INTRODUCTION

The purpose of the endocrine system is to accomplish, with the nervous system
1. integration
2. homeostasis
3. growth
4. control digestion and its functions
5. control the production and release of energy
6. control of the composition and volume of
7. allows for adaptation to hostile environments

This is accomplished by endocrine or DUCTLESS glands which secrete HORMONES. These are chemical agents which are released by a cell or group of cells and have a physiological influence on another cell or group of cells. The area in which the hormone has its effect is called the TARGET AREA.

Hormones may be
a. steroids
2. derivatives of a amino acids
3. polypeptides
4. glycoproteins

Eicosanoids - leukotriines and prostaglandins

Paracrines – vs Autocrines
Whatever their chemical nature hormones combine with receptor areas at the target site and modify the production or action of specific cellular enzymes

Hormones can affect the metabolism of their target area by
1. altering plasma membrane permeasbility
2. stimulating the synthesis of proteins
3. activates/deactivates enzymes
4. inducing secretory action
5. stimulates cell division

HORMONE RECEPTORS

Although hormones travel throughout the body in the bloodstream, it is attracted to only a specific group of cells called its target cells. Hormones like neurotransmitters influences their target areas by chemically bonding to integral membrane proteins or glycoproteins called receptors. Only the target cells with the specific receptors will respond to the given hormone and no other.

A target area has 2000 - 100,000 receptor areas for a particular hormone. When a hormone is present in excess the number of ell receptors may decrease. This is called down regulation. The decreases the responsiveness of the target’ cells to the hormone.

Conversely when the amount of available hormone is inadequate, the number of receptor sites may increase, known as up-regulation making the target area more sensitive to the hormone.
Circulating and Local Hormones

Hormones that pass into the blood stream and act on distant target areas are referred to as **circulating hormones** or **endocrines**. Hormones that act locally without entering the blood stream are referred to as **local hormones**. Those that act on neighboring tissues are called **paracrines**. Local hormones that act on the same cell that secreted them are **autocrines**.
CHEMICAL CLASSIFICATION

Hormones secreted by a wide range of endocrine glands have diverse chemical structures. They all can be grouped into three categories.

1. **AMINES** - derivatives of amino acids
2. **POLYPEPTIDES AND GLYCOPROTEINS** - such as ADH, insulin and TSH
3. **STEROID** - cortisol and testosterone. These are derived from cholesterol

ACTIVATION AND INACTIVATION OF HORMONES

Hormones molecules that affect the metabolism of target areas are often derived from less active **PARENT** molecules, also called **PRECURSOR** molecules. In the case of polypeptide hormones, the precursor may be a long chained **prohormone** that is cut and spliced to make a longer chained molecule. **INSULIN** for example is produced from proinsulin within the Islet cells of the pancreas.

In some instances the molecule secreted by the endocrine gland, and considered to be the hormone of the gland, is actually inactive in the target cells. In order to become active, the target cells must modify the chemical structure of the hormone. **Thyroxine (T4)** must be changed to **T3** within the target cells in order to affect the metabolism of the cells.

Similarly, testosterone and Vitamin D3 are converted together into active molecules within the target cells. These chemicals are all designated as **prehormones**.

The concentration of hormones or prehormones in blood primarily affects the rate of secretion of the endocrine glands - hormones do not accumulate within the blood. Hormones contain a **HALF-LIFE** which is rather short, ranging from a few minutes to several hours for most hormones. Most are removed by the liver, in the bile and through the urine.

HORMONAL INTERACTIONS:
1. **PERMISSIVE EFFECT** - the effect of one hormone on a target area requires a previous or simultaneous exposure to another hormone

2. **SYNERGISTIC EFFECT** - the effects of two or more hormones complement each other in such a way that the target area responds to the sum of the hormones involved

3. **ANTAGONIST EFFECT** - the effect of one hormone on a target area is opposed by another hormone

**MECHANISM OF ACTION**

1. **detector** - usually the nervous system or gland
2. **signaling device** - usually the nervous system
3. **secretory cells**
4. **target area**
5. **negative feedback control**

The detectors can be INTERNAL detecting the rise/fall of a hormone or external detecting EXTERNAL stimuli
INTRACELLULAR HORMONE MEDIATORS

Almost all amine, polypeptide and glycoprotein hormones cannot pass through the lipid barrier of target cell membranes. Although some of these hormones may enter through PINOCYTOSIS, most of the effects of these hormones are believed to the result from interaction of these hormones with receptor proteins on the outer surface of the target cell membrane.

