Carbohydrate digestion & absorption

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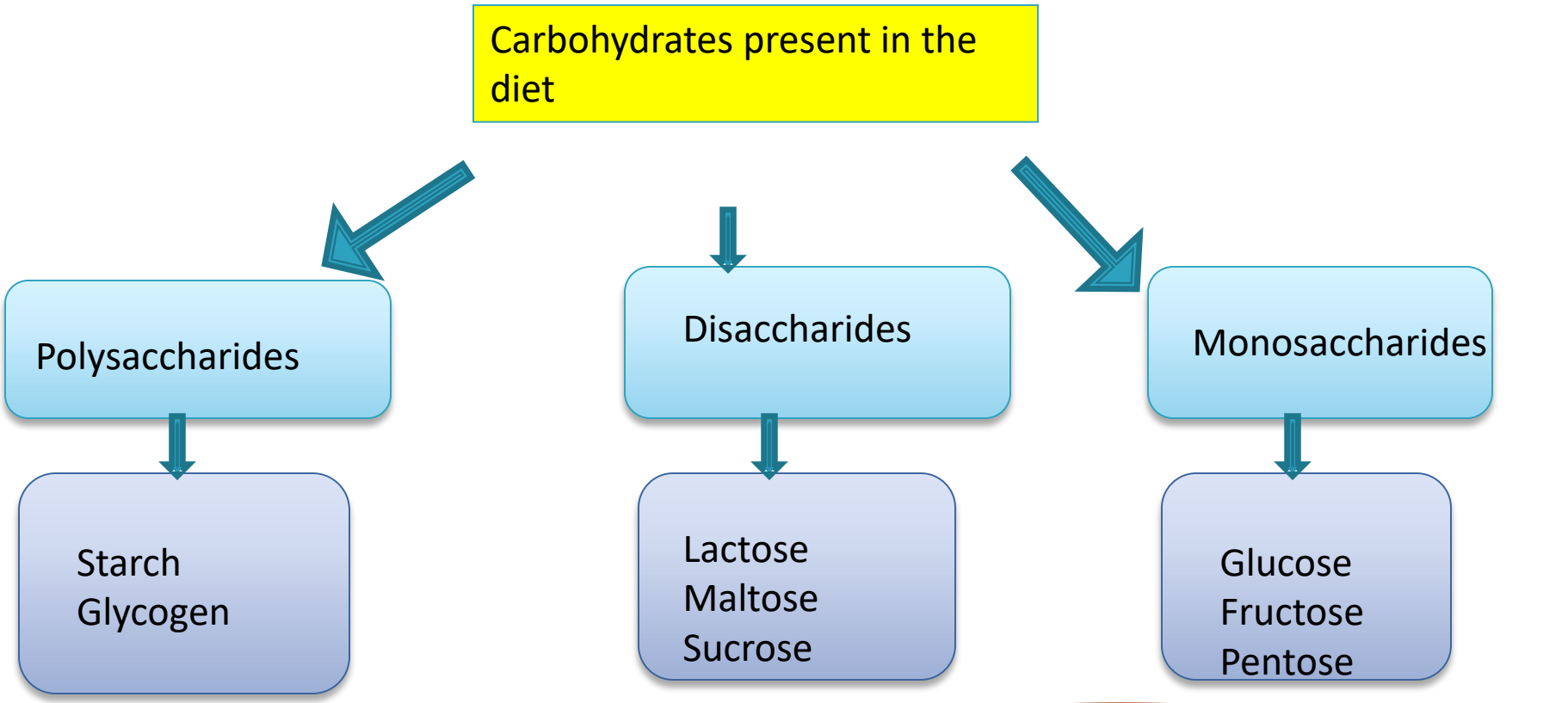
Objectives

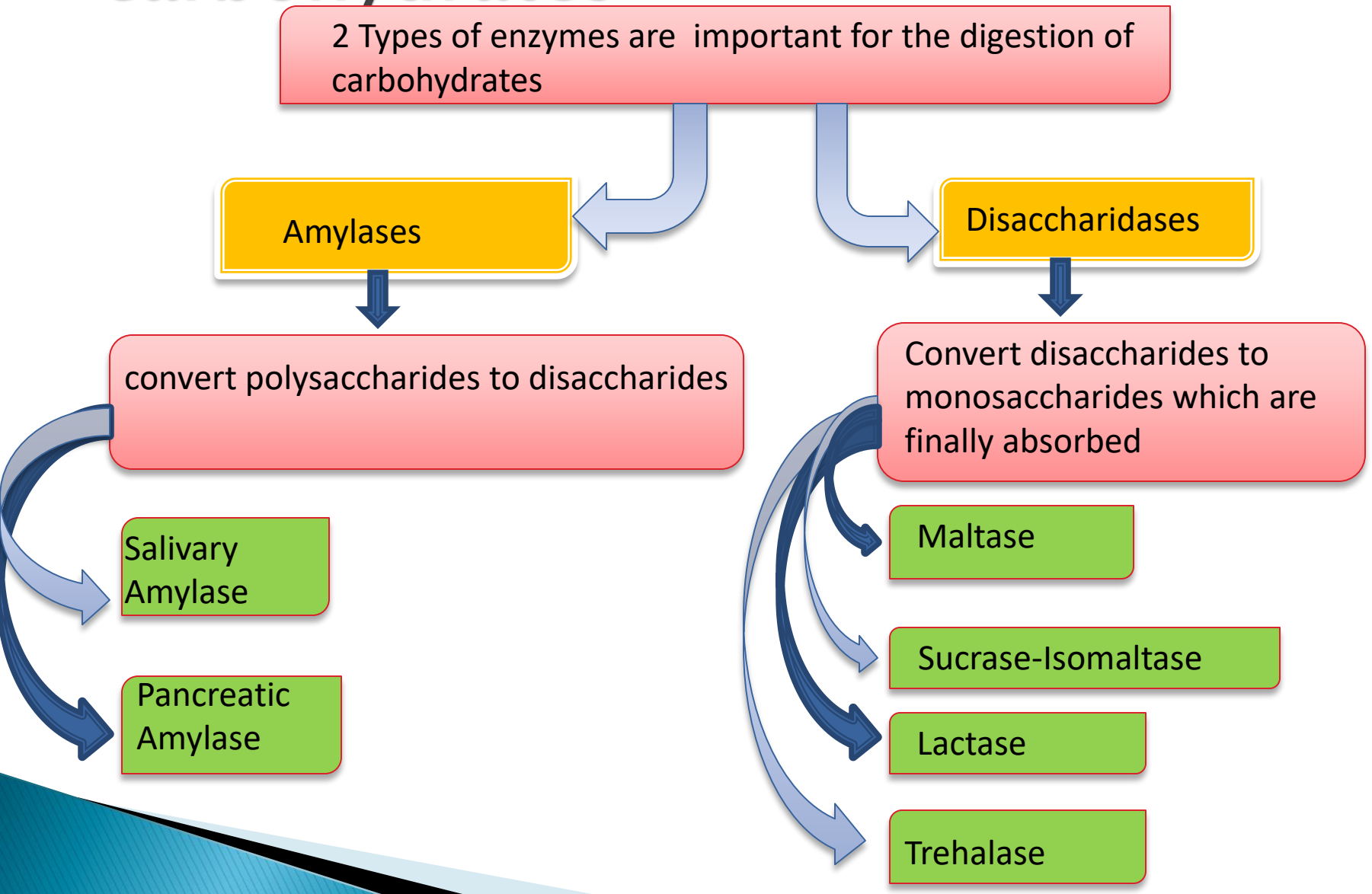
- Digestion of carbohydrates
- Absorption of carbohydrates
- Clinical significance

Digestion and absorption of carbohydrates



In GIT, all complex carbohydrates are converted to simpler monosaccharide form which is the absorbable form.

