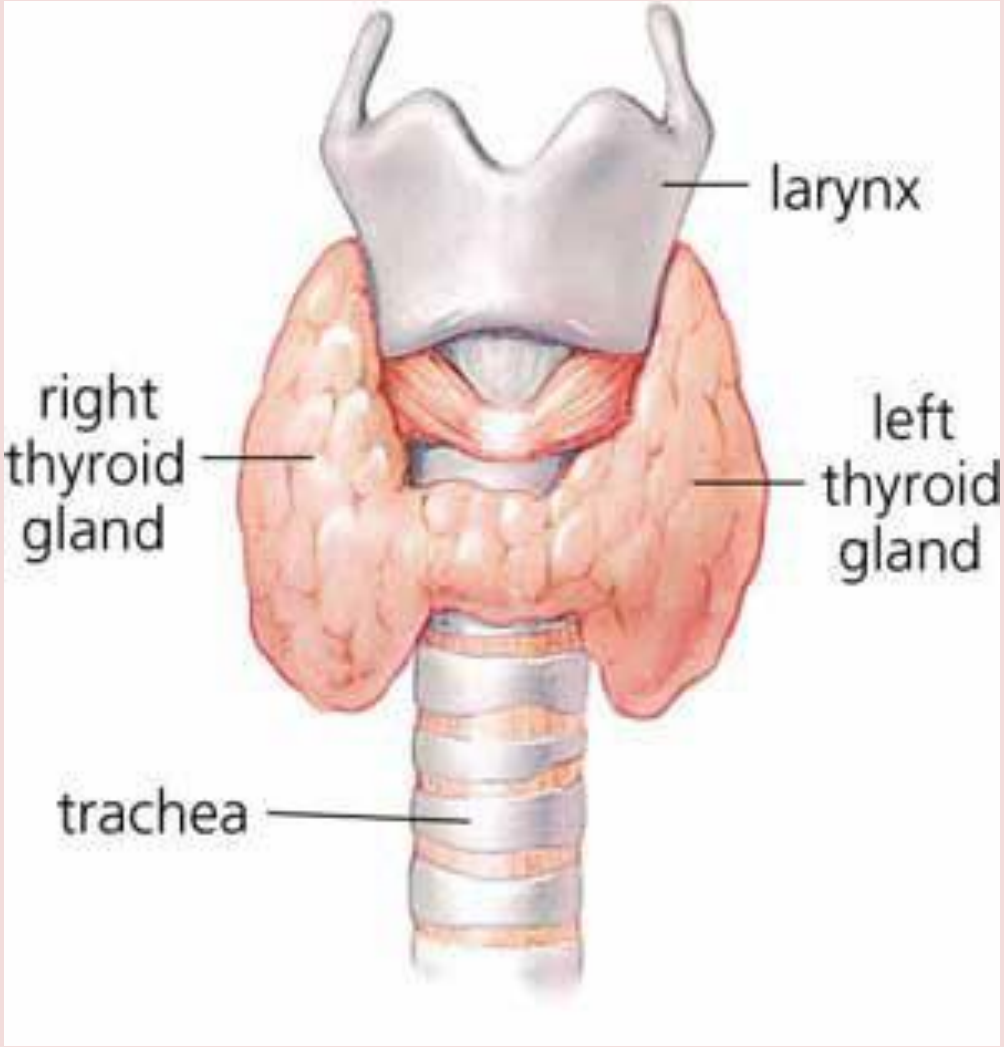
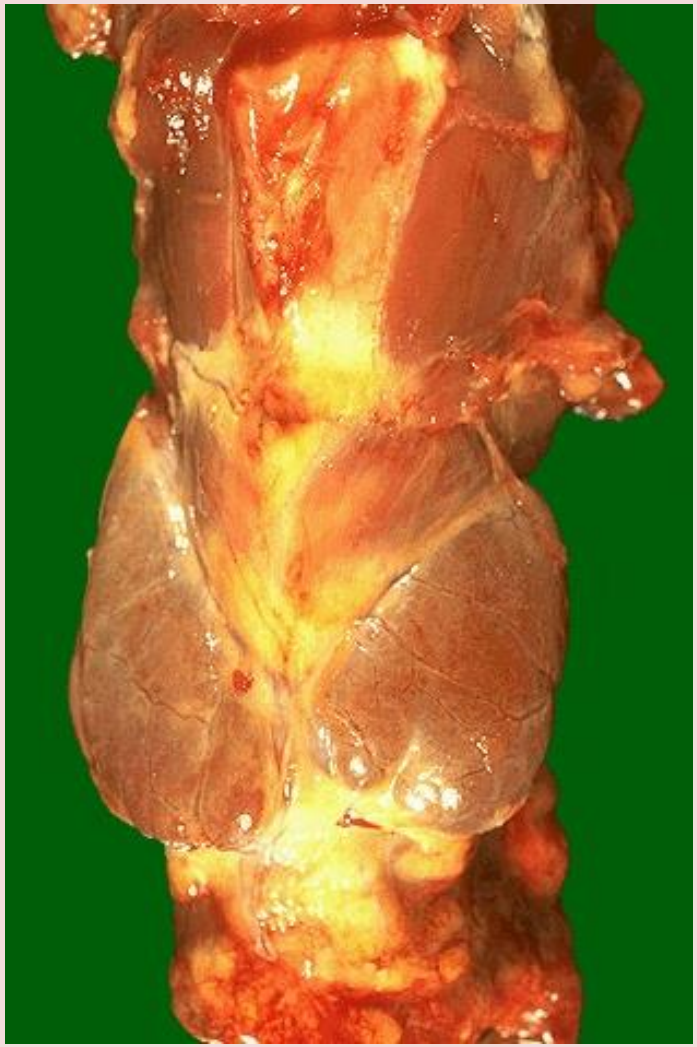
THYROID GLAND

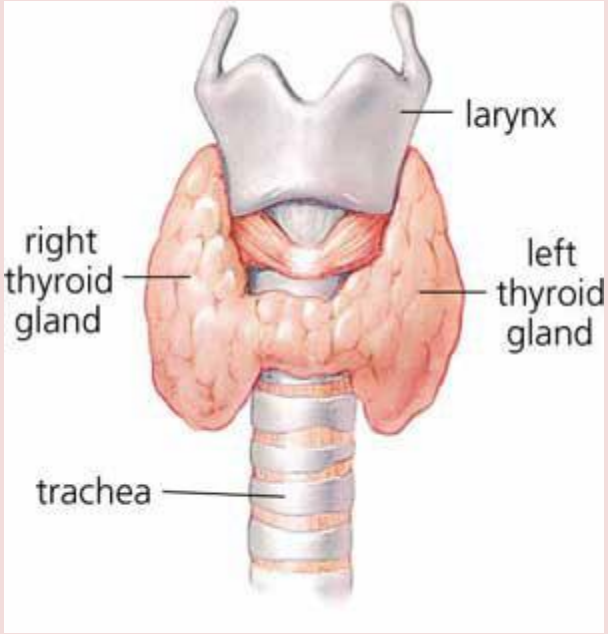




Thyroid gland

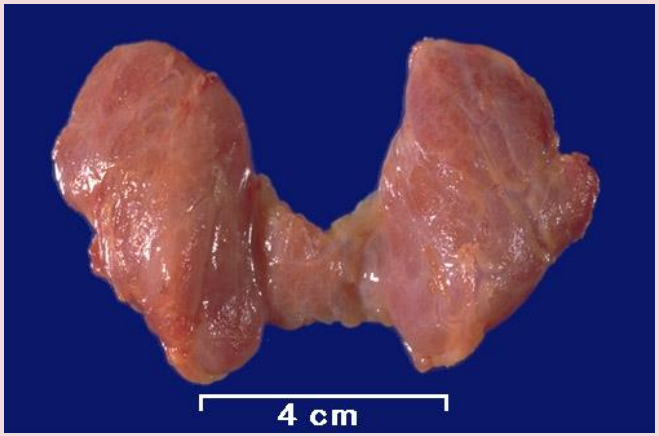
- **One of the largest endocrine glands in the body (≈ 20gms).**

- **Its size depends on:**
  1. **age …increase age α ↑ size.**
  2. **sex … female > male.**
  3. **physiological condition … (pregnancy, lactation)**

