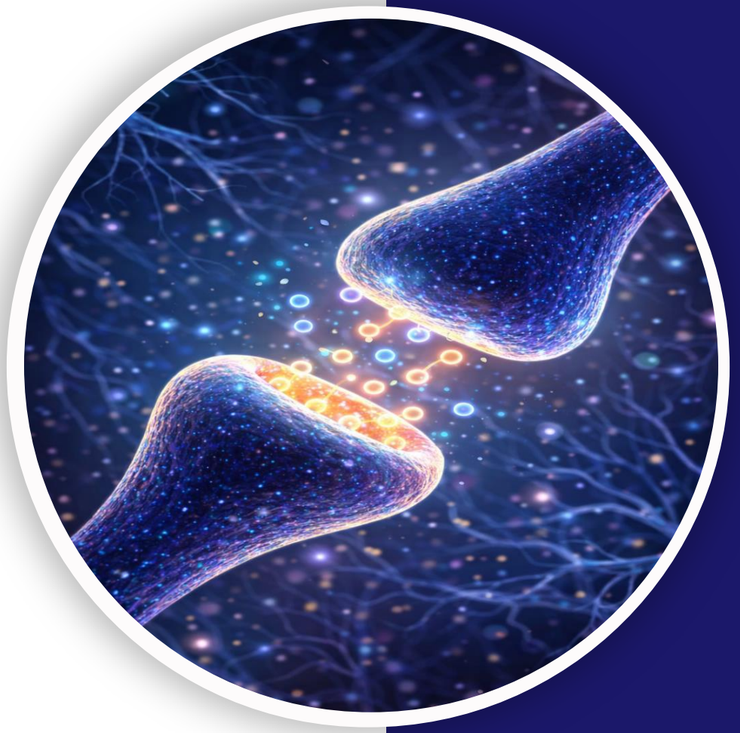
بسم الله الرحمٰن الرحيم

(وَفَوْقَ كُلِّ ذِي عِلْمٍ عَلِيمٌ)



Endocrine Physiology | MID L5

Thyroid metabolic Hormone

Reviewed by : Razan Al-Adhami
Leen Abukhalaf

Thyroid Metabolic Hormones

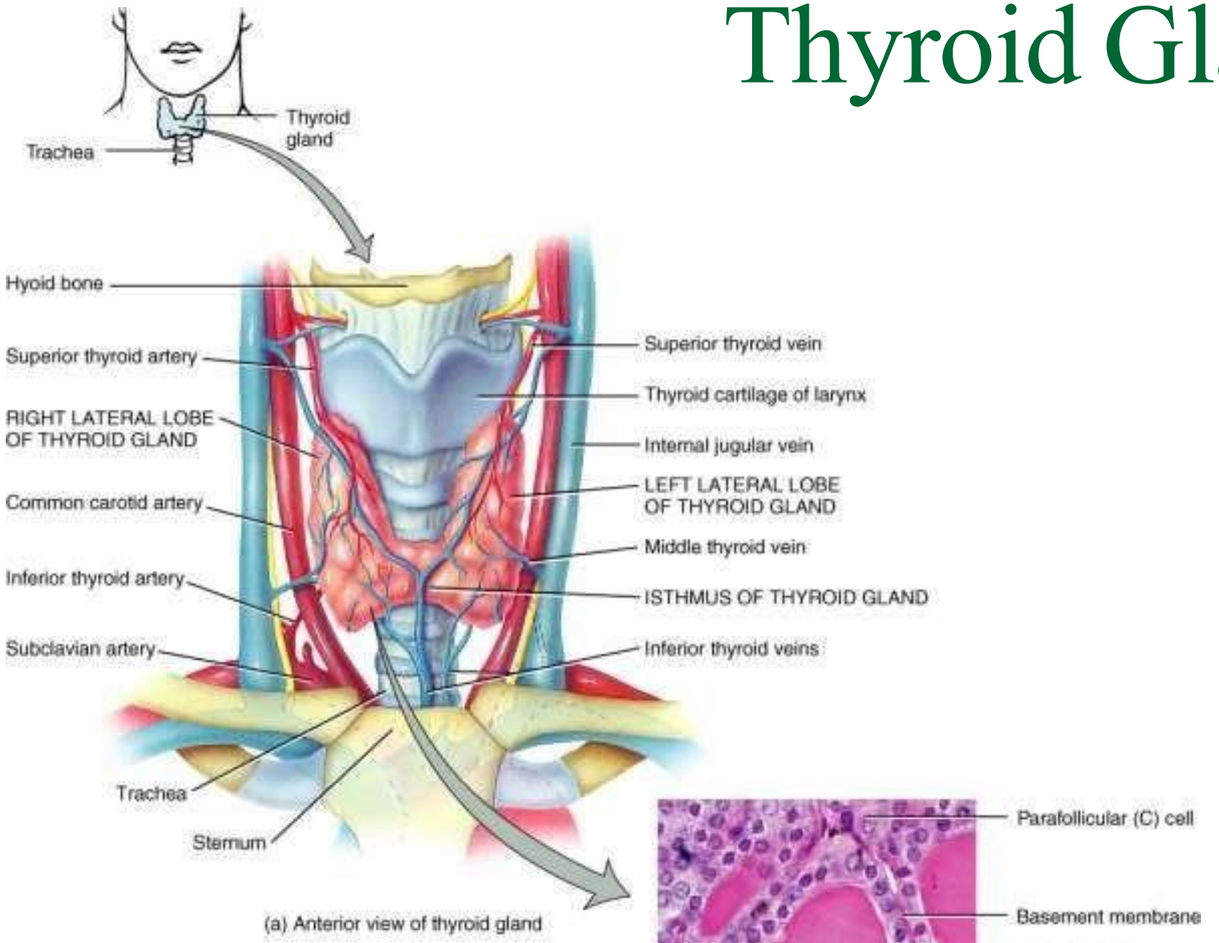
Objectives

- Illustrate thyroid hormone synthesis, storage and secretions
- Describe factors that control the synthesis, storage, and release of thyroid hormones.
- Explain the actions of thyroid hormones on development and metabolism.
- Review the mechanism of thyroid hormone actions
- Understand the causes and consequences of over- secretion and under-secretion of thyroid hormones. Explain why either condition can cause an enlargement of the thyroid gland.

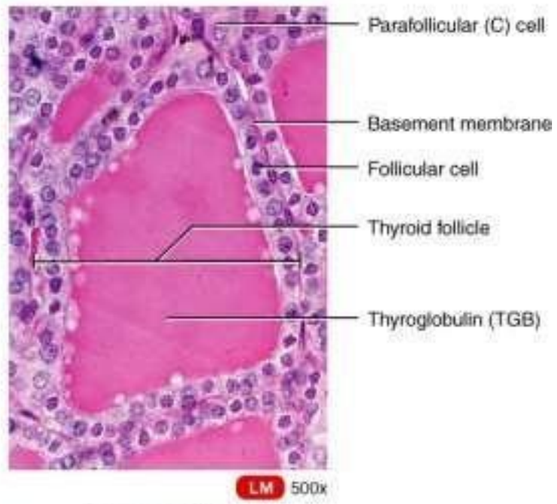
Thyroid Gland

- Located inferior to larynx
- 2 lobes connected by isthmus
- Thyroid follicles produce thyroid hormones
  - Thyroxine or tetraiodothyronine (T_4)
  - Triiodothyronine (T_3)
    - Both increase BMR (basal metabolic rate), stimulate protein synthesis, increase use of glucose and fatty acids for ATP production (unlike GH that uses the fatty acid as energy source)
      - Theoretically, this may result in low glucose concentration in case of hyperthyroidism, but actually that didn't happen; because glucose concentration is under the control of other mechanisms & other hormones.
- Parafollicular cells or C cells produce calcitonin (does not secret hormones that control metabolism rather secret hormones that control calcium)
  - Lowers blood Ca^{2+} by inhibiting bone resorption

Thyroid Gland



(a) Anterior view of thyroid gland



(b) Thyroid follicles

18.10

Remember: The shape of the follicular cells depends on its activity, when it's actively secreting colloid, it's more columnar-cuboidal and when it's inactive it's flattened-low cuboidal

Production of Thyroid Hormones -check the figure in slide 11-

1. Iodide (I^-) (thyroid wants iodine but in the plasma its iodide) actively transported into the follicle and secreted into the colloid.

- Iodide(free form) is transported to cells **with Na^+** – exploiting Na^+ concentration gradient – via "iodine pump" which considers as **secondary active transport** due to very high concentration of (I^-) inside the follicles.

2. **Oxidation:** Oxidized to iodine (I^o) by *peroxidase* using H_2O_2, this takes place because "iodination" is done by iodine not iodide.
3. **Iodination:** Iodine attached to **tyrosine** within thyroglobulin chain.
   - Attachment of 1 iodine produces monoiodotyrosine (MIT).
   - Attachment of 2 iodines produces diiodotyrosine (DIT).
4. MIT and DIT or 2 DIT molecules coupled together.

- Tyrosine molecule can't be iodide with more than two iodine, so coupling occur to from **T3 (MIT+DIT) or T4 (DIT+DIT),** reverse T3 may be synthesized also but in low concentration (will be discussed later).

To sum up: Iodide --transported-> **inside follicle** --oxidised--> **Iodine** -- Iodination of tyrosine --> **MIT or DIT** -- coupling --> **T3 or T4.**

Production of Thyroid Hormones (continued)

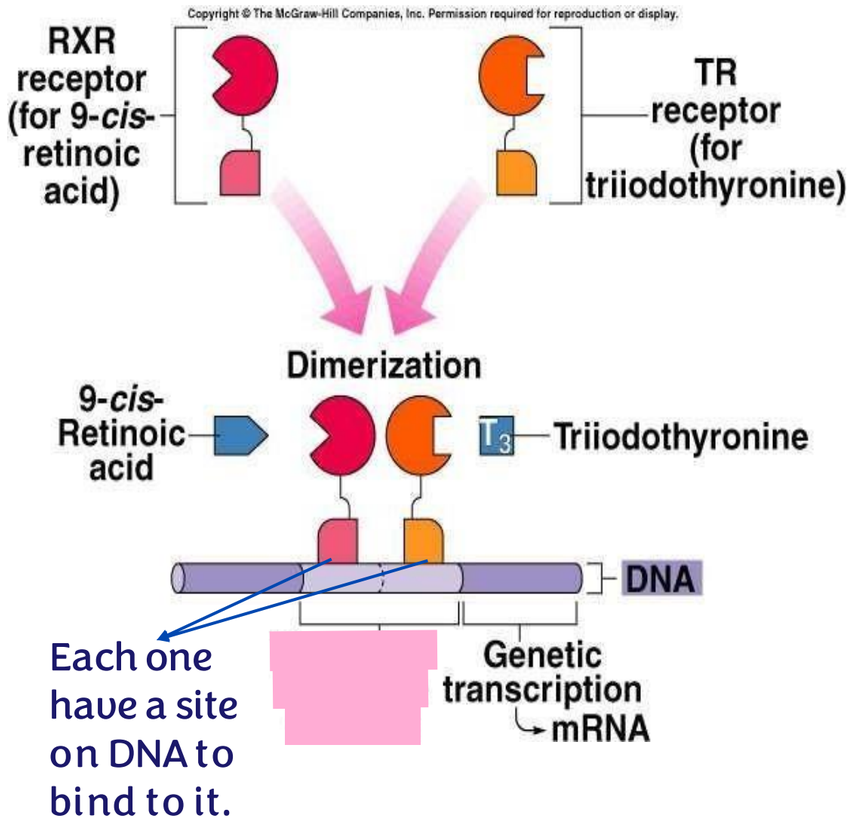
- T_3 and T_4 produced.
- TSH stimulates pinocytosis into the follicular cell.
  - TSH stimulates all processes of T3 & T4 synthesis & the growth of the gland (More TSH = More growth of Thyroid Gland (goiter)). Meaning it stimulates iodine pump, peroxidation, iodination, coupling, and pinocytosis.
  - Enzymes hydrolyze/lysosomes break down vesicle releasing T_3 and T_4 (mainly T4 80% or more) from thyroglobulin.
- Attached to TBG and released into blood cause lipid soluble so need a protein carrier.
  - Thyroxine-binding Globulin (specific)
  - Albumin (nonspecific)
  - Pre-albumin (nonspecific)

Control of thyroid hormone secretion

- Thyrotropin-releasing hormone (TRH) from hypothalamus
- Thyroid-stimulating hormone (TSH) from anterior pituitary
- Situations that increase ATP demand also increase secretion of thyroid hormones
- Regulation &control of thyroid hormones are not necessarily from hormonal negative feedback, the effect that is generated from thyroid hormones may affect; in case of low body temperature(cold), the TRH, TSH and thyroxin will be elevated, why? Cause thyroxin maintains the temperature –by increasing BMR and energy- which in turn decreases thyroidal hormones via a negative feedback loop
- Worthy to mention that the major effect for ATP is heat production, and the minor effect is to produce energy, accordingly people who live in cold weather have higher level of thyroxine &vice versa.

