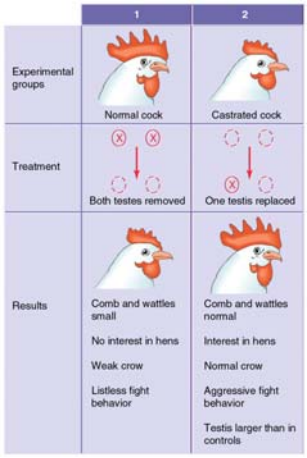
Lecture 14&15
18&20 Feb 2008

Vertebrate Physiology
ECOL 437 (MCB/VetSci 437)
Univ. of Arizona, spring 2008

Kevin Bonine & Kevin Oh

1. Endocrine Physiology (Ch14)
2. Reproductive Physiology (Ch15)




http://eebweb.arizona.edu/eeb_course_websites.htm

1

Housekeeping, 18 February 2008

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Upcoming Readings
Today: Ch14
Wed 20 Feb: Ch14&15
LAB Wed 20 Feb: 4 readings on website
Fri 22 Feb: no lecture, work on proposal
Monday 25 Feb: Ch15, Ch17?
Wed 27 Feb: Research Question Due
Wed 27 Feb: Ch17
LAB Wed 27 Feb: muscle readings on website
Fri 29 Feb: Ch17

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Lab discussion leaders: 20 Feb
1pm – Virsheena, Mathew S. Arturo
3pm – Kat, Clif, Amber

Lab discussion leaders: 27 Feb
1pm – Steve & Cassia
3pm – Kevin & Jennifer

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The Edges of Life Lecture Series

## The Edges of Life – 7pm at Centennial Hall

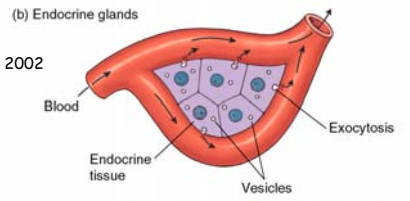
Wednesday, February 20
Life's Human Edge: Changing Perspectives on the End of Life
Michael Gill, Associate Professor, Philosophy
Nothing looms with more certainty than the final edge of one's own life. But in fact, the edge between life and death is anything but clear. This lecture will address the attempts that have been made to define the line between life and death and will explore the biological, legal, ethical, and spiritual debates that have raged around that line.

Wednesday, March 5
Life's Technological Edge: The Singularity is Near: When Humans Transcend Biology
Ray Kurzweil, *via Teleportec Teleporter*
Founder, Chairman and Chief Executive Officer, Kurzweil Technologies
Humanity is on the edge of a vast transformation, when what it means to be human will be both enriched and challenged. Inventor and futurist Ray Kurzweil will introduce this radically optimistic singularity, an era when we break our genetic shackles to create a nonbiological intelligence trillions of times more powerful than today. In this new world, humans will transcend biological limitations to achieve entirely new levels of progress and longevity.
*This lecture co-sponsored by: UA College of Engineering and UA College of Science*

## These do not count as physiology lectures. 3


Martin Wikelski, Princeton

# Endocrine System

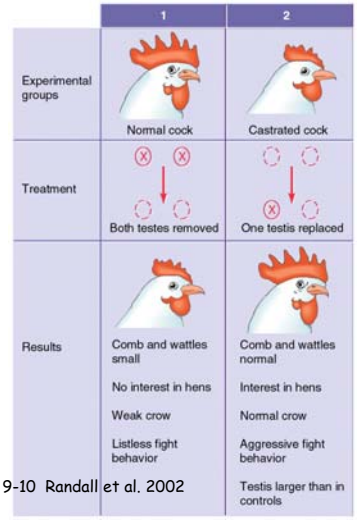



9-9 Randall et al. 2002

-Hormone action often amplified by second messenger cascade

-Hormones often released in very low concentrations

Made them difficult to study!




9-10 Randall et al. 2002

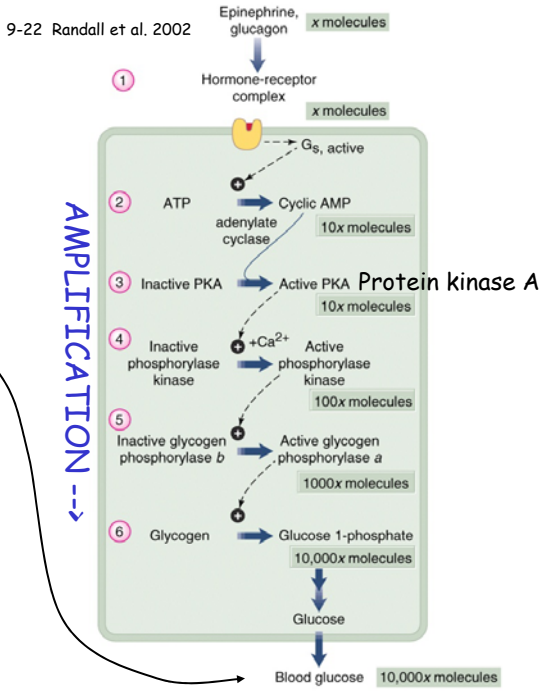
1. cAMP

Glycogenolysis Example: (glycogen-> glucose)

-Epi (muscle)

-Glucagon (liver)

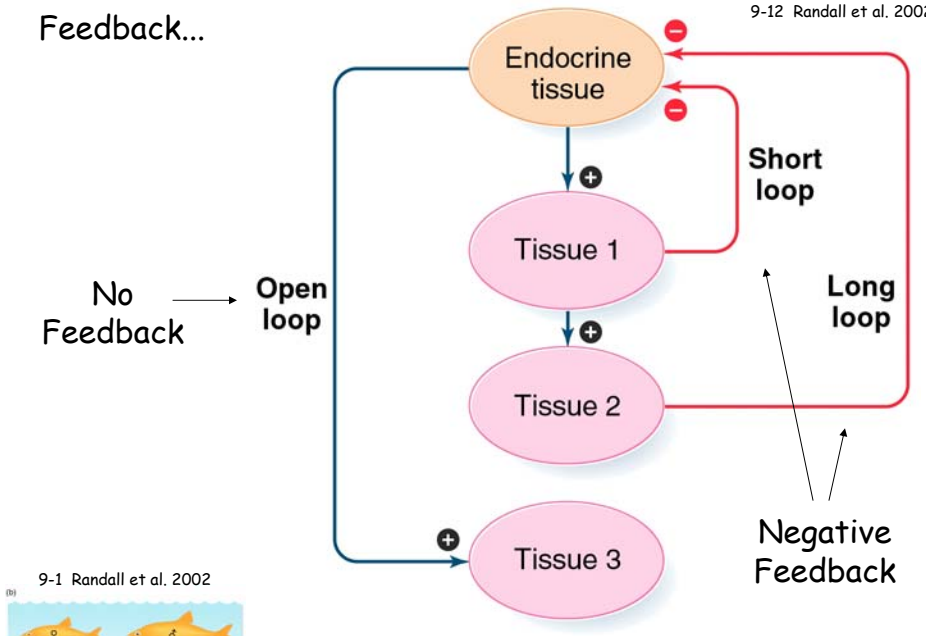
Goal is to increase blood [glucose]



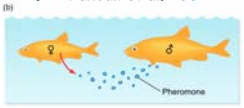

9-22 Randall et al. 2002

Feedback...

