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بالرسالة صفحات

لم ترد بالأصل

**THE EVALUATION OF THE EFFECT OF
THYROID HORMONES ON SERUM LEPTIN
LEVEL**

THESIS

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INTRODUCTION

INTRODUCTION

Thyroid Gland

The thyroid gland synthesizes, stores, and secretes thyroid hormones, which are necessary for normal growth and development, and plays a vital role in numerous diverse metabolic processes. The gland is not essential for life, but in its absence, there is poor resistance to cold, mental and physical slowing, and in children, mental retardation and dwarfism. Conversely, excess thyroid hormone secretion leads to body wasting, nervousness, tachycardia, tremors, and heat production.⁽¹⁾

Thyroid function is controlled by the thyroid-stimulating hormone (TSH, thyrotropin) of the anterior pituitary, the secretion of this tropic hormone is in turn regulated in part by a direct inhibitory feedback of high circulating thyroid hormone levels on the pituitary and in part via neural mechanisms operating through the hypothalamus. In this way, changes in the internal and external environment bring about appropriate adjustments in the rate of thyroid secretions.⁽¹⁾

The thyroid gland secretes thyroxine mainly (T₄) as well as smaller amounts of tri-iodothyronine (T₃), both of which modulate energy utilization and heat production and facilitate growth.⁽²⁾

Dietary iodine is essential for synthesis of thyroid hormones. The recommended daily iodine intake is 150 $\mu\text{g/day}$. Iodine is converted to iodide in the stomach; after rapid absorption from the gastrointestinal tract, iodide is distributed in the extracellular fluids. The thyroid follicular cells actively transport iodide from the blood stream across the follicular cell basement membrane. The trapped iodide is enzymatically oxidized by thyroid peroxidase; thyroid peroxidase also mediates the iodination of the tyrosine residues in thyroglobulin to form moniodotyrosine (MIT) and di-iodotyrosine (DIT). The iodotyrosine molecules couple to form thyroxine (3, 5, 3', 5'-tetraiodothyronine) or tri-iodothyronine (3, 5, 3'-tri-iodothyronine). Once iodinated, thyroglobulin containing newly formed T4 and T3 is stored in the follicles. Secretion of free T4 and T3 into the circulation occurs after proteolytic digestion of thyroglobulin. Thyroid hormone secretion is stimulated by thyroid stimulating hormone (TSH).⁽²⁾

The hypothalamic thyrotropin-releasing hormone (TRH) is transported via the hypothalamic hypophyseal portal system to the anterior pituitary gland where it binds to thyrotroph receptors and stimulates the synthesis and release of TSH. TSH in turn increases thyroidal iodide uptake and iodination of thyroglobulin, it also releases T3, and T4 from the thyroid gland by increasing hydrolysis of thyroglobulin. TSH also stimulates thyroid cell growth. Excess secretion

of TSH overtimes results in thyroid enlargement (Goiter). TRH and TSH release are under negative feed back inhibition by circulating levels of T4 and T3.⁽¹⁾

Thyroxine and T3 are highly bound to carrier proteins in the serum. The unbound or free fractions are the biologically active fractions and represent only 0.04% of the total T4 and 0.4% of the total T3. The three major proteins that transport thyroid hormones are thyroxine binding globulin (TBG) thyroxine binding prealbumin (TBPA) and albumin.⁽²⁾

Approximately 40% of the secreted T4 is deiodinated by the liver and other peripheral tissues to yield T3, and about 45% is deiodinated to yield reverse T3 (rT3). Therefore, with a normal T4 production of 100 nmol (80µg) daily, approximately 40 nmol of T3 and 45 nmol of rT3 would be produced by peripheral deiodination. Form the estimated daily production rates for T3 (30 µg) and rT3 (30 µg), it is evident that at least 85% of normal T3 production and essentially all of rT3 production can be accounted for by peripheral deiodination of T4 rather than by direct secretion by the thyroid gland (figure I).⁽³⁾

