

Endokrinologie I

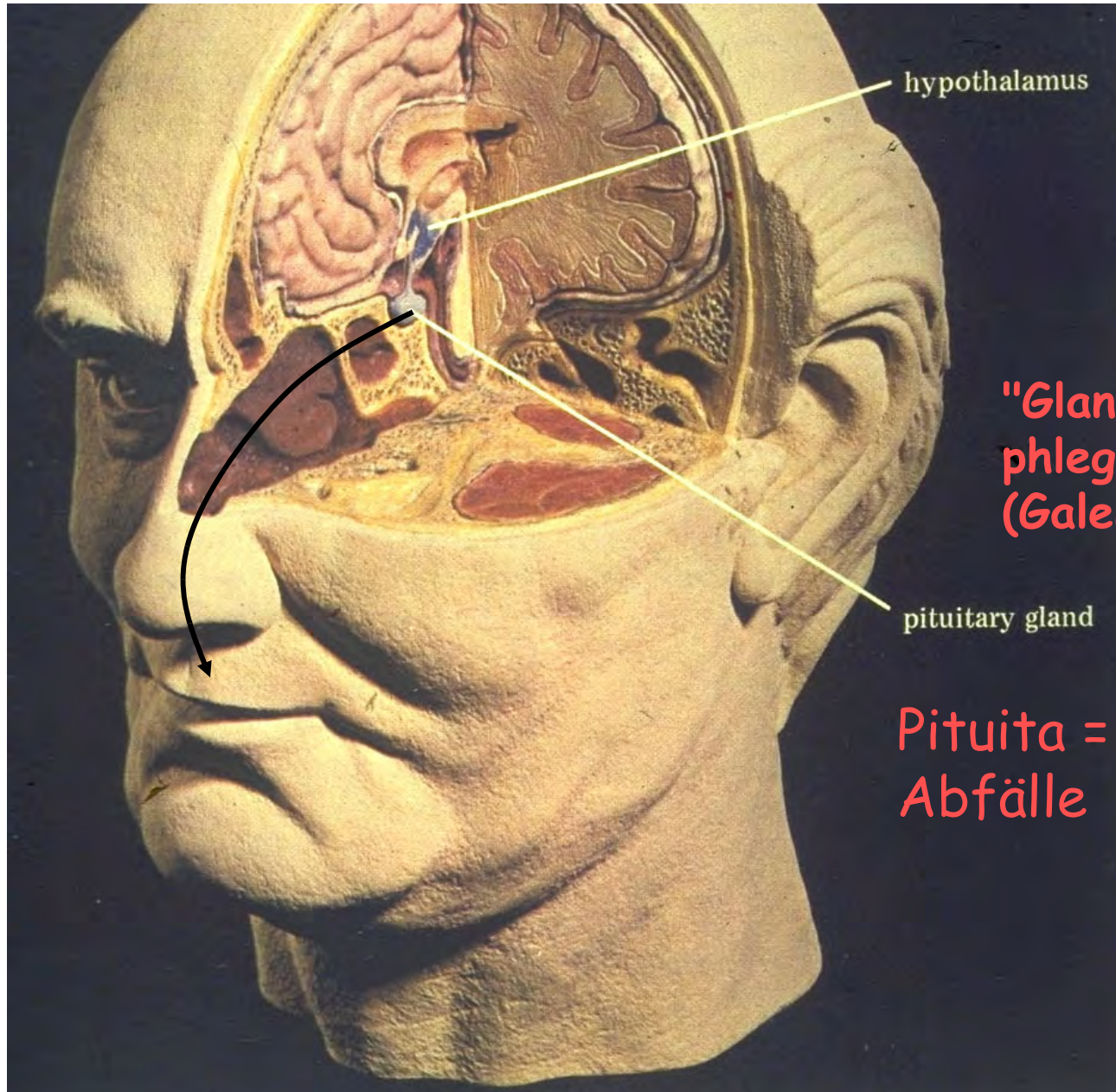
Endokrines System – Hormone: allgemein

- ***Definition***
 - *Begriffe*
 - *Abgrenzung*
- ***Hierarchie***
 - *Hypothalamus-Hypophysen-Achse*
 - *Endokrine Drüsen*
- ***Regelkreis***
 - *Negative Rückkopplung*
- ***Hormone***
 - *Chemische Struktur*
 - *Rezeptor/Signalkaskade*
 - *Wirkungseintritt*
 - *Wirkungsdauer*

Endokrines System – Hormone: speziell

- ***Hypophyse***
(Adenohyphyse)
- ***Schilddrüse***
- ***Nebennierenrinde***
- ***Gonaden***
- ***Nebenschilddrüse***
- *Wachstumshormon*
- *Glandotrope Hormone*
- *Schilddrüsenhormone*
(T₃/T₄)
- *Mineralocorticoide*
- *Glucocorticoide*
- *Sexualhormone*
- *Sexualhormone*
- *Parathormon*
- *(Calcitonin, Calcitriol)*

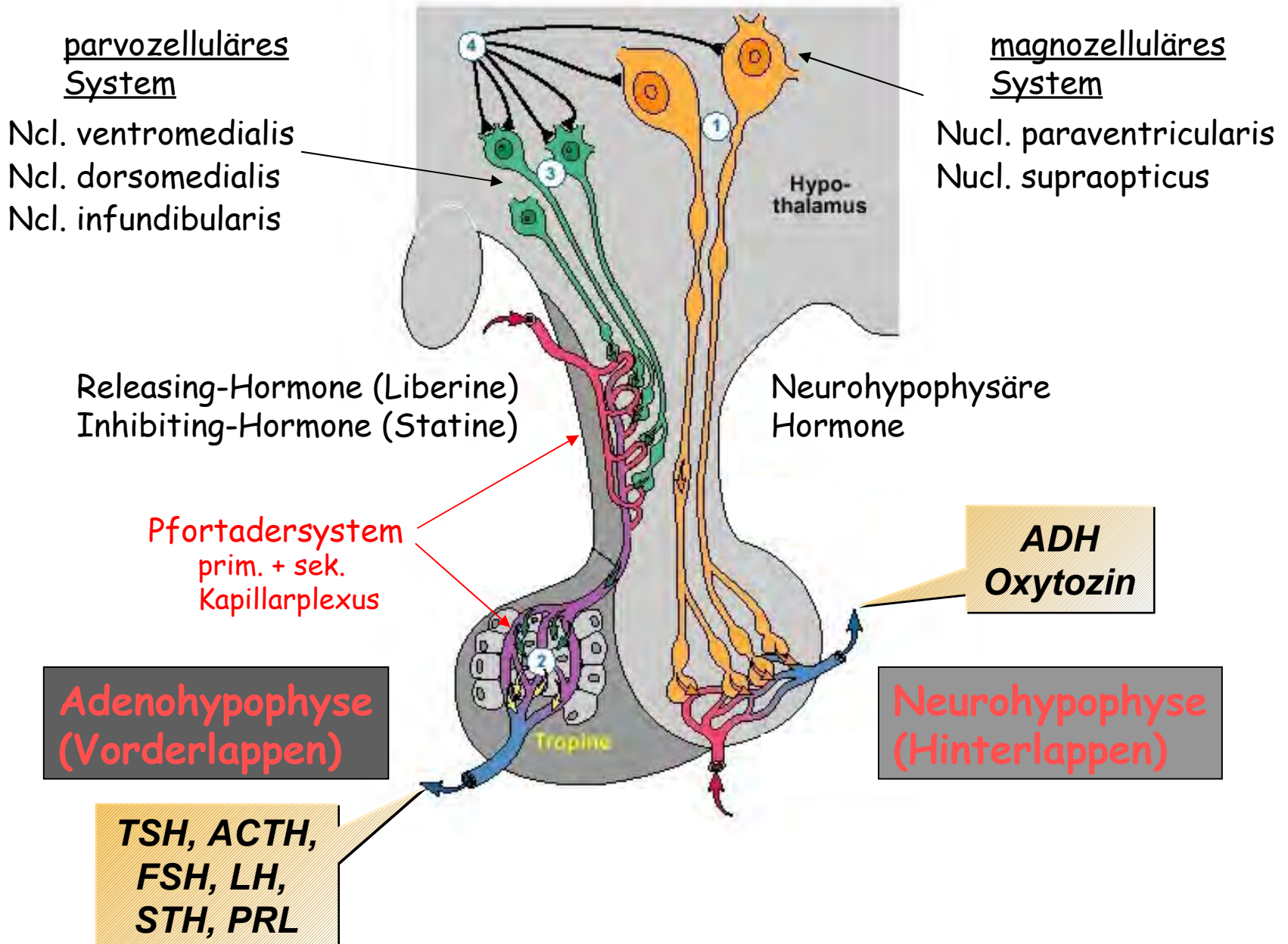
Hypothalamus-Hypophysen-Achse



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Hypothalamus und Hypophyse



Hypothalamus-Hypophysen-Achse: Releasing/Inhibiting-Hormone

Hypothalamische Hormone

*Thyreotropin-Releasing Hormon
(TRH), Syn: Thyreoliberin*

*Corticotropin-Releasing Hormon
(CRH), Syn: Corticoliberin*

*Gonadotropin-Releasing Hormon
(GnRH), Syn: Gonadoliberin*

*Growth-Hormone-Releasing Hormon
(GHRH, GHR), Syn: Somatoliberin*

*Somatostatin (ST), Syn: Growth Hormone
Release Inhibiting Hormone (GHRH),
Somatotropin-inhibierendes H. (SIH)*

*Prolactin-Release-Inhibiting
Factor (PIF), Syn: **Dopamin***

Adenohypophysäre Hormone

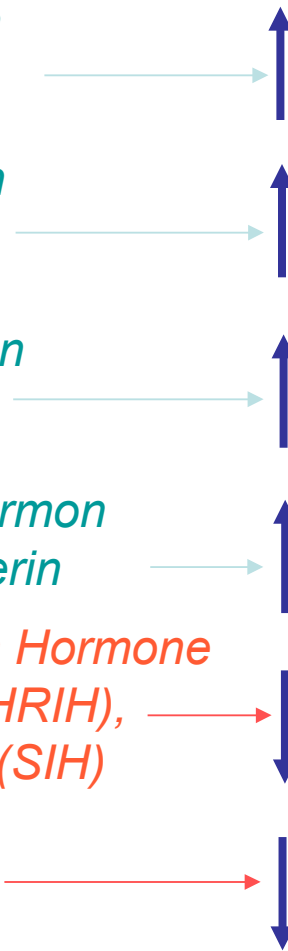
*Thyreoida-stimulierendes Hormon
(TSH)*

*Adrenocorticotropes Hormon
(ACTH)*

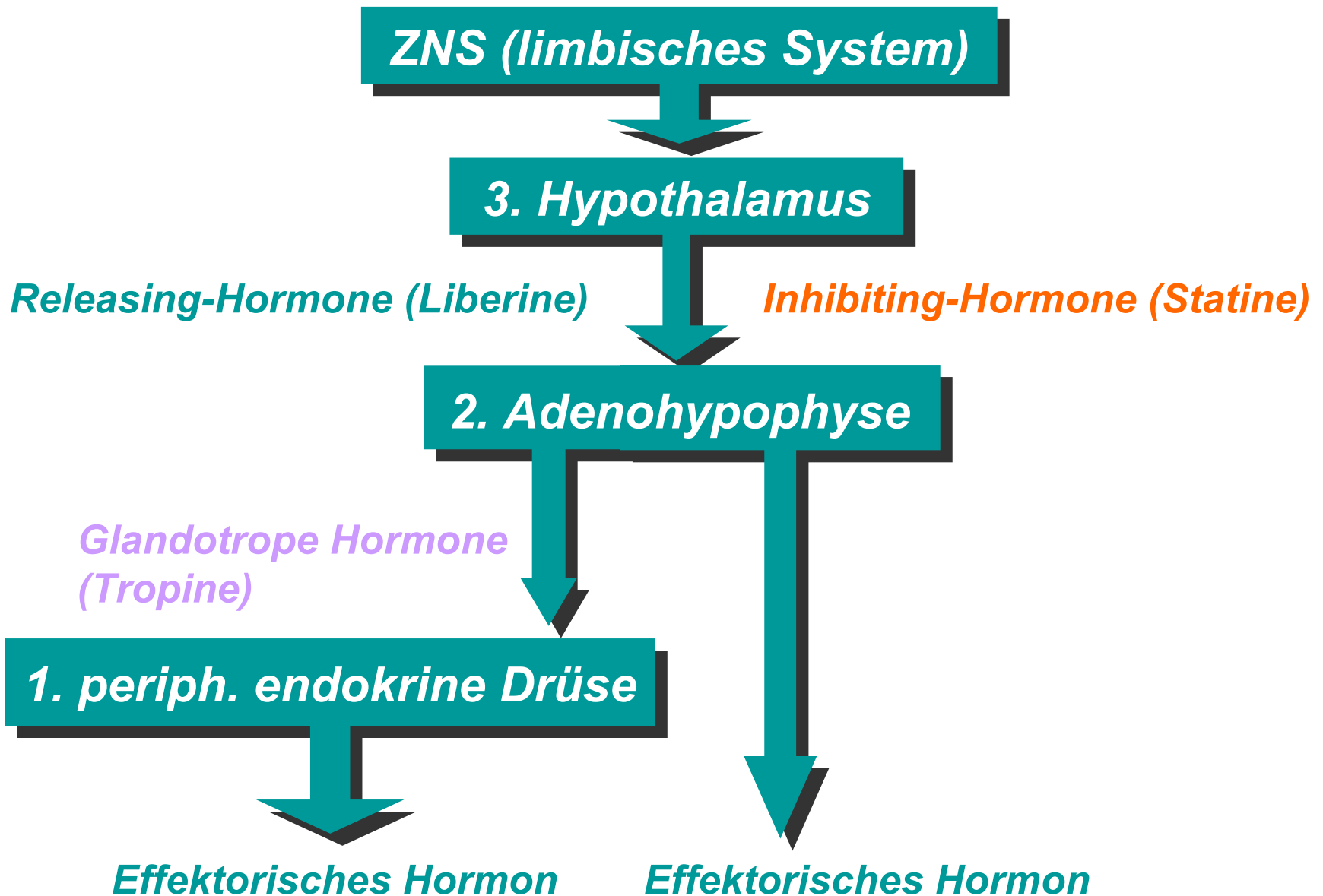
*Follikel-stimulierendes H. (FSH)
Luteinisierendes Hormon (LH)*

*Somatotropes Hormon (STH),
Syn: Growth Hormone (GH)
Somatotropin*

Prolaktin (PRL)



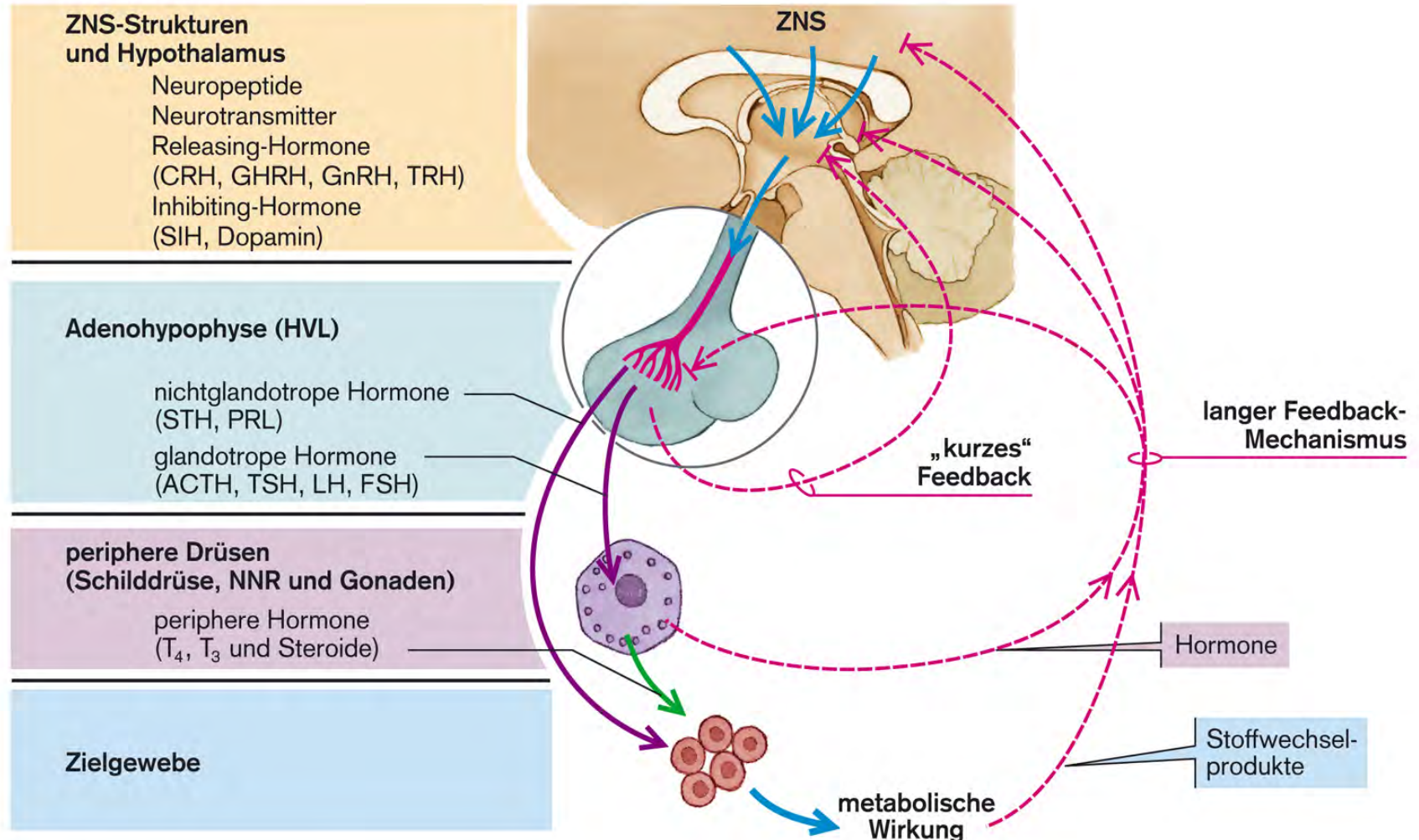
Endokrine Instanzen



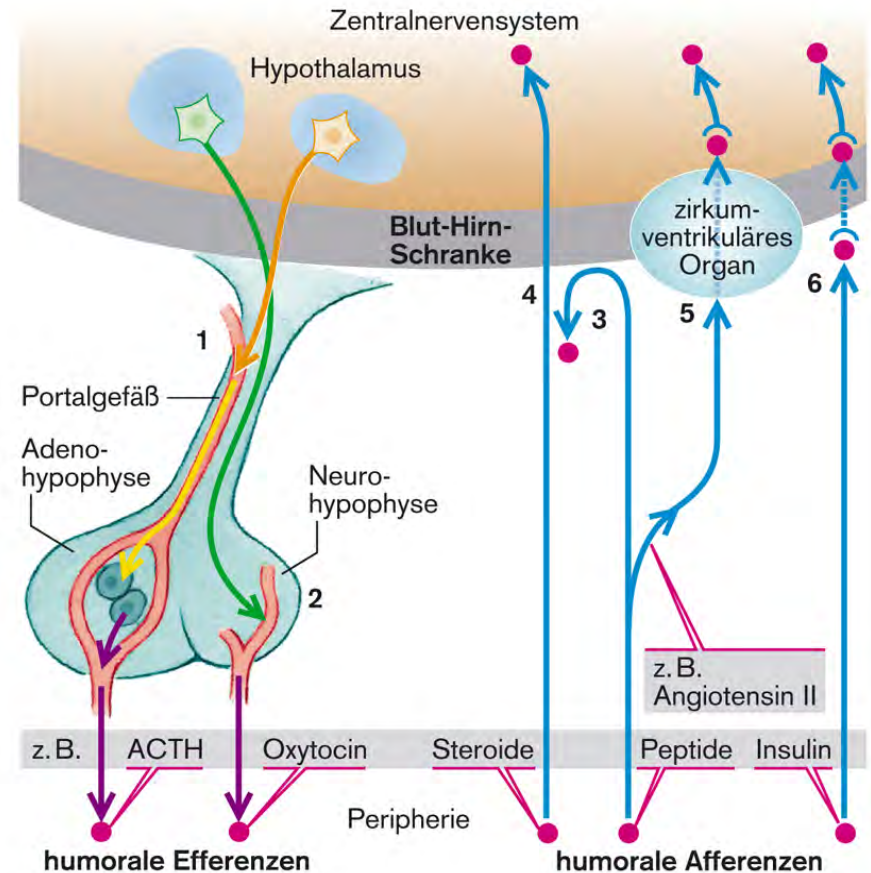
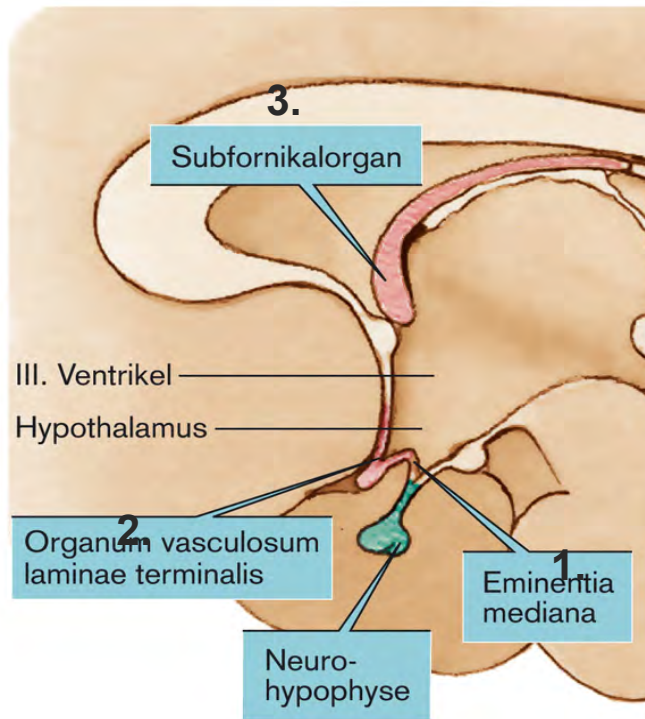
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Regulation der Hormonspiegel: negative Rückkopplung

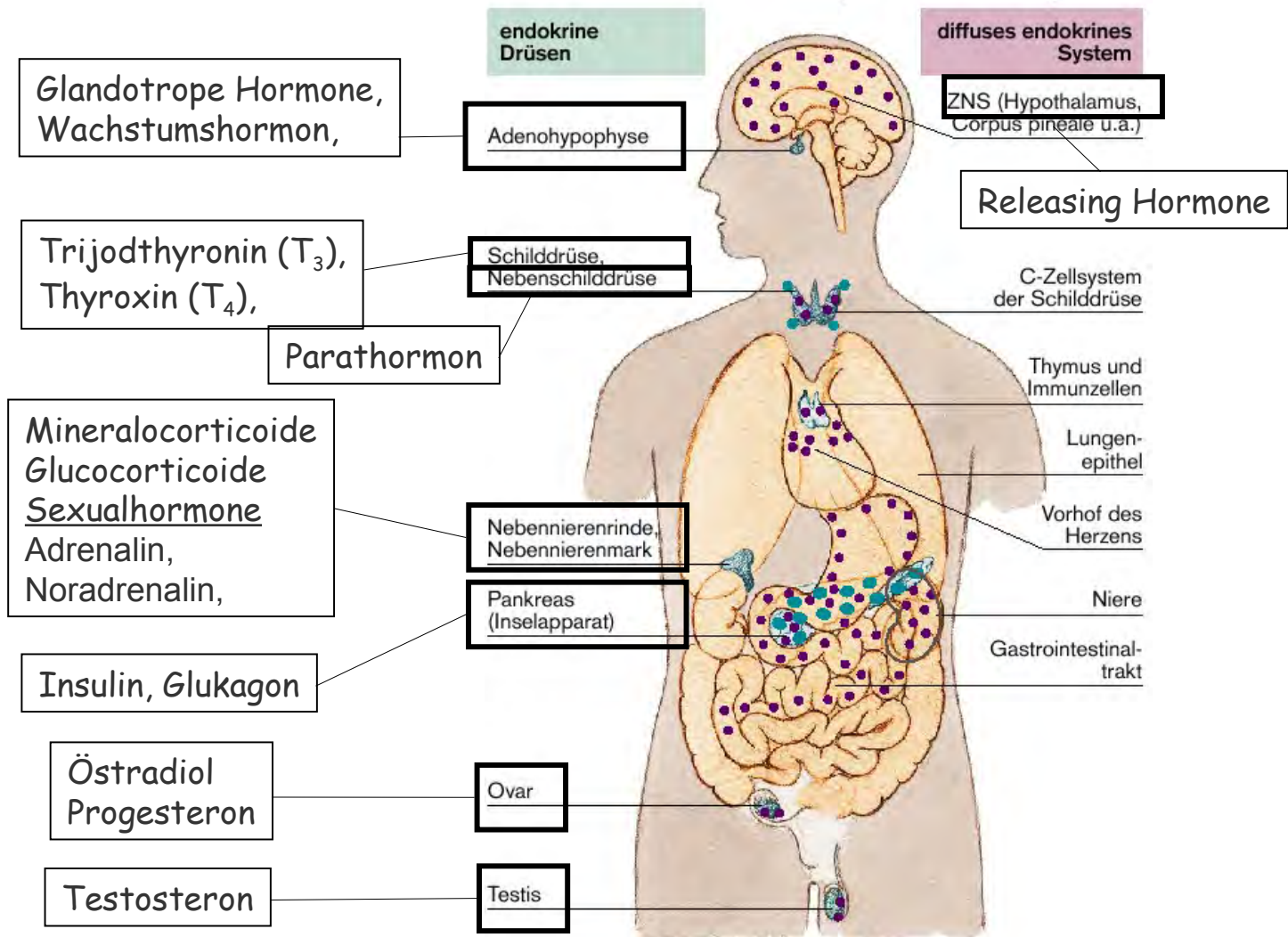


Zirkumventrikuläre Organe: negative Rückkopplung



Georg Thieme Verlag, Stuttgart
 Klinke, Pape, Silbernagl: Physiologie, 5. Auflage 2005
 A. postrema

Endokrines System – Hormone: Übersicht



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Hormonrezeptoren

1. Membranassoziierte Rezeptoren

(Peptidhormone, Katecholamine)

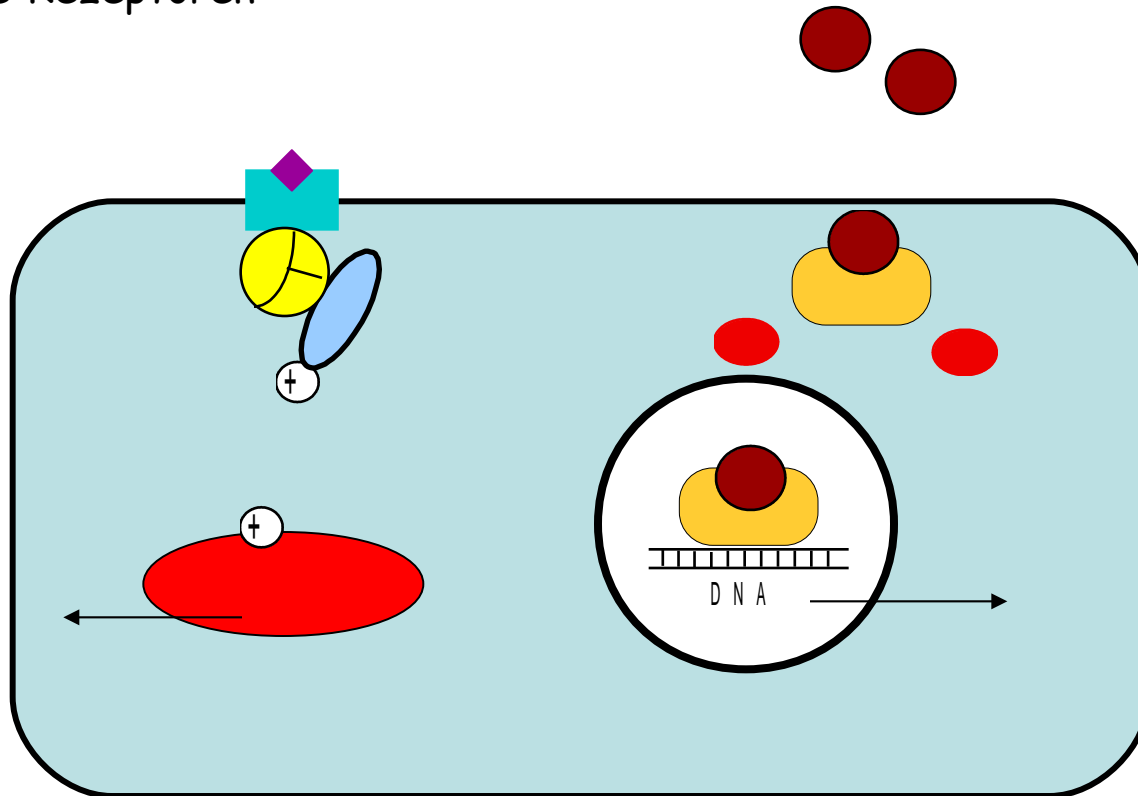
-G-Protein-gekoppelte
Rezeptoren

-Tryosinkinase-Rezeptoren

2. Intrazelluläre Rezeptoren (Steroide, SD-Hormone)

-Steroidhormon-Rezeptorfamilie

(regulierbare Transkriptionsfaktoren)

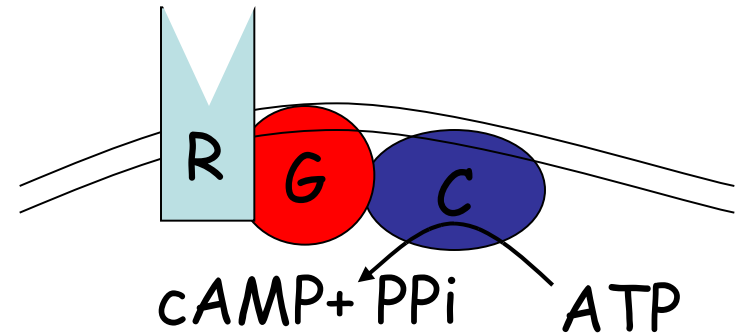


1. Membranassoziierte Hormonrezeptoren

a. G-Protein-gekoppelte Rezeptoren

z.B. β -adrenerger Rezeptor
(Adrenalin)

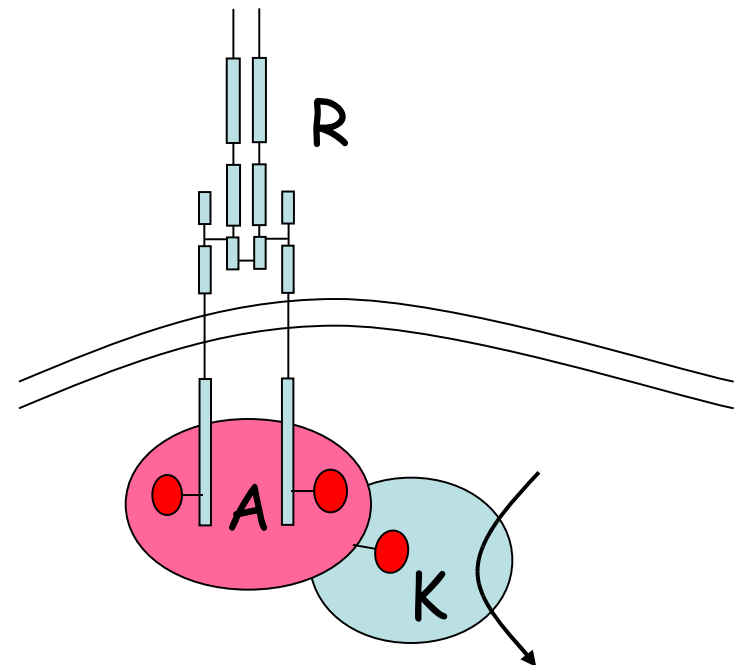
Ankopplung über G-Proteine an 2nd
Messenger-Systeme



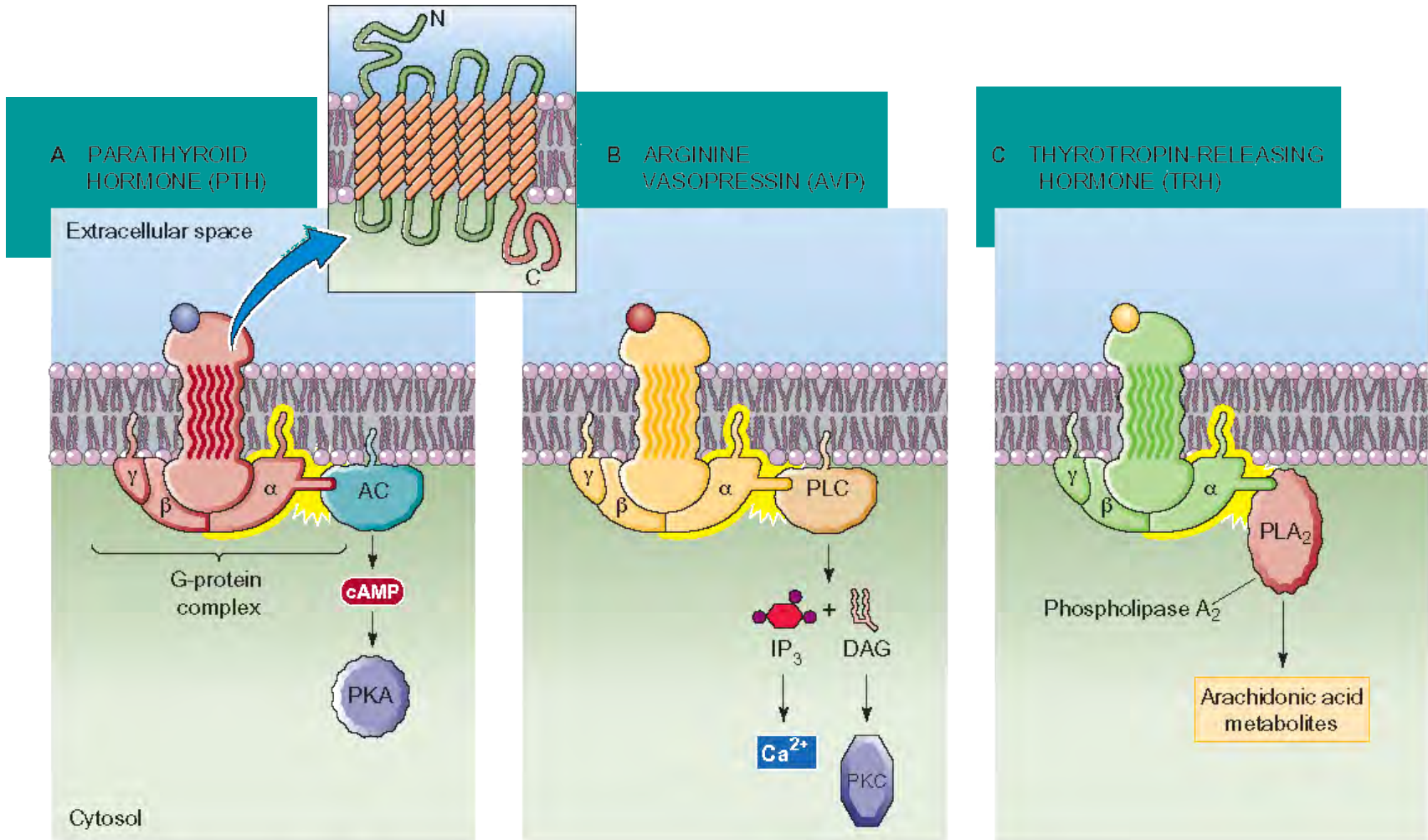
b. Rezeptoren mit Tyrosinkinase-Aktivität (autophosphorylierend)

z. B. Insulinrezeptor

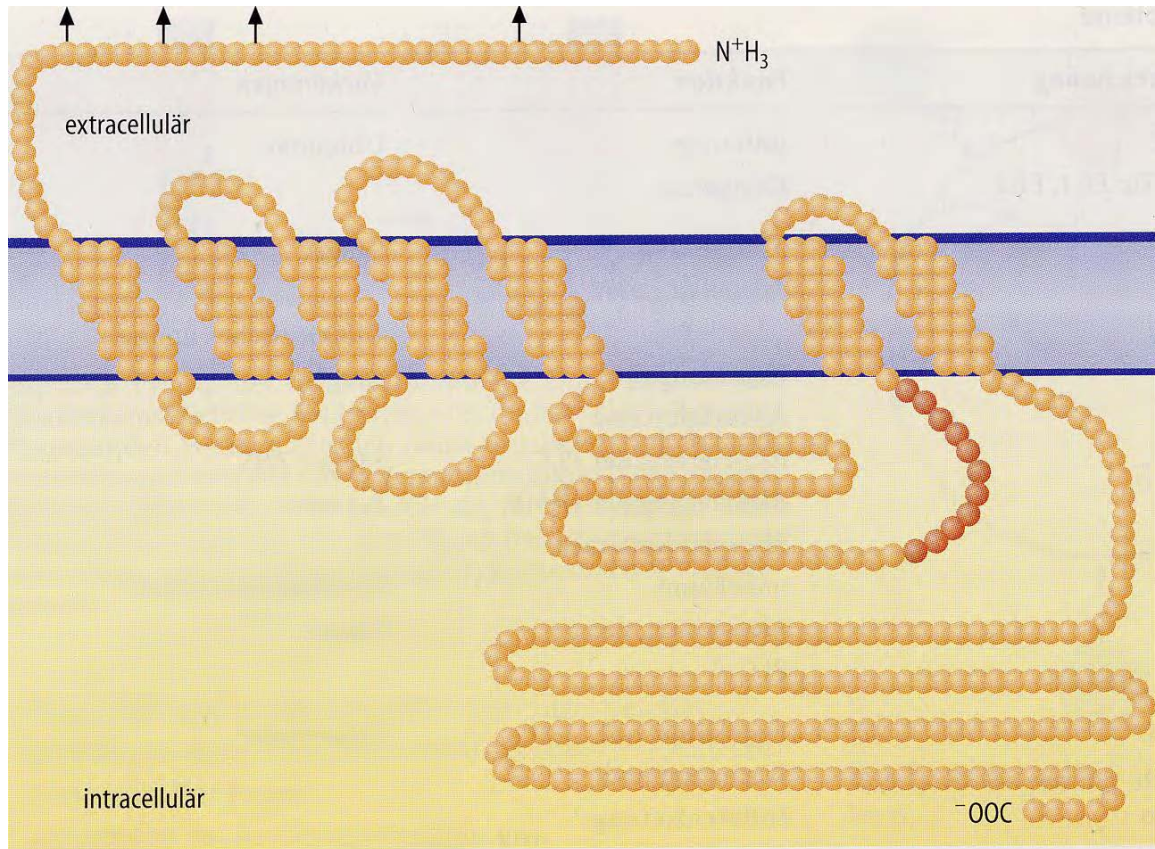
Ankopplung über intrazelluläre Proteine



G-Protein-gekoppelte Rezeptoren: Signalkaskaden



G-Protein-gekoppelte Rezeptoren: 7 Transmembrandomänen



Glukagon-R.

