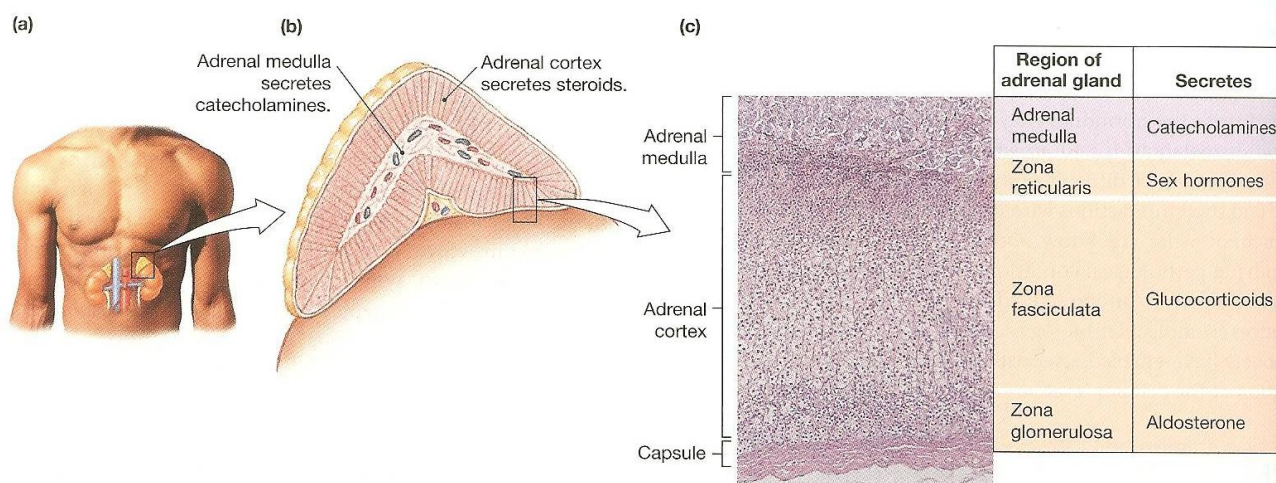
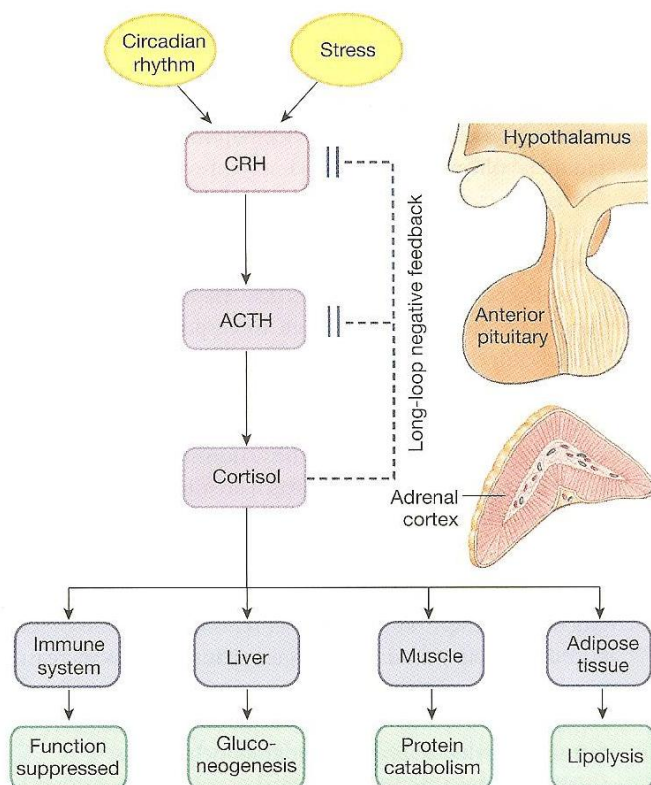


ADRENAL GLUCOCORTICOIDS (Cortisol)