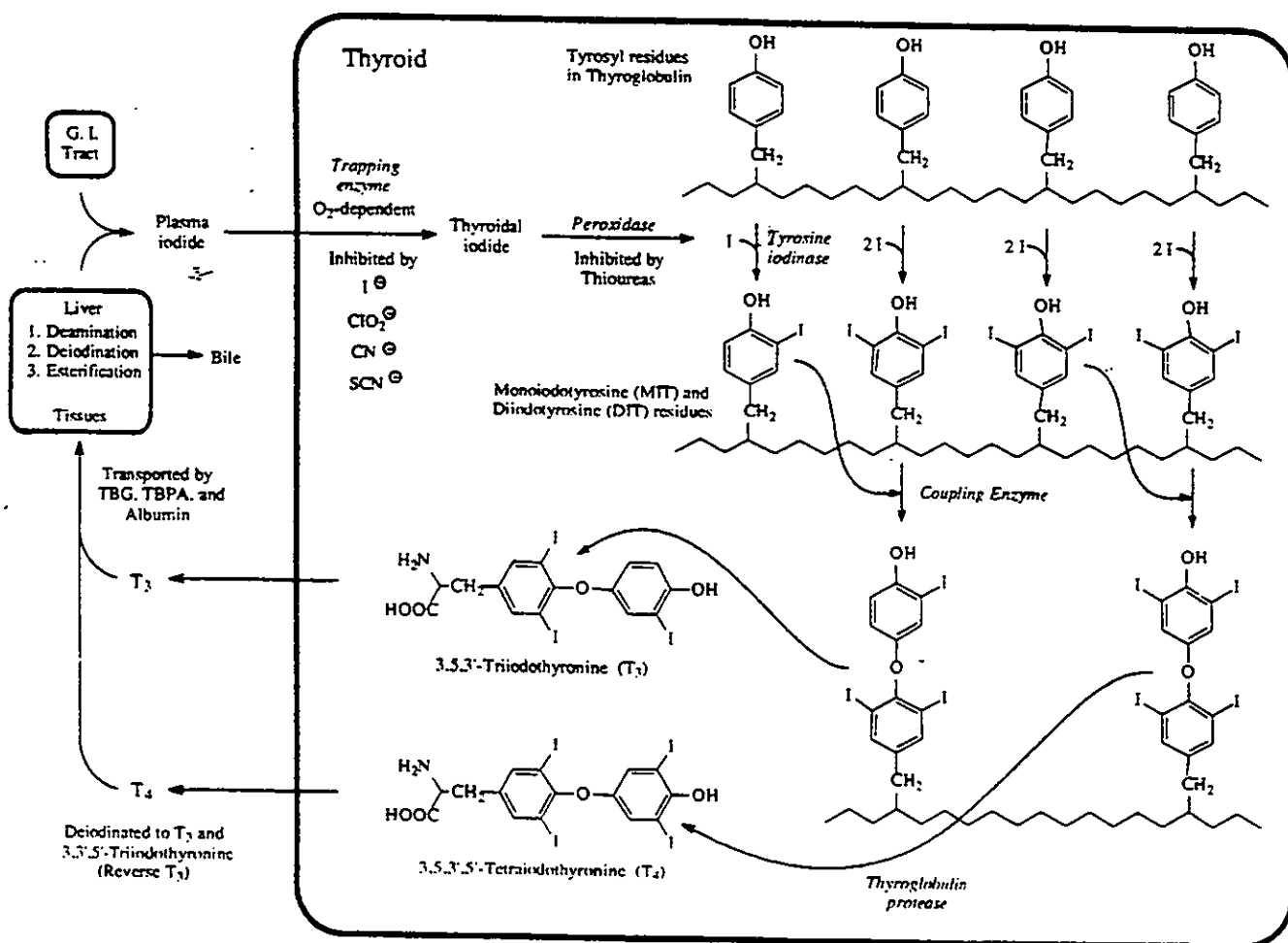


Figure 1: The metabolism of iodine, emphasizing formation and secretion of the thyroid hormones. U indicates block in the pathway. Iodine transport is inhibited by anions such as thiocyanate (SCN^-), perchlorate (ClO_4^-), and pertechnetate (TcO_4^-). The oxidation and organic binding of iodide to thyroglobulin is blocked by thiourenes, sulfonamides, and high concentrations of iodide.⁽³⁾

The physiologic effects of thyroid hormones are summarized in the following table.⁽²⁾

1. Cardiovascular effects	Increased heart rate and cardiac output
2. Gastrointestinal effects	Increased gut motility
3. Skeletal effects	Increased bone turnover and resorption
4. Pulmonary effects	Maintenance of normal hypoxic and hypercapnic drive in the respiratory center
5. Neuromuscular effects	Increased muscle protein turnover and increased speed of muscle contraction and relaxation
6. Lipids and carbohydrate metabolism effects	Increased hepatic gluconeogenesis and glycogenolysis as well as intestinal glucose absorption; increased cholesterol synthesis and degradation, increased lipolysis
7. Sympathetic nervous system effects	Increased numbers of beta-adrenergic receptors in the heart, skeletal muscle, lymphocytes, and adipose cells. Decreased cardiac alpha-adrenergic receptors, increased catecholamine sensitivity.
8. Hemopoietic effects	Increased red blood cell 2, 3 diphosphoglycerate, facilitating oxygen dissociation from hemoglobin with increased oxygen available to tissues.
9. Calorigenic effect	Increased oxygen consumption of almost all metabolically active tissues except brain, testes, uterus, lymph nodes, spleen, and anterior pituitary.
10. Effects on growth and development	Essential for normal growth and skeletal maturation. Potentiates the effect of growth hormone on the tissues