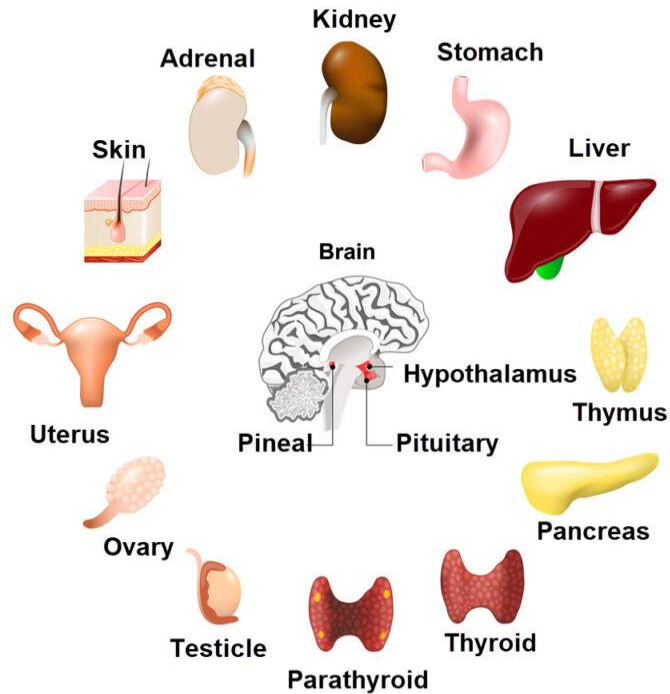


# ENDOCRINE SYSTEM



## Disorders of endocrine system 1

Assistant Professor Ivica Petrovic

Department of Pathophysiology

# Lecture

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1. General endocrinology
  1. Function of endocrine system
  2. Hormones
  3. Organisation
  4. Circadian rhythm
- Special endocrinology
  1. Disorders of hypothalamus
  2. Disorders of hypophyses
  3. Disorders of thyroideidea

# General endocrinology

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# - Function of endocrine system -

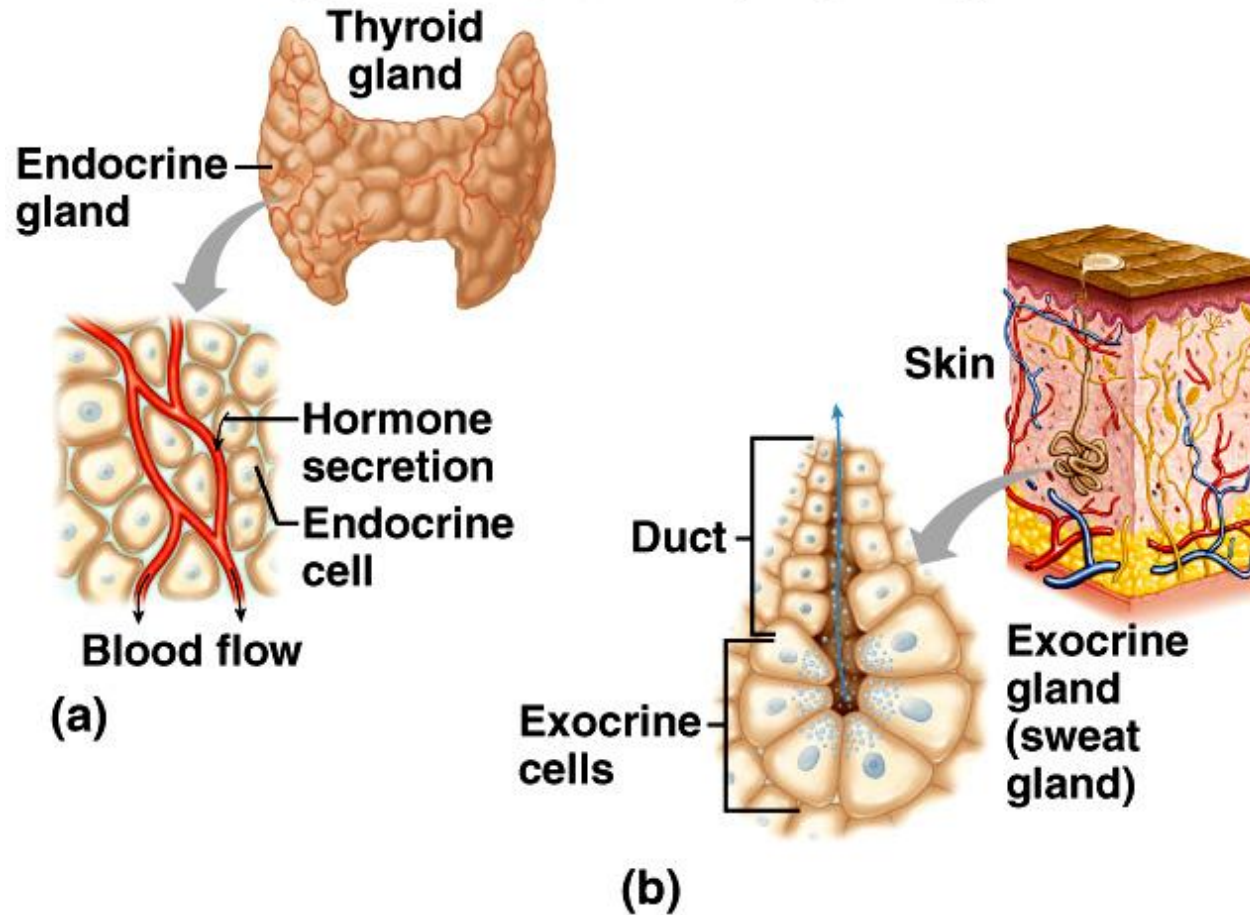
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1. Maintaining homeostasis
2. Growth and body development
3. Reproduction
4. Stress response
5. Energy metabolism
6. Maintaining the balance of water and acid-base status

# - Endocrine vs exocrine glands -

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# - Hormones -

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- Molecules produced by the endocrine gland or tissue which is secreted out of the cells and is transported to its target cells by a circulating blood
- Hormones influence their target cells to respond in a specific way, normally to the benefit of the organism (in physiological conditions and also during the state of compensatory response).
- The hormone transfer the message from one part of the body to another, and can therefore be considered as a messenger molecule.

# General endocrinology

## - Hormones -

**Table 1.1** The principal hormones, together with their chemical group and main sites of synthesis.

Hormone type	Examples	Main endocrine gland
Amino acid or amino acid derived	Thyroxine (T4) and tri-iodothyronine (T3)	Thyroid
	Adrenaline and noradrenaline	Adrenal medulla
	Dopamine	Hypothalamus
Polypeptide	Insulin	Pancreas
	Glucagon	Pancreas
	Vasopressin	Neurohypophysis
	Oxytocin	Neurohypophysis
	Corticotrophin	Adenohypophysis
	Calcitonin	Thyroid parafollicular cells
	Parathormone	Parathyroid glands
	Somatostatin	Hypothalamus
	Corticotrophin-releasing hormone	Hypothalamus
	Thyrotrophin-releasing hormone	Hypothalamus
	Gonadotrophin-releasing hormone	Hypothalamus
	Inhibin	Testis, ovary
	Activin	Ovary
	Angiotensin II	Blood
	Atrial natriuretic peptide	Heart
Protein	Somatotrophin	Adenohypophysis
	Prolactin	Adenohypophysis
	Erythropoietin	Kidney
	Cytokines (various, e.g. interleukins)	Immune system cells
	Leptin	Adipose tissue
	Ghrelin	Stomach

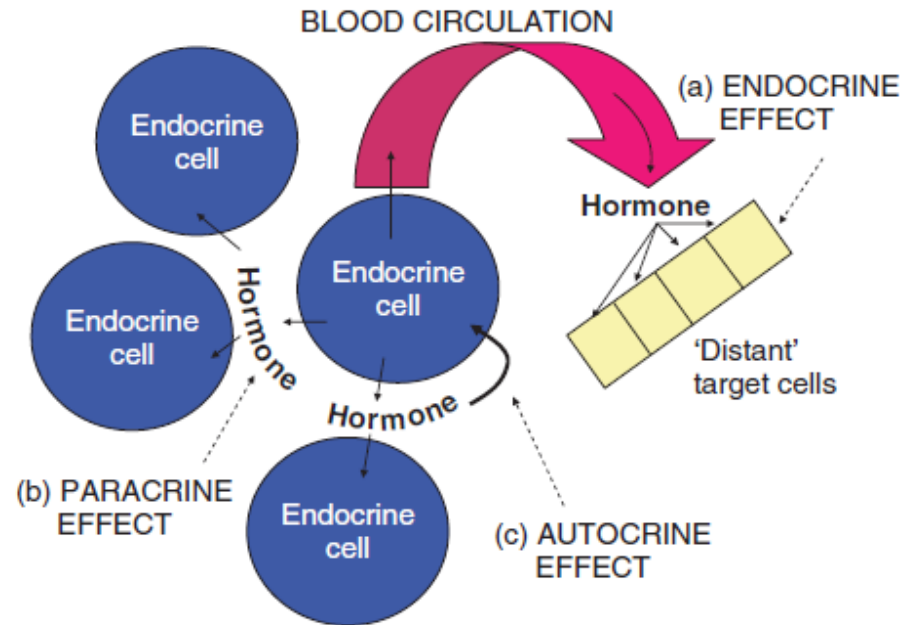
**Table 1.1** The principal hormones, together with their chemical group and main sites of synthesis.

