Figure 8.11

Glucose is now in the cell and there is an energy investment phase where we are going to use 2ATP to put 2 phosphates on glucose. In the Energy generation phase, we will give those 2ATP back up and we are going to split the glucose into 3 carbon compounds and we ultimately we are going to get 2 pyruvates.
Pyruvate: 3 carbons alpha kidoacid.
At the end glucose a 6 carbon sugar becomes 2 pyruvates (2, 3 carbons compounds)
The net will be of two molecules of ATP. We use two but we will going to get back 4, so the net production is 2 ATP. We also produce some NADH which can be used to produce some more ATP.

Figure 8.12: Glucose 6-Phosphate
Glucose become phosphorylated by an enzyme called Herokinase and another called Glucokinase. They both catalyzed the same reaction that is the transfer of the gamma phosphate, the last one of ATP onto the C6 hydroxyl. Once that phosphate is put on glucose, its trapped and this means that its "irreversible phosphorylation of glucose".
Hexokinase: is the enzyme in all of our tissue, brain, nerve, tissue, etc.
Glucokinase: is an isoform of Hexokinase that is specific for the liver.

Figure 8.13 Difference between Hexokinase and Glucokinase
Glucokinase: so we just ate a big bowl of rice and there is a lot of glucose on that rice and what happens is that all that glucose comes through the liver which does a control check. The liver holds back a lot of the process of glucose after digestion and in doing so it has to process a lot of it. So Glucokinase has a HIGH Vmax, it can phosphorylate many glucoses per time. But it also has a HIGH Km value which means that it doesn't bind glucose very tighly and having this high Km means that has a lower affinity for glucose.
Hexokinase: in contrast they have a much lower Vmax but a very low Km and this is because there is not as much glucose to be process by these other tissues and having this low Km means that has a higher affinity for glucose, which means that is able to spot trace amounts of glucose in the cell.
Figure 8.14 Glucokinase Regulation
Glucokinase is indirectly inhibited by fructose 6-phosphate, which is in equilibrium with glucose 6-phosphate, a product of glucokinase and is indirectly stimulated by glucose (a substrate of glucokinase).
Glucose has a chaperone and that is GKRP which is Glucokinase regulatory protein in the liver which grabs it and has it inactive until glucose comes.

Figure 8.15 Fructose 6-Phosphate
Reaction number 2: glucose 6-phosphate is isomerized to fructose 6-phosphate by phosphoglucose isomerase.

Phosphoglucose isomerase is an aldo-keto-isomerase. There is an equilibrium between both substances.

Figure 8.16 Fructose 1,6-Bisphosphate

Fructose 6-phosphate is phosphorylated by an enzyme called phosphofructokinase-1 (PFK-1) so now we have fructose 1,6-Bisphosphate. This enzyme is inactive by itself and it need to be activated by Fructose 2,6-bis-phosphate.

Bisphosphate means two phosphates but in different positions. After we have the Bisphosphate now comes Aldolase which splits the 6 Carbon compound into two 3 carbon compounds and they are Dihydrozyacetone phosphate and Glyceraldehyde 3-phosphate.

Now we have the last enzyme here which is triose phosphate isomerase which catalyzes interconverting between DHAP and glyceraldehyde 3-phosphate. The DHAP must be isomerized to glyceraldehyde 3-phosphate for further metabolism by the glycolytic pathway.

Figure 8.17 PKK-1 Regulation

Fructose 6-phosphate must first be converted to fructose 1,6-bisphosphate. Insulin/glucagon are both secreted by pancreas and in response to feeding or starvation.

Starvation: your glucose level goes down and brains send signal that we need more because it doesn't want our muscles to burn all the fuel so it wants to stop this glycolytic process, to slow it down. So pancreas releases Glucagon which binds to GPCR activates adenylyl cyclase to make cAMP which activates protein kinase A which phosphorylates this PKF-2 and makes it inactive and without this you cannot make fructose 2,6-bisphosphate which is the one that activates PKF-1 to send fructose 1,6-bisphosphate down through glycolysis.

Feeding: just ate and have lots of glucose so pancreas send Insulin binds to Insulin receptor tyrosine kinase which activates the PI3K kinase which then activates protein Kinase B and this activates the glucose transporters to go to the membrane and what it also does is that activates this enzymes called phosphatases and they hydrolyzes all of the phosphates created by the Glucagon signaling. The insulin activated protein phosphatase will remove the phosphate so dephosphorylated on the PFK-2 and when this happens the PFK-2 will become active and which favors formation of fructose 2,6-bisphosphate and will activate PFK-1 which leads to an increased rate of glycolysis.

GLYCOLYSIS OCCURS IN THE CYTOPLASM

Figure 8.18 NADH and ATP formation