Physiology & Biochemistry Team

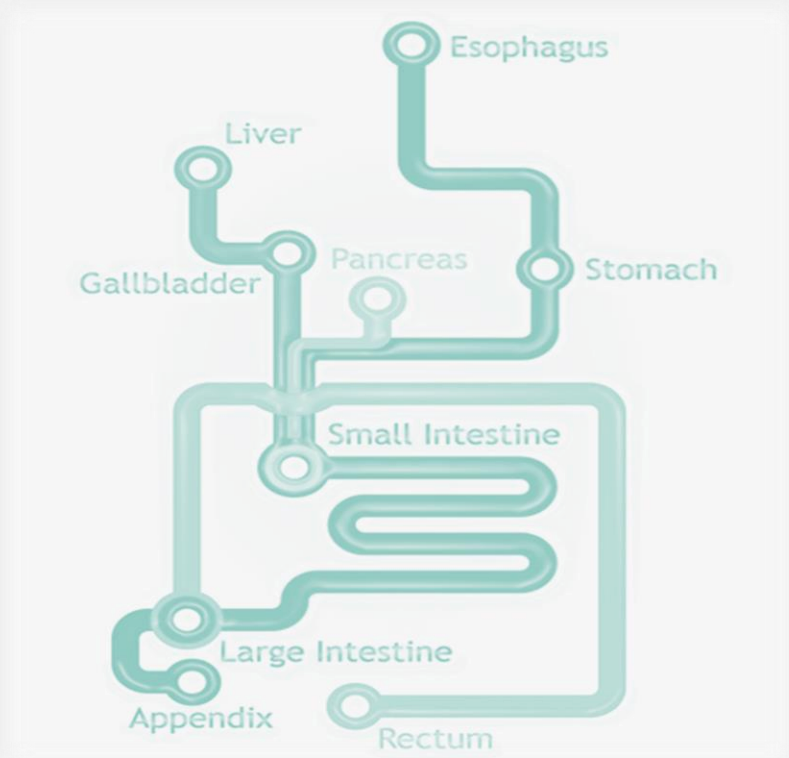
LECTURES

Physiology :

I. Small intestine
II. pancreas

Biochemistry:

I. Lipid digestion.
II. proteins and carbohydrates Digestion.



Physiology Team

Biochemistry Team

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Slides Important Male's notes Female's notes Explanation

Motility In The Small Intestine

Divisions of the Small Intestine:

1. Duodenum
2. Jejunum
3. Ileum

Motility in the Small Intestine :

The movements of the small intestine can be divided into:

- *Segmenting (Mixing) contractions*
- *Propulsive contractions (Peristalsis)*
- *Migrating motor complex*
- *Antiperistalsis*
- *Peristaltic rush*

Physiology Team

Motility In The Small Intestine

Biochemistry Team

Mixing (Segmentation) Contractions	•When a portion of small intestine becomes distended, the segmentation contraction is activated by ENS to divide the intestine into spaced segments which last for fraction of min , and have the appearance of a chain of sausages. •As one set of segmentation contractions relaxes, a new set often begins at points between the previous ones. •The segmentation contractions become weak when the excitatory activity of ENS is blocked by the drug atropine. •The significance of segmentation contractions: ✓Blend different juices with the chyme ✓Bring products of digestion in contact with absorptive surfaces
Propulsive Movements (Peristalsis)	•Organizes propulsion of material over variable distances. •Usual stimulus is distention. •They are faster in the proximal intestine and slower in the terminal intestine (velocity 0.5 to 2.0 cm/sec), and the *net movement along the small intestine normally averages only 1 cm/min (3 to 5 hours are required for passage of chyme from the pylorus to the ileocecal valve).* •Myenteric plexus is important for these movements. •They can be blocked by the drug atropine.
Migrating motor complex (MMC):	•It is bursts of depolarization accompanied by peristaltic contraction that begins in empty stomach during interdigestive period, travels along the small intestine to reach ileocaecal valve after 1.5-2 hrs, where it disappears and a new wave of starts. •Activity stops as soon as food is ingested. •Propels any remnants in stomach & small intestine into the colon.
Antiperistalsis	In the opposite direction (from anus to mouth) occurs between stomach and duodenum to allow more time for neutralization of chyme and between ileum and caecum to allow time for absorption. (Normally occurs during vomiting)

Movement Of The Villi

The villous movement consists of :

1. fast shortening and slow lengthening.
2. side to side movements.

•Villous contractions are initiated by local nervous reflexes in response to chyme in small intestine.

•They are stimulated by villikinin hormone (specific hormone for the movement of the villi) released by intestinal mucosa when it comes in contact with digestive products.

•They facilitate absorption and lymph flow from central lacteals into lymphatic system. (help in ↑ lymph flow)

Control Of Intestinal Motility

1. Neural factors:

- Vagal excitation increases intestinal and villous movements.
- Sympathetic excitation decreases intestinal and villous movements.

Gastroileal reflex *(initiated from the stomach and affect the ileum)* is:

- initiated by gastric distension.
- Impulses are conducted through myenteric plexus to initiate a fast peristaltic wave passing to the ileum.
- The ileocaecal valve relaxes allowing chyme to pass into cecum. This reflex is mediated by vagus nerve .

2- Hormonal factors

- Gastrin, CCK, insulin and serotonin stimulate intestinal motility.
- Gastrin and CCK relax ileocaecal sphincter. *(have an action like vagus nerve by relaxing ileocaecal sphincter and stimulating intestinal motility)*
- *(CKK decrease stomach motility but increase intestinal motility)*
- Motilin secreted from duodenum stimulates intestinal motility and regulate MMC.
- Secretin and glucagons inhibits intestinal motility and contract ileocaecal sphincter. *(similar to sympathatic action)*
- Villikinin stimulates movement of the villi.

Secretion Of The Small Intestine

	Brunner's Glands	The Crypts of Lieberkühn
Location	located in the wall of the first few centimeters of the duodenum.	small pits which lie between intestinal villi
Secretion	**Secretion of Mucus :** They secrete large amounts of alkaline mucus, which contains a large amount of bicarbonate ions •Mucus protects the mucosa.	**Secretion of Intestinal Juices (Succus Entericus):** The surfaces of both the crypts and the villi are covered by an epithelium composed of 2 types of cells: 1. Goblet cells → secrete mucus. 2. Enterocytes→ A. secrete large quantities of H_2O and electrolytes and over the surfaces of adjacent villi B. reabsorb H_2O, electrolytes & end products of digestion

Secretion Of The Small Intestine

Succus Entericus: (intestinal juice)

It is secreted from intestinal crypts.

Volume: 1800 ml/day.

pH: 7.5-8. (alkaline) It participates in the neutralization of acid chyme delivered from stomach. (important because the intestinal mucosa is not protected against HCL and to provide the optimal pH necessary for action of enzymes -pancreatic or intestinal enzymes-.)

Composition: (in addition to water)

- 0.6 % organic (mucus and enzymes)
- 1 % inorganic substance. (electrolytes)

Most of the enzymes are found in:

- the **brush border**
- the **cytoplasm of the enterocytes** (intracellular digestion).

The enzymes that secreted into the lumen are:

- enteropeptidase
- amylase (pancreatic)

Control Of Secretion

Brunner's gland secretion	Intestinal juice secretion
Is stimulated by: •Hermonal: Secretin (inhibitory to the motor function but stimulate secretion) •Tactile (tactile=coming of the chym in contact with the mucosa) •Neural: vagal stimulation .	Is stimulated by: A. Distension, tactile and vagal stimulation. B. Hermonal: gastrin, secretin, CCK, glucagons, enterocrinin. Sympathetic stimulation exerts an inhibitory effect.

Carbohydrates

Carbohydrate digestion:

Carbohydrate digestion is rapid. Which is why it is recommended to eat proteins for breakfast so that you'll feel full for a longer while than say if you eat donuts. Generally completed by the time the gastric contents reach the junction between the duodenum and jejunum. The site of digestion: mouth and intestinal lumen.

Dietary carbohydrates:

Polysaccharides (mainly)	Oligosaccharides	Disaccharides	Monosaccharides
• Starch (plants) • Glycogen (animal) • Cellulose (plants)		• Sucrose • Lactose • Maltose	Little amounts

Contain α (1-4) and α (1-6) bonds. Chemically similar in structure

Contain β (1-4) bonds.

Digestion Of Carbohydrates

Digestion by Pancreatic Amylase:

- Pancreatic α-amylase is almost identical with the α-amylase of saliva but more powerful. Within 15 to 30 minutes after the chyme mixes with pancreatic juice, all the carbohydrates will have become digested.
- The carbohydrates are almost totally converted into maltose or other very small glucose polymers before passing beyond the duodenum or upper jejunum.

The enterocytes lining the villi of the small intestine contain four enzymes

1. Lactase
2. Sucrase
3. Maltase
4. a-dextrinase

- which are capable of splitting the disaccharides lactose, sucrose, and maltose, plus other small glucose polymers, into their constituent monosaccharides (the absorptive form).
- These enzymes are located in the enterocytes covering the intestinal microvilli brush border, so that the disaccharides are digested as they come in contact with these enterocytes.

Enzymes for digestion of Carbohydrates

ENZYME	α –amylase (both salivary and pancreatic)	Disaccharidases (intestinal)	Isomaltase and α (1,6) glucosidase (intestinal)
SUBSTRATE	Polysaccharides	Disaccharides	Branch points of oligo/disaccharides

α –amylase

Hydrolysis of : α (1,4) glycosidic bonds

Products : Mixture of short oligosaccharides

Disaccharides : Maltose and isomaltose

- *No dietary carbohydrate digestion occurs in the stomach* (high acidity of stomach inactivates salivary α-amylase).
- *Pancreatic* α-amylase continues the process of starch and glycogen digestion in the small intestine (secreted by pancreas, worked in the small intestine).

Serum level of α –amylase (25-125 U/L)

- Normal levels in the serum
- Normally found in very small amounts

in the serum as it is not its side of action.

Its levels start to rise within a few hours

↓

Reaches peak within 12- 72 hrs

↓

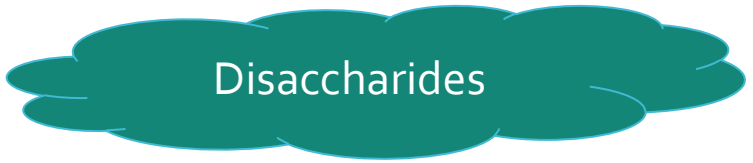
Returns to normal within a few days. Won't be detected in a week.