Hormone type	Examples	Main endocrine gland
Glycoproteins	Thyrotrophin	Adenohypophysis
	Luteinising hormone	Adenohypophysis
	Follicle-stimulating hormone	Adenohypophysis
Steroids	Aldosterone	Adrenal
	Cortisol	Adrenal
	Testosterone	Testis
	17 $\beta$ -oestradiol	Ovary
	Oestrone	Ovary
	Progesterone	Ovary
	Calcitriol	Kidney

# - Effect of hormones -

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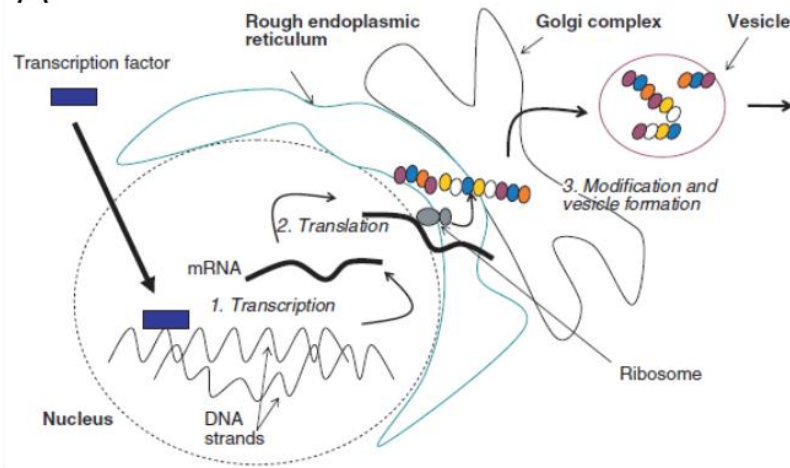
1. Autocrine effect
2. Paracrine effect
3. Endocrine effect





# - Types of secretion -

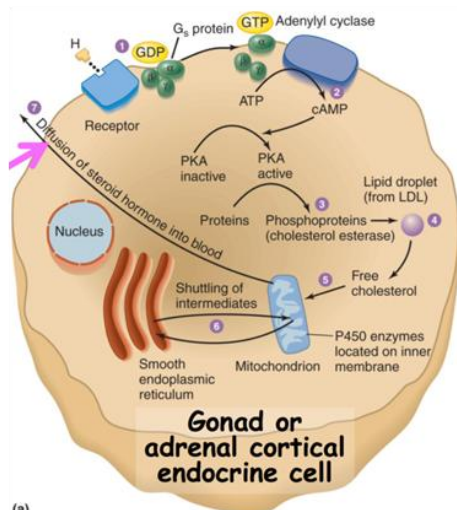
A



Without  
depot in  
blood

Blood  
vessel

B



(a)

Depot in  
blood

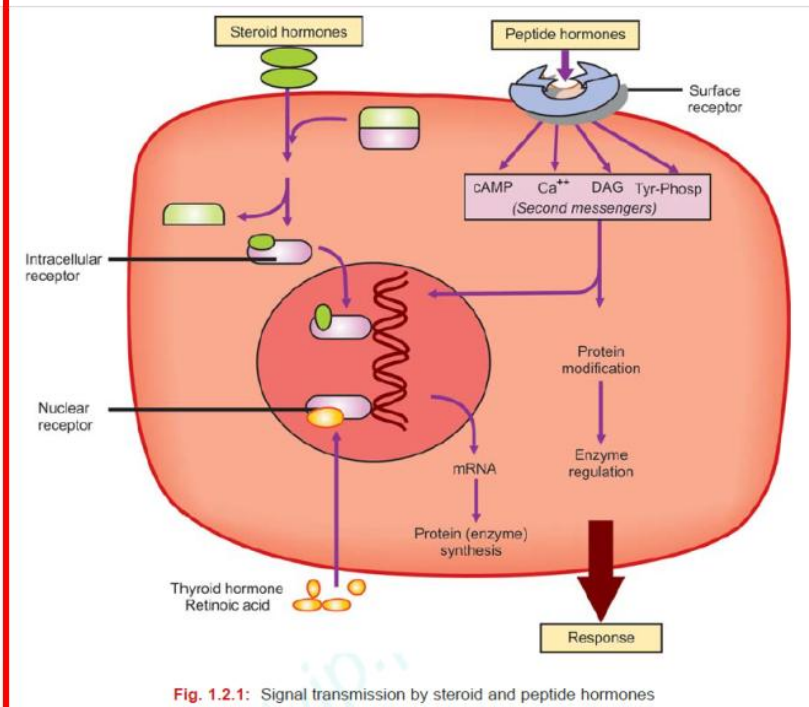
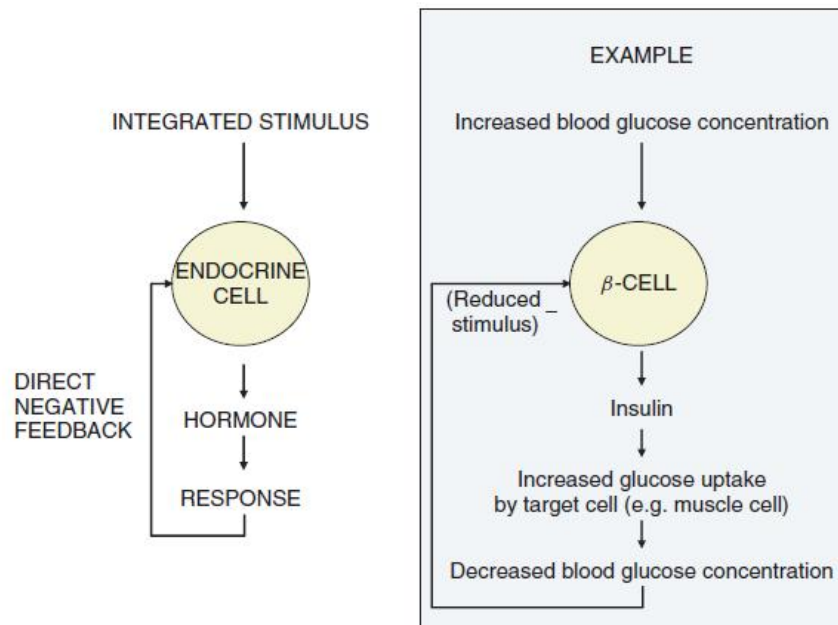


Fig. 1.2.1: Signal transmission by steroid and peptide hormones

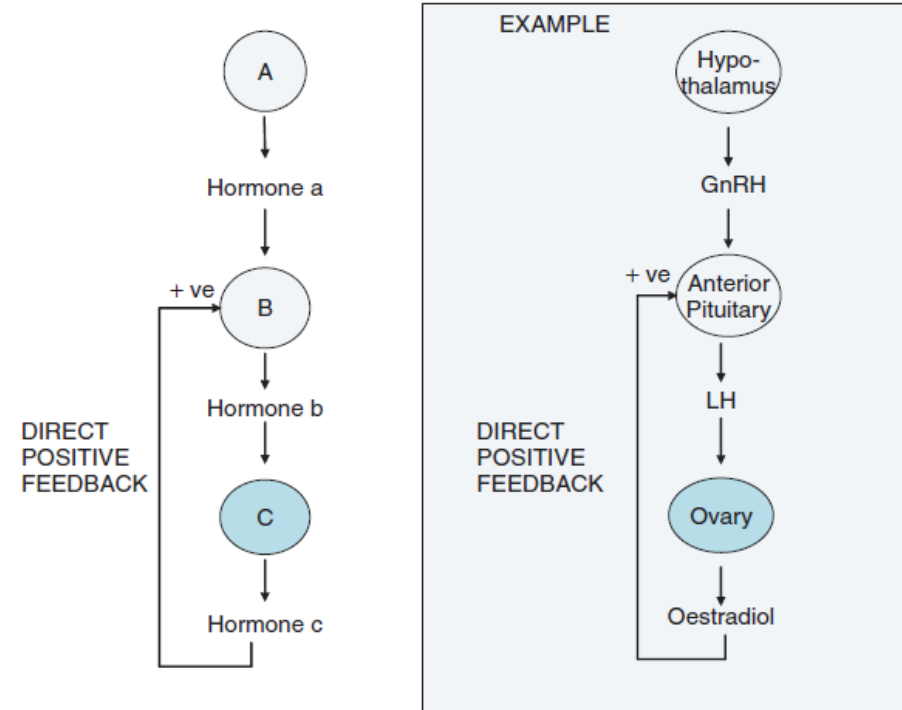
# - Secretion control mechanisms -

## Negative feedback



**Figure 1.16** Diagram illustrating (left) the general principle of a direct negative feedback loop, and (right) an example provided by the hormone insulin.

