Endocrine System: Overview

- Endocrine system – the body’s second great controlling system which influences metabolic activities of cells by means of hormones
Endocrine System: Overview

- Endocrine glands – pituitary, pineal, thyroid, parathyroid, thymus, and adrenal
- The pancreas and gonads produce both hormones and exocrine products
- Other tissues and organs that produce hormones – adipose cells, pockets of cells in the walls of the small intestine, stomach, kidneys, and heart

Endocrine System: Overview

- The hypothalamus has both neural functions and releases hormones
- Interacts with the nervous system & immune system to regulate & coordinate body activities (maintains homeostasis)
Autocrines and Paracrines

- Autocrines – chemicals that exert effects on the same cells that secrete them
- Paracrines – locally acting chemicals that affect cells other than those that secrete them
- These are not considered hormones since hormones are long-distance chemical signals
Hormones

- Hormones – chemical substances secreted by cells into the extracellular fluids
  - Regulate the metabolic function of other cells via receptors
  - Have lag times ranging from seconds to hours
  - Tend to have prolonged effects
  - Are classified as amino acid-based hormones, or steroids
- Eicosanoids – biologically active lipids with local hormone–like activity

Types of Hormones

- Amino acid based
  - Amines, thyroxine, peptide, and protein hormones
- Steroids – gonadal and adrenocortical hormones
- Eicosanoids – include
  - Leukotrienes
  - Prostacyclin
  - Thromboxanes
  - Prostaglandins
Hormone Action

- Hormones alter target cell activity by one of two mechanisms
  - Second messengers:
    - Regulatory G proteins
      - cAMP mechanism
    - Amino acid–based hormones
      - PIP₂ – Ca²⁺ mechanism
  - Direct gene activation
    - Steroid & thyroid hormones
    - Tyrosine Kinase activation
  - The precise response depends on the type of the target cell
What is a G protein?

- Belongs to a family of guanine nucleotide binding proteins
- Consists of 3 subunits (α, β, and γ)
- Inactive when GDP is bound
- Active when GTP is bound
What is cAMP?

- Formation and breakdown of cAMP
- ↑ cAMP levels by activating adenylate cyclase
- ↓ cAMP levels by inhibiting adenylate cyclase

2nd messenger cAMP mechanism (↑ cAMP)

- Hormone (first messenger) binds to its receptor, which then binds to a G protein (α subunit)
- The G protein is then activated as it binds GTP, which displaces GDP
- Activated G protein activates the effector enzyme adenylate cyclase (AC)
- AC generates cAMP (second messenger) from ATP
- cAMP activates protein kinases, which then cause cellular effects
Cellular Effects

- Hormones produce one or more of the following cellular changes in target cells
  - Alter plasma membrane permeability
  - Stimulate protein synthesis
  - Activate or deactivate enzyme systems
  - Induce secretory activity
  - Stimulate mitosis

2nd messenger cAMP mechanism (↑ cAMP)
- Glucagon binds to its receptor which uses the 2nd messenger cAMP
- cAMP activates protein kinases like glycogen phosphorylase (see diagram)

**Another 2nd messenger**

- Formation and breakdown of cGMP
2nd messenger cAMP mechanism (↓ cAMP)

Enzyme Amplification

Small stimulus

Number of molecules

1
100 each
10,000
1,000,000

Hormone

cAMP and protein kinase

Activated enzymes

Metabolic product

Great effect
Protein phosphorylation

- is a common means of information transfer. Many second messengers elicit responses by activating protein kinases. These enzymes transfer phosphoryl groups from ATP to specific serine, threonine, and tyrosine residues in proteins.