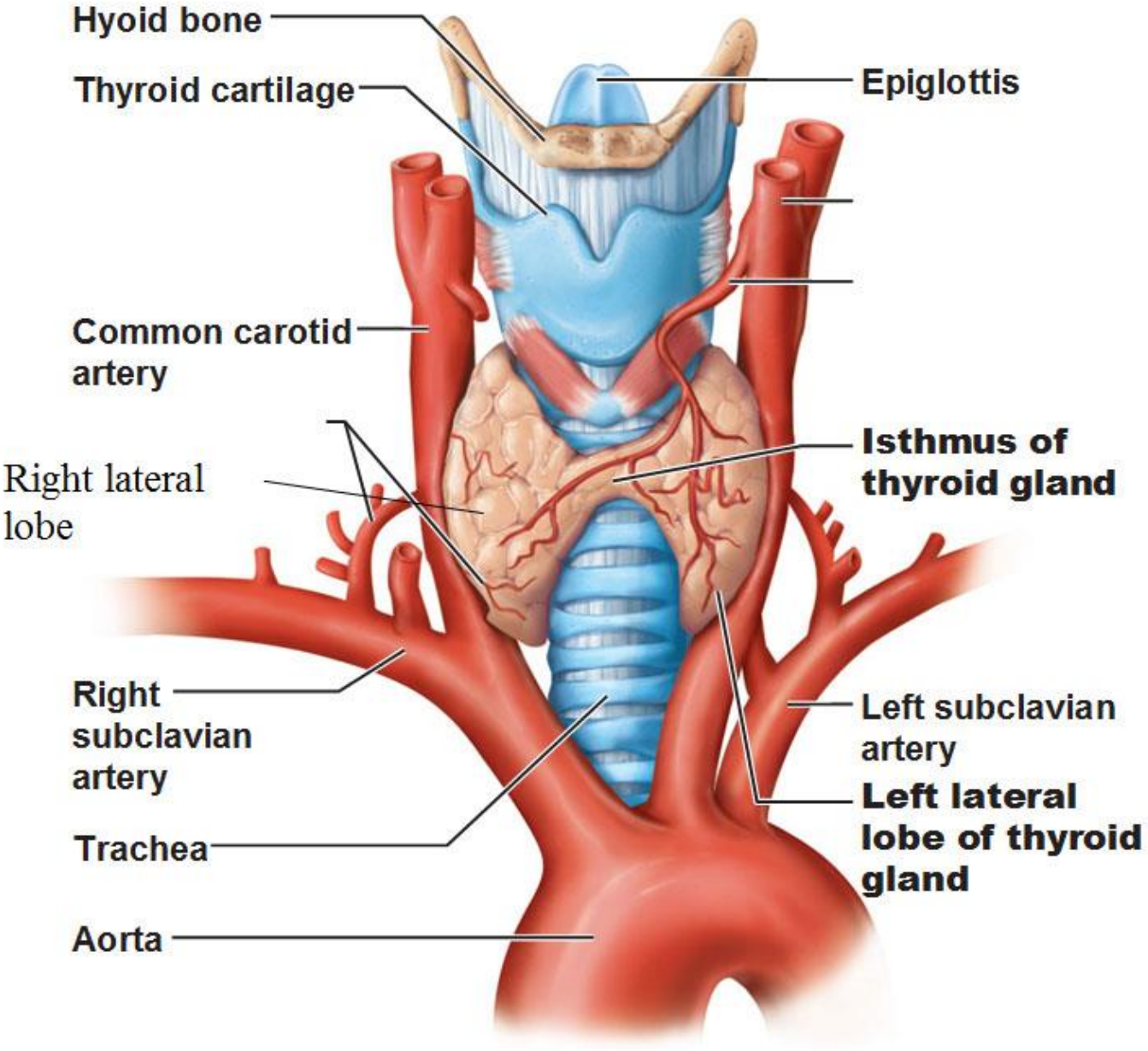


- **Site:**

Located in the neck just below the larynx, on either side of & anterior to the trachea, joined by isthmus near 2nd, 3rd and 4th tracheal ring.



The Thyroid Gland



Gross anatomy of the thyroid gland, anterior view

- Thyroid gland is well vascularized (rich in blood supply).

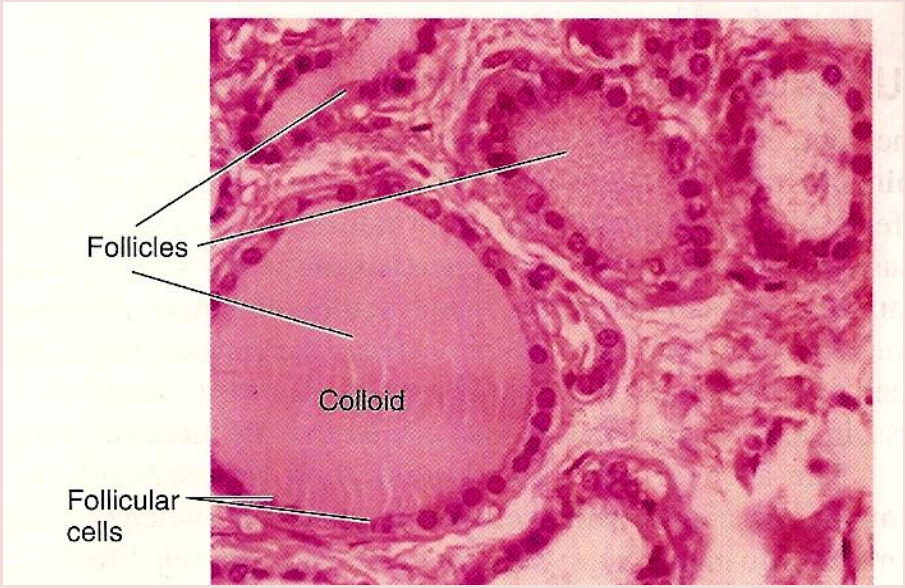
- **Embryologically:**

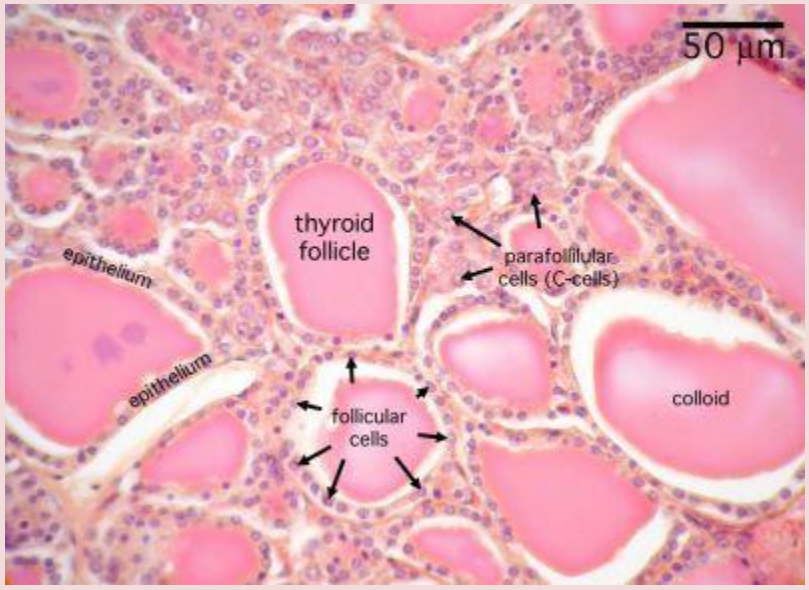
  Originates from an envagination of the floor of the pharynx.

- Formed of 2 lobes (Rt & Lt), that are connected by band of tissue called “isthmus”.

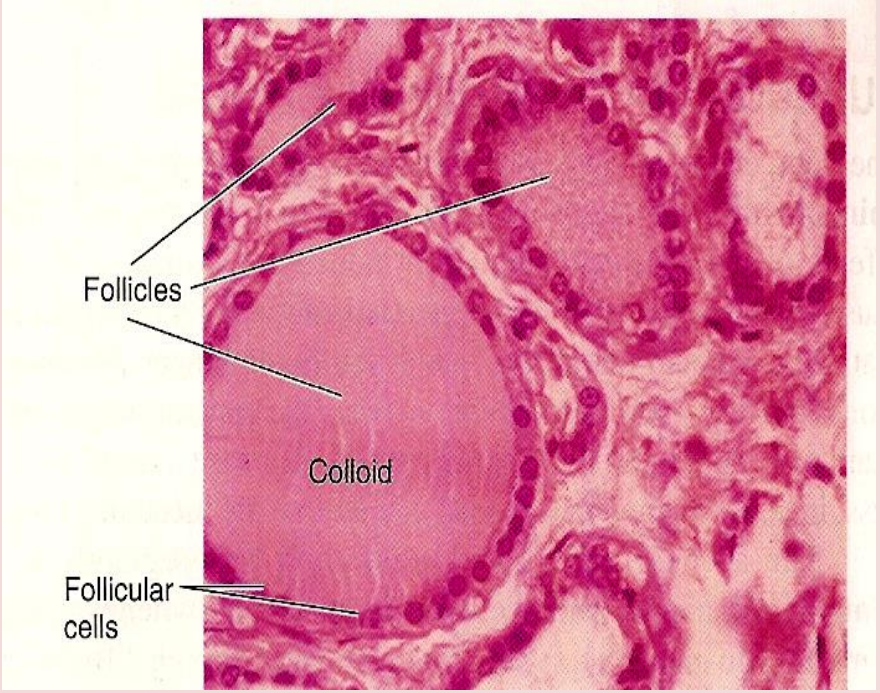
Histology

1. **Multiple Follicles (Acini):**
   - ♦ **Follicular vesicles are the functional unit.**
   - ♦ **Follicles are Thousands in no.**
   - ♦ **Each follicle ≈ 100 to 300 µmeters in diameter.**
   - ♦ **Each follicle is spherical in structure. Mitochondria and Golgi apparatus is present in large Number during hyperactivity.**
   - ♦ **Follicular wall is lined with a single layer of cuboidal epithelioid cells that secrete into the interior of the follicles.**