Since the hormones do not have to enter the target cells to mediate their effects, other molecules must mediate the actions of these hormones within the target cells. If you think of hormones as MESSENGERS from the endocrine glands, the intracellular mediators of the hormone's action can be called the SECOND MESSENGERS.

2ND MESSENGER

1. all amino acids based hormones are mediated via a 2nd messenger that is generated by hormones (1st messengers) binding to the plasma membrane
2. camp is the most common and most well known

The levels of camp release are dependent upon

1. hormone reception
2. a signal transducer (G-proteins)
3. effector enzyme (adenylate cyclase)
4. G-protein is activated with GTP and inactive with GDP
**Action**

a. 1st messenger binds to specific plasma membrane receptor

b. membrane receptor changes shape and binds to G-protein which is activated and binds to GTP

c. Activated G-protein displaces the GDP

d. Activated G-protein moves along the membrane binds to and activates *Adenylate Cyclase* that hydrolyzes the bound GTP to GDP via GTPase rendering G-protein inactive

e. Adenylate cyclase catalyzes ATP to camp

f. Camp diffuses into cell and activates *protein kinase*

g. *Protein kinase* produces effects

h. Process called *Amplification effect*

**Each Adenylate cyclase**

- Release large quantities of camp
- That causes large releases of protein kinase
- Causes the catalysis of 100’2 of reactions

These reactions are dependent on:

- Target cell type
- Specific protein kinases
- Hormones that acts as 1st messenger
- Camp is degraded by *phosphodiesterase*
**PIP₂ Calcium Signal mechanism**

Some hormones released through a different 2\(^{nd}\) messenger mechanism. In this mechanism Ca\(^{++}\) acts as the final mediator

**PIP₂ Calcium Signal Action**

1. 1\(^{st}\) messenger docks with receptor site on cell membrane
2. binds to inactive G-protein
3. G-protein is activated as GTP binds displacing GDP
4. Active G-protein binds to and activates membrane **Phospholipase enzyme** and then becomes inactive
5. Phospholipase splits a plasma membrane bound phospholipid

\(\text{PIP}_2 \rightarrow \text{phosphatidyl inositol biphosphate} \) into

**Diacylglycerol and IP\(_3\) inositol triphosphate**

6. Both PIP\(_2\) and IP\(_3\) act as 2\(^{nd}\) messengers
7. Diacylglycerol activates specific protein kinases
8. IP\(_3\) triggers the release of Ca\(^{++}\) from the endoplasmic reticulum
9. the Liberated calcium then becomes the 2\(^{nd}\) messenger
   a. activates enzymes and plasma membrane channels
   b. binds with **calmodulin**
   c. calcium + calmodulin produces enzymes to form an amplification responses

**Direct gene Activation**

1. Steroids are lipid soluble and can diffuse into target cells
2. inside the bind with intracellular receptors activated by coupling
3. activatyed receptor complex then moves to the nuclear chromatin
4. hormone then boinds to specific DNA associated with the receptor protein
5. interaction turns on genes → DNA transcription → mRNA --> > proteint synthesis
Target Cell Activation

Dependent upon

1. blood levels of hormone
2. relative number of G receptors for that hormone on the target cells
3. affinity between hormone and receptor
4. **EG:** target cells form more receptor sites in response to rising blood levels of a specific hormone they respond to \(= \) UP REGULATION
5. **EG:** prolonged exposure to high hormone concentrations desensitizes target cells causing them to be less responsive. This causes a loss of receptor sites \(= \) DOWN REGULATION

Hormone Groups

Somatotrophs – HGH
Thyrotrophs – TSH
Gonadotrophs – FSH LH ICSH
Lactotrophs – PRL
Corticotrophs ACTH

**PROSTAGLANDINS**

The **PROSTAGLANDINS** are autoendocrine regulatory molecules derived from **ARACHIDONIC ACID** and exert their effects within the same tissue in which they are produced. They act as complements and supplement to the endocrine and nervous systems.