9-12 Randall et al. 2002




9-1 Randall et al. 2002

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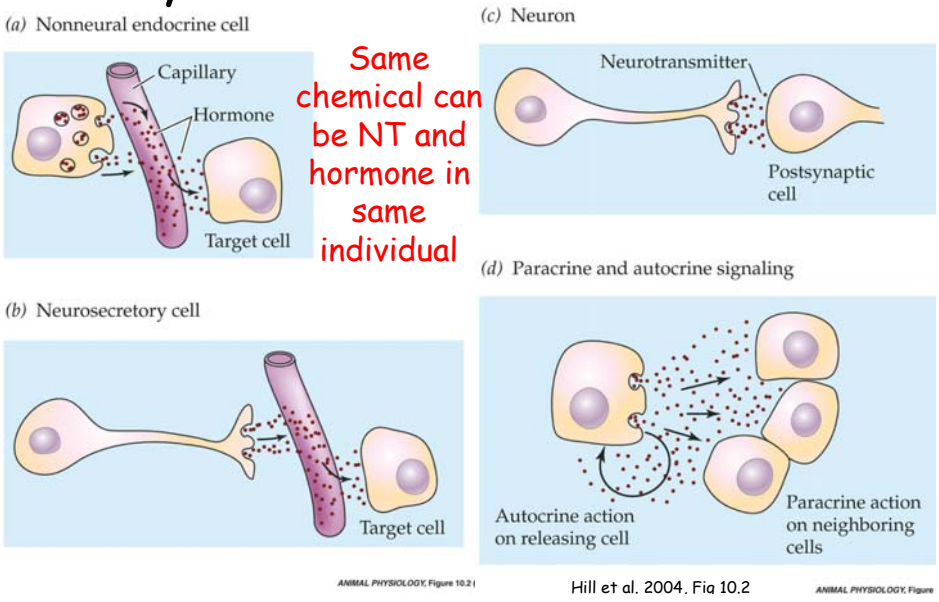
# Endocrine and Nervous Systems Have Similarities




Same chemical can be NT and hormone in same individual

Hill et al. 2004. Fig 10.2

Chap 14. Endocrine System – Glands and Hormones

Secretions with consequences

All cells secrete, but
Specialized secretory cells grouped into glands
Secrete same specialized substance (e.g., hormone)

Nervous System neurotransmitter acts near and fast

Other secretions, such as hormones, may act more distantly and over a longer time period

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Categories of cellular secretions:

Autocrine – affect the secreting cell directly

Paracrine – affect neighboring cells
e.g., histamine and inflammation

Endocrine – release into bloodstream

Exocrine – release onto epithelial surface
e.g., sweat onto skin, bile into digestive system

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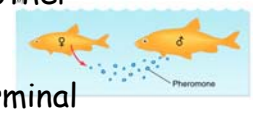
Pheromone – exocrine secretion to signal other individuals




Neuroendocrine – secretion from axon terminal into blood stream

Neurosecretory cells and Neurohormones
control much of the endocrine system

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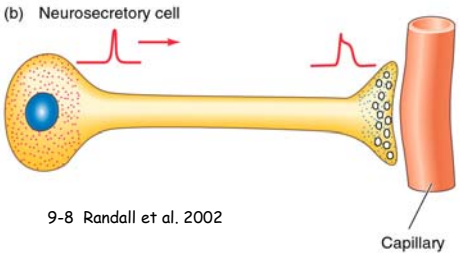
## Neuroendocrine Systems

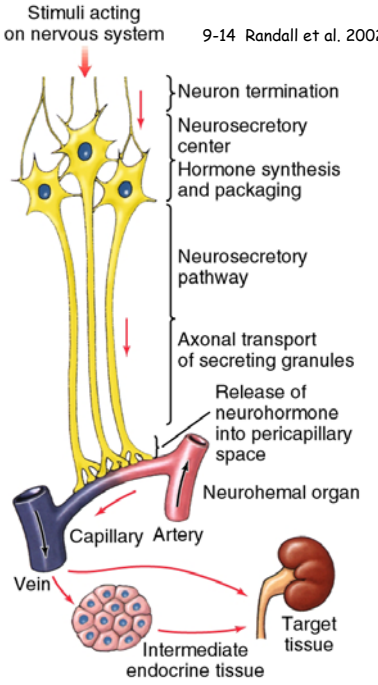
Neurosecretory cells secrete neurohormones

Axon terminates into neurohemal organ

Neurohormones often [effect] other glands/hormones



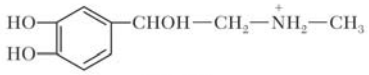

9-8 Randall et al. 2002




9-14 Randall et al. 2002

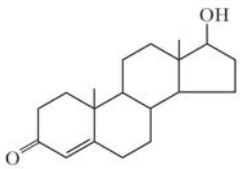
# Three Hormone Classes

1 Amines – small
(e.g., epinephrine, norepi, thyroid, melatonin)



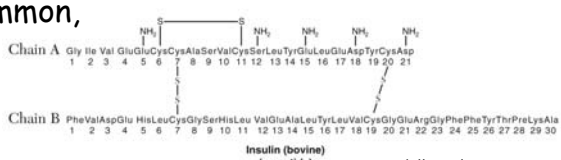

Epinephrine
(an amine)

2 Steroid Hormones from cholesterol
(e.g., testosterone, estrogen)




Testosterone
(a steroid)

3 Peptide Hormones – common, large, complex
(e.g., insulin, ADH, GH)




Insulin (bovine)
(a peptide)

9-11 Randall et al. 2002

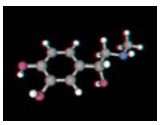
**TABLE 14.2** Peptide, steroid, and amine hormones

| Property | Peptides | Steroids | Amine hormones: Catecholamines | Amine hormones: Thyroid hormones | Amine hormones: Melatonin |
|---|---|---|---|---|---|
| Site of secretion | Most sites in Table 14.1, except adrenal cortex and medulla, thyroid gland, and pineal gland | Adrenal cortex, gonads, and placenta | Adrenal medulla | Thyroid gland | Pineal gland |
| Structure | Chains of amino acids | Derived from cholesterol | Derived from tyrosine | Derived from tyrosine and iodine | Derived from tryptophan |
| Solubility | Water-soluble | Lipid-soluble | Water-soluble | Lipid-soluble | Water-soluble |
| Synthesis and storage | Synthesized at rough ER, processed in Golgi apparatus; stored in vesicles in advance of use | Synthesized on demand in intracellular compartments; not stored | Synthesized in the cytoplasm and stored in vesicles ("chromaffin granules") | Made prior to use and stored in a colloid island within the gland | Synthesized in the cytoplasm and stored in vesicles |
| Secretion | Exocytosis | Simple diffusion through cell membrane | Exocytosis | Simple diffusion through cell membrane | Exocytosis |
| Transport | Dissolved in plasma; some bound to carrier proteins | Bound to carrier proteins | Dissolved in plasma | Bound to carrier proteins | Dissolved in plasma |
| Half-life | Minutes | Hours | Seconds to minutes | Days | Minutes |
| Location of receptor molecules | Surface of target cell membrane | Cytoplasm or nucleus (some steroids bind to cell-surface receptors) | Surface of target cell membrane | Nucleus | Surface of target cell membrane |
| Action at target cell | Activate second-messenger systems or alter membrane channels | Alter gene expression; activated genes initiate transcription and translation | Activate second-messenger systems | Alter gene expression; activated genes initiate transcription and translation | Activate second-messenger systems |
| Response of target cell | Change activity of preexisting proteins, some of which may induce new protein synthesis | Synthesize new proteins; some may change activity of preexisting proteins | Change activity of preexisting proteins | Synthesize new proteins | Change activity of preexisting proteins |

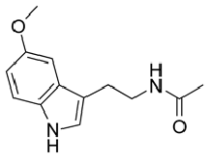
Sources: After Sherwood 2004; Silverthorn 2004; and Widmaier, Raff, and Strang 2004.

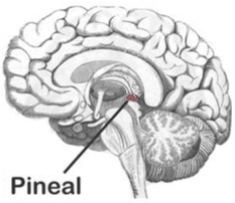
Hill et al. 2004 ANIMAL PHYSIOLOGY, Table 14.2 © Sinauer Associates, Inc.

