PRODUCT MONOGRAPH

[Pr]Auro-Liothyronine

(liothyronine sodium)

USP

Tablets, Oral

5 mcg and 25 mcg Liothyronine [as Liothyronine Sodium]

HYPOTHYROIDISM THERAPY

Auro Pharma Inc.
3700 Steeles Avenue West, Suite # 402
Woodbridge, Ontario, L4L 8K8,
Canada

Date of Preparation:
JUL 17, 2024

Submission Control No: 272838

Table of Contents

INDICATIONS AND CLINICAL USES

Auro-Liothyronine is indicated in conditions of inadequate endogenous thyroid production. These include:

a. HYPOTHYROIDISM: As replacement or supplemental therapy in patients with hypothyroidism of any etiology except transient hypothyroidism during the recovery phase of subacute thyroiditis

b. SIMPLE (non-toxic) GOITER: Auro-Liothyronine may be tried therapeutically in an attempt to reduce the size of such a goiter, the rationale being that suppression of pituitary thyroid stimulating hormone (TSH) removes at least one of the growth-promoting factors.

Auro-Liothyronine may be used in the T3 suppression test to differentiate suspected hyperthyroidism from euthyroidism. (See special instructions under ADMINISTRATION AND DOSAGE),

Auro-Liothyronine can be used in patients allergic to dessicated thyroid or thyroid extract derived from pork or beef.

Use in Pregnancy: See WARNINGS and PRECAUTIONS

Use in Nursing Mothers: See WARNINGS and PRECAUTIONS

Pediatric Use: See WARNINGS and PRECAUTIONS and DOSAGE and ADMINISTRATION

Geriatic Use: See WARNINGS and PRECAUTIONS and DOSAGE and ADMINISTRATION

CONTRAINDICATIONS

- Patients with an apparent hypersensitivity to liothyronine sodium or any of the inactive product constituents.

- Patients with uncorrected adrenal insufficiency, as thyroid hormones increase tissue demands for adrenocortical hormones and may thereby precipitate acute adrenal crisis (see WARNINGS AND PRECAUTIONS).

- Patients with acute myocardial infarction

WARNINGS AND PRECAUTIONS

Serious Warnings and Precautions
The use of Auro-Liothyronine or other thyroid preparations in the therapy of obesity is

unjustified and dangerous. In euthyroid patients, doses within the range of daily hormonal requirements are ineffective for weight reduction. Larger doses may produce serious or even life-threatening manifestations of toxicity, particularly when given in association with sympathomimetic amines such as those used for anorectic effects.

General

Liothyronine sodium has a narrow therapeutic index and has a relatively short half-life compared to levothyroxine. Regardless of the indication for use, careful dosage titration is necessary to avoid the consequences of over or under treatment. These consequences include, among others, effects on growth and development, cardiovascular function, bone metabolism, reproductive function, emotional state and gastrointestinal function. Many drugs interact with liothyronine sodium, necessitating adjustments in dosing to maintain therapeutic response (See DRUG INTERACTIONS).

Cardiovascular

Since Auro-Liothyronine is a metabolic stimulant, it is not recommended for use in patients with compromised cardiovascular systems, particularly the coronary arteries. These include patients with angina pectoris, ischemic states, and the elderly (in whom there is a greater likelihood of occult cardiac disease). However, if it is used in such patients, caution should be exercised and the starting dosage should never be more than 5 mcg daily. If the dosage is increased, it should be in increments of no more than 5 mcg daily at two-week intervals. (See DOSAGE and ADMINISTRATION).

Endocrine and Metabolism

Patients with Nontoxic Diffuse Goiter or Nodular Thyroid Disease

Exercise caution when administering liothyronine sodium to patients with nontoxic diffuse goiter or nodular thyroid disease in order to prevent precipitation of thyrotoxicosis. Based on shared pharmacological effect of thyroid hormones, the below caution may apply to liothyronine:
In those patients, particularly the elderly or those with underlying cardiovascular disease, levothyroxine therapy is contraindicated if the serum TSH level is already suppressed due to the risk of precipitating overt thyrotoxicosis. If the serum TSH level is not suppressed, levothyroxine should be used with caution in conjunction with careful monitoring of thyroid function for evidence of hyperthyroidism and clinical monitoring for potential associated adverse cardiovascular signs and symptoms of hyperthyroidism.