The precursor molecule to all prostaglandins is arachidonic acid which is released by phospholipids in the cell membrane under hormonal or neural stimulation. This substance has but two major pathways

1. converted by an enzyme cyclooxidase into a prostaglandin
2. conversion to lipooxygenase into leukotrienes.

The prostaglandins are produced by almost every organ and have been implicated in a wide variety of functions. Their study can be somewhat confusing since the same prostaglandins can have opposite effects in different organs.

The E series prostaglandins \textbf{PGE} relax the smooth muscle in the urinary bladder, bronchioles, intestines and uterus but cause contraction of smooth muscle in the blood vessels.

Overall they are involved in
1. the inflammatory response
2. reproductive system
3. gastrointestinal tract
4. respiratory system
5. blood vessels
6. blood clotting
7. kidneys
PITUITARY GLAND

The pituitary gland is gland of two major components:
1. adenohypophysis - anterior pituitary
2. neurohypophysis - posterior pituitary.

The neurohypophysis secretes hormones that are released by the hypothalamus and the adenohypophysis secretes its own hormones in response to regulation from the hypothalamus. The secretions of the pituitary gland are controlled by the hypothalamus as well as by negative feedback influences on the part of the target glands.
**POSTERIOR PITUITARY**

The posterior pituitary releases two hormones;

1. oxytocin
2. vasopressin or antidiuretic hormones

These hormones are actually produced in neuron bodies of the hypothalamus. Oxytocin is regulated by the PARAVENTRICULAR NUCLEI and vasopressin is regulated by the SUPRAOPTIC NUCLEI. These hormones are transported along axons in the hypothalamic-hypophyseal tract and converted to their appropriate chemicals in the neurohypophysis by a process called NEUROSECRETORY TRANSDUCTION.

The release of these hormones is controlled by NEUROENDOCRINE REFLEXES. For Oxytocin the pathways is as follows

**OXYTOCIN**

Predominantly found in the female and affects smooth muscle contraction of the uterus and control over the myoepithelial tissue of the breast during milk ejection. It works in conjunction with PROLACTIN from the anterior pituitary, which controls the production of milk.
OXYTOCIN RESPONSE - EJECTION REFLEX

child at breast

stimulation at nipple

impulses to hypothalamus

stimulation of the paraventricular nuclei

production of oxytocin

neurosecretory transduction

release from neurohypophysis

target: myoepithelium

Myoepithelial contraction

release of PRF

Anterior Pituitary

release of prolactin

mammary glands

production of milk

MILK EJECTION
The secretion of ADH is stimulated by osmoreceptors in the hypothalamus in response to a rise in osmotic pressure, or a decrease in total serum water volume. It is INHIBITED by left atrium STRETCH RECEPTORS which detect a rise in the total blood volume.
The anterior pituitary consist of three portions

1. **pars distalis**
2. **pars tuberalis** - this and connected to infundibulum
3. **pars intermedia** - between anterior and posterior

The anterior pituitary produced 6 major hormones, all with regulation from the hypothalamus. These are:

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Target</th>
<th>Releasing Factor</th>
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<tbody>
<tr>
<td>GROWTH HORMONE</td>
<td>BODY</td>
<td>HGHRF; HGHIF</td>
</tr>
<tr>
<td>THYROTROPIC STIMULATING HORMONE</td>
<td>THYROID GLAND</td>
<td>TSHRF</td>
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<tr>
<td>ACTH</td>
<td>ADRENALS</td>
<td>ACTHRF</td>
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<tr>
<td>FSH</td>
<td>OVARIES/TESTES</td>
<td>FSHRF</td>
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<td>LH/ICSH</td>
<td>OVARIES/TESTES</td>
<td>LHRF/ICSHRF</td>
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<tr>
<td>PROLACTIN</td>
<td>MAMMARIES</td>
<td>PRF/PIF</td>
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**SUMMARY OF ANTERIOR PITUITARY**

<table>
<thead>
<tr>
<th>CELLS</th>
<th>TROPINS</th>
<th>HORMONE</th>
<th>HYPOTHALAMIC-HYPOPHSIOTROPHS</th>
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<tbody>
<tr>
<td>Somatotrophs</td>
<td>Somatotropin</td>
<td>HGH</td>
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<td>Lactotrophs</td>
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<td>Gonadotrophs</td>
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</table>
HUMAN GROWTH HORMONE