Amines – small
(e.g., epi, norepi, thyroid)

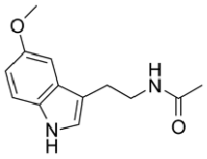


Modified Amino Acids
1. catecholamines (epi, norepi, dopamine; tyrosine)
2. thyroid (lipid soluble; tyrosine)
3. melatonin (tryptophan)

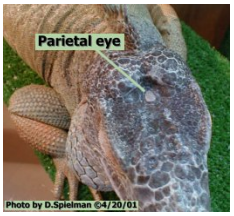





## Melatonin



Pineal Gland
Retina
GI Tract

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Produced at Night
Circadian Rhythms
Suprachiasmatic Nucleus (SCN) in Hypothalamus

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Antioxidant
Immune System Support
Suppress libido? (LH, FSH)

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Glandular Secretion

Response to stimulus

Hormone
Neurotransmitter
Action Potential
etc. (e.g., osmolarity and ADH)

Storage before Secretion

Large molecules easily stored because can't leave readily

Small molecules often stored bound to accessory proteins

Some molecules actively/continuosly taken into vesicles

Steroid hormones (lipid soluble) tend to leak out soon

Hydrophobic steroid and thyroid hormones move in blood, bound to carrier proteins

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**TABLE 14.1 Summary of major blood-borne hormones in mammals** *(Part 1)*

| Endocrine tissue | Hormone | Class of molecule | Main functions |
|---|---|---|---|
| Adrenal cortex | Aldosterone (mineralocorticoid) | All steroids | Stimulates Na reabsorption and K secretion in kidney |
| | Androgens | | Act on bone to cause growth spurt at puberty; increase sex drive in females by action on brain |
| | Glucocorticoids | | Part of stress response; affect metabolism of many tissues to increase blood glucose and cause protein and fat catabolism |
| Adrenal medulla | Epinephrine and norepinephrine | Catecholamines | Part of stress response; influence cardiovascular function and organic metabolism of many tissues |

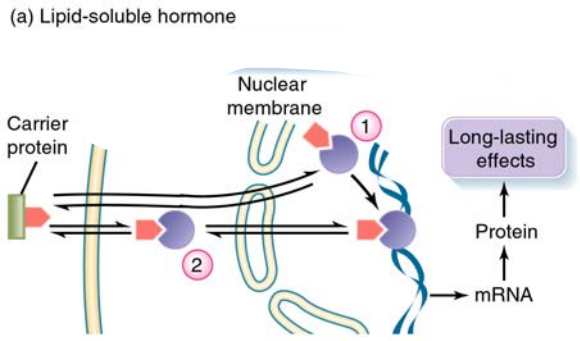
Sources: After Bentley 1998; Henderson 2000; Schmidt-Nielsen 1997; Sherwood 2004; Silverthorn 2004; and Widmaier, Raff, and Strang 2004.

ETC…

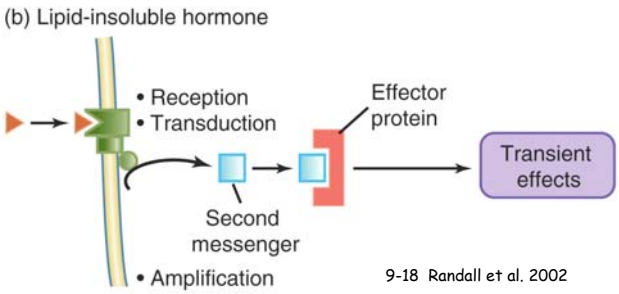
Hill et al. 2004

*ANIMAL PHYSIOLOGY*, Table 14.1 (Part 1) © Sinauer Associates, Inc.

1. Lipid Soluble




2. Lipid Insoluble




9-18 Randall et al. 2002

# Hormone *ACTION!*
## Where are the receptors?

1. Lipid Soluble
   - Steroid and Thyroid Hormones (~long-lived)
   - Through Membrane
   - Bind cytoplasmic receptors, then to Nucleus
   - Directly affect transcription (therefore long-term)

2. Lipid Insoluble
   - Bind cell-surface receptors
   - Often one or more 2nd messengers
   - Amplification

   ~ Rapid, short-duration responses

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Hormone *ACTION!* (receptors etc.)

2. Lipid Insoluble Hormones and Intracellular Signaling

A few receptors with direct catalytic activity, but most via 2nd messengers :

Possible 2nd messengers:

1. cAMP, cGMP (cyclic nucleotide monophosphates)
2. $IP_3$, DAG (diacylglycerol; inositol phospholipids)
3. $Ca^{2+}$ ions

General Model of Hormone Binding and Intracellular Signaling:

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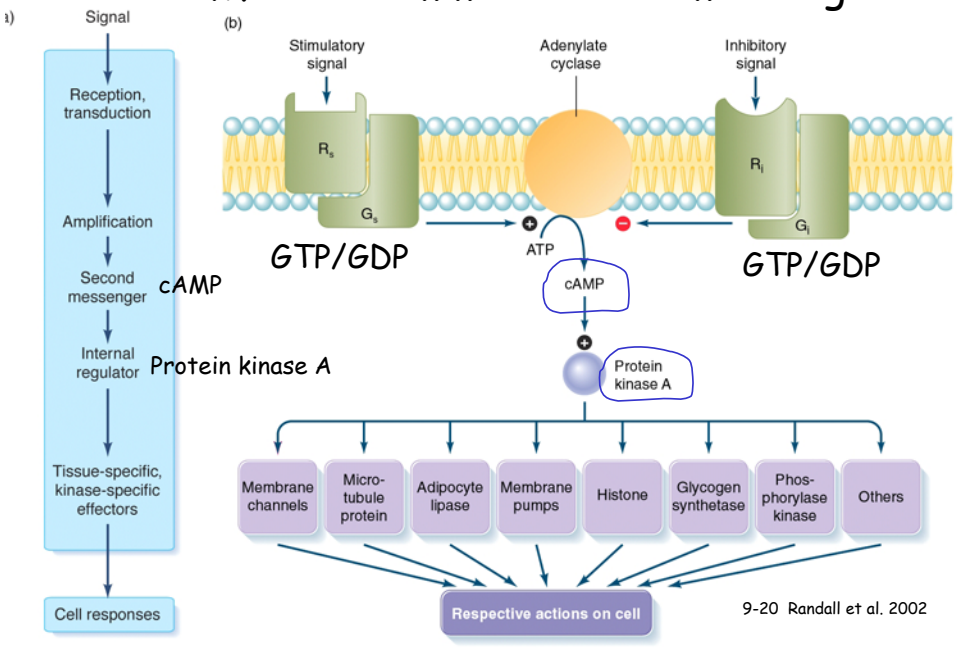
# cAMP is a common second messenger




9-20 Randall et al. 2002

Randall et al. 2002

*Table 9-6* Some hormone-induced responses mediated by the cAMP pathway

| Signal | Tissue | Cellular response |
|---|---|---|
| **Stimulatory** | | |
| Epinephrine ($\beta$-adrenoreceptors) | Skeletal muscle | Breakdown of glycogen |
| | Fat cells | Increased breakdown of lipids |
| | Heart | Increased heart rate and force of contraction |
| | Intestine | Fluid secretion |
| | Smooth muscle | Relaxation |
| Thyroid-stimulating hormone (TSH) | Thyroid gland | Thyroxine secretion |
| ADH (vasopressin) | Kidney | Reabsorption of water |
| Glucagon | Liver | Breakdown of glycogen |
| Serotonin | Salivary gland (blowfly) | Fluid secretion |
| Prostaglandin $I_2$ | Blood platelets | Inhibition of aggregation and secretion |
| **Inhibitory** | | |
| Epinephrine ($\alpha_2$-adrenoreceptors) | Blood platelets | Stimulation of aggregation and secretion |
| | Fat cells | Decreased lipid breakdown |
| Adenosine | Fat cells | Decreased lipid breakdown |

*Source:* Berridge, 1985.