Myxedematous patients are sensitive to thyroid substances; dosage should be started at a very low level and increased gradually (See DOSAGE and ADMINISTRATION).

Diabetes

Patients with diabetes mellitus may require upward adjustments of their antidiabetic therapeutic regimens when treated with liothyronine sodium (see **DRUG INTERACTIONS**).

Hypothalamic/Pituitary Hormone Deficiencies

In patients with secondary or tertiary hypothyroidism, additional hypothalamic/pituitary hormone deficiencies should be considered, and, if diagnosed, treated (see **WARNINGS AND PRECAUTIONS, Autoimmune Polyglandular Syndrome** for adrenal insufficiency).

Severe and prolonged hypothyroidism can lead to a decreased level of adrenocortical activity commensurate with the lowered metabolic state. When thyroid replacement therapy is administered, the metabolism increases at a greater rate than adrenocortical activity. This can precipitate adrenocortical insufficiency. Therefore, in severe and prolonged hypothyroidism, supplemental adrenocortical steroids may be necessary.

In rare instances, the administration of thyroid hormone may precipitate a hyperthyroid state or may aggravate existing hyperthyroidism. In infants, excessive doses of thyroid hormone preparations may produce cradiosynostosis.

Morphologic hypogonadism and nephrosis should be ruled out before the drug is administered. If hypopituitarism is present, the adrenal deficiency must be corrected before Auro-Liothyronine therapy is started.

Bone Mineral Density

In women, long-term thyroid hormones therapy has been associated with increased bone resorption, thereby decreasing bone mineral density, especially in post-menopausal women on greater than replacement doses or in women who are receiving suppressive doses of thyroid hormones. Therefore, it is recommended that patients receiving thyroid hormones be given the minimum dose necessary to achieve the desired clinical and biochemical response.

Immune

Autoimmune Polyglandular Syndrome

Occasionally, chronic autoimmune thyroiditis may occur in association with other autoimmune disorders such as adrenal insufficiency, pernicious anemia, and insulin dependent diabetes mellitus. Patients with concomitant adrenal insufficiency should be treated with replacement glucocorticoids prior to initiation of treatment with liothyronine sodium. Failure to do so may precipitate an acute adrenal crisis when thyroid hormone therapy is initiated, due to increased metabolic clearance of glucocorticoids by thyroid hormone.

Special Populations

Pregnancy: Thyroid hormones do not readily cross the placental barrier. The clinical experience to date does not indicate any adverse effect on fetuses when thyroid hormones are administered to pregnant women. Thyroid replacement therapy to hypothyroid women should not be discontinued during pregnancy.

Pregnant women who are maintained on liothyronine sodium should have their TSH measured periodically and liothyronine sodium dose adjusted as necessary. Serum TSH level should be obtained six to eight weeks postpartum to adjust dosage.

There are no available animal data informing the drug-associated risk.

Breast-feeding: Thyroid hormones are excreted in human milk in minimal amounts. Because liothyronine is excreted in human milk, caution should be exercised.

Pediatrics: Clinical assessment of growth, development, and thyroid status should be monitored frequently (See DOSAGE AND ADMINISTRATION).

Geriatrics: Clinical studies of liothyronine sodium did not include sufficient numbers of subjects aged 65 and over to determine whether they respond differently from younger subjects. This drug is known to be substantially excreted by the kidney, and the risk of toxic reactions to this drug may be greater in patients with impaired renal function. Because elderly patients are more likely to have decreased renal function, care should be taken in dose selection and renal function should be monitored. Because of the increased prevalence of cardiovascular disease among the elderly, liothyronine therapy should not be initiated at the full replacement dose (See DOSAGE and ADMINISTRATION).

Monitoring and Laboratory Tests
Periodic measurements of thyroid function tests should be performed guided by the patient's condition.