Protein + ATP + Protein kinase → Protein + ADP + phosphate
**2nd messenger PIP$_2$ – Ca$^{2+}$ mechanism**

- Hormone binds to the receptor and activates G protein (α subunit)
- G protein binds and activates phospholipase C (PL-C)
- PL-C splits the phospholipid PIP$_2$ into diacylglycerol (DAG) and IP$_3$ (both act as second messengers)
- DAG activates protein kinases (PKC) which phosphorylates & activates proteins
- IP$_3$ triggers release of Ca$^{2+}$ stores
- Ca$^{2+}$ (third messenger) alters cellular responses
Tyrosine Kinase

Steroid and Thyroid Hormones

- This interaction prompts DNA transcription to produce mRNA
- The mRNA is translated into proteins, which bring about cellular effects
Hormone Effects on Gene Activity

(a) Steroid hormone

1. Diffusion through membrane lipids
2. Binding of hormone to cytoplasmic or nuclear receptors
3. Alteration of cellular structure or activity
4. Receptor-hormone complex binds to DNA
5. Binding of hormone - receptor complex to DNA
6. Gene activation

(b) Thyroid hormone

1. Diffusion or transport across cell membrane
2. Binding to receptors at mitochondria and nucleus
3. Increased ATP production
4. Alteration of cellular activity
5. Binding to DNA

New protein

**Target Cell Specificity**

- Hormones circulate to all tissues but only activate cells referred to as target cells
- Target cells must have specific receptors to which the hormone binds
- These receptors may be intracellular or located on the plasma membrane

**Target Cell Specificity**

- Examples of hormone activity
  - ACTH receptors are only found on certain cells of the adrenal cortex
  - Thyroxin receptors are found on nearly all cells of the body
Target Cell Activation

- Target cell activation depends on three factors
  - Blood levels of the hormone
  - Relative number of receptors on the target cell
  - The affinity of those receptors for the hormone
- Up-regulation – target cells form more receptors in response to the hormone
- Down-regulation – target cells lose receptors in response to the hormone

Modulation of Target Cell Sensitivity

(a) Up-regulation
- Low receptor density
  - Weak response
- Increased receptor density
  - Increased sensitivity
  - Stronger response

(b) Down-regulation
- High receptor density
  - Strong response
- Reduced receptor density
  - Reduced sensitivity
  - Diminished response
Hormone Concentrations in the Blood

- Hormones circulate in the blood in two forms – free or bound
  - Steroids and thyroid hormone are attached to plasma proteins
  - All others are unencumbered or free

- Concentrations of circulating hormone reflect:
  - Rate of release
  - Speed of inactivation and removal from the body

- Hormones are removed from the blood by:
  - Degrading enzymes
  - The kidneys
  - Liver enzyme systems
**Interaction of Hormones at Target Cells**

- Three types of hormone interaction
  - Permissiveness – one hormone cannot exert its effects without another hormone being present
  - Synergism – more than one hormone produces the same effects on a target cell
  - Antagonism – one or more hormones opposes the action of another hormone

**Control of Hormone Release**

- Blood levels of hormones:
  - Are controlled by negative feedback systems
  - Vary only within a narrow desirable range
- Hormones are synthesized and released in response to:
  - Humoral stimuli
  - Neural stimuli
  - Hormonal stimuli
Humoral Stimuli

- Humoral stimuli – secretion of hormones in direct response to changing blood levels of ions and nutrients
- Example: concentration of calcium ions in the blood
  - Declining blood Ca\(^{2+}\) concentration stimulates the parathyroid glands to secrete PTH (parathyroid hormone)
  - PTH causes Ca\(^{2+}\) concentrations to rise and the stimulus is removed
Neural Stimuli

- Neural stimuli – nerve fibers stimulate hormone release
  - Preganglionic sympathetic nervous system (SNS) fibers stimulate the adrenal medulla to secrete catecholamines

Hormonal Stimuli

- Hormonal stimuli – release of hormones in response to hormones produced by other endocrine organs
  - The hypothalamic hormones stimulate the adenohypophysis
  - In turn, pituitary hormones stimulate targets to secrete still more hormones
Hormonal Stimuli

Nervous System Modulation

- Remember: the nervous system modifies the stimulation of endocrine glands and their negative feedback mechanisms
Nervous System Modulation

- The nervous system can override normal endocrine controls
  - For example, control of blood glucose levels
    - Normally the endocrine system maintains blood glucose
    - Under stress, the body needs more glucose
    - The hypothalamus and the sympathetic nervous system are activated to supply ample glucose

