Commentary

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A Report on Gluconeogenesis

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Although glucose is an important metabolite in human metabolism, it is not always present in sufficient amounts in the diet. As a result, there is a mechanism that transforms other nutrients into glucose. Gluconeogenesis is the name for this process.

At least half of the calories consumed by the brain must be in the form of glucose. Glucose is a precursor for the production of nucleotides, glycoproteins, and glycolipids, as well as other sugars. NADPH, which provides reducing power for biosynthesis and detoxification, requires glucose to be replenished.

The reversal of glycolysis, with many workarounds for the irreversible events in that pathway, is known as gluconeogenesis. The reactions that are common to both glycolysis and gluconeogenesis are represented in blue, while those that are unique to gluconeogenesis are highlighted in red. Because pyruvate and oxaloacetate are both beginning places for red arrows, any pathway that produces any of these, or indeed any other glycolysis step, might provide substrate carbon for gluconeogenesis. Green arrows represent these paths in this diagram.

Protein, both dietary and endogenous, is the primary source of substrate for gluconeogenesis. Protein is broken down into its individual amino acids first. Glucogenic amino acids are those that can be metabolised to pyruvate or any of the TCA cycle intermediates and hence serves as substrates for gluconeogenesis.

Acetyl-CoA or acetoacetate are formed from leucine, lysine, and aromatic amino acids. These amino acids are considered ketogenic because acetoacetate is a ketone body and acetyl-CoA can be transformed to ketone bodies. While it was once believed that ketogenic amino acids could not be converted to glucose in human metabolism, this is no longer the case, as the ketone body acetone can be converted to pyruvate. Nonetheless, ketogenic amino acids are expected to play a modest role in glucose regeneration.

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We already know that most processes are shared with glycolysis, so we only need to investigate the limited number of events that are unique to gluconeogenesis. The conversion of the phosphate group from phosphoenolpyruvate (PEP) to ATP is the final step in glycolysis. Because the following rearrangement of pyruvate from the enol to the keto form is very exergonic, this reaction is irreversible. To convert pyruvate back to PEP in gluconeogenesis, there are two enzymatic steps: (1) carboxylation of pyruvate to pyruvate carboxylase converts oxaloacetate to PEP, and phosphoenolpyruvate carboxykinase converts oxaloacetate to PEP.

We can fix CO_2 biologically, just like plants, using the pyruvate carboxylase reaction. However, before we try to claim Kyoto treaty credit for this skill, we must keep in mind that the same CO_2 molecule is released again in the next phase. The entire point of transitory CO_2 fixation is to allow for this eventual reaction, as is depicted in slide.

The pyruvate carboxylase reaction is split into two stages, which are carried out in two different active sites of a single enzyme molecule in human metabolism. Both activities are located on distinct enzyme molecules in E. coli. Biotin carboxylase is the first enzyme activity, which binds CO_2 to the coenzyme biotin. The graphic (generated from 3G8C.pdb) depicts the locations of the reactants, as well as a few key amino acid residues, within the *E. coli* enzyme's active site. Bicarbonate and ATP are involved in the process; the terminal phosphate group of ATP would fit between ADP, arginine 292, and bicarbonate. The next slide depicts the roles of arginine 338 and glutamate 296.