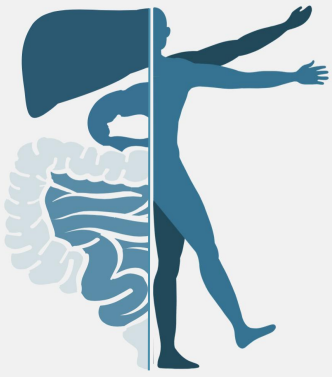
Physiology Team 439

MED439
KING SAUD UNIVERSITY

Revised & Approved
Bassam Alasmari
Rania Almutiri



Physiology of Pancreas

Objectives:

- Discuss the functional anatomy of the pancreas, its division into endocrine and exocrine organ and the role of each.
- Describe the role of the pancreas in digestion.
- Discuss the components of pancreatic juice and their role in digestion.
- List the proteolytic enzymes synthesized by the pancreas and their target.
- Discuss the mechanism of secretion of bicarbonate-rich secretions by the pancreas.
- Describe the mechanism of secretion and activation of pancreatic enzymes.
- Discuss the hormonal & neural mechanisms regulating pancreatic secretion.
- Potentiation of the secretory response
- Name and describe the phases of pancreatic secretion.

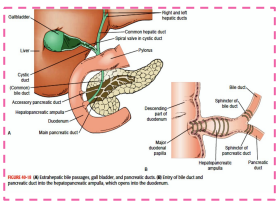
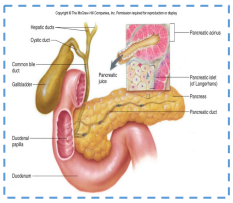
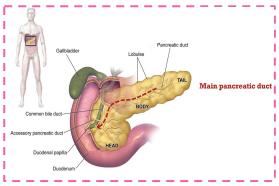
Color index:

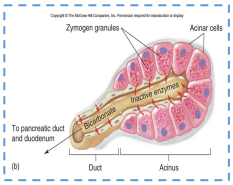
- Important.
- Girls slide only.
- Boys slide only.
- Dr's note.
- Extra information.

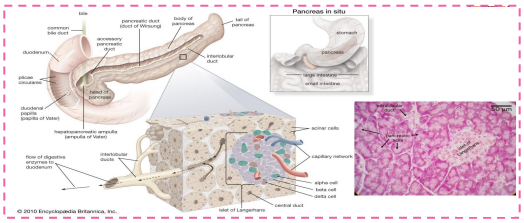
Editing File

Functional Anatomy

- The pancreas is an elongated organ that lies in the epigastric and left upper quadrant of the abdomen.
- It lies behind and below the stomach
- The Pancreas which lies parallel to and beneath the stomach is a large compound gland with most of its internal structure similar to that of the salivary glands.
- The pancreatic digestive enzymes are secreted by pancreatic acini, and large volumes of sodium bicarbonate solution are secreted by the small ductules and larger ducts leading from the acini.
- Pancreatic juice is secreted in response to the presence of chyme in the upper portions of the small intestine
- The pancreas is a mixed gland; endocrine & exocrine...





Click here to check an extra helpful picture

Pancreas has a:
Head: towards spleen (in the concavity of the duodenum)
Tail : towards duodenum (reach the spleen)
Body : extend to the left of the epigastric region
Parallel to stomach :
pancreatic duct that comes together with the bile duct.

There are many Anatomical variations that are important surgically.
There is small accessory arise from Pancreatic duct that opens a bit superior to the main duct and can be seen in certain people.

What is the difference between endocrine and exocrine glands?

Exocrine*

- Exo ? Because it secret its products into a duct system that takes it somewhere else " in GI system will secret into duct system then into duodenum".
- Constitute 90% of pancreas
- lined by Epithelial acinar cell ⇒ secretes Digestive enzymes of pancreas and these flow through ducts which lined by a different type of epithelial cells caned ductal cells.
- both يصبون ⇒ in the duct system of the pancreas, which then empties in the duodenum
- Made of acinar (grap like) & ductal cells Acinar cells are the factory of pancreatic enzymes
- Secretes digestive enzymes, HCO_3^- and water into duodenum

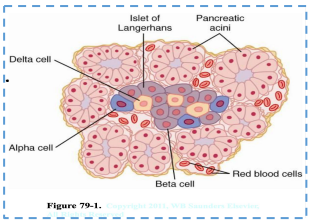
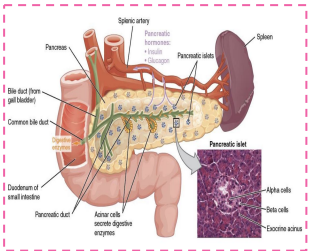


Figure 79-1.



Endocrine

- End ? Because it Produce hormones and secret it directly in the blood
- Constitute 2% of pancreas
- Made of islets of langerhans Islands of specialized types of endocrine cells alpha and beta, each of them secret a hormone.
  Alpha cells> glucagon
  Beta cells> insulin
- Islet of Langerhans contain alpha, beta and delta cells.
- In addition to the digestive functions it Secretes important hormones directly into blood such as :
  1- Insulin : 60% from beta cells and is crucial for normal regulation of glucose, lipid and protein metabolism
  2- Glucagon : 25% from alpha cell and is crucial for normal regulation of glucose, lipid and protein metabolism
  3- Somatostatin : 10% from delta cell

Pancreatic Secretions "juice"

The major functions of pancreatic secretion*/The role of the pancreas*

1

To neutralize the acids in the duodenal chyme to optimum range (PH= 7.0-8.0) for activity of pancreatic enzymes./Neutralize duodenal acidity arriving from stomach.
All pancreatic enzymes that get released from the pancreatic duct towards lumen of duodenum are in their inactive form. They are activated by pancreatic juice, which has high levels of bicarbonate that will neutralize the pH. It increases pH from 3-4 to 7-8. Once the pH becomes 7-8, pancreatic enzymes can be activated.

