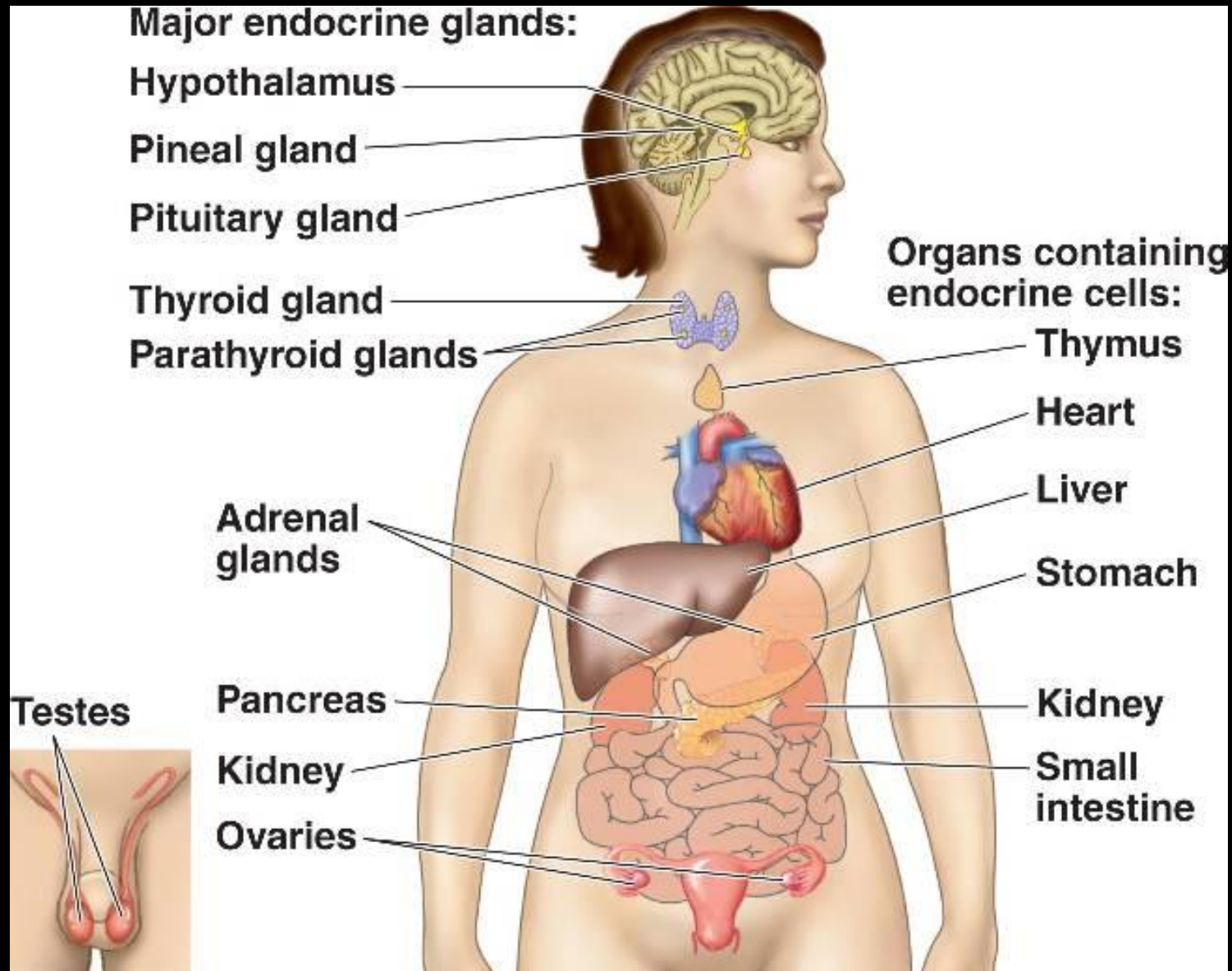


Endokrinní soustavy



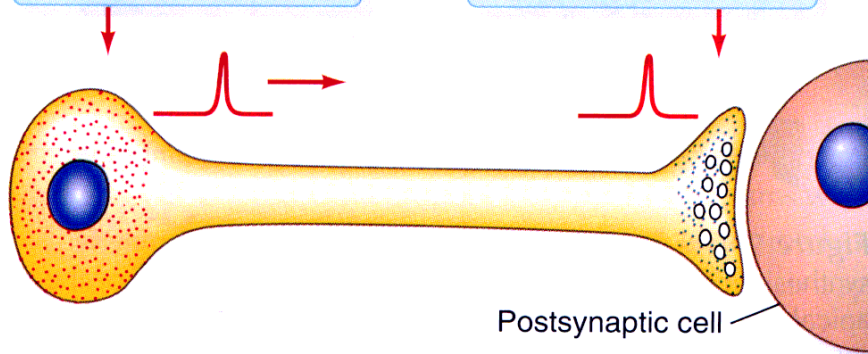
(a) Neuron

Stimulus induces

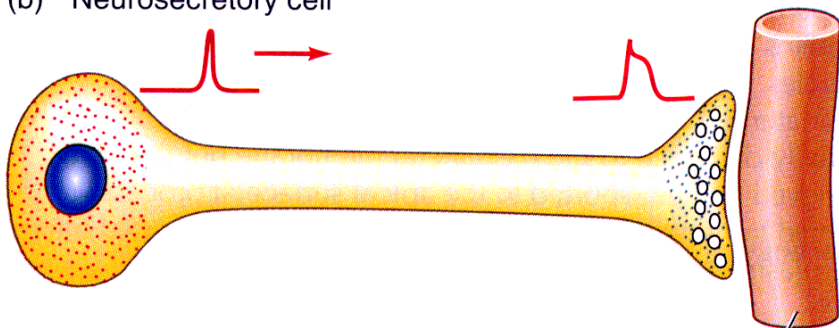
- (1) permeability increase
- (2) Na^+ , Ca^{2+} influx
- (3) depolarization

Secretion follows

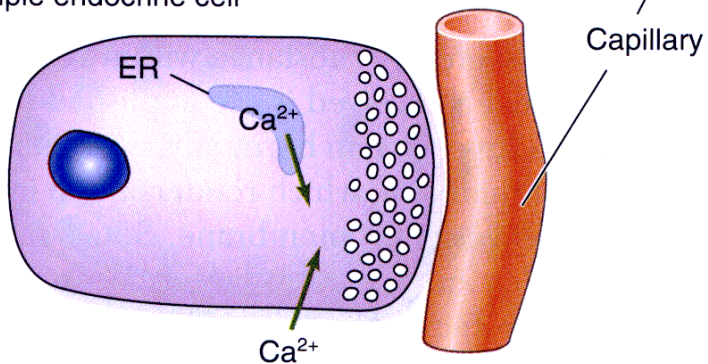
- (1) depolarization
- (2) permeability increase
- (3) Ca^{2+} influx



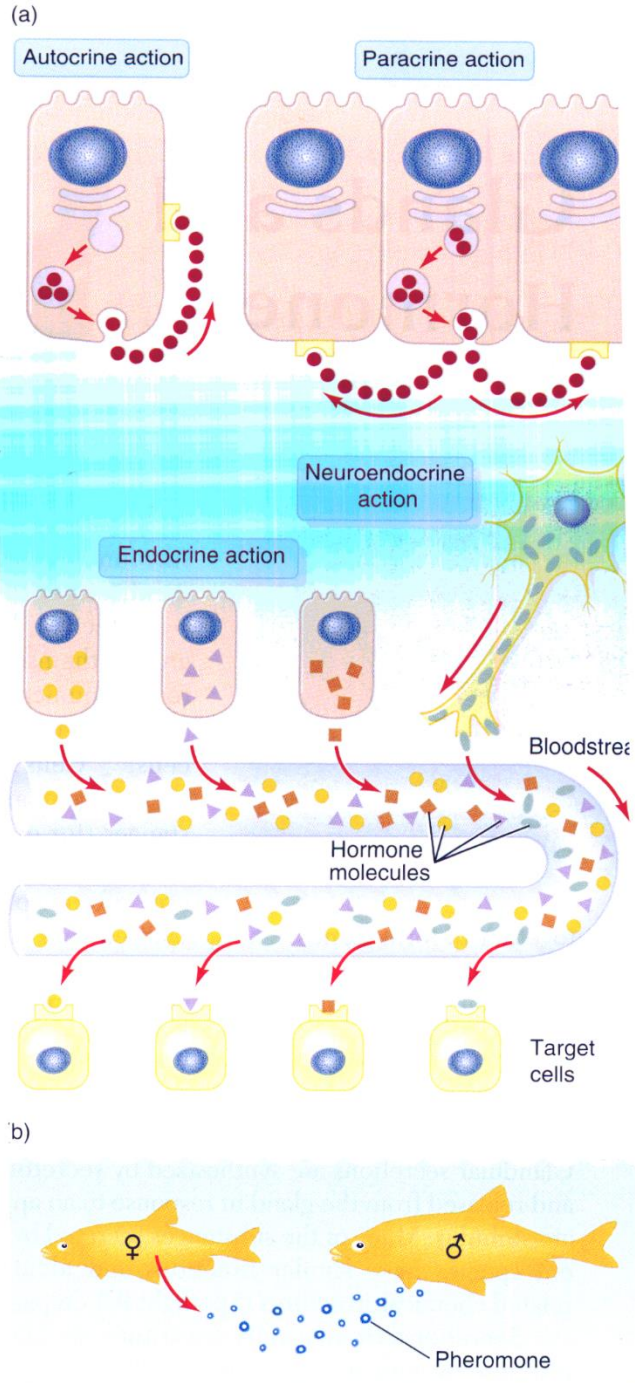
(b) Neurosecretory cell



(c) Simple endocrine cell



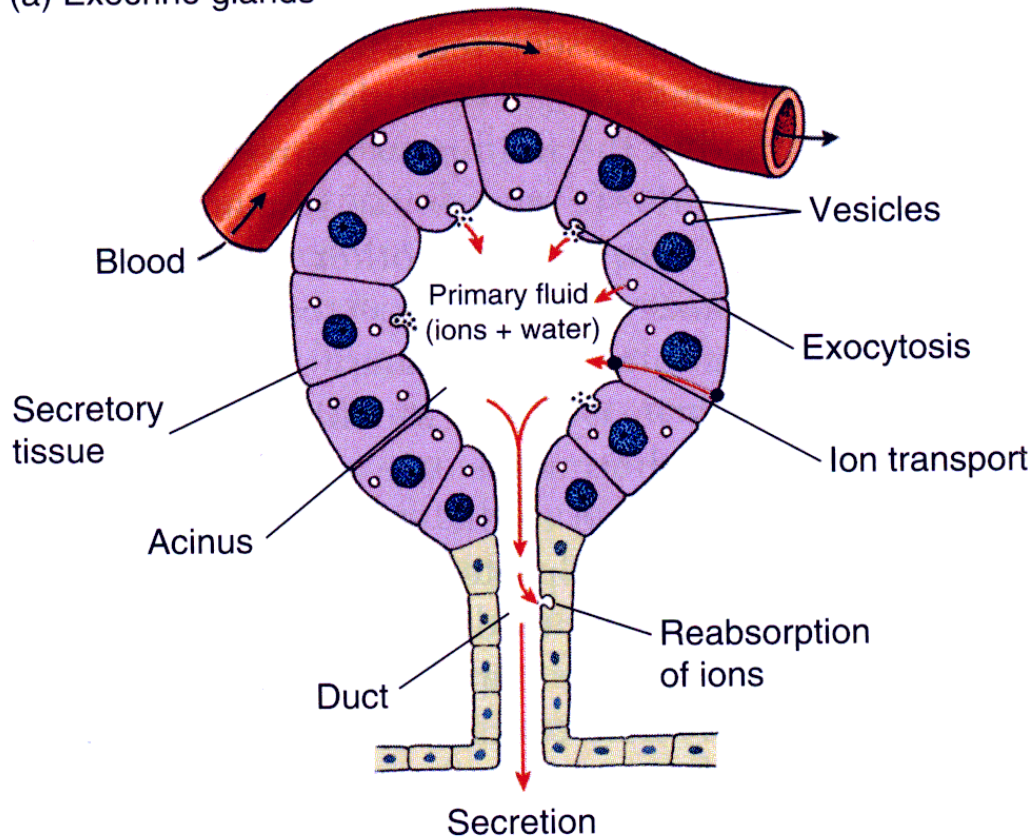
Rozdíly mezi nervovou a endokrinní buňkou



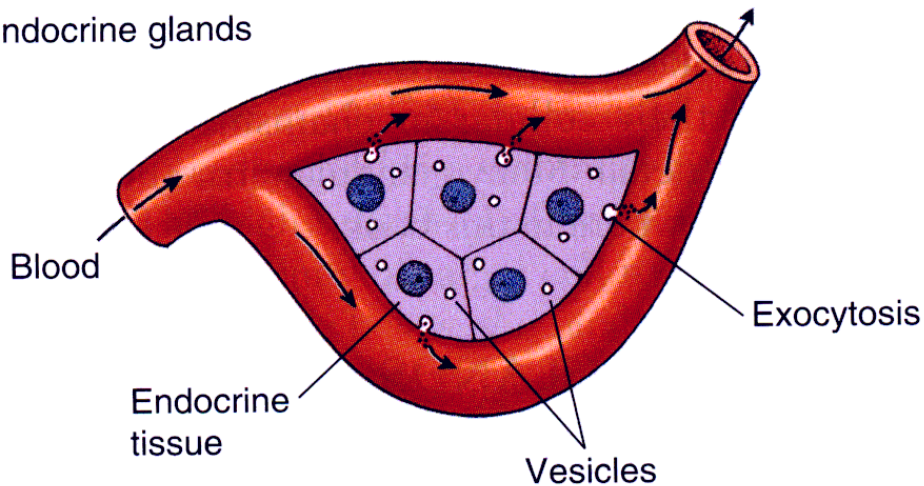
Různé způsoby endokrinní komunikace mezi buňkami

Srovnání exokrinní a endokrinní žlázy

(a) Exocrine glands



(b) Endocrine glands



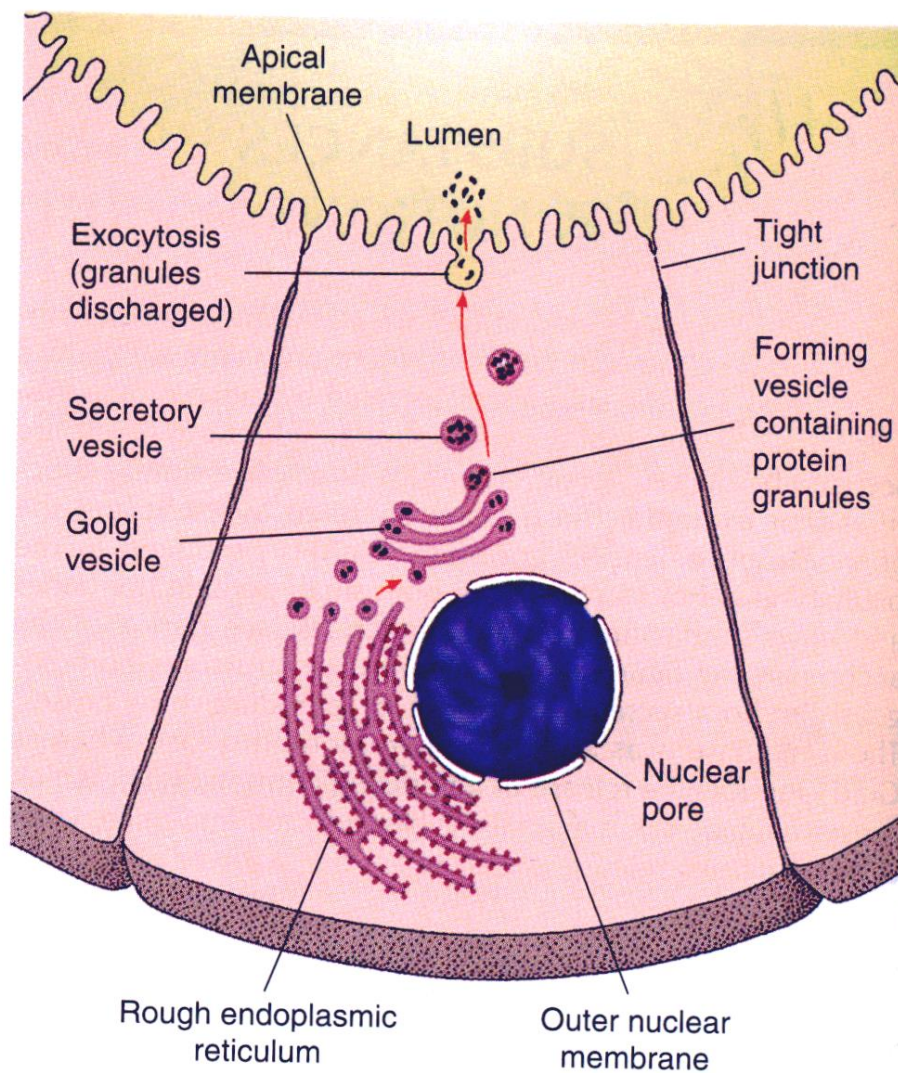


Schéma syntézy sekretorických bílkovin

Figure 9-2 Secretory proteins are synthesized in the rough endoplasmic reticulum, transferred in vesicles to the Golgi complex, and from there move to the apical surface. After the proteins are concentrated in secretory vesicles, the vesicles move to and fuse with the apical plasma membrane, discharging their contents into the lumen of the gland by exocytosis.

Syntéza peptidických hormonů - shrnutí

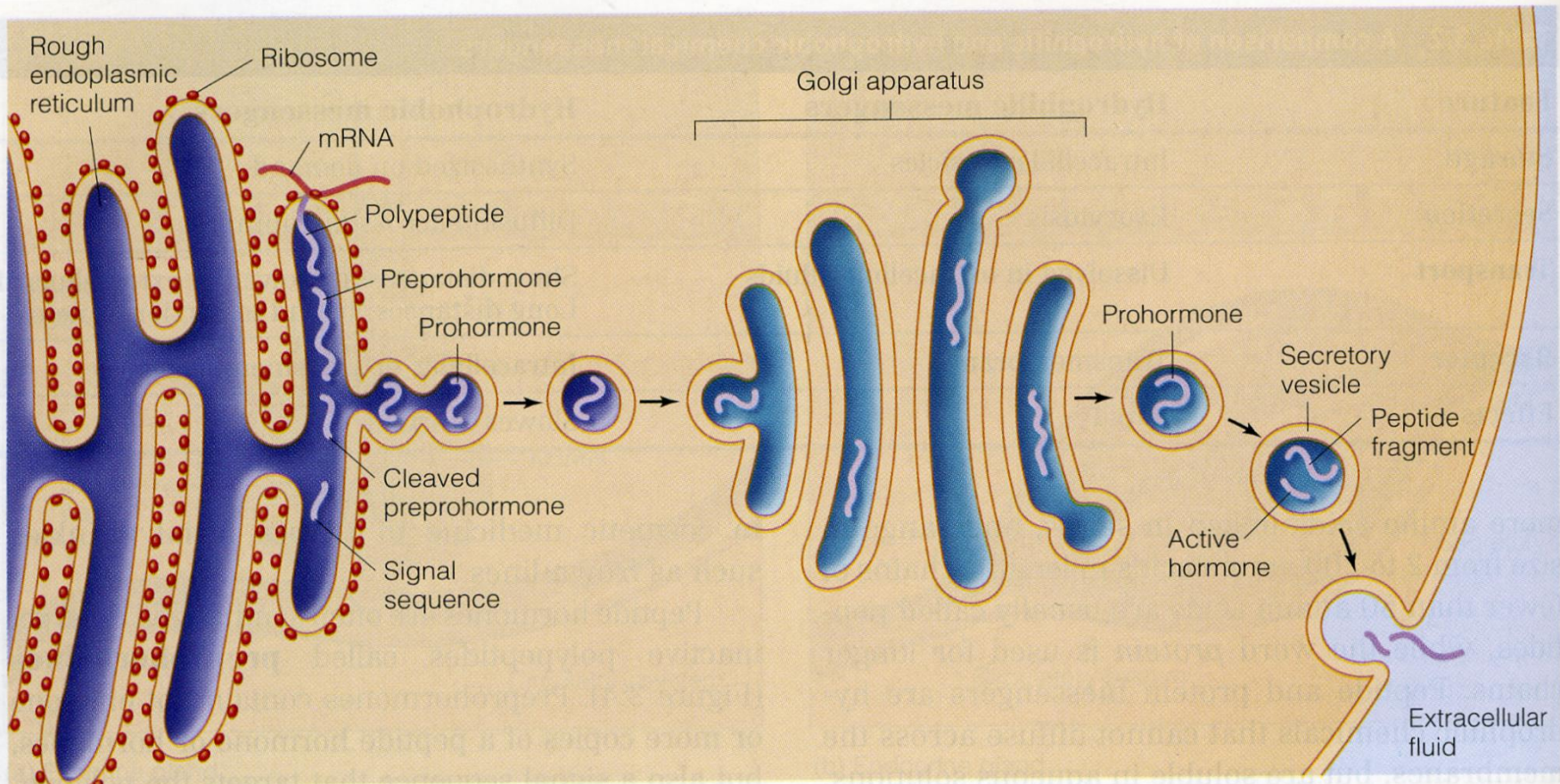
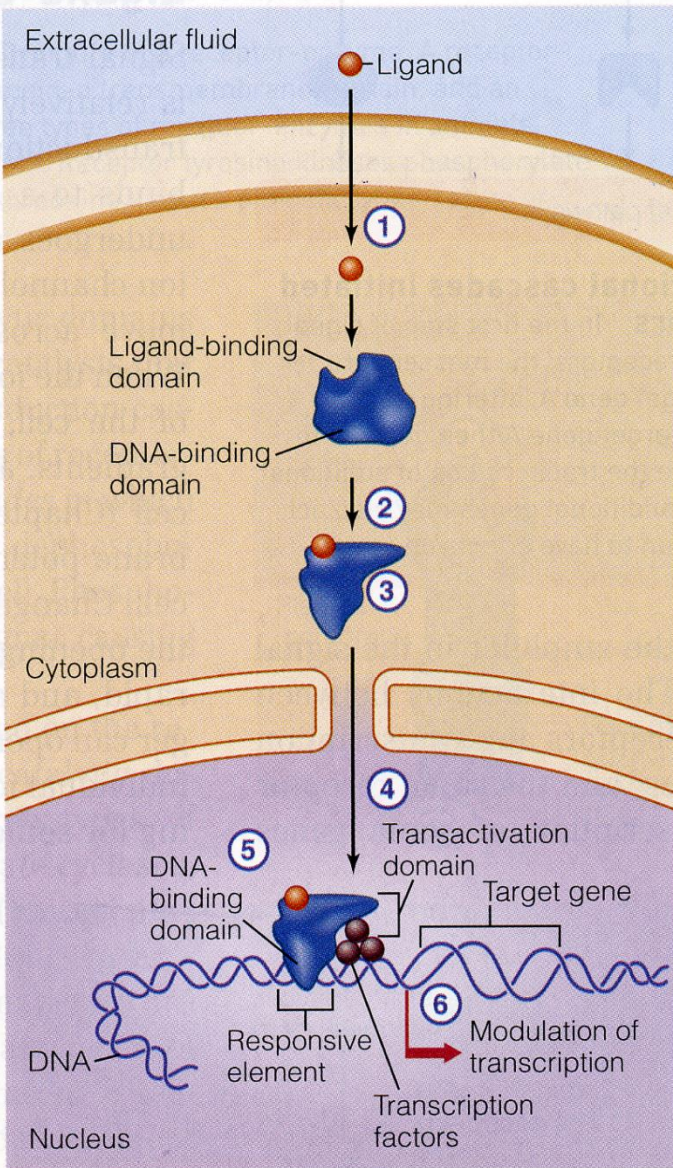


Figure 3.4 Synthesis of peptide hormones

Peptide hormones are synthesized by ribosomes on the rough endoplasmic reticulum, often as large preprohormones. The preprohormone enters the rough endoplasmic reticulum, where the signal sequence is cleaved off. The resulting prohormone is packaged into vesicles that move to the Golgi apparatus for

further processing and sorting. In the Golgi apparatus, the prohormone is packaged into secretory vesicles, where it is cleaved into active hormone and one or more peptide fragments. The secretory vesicle fuses with the plasma membrane, releasing its contents by exocytosis.

Mechanismus působení lipofilních hormonů



- 1 Hydrophobic ligands pass through the cell membrane.
- 2 Inside the cell, the ligand binds to the ligand-binding domain of the intracellular receptors.
- 3 Ligand binding changes the shape of the receptor.
- 4 The receptor-ligand complex translocates to the nucleus.
- 5 The DNA-binding domain of the receptor binds to responsive element DNA sequences, and the transactivating domain interacts with other transcription factors bound in this region.
- 6 Together, these transcription factors alter the rate of transcription of the target genes into mRNA.

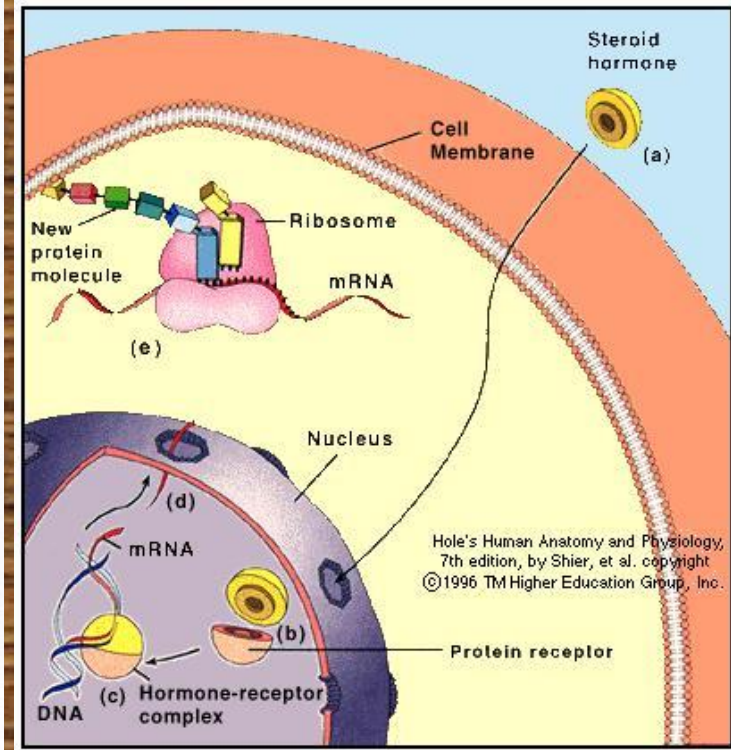
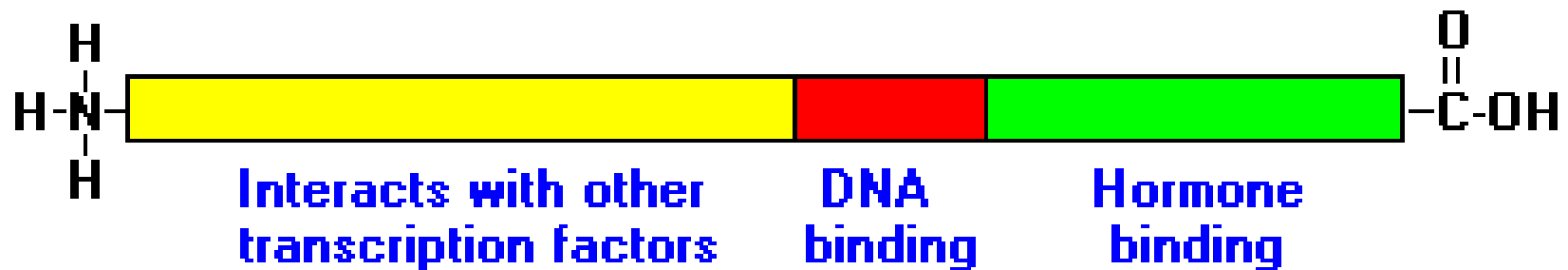
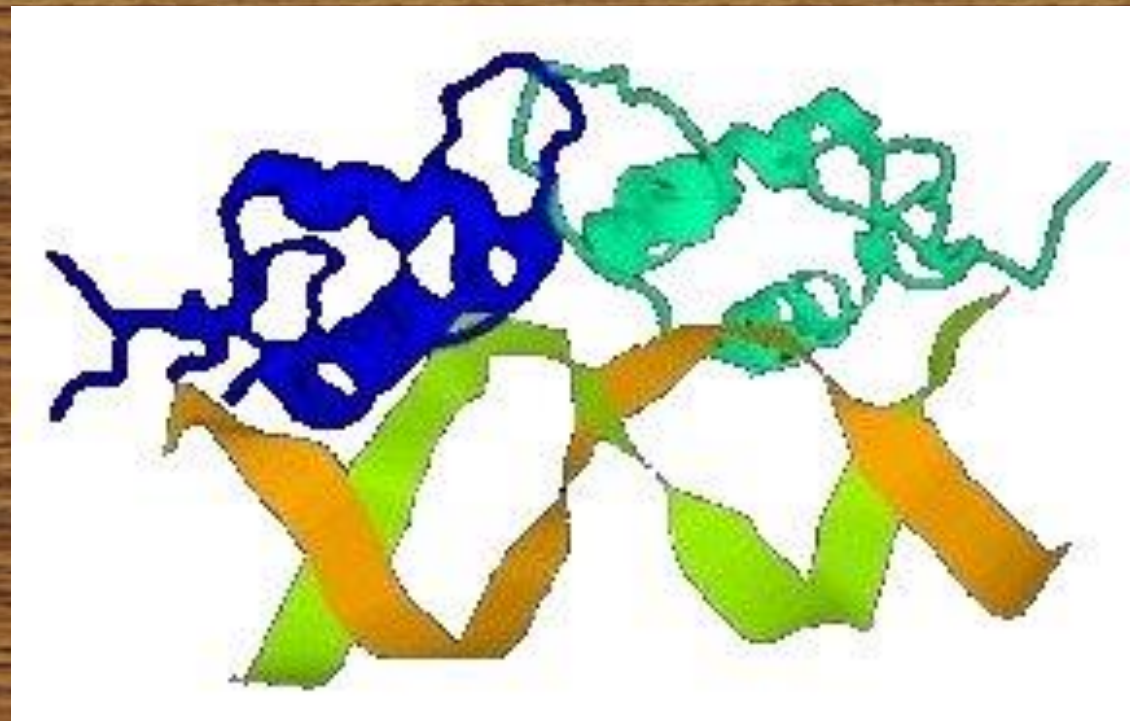


Figure 3.17 Signal transduction by intracellular receptors

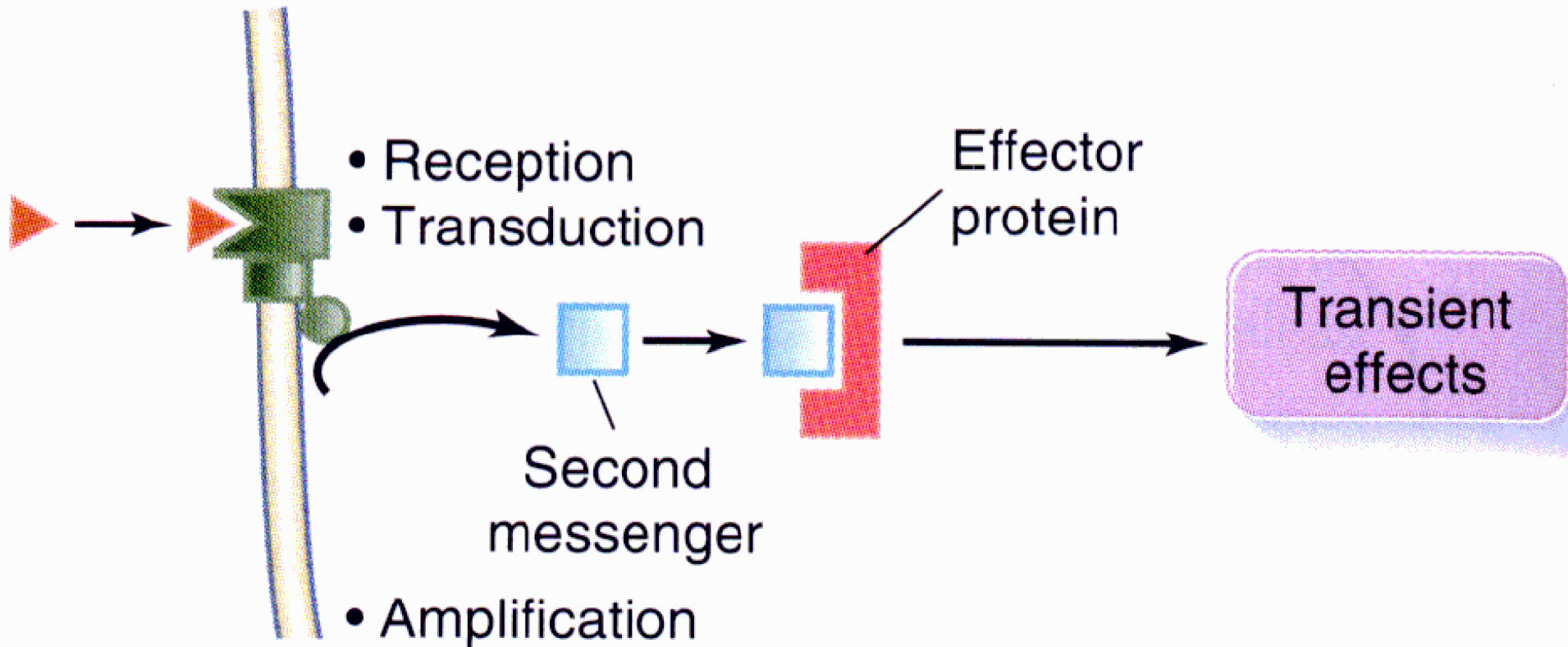
Struktura bílkovinných intracelulárních receptorů



