Digestion and absorption of carbohydrates

Digestion of carbohydrates:

The principal sites of dietary carbohydrates digestion are the mouth and intestinal lumen. There is little monosaccharaides present in diets of mixed animal and plant origin. Therefore the enzymes needed for degradation of most dietary carbohydrates are primarily endoglycosidases that hydrolyze oligosaccharides and polysaccharides and disaccharides.

Hydrolysis of glycosidic bond is catalyzed by a family of glycosidases that degrade carbohydrates into their reducing sugar components.

A / Digestion of carbohydrates begins in the mouth:

The major dietary polysaccharides are of plant (starch) and animal (glycogen) origin. During mastication, salivary α-amylase acts briefly on dietary starch and glycogen in a random manner, hydrolyzing some α (1-4) bonds.

Note: There are both α (1-4) and β (1-4) endoglucosidases in nature, but humans do not produce and secrete the latter in the digestive juices. Therefore they are unable to digest cellulose.

Because branched amylopectin and glycogen contain α(1-6)bonds , which α-amylase cannot hydrolyze , the digest resulting from its action contains a mixture of short , branched oligosaccharides or dextrin's .

Carbohydrates digestion halts temporarily in the stomach, because the high acidity inactivates the salivary α-amylase.

B / Further digestion of carbohydrates by pancreatic enzymes occurs in the small intestine.

When the acidic stomach contents reach the small intestine, they are neutralized by bicarbonate secreted by the pancreas, and pancreatic α-amylase continues the process of starch digestion.

C / Final carbohydrates digestion by enzymes synthesized by the intestinal mucosal cells

The final digestive processes occur at the mucosal lining of the upper jejunum , declining as they proceed down the small intestine and include the action of several disaccharidases and oligosaccharidases . For example, isomaltase cleaves the α(1-6) bond in isomaltose and maltase cleaves maltose , both producing glucose , sucrase cleaves sucrose producing glucose and fructose , and lactase cleaves lactose producing galactose and glucose . These enzymes are secreted through, and remain associated with, the luminal side of the brush border membranes of the intestinal mucosal cells.

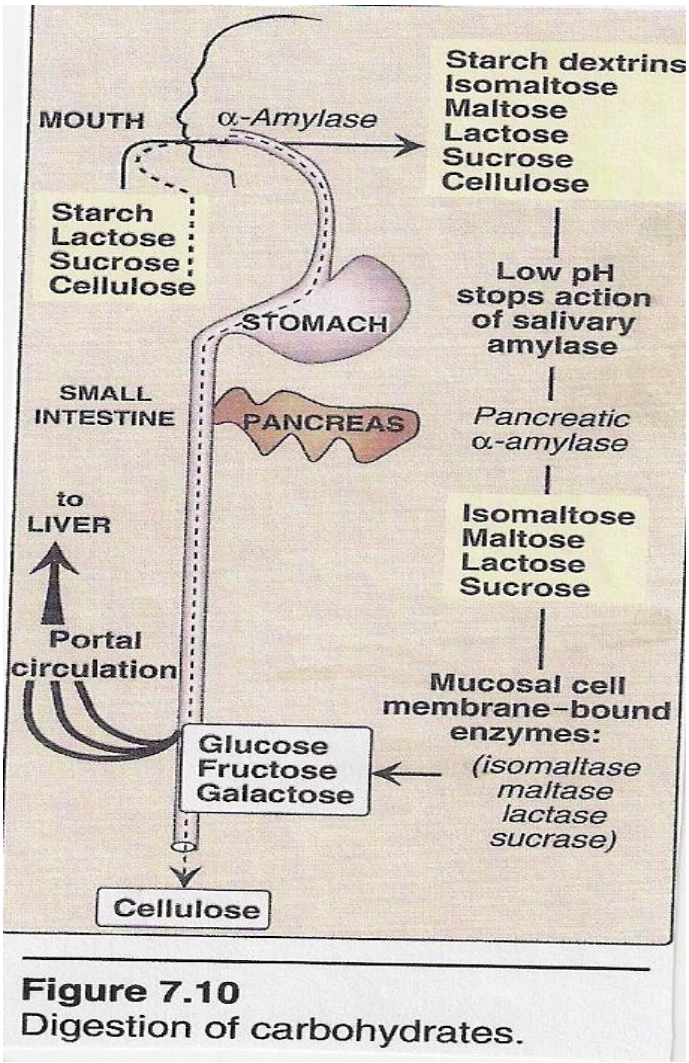

Figure 7.10
Digestion of carbohydrates.