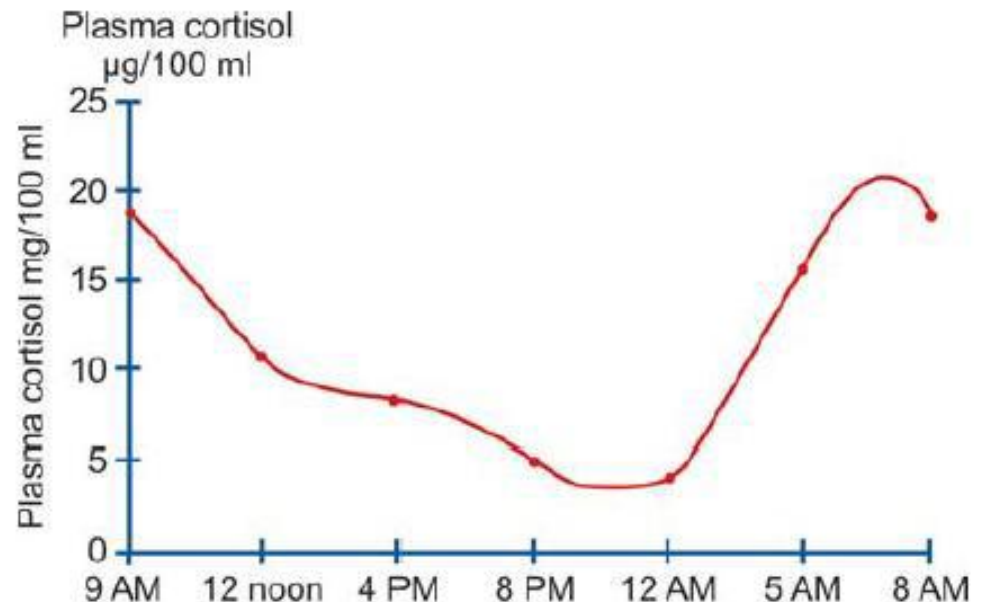
## Positive feedback



# - Circadian rhythm -

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- Individual patterns of secretion of each hormone
- Important time when to check the concentration
- Pooled serum
- Brownil blood sampling



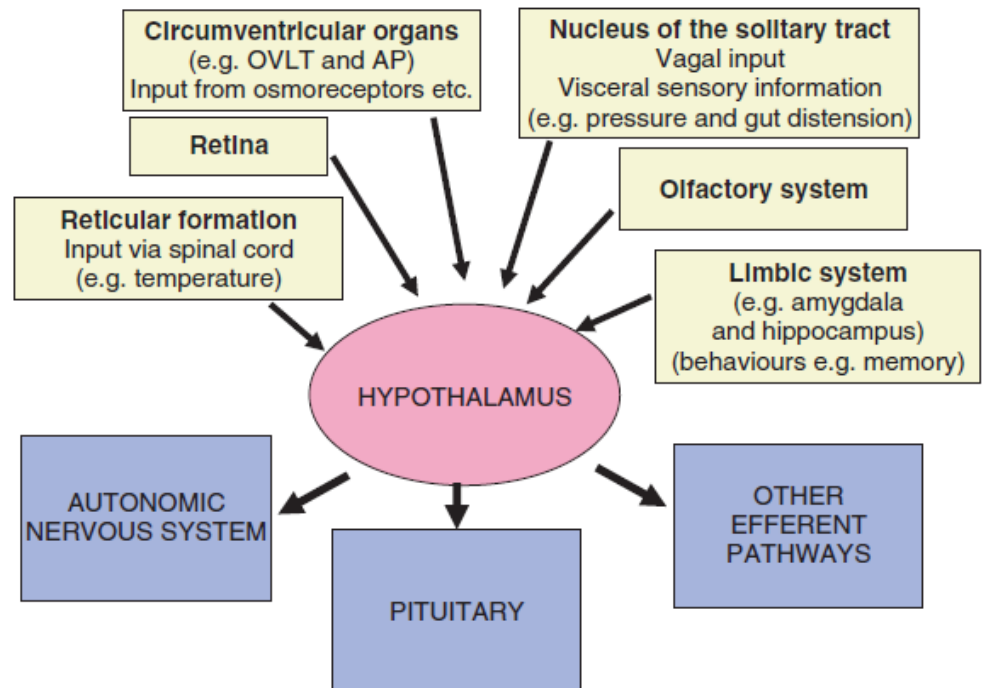
# Special endocrinology

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# Neuroendocrine interaction

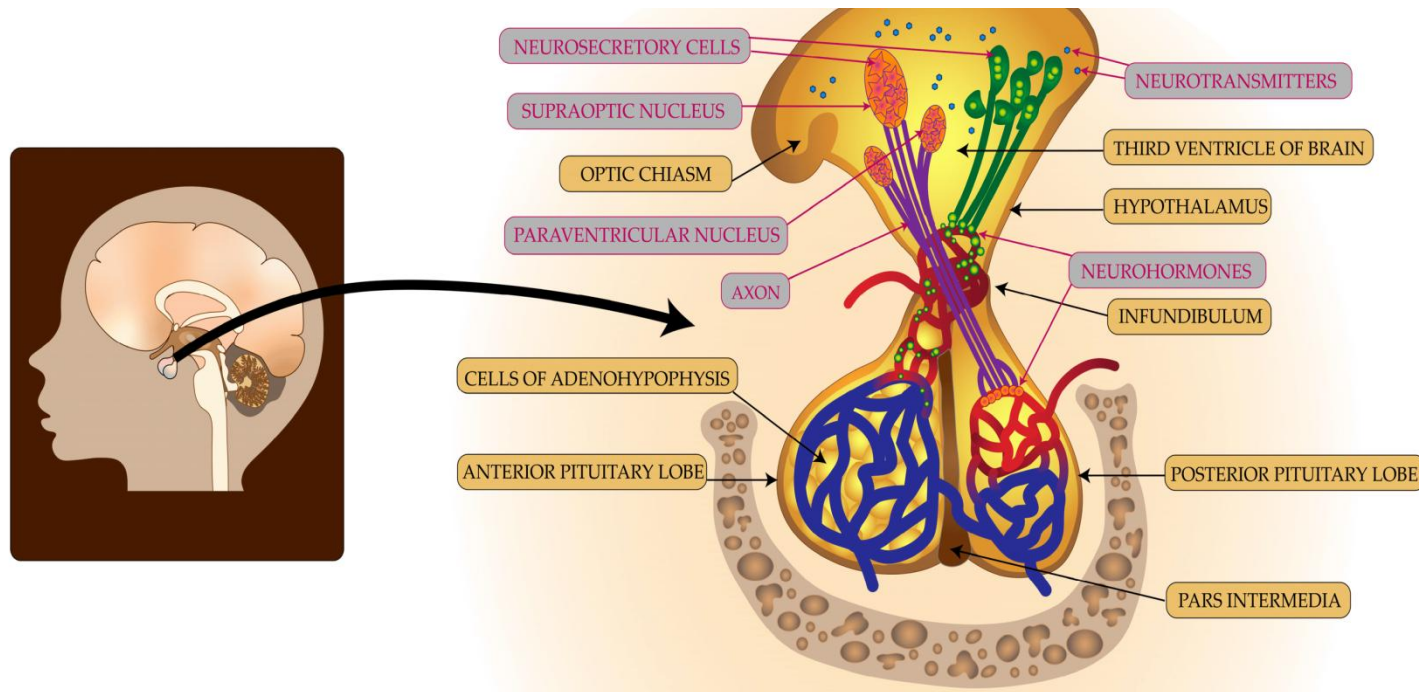
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- Hypothalamus - common part of CNS and endocrine system
- Very important for neuroendocrine interaction and synchronized action



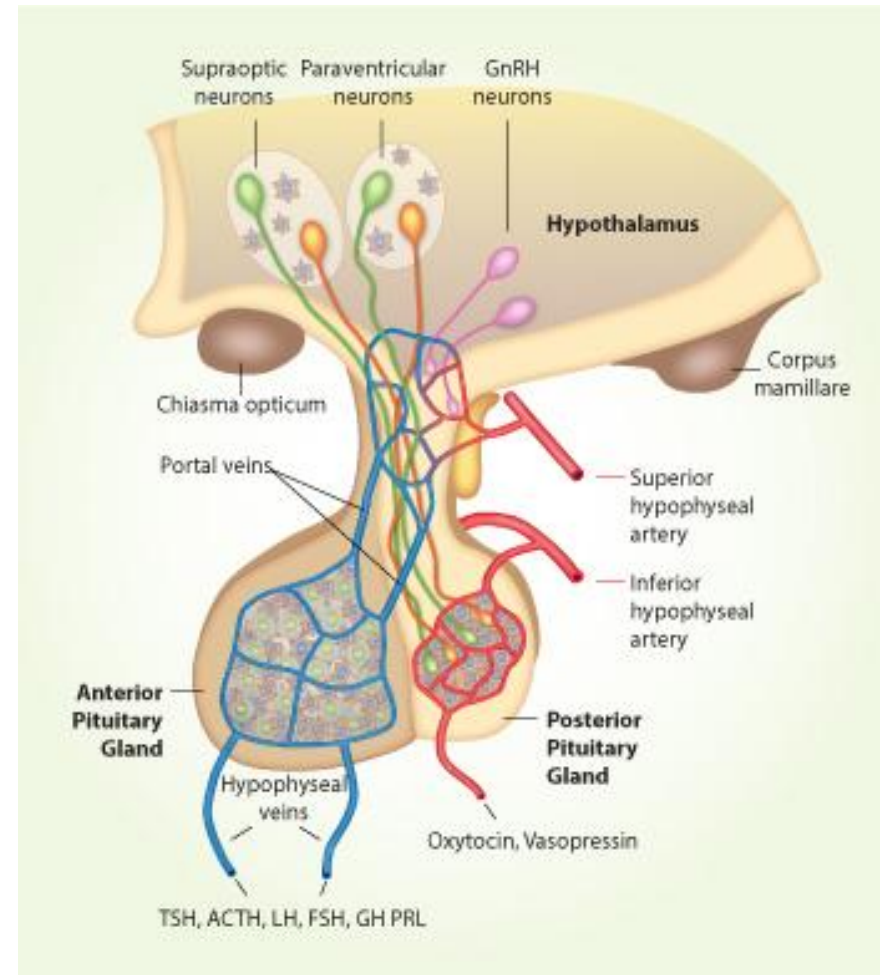
# Hypothalamo-pituitary axis

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# Hypothalamo-pituitary axis

- Portal vein system
- Very low concentrations of releasing hormone or factors
- They are not routinely determined from blood