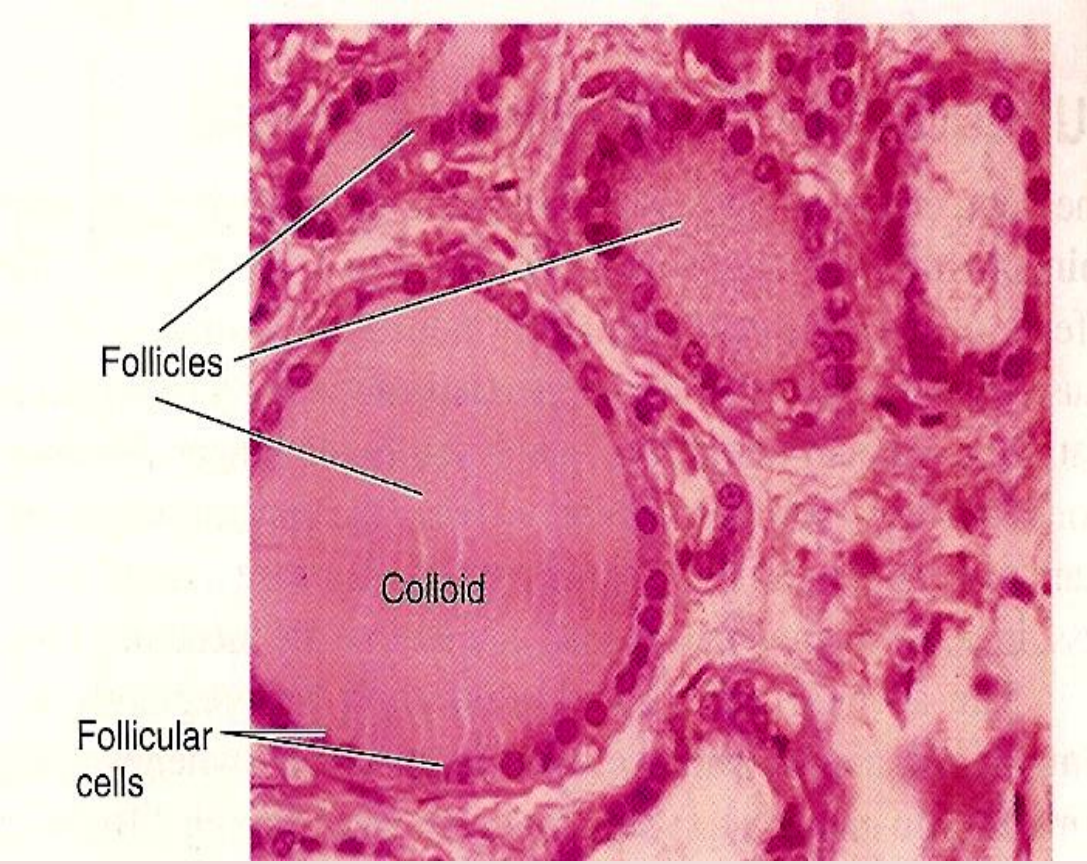
- **Each follicle is filled with pink- staining proteinaceous material called colloid.**



- **When the gland is INACTIVE:**
  colloid is abundant, follicles are large, & lining cells are flat.

- **When the gland is ACTIVE:**
  follicles are small, lining cells are cuboidal or columnar, & the edge of colloid is forming many small "reabsorption lacunae".

- **Each follicle is surrounded by a good & rich blood supply.**
- **Individual thyroid cells rest on a basal lamina that separates them from the adjacent capillaries.**
- **Prominent endoplasmic reticulum (a common feature in most glandular cells), & secretory droplets of thyroglobulins.**



Thyroid gland secretions

- **2 important thyroid hormones:**
  - **Thyroxine (T_4) or tetra-iodothyronine**
  - **Tri-iodothyronine (T_3)**

• Iodine is an essential raw material for the synthesis of thyroid hormone. The richest source of dietary iodine is sea food, but it is also occur in vegetables, milk. Iodized salt containing up to 0.05% iodine. All iodine is converted to iodite in the gut and then it is rapidly absorbed from the gastro intestinal tract into the blood.

• The follicular cells of thyroid gland synthesize the glycoprotein (thyroglobulin) which is stored as colloid in the follicle. Thyroglobulin is synthesized on the ribozomes of the RER of the follicular cells. Carbohydrate is added to protein in Golgi complex, forming glycoprotein. Then it is discharged into the follicular lumen by exocytosis.

Thyroid gland secretions

- 2 important thyroid hormones:
  - Thyroxine (T_4) or tetra-iodothyronine
  - Tri-iodothyronine (T_3)

- Secreted by **Follicular cells.**
- Can be stored in thyroid gland for couple of months (2-3 months).
- Having significant effect on increase in metabolic rate of the body.
- Thyroid hormones are produced from **inorganic iodine** and amino acid **Tyrosine**.
- Food iodine → Gut iodite → Blood iodite → Thyroid Gland → Oxidised to Iodine by peroxidase → Joins to Tyroxine to form MIT, DIT and Tetra iodothyronine
- → Combine to Thyroglobulin to form Thyroxine.