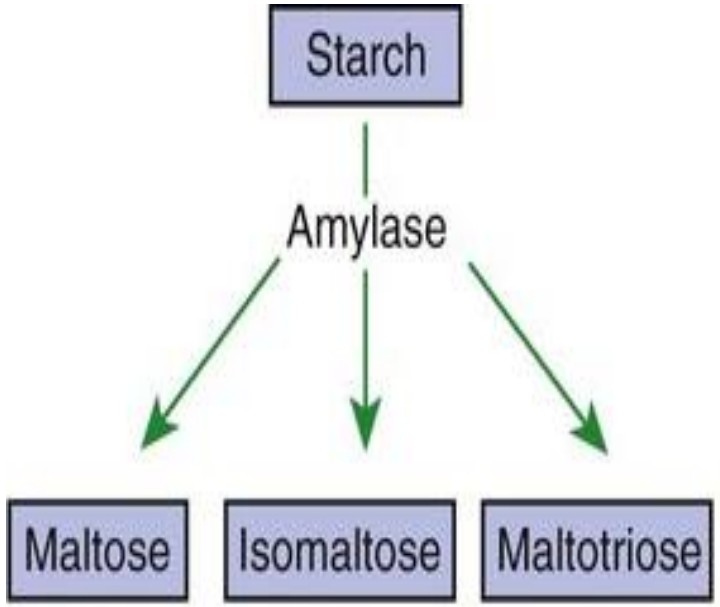
Details of digestion of carbohydrates



Digestion in the Mouth

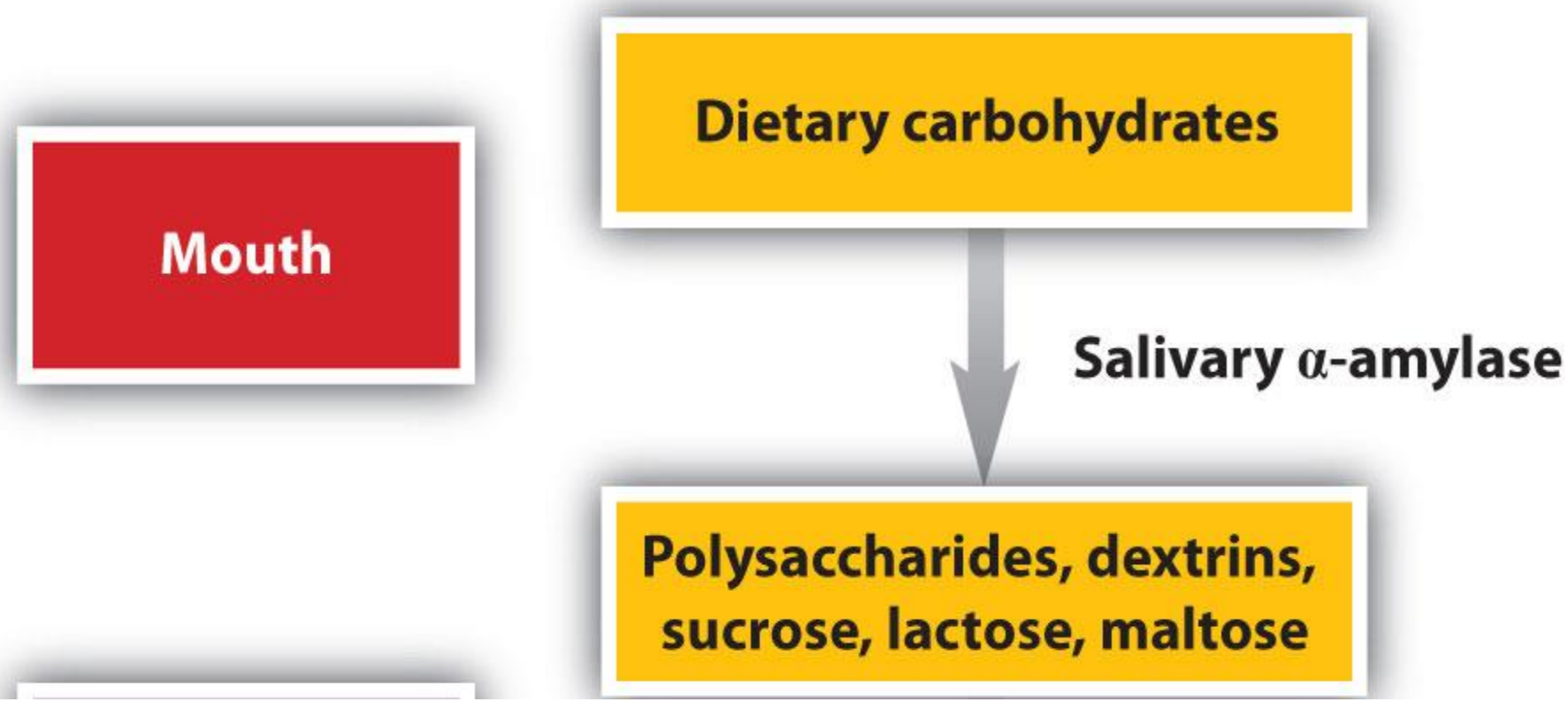
Digestion of Carbohydrate starts in the mouth, upon contact with saliva during mastication.

Saliva contains a carbohydrate splitting enzyme called *salivary amylase* , also known as *ptyalin*. Product of digestion are maltose, maltotriose & isomaltose.



ACTION OF PTYALIN(SALIVARY AMYLASE)

- Location: mouth
- Ptyalin is α-amylase and requires Cl^- ion for activation with an optimum pH of 6.7 (Range 6.6 to 6.8).
- The enzyme hydrolyses α-1→ 4 glycosidic linkages at random deep inside polysaccharide molecules of starch & glycogen. It has no action on β1-4 bond present in cellulose.
- However, ptyalin action stops in the stomach when the pH falls to 3.0.



- **Shorter duration of food in mouth.**
- **Thus it is incomplete digestion of starch or glycogen in the mouth.**

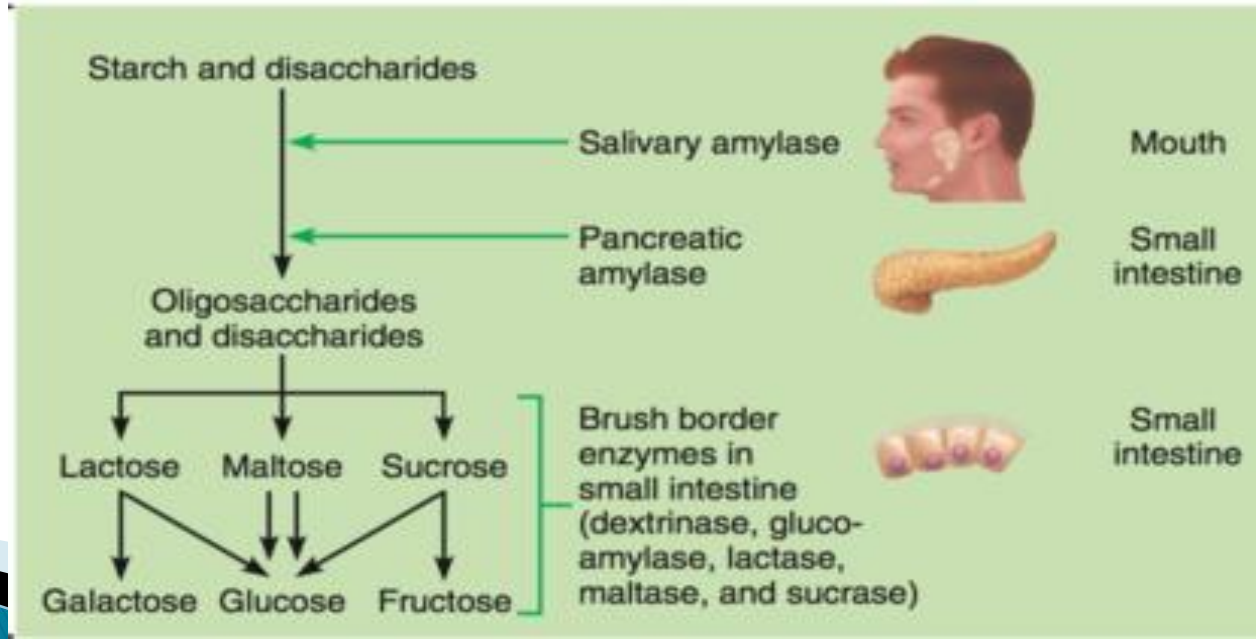
Digestion in the Stomach

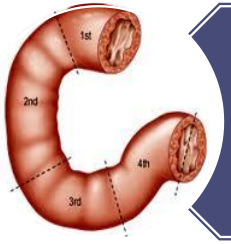
- There is no enzyme to break the glycosidic bonds in gastric juice.
- However, HCl present in the stomach causes hydrolysis of sucrose to fructose and glucose.