# Glandular Secretion

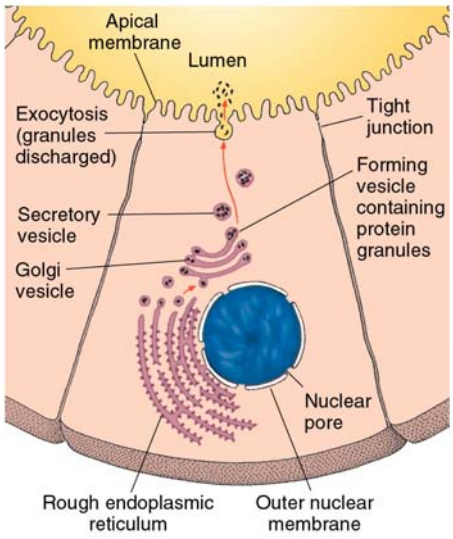
Secretory Vesicles

Exocytosis – common release mechanism

Regulation via Calcium –

free cytosolic calcium concentration is correlated with exocytosis (including of NT, hormones, etc.)

9-2 Randall et al. 2002

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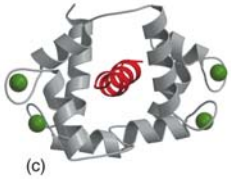
$Ca^{2+}$ ions

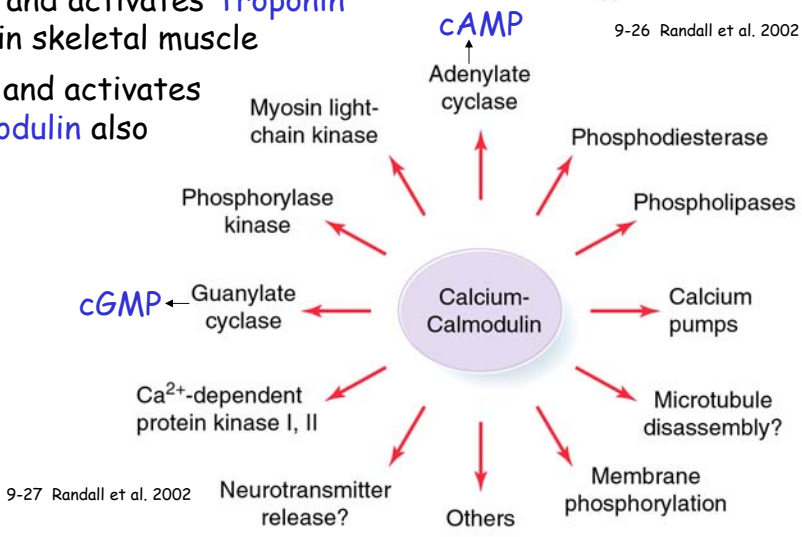
- Intracellular stores (ER)
- Influx from extracellular fluid

1- binds and activates Troponin C in skeletal muscle

2- binds and activates Calmodulin also




9-26 Randall et al. 2002




9-27 Randall et al. 2002

# Multiple Cleavage Events to Make Active Hormone

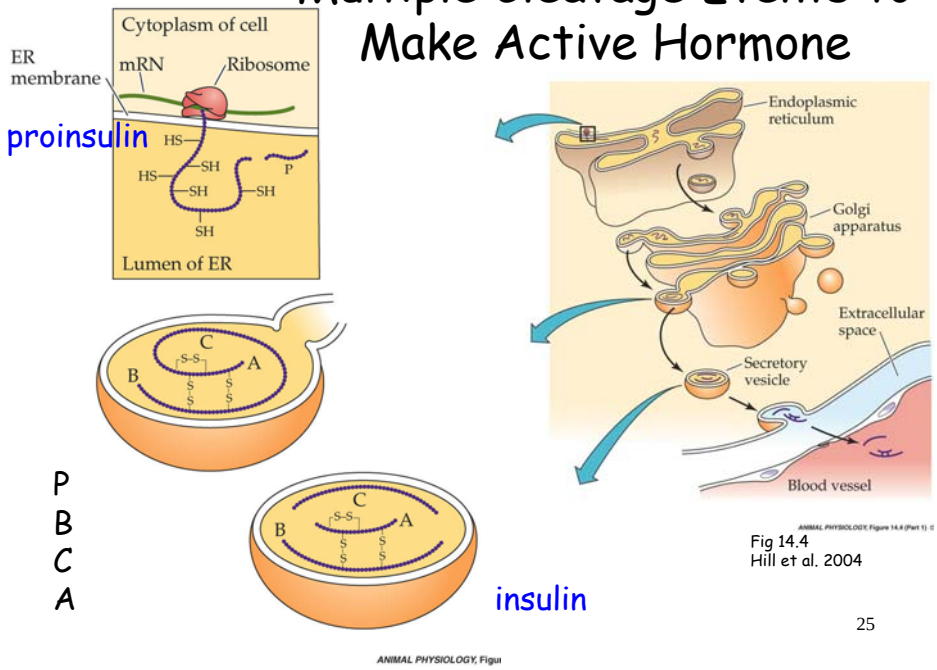



Fig 14.4
Hill et al. 2004

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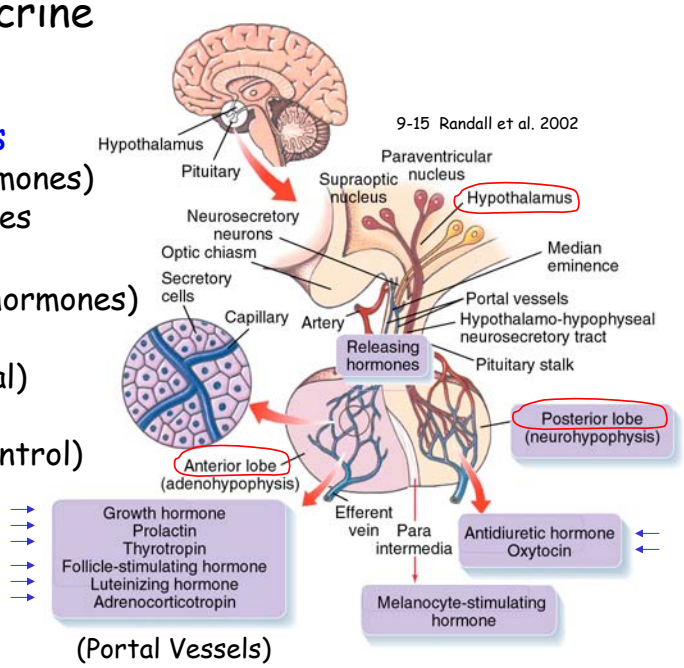
# Major Endocrine Players

## Hypothalamus
- (~7 neurohormones)
- mostly peptides

## Pituitary (9 hormones)
1. *Anterior*
   (~nonneuronal)
2. *Posterior*
   (~neuronal control)




9-15 Randall et al. 2002

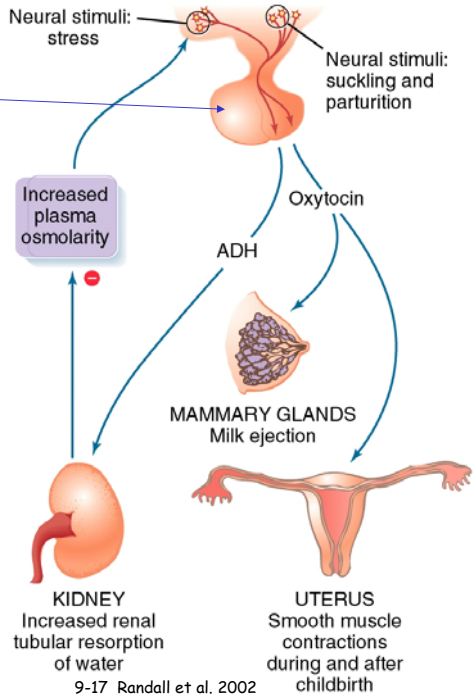
# Posterior Pituitary

Oxytocin – uterine contractions, milk ejection

Antidiuretic Hormone (ADH) - water retention in kidney

Both are peptide hormones

Both are similar and highly conserved…



9-17 Randall et al. 2002

# Posterior Pituitary

Randall et al. 2002

*Table 9-4* Variant forms of neurohypophyseal nonapeptide hormones

| Peptide | Positions of amino acid residues* 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | Animal group |
|---|---|---|---|---|---|---|---|---|---|---|
| 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 ADH |
| 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 |
| Glumitocin | 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.