1. accelerates amino acid anabolism ---< increasing protein synthesis
2. increases lipolysis
3. increases fat catabolism changing use of carbs and prots to fats
4. decreases sugar utilization
5. influences liver to secrete somatomedin or insulin-like growth factor [IGF] which increases sugar utilization
6. increases cellular utilization of amino acids for growth

**Diabetogenic Effect**

The somatotrophs of the anterior pituitary release bursts of HGH every few hours. Their release is controlled by two hypophysiotropic hormones:

1. HGHRF (somatocrinin)
2. HGHIF (somatostatin)

Low blood sugar stimulates HGHRF to be released and passed to Anterior pituitary

Anterior pituitary in responses to HGHRF releases HGH

The release of HGH and IGF increase liver glycogenolysis cuasing increases in serum sugar

Sugar level rises

Increases in sugar inhibit HGHRF

Abnormally high sugar level causes the release of HGHIF and reduce HGHRF

HGHIF inhibit anteriro pituitary HGH production

Low HGH causes sugar to be released more slowly

Serum sugar levels steady off falling to normal
**THYROID GLAND**

**STRUCTURE AND LOCATION**

The thyroid gland is located over the larynx as a bilobed gland connected by a structure called the Isthmus. It is a follicular gland and the only gland capable of hormonal storage in the body.

On the microscopic level the thyroid gland consists of spherical structures called follicles lined with simple cuboidal cells called principle cells. These cells function in the synthesis of the principle thyroid hormones.

The interior of the follicle is the colloid which is a protein-rich fluid which contains stored hormone. Between the follicles are the parafollicular cells which function in the synthesis of thyrocalcitonin.

**Hormone Production**

The thyroid follicles actively accumulate iodide from the bloodstream and secrete it into the colloid. Once the iodide is in the colloid it is oxidized into iodine and attached to tyrosine in specific combinations. These combinations are then bound to globulin to form thyroxin-thyroglobulin which is the storage form.
The various combinations are

\[ \begin{align*}
T_1 & \quad \text{monoiodotyrosine [mit]} \\
T_2 & \quad \text{diiodotyrosine [dit]} \\
T_3 & \quad \text{triiodotyrosine} \\
T_4 & \quad \text{tetraiodotyrosine [Thyroxin]} \\
\end{align*} \]

MIT and DIT are initially formed and enzymes within the colloid couple MIT and DIT together to for T3 and T4. These too are coupled with thyroglobulin. Upon stimulation with TSH the principle cells will take up a volume of colloid by pinocytosis and hydrolyze the T3 and T4 from the thyroglobulin and secrete T3 and T4 into the bloodstream.

**Transport**

T3 and T4 are not water soluble so they are transported in the circulation via plasma proteins, particularly, globulin. The associated protein is TBG - Thyroid bonding globulin. T4 binds 10 times greater than T3, and only free T4 can enter the target areas. The TBG provides a reserve when the free % diminishes. In the target cells in order for the thyroid hormones to exert its effect the T4 must be converted to T3.
1. NO SPECIFIC TARGET AREA - MANIFESTED THROUGHOUT THE BODY
2. ENHANCES PROTEIN SYNTHESIS
3. ENHANCES NORMAL GROWTH OF TISSUE AND TISSUE DIFFERENTIATION
4. CALORIGENIC EFFECT - ENHANCES OXYGEN UTILIZATION
5. REGULATES BASAL METABOLIC RATE
6. REGULATES LIPID METABOLISM
7. ACCELERATES INTESTINAL CARBOHYDRATE METABOLISM
8. AIDS IN DISSOCIATING OXYGEN FROM HEMOGLOBIN
9. CONTROLS FEMALE REPRODUCTIVE RHYTHMICITY
**THYROCALTITONIN**

The parafollicular cells of the thyroid secrete THYROCALTITONIN [TCT]. Under specific circumstances TCT promotes a decrease in blood calcium levels by increasing OSTEOBLASTIC ACTIVITY and decreasing osteoclastic ACTIVITY. It antagonizes the effects of the Parathyroid gland and Vitamin D3.

**PARATHYROID GLAND**

The parathyroid glands are located behind the thyroid gland are comprised of 4-5 lobes.