- Sucrose $\xrightarrow{HCl}$ Fructose + Glucose

Digestion in Small Intestine

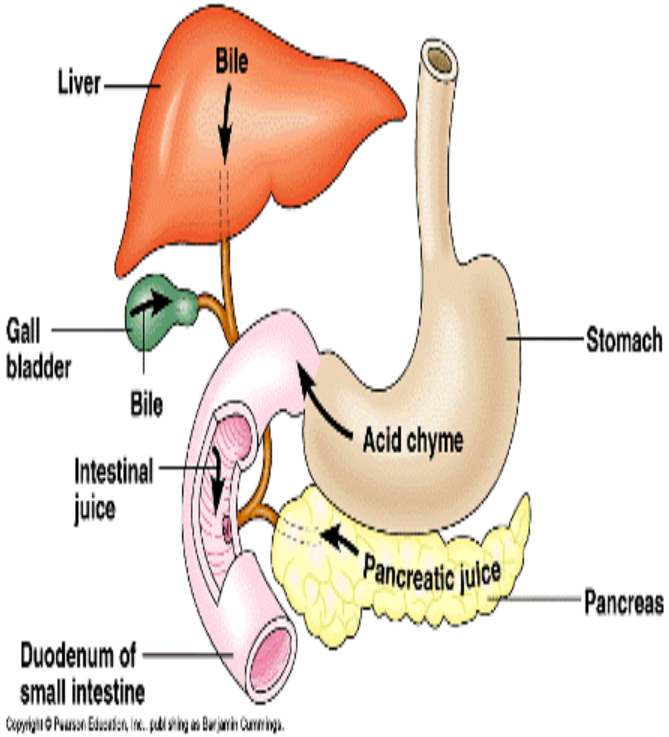
- **Main digestion takes place in the small intestine by pancreatic amylase**
- **Digestion is completed by pancreatic amylase because food stays for a longer time in the intestine.**





Digestion in Duodenum

- Food bolus reaches the duodenum from the stomach,it stimulates mucosal cells of duodenum to release secretein & cholecystokinin(local peptide hormones).
- They stimulate the pancreas to release pancreatic juice into the intestine.



Digestion in Small Intestine

- Secretein : neutralizes acidic chyme by stimulating release of bicarbonate.
- Cholycystokinin : stimulates release of digestive enzymes pancreatic α-amylase.

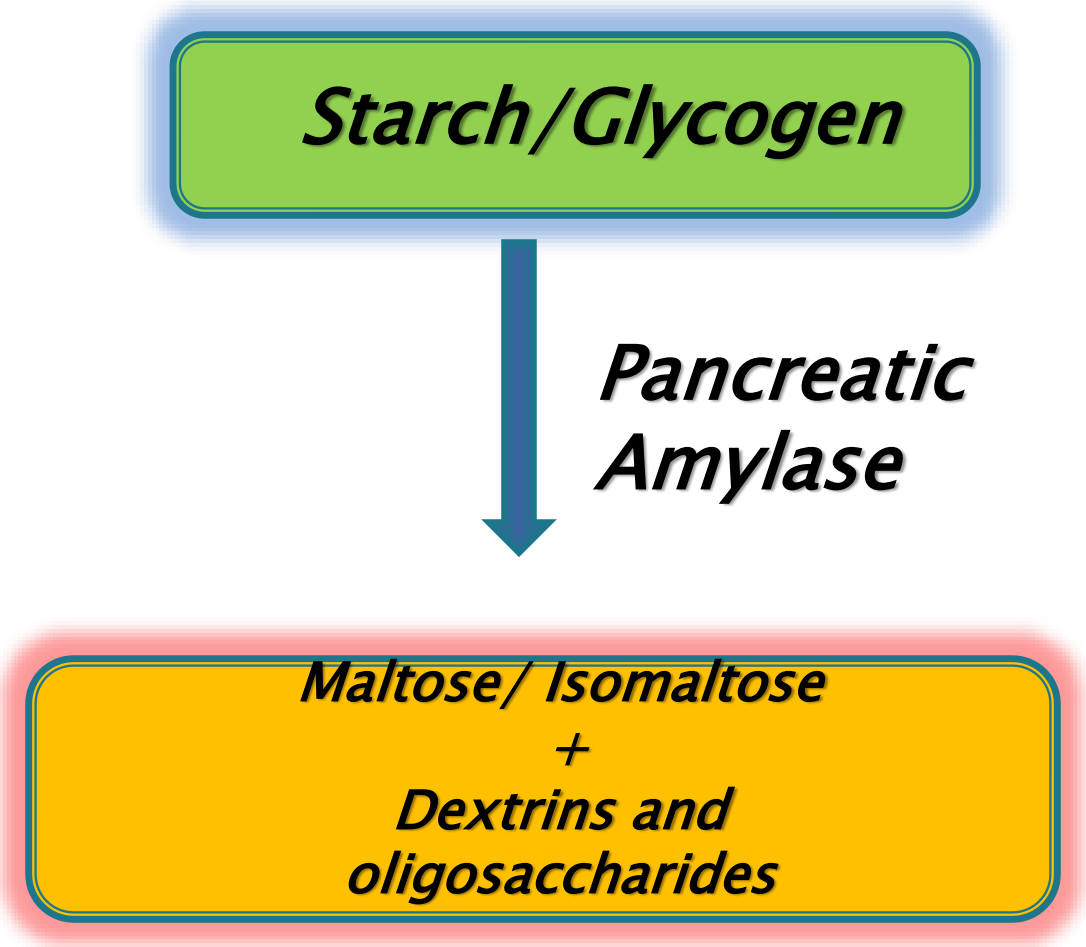
There are two phases of intestinal digestion.

