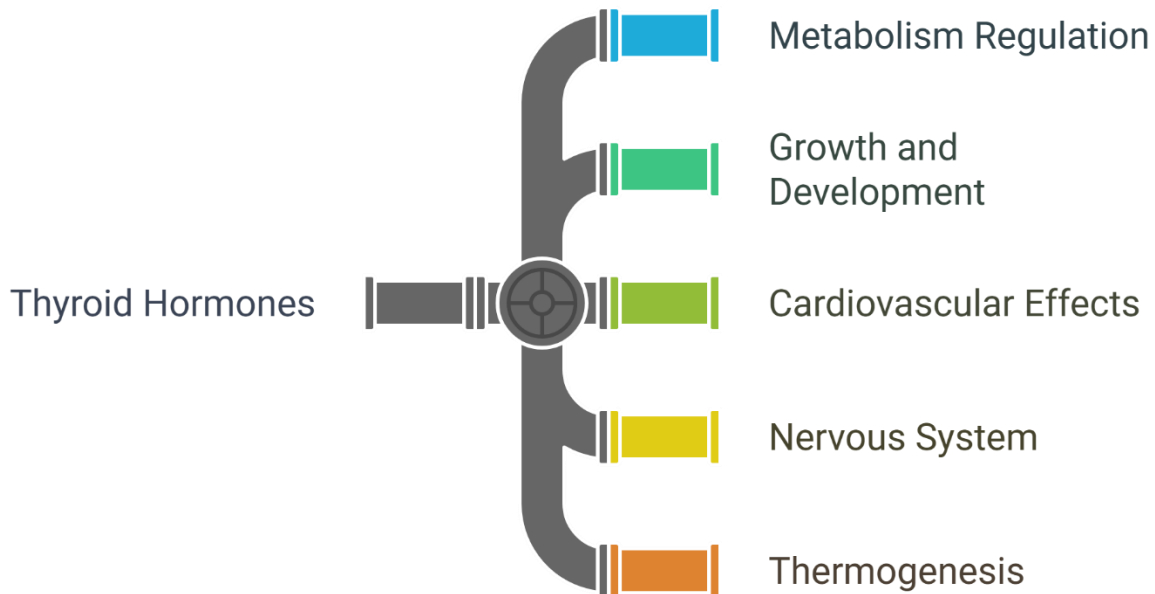


Hormones and Hormone Antagonists

Thyroid Hormones

Physiological Role:

- The two primary thyroid hormones are **T3 (triiodothyronine)** and **T4 (thyroxine)**, produced by the thyroid gland under the regulation of **TSH (thyroid-stimulating hormone)** from the pituitary.



1. Metabolism Regulation:

- Increase basal metabolic rate (BMR)
- Stimulate mitochondrial activity, oxygen consumption, and ATP production
- Promote glucose absorption, glycogenolysis, gluconeogenesis, lipolysis, and cholesterol metabolism

2. Growth and Development:

- Crucial for fetal brain development and skeletal growth
- Synergize with growth hormone

3. Cardiovascular Effects:

- Increase heart rate, cardiac output, and myocardial contractility by enhancing beta-adrenergic receptor expression

4. Nervous System:

- Influence mood, memory, reflexes, and sensory systems

- Essential for myelination in infants

5. **Thermogenesis:**

- Increase heat production via stimulation of Na^+/K^+ ATPase and mitochondrial uncoupling proteins

Pathological Role:

1. **Hyperthyroidism (Thyrotoxicosis):**

- Elevated T3/T4 levels → weight loss, heat intolerance, tachycardia, anxiety, tremors, exophthalmos (in Graves' disease)
- **Causes:** Graves' disease (autoimmune), toxic multinodular goiter, thyroid adenoma

2. **Hypothyroidism:**

- Low T3/T4 levels → fatigue, weight gain, cold intolerance, bradycardia, constipation, depression
- **Causes:** Hashimoto's thyroiditis (autoimmune), iodine deficiency, post-thyroidectomy

Clinical Uses:

- **Levothyroxine (T4):** Standard therapy for hypothyroidism
- **Liothyronine (T3):** Used in certain cases like myxedema coma
- **Thyroid hormone suppression therapy:** To suppress TSH in thyroid cancer patients or benign nodular thyroid disease

Anti-thyroid Drugs

Physiological Mechanism (Mode of Action):

Anti-thyroid drugs inhibit the synthesis or function of thyroid hormones.