Chemical nature of thyroid hormones

- Tyrosine

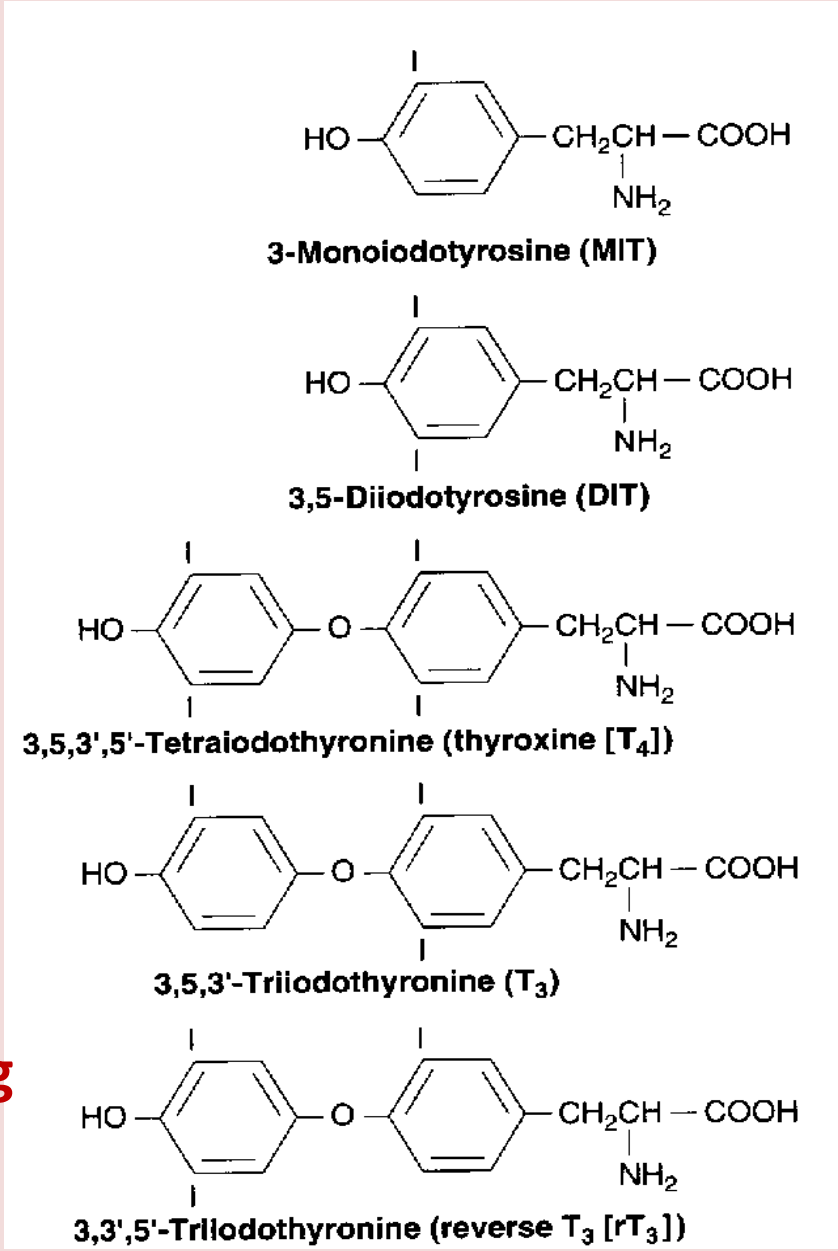
- **Monoiodotyrosine (MIT)**

- **Diiodotyrosine (DIT)**

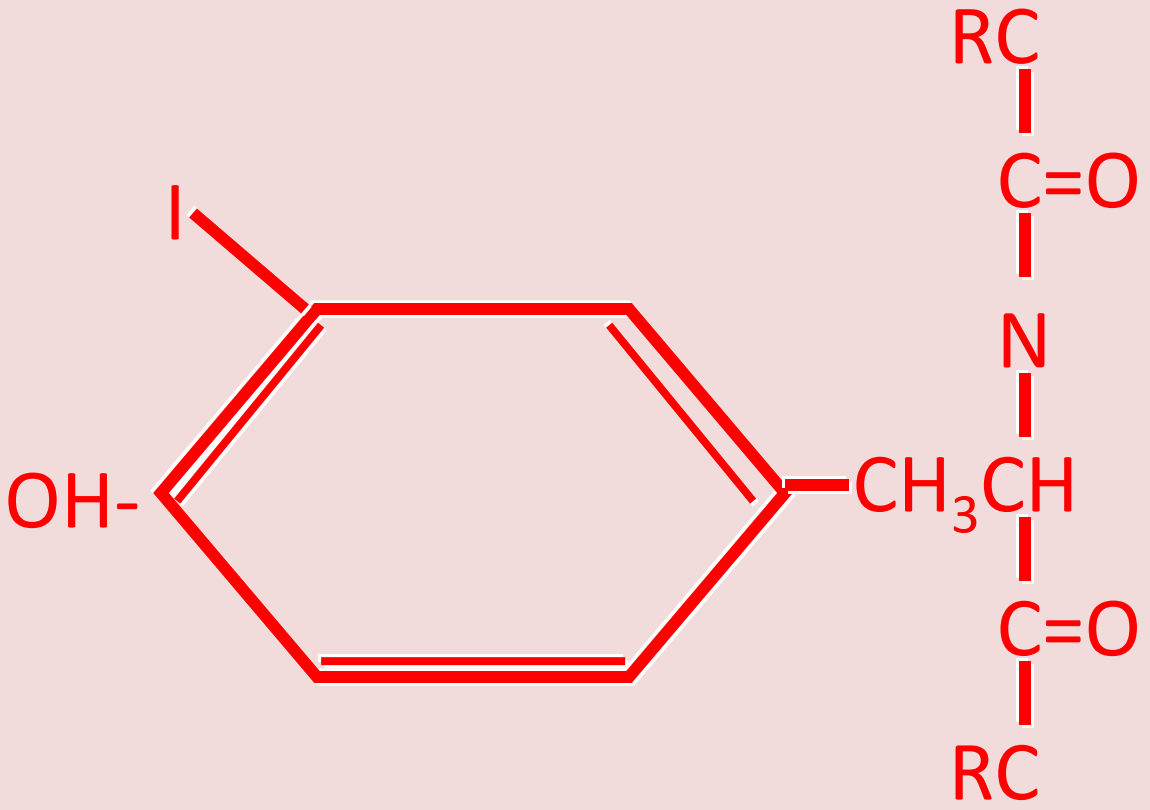
- **Tetraiodothyronine (T_4)**

- **Triiodothyronine (T_3)**

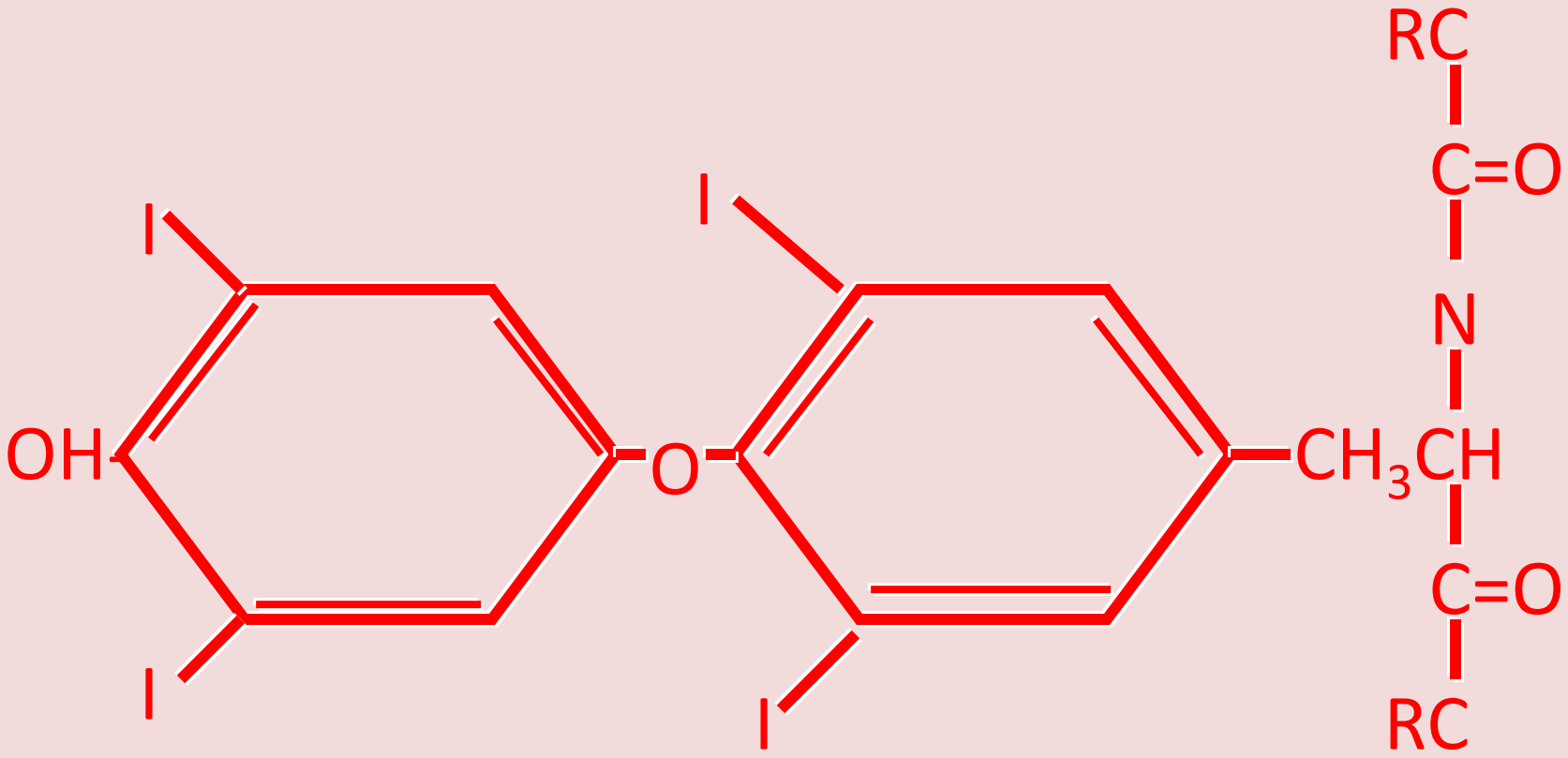
Adult thyroid follicle contains 20 µg of Thyroxine and secretes about 1 µg of Thyroxine per day.