Clinical significance of rising circulating levels of α- amylase activity:

- Diagnosis of **acute pancreatitis** (*damage of pancreatic cells leads to release and activation of the intracellular enzymes into the blood*)

Final digestion of carbohydrates by intestinal enzymes in the small intestine

Enzymes : Secreted by and remain associated with the brush border membranes of the intestinal mucosal cells.
Location of their action : Mucosal lining of jejunum
They include : 1- Disaccharides 2- A(1-6) glucosidase for branched oligosccharides



Enzymes	Substrate	Product
isomaltase	isomaltose	2 Glucose
Maltese	Maltose	2 Glucose
Sucrase	Sucrose	Glucose + Fructose
Lactase (β – galactosidase)	Lactose	Glucose + Galactose

Dietary **cellulose** cannot be digested in the **absence of the enzyme that can cleave β (1-4) bonds**. It passes through the GIT intact.

Despite that, it has many beneficial effects. Cellulose remains throughout as it is.

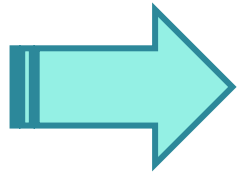
Absorption of monosaccharides by intestinal mucosal cells

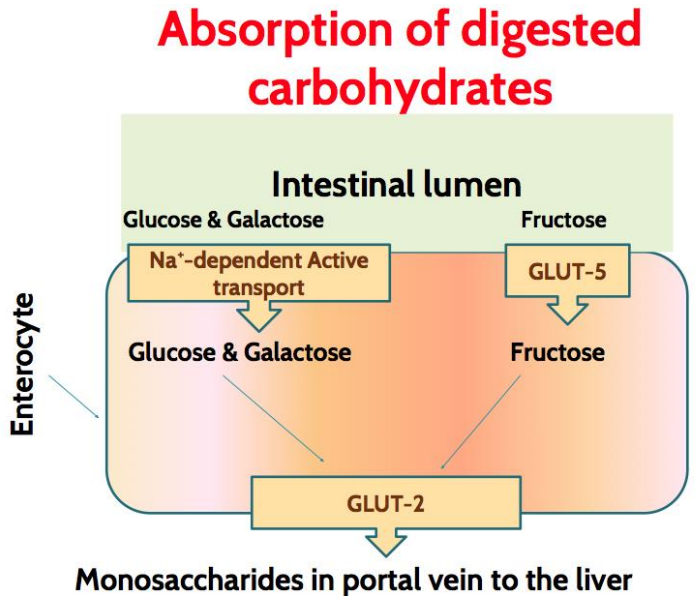
- Location: Duodenum and upper jejunum
- *Insulin not required for uptake of glucose by intestinal cells.
- **Different monosaccharides have different mechanisms of absorption

1-Facilitated diffusion (GLUT-mediatd)

2-Active transport (energy dependant): cotransport with Na^+

3-Both are needed. No simple diffusion.





Abnormal digestion of disaccharides (e.g., lactose)

"Lactose intolerance – lactose(β –galactosidase) deficiency"

Bacterial fermentation of undigested compunds in the large intestine → CO_2, H_2 gas → Abdominal cramping, diarrhea, distention and flatulence

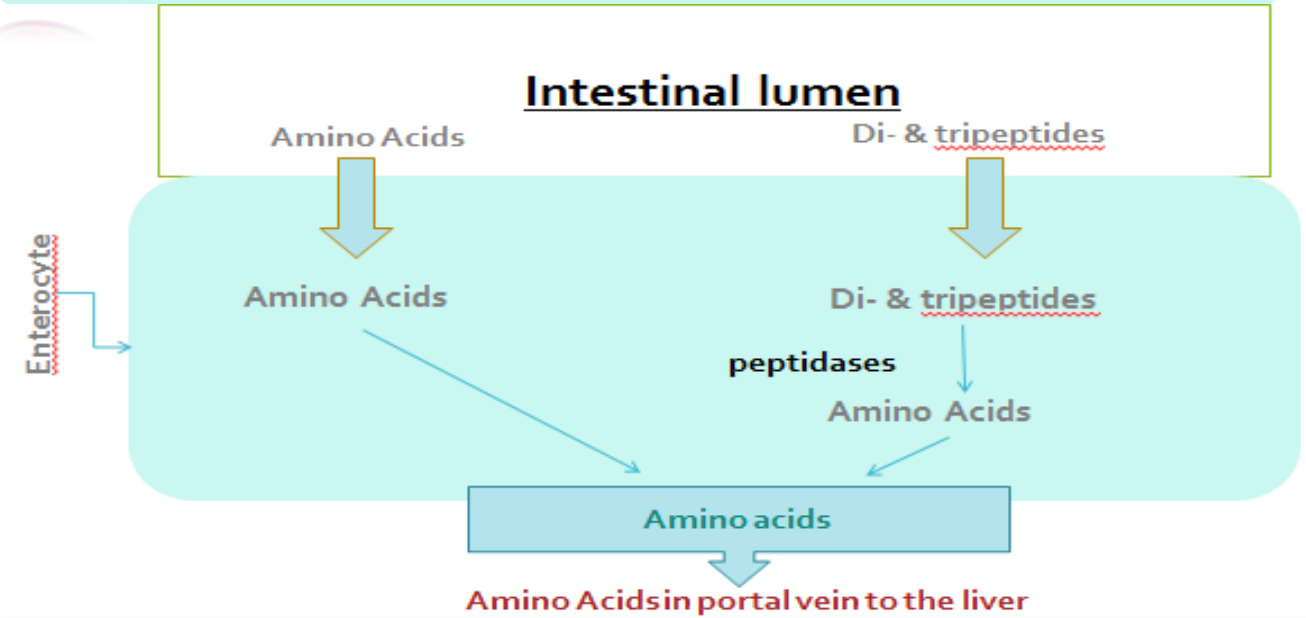
Lactase deficiency → Undigested carbohydrates in the intestine → Osmotic diarrhea (osmotic dragging

Proteins

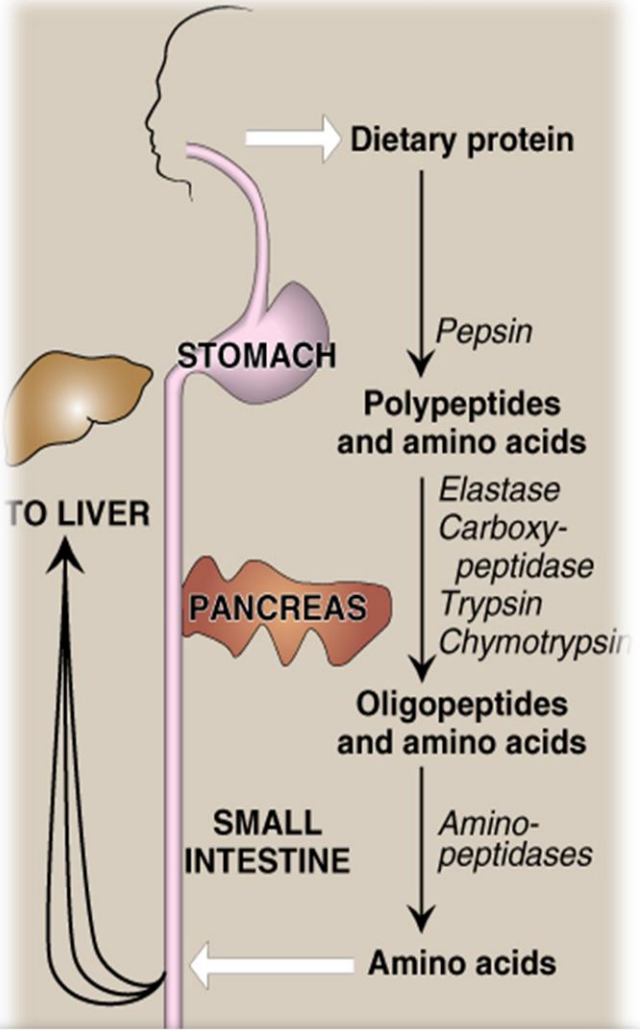
Protein Digestion

- Dietary proteins constitute 70-100 g/day.
- Proteins are generally too large to be absorbed by the intestine.
- They must, therefore, be hydrolyzed to their constituent amino acids, which can be absorbed.

Digestion and absorption of protein (see the next slide)



Take a look



- Aminopeptidase work on peptide chain with amino acid terminal.
- Oligopeptidases work on peptides with small chains.
- di and tripeptides are absorpted as such and digested Intracellulary by di and tripeptidases.

Digestion of Proteins

Stomach

- The gastric juice contains 3 components important for protein digestion:
  - Hydrochloric acid
  - Pepsin
  - Rennin (in neonates and infants)
- Protein digestion by stomach ----→ Polypeptides
- Digested by pepsin (active at pH 2 – 3).
- Pepsin also digests collagen protein of intracellular connective tissue in meat, thus allowing digestive enzymes to reach meat protein.
- Pepsin is responsible for only 10% – 20% of protein digestion.

Pancreas

- The pancreatic secretion contains a group of pancreatic proteases (secreted as inactive zymogens)
- Each of these enzymes has different specificity for the cleavage sites.

Small Intestine

- Oligopeptides that result from the action of pancreatic proteases are cleaved into free amino acids and smaller peptides (di- & tri-peptides) by **intestinal aminopeptidase** (an exopeptidase on the luminal surface of the intestine) Only a small percentage of the proteins are digested all the way to their constituent AA (Amino acids) by the action of→ pancreatic juices (trypsin, chemotrypsin & elastase).
- Most remain as dipeptides and tripeptides.
- Most protein digestion occurs in the duodenum and jejunum by Aminopeptidases, Oligopeptidases Intracellular di and tripeptidases for splitting small peptides into amino acids.

Lipids

Dietary Lipids :

Dietary lipids intake is ~81 g/day

-Triacylglycerol is ~ 90%

- The remainder includes (10%):
  - Cholesterol
  - Cholesteryl ester (cholestrol with fatty acids)
  - Phospholipids
  - Glycolipids
  - Free fatty acids

Lipid Digestion: Sites and Enzymes

❖ **Sites:**

1-The stomach

2-The small intestine

❖**Enzymes:**

1. Act in stomach: (The effects of lingual and gastric lipases on TAG)

Mouth: Lingual lipase (first enzyme act on the food it is coming from base of salivary gland).