1. **Thionamides:**

- **Methimazole, Propylthiouracil (PTU)**
- Inhibit thyroid peroxidase → block oxidation of iodide, iodination of tyrosine, and coupling of iodotyrosines → decreased T3 and T4 synthesis
- PTU also inhibits peripheral conversion of T4 to T3

2. **Iodide (Lugol's solution):**

- High doses inhibit thyroid hormone release (Wolff–Chaikoff effect)

3. **Radioactive Iodine (I-131):**

- Accumulates in thyroid → emits beta particles → gland ablation

4. **Beta-blockers (e.g., Propranolol):**

- Control symptoms (palpitations, tremors)
- Propranolol also blocks peripheral conversion of T4 to T3

Pathological Context (Clinical Indications):

- **Graves' Disease:** Autoimmune hyperthyroidism
- **Toxic Nodular Goiter**
- **Thyroid Storm (thyrotoxic crisis):** PTU is preferred for rapid T3 inhibition
- **Preoperative Preparation:** To render patient euthyroid before thyroidectomy

Clinical Uses and Considerations:

- **Methimazole:** Preferred due to once-daily dosing and less hepatotoxicity
Contraindicated in first trimester of pregnancy
- **PTU:** Preferred in 1st trimester of pregnancy and thyroid storm
Risk of hepatotoxicity
- **Adverse Effects:** Agranulocytosis, rash, hepatotoxicity (more with PTU)
- **Iodine and I-131:** Used for long-term treatment or ablation in resistant cases

Parathormone (Parathyroid Hormone - PTH)

Physiological Role:

Produced by the **chief cells of parathyroid glands**, PTH plays a crucial role in **calcium and phosphate homeostasis**.

1. **Bone:**

- Stimulates osteoblasts to produce RANKL → activates osteoclasts → bone resorption → increases serum calcium

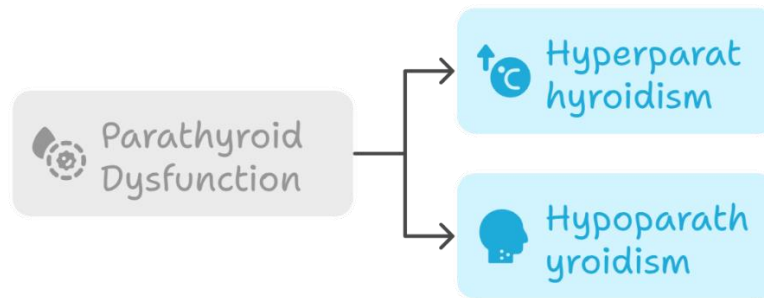
2. **Kidney:**

- Increases calcium reabsorption (distal tubules)
- Decreases phosphate reabsorption (proximal tubules) → phosphaturia
- Stimulates renal 1-alpha hydroxylase → converts 25(OH)D to active 1,25(OH)₂D (calcitriol)

3. **GI Tract:**

- Indirectly increases calcium and phosphate absorption via increased calcitriol

Pathological Role:



1. Hyperparathyroidism:

- **Primary** (adenoma): $\uparrow$ PTH, $\uparrow$ Ca²⁺, $\downarrow$ PO₄³⁻ → bone pain, kidney stones, GI disturbances, neuropsychiatric symptoms ("bones, stones, groans, and moans")
- **Secondary** (chronic kidney disease): $\uparrow$ PTH due to hypocalcemia and hyperphosphatemia
- **Tertiary**: Autonomous PTH secretion after prolonged secondary hyperparathyroidism

2. Hypoparathyroidism:

- $\downarrow$ PTH → hypocalcemia → tetany, seizures, Chvostek and Trousseau signs
- Causes: post-surgical, autoimmune, DiGeorge syndrome

Clinical Uses:

- **Teriparatide**: Recombinant PTH analog used in **osteoporosis** (intermittent dosing increases bone formation)
- Monitoring of PTH used in chronic kidney disease and during parathyroidectomy

Calcitonin

Physiological Role:

- Produced by **parafollicular (C cells)** of the thyroid gland
- **Counter-regulatory hormone** to PTH
- **Functions:**
 - Inhibits osteoclast activity → decreases bone resorption
 - Increases renal excretion of calcium and phosphate
- **Overall effect**: Lowers plasma calcium levels (minor role in humans compared to PTH and Vitamin D)

Pathological Role:

- **Medullary Thyroid Carcinoma:** Tumor of parafollicular C cells → high calcitonin levels (used as tumor marker)
- **Not commonly involved in calcium homeostasis disorders**

Clinical Uses:

1. Salmon Calcitonin (synthetic):

- Used in **Paget's disease**, **osteoporosis**, and **hypercalcemia of malignancy**
- Administered via nasal spray or injection

2. Limitations:

- Tachyphylaxis (reduced effect with prolonged use)
- Less potent than bisphosphonates
- Side effects: Nausea, flushing, nasal irritation (nasal spray)

Vitamin D

Physiological Role:

Vitamin D is a **fat-soluble prohormone** that plays a central role in **calcium and phosphate metabolism**.

Forms:

- **D2 (ergocalciferol)** – from plants
- **D3 (cholecalciferol)** – synthesized in skin from 7-dehydrocholesterol under UVB sunlight

Activation Process:

1. **Liver:** Cholecalciferol → 25-hydroxyvitamin D (25(OH)D) [inactive storage form]
2. **Kidney:** 25(OH)D → 1,25-dihydroxyvitamin D (1,25(OH)₂D / Calcitriol) by **1α-hydroxylase**, stimulated by PTH

Functions of Calcitriol (Active form):

- **Intestine:** Increases absorption of **calcium and phosphate**
- **Bone:** In conjunction with PTH, mobilizes calcium when needed
- **Kidney:** Helps in calcium reabsorption (minor effect)
- **Parathyroid Glands:** Negative feedback to reduce PTH secretion

Pathological Role:

- **Deficiency:**



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