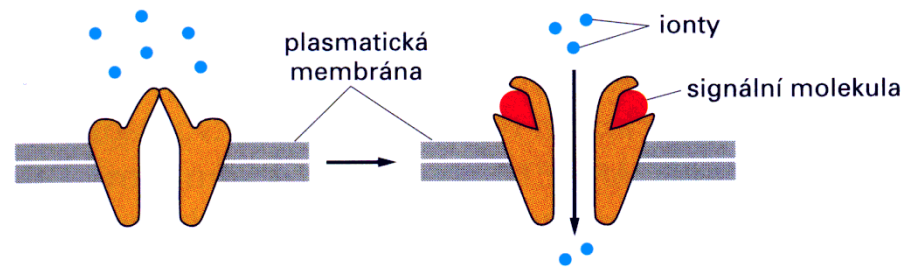
Příklad intracelulárního receptoru pro glukokortikoidy



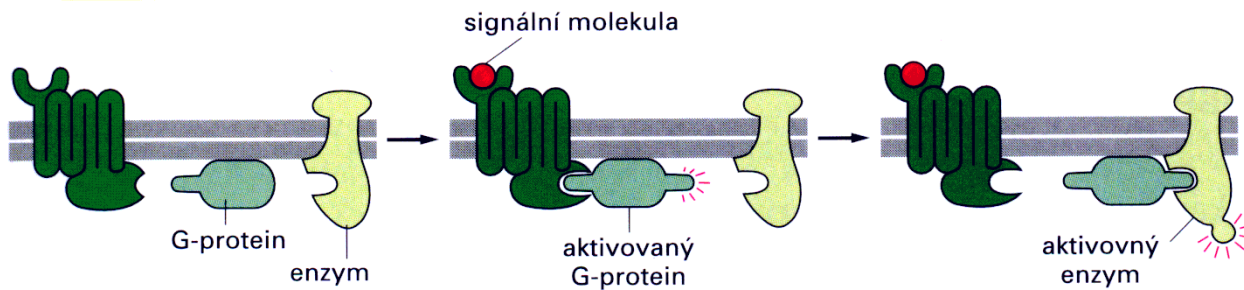
Mechanismus působení hydrofilních hormonů



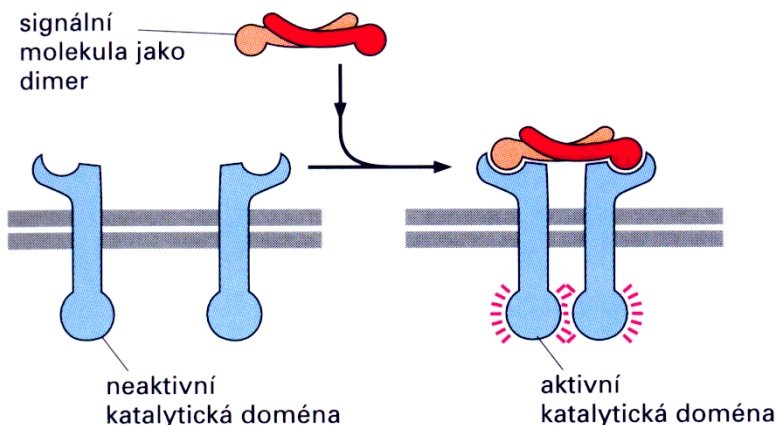
(A) receptory spojené s iontovým kanálem



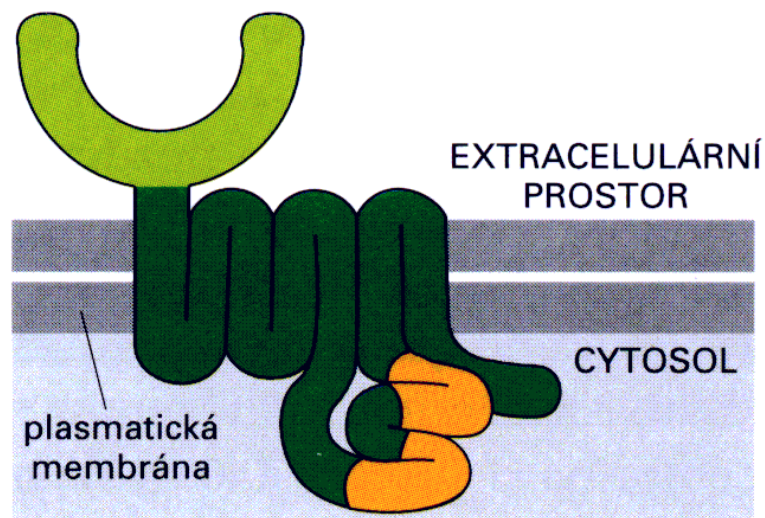
(B) receptory vázané na G-protein



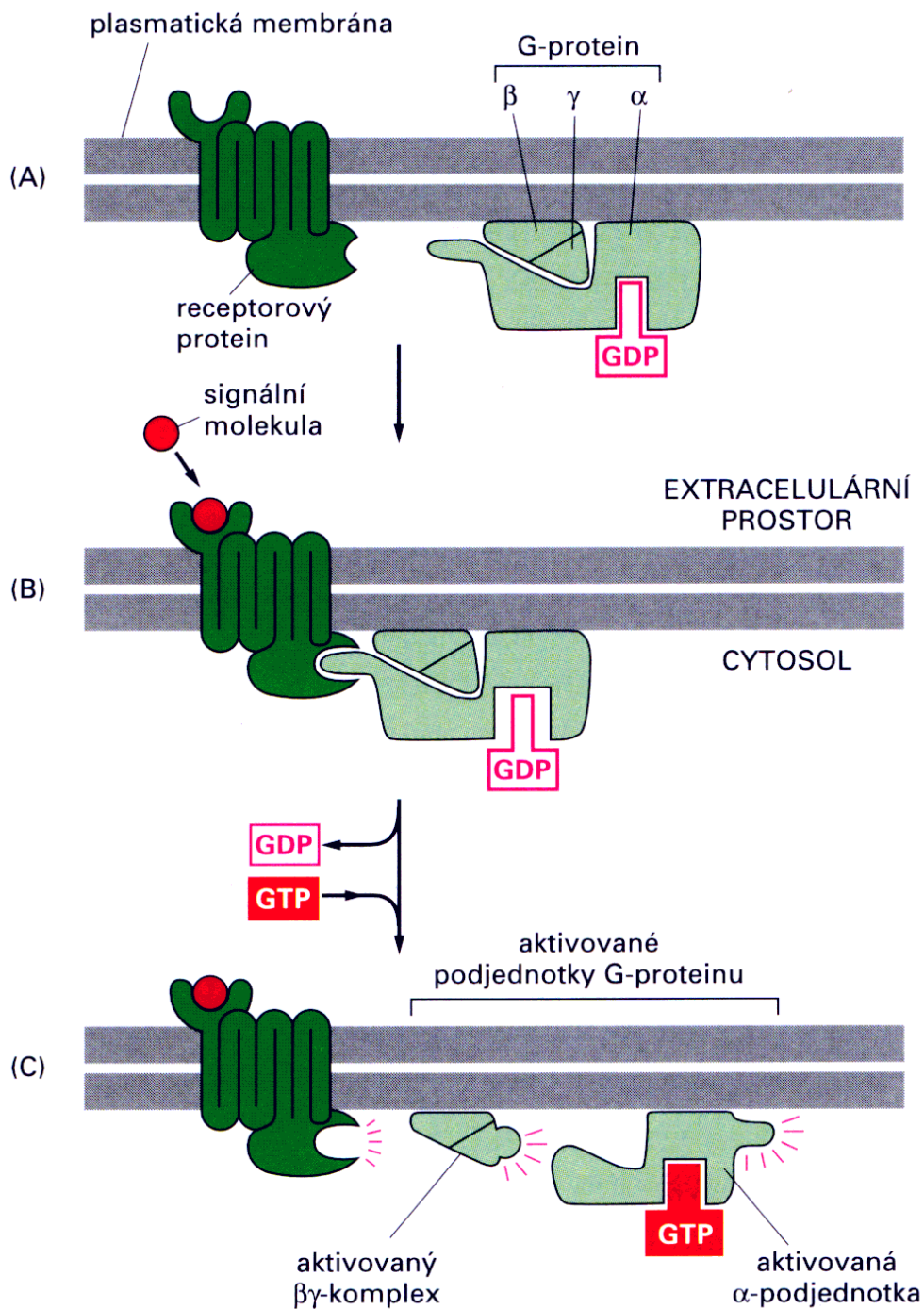
(C) receptory vázané na enzym



Obrázek 15-12 Tři třídy povrchových buněčných receptorů. (A) Receptor spojený s iontovým kanálem se otevírá (nebo zavírá) v odpověď na navázání svého ligandu. (B) Když receptor spojený s G-proteinem naváže svůj ligand, je signál nejprve předán GTP-vázajícímu proteinu (G-proteinu), který se spojí s receptorem. Potom tento protein receptor opustí a aktivuje cílový enzym (nebo iontový kanál) v plasmatické membráně. Pro jednoduchost je zde G-protein ukázán jako jediná molekula; jak uvidíme, jde ve skutečnosti o komplex tří podjednotek, které mohou disociovat. (C) Receptor spojený s enzymem váže svůj extracelulární ligand a přitom aktivuje enzym na druhém konci receptoru, na opačné straně membrány. Ve všech třech případech navázání signální molekuly způsobí v receptoru změnu, která se štafetově šíří dále spolu se zprávou, aniž by samotná signální molekula musela vstoupit do buňky.

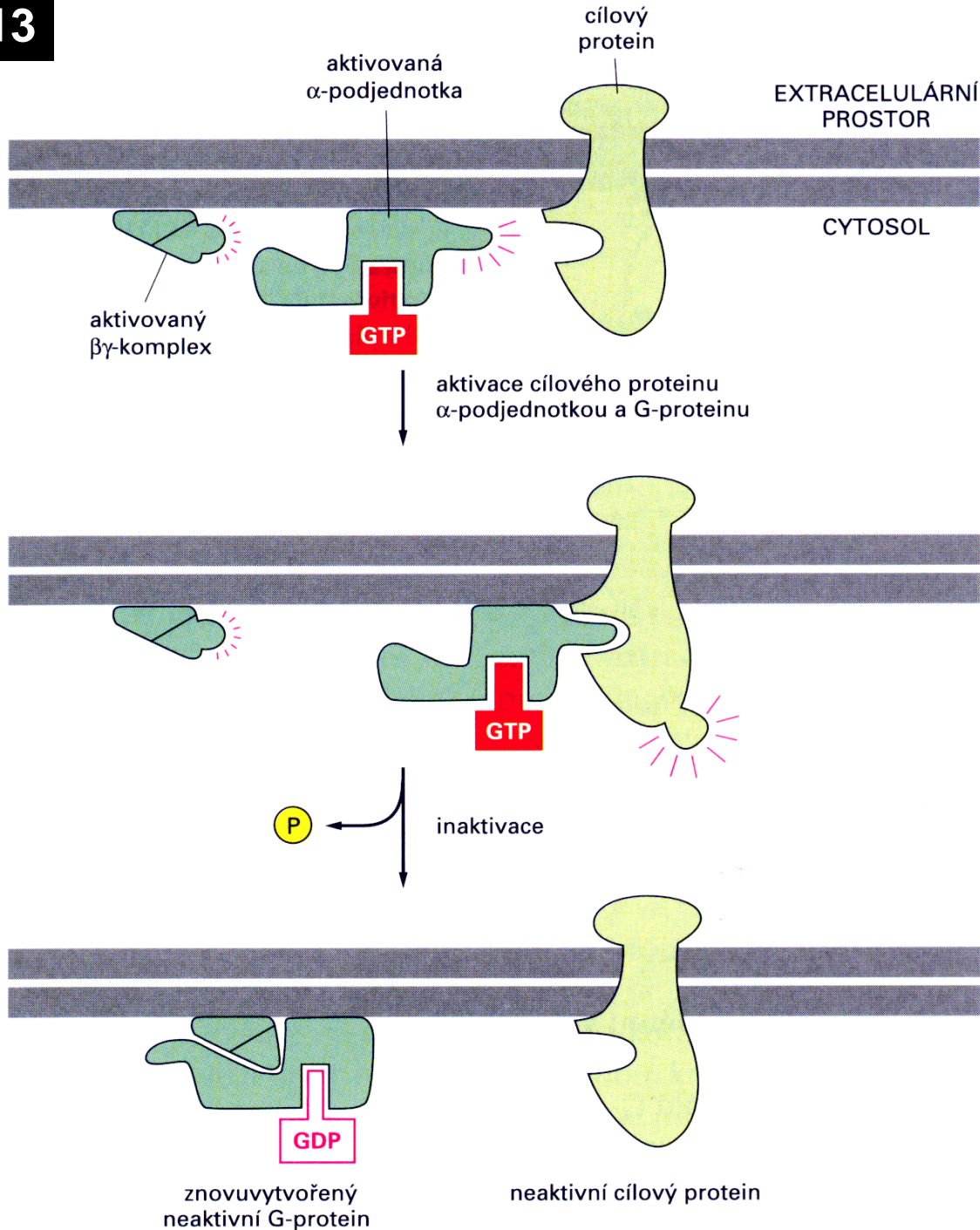


Obrázek 15-14 Schéma receptoru spojeného s G-proteinem. Cytoplasmatické části receptoru, které jsou hlavně zodpovědné za vazbu ke G-proteinu, jsou ukázány *oranžově*. Receptory, které vážou proteinové signální molekuly, mají vně buňky velkou doménu vázající ligandy, tvořenou částí polypeptidového řetězce, který je na obrázku *světle zeleně*. Receptory pro malé signální molekuly, jako např. pro adrenalin, však mají malé extracelulární domény; vazebné místo pro ligand je často tvořeno hluboko v membráně částmi transmembránových segmentů (na obrázku nejsou vidět).



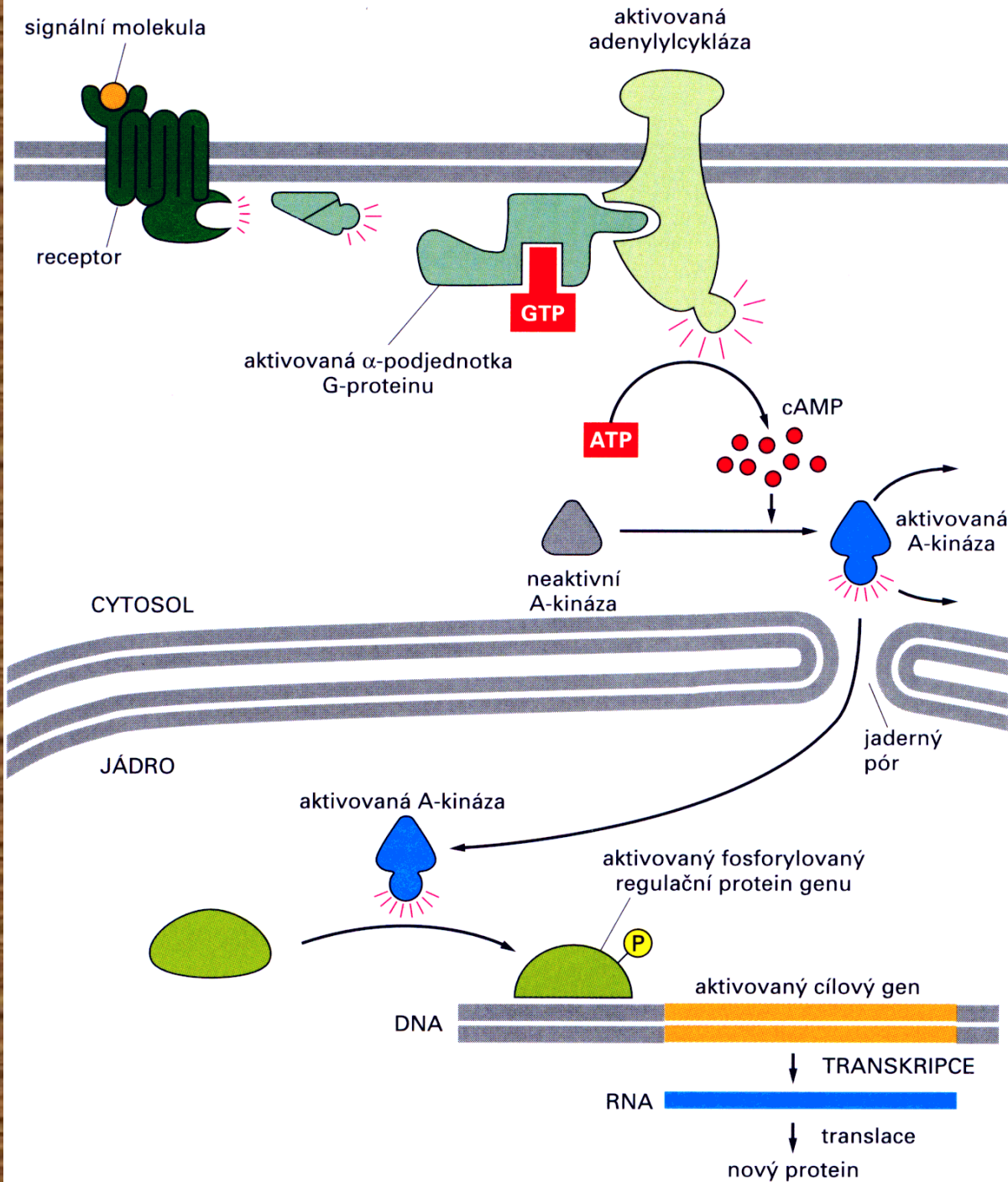
Obrázek 15-15 G-proteiny se při aktivaci rozpadají na dva signální proteiny.

(A) V nestimulovaném stavu jsou receptor i G-protein inaktivní a pravděpodobně se ani nedotýkají. (B) Aktivace receptoru extracelulární signální molekulou umožní G-proteinu spojit se s receptorem. (C) Navázání aktivovaného receptoru umožní α -podjednotce G-proteinu vyměnit svůj GDP za GTP. To způsobí rozpad G-proteinu na aktivovanou podjednotku α a na komplex $\beta\gamma$, které difundují podél cytoplasmatického povrchu plasmatické membrány, dokud nenajdou své cílové proteiny, jak je vidět na obr. 15-16. Receptor zůstává aktivní, dokud je k němu navázána externí signální molekula, a může takto katalyzovat aktivaci stovek či tisíců molekul G-proteinu.

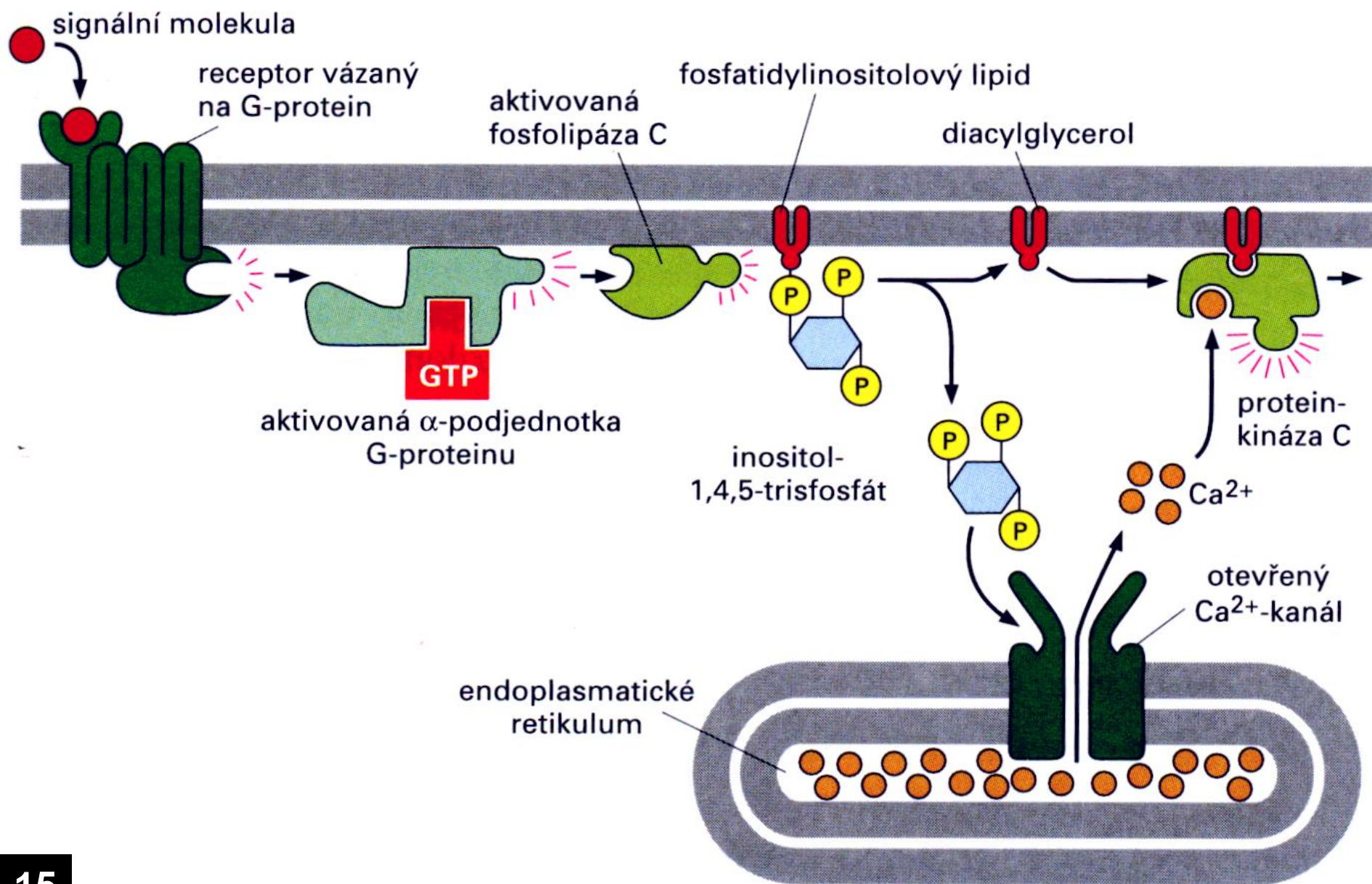


Obrázek 15-16 α -Podjednotka G-proteinu se sama deaktivuje hydrolýzou svého GTP. Když aktivovaná α -podjednotka najde a naváže svůj cílový protein, aktivuje ho (nebo v některých případech deaktivuje; na obrázku tato možnost není naznačena) na celou dobu, po kterou oba proteiny zůstanou spolu. Po několika sekundách je GTP vázaný na α -podjednotce GTPázovou aktivitou této podjednotky hydrolyzován na GDP. To inaktivuje α -podjednotku, která se oddělí od svého cílového proteinu a znovu se spojí s komplexem $\beta\gamma$ za opětného vzniku neaktivního G-proteinu. G-Protein je nyní připraven spojit se s dalším receptorem, jako na obrázku 15-15B. Jak aktivovaná podjednotka α (jak je ukázáno) tak volný komplex $\beta\gamma$ mohou regulovat cílové proteiny.

Působení hydrofilních hormonů - signální dráha aktivovaná cAMP



Působení hydrofilních hormonů signální dráha aktivovaná fosfolipázou C

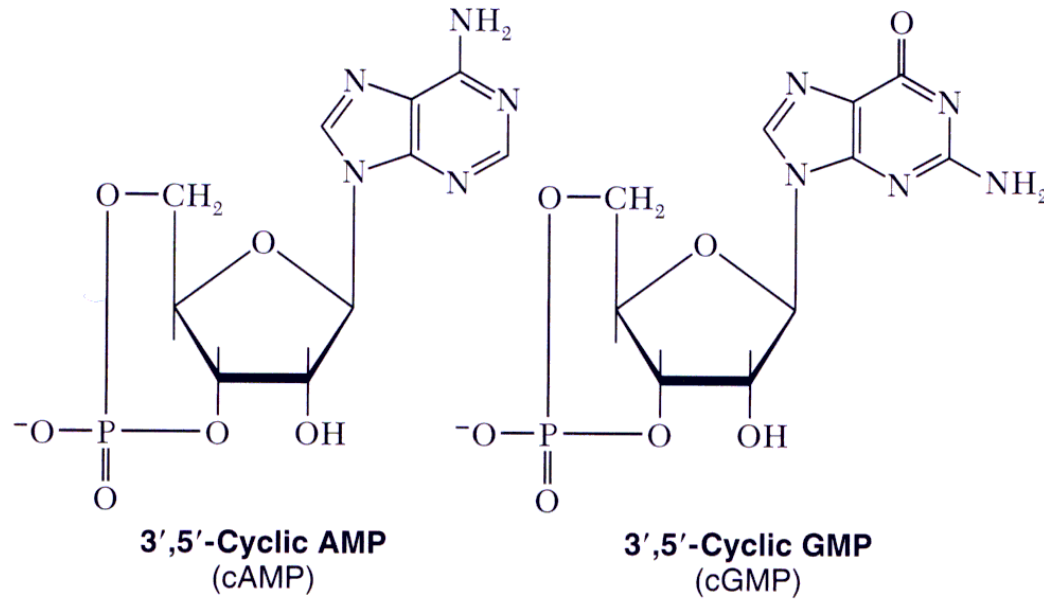


http://highered.mheducation.com/sites/0072943696/student_view0/chapter10/animation_mechanism_of_steroid_hormone_action_quiz_1.html

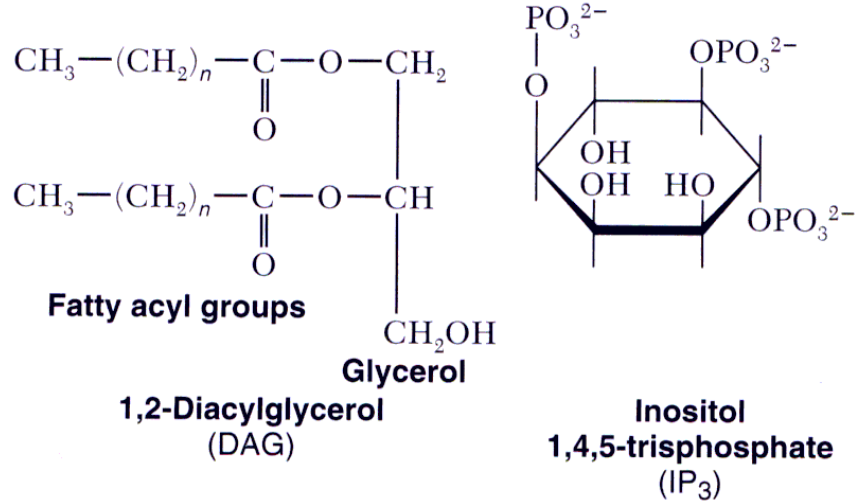
<https://www.youtube.com/watch?v=Nt2r5R0ZO5U>