- I. Adrenal Gland Secretions
 - a. Adrenal Medulla
 - i. Secretes Catecholamine's (Epinephrine)
 - b. Adrenal Cortex
 - i. Secretes Steroid hormones
 - ii. Cortisol is the main glucocorticoid secreted by the adrenal cortex



- II. Cortisol Secretion is Controlled by CRH ACTH
 - a. Control Pathway of Cortisol secretion is known as Hypothalamic-pituitary-adrenal (HPA) pathway
 - b. CRH: Corticotropin-releasing hormone
 - i. Secreted into hypothalamic-hypophyseal portal system to be transported into anterior pituitary gland (tropic gland)
 - c. ACTH: Adrenocorticotrophic Hormone
 - i. Activated by CRH presence in anterior pituitary
 - ii. ACTH acts on the adrenal cortex to promote synthesis and release of Cortisol
 - d. Cortisol
 - i. Acts as a Long Loop Negative Feedback signal, inhibiting CRH and ACTH secretions when adequate Cortisol levels are achieved
 - ii. Cortisol secretion peaks in the morning and diminishes at night
 - iii. Cortisol secretion increases with stress



III. Cortisol effect's on Tissue

- a. Most important metabolic effect of Cortisol is its protective effect against *hypoglycemia*
 - i. In the absence of Cortisol, glucagon (peptide hormone that allows glucose secretion from glycogen) is unable to respond to low levels of glucose
- b. Promotes Gluconeogenesis
 - i. Gluconeogenesis of the liver
 - ii. Increases systemic blood glucose concentrations by having the liver produce/release excess glucose
- c. Cortisol Causes Breakdown of Skeletal Muscle Proteins
 - i. Breaks down skeletal muscle proteins to provide substrates for Gluconeogenesis
- d. Enhances Lipolysis
 - i. Enhances adipose breakdown so free fatty acids are available to peripheral tissues for energy use
- e. Suppresses the Immune System
- f. Causes Negative Calcium Balance
 - i. Decreases intestinal calcium absorption
 - ii. Increases renal calcium excretion
 - iii. Causing calcium loss from body
 - iv. Catabolic in bone tissue; breaking down the calcified bone matrix
- g. Brain Function
 - i. Excess or deficiency causes mood swings or memory/learning altercations

Cortisol	
Organ/System	Effect
Immune system (including thymus)	Decreases
Plasma Glucose	Promotes/Increases
Muscle	Catabolism
Adipose	Catabolism

IV. Exogenous Stimulation of Glucocorticoid Pathway

a. Exogenous Cortisol

i. Causes atrophy of

1. Adrenal Cortex
2. Anterior Pituitary

[Hypothalamus probably not effected]

b. Exogenous ACTH

i. Causes atrophy of

1. Anterior Pituitary

[Adrenal Cortex does not atrophy because it is still being stimulated by Cortisol, even though its exogenous; Hypothalamus probably not effected]

V. Hypercortisolism

Definition: Ecess Cortisol in the body

Symptoms: Excess gluconeogenesis causes hyperglycemia (diabetes like symptoms), tissue wasting, excess Cortisol deposits in trunk and face

Classic Symptoms: Thin arms and legs; obesity in the trunk, and a moon face with plum red cheeks

Cushing Disease: Hypercortisolism from any cause

a. Hyperadrenocorticism (Primary Disorder)

- i. Adrenal tumor that autonomously secretes Cortisol
- ii. Not under control of pituitary ACTH
- iii. Primary Hypercortisolism
 1. Causes long negative feedback loop to shut off CRH and ACTH



b. Cushing Syndrome

- i. Pituitary tumor that autonomously secretes ACTH
- ii. Excess ACTH thus causing over-secretion of Cortisol
- iii. Second Hypercortisolism

c. Ectopic ACTH Syndrome

- i. Non-pituitary ACTH producing tumor

VI. Hypocortisolism

Definition: Hyposecretion of Cortisol

a. Addison's Disease

- i. Hyposecretion of all adrenal steroid hormones
- ii. Usually induced by autoimmune destruction of adrenal cortex