1. Digestion due to pancreatic amylase
2. Digestion due to intestinal brush border enzymes.

Digestion due to pancreatic amylase

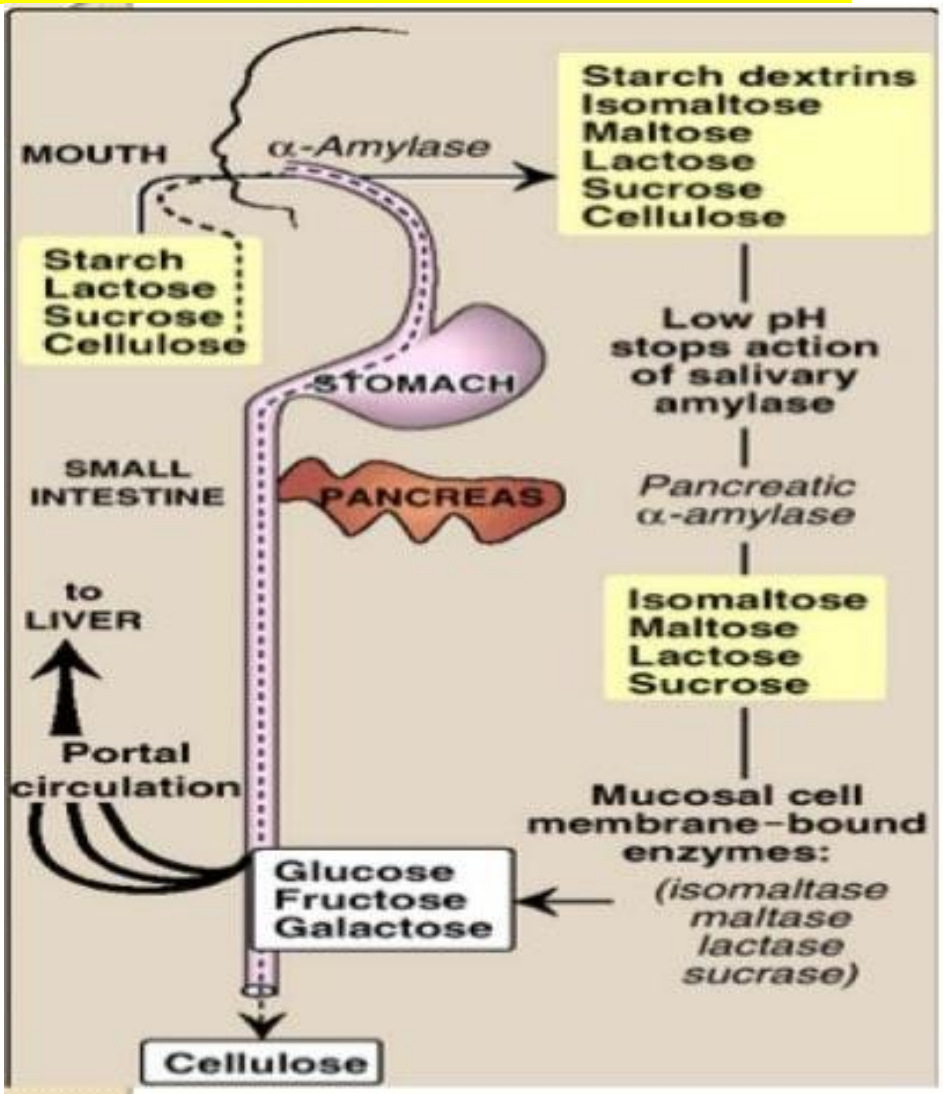
- It is an *α- Amylase*
- *Optimum pH=7.1*
- *Like ptylin, it requires* Cl^- ion for its activity.
- It hydrolyses α-1→ 4 glycosidic linkages situated well inside polysaccharide molecules.
- Note: *Pancreatic amylase, an isoenzyme of salivary amylase, differs only in the optimum pH of action. Both the enzymes require Chloride ions for their actions (Ion activated enzymes).*

Reaction catalyzed by pancreatic amylase



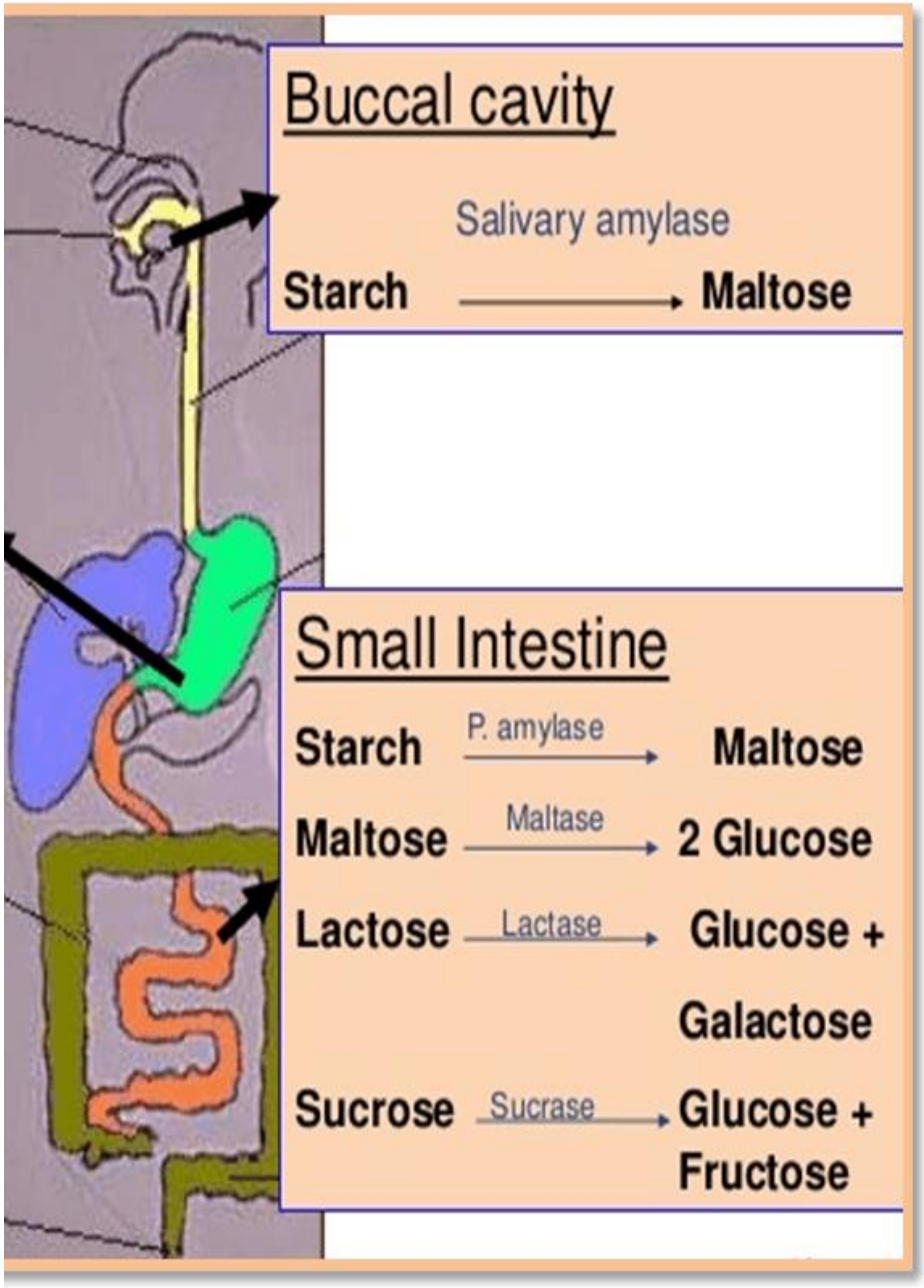
Digestion duo to intestinal brush border enzymes

- Enzymes present in intestinal brush border are disaccharidases except dextrinase .
- Dextrinase hydrolyze α(1-6) bonds at the branch point of limit dextrin.
- Intestinal amylase hydrolyze terminal α-1-4 linkages in polysaccharides.



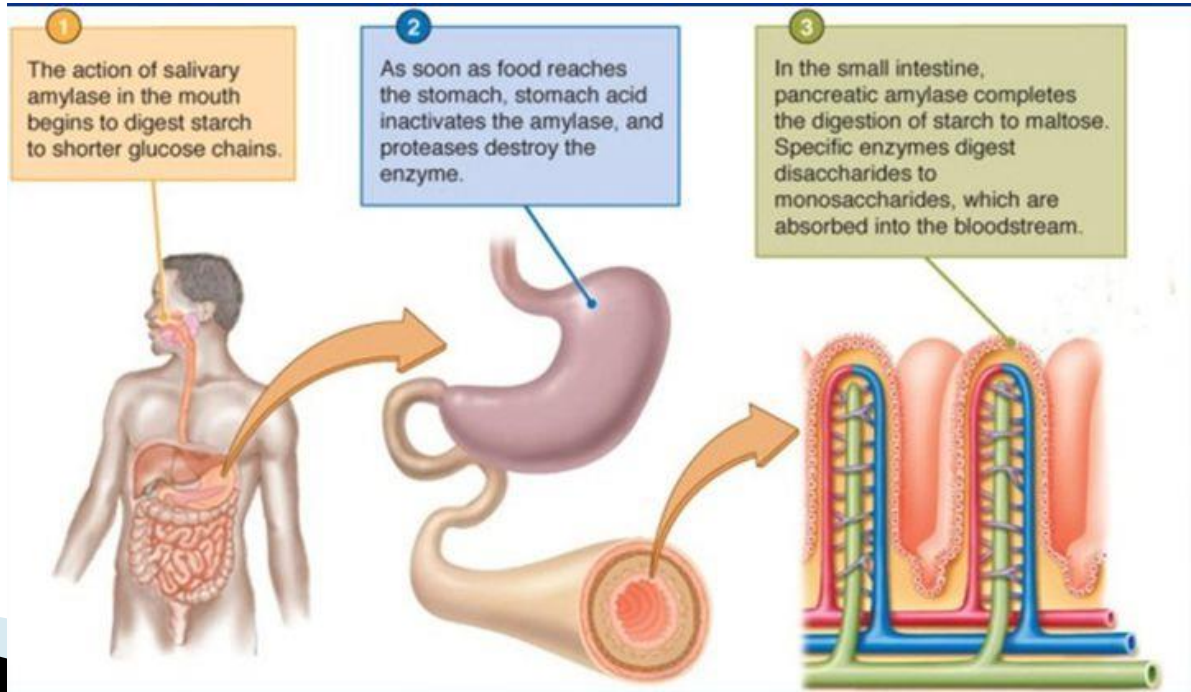
The different disaccharidases present in the intestinal epithelial brush border are :