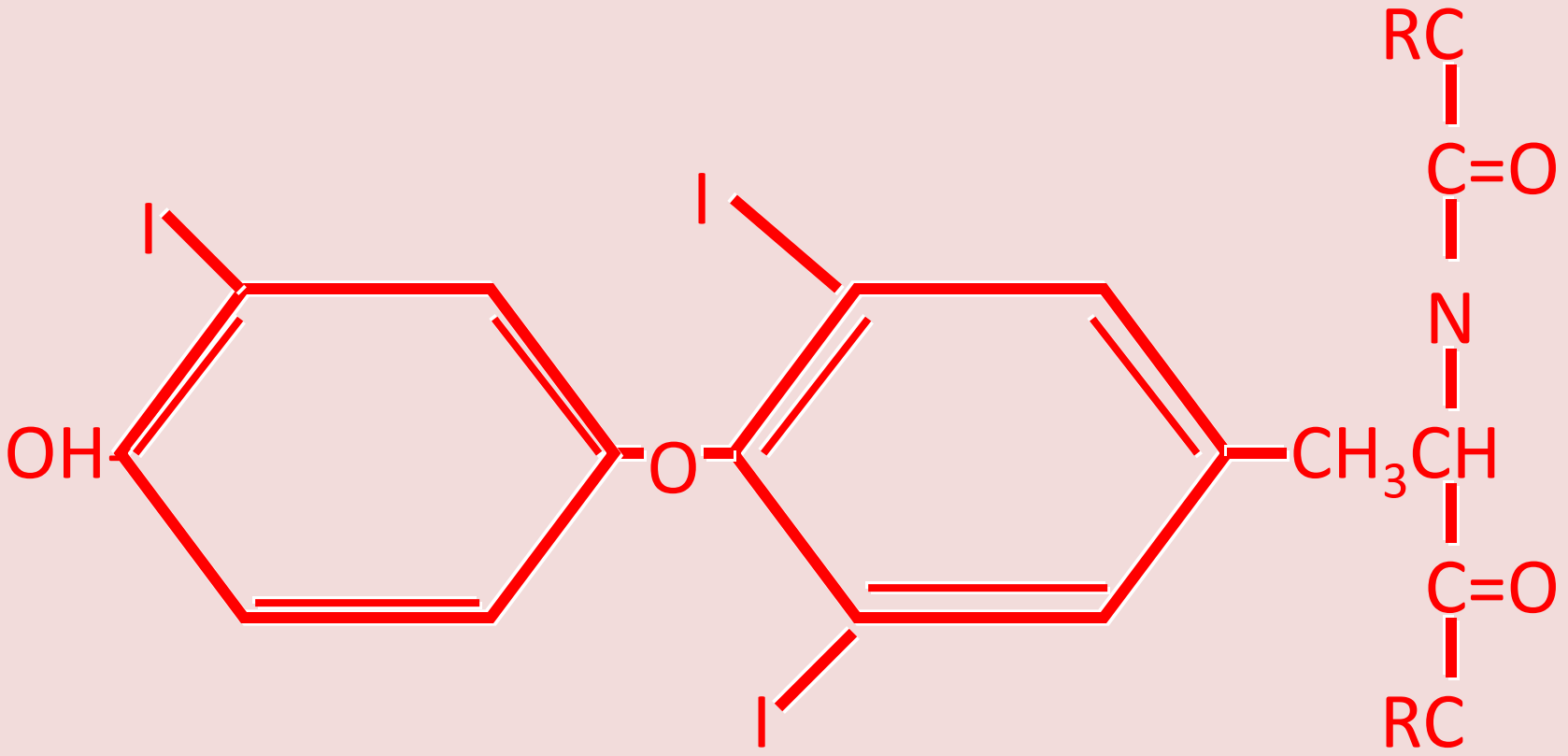
Thyroid Hormone Synthesis



- **Thyroperoxidase attaches Iodine to 3 position---MIT**
- **T3 and T4 are formed by coupling reactions. The combination of two DIT molecules produces T4 and combination of one MIT molecule with one DIT molecule produces T3. Both are in peptide linkage with thyroglobulin.**



- **Thyroperoxidase attaches ring from one DIT to adjacent DIT = Thyroxine (T_4)**



- **Thyroperoxidase attaches ring from one MIT to adjacent DIT = Triiodothyronine (T_3)**

Functions of thyroid hormones

- **Generally, THs:**

1. **Increases metabolic rate.**
   Stimulates Glucose absorption from intestine, causes hyperglycemia, Mobilises glycogen from liver, promotes gluconeogenesis, stimulates Glycogen Metabolism, Stimulates increased consumption of glucose, fatty acids and other molecules.
2. **Increases metabolic heat, by increase mitochondrial no & activity → increase in ATP,**
3. **Stimulates rate of cellular respiration by:**
   - **Production of uncoupling proteins.**
   - **Increase active transport by Na^+/K^+ pumps.**
   - **Stimulates O_2 consumption of most of cells in the body and accelerate the energy production (B.M.R.).**

Other Functions of thyroid hormones

4. Necessary for normal body growth & maturation.
5. Promotes maturation of nervous system. It controls normal emotional responsiveness. It influence the levels and activity of the central, peripheral and autonomic nervous system.
6. Stimulates RNA production and protein synthesis.
7. Help regulating lipid & Cholesterol metabolism.
8. Accelerate the Heart Beat (Action on S.A. node)
9. Help in milk output.
10. Increases nitrogen excretion, Urine volume, Increases the excretion of salt and Creatine.
11. Regulates the calcium and phosphorus metabolism.
12. It act on voluntary muscles. Hyperthyroidism may cause catabolism of muscles. Hormone shows increased activity at puberty, during menstruation, pregnancy and lactation. After menopause it tends to atrophy.

Abnormal thyroid hormones secretions

I: Hyperthyroidism (thyrotoxicosis)

- Hyperthyoidism $\rightarrow \uparrow$ THs.
- Could be:

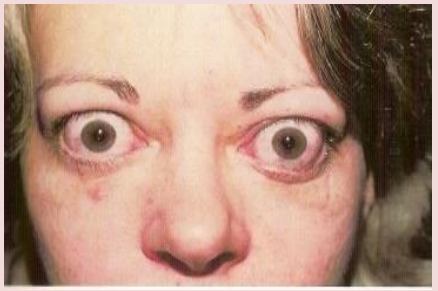
$\uparrow$ TRH $\rightarrow \uparrow$ TSH $\rightarrow \uparrow T_3$ & T_4.

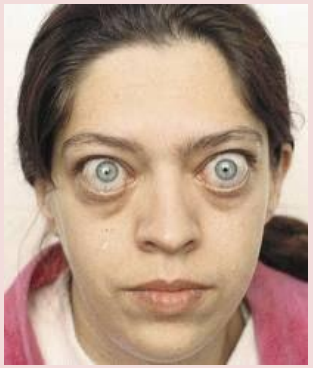
- **Follicular cells become overactive.**

- **Females > males (4:1).**

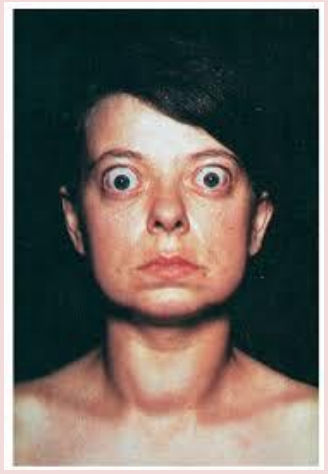
I: Hyperthyroidism … 'Grave's disease' Thyrotoxicosis

- 90% of hyperthyoidism is due to "Grave's disease"/ Toxic goitre/Thyrotoxicosis.
- GD is an autoimmune disease → ↑ thyroid stimulating antibodies IgG.
- **Symptoms of GD:**
  - **Exophthalmic eye**, due to retro-orbital oedema (deposition of fat). Protruding eyeball with a staring look.

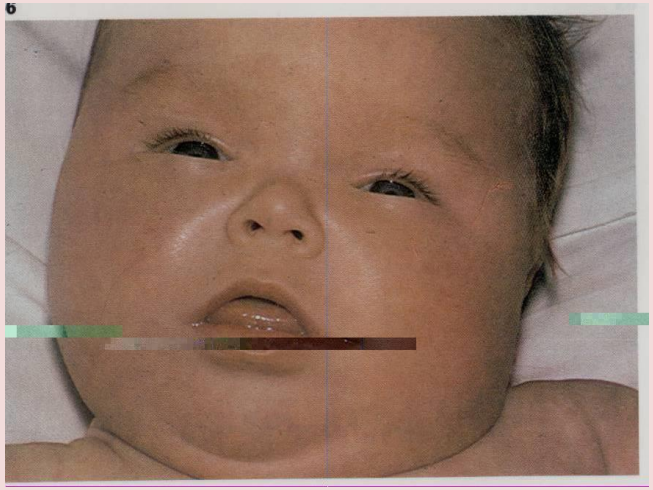




- **Lid lag**, due to weakness of extraoccular muscles.
- Anxiety & restlessness. Mentaly sharp, emotional, high blood sugar, heart rate increased, Sleeplessness.
- ↑ appetite, ↓ weight & diarrhea.
- Intolerence to heat.