Mechanism of Thyroid Hormone Action

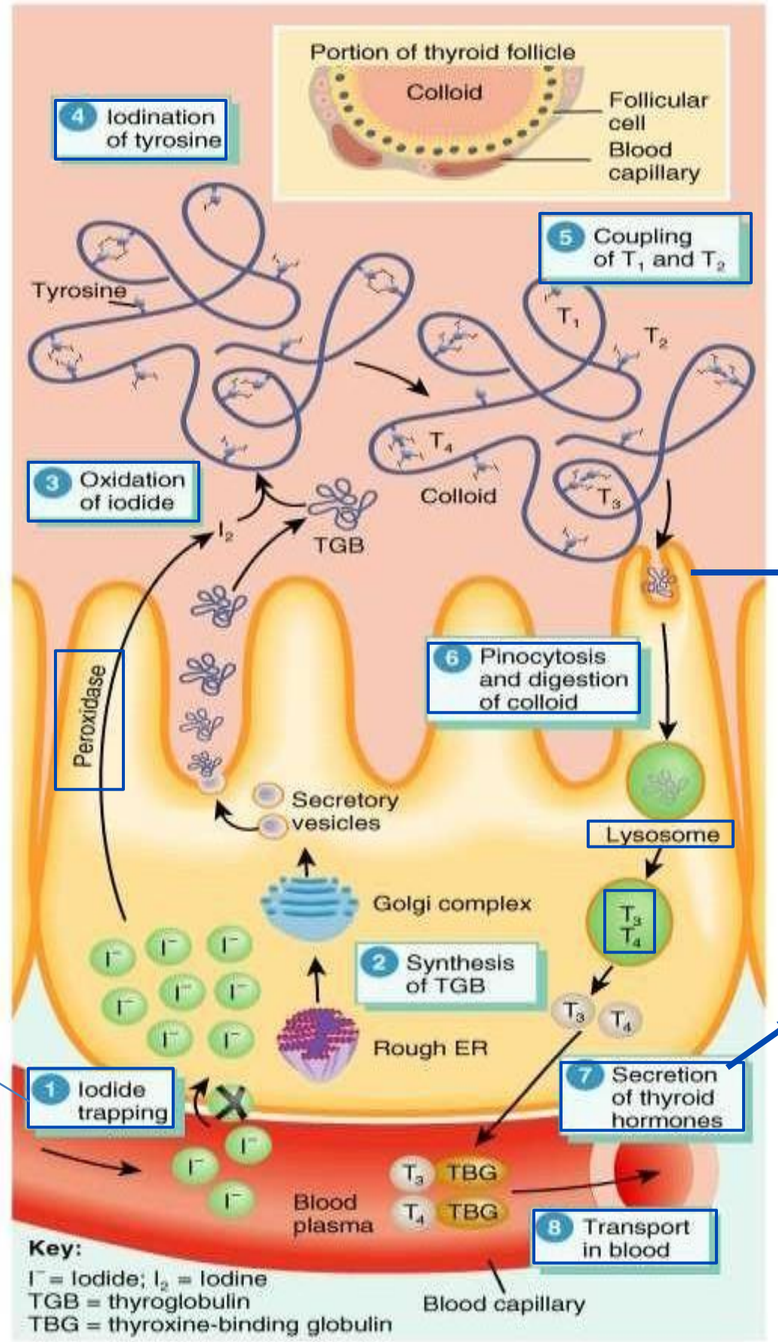
- T_4 passes into cytoplasm and is converted to T_3.
- Receptor proteins located in nucleus, there are two receptors, each one is a **dimer** so each have two binding sites, one that binds to T3 and T4 and one to DNA and they differ in the other one:
  1. $\overline{T_3}$ binds to ligand-binding domain.
  2. Other half-site is vitamin A derivative (**9-cis-retin**oic) acid.
     - DNA-binding domain can then bind to the half-site of the HRE (Hormone response element).
     - Two partners can bind to the DNA to activate HRE.
       - Stimulate transcription of genes.



- This process is the slowest in the body because it result in a protein production(half life of T4 is 7 days and T3 1 day(these are considered very long half life's)).
- All cells in the body have thyroid receptors.

Synthesis of thyroid hormones:

Make sure that you understand all the details in the figure, watching the lecture is recommended!



Thyroglobulin enters the cell via pinocytosis (endocytosis)

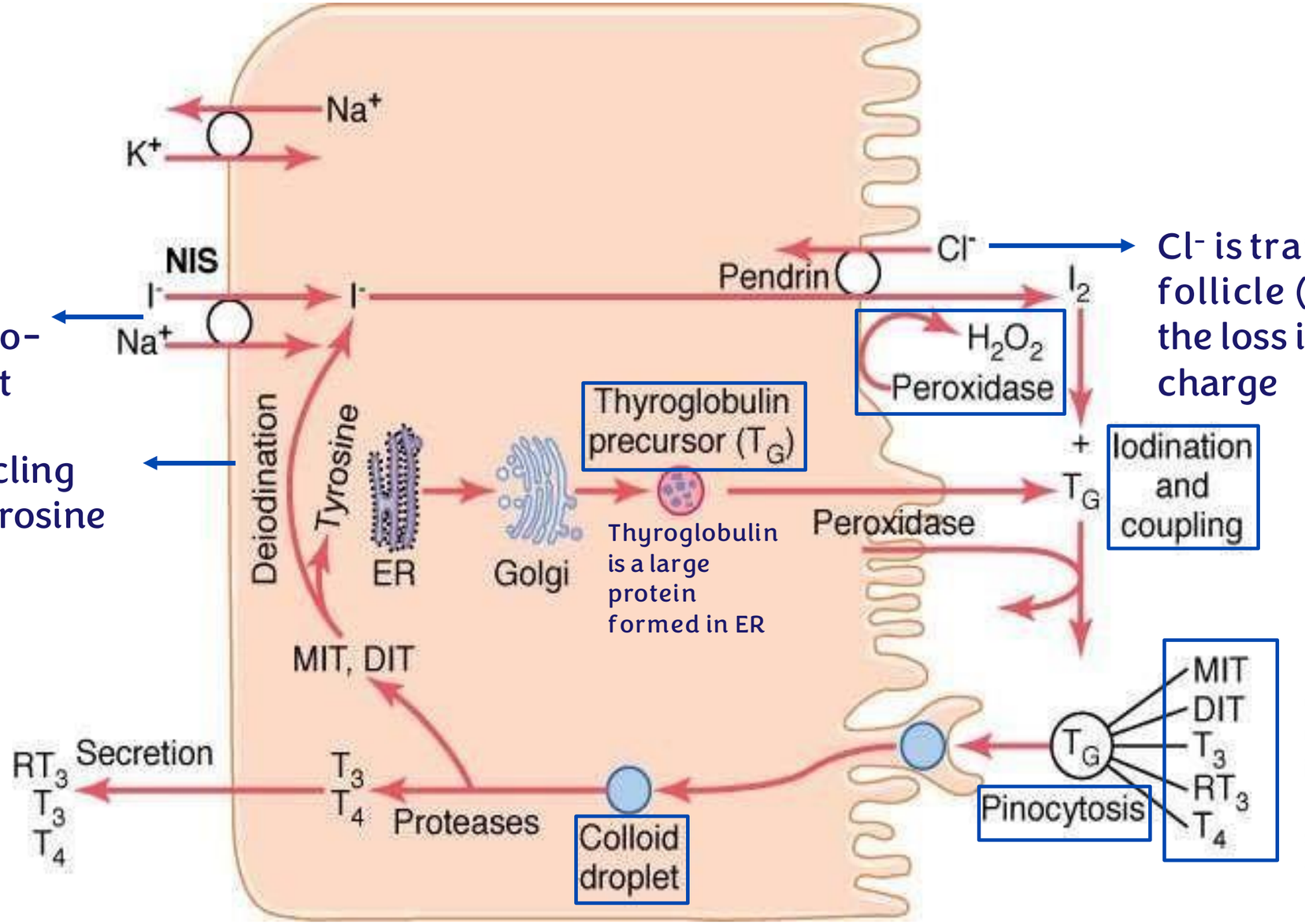
Pinocytosis, also known as cell drinking or fluid endocytosis, is a type of endocytosis where a cell takes in extracellular fluid and dissolved solutes by engulfing them in small vesicles

Iodide is found in plasma, enters cell by active transport (iodide pump)

T3&T4 bind to thyroid-binding globulin as a carrier

Make sure that you understand all the details in the figure

Thyroid Hormone Synthesis s Secretion



Cl- is transported into the follicle (cell) to balance the loss in the negative charge

Reverse T3 VS T3, they differ structurally (T3 is 3,5-3') and (RT3 is 3',5',3)
RT3 is not effective at stimulating metabolism

Thyroid Hormones

- T_3 (triiodothyronine) around 4-5 times more effective than T4- so in the peripheral tissue T4 is converted to T3-.
- T_4 (thyroxine) - Pro-hormone for T3-.

- *Pro*-hormone & *Pre*-hormone serve for the same purpose, unless there is an intermediate in the conversion process.

Thyroid Hormone Synthesis & Secretion

Prof.Faisal mentioned almost all the information in the table, so they all are required (:

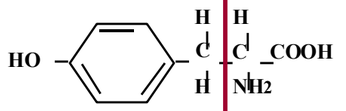
TG=thryoglobulin(synthesis in ER, packaged by golgi and goes to thyroid lumen)

	EVENT	SITE	ENZYME	INHIBITOR
1	Synthesis of TG; extrusion into follicular lumen	Rough ER, Golgi apparatus		
2	I^- pump	Basal membrane		Perchlorate, thiocyanate
3	Oxidation of $I^- \rightarrow I_2$ membrane	Apical (luminal)	Peroxidase	Propylthiouracil (PTU)
4	Organification of I_2 into MIT & DIT	Apical Membrane	Peroxidase	Propylthiouracil
5	Coupling reaction of MIT & DIT into T_3 & T_4	Apical Membrane	Peroxidase	Propylthiouracil
6	Endocytosis of TG	Apical Membrane		
7	Hydrolysis of T_4,T_3,RT_3,MIT, DIT; T_4,T_3, & RT_3 enter circulation	Lysosomes	Proteases	
8	Deiodination of residual MIT & DIT. Recycling of I^- & Tyrosine	Intracellular	Deiodinase	

These inhibitors could be utilized as drugs in case of hyperthyroidism (will be discussed more in Pharmacology).

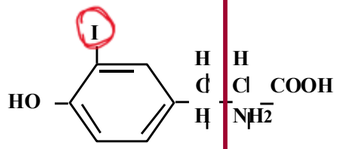
Binding of THs & Precursors to TG During Hormone Synthesis

Tyrosine

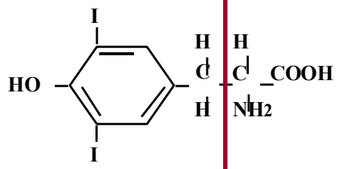


3-Monoiodotyrosine (MIT)

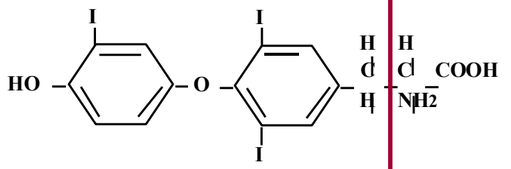
Iodination at position 3



3,5-Diiodotyrosine (DIT)

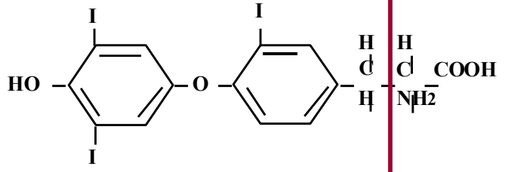


3,5,3'-Triiodothyronine (T3)

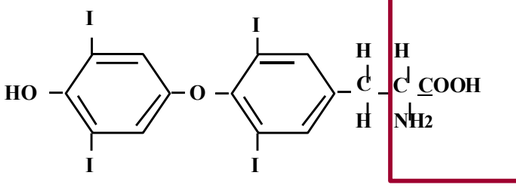


3,3',5'-Triiodothyronine (RT3)

RT3 is not effective as T3, also it present in very low concentration, unlike T3.

