Biochemical Aspects of Digestion of Lipids, Proteins, and Carbohydrates

part 2

الفريق الطبي الأكاديمي

Done By :- AHMAD ALSAHELE
Slide 8

Carbohydrate digestion

Dietary carbohydrates:

- **Polysaccharides:**
  1. Containing α(1,4) / α(1,6) bonds: Starch from plants & Glycogen from animals
  2. Contains β(1,4) bonds: Cellulose *from plants

  *It can’t be digested in humans due to the absence of the enzyme that can cleave β(1,4) bonds.*

- **Oligosaccharides**
- **Disaccharides**
  1. Sucrose
  2. Lactose
  3. Maltose
- **Monosaccharides:** Little amounts

Have few sugar molecules (glucose for example) linked together and it may have branches.

The doctor says that the number is between 6-15 but actually it may be even 3.

If it contains only D-glucose it is considered **dextrin**

Have alpha1-4 or alpha1-6 linkages or both & have 2 sugar units after a1-6 & it is hydrolyzed by dextrinase with activity on both types of linkages (a-amylase & isomaltase have one activity only).

Limit dextrin have 1 sugar unit before a1-6, 2 sugar units after a1-6, the molecule have only one a1-6 bond.

*Note: After the search, it was found that what the doctor said was not precise, but what is between the comma and period is the most logical at this time, so the doctor's information must be confirmed.*

- Digestion result in mono and disaccharides & alpha-dextrins (oligosaccharides)
- Alpha1-4 ➔ in sequences / alpha1-6 ➔ at branches

- Digestion is rapid.
- Generally completed by the time the gastric contents reach the junction of the Duodenum & Jejunum.

- No digestion occurs in the stomach because the high acidity (low pH) inactivates the **Salivary α-amylase.**
- **Pancreatic α-amylase** continues the process of Starch & Glycogen digestion in Small Intestine.
Sites for digestion of dietary carbohydrates:

- The mouth
- The intestinal lumen

Slide 9

Enzymes for Digestion of Dietary Carbohydrates

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>α-amylase</th>
<th>Disaccharidas</th>
<th>Isomaltase &amp; α(1,6) glucosidase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Substrate</td>
<td>Polysaccharides</td>
<td>Disaccharides</td>
<td>Branch points of oligo- &amp; di- saccharides</td>
</tr>
<tr>
<td>Type</td>
<td>Both pancreatic &amp; salivary</td>
<td>intestinal</td>
<td>intestinal</td>
</tr>
</tbody>
</table>

They are considered isoenzymes which mean different forms of the same enzyme. They can be differentiated only by electrophoresis. Other examples (للإطلاع) creatine phosphokinase & alkaline phosphatase

1. **α-Amylases**

   Normal level in serum → 25-125 U/L The clinical significance of rising circulating levels of α-amylase activity is a diagnosis of **Acute Pancreatitis**

   - Start to rise → Few hours
   - Peak → 12-72 hours
   - Returns to Normal → Few days

   *Damage of pancreatic cells which leads to release & activation of intracellular enzymes into the blood.

2. **Intestinal enzymes** are secreted by & remain associated with the luminal side of the brush border membranes of the intestinal mucosal cells in mucosal lining of the Jejunum.
**Intestinal Di-saccharidases**  
They are responsible for the final digestion of lipids

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Isomaltase</th>
<th>Maltase</th>
<th>Sucrase</th>
<th>Lactase (β-galactosidase)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Substrate</td>
<td>Isomaltose</td>
<td>Maltose</td>
<td>Sucrose</td>
<td>Lactose</td>
</tr>
<tr>
<td>Product</td>
<td>2 Glucose</td>
<td>2 Glucose</td>
<td>Glucose &amp; Fructose</td>
<td>Glucose &amp; Galactose</td>
</tr>
</tbody>
</table>

From 3 sources:
- Mono- & di- & poly-saccharides

**Absorption of Monosaccharides**

Absorption of Monosaccharides by Intestinal Mucosal Cells occurs in the Duodenum & upper Jejunum.

Different Monosaccharides have different mechanisms of absorption:
1. Facilitated diffusion) GLUT-mediated)
2. Active transport) Energy-dependent): Co-transport with Na+

Note: Insulin is not required for the uptake of glucose by intestinal cells

GLUT-5 & Na+ dependent active transporters present at apical surface of enterocytes (intestinal epithelial cells) while GLUT-2 at basal surface
Hormonal control of digestion in small intestine

- The digestion in small intestine is hormonally controlled.
- Two small peptide hormones are released from cells of the upper part of small intestine:

<table>
<thead>
<tr>
<th>The Gut Hormone</th>
<th>Cholecystokinin (CCK)</th>
<th>Secretin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stimulus of secretion</td>
<td>The presence of partially digested lipids &amp; proteins in the <strong>Upper Small Intestine</strong>.</td>
<td>Low pH (high acidity) of the Chyme entering the Intestine.</td>
</tr>
</tbody>
</table>
| Effects | • Stimulates the release of Pancreatic Enzymes.  
• Stimulates the contraction of Gall Bladder & release of bile  
• ↓ Gastric motility ➞ slower release of gastric contents into Small Intestine so more time to digestion. | Stimulates the Pancreas to release a watery solution high in HCO3 to neutralize the pH of the intestinal contents. (to reach the optimum pH for digestive activity by pancreatic enzymes) |

Especially:
- zymogens
- pancreatic α-amylase
- pancreatic lipases

That means there is no denaturation due to high acidity.

Abnormalities in Digestion/ Absorption

**Abnormalities in Lipid & protein Digestion/ Absorption**

- Liver and Gall Bladder diseases
- Intestinal diseases) Intestinal resection- shortened bowel motion)
- Pancreatic insufficiency) Chronic pancreatitis • CF • surgical removal of the pancreas): There is incomplete digestion & absorption of fat & protein ➞
  - Steatorrhea” & undigested proteins in the feces.
*The excretion of fat with the feces because of reduced absorption of fat by intestine

**Abnormal digestion of disaccharides:** Lactose intolerance (Lactase deficiency)

- Lactase (β-galactosidase) deficiency leads to undigested carbohydrate in large intestine → **Osmotic Diarrhea**.
- Bacterial fermentation of the undigested compounds lead to accumulation of CO2 & H2 gases → **Abdominal cramps**, diarrhea, and distention (flatulence).

The most common problem in digestion is lactose intolerance which could be inherited (problem in genes) or congenital (injury during gestation) or acquired (injury in the intestine).

Lactose intolerance → problem in lactose digestion → no lactose digestion → no absorption → accumulation of lactose → lactose metabolized by bacteria of the normal flora → produce organic acids and gases → they make you feel cramped and pained.

Because lactase is immature in lactose intolerant infants after first 6 months so the infant will do screaming because of the pain that produced by the above mechanism and we will notice there is swelling (because of gases) and diarrhea (because of osmosis).

The problem is more prevalent when taken with milk or any other lactose rich food.

How the problem can be diagnosed? By **Hydrogen Breath Test** (because bacteria produce gases (H2) & organic acids (H+) we can detect the problem by the rise of hydrogen concentration in the body above normal value).

**Fibrosis Cystic**

- **Autosomal recessive disorder due to mutation of CFTR** gene.
- CFTR protein is a Cl channel on epithelium.
- **It affects the lungs mainly, and also pancreas, liver, and intestines.**
- Characterized by abnormal transport of Cl & Na across an epithelium, leading to thick, viscous secretions.
- Defects leads to decreased secretion of Cl and increased reabsorption of Na & H2O.
- In pancreas, decreased hydration results in, thickened secretions which can’t reach the intestine, causing Pancreatic insufficiency.
Abnormality in protein digestion: **Celiac Disease (Celiac sprue)**

- It is a disease of malabsorption resulting from **immune-mediated damage to the Small Intestine in response to ingestion of Gluten**.
- Gluten is a protein found in **wheat, rye & barley**.

**Summary**

**All the digestive enzymes in GI tract**

<table>
<thead>
<tr>
<th>Lipid</th>
<th>Protein</th>
<th>COH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouth</td>
<td>Lingual lipase</td>
<td>Salivary α-amylase</td>
</tr>
<tr>
<td>Stomach</td>
<td>• Gastric lipase</td>
<td>• Renin</td>
</tr>
<tr>
<td></td>
<td>• Pepsin</td>
<td>Infants</td>
</tr>
<tr>
<td></td>
<td></td>
<td>adults</td>
</tr>
<tr>
<td>Pancreas</td>
<td>• Lipase &amp;co-lipase</td>
<td>• Trypsin</td>
</tr>
<tr>
<td></td>
<td>• Cholesteryl esterase</td>
<td>• Elastase</td>
</tr>
<tr>
<td></td>
<td>• Phospholipase A₂</td>
<td>• Chymotrypsin</td>
</tr>
<tr>
<td></td>
<td>• Lysophospholipase</td>
<td>• Carboxypeptidase</td>
</tr>
<tr>
<td>Intestine</td>
<td>Short &amp; medium-chain fatty acids are absorbed directly in intestine to portal circulation. Long-chains are resynthesized and secreted into systemic circulation</td>
<td>• Pancreatic α-amylase</td>
</tr>
</tbody>
</table>

- This disease is considered as an allergy to gluten
- The treatment is a free gluten diet

**DON'T WATCH THE CLOCK; DO WHAT IT DOES. KEEP GOING. — SAM LEVENVSON**