Stomach: Gastric lipase (gastric mucosa):

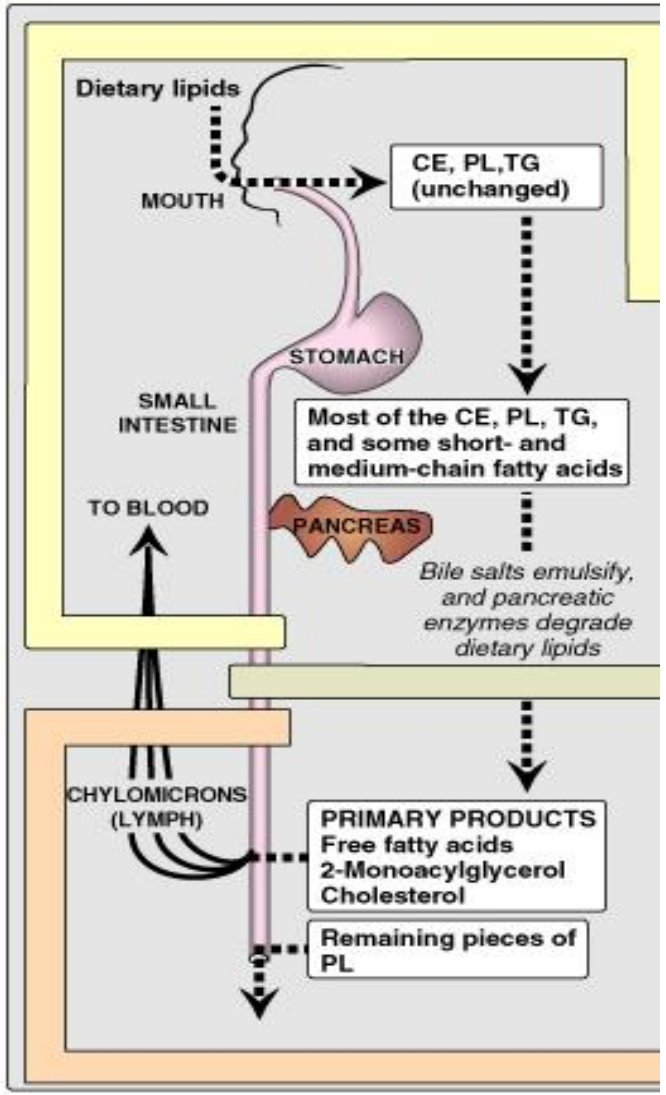
+They act only on short and medium length fatty acids (<12 carbon fatty acid chains, e.g. milk).

So,It is essential for neonates and not that important for adults unless they have pancreatic insufficiency(e.g.cystic fibrosis)

2. Act in small intestine: (is preceded by emulsification + hormonally controlled)

Pancreatic enzymes

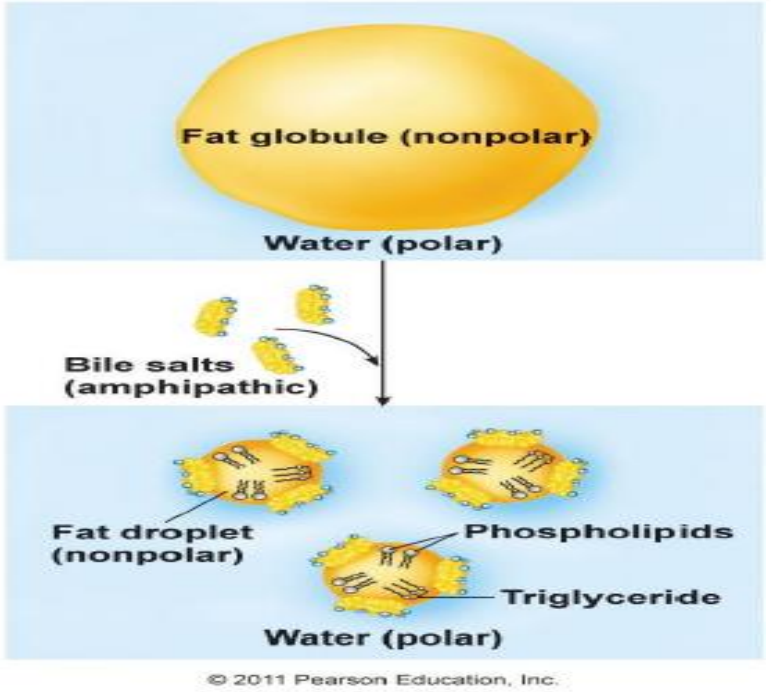
- -Lipase and co-lipase
- -Cholesterol esterase
- -Phospholipase A2
- -Lysophospholipase



Digestion Of Fats

•**Bile salts and lecithin** in the bile allow fat globules to be easily broken into smaller pieces by intestinal agitation and help fat digestion by make the fat globules readily fragmentable with the water in the small intestine (*emulsification of fat).

- Bile salts break the fat globules into very small sizes, so that the water-soluble digestive enzymes can act on the globule surfaces.
- The most important enzyme in TG digestion is pancreatic lipase



*Emulsification increases the surface area of lipid droplets (enzyme can act only on the surface so when we break it to small droplet, the enzyme can act on much more molecules so the surface area increases) ,therefore the digestive enzymes can effectively act.

Mechanisms:

1-Mechanical mixing by peristalsis

the rhythmic contractions and relaxations to mix the lipids with the bile salts.

2. Detergent effect of bile salts: (by hydropholic end they interact with water & hydrophopic end they interact with lipids)

Bile salts interact with lipid particles and aqueous duodenal contents, stabilizing the particles as they become smaller, and preventing them from coalescing

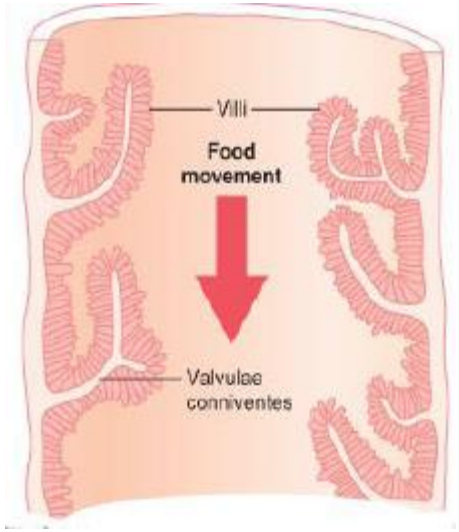
Main End products of lipid digestion:

- **2-Monoacylglycerol**
- **Cholesterol**
- **Free fatty acids**

Basic Principles Of lipid Absorption

Absorptive Surface of the small intestinal wall

- The absorptive surface of the small intestinal mucosa, showing many folds called valvulae conniventes, well developed in the duodenum and jejunum.
- They increase the surface area of the absorptive mucosa about ***3-fold.***
- The presence of villi on the mucosal surface enhances the total absorptive area another ***10-fold.***
- The epithelial cell on each villus is characterized by a brush border, consisting of as many as 1000 microvilli (increases the surface area another ***20-fold.***



1-Absorption of lipids (**by Intestinal Mucosal Cells):**

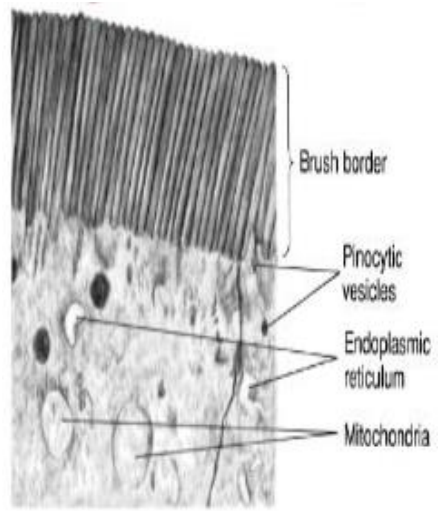
Mixed micelles*:

Disc-shaped clusters of amphipathic lipids.

Arranged with their hydrophobic groups on the inside and their hydrophilic groups on the outside.

Micelle includes end products of lipid digestion, bile salts and fat-soluble vitamins

Short- and medium-chain fatty acids do not require mixed micelle for absorption by intestinal cells (They directly go to the portal circulation)



Resynthesize of Lipids and assembly of Chylomicrons by Intestinal Mucosal Cells

Resynthesis of Lipids by Intestinal Mucosal Cells

1. Activation of long chain fatty acids into acyl CoA

2. - Synthesis of TAG from monoacylglycerol
   - Cholesterol ester from cholesterol
   - Phospholipids from glycerylphosphoryl base

3. Short- and medium-chain fatty acids are not converted into their CoA derivatives. Instead, they are released into portal circulation, carried by serum albumin

Assembly of Chylomicrons by Intestinal Mucosal Cells

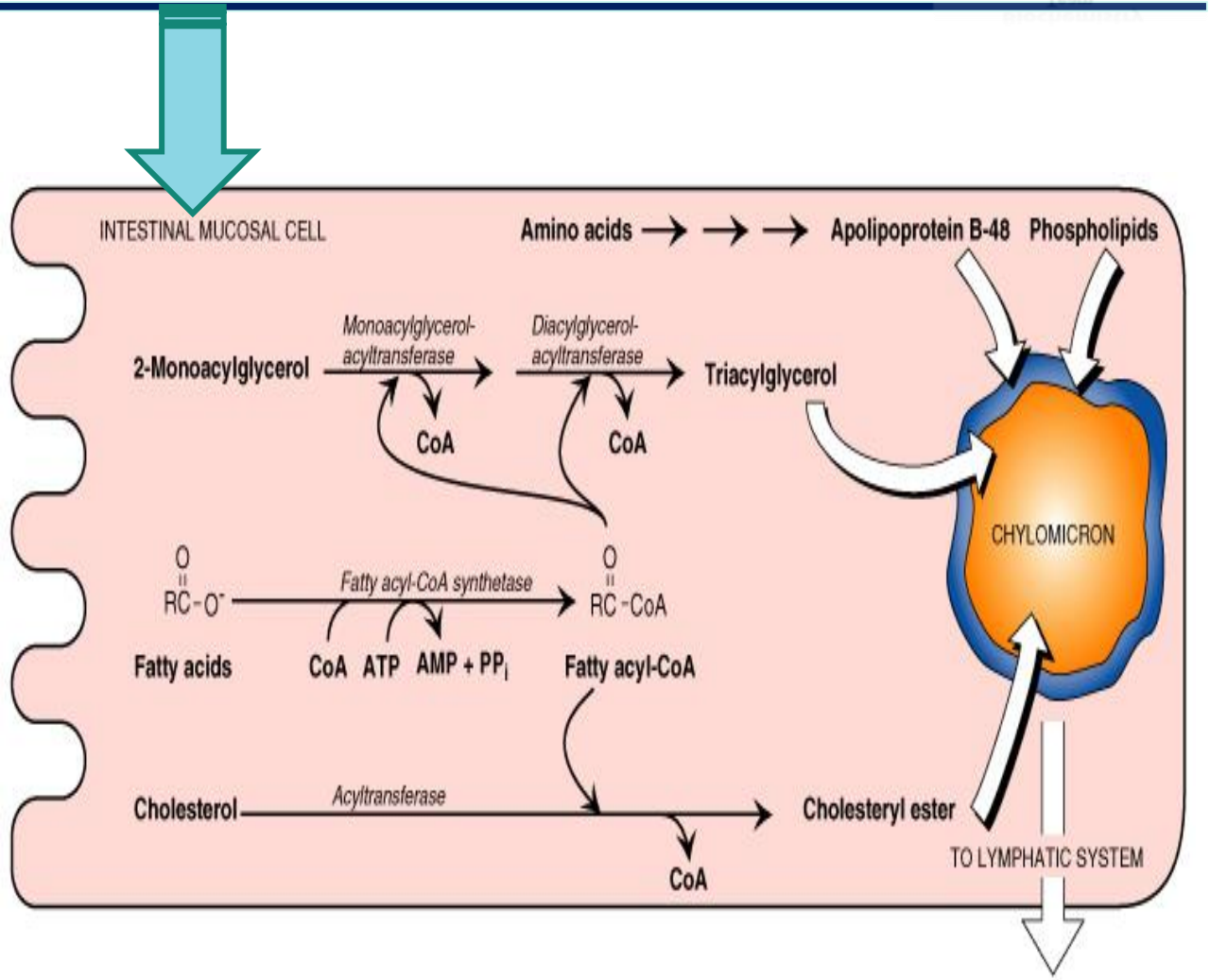
Newly synthesized TAG and cholesteryl ester are packaged as lipid droplets surrounded by thin layer of:

1. Apolipoprotein B-48 (apo B-48)
2. Phospholipids
3. Free cholesterol

Secretion of Chylomicrons by Intestinal Mucosal Cells

-By exocytosis into lymphatic vessels around villiof small intestine (lacteals) then enter into systemic circulation

-Milky-appearance of serum after lipid-rich meal



Abnormalities in Lipid Digestion/Absorption

- **Pancreatic insufficiency**
  - 1-chronic pancreatitis
  - 2-cystic fibrosis
  - 3-surgical removal of the pancreas
- **Intestinal diseases:**
  - e.g., Intestinal resection (shortened bowl)
- **Liver and gall bladder diseases**

→ incomplete digestion & absorption of fat & protein → abnormal appearance of lipids (steatorrhea) & undigested protein in the feces (Malabsorption syndrome).

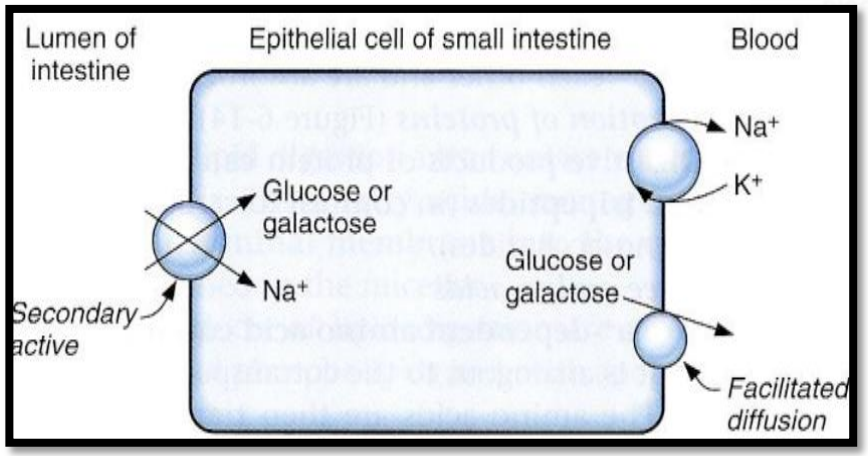
- The name ***cystic fibrosis*** refers to the characteristic scarring (fibrosis) and cyst formation within the pancreas, first recognized in the 1930s
- It affects most critically the lungs, and also the pancreas, liver, and intestine. It is characterized by abnormal transport of chloride and sodium across an epithelium, leading to thick, viscous secretions.
- Autosomal recessive disorder due to mutation to the CF Transmembrane Conductance Regulator (CFTR) gene
- CFTR protein is a chloride channel on epithelium
- Defect leads to decreased secretion of chloride and increased reabsorption of sodium and water
- In the pancreas, decreased hydration results in thickened secretions which cannot reach intestine, causing pancreatic insufficiency We give the patient enzyme supplements and vitamins

Absorption

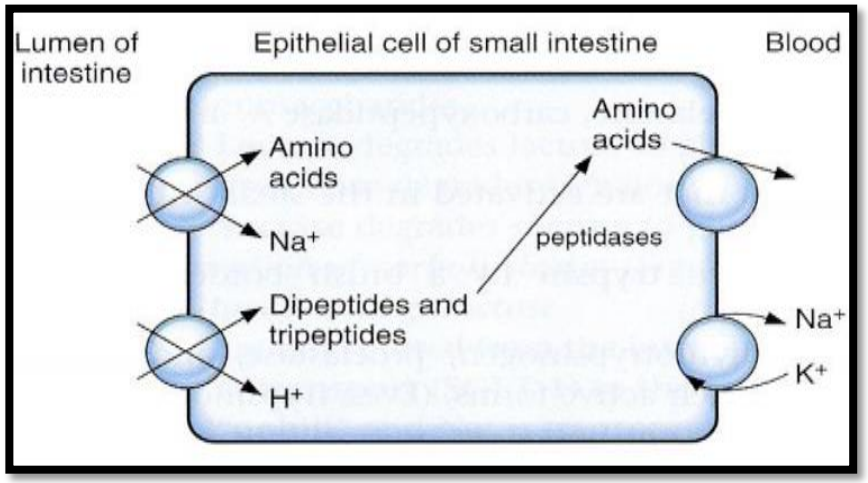
	Carbohydrates	Proteins	Fats	Vitamins
Absorption form	**Mono-saccharides** + small fraction **Disaccharides**	**Dipeptides** + **Tripeptides** + few free **Amino Acids**	Break down to **Fatty acids** + **Monoglycerides** then carried by bile micelles*	_____
How absorption occur + its mechanisms	*Glucose and Galactose → 2ry active transport with Na+ *Fructose → facilitated diffusion on Na+ via lumenal membrane *Pentose → via passive diffusion	***D- AA**→ passive diffusion. ***L- AA**→ 2ry active transport. ***Di and tripeptides** →active transport protein carrier by crossing the brush border. They are hydrolyzed by brush border and cytoplasmic oligopeptidases. ***AA** → facilitated Transport by leaving the cell at the basolateral membrane.	*In presence of bile micelles →about 97% of the fat is absorbed *In absence of the bile micelles → only 40 to 50% fat is absorbed. _____ *Bile micelle: small spherical, cylindrical Globule of 20 - 40 molecules of bile salts** ** Bile salts: amphipathic molecules, composed of {sterol nucleus (fat-soluble)} + {polar group (water-soluble), ((its –ive charge make entire micelle dissolve in water of GI fluids))} _____ #Triglycerides aggregate into globules along with the absorbed cholesterol and phospholipids. #B-lipoprotein coat part of the surface of each globule to form chylomicrons. #It diffuses to side of the cell and is excreted by exocytosis into the central lacteal of villi, to lymph, then to thoracic duct.	**#Fat-soluble vitamins** (A, D, E, & K) are absorbed along with other lipids → by micelles. **#Water-soluble vitamins** (C, B1, B2, B6, and folic acid) are absorbed →by Na+-dependent cotransport #Vitamin B12 is absorbed in the ileum and → requires intrinsic factor _____ **Gastrectomy** = loss of parietal cells and loss of intrinsic factor → pernicious anemia!

