Ch14

P571 10

I think the shortening would not benefit the cell. The reactions deleted could generate 2 ATP molecules. This shorten decrease the production of ATP in glycolysis, hence it makes cells could not create as much energy as wild type.

P571 11

It couldn’t carry out strenuous physical activity and generate ATP at a high rate by glycolysis. The devoid of lactate dehydrogenase prevent the generation of lactose and consuming of NADH. For the ATP production is exclusively by lactic acid fermentation, NADH cannot be converted to NAD+. In this case, the rate of [NADH]/[NAD+] become vastly higher. This will cause the increase of the free energy of reaction generating NADH (like 3-PGAld to1,3-BPG) and make the reaction cannot occur. Because of the accumulate of reactants, the glycolysis will finally stop.

P571 16

During strenuous physical activity, carbohydrates are rapidly converted to water and CO2 and release energy. So, cells need enough carbohydrate to confirm metabolism working normally. Niacin can be used to synthesis NAD+. NAD+ works as coenzyme in glycolysis. It is converted to NADH and then back to NAD+ in following reactions. Therefore, NAD+ and NADH is not consumed during glycolysis and following biological oxidation. Hence, human doesn’t need as much niacin as carbohydrates.

P615 4

(a) PFK-1 is an allosteric enzyme. In addition to substrate binding sites, the allosteric enzymes also contain regulatory sites. ATP acts not only a substrate but also a negative allosteric regulator. It binds to regulatory sites to change the catalysis properties of the enzymes.

(b) It is a negative feedback regulation. The high rate of ATP indicates that cell is energy abundant. It mains that cell doesn’t need to generate lots of energy and hence lower the velocity of glycolysis.

(c) High concentration of ADP indicates that cell needs more energy. Using of ATP increase the generation of ADP. So, it is easy to comprehend that high [ADP] decrease the inhibition of ATP to PFK-1. Maybe it is regulated by the ratio [ATP]/[ADP].

P616 5

(1) In cells, most glucose is phosphorylated by hexokinase and generate glucose-6-phosphate. So, the concentration of glucose is much lower in cytosol.

(2) Because there are specific glucose transporters on cell membrane. They specifically transport glucose into the cell. Hence, they cannot transport glucose-6-phosphate into the cell. Obviously, glucose-6-phosphate cannot diffuse into cells. So, glucose-6-P is not capacity to enter cytosol and is useless.

P616 8

(a) increase

(b) decrease

(c) increase

**Extra question:**

1. Write the net reaction for the following processes.

glucose → 2 pyruvate (glycolysis) 1Glc + 2ADP+ 2Pi + 2NAD+ → 2pyruvate + 2ATP + 2NADH + 2H+

glucose → 2 lactate (fermentation) 1Glc + 2ADP+ 2Pi → 2lactate + 2ATP

Compare the processes in terms of these characteristics:

a. Starting carbohydrate

b. Final carbon product

c. Yield of ATP

d. Yield of NADH

|  |  |  |
| --- | --- | --- |
|  | Glycolysis | Fermentation |
| Starting carbohydrate | glucose | glucose |
| Final carbon product | Pyruvate | lactate |
| Yield of ATP | 2 | 2 |
| Yield of NADH | 2 | 0 |

2. The compound 2,3-bisphosphoglycerate (2,3-BPG) acts as a coenzyme in the glycolytic reaction catalyzed by phosphoglycerate mutase. Through in most cells 2,3-BPG is present only in race amounts - enough to act in its role as coenzyme - it is present in relatively high concentration in erythrocytes, where it acts as a regulator of the affinity of hemoglobin for oxygen. Because erythrocytes synthesize and degrade 2,3-BPG via a detour from the glycolytic pathway, the rate of glycolysis and therefore the rate of generation of glycolytic intermediates has an impact on the concentration of 2,3-BPG. It follows that defects in the glycolytic pathway in erythrocytes can affect the ability of hemoglobin to carry oxygen.

a. How would the concentration of 2,3-BPG, and therefore the affinity of hemoglobin for oxygen, be affected in erythrocytes with a deficiency of hexokinase?

The deficiency of hexokinase causes the damaging of glycolysis pathway. The precursor of 2,3-BPG cannot be generated and therefore lower the concentration of 2,3-BPG. So, the affinity of hemoglobin for oxygen will increase.

b. How would a pyruvate kinase deficiency affect hemoglobin’s affinity for oxygen?

The deficiency of pyruvate causes the accumulation of 2-BPG and therefore make the equilibrium tend to form more 2,3-BPG. So, the affinity of hemoglobin will decrease.