Tři třídy druhých posílů

CYCLIC NUCLEOTIDES



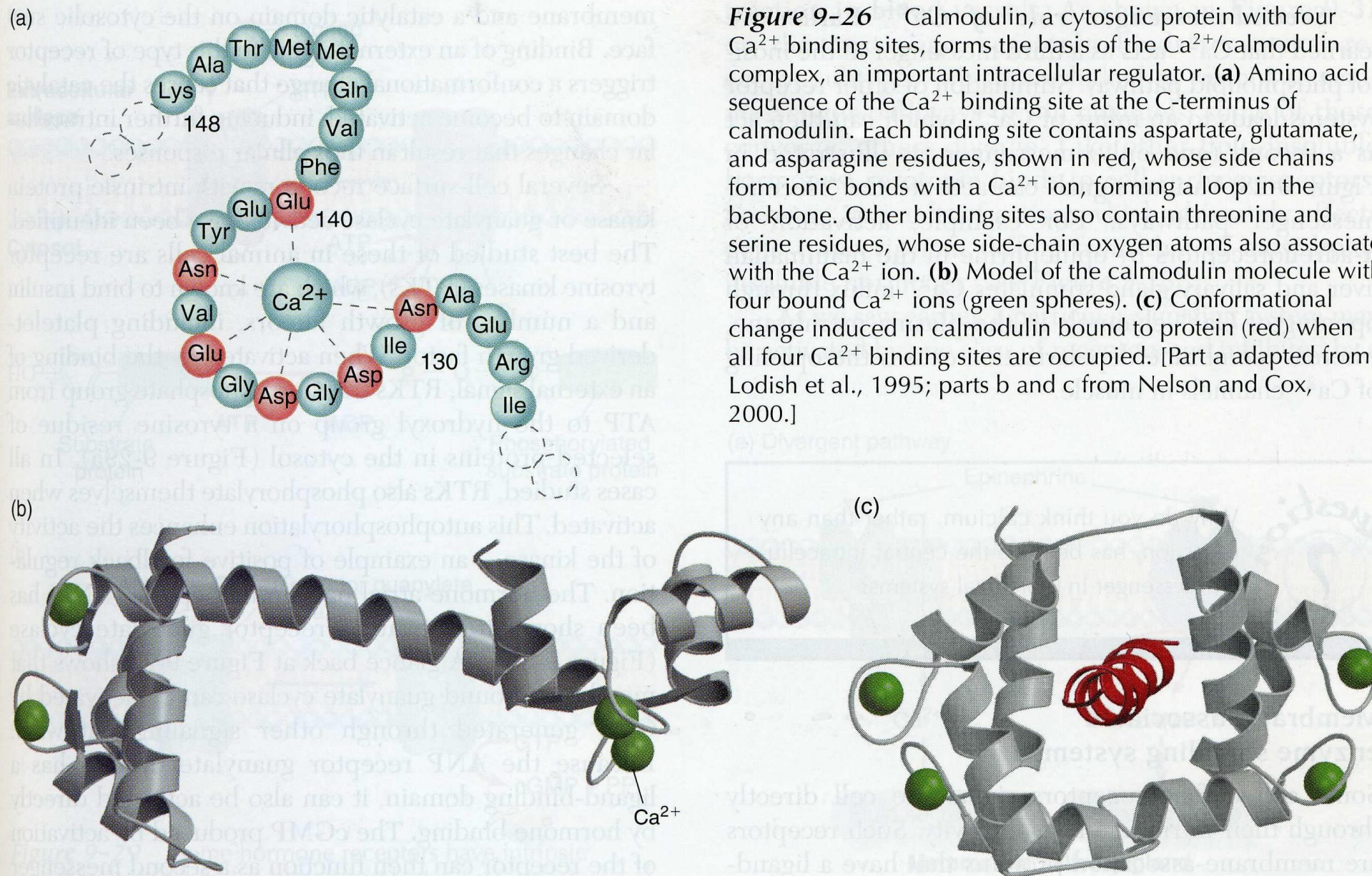
INOSITOL PHOSPHOLIPIDS



CALCIUM ION



Kalmodulin a jeho interakce s vápenatými ionty



**Kalmodulin/Ca⁺⁺
systém zasahuje
do celé řady
procesů**

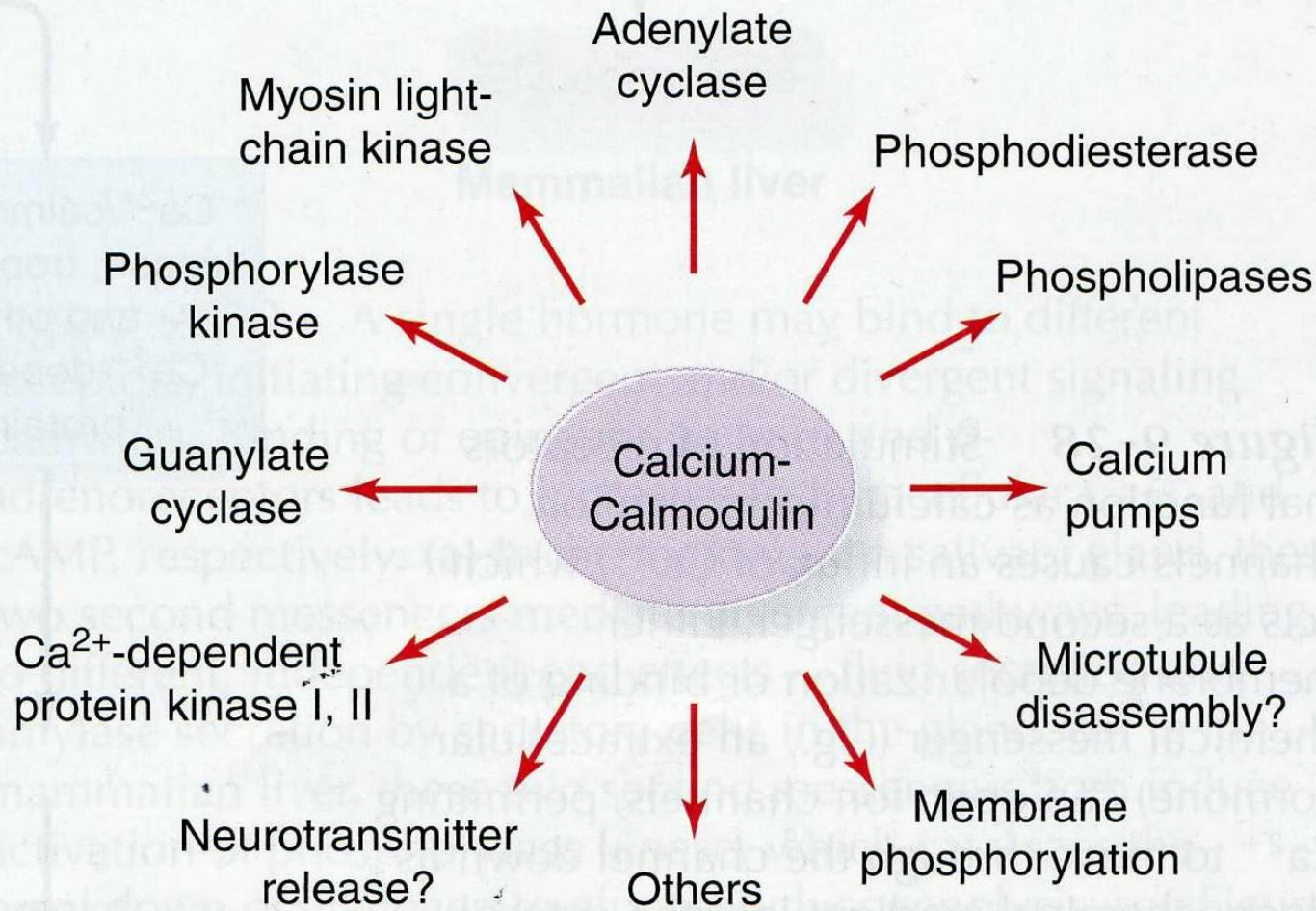
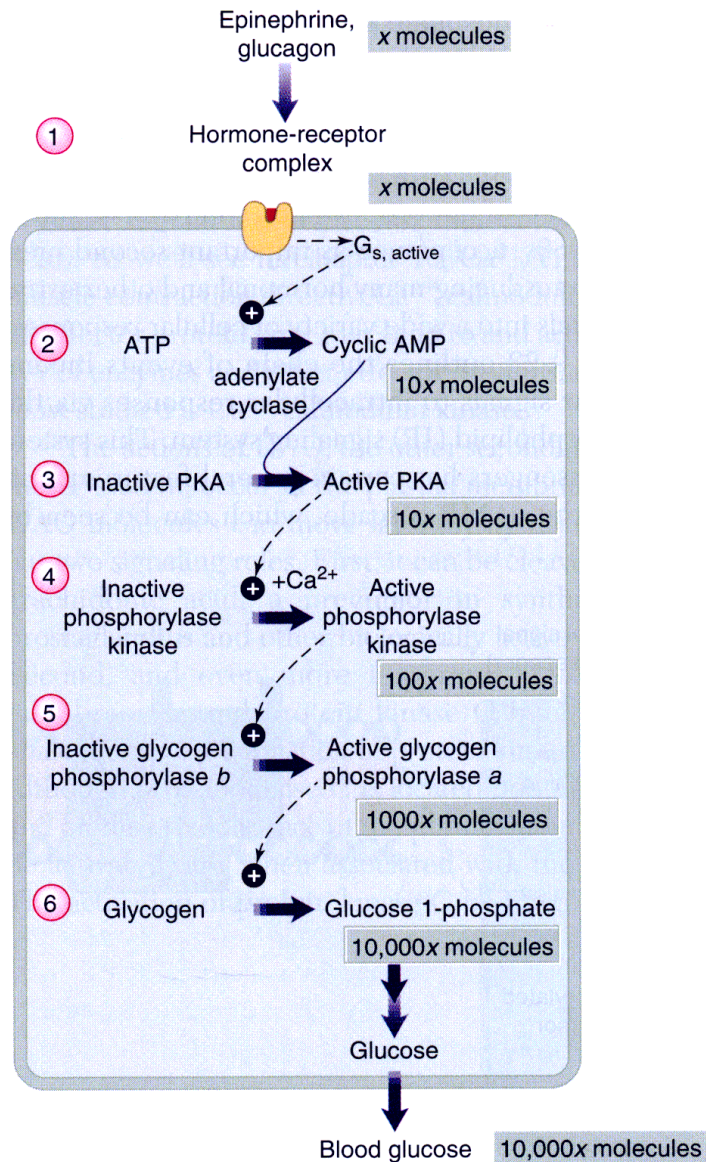
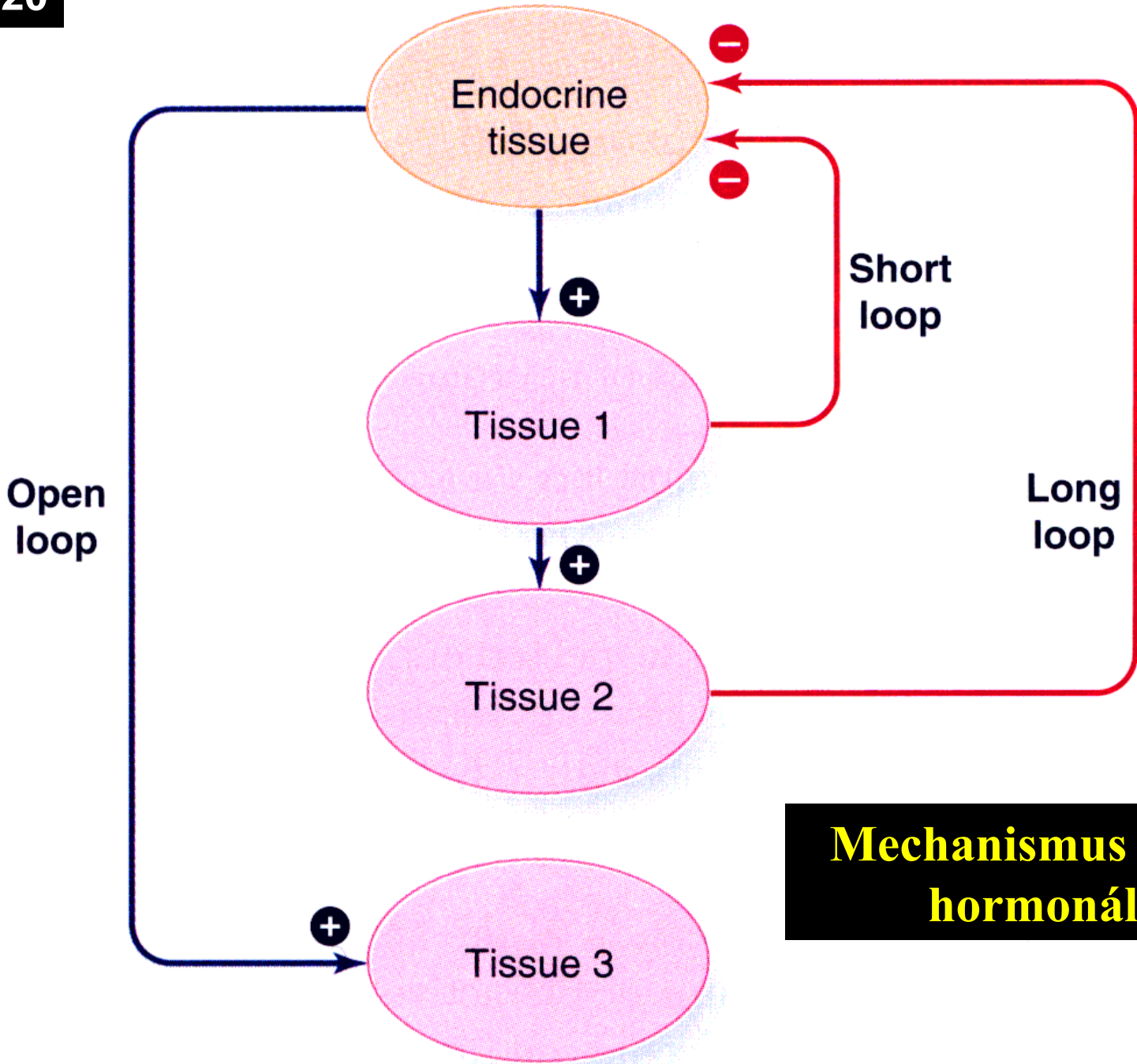


Figure 9-27 Ca²⁺/calmodulin regulates many processes and enzymes within cells. Among these are adenylate cyclase and guanylate cyclase, which catalyze formation of the cyclic nucleotide second messengers. [Adapted from Cheung, 1979.]



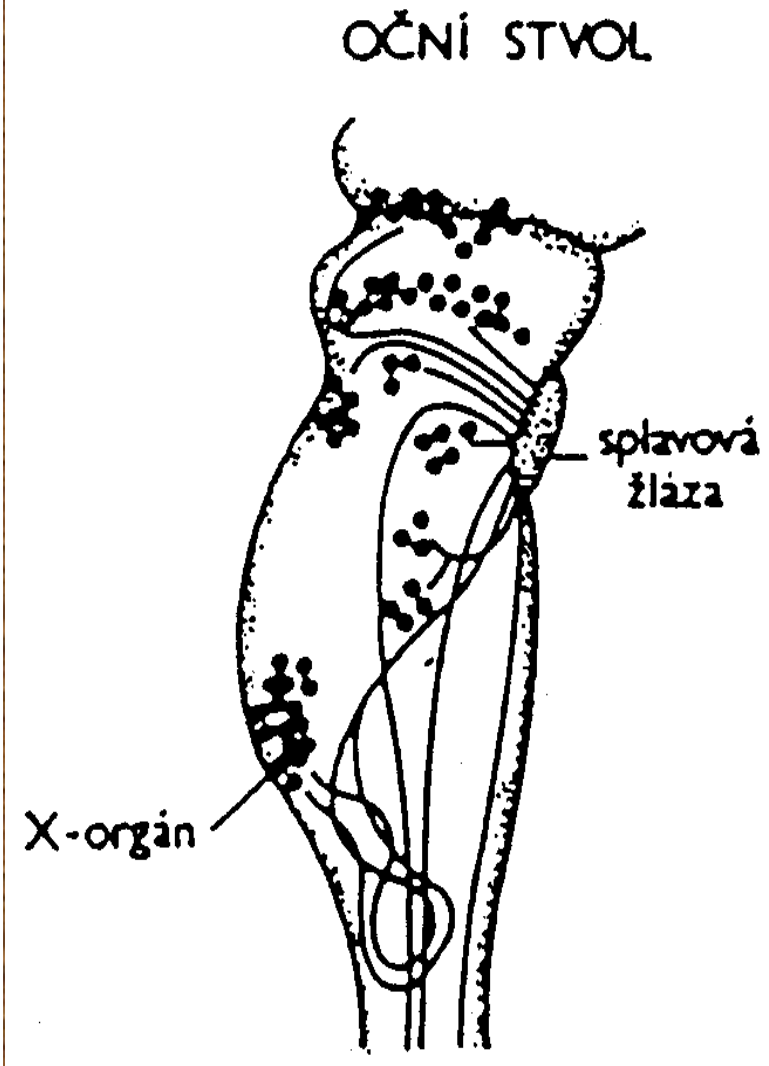
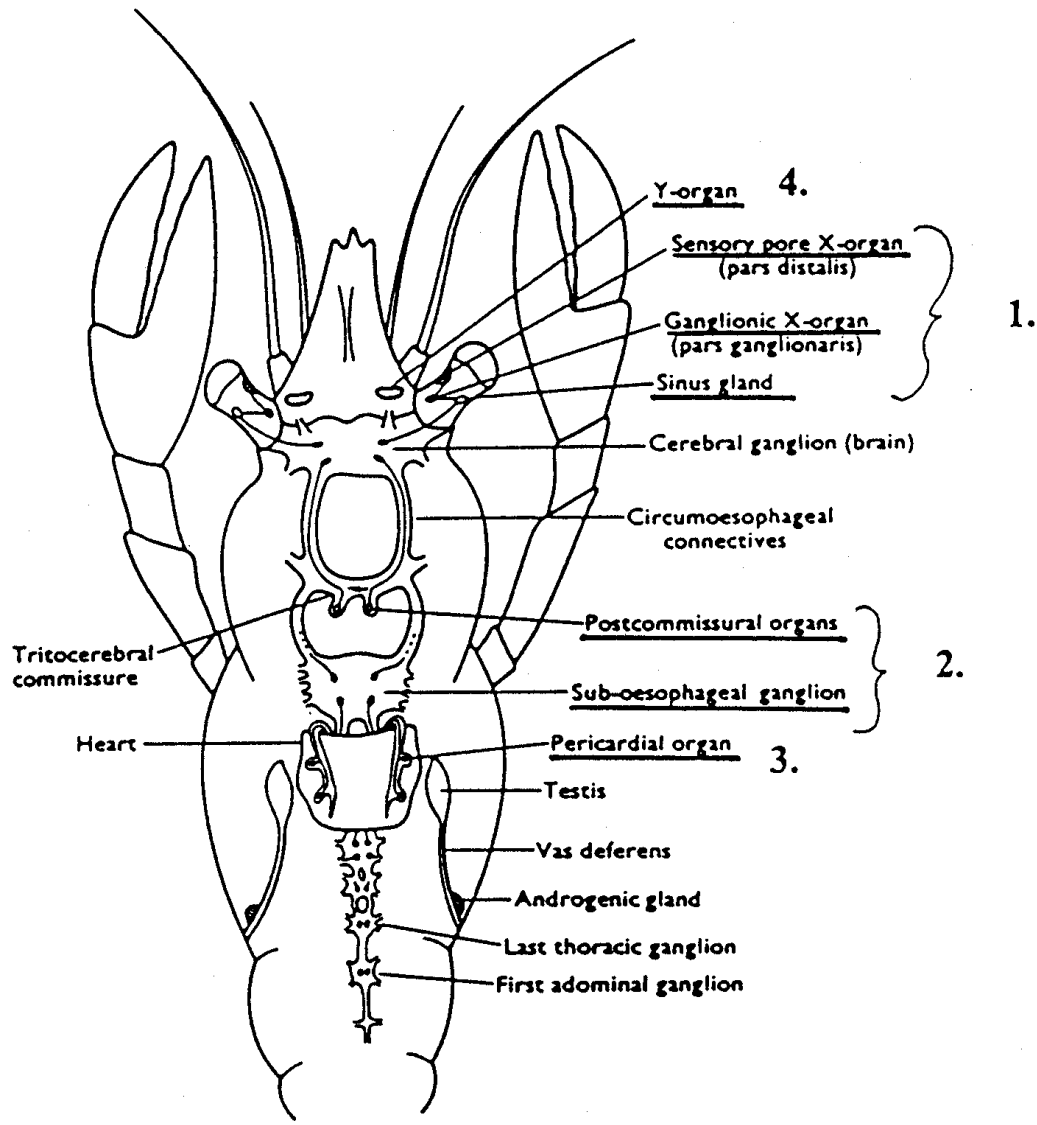
Příklad amplifikace – kaskádovitého působení hormonů

Figure 9-22 Epinephrine and glucagon stimulate breakdown of glycogen to glucose (glycogenolysis) in muscle and liver, respectively. When epinephrine binds to β -adrenoreceptors it triggers a sequence of reactions in which several enzymes are converted from an inactive to an active form. As a result of this enzyme cascade, the original signal is greatly amplified. [From Nelson and Cox, 2000]

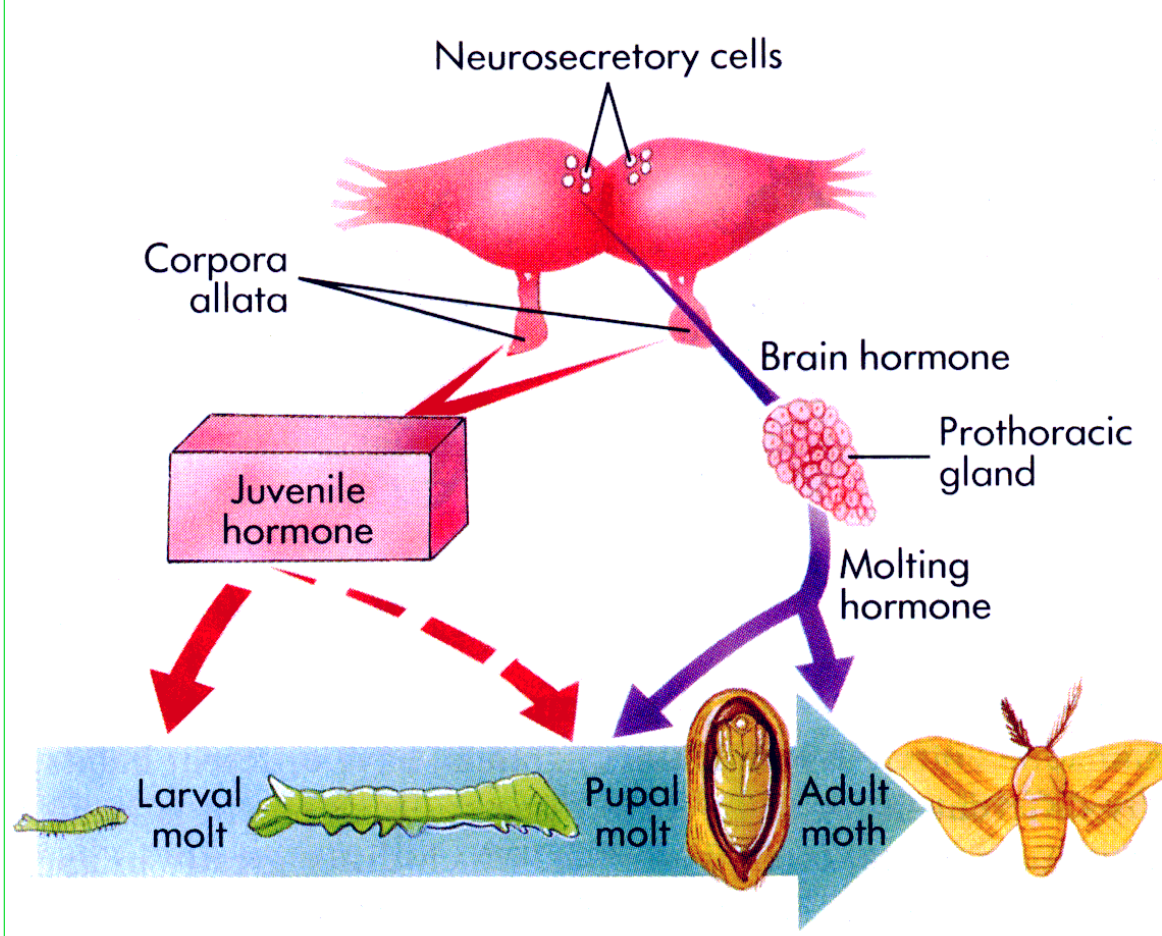
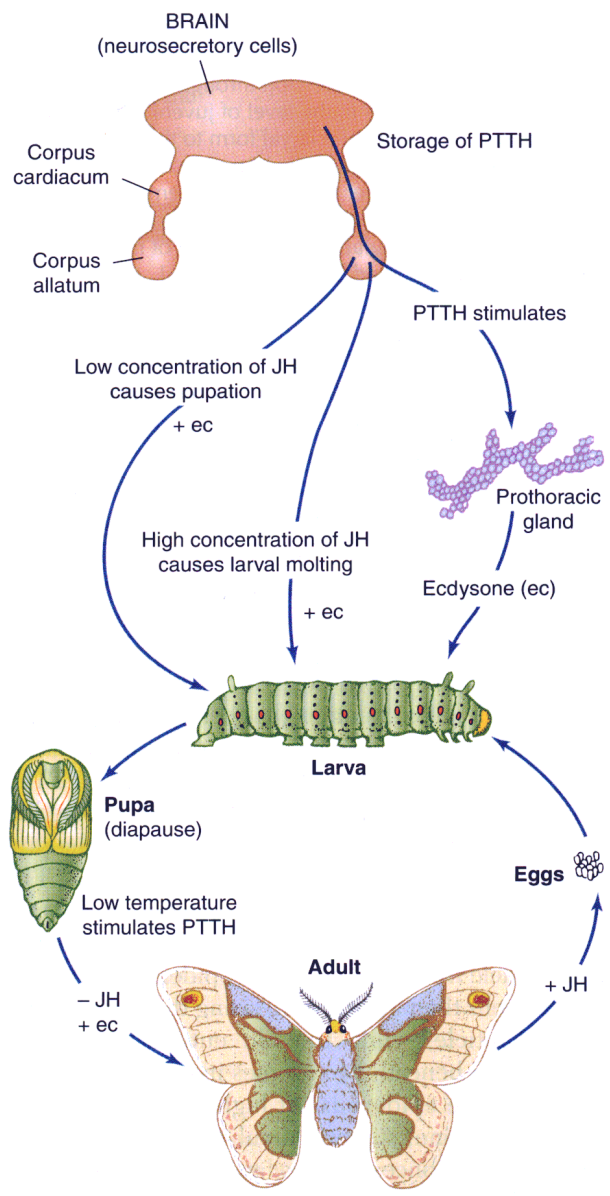


Mechanismus zpětné vazby v hormonálním řízení

Hormonální soustava korýšů



Hormonální soustava hmyzu



- 1. Ekdysteroidy** - ekdyson, 20-hydroxyekdyson (20-E), makisteron A (=24-metyl-20E), 2-deoxyekdyson, 26-hydroxyekdyson a další
- 2. Juvenilní hormony** - JH-I, JH-II, JH-III, JH-0, 4-metyl-JH-I, kyselina juvenilního hormonu
- 3. Peptidické neurohormony**

I. Hormony řídící metabolismus a homeostázu

1. Adipokinetické hormony (AKH) a hypertrehalosemické hormony
2. Diuretické hormony
3. Antidiuretické hormony
4. Chloride transport stimulating hormone a ion transport peptide

II. Hormony řídící metamorfózu, vývoj a růst

1. Prothoracikotropní hormon (PTTH) a bombyxin
2. prothoracikostatický hormon (PTSH)
3. Allatostatiny a allatotropin
4. PBAN I, II, III (pheromone biosynthesis activating neuropeptide)
5. Ekložní hormon a *ecdysis triggering hormone (ETH)*
6. Burzikon
7. Faktory regulující puparizaci much
8. Diapauzní hormon

III. Hormony řídící pohlavní funkce

1. stimulační gonádotropní neurohormony (gonadotropiny) - ovary maturing parsin (OMP) a egg development neurohormone (EDNH) (=ovarian ecdysteroidogenic factor)
2. inhibiční neurohormony (antigonadotropiny, folikulostatiny) – neuroparsin, oostatické hormony a TMOF (trypsin-modulating oostatic factor).

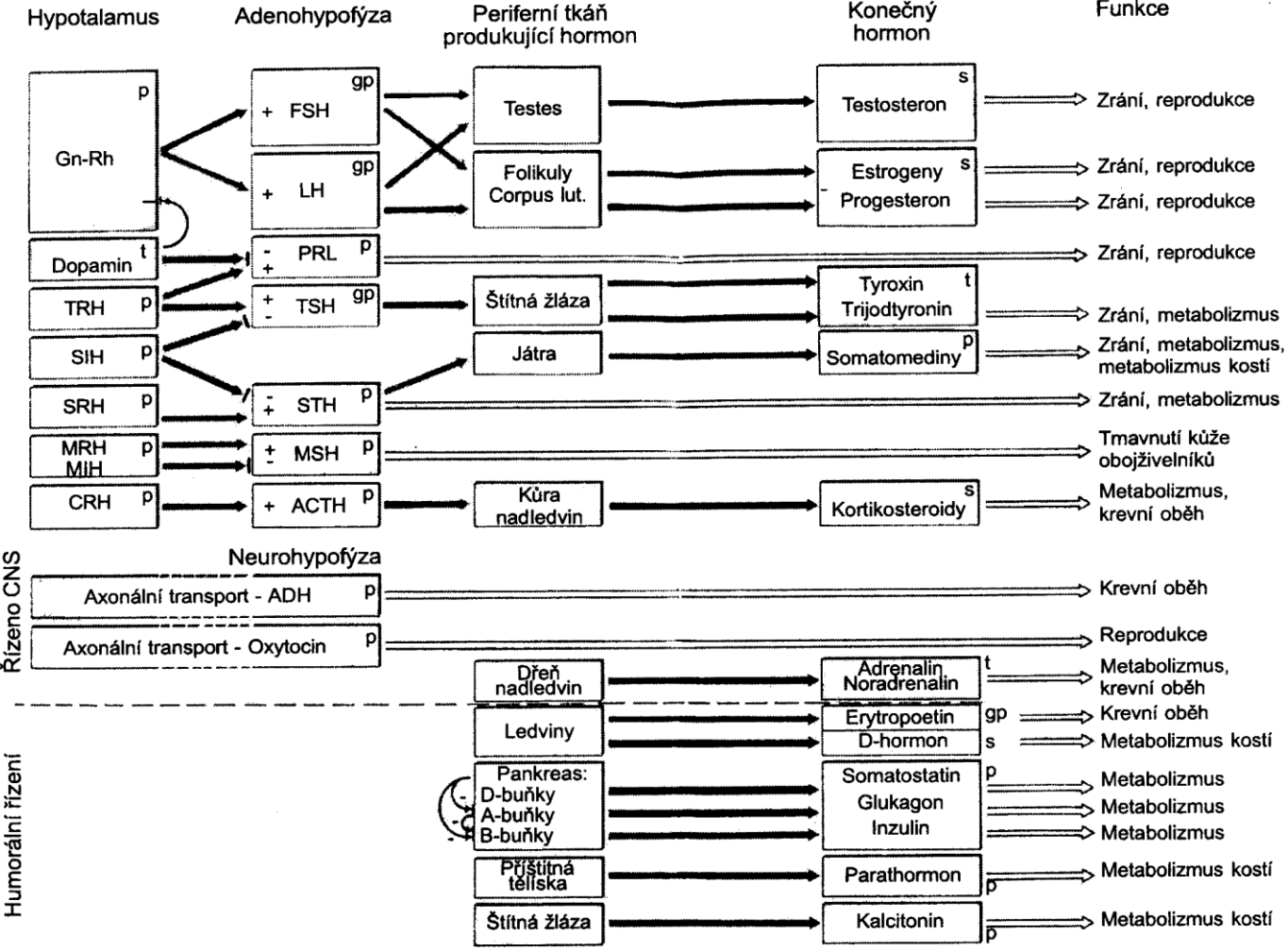
VI. Hormony modifikující svalovou kontrakci (myotropní peptidy)

1. Proctolin
2. Kardiostimulační hormony - crustacean cardioactive peptide (CCAP), corazonin
3. Skupiny myotropních neurohormonů - myokininy, sulfakininy, pyrokininy, tachykininy, myoinhibiční peptidy, periviscerokininy, FMRF-amid

V. Hormony řídící barvoměnu (chromatotropiny)

1. PDF - pigment dispersing factor
2. MRCH - melanization and reddish coloratig hormone (identický s PBAN)

Přehled endokrinní soustavy člověka

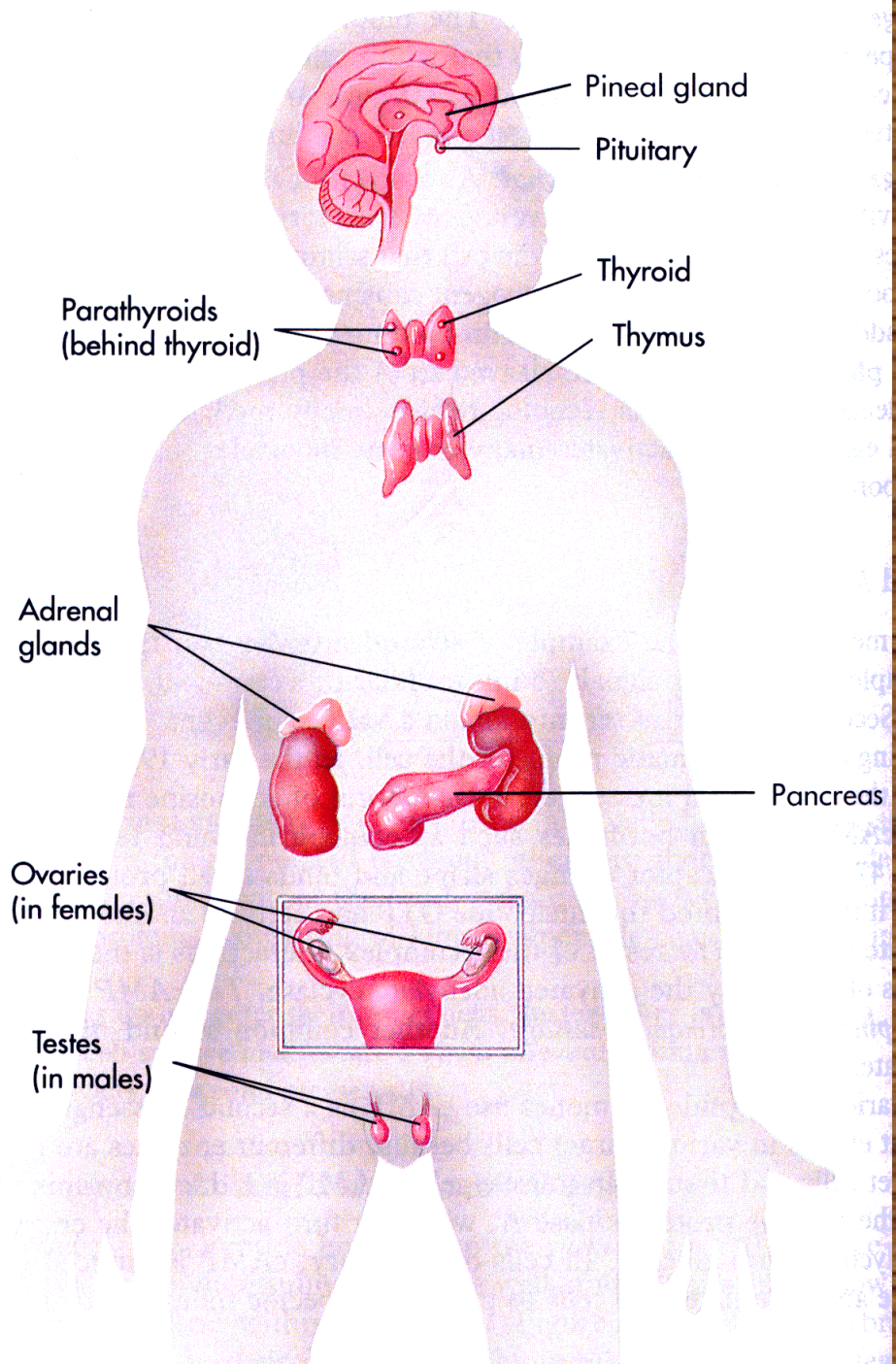


Použitá jména a zkratky hormonů:

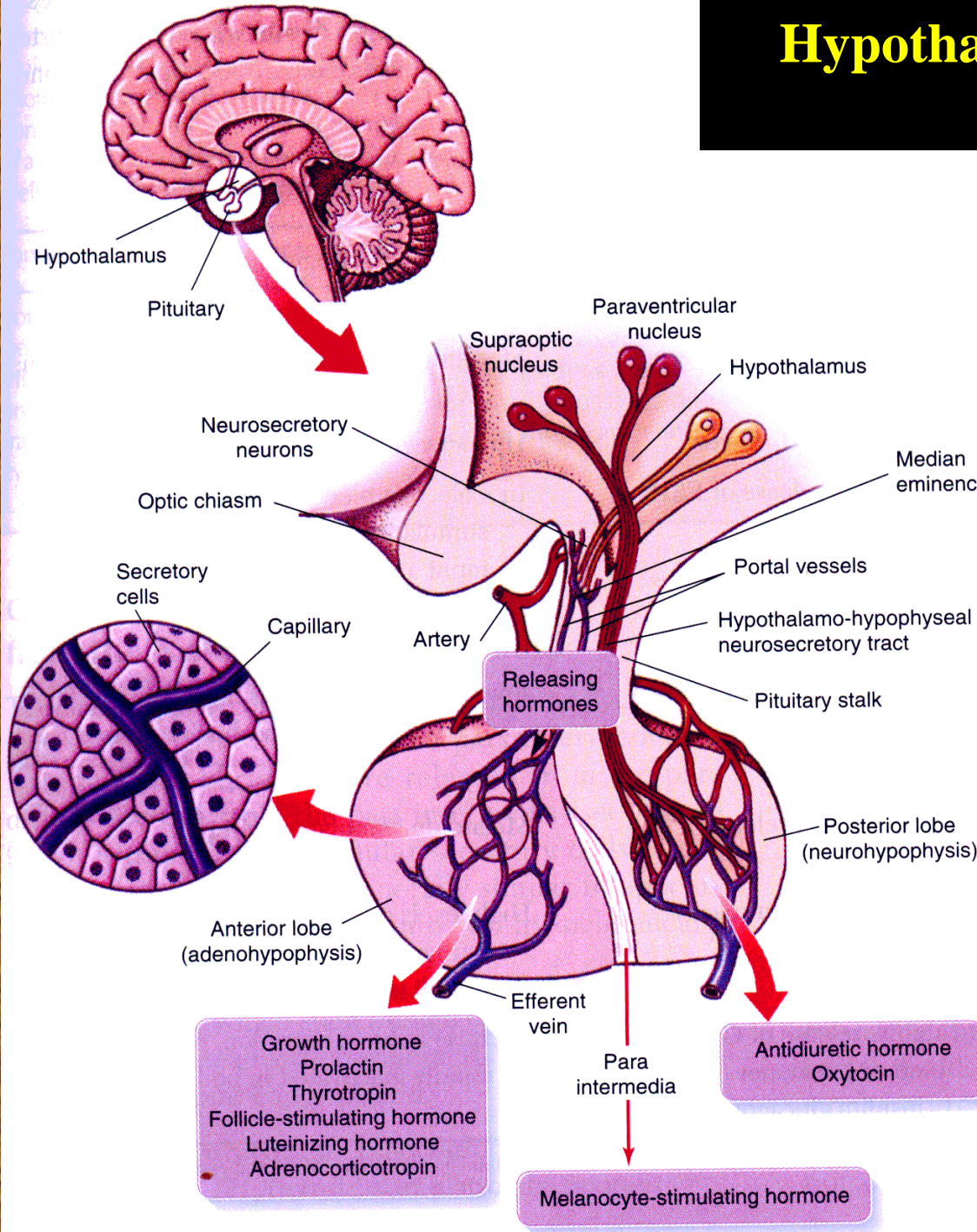
Hypotalamus:		Adenohypofýza		Neurohypofýza	
Kortikoliberin	CRH	Kortikotropin	ACTH	Oxytocin	
Gonadoliberin	Gn-RH	Folítropin	FSH	Adiuretin	ADH
Melanoliberin	MRH	Lutropin	LH		
Melanostatin	MIH	Melanotropin	MSH		
Prolaktostatin = Dopamin	PIH	Somatotropin	STH		
Somatoliberin	SRH	Tyotropin	TSH		
Somatostatin	SIH	Prolaktin	PRL		
Tyreoliberin	TRH				

Obr. 15.6. Přehled hlavních hormonálních os člověka. (p) proteinové hormony, (s) steroidní, (t) odvozené od tyrozinu, (+) stimulační vliv, (-) inhibiční vliv.