# Functions of hypothalamus

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## Endocrine

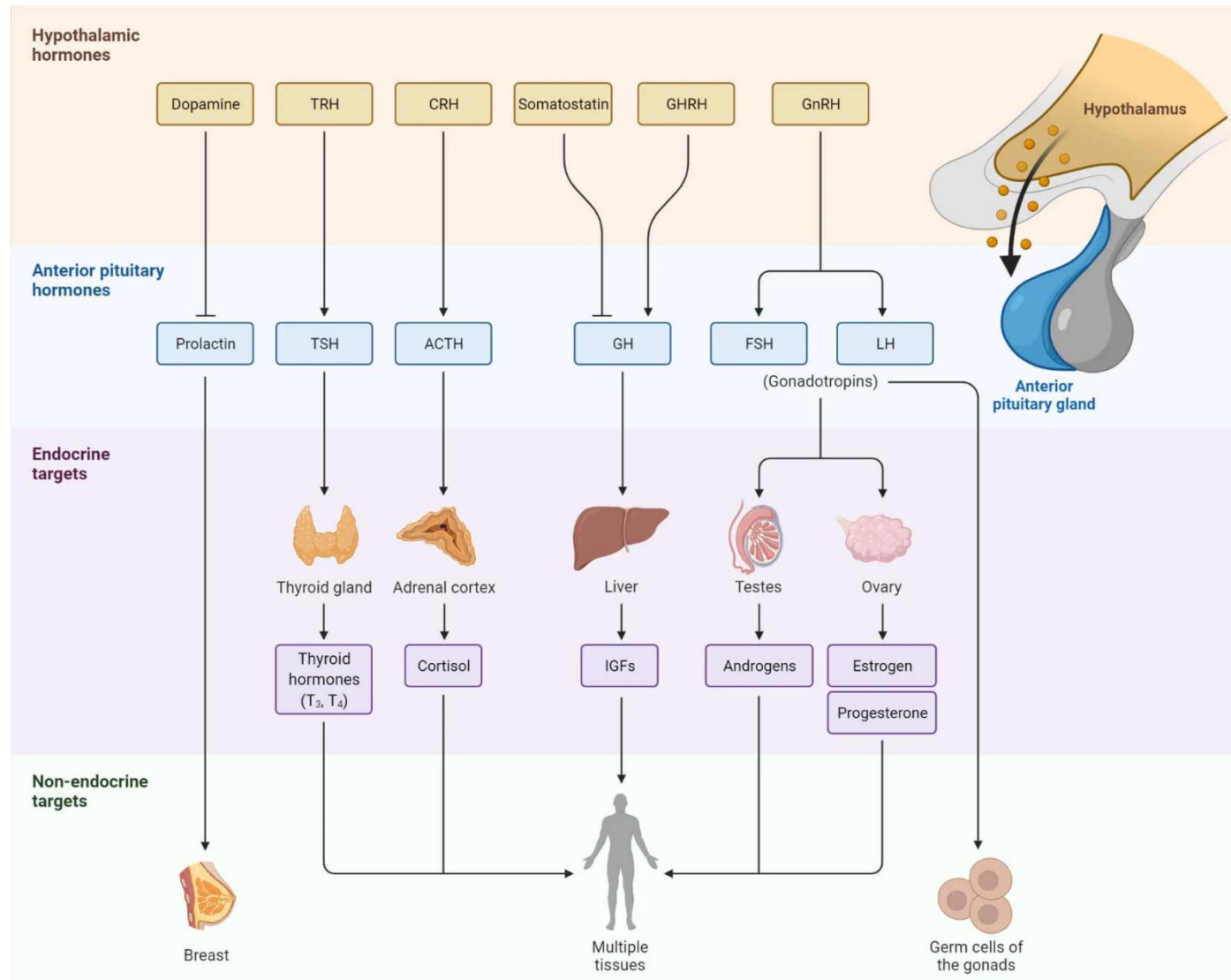
- Secretion of stimulatory molecules
  - Corticotropin releasing factor
  - Thyrotropin releasing factor
  - Gonadotropin releasing factor
  - Somatotrophic releasing factor
- Secretion of inhibitory molecules
  - Somatostatin
  - Dopamine
- Pars posterior hypophysis
  - Vasopressin
  - Oxytocin

## Nonendocrine

- Regulation
  - Sleeping
  - Sexual behavior
  - Temperature
  - Thirst
  - Hunger



# Endocrine function of hypothalamus



# Disorders of hypothalamus

# Disorders of the hypothalamus

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- 1. Changes in the functions of the pituitary gland (in hormonal secretions - endocrine disorders)**
- 2. Nonendocrine disorders:**
  - Behavior
  - Appetite regulation
  - Regulation of body temperature

# Disorders of the hypothalamus

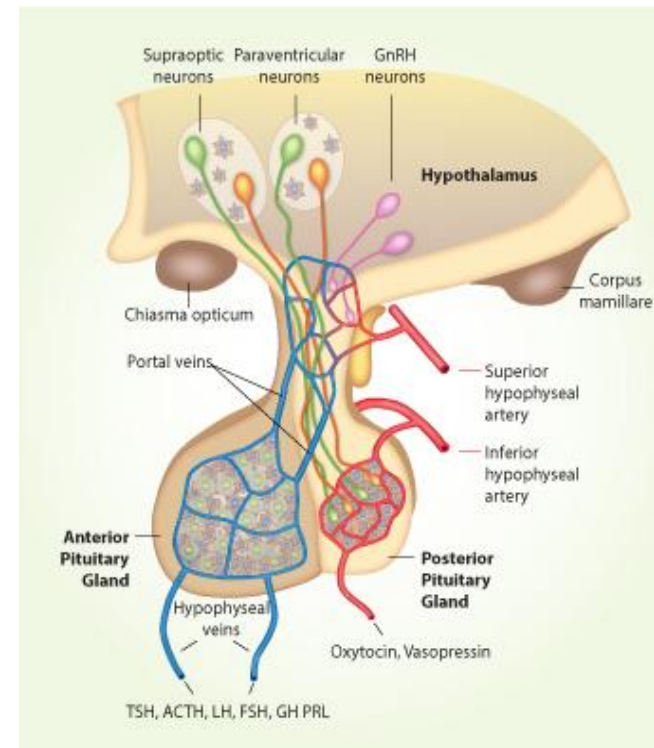
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- **Pituitary dysfunction**
- Neuropsychiatric and behavioral disorders (sleep disorders, consciousness disorders, water intake disorders, behavioral disorders, etc.)
- Disorders of the regulation of the autonomic nervous system (cardiac arrhythmias, sphincter disorders...)
- Metabolic regulation disorders (temperature regulation disorders, food intake disorders, etc.)

# Disorders of the hypothalamus

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- **The syndrome of "pituitary isolation"** (for ex. during lesion of the pituitary stalk) is manifested by:
  1. Hypothalamic hormone deficiency with absence of pituitary hormone secretion
  2. Diabetes insipidus



# The syndrome of "pituitary isolation"

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The result of **destructive processes in the sellar region**, most often in the area of the pituitary gland

**Etiology:** head injuries, surgical interventions, tumors or granulomas

Clinical manifestations:

1. **Diabetes insipidus** (vasopressin deficiency)
2. **Hypophysiotropic hormone deficiency:**
  - CRF (deficiency of ACTH and cortisol)
  - GnRH (deficiency of FSH and LH, menstrual disorders)
  - TRH (deficiency of TSH and T3 and T4)
  - GHRH (GH deficiency)
  - Dopamine (loss of inhibitory effect and increase in PRL secretion)

# Hypothalamic hormone deficiency

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- **Hypothalamic hormone deficiency can be:**
  1. isolated (one hormone missing) or
  2. multiple (missing multiple hormones)
- **Etiology:**
  1. genetic and developmental disorders
  2. tumors
  3. granulomas
  4. brain injuries (traffic trauma)
  5. chemotherapy and radiotherapy of tumors

# Isolated hypothalamic hormone deficiency

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1. **Isolated GnRH deficiency (the most common disorder) leading to hypogonadotropic hypogonadism:-**
  - in children it leads to Pubertas tarda (delayed puberty)
  - in adults to hypogonadism (secondary amenorrhea, decrease in libido, erectile dysfunction, and after a long time loss of secondary sexual characteristics)
- **Kallmann syndrome (Sy Kallmann):**

a developmental disorder in which the disorder of the development of GnRH neurons connected with the disorder of the olfactory lobe

  - clinical presentation: hypogonadotropic hypogonadism and anosmia/hypoosmia (lack of/reduced sense of smell)



# Isolated hypothalamic hormone deficiency

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- 2. Isolated TRH deficiency leads to central hypothyroidism (pituitary hypothyroidism)**
  - there is an inadequate synthesis of TSH, and therefore the synthesis of thyroid hormones (T3 and T4)
- 3. Isolated GHRN deficiency leads to GH (growth hormone) deficiency**
  - the consequence is low but symmetrical growth of children
- 4. Isolated CRH deficiency leads to decreased synthesis of ACTH, and then decreased cortisol production**
- 5. Dopamine deficit from the hypothalamus will lead to increased production of PRL (hyperprolactinemia)**

# Isolated hypothalamic hormone hypersecretion

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1. **Hypersecretion of GnRH leads to Pubertas praecox (early puberty) activation of the pituitary-gonadal axis too early:**
  - appearance of the menstrual cycle in girls before the age of 8
  - appearance of spermatogenesis in boys before 9 or 10 years of age
- **Etiology: hypothalamic tumors (most often GnRH secreting hamartoma)**

