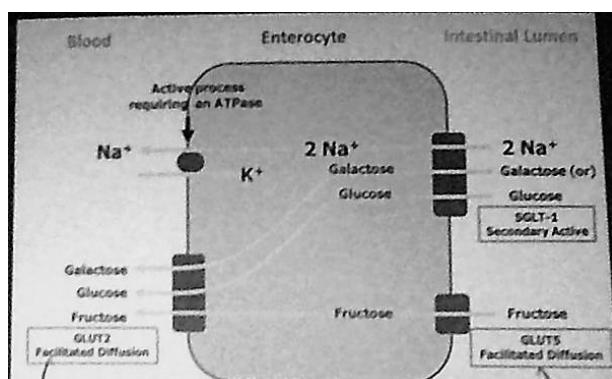




1. Sodium is pumped out of the enterocyte by the sodium potassium ATPase pump
2. Sodium conc is decreased so sodium diffuses into the cell from the lumen across the SGLT-1 with glucose or galactose
3. The glucose and galactose and fructose then diffuse out of the cell through the GLUT 2 carrier into the blood stream
4. Fructose diffuses into the cell by the GLUT 5 transporter

Once absorbed the monosaccharides can be sent to tissues where they are needed/converted into glycogen/stored as fat.

- Protein absorption

Protein digestion occurs in the stomach where it is denatured by the acid and digested by pepsin (endopeptidase) into peptides. In the small intestine pancreatic enzymes such as trypsin, chymotrypsin (exopeptidases) digest the peptides from one end into amino acids.

Once in the lumen of the small intestine the amino acids can diffuse by facilitated diffusion with sodium along the sodium concentration gradient, also normal FD and passive FD. Di and tripeptides are also able to enter the cell by FD and are digested within the enterocyte. There is a different transporter for each of the 20 amino acids.

- Absorption of ions and water

It is important that the osmolality of body compartments does not change greatly, as it can lead to bloating or dehydration. The gut plays an important role in ensuring that the water and electrolyte levels are kept in balance. We secrete 1-1.5 litres of water into the gut so we must have a good method of reabsorption, 95% of this occurs in the small intestine where water molecules can move by osmosis between enterocytes or through aquaporins. The Jejunum of the small intestine has larger surface area of villi and the enterocytes have looser tight junctions which allows faster H₂O transport.

Ions are very important to be absorbed as their absorption is interdependent with that of the nutrients. Sodium is highly important as it allows the absorption of many substances as well as creating the osmotic gradient for water absorption.

Sodium is absorbed into the gut by facilitated diffusion along a concentration gradient established by a sodium potassium atpase pump which moves 2 Na⁺ out of the enterocyte and into the blood which allows sodium to diffuse into the cell along the concentration gradient either through protein channels or through cotransport.

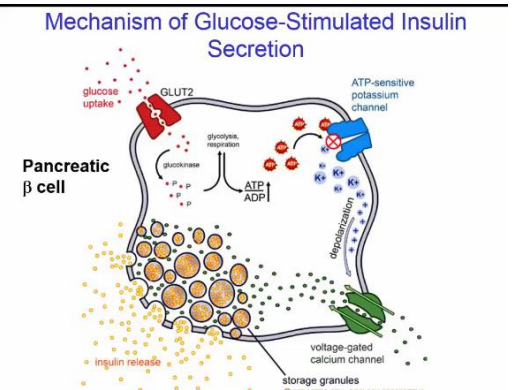
Water follows the sodium ion gradient and so moves by osmosis into the blood, this increases the solute concentration of the lumen so ions can diffuse into enterocytes. If there is not this ion gradient then water remains in the lumen leading to diarrhoea. The cholera bacterium causes diarrhoea by producing a toxin which attacks the ion channels of Na⁺ and Ca²⁺ causing the ions to move into the lumen and draw water with them.

Importance of insulin:

- Pre proinsulin to proinsulin then c chain released to release insulin
- Released constantly at basal level, very high when eating. Release tells the body we have eaten so it should use the glucose and store it

Glucose stimulated insulin secretion

1. **Glut 2** transporter on Beta cells raising extracellular glucose so glucose diffuses into the beta cell through the glut 2 transporter
2. Glucose is converted to glucose 6 phosphate in the cell and broken down to yield atp
3. the ATP can be used to close ATP sensitive potassium ion channels = depolarisation
4. Voltage dependent calcium ion channels open upon depolarisation = calcium influx the calcium causes the release of insulin from the vesicles into the blood.



Insulin – anabolic function

- Increases glycogen and fat and protein synthesis stops glucose production

Glucose uptake is allowed by insulin as it increases the number of glut4 receptors in cell membranes so more glucose can diffuse into cells. Without insulin no glut4 receptors appear on the cells so the people are hyperglycaemic as the glucose stays in the blood

Glucagon

- Single polypeptide
- Increases glycogenolysis
- Increases gluconeogenesis
- Increases fat breakdown in both liver and adipose tissue
- **Catabolic**

Hypoglycaemia releases glucagon, adrenaline, glucocorticoids, growth hormone all release glucose into the blood

Diabetes mellitus

- Type I – childhood onset and muscle wastage, can't utilise glucose so hyperglycaemia and has ketosis in the blood

It is an autoimmune destruction of the beta islet cells lead to absolute insulin deficiency, however it is idiopathic and has definite environmental factors, potentially infections or neonatal milk consumption that worsen a genetic predisposition. Liver does not receive insulin signal so releases glucose as it perceives that it is in a starved state.

Type II – become insulin resistant due to high circulating levels of insulin as a result of over consumption