Angiotensin-R.

ADH-R.

ACTH-R.

TSH-R.

Gonadotropin-R.

β -adrenerge-R.

α -adrenerge R.

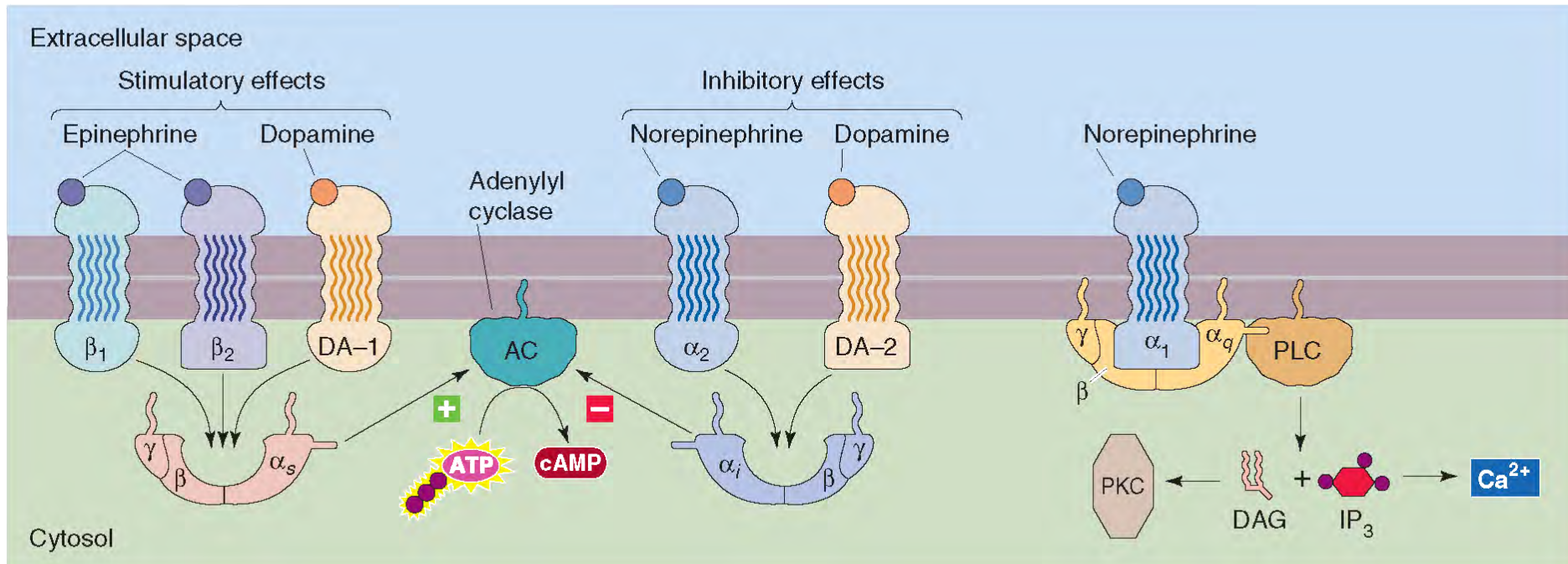
muskarin. ACh-R.

Dopamin-R.

Serotonin-R.

$GABA_B$ -R.

G-Protein-gekoppelte Rezeptoren: Beispiele

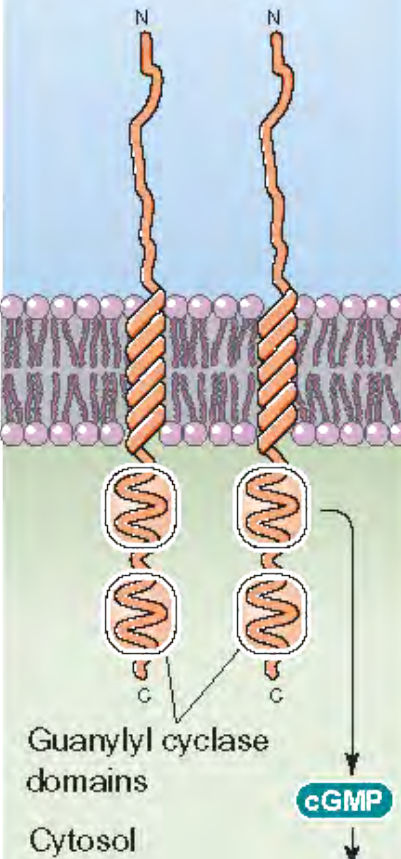


Weitere Peptid-Rezeptoren:

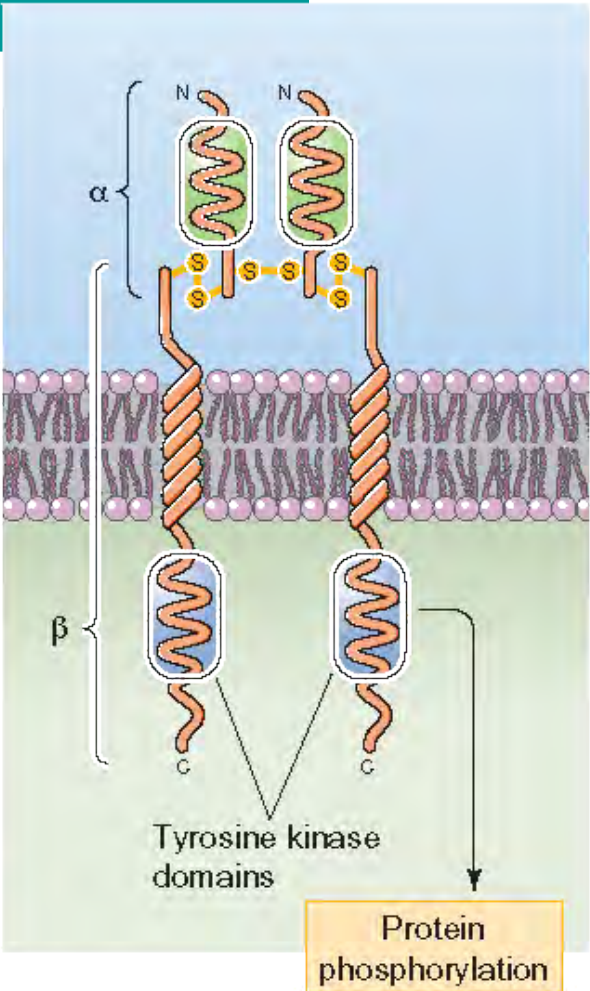
Guanylylcyclase und Thyrosinkinassen

D ATRIAL NATRIURETIC PEPTIDE (ANP)

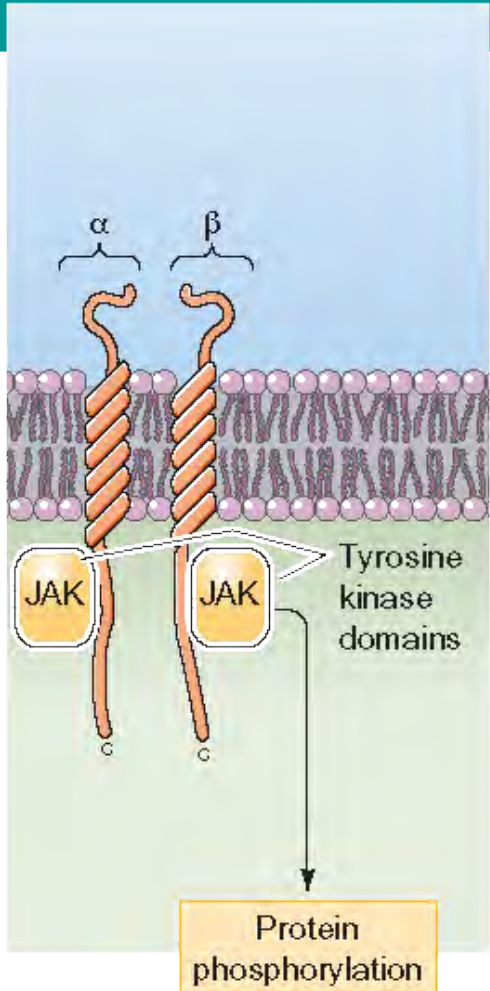
Extracellular space



E INSULIN



F GROWTH HORMONE (GH)



Hormonrezeptoren

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(Peptidhormone, Katecholamine)

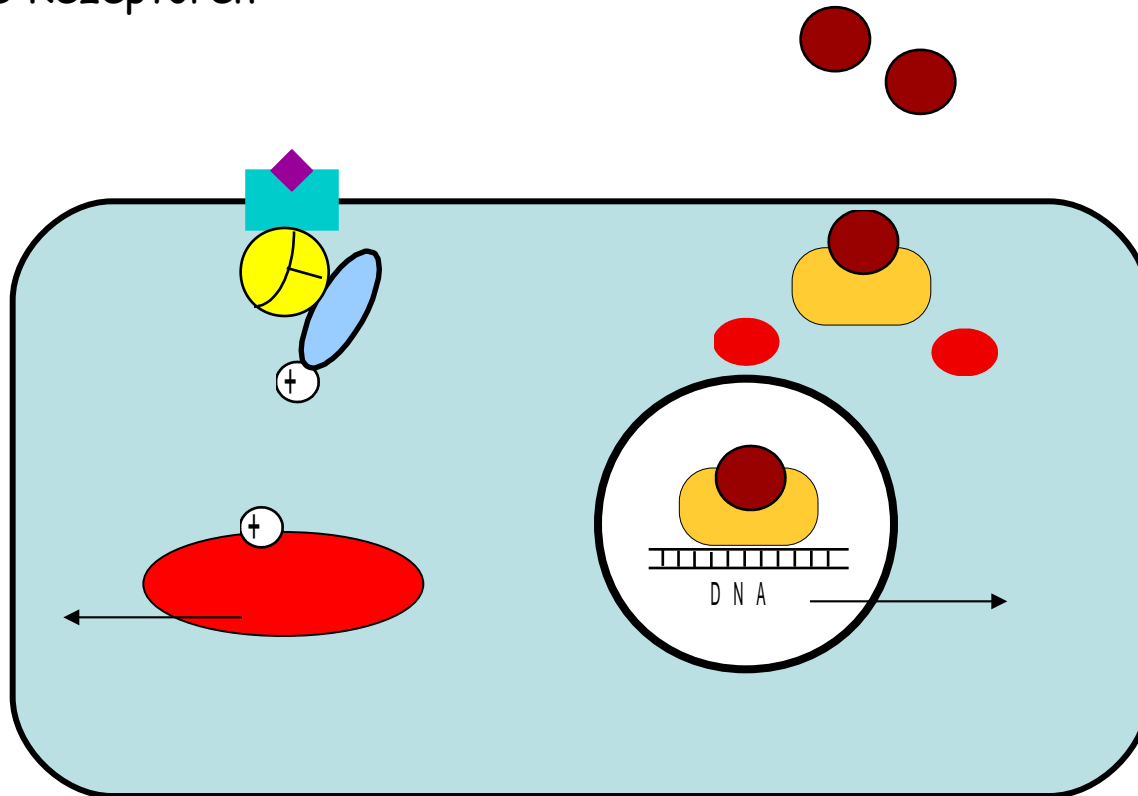
-G-Protein-gekoppelte
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-Tryosinkinase-Rezeptoren

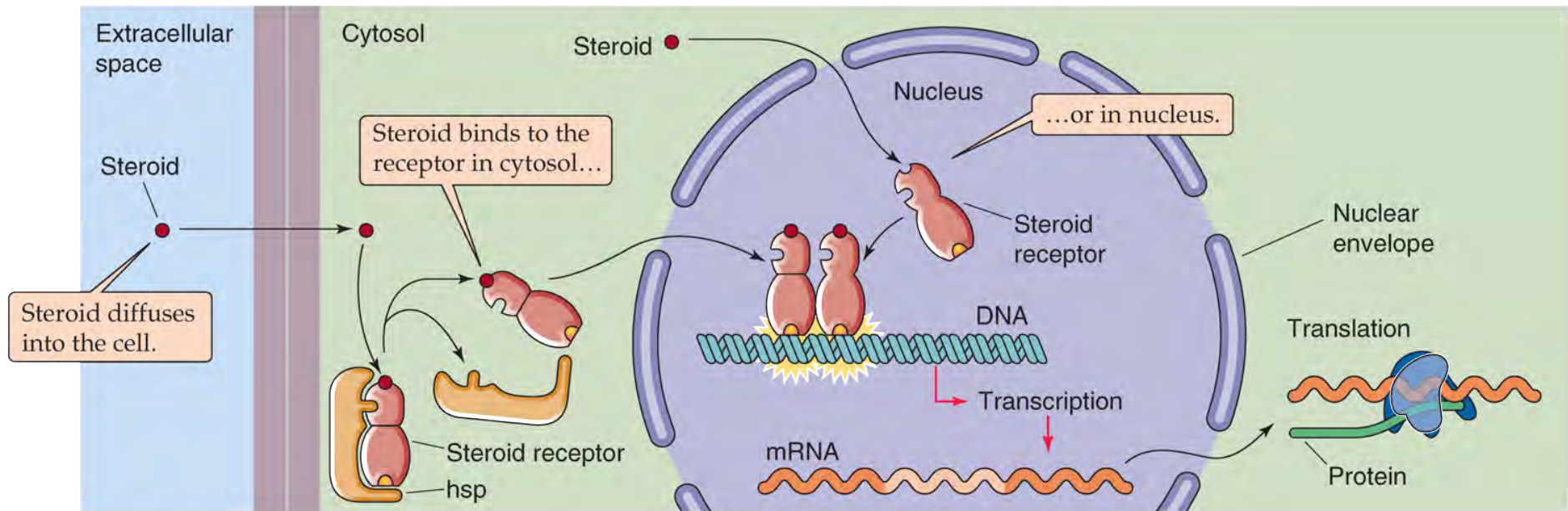
2. Intrazelluläre Rezeptoren (Steroide, SD-Hormone)

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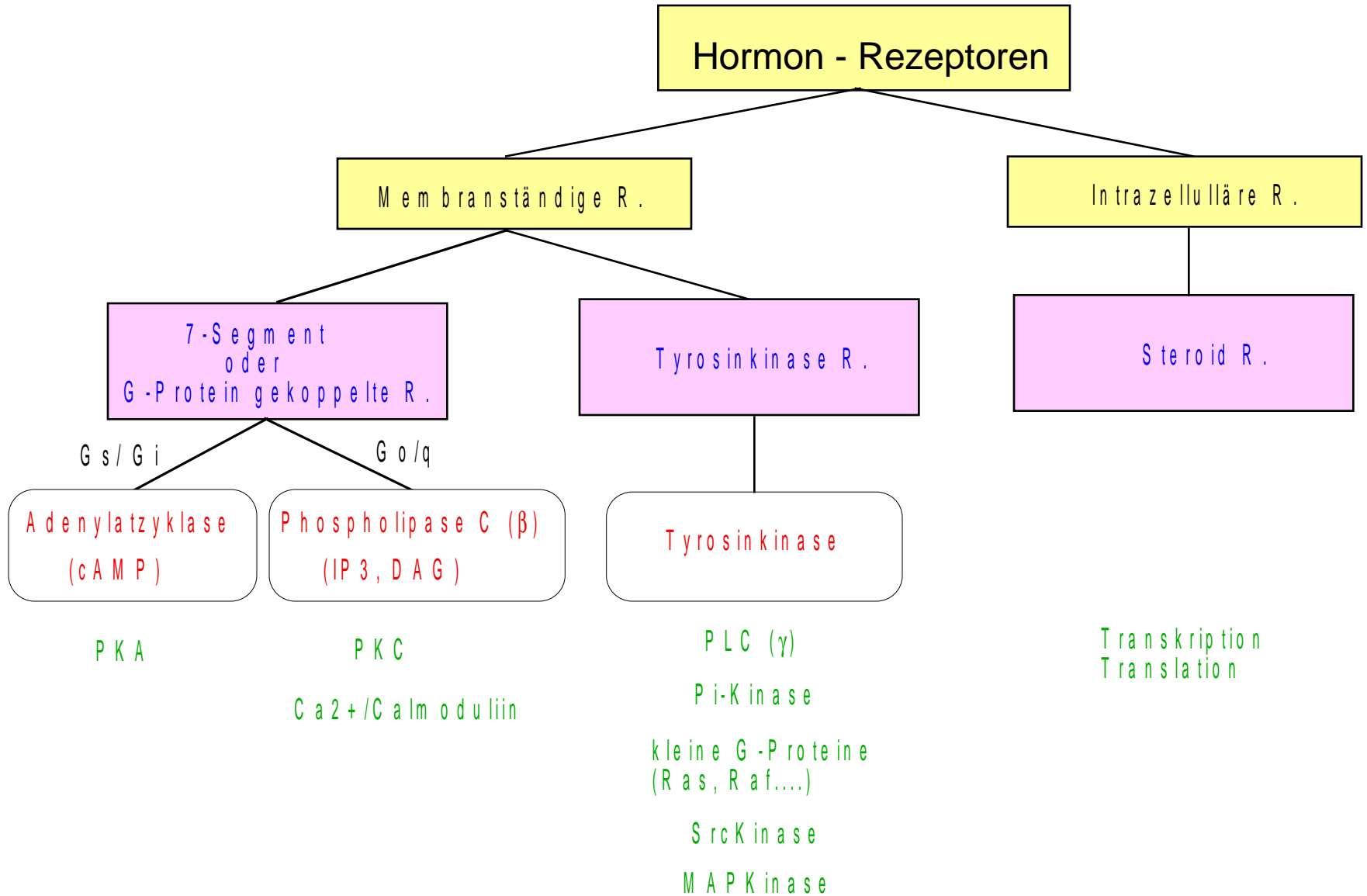
(regulierbare Transkriptionsfaktoren)



Steroid-Hormone: Intrazelluläre Signaltransduktion



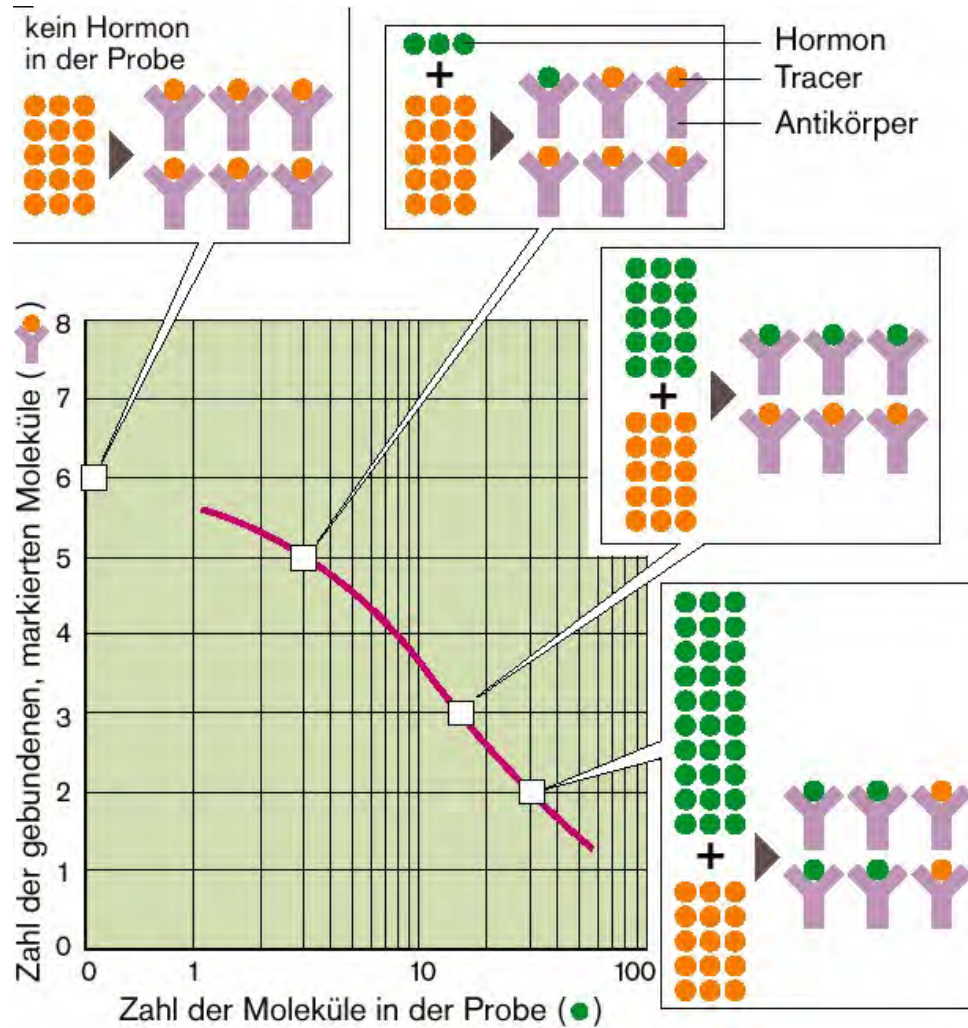
Zusammenfassung - Hormonrezeptoren



Chemische Struktur und Eigenschaften

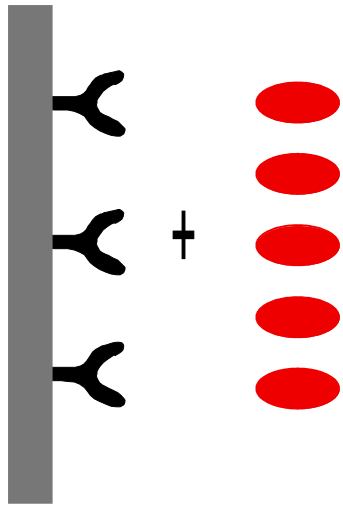
Klasse:	Peptide:	Aminosäuren:	Schilddrüsen-H.:	Steroide:
Beisp.:	Insulin, ADH	Adrenalin	Trijodthyronin (T3)	Cortisol
Chemie:	1-191 AS <i>hydrophil,</i> <i>nicht membrangäng.</i>	Tyrosinderivate <i>hydrophil,</i> <i>nicht membrangäng.</i>	Poly-I-Tyrosinder. <i>hydrophob,</i> membrangängig	Cholesterinderivate <i>hydrophob,</i> membrangängig
Synthese:	Peptidbiosynthese	enzymatisch	enzymatisch	enzymatisch
Sekretion:	Exozytose	Exozytose	Diffusion	Diffusion
HWZ (Plasma):	Min.-Stunden	Sekunden	Tage	Stunden
Rezeptor- lokalis.:	Membran (extraz.)	Membran (extraz.)	Zellkern	Zytosol, Zellkern
Wirkung:	2nd Messenger	2nd Messenger	Transkription	Transkription
Zeitskala:	Minuten-Stunden	Sekunden-Min.	Tage	Stunden-Tage

Nachweisverfahren: Radioimmunassay

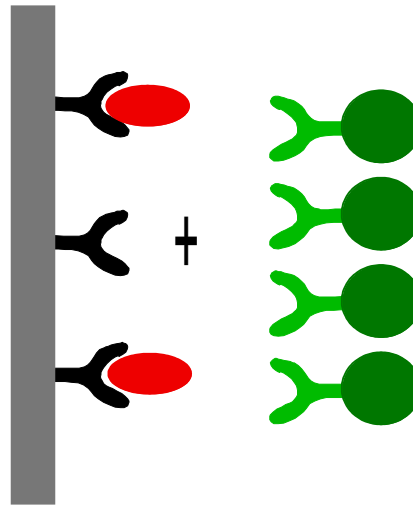


Immunometrisches Verfahren (Festphase)

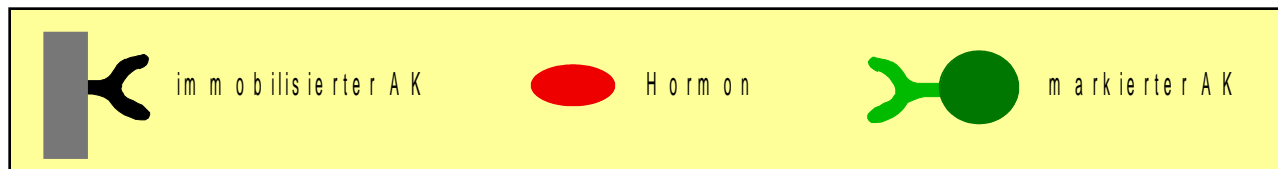
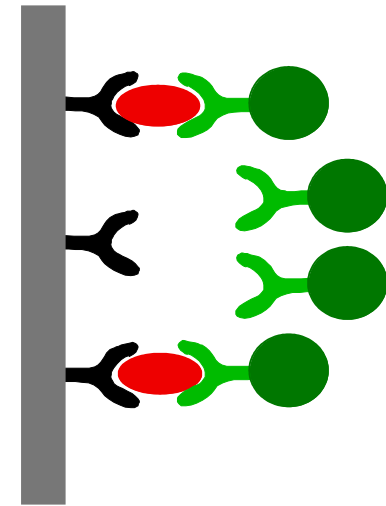
1. Schritt



2. Schritt



3. Schritt



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Die Neurohypophyse: ADH (Vasopressin) / Oxytozin

Neurohypophyse

N u c l. p a r a v e n t r i c u l a r i s

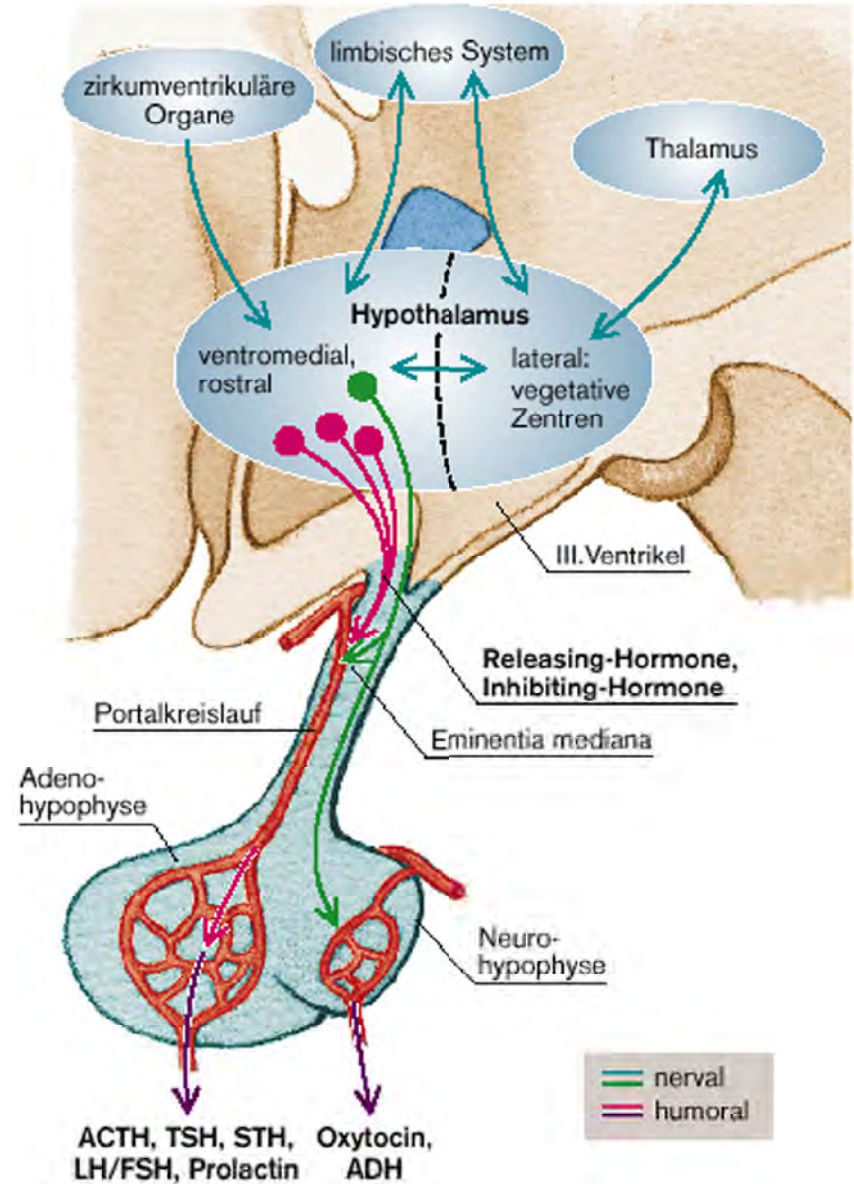
N u c l. s u p r a o p t i c u s

Adenohypophyse

N u c l. v e n t r o m e d i a l i s

N u c l. d o r s o m e d i a l i s

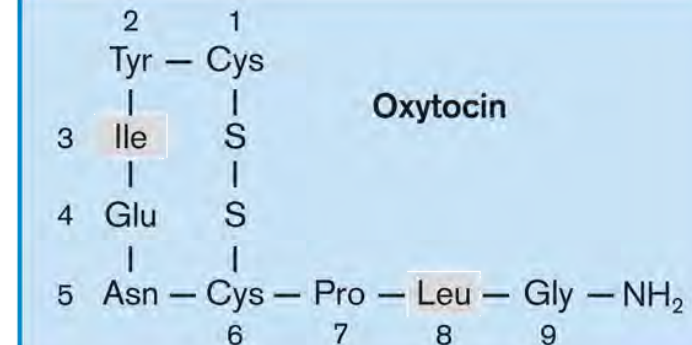
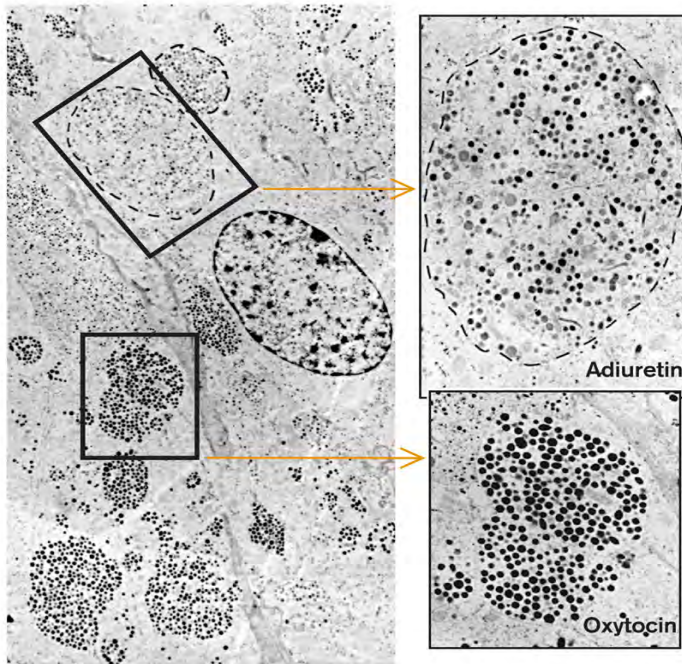
N u c l. i n f u n d i b u l a r i s