Why is it important to neutralize acid arriving at the duodenum from the stomach?
Pancreas يصب in the duodenum "which contain chyme from stomach, and highly Acidic" And the duodenal mucosa is not prepare to deal with this Acidity. So, pancreas has role in neutralize this chyme that is arriving from the stomach.
Secondly, need to
continue digesting the nutrients in the chyme.
- does the Alkalinization is only important to protect duodenal mucosa?

Every enzyme has an Ideal pH where it can work. Pancreatic enzymes work at alkaline pH → So, The environment needs to become Alkalinization in order for these enzymes to work properly in the lumen of the duodenum. So to Summarize:
1\ To protect duodenal mucosa from the acidity.
2\ To provide the proper environment for pancreatic enzymes to work.

2

To prevent damage to duodenal mucosa by acid & pepsin.

3

To produce enzymes involved in the digestion of dietary carbohydrate, fat, and protein./To digest dietary nutrients

Constituents of pancreatic secretion "juice"

Only in girls slides

Pancreatic secretion (pH = 7.6 to 9.0)

Enzymes

- For protein digestion → Trypsin, Chemotrypsin, Carboxypolypeptidase
- For carb digestion → Pancreatic amylase (Similar to salivary amylase "in the mouth")
- For DNA & RNA → Nucleases
- For fat digestion → Pancreatic lipase, Phospholipase, Cholesterol esterase

Aqueous solution

Rich in bicarbonate
Water + bicarbonate
Alkaline

Making up the pancreatic secretions is alkaline in nature.

Characteristics of Pancreatic Enzymes*

Only in boys slides

Proteolytic (For protein)

Endopeptidases:
Trypsin(ogen)
Chymotrypsin(ogen)
(Pro)elastase

Exopeptidase:
(Pro)carboxypeptidase

Amylolytic
(For carbohydrates and lipids)

α-Amylase
Lipases
(Pro)phospholipase A2
Carboxylester hydrolase
(cholesterol esterase)

Nucleolytic
not abundant
(For RNA and DNA)

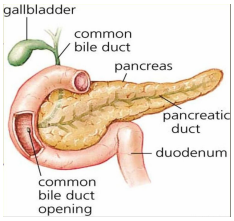
Ribonuclease
Deoxyribonuclease

The suffix -ogen or prefix pro- indicates the enzyme is secreted in an inactive form

Flow of Pancreatic Secretion into Duodenum*

Click here to check an extra helpful picture Only in boys slides

- The combined product of enzymes and NaCo3 flows through a long pancreatic duct
- Pancreatic duct joins the hepatic duct immediately before it empties into the duodenum through the papilla of Vater, surrounded by sphincter of Oddi



Pancreatic Secretions

Only in girls slides

Acinar cell	Ductal cell
secrete the pancreatic digestive enzymes	secrete NaHCO3 solution
Secrete a protein-rich (digestive enzymes) secretion in an isotonic plasma-like fluid.	Secretes a HCO3- rich fluid that alkalinizes & hydrates the protein-rich secretion of acinar cells ([HCO3] =145mEq/L).
Constitute 25% of total pancreatic secretion.	Constitute 75% of pancreatic secretion.Secreted by s cells of the duodenum, stimulate ductal cells to produce bicarb rich solution
Stimulated by CCK & Ach.	Stimulated by Secretin. Effects of Secretin are potentiated by CCK & Ach.

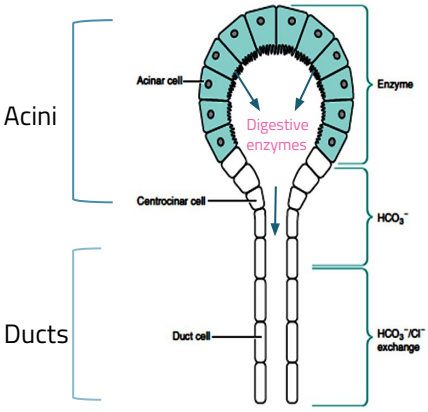


Fig. 5.4
Secretory unit showing the cellular locations of the different secretions.

Because it's rich in digestive enzymes and the solution is low, they are thick. So, to flow into the duct need solution to carry it, will come from ductal cells which will secret bicarb rich solution to wash out these digestive enzymes and carry it through the duct all system until they arrive the duodenum.

Only in girls slides

Why don't these enzymes (secreted from acini) digest the pancreas?

1. Secreted as proenzymes (inactive form) which get activated in the lumen of the intestine.
2. The same cells secrete a substance "trypsin inhibitor". Discussed Later

Only in boys slides

- Pancreatic secretions contain many enzymes for digesting proteins, carbohydrates, and fats, and large quantities of HCO3 ions
- The most important pancreatic enzymes for digesting proteins are:

Trypsin

Chymotrypsin

Trypsin and chymotrypsin split whole and partially digested proteins into peptides of various sizes but **do not cause release of individual amino acids**.
they give off oligopeptide, dipeptide but NOT amino acids

Carboxypolypeptidase

Carboxypolypeptidase **splits some peptides into individual amino acids**, thus completing the digestion of some proteins to amino acids.

Trypsin, chymotrypsin and carboxypolypeptidase are released from the pancreatic duct in their inactive form (otherwise they can digest the pancreatic duct and tissue).