Basic Principles Of Gastrointestinal Absorption

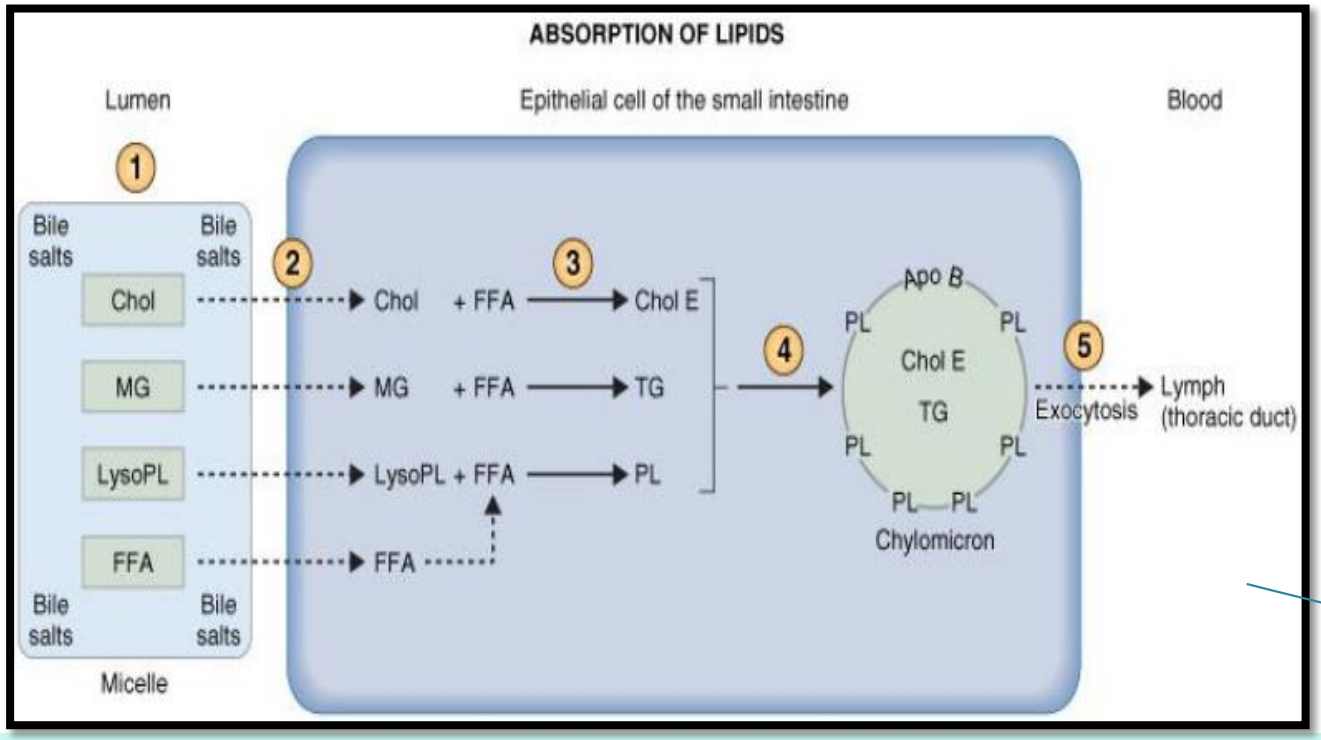
Carbohydrates Absorption



Proteins Absorption



Fat Absorption



Hormonal control of absorption & secretions of electrolytes

Important

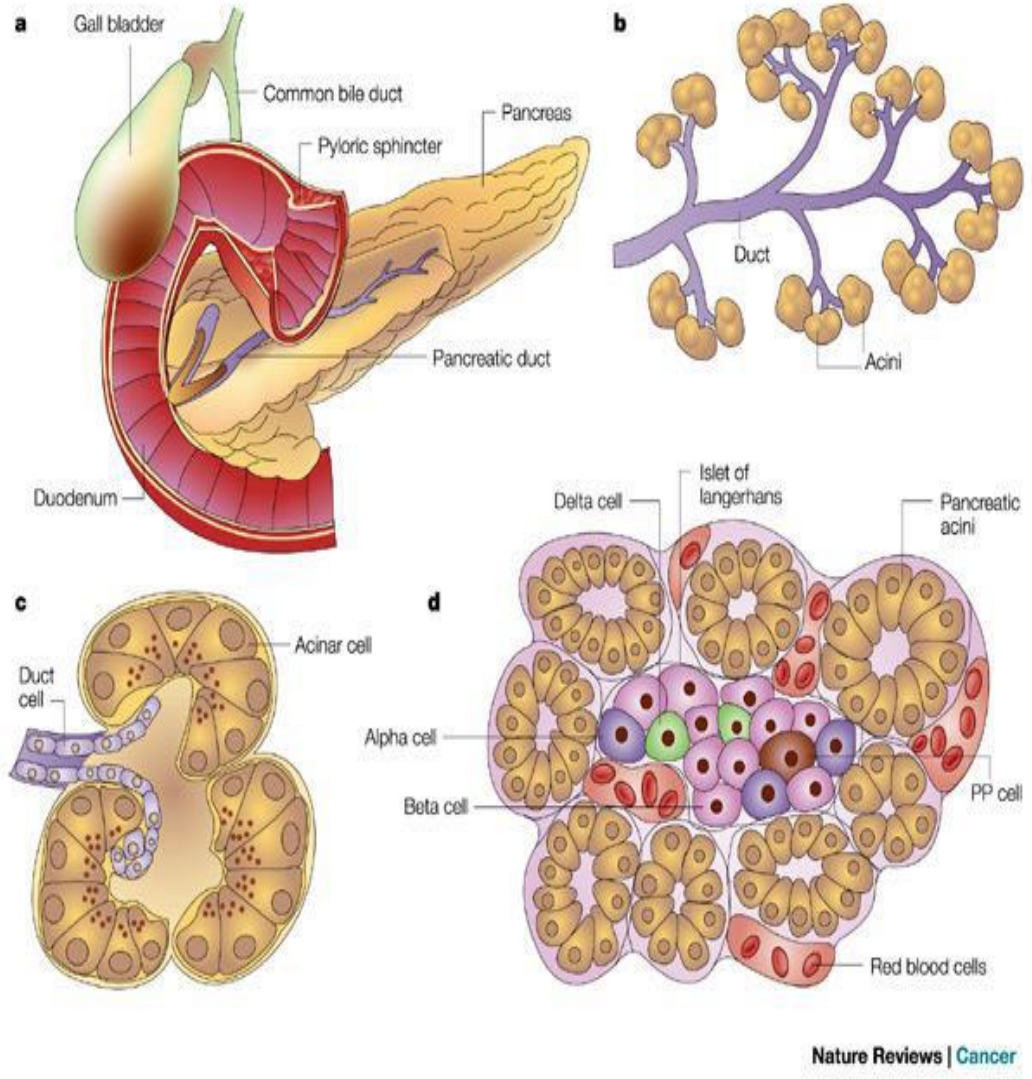
Hormone	Absorption	In the
Glucocorticoid	↑ absorption of H_2O & ions	(small & large intestine)
Somatostatin	↑ H_2O & ions absorption	(ileum & colon)
Epinephrine	↑ NaCl absorption	(ileum)
Aldosterone	↑ synthesis of Na+ channel	(colon)
Catecholamines	↓ intestinal secretion	intestine

Pancreas

- **The pancreas, which lies parallel to and beneath the stomach is composed of:**

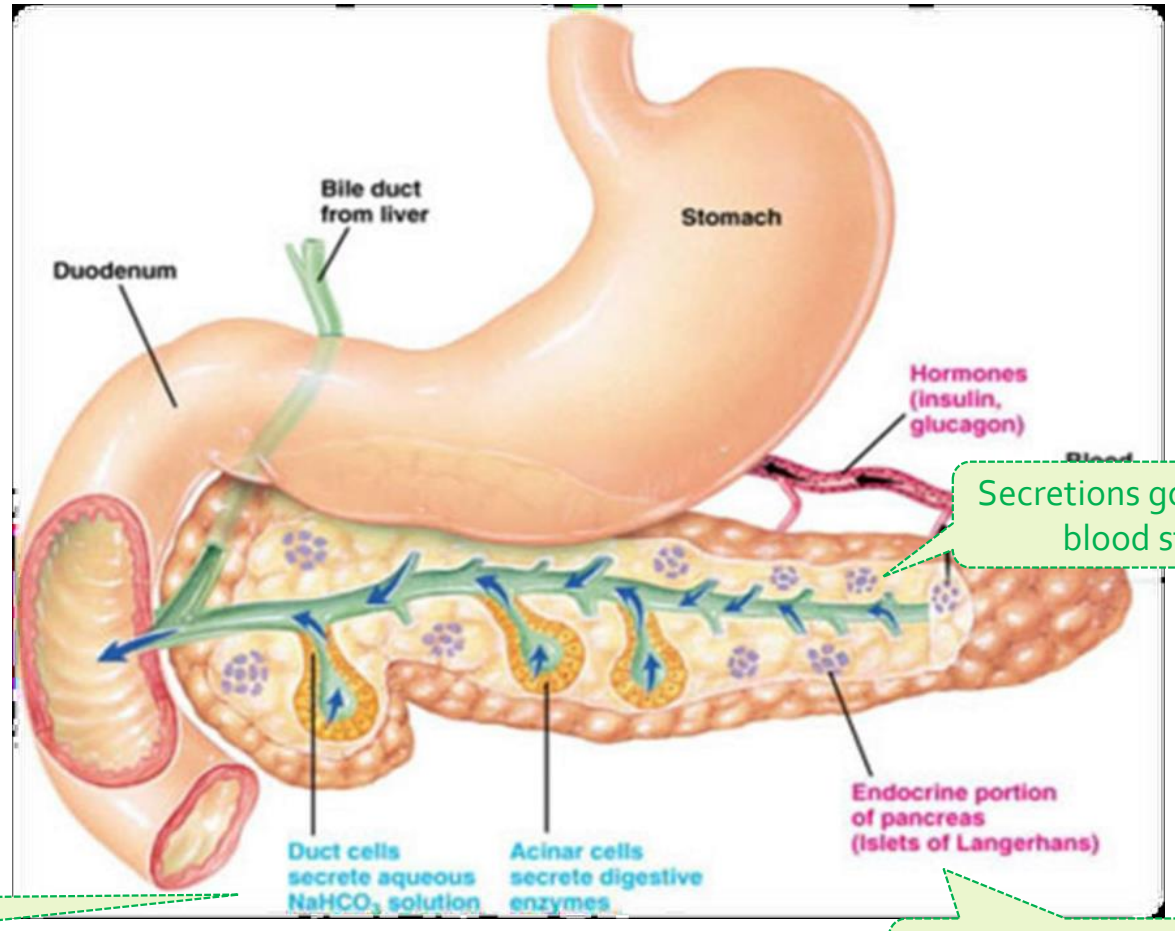
1. The endocrine islets of Langerhans which secrete insulin(60% from beta cells), Glucagone(25% from alpha cells) and Somatostatin(10% from delta cells)
2. Acinar gland tissues which produce pancreatic juice (the main source of digestive enzymes).

- The cells lining the acini are serous cells containing ZYMOGEN granules.
- Most of its structure are similar to that of Salivary glands.



Cont..

The main pancreatic duct joins into bile duct at ampulla of vater that is surrounded by sphincter of Oddi. Sphincter of Oddi controls the passage of pancreatic secretion and bile flow.
Pancreatic duct and bile duct join each other and open by a single duct into duodenum.



Secretions go directly to blood stream

Secretions pass in pancreatic duct then duodenum

Secretions go directly to blood stream

Pancreatic Secretion

- Pancreatic juice is secreted in response to the presence of CHYME in the upper portions of the small intestine.