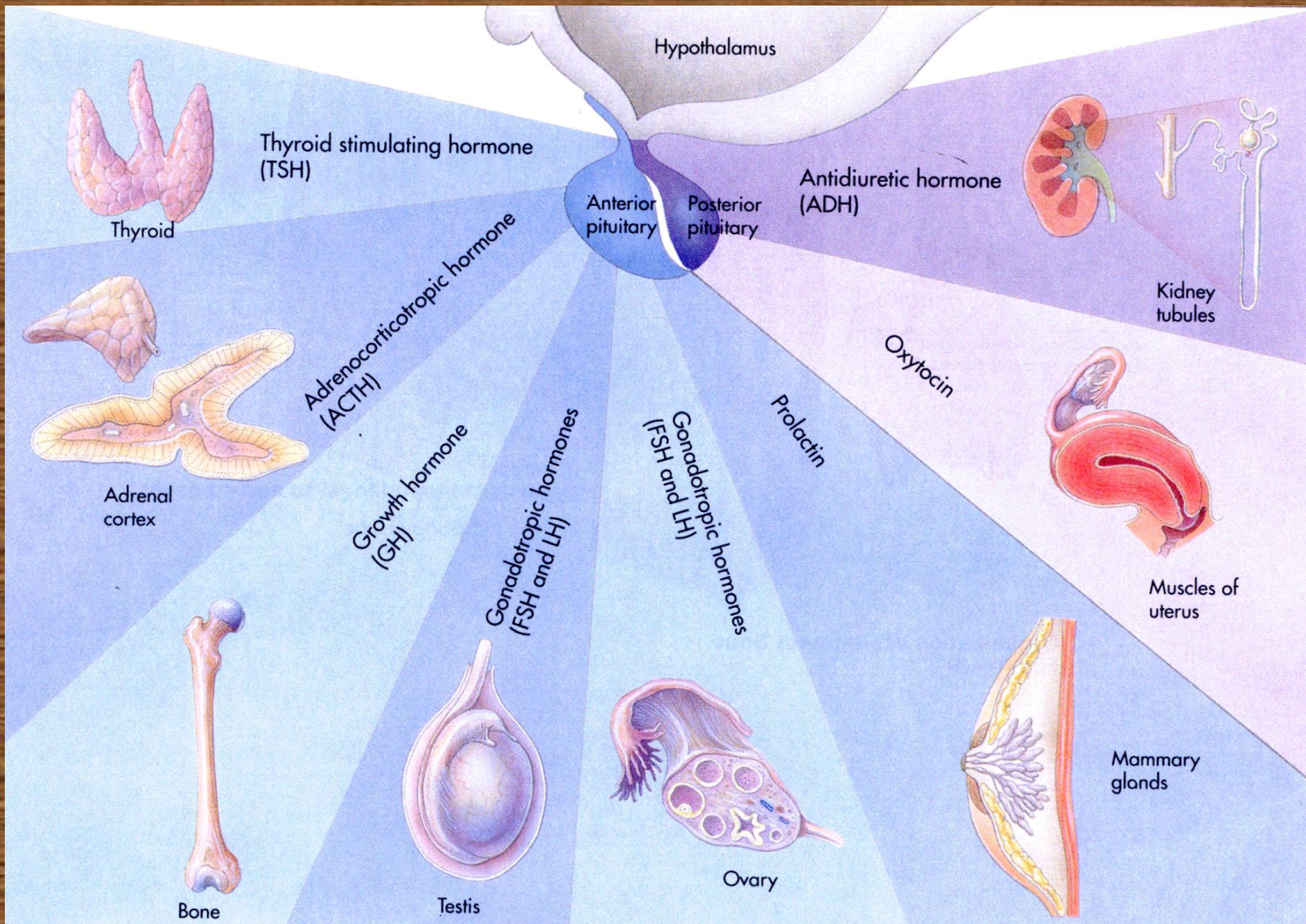
Endokrinní soustava člověka



Hypothalamo-hypofyzární soustava



Hormony hypofýzy



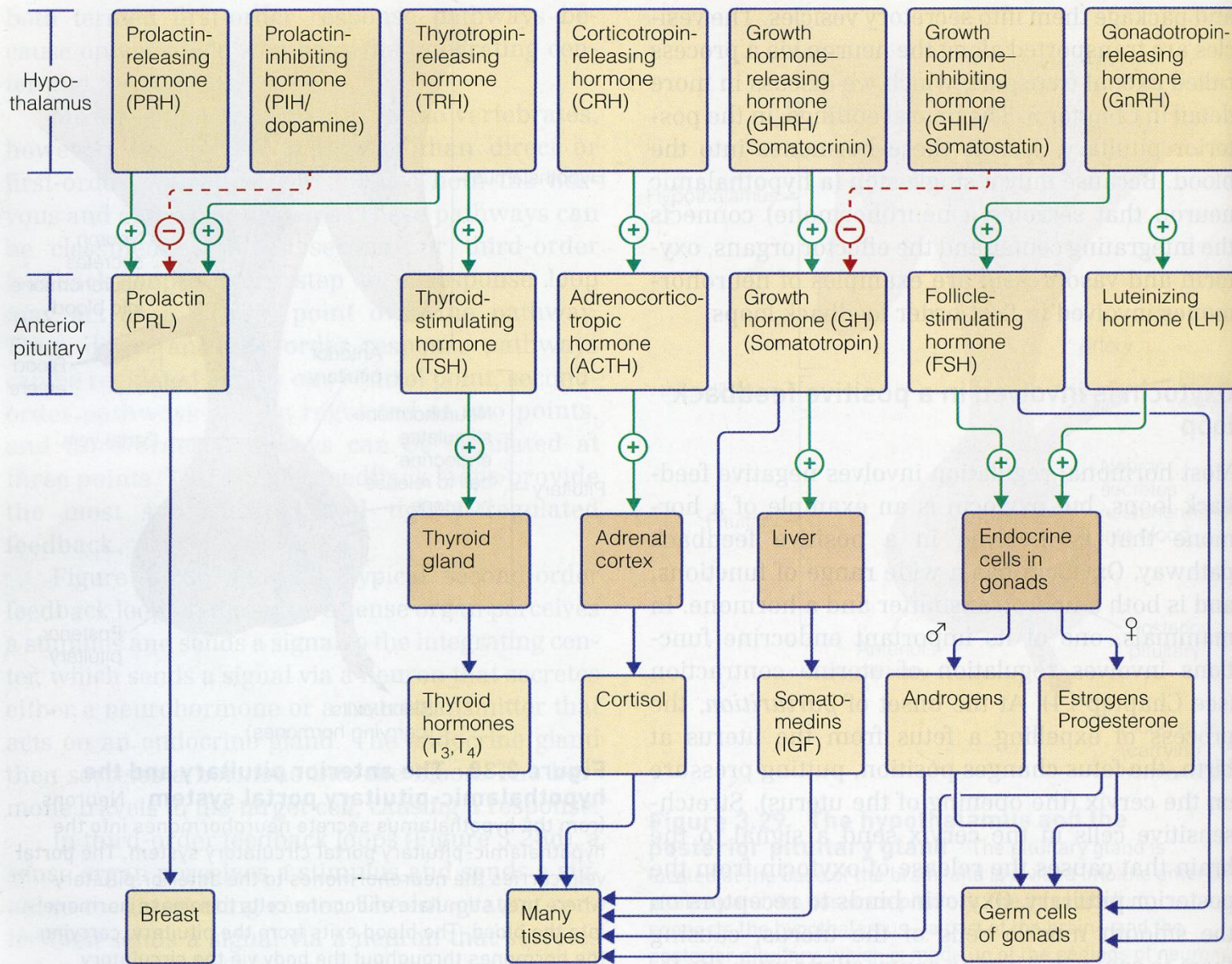
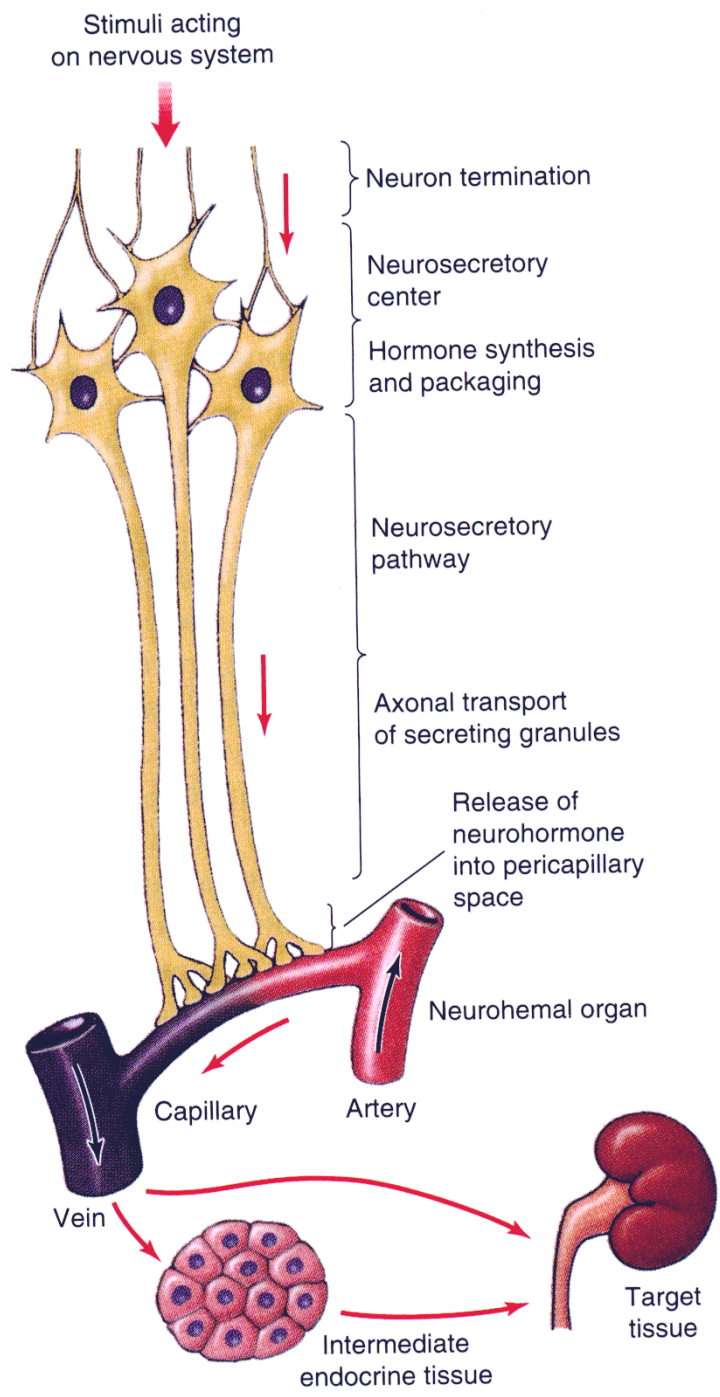


Figure 3.31 The relationship between the hypothalamic hormones and the hormones of the anterior pituitary The hypothalamus secretes releasing or inhibiting neurohormones into the hypothalamic-pituitary portal system. These neurohormones act on the endocrine cells of the anterior pituitary to

stimulate or inhibit the release of the pituitary hormones. The circulatory system carries these hormones to their target tissues, causing a response. Some of these target tissues are endocrine glands, which secrete hormones into the blood. The circulatory system carries these hormones to their target tissues, causing a response.



Sekrece hormonů z adenohipofýzy

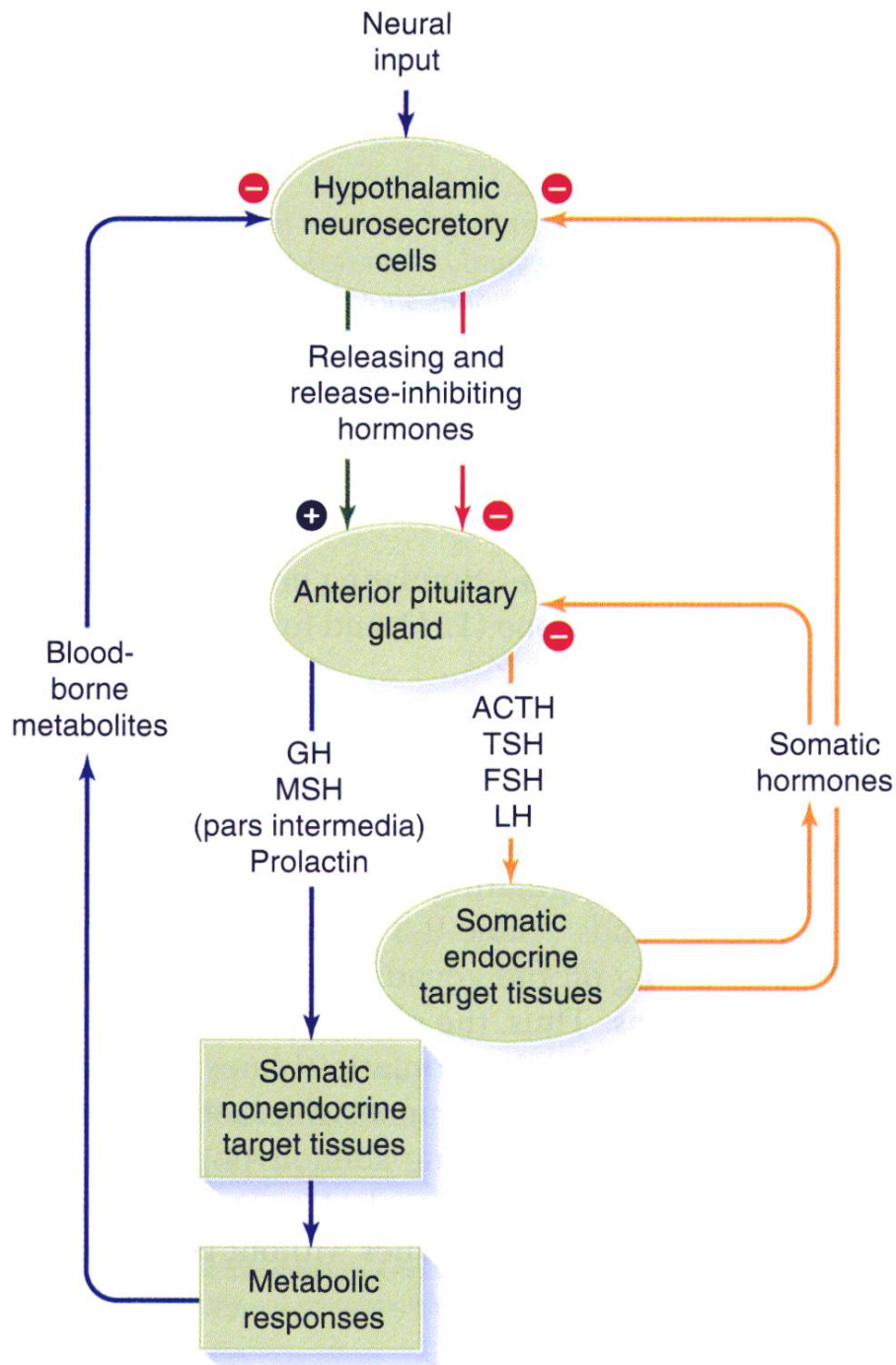
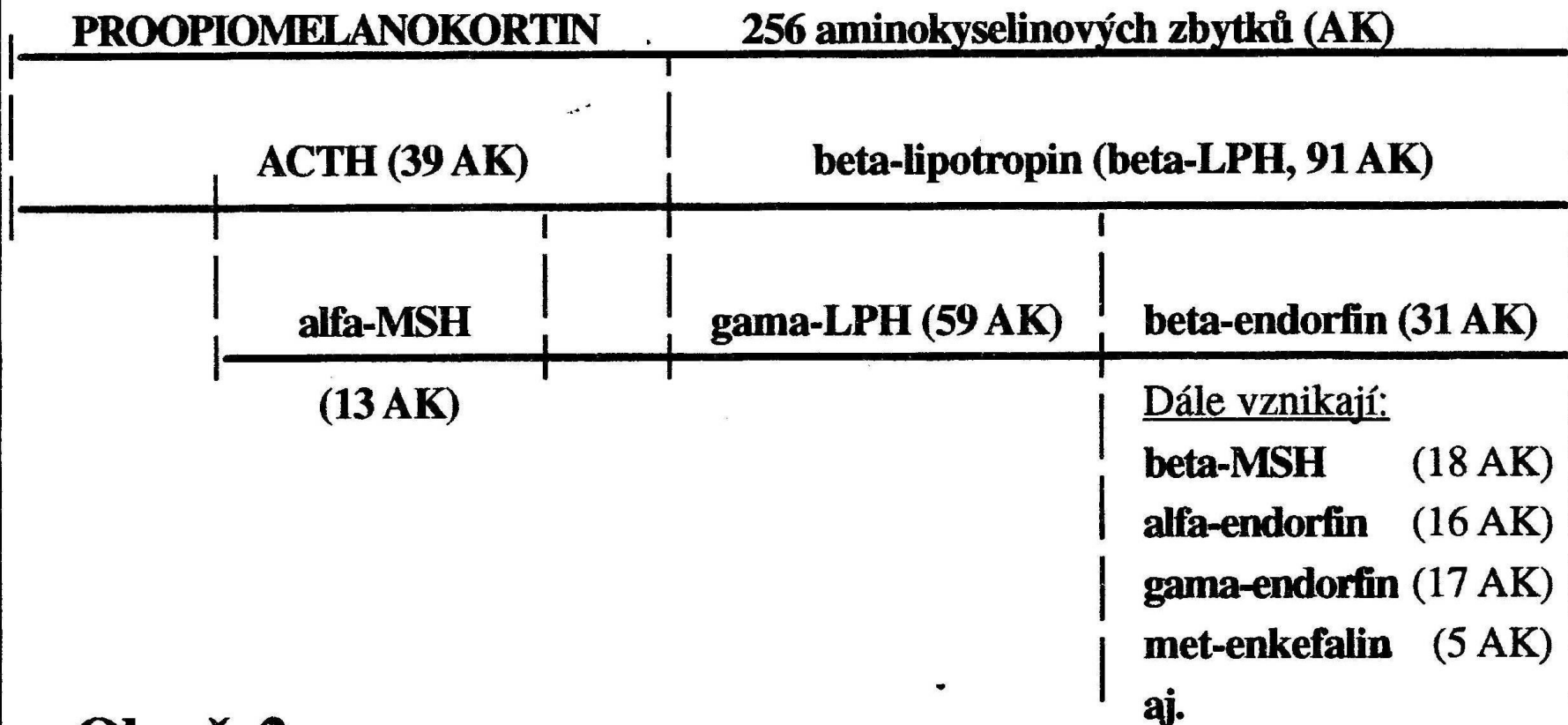


Schéma zpětnovazebné regulace výdeje adenohipofyzárních hormonů



Obr. č. 2:

Schématické znázornění souvislostí mezi některými peptidy,

vznikajícími v adenohipofýze (uvedené látky mají odpovídající si pořadí AK)

(upraveno podle Šterzla, J., 1988)

Physiological Processes

Table 9-3 Tropic hormones of the anterior pituitary gland

Hormone	Structure	Target tissue	Primary action in mammals	Regulation*
Adrenocorticotrophic hormone (ACTH)	Peptide	Adrenal cortex	Increases synthesis and secretion of steroid hormones by adrenal cortex	Cortical-releasing hormone (CRH) stimulates release; ACTH slows release of CRH
Follicle-stimulating hormone (FSH)	Glycoprotein	Ovarian follicles (female); seminiferous tubules (male)	In female, stimulates maturation of ovarian follicles; in male, increases sperm production	GnRH stimulates release; inhibin and steroid sex hormones inhibit release
Luteinizing hormone (LH)	Glycoprotein	Ovarian interstitial cells (female); testicular interstitial cells (male)	In female, induces final maturation of ovarian follicles, estrogen secretion, ovulation, corpus luteum formation, and progesterone secretion; in male, increases synthesis and secretion of androgens	GnRH stimulates release; inhibin and steroid sex hormones inhibit release
Thyroid-stimulating hormone (TSH)	Glycoprotein	Thyroid gland	Increases synthesis and secretion of thyroid hormones	TRH induces secretion; thyroid hormones and somatostatin slow release

*See Table 9-2 for key to abbreviations.

Úloha rústového hormonu

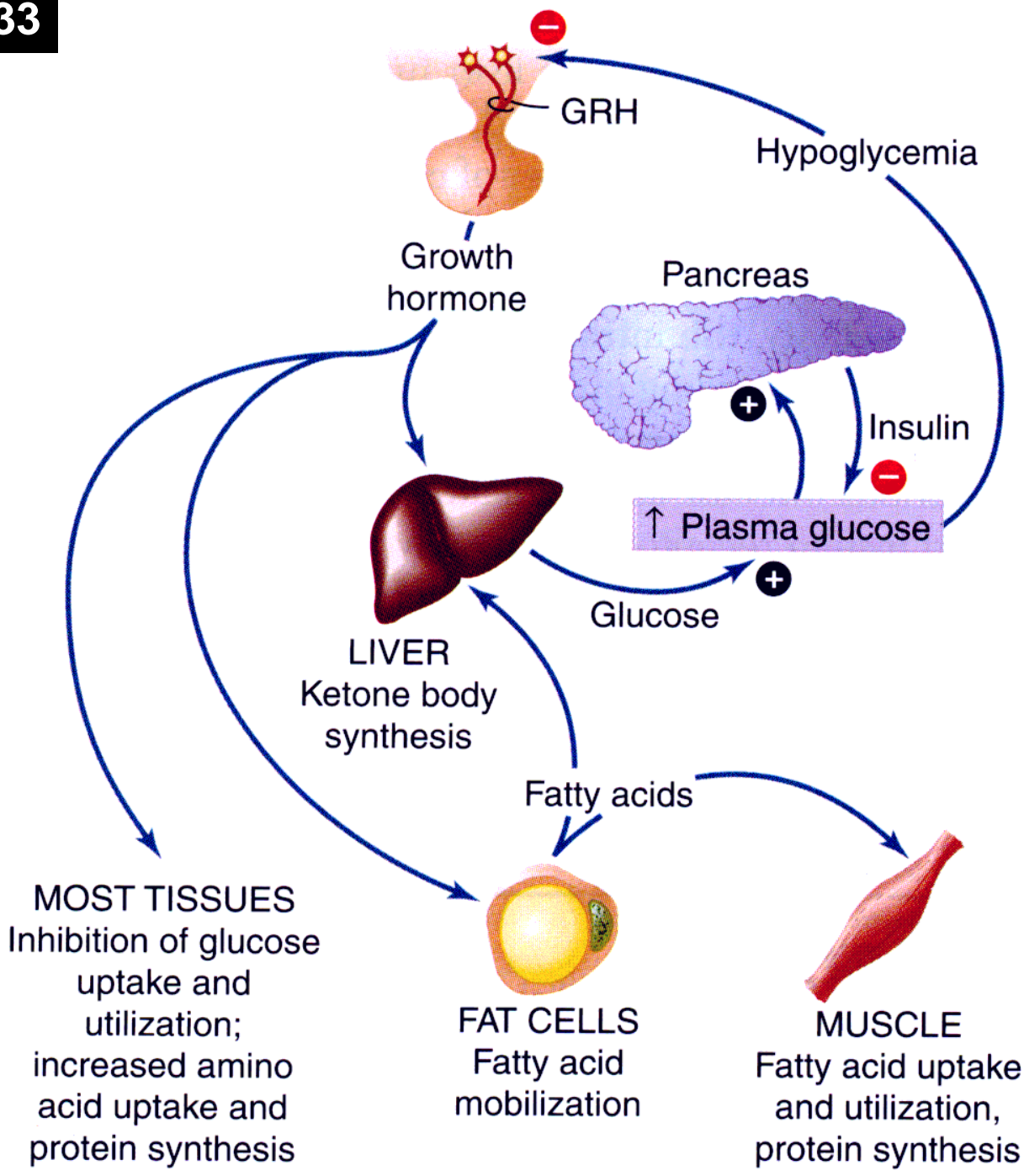


Figure 9-44 Many of the actions of growth hormone are antagonistic to those of insulin and similar to those of glucagon. Output of insulin from pancreatic beta cells occurs in response to high blood glucose, as after a meal. Growth hormone (GH) is released, usually several hours after a meal or after prolonged exercise, in response to insulin-induced hypoglycemia. Growth hormone causes lipolysis and the uptake of fatty acids by muscle tissue for energy and by the liver for ketone body synthesis. The GH-induced general depression of glucose uptake (except in the central nervous system) leads to a rise in plasma glucose, which then stimulates insulin secretion. The insulin stimulates glucose uptake into cells and thus counteracts GH-induced hyperglycemia.

Neurohypofýza a jí vylučované hormony

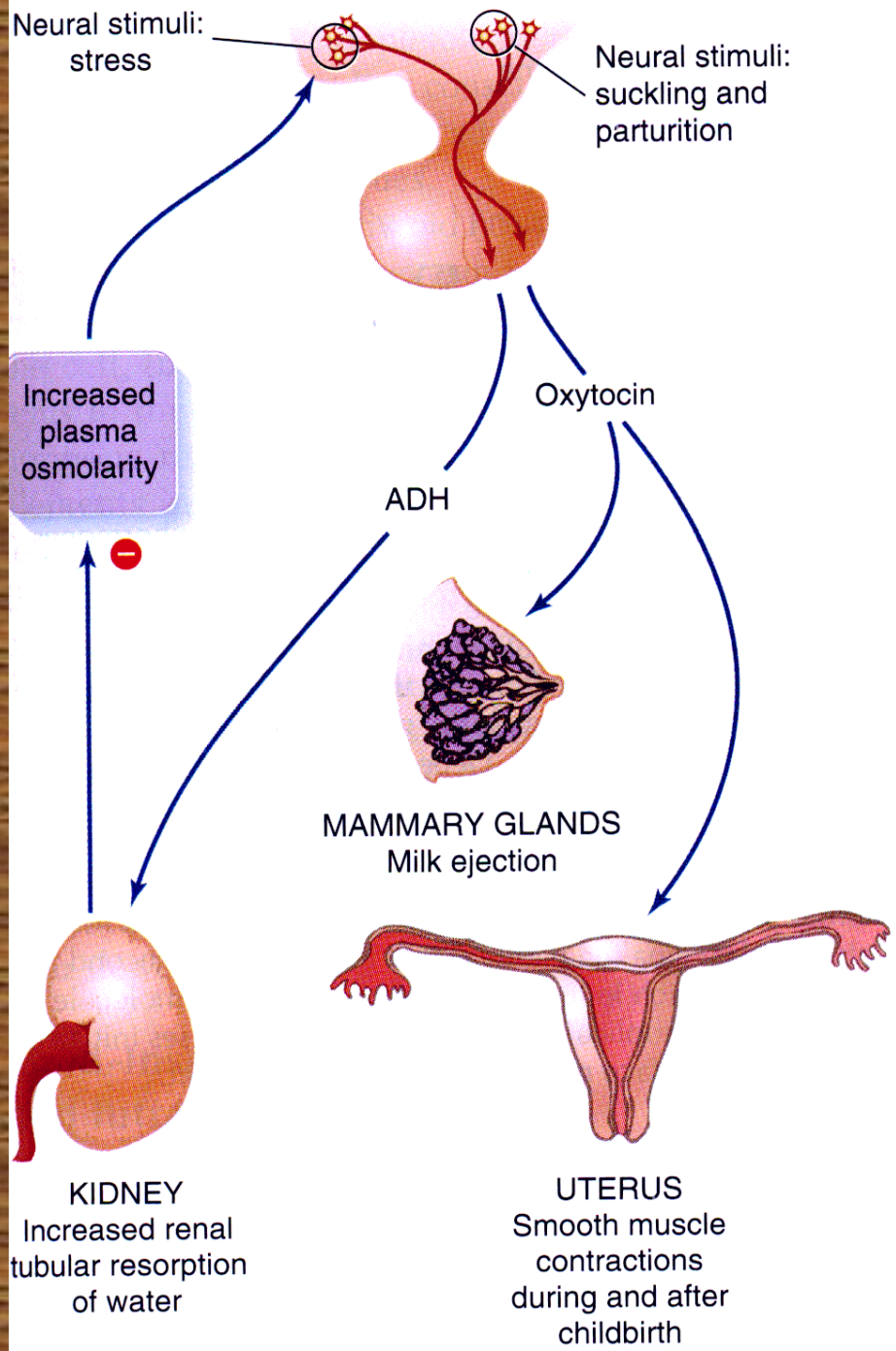


Table 9-4 Variant forms of neurohypophyseal nonapeptide hormones

Peptide	Positions of amino acid residues*									Animal group	
	1	2	3	4	5	6	7	8	9		
Lysine vasopressin	Cys	Tyr	Phe	Gln	Asn	Cys	Pro	Lys	Gly	(NH ₂)	Pigs and relatives
Arginine vasopressin	Cys	Tyr	Phe	Gln	Asn	Cys	Pro	Arg	Gly	(NH ₂)	Mammals
Oxytocin	Cys	Tyr	Ile	Gln	Asn	Cys	Pro	Leu	Gly	(NH ₂)	Mammals
Arginine vasotocin	Cys	Tyr	Ile	Gln	Asn	Cys	Pro	Arg	Gly	(NH ₂)	Reptiles, fishes, and birds
Isotocin	Cys	Tyr	Ile	Ser	Asn	Cys	Pro	Ile	Gly	(NH ₂)	Some teleosts
Mesotocin	Cys	Tyr	Ile	Gln	Asn	Cys	Pro	Ile	Gly	(NH ₂)	Reptiles, amphibians, and lungfishes
Glomitocin	Cys	Tyr	Ile	Ser	Asn	Cys	Pro	Gln	Gly	(NH ₂)	Some elasmobranchs

*The cysteine residues in positions 1 and 6 of each peptide are bridged by a disulfide bond.