Pancreatic Enzymes

When first synthesized in the pancreatic cells, the proteolytic digestive enzymes are in the inactive forms:

1. Trypsinogen
2. Chymotrypsinogen
3. Procarboxypolypeptidase

These enzymes become activated only after the are secreted into the intestinal tract

Trypsinogen is activated by:

- Enteropeptidase (enterokinase), an enzyme secreted by the brush border of the small intestine/ the intestinal mucosa when chyme comes in contact with the mucosa
- Trypsinogen can be autocatalytically activated by trypsin formed from previously secreted trypsinogen

2 & 3 Chymotrypsinogen and procarboxypolypeptidase are activated by trypsin to form chymotrypsin and carboxypolypeptidase

In the lumen of the duodenum in the enterocytes of the intestine, there is an enzyme called Enterokinase on the brush border. When the pancreatic enzymes reach the lumen, trypsinogen get activated by enterokinase and gives us trypsin.
Trypsin can autocatalysis the activation of itself and catalysis the activation of other pro-enzymes. Trypsin is the key enzyme, if I inhibit it, I can inhibit the activation of all these enzymes along with trypsinogen as well.

Trypsin Inhibitor

Secretion of Trypsin Inhibitor Prevents Digestion of the Pancreas Itself. How?

Proteolytic enzymes of the pancreatic juice do not become activated until after they have been secreted into the intestine because the trypsin and other enzymes would digest the pancreas itself

The same cells that secrete proteolytic enzymes into the acini of the pancreas secrete another substance called trypsin inhibitor, which is formed in the cytoplasm of the granular cells, and it prevents activation of trypsin both inside the secretory cells and in the acini and ducts of the pancreas

Because trypsin activates the other pancreatic proteolytic enzymes, therefore trypsin inhibitor prevents activation of other enzymes as well

How do we guarantee that enzymes stay in their inactive form at the pancreatic duct and tissues? trypsin inhibitor released by acinar cells
Trypsin will be inhibited first, then the rest will stay in their inactive form until they reach the lumen of duodenum and trypsin inhibitor becomes degraded.

Activation of Trypsinogen in duodenal Lumen*/ Activation of Pancreatic enzyme precursors in small intestine*(M)

Precursor

Active enzyme

Activation of Gastrointestinal Proteases

(A) Stomach

Pepsinogen → (Low PH) → Pepsin

(B) Small intestine

Trypsinogen → (enterokinase (brush border), trypsin) → Trypsin

Trypsin → (+) → Trypsin

Trypsinogen	Chymotryposinogen	Proelastase	Procarboxypeptidase A or B	Prophospholipase A
↓ trypsin	↓ trypsin	↓ trypsin	↓ trypsin	↓ trypsin
Trypsin	Chymotrypsin + peptide	Elastase+ Peptide	Carboxypeptidase A or B +peptide	Phospholipase A + peptide

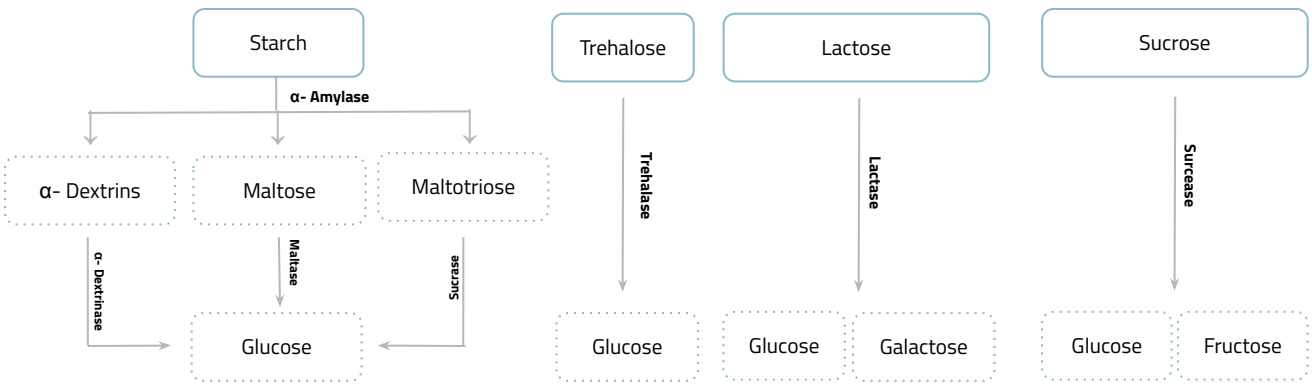
When these enzymes are released into the lumen of duodenum, they get exposed to enteropeptidase/enterokinase also known as the brush border enzyme that comes from the mucosal epithelial cells of duodenum. They work on trypsinogen **first** and activate it, then trypsin will activate other trypsinogen and the other enzymes (chymotrypsinogen and procarboxypeptidase)

Pancreatic secretion

Only in boys slides

The pancreatic enzyme for digesting carbohydrates is pancreatic amylase, which hydrolyzes starches, glycogen and most other carbohydrates (except cellulose) to form mostly disaccharides and a few tri-saccharides

Carbohydrate Digestion



α- amylase is the only enzyme needed from pancreas to break down starch into a disaccharide form. If we ingest a disaccharide we don't need an enzyme from the pancreas since it can be digested to a monosaccharide by brush border enzymes of mucosal luminal layer of epithelium.

Remember carbs should be in a monosaccharide form to be absorbed.