ADVERSE REACTIONS

Adverse reactions associated with liothyronine sodium are primarily those of hyperthyroidism, due to therapeutic overdosage (see WARNINGS and PRECAUTIONS, General and Overdosage). They include the following:

General: excessive sweating, fatigue, increased appetite, weight loss, heat intolerance, fever.
Cardiovascular: cardiac arrhythmias, angina pectoris, palpitations, tachycardia, increased pulse, increased blood pressure, heart failure
Central Nervous System: headache, nervousness, hyperactivity, anxiety, irritability, emotional lability, insomnia
Hypersensitivity: rare instances of allergic skin reactions have been reported
Endocrine: decreased bone mineral density
Gastrointestinal: diarrhea
Musculoskeletal: tremors, muscle weakness
Reproductive: menstrual irregularities
Respiratory: dyspnea

Medication should be interrupted until symptoms disappear, then resumed in smaller doses. Therapy can usually be resumed after one or two days.

DRUG INTERACTIONS

Overview:
Any agent that alters thyroid hormone synthesis, secretion, distribution, effect on target tissues, metabolism, or elimination may alter the optimal therapeutic dose of Auro-Liothyronine. A listing of drug-thyroidal axis interactions is contained in the Table below:

Drug or Drug Class	Effect
Drugs that alter thyroid hormone secretion Drugs that may decrease thyroid hormone secretion, which may result in hypothyroidism	
Aminoglutethimide Amiodarone Iodide (including iodine-containing radiographic contrast agents) Lithium Thioamides - Methimazole - Propylthiouracil (PTU) - Carbimazole Sulfonamides Tolbutamide	Long-term lithium therapy can result in goiter in up to 50% of patients, and either subclinical or overt hypothyroidism, each in up to 20% of patients. The fetus, neonate, elderly and euthyroid patients with underlying thyroid disease (e.g., Hashimotos's thyroiditis or with Grave's disease previously treated with radioiodine or surgery) are among those individuals who are particularly susceptible to iodine-induced hypothyroidism. Oral cholecystographic agents and amiodarone are slowly excreted, producing more prolonged hypothyroidism than parenterally administered iodinated contrast agents. Long-term aminoglutethimide therapy may minimally decrease T4 and T3 levels and increase TSH, although all values remain within normal limits in most patients.
Drugs that may decrease T3 absorption, which may result in hypothyroidism	
Bile Acid Sequestrants - Cholestyramine	Cholestyramine binds orally administered thyroid hormones in the intestine, impairing their absorption. *In vitro* studies indicate that the binding is not easily overcome. Therefore, four to five hours should elapse between administration of cholestyramine and thyroid hormones.
Drugs that may alter T3 serum transport	
Drugs that may increase serum TBG Concentration Clofibrate Estrogen-containing Oral Contraceptives Estrogens (oral) Heroin/Methadone 5-Fluorouracil Mitotane Tamoxifen	Drugs that may decrease serum TBG Concentration Androgens/Anabolic Steroids Asparaginase Glucocorticoids Slow-Release Nicotinic Acid
Miscellaneous	
Oral Anticoagulants	Thyroid hormones appear to increase the catabolism of vitamin K-dependent clotting factors. Patients stabilized on oral anticoagulants may require dosage adjustment, through close monitoring of Prothrombin time, when thyroid therapy is started. No special precautions appear to be necessary when oral anticoagulant therapy is begun in a patient already stabilized on maintenance thyroid therapy.
Tricyclic Antidepressants	Use of thyroid products with imipramine and other tricyclic antidepressants may increase receptor sensitivity and enhance antidepressant activity; transient cardiac arrhythmias have been observed. Thyroid hormone activity may also be enhanced.