Hypothyroidism: cretenism



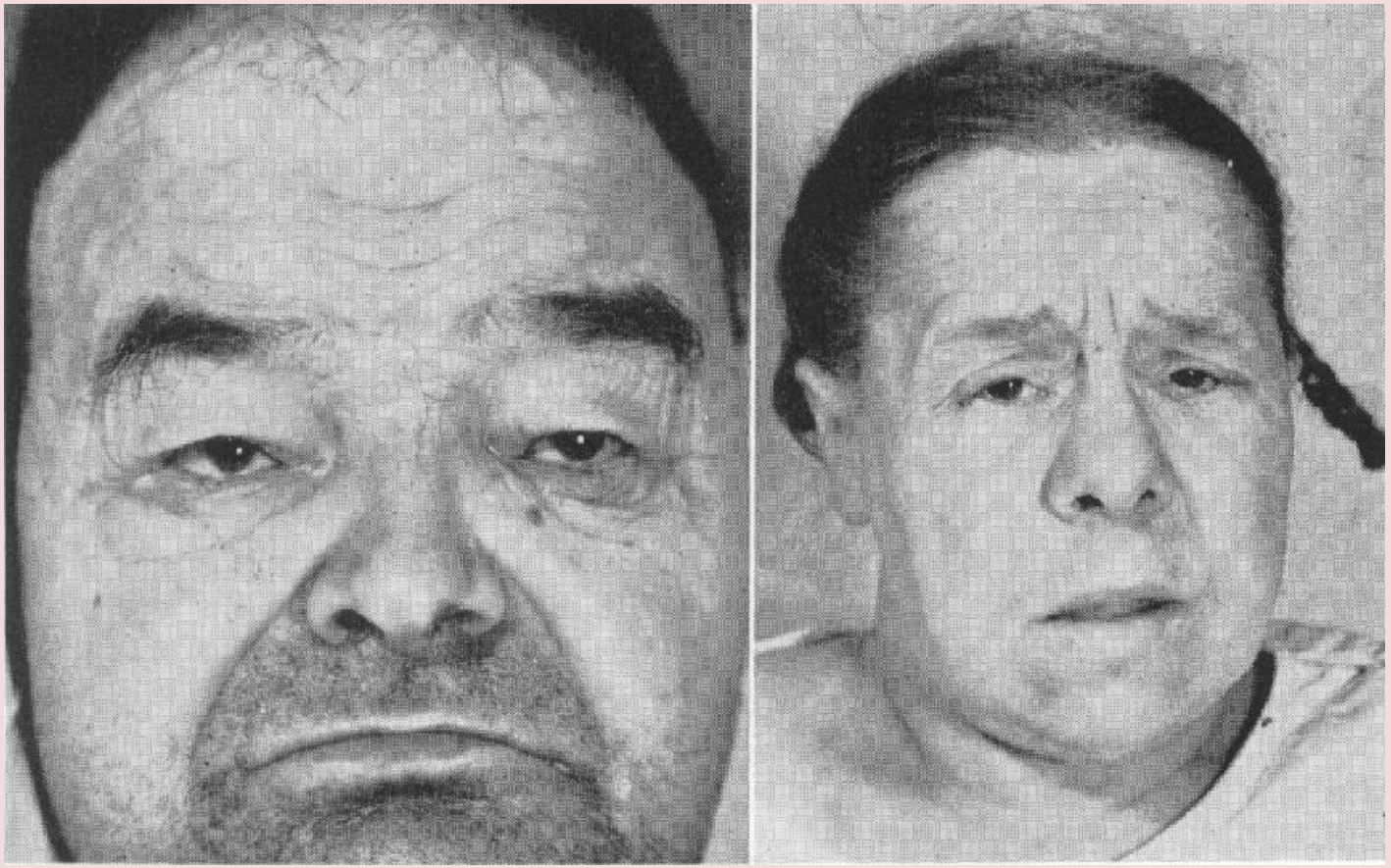
- This is uncommon disease of childhood due to failure of thyroid to synthesize thyroid hormones →
- **Newborn**
  - **Lack of myelination**
- **Children**
  - **Retarded Growth, Dentition, standing, walking, speech etc are delayed, Disproportionate body.**
  - **Stunted growth, deformed bones and teeth, rough and dry skin, idiotic look, protruding tongue, saliva dribbling, pot bellied, often deaf and dumb, low BMR and low resistance, susceptible to cold.**

Myxedema

- **Hypothyroidism in adult.**
- **- Clinically:**
  - dizziness **& subtle,** Muscle weakness, Mental slowness.
  - **lethargy & weakness with slow speech,** Impaired renal function.
  - **cold intolerance with cool & rough skin**
  - **menstrual problems & psychosis**
  - **cardiac changes: ↓ cardiac output, hypertrophy, (myxedema heart)**
  - **deposition of mucopolysaccharides in connective tissue**
  - **atherosclerosis (↑ cholesterol)**
  - **swollen & puffy appearance of body,**

    due to deposition of protein-carbohydrate complexes 'mucopolysaccharides' & fluid in subcutaneous tissue.

Myxedema



Goitre:

It is non-inflammatory enlargement of thyroid gland. In simple goitre usually with no hypofunction or hyperfunction of the gland. The simple goitre may be of the following types.

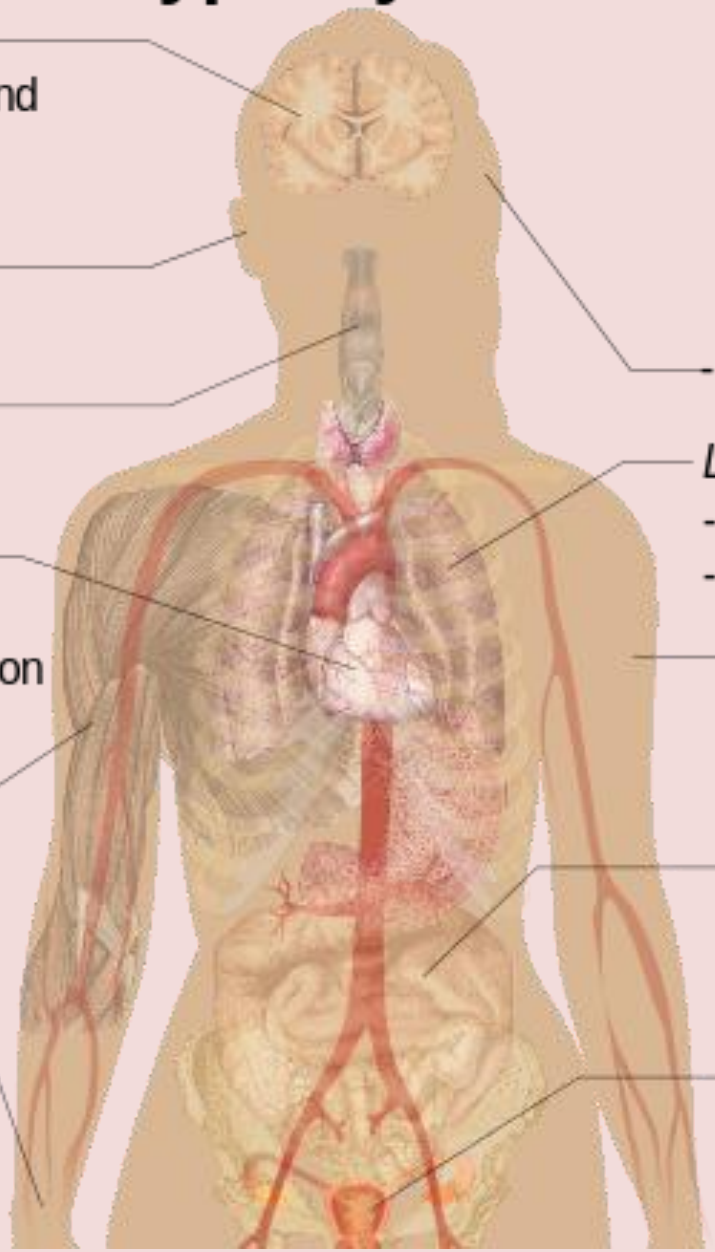
1. Colloid goitre: It is a deficiency disease caused by an inadequate supply of iodine in diet. Thevesicles are distended with cuboidal epithelial cells. There is no hypertrophy (increase in muscles).

2. Diffuse parenchymatous goitre: there is hypertrophy and multiplication of epithelial cells.

3. Hokkaido goitre: very high concentration of iodine may also causes hypothyroidism by inhibiting iodine organification. In hokkaido, the Japanese people consume large amount of seaweed providing 8 to 25 mg iodine per day. This interferes the release of T3 and T4 from the thyroid gland.