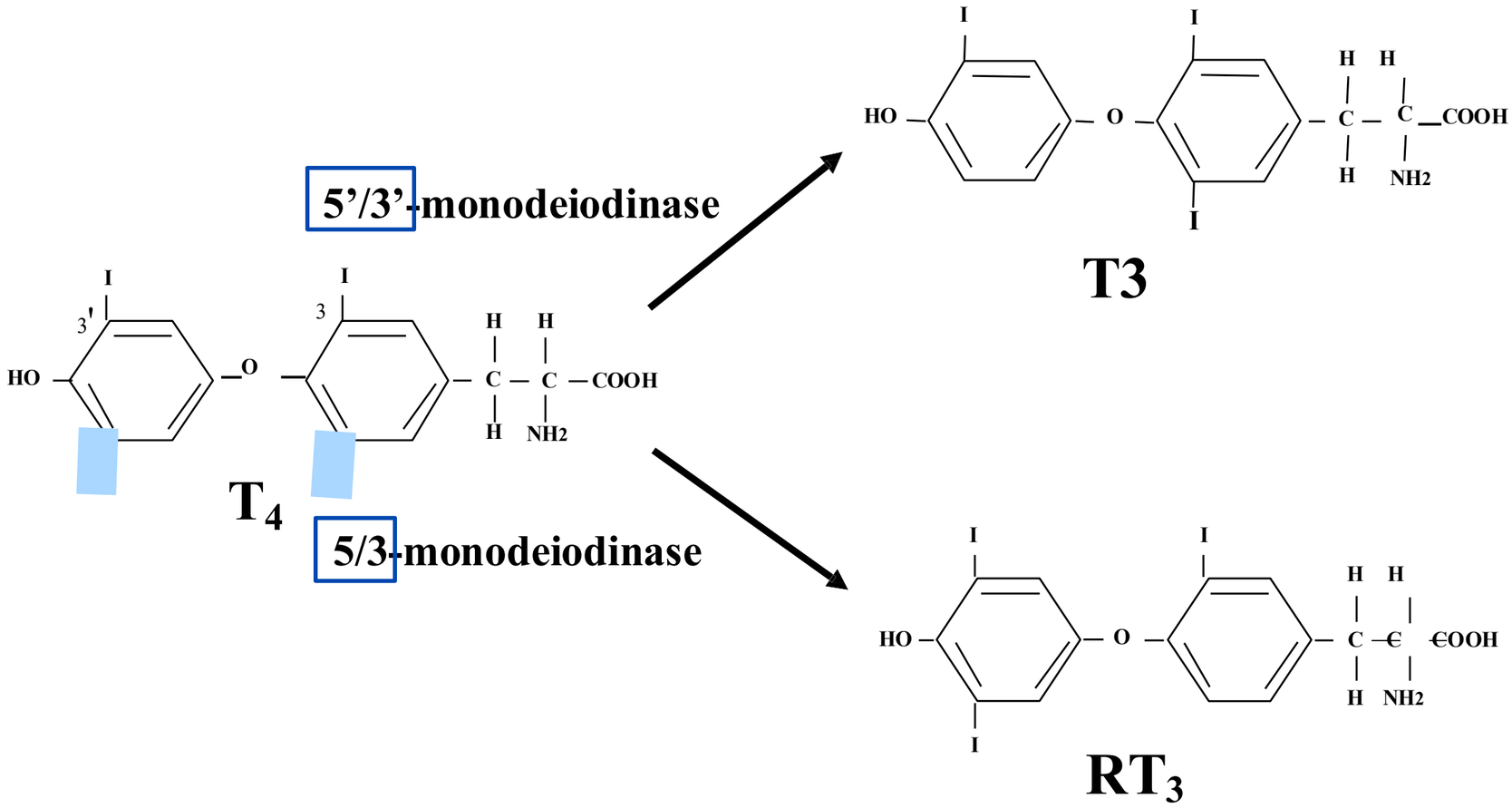


Thyroxine (T_4)



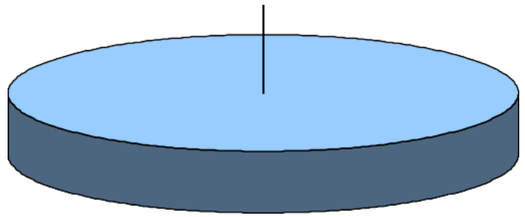
Thyroglobulin Molecule

Metabolism of Thyroxine by Deiodination in Peripheral Tissues



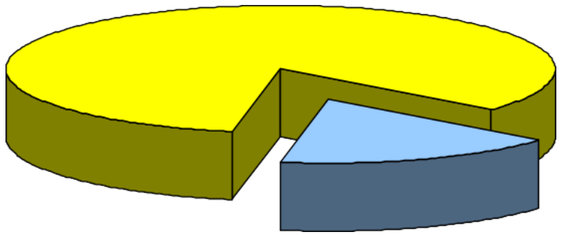
Thyroid Hormone Production

NOTE: Most of T4 is deiodinated (70 ug of 80 ug). So most secretions is T4!



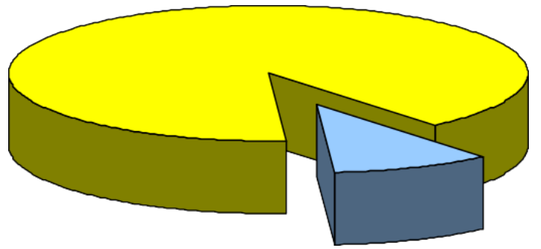
T_4

80 ug/day



T_3

31 ug/day



RT_3

38 ug/day

Secreted by thyroid

Deiodination of T_4 in peripheral tissues

Study the whole table & focus on information written in red.

Circulating Transport Proteins

Transport Protein	Principle Hormone Transported
- Specific	
Corticosteroid binding globulin (CBG, transcortin)	Cortisol, aldosterone
Thyroxine binding globulin (TBG)	Thyroxine, triiodothyronine
Sex hormone-binding globulin (SHBG)	Testosterone, estrogen
- Nonspecific	
Albumin	Most steroids, thyroxine, triiodothyronine
Transthyretin (prealbumin)	Thyroxine, some steroids

Steady-State Effects of Variations in Plasma [TH- Binding Proteins]

Condition	Concentrations of Binding Proteins	Total Plasma T_4, T_3, RT_3	Free Plasma T_4, T_3, RT_3	Plasma TSH	Clinical State
Hyperthyroidism	Normal	High	High	Low	Hyperthyroid
Hypothyroidism	Normal	Low	Low	High	Hypothyroid
Estrogens, pregnancy	High	High	Normal	Normal	Euthyroid
Liver disease, glucocorticoids, androgens	Low	Low	Normal	Normal	Euthyroid

Why Normal? Original it was low, it increased TSH which stimulated gland to grow bigger to compensate for low T3,T4 and when it became bigger the thyroxin amount became normal (gland size is bigger—goiter)

NOTES:

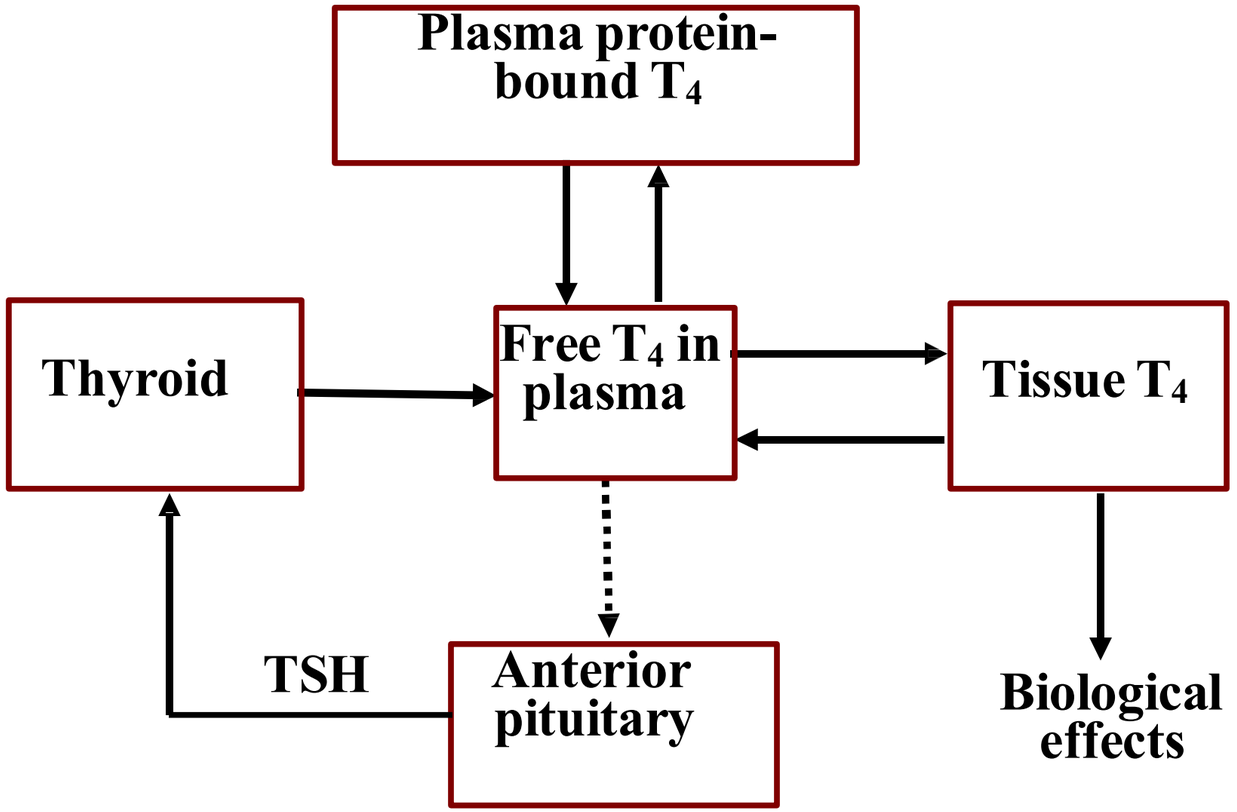
1 Both hypothyroidism & hyperthyroidism may cause **goiter** (increase in size of the gland).

2 In euthyroid, T3&T4 levels are normal due to over stimulation of thyroid gland as a compensation of decreased function (Euthyroid = subclinical hypothyroid).

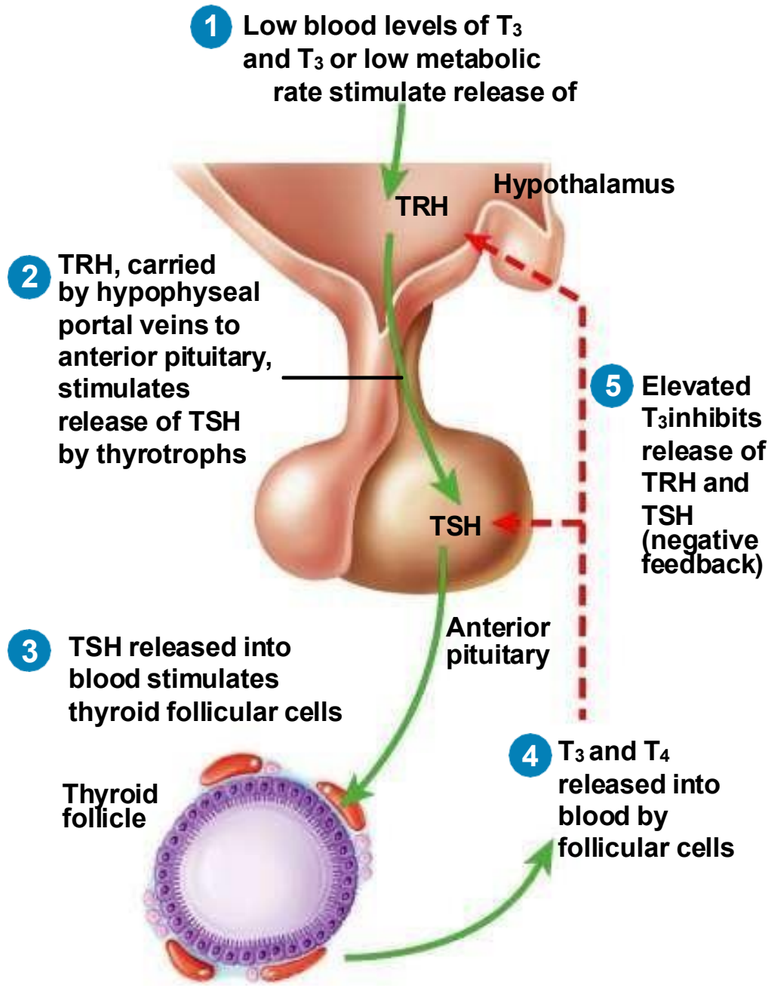
3 decision is done according to "free plasma hormones" concentration".