Drug or Drug Class	Effect
Drugs that alter thyroid hormone secretion Drugs that may decrease thyroid hormone secretion, which may result in hypothyroidism	
Insulin or Oral Hypoglycemics	Initiating thyroid replacement therapy may require increases in insulin or oral hypoglycemic requirements. The effects seen are poorly understood and depend upon a variety of factors such as dose and type of thyroid preparations and endocrine status on the patient.
Digitalis	Thyroid preparations may potentiate the toxic effects of digitalis. Thyroid hormonal replacement increases metabolic rate, which requires an increase in digitalis dosage.
Cytokines -Interferon-alpha -Interleukin-2	Therapy with interferon-alpha has been associated with the development of antithyroid microsomal antibodies in 20% of patients and some have transient hypothyroidism, hyperthyroidism, or both. Patients who have antithyroid antibodies before treatment are at higher risk for thyroid dysfunction during treatment. Interleukin-2 has been associated with transient painless thyroiditis in 20% of patients. Interferon- beta and gamma have not been reported to cause thyroid dysfunction.
Growth Hormones - Somatrem - Somatropin	Excessive use of thyroid hormones with growth hormones may accelerate epiphyseal closure. However, untreated hypothyroidism may interfere with growth response to growth hormone.
HMG-CoA reductase inhibitors (statins) - Lovastatin	Some statins may increase thyroid hormone requirements. It is unknown if this occurs with all statins. Close monitoring of thyroid function and appropriate thyroid hormone dose adjustments may be necessary when thyroid hormones and statins are co-prescribed.
Ketamine	When administered to patients on a thyroid preparation, the parenteral anesthetic may cause hypertension and tachycardia. Use with caution and be prepared to treat hypertension, if necessary.
Methylxanthine Bronchodilators - (e.g., Theophylline)	Decreased theophylline clearance may occur in hypothyroid patients; clearance returns to normal when the euthyroid state is achieved.
Radiographic agents	Thyroid hormones may reduce the uptake of ^{123}I, ^{131}I, and ^{99}mTc.
Sympathomimetics/Catecholamines	Concurrent use may increase the effects of sympathomimetics, catecholamines or thyroid hormone. Thyroid hormones may increase the risk of coronary insufficiency when sympathomimetic or catecholamine agents are administered to patients with coronary artery disease.

Drug or Drug Class	Effect
Drugs that alter thyroid hormone secretion Drugs that may decrease thyroid hormone secretion, which may result in hypothyroidism	
Diazepam Ethionamide Metoclopramide Para-aminosalicylate sodium Perphenazine Resorcinol (excessive topical use)	These agents have been associated with thyroid hormone and/or TSH level alterations by various mechanisms.

DRUG/LABORATORY TEST INTERACTIONS

Since liothyronine is not as firmly bound to serum protein as thyroxine, the PBI usually remains at levels below normal during full replacement therapy. As with all thyroid preparations, thyroid gland-function reflected by ^{131}I uptake may be depressed by Auro-Liothyronine, particularly when dosage exceeds 75 mcg daily. This effect disappears rapidly, and useful ^{131}I uptake values may be obtained usually within two weeks following discontinuance of the drug.

The following drugs or moieties are known to interfere with laboratory tests performed in patients on thyroid hormone therapy: androgens, corticosteroids, estrogens and estrogen-containing oral contraceptives, iodine-containing preparations and salicylates.

Changes in TBG concentration should be taken into consideration in the interpretation of T4 and T3 values, which necessitates measurement and evaluation of unbound (free) hormone and/or determination of the free-T4 index (FT4I). Pregnancy, infectious hepatitis, estrogens and estrogen-containing oral contraceptives, and acute intermittent porphyria increase TBG concentration. Decreases in TBG concentrations are observed in nephrosis, severe hypoproteinemia, severe liver disease, acromegaly and after androgen or corticosteroid therapy. Familial hyper- or hypo-thyroxine binding- globulinemias have been described.

The binding of thyroxine by thyroxine-binding prealbumin (TBPA) is inhibited by salicylates.

Medicinal or dietary iodine interferes with all in vivo tests of radioiodine uptake, producing low uptakes which may not be reflective of a true decrease in hormone synthesis.

Drug-Food Interactions: interactions with food products have not been established.

Drug-Herb Interactions: interactions with herbal products have not been established.

DOSAGE AND ADMINISTRATION

Dosing Considerations

Optimum dosage is usually determined by the patient's clinical response. Confirmatory tests include: Radioactive Iodine T_3 Resin Uptake, ^{131}I-T3 Red cell Uptake, BMR, and the Achilles Tendon Reflex Test. Comparatively small doses (as little as 5 mcg daily) may be fully effective in some cases; however, in other instances, a satisfactory clinical response may not be achieved

until recommended dosage levels have been exceeded. Daily doses of up to 150 mcg may be required in a few resistant patients.

