**Ch20**

P584

**20. Pathway of Atom in Gluconeogenesis**

(a) The bicarbonate binds to pyruvate and form the carboxyl group attaching to methylene. Then, the labeled carboxyl group is removed catalyzed by PEP carboxykinase and the oxaloacetate is converted to PEP. So, the labeled carbon won’t appear in the final carbohydrate.

(b) The labeled carbon atoms appear on **1-C** of pyruvate, oxaloacetate, PEP, 2-phosphoglycerate, 3-phosphoglycerate, 1,3-diphosphoglycerate and 3-phosphoglyceraldehyde, dihydroxyacetone phosphate. And in the **3 or 4-C** of fructose 1,6-diphosphate, fructose 6-phosphate, glucose 6-phosphate and glucose.

**22. Relationship between Gluconeogenesis and Glycolysis.**

a. Some reactions in glycolysis have very low Gibbs free energy change and they are almost irreversible. If gluconeogenesis and glycolysis are totally reverse reaction, the generation will be block at such reactions.

b. Also, if gluconeogenesis uses same enzyme to catalyze the reactions, it will be impossible for cells to regulate the velocities of two reactions.

**27. Relationship between Fructose 1,6-Biphosphatase and Blood Lactate Levels**

Through Cori cycle, muscle release lactate in to the blood and lactate are transported to liver and becomes the material of gluconeogenesis. In liver, the defect of fructose 1,6-diphosphatase inhibit gluconeogenesis hence cause the accumulation of lactate. And further increase the concentration of lactate in blood.

P631

**14. Metabolic Effects of Mutant Enzymes**

(a) Loss of the cAMP-binding site on the regulatory subunit of protein kinase A (PKA) makes that PKA cannot be activated by cAMP. Therefore, PKA isn’t capable to phosphorylate proteins like glycogen phosphorylase b or PFK-2 responding to glucagon or epinephrine and further make glucose concentration is hard to increase.

(b) Without protein phosphatase inhibitor, PP1 is activated and hence dephosphorylate glycogen synthase and glycogen phosphorylase a. In this case, the synthesis of glycogen is activated and the degradation of glycogen is inhibited.

(c) Phosphorylase b kinase could convert phosphorylase b to phosphorylase a that is active. Hence, overexpression of kinase increases the rate of glycogen degradation.

(b) Glucagon could bind to its receptor on hepatocyte and stimulate the degradation of glycogen. Defect of receptor lead to incapability to increase blood glucose and hence cause fatal low blood glucose.

**15. Hormonal Control of Metabolic Fuel**

Between evening meal and breakfast, drop of blood glucose inhibits the excretion of insulin whereas activates the excretion of glucagon. In liver, glucagon binds to its receptor and activate phosphorylase through GPCR signal pathway and stimulate degradation of glycogen. Also, it lowers the concentration of Fructose 2,6-diphosphate in cytosol and hence activate gluconeogenesis. All of above helps to increase blood glucose.

P828

**1. Segregation of Metabolism in Organelles**

(a) By segregating biochemical reaction in different organelles, it becomes easy for cell to regulate different reactions.

(b) Segregation makes metabolism intermediates have different concentration in different organelles. Such as NAD+/NADH concentration is much higher in mitochondria test than in cytosol.

(c) Avoid chaos of reaction. Enzyme demanded in the reaction just exists in the certain organelles. This makes the reaction is ordered.

**2. Phase of Photosynthesis**

The significance of this observation is that it indicates that photosynthesis has two phases, the light phase and the dark phase. It is related to light reaction od photosynthesis because dark phase uses the energy and reducing power to fix CO2. After a brief time, the energy and reducing power is stored in glucose. The energy and reducing power is not enough to fix CO2.

**Extra question:**

1. Many bacterial species can carry out the reactions of gluconeogenesis. Would you expect to find the gluconeogenic enzyme glucose 6-phosphatase in such bacteria? Why or why not?

I expect that glucose 6-phosphatase doesn’t exist in bacteria. Because bacteria are single-cell organisms, so the glucose is just used to build their own body. They don’t need to release glucose to the surrounding.

2. Under what circumstances does the bifunctional protein phosphofructokinase-2 /fructose 2,6-bisphosphatase (PFK-2/FBPase-2) become phosphorylated, and what are the consequences of its phosphorylation to the glycolytic and gluconeogenic pathways?

When hormones like glucagon or epinephrine interact with their receptor on the membrane, they active PKA and phosphorylase the enzymes. The consequence is that the concentration of fructose 2,6-diphosphate is lower hence inhibit glycolysis and activate gluconeogenesis. Further increase blood glucose.