH), a hypothalamic protein of origin. Endogenous thyroid hormone secretion is suppressed when TSH concentration is high. TSH is secreted in response to thyroid stimulating effects of TRH. The mechanism by which thyroid hormones exert their physiologic action are not well understood. These hormones enhance oxygen consumption by most tissues of the body, increase the basal metabolic rate, and the metabolism of carbohydrates, lipids, and proteins. Thus, they exert a profound influence of the peripheral tissues of the body in converting stored energy into usable forms of energy. Normally, a normal thyroid gland contains approximately 200 mcg of levothyroxine (T4) per gram of tissue and 15 mcg of liothyronine (T3) per gram.

The metabolic fate of T4 and T3 is dependent upon the individual. Approximately 90 percent of peripheral T4 is converted to T3 by a complex series of metabolic processes. The remaining 10 percent of peripheral liothyronine comes from monodeiodination of levothyroxine at the 5-position (inner ring) also results in the formation of reverse liothyronine (T3), which is catabolically inactive. Liothyronine (T3) levels are low in the fetus and newborn and rise within 6 to 12 hours of birth. Vascular innovation, hepatic crisis, renal failure, surgical stress, and chronic illnesses representing what has been called the "13 thyroid syndrome."  

**Pharmacokinetics** — Animal studies have shown that T4 is only partially absorbed from the gastrointestinal tract. The degree of absorption is dependent upon the individual, the source of the preparation, including changes in protein, and soluble dietary factors. All of which bind thyroid hormone and thereby make it unavailable for diffusion. Only 41 percent is absorbed when given in a gelatin capsule as opposed to 74 percent absorption when given with an albumin, according to other reports. Absorption of T3 has been determined to be 49 percent of the amount administered. In patients with thyroiditis, approximately 70 percent of the administered T3 is absorbed. In those patients without thyroiditis, approximately 90 percent of peripheral liothyronine comes from monodeiodination of levothyroxine.  

Monodeiodination of liothyronine at the 5-position results in the formation of reverse liothyronine (T3), which is biologically inactive. The natural preparations are absorbed in a manner similar to the synthetic hormones. More than 99 percent of circulating hormones are bound to serum proteins, including thyroid-binding globulin (TBg), thyroid-binding proteins (TBPA), and albumin (Tb), whose capacities and affinities vary for the hormones. The high affinity of levothyroxine (T4) for both Tbg and TBPA is compared to liothyronine (T3) partially explains the higher serum levels and longer half-life of the former hormone. Both protein-bound hormone exist in relative equilibrium with minute amounts of free hormone, the latter accounting for the metabolic activity. Distribution of levothyroxine (T4) occurs at a number of sites, including liver, kidney, and other tissues. The conjugated hormone, in the form of glucuronide or sulfate, is found in the bile and urine where it may complete an enterohepatic circulation. Eighty-five percent of levothyroxine (T4) metabolized daily is deiodinated.

**Indications and Usage** — NP Thyroid™ tablets (USP) are indicated: 1. As replacement or supplemental therapy in patients with hypothyroidism due to primary deficiencies of thyroid(ie, congenital hypothyroidism, myxedema, and primary hypothyroidism in patients of any age) (children, adults, the elderly, or those living in tropical countries) or secondary deficiencies (due to pituitary or hypothalamic insufficiency). 2. As replacement therapy in patients with thyroiditis, and in those patients receiving a therapeutic trial of the drug. 3. As diagnostic agents in suppression tests to differentiate suspected mild hyperthyroidism or hypothyroidism of maternal origin.  

**Contraindications** — Thyroid hormone preparations are generally contraindicated in patients with diagnosed or suspected undulant or acute oral sulfonamides, and apparent hyperthyroidism to any of their active or odorous constituents. There is no well documented evidence from the literature, however, of true allergic or idiosyncratic reactions to thyroid hormones.

**Warnings** — Drugs with thyroid hormone activity, alone or together with other therapeutic agents, have been used for the treatment of obesity. In euthyroid patients, those within the range of daily hormonal requirements are insufficient for weight reduction. Larger doses may produce nausea or (even the threatening manifestations of toxicity, particularly when given in association with sympathomimetic amines such as Amphetamines due to the similar effects. The use of thyroid hormones in the therapy of obesity, alone or combined with other drugs, is unjustified and has shown be ineffective. Neither is their use justified for the treatment of male or female infertility unless this condition is accompanied by hypothyroidism.  

**Precautions** — General — Thyroid hormones should be used with great caution in a number of circumstances where the integrity of the cardiovascular system, particularly the coronary arteries, is suspect. These include patients with angina pectoris or the elderly, in whom there is a greater likelihood of coronary disease; patients with glaucoma; nephritis, cirrhosis, myxedema, and primary hypothyroidism in patients of any age (children, adults, the elderly, or those living in tropical countries) or secondary deficiencies (due to pituitary or hypothalamic insufficiency).  

**Dosage and Administration** — In the past, the usual suppressive dose of levothyroxine (T4) is 1.56 mcg/kg of body weight per day given for 7 to 10 days. These doses usually yield TSH levels of 0.05 to 0.2 mU/L. However, because of the inaccuracy of the TSH assay, the following guidelines are suggested for the use of thyroid hormone in children. The initial dose of thyroid hormone in children with primary hypothyroidism is 15 mcg/kg of body weight per day. Approximately one-third of the total daily dose may be administered at bedtime. Children with severe hypothyroidism may need larger initial dosages. The daily dose is increased by 15 mcg/kg of body weight per day at 2 to 3 weekly intervals, or until the desired serum TSH level is achieved. Additional dosage adjustments are made based on the child's response assessed on the basis of clinical and laboratory findings.  

**Adverse Reactions** — Adverse reactions other than those indicative of hyperthyroidism because of therapeutic overdosage, either initially or during the maintenance period, are rare (see OVERDOSAGE).  

**Drug/Laboratory Test Interactions** — The following drugs or moieties are known to interfere with laboratory tests performed in patients on thyroid hormone therapy: androgens, corticosteroids, estrogens, oral contraceptives containing estrogens, iodine-containing preparations, and salicylates. The effects seen are poorly understood and depend upon a variety of factors such as dose and type of thyroid preparations and endocrine states of the patient. Increases in TBg concentration should be taken into consideration in the interpretation of T4 and T3 values. In such cases, the unbound (free) hormone should be measured. Pregnancy, estrogens, and estrogen-containing oral contraceptives increase TBg. TBg may also be increased during infectious diseases. Decreases in TBg can occur in neoplastic, anorexia, and after androgen or corticosteroid therapy. Familial hyper- or hypothyroidism binding globulins have been described. In the presence of deficient TBg, binding activity is reduced.  

**NP Thyroid™ Tablets**

**Pharmacology**

**T4**

**T3**

**TSH**

**Thyroid Cancer**

Hyperthyroidism

**Indications and Usage**

**Contraindications**

**Warnings**

**Precautions**

**Dosage and Administration**

**Adverse Reactions**

**Drug/Laboratory Test Interactions**

**NP Thyroid™ Tablets**

**Pharmacology**

**T4**

**T3**

**TSH**

**Thyroid Cancer**

Hyperthyroidism

**Indications and Usage**

**Contraindications**

**Warnings**

**Precautions**

**Dosage and Administration**

**Adverse Reactions**

**Drug/Laboratory Test Interactions**