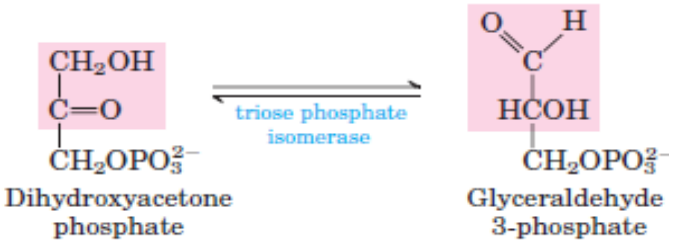
converted to an aldotiose; glceraldehyde-3-phosphate reversibly and rapidly by the enzyme "Triose phosphate isomerase"

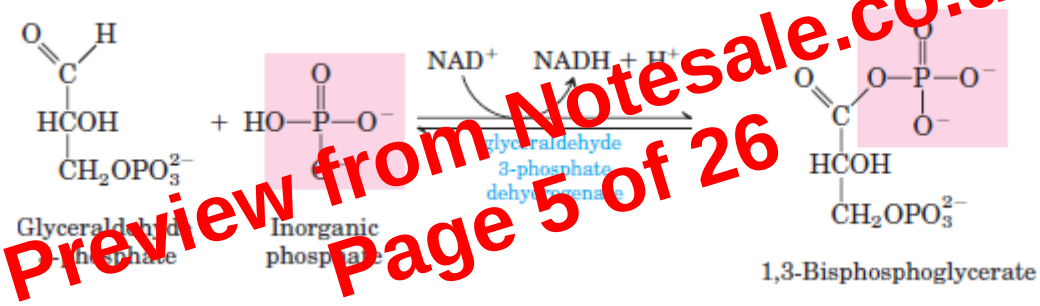


The Pay-off Phase of Glycolysis

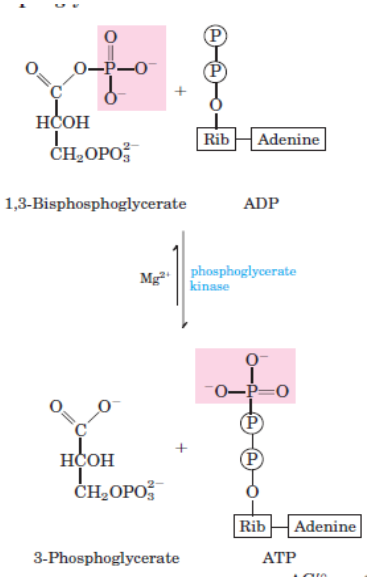
This are the energy conserving phosphorylation steps of glycolysis; in which energy is conserved in form of ATP & NADH

- **Step6: Oxidation of Glyceraldehyde 3-Phosphate to 1,3-Bisphosphoglycerate: The first step in the payoff phase is the oxidation of glyceraldehyde 3-phosphate to 1,3-bisphosphoglycerate, catalyzed by "glyceraldehyde 3-phosphate dehydrogenase". The aldehyde group of glyceraldehyde 3-phosphate is oxidized to a carboxylic acid anhydride with phosphoric acid. This type of anhydride is called "acyl phosphate".**

  The dehygrogenase enzyme is inhibited by iodoacetate, arsenate and heavy metal



- **Step7: Phosphoryl transfer from 1,3-bisphosphoglycerate to ADP: the phosporyl group on C1 of 1,3-bisphosphoglycerate is transferred reversibly to ADP, a substrate level phosphorylation reaction that converts ADP to ATP, and leaves 3-phosphoglycerate behind. The enzyme that catalyze this energy yielding rection is "Phosphoglycerate kinase"**



into the blood via GLUT2. Glucose uptake from the blood is carried out by a family of glucose transporters(GLUT). In RBC, GLUT1 is the glucose transporter, in hepatocytes; the glucose transorter is GLUT 1 & GLUT2, in brain neurons; GLUT3 is the glucose transporters. GLUT1,2 & 3 are always present in the plasma membrane.

In contrast, the glucose transporter in cells of skeletal muscle, cardiac muscle and adipose tissue is GLUT4 and is always found intracellularly secluded in vesicles and moves into plasma membrane only at the signal of the insulin hormone; secreted by the pancreatic β-cells.

Patients with insulin-dependent diabetes mellitus have too few β-cells (or autoimmune disorders) and insufficient insulin secretion, thus a drastically reduced glucose uptake by GLUT4 transporters of muscles. Muscles thus need to switch to stored lipids as its principal source of fuel, thus a significant increase in ketone production, excess of which can be life threatening in ketoacidosis condition.