Source: Frieden and Lipner, 1971.

Štítná žláza a thyroïdní hormony

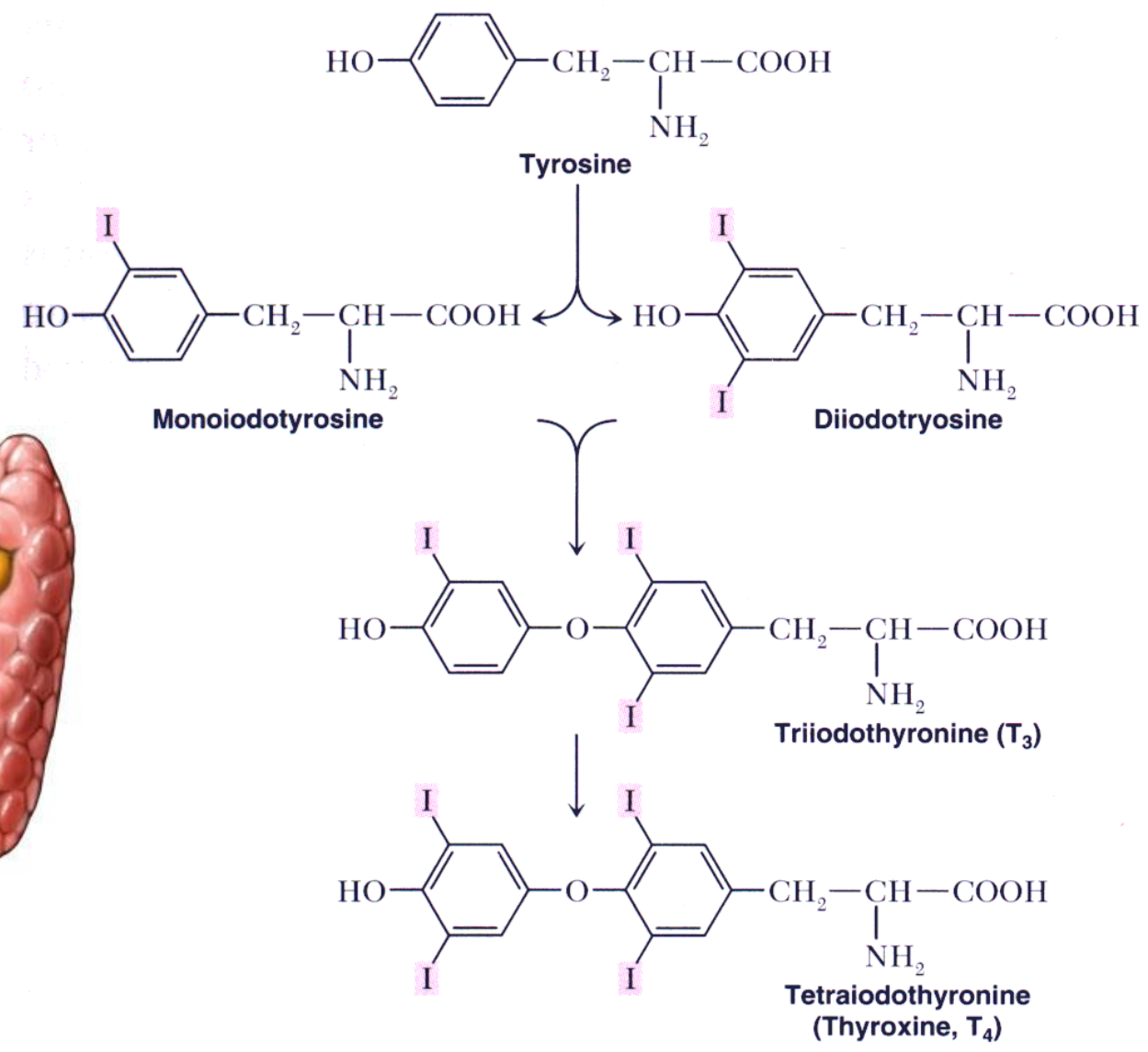
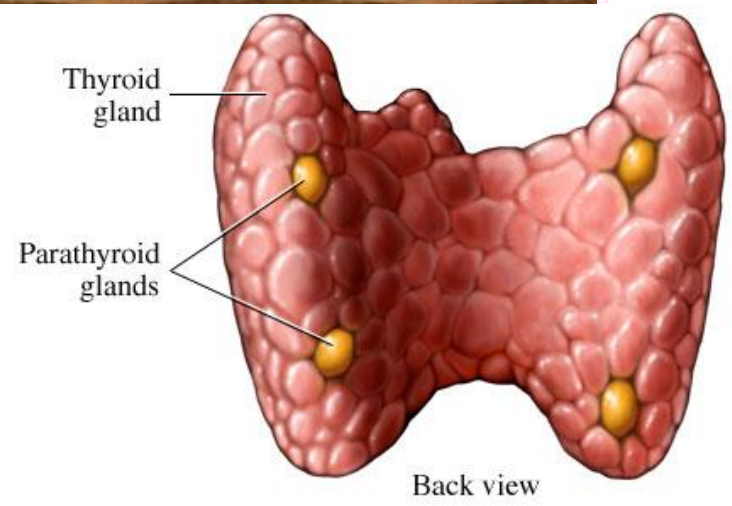
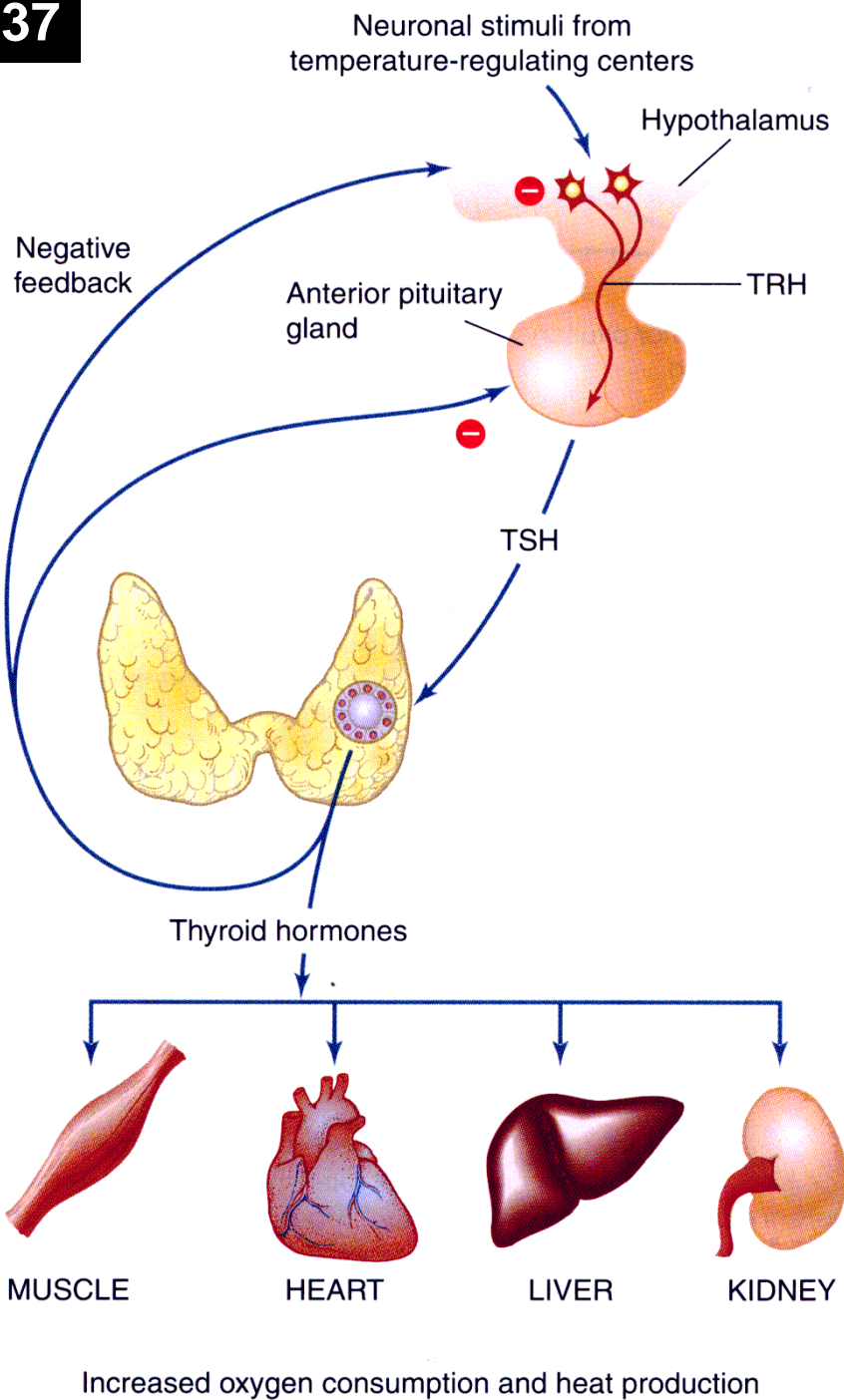
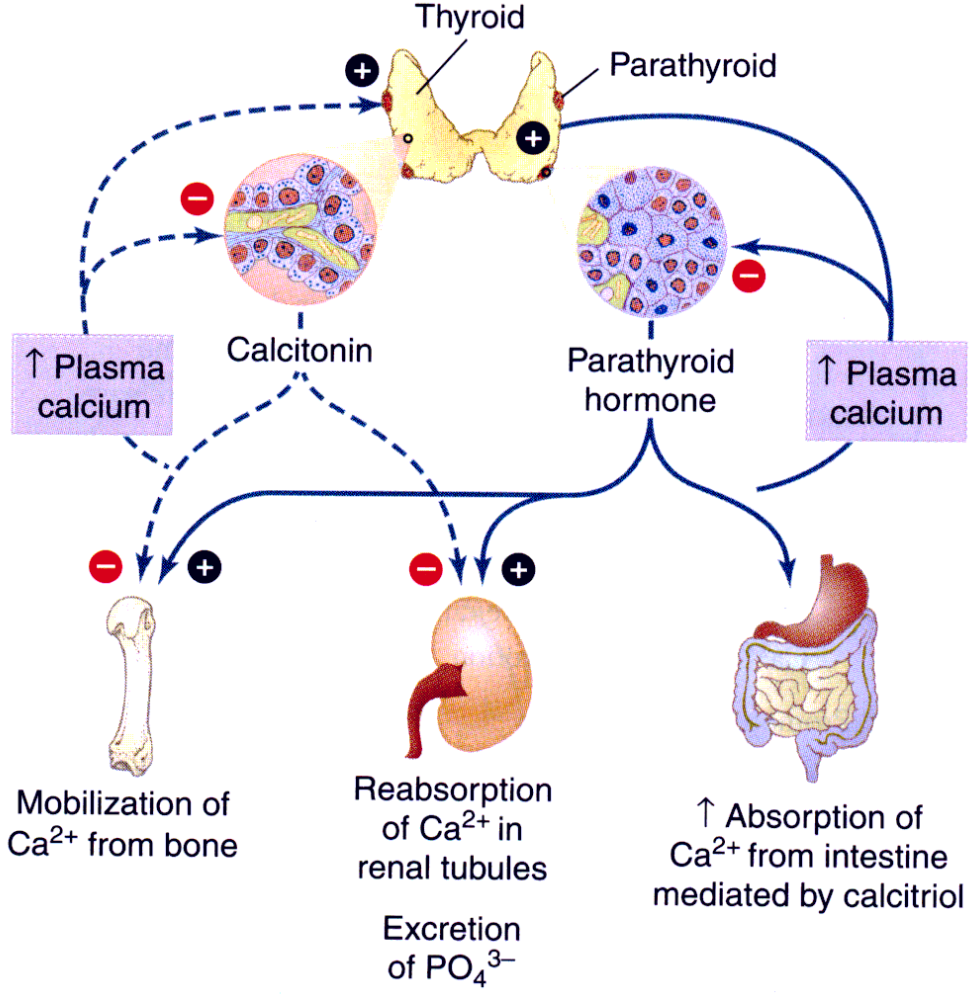


Figure 9-41 Thyroid hormones are produced from iodinated derivatives of the amino acid tyrosine. Condensation of the tyrosine derivatives yields 3,5,3'-triiodothyronine (T₃) and thyroxine (T₄). T₃ is also produced by removal of one iodide from thyroxine.



Štítná žláza a její hormony

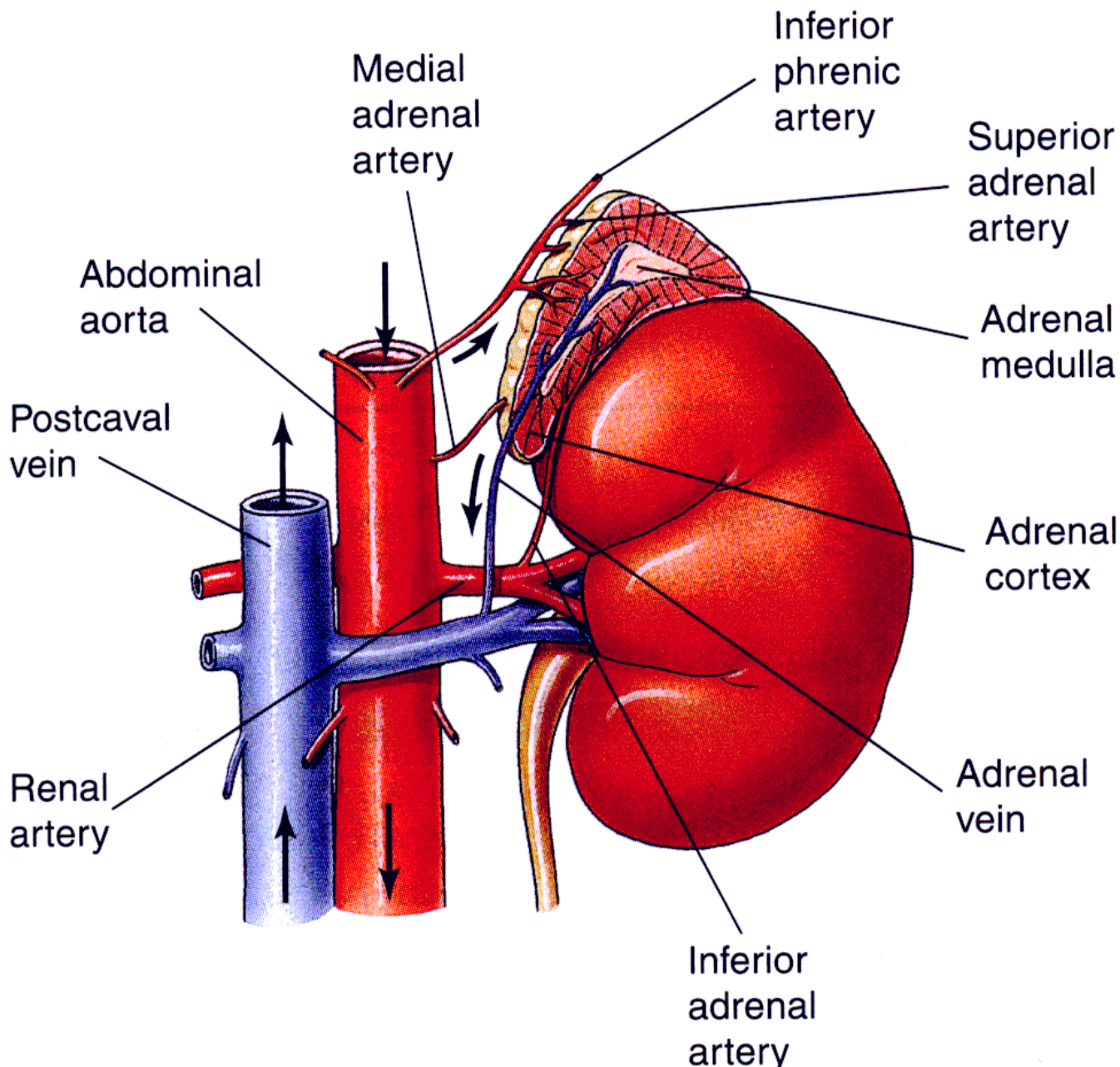
Figure 9-42 Thyroid hormones, which regulate metabolism in various tissues, are themselves regulated by neuronal stimuli and negative feedback. Low skin temperature and stress stimulate the release of TSH-releasing hormone (TRH) from hypothalamic neurosecretory cells. TRH then stimulates secretion of thyroid-stimulating hormone (TSH) from the anterior pituitary gland. The thyroid responds by secreting the thyroid hormones, which cause increased metabolism in skeletal and cardiac muscle, liver, and kidney, leading to the metabolic generation of heat. Feedback inhibition by thyroid hormones apparently occurs at the levels of both the anterior pituitary and the hypothalamus. The follicle shown superimposed on the thyroid gland is drawn at a disproportionately large scale.



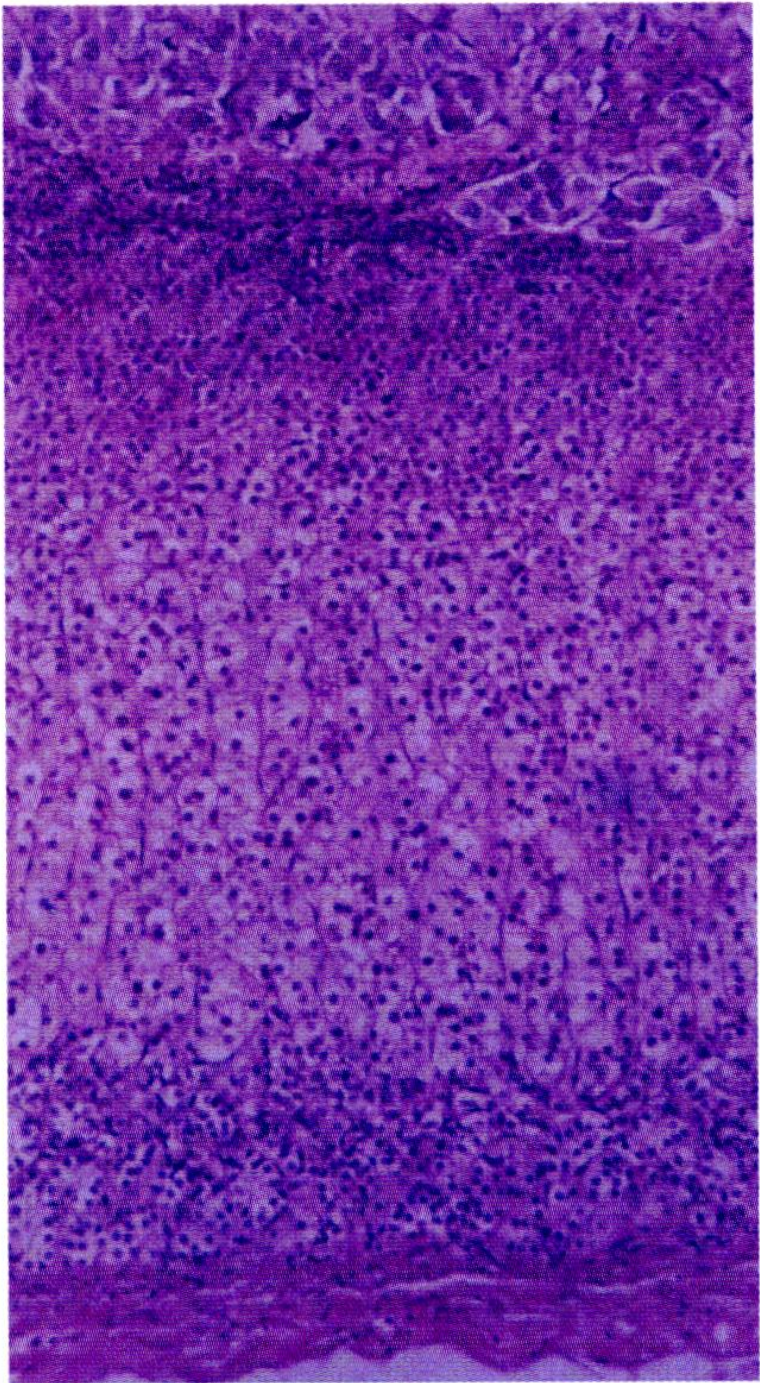
**Štítná žláza a
příštítná tělíska –
kalcitonin a
parathormon**

Figure 9-45 Calcitonin and parathyroid hormone (PTH) have opposite effects on plasma Ca²⁺ levels in mammals. Low levels of plasma Ca²⁺ stimulate the cells of the parathyroid glands to release PTH, which has several actions, all tending to increase plasma Ca²⁺. High concentrations of Ca²⁺ in the blood stimulate parafollicular cells in the thyroid gland to release calcitonin, which acts to increase plasma Ca²⁺. Calcitriol, the active hormonal form of vitamin D, also increases intestinal absorption of Ca²⁺.

Left Adrenal Gland



Anatomie kůry nadledvin



Adrenal medulla

Zona reticularis

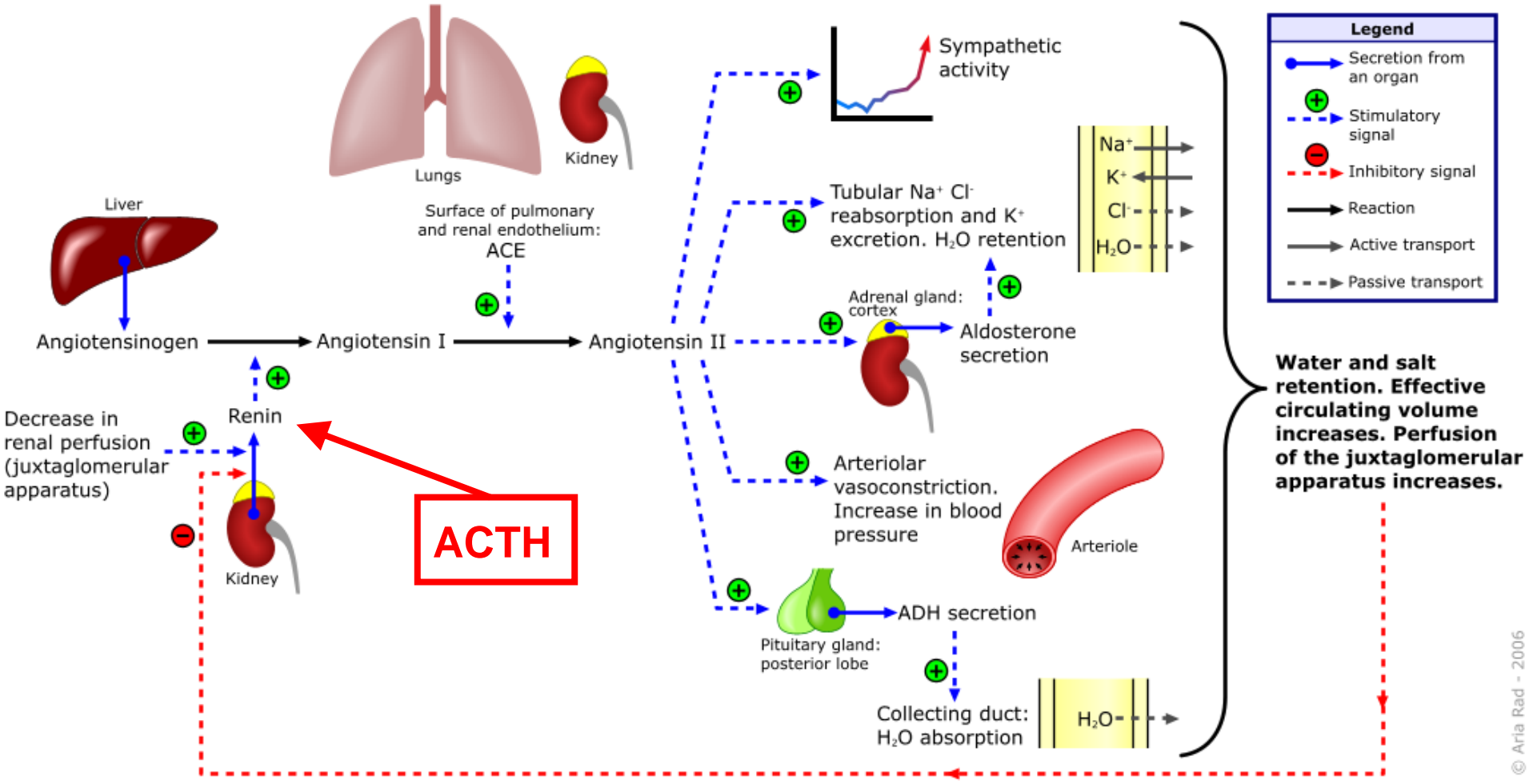
Zona fasciculata

Zona glomerulosa

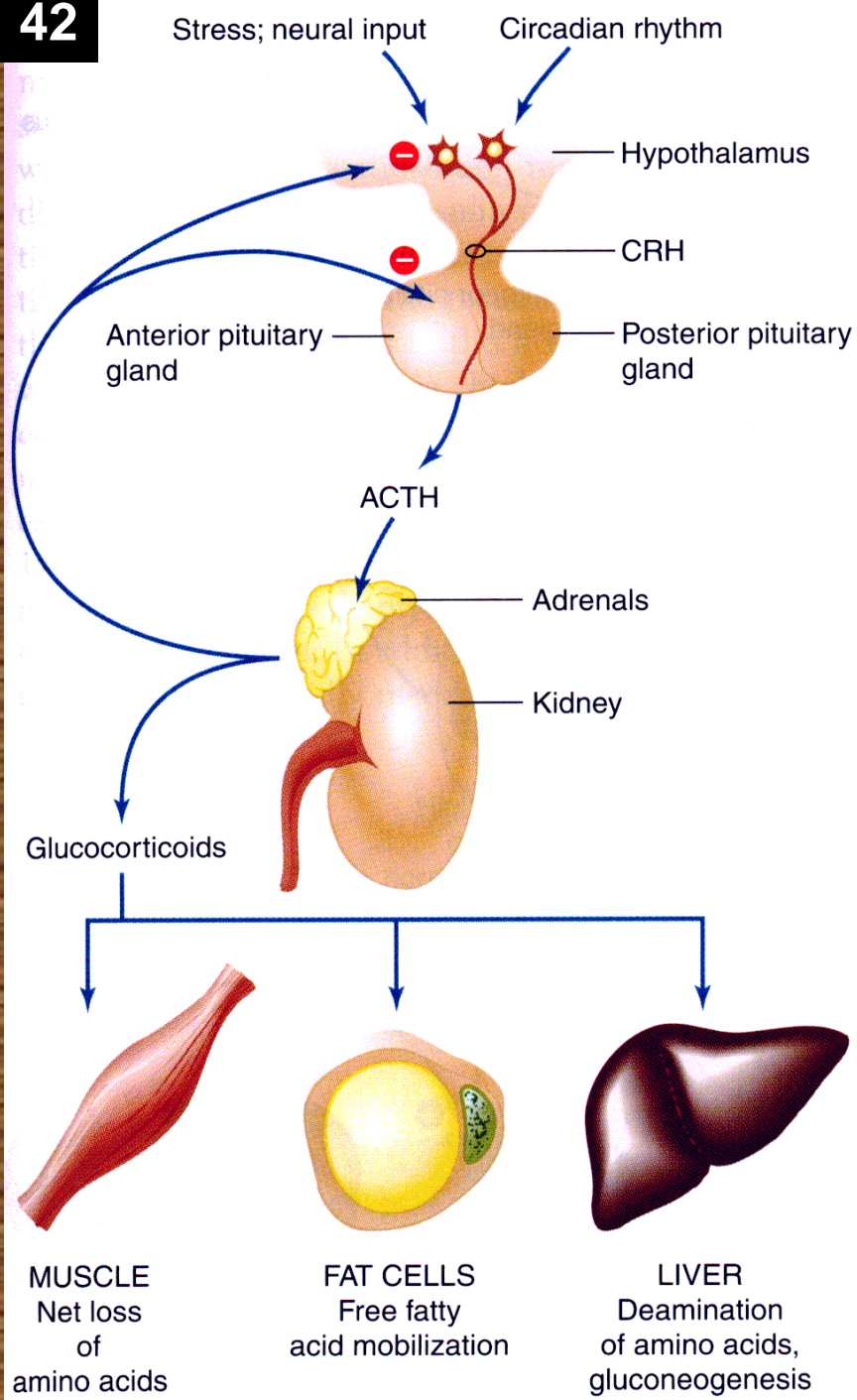
Capsule

Adrenal cortex

Renin-angiotensin-aldosterone system



Legend:
 ACE – angiotenzin converting enzyme
 ADH –antidiuretic hormone

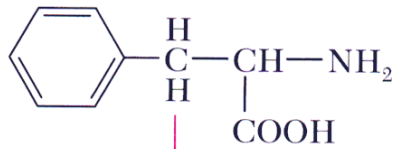


Glukokortikoidy a jejich úloha

Figure 9-40 The secretion of glucocorticoids, and hence their effects on target tissues, is regulated by neuronal stimuli and negative feedback. Neuronal stimuli induce the release of corticotropin-releasing hormone (CRH) from hypothalamic neurosecretory cells. The resulting release of adrenocorticotropic hormone (ACTH) from the anterior pituitary gland stimulates the secretion of glucocorticoids by the adrenal cortex. These steroids produce an increase in blood glucose and liver glycogen by stimulating conversion of amino acids and fats to glucose. Negative feedback by the glucocorticoids to both the pituitary and the hypothalamus may limit ACTH release.

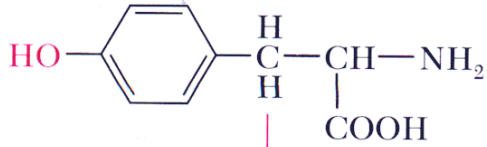
Katecholaminy a jejich syntéza

Phenylalanine



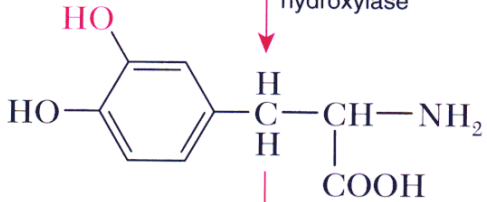
Phenylalanine hydroxylase

Tyrosine



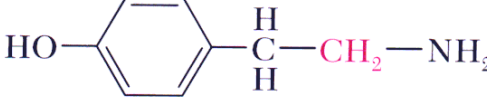
Tyrosine hydroxylase

Dopa



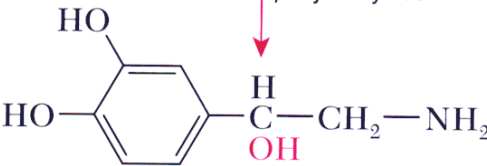
Dopa decarboxylase

Dopamine



Dopamine β -hydroxylase

Norepinephrine



Phenylethanolamine *N*-methyltransferase (glucocorticoids \uparrow)

Epinephrine

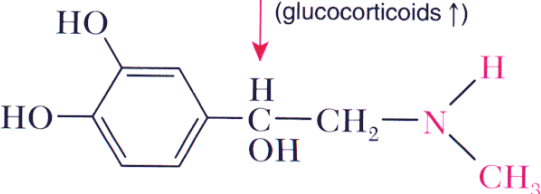


Figure 9-35 The catecholamines—dopamine, norepinephrine, and epinephrine—are synthesized from phenylalanine and tyrosine. Glucocorticoids produced by the adrenal cortex increase the activity of phenylethanolamine *N*-methyltransferase, thereby promoting the conversion of norepinephrine to epinephrine.