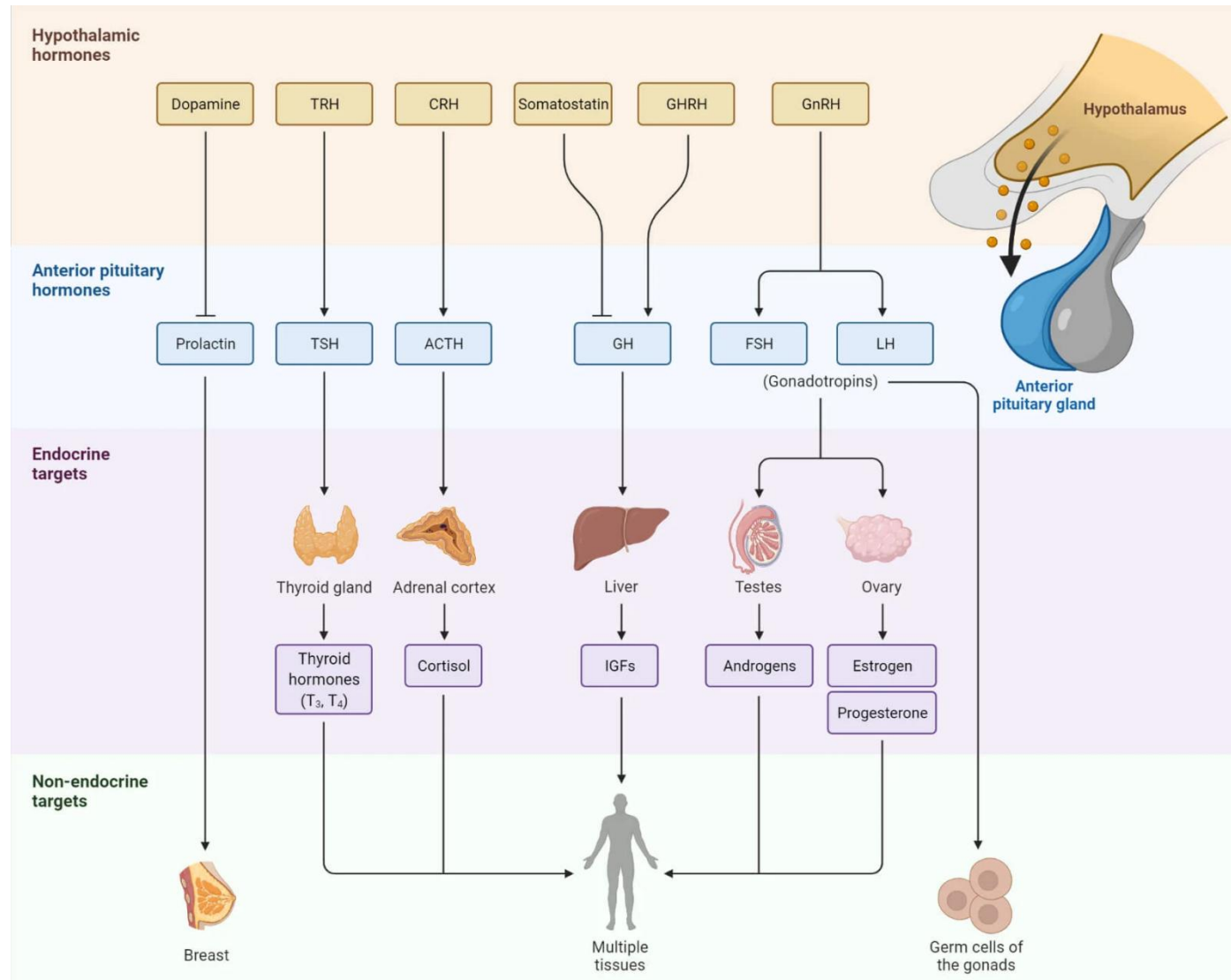
# Isolated hypothalamic hormone hypersecretion

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2. **Hypersecretion of GHRN leads to increased production of growth hormone (acromegaly)**
  3. **Hypersecretion of CRH leads to hypercorticism - one form of Cushing's syndrome (Mb Cushing)**
- **The most common causes of both forms of hypothalamic hypersecretion are:**
    - tumors of the hypothalamus (GHRN and CRH secreting tumor) and
    - paraneoplastic syndromes in carcinoma of the bronchus and pancreas (which ectopically produce CRF and GHRH).

# Disorders of hypophyses

# Endocrine function of hypothalamus



# Disorders of the adenohypophysis

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- 1. Disorders of the anterior lobe of the pituitary gland:**
  - reduced function (hypopituitarism)
  - increased function
- 2. Disorders of the posterior lobe of the pituitary gland:**
  - reduced function
  - increased function

# Disorders of the adenohypophysis

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- **Decreased function of the adenohypophysis:**
  - Panhypopituitarism
  - monotropic hypopituitarism
  - (prolactin or **STH<LH and FSH<TSH<ACTH**)
- **Increased function of the adenohypophysis:**
  - increased growth hormone secretion
  - increased secretion of prolactin
  - increased secretion of TSH
  - increased secretion of ACTH

# **Etiology of hypopituitarismus**

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- 1. Disorders at the hypothalamic level**
- 2. Disorders in the pituitary gland itself:**
  - vascular disorders (postpartal necrosis, vasculitis, aneurysm)
  - tumors
  - infections (tuberculosis, lues meningitis)
  - iatrogenic causes (surgical procedures, radiation)
- 3. Idiopathic disorders (of unknown cause)**



# Consequences of adenohypophysis hormone deficiency

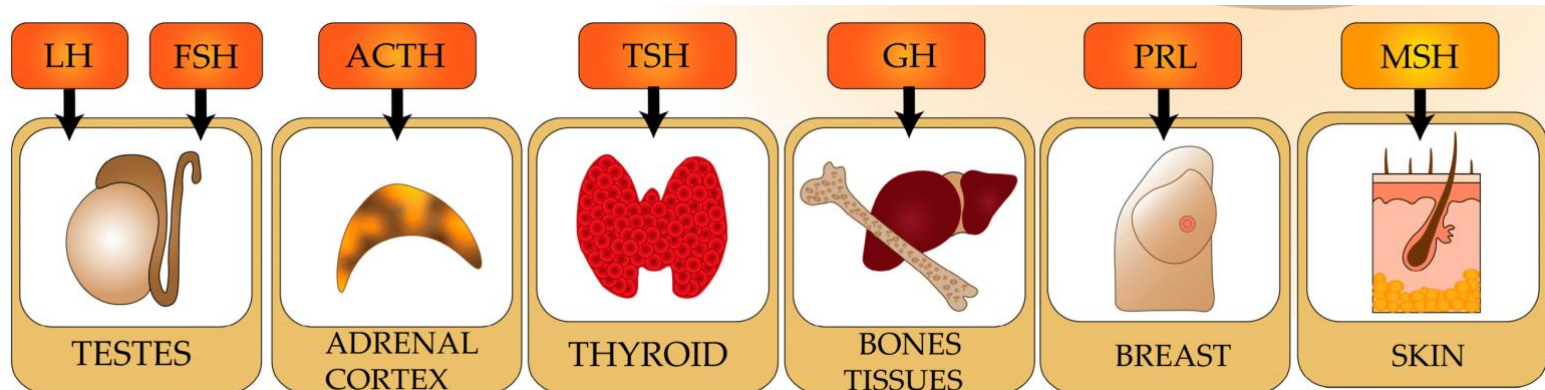
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- They arise when 70-75% of the anterior lobe of the pituitary gland is destroyed
- Depends on:
  - the age of the patient
  - the speed of occurrence and duration of the disturbance
  - a hormone whose secretion is reduced
- **STH < LH and FSH < TSH < ACTH**

# Consequences of adenohypophysis hormone deficiency

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- Lack of growth hormones
- Lack of gonadotropins (LH and FSH)
- Lack of ACTH
- Lack of TSH
- Lack of prolactin



# Consequences of adenohypophysis hormone deficiency in childhood

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- Lack of growth hormone or secretion of a non-functional hormone
  - causes short, symmetrical, dwarf growth: pituitary (pituitary) nanosomia
- Growth hormone deficiency is rarely encountered as an isolated disorder
  - It is usually associated with gonadotropin deficiency
    - in this case, the epiphyseal plates at the ends of the bones do not close during puberty and growth is prolonged, but always substantially behind/low the normal

# Consequences of adenohypophysis hormone deficiency: LH and FSH

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- 1. In childhood or before and during puberty:**  
absence of changes characteristic of puberty  
(secondary sexual characteristics and infertility)
- 2. In adults:** secondary hypogonadism in:
  - woman: secondary amenorrhea (loss of menstruation), signs of reduced estrogen action (atrophy of the breasts, vaginal epithelium and uterus)
  - men: signs of decreased testosterone activity (decrease in libido, potency, muscle tone and hairiness)

# Consequences of adenohypophysis hormone deficiency: TSH, ACTH, prolactin

- A lack:
  1. TSH: secondary hypothyroidism
  2. ACTH: secondary adrenal hypofunction  
(difference from Addison's disease:  
aldosterone secretion is normal)
  3. prolactin: absence of lactation after  
childbirth, e.g. postpartum pituitary necrosis  
caused by hemorrhagic shock (Sheehan's  
syndrome)

# Increased function of the adenohypophysis

---

- The most common cause: functional tumors of the pituitary gland (microadenomas and macroadenomas) that secrete hormones
- Usually the increased amount of only one hormone
  - most often prolactin, growth hormone or ACTH
  - very rarely glycoprotein hormones of the pituitary gland: TSH, FSH and LH