Similar, highly conserved peptide hormones

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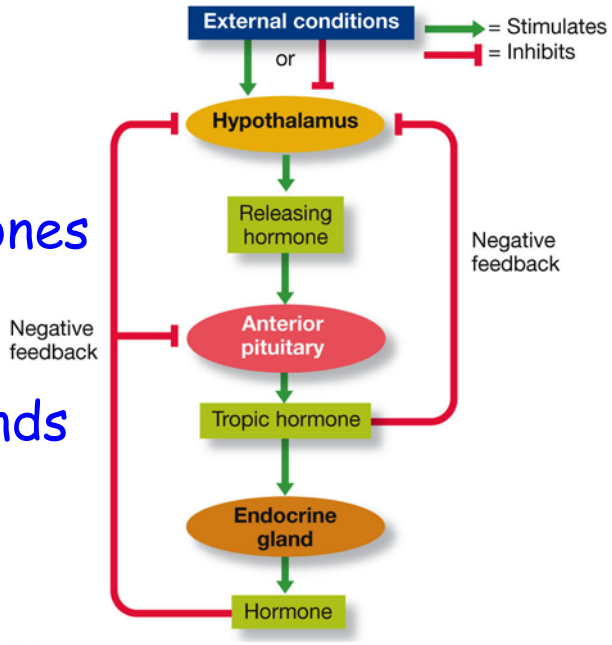
## Tropic Hormones

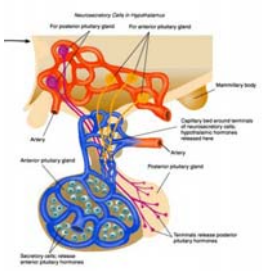
## Multiple Glands

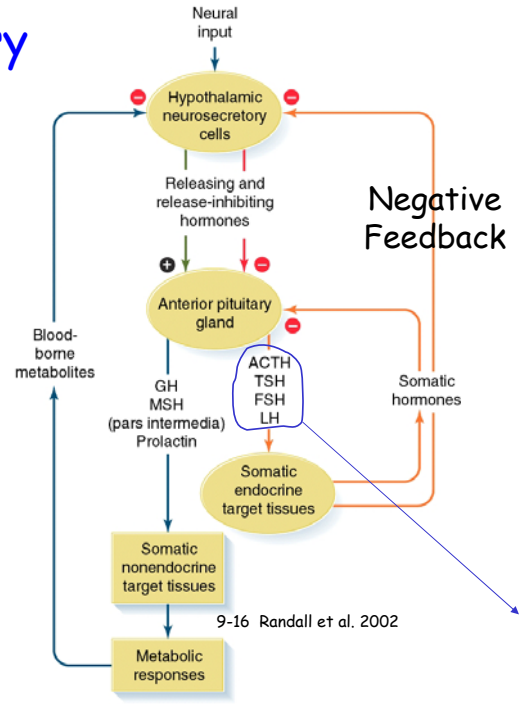



## Anterior Pituitary

*Tropic* hormones = act on other endocrine tissues

(e.g., adrenal cortex, thyroid, gonads)





# Tropic Hormones of Anterior Pituitary

*Table 9-3* Tropic hormones of the anterior pituitary gland

| Hormone | Structure | Target tissue | Primary action in mammals | Regulation* |
|---|---|---|---|---|
| Adrenocorticotropic hormone (ACTH) 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) 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) 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) 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.

Randall et al. 2002

In response to cold, fright, pain etc…

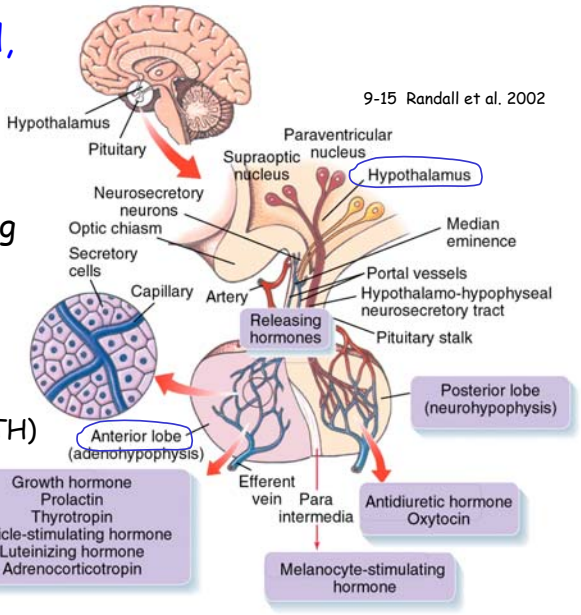
Hypothalamus
-corticotropin-releasing hormone

Portal vessel

Anterior Pituitary
-Adrenocorticotropin (ACTH)

Blood Stream

Adrenal Cortex
(corticosterone; stress hormones)




9-15 Randall et al. 2002

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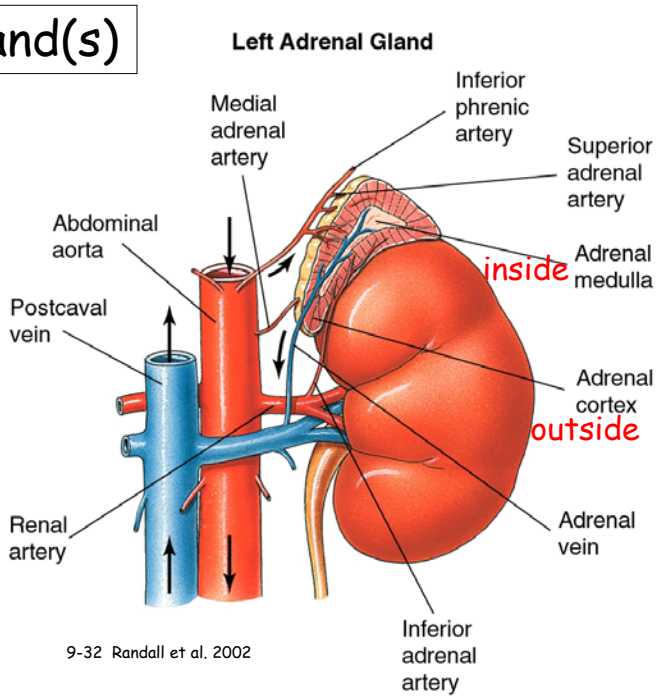
# Adrenal Gland(s)