1. Maltase: five maltases are found in human intestinal epithelial cells. Maltase 5 can act as isomaltase.
2. Sucrase
3. Lactase

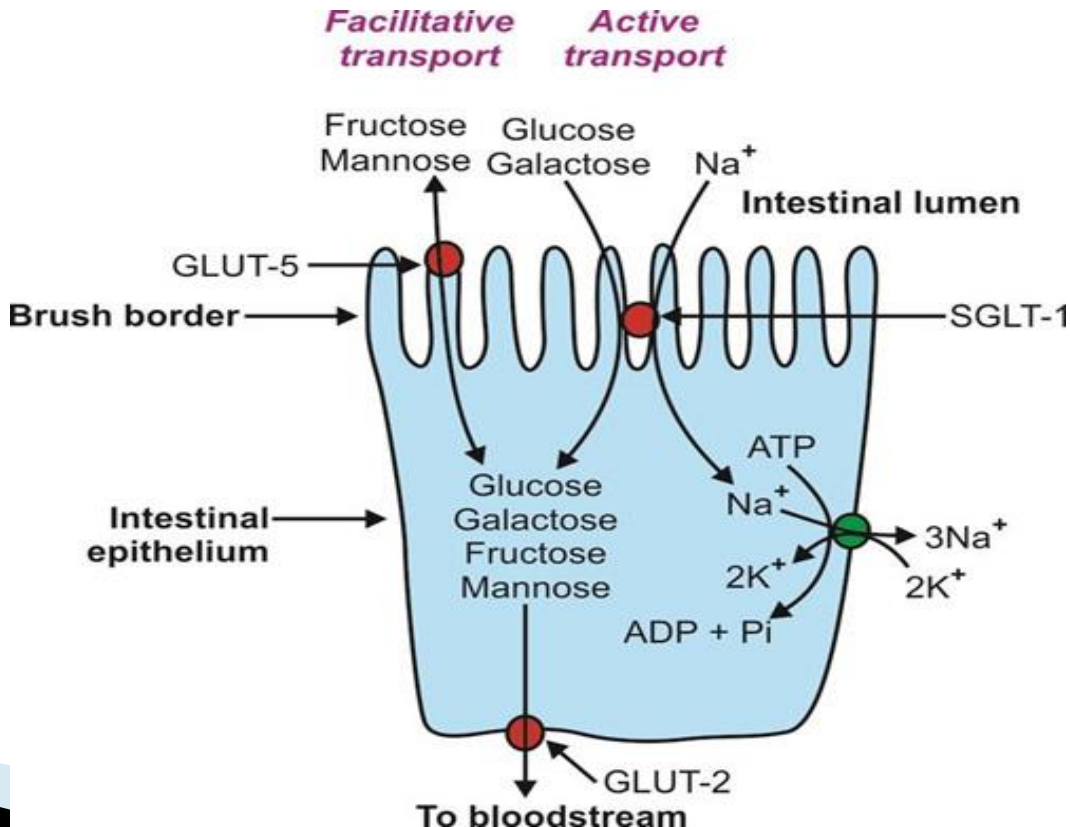


Absorption of carbohydrates

- Carbohydrates are absorbed as monosachharides from intestinal lumen through epithelial cells into the circulation.
- Absorption mostly takes place in duodenum & upper jejunum.

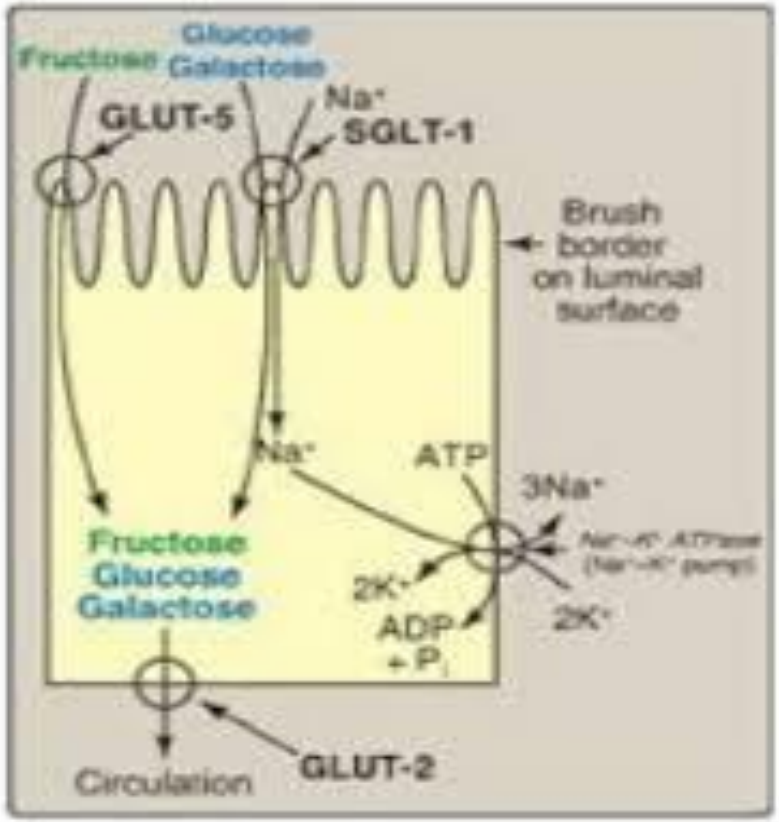


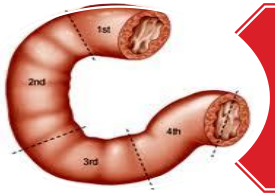
- There are two mechanisms for absorption of monosaccharides.
- 1. Active transport against concentration gradient.
- 2. Facilitative transport(with concentration gradient.



Transport of carbohydrates

- Transport of glucose & galactose across the brush border membrane of mucosal cells occur by active transport, energy requiring process that requires specific transport protein & the presence of Na^+ ion.