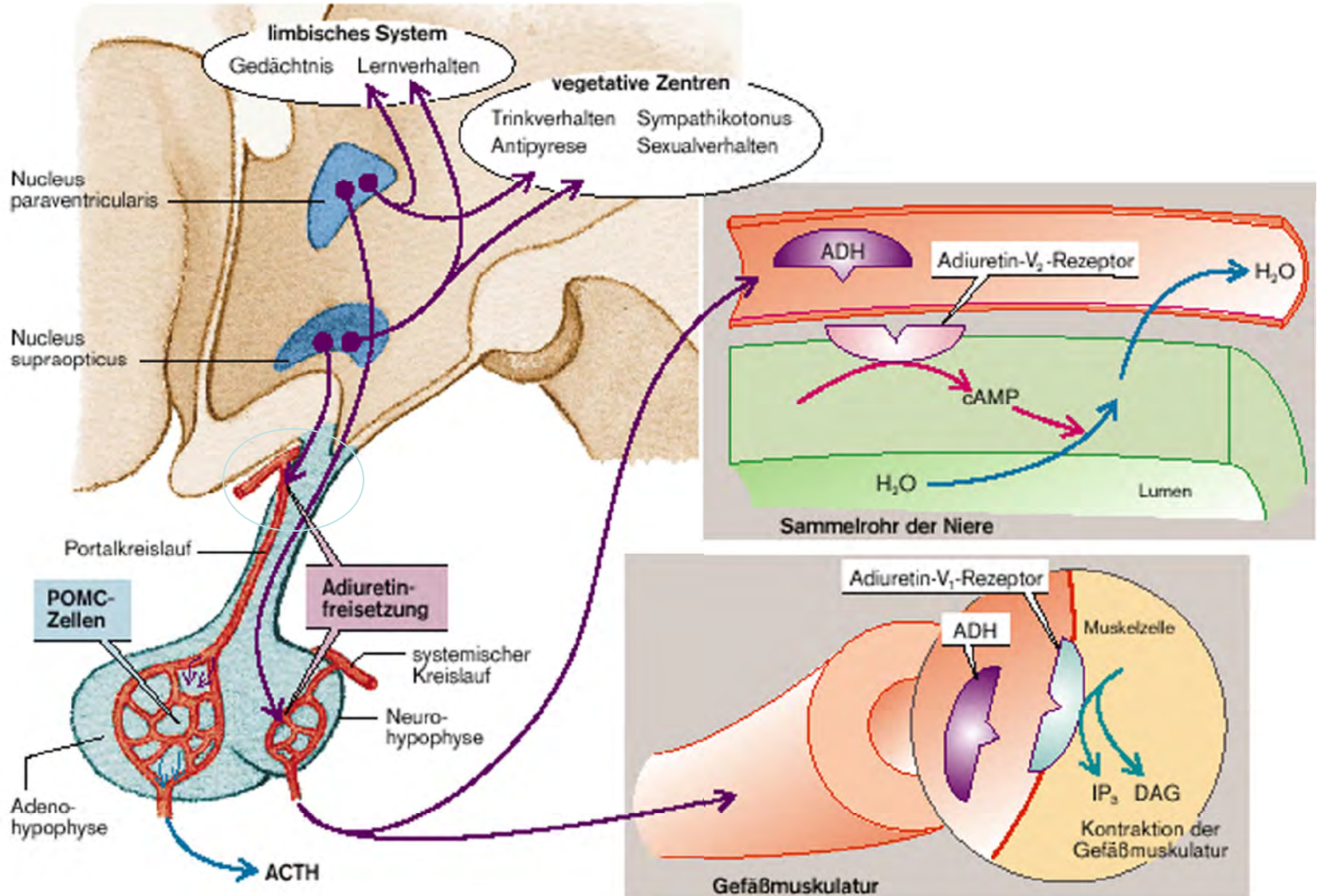
ADH und Oxytozin: strukturell ähnliche Oligopeptide

Neurohypophyse
=

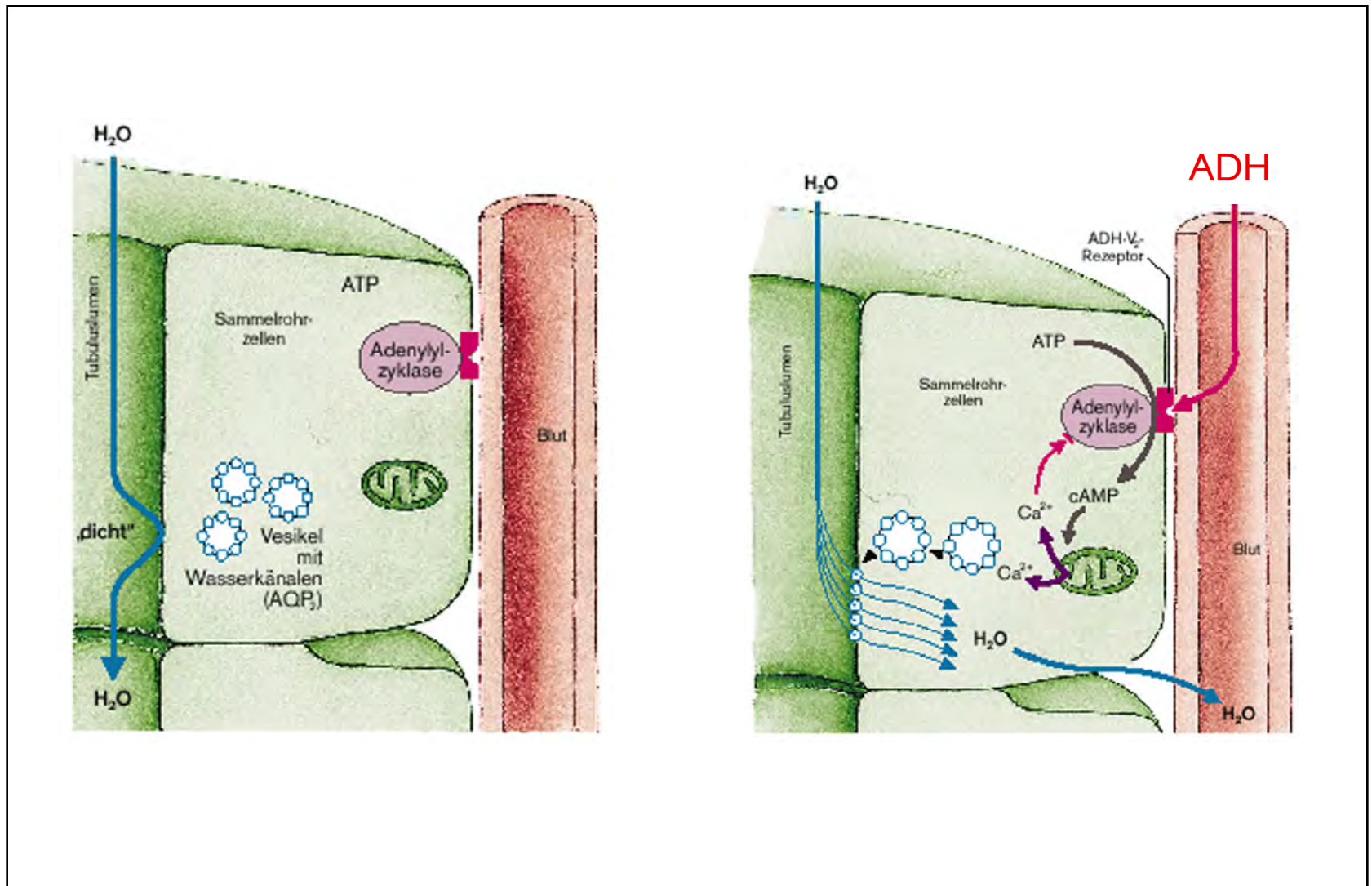
**Terminale von Neuronen
des Ncl. paraventricularis
bzw. Ncl. supraopticus
im Hypothalamus**



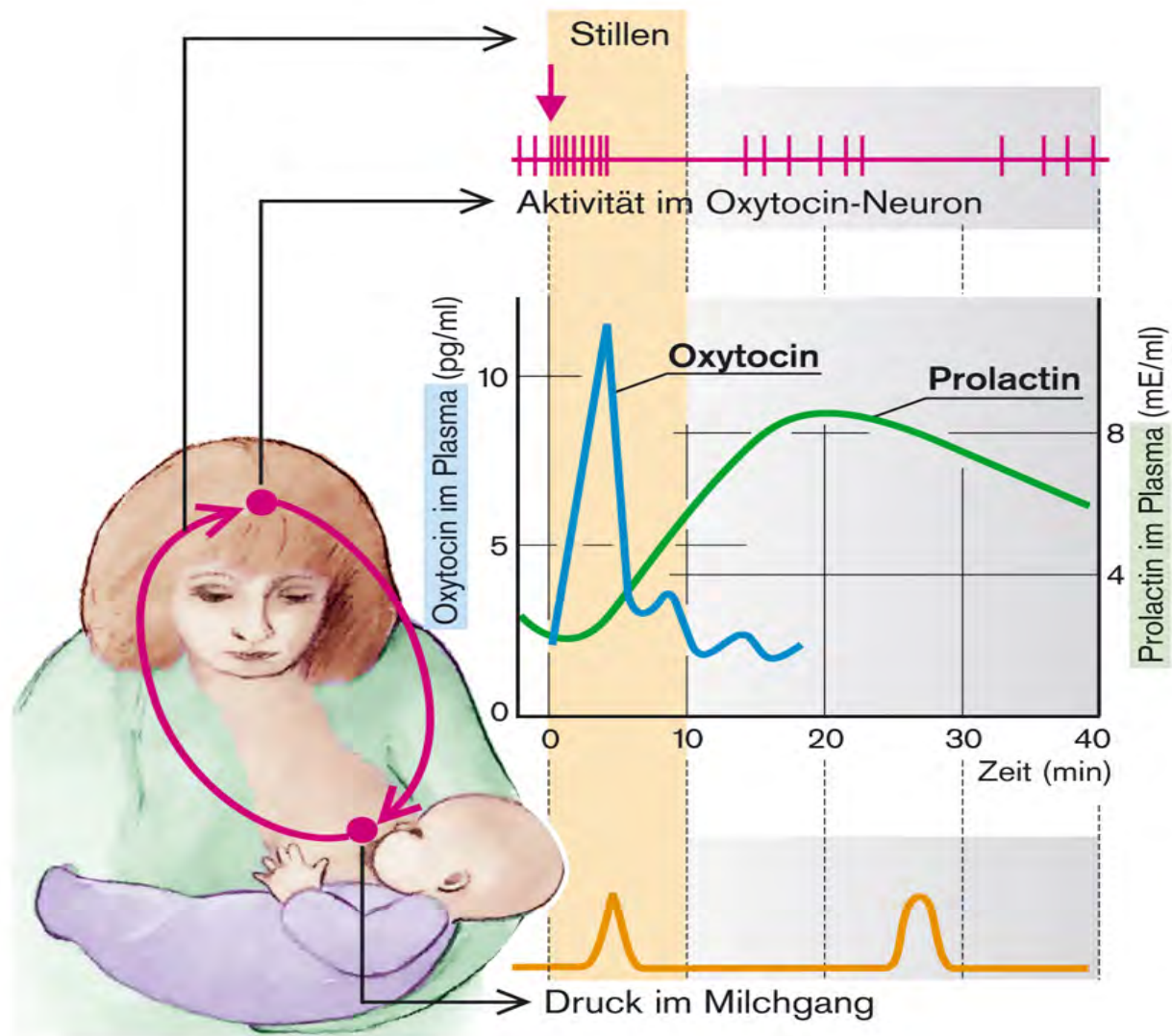
ADH (Neurohypophyse): Zielorgane



Wirkung von ADH (Vasopressin) im Nephron



Oxytozin-Wirkung beim Stillen



Endokrines System – Hormone: speziell

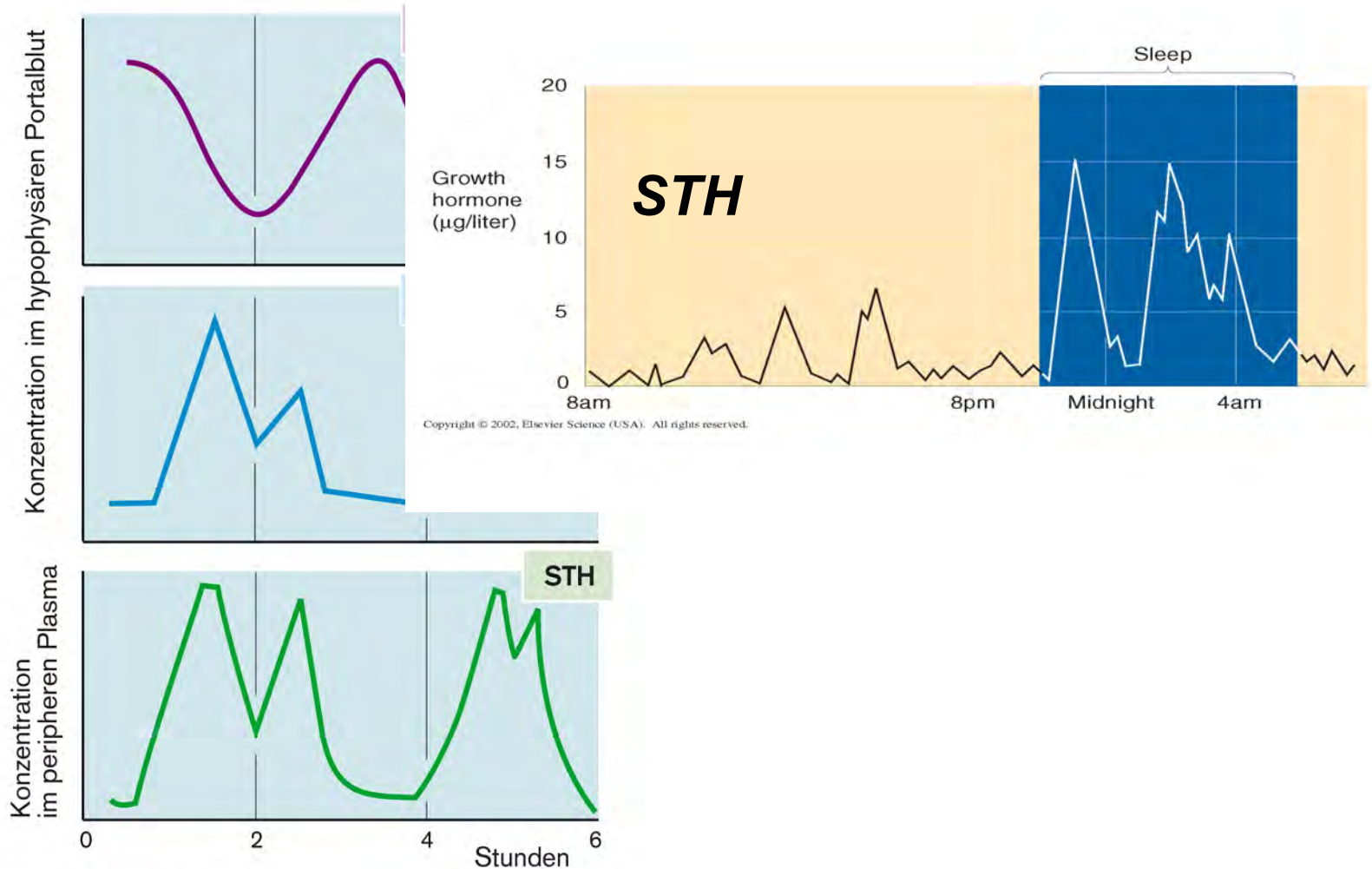
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Somatotropes Hormon (STH)

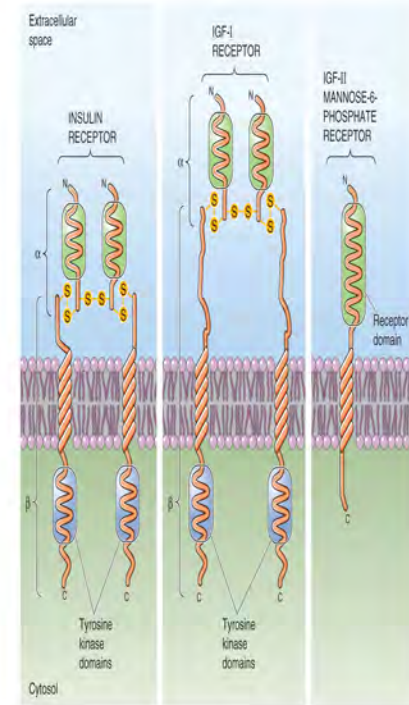
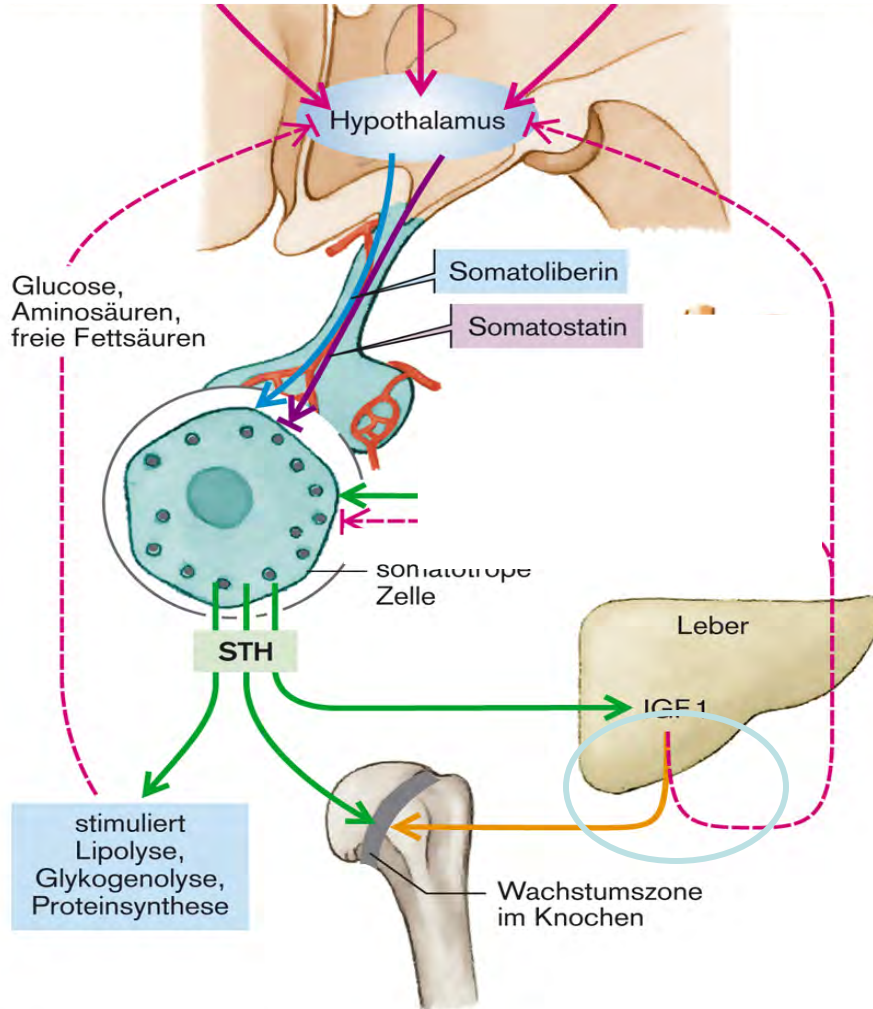
The collage consists of several key elements:

- Top Left:** A diagram of the hypothalamic-pituitary axis. It shows the hypothalamus with the supraoptic nucleus and the pituitary gland with the anterior and posterior lobes. Labels include: "Somatotropes cells in the anterior pituitary secrete growth hormone (GH) and a 19-amino acid peptide that regulates the somatotrophic growth axis.", "Cells in the paraventricular region release somatostatin, a hormone that is a potent inhibitor of growth hormone (GH) secretion, via the posterior pituitary.", "Hypothalamus", "Anterior lobe of pituitary", "Posterior lobe of pituitary", "Somatotrophic axis", "Somatostatin inhibits the release of GH by somatotrophic cells.", "Copyright © 2002, Elsevier Science (USA). All rights reserved."
- Top Right:** A diagram of a cell membrane showing two receptors labeled "GHRH" (green) and "SIH" (red).
- Bottom Left:** A microscopic image of a cell, likely a somatotrophic cell.
- Bottom Center:** A diagram of a glandular structure, possibly the pituitary gland, with the label "STH" below it.
- Bottom Right:** A callout box with a yellow background and black border containing the text: "anabol, Steuerung des Längenwachstums".

Somatotropes Hormon: sekretorische Episoden (verstärkt in Tiefschlafphasen)

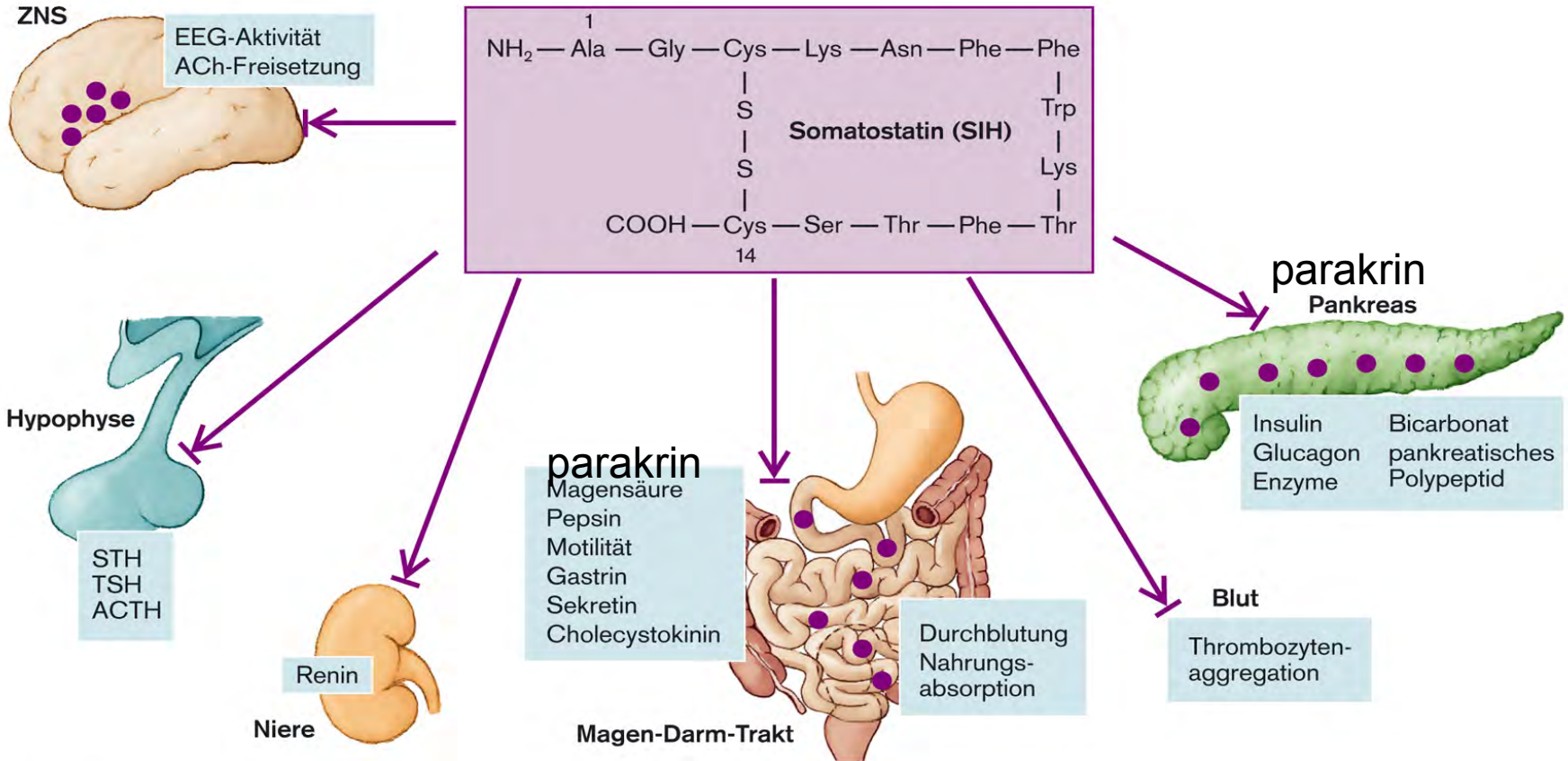


STH – IGF - Achse



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Somastatin (SIH): inhibierende Wirkungen



Vor Pubertät:

STH-Mangel

=>

***Hypophysärer
Zwergwuchs***

STH-Überschuß

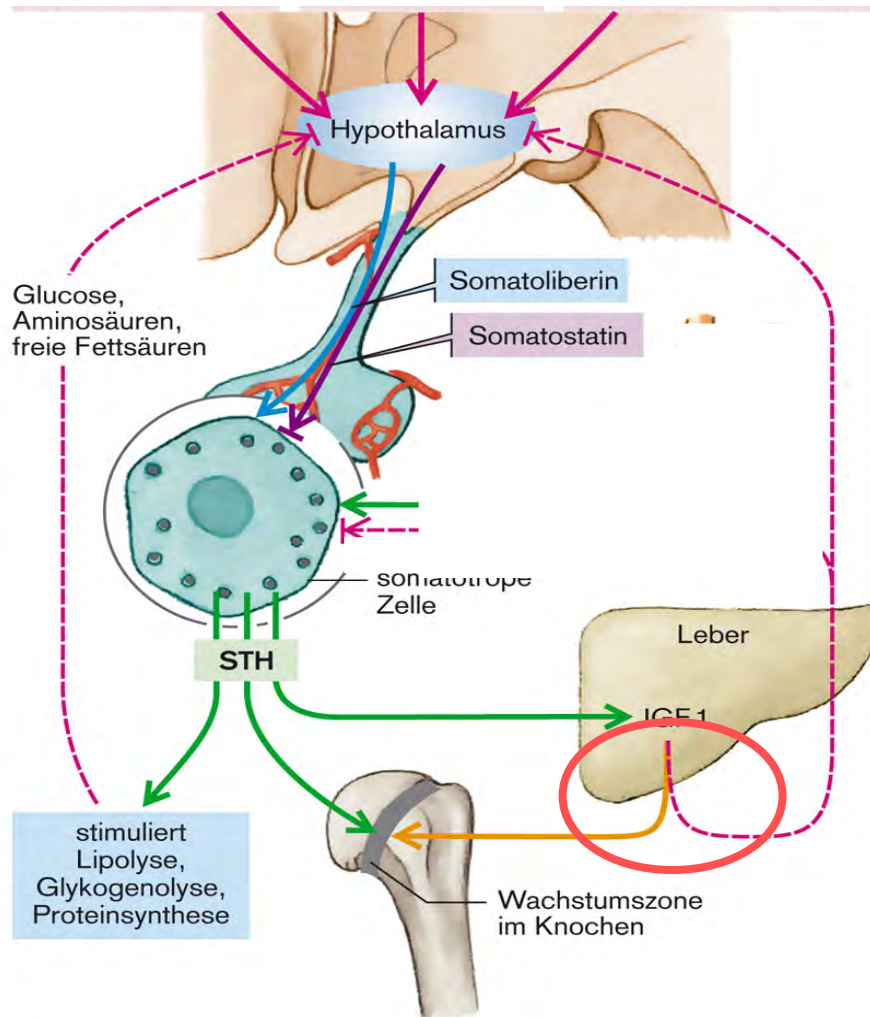
=>

***Hypophysärer
Riesenwuchs***



Abb. 272. 30jähriger Mann mit idiopathischem hypophysärem Zwergwuchs. Körpergröße 140 cm, extremer sexueller Infantilismus mit doppel-seitigem Kryptorchismus. Sehr kleine, aber sonst unauffällige Sella, keine Augensymptome. Skelettalter entspricht einem 10jährigen. Zeichen der sekundären Nebennierenrindensuffizienz.

Diagnose: „hypophysärer Kleinwuchs“ IGF 1 - Mangel



Nach Pubertät:

STH-Überschuß

=>

Akromegalie



Abb. 274. 32-jähriger Mann mit typischen akromegalen Veränderungen des Gesichts und der Hände. Körpergröße 197 cm, Schuhgröße 48, Taillenumgröße Exkavation der Sella turcica; keine Gesichtsfeldausfälle. Behandlung: transkraniale Tumorresektion + Radiotherapie der Hypophysen (1969) + Hormontherapie.

Hypophysenadenom mit Panhypopituitarismus

normale Hypophyse

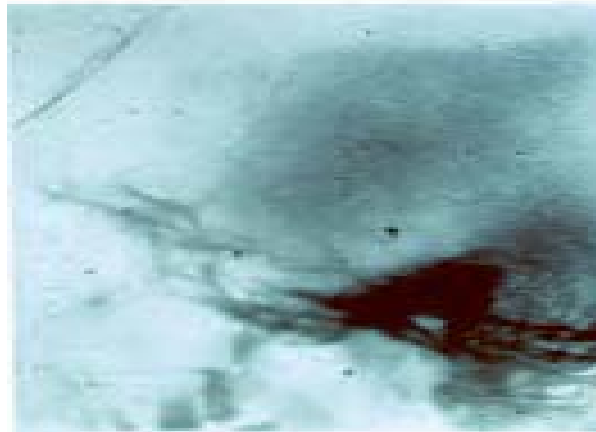
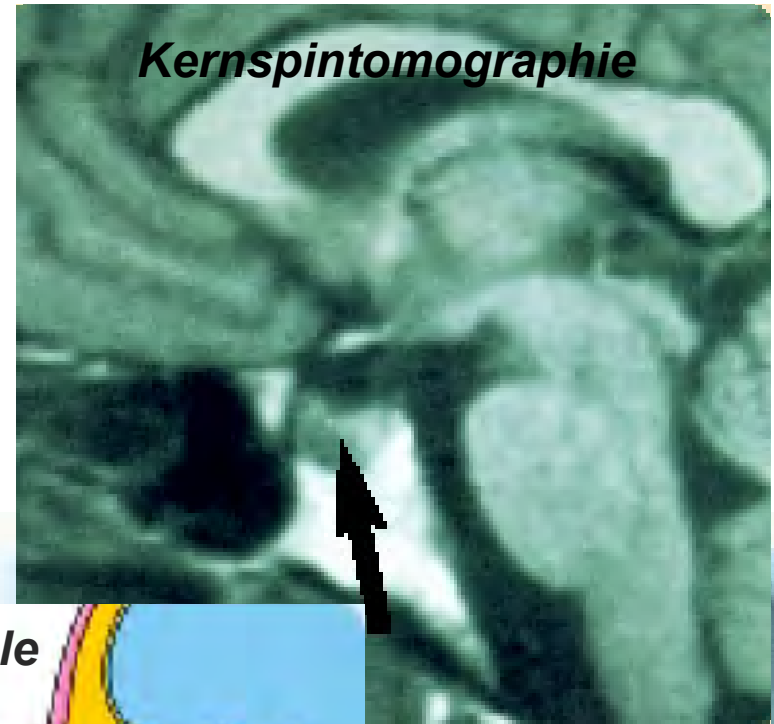
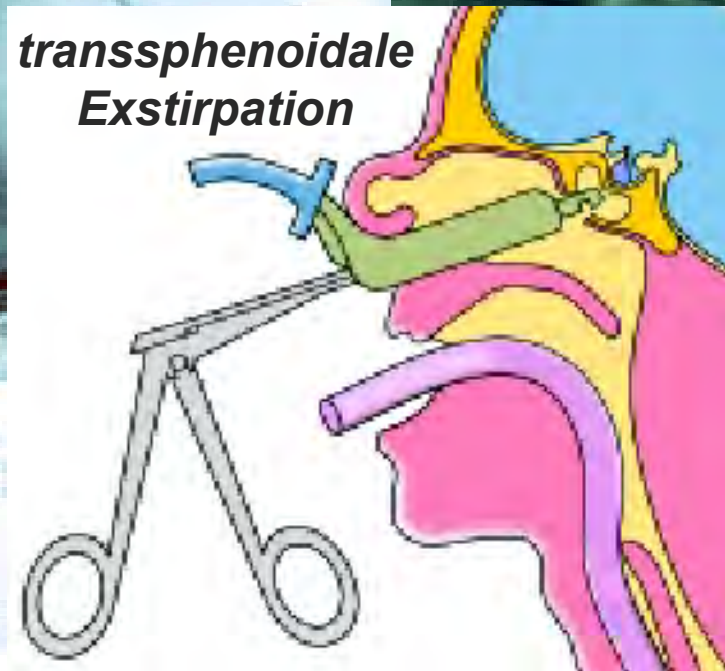
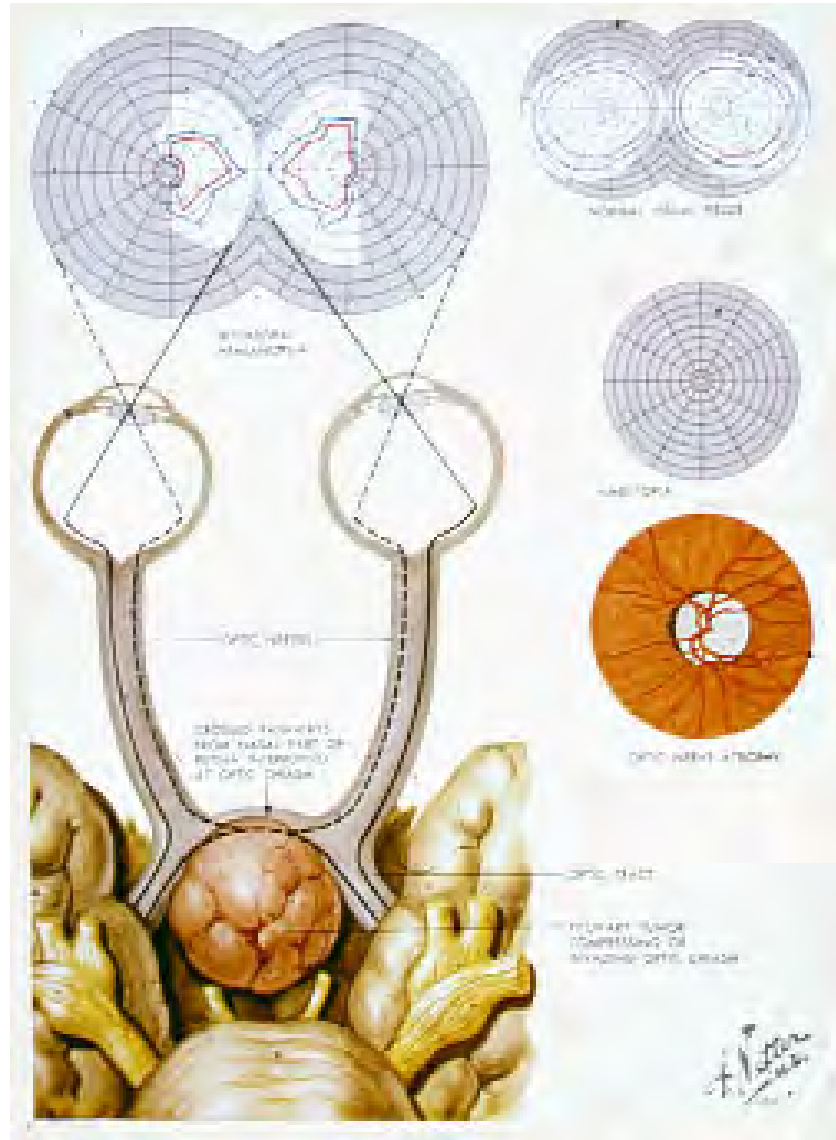