Syntéza (1) a sekrece (2) katecholanimů

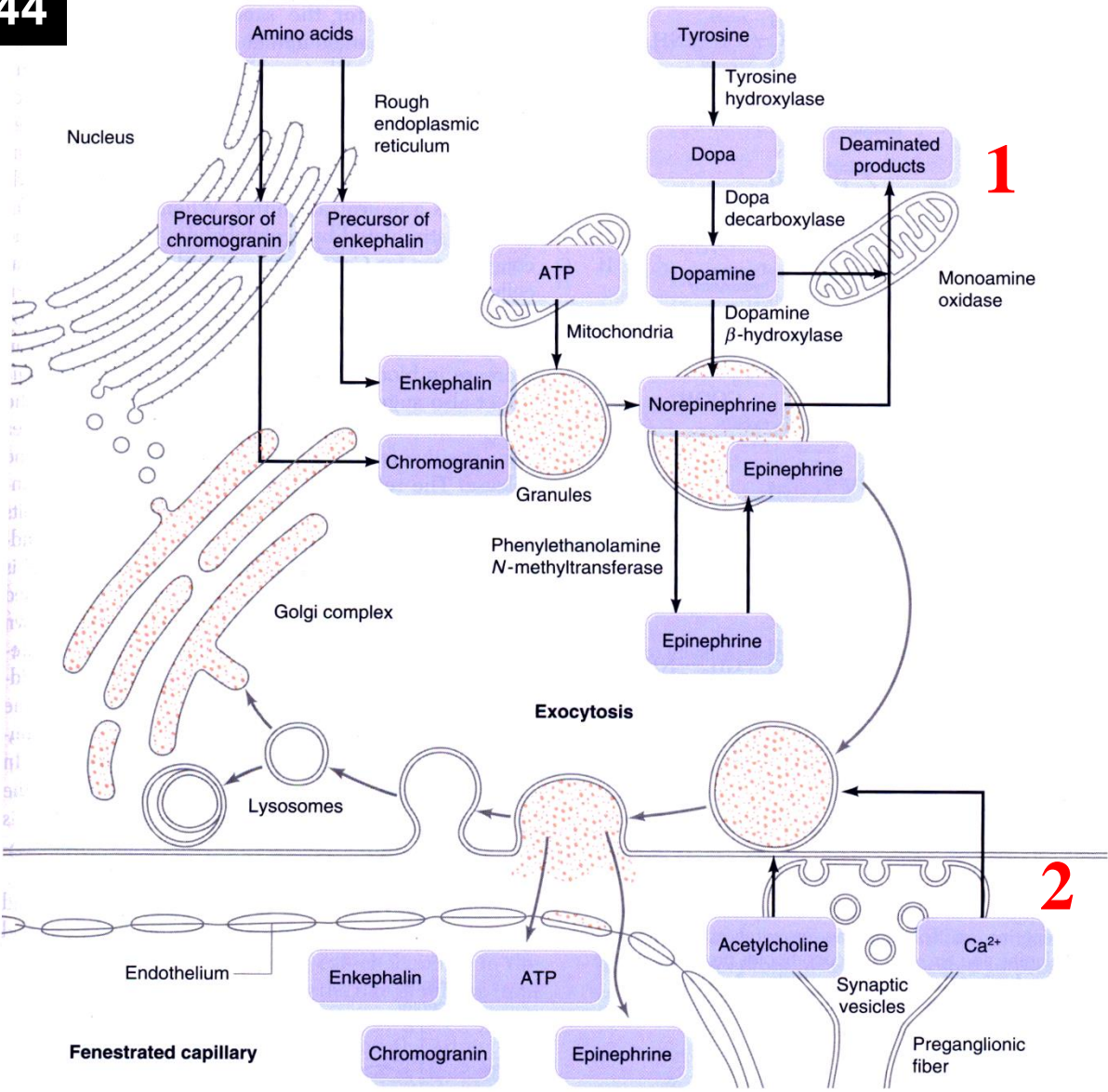


Figure 9-34 Secretory vesicles in the chromaffin cells of the adrenal medulla contain catecholamines, enkephalin, ATP, and chromogranin, all of which are synthesized in different cellular compartments. In epinephrine-producing cells (shown here), norepinephrine leaves the secretory vesicles, is converted to epinephrine, and then is reincorporated into the vesicles. Stimulation of chromaffin cells by acetylcholine,

which is liberated from the terminals of preganglionic sympathetic nerve fibers, triggers release of the granule contents by exocytosis. The neuronal stimulus increases the membrane permeability for Ca²⁺, leading to the increased intracellular Ca²⁺ required for exocytosis. [Adapted from Matsumoto and Ischii, 1992.]

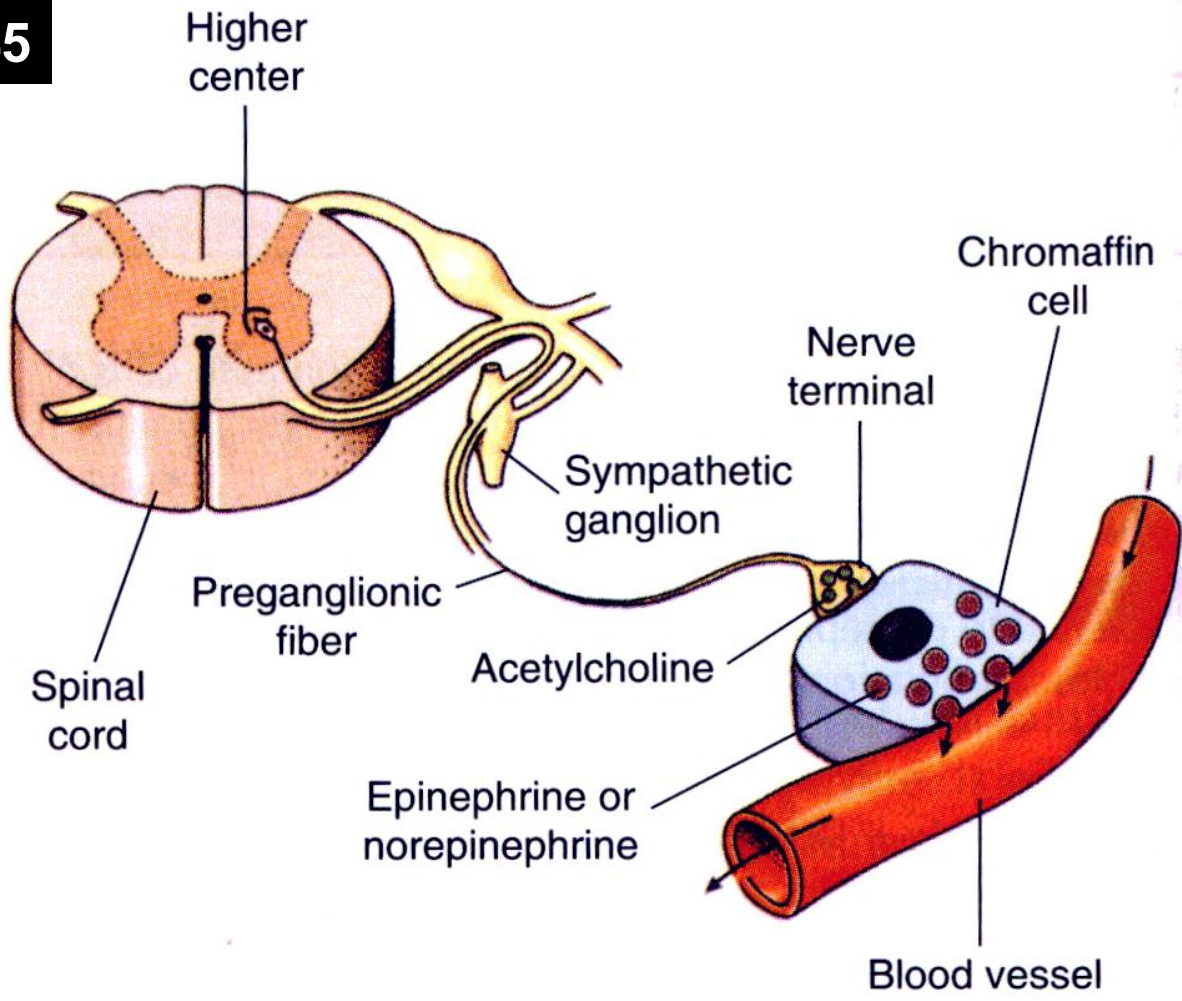
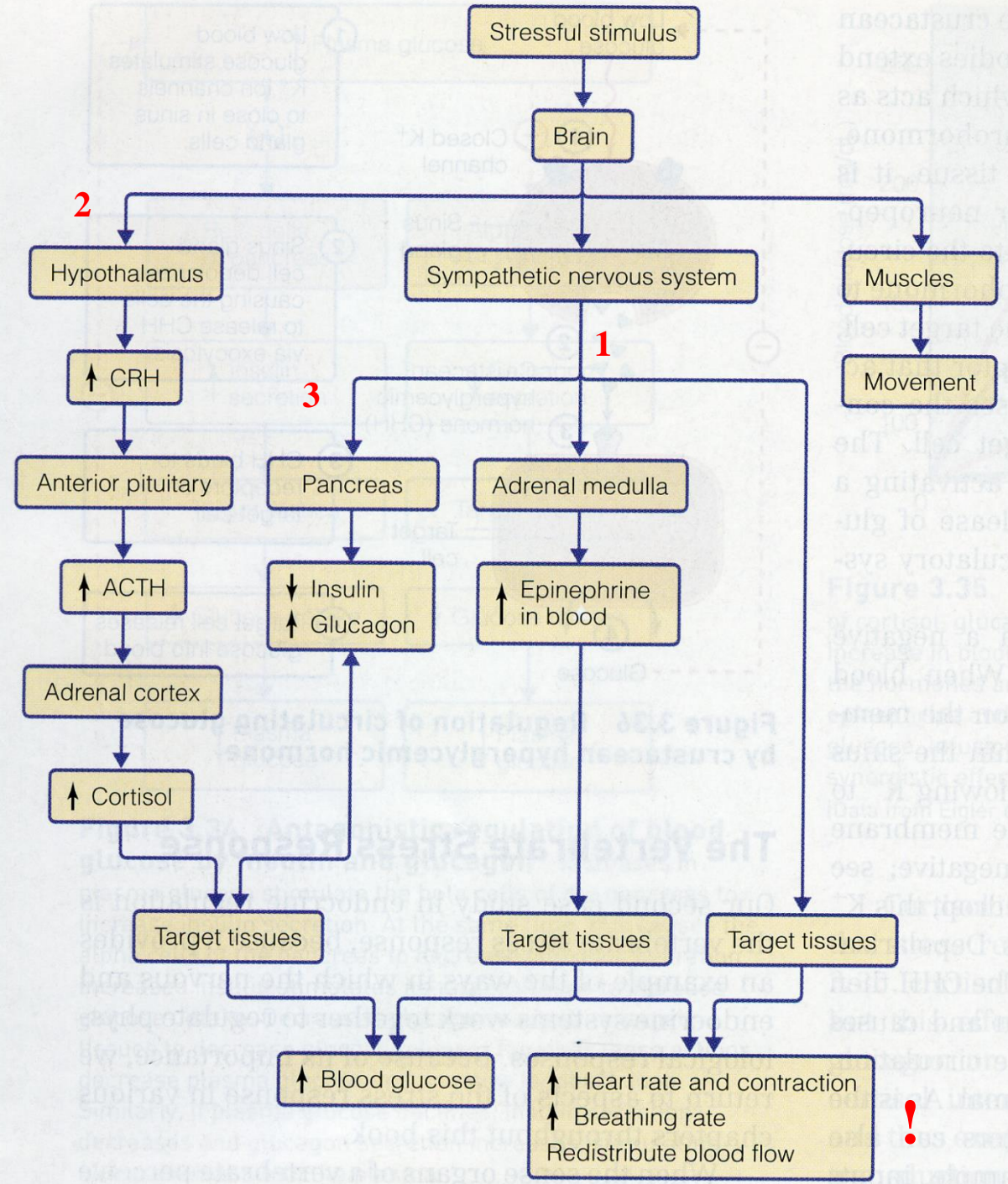


Schéma sekrece katecholaminů z dřeně nadledvin

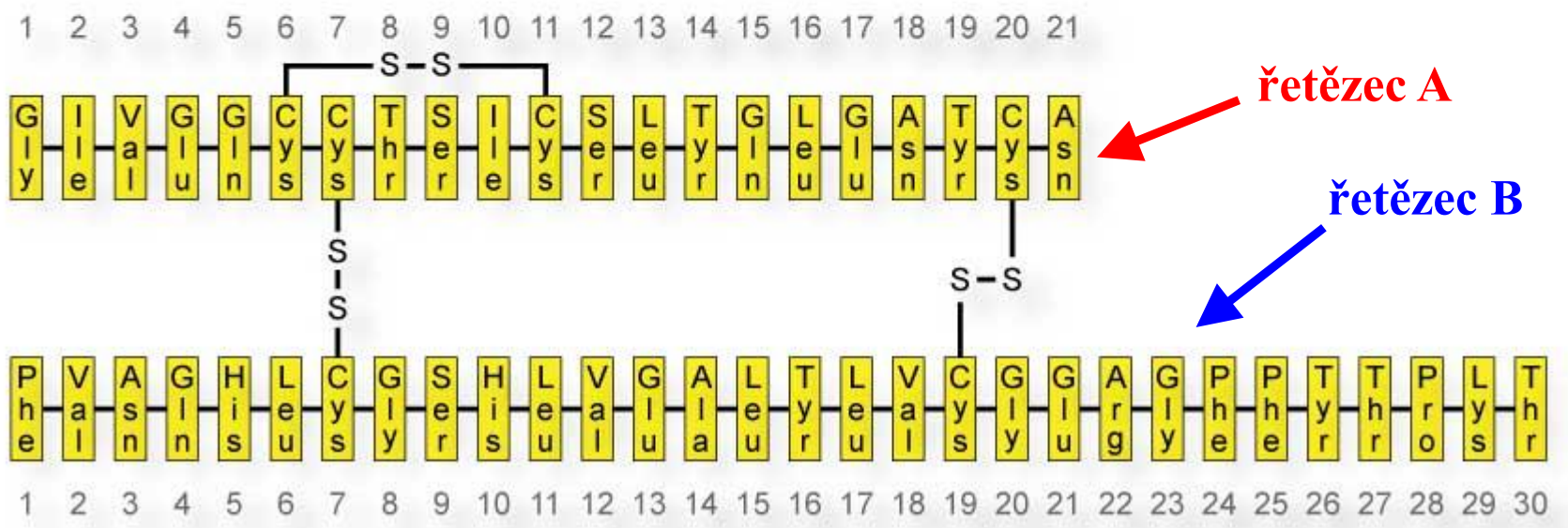
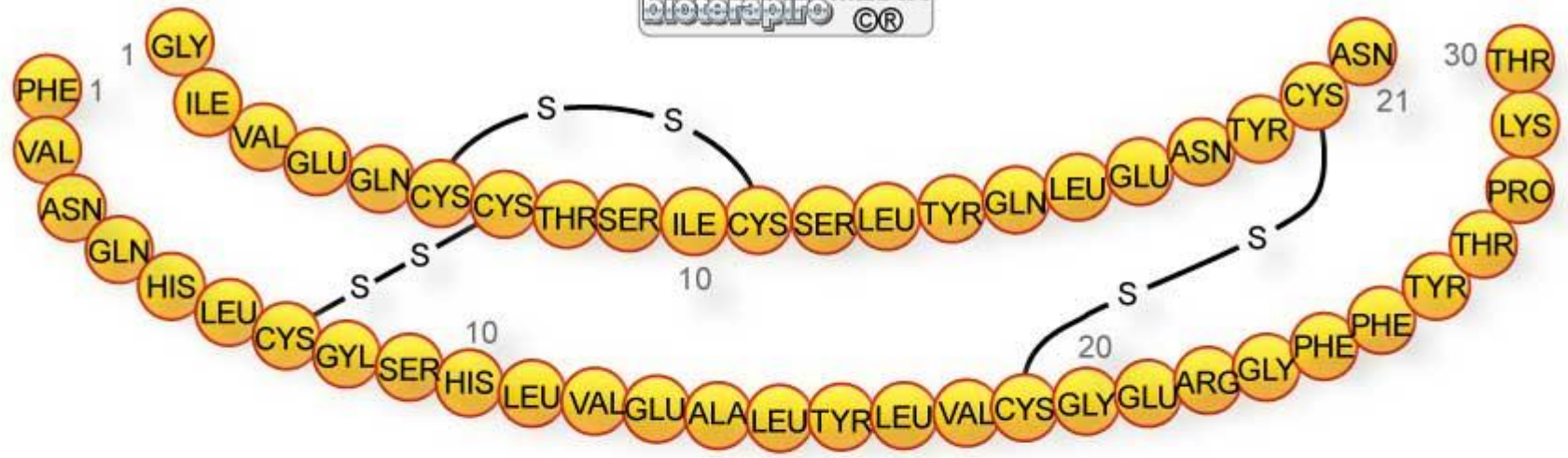
Figure 9-36 Catecholamine secretion by the adrenal medulla is regulated by neurons. Sympathetic nerve axons originating in the spinal cord pass through the sympathetic ganglia without forming synapses, but then synapse on the catecholamine-producing cells. Acetylcholine liberated from these preganglionic nerve terminals stimulates the secretion of medullary hormones.

Reakce organismu na stres: průběh poplachové reakce

Červená čísla představují pořadí v jakém jsou jednotlivé dráhy spouštěny



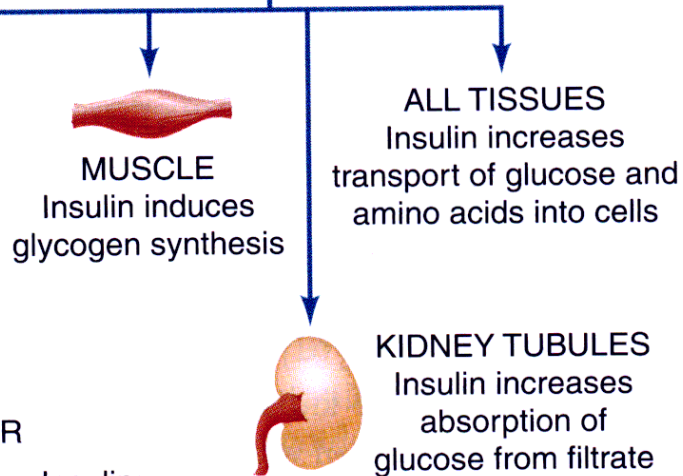
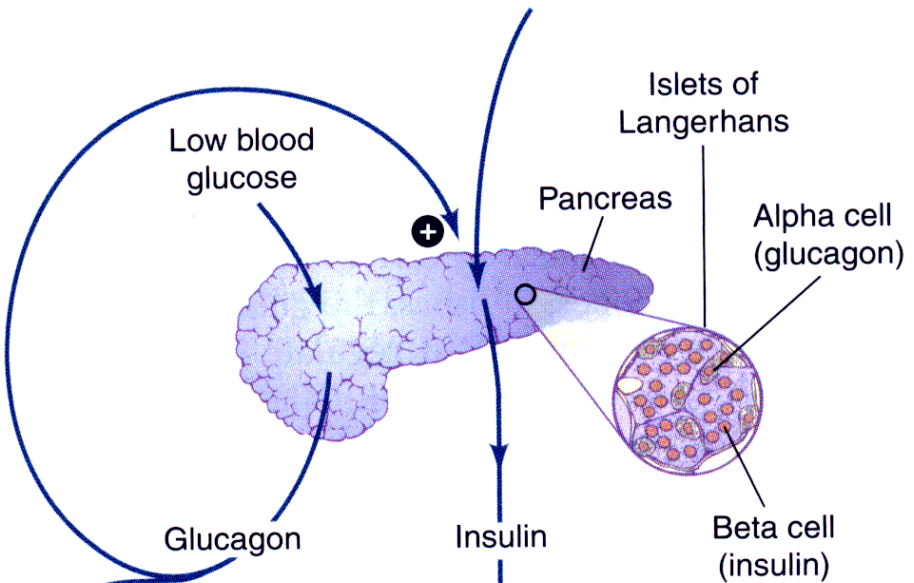
Struktura inzulínu



řetězec A

řetězec B

High blood glucose;
gastrointestinal
hormone secretion (GIP)



Pankreatické hormóny – inzulín a glukagón

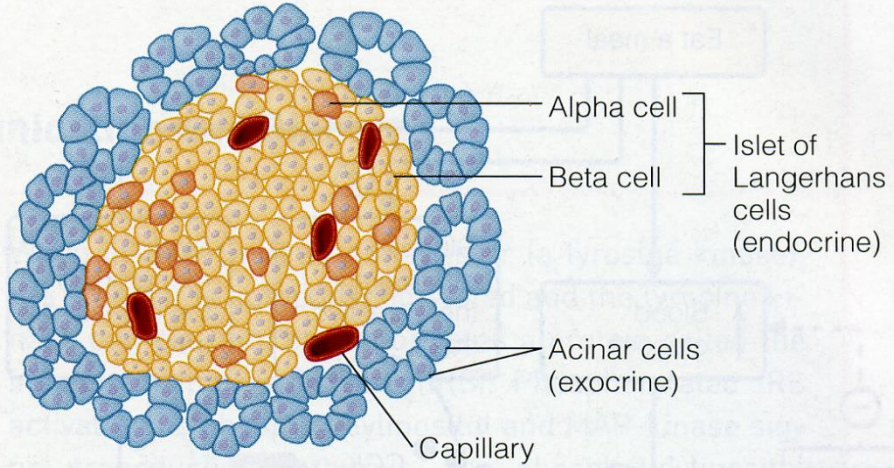


Figure 3.32 The mammalian pancreas The pancreas consists of both exocrine and endocrine tissues. The islets of Langerhans contain cells called beta cells that secrete the hormone insulin and cells called alpha cells that secrete the hormone glucagon.

Cholesterol – prekursor steroidních hormonů

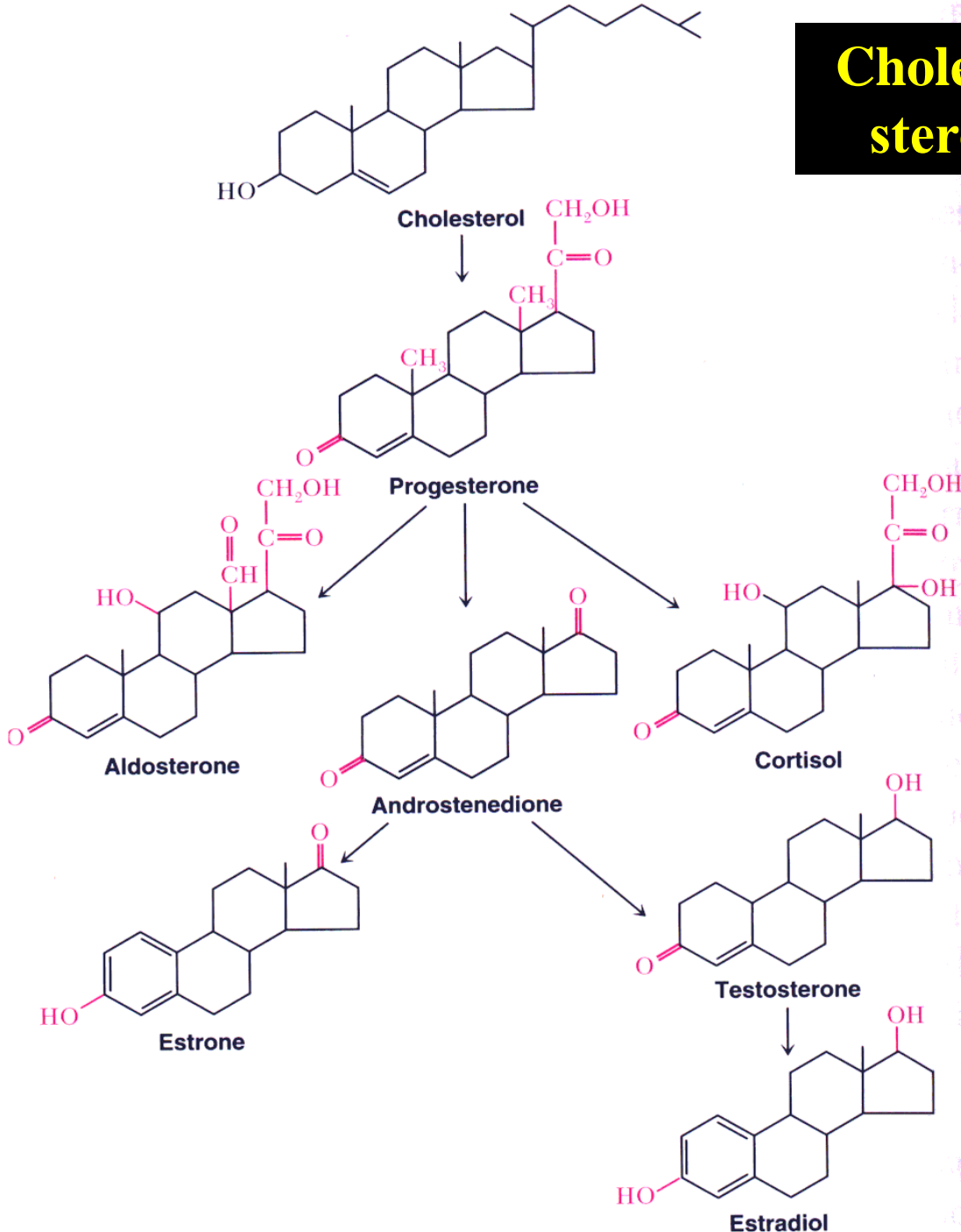
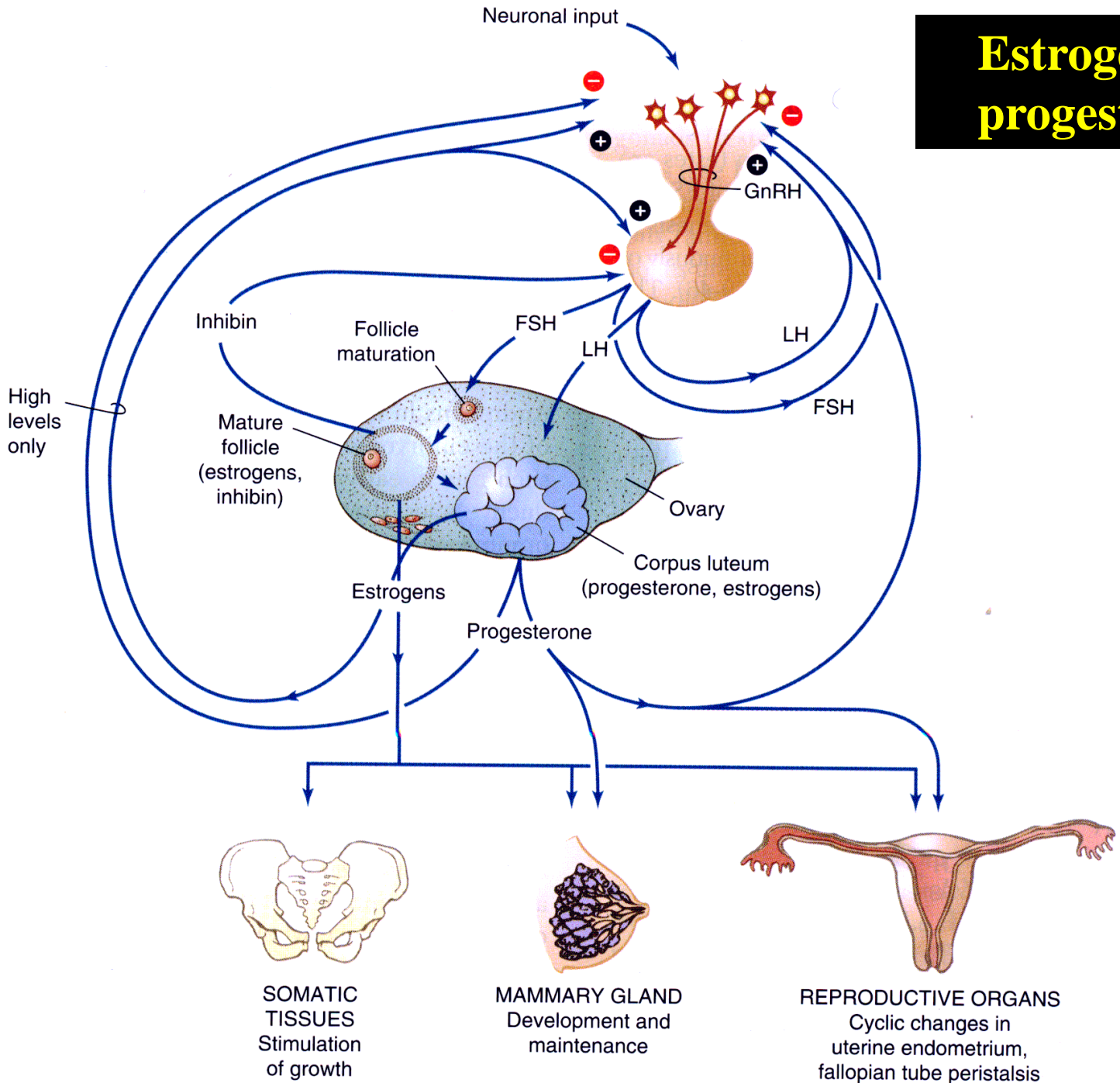


Table 9-9 Important mammalian reproductive hormones

Hormone	Tissue of origin	Structure	Target tissue	Primary action	Regulation
Primary sex hormones					
Estradiol-17 β (estrogens)	Ovarian follicle, corpus luteum, adrenal cortex	Steroid	Most tissues	Promotes development and maintenance of female characteristics and behavior; oocyte maturation, and uterine proliferation	Increased FSH and LH levels stimulate secretion
Progesterone	Corpus luteum, adrenal cortex	Steroid	Uterus, mammary glands	Maintains uterine secretion; stimulates mammary duct formation	Increased LH and prolactin levels stimulate secretion
Testosterone (androgens)	Testes (Leydig cells), adrenal cortex	Steroid	Most tissues	Promotes development and maintenance of male characteristics and behavior and spermatogenesis	Increased LH level stimulates secretion
Other Hormones					
Oxytocin	Posterior pituitary	Nonapeptide	Uterus, mammary glands	Promotes smooth muscle contraction and milk ejection	Cervical distention and suckling stimulate release; high progesterone inhibits release
Prolactin (PL)	Anterior pituitary	Peptide	Mammary glands (alveolar cells)	Increases synthesis of milk proteins and growth of mammary glands; elicits maternal behavior	Continuous secretion of PL-inhibiting hormone (PIH) normally blocks release; increased estrogen and decreased PIH secretion permit release

Estrogeny a progesteron



Testosterone

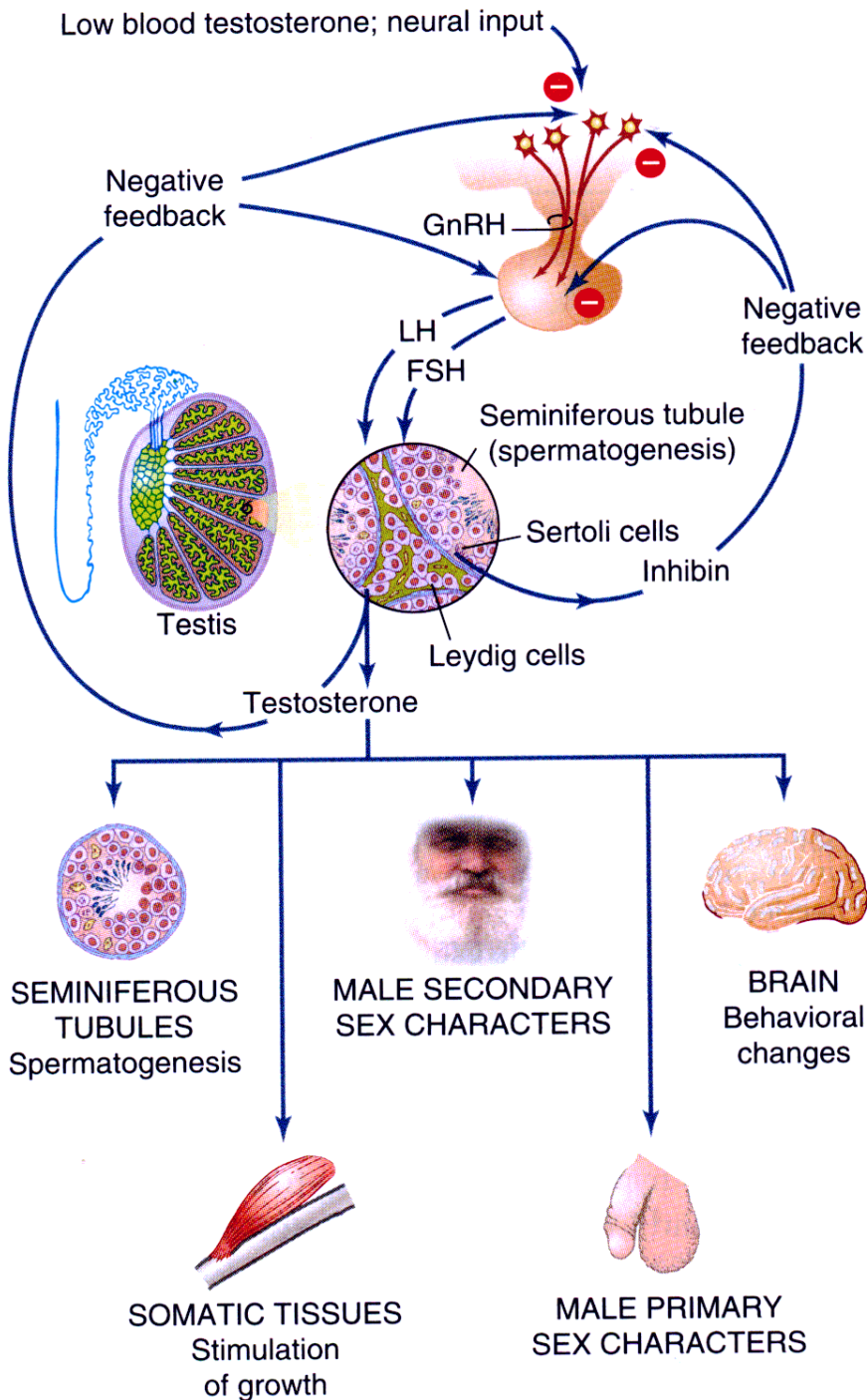


Figure 9-46 Testosterone, the primary sex hormone in males, has numerous actions and is regulated by neuronal stimuli and feedback control. A decrease in blood levels of testosterone stimulates the secretion of gonadotropin-releasing hormone (GnRH), which promotes the release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Some of the actions of testosterone are indicated at the bottom of the figure. High testosterone levels and inhibin, also secreted by the testes, inhibit FSH secretion both directly and indirectly.