- **The major functions of pancreatic secretion:**

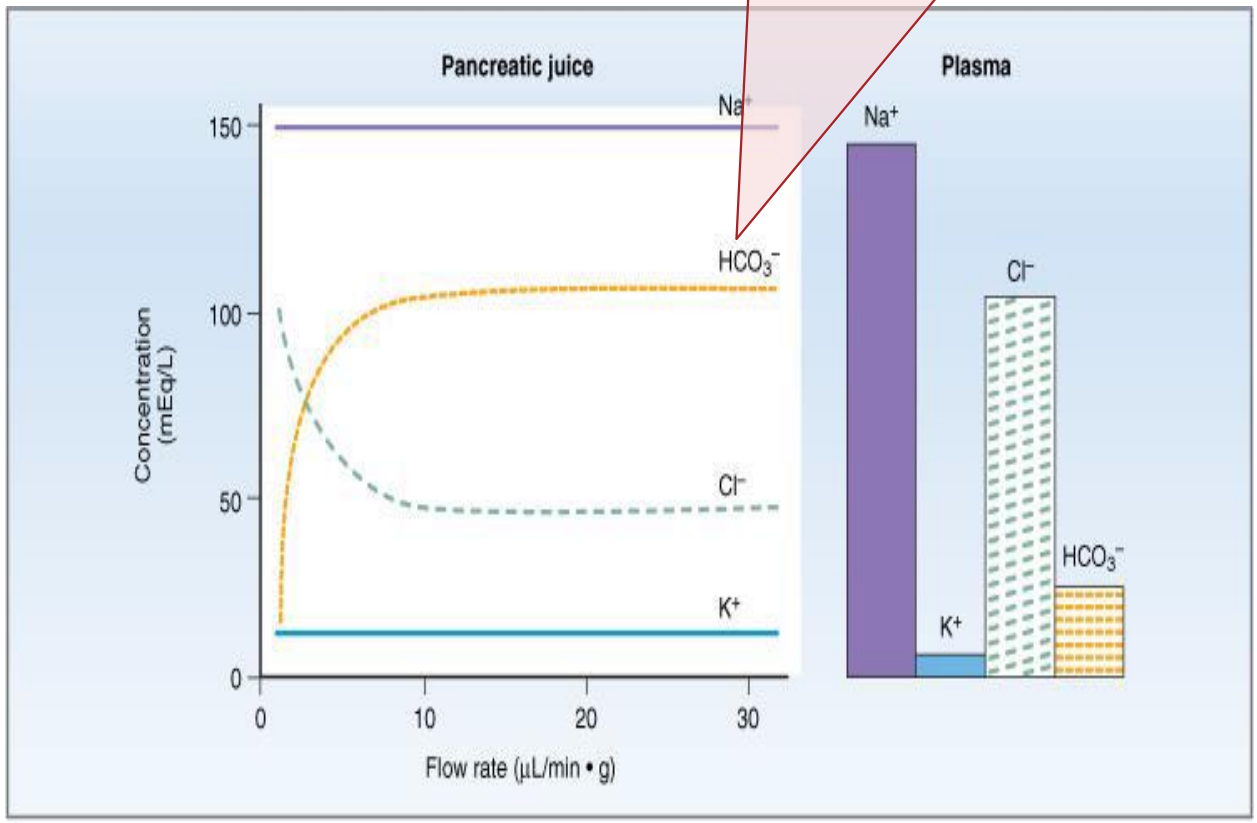
1. To neutralize the acids in the Duodenal chyme to optimum range for pancreatic enzyme activity (pH=7.0-8.0) . By alkaline secretion.
2. To produce enzymes involved in the digestion of dietary carbohydrate, fat, and protein.It can even digest DNA & RNA.
3. Mucosal protection against acid and pepsin

- **The electrolytes**
- They are produced from the epithelial cells of the ductules and ducts and include cations Na+, K+, Ca++ and anions HCO_3- and Cl-.
- The greater bulk of electrolytes is in the form of $NaHCO_3$. High concentration of $NaHCO_3$ produces alkalinity.
- HCO_3- concentration increases with increasing secretion rate.

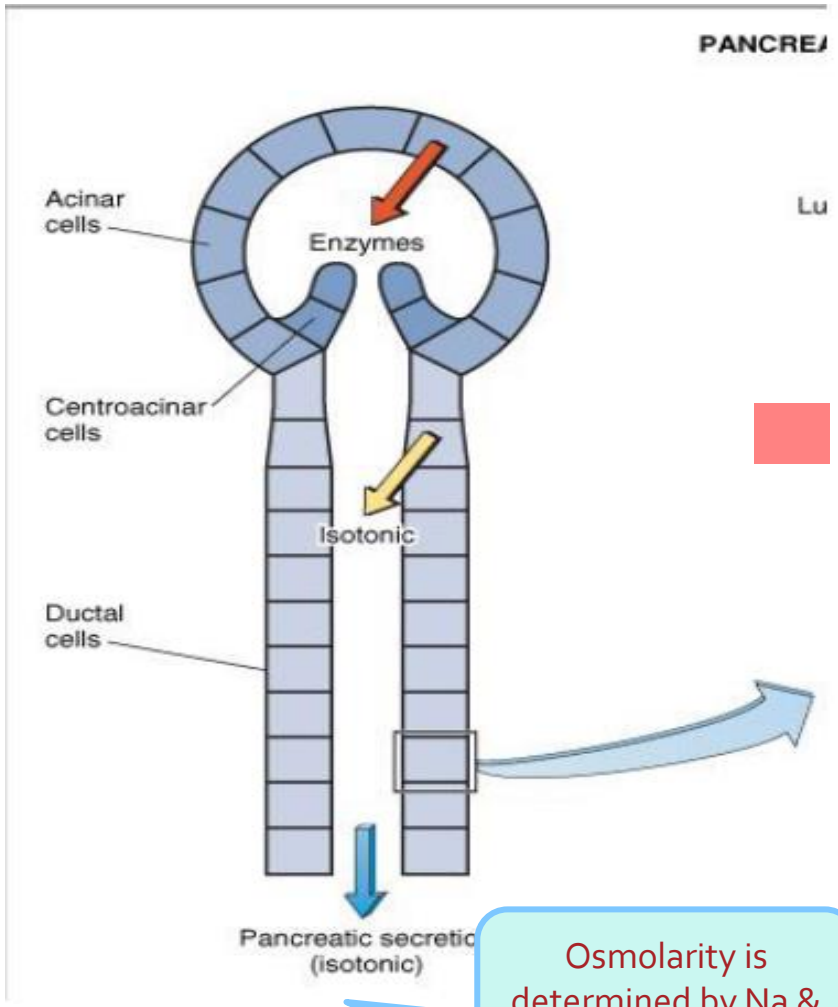
Pancreatic Secretion & Flow Rate

Properties	
Volume	1.2 – 1.51/day
Osmolarity	Isotonic
pH	8 (Alkaline) •For acid neutralization •Providing optimum pH for the action of enzymes.
Composition	1% inorganic materials (electrolytes) 1-2% organic materials mostly enzymes The rest is water.

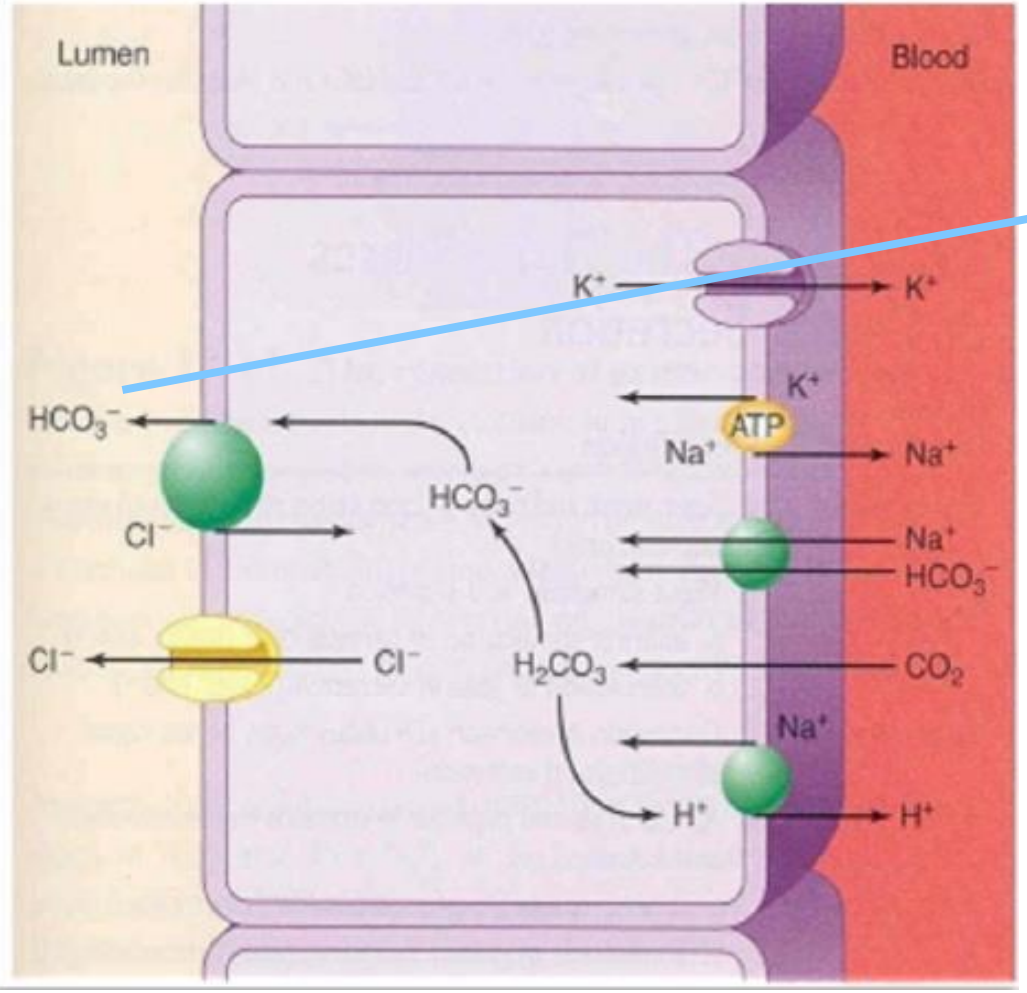
Bicarbonate secretion increases as the flow increases