Three Methods of Hypothalamic Control
Major Endocrine Organs: Hypophysis

- Hypophysis (pituitary gland) – two-lobed organ that secretes nine major hormones
- Neurohypophysis – pars nervosa (posterior portion), infundibulum, and the median eminence.
  - Receives, stores, and releases hormones from the hypothalamus
- Adenohypophysis – pars distalis (anterior portion), pars intermedia (intermediate portion), and the pars tuberalis
  - Synthesizes and secretes a number of hormones

Anatomy and Orientation of the Hypophysis

![Anatomy and Orientation of the Hypophysis](image)
Anatomy and Orientation of the Hypophysis

Hypothalamic Control of Hypophysis
Pituitary-Hypothalamic Relationships: Neurohypophysis (posterior portion)

- The neurohypophysis is a downgrowth of hypothalamic neural tissue
- Has a neural connection with the hypothalamus (hypothalamic-hypophyseal tract)
- Nuclei of the hypothalamus synthesize oxytocin and antidiuretic hormone (ADH)
- These hormones are transported to the posterior pituitary

Pituitary-Hypothalamic Relationships: Adenohypophysis (anterior portion)

- The adenohypophysis of the pituitary is an outpocketing of the oral mucosa
- There is no direct neural contact with the hypothalamus
There is a vascular connection, the hypophyseal portal system, consisting of:

- The primary capillary plexus
- The hypophyseal portal veins
- The secondary capillary plexus
Adenohypophyseal Hormones

- The six hormones of the adenohypophysis:
  - Abbreviated as GH, TSH, ACTH, FSH, LH (ICSH), and PRL
  - Regulate the activity of other endocrine glands
- In addition, pro-opiomelanocortin (POMC):
  - Has been isolated from the pituitary
  - Is split into ACTH, opiates, and MSH
Activity of the Adenohypophysis

- The hypothalamus sends a chemical stimulus to the anterior pituitary
  - Releasing hormones (RH) stimulate the synthesis and release of hormones
  - Inhibiting hormones (IH) shut off the synthesis and release of hormones
Activity of the Adenohypophysis

- The tropic hormones that are released are:
  - Thyroid-stimulating hormone (TSH)
  - Adrenocorticotropic hormone (ACTH)
  - Follicle-stimulating hormone (FSH)
  - Luteinizing hormone (LH) also known as interstitial cell stimulating hormone (ICSH)

Growth Hormone (GH)

- Produced by somatotropic cells of the pars distalis that:
  - Stimulate most cells, but target bone and skeletal muscle
  - Promote protein synthesis and encourage the use of lipids for fuel (glucose sparing)
  - Most effects are mediated indirectly by somatomedins
**Growth Hormone (GH)**

- Antagonistic hypothalamic hormones regulate GH
  - Growth hormone–releasing hormone (GHRH) stimulates GH release
  - Growth hormone–inhibiting hormone (GHIH) inhibits GH release

**Metabolic Action of Growth Hormone**

- GH stimulates liver, skeletal muscle, bone, and cartilage to produce insulin-like growth factors
- Direct action promotes lipolysis and inhibits glucose uptake
Metabolic Action of Growth Hormone (GH)

Growth Hormone Disorders

- Hypersecretion:
  - Giantism
  - Acromegaly
- Hyposcretion:
  - Dwarfism
Growth Hormone Abnormalities

Thyroid Stimulating Hormone (Thyrotropin)

- Stimulates the normal development and secretory activity of the thyroid
- Triggered by hypothalamic peptide thyrotropin-releasing hormone (TRH)
- Rising blood levels of thyroid hormones act on the pituitary and hypothalamus to block the release of TSH
Adrenocorticotropic Hormone (Corticotropin)

- Stimulates the adrenal cortex to release corticosteroids
- Triggered by hypothalamic corticotropin-releasing hormone (CRH) in a daily rhythm
- Internal and external factors such as fever, hypoglycemia, and stressors can trigger the release of CRH