Signs and symptoms of

Hypothyroidism



Psychological
- Poor memory and concentration

- Poor hearing

Pharynx
- Hoarseness

Heart
- Slow pulse rate
- Pericardial effusion

Muscular
- Delayed reflex relaxation

Extremities
- Coldness
- Carpal tunnel syndrome

General
- Fatigue
- Feeling cold
- Weight gain with poor appetite

- Hair loss

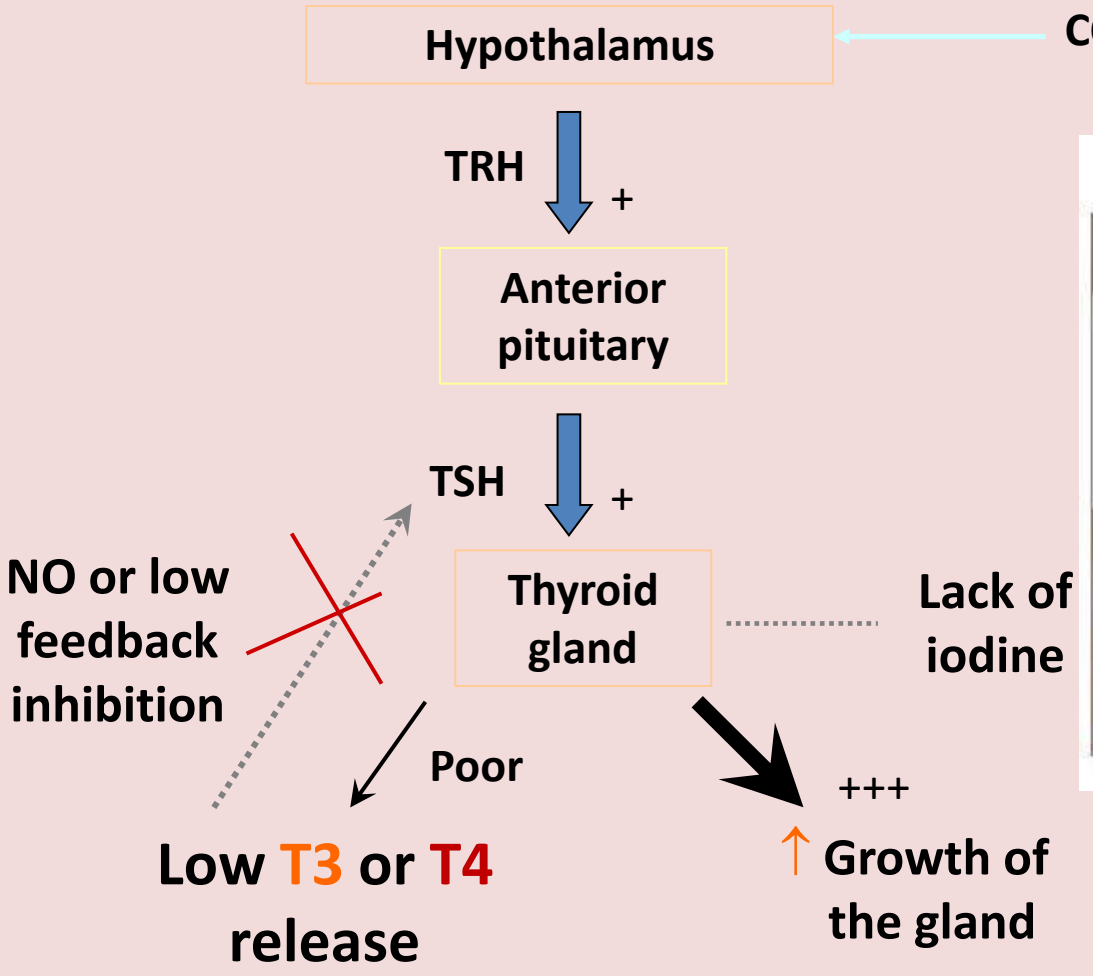
Lungs
- Shortness of breath
- Pleural effusion

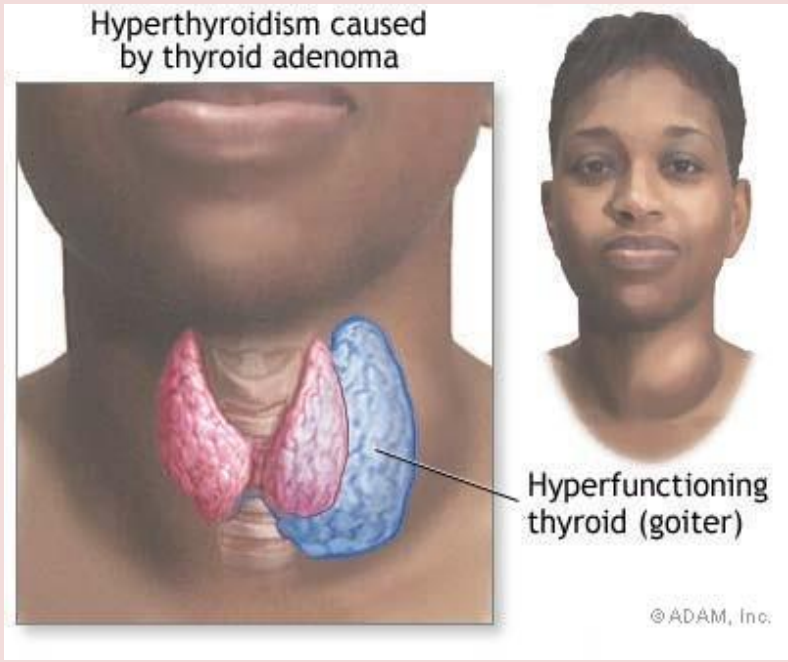
Skin
- Paresthesia
- Myxedema

Intestines
- Constipation
- Ascites

Reproductive system
- Menorrhagia

How goiter ‘swollen neck’ is formed? With lack of iodine





• Thyrocalcitonin

-Thyrocacitonin Secreted by **Parafollicular cells.**

-- Regulate the Blood Calcium and phosphorus level, antagonistic to that of parathyroid hormone.