Secretion of Bicarbonate Ions into Pancreatic Juice



Osmolarity is determined by Na & Cl concentrations



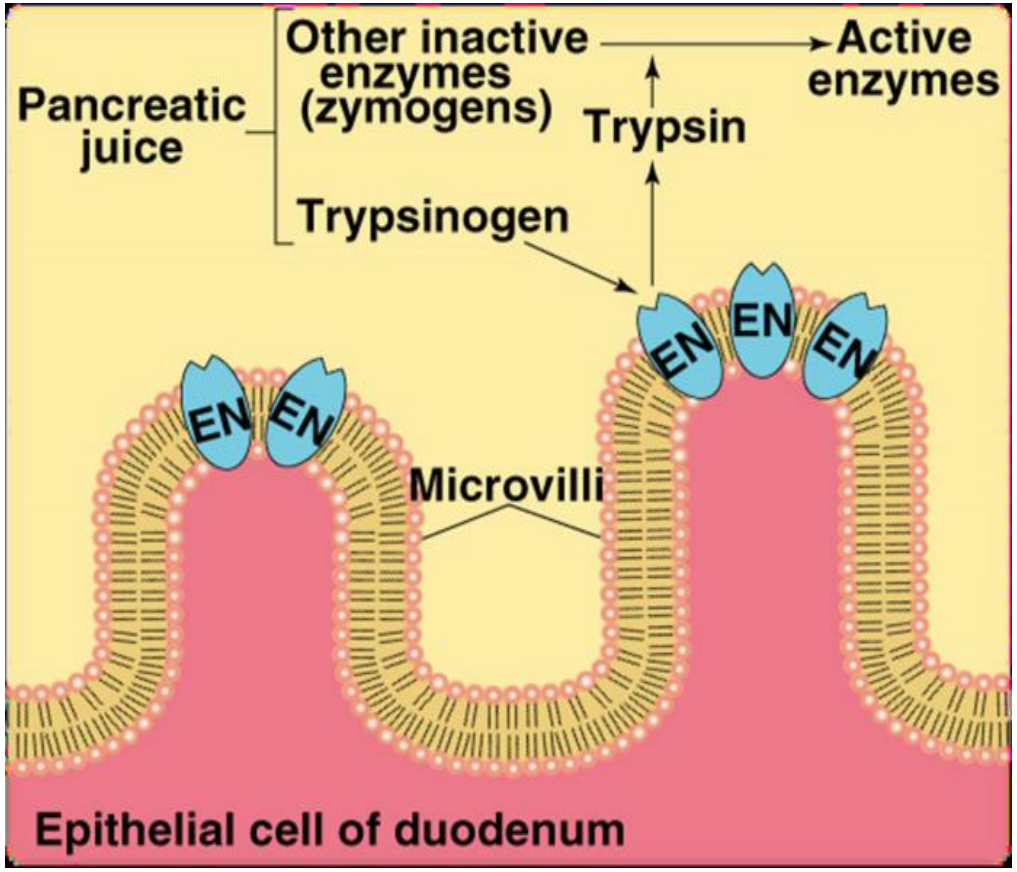
HCO_3 is secreted into the lumen so it will be added to the pancreatic juice.

Pancreatic Enzymes

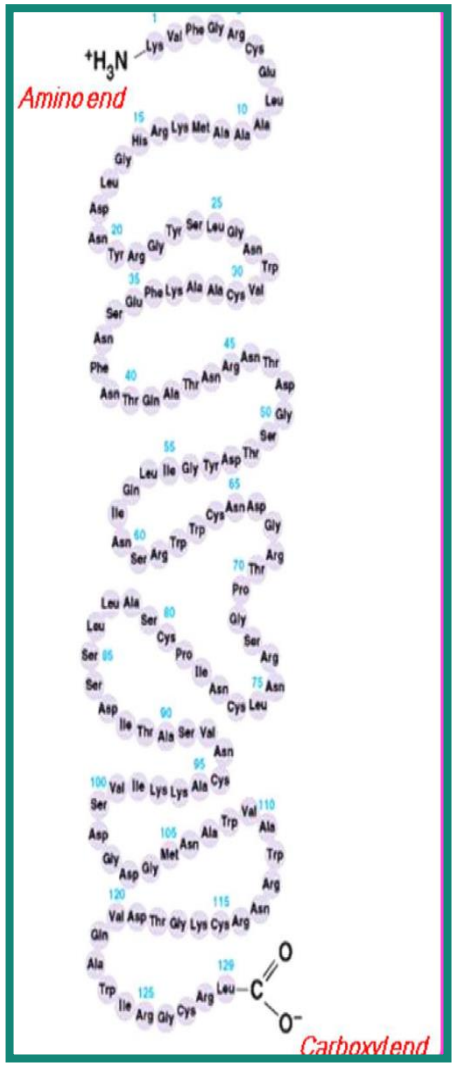
The pancreas secrets enzymes that act on all major types of food stuffs.

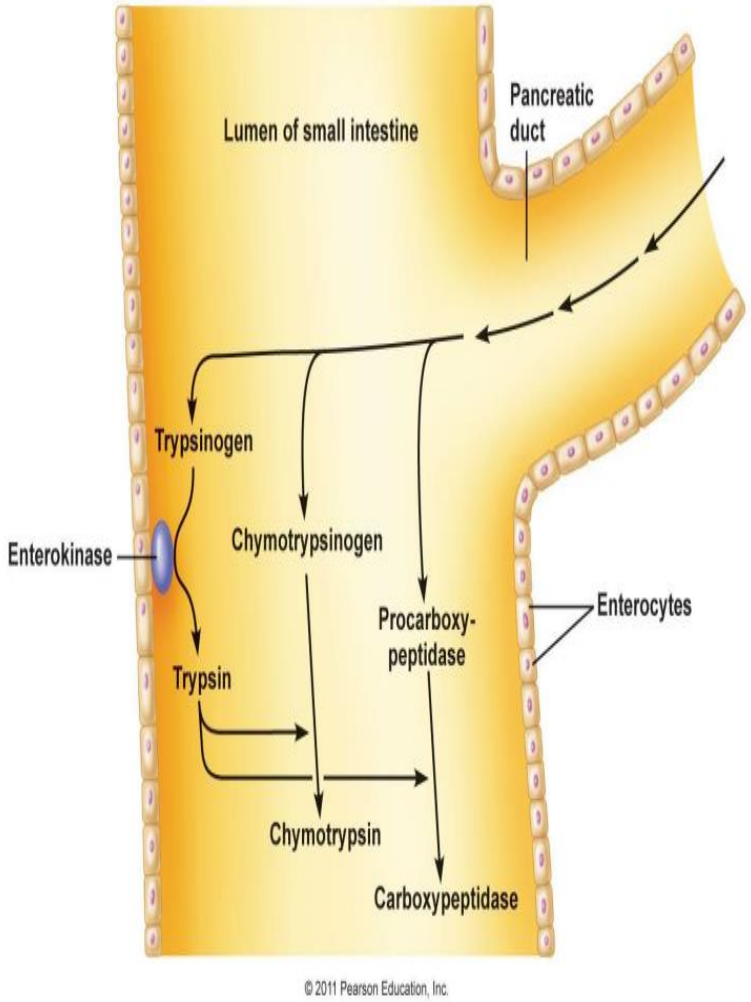
1- Pancreatic proteolytic enzymes (proteases)

- Trypsin, chymotrypsin, elastase, carboxypeptidase(Carboxypolypeptidase).
- They are secreted in inactive form (so they don't digest pancreatic tissue) and activated in intestinal lumen.
- Trypsinogen is activated into trypsin by the enzyme enteropeptidase (enterokinase), secreted by duodenal mucosal cells. Enterokinase is not a proteolytic enzyme but it activates trypsin which in turn activates all pancreatic proteolytic enzymes.
- **Trypsinogen can be autocatalytically activated by trypsin formed from previously secreted trypsinogen**
- Trypsin activates chymotrypsinogen to chymotrypsin, proelastase to elastase and procarboxypeptidase into carboxypeptidase.



- Trypsin, chymotrypsin and elastase are endopeptidases (working in the middle of the chain), splitting protein into shorter peptide chains.(don't cause a release of individual AA)
- Carboxypeptidase is an exopeptidase (working on terminal branches) which splits off amino acids at the carboxyl terminus of the peptide. (cause a release of AA)
- Trypsin inhibitor is present in cytoplasm of glandular cells. It inhibits activation of trypsin in acini and ducts of the pancreas.
- Absence of trypsin inhibitor can result in pancreatic auto-digestion which is a fatal condition.





Pancreatic Enzymes

2- Pancreatic amylase

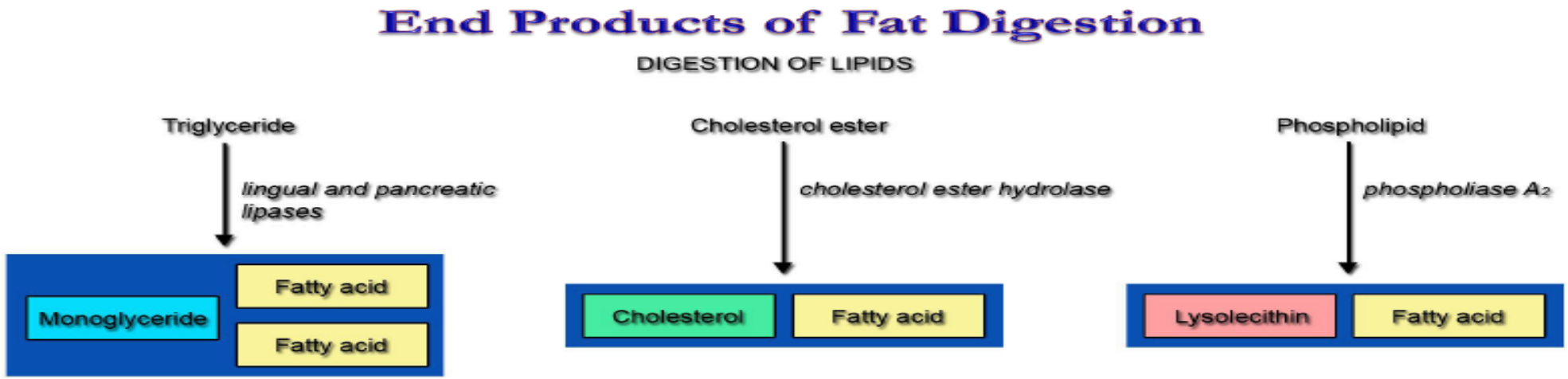
It splits starch to maltose, maltotriose and dextrins except cellulose . Pancreatic amylase is similar to salivary amylase however, food lasts for a longer period here thus, giving pancreatic amylase a chance to digest for a longer duration.

3- Enzymes for fat digestion

a) Pancreatic lipase is the most important fat splitting enzyme. It breaks TG into MG and FA in the presence of bile salts and co-lipase (they are only facilitatory, they don't digest lipids.)