Gonadotropins

- Gonadotropins – follicle-stimulating hormone (FSH) and luteinizing hormone (LH)
  - Regulate the function of the ovaries and testes
  - FSH stimulates gamete (egg or sperm) production
  - Absent from the blood in prepubertal boys and girls
  - Triggered by the hypothalamic gonadotropin-releasing hormone (GnRH) during and after puberty
Functions of Gonadotropins

- In females
  - LH works with FSH to cause maturation of the ovarian follicle
  - LH works alone to trigger ovulation (expulsion of the egg from the follicle)
  - LH promotes synthesis and release of estrogens and progesterone

- In males
  - LH stimulates interstitial cells of the testes to produce testosterone
  - LH is also referred to as interstitial cell-stimulating hormone (ICSH)
### Prolactin (PRL)

- In females, stimulates the development of mammary glands, initiates & maintains milk production by the breasts
- In males, enhances testosterone production
- Triggered by the hypothalamic prolactin-releasing hormone (PRH)
- Inhibited by prolactin-inhibiting hormone (PIH)

### Prolactin (PRL) cont.

- Blood levels rise toward the end of pregnancy
- Suckling stimulates PRH release and encourages continued milk production
Pituitary-Hypothalamic Relationships: Neurohypophysis

- There is a neural connection, the hypothalamic-hypophyseal tract system
Hypothalamic Control of Hypophysis

The Pars Nervosa & Hypothalamic Hormones

- Pars nervosa – made of axons of hypothalamic neurons, stores antidiuretic hormone (ADH) and oxytocin (OT)
- Both are synthesized in the hypothalamus
- ADH influences water balance & causes vasoconstriction
  - Uses cAMP mechanism
- OT stimulates smooth muscles associated with reproductive structures
  - Uses PIP$_2$-Ca$^{2+}$ mechanism
ADH

- ADH helps to avoid dehydration or water overload
  - Prevents urine formation
- Osmoreceptors monitor the solute concentration of the blood
- With high solutes, ADH preserves water
- With low solutes, ADH is not released, thus causing water loss
- Alcohol inhibits ADH release and causes copious urine output

ADH Disorders

- Hypersecretion:
  - Syndrome of Inappropriate ADH (SIADH)
- Hypossecretion:
  - Diabetes Insipidus
Oxytocin

- Regulated by a positive feedback mechanism to OT in the blood
- In women:
  - This leads to increased intensity of uterine contractions, ending in birth
  - OT triggers milk ejection (“letdown” reflex)
- Synthetic and natural OT drugs can be used to induce or hasten labor

Oxytocin

- In males:
  - Involved in ejaculation and sperm transport
  - Plays a role in sexual arousal and satisfaction in males and nonlactating females
Pineal Gland

- Small gland hanging from the roof of the third ventricle of the brain
- Secretory product is melatonin
- Melatonin is involved with:
  - Cyclic activities
  - Physiological processes that show rhythmic variations (body temperature, sleep, appetite)
Thyroid Gland

- The largest endocrine gland, located in the anterior neck, consists of two lateral lobes connected by a median tissue mass called the isthmus
- Composed of follicles that produce the glycoprotein thyroglobulin
- Colloid (thyroglobulin + iodine) fills the lumen of the follicles and is the precursor of thyroid hormone
- Other endocrine cells, the parafollicular cells (C cells), produce the hormone calcitonin
**Thyroid Hormone**

- Thyroid hormone – major metabolic hormone
- Consists of two related iodine-containing compounds
  - $T_4$ – thyroxine; has two tyrosine molecules plus four bound iodine atoms
  - $T_3$ – triiodothyronine; has two tyrosines with three bound iodine atoms

**Effects of Thyroid Hormone**

- TH is concerned with:
  - Glucose oxidation
  - Increasing metabolic rate
  - Heat production
- TH plays a role in:
  - Maintaining blood pressure
  - Regulating tissue growth
  - Developing skeletal and nervous systems
  - Maturation and reproductive capabilities
**Transport and Regulation of TH**