Pancreatic secretion

Only in boys slides

The main enzymes for fat digestion are:

1. Pancreatic lipase
2. Cholesterol esterase
3. Phospholipase

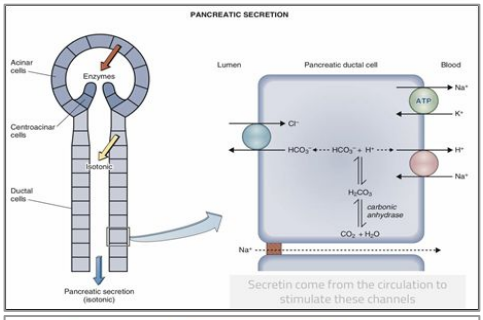
Digestion of Lipids

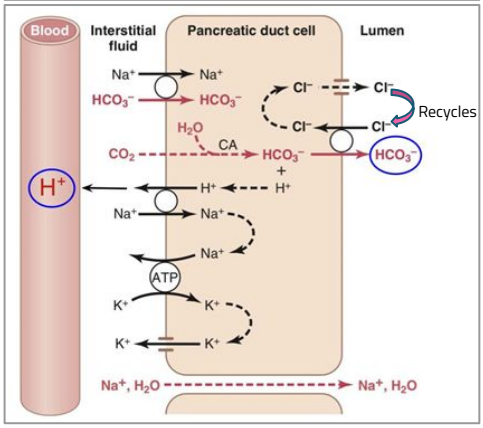
Triglycerides	Cholesterol ester	Phospholipid
↓ Lingual and pancreatic lipases	↓ Cholesterol ester hydrolase	↓ Phospholipase A_2
Monoglyceride + Fatty acid + Fatty acid	Cholesterol + Fatty acid	Lysolecithin + Fatty acid

Mechanism of Bicarbonate (HCO_3^-) Secretion:

Explained by both doctors

1. CO_2 and H_2O combine in ductal cells to form H_2CO_3.
2. H_2CO_3 dissociated into H^+ and HCO_3^-.
3. H^+ is transported into blood by Na^+-H^+ exchanger at basolateral membrane of ductal cells. makes blood around pancreas acidic "Acidic tide"
4. HCO_3^- is secreted into pancreatic juice by Cl^--HCO_3^- exchanger at apical membrane of ductal cells.





Absorption of H^+ causes acidification of pancreatic venous blood.
water will flow to neutralize osmolarity

Our body 60% water and has CO2 from metabolism, blood..
If both brought together in presence of CA= carbonic acid, which dissociate immediately into bicarb and H+
Bicarb leave the cell into through the lumen side in exchange for Cl- (from acinar cells or recirculation from inside the cell into out) CL- recycle for this to happen.
H+ leave the cells into the blood in exchange for Na+ (which move down its gradient because it's higher in outside than inside the cells "due to Na+ K+ atpase "). So this is secondary active transport.

In Cystic fibrosis
One of the Cl- channels called cystic fibrosis transmembrane conductor.
In certain people has genetic mutation in this Cl- channel, this mean it will not do its job, there will not be recycling Cl-, so no secretion of bicarb.
So the secretion "from pancreas" will be thick, The risk of blockage of these ducts system " of pancreas" increases, so these people can develop pancreatitis "due to blockage of the ducts from thick juices". In addition to pancreatitis, they also suffer that the secretion of the lungs are thick causing obstructive problems in the lungs.
So this is what happen in cystic fibrosis, their secretion become thick, blockage in the pancreas that can lead to pancreatitis. In the lung, obstruction of the airways.

Only in boys slides

Pancreatic secretions and Bicarbonate ions (HCO_3^-)

- The pancreas secrets about 1 L/day of HCO_3^- rich fluid from the epithelial cells of the ductules and ducts.
- The osmolarity of pancreatic fluid is equal to that of plasma. Isotonic
- HCO_3^- concentration increases with increasing secretion rate. If you activate the pancreatic tissue to release more pancreatic enzymes -> bicarbonate concentration and production.

Flow Rate and Pancreatic Secretion

Graph explained by both doctors

Stimulating the pancreatic tissue will benefit the pancreatic secretion.
Why? because it increases bicarbonate secretion and decreases Cl^- secretion. more activation = more bicarbonate = more flow rate.
Na^{+2} and K^+ are not affected by the flow rate of pancreatic secretion.
While, $HCO3^-$ and Cl^- are affected by the flow rate.

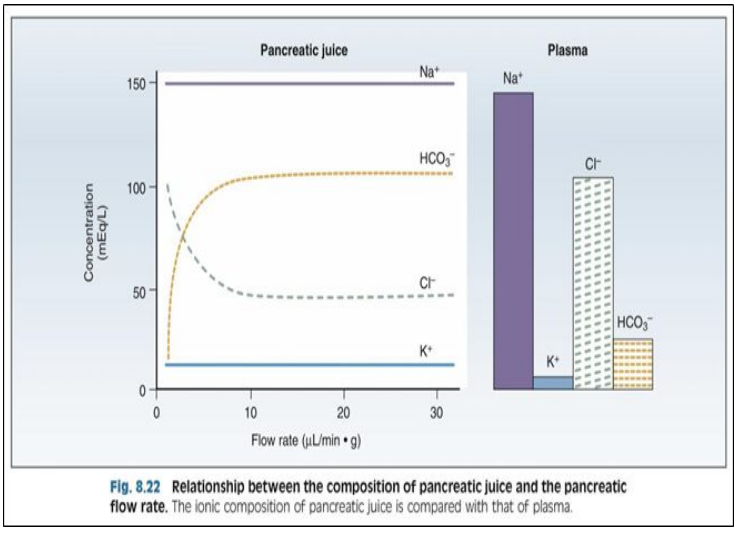


Fig. 8.22 Relationship between the composition of pancreatic juice and the pancreatic flow rate. The ionic composition of pancreatic juice is compared with that of plasma.