On the microscopic level there are two types of epithelial cells. The cells that synthesize the parathyroid hormone are called **CHIEF CELLS** and are scattered among the OXYPHIL cells. The **oxyphil** cells support the chief cells are believed to produce reserve quantities of the parathyroid hormone.

**MAJOR FUNCTIONS**

The PARATHYROID HORMONE PTH promotes a rise in blood calcium levels by working on the bones:

1. increasing osteoclastic activity
2. decreasing osteoblastic activity
3. decrease bone desity
4. increase serum Ca++ levels

This gland exhibits a humoral effect. No connection with hypothalamus or pituitary. It samples the blood directly.

as well as the kidneys and intestines
Decreasing Calcium levels in the bloodstream

humorally effects parathyroid gland

bone calcium release

kidney calcium conservation

intestinal calcium absorption
ADRENAL GLANDS

The adrenal glands sit atop the kidneys
are also referred to as the suprarenal glands.
Each gland is about 50 mm long and 30 mm wide and generally pyramidal in shape.
each consists of an outer ADRENAL CORTEX and an inner ADRENAL MEDULLA.

Adrenal Medulla

The adrenal medulla is composed of tightly linked packed clusters of cells called CHROMAFFIN CELLS.
These are generally arranged around blood vessel and receives innervation directly from the ANS division of the nervous system
The adrenal medulla produces a class of hormones called catecholamines specifically two major ones;
    1. NOREpinephrine
    2. EPinephrine
in a ratio of 1:4.
They are amines and are derived from the non-essential amino acid TYROSINE.
    o The effects of these hormones are similar to those that are brought about by activation of the sympathetic NS, except the hormonal effects last about 10x longer.
They will

1. INCREASE CARDIAC OUTPUT
2. INCREASE HEART RATE
3. DILATE CORONARY BLOOD VESSELS,
4. INCREASE MENTAL ALERTNESS
5. INCREASE METABOLIC RATE
6. INCREASE RESPIRATORY RATE

Norepinephrine has the same effects but to a lesser degree.

Many stressors activate the adrenal medulla as well as the adrenal cortex and are involved in the **FLIGHT OR FIGHT** responses of the body.

They will recruit large quantities of energy in very short periods of time for maximum expenditure.
Adrenal Cortex

The adrenal cortex makes up the bulk of the adrenal gland and is histologically broken into three major divisions from outer to inner they are

<table>
<thead>
<tr>
<th>REGION</th>
<th>HORMONE</th>
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<tbody>
<tr>
<td>ZONA GLOMERULOSA</td>
<td>MINERALCORTICOIDES</td>
</tr>
<tr>
<td>ZONA FASCICULATA</td>
<td>GLUCOCORTICOIDES</td>
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<tr>
<td>ZONA RETICULARIS</td>
<td>ANDROGENS</td>
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</tbody>
</table>

BASIC SYNTHESIS

Zona Glomerulosa

Cholesterol

\[ \text{pregnenolone} \]

\[ \text{progesterone} \]

\[ \text{deoxycorticosterone} \]

\[ \text{corticosterone} \]

\[ \text{aldosterone} \]
Stress and the Adrenal Gland

All forms of stress produce effects that stimulate the adrenal cortex. The stressors, whatever they may be, stimulate the PITUITARY ADRENAL AXIS. Under stressful conditions, there is an increase in the release of ACTH and thus increased secretion of the corticosteroids from the adrenal cortex.

Stress is defined as **any condition which stimulates the pituitary adrenal axis**. Even pleasant changes in one's life can be as stressful as unpleasant ones. The body responds with a non-specific response called the **GENERAL ADAPTATION SYNDROME** (GAS) in which there are three stages
1. **ALARM REACTION** - adrenal glands activated
2. **STAGE OF RESISTANCE** – readjustment and acclimation occurs
3. **STAGE OF EXHAUSTION** if readjustment does not occur leading to sickness and possible death.

![PITUITARY ADRENAL AXIS STIMULATION Diagram](image)
ZONA GLOMERULOSA

The zona glomerulosa releases a series of hormones which come under the class of MINERALCORTICOIDS. These include

1. aldosterone
2. deoxycorticosterone

They work in conjunction with the kidneys to control and regulate the total extracellular water volume related to serum levels of sodium ions. The sensor is located in the macula lutea of the kidney's distal convoluted tubules is a particular structure called the JUXTAGLOMERULAR APPARATUS.