Schematické znázornění hormonálního řízení menstruačního cyklu

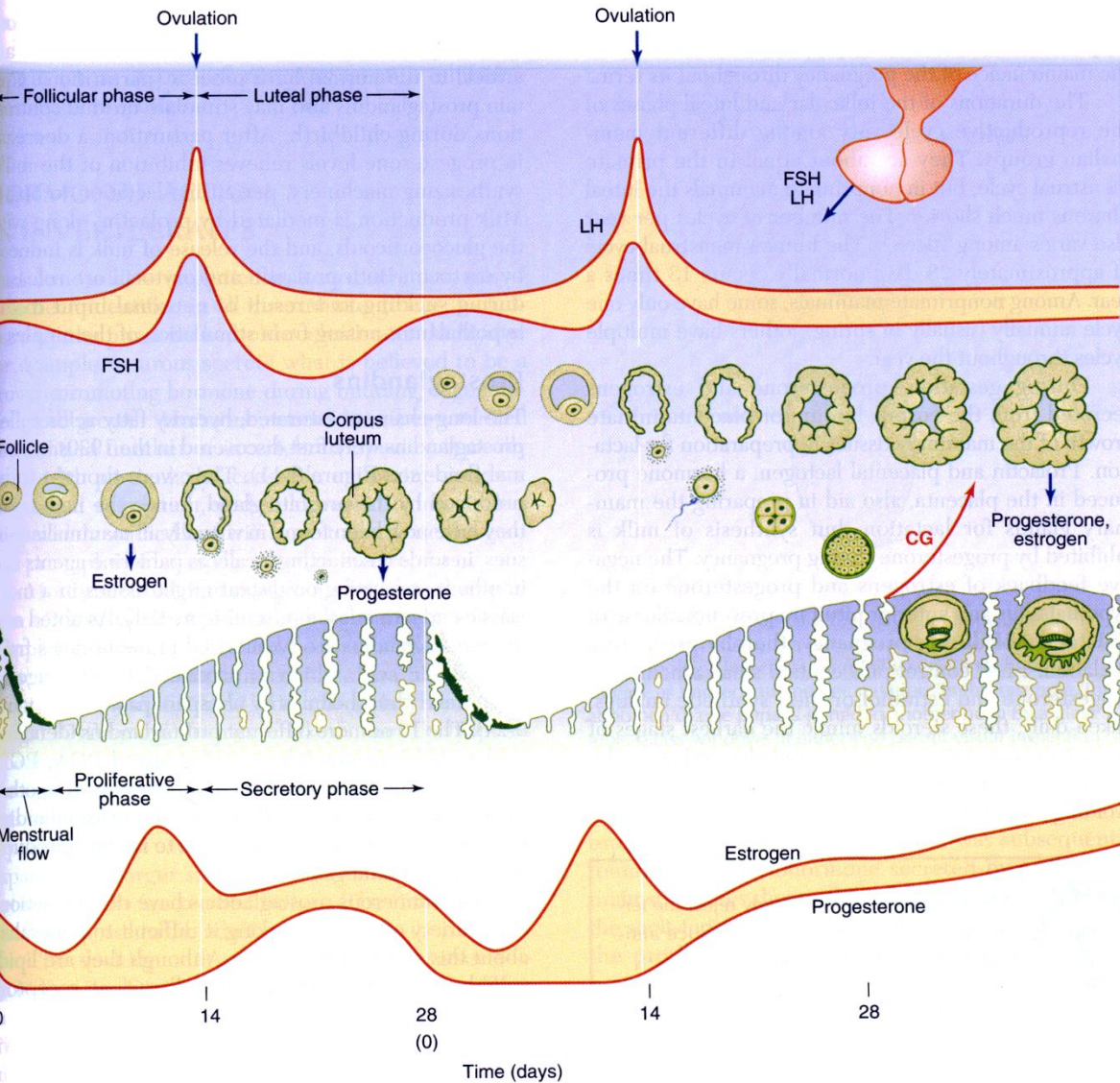


Figure 9-48 The primate menstrual cycle is regulated by periodic changes in the levels of the gonadotropins, estrogens, and progesterone. Before ovulation, follicle-stimulating hormone (FSH) promotes maturation of ovarian follicles, which secrete estrogen. High estrogen levels cause a surge of luteinizing hormone (LH), which triggers ovulation from one follicle. LH promotes development of the corpus luteum and induces it to secrete progesterone and some estrogen. In the absence of implantation (left), the progesterone and estrogen levels peak and then fall, initiating menstruation. The

subsequent decrease in estrogen, progesterone, and inhibin levels allows pituitary secretion of FSH and LH to increase again, thus initiating a new cycle. If implantation and pregnancy occur (right), secretion of chorionic gonadotropin (CG) by the placenta "rescues" the corpus luteum, which maintains secretion of estrogen and progesterone for the first two to three months of pregnancy in humans. Thereafter, the placenta itself secretes estrogens and progesterone. [Adapted from McNaught and Callander, 1975.]

Řízení hladiny krevní glukózy

hypoglykemizující hormon

- inzulín

hyperglykemizující hormony

- katecholaminy – adrenalin a noradrenalin
- glukagon
- glukokortikoidy – kortizol a kortikosteron
- růstový hormon – somatotropin

Řízení syntézy bílkovin a růstu

- **růstový hormon**
- **thyreoidní hormony T3 a T4**
- **pohlavní hormony**
- **inzulín**
- **aldosteron**

Řízení produkce tepla

- **nordrenalin – hlavní hormon netřesové termogeneze**
- **adrenalin**
- **tyroxin**
- **skupina hormonů se slabším účinkem: ACTH, kortizon, inzulín, STH, glukagon**

Řízení výměny iontů a vody

- **antidiuretický hormon – vasopresin**
- **aldosteron**
- **parathyroidní hormon**
- **kalcitonin**

Hormony regulující výměnu vody a iontů v těle

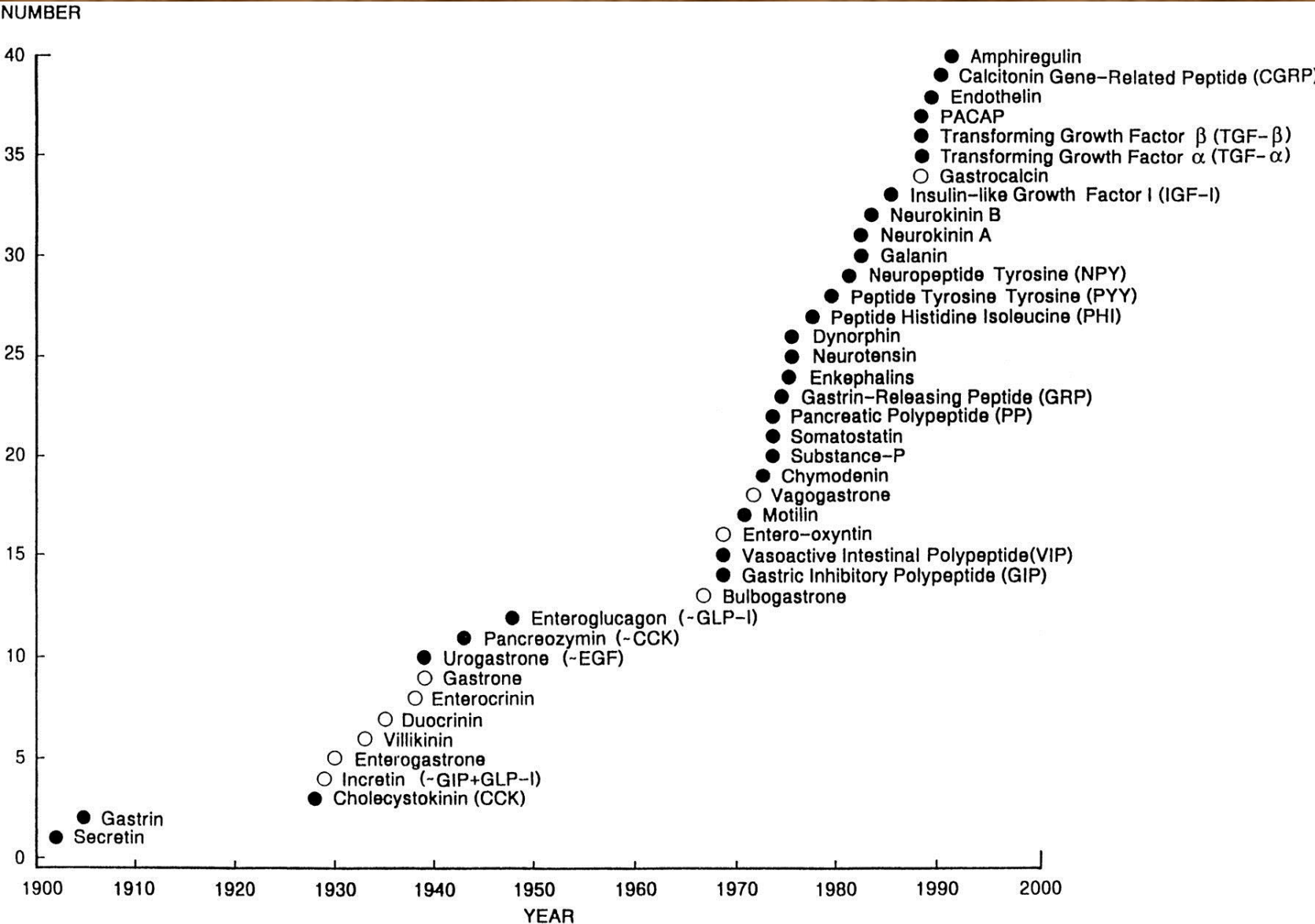
Table 9-8 Mammalian hormones involved in regulating water and electrolyte balance

Hormone	Tissue of origin	Structure	Target tissue	Primary action	Regulation
Antidiuretic hormone (ADH, vasopressin)	Posterior pituitary	Nonapeptide	Kidneys	Increases water reabsorption	Increased plasma osmotic pressure or decreased blood volume stimulates release
Atrial natriuretic peptide (ANP)	Heart (atrium)	Peptide	Kidneys	Reduces Na ⁺ and water reabsorption	Increased venous pressure stimulates release
Calcitonin	Thyroid (parafollicular cells)	Peptide	Bones, kidneys	Decreases release of Ca ²⁺ from bone; increases renal Ca ²⁺ and PO ₄ ³⁻ excretion	Increased plasma Ca ²⁺ stimulates secretion
Mineralocorticoids (e.g., aldosterone)	Adrenal cortex	Steroid	Distal kidney tubules	Promotes reabsorption of Na ⁺ from urinary filtrate	Angiotensin II stimulates secretion
Parathyroid hormone (PTH)	Parathyroid gland	Peptide	Bones, kidneys, intestine	Increases release of Ca ²⁺ from bone; with calcitriol increases intestinal Ca ²⁺ absorption; decreases renal Ca ²⁺ excretion	Decreased plasma Ca ²⁺ stimulates secretion

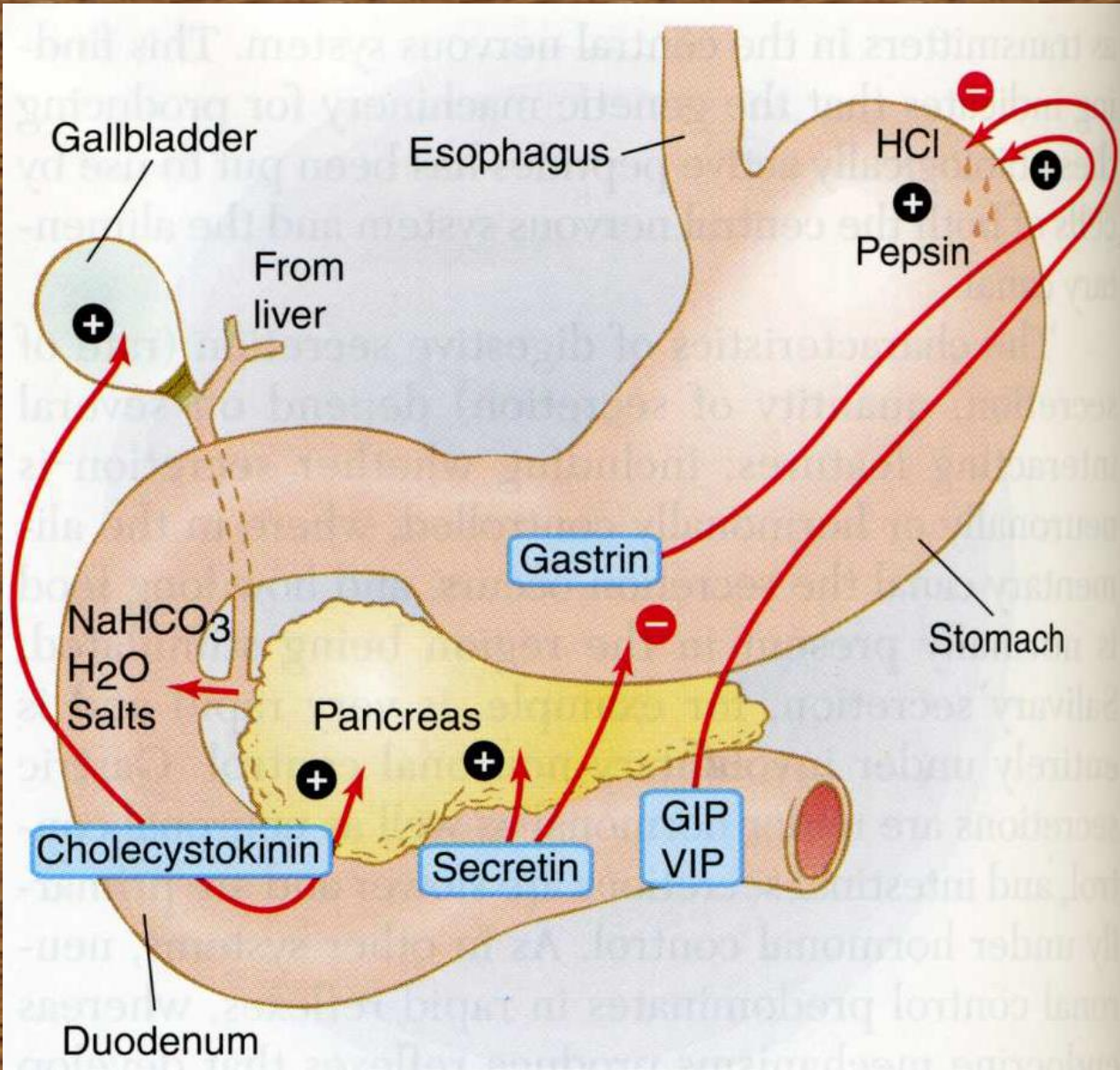
Tkáňové hormony - gastrointestinální peptidy

- **Gastrin**
- **Bombesin**
- **Somatostatin**
- **Pankreatický polypeptid (PP).**
- **Vasoaktivní intestinální polypeptid**
- **Cholecystokinin (cholecystokinin-pankreozymin = CCK-PZ)**
- **Chymodenin**
- **Enteroglukagon**
- **Gastric inhibitory peptide (GIP, enterogastron)**
- **Motilin**
- **Sekretin**
- **Villikinin**
- **Hepatokinin**
- **Neurotenzin**
- **Ghrelin - hormon hladu**

60 Objev a identifikace tkáňových gastrointestinálních hormonů



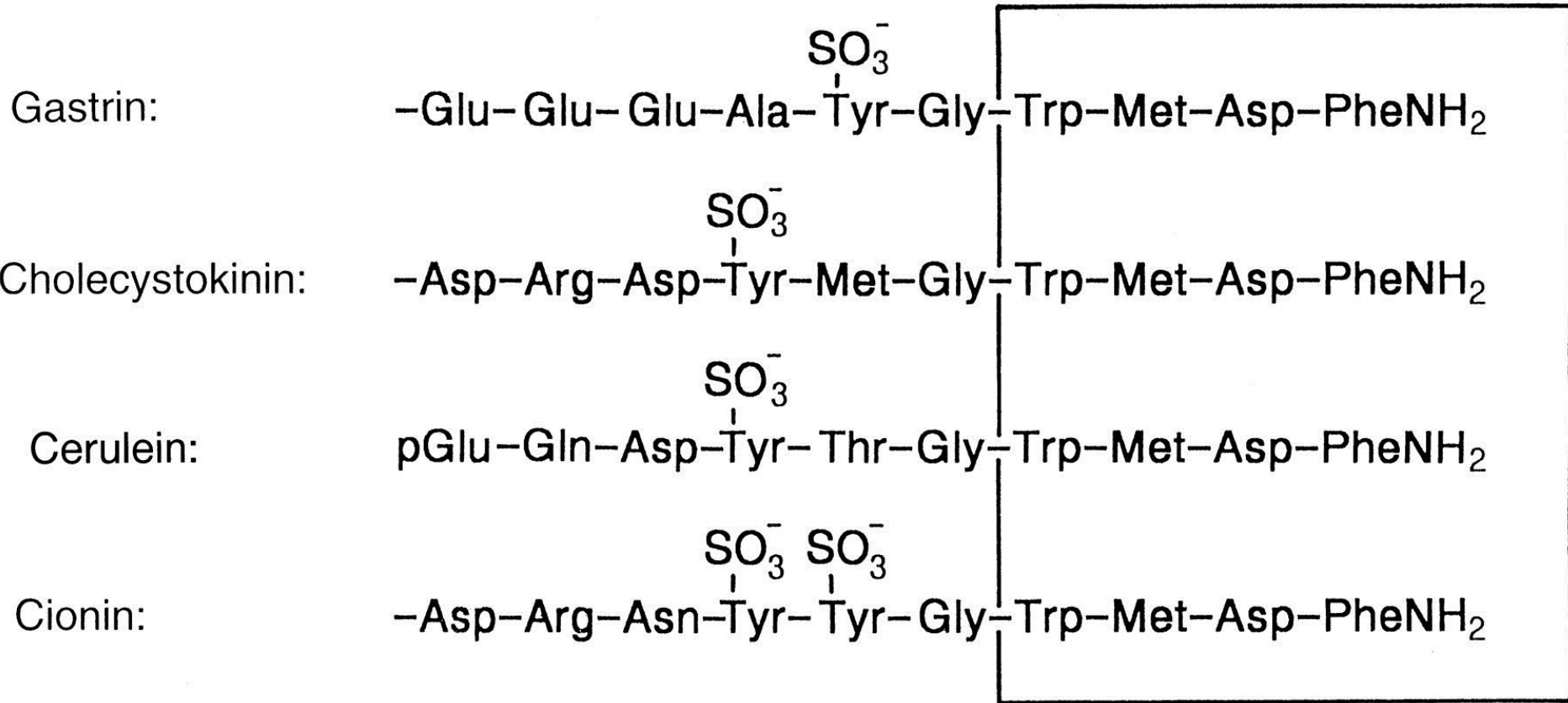
Účinek hlavních gastrointestinálních tkáňových hormonů



GIP – gastric inhibitory peptide

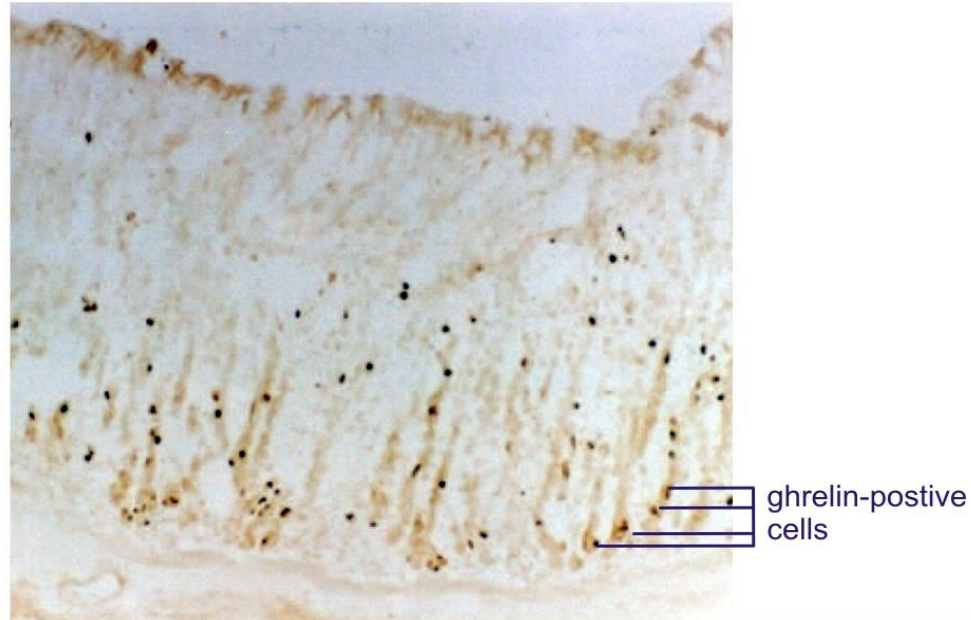
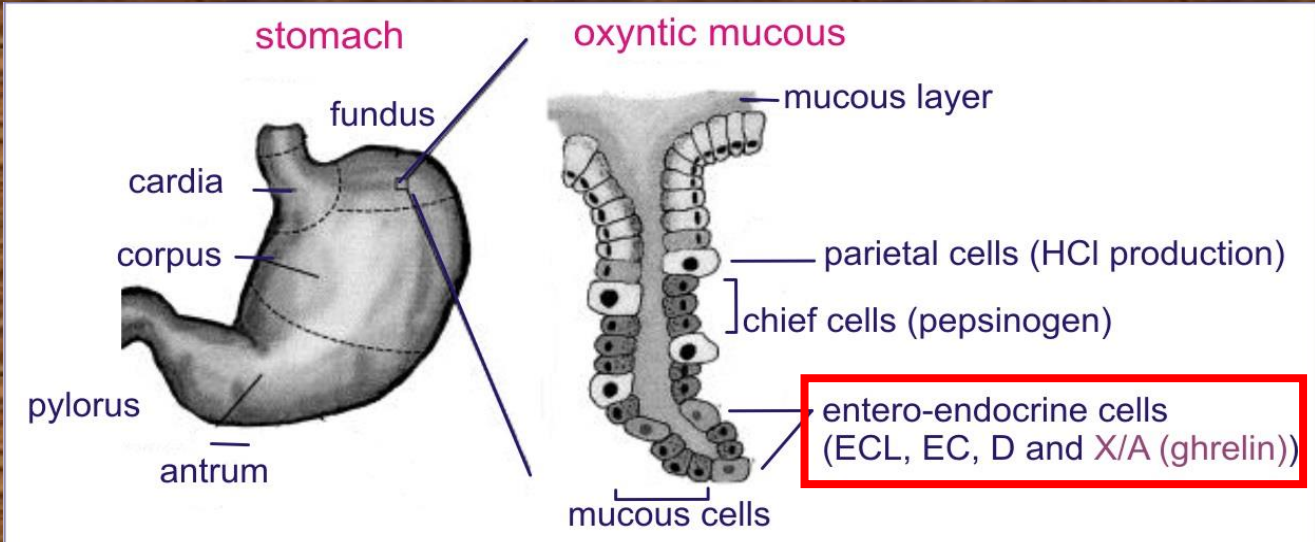
VIP – vasoactive intestinal peptide

Strukturní homologie členů gastrinové rodiny

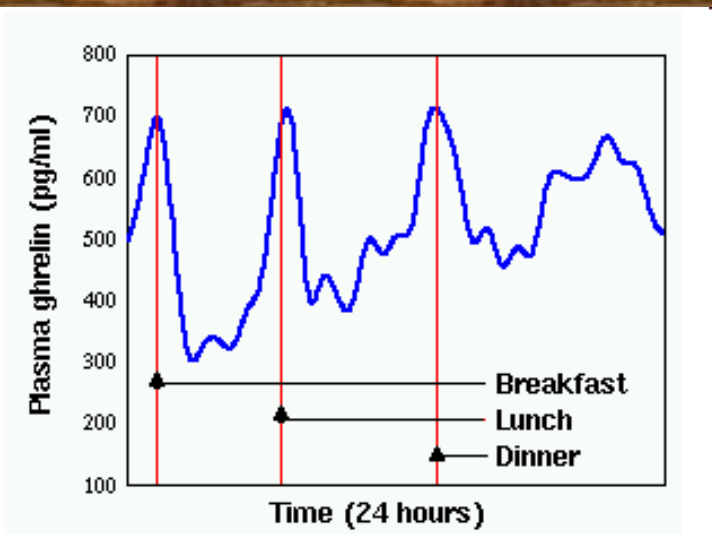
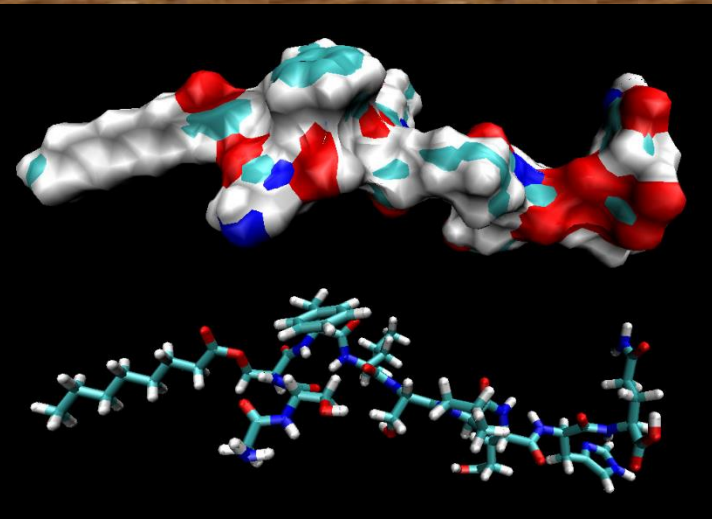


Ghrelin – hormon hladu

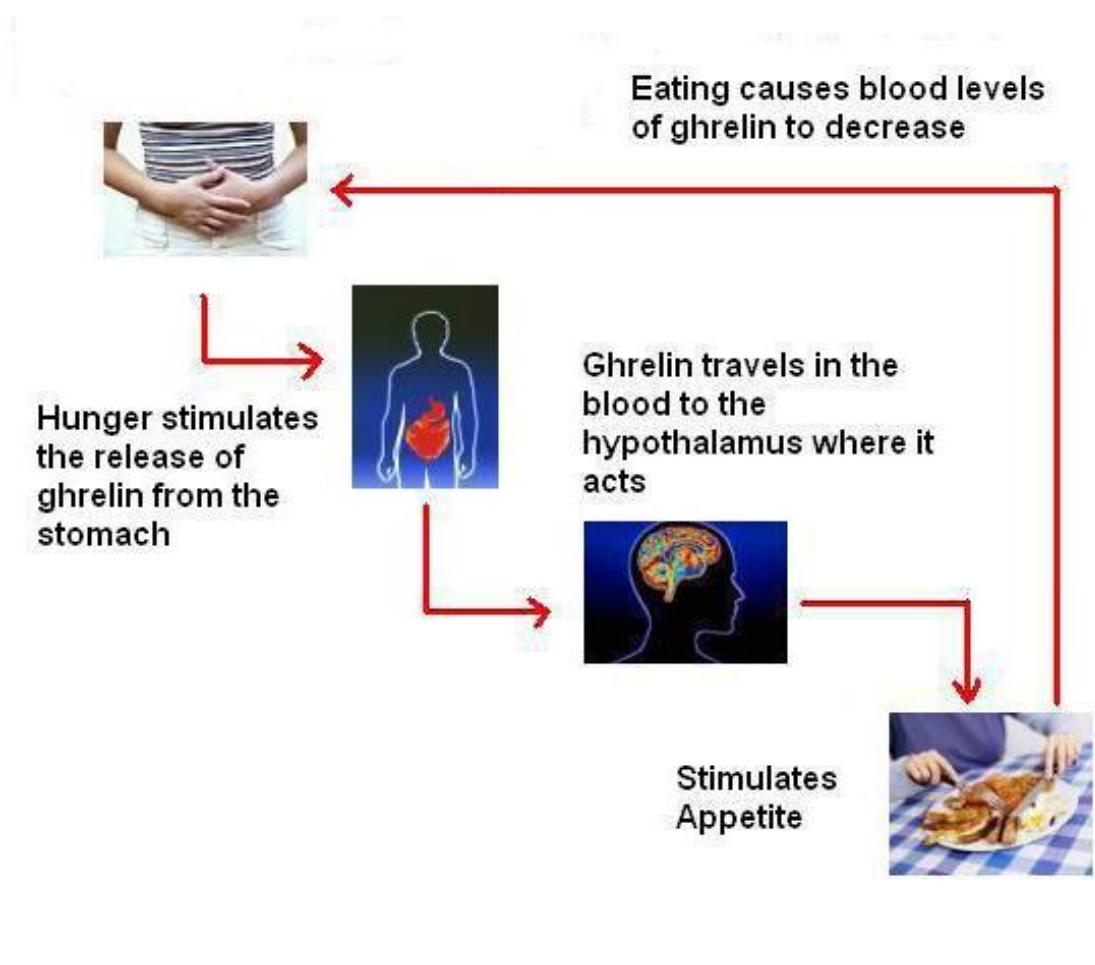
Ghrelin je produkován v žaludku (v malé míře i ve slinivce, mozku, ledvinách a placentě)



Ghrelin – stimuluje chuť k jídlu prostřednictvím centra hladu/sytosti v hypothalamu



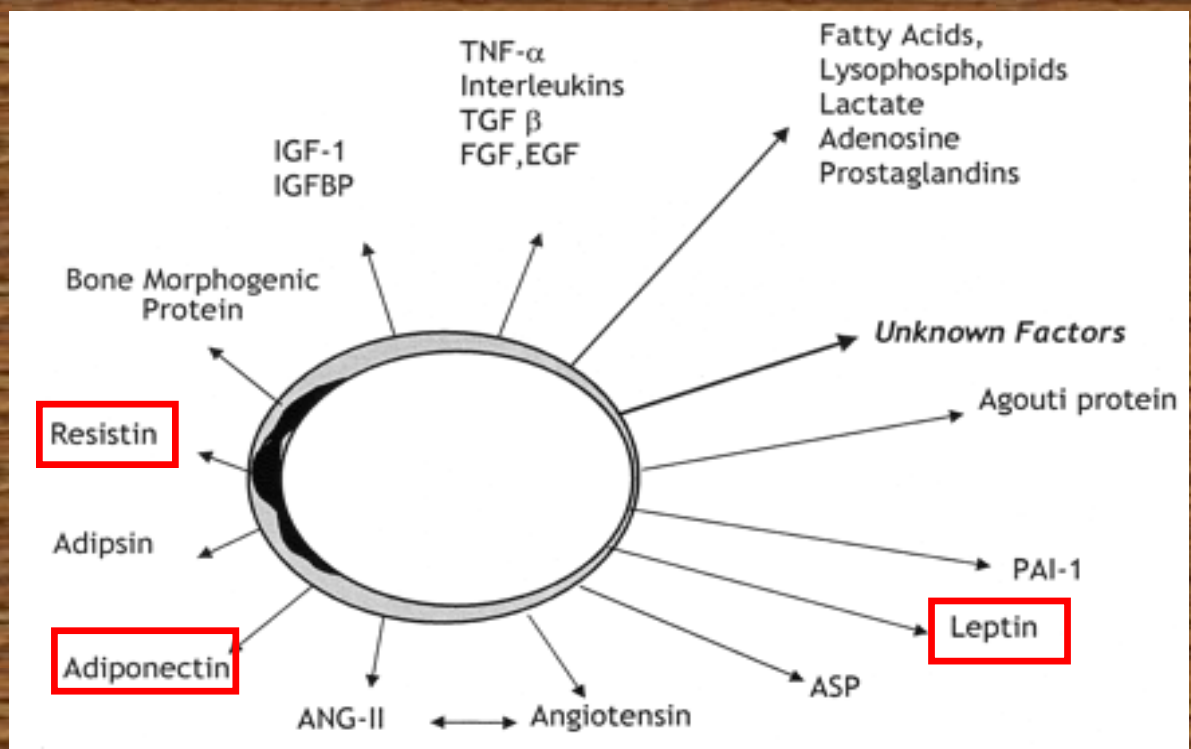
Adapted from Cummings et al. Diabetes 50:1714, 2001.