Recommended Dose and Dosage Adjustment

It is not necessary to divide daily maintenance doses. Although Auro-Liothyronine has a rapid cut-off, its metabolic effects persist for a few days following discontinuance.

Mild Hypothyroidism: Recommended starting dose is 25 mcg daily. Daily dosage then may be increased by 12.5 or 25 mcg every one or two weeks. Usual maintenance dosage is 25 – 75 mcg daily. Smaller doses may be fully effective in some patients, while 100 mcg may be required in others.

Myxedema: Myxedematous patients are very sensitive to thyroid substances; dosage should be started at a very low level and increased gradually.

Recommended starting dosage is 5 mcg daily. This may be increased by 5 or 10 mcg daily every one or two weeks. Usual maintenance dosage is 50 to 100 mcg daily.

Cretinism: Since the mother provides little or no thyroid hormone to the fetus, infants with thyroid dysfunction will require replacement therapy from birth. Treatment should be initiated as early as possible to avoid permanent physical and mental changes.

Recommended starting dosage is 5 mcg daily, with a 5 mcg increment every three to four days until the desired response is achieved. Infants a few months old may require only 20 mcg daily for maintenance. At one year, 50 mcg daily may be required. Above 3 years, full adult dosage may be necessary.

Simple (non-toxic) Goiter: recommended starting dosage is 5 mcg daily. This dosage may be increased by 5 to 10 mcg daily every one to two weeks. When 25 mcg daily is reached, dosage may be increased every week or two by 12.5 or 25 mcg. Usual maintenance dosage is 75 mcg daily.

Special Populations

Pediatrics: Treatment should be initiated immediately upon diagnosis and maintained for life, unless transient hypothyroidism is suspected, in which case, therapy may be interrupted for 2 to 8 weeks after the age of 3 years to reassess the condition. Gradual cessation of therapy is justified in patients who have maintained a normal TSH during those 2 to 8 weeks. Therapy should be started with 5 mcg daily and increased only by 5 mcg increments at the recommended intervals. See WARNINGS and PRECAUTIONS.

Geriatrics: Therapy should be started with 5 mcg daily and increased only by 5 mcg increments at the recommended intervals. In general, dose selection for an elderly patient should be cautious, usually starting at the low end of the dosing range, reflecting the greater frequency of decreased hepatic, renal, or cardiac function, and of concomitant disease or other drug therapy.

See WARNINGS and PRECAUTIONS.

Switching to Auro-Liothyronine from thyroid, L-thyroxine or thyroglobulin medication

When switching a patient to Auro-Liothyronine from thyroid, L-thyroxine or thyroglobulin, discontinue the other medication, initiate Auro-Liothyronine at a low dosage, and increase gradually according to the patient's response. When selecting a starting dose, bear in mind that this drug has a rapid onset of action, and that residual effects of the other thyroid preparation may persist for the first several weeks of therapy.

Special Instruction for T_3 Suppression Test: When ^{131}I thyroid uptake is in the borderline-high range, administer 75-100 of Auro-Liothyronine daily for 7 days, then repeat ^{131}I thyroid uptake test. In the hyperthyroid patient, 24-hour ^{131}I thyroid uptake will not be affected significantly. In the euthyroid patient, 24-hour ^{131}I thyroid uptake will drop to less than 20%.

Missed Dose
The missed dose should be taken as soon as possible. If it is almost time for the next dose, the missed dose should not be taken. Instead, the next regularly scheduled dose should be taken. Doses should not be doubled.

OVERDOSAGE

For management of a suspected drug overdose, contact your regional poison control centre.

Symptoms: see **WARNINGS AND PRECAUTIONS, General** and **ADVERSE REACTIONS.** Angina pectoris or congestive heart failure may be induced or aggravated. Shock may also develop. Massive overdosage may result in symptoms resembling thyroid storm. Chronic excessive dosage will produce the signs and symptoms of hyperthyroidism.

Treatment: Dosage should be reduced or therapy temporarily discontinued if signs and symptoms of overdosage appear. Treatment may be reinstituted at a lower dosage. In normal individuals, normal hypothalamic-pituitary-thyroid axis function is restored in 6 to 8 weeks after thyroid suppression.