Glucose transporters

Glucose transporters

2 types

Na^+ dependent transporter

Also called

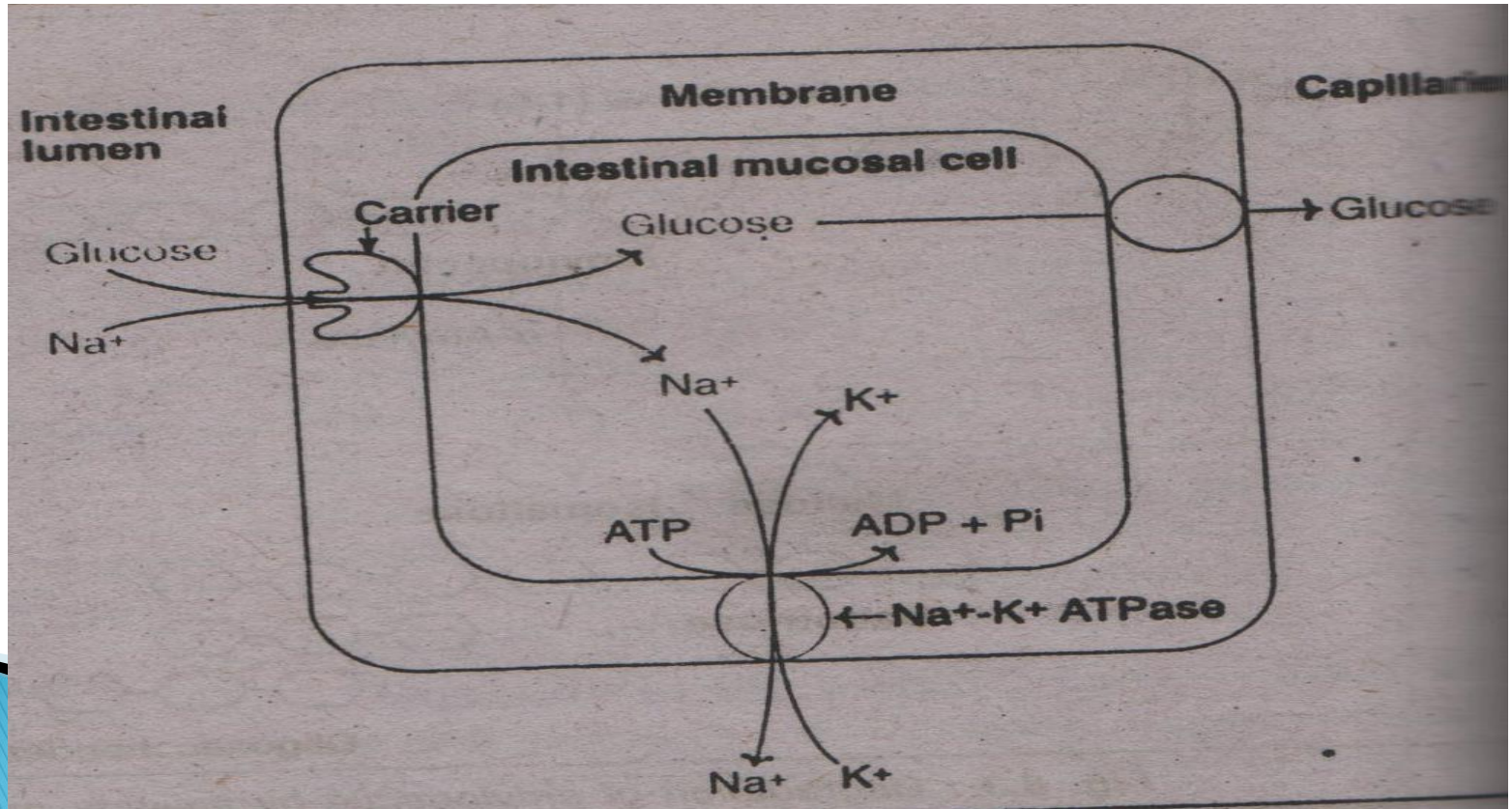
SGLT

Na^+ independent transporter

Also called

GLUT

- A sodium dependent glucose transporter(SGLT-1) binds both glucose & Na^+ at separate sites and transport them.
- Glucose is transported against its conc gradient while Na^+ is transported along its conc gradient.
- Energy is obtained from Na^+-K^+ ATPase involved in the transport of Na^+ in exchange with K^+ against conc gradient.

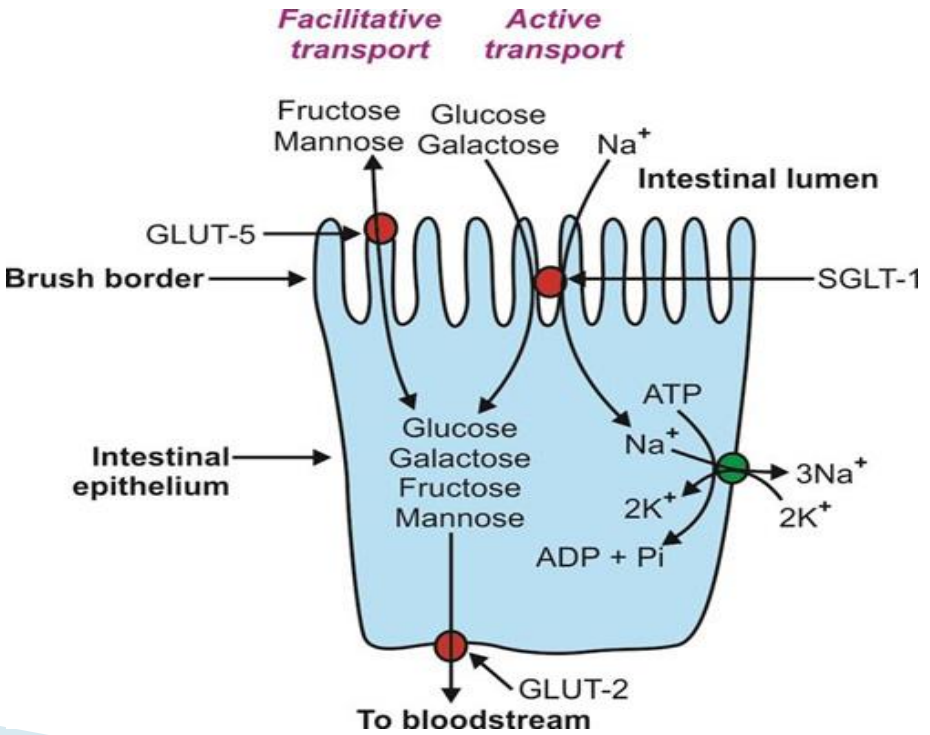


Oral rehydration therapy(ORT): ORT is the most common treatment of diarrhea. The oral rehydration fluid contain glucose and sodium. Intestinal absorption of Na^+ is facilitated by the presence of glucose.

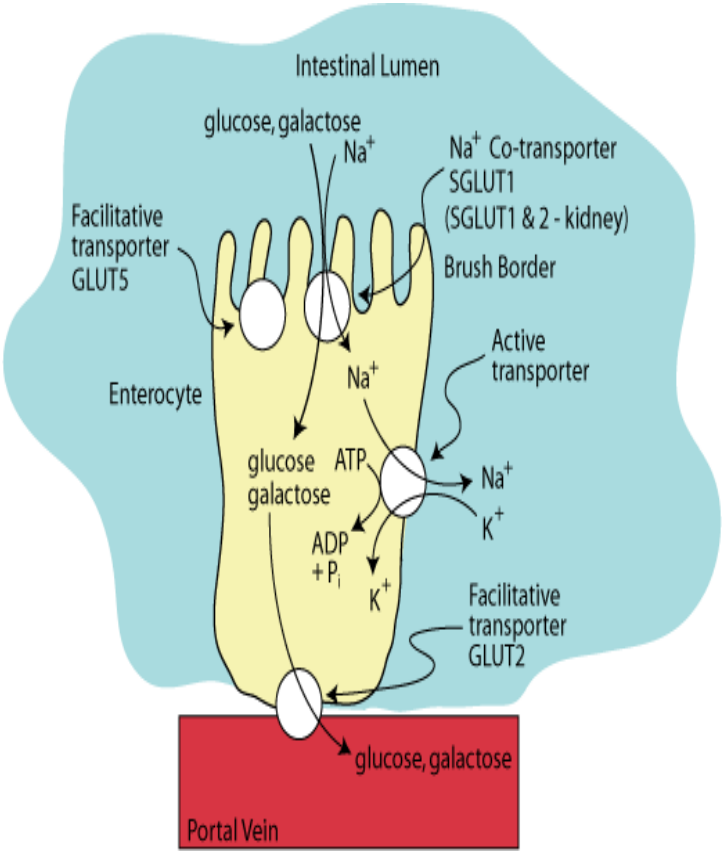
Clinical significance

- In deficiency of SGLT- 1, glucose is left unabsorbed and is excreted in feces. *Galactose is also malabsorbed.*
- In deficiency of SGLT- 2, the filtered glucose is not reabsorbed back, it is lost in urine, causing *glycosuria*.

- Fructose & mannose are transported across the brush border by a Na independent facilitative diffusion, involving another glucose transporter GLUT-5.
- Glucose and galactose can also use same transporter if conc gradient is favorable.



- Sodium independent transporter(GLUT-2) facilitates transport of sugars out of mucosal cells into the portal circulation to the liver.