- $T_4$ and $T_3$ bind to thyroxine-binding globulins (TBGs) produced by the liver
- Both bind to target receptors, but $T_3$ is more potent and has a shorter half-life than $T_4$
- Peripheral tissues convert $T_4$ to $T_3$
- Mechanisms of activity are similar to steroids
- Regulation is by negative feedback
- Hypothalamic thyrotropin-releasing hormone (TRH) can overcome the negative feedback

**Synthesis of Thyroid Hormone**

- Thyroglobulin is synthesized and discharged into the lumen
- Iodides ($I^-$) are actively taken into the cell, oxidized to iodine ($I_2$), and released into the lumen
- Iodine attaches to tyrosine, mediated by peroxidase enzymes, forming $T_1$ (monoiodotyrosine, or MIT), and $T_2$ (diiodotyrosine, or DIT)
Synthesis of Thyroid Hormone

- Iodinated tyrosines link together to form T₃ and T₄.
- Colloid is then endocytosed and combined with a lysosome, where T₃ and T₄ are cleaved and diffuse into the bloodstream.
Calcitonin (CT)

- A peptide hormone produced by the parafollicular, or C, cells
- Lowers blood $Ca^{2+}$ levels, especially in children
- Antagonist to parathyroid hormone (PTH)
Thyroid Disorders

(a) 

(b) 

Copyright © 2004 Pearson Education, Inc., publishing as Benjamin Cummings.
Thyroid Disorders – Endemic Goiter

Calcitonin

- CT targets the skeleton, where it:
  - Inhibits osteoclast activity (and thus bone resorption) and release of Ca\(^{2+}\) from the bone matrix
  - Stimulates Ca\(^{2+}\) uptake and incorporation into the bone matrix
  - Regulated by a humoral (Ca\(^{2+}\) ion concentration in the blood) negative feedback mechanism
Parathyroid Glands

- Tiny glands embedded in the posterior aspect of the thyroid
- Cells are arranged in cords containing oxyphil and chief cells
- Chief (principal) cells secrete PTH also referred to as parathormone
- PTH increases blood Ca\(^{2+}\) levels
Effects of Parathyroid Hormone

- PTH release increases Ca²⁺ in the blood as it:
  - Stimulates osteoclasts to digest bone matrix
  - Enhances the reabsorption of Ca²⁺ and the secretion of phosphate by the kidneys
  - Increases absorption of Ca²⁺ by intestinal mucosal
- Rising Ca²⁺ in the blood inhibits PTH release
Thymus

- Lobulated gland located deep to the sternum
- Major hormonal products are thymopoietins and thymosins
- These hormones are essential for the maturation and proliferation of T lymphocytes (T cells) of the immune system

Pancreas

- A triangular gland, which has both exocrine and endocrine cells, located behind the stomach
- Acinar cells produce an enzyme-rich juice used for digestion (exocrine product)
- Pancreatic islets (islets of Langerhans) produce hormones (endocrine products)
The islets contain four cell types:

- Alpha ($\alpha$) cells that secrete glucagon
- Beta ($\beta$) cells that secrete insulin
- Delta ($\delta$) cells that secrete somatostatin (growth-hormone inhibiting hormone)
- F cells that secrete pancreatic polypeptide
**Glucagon**

- A 29-amino-acid polypeptide hormone that is a potent hyperglycemic agent
- Its major target is the liver, where it promotes:
  - Glycogenolysis – the breakdown of glycogen to glucose
  - Gluconeogenesis – synthesis of glucose from lactic acid and noncarbohydrates
  - Release of glucose to the blood from liver cells

**Insulin**

- A 51-amino-acid protein consisting of two amino acid chains linked by disulfide bonds
- Synthesized as part of proinsulin and then excised by enzymes, releasing functional insulin
- Insulin:
  - Lowers blood glucose levels
  - Enhances transport of glucose into body cells
  - Counters metabolic activity that would enhance blood glucose levels
**Effects of Insulin Binding**

- The insulin receptor is a tyrosine kinase enzyme
- After glucose enters a cell, insulin binding triggers enzymatic activity that:
  - Catalyzes the oxidation of glucose for ATP production
  - Polymerizes glucose to form glycogen
  - Converts glucose to fat (particularly in adipose tissue)

**Regulation of Blood Glucose Levels**

- The hyperglycemic effects of glucagon and the hypoglycemic effects of insulin
Insulin Disorders

- Hyperinsulinism – excessive insulin secretion, resulting in hypoglycemia
  - Symptoms can include such things as headache, dizziness, weakness, and emotional instability. In severe cases there may be convulsions, coma, and death.
  - The cause of oversecretion of insulin may be organic, i.e., a tumor of the pancreas, impaired liver function, or endocrine disorders, or it may be functional, e.g., unusual muscular exertion, pregnancy, or lactation.

