

# Metabolism case study essay sample



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Enzymes act as a catalyst by speeding up or slowing down processes in the body. A protein enzyme has a particular shape that contains an active site. An active site is where a substrate fits into place to have its bonds either built or broken. At which time the products or product produced are released. The enzyme remains to continue to act on any available substrate.

### Hereditary Fructose Intolerance

AldolaseB is found mainly in the liver.

The second step in fructose metabolism is where aldolaseB breaks down fructose-1-phosphate into glyceraldehyde and dihydroxyacetone phosphate. If there is a deficiency in aldolaseB, it slows down the metabolism of fructose allowing for a buildup in the liver. A toxic build up of fructose in the liver can cause the death of liver cells. Hereditary Fructose Intolerance

If fructose-1-phosphate is not being broken down by aldolaseB its products (glyceraldehyde and dihydroxyacetone phosphate) are not available to perform glycolysis in which the end product is ATP. Also, the phosphate is bound to the Fructose-1-Phosphate and not available to the cells to produce ATP reducing the available ATP for the cells to use even more. Also fructose-1-phosphate signals for glucokinase to stay in the cytoplasm of the cell, this does not allow for the liver to release glucose to help stabilize low blood sugars. Fructose Metabolism

Fructose has a phosphate added to it by fructokinase into Fructose-1-Phosphate. Fructose-1-phosphate is the substrate for aldolaseB where it is converted to glyceraldehyde and dihydroxyacetone phosphate.

Glyceraldehyde and dihydroxyacetone phosphate are used in glycolysis or the production of glycogen. Lock and Key Model

### Activation Energy

What would happen if the Cori Cycle only happened within a single cell. The cycle would be unending repeating its self over and over, making it a futile cycle. Eventually using up the ATP available to the cell, because the process uses more ATP than it produces. Eventually, the cell would die from lack of ATP. Hypothetically if  $\alpha$ -ketoglutrare dehydronase (Oxoglutarate dehydronase complex) were defective The citric acid cycle would stop at the third stage.

Specifically affected would be  $\alpha$ -ketoglutrare, and it would not be processed to produce the Succinyl-CoA, CO<sub>2</sub>, or NADH. The citric acid cycle would not proceed any further, and no further products would be formed. This action would reduce the amount of NADH provided for the electron transport chain, as well as the one FADH<sub>2</sub>. Resulting in a greatly reduce the amount of ATP that would be produced by the electron transport chain. Coenzyme Q10 (coQ10)

On the inner membrane of the mitochondrion, you find a very high concentration of coQ10. CoQ10 is the part of the electron transport chain that carries the electrons through complex's I, II, and III. CoQ10 is essential as it is the only molecule that can carry the electron particles through the complexes. Once the electrons arrive in complex III they are transported to complex IV by cytochromeC. Where they are given to oxygen to produce H<sub>2</sub>O The energy generated by the electrons allows for Complex I, III, and IV

to pump the hydrogen across the inner membrane of the mitochondria. This causes a high concentration of  $H^+$  in the inner membrane space. The  $H^+$  wants to go to a low concentration, so it does so through the ATP synthase and the energy produced by this is what the ATP synthase uses to do oxidative phosphorylation and produce ATP from ADP.

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