Hormony tukové tkáně

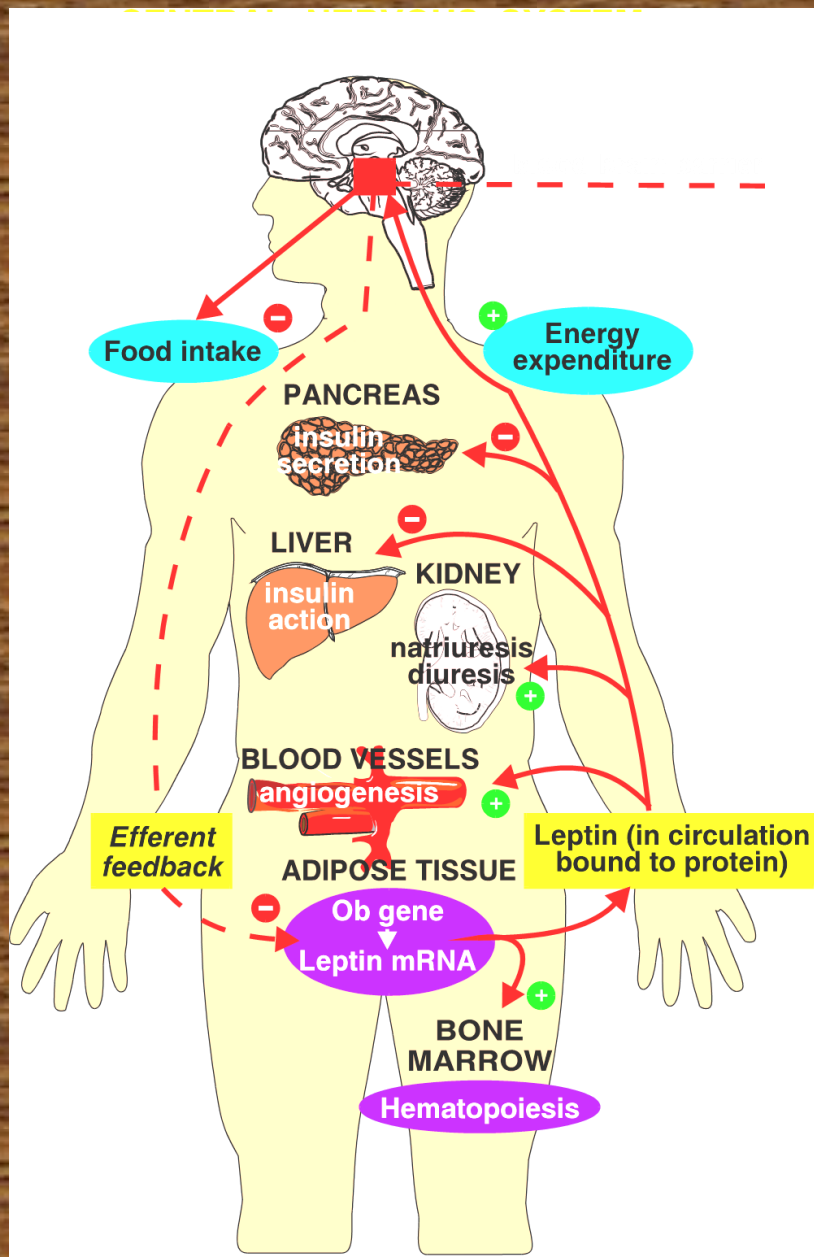
- **Leptin - hormon sytosti**
- **Adiponectin**
- **Resistin**

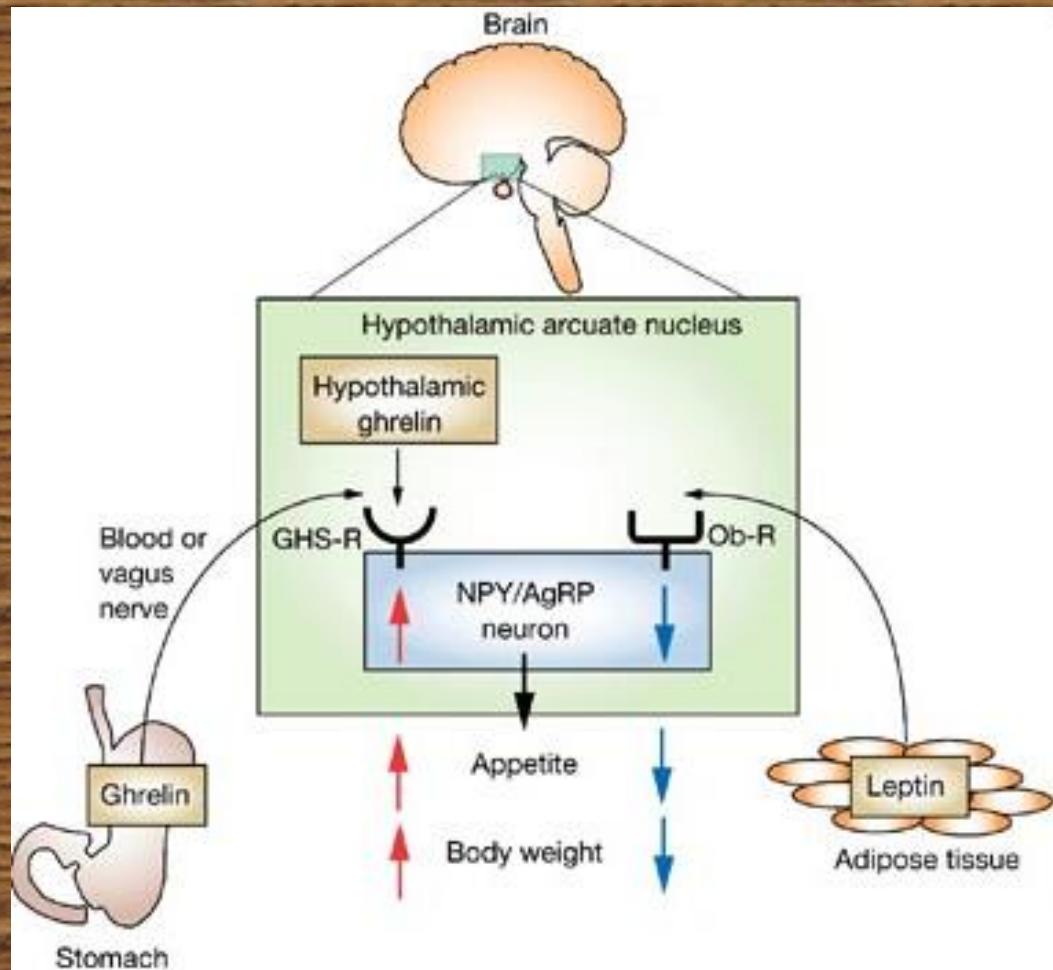
Tuková tkáň jako endokrinní orgán



Leptin – hormon sytosti

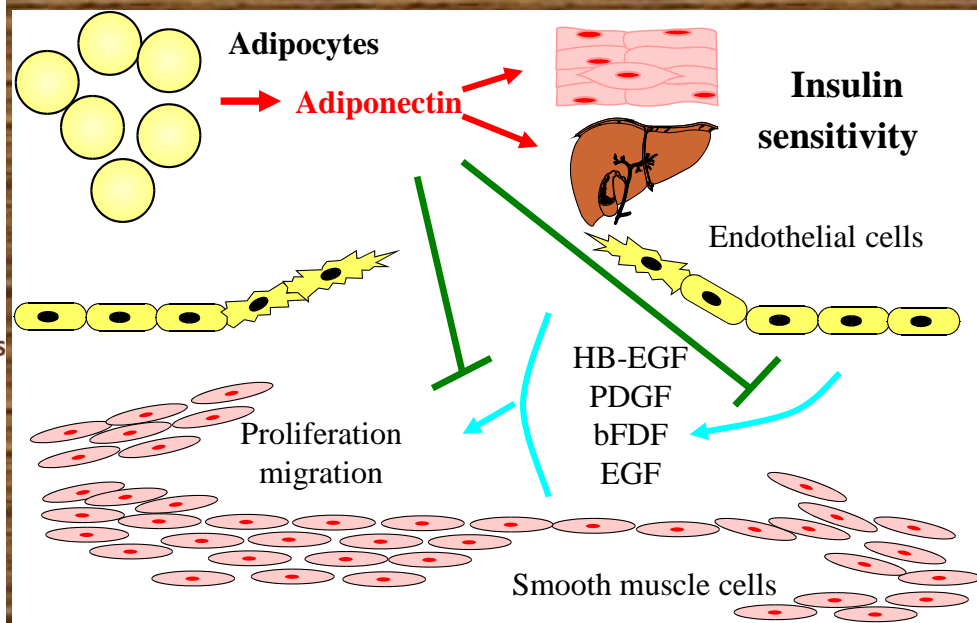
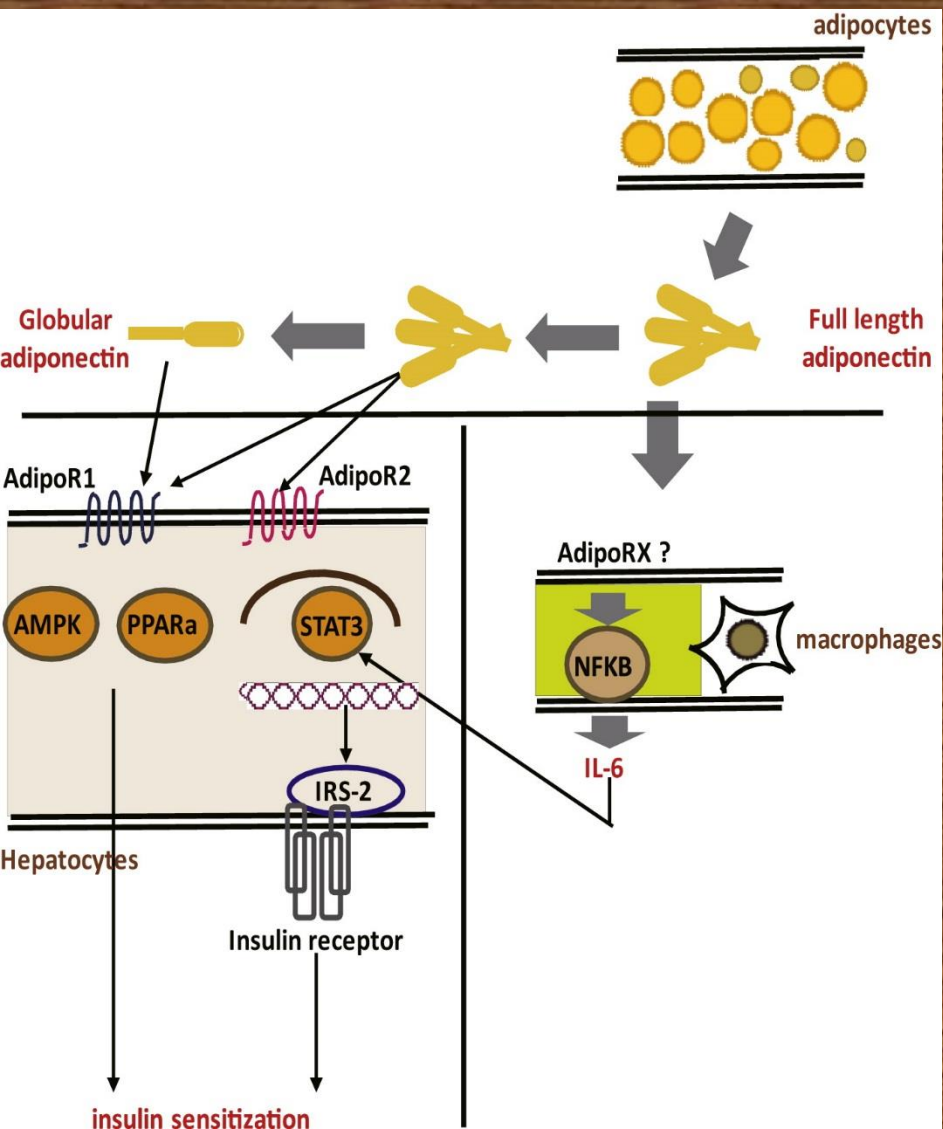
- Proteinový hormon produkovaný adipocyty
- Hladiny leptinu pozitivně korelují s obsahem tuku v organismu
- Regulátor příjmu potravy a energetického výdeje
- Jeho chybění vede k morbidní obezitě, nicméně u běžných obézních pacientů je jeho hladina vysoká
- Leptin není fylogeneticky primárně určen k tlumení příjmu potravy, ale k adaptaci organismu na dlouhodobé hladovění





Adiponectin

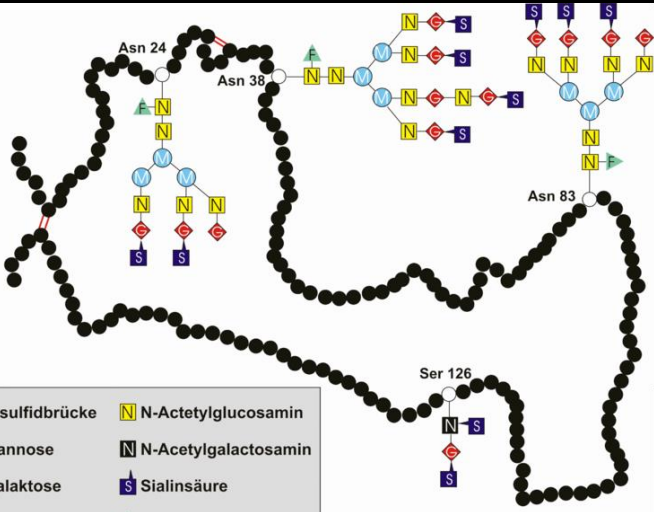
Adiponectin zvyšuje inzulínovou senzitivitu zvýšenou oxidací tuků, čímž snižuje hladinu triglyceridů v játrech a svalecth



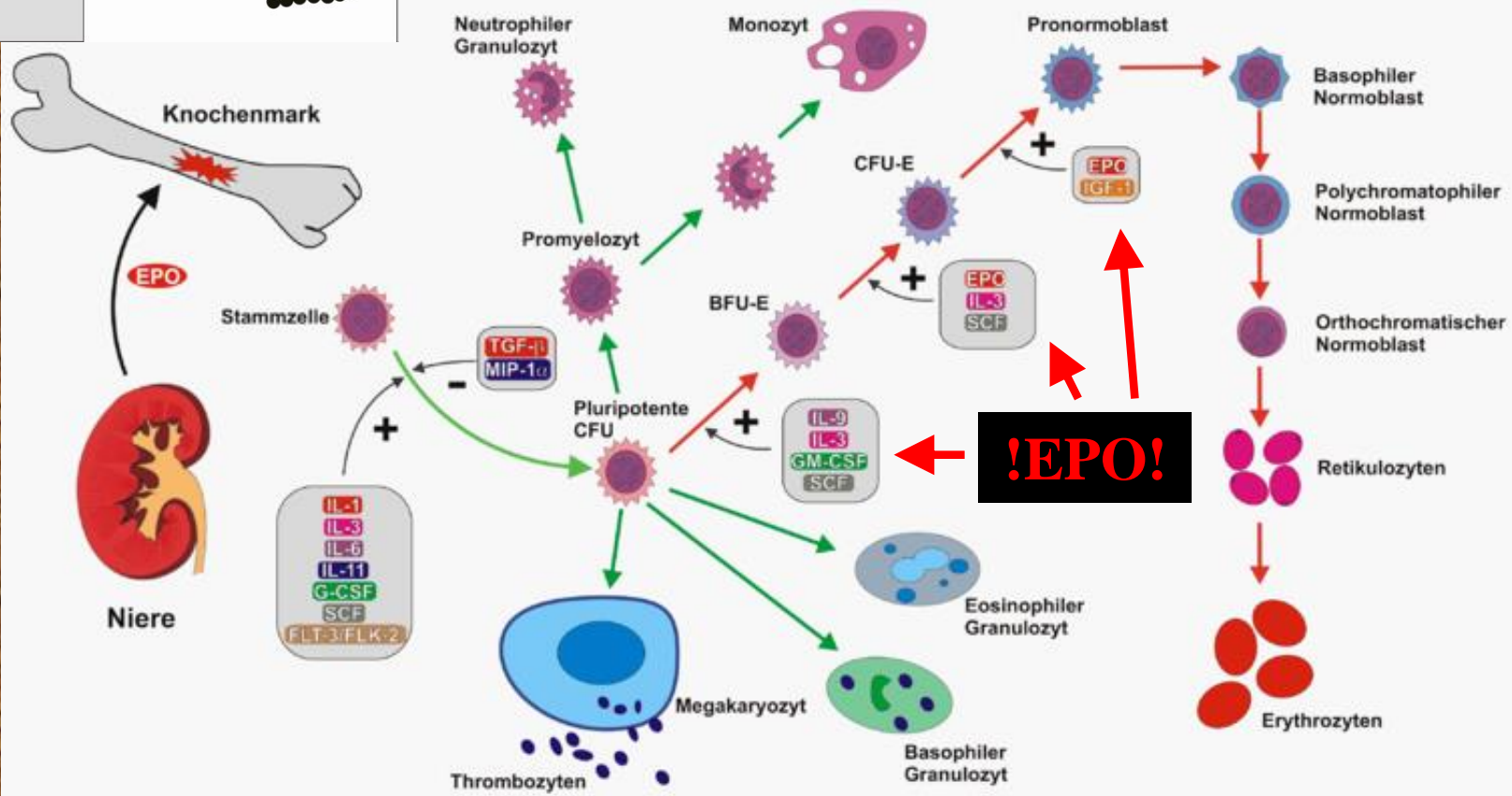
Tkáňové hormony ledvin

- **Renin**
- **Angiotenzin I a II**
- **Erythropoetin**
- **Calcitriol (hormon D)**

Erythropoetin



Erythropoetin (EPO) - hormon stimulující tvorbu erytroblastů



Tkáňové hormony jater

- **Kininy (kalidin a bradykinin)**
- **Erytropoetin**
- **Somatomediny**
- **Angiotenzin I**

Tkáňové hormony plic

- **Angiotenzin II**
- **Histamin**
- **Serotonin**
- **Prostaglandiny**

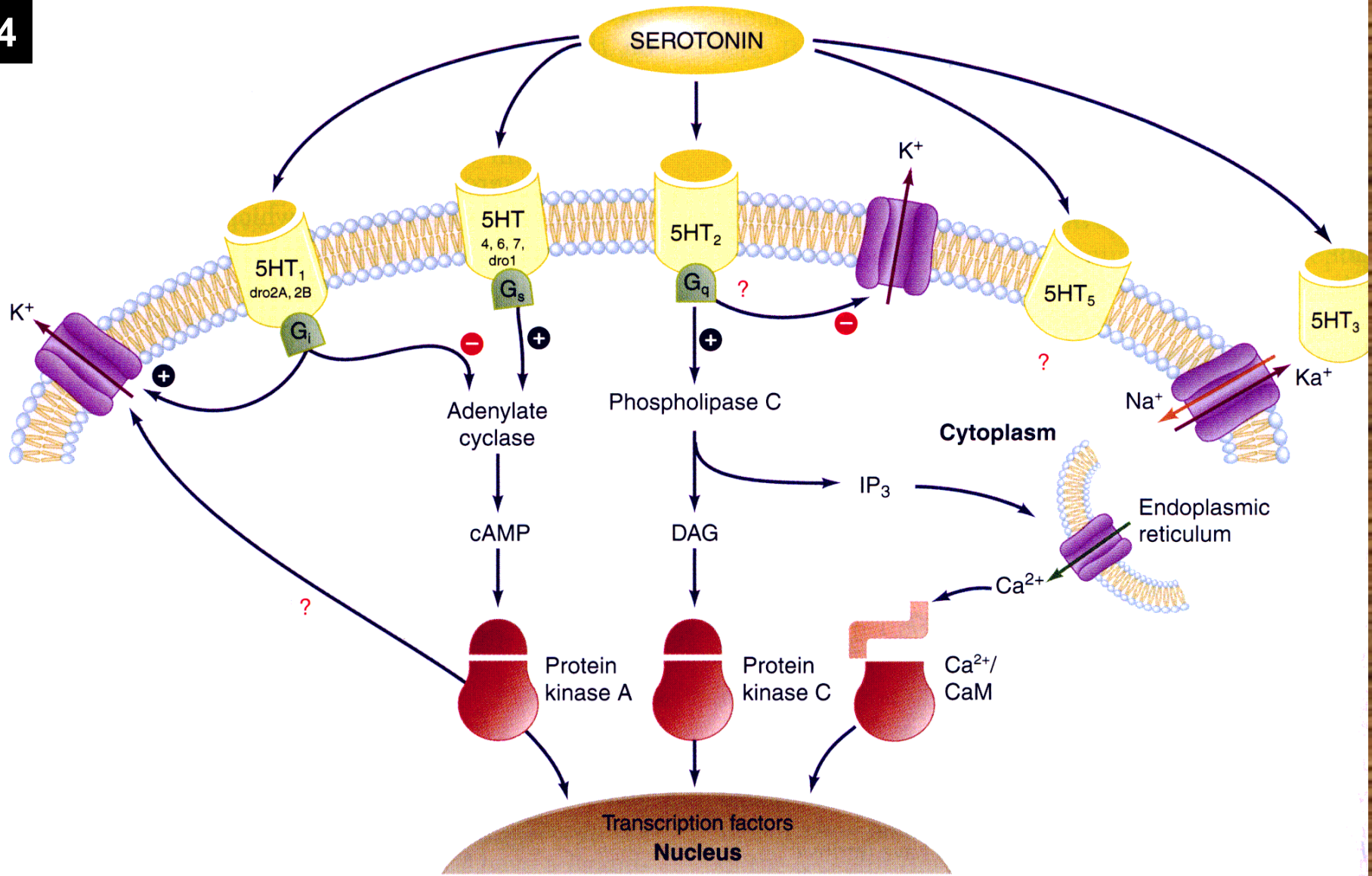


Figure 9-31 Serotonin binds to multiple receptors, which are linked to convergent and divergent second-messenger pathways. Binding of serotonin, also known as 5-hydroxytryptamine (5-HT), to some receptors leads to production of cAMP, diacylglycerol (DAG), or inositol trisphosphate (IP₃), all of which can mediate the same cellular responses in cells of different

tissues, or even in the same cells. The various receptors illustrated represent subclasses of the serotonin receptor family (dro = *Drosophila*). G_i = inhibitory G proteins; G_s = stimulatory G proteins; G_q = pertussis toxin-insensitive G proteins; Ca²⁺/CaM kinase = Ca²⁺/calmodulin-dependent protein kinase. [Adapted from Saudou and Hen, 1994.]

Hormony produkované krevními elementy

- **Histamin**
- **Serotonin**
- **Leukotrieny**
- **Kininy - kalidin a bradykinin**
- **Heparin**

Hormony produkované neurony

- Endorfiny
- Enkefaliny (Leu- a Met-enkefalin)

α -Endorphin: Tyr-Gly-Gly-Phe-Met-Thr-Ser-Glu-Lys-Ser-Gln-Thr-Pro-Leu-Val-Thr-OH

β -Endorphin: Tyr-Gly-Gly-Phe-Met-Thr-Ser-Glu-Lys-Ser-Gln-Thr-Pro-Leu-Val-Thr-Leu-Phe-Lys-Asn-Ala-Ile-Ile-Lys-Asn-Ala-Tyr-Lys-Lys-Gly-Glu-OH

γ -Endorphin: Tyr-Gly-Gly-Phe-Met-Thr-Ser-Glu-Lys-Ser-Gln-Thr-Pro-Leu-Val-Thr-Leu-OH

Mode of action

- Effects located in the Central Nervous System
- Specific receptors in the brain for different narcotics lead to different side effects

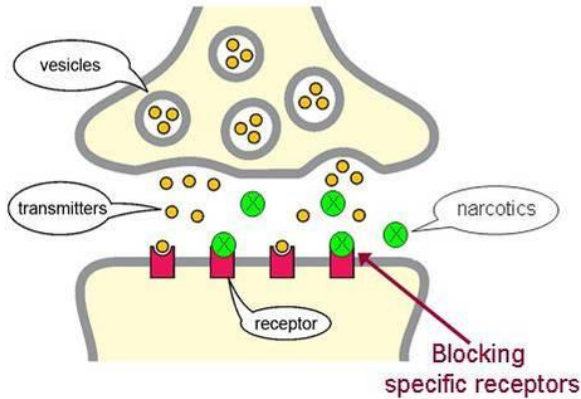


Action on:

μ -receptor (*Endorphins*)
 ⇨ Analgesia Euphoria

κ -receptor (*Dynorphines*)
 ⇨ Analgesia Sedation

δ -receptor (*Enkephalins*)
 ⇨ Analgesia Dysphoria

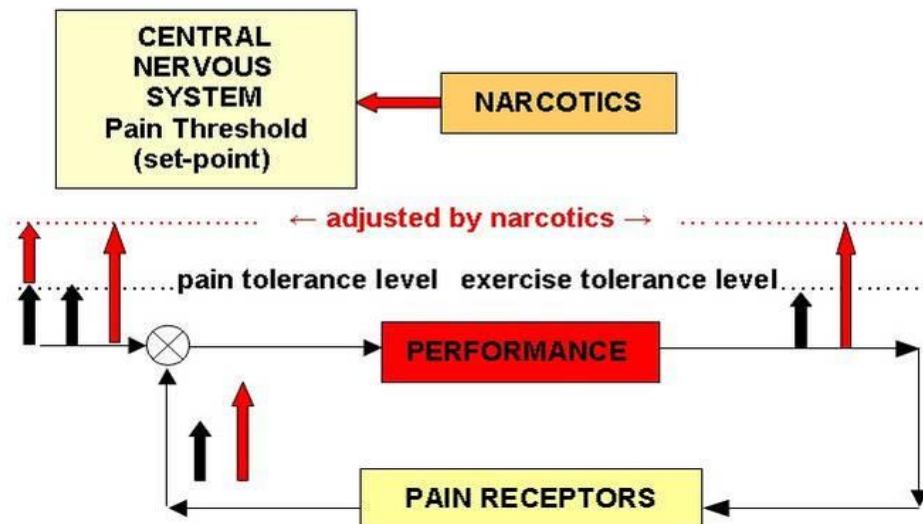


Müller-Esterl, Biochemie, 2004
 © Spektrum Akademischer Verlag, Heidelberg

Působení tkáňových hormonů mozku: obsazují specifické receptory a zvyšují tak prahovou hladinu bolesti a výkonnosti

Abuse of narcotics in sports

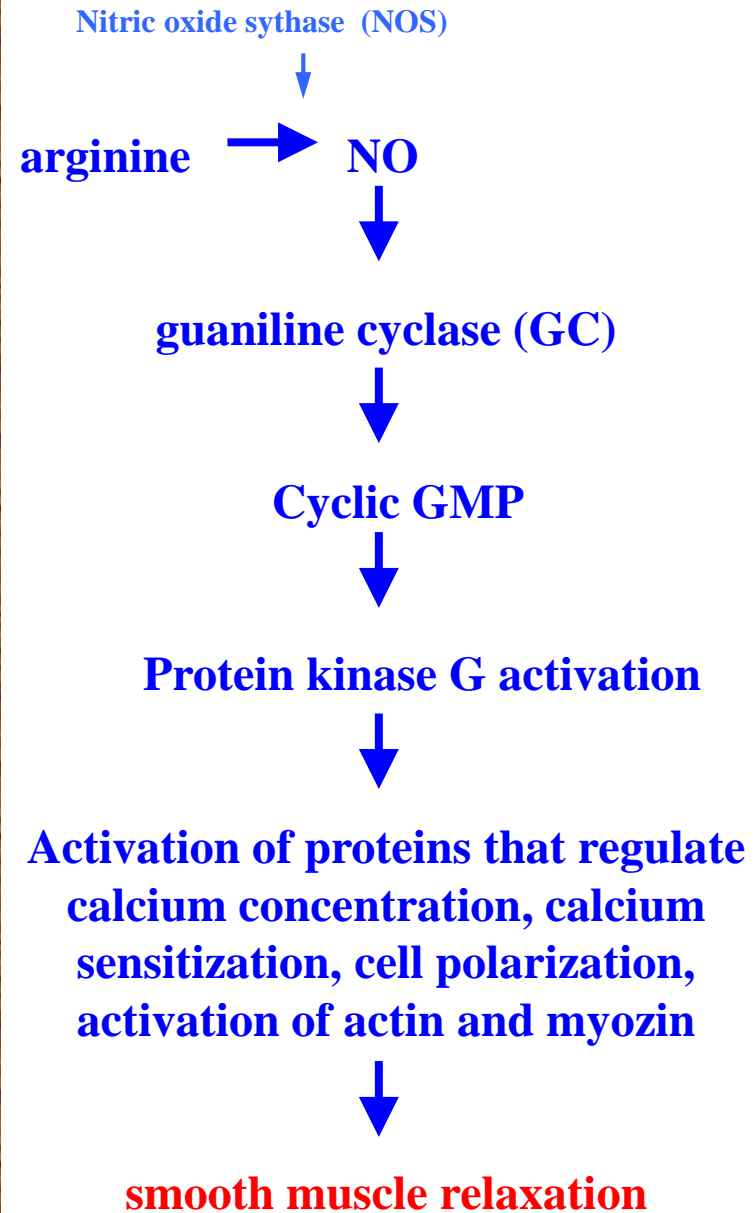
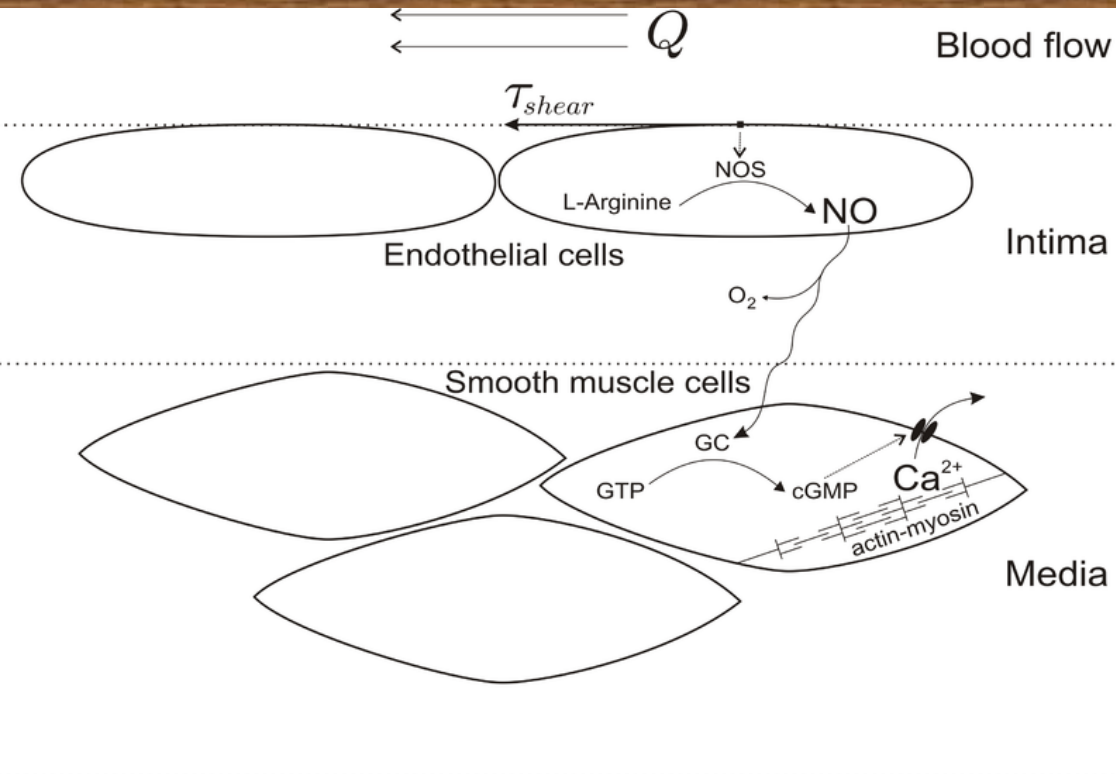
⇨ *Enhancing performance effects of analgesic narcotics by increased tolerance to pain*



Hormony srdce a cév

- **Atriový natriuretický faktor (ANF)**
- **NO - oxid dusnatý**

79 Fyziologická role oxidu dusnatého



Hodně štěstí u zkoušky...