In cancerous and milignant cells, glucose uptake and glycolysis is noted to increase remarkably due to hypoxia and overt genetic expression of glycolysis enzymes and certain growth factors

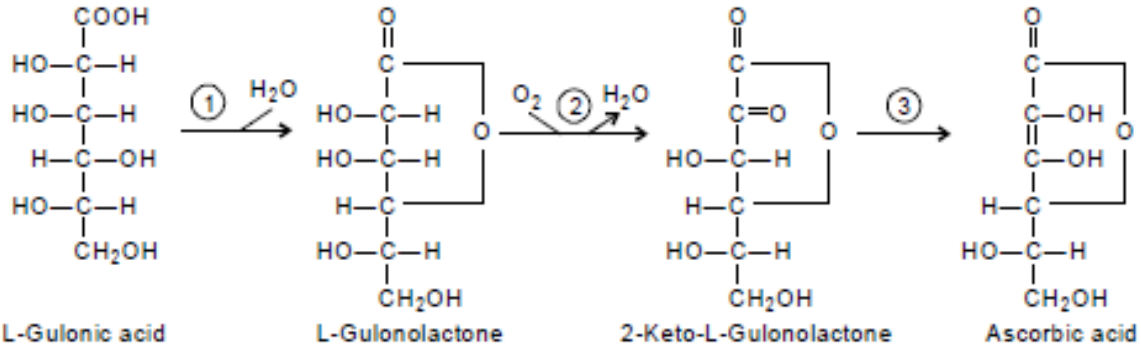
- **Other monosaccharides enter the glycolytic pathway at several points: a variety of D-hexose including fructose, galactose, mannose can be siphoned into glycolysis.**

  - **FRUCTOSE: catabolism of fructose in the muscle, kidney and small intestine begins with enzyme "Hexokinase" which phosphorylates it at C6 and yield fructose-6-phosphate which can proceed glycolysis**

  However, in the liver an isozyme of hexokinase; "Fructokinase" which phosphorylates at C1 instead converts fructose to fructose-1-phosphate. Then another hepatic enzyme "Aldolase-B" cleaves the product to dihydroxyacetone phosphate (which can proceed glycolysis) and glyceraldehyde which on action of nother enzyme "Triose kinase" is converted to glyceraldehyde-3-phosphate; which proceeds with glycolysis.

$$\text{Fructose} + \text{ATP} \xrightarrow{Mg^{2+}} \text{fructose 6-phosphate} + \text{ADP}$$

IN THE INTESTINE & MUSCLE



1 = Lactonase

2= Gulonolactone oxidase

GLYCOGEN METABOLISM IN ANIMALS.

Glycogen is the polymeric form in which animals stores excess glucose

It has earlier been explained that glycogen is catabolized by phosphorolysis by the glycogen-phosphorylase enzyme and the debranching enzyme [α(1→6) to α(1→4) glucan transferase]; which help at the branch points; hence we shall focus more on glycogen synthesis, regulation and inborn disorders of glycogen storage.

Glycogen synthesis takes place virtually in all tissues. How?

1. **The start of glycogen synthesis is formation of glucose-6-phosphate as in step 1 of glycolysis.**

Some glucose may not take the straight shunt to G6P, for example in erythrocyte, the glucose is first converted to lactate glycolytically, then the lactate is taken up by the liver and converted to G6P by gluconeogenesis

2. The glucose-6-phosphate is reversibly converted to to glucose-1-phosphate by the enzyme "phosphogluco-mutase". In the liver, glucose may be converted straight to glucose-1-phosphate by the hexokinase isoform "glucokinase".

$$\text{Glucose 6-phosphate} \rightleftharpoons \text{glucose 1-phosphate}$$

3. **Then the glucose-1-phosphate is converted to UDP-glucose by the action of "UDP-glucose pyrophosphorylase"**

$$\text{Glucose 1-phosphate} + \text{UTP} \longrightarrow \text{UDP-glucose} + PP_i$$

4. **UDP-glucose then becomes the immediate donor of glucose residue in the reaction catalysed by "glycogen synthase"; which catalyzes the transfer of glucose residue from UDP-glucose to the non-reducing end of a growing glycogen molecule chain, thus increasing the glycogen molecule by one glucose in an α(1→4) glycosidic linkage.**

It is imperative to know that glycogen synthase don't form branches, thus another enzyme "α(1→4) to α(1→6) transglycolase" otherwise called "glycosyl(4→6)transferase catalyzes the

Reference

This study guide is a simplified and compressive presentation of the topic compiled from Success biochemistry academy lecture note and some other trusted biochemistry authority and classical textbook. All pieced together in a simplified easily digestible form for all and sundry.

Classical textbooks whose phrases and picture reflect in this writeup are:

1. **Lehningher principles of biochemistry, 6th edition, David Nelson & Micheal Cox**
2. **Medical Biochemistry by N. Mallikarjuna Rao**
3. **Harper's illustrated biochemistry, 26th edition. Murray, Granner, Mayer, Rodwell**
4. **Textbook of medical biochemistry. MN Chatterjea, Rana shinde**
5. **Biochemistry: the chemical reactions of living cells. David Metzler**
6. **Fundamentals of biochemistry life at molecular level. Donald & Judith Voet, Charlotte Pratt**