Ch19

**4. Degree of Reduction of Electron Carriers in the Respiratory Chain**

(a)

All of above are reduced

(b)

All of above are reduced

(c)

All of above are oxidized

(d)

The normal condition of electron transferring chain.

**5. Effect of Rotenone and Antimycin A on Electron Transfer**

(a) Rotenone block the electron transferring from NADH to Q, which will cause the respiratory chain start from NADH. Hence, the cell not only could not generate enough energy, but also accumulate a large amount of NADH and increase the ratio of [NADH]/[NAD+]. This will inhibit reactions creating NADH in citric acid cycle.

(b) Antimycin, a respiratory chain inhibitor, block electronic current between Cyt b and Cyt c1. Similar to rotenone, it lowers the generation of ATP and further inhibit TCA.

(c) Antimycin is a more potent poison. Rotenone only block electrons from NADH whereas antimycin block electrons from both NADH and FADH2. So, antimycin almost fully inhibits respiratory chain. Obviously, antimycin is more toxic.

**7. Effects of Valinomycin on Oxidative Phosphorylation**

Valinomycin can combine with K+ and carries it across inner mitochondrial membrane. Once respiratory chain pump one proton out of membrane, valinomycin bring one K+ from intermembrane space. In this case, the electrochemical potential across inner membrane would reach an equilibrium. Hence, though there is proton gradient across inner membrane, proton cannot move through FoF1­ ATPase because electrochemical is equilibrium to proton gradient. Therefore, ATP synthesis is blocked and the rate of electron transfer will increase. Furthermore, the result is an increase of H+ gradient and release of heat.

**13. High Blood Alanine Level Associated with Defects in Oxidative Phosphorylation**

Defects of oxidative phosphorylation cause the accumulation of NADH and FADH2. NADH inhibits the activity of pyruvate dehydrogenase complex. Pyruvate cannot be converted to acetyl-CoA hence it will accept amino group to form alanine. Then alanine will be released into blood and transported to hepatocyte to synthesis glucose.

**19. The Pasteur Effect**

(a) Because pyruvate can be further oxidized in cyclic acid cycle and release more energy with the presence of oxygen.

(b) Because cell can produce much more ATP with oxygen, so less glucose is demanded.

(c) For instance, PFK-1 is regulated by ATP and citrate. Increase of oxygen cause the increasing of ATP and citrate. So, PFK-1 will be inhibited. Thus, lower the rate of glycolysis.

Ch19-2

**6. Limited ATP Synthesis in the Dark**

Short-time illumination to chloroplast simulate the process of photosynthesis. During the process, proton gradient is built. Though illumination disappears, the proton gradient will maintain. Addition of ADP and Pi could use this gradient to form ATP.

**8. Effect of Venturicidin on Oxygen Evolution**

The evolution of oxygen will rapidly decrease. Venturicidin blocking the passing of H+, hence significantly increase H+ gradient. Thus, it becomes harder for photosystem to release H+ into thylakoid lumen. So, the transferring of electrons is blocked. Obviously, photolysis of water is inhibited.

However, 2,4-DNP carry H+ from thylakoid lumen to stoma. Protons couldn’t pass CFoF1 ATPase thus block the synthesis of ATP. For 2,4-DNP, it doesn’t block the passing of H+, so the rate of photosynthesis electron transfer will be increase.

**Extra question:**

Design an experiment to determine whether a chemical is an electron-transport-chain inhibitor or an inhibitor of ATP synthase.

Suspend mitochondria in a buffered medium. Use oxygen electrode to detect consumption of oxygen. Then remove mitochondria and determent the generation of ATP.

After addition of chemical, if the consumption of oxygen increases and the generation of ATP stops, we speculate that the chemical is a kind of *uncoupler.*

If both consumption of oxygen and generation of ATP are inhibited, we can add uncoupler like 2,4-DNP in the suspensions. If the consumption of oxygen back to normal, the chemical maybe a kind of *ATP synthase inhibitor*. If the consumption of oxygen is still inhibited, the chemical maybe a kind of *electron-transport-chain inhibitor*.