Female Dr Explanation:
Na+ and K+ = the same.
Cl-= plasma > pancreatic juice.
Bicarb = plasma < pancreatic juice.

Does the the velocity of the flow of the juice affect the composition of the electrolyte ?
Increase the flow rate lead to :
- Na^+ and K^+ = no changes
- increase HCO_3^- level
- decrease Cl^-

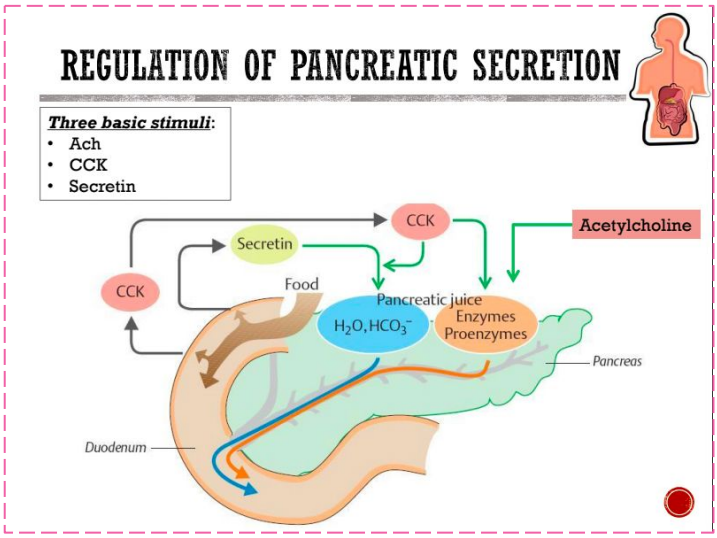
When the pancreatic flow rate changes, the Na^+ and K^+ concentrations in the pancreatic juice remain constant, whereas the concentrations of $HCO3^-$ and Cl^- change. In pancreatic juice, there is a reciprocal relationship between the Cl^- and $HCO3^-$ concentrations which is maintained by the Cl^- - $HCO3^-$ exchanger in the apical membrane of ductal cells. At the highest pancreatic flow rates $HCO3^-$ concentration of pancreatic juice is highest (and much greater than plasma HCO^-) and the Cl^- concentration is lowest. At lowest flow rates, $HCO3^-$ is lowest and Cl^- is highest.

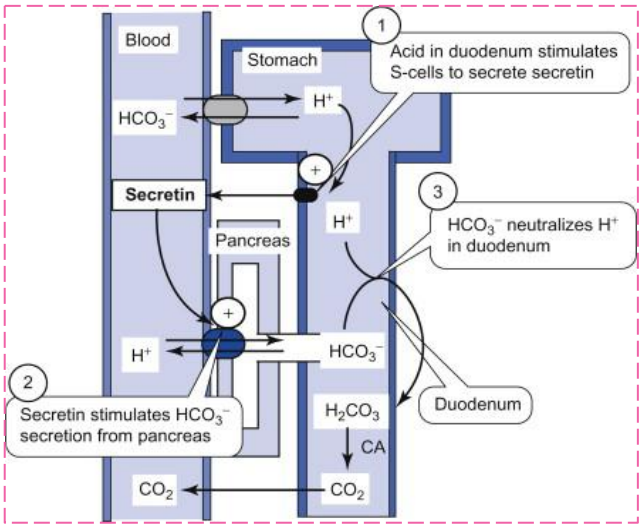
Neural and Hormonal Control of Pancreatic Secretion(Regulation)

	Parasympathetic	Secretin	Cholecystokinin(CCK)
Stimulation	Stimulation (through Ach on acinar cells) results in an increase in enzyme secretion-fluid and $HCO3^-$ *	- Tends to stimulate a HCO3- rich secretion by activating ductal cells.* mainly -Secretin Stimulates Secretion of Copious Quantities of Bicarbonate Ions—Neutralization of Acidic Stomach Chyme.*	-Stimulates a marked increase in enzyme secretion by stimulating the acinar cells. -Its Contribution to Control of Digestive Enzyme Secretion by the Pancreas./ CCK → ↑ pancreatic digestive enzyme secretion.
Secretion	Acetylcholine: released from the parasympathetic vagus nerve endings and from other cholinergic nerves in the enteric nervous system.*	Secretin is a 27 amino acid polypeptide secreted by the duodenal and upper jejunal mucosa (S cells) when highly acidic chyme enters the small intestine./ When luminal pH<4.5 Stimulated by H^+	CCK is a 33-amino acid polypeptide secreted by the duodenal and upper jejunal mucosa (I cells) when food enters the small intestine.
Mechanism		1-Secretin is present in an inactive form, prosecretin (in S cells in the mucosa of the duodenum and jejunum). 2-When acid chyme with pH less than 4.5-5.0 enters the duodenum from the stomach, it causes duodenal mucosal release and activation of secretin, which is then absorbed into the blood. 3-Secretin causes the pancreas to secrete large quantities of fluid containing a high concentration of HCO3- (up to 145mEq/L = ~5X normal) but a low concentration of Cl- $HCl + NaHCO_3 \rightarrow NaCl + H_2CO_3$ **H_2CO_3 dissociates Immediately into CO_2 and H_2O.** By this rxn we get rid of the acidity in the lumen which comes from chyme from stomach	1-The presence of food in the upper small intestine causes cholecystokinin to be released from the I cells in the mucosa of the duodenum and upper jejunum. 2-Release of cholecystokinin results especially from the presence of proteoses and peptones (products of partial protein digestion) and long-chain fatty acids in the chyme./ Stimulated by the presence of fat and protein degradation products (proteoses & peptides). 3-Cholecystokinin, like secretin, passes by way of the blood to the pancreas and causes secretion of pancreatic digestive enzymes by the acinar cells. This effect is similar to that caused by vagal stimulation but even more pronounced, accounting for 70-80% of the total secretion of the pancreatic digestive enzymes after a meal.