Fig. 276. a) Normale Sella turcica
bei einem 40-jährigen Mannes mit
Zustand:



Exstirpation der Keil-
beinhypophyse

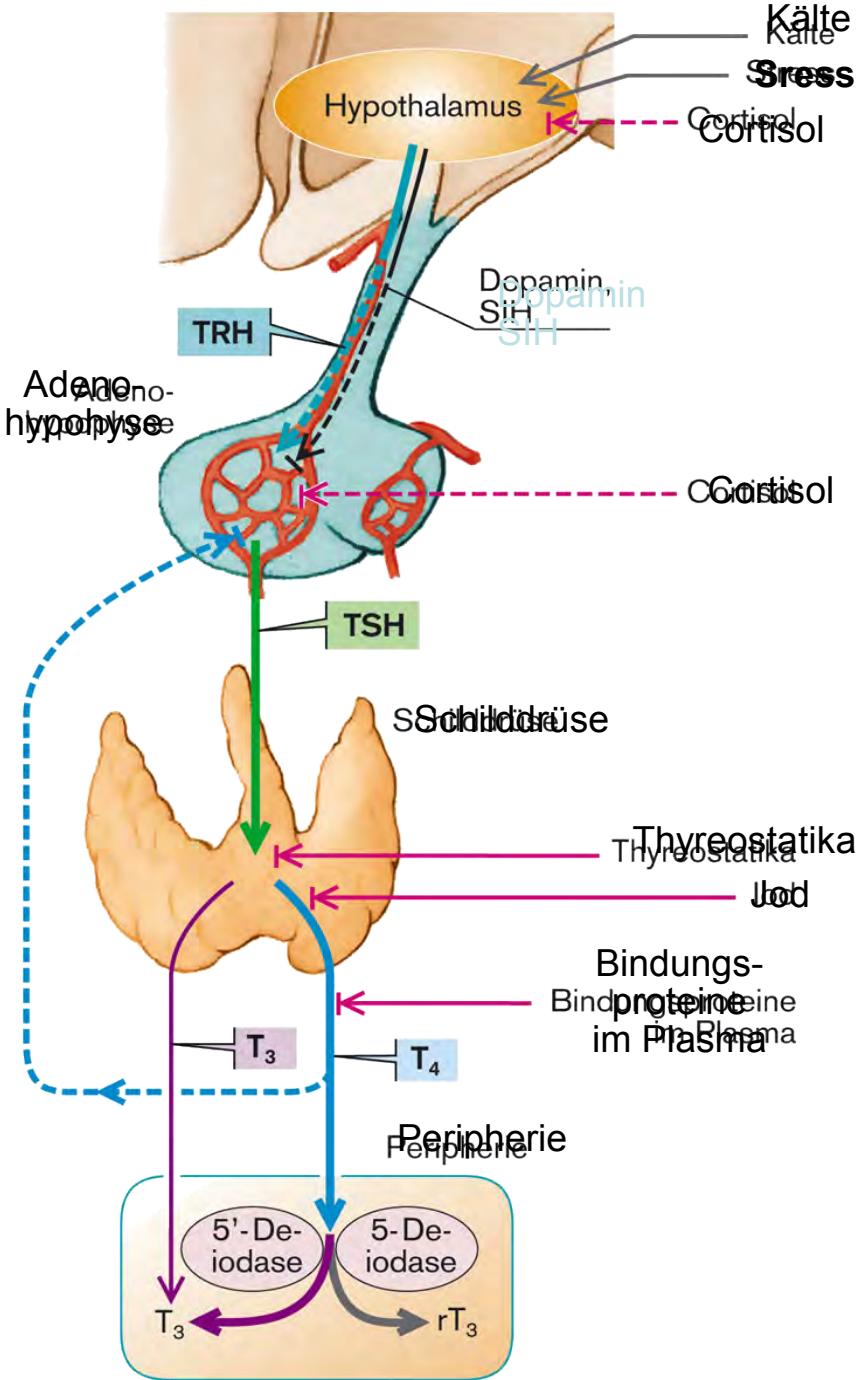
Akromegalie: bitemporale Hemianopsie



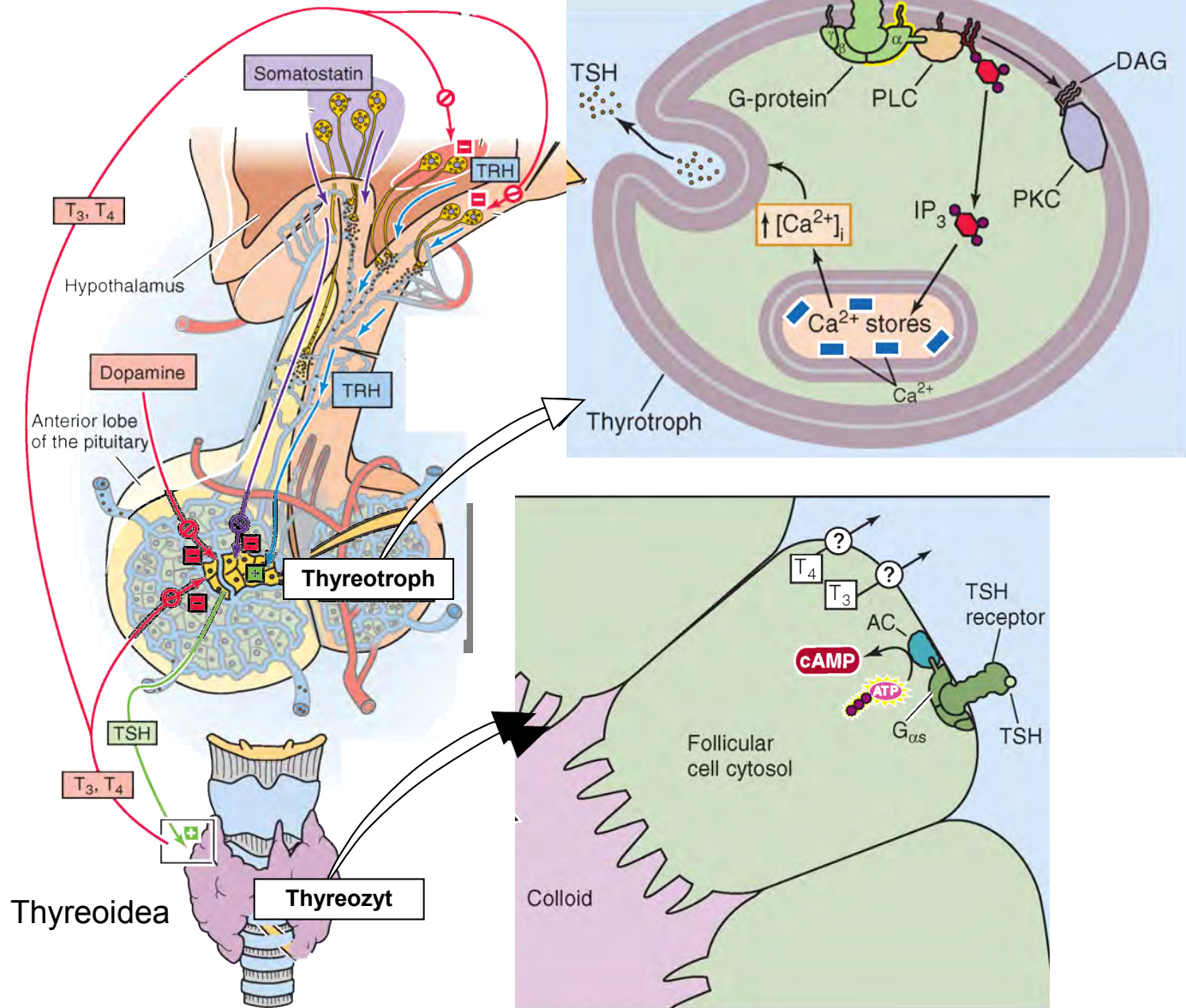
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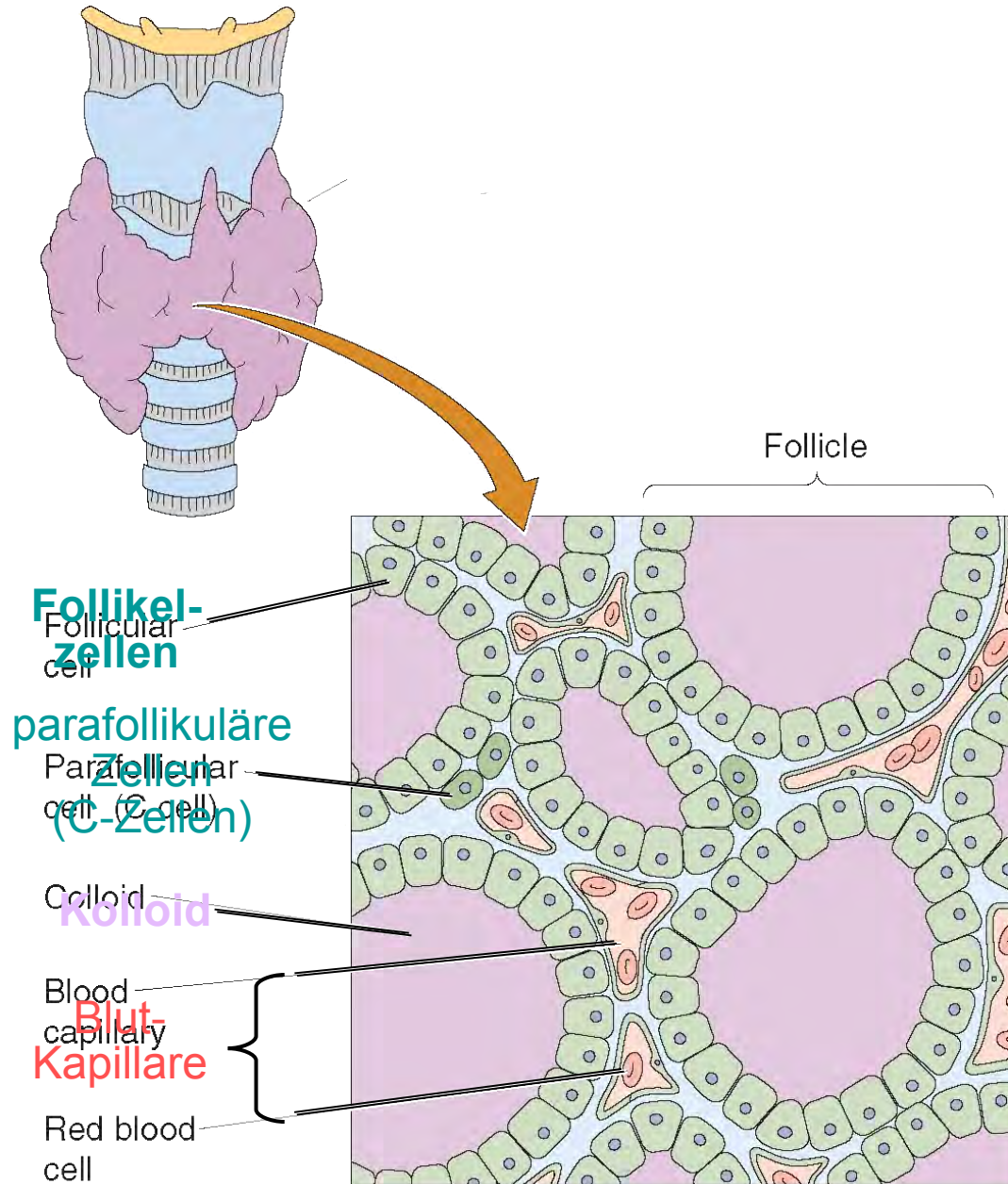
Schilddrüse: Überblick



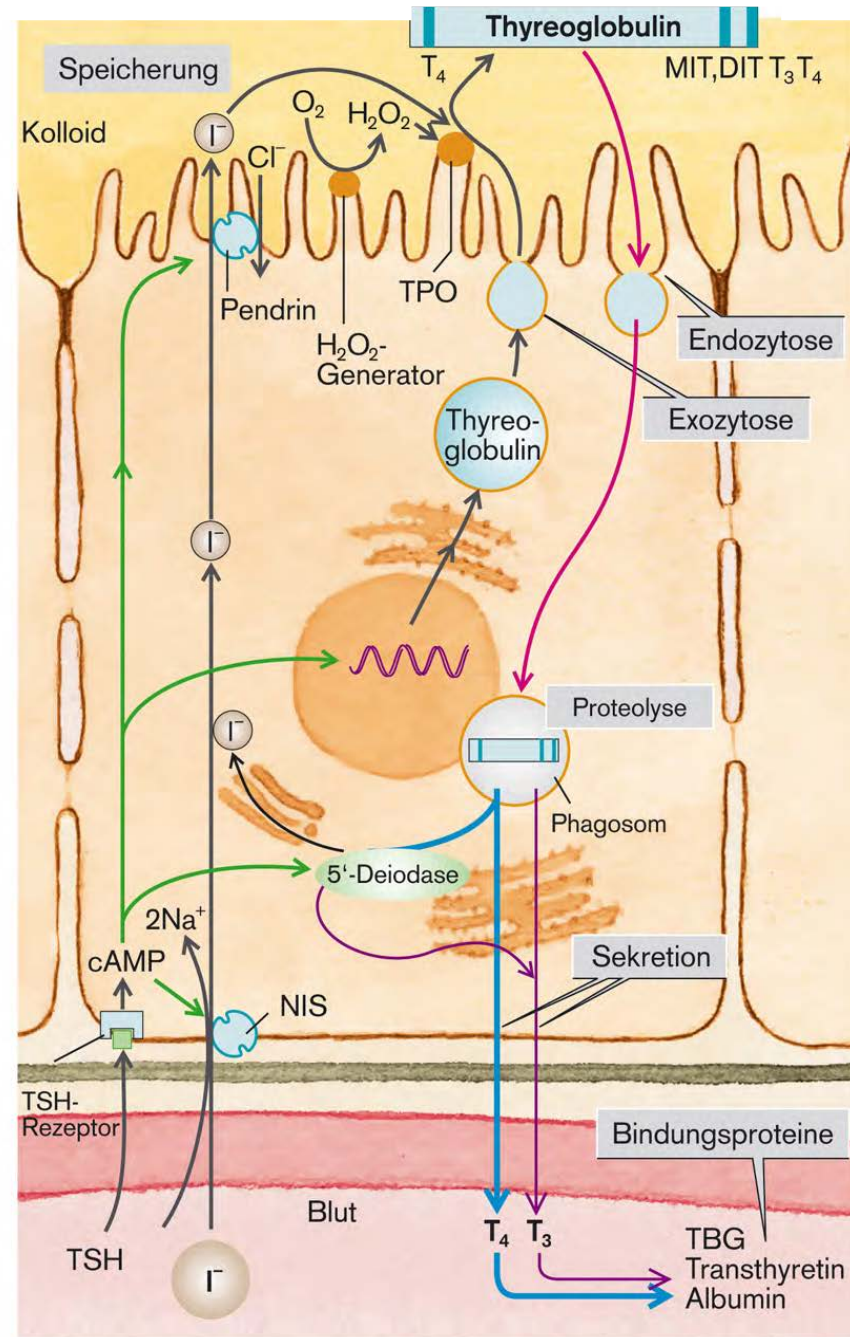
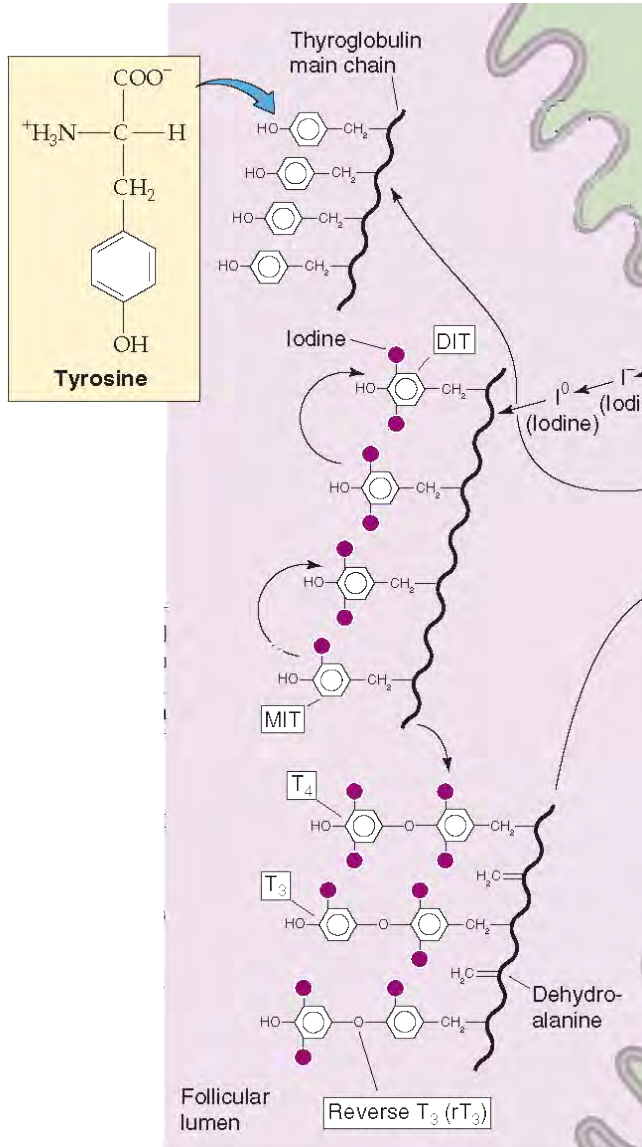
Schilddrüse: Signaltransduktion



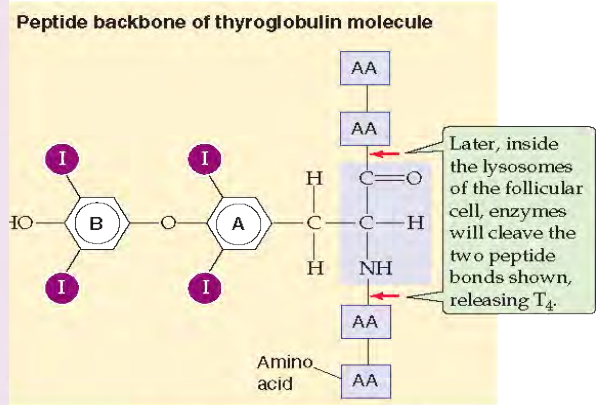
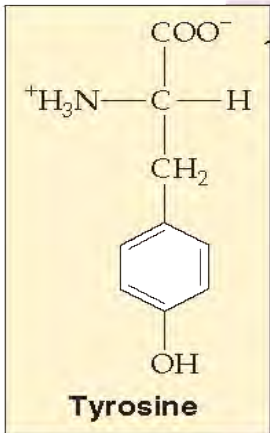
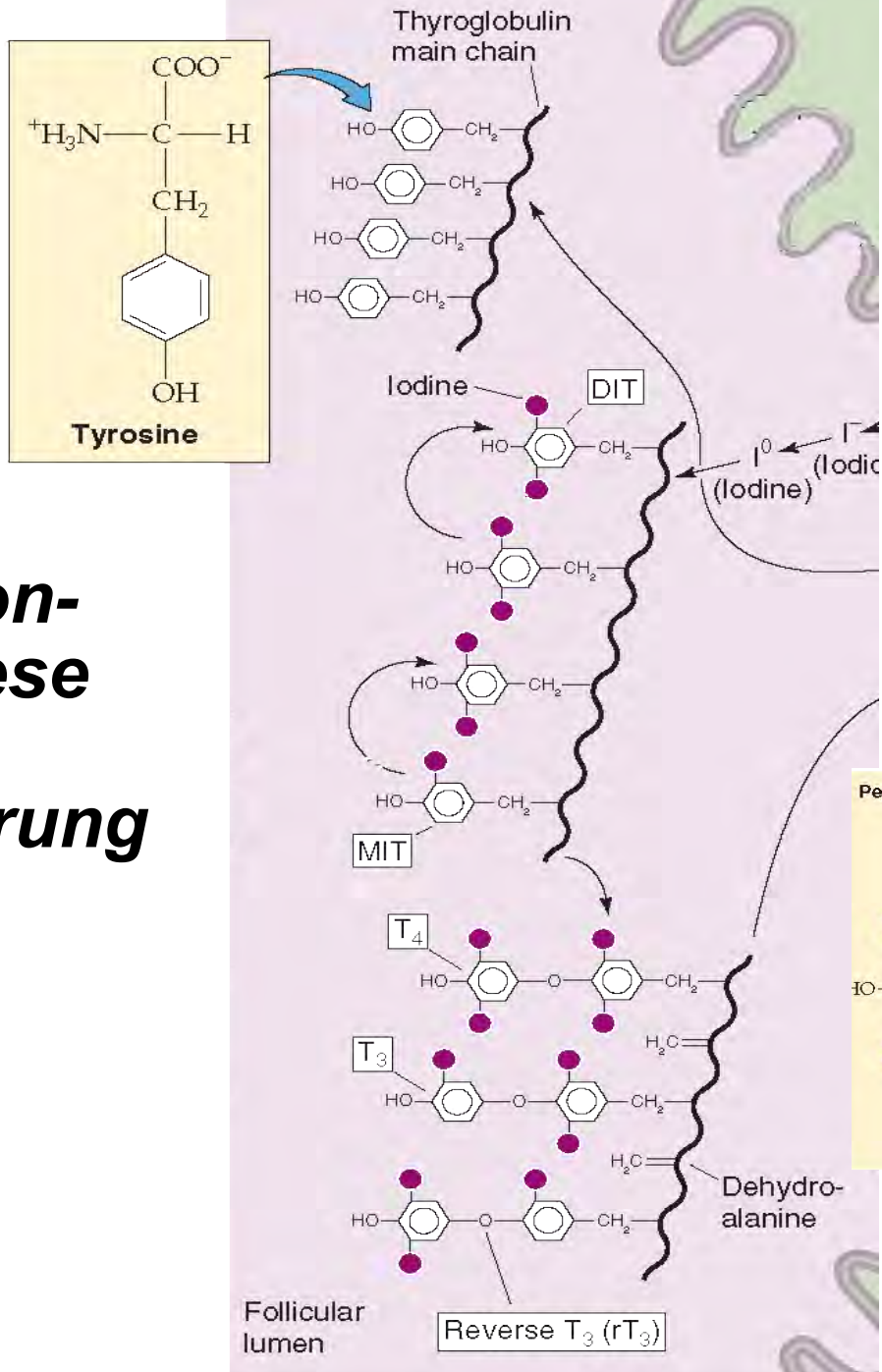
Schilddrüsenparenchym



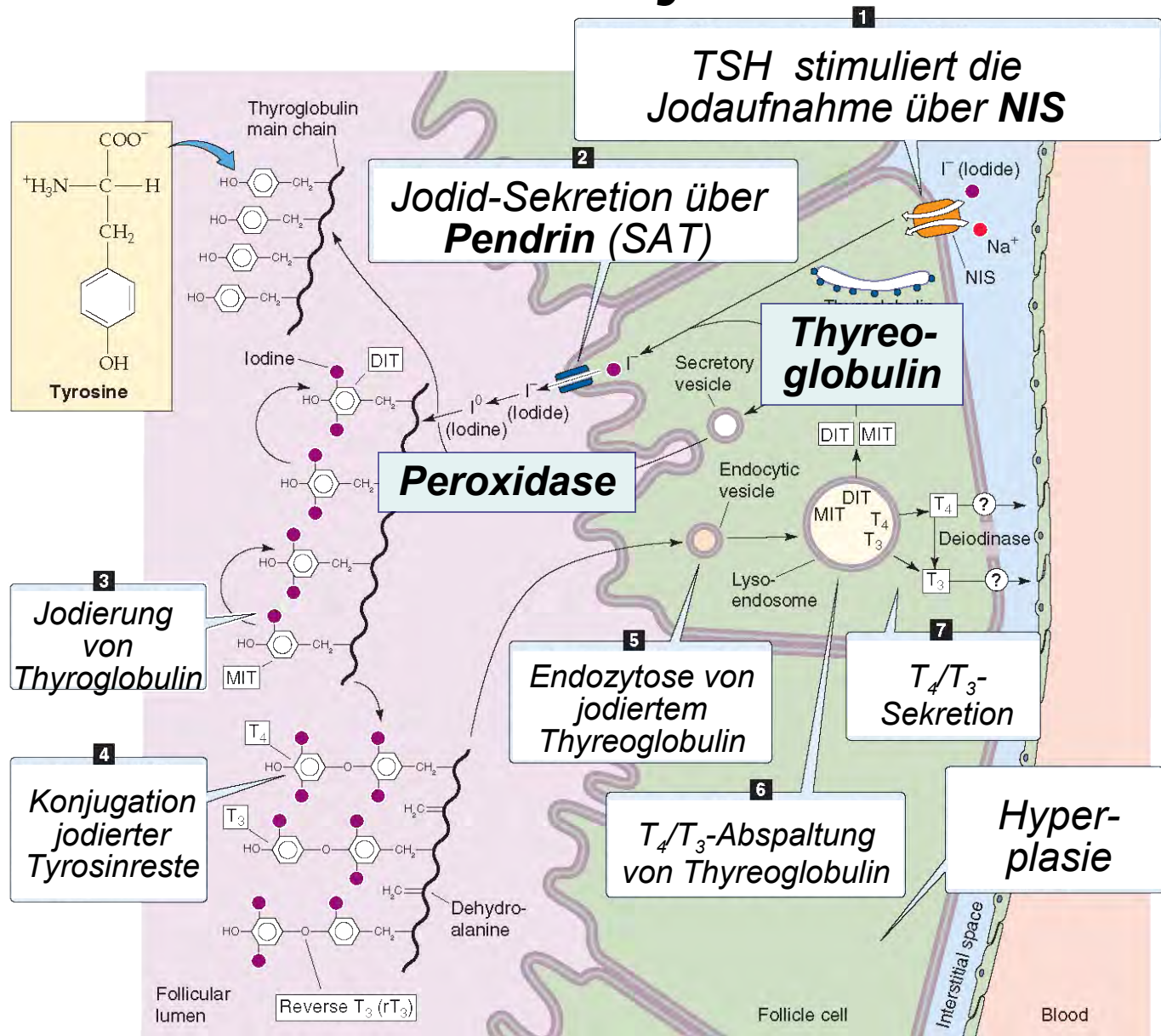
Hormon-Synthese und Speicherung



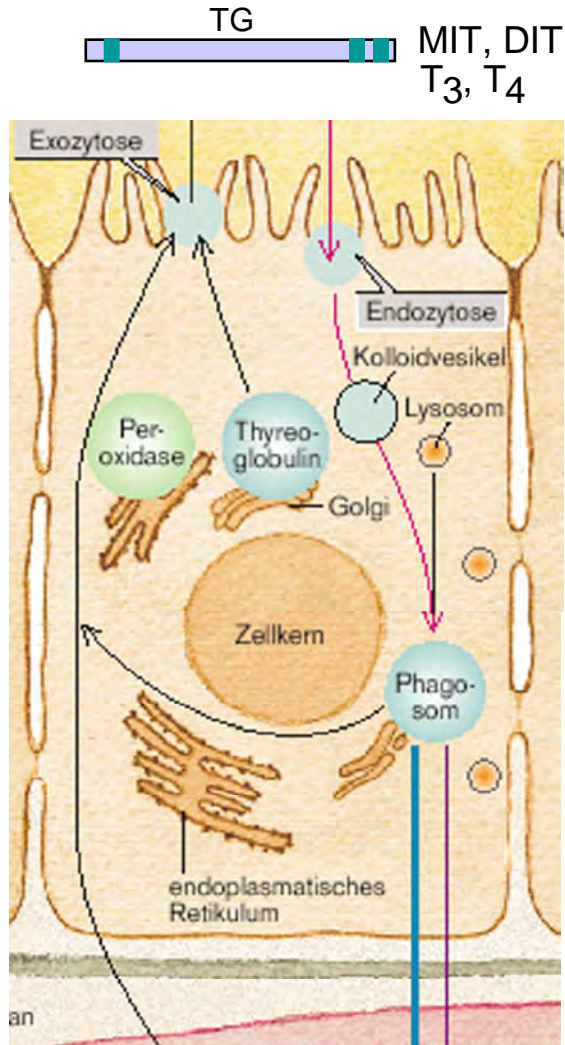
Hormon-Synthese und Speicherung



TSH-Wirkungen im Schilddrüsen-Parenchym



Sekretion von T3/T4 und Transport im Blut



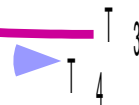
	T ₃	T ₄
Produktionsrate (nmol/d)		
aus der Schilddrüse	45–60	110
aus Gewebsmetabolismus	15%	100%
	85%	–
Konzentration im Plasma		
gesamt (nmol/l)	1,5–2,0	80–100
frei (pmol/l)	3,0	20,0
Halbwertszeit (Tage)	1	7
Clearance-Rate (MRC/d)	20–25	1,2
Bindung an		
TBG	40	70
TBPA	25	10
Albumin	35	20
(% des Hormons im Plasma)		
„biologische Aktivität“	1	0,2–0,3

Im Plasma Bindung an:

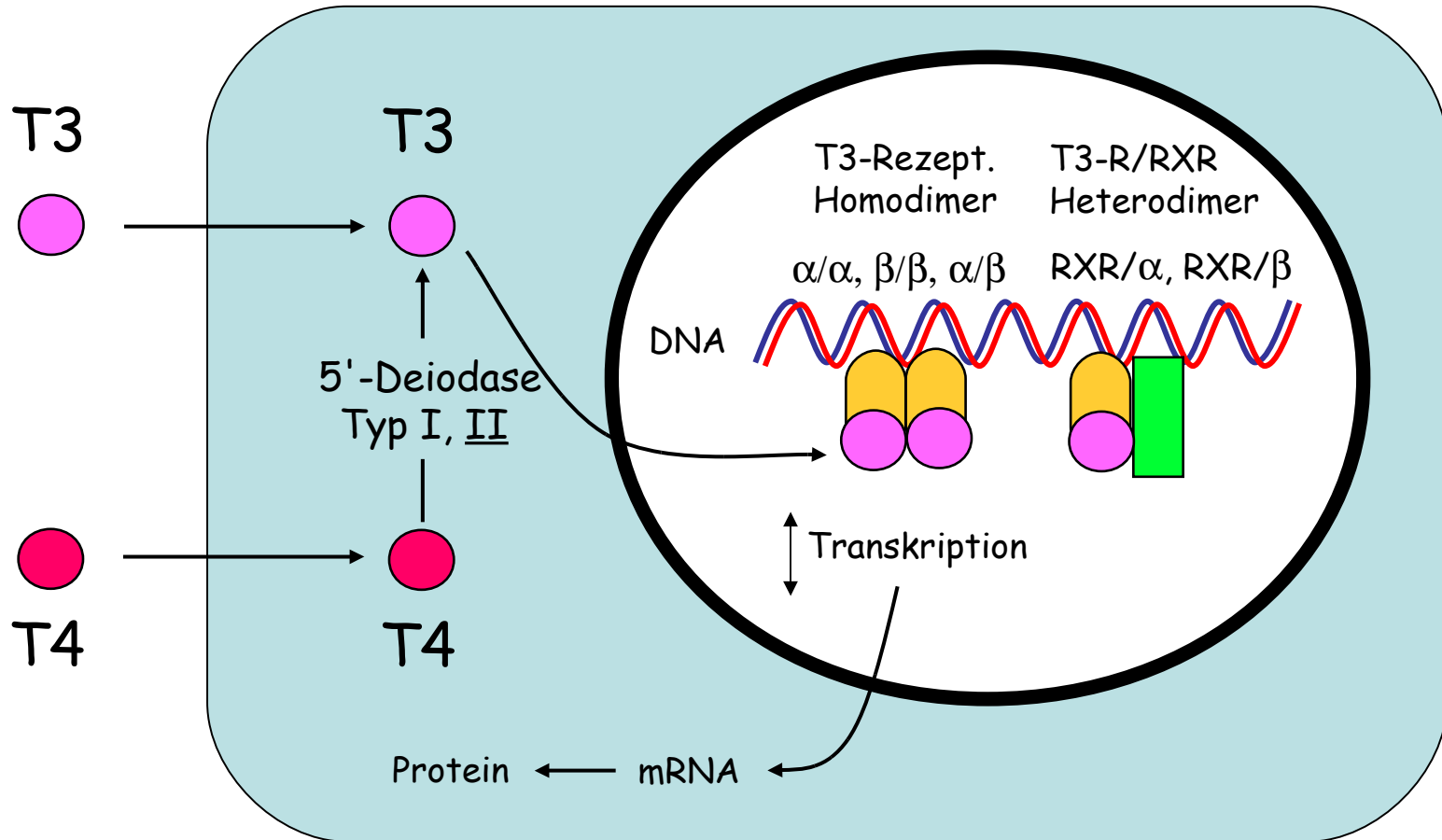
TBG (thyroxinbindendes Globulin)

TBPA (thyroxinbindendes Präalbumin, Syn.: Transthyretin)