# Increased function of the adenohypophysis

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1. Increased secretion of growth hormones:
  - before puberty: gigantism (Before fusion of epiphyseal plates of long bones)
  - after puberty: acromegaly (After fusion of epiphyseal plates of long bones:)
2. Increased secretion of prolactin
3. Increased gonadotropin secretion before puberty:
  - true precocious puberty
4. Increased secretion of ACTH: Cushing's disease
5. Increased secretion of TSH: secondary hyperthyroidism

# Increased function of the adenohypophysis

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- Acromegaly:
  - Enlargement of the short bones of the hand and foot-
  - Enlargement of the membranous bones of the face and skull
  - Growth of cartilage and bone:
    1. bone tissue is created in the subperiosteum,
    2. articular cartilage thickens and degenerates
    3. due to thickening of the cartilage of the larynx and airways: the color of the voice changes
- Enlargement of internal organs: liver, spleen, kidneys (visceromegaly)
- Cardiomegaly (due to the effect of STH and hypertension)
- Reduced utilization of glucose (hyperglycemia)



# Hyperprolactinaemia

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- Definition: Elevated level of PRL in the blood
- Etiology:
  - Adenoma of pituitary cells that produce prolactin
  - Dopamine deficiency
    1. pituitary isolation syndrome,
    2. antidopaminergic drugs
  - Other:
    1. excess estrogen,
    2. primary hypothyroidism,
    3. chronic renal insufficiency
- Pathogenesis: autonomous secretion of PRL in the adenoma, loss of inhibitory effect via D2 receptors

# Hyperprolactinaemia

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- Clinical picture:
- headaches, visual field disturbance
  - + Women:
    - menstrual disorder (oligo to amenorrhea)
    - Infertility
    - Galactorrhea
  - + Men:
    - libido disorder
    - sexual dysfunction

# Neurohypophysis function disorders:

## ADH

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- Decrease of ADH secretion (Diabetes insipidus)
  - Etiology: damage to the hypothalamus and pituitary gland (due to tumor growth, inflammatory process, vascular disorder, skull base fracture)
  - Pathogenesis: the cells of the kidney's collecting ducts are impermeable to water, they secrete large amounts of unconcentrated urine (5-15 l), with low specific gravity ( $<1.007$ ).
  - Clinical picture: polyuria and nocturia (nocturnal urination) and polydipsia (increased thirst, accompanied by increased water intake)

# Neurohypophysis function disorders:

## ADH

---

- Decrease or cessation of ADH secretion (Diabetes insipidus)
- 2 types:
  1. Hypothalamic (central) type: insufficient production of ADH (complete or partial, partial)
  2. Nephrogenic (peripheral) type: insensitivity/resistance of renal tubules to ADH

# Neurohypophysis function disorders:

## ADH

---

- "Syndrome of inadequate ADH secretion" (excessive secretion of ADH): Independent of plasma osmolarity
- Occurs due to tumors that secrete ADH or the effect of some drugs
  - effect:
  - water retention, with hemodilution and hyponatremia
  - increase in plasma volume and glomerular filtration, which causes reduced secretion of aldosterone (loss of sodium in the urine, which increases hyponatremia)
  - clinical symptoms: depend on sodium concentration in plasma

# Disorders of thyreoidea

# Thyroid gland

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- Located below and in front of the larynx
- It has two lobes connected by an isthmus
- Thyroid follicles produce thyroid hormones
  - Thyroxine or tetraiodothyronine (T<sub>4</sub>)
  - Triiodothyronine (T<sub>3</sub>)
  - Both increase the intensity of basal metabolism, stimulate protein synthesis, increase the use of glucose and fatty acids for the production of ATP
- Parafollicular cells or C-cells produce calcitonin–  
Reduces Ca<sup>2+</sup> in the blood by inhibiting bone reabsorption

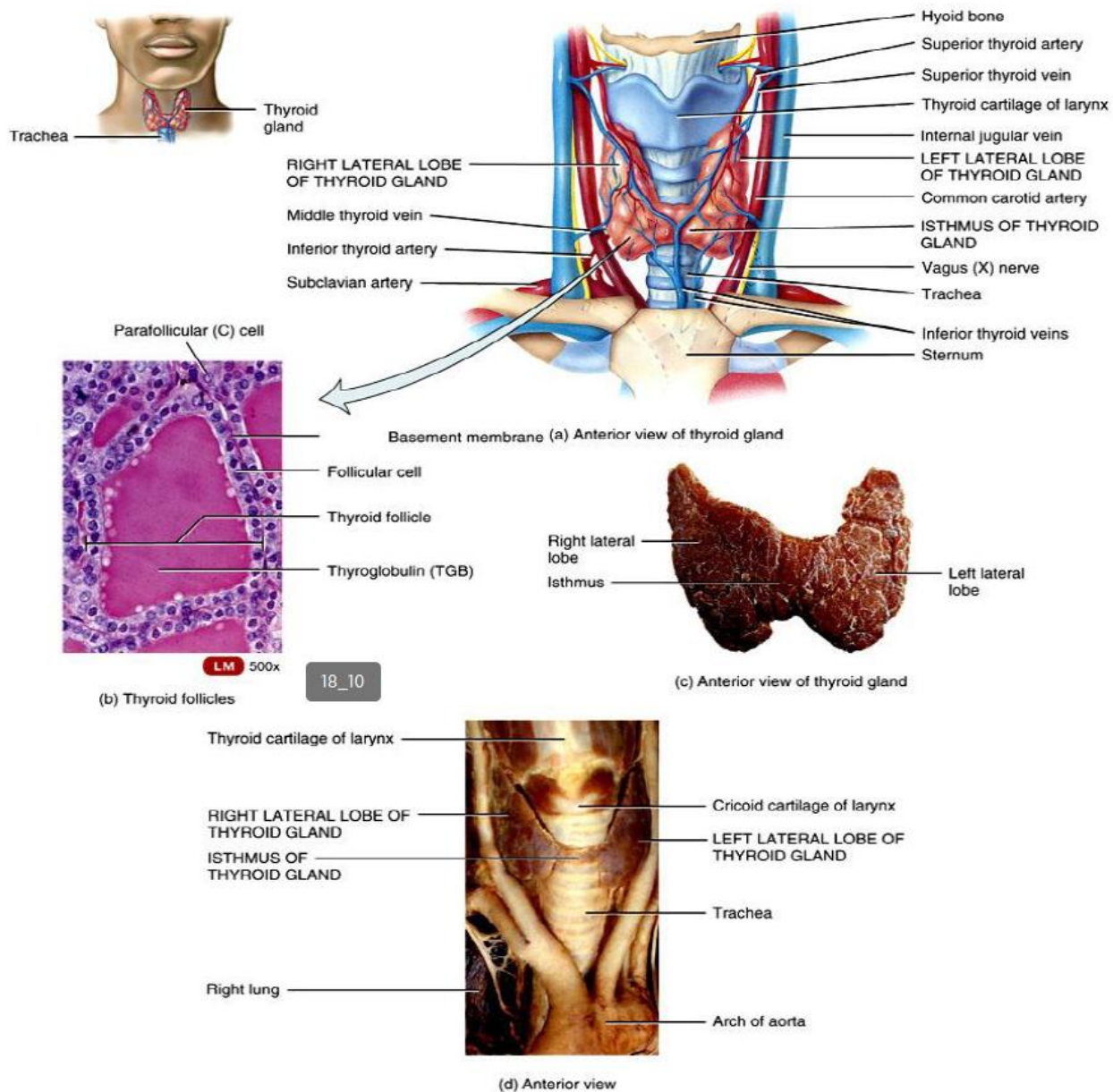


Figure 18.10 Tortora - PAP 12/e  
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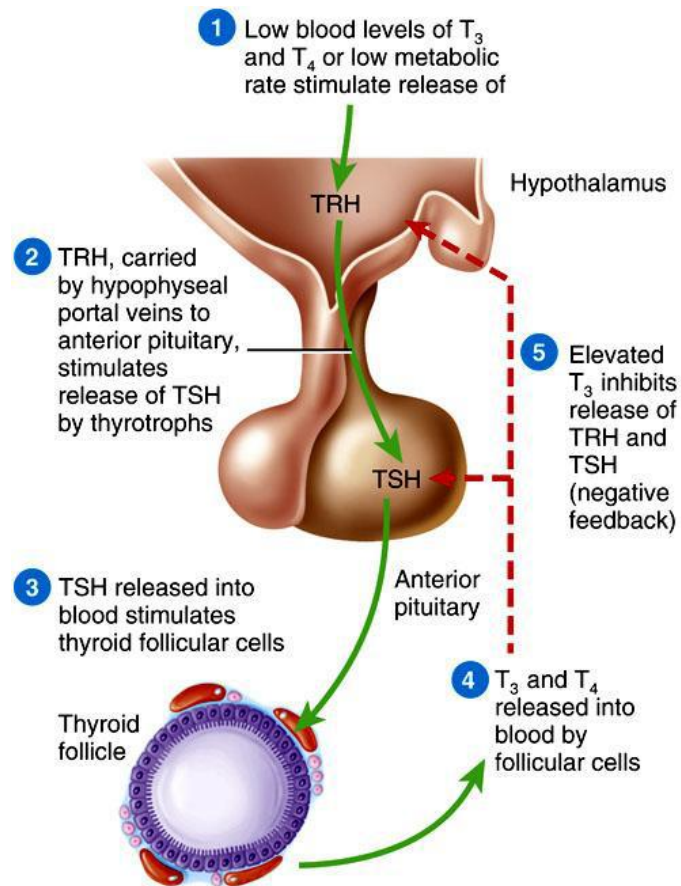