D /Absorption of monosaccharaides by intestinal mucosal cells.

The duodenum and upper jejunum absorb the bulk of the dietary sugars. Insulin is not required for the uptake of glucose by intestinal cells. However, different sugars have different mechanisms of absorption. For example , galactose and glucose are transported from intestinal lumen to intestinal cells by an active , energy requiring process that requires a concurrent uptake of sodium ions, the transport protein is the sodium-dependent glucose cotransporter 1 (SGLT-1). Fructose uptake requires a sodium-independent monosaccharaide transporter (GLUT-5) for its absorption.

All three monosaccharaides are transported from intestinal mucosal cell into the portal circulation by yet another transporter, GLUT-2.

E / Abnormal degradation of disaccharides.

The overall process of carbohydrate digestion and absorption is so efficient in healthy individuals that ordinarily all digestible dietary carbohydrate is absorbed by the time the ingested material reaches the lower jejunum. However, because predominantly monosaccharaides are absorbed, any defect in a specific disaccharidase activity of the intestinal mucosa causes the passage of undigested carbohydrate into the large intestine. As a consequence of the presence of this osmotically active material, water is drawn from the mucosa into the large intestine, causing osmotic diarrhea. This is reinforced by the bacterial fermentation of the remaining carbohydrate to two and three carbon compounds (which also osmotically active) plus large volumes of CO2 and H2gas, causing abdominal cramps, diarrhea, and flatulence.

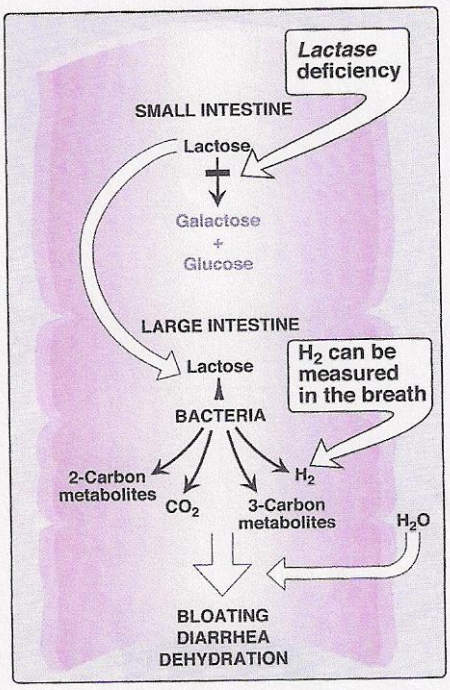

Figure 7.11
Abnormal lactose metabolism.

***Digestive enzyme deficiencies:** Hereditary deficiencies of the individual disacchharidases have been reported in infants and children. Alteration in disaccharide degradation can also be caused by a variety of intestinal diseases, malnutrition, or drugs that injure the mucosa of the small intestine. For example brush border enzymes are rapidly lost in normal individuals with severe diarrhea, causing a temporary acquired enzyme deficiency. Thus, a patients suffering from such a disorder cannot drink or eat significant amounts of dairy products or sucrose without exacerbating the diarrhea.

***Lactose intolerance:** More than three quarters of the world's adults are lactose intolerant. This is particularly manifested in certain races. For example, up to ninety percent of adults of African or Asian descent are lactase deficient and therefore, are less able to metabolize lactose than individuals of Northern European origin.

Treatment for this disorder is to reduce consumption of milk while eating yogurts and cheeses, as well as green vegetables such as broccoli, to ensure adequate calcium intake, to use lactase treated products; or to take lactase in pill form prior to eating.

***isomaltase-sucrase deficiency:** This disorder is found in about ten percent of Greenland's Eskimos, whereas two percent of North Americans are heterozygous for the deficiency. Treatment includes the withholding of dietary sucrose, and enzyme replacement therapy.

***Diagnosis:** Identification of a specific enzyme deficiency can be obtained by performing oral tolerance tests with the individual disaccharides. Measurement of hydrogen gas in the breath is reliable test for determining the amount of ingested carbohydrate not absorbed by the body, but which is metabolized instead by the intestinal flora.