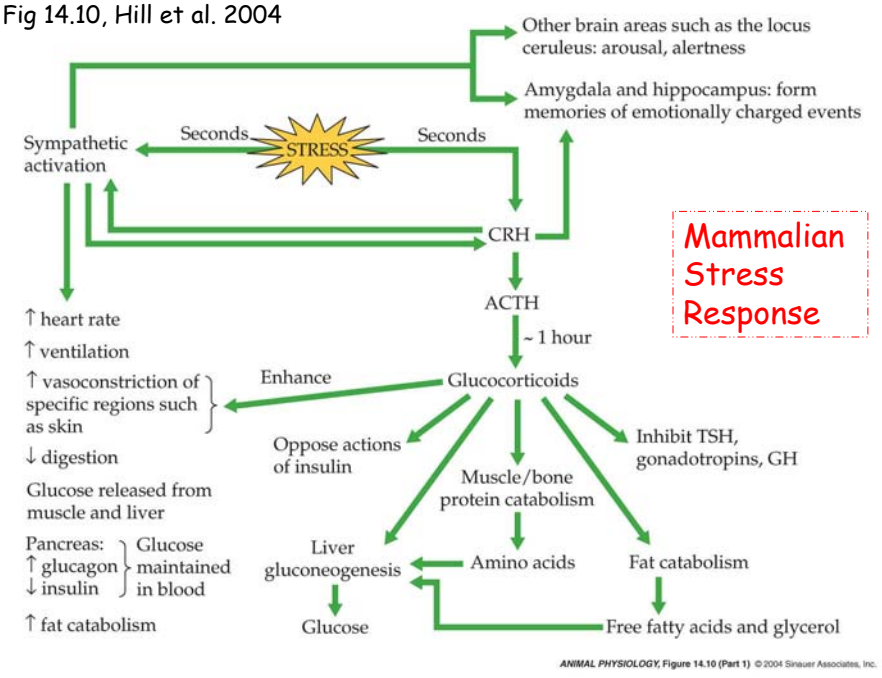
Catecholamines
(from medulla)

Glucocorticoids
(from cortex)




9-32 Randall et al. 2002

Fig 14.10, Hill et al. 2004




Mammalian Stress Response

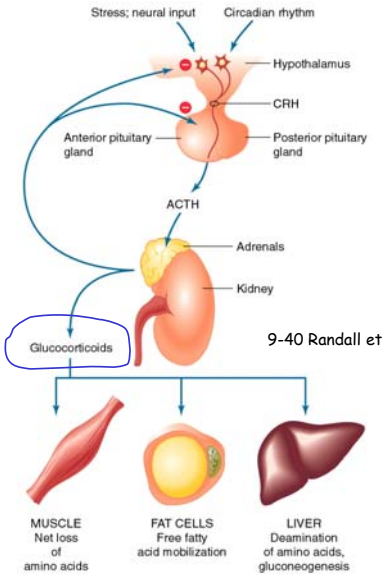
## Glucocorticoids

(from adrenal cortex)

Includes 'Stress Hormones'
Cortisol, cortisone, corticosterone

Released in response to ACTH (Adrenocorticotropin) from Anterior Pituitary

Steroid hormones (lipid soluble) derived from cholesterol




9-40 Randall et al. 2002

(mobilize a.a.s and glucose, suppress immune system etc.)

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Vasopressin from Anterior Pituitary

Blood loss:
↑ vasopressin → ↑ water reabsorption at kidney
↑ aldosterone → ↑ Na reabsorption at kidney

↓

↑ fluid retention

↓

↑ blood volume

↓

↑ blood pressure

Negative Feedback Loop

Fig 14.10, Hill et al. 2004

More on aldosterone and other kidney hormones later

## Water and Electrolyte Balance Hormones

Randall et al. 2002

*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 cAMP pathway | 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 |

Post. Pit., signal from Hypothal

Calcitonin decrease $Ca^{++}$ in blood; PTH increase $Ca^{++}$ in blood

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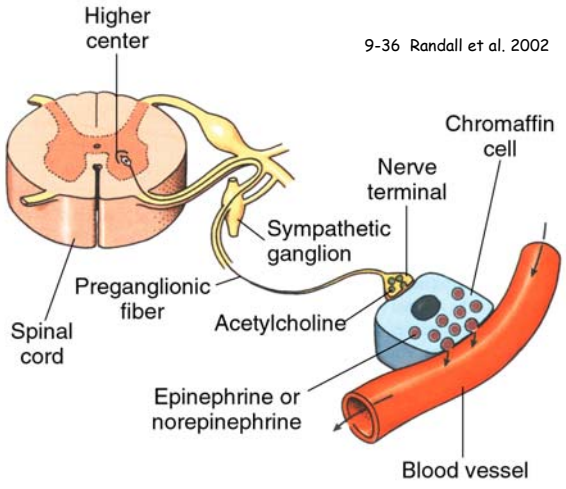
Fight/flight, exertion

Catecholamines

(adrenal medulla)

- Epi, Norepi

Secretion regulated by sympathetic preganglionic cholinergic neurons




9-36 Randall et al. 2002

Catecholamine receptors are Alpha and Beta adrenoreceptors

Alpha – smooth muscle contraction

Beta1 – cardiac muscle stimulant

Beta2 – broncho/vaso dilation

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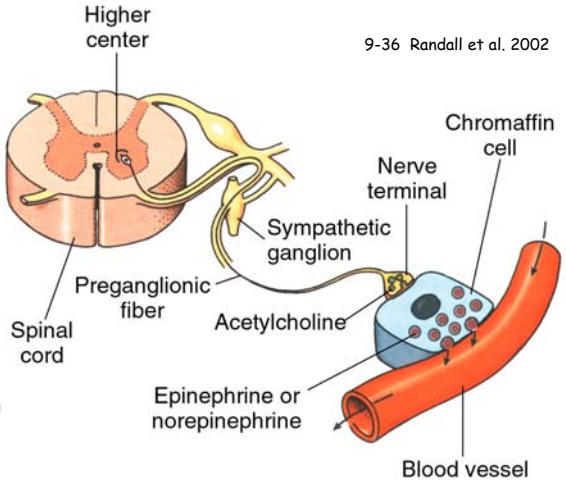
# Fight/Flight

Catecholamines from adrenal medulla (e.g. epinephrine)




9-36 Randall et al. 2002

Secretion regulated by sympathetic neurons

Response includes bronchodilation, vasodilation to major muscles, vasoconstriction to stomach and skin, increased heart rate, mobilization of resources

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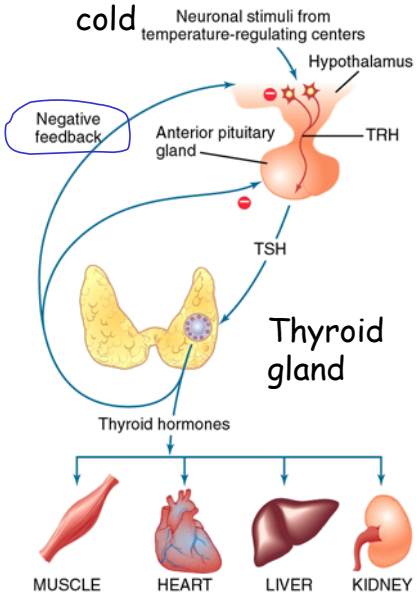
# Thyroid Hormone (aka Thyroxine)

$T_3$ and $T_4$ (# of iodines)

lipid soluble

Development, maturation, protein synthesis, metabolism

Being cold can stimulate




9-42 Randall et al. 2002

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# Thyroxine and Thyroid Gland

Hypothyroid Goiter
(e.g. not enough iodine in diet)

Hyperthyroid Goiter
(e.g. 1. thyroxine receptors on hypothalamus or anterior pituitary don't work,
or
2. receptor for thyroid stimulating hormone is activated by autoimmune antibody )