4 Estrogen & testosterone are considered as growth hormone like hormone which results in high concentration of proteins.

Interrelationships Between Free & Bound T4



- These interrelationships occur to maintain molecules' concentration & effect in the normal physiological range.
- They all are mentioned by Prof.Faisal so make sure you master them too (:



Summary

Low blood levels of T3 and T4 >> Stimulate release of TRH from the hypothalamus >> TRH stimulate release of TSH from anterior pituitary >> TSH stimulate release
and synthesis of T3 and T4 from thyroid.

Actions of Thyroid Hormones:

T4: Increase basal metabolic rate to increase ATP usage for energy

Stimulate synthesis of Na^+/K^+ ATPase [that is a protein]

Other protein synthesized can be beta adrenergic receptors in the heart which becomes more sensitive to epinephrine and norepinephrine so there will be an increase in heart rate + contractility + cardiac output.

Increase body temperature (calorigenic effect)

About 75% of the energy released from ATP is lost as heat, while only about 25% is converted into useful mechanical work.(Best efficiency is in the heart.)

so hyperthyroidism patients are characterized by having heat intolerance [feeling hot in cold weather] unlike hypothyroidism patients who are cold intolerant.[feeling cold in hot weather].

Stimulate protein synthesis [thyroid act on many different cells and proteins produced are cell specific]

Increase the use of glucose and fatty acids for ATP production

Stimulate lipolysis so there be increase in FFA. But for hyperthyroid patients' blood cholesterol, LDL, VLDL, and TAG will be low - doesn't increase atherosclerosis, but there will be tachycardia (hyperthyroidism + B-adrenergic stimulation).

Hyperthyroidism and Hypothyroidism
In the concept of hyperthyroidism and hypothyroidism, you find that hyperthyroidism occurs in thin people due to the increased utilization of glucose and fatty acids.
You notice that they eat a lot, but they do not get fat because of the high metabolic rate.
It stimulates lipolysis, which is the breakdown of lipids to produce energy.
Therefore, they have low cholesterol levels in their blood.
Then, in hypothyroidism, they have high levels of cholesterol in their blood, and since they have high levels of cholesterol, lipids are precipitated under the skin, so we call it myxedema.
The doctor also talked about precipitation of fluid around the heart, and he said that this is not the case here.

Enhance some actions of catecholamines due to increase in synthesis of beta receptors

Thyroid hormones enhance the actions of catecholamines because thyroid hormones stimulate protein synthesis.
One of the proteins synthesized is the β receptor.
These receptors are more abundant in the heart.
When their number increases, the heart becomes more sensitive to epinephrine and norepinephrine (catecholamines).
That is why patients suffer from a high heart rate (tachycardia).
Therefore, thyroid hormones increase:
Heart rate
Cardiac output
Contractility of the heart

Regulate development and growth of nervous tissue and bones [v.imp]

Congenital Hypothyroidism :

In congenital hypothyroidism, the newborn's thyroid gland fails to produce thyroxine. During pregnancy, the fetus receives maternal thyroid hormone via the placenta as thyroxine is lipophilic and can pass. However, after birth, the infant becomes dependent on their own thyroid function.

Without treatment, lack of thyroid hormone leads to:

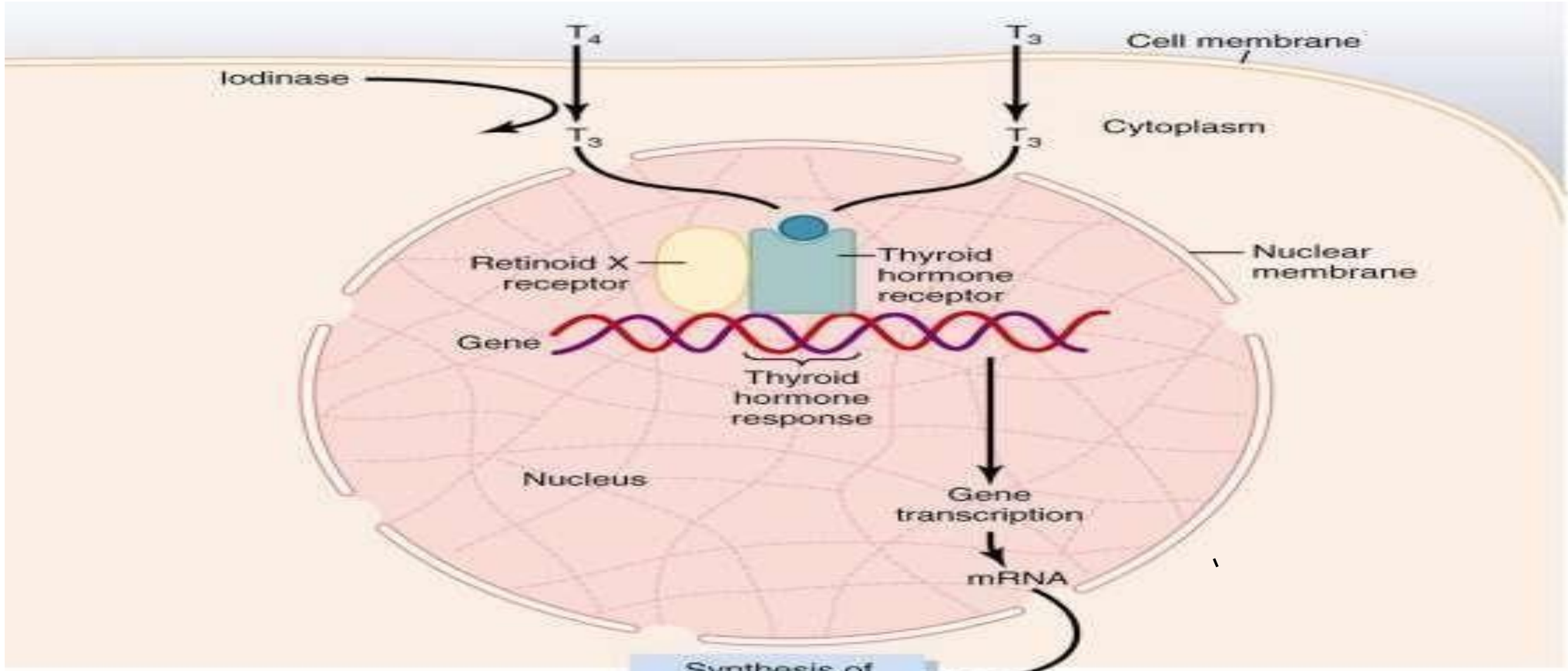
- Mental retardation due to CNS immaturity (CNS needs 2 years after birth to mature and needs thyroid hormones)
- Uncontrolled urination and defecation
- Cretinism or dwarfism [thyroid hormones needed for growth too]

Treatment:
Newborn screening for T3 and T4 should be done immediately after birth for early diagnosis
If diagnosed, prompt treatment with thyroxine tablets ensures normal growth and CNS development, allowing the child to live a normal life.

Thyroid hormones excite peripheral nerves->hyperthyroid patients are restless, nervous, high appetite, high metabolism, and easily irritable.
They also have tremor, it can be tested by placing a paper on the back of their hands (pronation position), this position), this symptom overlaps with different conditions.

Newborn Screening
Right after delivery, they measure T3 and T4 during newborn screening.
If they are low, the child needs thyroxine for the rest of his life.
The doctor mentioned that the dose is 100 micrograms.
If the child receives thyroxine, he will grow normally.
If T3 and T4 increase, they will produce negative feedback on the anterior pituitary and the hypothalamus, causing them to decrease their secretion.

Actions of Thyroid Hormones



Mechanism of Action of Thyroid Hormones
Thyroid hormones are lipid soluble.
They enter the nucleus and bind to their receptors.
We have T3/T4 binding sites together with vitamin A derivatives.
Then they move to the DNA and bind to the Hormone Response Element (HRE).
They form new proteins.
These proteins could be:

- Growth proteins
- CNS maturation proteins
- β receptors in the cardiovascular system

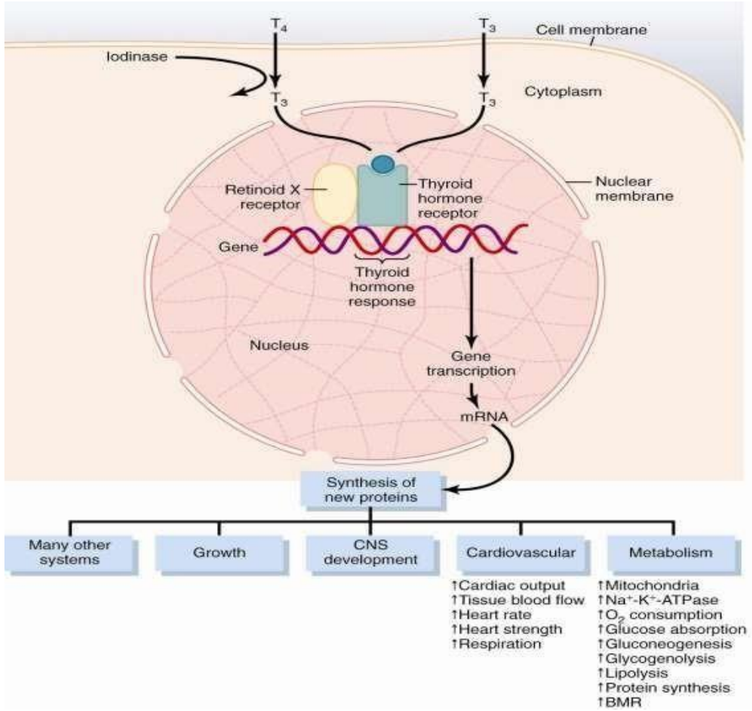
These β receptors increase the heart rate, cardiac output, and the contractility of the heart.
There is also increased synthesis of:

- Sodium-potassium ATPase
- Mitochondria

Mitochondria stand for ATP production.
Then we have:

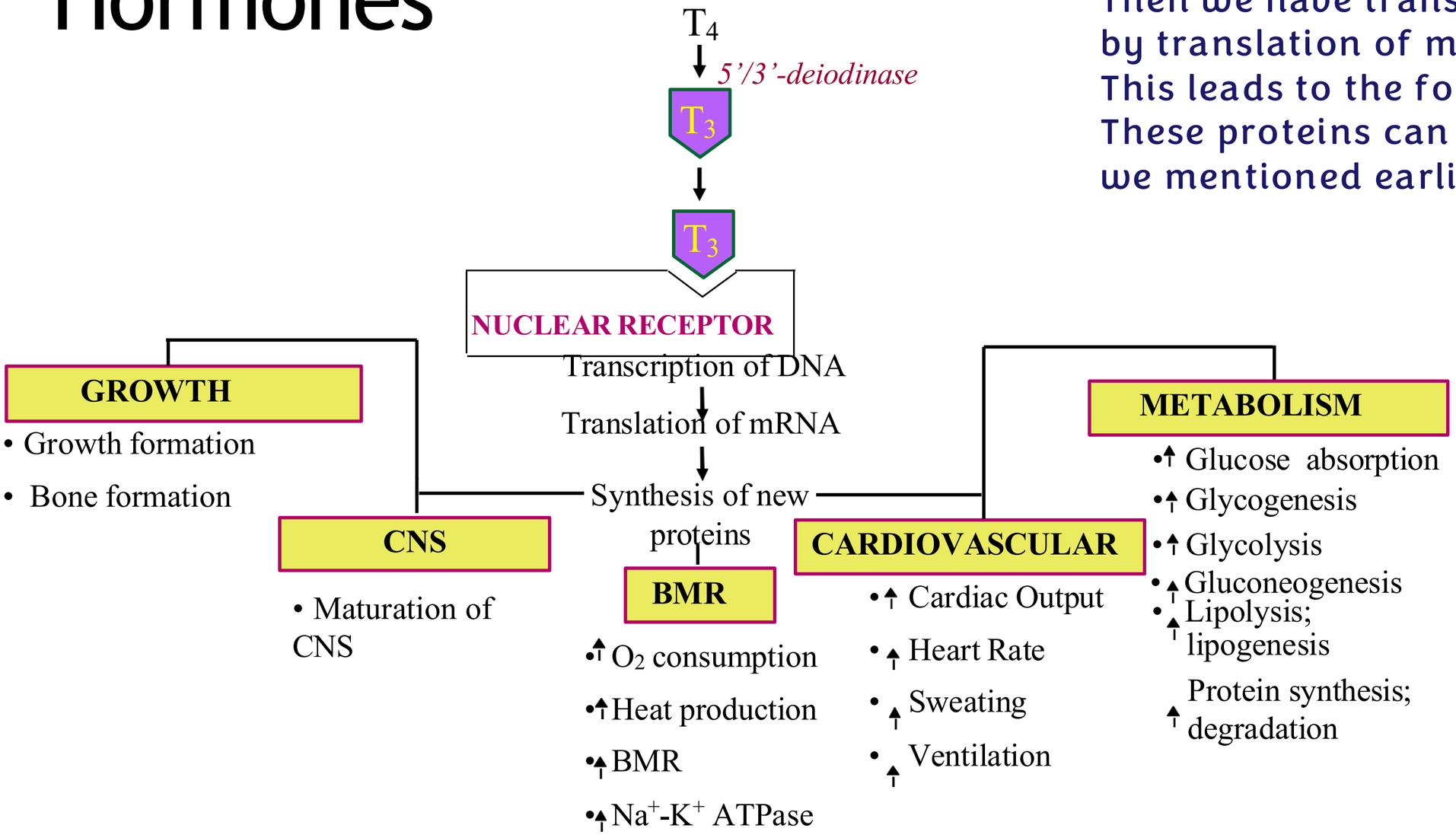
- Glycogenolysis
- Gluconeogenesis
- Lipolysis

There is also increased absorption of glucose to be used for energy.
Lipolysis is the breakdown of lipids, so patients have low cholesterol levels in their blood.



Actions of Thyroid Hormones

T4 is transformed into T3.
Then we have transcription of DNA followed by translation of mRNA.
This leads to the formation of new proteins.
These proteins can be multiple things, just as we mentioned earlier.



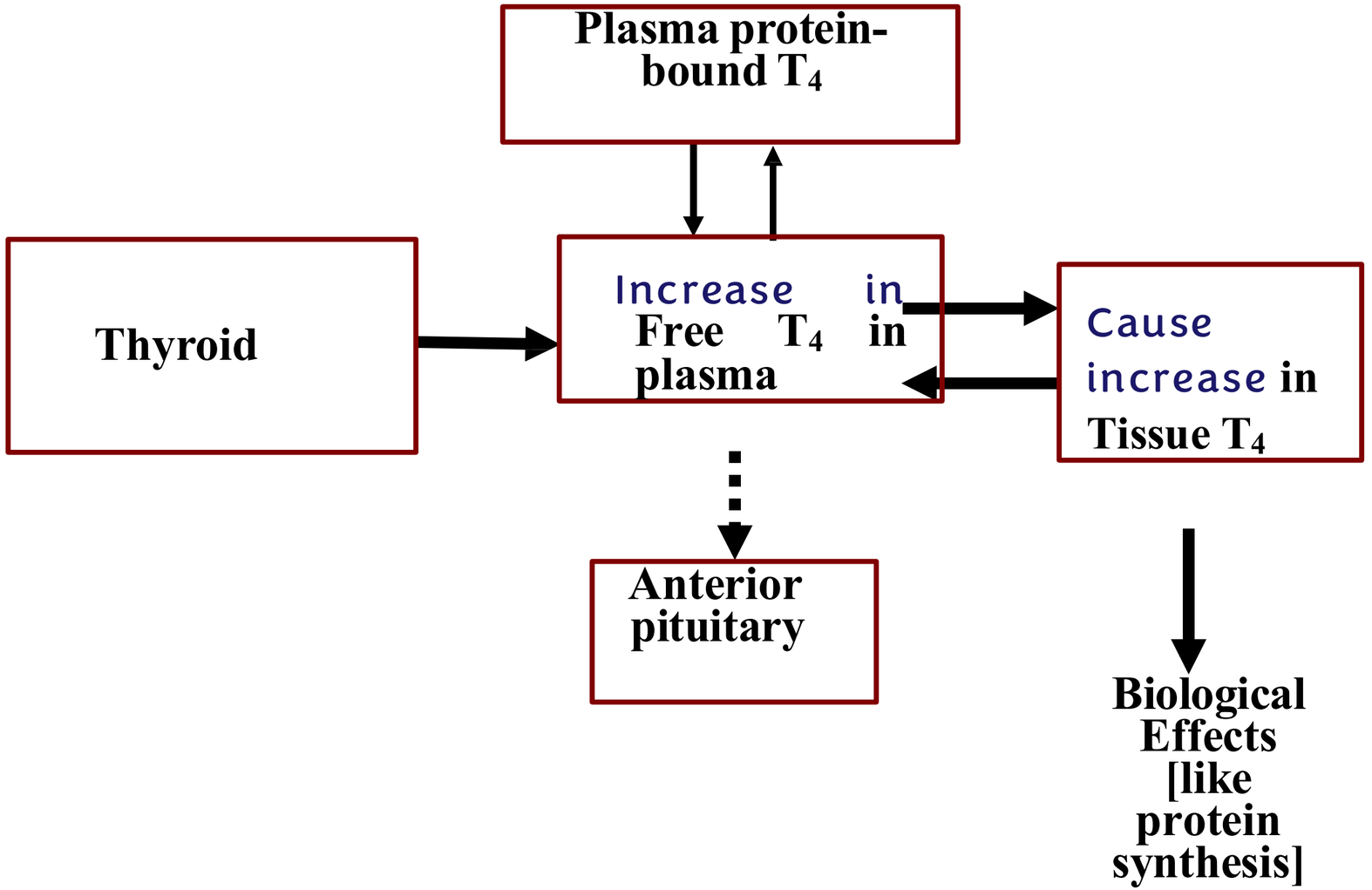
Actions of T_3

- Stimulates protein synthesis. Like GHLH
- Promotes maturation of nervous system in first 2 years of age
- Stimulates rate of cellular respiration by:
  - Production of uncoupling proteins [UCP] in mitochondria [recall metabolism: UCP3 in skeletal muscle]
  - Increase active transport by Na^+/K^+ pumps.
  - Lower cellular [ATP].

The action of thyroid hormone includes lower cellular ADP because of the high consumption of ATP for energy.
Increased metabolic rate equals increased metabolic heat.

- Increases metabolic heat.
- Increases metabolic rate.
  - Stimulates increased consumption of glucose, fatty acids and other molecules.

Interrelationships Between Free s Bound T_4--Increased T_4 Secretion



From the thyroid, there is a free T4 in the plasma.
The free T4 feeds back to the anterior pituitary and also feeds back to the hypothalamus.
The free T4 in the plasma acts on the tissues.

Steady-State Effects of Variations in Plasma [TH-Binding Proteins]

Condition	Concentrations of Binding Proteins	Total Plasma T_4, T_3, RT_3	Free Plasma T_4, T_3, RT_3	Plasma TSH	Clinical State
Hyperthyroidism	Normal	High	High	Low	Hyperthyroid
Hypothyroidism	Normal	Low	Low	High	Hypothyroid
Estrogens, pregnancy	High	High	Normal	Normal	Euthyroid
Liver disease, glucocorticoids, androgens	Low	Low	Normal	Normal	Euthyroid

Thyroid Function Summary

In both hypothyroidism and hyperthyroidism, the levels of thyroid-binding proteins remain normal.

In primary hypothyroidism:
Free T3 and T4 levels are low
TSH is high, due to loss of negative feedback on pituitary gland
In primary hyperthyroidism:
Free T3 and T4 are high
TSH is low, also due to negative feedback

If the abnormality is due to the pituitary (secondary thyroid disease), TSH and T3/T4 levels will be appropriately matched:

For example, high T3/T4 with high TSH, or low T3/T4 with low TSH – both suggest pituitary dysfunction

Key Rule:
Always measure both TSH and free T3/T4 together for accurate diagnosis – this saves time and helps differentiate between primary and secondary thyroid
*primary: thyroid dysfunction\ secondary: pituitary dysfunction

TBG (Thyroxine-Binding Globulin):

In cases of liver disease, patients have a very low concentration of Thyroxine-Binding Globulin (TBG) because TBG is formed in the liver.
Because of the higher TSH, the thyroid cells become bigger.
The thyroid gland becomes bigger and starts to secrete a normal amount of thyroxine.
This thyroid may have a goiter.
Goiter does not mean hypothyroidism or hyperthyroidism.
You determine whether the patient has hypo- or hyperthyroidism by measuring the level of thyroxine.

Metabolic Effects of Thyroid Hormones

Parameter	↓ T_3, T_4 Hypothyroidism	↑ T_3, T_4 Hyperthyroidism
Basal metabolic rate	↓	↑
Carbohydrate metabolism	↓ Gluconeogenesis ↓ Glycogenolysis Weight gain, laziness	↑ Gluconeogenesis ↑ Glycogenolysis ↑ Glycolysis
	Normal serum [glucose]	Normal serum [glucose]
Protein metabolism	↓ Synthesis ↓ Proteolysis	↑ Synthesis ↑ Proteolysis Muscle wasting [becomes thin]
Lipid metabolism	↓ Lipogenesis ↓ Lipolysis ↑ Serum [cholesterol]	↑ Lipogenesis ↑ Lipolysis ↓ Serum [cholesterol]
Thermogenesis	↓	↑ increase in heat production

[NO change in blood glucose as blood glucose is regulated by many hormones like growth hormone, glucagon and insulin]

Protein metabolism

↓ Synthesis
↓ Proteolysis

↑ Synthesis
↑ Proteolysis

Muscle wasting
[becomes thin]

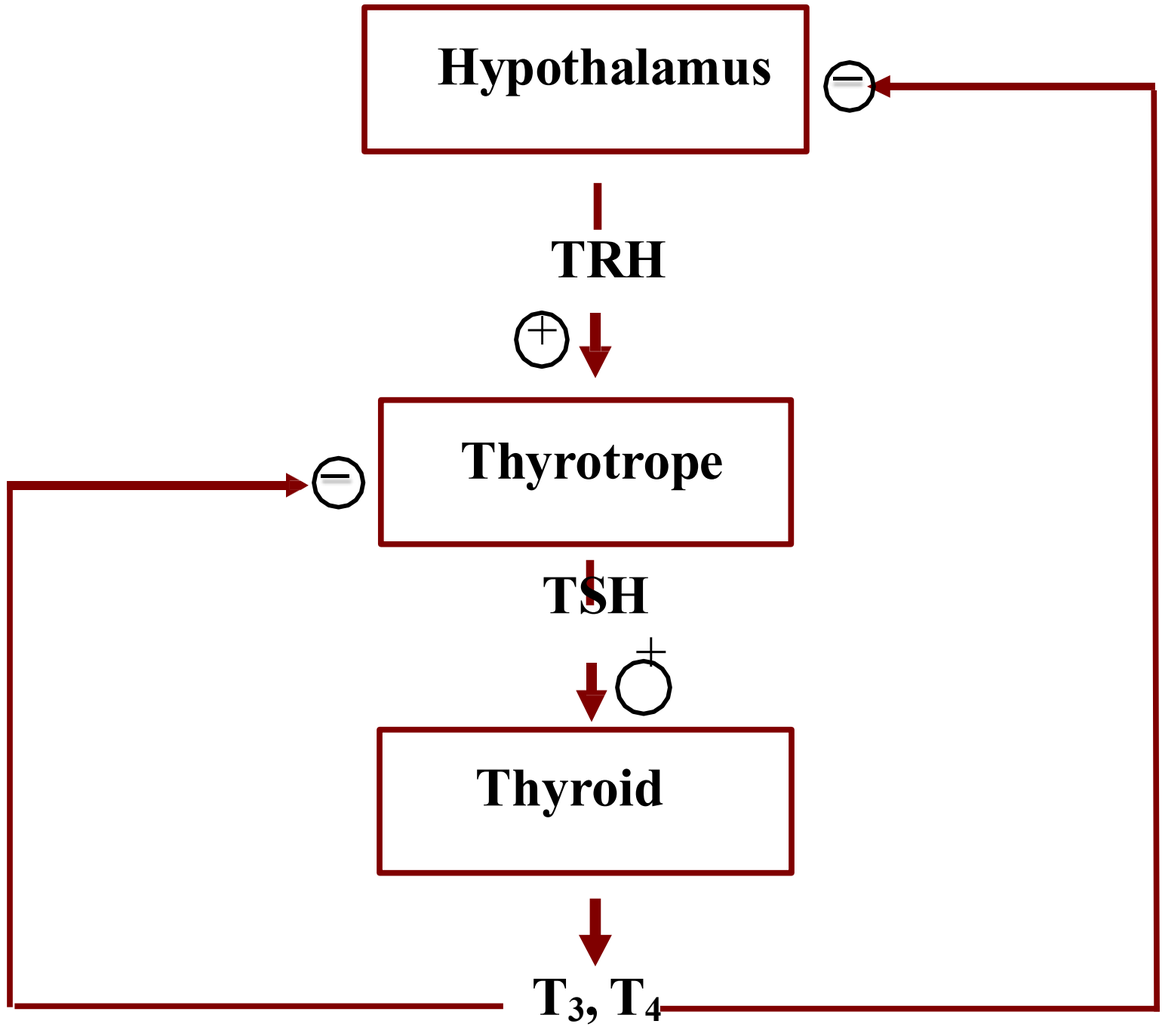
Lipid metabolism

↓ Lipogenesis
↓ Lipolysis
↑ Serum [cholesterol]