# Control of thyroid hormone secretion

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Is achieved through

1. Thyrotropin-releasing factor (hormone) (TRF or TRH) from the hypothalamus
2. Thyroid-stimulating hormone (TSH) from the adenohypophysis
3. Some conditions in which the need for ATP/energy is increased - lead to an increase in the secretion of thyroid hormones



#### Actions of Thyroid Hormones:

- Increase basal metabolic rate
- Stimulate synthesis of  $Na^+/K^+$  ATPase
- Increase body temperature (calorigenic effect)
- Stimulate protein synthesis
- Increase the use of glucose and fatty acids for ATP production
- Stimulate lipolysis
- Enhance some actions of catecholamines
- Regulate development and growth of nervous tissue and bones

# Thyroid function

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Is achieved through several steps:

1. synthesis and secretion of hormones
2. transfer of hormones to target cells
3. binding to receptors on target cells
4. biological effect of thyroid hormones
5. breakdown and elimination of hormones
6. regulation of the concentration of thyroid hormones (feedback)

# Effect of TSH on thyrocytes

---

- TSH binds to the TSH receptor and activates enzymes:
  1. cAMP triggers:
    - active transport of iodide
    - synthesis of thyroglobulin (Tg) in the ER
  2. thyreoperoxidase (TPO) enables:
    - conversion of iodide into iodine
    - incorporation of iodine into tyrosine and Tg (colloid)
  3. lysosomal enzymes release:
    - T4/T3

# The influence of the amount of iodine on the function of the thyroid gland

- Increased intake of iodine:
  - initially leads to an increased synthesis of thyroid hormones (up to an optimal level)
  - After that, it causes inhibition of iodine organization and hormone synthesis (Wolff-Chaikoff effect) In euthyroid persons, this effect is transient
- In people with latent Graves' disease and polynodous goiter, it can cause the Iodine-Basedow effect
- In people with autoimmune thyroiditis or dyshormonogenesis: iodide-induced hypothyroidism

# Secretion and transport of thyroid hormones

---

- Thyroxine (T<sub>4</sub>)- about 100 nmol is excreted daily
- Triiodothyronine (T<sub>3</sub>)- about 5 nmol is excreted daily - the largest part of T<sub>3</sub>, 80%, is created by peripheral conversion of T<sub>4</sub>
- More than 99% of T<sub>4</sub> and T<sub>3</sub> bound to plasma proteins (enable depot of thyroid hormones)
- Transport proteins:-thyroxine binding globulin (TBG): 70%-albumin: 15-20%, prealbumin: 10-15%

# Effect of thyroid hormones

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- by binding to nuclear receptors, they activate genes that control energy production
- bind to mitochondria, increasing ATP production
- have a caloric effect

# Effect of thyroid hormones

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- they stimulate the function of all organs
- in small doses they act anabolically, but in large doses they act catabolically
- are necessary for brain development and maintaining body temperature,
- increase the consumption of energy and oxygen and the creation of reactive forms of oxygen
- have pronounced inotropic and chronotropic effects on the heart
- speed up intestinal motility
- increase the resorption of bone mass
- speed up muscle contraction and relaxation



# Thyroid gland function disorders

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- **Hypofunction of the thyroid gland**  
(hypothyroidism)
- **Hyperfunction of the thyroid gland**  
(hyperthyroidism)

# Hypothyroidism

---

- **Hypothyroidism** is:
  - a condition resulting from the lack of effects of thyroid hormones due to:
    1. reduced production and release of hormones
    2. resistance of peripheral tissues to the effects of hormones
- **Myxedema** (severe hypothyroidism) refers to skin and subcutaneous tissue changes seen in patients with severe hypothyroidism.

# Hypothyroidism

---

- Etiological division of hypothyroidism:
- **Congenital hypothyroidism:**
  - Aplasia, hypoplasia and ectopia of the thyroid gland
  - Defects in the biosynthesis and/or action of hormones
- **Acquired hypothyroidism:**
  - Autoimmune disease of the thyroid gland (autoimmune thyroiditis)
  - Severe deficit of iodine in the diet
  - Thyroid ablation (surgical interventions, radiation, etc.)
- **Medicated hypothyroidism:**
  - Iodine, PTU, MMI, K-perchlorate, Thiocyanate

# Pathophysiological consequences of thyroid hormone deficiency

---

- slow breakdown of mucopolysaccharides, they infiltrate the dermis, retain water and cause edema (myxedema)
- slow conversion of carotene into vitamin A, accumulation of carotenoid pigment in the skin (yellowish color)
- slowed metabolism (slowed intellectual and motor functions, hypothermia, dry and cold skin, patients gain weight)
- reduced synthesis of beta-adrenergic receptors, reduced sympathetic tone (bradycardia, reduced cardiac output, reduced intestinal motility and constipation, reduced muscle tone and hyporeflexia)
- findings of bradycardia and hypothermia - think of hypothyroidism

# Clinical picture of hypothyroidism

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- Hypothyroidism is an insidious disease that develops slowly, accustoms both the patient and his environment to its existence, brutally numbs the patient, and lulls them all into apathy.

# Etiological division of hyperthyroidism

---

- Etiological division of hyperthyroidism:
  1. Primary hyperthyroidism
    - Autoimmune disease of the thyroid gland (M. Graves, diffuse goiter with orbitopathy)
    - Toxic adenoma (Adenoma toxicum)
    - Multinodular toxic goiter
  2. Secondary hyperthyroidism
    - TSH producing pituitary adenoma
    - Resistance to thyroid hormones
  3. Other
    - Subacute thyroiditis
    - Iatrogenic (use of L-thyroxine)

# Pathophysiological consequences of excess thyroid hormones

---

1. Hypermetabolic state
2. increased synthesis of beta-adrenergic receptors, symptoms and signs of increased sympathetic tone
3. increased concentration of TSHR antibodies and extrathyroid manifestations (thyroid orbitopathy and dermatopathy)

# Pathophysiological consequences of excess thyroid hormones

---

1. Hypermetabolic state:
  - increased energy needs
  - utilization of energy reserves (despite the intake, the patient lose his weight)
  - increased heat generation (skin warm and moist)
  - increased breakdown of glycogen, proteins and fats (glyconeogenesis)



# Pathophysiological consequences of excess thyroid hormones

---

2. Increased synthesis of beta-adrenergic receptors, symptoms and signs of increased sympathetic tone:
  - fast pulse
  - increased cardiac output,
  - reduced peripheral resistance (divergent increase in blood pressure)
  - increased muscle tone, hyperreflexia and tremors
  - accelerated GI motility and diarrhea

# Pathophysiological consequences of excess thyroid hormones

---

3. Increased concentration of TSHR antibodies and extrathyroid manifestations (thyroid orbitopathy and dermatopathy)
  - thyroid orbitopathy (inflammation of extraocular muscles, fat and connective tissue)
  - thyroid dermatopathy (inflammation of the dermal layer of the skin, accumulation of mucopolysaccharides)

# Thyrotoxicosis vs hyperthyroidism vs hypermetabolic state

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1. Thyrotoxicosis is a condition in which the concentration of thyroid hormones in the blood is increased due to:
  - increased production in the thyroid gland
  - released after the destruction of the thyroid gland
  - iatrogenic
2. Hyperthyroidism is a condition in which the concentration of thyroid hormones in the blood is increased due to their increased production in the thyroid gland (only)

# Thyrotoxicosis vs hyperthyroidism vs hypermetabolic state

---

- Hypermetabolic state is a condition in which the concentration of thyroid hormones in the blood is increased due to increased basal metabolism:
- in physiological conditions:
  - after taking food
  - during pregnancy
  - due to physical activity
  - due to sympathetic activation
- in pathological conditions:
  - thyrotoxicosis
  - fever
  - Pheochromocytoma
  - Tumors
  - diabetic ketoacidosis

# Autoimmune Thyroid Disease (AITD)

---

- AITD is a multifactorial disease, the clinical form of which is the result of the interaction of genetic and environmental factors
- Autoimmune disease: response of the immune system to its own antigens (thyroid glands)
- inflammatory disease mediated by immune mechanisms
- Why does an immune response develop against one's own thyroid antigens?