Neural and Hormonal Control of Pancreatic Secretion(Regulation)

*Pictures are from girls slides





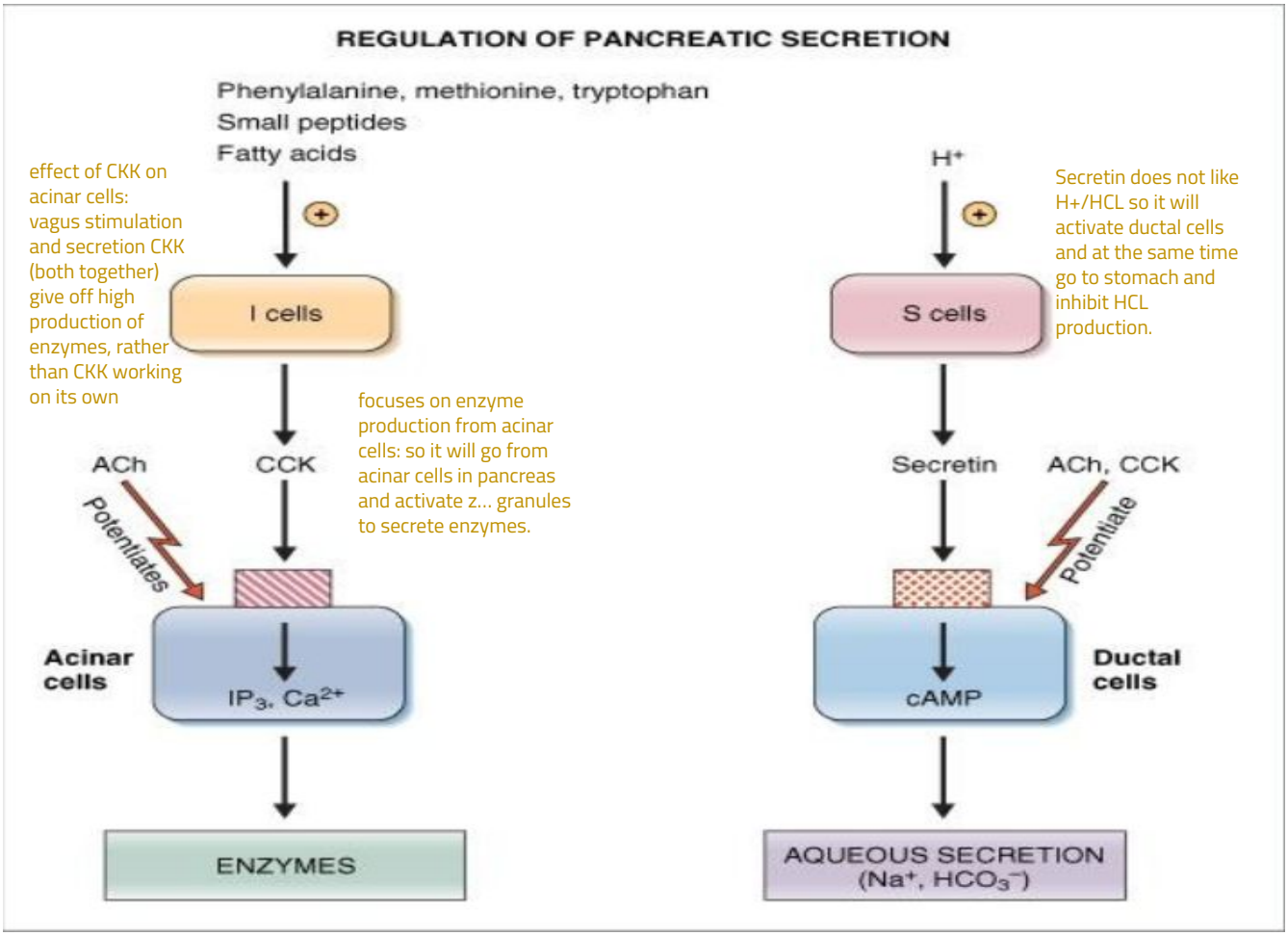
Phases of Pancreatic secretion

Phase	Stimulus	Mediators	Percentage of maximum enzyme secretion*
Cephalic phase I am preparing to eat food (smell + see + start eating " before reach the esophagus ")	Smell, taste,chewing and swallowing	Release of Ach by the vagal nerve	20% of pancreatic enzymes
Gastric phase	Protein, gastric distention	Release of Ach by the vagal nerve	5-10 % Of pancreatic secretion
Intestinal phase **most important** Once the chyme is emptied into the duodenum	Acid in chyme, fatty acid	-Mainly in response to secretin -Through hormonal stimulation (secretin & CCK).	70-75 % Of pancreatic secretion

Multiplicative or Potentiation Effects of Different Pancreatic Secretion Stimuli

*Text was found in boys slides but picture was found in girls and boys slides

- Pancreatic secretion normally results from the combined effects of the multiple basic stimuli, not from one alone (potentiate each other).
- Acetylcholine and cholecystokinin stimulate the acinar cells of the pancreas, causing production of large quantities of pancreatic digestive enzymes but relatively small quantities of water and electrolytes to go with the enzymes.
- Secretin, in contrast to the first two basic stimuli, stimulates secretion of large quantities of H2O and NaHCO3 solution by the pancreatic ductal epithelium.
- When all different stimuli of pancreatic secretion (acetylcholine, cholecystokinin, and secretin) occur at once, then the total secretion is far greater than the sum of the secretions caused by each stimulus separately. The stimuli are said to "multiply" or "potentiate" one another.
- Usually, pancreatic secretions are the result of multiple stimuli rather than one stimulus.