↑ Lipogenesis
↑ Lipolysis
↓ Serum [cholesterol]

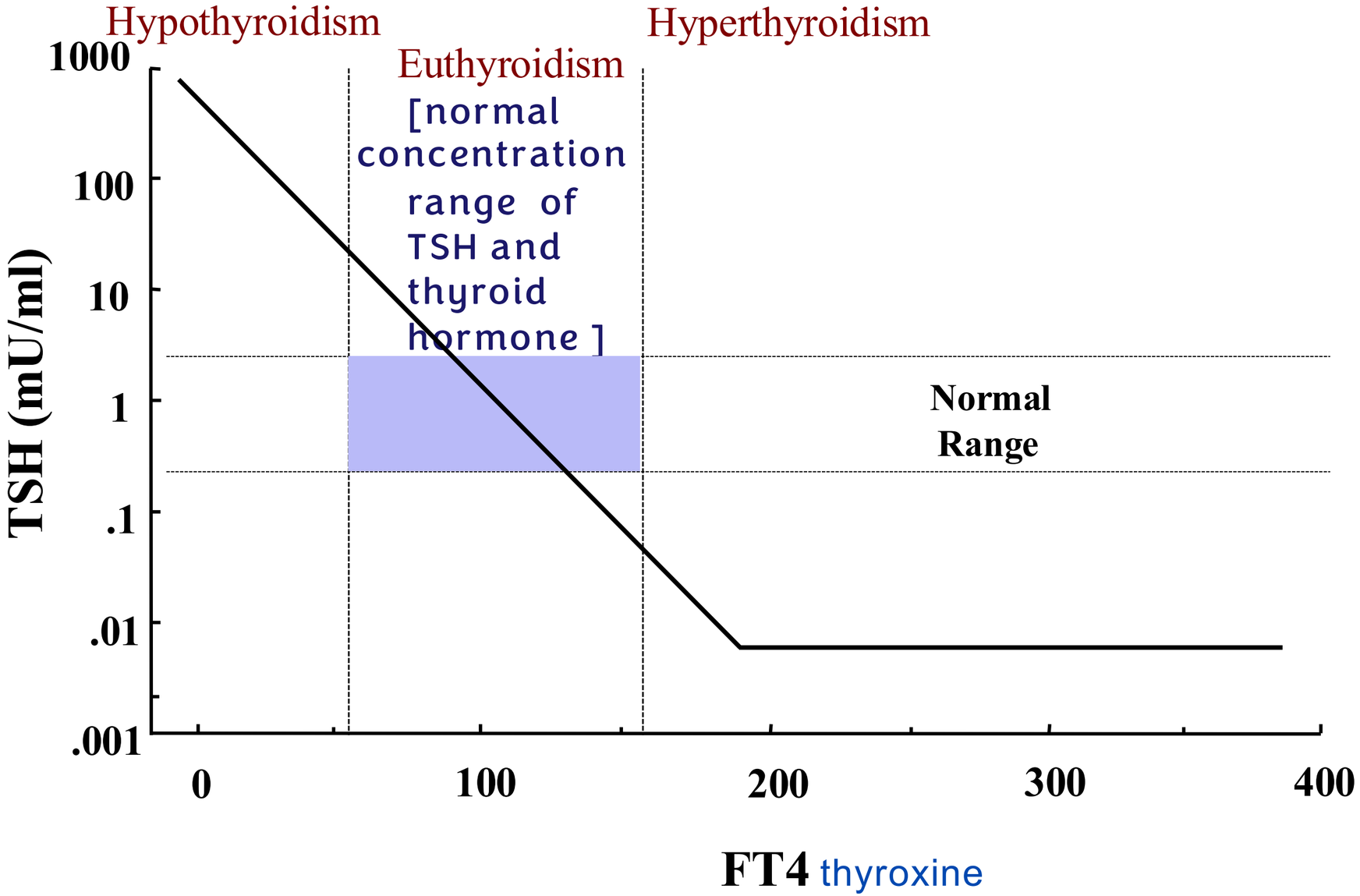
This cause accumulation of fat under the skin
>> myxedema
Not same as pitting edema that is accumulation of fluid in interstitial fluid
[recall introduction to physiology]
Myxedema indicate thyroid disease.
Pitting edema indicate heart disease.

Hypothalamohypophyseal-Thyroid Axis



T3 and T4 give negative feedback to the hypothalamus and the anterior pituitary.
Whenever they increase, they produce negative feedback.
Another type of feedback is controlled by the effects of thyroxine itself.
For example:
Heat
Weight
Metabolic rate

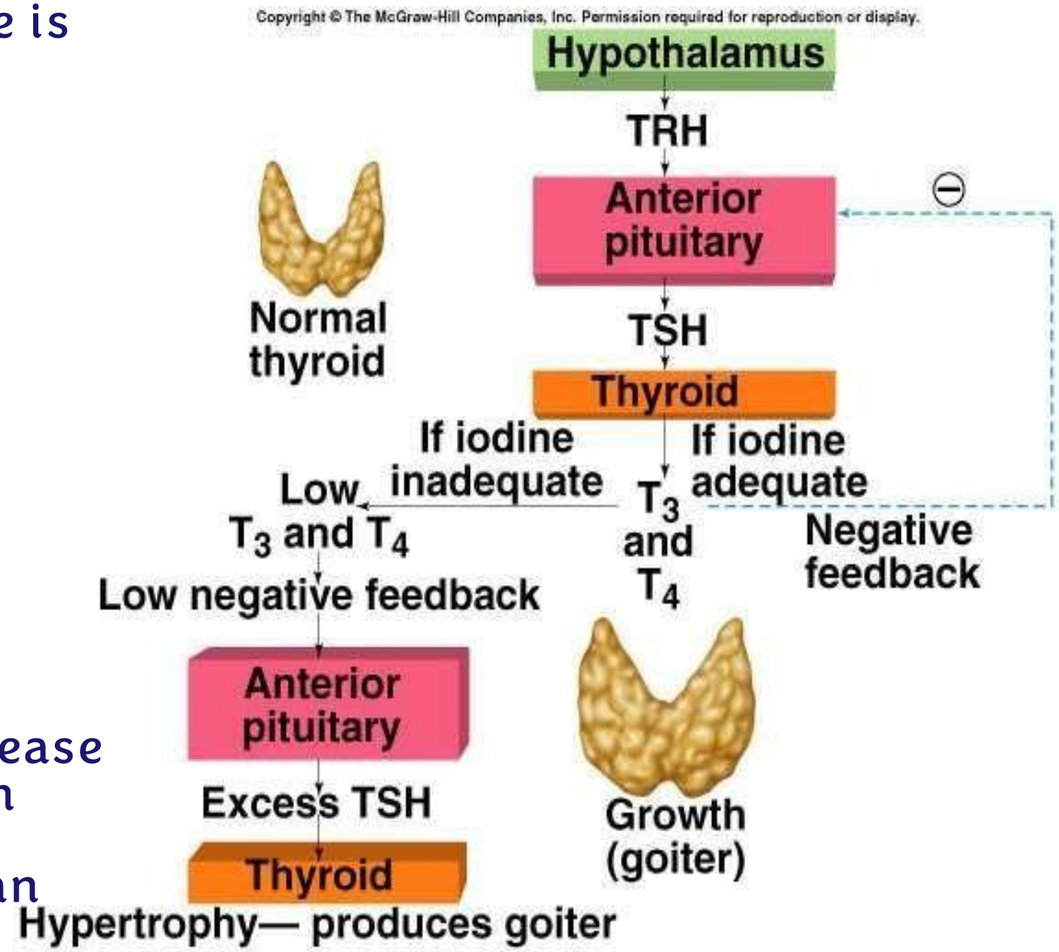
Relationship Between Plasma TSH s Free T4



Diseases of the Thyroid

Can be endemic (esp in high altitudes) but nowadays is rare because iodine is present in any salt

- Iodine-deficiency (endemic) goiter:
  - Abnormal growth of the thyroid gland.
    - In the absence of sufficient iodine, cannot produce adequate amounts of T_4 and T_3.
      - Lack of negative feedback inhibition.
        - Stimulates TSH, which causes abnormal growth.



iodine abnormality won't allow for normal thyroid hormones concentration, but an increase in TSH would be present (remember here both thyroid and pituitary are functional) but this constant stimulation of the thyroid by TSH can cause goiter.

Mutation in peroxidase enzyme (responsible for iodine oxidation) can also cause goiter

Causes of Thyrotoxicosis

- **Primary hyperthroidism**
  - Graves' disease (most common form of thyrotoxicosis)
    autoimmune disease that produce thyroid stimulating immunoglobulin (TSI) which mimic TSH causing increase in thyroxin which cause negative feedback on TSH decreasing TSH
    >> patient has high blood thyroxin and low TSH
  - Toxic multinodular goiter
  - Toxic adenoma
  - Thyroid carcinoma
- **Thyrotoxicosis without hyperthyroidism**
  - Thyroiditis
  - Iatrogenic : formed by doctor [like giving a drug]
- **Secondary hyperthyroidism**
  - TSH-secreting pitutary adenoma
  - Hypothalamic disease

Prevalent Endocrine Disorders in the Adult

Disorder	Prevalence
• Type II DM	~8%
• Hypothyroidism	5-10%, ♀ 0.5-2%, ♂
• Graves' disease	1-3%, ♀ .1%, ♂ . More in females
• Thyroid nodules	5%
• Osteoporosis	5%, ♀ 1%, ♂
• Hyperparathyroidism	0.1-0.5%, ♀ > ♂