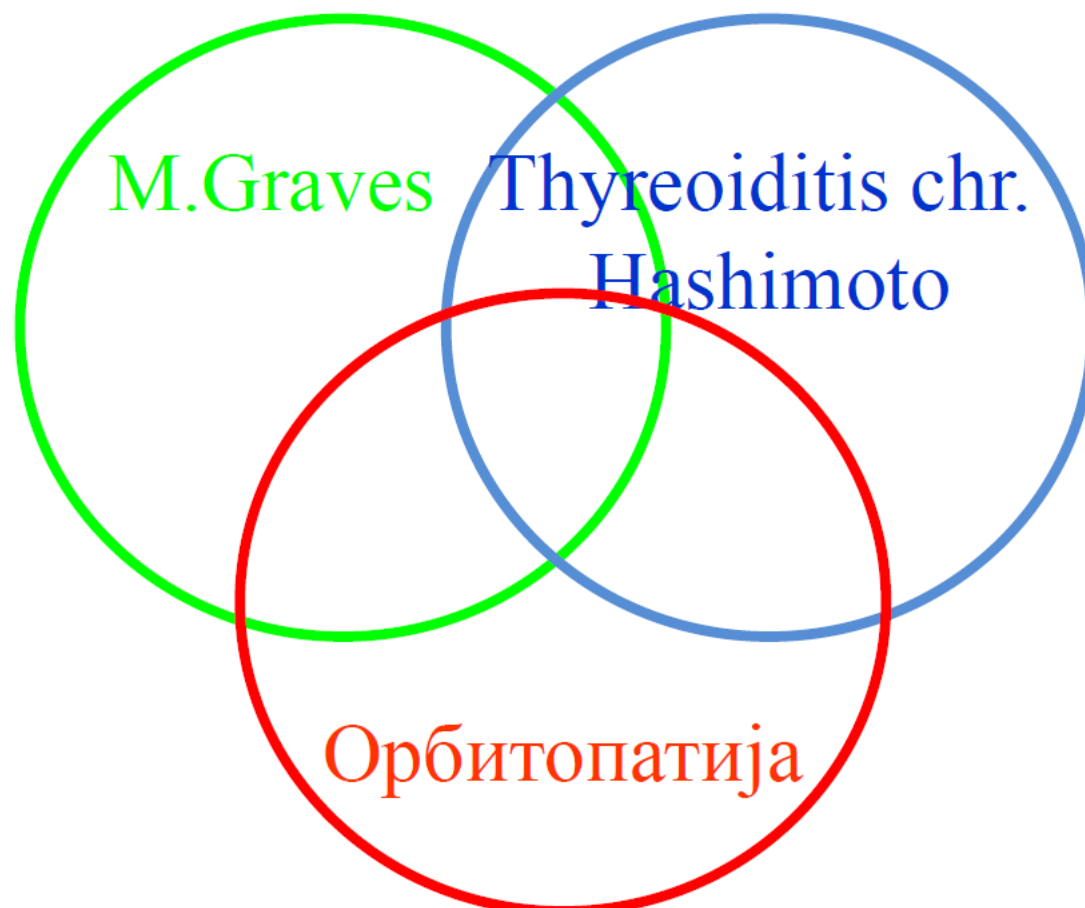
# Disorders of central and peripheral tolerance

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- Central tolerance disorder
- AIRE (English AutoImmune REGulator): a transcription factor that regulates the expression of important autoantigens on thymic medullary epithelial cells
- Disorder of peripheral tolerance
  - loss of anergy (better processing of antigens, expression of costimulatory molecules, released cytokines)?
  - deletion disorder (activation-induced cell death, i.e. apoptosis)?
  - disorder of suppression (functions of T regulatory lymphocytes, lack of inhibitory cytokines IL-10 and TGFb)?

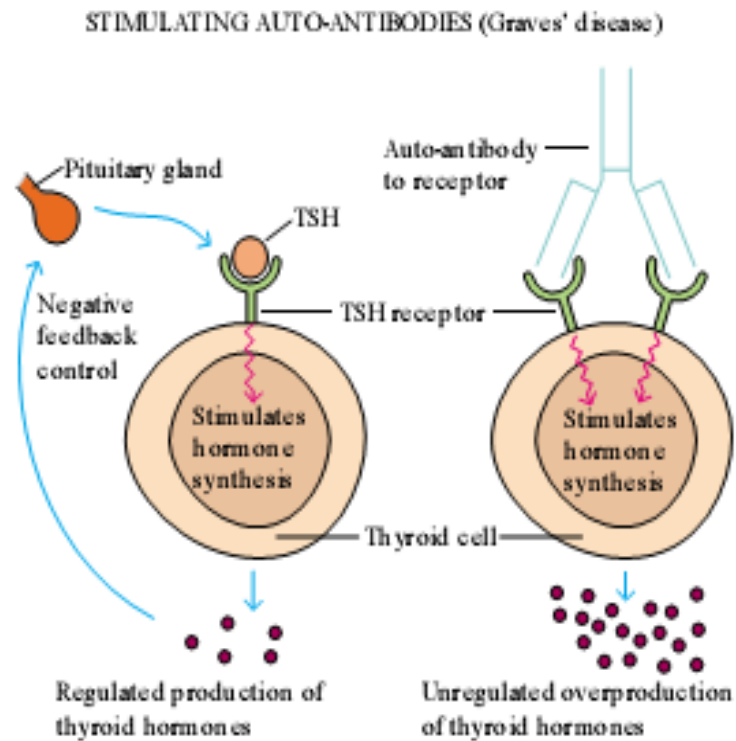
# Autoimmune Thyroid Disease (AITD)

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# M. Graves

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# M. Graves

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- AntiTSH R Ab (antibodies to the receptor for TSH)
- Long-term stimulation of TSH receptors on thyrocytes after binding of AntiTSH R At
- Hyperfunction with hyperplasia of thyroid tissue
- Possible occurrence:
  - thyroid ophthalmopathy
  - dermatopathies

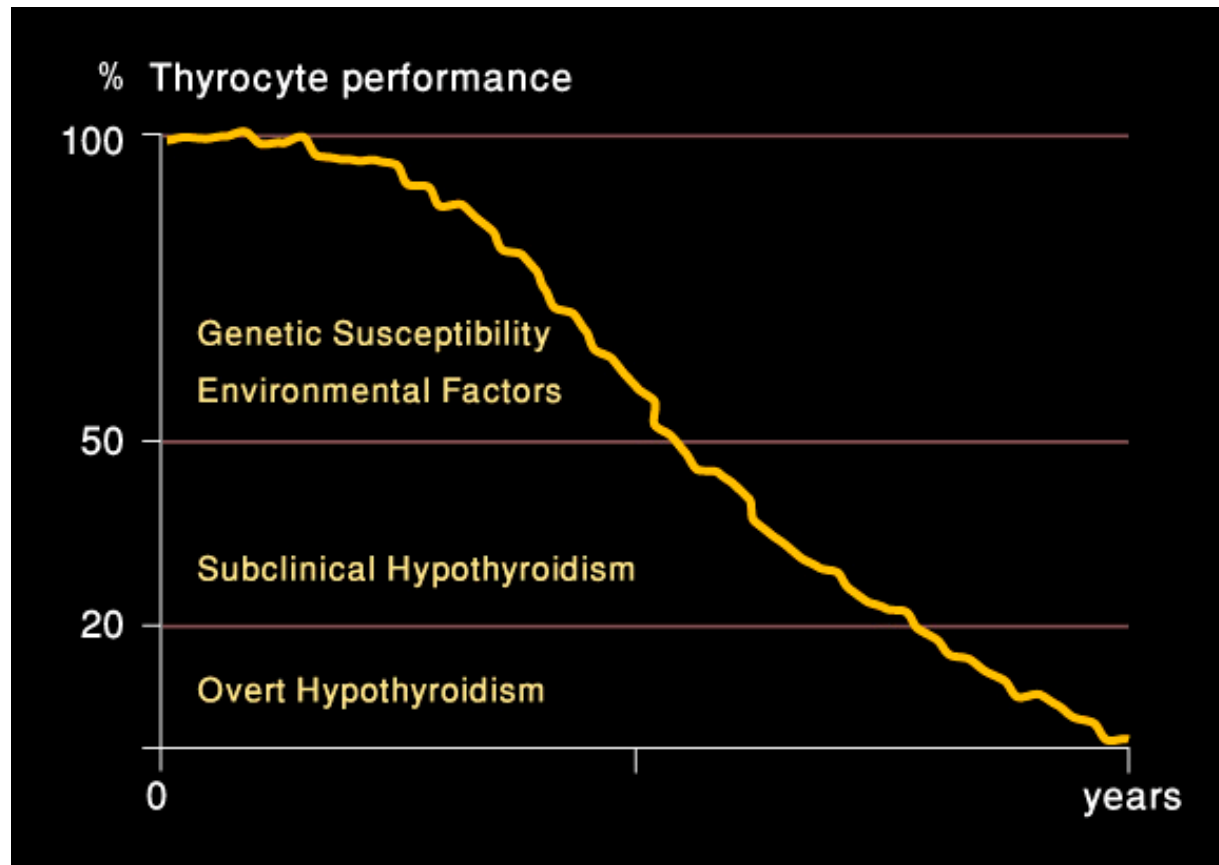
# Thyreoiditis chronica Hashimoto

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- Hashimoto thyroiditis
- Effector mechanisms of cellular immunity (cytotoxic T lymphocytes), which damage thyrocytes by type IV hypersensitivity (late type) by release of perforin and granzyme
- Induction of thyrocyte apoptosis (T lymphocytes express FasL which interacts with Fas on thyrocytes and induces thyrocyte apoptosis)
- Effector mechanisms of type II hypersensitivity - Antithyroid antibodies, which damage thyrocytes by complement activation – ADCC

# The development of Hashimoto thyroiditis

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# Thyroid function disorders in autoimmune atrophic Hashimoto's

- Three possibilities:
  1. initially without functional disorders, a possible phase of thyrotoxicosis, and later subclinical and clinically manifest hypothyroidism
  2. gradual reduction of thyroid function, with the emergence of subclinical and clinically manifest hypothyroidism
  3. slow autoimmune process, with gradual destruction of thyroid tissue, without thyroid function disorders

# Tumors of the thyroid gland

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- Benign
  - Follicular adenomas
- Malignant
  1. Origin of follicular cells
    - Papillary carcinoma
    - Follicular carcinoma
    - Poorly differentiated carcinoma-
    - Undifferentiated carcinoma (anaplastic)
  2. Origin of C-cells
    - Medullary carcinoma
- Of primary non-epithelial origin-Lymphomas, sarcomas
- Secondary

# Inflammation of the thyroid gland

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- Thyroiditis: inflammation of the thyroid gland
- Division by duration:
  1. Acute: bacterial pathogens
  2. Subacute: viruses that cause inflammation
  3. Chronic: autoimmune inflammation

# Lecture

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1. General endocrinology
  1. Function of endocrine system
  2. Hormones
  3. Organisation
  4. Circadian rhythm
- Special endocrinology
  1. Disorders of hypothalamus
  2. Disorders of hypophyses
  3. Disorders of thyroideidea