Factors affecting rate of absorption

- State of mucous membrane & time of contact:
- Hormones:

Thyroid, adrenal cortex hormones, anterior pituitary, insulin.

- Vitamins : thiamin, pyridoxine and pantothenic acid.
- Deficiency of carrier proteins.
- Drugs:
- Active transport of glucose is inhibited by cardiac glycoside oubain (Inhibitor of Na pump).
- Phlorizin is also inhibitor.
- Phloretin is inhibitor of GLUT-1& GLUT4.
- Dinitrophenol barbiturate

Uptake of glucose in peripheral cells

- Mechanism: facilitated diffusion.
- There are 7 important glucose transporter for uptake of glucose into special cells.
- They have been numbered from 1 to 7 (GLUT 1 to GLUT 7).
- They are biologically important.

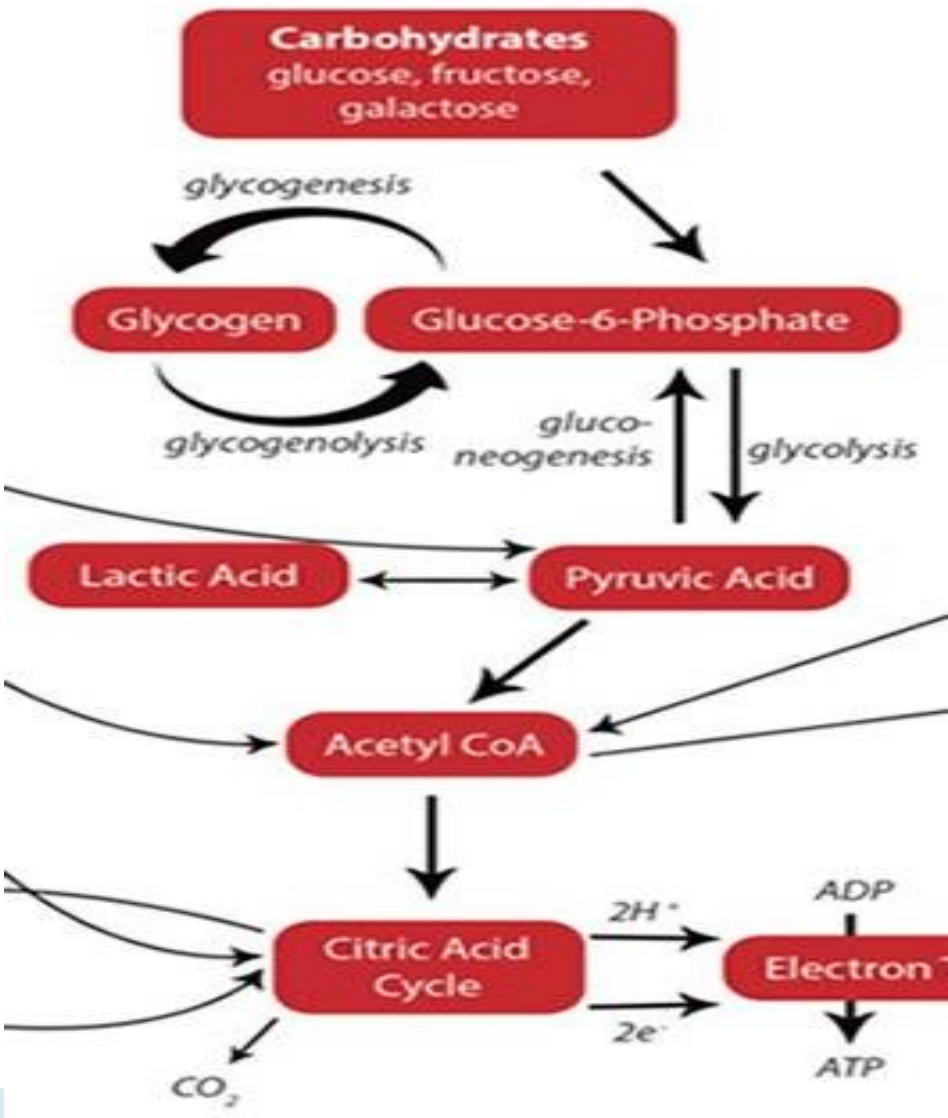
Tissue specific glucose transporter	Tissue distribution	Functions	Clinical significance
GLUT-1 (great affinity for glucose)	Present in almost all cells with an abundance in RBC.	Na-independent	Cancer cells express high level of GLUT-1, so they can internalize more of glucose, which is used as a source of energy for rapidly dividing cells.
GLUT-2 (low affinity for glucose, it can transport only when there is glucose load in the body)	Present in intestine, liver and pancreas.	Releases insulin by movement of glucose into β-cells of pancreas. (Acts as a sensor for the release of insulin by pancreas.) Promotes uptake of glucose in liver cells, lowering down blood glucose.	Diabetes Mellitus.

GLUT 3	Brain cells, all other cells of body		Cancer cells express high level of GLUT-3, so they can internalize more of glucose, which is used as a source of energy for rapidly dividing cells
GLUT 4	Adipose tissue, skeletal muscles, cardiac muscles	The only transporters which are under the influence of insulin. Insulin promotes uptake of glucose in the tissues by mobilizing the transporters to the cell surface whenever there is high glucose concentration in the blood	

GLUT 5 (least affinity for glucose)	Intestine at the luminal surface, testicles, seminal vesicles	Mainly for the transport of fructose	
GLUT 6		Non-functional transporter product of a pseudo gene. No role in absorption of glucose in peripheral cells.	
GLUT 7	Surface of endoplasmic reticulum	Transportation of glucose across the membrane of endoplasmic reticulum.	
SGLT 1	Kidney, intestine	For the absorption of glucose.	In cases where SGLT 1 is deficient, glucose is left unabsorbed and is excreted in faeces.
SGLT 2	Kidney	For the re-absorption of glucose	If deficient, filtered glucose is not re-absorbed and is

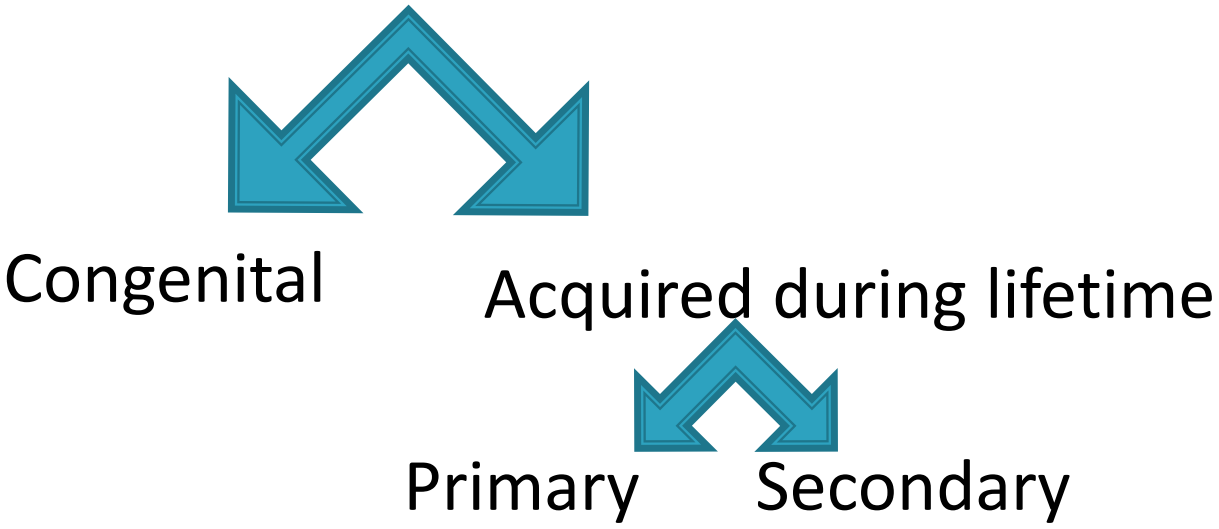
Metabolic fate of carbohydrate

- Monosaccharides are converted to Glucse-6-phosphate and enter to different metabolic pathways.