Summary of Gastrointestinal Hormones

Hormone	Hormone family	Site of secretion	Stimuli for secretion	Actions
Gastrin	Gastrin-CCK	G cells of the stomach	-Small peptides and amino acids -Distention of the stomach -Vagal stimulation (GRP)	-↑Gastric H+ secretion -Stimulates growth of gastric mucosa
Cholecystokinin(CCK)	Gastrin-CCK	I cells of the duodenum and jejunum	-Small peptides and amino acids -Fatty acids Need emulsification by bile salts (emulsifying agent) which help lipases from pancreas in digestion	-↑Pancreatic enzyme secretion -↑Pancreatic HCO3- secretion -Stimulates contraction of the gallbladder and relaxation of the sphincter of oddi -Stimulates growth of the exocrine pancreas and gallbladder -Inhibits gastric emptying
Secretin	Secretin-glucagon	S cells of the duodenum	-H+ in the duodenum -Fatty acids in the duodenum	-↑Pancreatic HCO3- secretion -↑Biliary HCO3- secretion -↓Gastric H+ secretion -Inhibits trophic effect of gastrin on gastric mucosa
Gastric inhibitory peptide (GIP) Glucose dependent insulin tropic factor	Secretin-glucagon	Duodenum and jejunum	-Fatty acids -Amino acids -Oral glucose	-↑Insulin secretion from pancreatic β cells كأنه قاعد يقول للجسم : استعد فيه دفعة اكل جاية، فيه جلوكوز يحتاج انسولين -↓Gastric H+ secretion

Summary of Digestion of Food Types

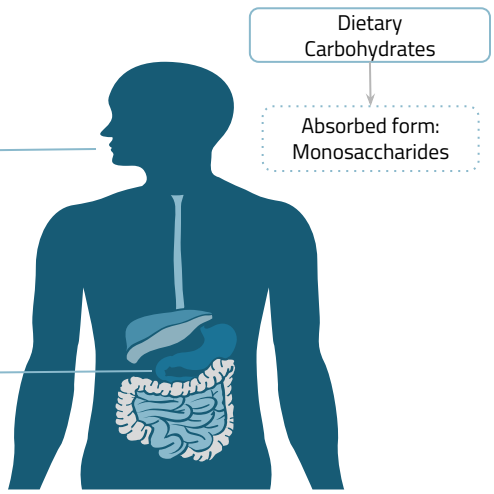
A-Digestion of Carbohydrates (from mouth to duodenum)

Carbohydrates in diet

- Most (2/3) Plant polysaccharides "Starch"
- Glycogen Cellulose "fiber"-not digested by humans
- Remaining are disaccharides; Sucrose (table sugar) Lactose (milk sugar)
  - Sucrose is made of → Glucose+Fructose
  - Lactose is made of → Glucose+Galactose

1-Mouth (20-40%): Starch —(Salivary amylase)→ Oligosaccharides Maltose

2-Duodenum (50-80%): Starch Oligosaccharides —(Pancreatic amylase)→ Small glucose polymers Maltose

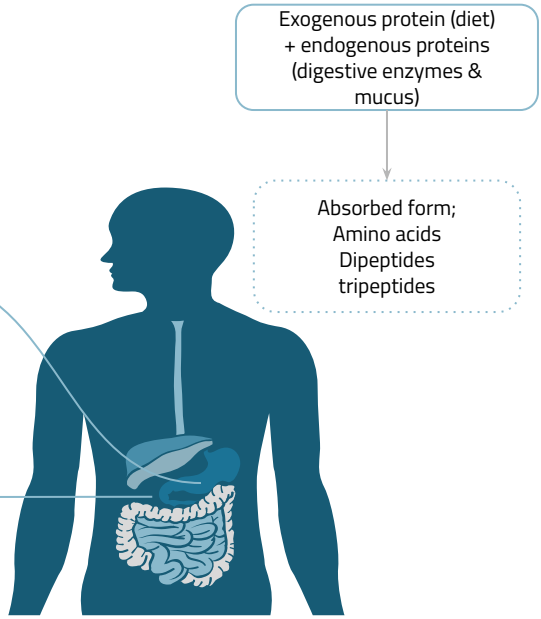


B-Digestion of Proteins (from mouth to Duodenum)

What is the difference between a protein and a peptide??

1-Start at stomach (10-20%): Proteins —(Pepsin HCL)→ Partially digested proteins polypeptides

2-Duodenum:
- Proteoses Peptones —(Trypsin Chemotrypsin)→ Polypeptides
- Polypeptides —(Carboxypeptidase)→ Releases some a.a. Smaller peptides (some Tri- & di-peptides)

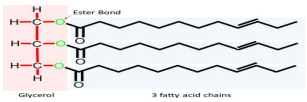


Summary of Digestion of Food Types

C-Digestion of Fat (From mouth to Duodenum)