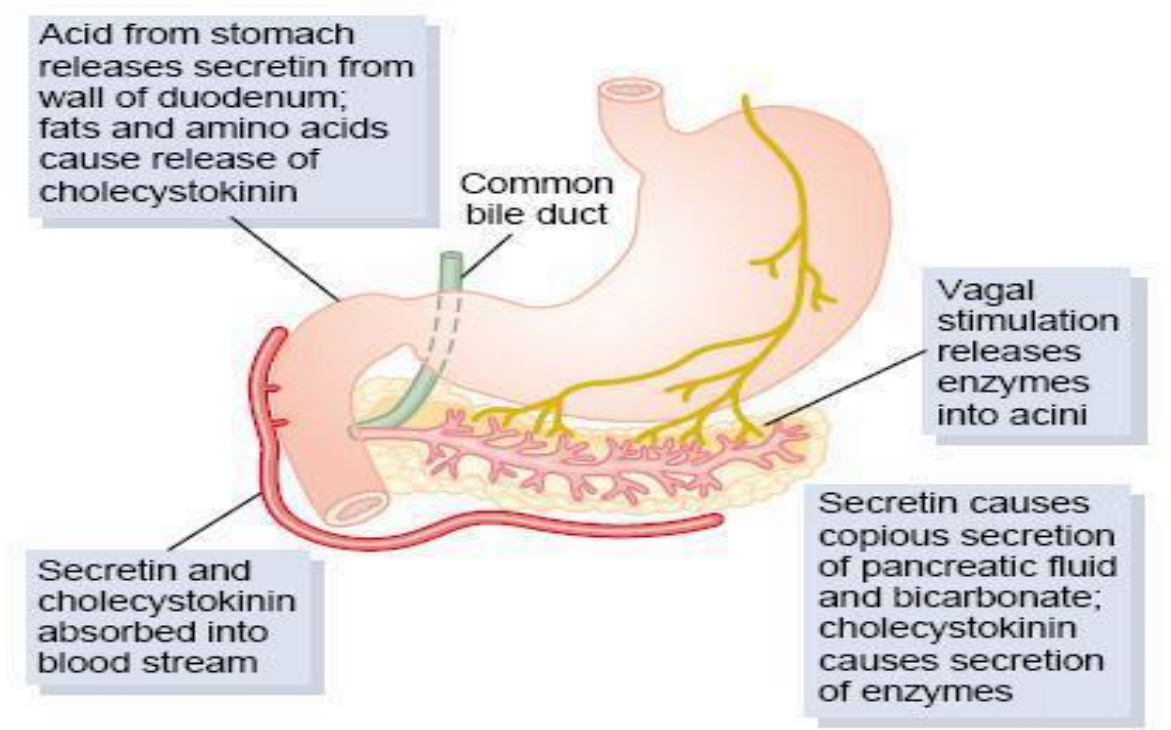
b) Cholesterol esterase which liberates cholesterol.

c) Phospholipase A2 which splits phospholipids into lysophospholipids & FA.

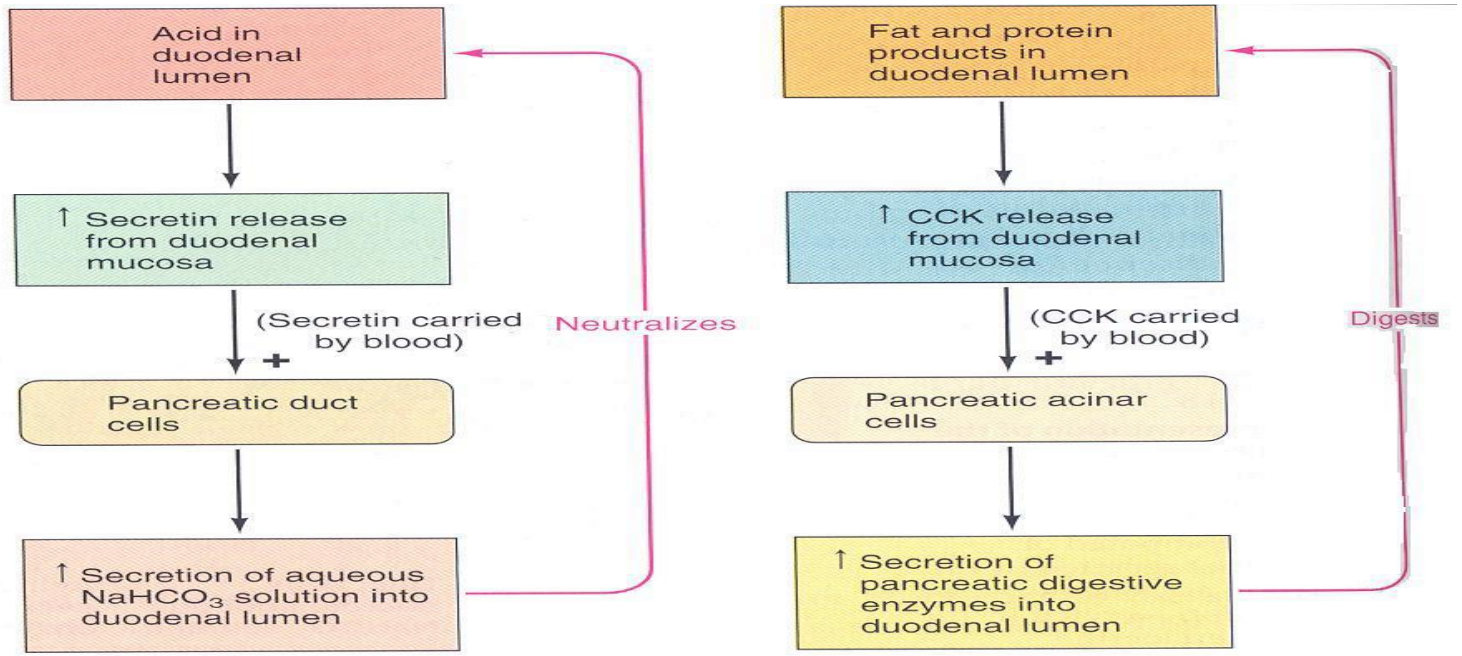


Pancreatic secretion is under neural & hormonal control

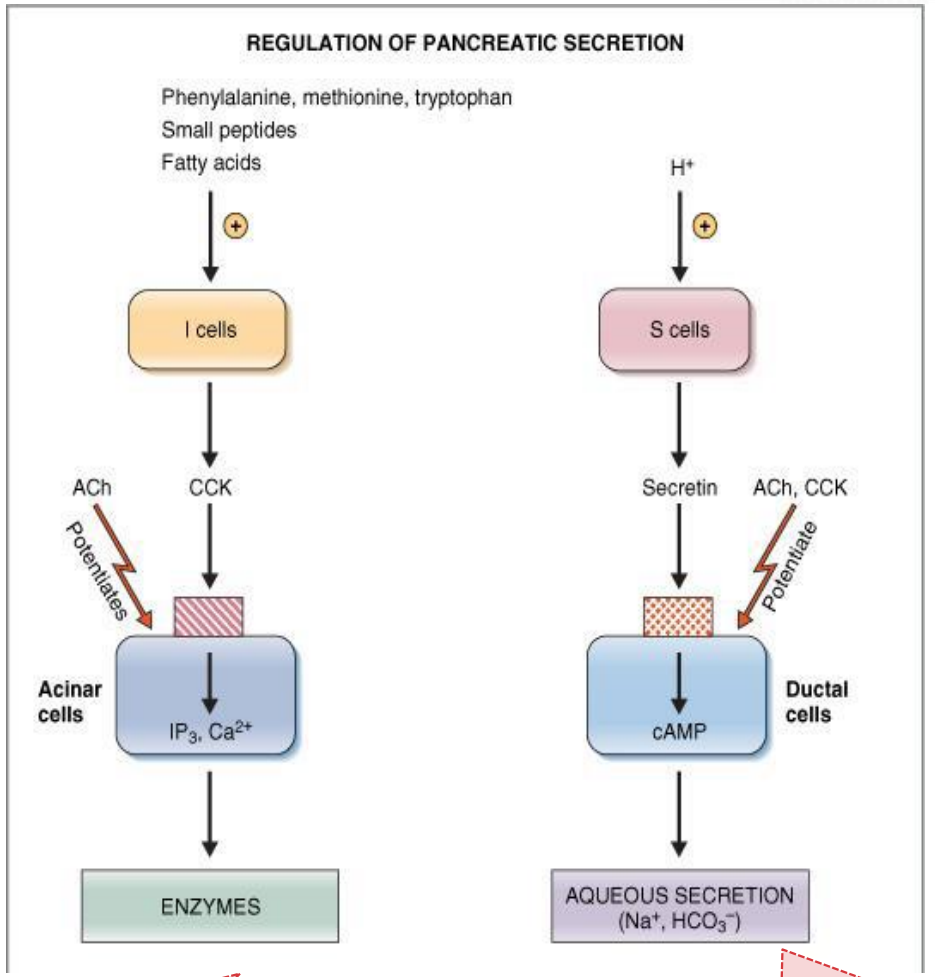
- Parasympathetic stimulation (through Ach on acinar cells) results in an increase in enzyme secretion-fluid and HCO_3-.
- Secretin tends to stimulate a HCO_3- rich secretion by activating ductal cells.
- Cholecystokinin (CCK) stimulates a marked increase in enzyme secretion by stimulating the acinar cells.
- Pancreatic secretion normally results from the combined effects of the multiple basic stimuli, not from one alone (potentiate each other).
- Secretin & CCK augment each other's effect.
- Ach and CCK stimulate acinar cells producing large
- quantity of Pancreatic enzyme and small quantity of
- water and electrolyte in contrast to Parasympthetic
- and secretin stimulation which will secrete large
- quantities of H_2O and $NaHCO_3$.
- Multiplication or potentiation effect occur when different pancreatic stimuli occur at once Then the total secretion is far greater than the sum of each and which is usually the setting.



Phases of pancreatic secretion



Phase	Stimulus	Mediator
Cephalic phase	Smell, taste, chewing and swallowing	Release of Ach and gastrin
Gastric phase	Protein, gastric distention	Vago-vagal reflex
Intestinal phase	Acid in chyme, fatty acids	Secretin, CCK and vago-vagal reflex



To digest food end products

To neutralize acidity

Important

	Secretin	Cholecystokinin (CCK)
Released from	S cells in upper intestinal mucosa	I cells in upper intestinal mucosa
Stimuli	Mainly acids (pH 4 or less) (PH<4.5-5)	Mainly by AA & FA & peptones ,proteoses
Effect on pancreas	Acts on pancreatic duct cells to stimulate secretion of HCO_3- and H_2O.	1. acts on pancreatic acinar cells to stimulate enzyme secretion. 2. Has trophic effect on pancreas.
Effect on stomach	Inhibits gastric motility, contracts pylorus and slows gastric emptying.	Stimulates gastric motility, contracts pylorus thus slows gastric emptying.
Effect on intestines	Inhibits intestinal motility & contracts ileocecal sphincter.	stimulates intestinal motility.
Effect on LES	Relaxation	Relaxation
Extra effects	1. Acts on biliary duct cells to stimulate hepatic bile flow and HCO_3- secretion. 2. Inhibits gastric acid secretion and gastrin release. 3. Stimulates pepsin secretion.	1. contracts gall bladder, relaxes sphincter of Oddi and causes bile discharge into intestine. 2. May be concerned with the mechanism of satiety.

Finally

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Remember that Smooth seas don't make skillful sailors*