Treatment of acute massive thyroid hormone overdosage is aimed at reducing gastrointestinal absorption of the drugs and counteracting central and peripheral effects, mainly those of increased sympathetic activity. Vomiting may be induced initially if further gastrointestinal absorption can reasonably be prevented and barring contraindications such as coma, convulsions, or loss of the gagging reflex. Treatment is symptomatic and supportive. Oxygen may be administered and ventilation maintained. Cardiac glycosides may be indicated if congestive heart failure develops. Measures to control fever, hypoglycemia, or fluid loss should be instituted if needed. Antiadrenergic agents, particularly propranolol, have been used advantageously in the treatment of increased sympathetic activity.

ACTION

There are two principal naturally occurring thyroid hormones, L-tetraiodothyronine (T_4, levothyroxine, L-thyroxine) and L-triiodothyronine (T_3, liothyronine).

The mechanisms by which thyroid hormones exert their physiologic actions are not well understood. It is generally believed that they exert most, if not all, of their actions through control of protein synthesis.

At moderate concentrations, thyroid hormones increase the synthesis of RNA and protein, followed by an increase in basal metabolic rate (BMR). They stimulate oxidative enzyme systems generally, and enhance the release of free fatty acids from adipose tissue. They also increase the intestinal absorption and peripheral utilization of glucose. At higher concentrations, thyroid hormones decrease protein synthesis, uncouple oxidative phosphorylation and increase breakdown of glycogen, lipids and protein.

CLINICAL PHARMACOLOGY

Liothyronine sodium is the synthetic levo form of triiodothyronine, with all pharmacologic activities of the natural substance. Its onset of action is rapid, occurring within a few hours. Maximum pharmacologic response occurs within two or three days, providing early clinical response.

Following oral administration, about 95% of the dose of thyronine is absorbed from the gastrointestinal tract in four hours. Liothyronine, not firmly bound to serum protein, is readily available to body tissues. Its biologic half-life is about 2 ½ days.

Liothyronine has a rapid cut-off of activity which permits quick dosage adjustment and facilitates control of the effects of overdosage should they occur.

COMPARITIVE BIOAVAILABILITY STUDIES

A randomized, single-dose (4 x 25 mcg), two-way crossover comparative bioavailability study of Auro-Liothyronine (Auro Pharma Inc.) and CYTOMEL (Pfizer Canada Inc.) was conducted in 72 healthy, adult, male subjects under fasting conditions. Comparative bioavailability data from the 68 subjects who completed both periods of the study are presented in the following table:

Summary Table of the Comparative Bioavailability Data

Liothyronine (4 x 25 mcg) Baseline corrected data Geometric Mean Arithmetic Mean (% CV)				
Parameter	Test[1]	Reference[2]	% Ratio of Geometric Means	90% Confidence Interval
AUC_{0-48h} (ng•h/mL)	78.67 81.67 (27.00)	77.38 79.89 (24.22)	101.7	99.4 - 104.00
AUCi (ng•h/mL)	88.77 93.10 (30.18)	88.89 92.64 (28.39)	99.9	97.0 - 102.9
C_{max} (ng/mL)	7.71 8.01 (27.78)	7.42 7.68 (25.98)	103.9	100.7 - 107.2
T_{max}[3] (h)	2.00 (1.33– 4.00)	2.33 (1.33– 4.02)		
$T_{½}$[4] (h)	16.98 (34.77)	17.73 (42.68)		

[1] Auro-Liothyronine (liothyronine as liothyronine sodium) tablets 25 mcg (Auro Pharma Inc.)
[2] CYTOMEL® (liothyronine as liothyronine sodium) tablets 25 mcg (Pfizer Canada Inc.)
[3] Expressed as the median (range) only.
[4] Expressed as arithmetic mean (% CV) only.