Albumin

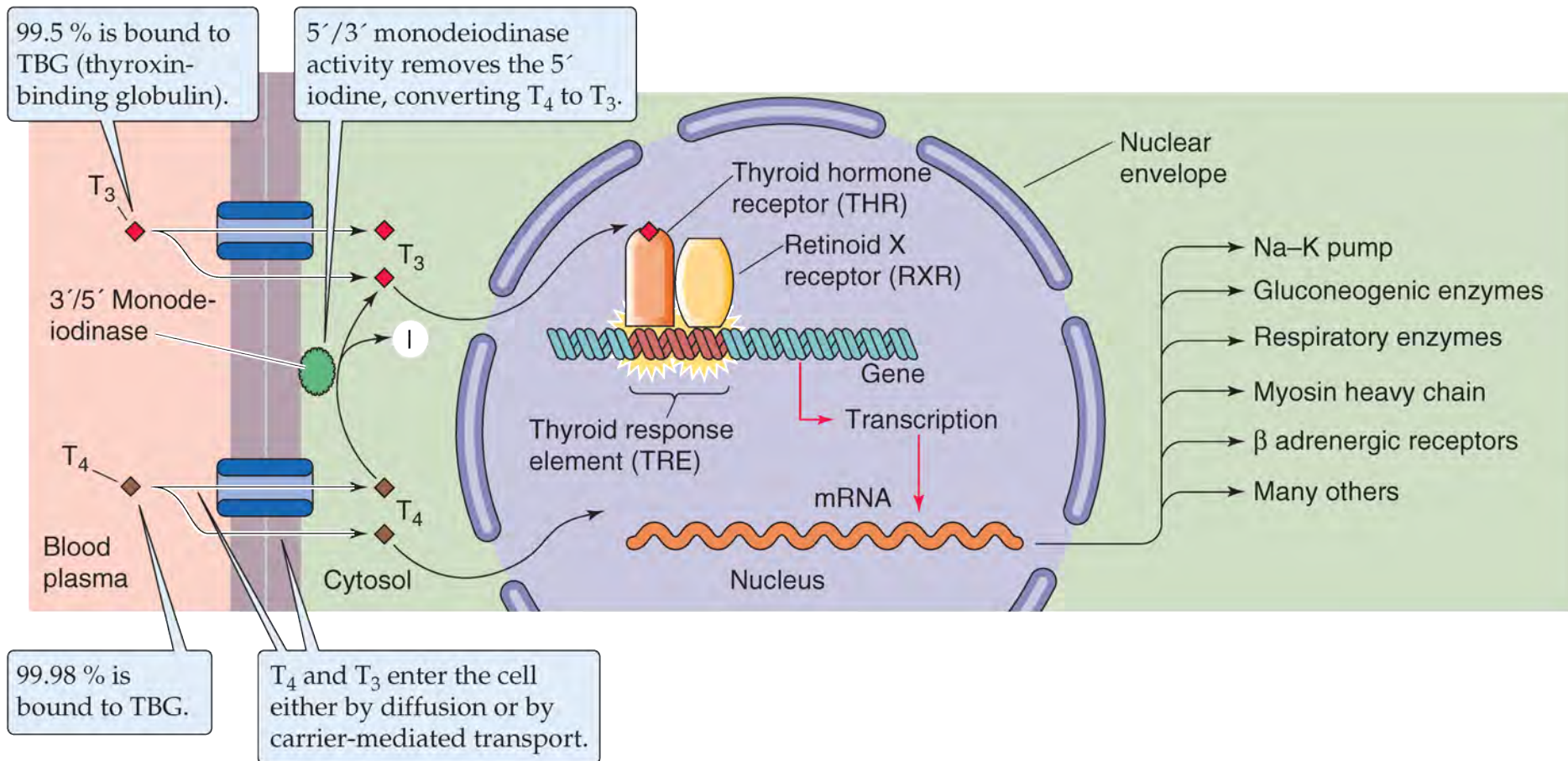


Wirkung von T3 (T4) an der Zielzelle



RXR=Retinsäure-X-Rezeptor

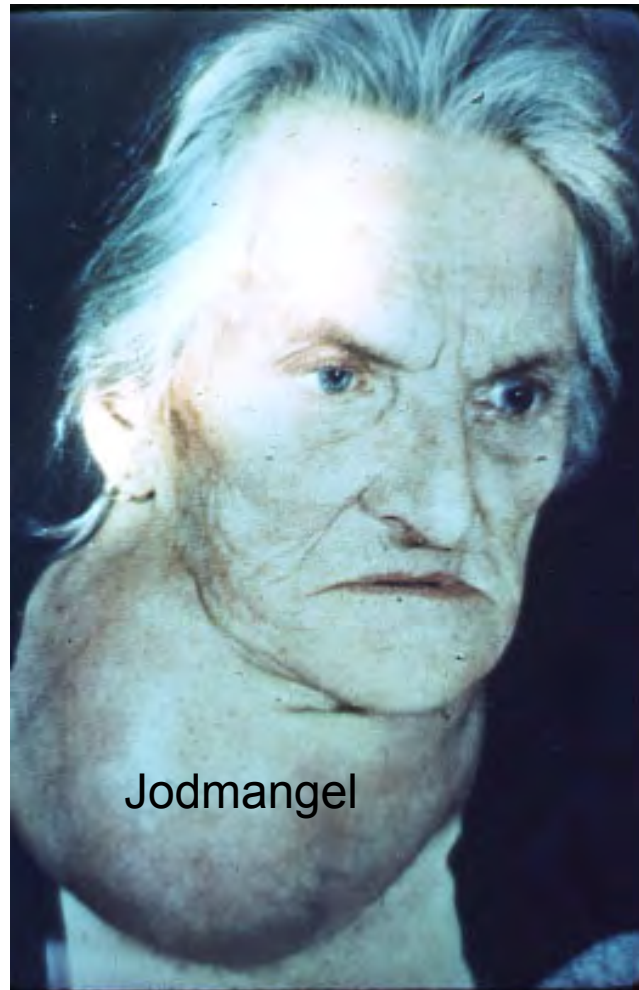
Intrazelluläre Wirkung von Trijod-Thyronin (T3)



Jodmangel-Struma: Hyperplasie aufgrund fehlender negativer Rückkopplung durch T_3/T_4

keine
negative
Rückkopplung

$T_4, T_3 \downarrow$



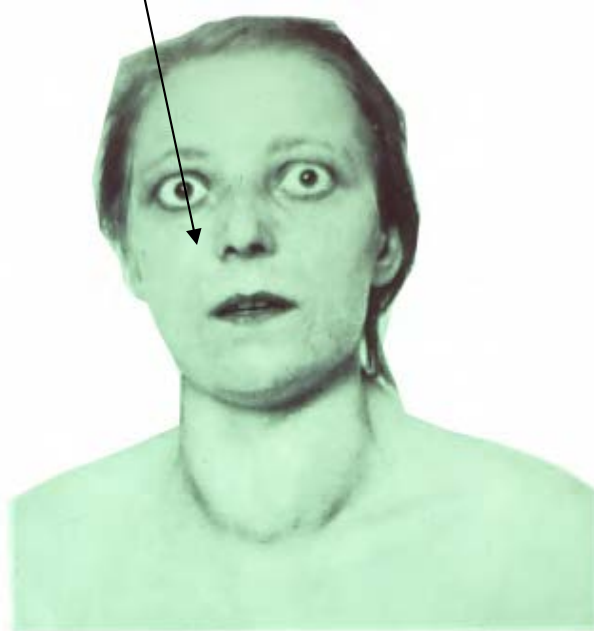
Jodmangel

TSH ↑

Hyperplasie

Schilddrüsen- Funktionsstörungen:

Exophthalmus



Hyperthyreose

TSH ↓

Myxödem



Hypothyreose

TSH ↑

Exkurs: Myxödem (Myx-, gr. μύξα: Schleim, Schleimhaut)

1) prätibiales Myxödem:
bei **M. Basedow**
mit: Exophthalmus, Struma
Infiltrative Dermopathie
(autoimmun, entzündlich)

2) Generalisiertes Myxödem:
(auch prätibial!)
bei **Hypothyreose**
Einlagerung von Hyaluronsäure
und Chondroitinsulfat
(verlangsamter Abbau)



Abb. 8.14 Zirkumskriptes prätibiales Myxödem bei Basedow-Hyperthyreose



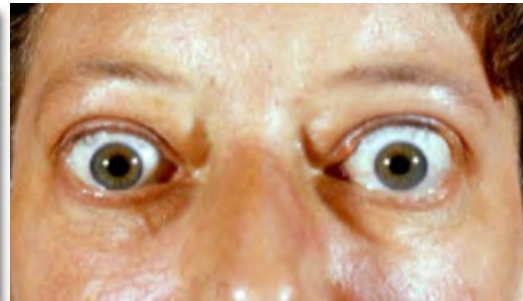
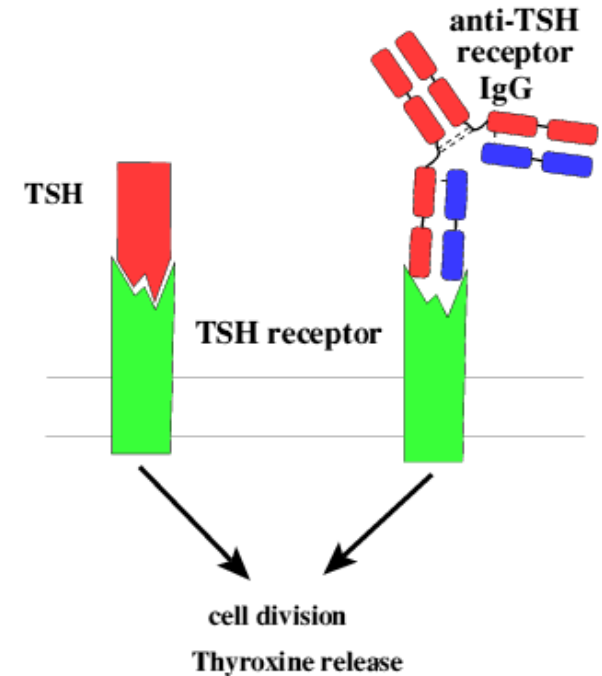
Abb. 8.15 Typisches Gesicht bei Myxödem. 61-jähriger Mann



Abb. 26.3 Prätibiales Myxödem bei Patientin mit Hypothyreose

Morbus Basedow (engl. Graves' disease)

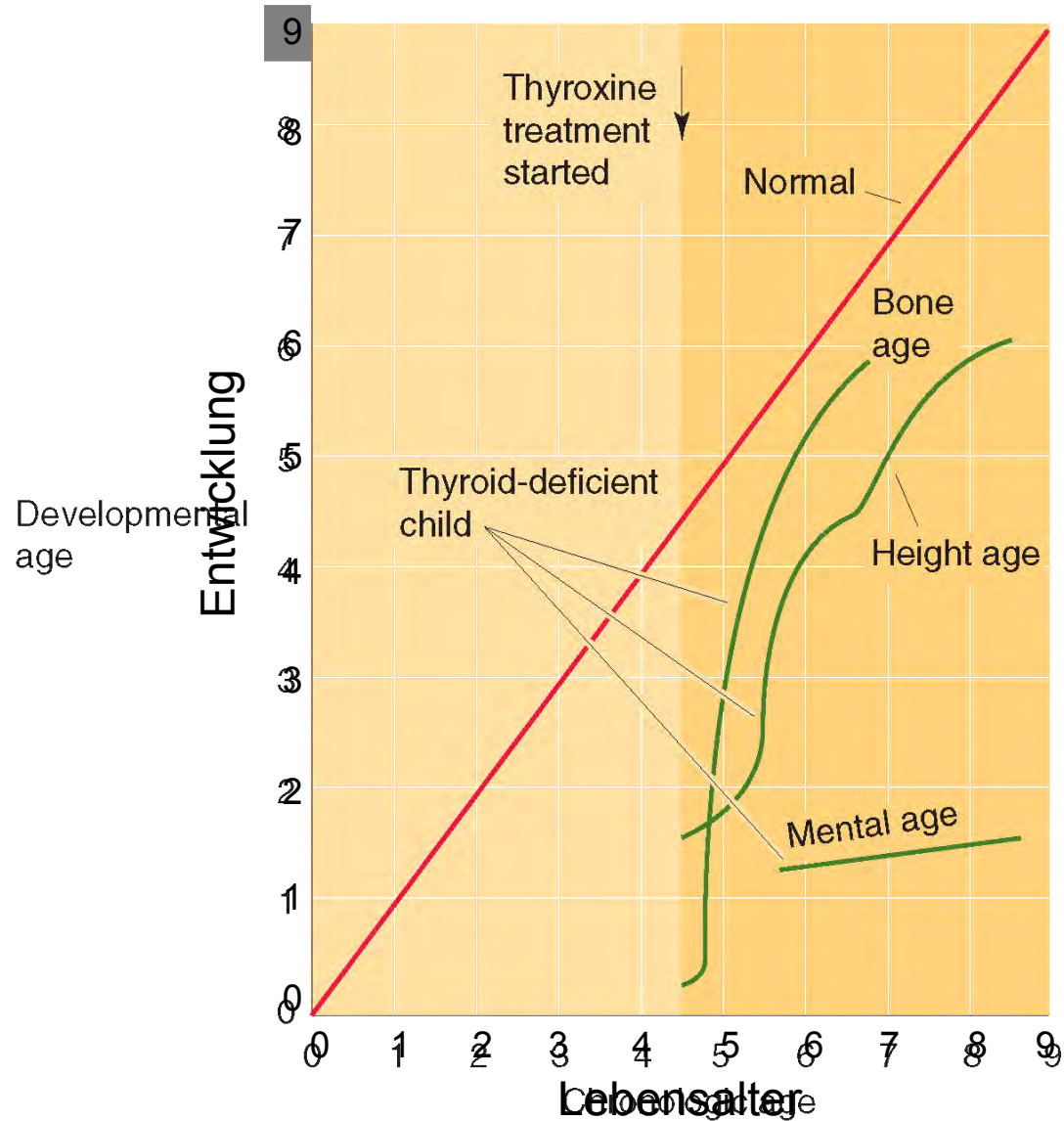
- Autoimmunerkrankung
 - aktivierende AK gegen TSH-Rezeptor
 - T3, T4 hoch, TSH niedrig
 - Gewichtsverlust, Müdigkeit
 - Tachykardie
 - Diffuse Struma
 - Exophthalmus
 - prätibiales Myxödem
- Merseburger Trias



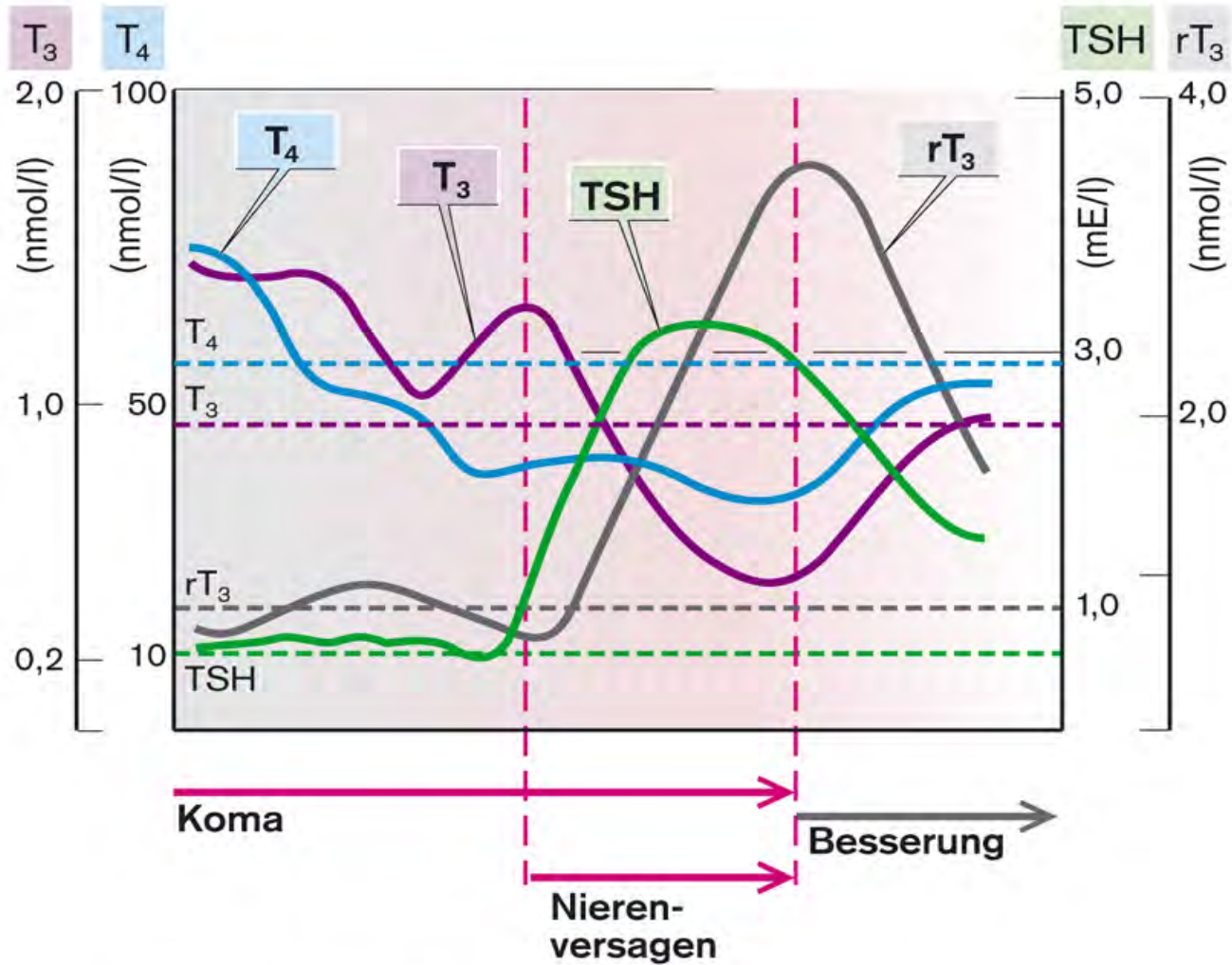
Schilddrüsenaplasie: “Kretinismus”



Schilddrüsenaplasie: Hormon-Substitution

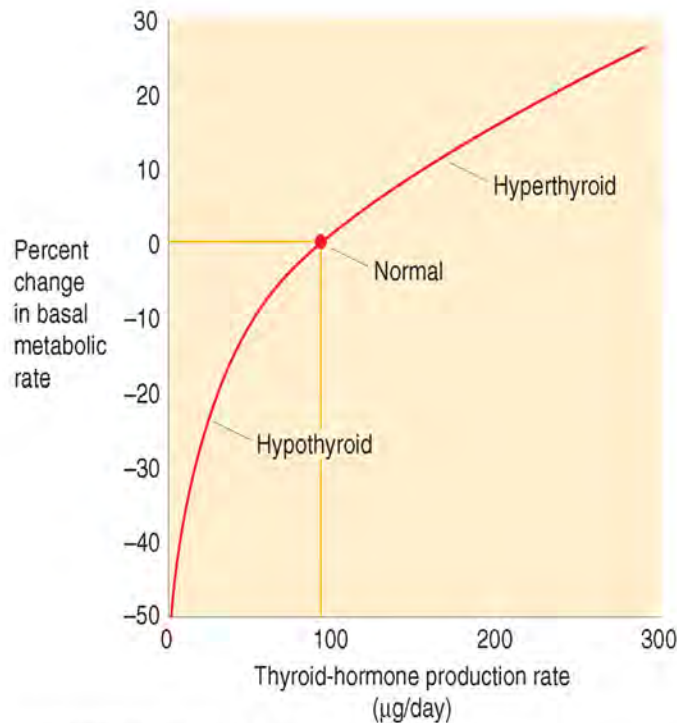


Sick Euthyroid Syndrome

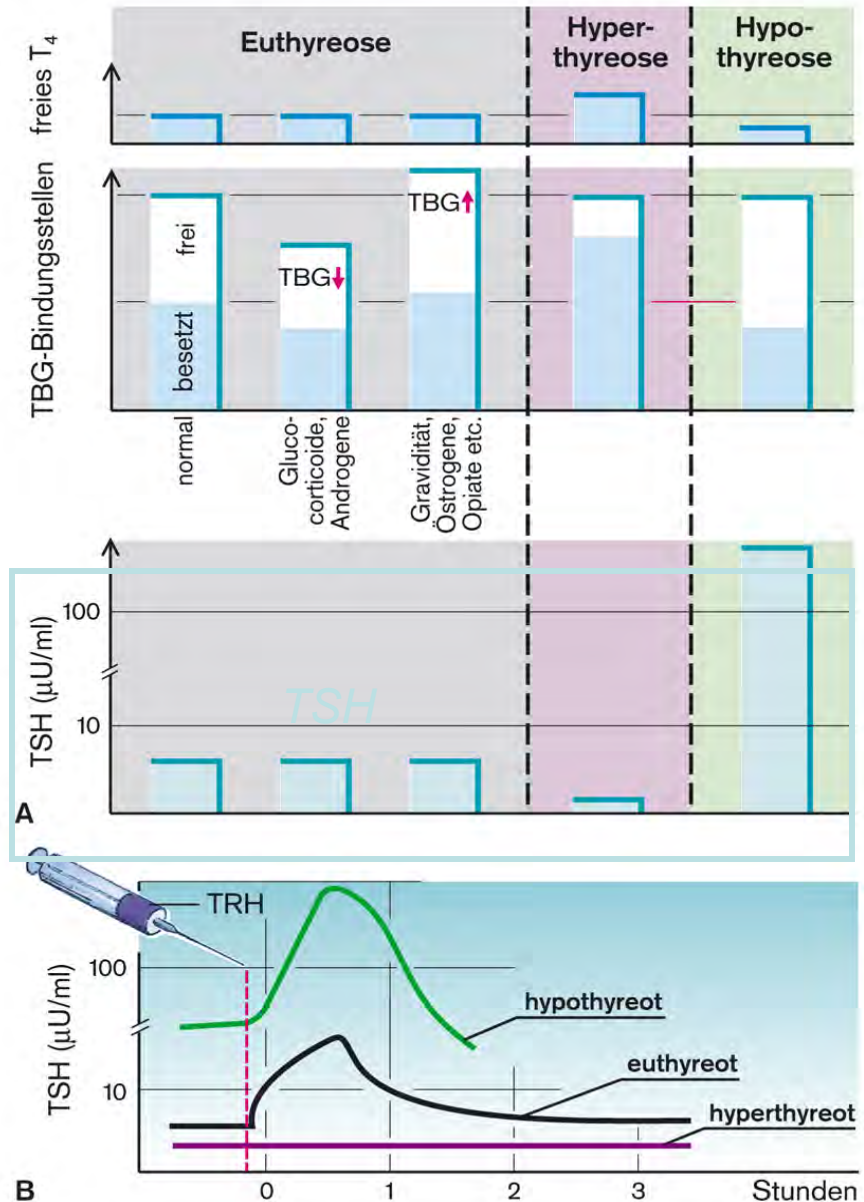


Schilddrüsenfunktion: GU und Blutwerte

Grundumsatz

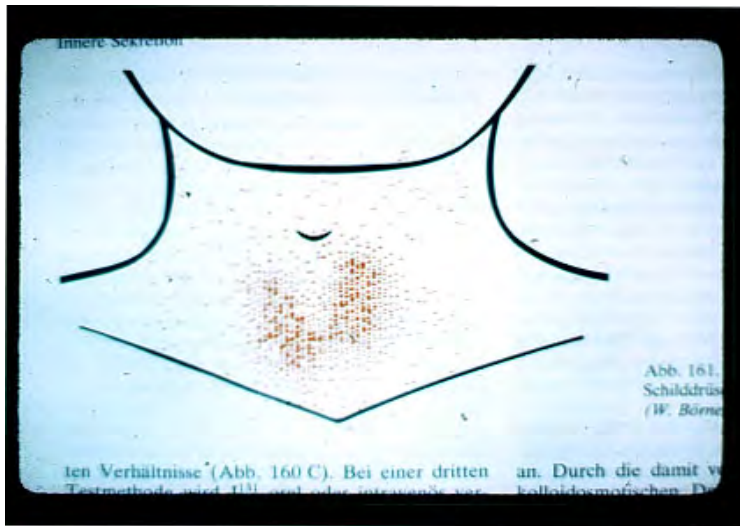


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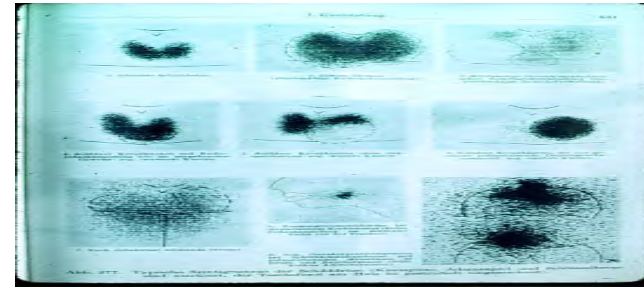
Schilddrüsenfunktion: Szintigramm

**normales
Szintigramm**



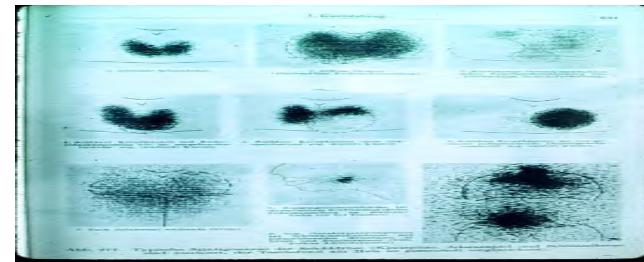
**speichernde
Struma**

**degenerierte
Struma**

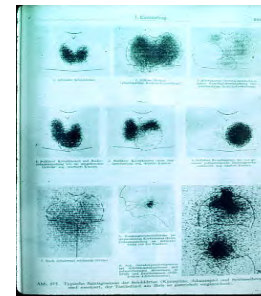


**„kalter“
Knoten**

**„heißer“
Knoten**



**retrosternale
Struma**

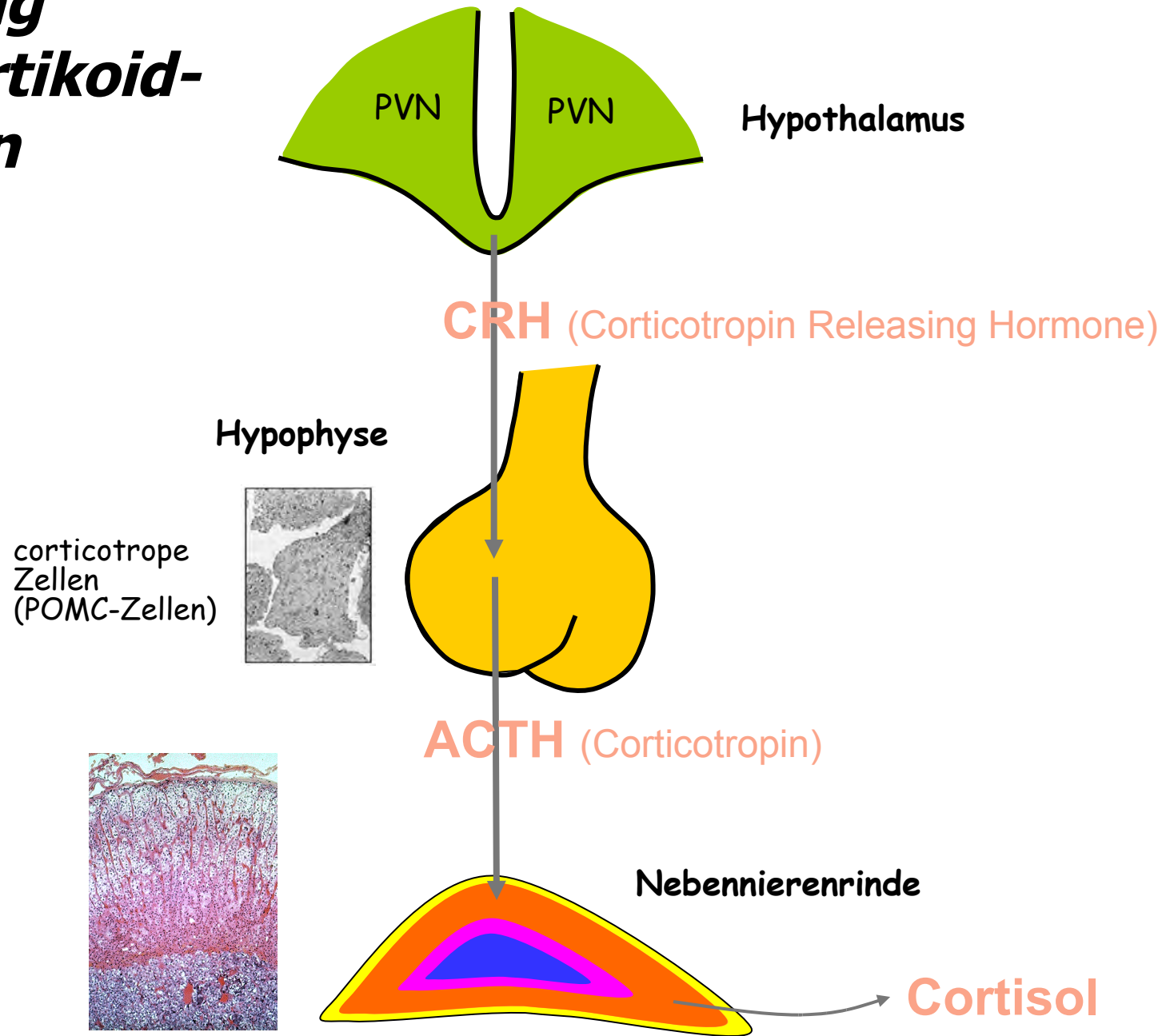


Endokrinologie II

Endokrines System – Hormone: speziell

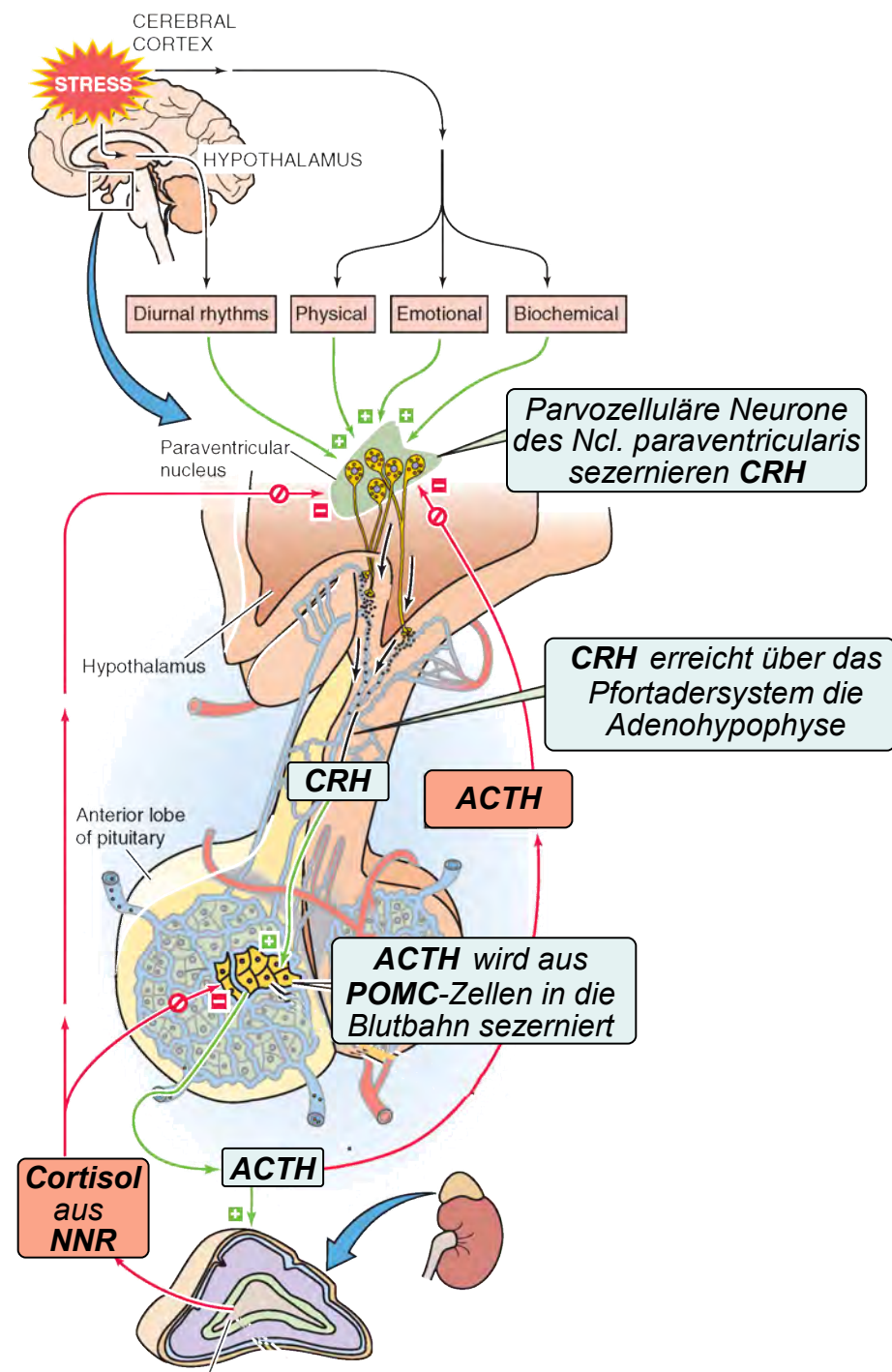
- ***Hypophyse***
(Adeno-/Neurohyphyse)
- ***Schilddrüse***
- ***Nebennierenrinde***
- ***Gonaden***
- ***Nebenschilddrüse***
- *Somatotropes Hormon*
- *Glandotrope Hormone*
- *ADH, Oxytocin*
- *Schilddrüsenhormone*
(T_3/T_4)
- *Mineralocorticoide*
- *Glucocorticoide*
- *Sexualhormone*
- *Sexualhormone*
- *Parathormon*
- *(Calcitonin, Calcitriol)*

Steuerung Glukokortikoid- Sekretion

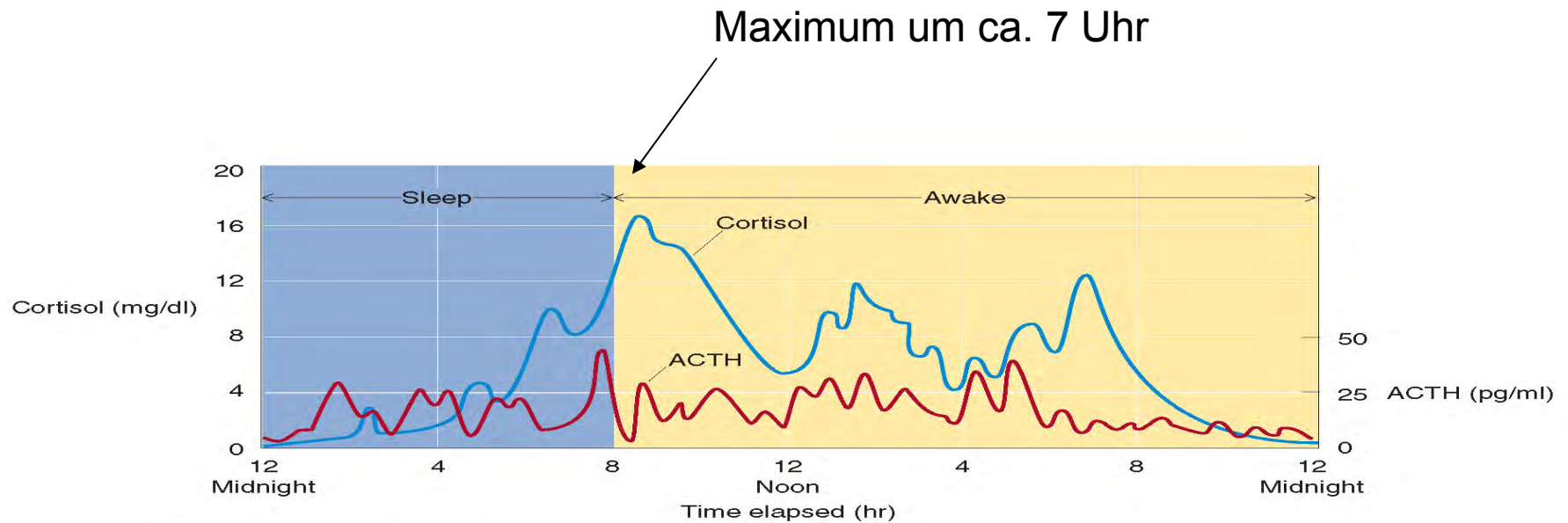


Hypothalamus- Hypophysen- NNR- Achse:

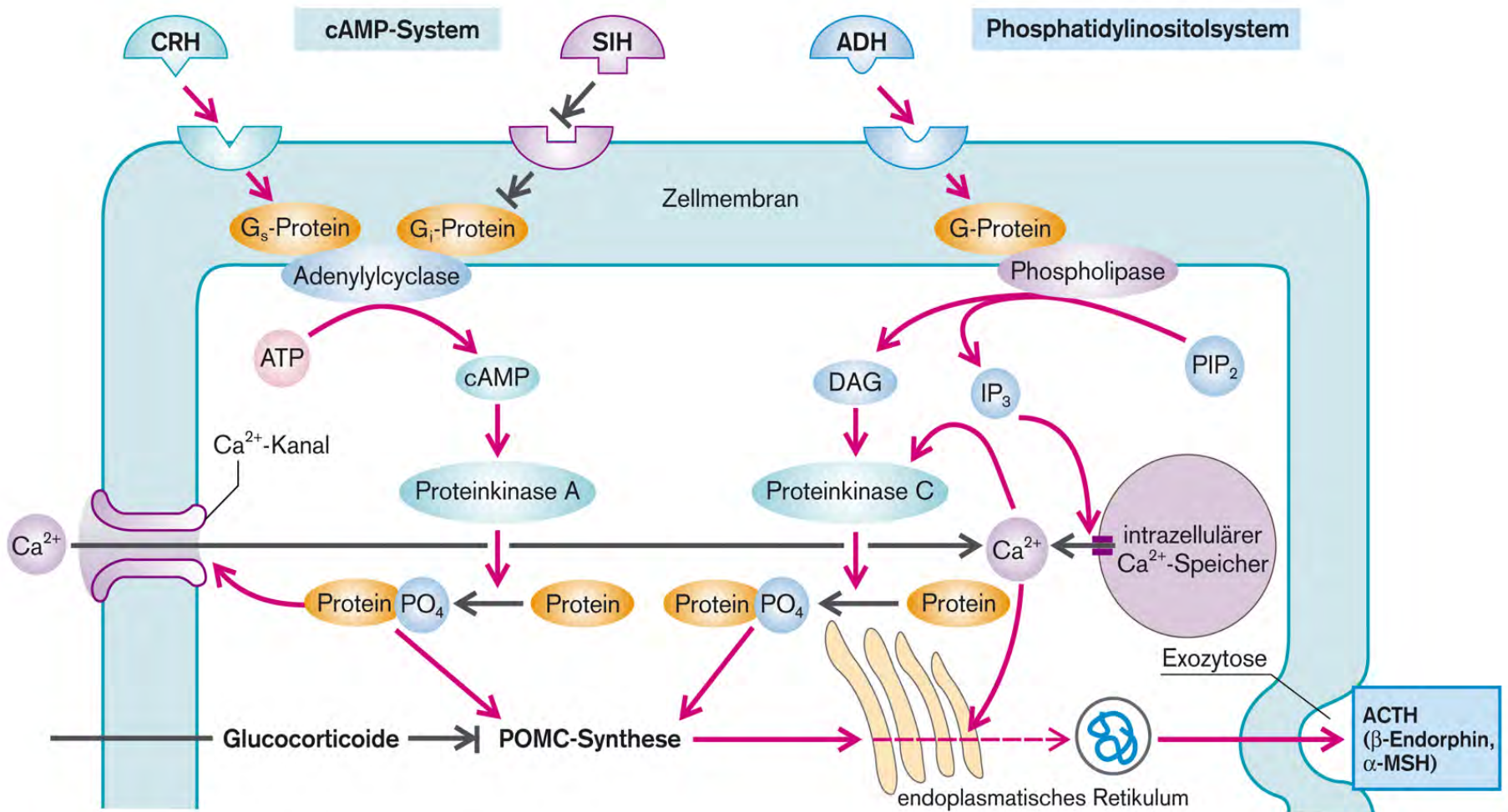
Glucocorticoide- Sekretion



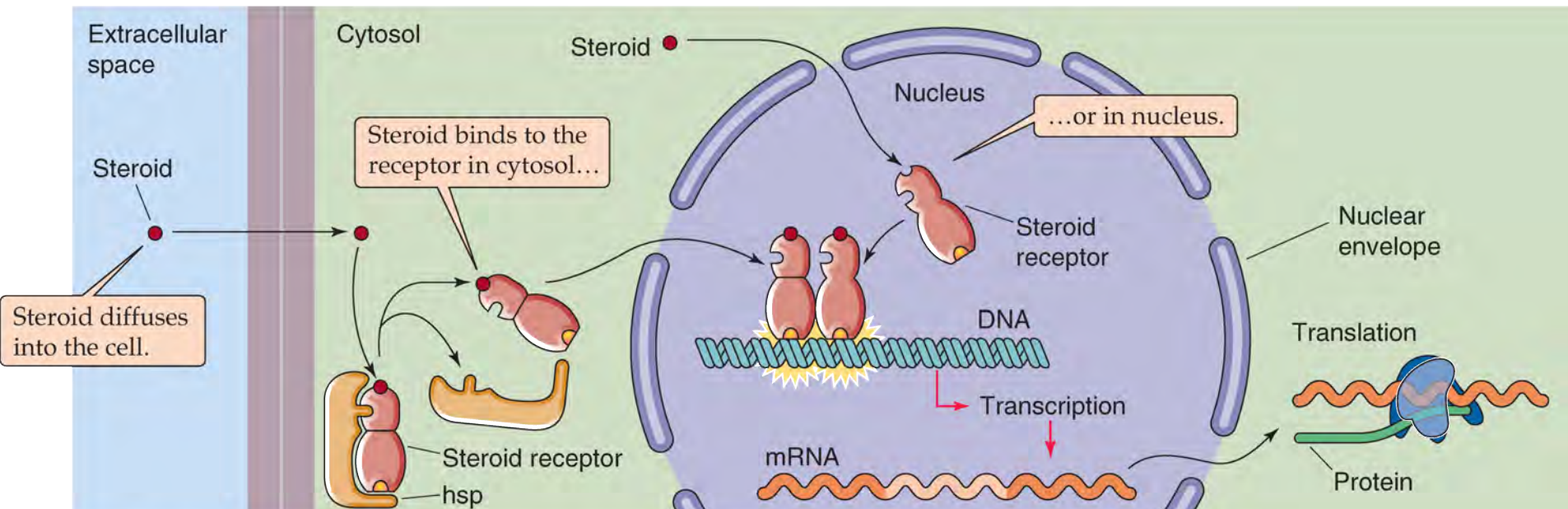
Rhythmische Ausschüttung: ACTH und Cortisol



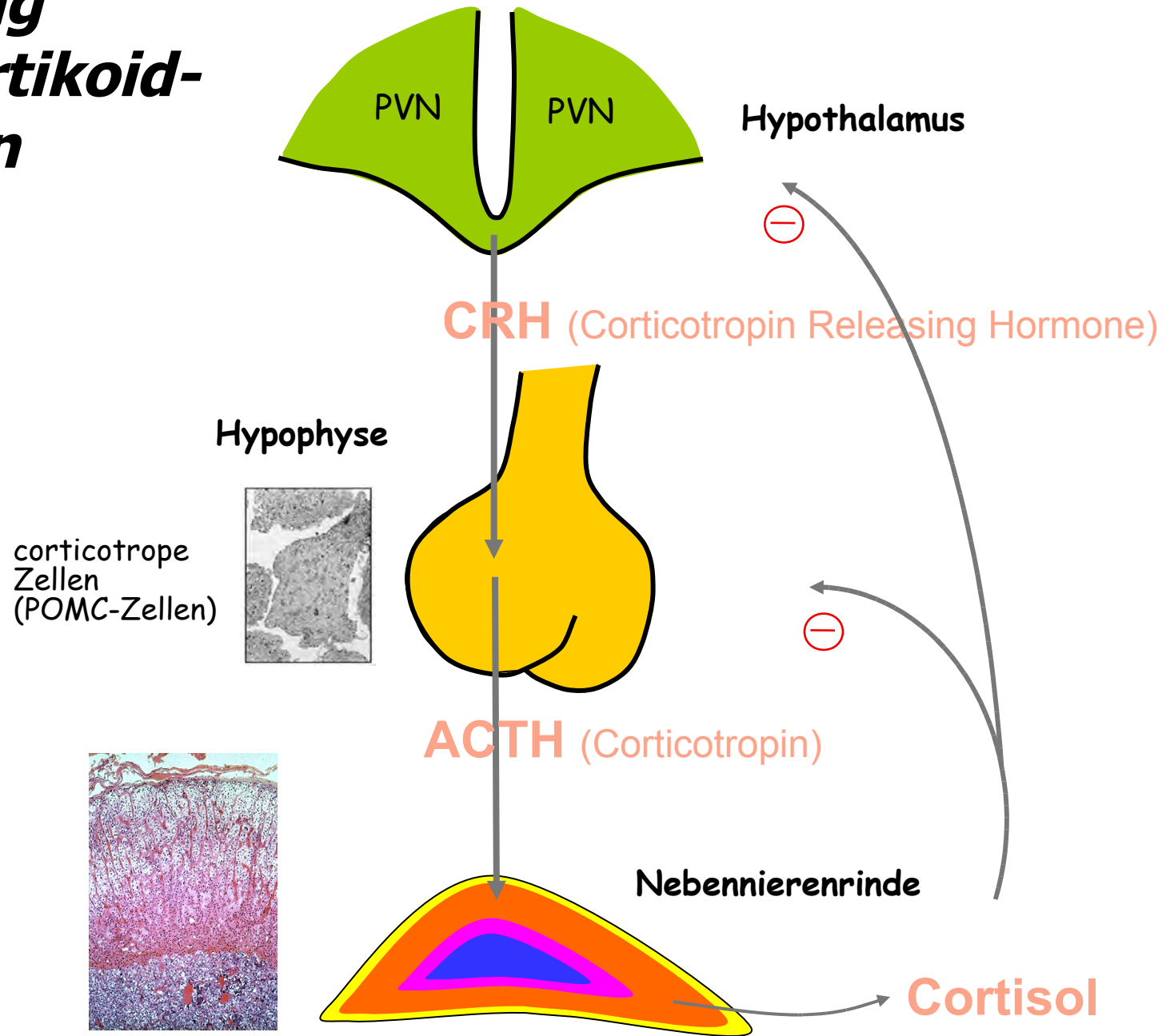
ACTH-Freisetzung aus POMC-Zellen (Pro-opiomelanocortin)



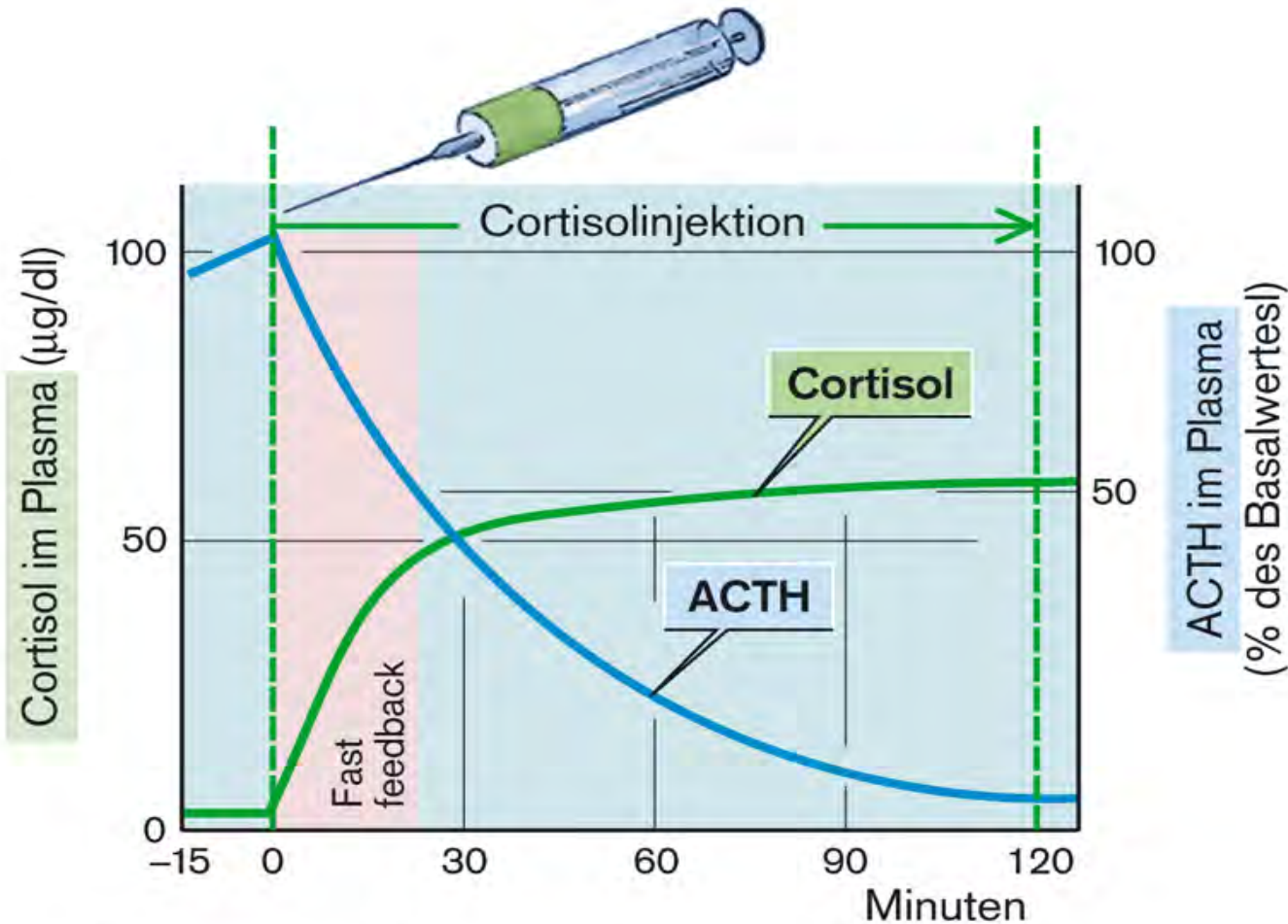
Signal-Transduktion der Steroid-Wirkungen



Steuerung Glukokortikoid- Sekretion



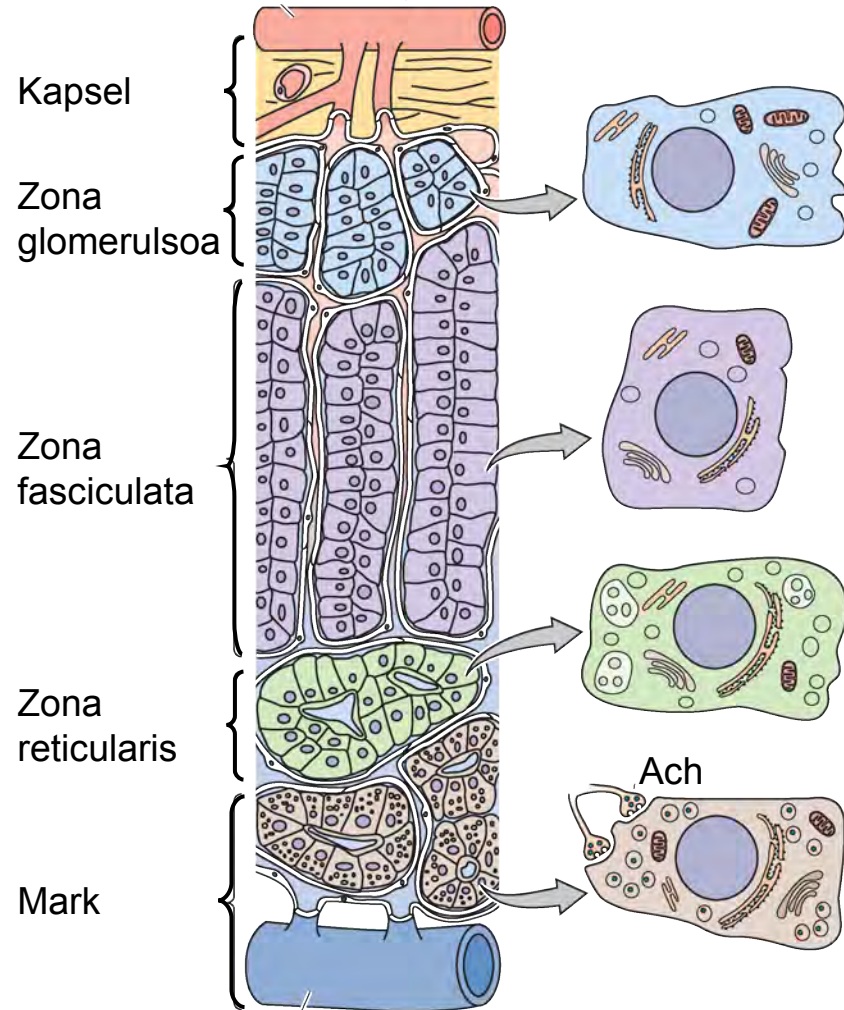
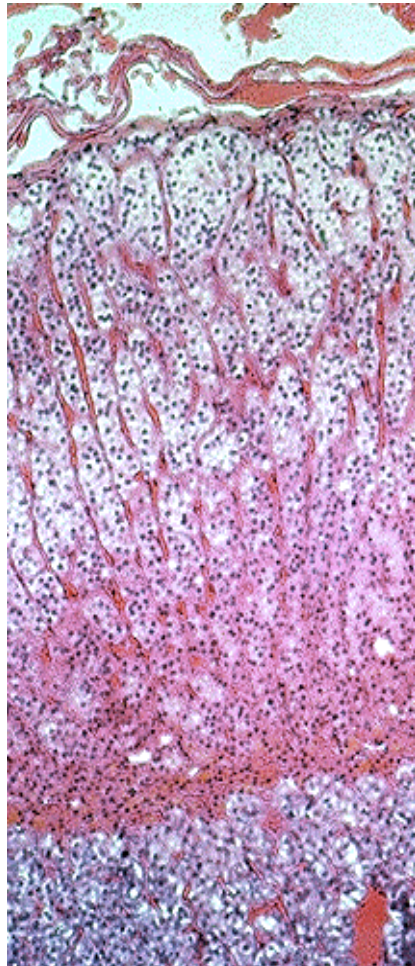
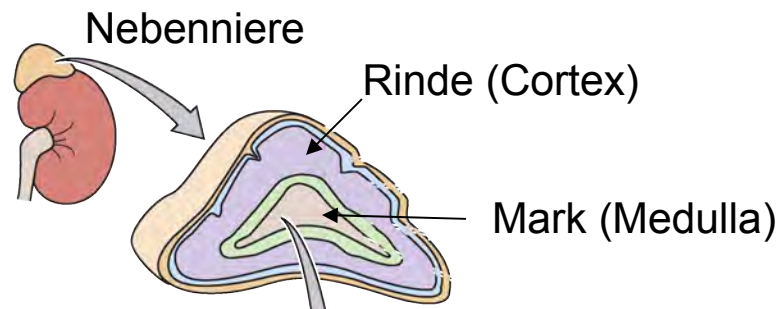
Feedback-Regulation der ACTH-Ausschüttung durch Cortisol



Georg Thieme Verlag, Stuttgart

Klinke, Pape, Silbernagl: Physiologie, 5. Auflage 2005

Nebenniere: Aufbau und Hormone



Hormone

Mineralocorticoide
(z. B. Aldosteron)

Glucocorticoide
(z. B. Cortisol)

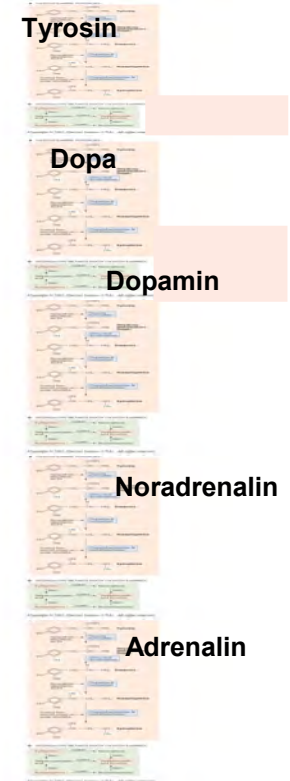
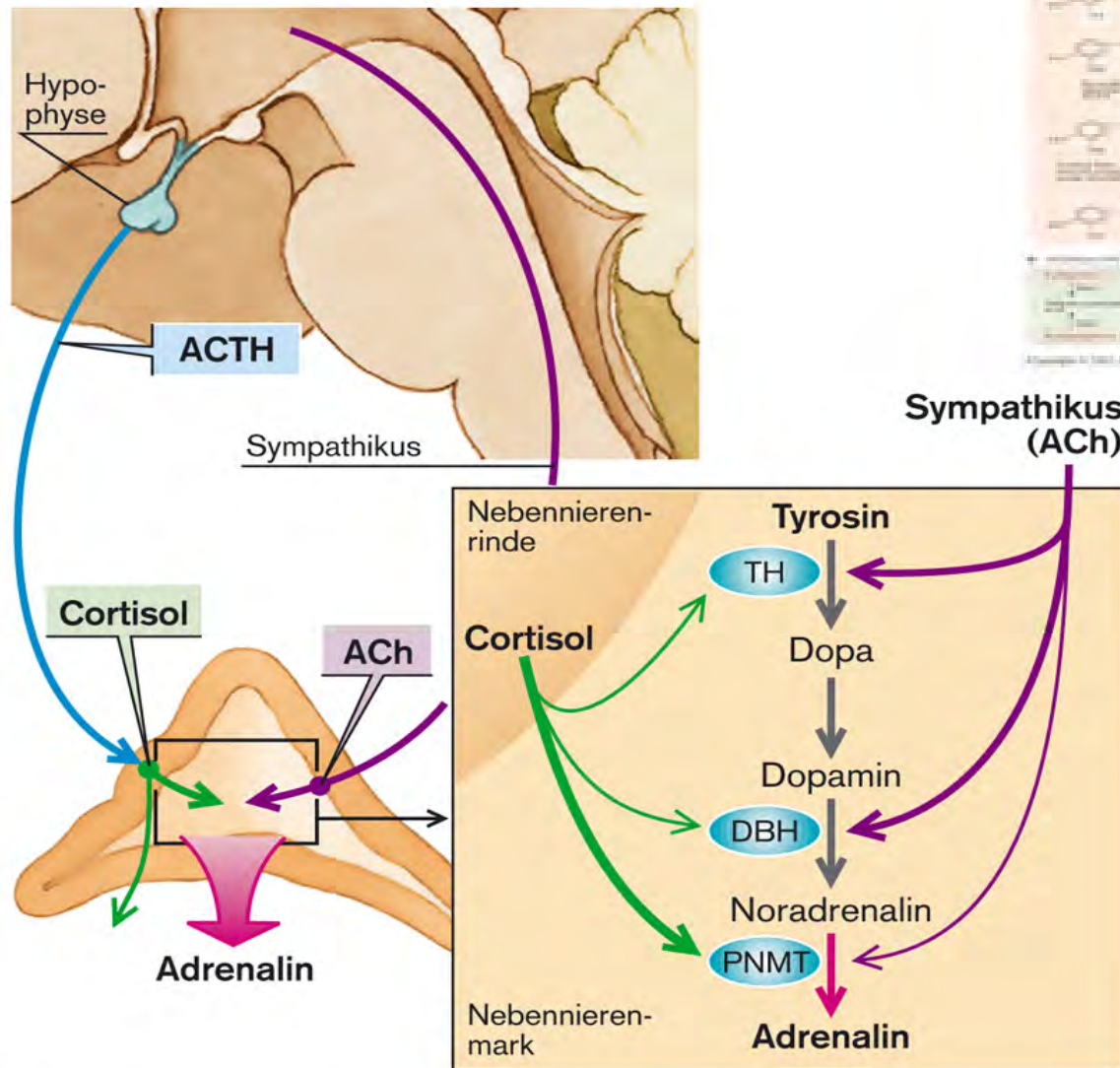
Androgene
(z. B. DHEA)

Catecholamine
(80% Adrenalin)

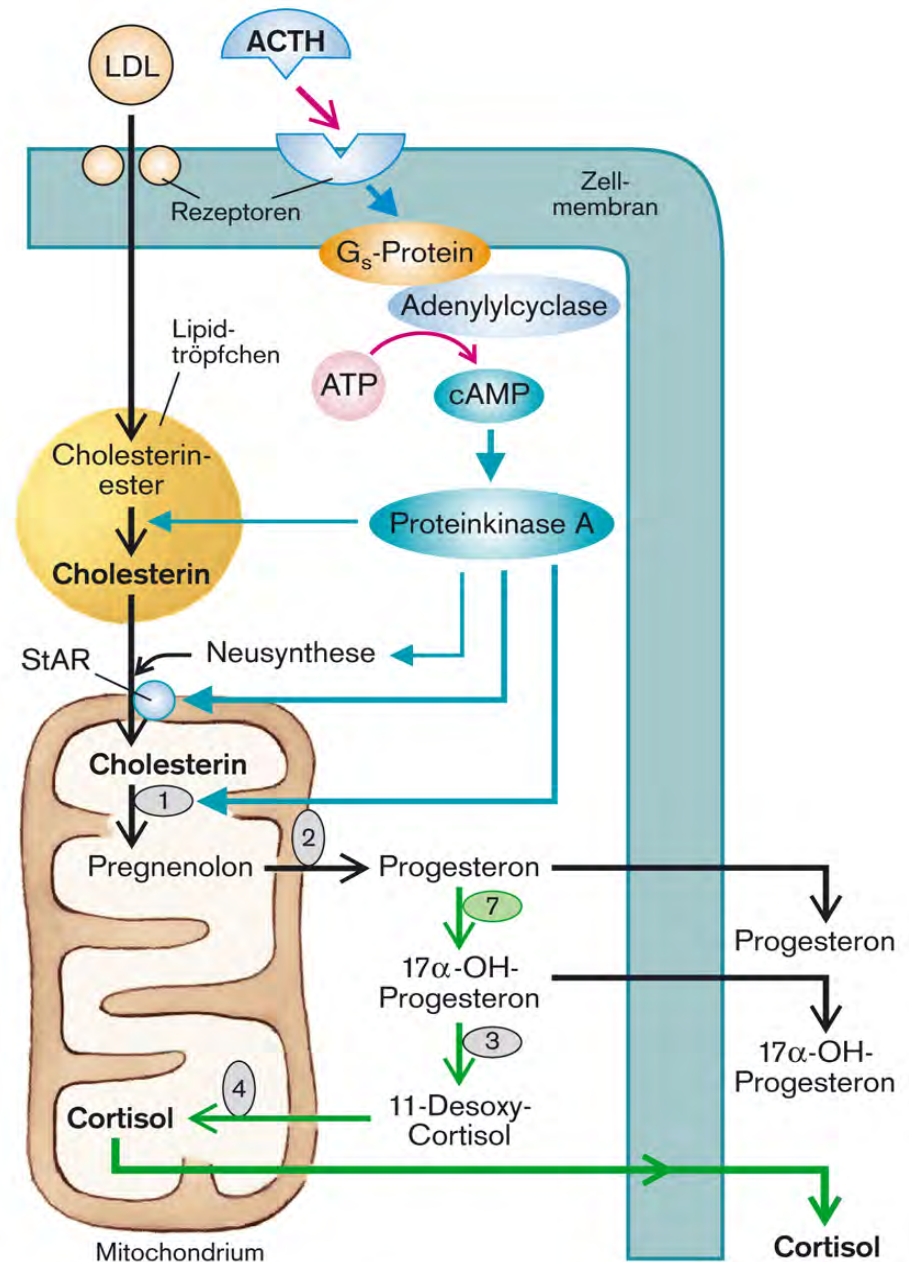
Permissiver
Effekt

Nebennierenmark:

Permissiver Effekt der (

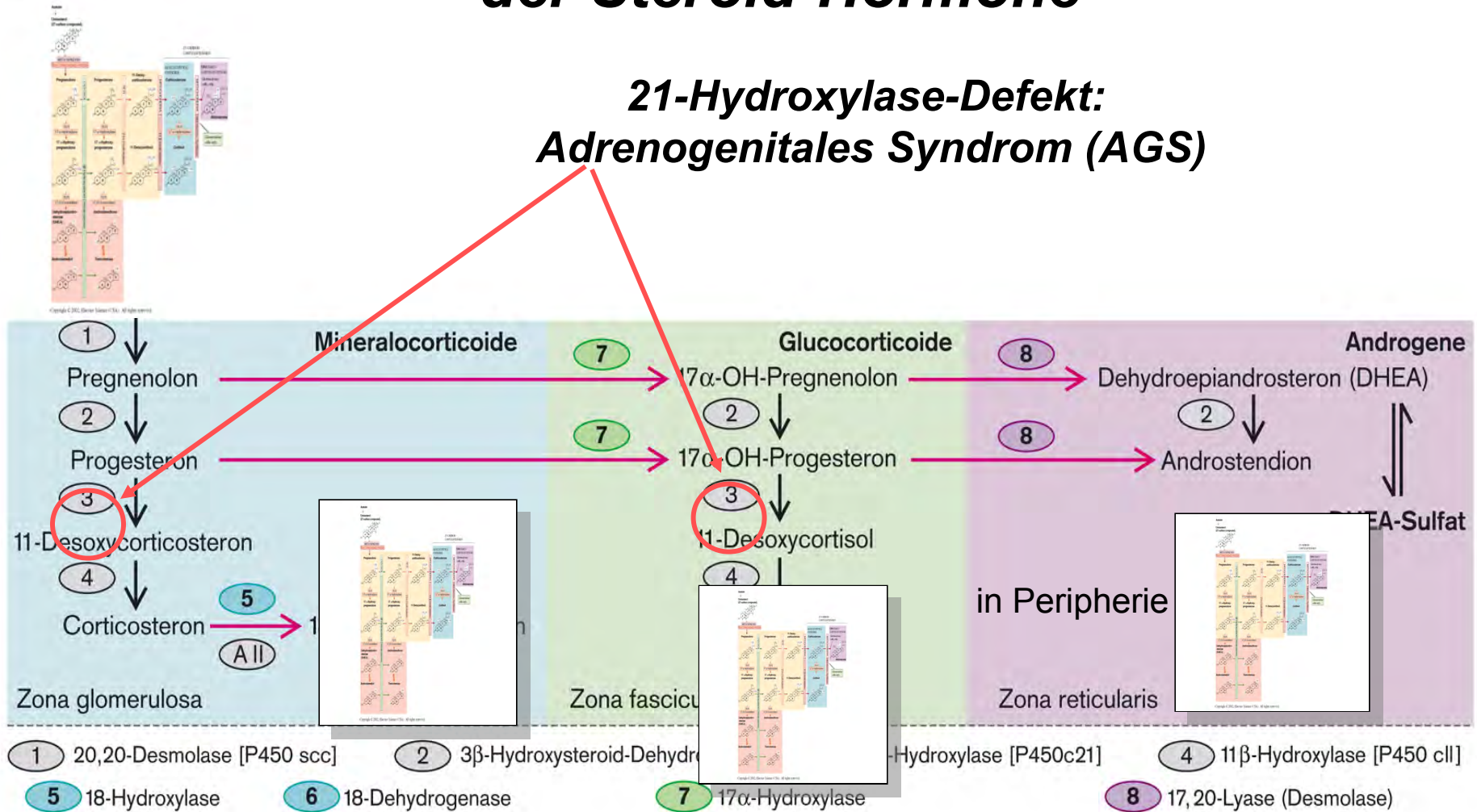


Nebennieren- Rinde: ACTH-Wirkung

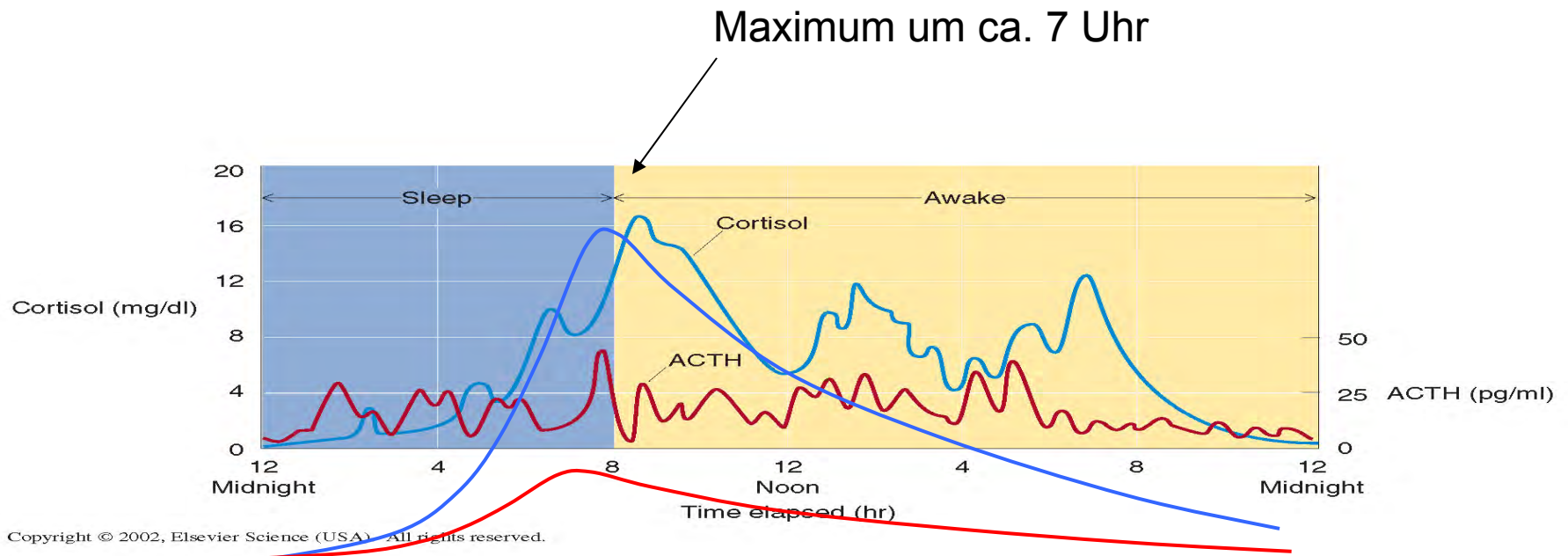


Synthese der Steroid-Hormone

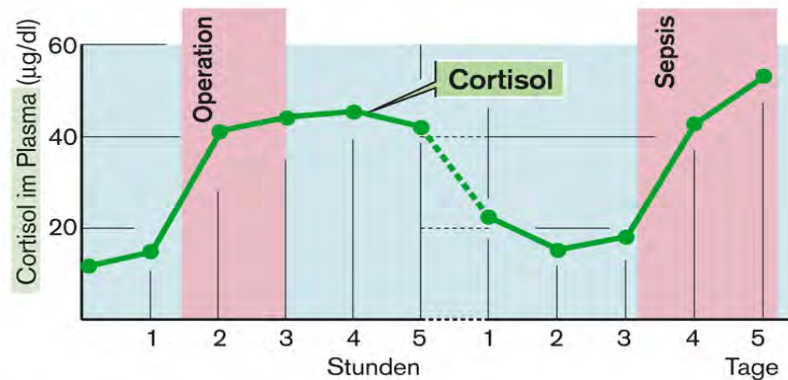
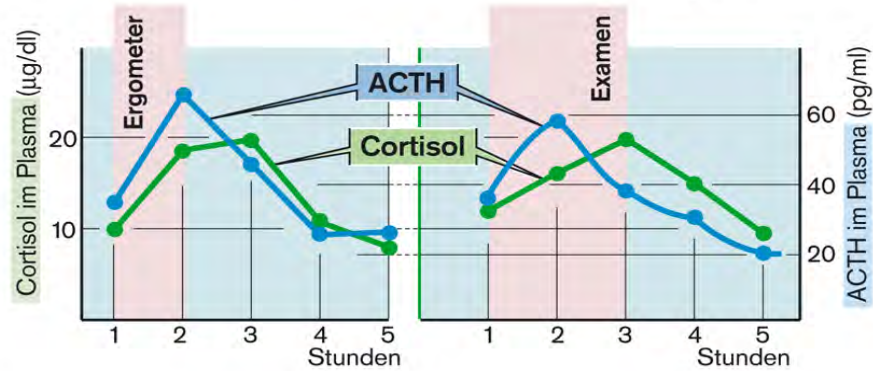
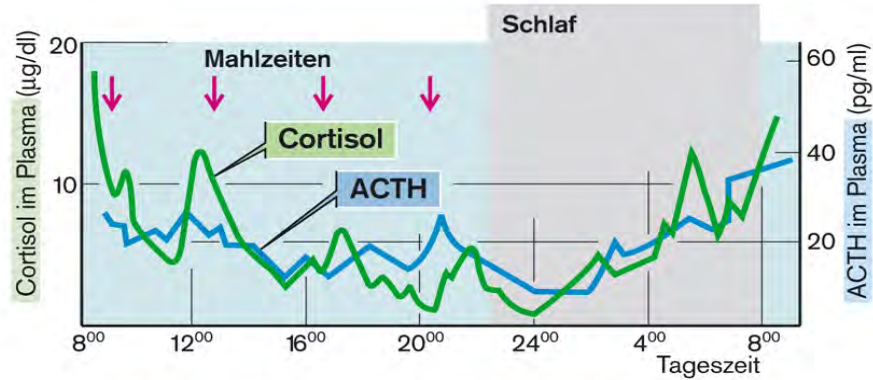
21-Hydroxylase-Defekt: Adrenogenitales Syndrom (AGS)