-

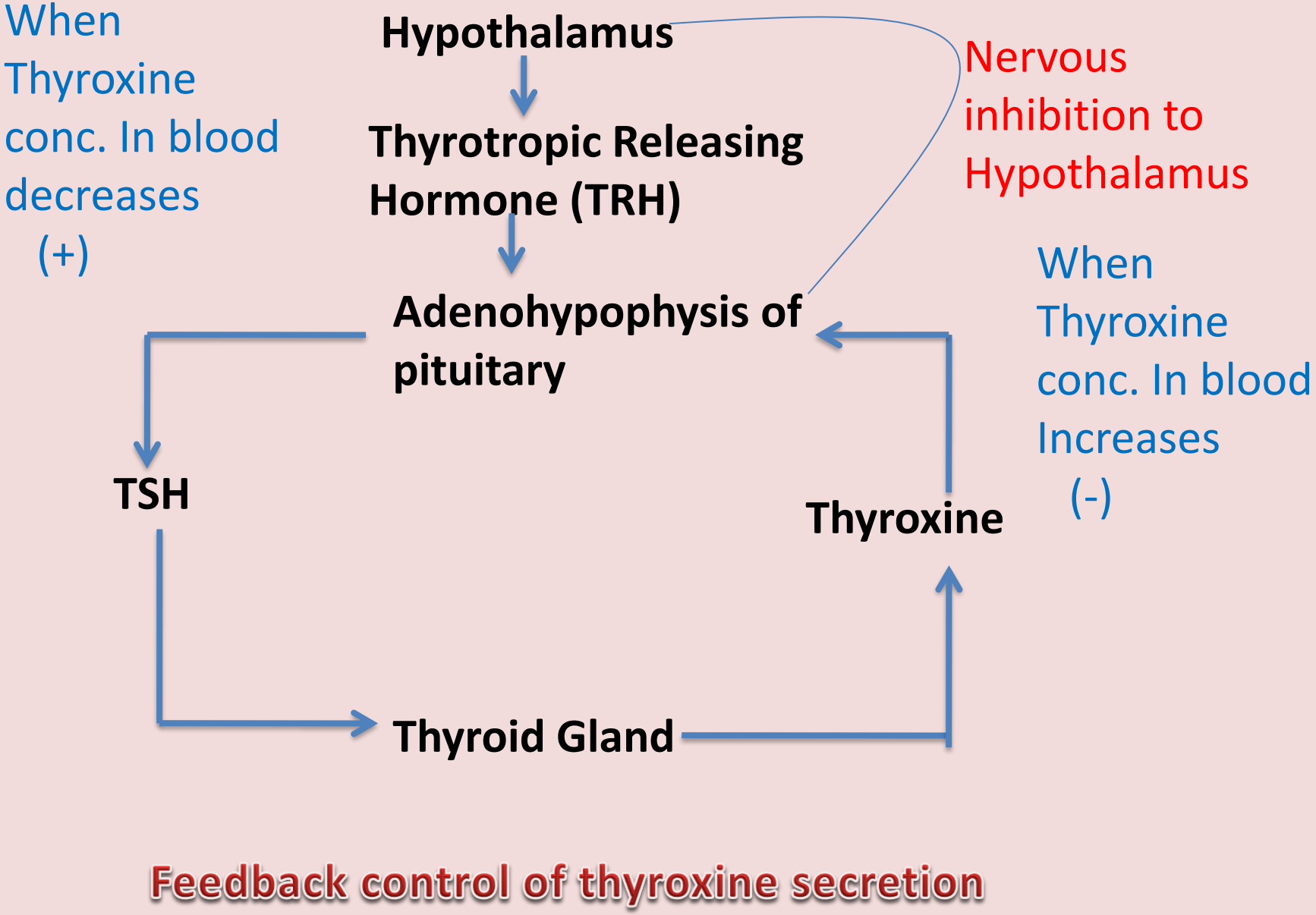
-

-Blood —Calcitonin→ Bone

-

-Bone —Parathormone→ Blood (Osteoporesis)

- Important hormone for Ca^{2+} metabolism & homeostasis. It is plasma calcium lowering hormone. It inhibit the reabsorption of bone calcium and protecting bone from osteoporosis.



Feedback control of thyroxine secretion

HYPOTHALAMUS

HYPOTHALAMIC-
PITUITARY
PORTAL SYSTEM

(-)

(-)

TRH
(+)

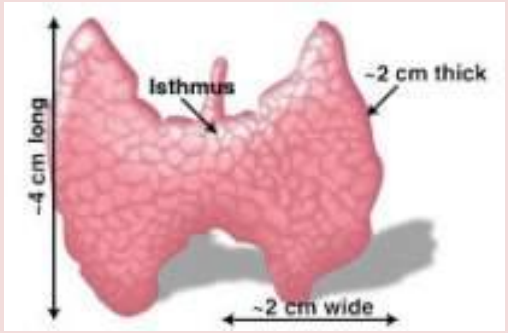
ANTERIOR
PITUITARY

POSTERIOR
PITUITARY

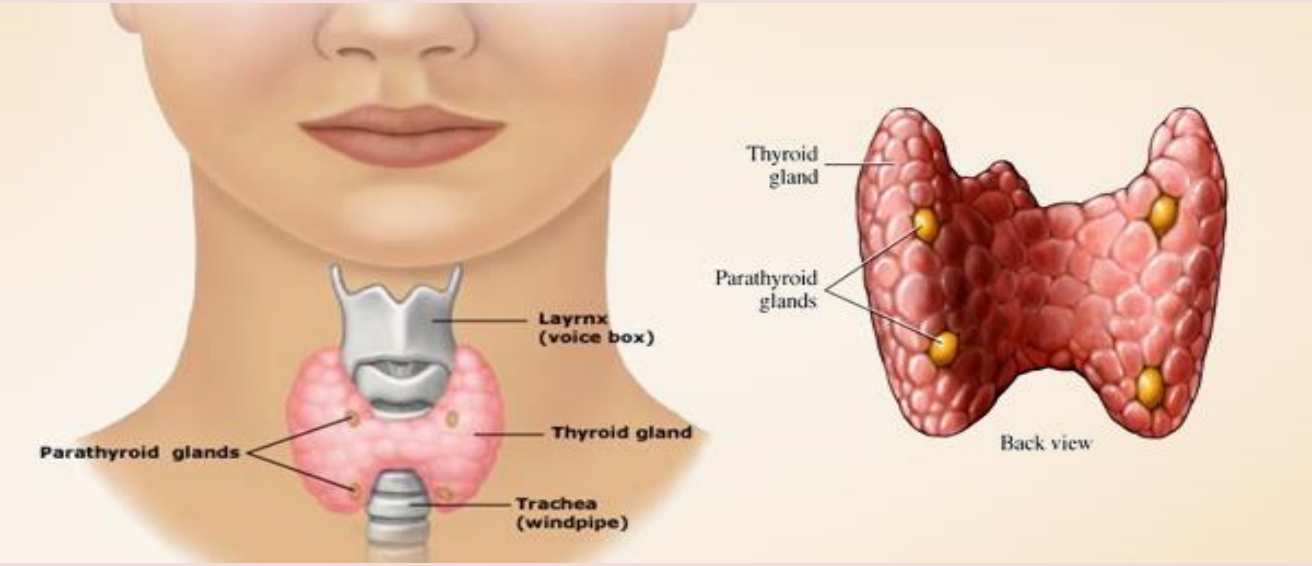
TSH

THYROID GLAND

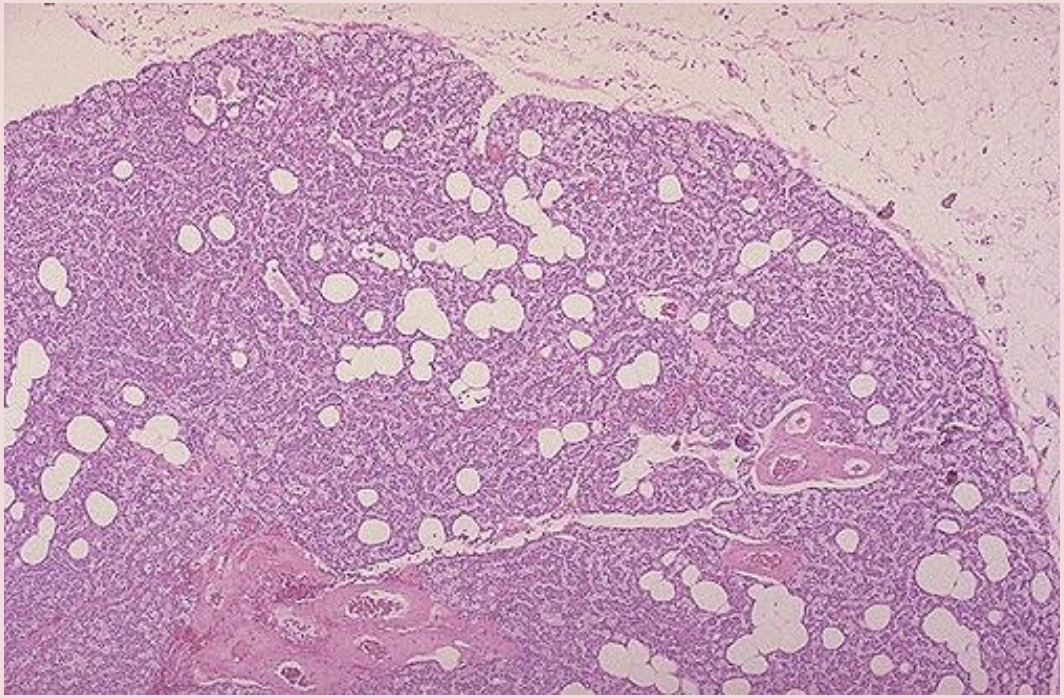
T4, T3 (T4 --> T3)



PARATHYROID GLAND



- Four oval, small endocrine glands situated close to Thyroid, Two on each lobe.
- Secretes the PTH/Parathormone/Collip's Hormone which maintain the metabolism of Calcium and Phosphate i.e. Movement of Ca and P from Bone to Blood.
- **Bone → Blood (Osteoporesis)**



- **Parathormone secretion is under Feedback control operated by calcium level in blood (Low Calcium level Stimulates the secretion while high Calcium level inhibits.**

Hyper-parathyroidism

- Primary Hyperparathyroidism:

Increase in PTH is due to parathyroid lesion (Adenoma/hyperplasia) → Results in Hypercalcaemia

PTH → Hypercalcaemia :

- ↑ **to mobilize Ca++ from bone to blood**
- ↑ **Ca++ reabsorption in the kidney**
- ↑ **Ca++ absorption in gut through vit .D.**
- ↑ **excretion of phosphate in urine .**

- Results into formation of **cavities in bones**, Bone pain (**osteomalacia**), Decalcification of bones results into **Osteoporesis** (**osteitis fibrosa cystica/brown tumor**), Bony deformations and fractures, Kidney stone.
- peptic ulcer, Metastatic calcification (blood vessels, soft tissue & & joints)

Hypo-parathyroidism

- Hypofunction of PTH results into decrease Calcium level in blood, It causes muscular twitching, cramps and spasms (sudden involuntary contraction of muscles) especially of hands, feet and face.


Thank you