A randomized, single-dose (4 x 25 mcg), two-way crossover comparative bioavailability study of Auro-Liothyronine (Auro Pharma Inc.) and CYTOMEL (Pfizer Canada Inc.) was conducted in 72 healthy, adult, male subjects under high fat, high calorie fed conditions. Comparative bioavailability data from the 64 subjects who completed both periods of the study are presented in the following table:

Summary Table of the Comparative Bio-availability Data

Liothyronine (4 x 25 mg) Baseline corrected data Geometric Mean Arithmetic Mean (CV %)				
Parameter	**Test[1]**	**Reference[2]**	**% Ratio of Geometric Means**	**90% Confidence Interval**
AUC_{0-48h} (ng•h/mL)	58.72 60.50 (23.55)	59.88 62.00 (24.49)	98.1	94.7 - 101.5
AUC_I (ng•h/mL)	64.53 67.76 (24.99)	66.05 67.70 (25.51)	97.7	93.8 - 101.7
C_{max} (ng/mL)	4.25 4.39 (25.14)	4.70 4.91 (29.48)	90.4	86.3 – 94.7
T_{max}[3] (h)	4.50 (2.00- 8.00)	4.25 (2.00-12.00)		
$T_{½}$[4] (h)	13.90 (27.78)	13.98 (28.34)		

[1] Auro-Liothyronine (liothyronine as liothyronine sodium) tablets 25 mcg (Auro Pharma Inc.)
[2] CYTOMEL® (liothyronine as liothyronine sodium) tablets 25 mcg (Pfizer Canada Inc.)
[3] Expressed as the median (range) only.
[4] Expressed as arithmetic mean (% CV) only.

SUPPLY

HDPE Container: 100's

5mcg: White to off White, round biconvex tablets debossed with "LT" one side of the tablet and "5" on other side.

25mcg: White to off White, round biconvex tablets debossed with "LT25" one side of tablet and break line on other side

COMPOSITION

Each AURO-LIOTHYRONINE tablet contains liothyronine 5 mcg (present as liothyronine sodium 5.17 mcg) or 25 mcg (present as liothyronine sodium 25.8 mcg); 25 mcg of liothyronine is equivalent to approximately 65 mg of dessicated thyroid or thyroglobulin and 0.1 mg of L-thyronine.

The nonmedicinal ingredients are: Calcium Sulfate, Corn Starch, Gelatin, Magnesium Stearate and Mannitol.

REFERENCES

1. PrCYTOMEL® (liothyronine sodium) Submission control number 206002, Product Monograph, Pfizer Canada ULC. (July 25, 2017)

PART III: CONSUMER INFORMATION

IMPORTANT: PLEASE READ

[Pr]Auro-Liothyronine
(Liothyronine Sodium Tablets)

This leaflet is Part III of a three-part "Product Monograph" published when Auro-Liothyronine was approved for sale in Canada and is designed specifically for Consumers. This leaflet is a summary and will not tell you everything about Auro-Liothyronine. Contact your doctor or pharmacist if you have any questions about this drug.

ABOUT THIS MEDICATION

What the medication is used for
Auro-Liothyronine (liothyronine sodium) is used for the treatment of:
- Mild to severe hypothyroidism (thyroid gland does not produce enough hormone);
- Enlarged thyroid gland (simple goiter)

What it does
Auro-Liothyronine (liothyronine sodium) is a synthetic form of a hormone produced by the thyroid gland called triiodothyronine.

When it should not be used
Auro-Liothyronine should not be used if you have:

- a hypersensitivity or allergy to thyroid hormones (liothyronine sodium) or to any nonmedicinal ingredients in Auro-Liothyronine;
- uncorrected adrenal gland under-activity;
- acute myocardial infarction (heart attack)

What the medicinal ingredient is
Liothyronine sodium

What the non medicinal ingredients are
Calcium Sulfate, Corn Starch, Gelatin, Magnesium Stearate, Mannitol.

What dosage form it comes in
Auro-Liothyronine is available as round, white to off-white tablets.
The tablets are available in two strengths: 5 micrograms and 25 micrograms. The 5 mcg tablets are white to off white, round, biconvex tablets debossed with "LT" one side of the tablet and "5" on other side. The 25 mcg tablets are white to off white, round biconvex tablets debossed with "LT25" one side of tablet and break line on other side.

WARNINGS AND PRECAUTIONS

Serious Warnings and Precautions
Auro-Liothyronine, like other thyroid hormones, should not be used for the treatment of obesity or for weight loss, either alone or with other medicines. At normal doses, Auro-Liothyronine does not reduce body weight and at higher doses, it can produce serious or even life-threatening side effects.