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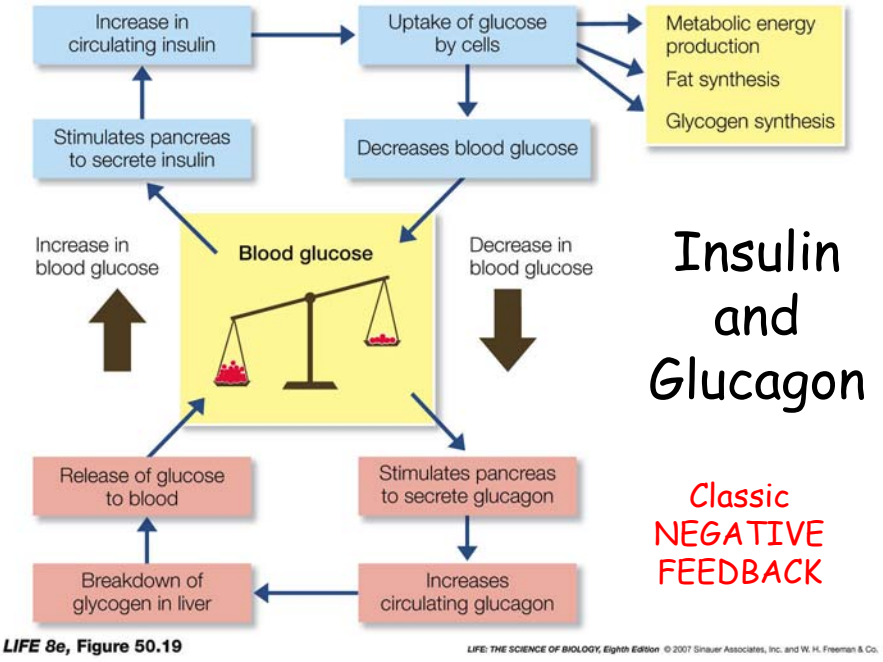


Insulin and Glucagon

Classic NEGATIVE FEEDBACK

## Insulin and Glucagon Regulate blood [glucose]

- Insulin in response to high [glu]
- Glucagon in response to low [glu]

Both from pancreatic gland:
Insulin from beta cells
Glucagon from alpha cells

Leads to glucose uptake into tissues ETC.

Causes glycogenolysis and glucose release from tissues (liver, muscles)

Type 1 Diabetes
-when beta cells decrease insulin production
Type 2 Diabetes
-when insulin receptor signal pathway defective

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# Growth Hormone

Metabolic and developmental effects

From Anterior Pituitary

Often has opposite effect of insulin

-Stimulates gluconeogenesis
-Reserves glucose for NS
-Promotes use of fatty acids as fuel

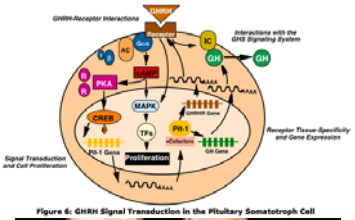


Figure 6: GHRH Signal Transduction in the Pituitary Somatotroph Cell

Works with Thyroid hormone in growth and development

abnormality

Gigantism / Acromegaly

## Growth Hormone

\+ -Gigantism
-Acromegaly

– Dwarfism

## Vertebrate Reproduction

Asexual
Parthenogenetic
Whiptail
Lizards

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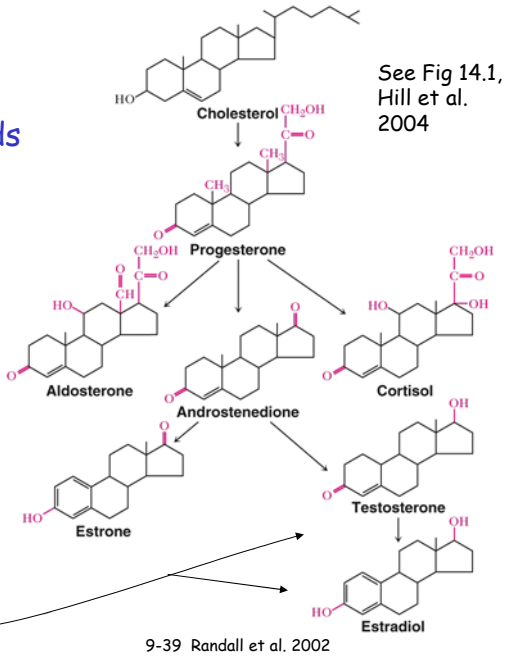
## Reproductive Hormones

Steroid Hormones from Adrenal cortex and Gonads

Hypothalamus
↓
GnRH
↓
Anterior Pituitary
↓
FSH, LH
↓
Gonads, Adrenal Cortex




See Fig 14.1, Hill et al. 2004

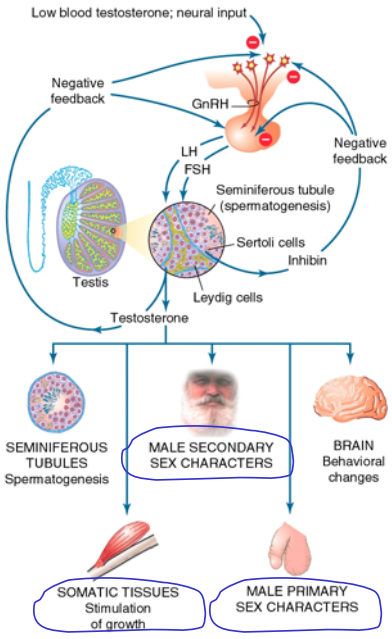
9-39 Randall et al. 2002

Male Sex Hormones

Testosterone and other androgens

Released from Leydig Cells in response to LH, FSH

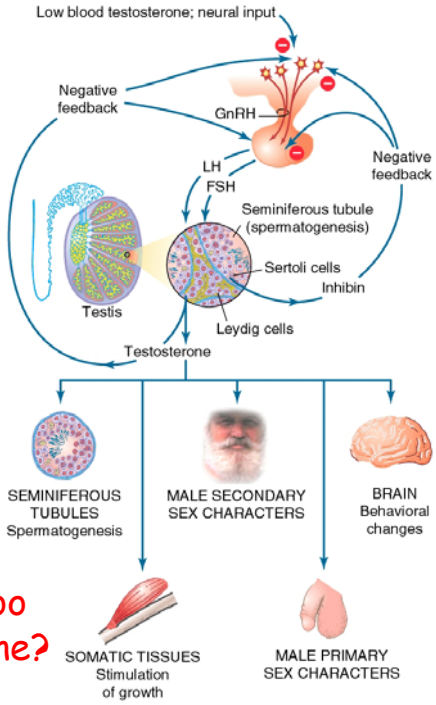
FSH binding to Sertoli cells stimulates spermatogenesis




9-46 Randall et al. 2002

# Steroid Abuse

Homeostasis & Negative Feedback




What happens if you take too much exogenous testosterone?

Female Sex Hormones

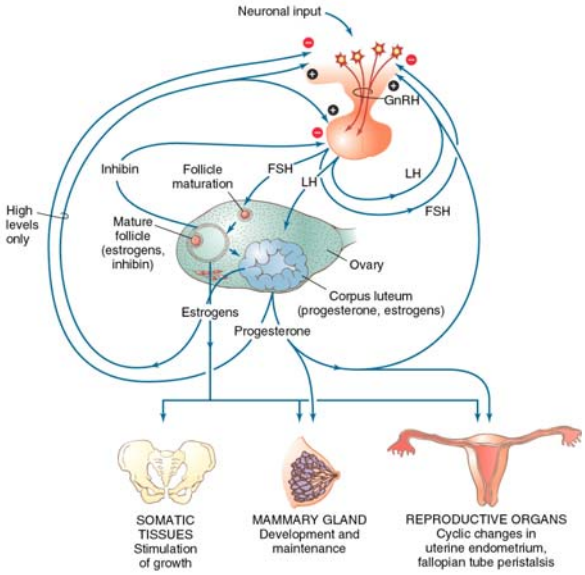
## Estrogens

Ova created and stored before birth (mammals and birds)

Repro cycle in 2 phases:

Follicular and Luteal

FSH stimulates beginning of follicular phase -> development of ovarian follicles




9-47 Randall et al. 2002



## Follicular Phase

1- FSH ⇑

Causing follicular maturation

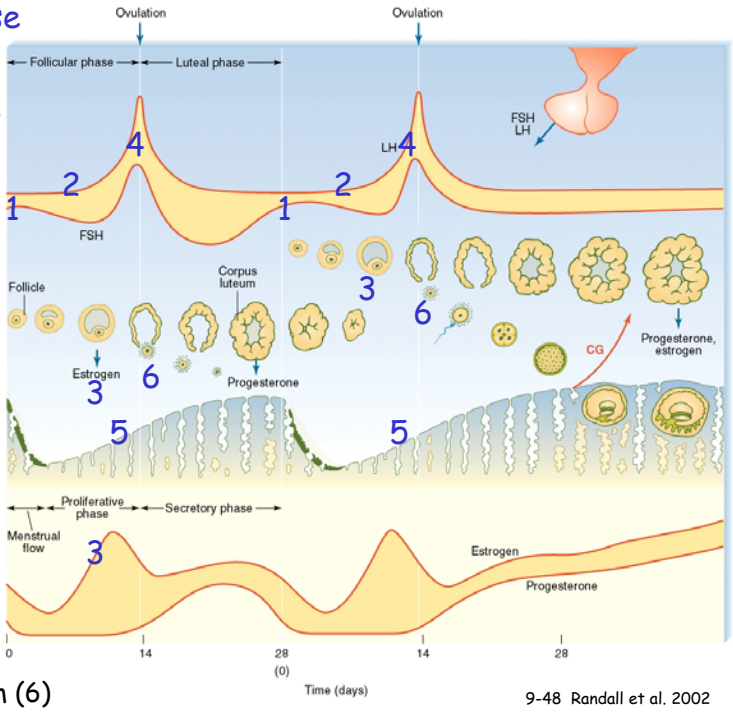
2- LH ⇑

FSH and LH help generate Estrogen (3)

This Estrogen causes hypothal and ant. pit. to release spike of LH and FSH leading to ovulation (4)

(endometrium 5)

= release of ovum (6)




9-48 Randall et al. 2002

## Luteal Phase

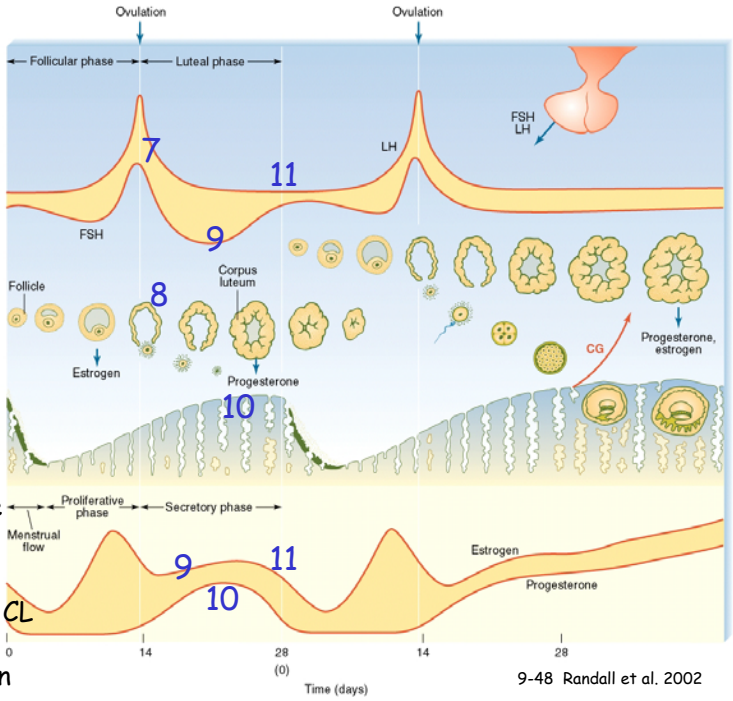
7- FSH, LH ⇓
Estrogen ⇓

8- LH changes ruptured follicle into corpus luteum

9- corpus luteum secretes estrogen and progesterone which inhibit FSH, LH release from anterior pituitary by slowing GnRH

10- progesterone leads to richer endometrium

11- without fert. CL degenerates and cycle begins again

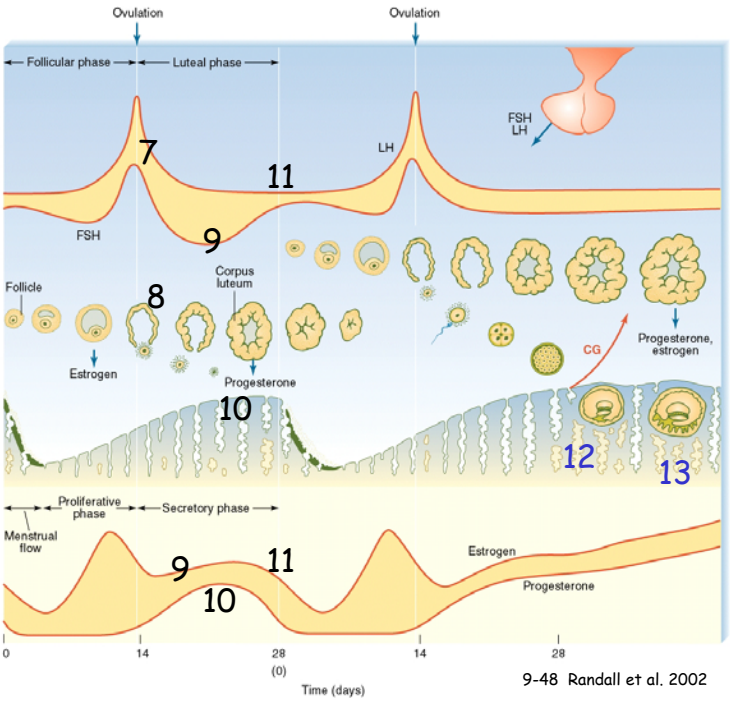



9-48 Randall et al. 2002

## Luteal Phase

12- with fert. Chorionic gonadotropin maintains CL, maintaining high levels of estrogen and progesterone; maintaining endometrium; follicular development inhibited

13- Placenta takes over hormone production




9-48 Randall et al. 2002

Dear Cecil:
I just hit 40 and still have an unanswered question from my teens that has always bothered me. Aerosmith in "Sweet Emotion" sings about being accused of impregnating a girl. At the end of the verse they sing, "can't catch me 'cause the rabbit done died," referring to a pregnancy test. Arguments start over just what this test entails. Most say the bunny will die, but can't agree as to why. Some say the rabbit will always die because they kill it before they take its blood (which seems pretty dumb). Some say they inject it with some fluid taken from the woman and it dies a horrible, convulsive death. And some say they have to dissect the rabbit after it has been injected. This was before those home test kits, but wasn't there a better way? --Joe Shredl, Colonial Heights, Virginia

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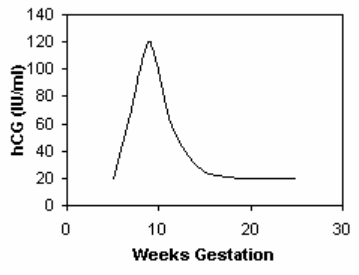
Chorionic gonadotropin
hCG
eCG

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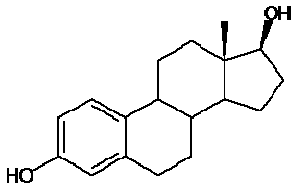
## Birth Control Pills?

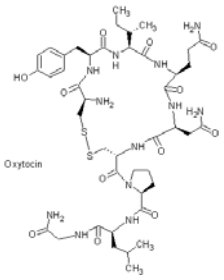



Progesterone and Estradiol – mimic early pregnancy and inhibit ovulation

## Parturition (Birth)

Oxytocin released in response to cervical stretch

Oxytocin causes uterine smooth muscle contractions




Positive Feedback Loop

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## Lactation

Decreased progesterone levels and presence of prolactin (milk production) and oxytocin (milk ejection) and other hormones

Antibodies
Vitamins

joey

Mechanosensory feedback

Dopamine inhibits prolactin secretion

marsupial

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Randall et al. 2002

*Table 9-9* **Important mammalian reproductive hormones**

| Hormone | Tissue of origin | Structure | Target tissue | Primary action | Regulation |
|---|---|---|---|---|---|
| **Primary sex hormones** | | | | | |
| Estradiol-17$\beta$ (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 |