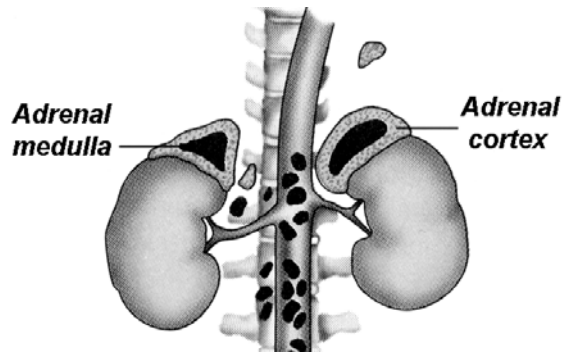


## INTRODUCTION

### A. Functional Anatomy

1. Two glands: adrenal cortex and adrenal medulla
2. Located on the superior pole of the kidneys



### B. Hormones

1. Adrenal cortex: steroid hormones (synthesized from cholesterol)
  - a. glucocorticoids (e.g. cortisol); named for their effects on carbohydrate metabolism but note they have many other effects
  - b. mineralocorticoids (e.g. aldosterone); named for their effect on mineral (Na, Cl, and K) excretion by the kidneys
  - c. sex hormones (e.g. testosterone); normally relatively low output compared to the male testes
2. Adrenal medulla: catecholamines
  - a. epinephrine (adrenalin)
  - b. norepinephrine (noradrenalin)

Note: Mineralocorticoid actions covered with the kidney; Sex hormone action covered with the reproductive system; Adrenal medulla hormone action covered with the autonomic nervous system, so these notes will concentrate on adrenal cortex glucocorticoids

## GLUCOCORTICOIDS

### A. Major Hormones

1. Cortisol
2. Corticosterone
3. Cortisone

### B. Physiological Actions (normal concentrations)

1. Gluconeogenesis: conversion of proteins to glucose
  - a. breakdown of proteins to amino acids in several tissues and release of the amino acids into the circulation
  - b. uptake of amino acids by the liver
  - c. synthesis of glucose from amino acids in the liver
  - d. release of glucose from the liver into the blood
2. Lipolysis: breakdown of lipids (stored in adipose tissue) into free fatty acids (FFA); FFA can be used as a metabolic substrate
3. Vascular reactivity: maintain responsiveness of vascular smooth muscle to catecholamines
4. Renal water excretion: maintains ability of the kidney to excrete water by a permissive effect on glomerular filtration
5. Resistance to "stress". Cortisol and related hormones are not essential for life but are necessary for the normal response to "stress"
6. Preservation of normal mentation

### C. Pharmacological Actions (high concentrations)

1. Anti-inflammatory (e.g. blocks deposition of fibrotic tissue)
2. Anit-allergic (reduces histamine release)
3. Anti-immune system (e.g. used in organ transplant patients to reduce rejection)

Note: The above actions make individuals receiving high doses of glucocorticoids especially susceptible to infection

## CONTROL OF GLUCOCORTICOID SECRETION

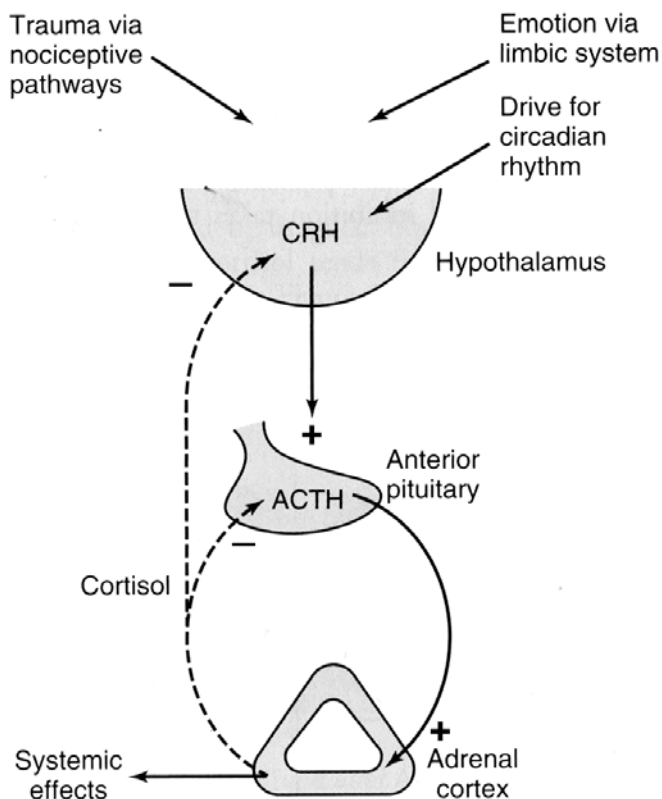
### A. Anterior Pituitary

1. Releases ACTH (adrenocorticotropic hormone)
2. Actions of ACTH
  - a. increase glucocorticoid synthesis and release
  - b. adrenal cortex hypertrophy (increase size) and hyperplasia (increase number of cells)

### B. Hypothalamus

1. Secretes CRH (corticotropin releasing hormone), which is transported to the anterior pituitary
2. CRH stimulates release of ACTH
3. Input to the hypothalamus influencing CRH release; many, among which are
  - a. neurons involved in mediating diurnal (circadian) rhythms (most ACTH is released in the several hours in early morning before and after waking)
  - b. collaterals from nociceptive pathways
  - c. limbic system neurons (associated with emotion)

### C. Feedback Control: long loop negative feedback



## PATHOPHYSIOLOGY OF OVERSECRETION

### A. Causes

1. Glucocorticoid producing tumor of the adrenal cortex
2. ACTH producing tumor of the anterior pituitary
3. Loss of pituitary sensitivity to glucocorticoid inhibition
4. Administration of excess corticosteroids

### B. Results: Cushing's Syndrome

1. Muscle wasting (protein breakdown > protein synthesis)
2. Poor wound healing (same as 1 above)
3. Hyperglycemia (gluconeogenesis)
4. Osteoporosis (resorption of bone matrix proteins)
5. Redistribution of body fat (round, puffy face)
6. Edema