BEFORE or WHILE YOU USE Auro-Liothyronine, talk to your doctor if you have the following condition:
- any heart problems, such as a heart attack, angina pectoris (chest pain)
- a history problem with thyroid (myxedema), or adrenal or pituitary gland
- kidney disease
- high blood sugar (diabetes)
- decreased development of sexual organs (hypogonadism)
- bone thinning (osteoporosis)
- pregnant or planning to become pregnant
- breast feeding
- blood clotting problem

INTERACTIONS WITH THIS MEDICATION

Before or while you use Auro-Liothyronine tell your doctor or pharmacist about all other medications you take including medications that you bought without a prescription, vitamins, and natural products. Particularly if you are taking the following:
- Blood thinner (anticoagulants)
- Medications for high cholesterol levels such as lovastatin, cholestyramine, clofibarate
- Medications for high blood sugar or insulin
- Medications that contain estrogen such as birth control pills
- Amiodarone
- Medicines that contain iodine such as radiographic contrast agents
- Heroin/methadone
- 5-fluorouracil, mitotane, tamoxifen
- Tricyclic antidepressant such as imipramine
- Digoxin (digitalis)
- Cyokines such as interferon-alpha, interleukin-2
- Growth hormones
- Ketamine
- Theophylline
- Diazepam
- Ethionamide
- Metoclopramide

- Perphanazine
- Resorcinol (topical products)
- Para-aminosalicylate sodium

Some medicines may interfere with blood tests done to determine thyroid hormone levels (thyroid function tests). It is important to inform your doctor of all medicines you are taking before and at the time of blood tests.

The following drugs are known to interfere with laboratory tests performed in patients on thyroid hormone therapy:

- Androgens (male hormones)
- Corticosteroids (eg., prednisone, dexamethasone)
- Estrogens and estrogen-containing oral contraceptives (birth control pills)
- Preparations containing iodine
- Salicylates (eg., aspirin)

PROPER USE OF THIS MEDICATION

You should take Auro-Liothyronine as prescribed by your doctor. Treatment is usually started with a low dose of Auro-Liothyronine, then increased gradually according to your response.

Overdose

If you think you, or a person you are caring for, have taken too much Auro-Liothyronine, contact a healthcare professional, hospital emergency department, or regional poison control centre immediately, even if there are no symptoms.

Missed dose

If you miss a dose, take it as soon as you remember on the same day. Skip that dose if you do not remember until the next day.

SIDE EFFECTS AND WHAT TO DO ABOUT THEM

Side effects with Auro-Liothyronine may include symptoms of hyperthyroidism as follows: Headache, excessive sweating, fatigue, increased appetite, weight loss, fever, nervousness, anxiety, irritability, insomnia, cardiac arrhythmias (heartbeats which are irregular or too fast), angina pectoris (chest pain), diarrhea and menstrual irregularities.

Tell your doctor about any other side effect that is not included above or if you have any question. Your doctor may ask you stop taking Auro-Liothyronine until the symptoms disappear, and re-start after one or two days at a lower dose.

HOW TO STORE IT

Store between 15° and 30° C (59° and 86°F)
The product should be stored in the original container.
Keep away from light and moisture.
Keep container firmly closed and away from the reach and sight of children**.**

Reporting Side Effects

You can report any suspected side effects associated with the use of health products to Health Canada by:

- Visiting the Web page on Adverse Reaction Reporting (https://www.canada.ca/en/health-canada/services/drugs-health-products/medeffect-canada/adverse-reaction-reporting.html) for information on how to report online, by mail or by fax; or
- Calling toll-free at 1-866-234-2345.

NOTE: Contact your health professional if you need information about how to manage your side effects. The Canada Vigilance Program does not provide medical advice.

MORE INFORMATION

- Talk to your healthcare professional
- Find the full Product Monograph that is prepared for healthcare professionals and includes this Consumer Information by visiting the Health Canada website (https://www.canada.ca/en/health-canada/services/drugs-health-products/drug-products/drug-product-database.html); the manufacturer's Website http://www.auropharma.ca, or by calling 1-855-648-6681.

This leaflet was prepared by Auro Pharma Inc.
Last revised: JUL 17, 2024