Fat in diet

- Mostly triglycerides



- Remaining: Cholesterol, Cholesterol esters, phospholipids

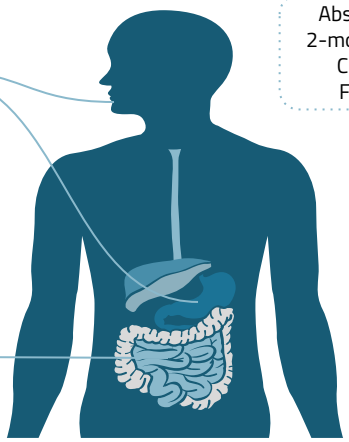
Dietary Fat → Absorbed form: 2-monoglycerides, Cholesterol, Fatty acids

1-Mouth & Stomach (10%)

TGs —(Lingual Lipase)→ Diglycerides, Fatty acids

2-Intestines

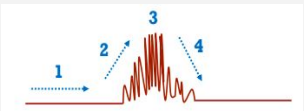
- TGs, DAGs —(Pancreatic Lipase)→ 2-MG, Fatty acids
- CE —(Cholesterol esterase)→ Chol, FA
- Phospholipids —(Phospholipase A2)→ Lysoph., FA



Migratory Motor Complex

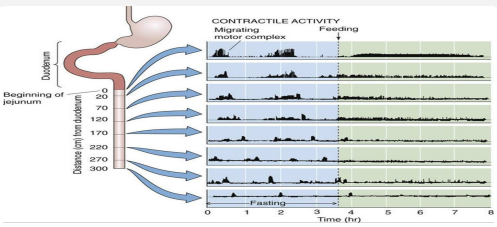
- We understood that the GI tract motility is involved in mixing and moving food along the tract in an orad to caudad direction.
  But what happens when there is no food in the system? During fasting for example?
  The intestine is relatively quiescent during fasting but exhibit a certain pattern of electric and motor activity called Interdigestive myoelectric complexes "Migrating Motor Complexes (MMC)"

- MMC is a term that describes the rhythmic contractions of the small intestine during the fasting state.It starts at the stomach and moves down to terminal ilium at intervals of 90-120 min.
  It Consists of four main phases:
  1. Prolonged quiescent period.
  2. A period of increasing AP and contractility.
  3. A period of peak electrical and mechanical activity.
  4. A period of declining activity.



- These are though to clear the intestine of its contents. Allows particles > 2mm to pass from stomach to duodenum.
- Motilin is thought to play a role in their generation.

Figure 41-6: Mechanical activity in the fasting and fed states. Shown are records of intraluminal pressure along the small intestine of a conscious dog. Before feeding (left side), the pattern is one of MMCs. Feeding triggers a switch to a different pattern, characterized by both segmental contractions that chum the contents and peristaltic contractions that propel the contents along the small intestines.



MCQ & SAQ:

Q1: Which of the following enzymes splits some peptides into individual amino acids?

A. Trypsin
B. Chymotrypsin
C. Carboxypolypeptide
D. Trypsin and chmotrypsin

Q2: Which one of the following activates proelastase into elastase:

A. Enteropeptidase
B. Trypsin
C. Pepsin
D. Secretin

Q3: Trypsin is Considered to be:

A. Endopeptidases Proteolytic
B. Exopeptidases Proteolytic
C. Amylolytic
D. Nucleolytic

Q4: The osmolarity of pancreatic fluid is equal to that of the:

A. Blood
B. Plasma
C. Extracellular interstitial fluid
D. Intracellular fluid

Q5: Secretin stimulates the secretion of which of the following?

A. HCO3-
B. HCL
C. Insulin
D. Mucous

Q6: Which of the following does not control pancreatic secretions?

A. Parasympathetic NS
B. CCK
C. Secretin
D. Gastrin

answer key:
1: C
2: B
3: A
4: B
5: A
6: D

1- What are the functions of pancreatic enzymes?

2- Why doesn't Trypsin digest the pancreas?

3- Explain the mechanism of bicarbonate secretion

4- List the phases of pancreatic secretions with mentioning their stimulus and mediators.

A1: To neutralize duodenal acidity coming from stomach, prevent damage to duodenal mucosa by acid and pepsin, and to produce enzymes involved in the digestion of dietary carbohydrate, fat, and protein.

A2: Because pancreatic enzymes do not become activated until after they have been secreted into the intestine, also because the pancreas secrete enzyme Inhibitors (Trypsin Inhibitor).

A3: Slide 8

A4: Slide 11

Leaders:

- Abdulaziz Alsuhaim.
- Yara Alasmari.

Co-leader:

- Mayasem Alhazmi.

Organizers:

- **Rawan baqader**
- Haya Alanazi
- Shaden Alobaid
- Mayasem Alhazmi.
- Basel Fakeeha
- Leen Almadhyani

Note takers:

- **Shaden Alobaid**
- **Raghad Albarrak**
- **Homoud Algadheb.**

Revisers:

- Abeer Awwad.

MEMBERS:

- Abdulaziz Alamri
- Basel Fakeeha
- Ahmad Alkhayatt
- Aljoud Algazlan
- Almaha Alshathri
- Abdulrahman Barashid
- Nada Bin Obeid
- Tarfa Alkaltham
- Yara alzahrani
- **Sarah AlQuwayz**
- Abdulrhman Alsuhaibany
- Abeer Awwad
- Mohammed alkathiri
- ziyad alhosan
- Rand Alrefaie
- **Khalid Altuwaijri**
- Omar Alhalabi
- Joud Alarifi
- Ibrahim Alnamlah
- Hamad Almousa
- **Abdullah Alburikan**
- Leen Almadhyani
- Abdullah Alanzan
- Bader Alrayes
- Faisal jazzar
- Khalid Almutlaq
- Yara Alomar
- Reem Alqahtani
- Aljoharah Albnyan
- Saud Alhasani
- Muneerah Alsadhan

Physiology439@gmail.com