Clinical significance of Digestion

- Lactose intolerance is the inability to digest lactose due to the deficiency of Lactase enzyme.
- **Causes**



Congenital Lactose intolerance

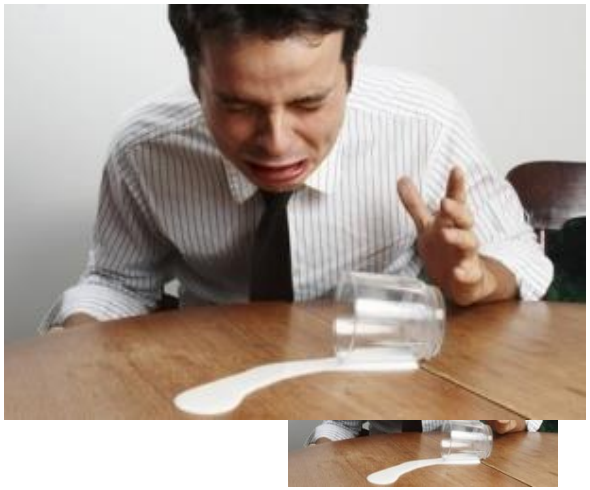
- It is a congenital disorder
- There is complete absence or deficiency of lactase enzyme.
- The child develops intolerance to lactose immediately after birth.
- It is diagnosed in early infancy.
- Milk feed precipitates symptoms.

Baby with Lactose Intolerance

Primary Lactase deficiency

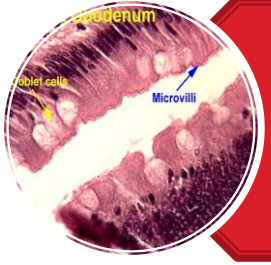
- Primary lactase deficiency develops over time
- There is no congenital absence of lactase but the deficiency is precipitated during adulthood.
- The gene for lactose is normally expressed upto RNA level but it is not translated to form enzyme.
- It is very common in Asian population.
- There is intolerance to milk + dairy products.

Adult with lactose intolerance



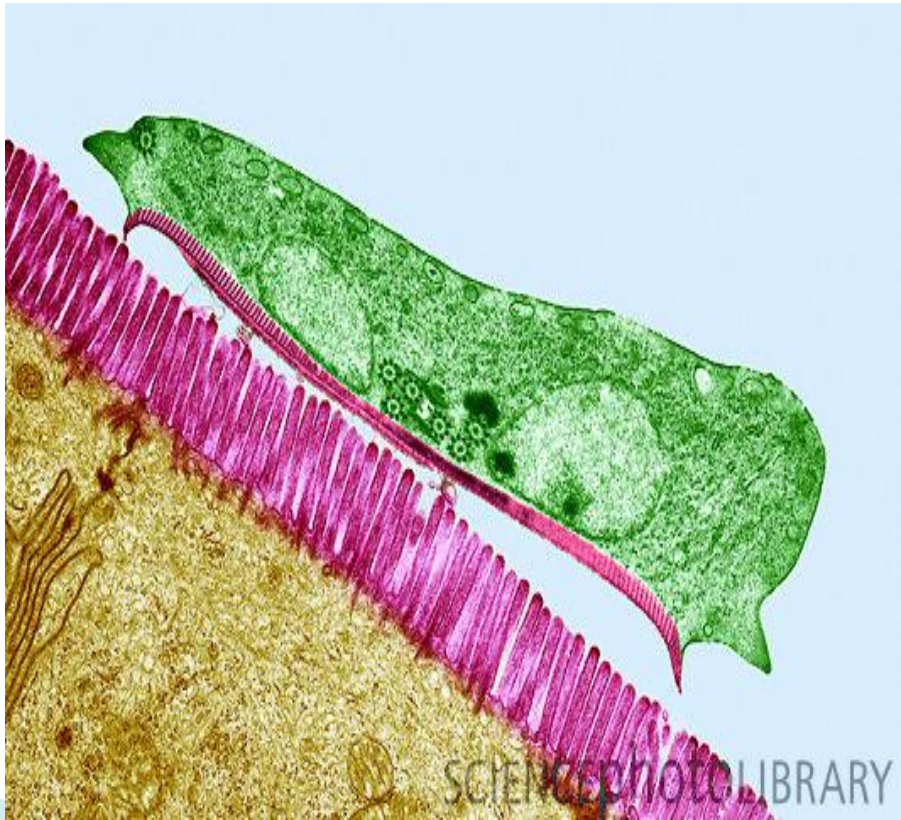
Secondary lactase deficiency

- It may develop in a person with a healthy small intestine during episodes of acute illness.
- This occurs because of mucosal damage or from medications or resulting from certain **gastrointestinal diseases**, **celiac disease, Crohn's disease, or chemotherapy**.
- This type of lactase deficiency can occur at any age but is more common in infancy.
- Another form of temporary lactose intolerance is lactose overload in infants.



Secondary lactase deficiency

- Exposure to **intestinal parasites** such as Giardia lamblia.
- In such cases the production of lactase may be permanently disrupted.



Rotavirus

- A very common cause of **temporary** lactose intolerance is gastroenteritis, particularly when the gastroenteritis is caused by **rotavirus.**

Clinical manifestations

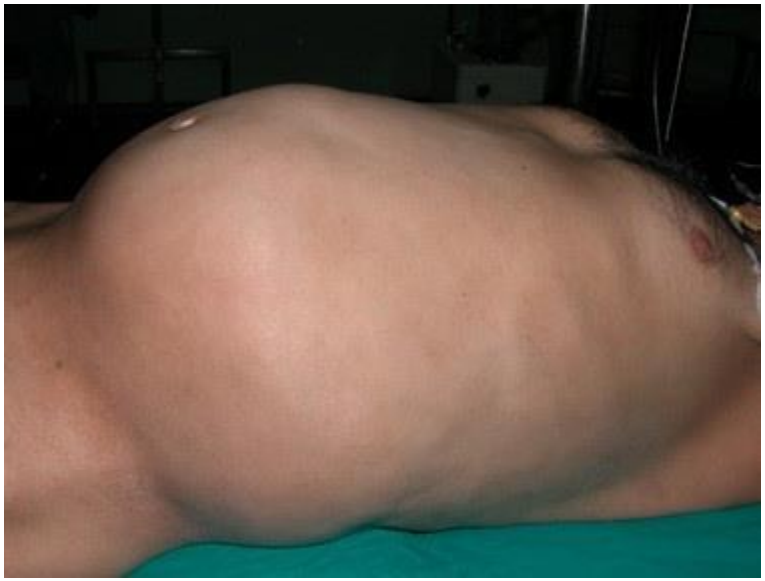
- In the form of abdominal cramps, distensions, diarrhea, constipation, flatulence upon ingestion of milk or dairy products.

Biochemical basis

- Undigested lactose in intestinal lumen is acted upon by bacteria and is converted to CO_2 , H_2 .producing flatulence, distension and abdominal cramps.

- Lactose is osmotic ally active .

- They withdraw H_2O from intestinal mucosal cell and cause osmotic diarrhea or constipation because of undigested milk.



Abdominal distension & Flatulence

Diagnosis

- *Two tests are commonly used:*
  -lactose detected in urine.
- **Hydrogen Breath Test**
- The person drinks a lactose-loaded beverage and then the breath is analyzed at regular intervals to measure the amount of hydrogen. Normally, very little hydrogen is detectable in the breath, but undigested lactose produces high levels of hydrogen. The test takes about 2 to 3 hours.

MANAGEMENT OF LACTOSE INTOLERANCE:

- Avoidance of dairy products.
- Although the body's ability to produce lactase cannot be changed, the symptoms of lactose intolerance can be managed with dietary changes.
- Most people with lactose intolerance can tolerate some amount of lactose in their diet. Gradually introducing small amounts of milk or milk products may help some people adapt to them with fewer symptoms.
- Partly digested dairy products can also be given.

REFRERNCE BOOKS:

- Lippincotts
- Chatterjea
- Hashmi
- Harpers


THANK
YOU