Pathophysiology of Thyroid Hormones

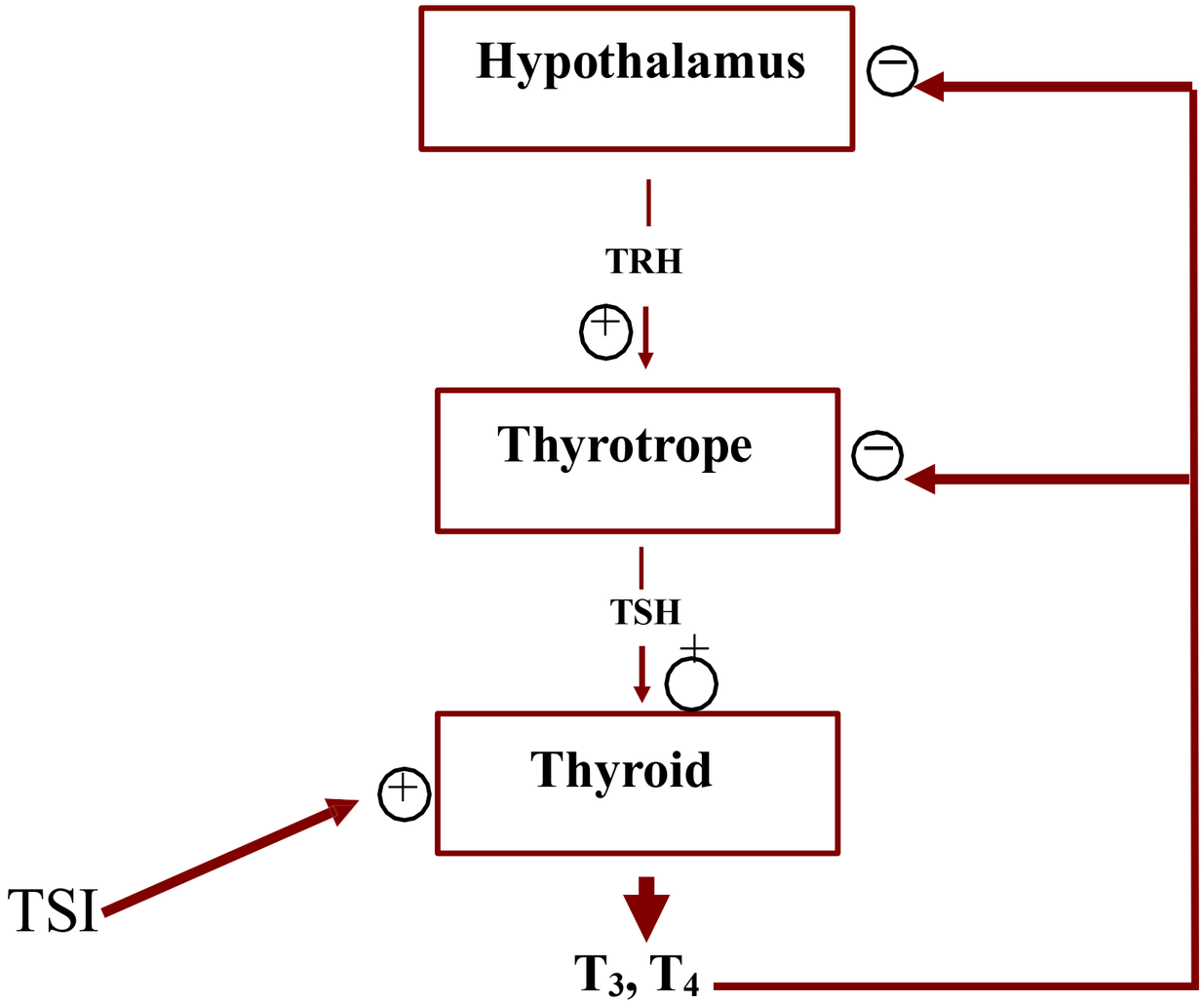
- **Graves' disease**
- Myxedema in hypothyroidism

Cretinism [dwarfism] occurs in hypothyroidism before puberty and in congenital hypothyroidism.

Again: before puberty; normal cognitive functions congenital \untreated; retardation\ abnormal cognitive functions.
After; metabolic abnormality, cognitive slowing, bradycardia, myxedema, and cold intolerance

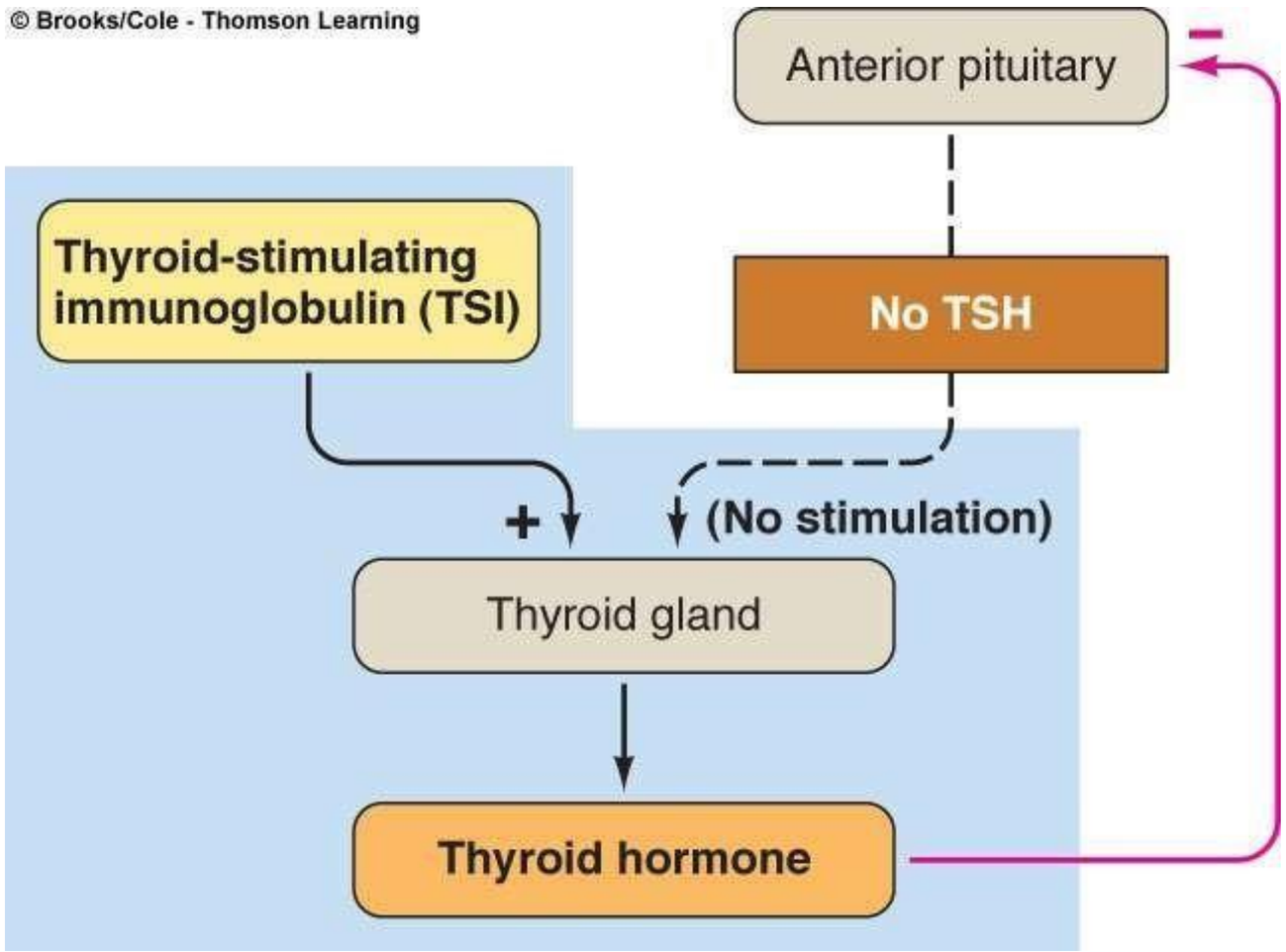
Graves disease is an autoimmune disease.
It produces a protein called Thyroid-Stimulating Immunoglobulin (TSI).
This globulin may be produced in the thyroid or somewhere else.
It stimulates more release of T3 and T4.
The increased T3 and T4 produce negative feedback on the hypothalamus.
Therefore, TSH becomes very low.
However, thyroxine remains very high because there is no control over the stimulus.

Hypothalamohypophyseal-Thyroid Axis in Graves' Disease



Patient will have high blood T3 and T4 with low TRH and TSH

Role of Thyroid-Stimulating Immunoglobulin in Graves' Disease



Signs s Symptoms of Thyrotoxicosis

Descending Order of Frequency

Symptoms	Signs
Hyperactivity, irritability, dysphoria (dissatisfaction)	Tachycardia, atrial fibrillation
Heat intolerance C sweating	Tremor [if paper placed above fingers , paper will shake . This symptom is not specific as it could be present in other diseases]
Palpitations	
Fatigue C weakness	
Nervousness [due to excitation of peripheral nerves]	Goiter
Weight loss [due to increase of metabolism] C increased appetite Diarrhea	Warm moist skin
	Muscle weakness, proximal myopathy
Oligomenorrhea	Lid retraction or lag
	Exopthalmos[protrusion of the eyeball from the eye]
	Gynecomastia

- Reason for Exopthalmos
- Autoantibodies (TSI - thyroid-stimulating immunoglobulins) activate fibroblasts in the orbit.
- These fibroblasts produce hyaluronic acid and collagen → tissue swelling.
- Inflammatory cells infiltrate muscles and connective tissue.
- Leads to enlargement of extraocular muscles and orbital fat, causing protrusion of the eyeball .

Signs s Symptoms of Hypothyroidism

Descending Order of Frequency

Symptoms	Signs
Weakness, tiredness	Dry coarse skin, cool extremities
Dry skin	Puffy face, hands, & feet (myxedema)
Feeling cold	Diffuse alopecia
Hair loss	Bradycardia
Difficulty concentrating & poor memory	Peripheral edema
Constipation	Delayed tendon reflex relaxation
Weight gain & poor appetite	Carpal tunnel syndrome[fat accumulation under skin in wrist area cause pressure in median nerve]
Dyspnea	
Hoarse voice	
Menorrhagia	
Paresthesia	

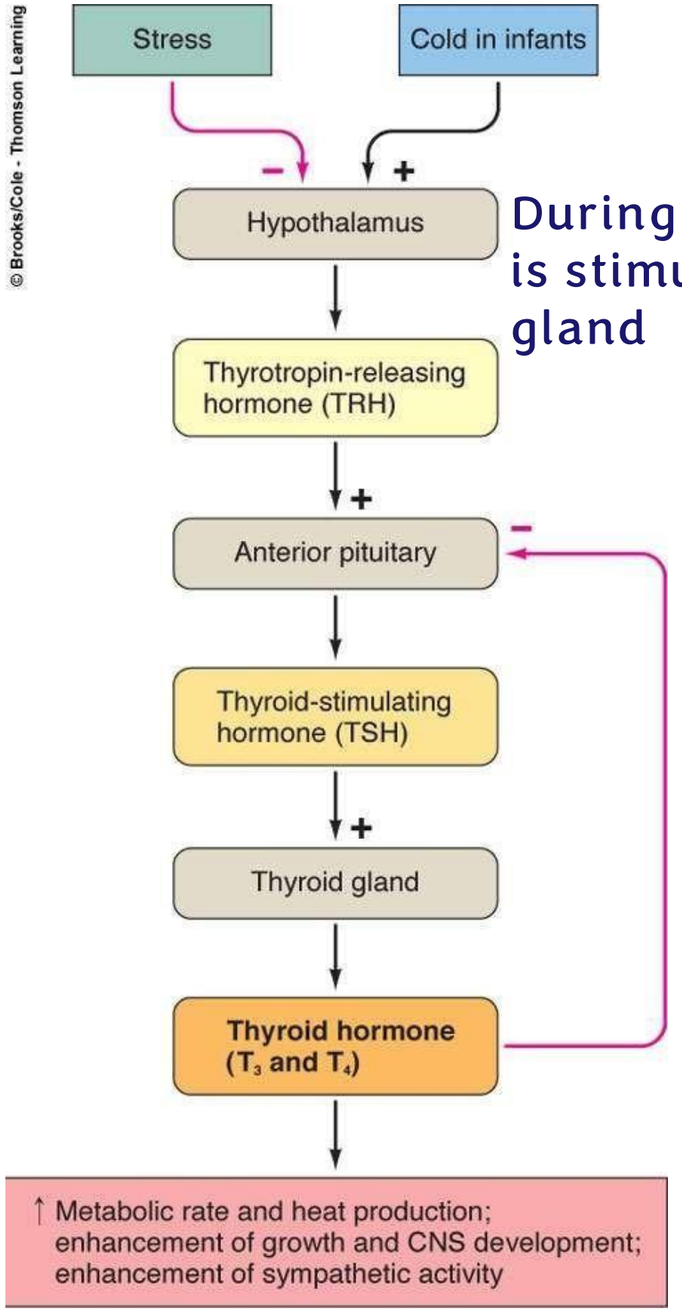
Sluggish: **S**=Sleepiness, fatigue, tiredness **L**=Loss of memory