This structure monitors the blood and sense several items

1. total serum sodium levels
2. total extracellular fluid volume
3. mean arterial blood pressure

In doing so it can regulate and recruit the uptake of sodium, consequently chloride and ultimately water by changing the tonicity of the blood. The principle pathway is referred to as the RENIN-ANGIOTENSIN PATHWAY.
RENNIN-ANGIOTENSIN PATHWAY

decrease in ECF volume and/or
Decrease in mean arterial BP and/or
decrease in NA concentration

stimulates the JGA

release of renin targets liver

converts angiotensinogen to angiotensin I

converted to angiotensin II (vasoconstrictor)

active reabsorption of Na ions
consequent Cl ions
water to offset tonicity

release of aldosterone

release of ACTH targets adrenals

targets pituitary

releases of ACTHRF

stimulates hypothalamus
Zona Fasciculata

This region produces a series of hormones which are called glucocorticoids. The examples are

1. cortisone
2. corticosterone
3. hydrocortisone
4. cortisol

These hormones have an influence on metabolism in that they are capable of directing which food material will be burned. Among their functions include:

1. capable of altering chemical control so that glucose production is increased at the expense of protein
2. in starvation amino acids are transformed into glucose via gluconeogenesis
3. controls lipolysis
4. stress is initiator to the system
5. controls lipogenesis
6. controls gluconeogenesis
7. controls glycogenolysis and glycogenesis
8. can increase blood pressure
9. aids as an anti-inflammatory agent by decreasing cell membrane permeability
10. counteracts the action of insulin
11. accentuates the action of the thyroid
12. can activate diabetogenic effect
GLUCOCORTICOID INSUFFICIENCY

1. INCREASES ACTH
2. VASCULAR SMOOTH MUSCLE BECOME UNRESPONSIVE
3. SKELETAL MUSCLE FATIGUES EASILY
4. SLOW EEG WAVES; PERSONALITY CHANGES
5. INCREASE GUSTATION
6. DECREASE GASTRIC ACTIVITY
7. DECREASE URINE OUTPUT = 100-200 ML/DAY
8. DECREASE IN WBC, RBC AND PLATELETS
9. DECREASE IN ADAPTATION SYNDROME
ZONA Reticularis

This region produces a series of hormones called the **ANDROGENS**.

These include

1. estrogen
2. testosterone

Their principle action is to increase the synthesis and decrease protein catabolism and thereby increase growth rate. They effect epiphyseal growth and principally aids in the secondary sexual characteristics,

1. DEVELOPMENT OF EXTERNAL GENITALIA
2. FULL GROWTH AND DEVELOPMENT ON INTERNAL GENITALIA
3. VOICE
4. BODY HAIR
5. BREAST TISSUE
6. MUSCLE AND BODY CONFORMATION
7. SEBACEOUS SECRETIONS
8. MENTAL ATTITUDE
9. AGGRESSIVENESS
10. OPPOSITE SEXUAL ATTRACTION.
The pancreas is both an endocrine and an exocrine organ. As an exocrine organ it provides some of the necessary enzymes needed for digestion. As an endocrine organ it contains the **ISLETS OF LANGERHANS**. There are 4 types of hormone secreting cells which comprise these islets:

1. alpha cells - secrete glucagon
2. beta cells - secrete insulin
3. delta cells - secrete somatostatin
4. F-cells - secrete pancreatic polypeptide within acinar tissue

**GLUCAGON**

1. functions to increase blood sugar by activating the liver to undergo
   a. glycogenolysis
   b. lipolysis
   c. conversion of glucose from lactic acid (gluconeogenesis)
2. regulates the entrance of sugar into the blood stream

**INSULIN**

1. produced by the beta cells
2. accelerates the utilization of sugar by tissue via
   a. glycogenesis
   b. lipogenesis
   c. removal of sugar from blood into muscles
3. decreases glycogenolysis and gluconeogenesis
Extraendocrine Producing Structures

Heart: ANP – Atrial naturietic peptide – decrease in BP, blood volume and serum Na
GI TRACT – Gastrin; enterocrinin; villikinin
Placenta – HcG
Kidney – rennin; erythropoirtin
Skin – cholecalciferol + UV = Vitamin D
Adipose – leptin ---> arenterin