Insulin Disorders

- Pre-diabetes → where fasting glucose levels are elevated above normal, but not high enough to be considered diabetes
  - Fasting results are between 100 to 125 mg/dl
    - Normal blood glucose levels in the range of 70 – 110 mg/dl (90 – 100 mg/dl is average)
  - Also referred to as impaired fasting glucose
  - If no intervention takes place, then type 2 diabetes will develop
Insulin Disorders

- Diabetes Mellitus (DM)
  - Results from hyposcretion or hypoactivity of beta cells (insulin)
  - The three cardinal signs of DM are:
    - Polyuria – huge urine output
    - Polydipsia – excessive thirst
    - Polyphagia – excessive hunger and food consumption
  - 3 further signs revealed by blood & urine tests include: hyperglycemia, glycosuria, ketonemia, ketonuria

---

Diabetes Mellitus (DM)

- Fasting blood glucose is 126 mg/dl or higher on three occasions
- 3 types:
  - Type 1 or insulin-dependent diabetes mellitus (IDDM)
  - Type 2 or non-insulin-dependent diabetes mellitus (NIDDM)
  - Gestational
Diabetes Mellitus (DM)

**Adrenal (Suprarenal) Glands**

- Adrenal glands – paired, pyramid-shaped organs atop the kidneys
- Structurally and functionally, they are two glands in one
  - Adrenal medulla – neural tissue that acts as part of the SNS
  - Adrenal cortex – glandular tissue derived from embryonic mesoderm
Adrenal Cortex

- Synthesizes and releases steroid hormones called corticosteroids
- Different corticosteroids are produced in each of the three layers
  - Zona glomerulosa – mineralocorticoids (chiefly aldosterone)
  - Zona fasciculata – glucocorticoids (chiefly cortisol)
  - Zona reticularis – gonadocorticoids (chiefly androgens)

Adrenal Gland
Adrenal Cortex – Zona Glomerulosa

Adrenal Cortex – Zona Fasciculata
Adrenal Cortex – Zona Reticularis

Adrenal Medulla
Mineralocorticoids

- Regulate electrolytes and H₂O in extracellular fluids
- Aldosterone – most important mineralocorticoid
  - Maintains Na⁺ balance by reducing excretion of sodium from the body
  - Stimulates reabsorption of Na⁺ and H₂O plus secretion of K⁺ by the kidneys
Aldosterone secretion is stimulated by:
- Rising blood levels of $K^+$
- Low blood $Na^+$
- Decreasing blood volume or pressure

**Mineralocorticoids**

**The Four Mechanisms of Aldosterone Secretion**

- Renin-angiotensin mechanism – kidneys release renin, which is converted into angiotensin II that in turn stimulates aldosterone release
- Plasma concentration of sodium and potassium – directly influences the zona glomerulosa cells
- ACTH – causes small increases of aldosterone during stress
- Atrial natriuretic peptide (ANP) – inhibits activity of the zona glomerulosa
Glucocorticoids (Cortisol)

- Help the body resist physical or emotional stress by:
  - Keeping blood sugar levels relatively constant
  - Maintaining blood volume and preventing water shift into tissue

- Cortisol provokes:
  - Gluconeogenesis (formation of glucose from noncarbohydrate sources)
  - Rises in blood glucose, fatty acids, and amino acids
Excessive Levels of Glucocorticoids

- Excessive levels of glucocorticoids:
  - Depress cartilage and bone formation
  - Inhibit inflammation
  - Depress the immune system
  - Promote changes in cardiovascular, neural, and gastrointestinal function