U=Unusually dry skin

G=Goiter **G**=Gradual personality change **I**=Increase in body weight

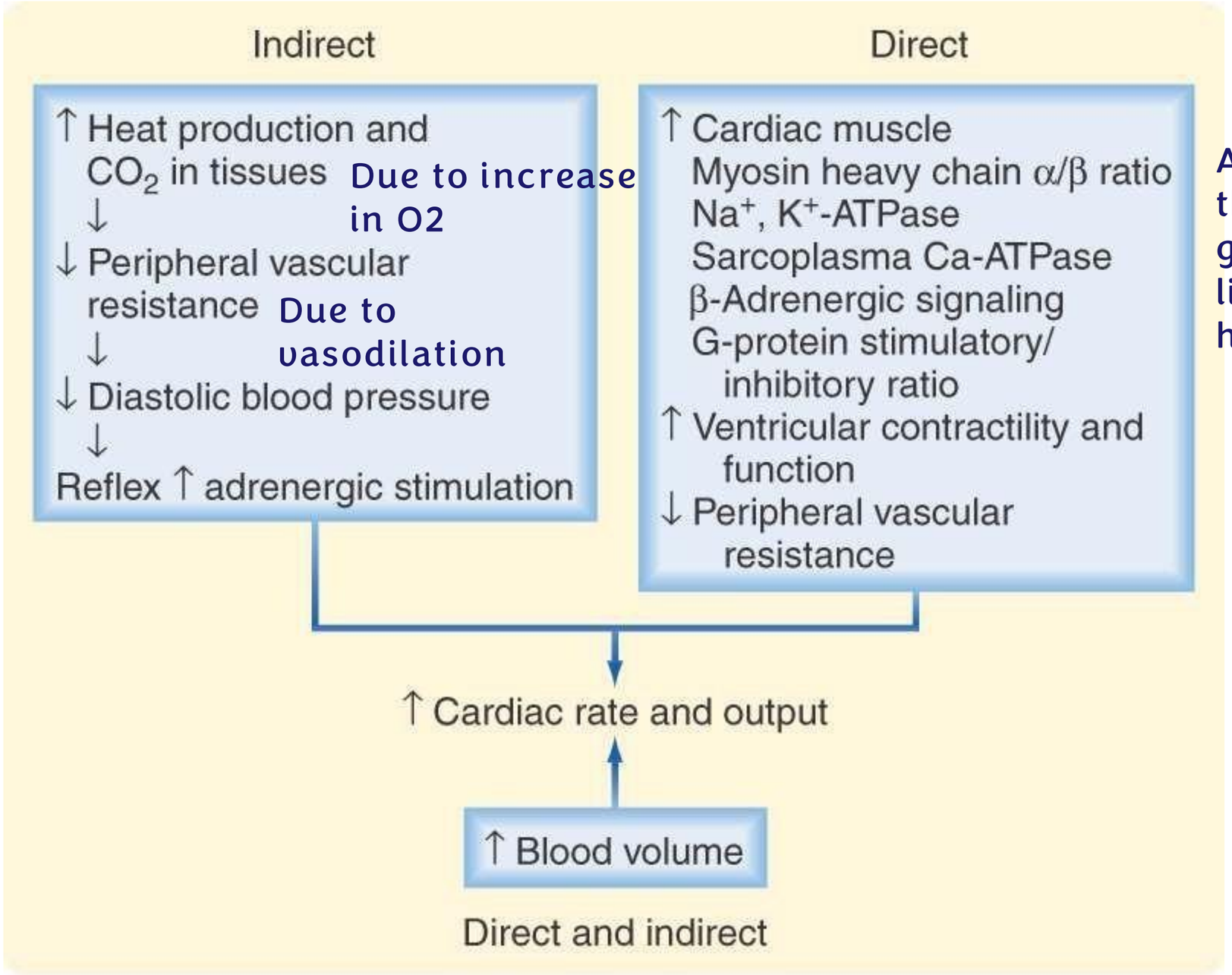
S=Sensitivity to cold **H**=Hair loss, sparseness of hair

Regulation of Thyroid Hormone Secretion



During cold weather there is stimulation of thyroid gland

Action of thyroid hormone can be



As thyroxin is growth like hormone

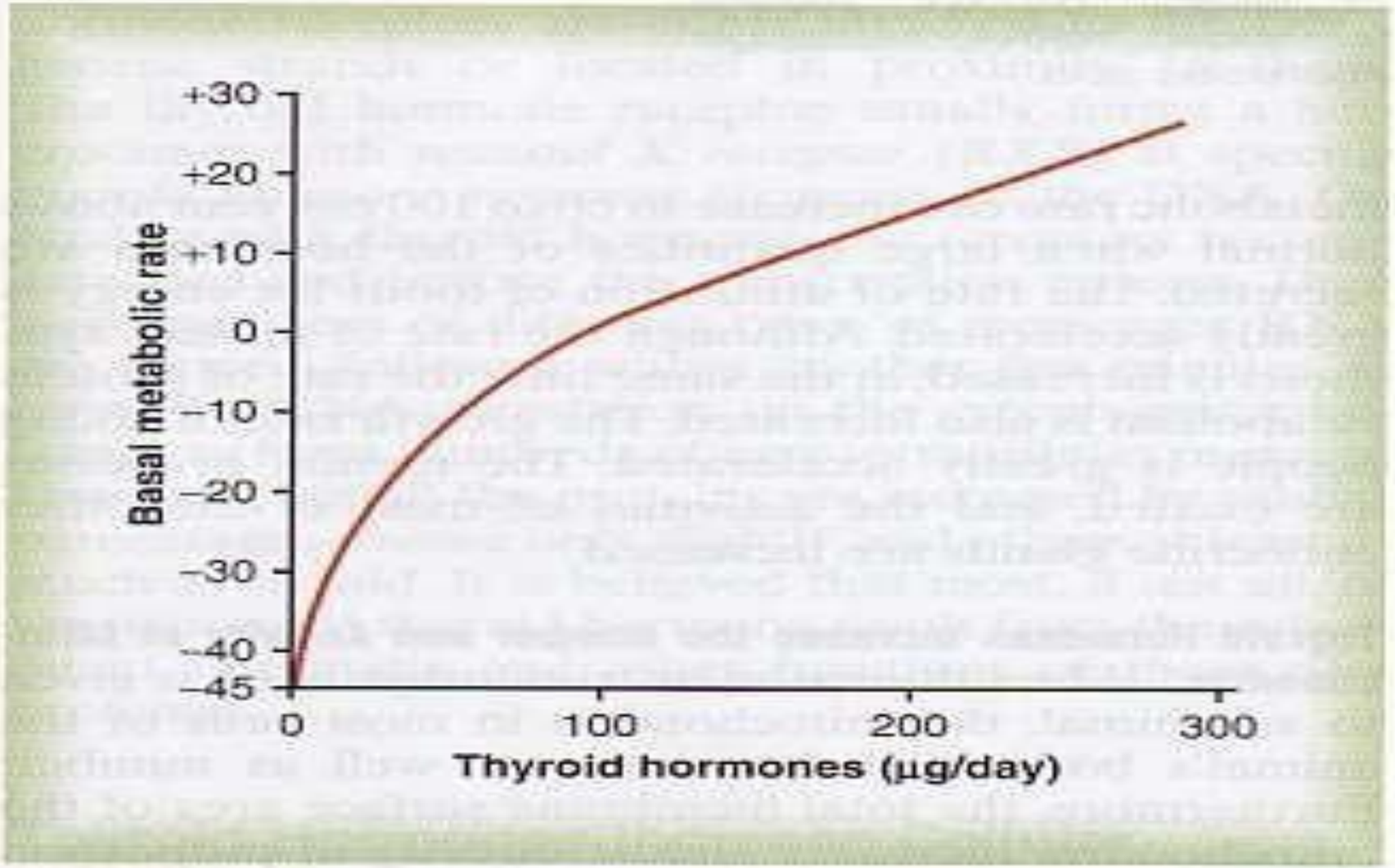


Figure 76–6

Approximate relation of daily rate of thyroid hormone (T_4 and T_3) secretion to the basal metabolic rate.

Increased thyroid hormone causes an increased metabolic rate.
The effect is delayed because thyroxine has a long half-life of about seven days. Therefore, its action is prolonged.

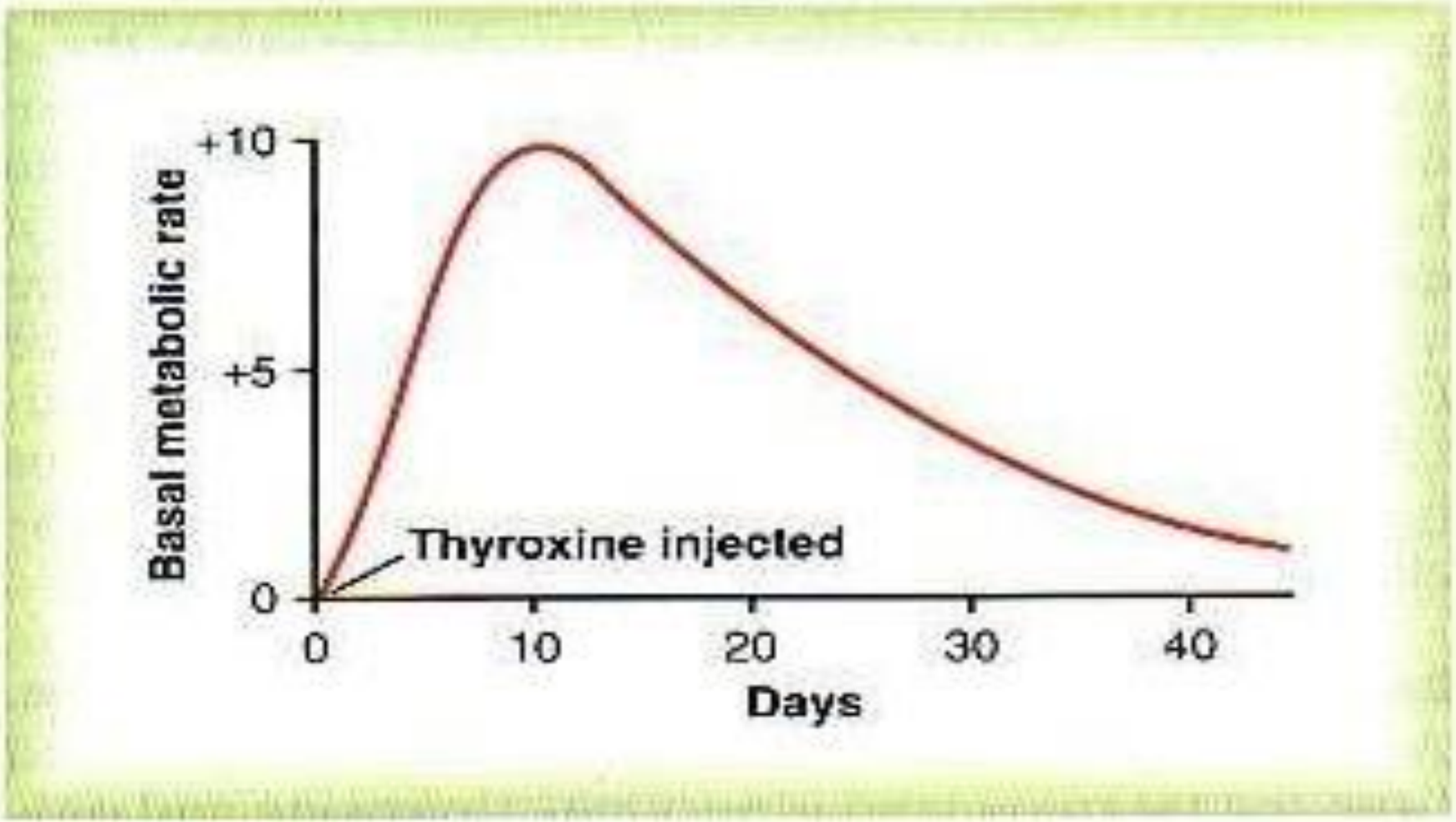


Figure 76–4

Approximate prolonged effect on the basal metabolic rate caused by administering a single large dose of thyroxine.

If another thyroxin dose is injected , BMR will continue to increase

Thank You

Physiology Quiz 5

رسالة من الفريق العلمي:

إنْ طال شوقُ العالمينَ لبعضهم

فالشوقُ نحوكَ لا يُحاطُ مداهُ

صَلَّى عليكَ اللّٰهُ ما رُفِعَ النَّدَاء

وتحرَّكت بـالباقياتِ شِـفاهُ

For any feedback, scan the code or click on it.

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			