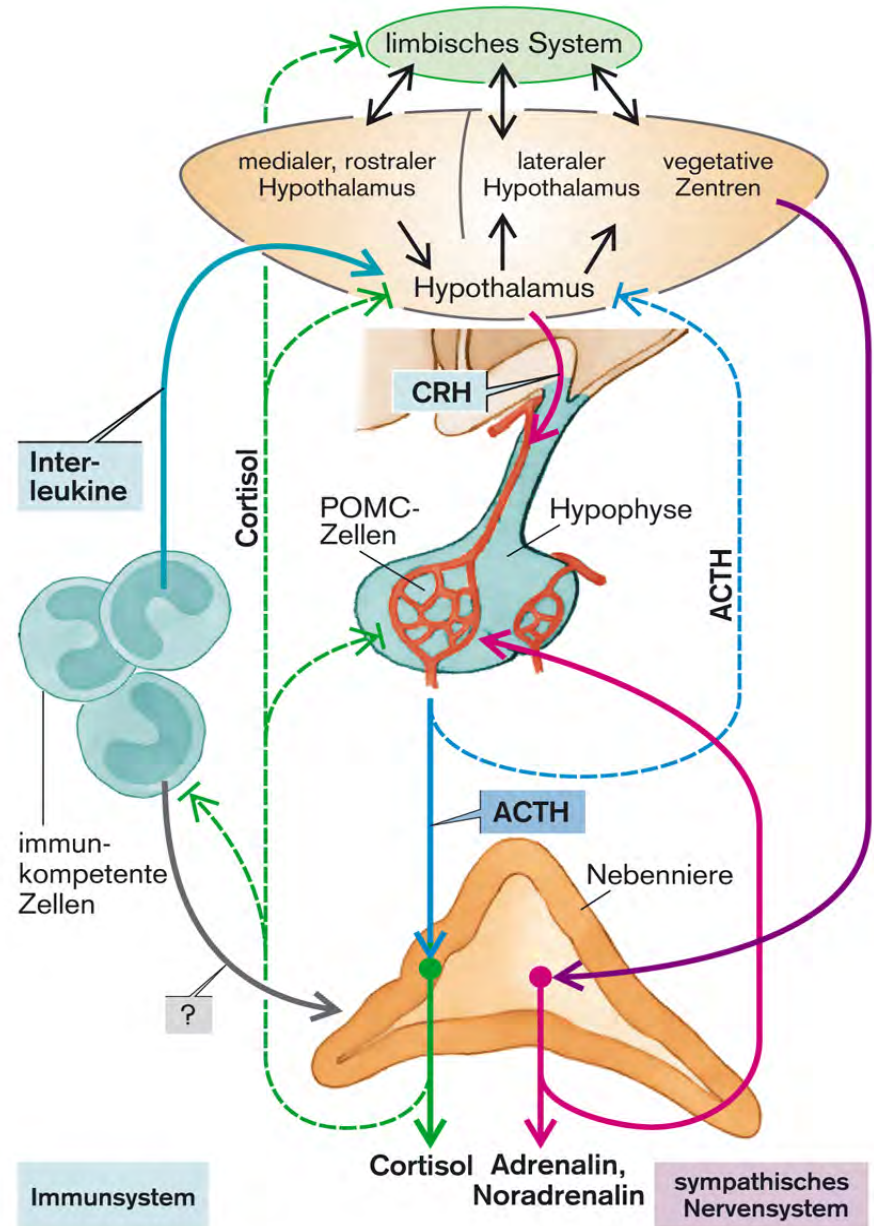
Rhythmische Ausschüttung: ACTH und Cortisol



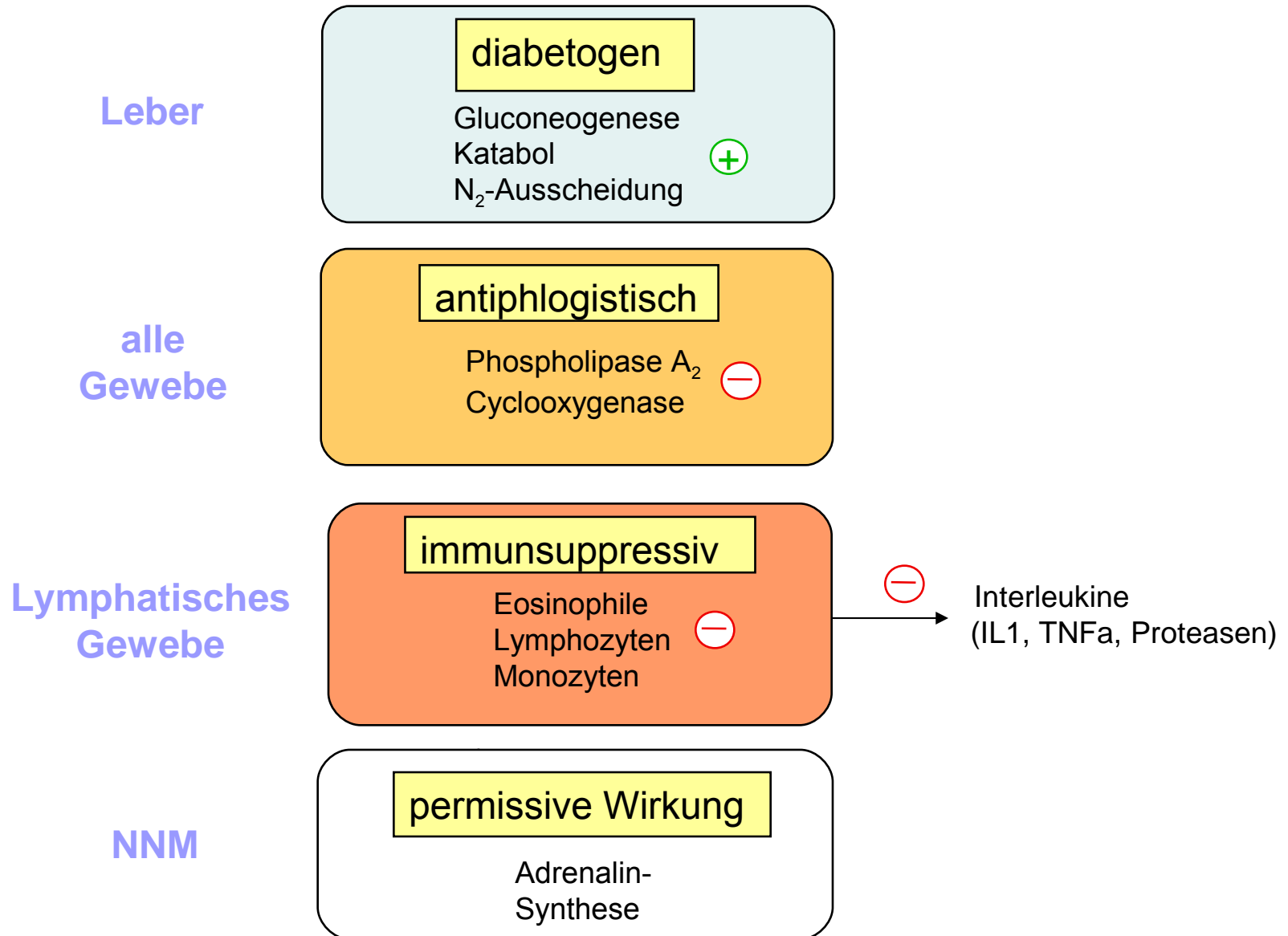
ACTH- und Cortisolsekretion:



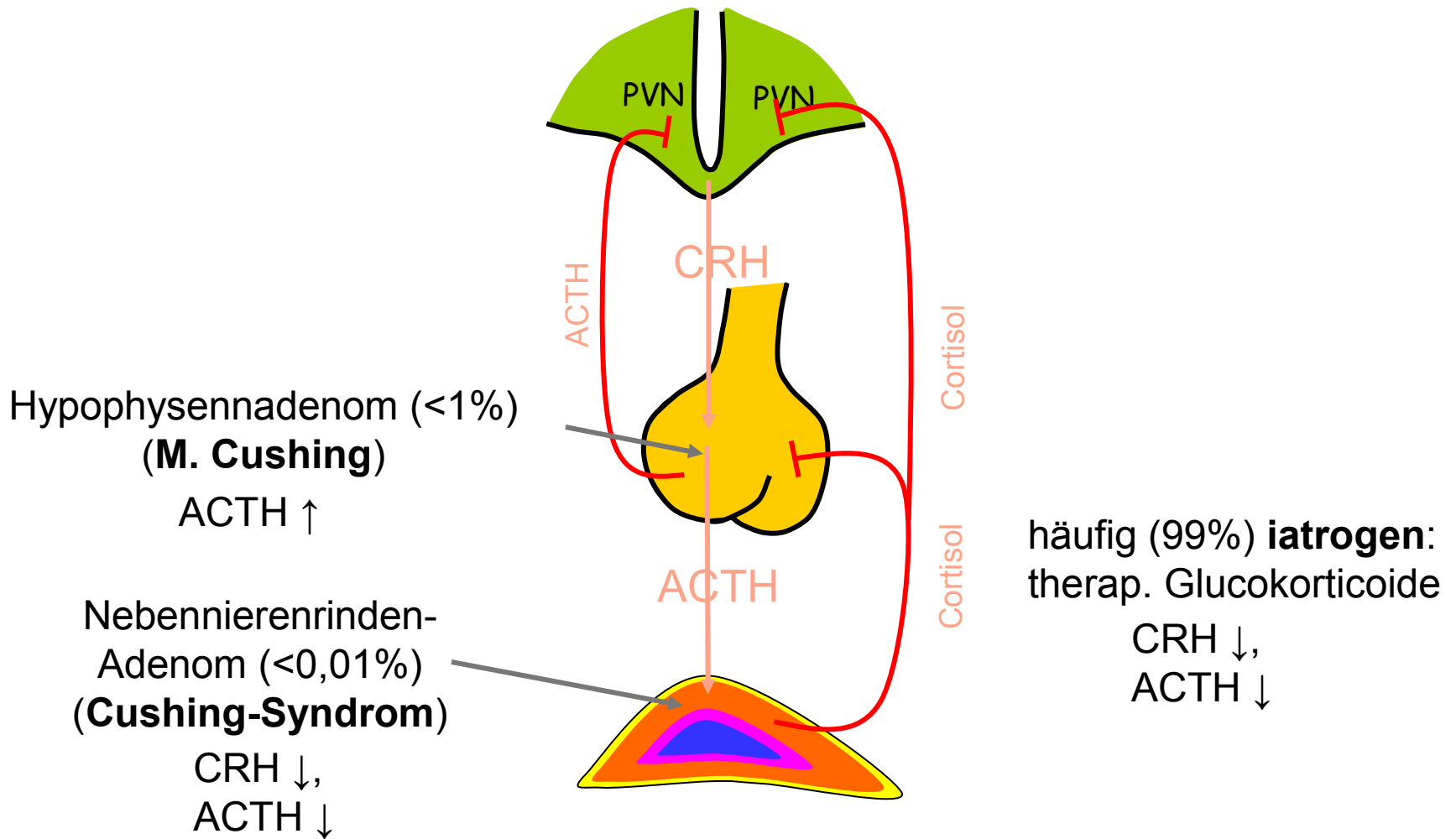
Interaktionen mit dem Immunsystem



Wirkung von Cortisol:



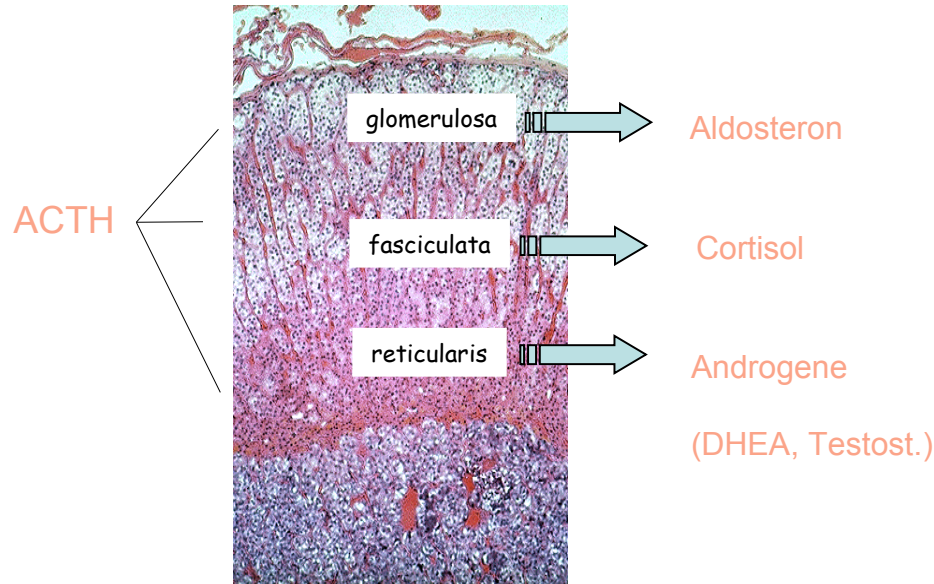
Hypercortisolismus



Hyperkortisolismus: Morbus Cushing

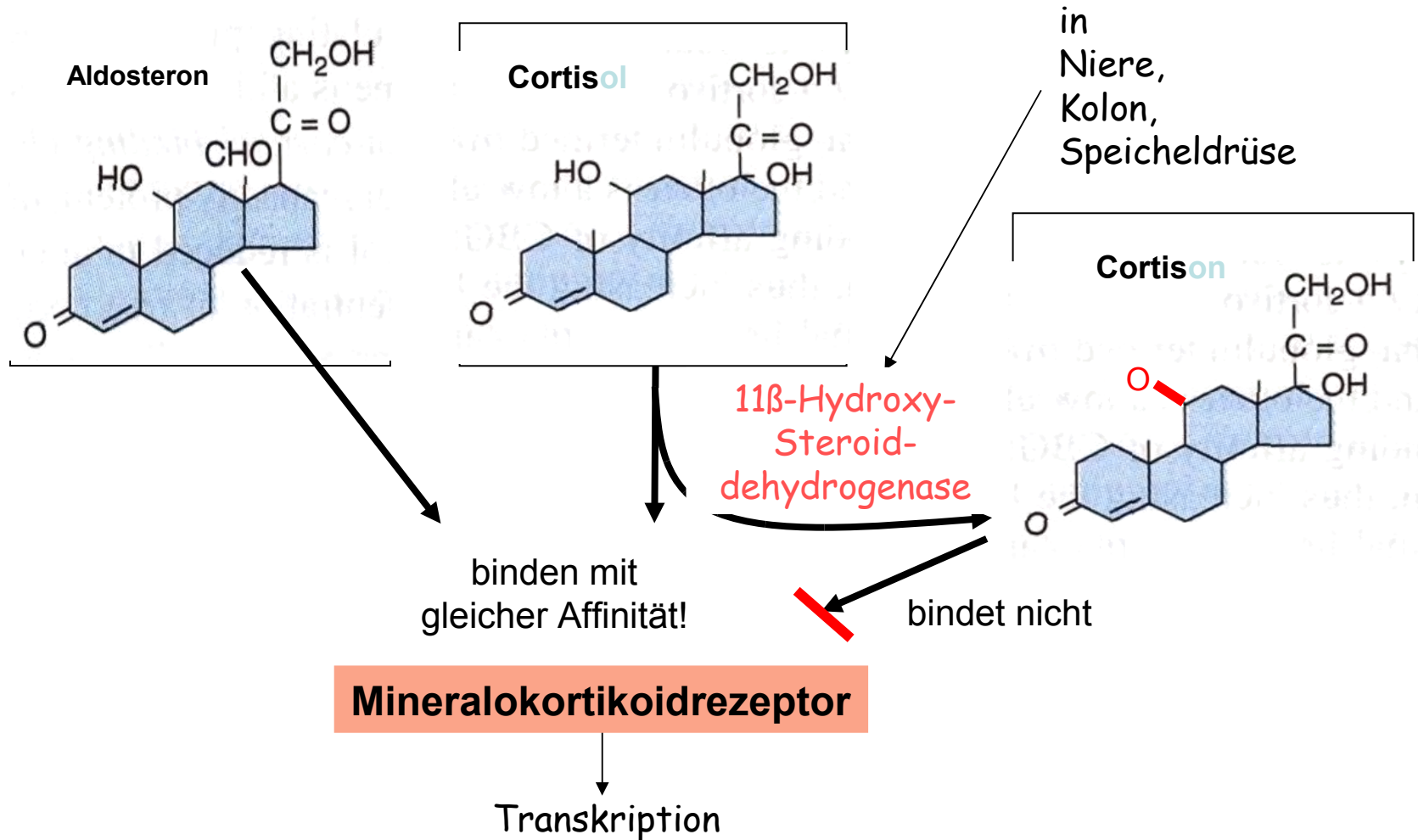


Virilisierung beim M. Cushing (Hypophysenadenom)



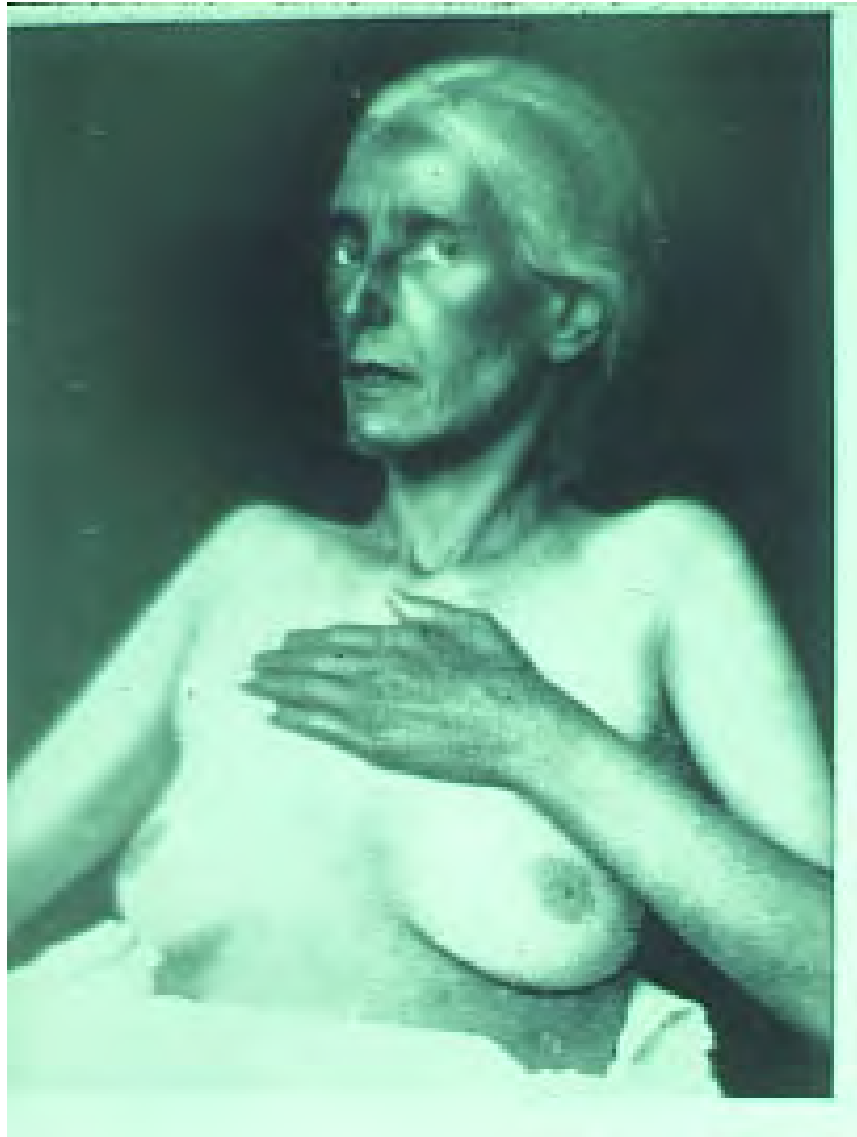
Beim primären und iatrogenen Hyperkortisolismus (Cushing-Syndrom) fehlen gewöhnlich Androgen- und Mineralokortikoid-Wirkungen !

Spezifität des Mineralokortikoid-Rezeptors

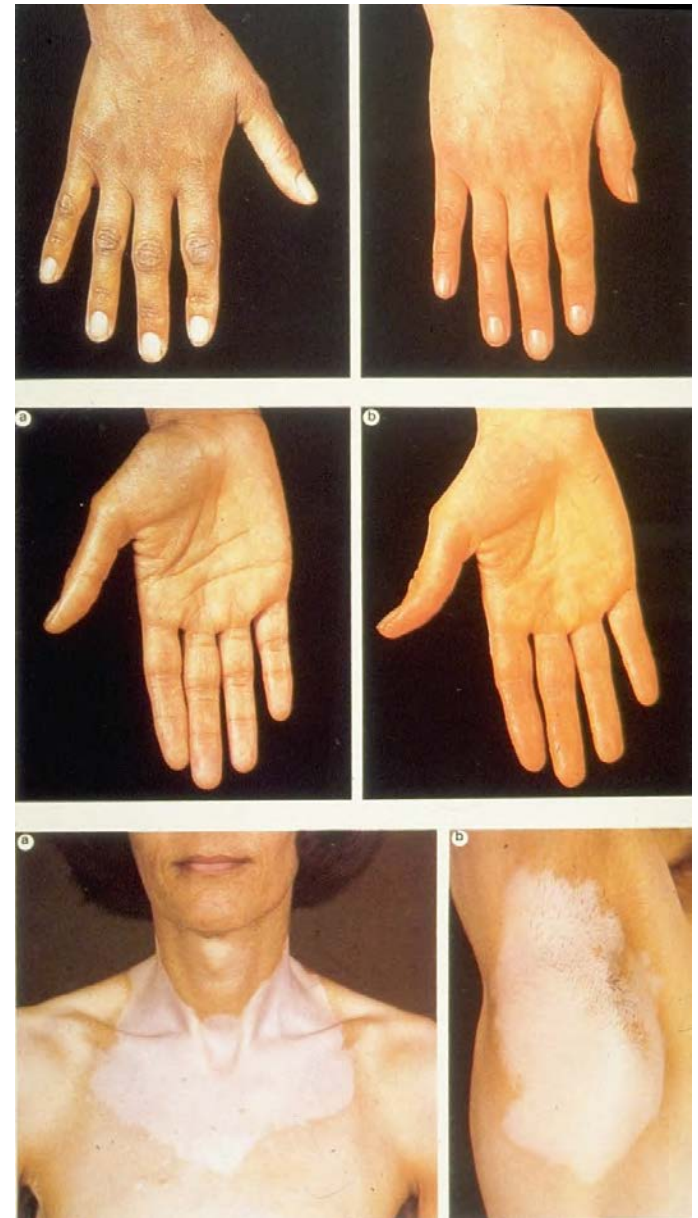
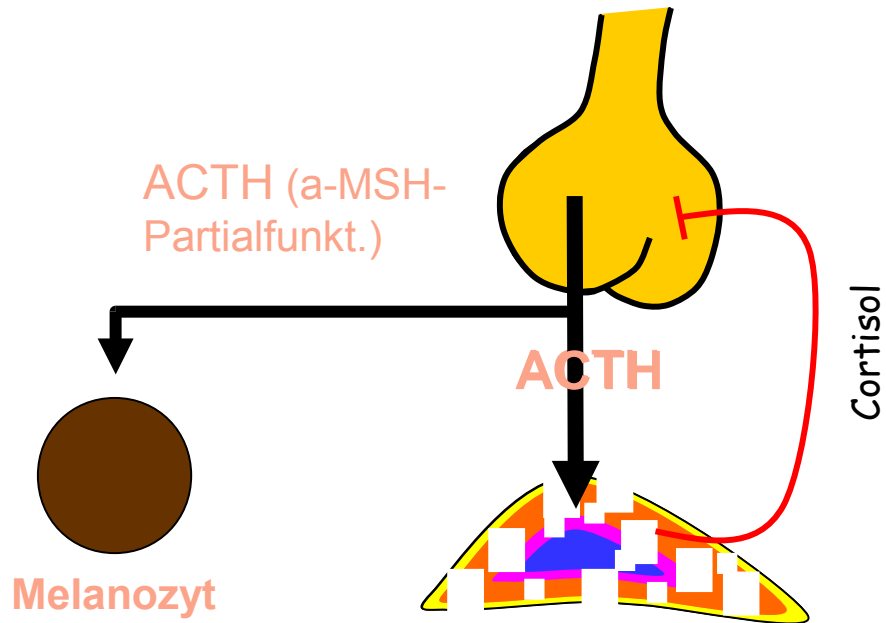


Hemmung der 11β-DH durch Glycyrrhizinsäure (Lakritze)!

Nebennierenrinden-Insuffizienz: Morbus Addison



M. Addison: Hyperpigmentierung

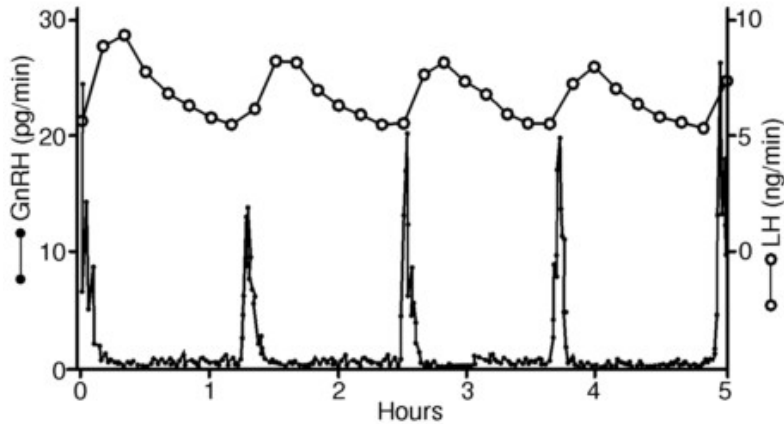


Endokrines System – Hormone: speziell

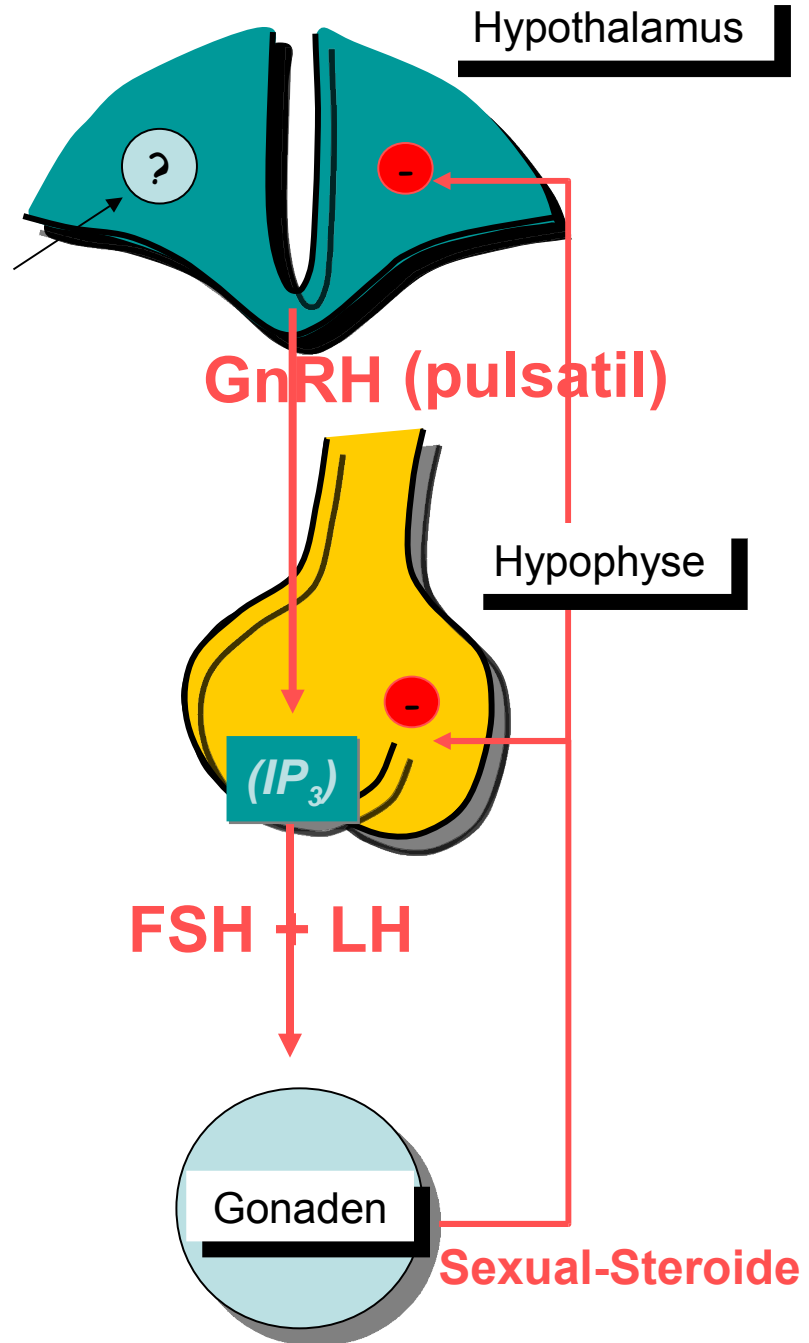
- ***Hypophyse***
(Adeno-/Neurohyphyse)
- ***Schilddrüse***
- ***Nebennierenrinde***
- ***Gonaden***
- ***Nebenschilddrüse***
- *Somatotropes Hormon*
- *Glandotrope Hormone*
- *ADH, Oxytocin*
- *Schilddrüsenhormone*
(T_3/T_4)
- *Mineralocorticoide*
- *Glucocorticoide*
- *Sexualhormone*
- ***Sexualhormone***
- *Parathormon*
- *(Calcitonin, Calcitriol)*

Hypothalamus- Hypophysen- Gonaden- Achse

hypothalamischer
Pulsgenerator



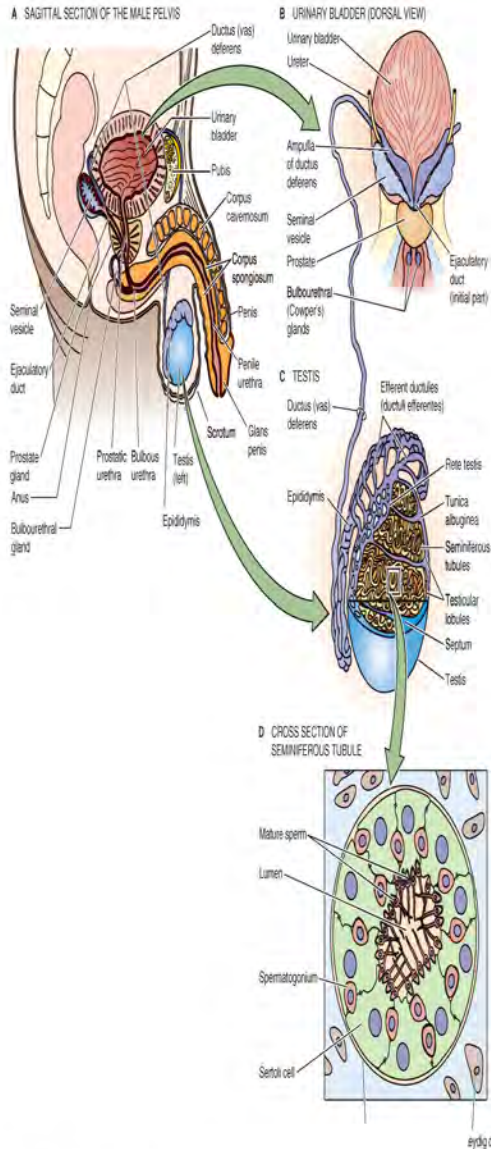
- Senkung der Pulsfrequenz durch gonadale Steroide (Östradiol, Progesteron) --> wichtiger neg. Feedback-Mechanismus
- GnRH-Neurone haben keine Rezeptoren für gonadale Steroide
- Effekt durch Naloxon aufhebbar --> Opioiderger Mechanismus (β -Endorphin)