Gonadocorticoids (Sex Hormones)

- Most gonadocorticoids secreted are androgens (male sex hormones), and the most important one is testosterone
- Androgens contribute to:
  - The onset of puberty
  - The appearance of secondary sex characteristics
  - Sex drive in females
- Androgens can be converted into estrogens after menopause
Adrenal Medulla

- Made up of chromaffin cells that secrete catecholamines (epinephrine & norepinephrine)
- Secretion of these hormones causes:
  - Blood glucose levels to rise
  - Blood vessels to constrict
  - The heart to beat faster
  - Blood to be diverted to the brain, heart, and skeletal muscle

Adrenal Medulla

- Epinephrine (E) is the more potent stimulator of the heart and metabolic activities
- Norepinephrine (NE) is more influential on peripheral vasoconstriction and blood pressure
Adrenal Disorders

POMC

\[
\begin{align*}
\gamma-MSH & \quad \text{ACTH} & \quad \beta\text{-lipotropin} \\
\alpha-MSH & \quad \text{CLIP} & \quad \gamma\text{-lipotropin} & \quad \beta\text{-endorphin} \\
\beta-MSH
\end{align*}
\]
Adrenal Disorders

(a)

(b)

The same boy, only 4 months later.
Paired ovaries in the abdominopelvic cavity produce estrogens and progesterone

They are responsible for:
- Maturation of the reproductive organs
- Appearance of secondary sexual characteristics
- Breast development and cyclic changes in the uterine mucosa

Gonads: Male

- Testes located in an extra-abdominal sac (scrotum) produce testosterone
- Testosterone:
  - Initiates maturation of male reproductive organs
  - Causes appearance of secondary sexual characteristics and sex drive
  - Is necessary for sperm production
  - Maintains sex organs in their functional state
Stress and the Adrenal Gland

- General Adaptation Syndrome (GAS)
  - Alarm phase – short-term responses
  - Resistance phase – long-term metabolic adjustments
  - Exhaustion phase – collapse of vital organs
Response to Stress

**Alarm Phase**

"Fight or Flight" Immediate short-term responses to crises

1. Mobilization of glucose reserves
2. Changes in circulation
3. Increases in heart and respiratory rates
4. Increased energy use by all cells

**Response to Stress**

**Resistance Phase**

Long-term metabolic adjustments

1. Mobilization of remaining energy reserves: Lipids are released by adipose tissue; amino acids are released by skeletal muscle
2. Conservation of glucose: Peripheral tissue (except neural) breaks down lipids to obtain energy
3. Elevation of blood glucose concentrations: Liver synthesizes glucose from other carbohydrates, amino acids, and lipids
4. Conservation of salts and water: loss of K⁺ and H⁺

**KEY**

- GH = Growth hormone
- GC = Glucocorticoids
- ACTH = Adrenocorticotropic hormone
- MC = Mineralocorticoids
- ADH = Antidiuretic hormone
Response to Stress

Exhaustion Phase

Collapse of vital systems

Causes may include:
- Exhaustion of lipid reserves
- Inability to produce glucocorticoids
- Failure of electrolyte balance
- Cumulative structural or functional damage to vital organs

Other Hormone-Producing Structures

- Heart – produces atrial natriuretic peptide (ANP), which reduces blood pressure, blood volume, and blood sodium concentration
- Brain – produces brain natriuretic peptide (BNP), which reduces blood pressure, blood volume, and blood sodium concentration
- Gastrointestinal tract – enteroendocrine cells release local-acting digestive hormones
- Placenta – releases hormones that influence the course of pregnancy
Other Hormone-Producing Structures

- Kidneys – secrete erythropoietin, which signals the production of red blood cells, renin and calcitriol
- Skin – produces cholecalciferol, the precursor of vitamin D
- Adipose tissue – releases leptin, which is involved in the sensation of satiety, and stimulates increased energy expenditure

Endocrine Functions of the Kidneys
Endocrine Functions of the Kidneys

Angiotensin converting enzyme (ACE)