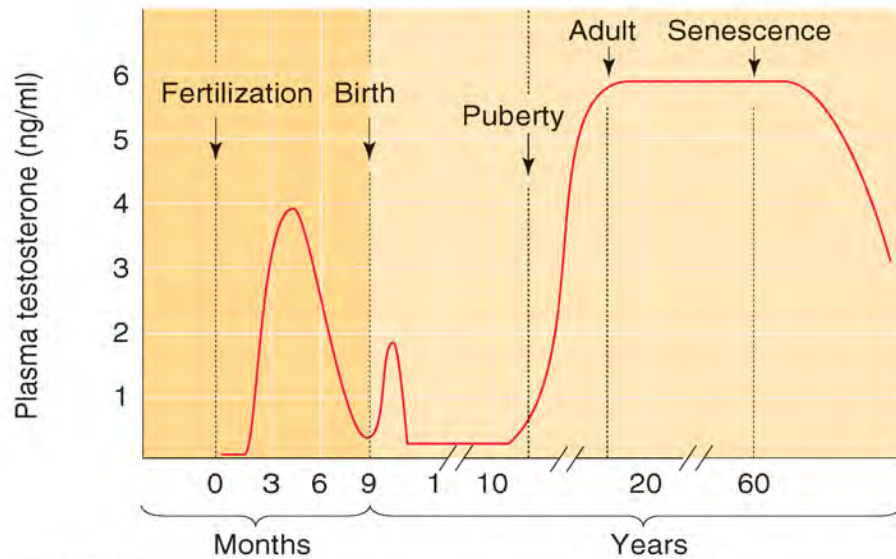
Endokrines System – Hormone: speziell

- ***Hypophyse***
(Adeno-/Neurohyphyse)
- ***Schilddrüse***
- ***Nebennierenrinde***
- ***Gonaden***
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- *Glandotrope Hormone*
- *ADH, Oxytocin*
- *Schilddrüsenhormone*
(T_3/T_4)
- *Mineralocorticoide*
- *Glucocorticoide*
- *Sexualhormone*
- *Androgene*
- *Östrogene*
- *Gestagene*

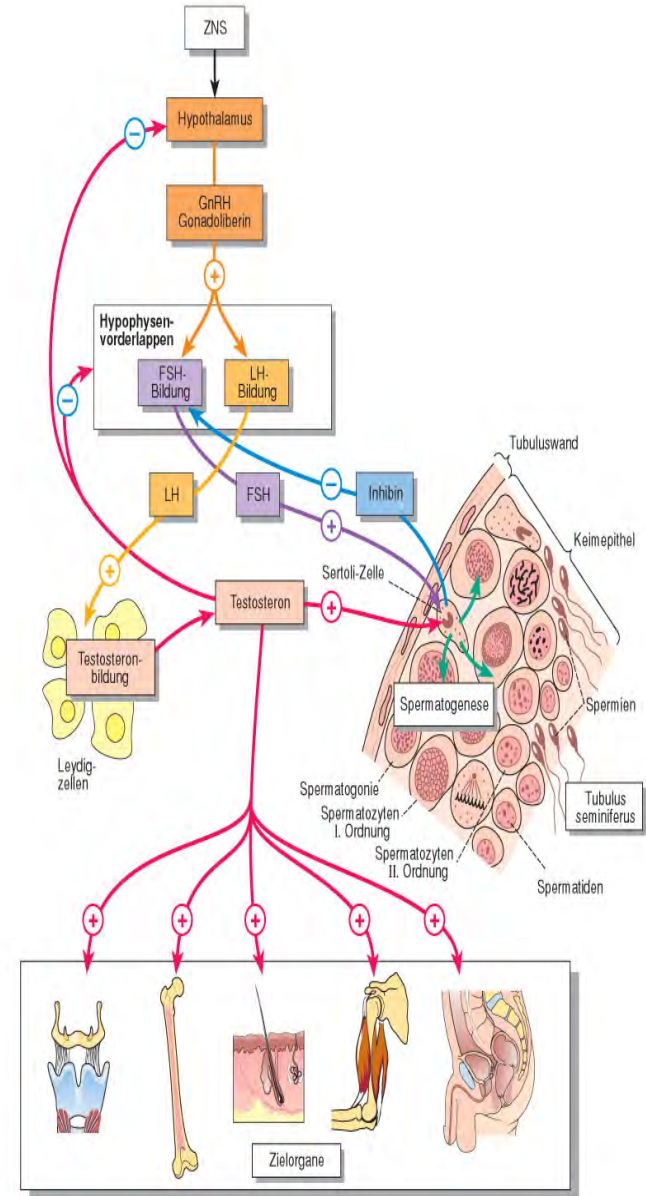
Zielzellen der gonadotropen Hormone



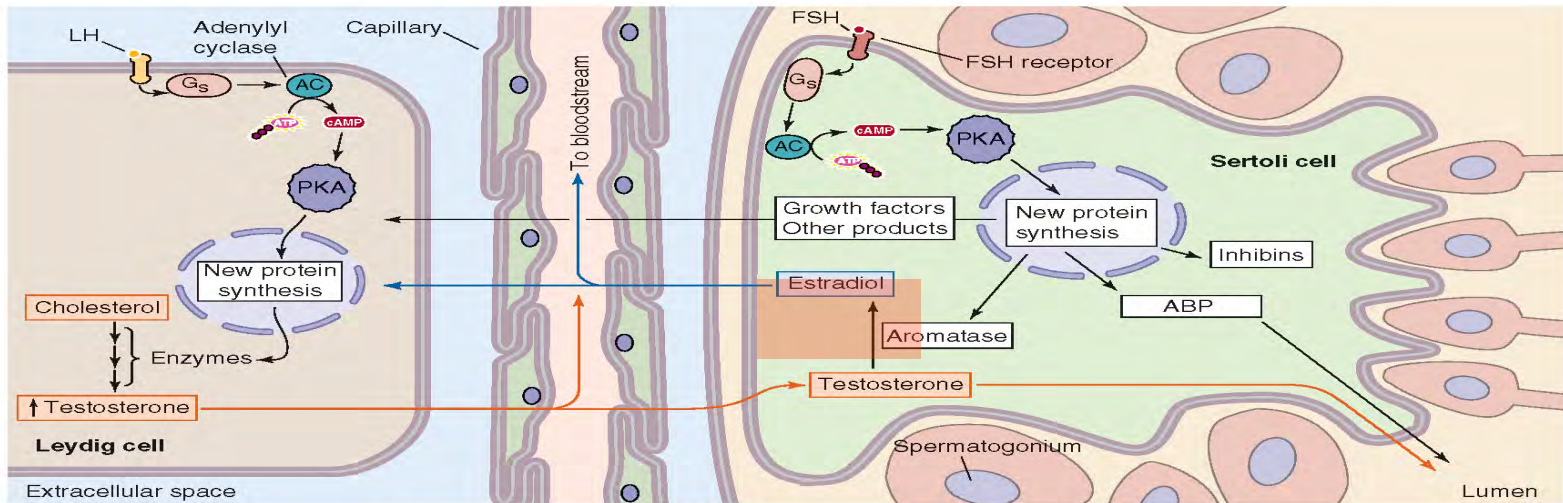
Spermatogenese und Testosteron- Sekretion



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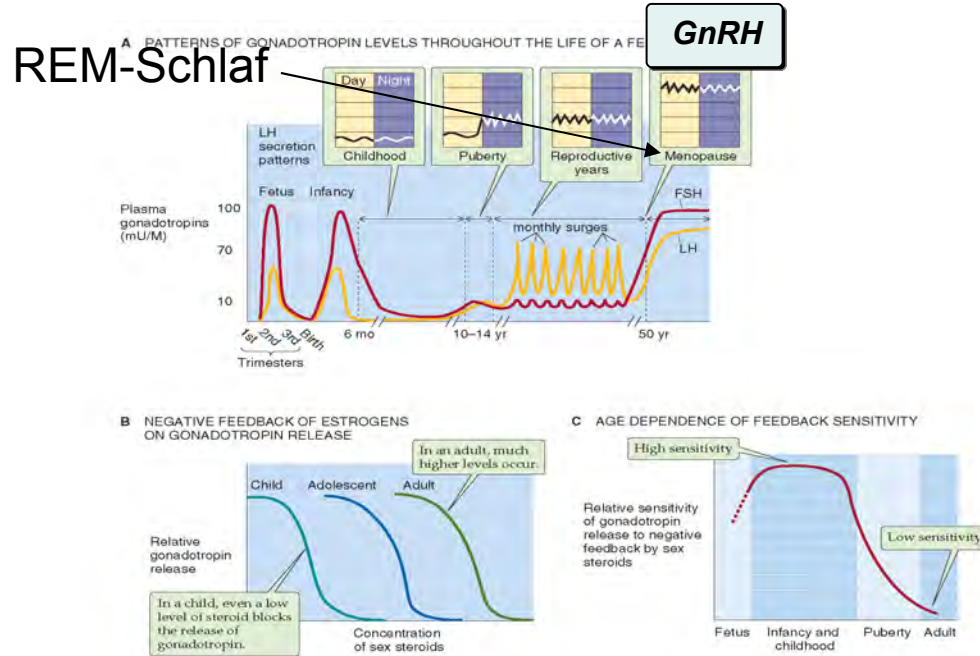
Steuerung und Interaktionen von Sertoli- und Leydig-Zellen



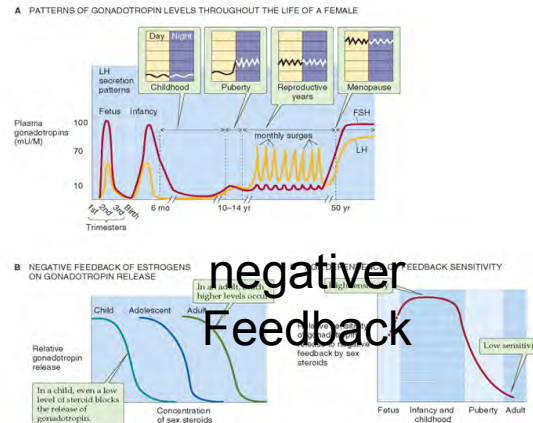
Endokrines System – Hormone: speziell

- ***Hypophyse***
(Adeno-/Neurohyphyse)
 - ***Schilddrüse***
 - ***Nebennierenrinde***
 - ***Gonaden***
- *Somatotropes Hormon*
 - *Glandotrope Hormone*
 - *ADH, Oxytocin*
 - *Schilddrüsenhormone*
(T_3/T_4)
 - *Mineralocorticoide*
 - *Glucocorticoide*
 - *Sexualhormone*
 - *Androgene*
 - *Östrogene (Östradiol)*
 - *Gestagene*
(*Progesteron*)

Pulsatile GnRH-Sekretion und Lebensalter

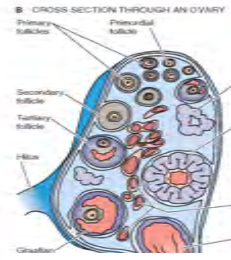
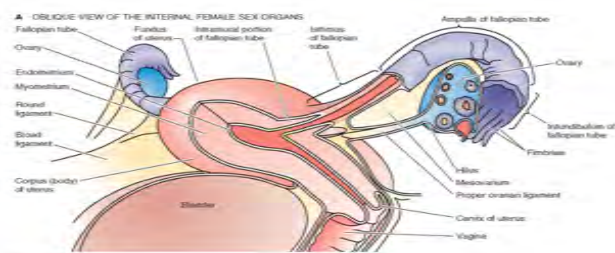


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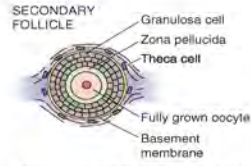
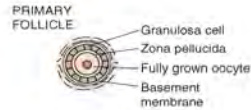
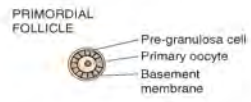


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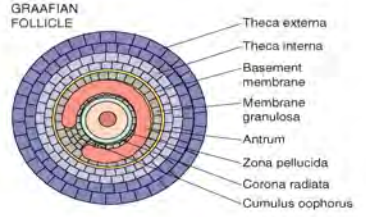
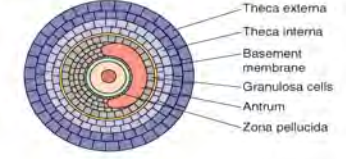
Monatszyklus und Follikelreifung



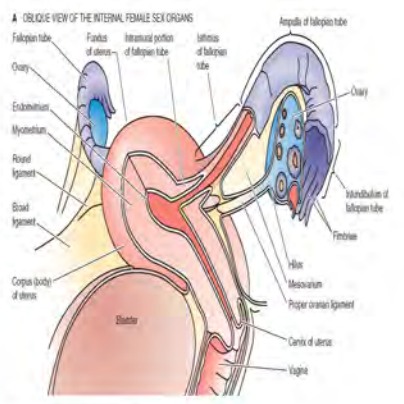
PRIMARY OOCYTE



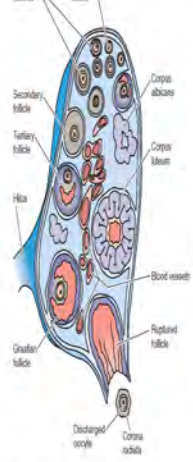
EARLY TERTIARY FOLLICLE



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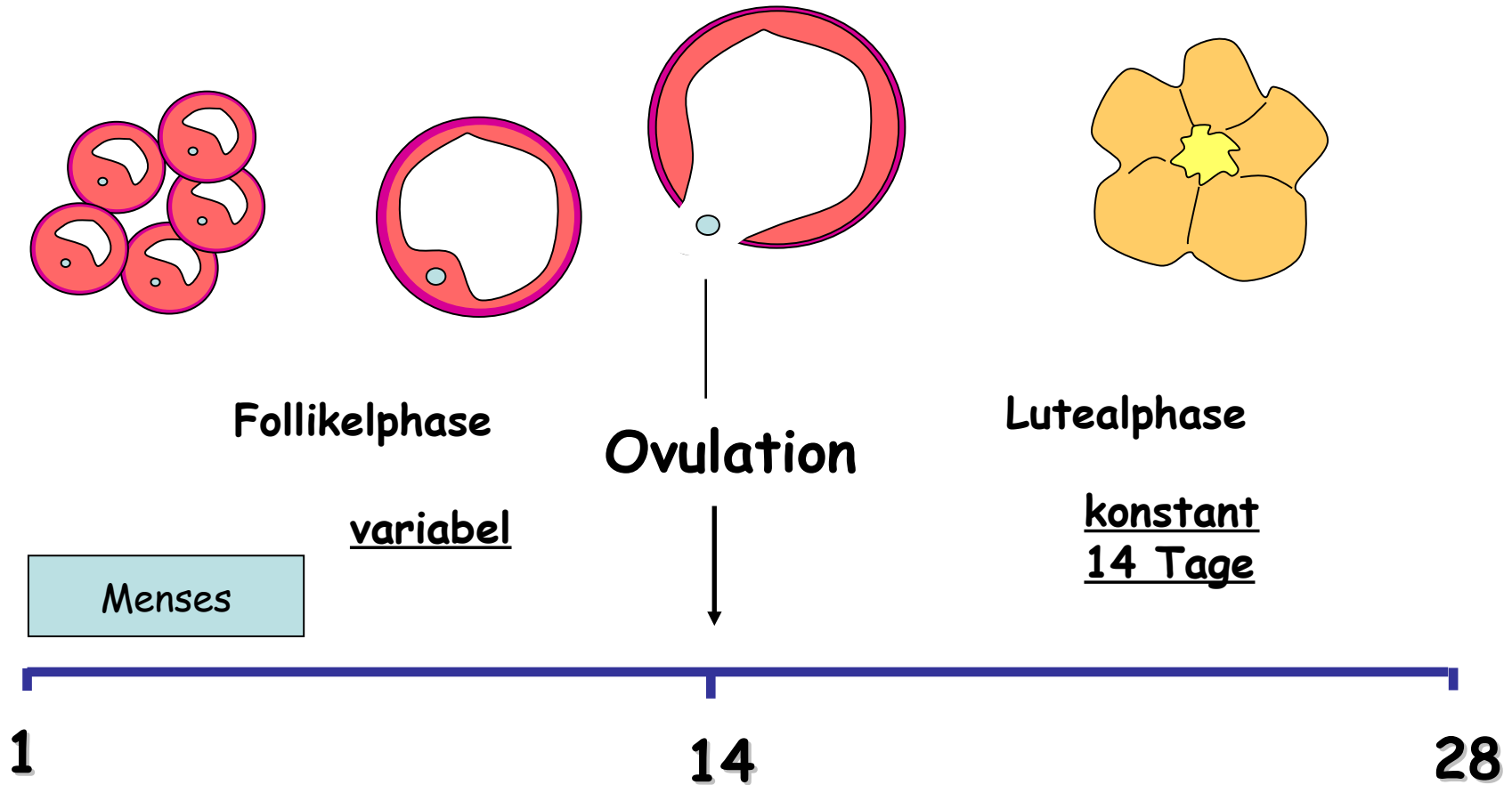


CROSS SECTION THROUGH AN OVARY



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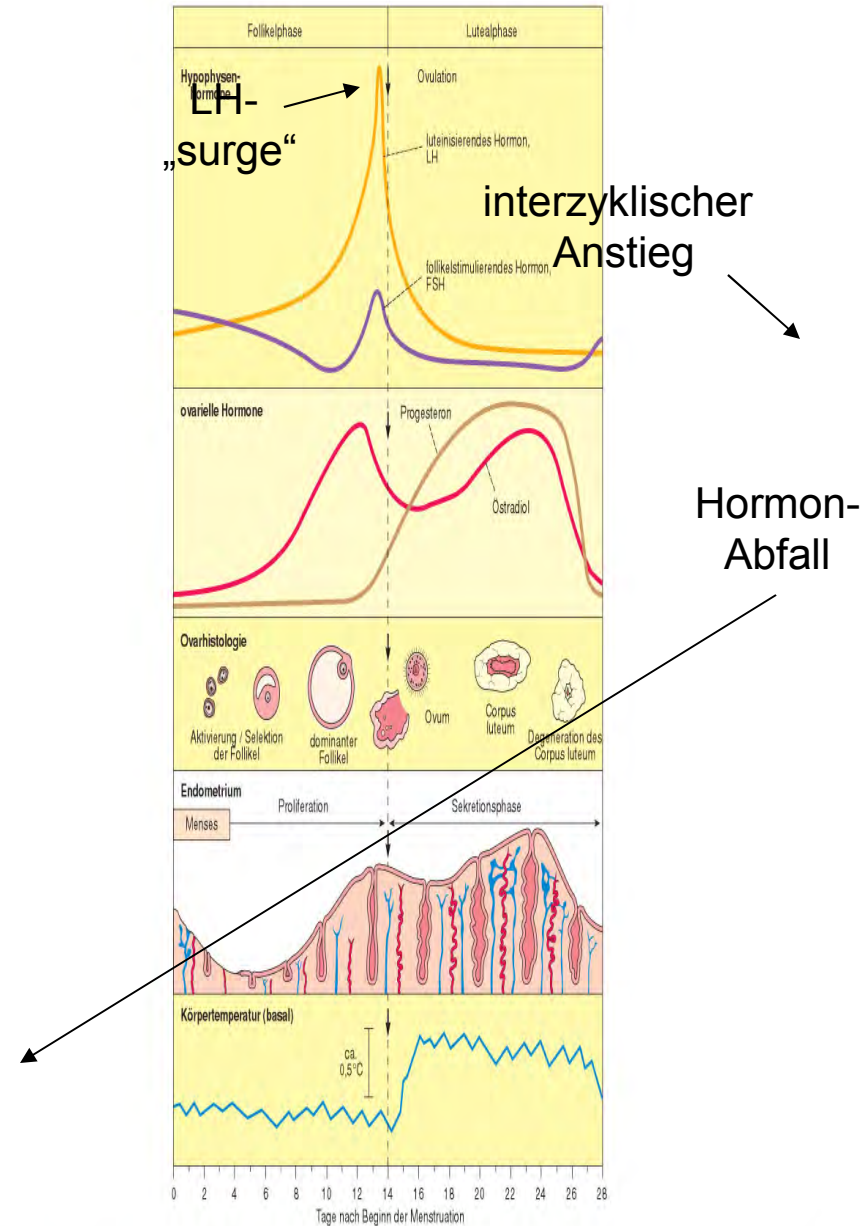
Menstruationszyklus



- Tag 1 ist der erste Tag der Monatsblutung
- normalerweise nicht mehr als 4 Tage Variation von Monat zu Monat
- Dauer der Blutung: 3-8 Tage

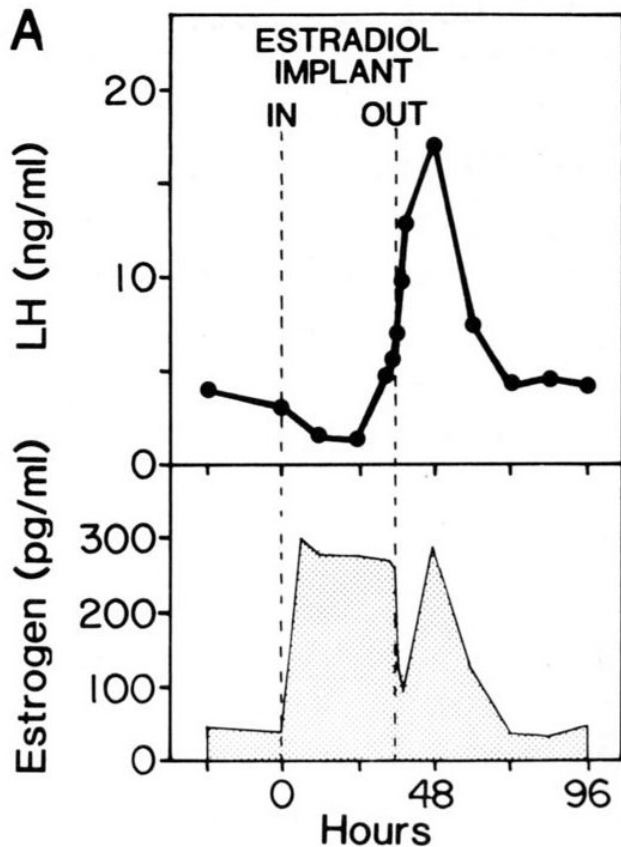
GK1!!

Menstruationszyklus: Hormon-Plasma-Spiegel und Follikelphasen

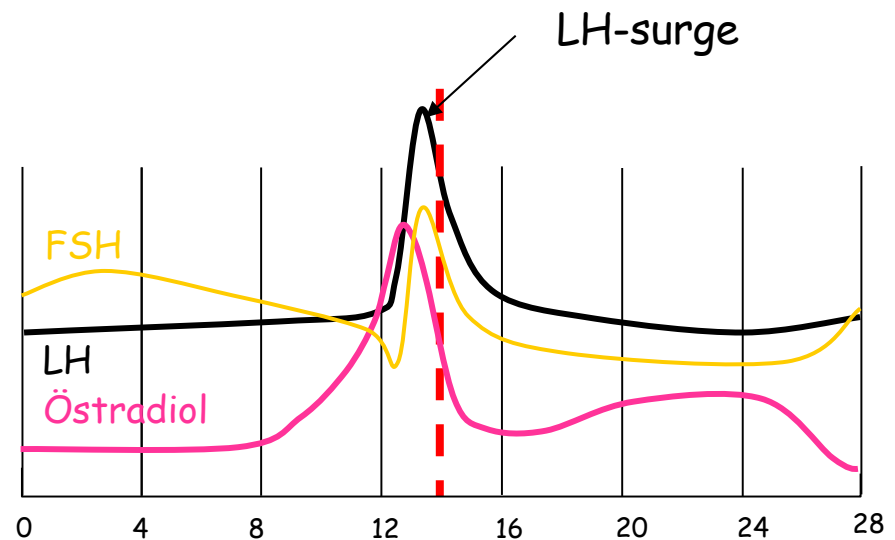


Prä-ovulatorischer positiver (!!) Feedback von Östradiol auf die LH-Sekretion

Hohlweg-Effekt:

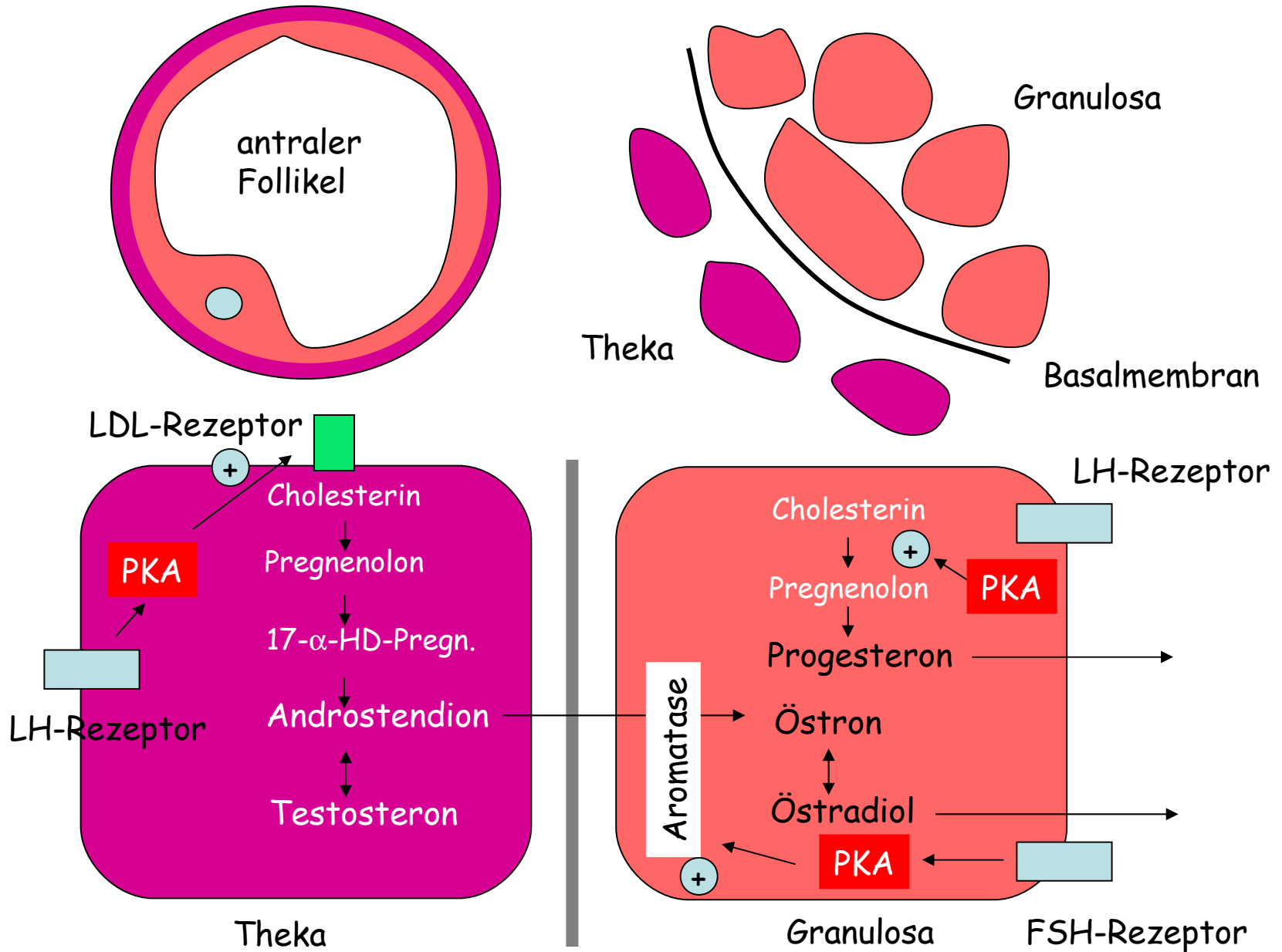


Anhaltend hohen Östradiol-Konzentrationen
(>150 pg/ml für wenigstens 36 Stunden)
→ Positiver Feedback

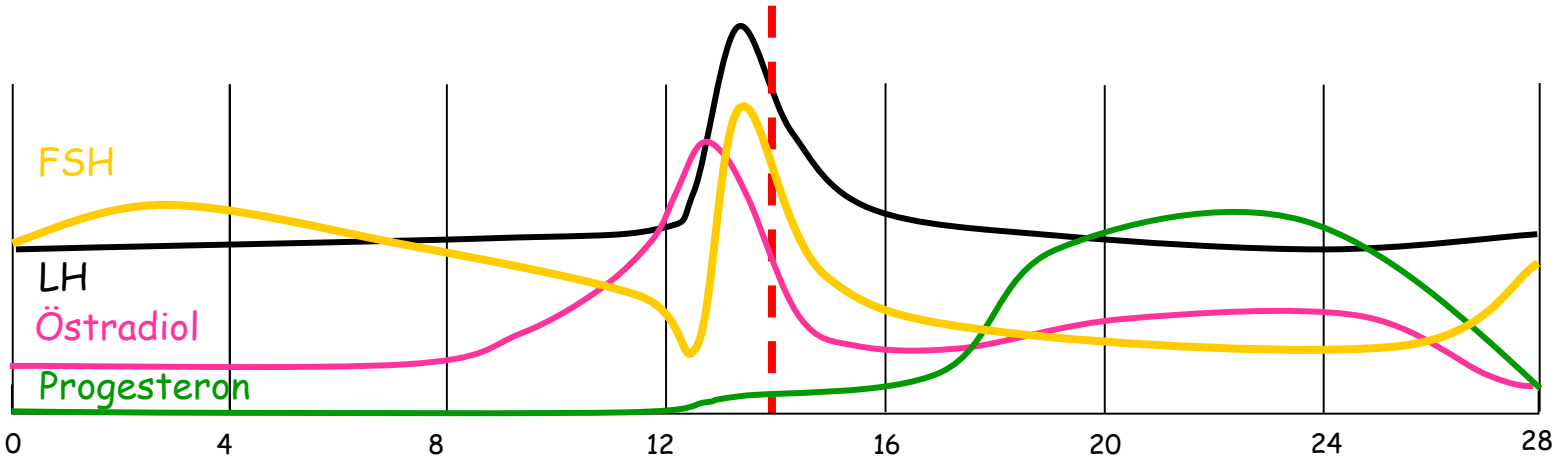


Positiver Feedback wahrscheinlich hypothalamisch
(über GnRH) als auch hypophysär wirksam.

Die Zwei-Zell-Theorie der föllikulären Östradiol- bzw. Progesteronbildung



Hormonspiegel während des Menstruationszyklus

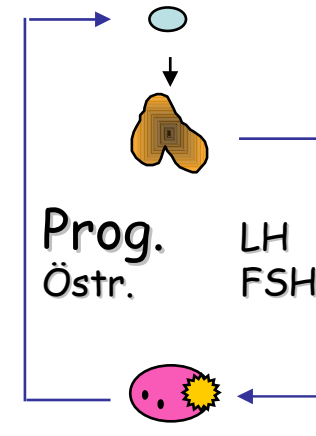
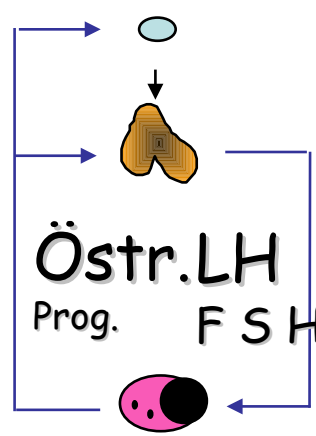
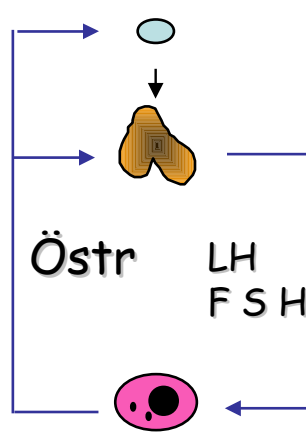
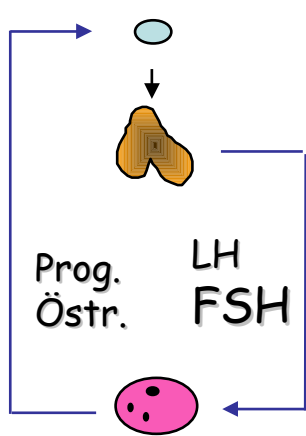


spätluteal
frühfollikulär

Mitte Follikelph.

Mittzyklisch

Mitte luteale Ph.



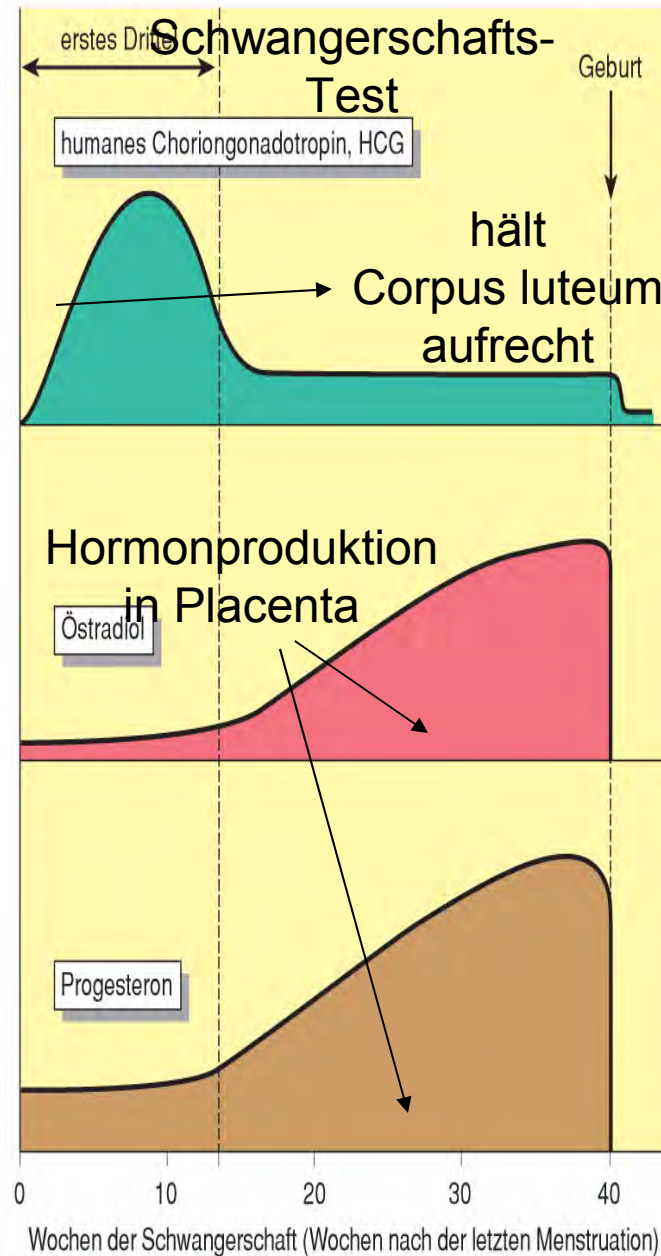
Östr. und Prog. sinken
Selektive Zunahme von
FSH=
Interzyklischer Anstieg

Östr. steigt:
neg. Feedback:
FSH fällt

2 Tage Östr.
>300 pg/ml
= positiver Feedback
= LH peak

Hohes Prog.
negativer Feedback
=LH/FSH niedrig
Prog. überwiegt Östr.

Schwangerschaft: HCG-Sekretion aus Blastozyste



Signalkaskaden im endokrinen System:

Bildungsort	G_s (cAMP↑)	G_i (cAMP↓)	G_{o/q} (IP₃)	TK-R.	TK.ass.-R.
Hypo- thalamus	GHRH	SIH	TRH GnRH		
	CRH	Dopamin (D ₂)			
Neuro- hypophyse	ADH (V ₂) Oxytocin		ADH (V ₁) Oxytocin		
Adeno- hypophyse	TSH ACTH FSH		Prolaktin		STH
Leber				IGF1/2	