Question n1

Oxidation-Reduction Reactions The NADH dehydrogenase complex of the mitochondrial respiratory chain promotes the following series of oxidation-reduction reactions, in which Fe^{3+} and Fe^{2+} represent the iron in iron-sulfur centers, Q is ubiquinone, QH_2 is ubiquinol, and E is the enzyme:

(1) $NADH + H^+ + \text{E-FMN} \longrightarrow NAD^+ + \text{E-FMNH}_2$

(2) $\text{E-FMNH}_2 + 2Fe^{3+} \longrightarrow \text{E-FMN} + 2Fe^{2+} + 2H^+$

(3) $2Fe^{2+} + 2H^+ + Q \longrightarrow 2Fe^{3+} + QH_2$

Sum: $NADH + H^+ + Q \longrightarrow NAD^+ + QH_2$

For each of the three reactions catalyzed by the NADH dehydrogenase complex, identify **(a)** the electron donor, **(b)** the electron acceptor, **(c)** the conjugate redox pair, **(d)** the reducing agent, and **(e)** the oxidizing agent.

Question n2

All Parts of Ubiquinone Have a Function In electron transfer, only the quinone portion of ubiquinone undergoes oxidation-reduction; the isoprenoid side chain remains unchanged. What is the function of this chain?

Question n3

The Pasteur Effect When O_2 is added to an anaerobic suspension of cells consuming glucose at a high rate, the rate of glucose consumption declines greatly as the O_2 is used up, and accumulation of lactate ceases. This effect, first observed by Louis Pasteur in the 1860s, is characteristic of most cells capable of aerobic and anaerobic glucose catabolism.

(a) Why does the accumulation of lactate cease after O_2 is added?

(b) Why does the presence of O_2 decrease the rate of glucose consumption?

(c) How does the onset of O_2 consumption slow down the rate of glucose consumption? Explain in terms of specific enzymes.

Question n4

. **Respiration-Deficient Yeast Mutants and Ethanol Production** Respiration-deficient yeast mutants (p^-; "petites") can be produced from wild-type parents by treatment with mutagenic agents. The mutants lack cytochrome oxidase, a deficit that markedly affects their metabolic behavior. One striking effect is that fermentation is not suppressed by O_2—that is, the mutants lack the Pasteur effect (see Problem 13). Some companies are very interested in using these mutants to ferment wood chips to ethanol for energy use. Explain the advantages of using these mutants rather than wild-type yeast for large-scale ethanol production. Why does the absence of cytochrome oxidase eliminate the Pasteur effect?

Question n5

Rate of ATP Turnover in Rat Heart Muscle Rat heart muscle operating aerobically fills more than 90% of its ATP needs by oxidative phosphorylation. Each gram of tissue consumes O_2 at the rate of 10.0 μmol/min, with glucose as the fuel source.

(a) Calculate the rate at which the heart muscle consumes glucose and produces ATP.

(b) For a steady-state concentration of ATP of 5.0 μmol/g of heart muscle tissue, calculate the time required (in seconds) to completely turn over the cellular pool of ATP. What does this result indicate about the need for tight regulation of ATP production? (Note: Concentrations are expressed as micromoles per gram of muscle tissue because the tissue is mostly water.)

Question n6

- $FADH_2$ and NADH are both electron carriers that bring electrons to the inner mitochondrial membrane to be used during the electron transport chain (ETC). $FADH_2$, however, produces less ATP than NADH. Why?

- Several inhibitors of the electron transport chain have been identified. Barbiturates interfere with complex I. Cyanide binds to the iron in the Heme groups in cytochrome Oxidase. Cyanide poisoning can be deadly. On the other hand, barbiturates are nontoxic. How can we explain this?

- Dinitrophenol (DNP) is an un-coupler, ***i.e.,*** has the ability to separate the flow of electrons and the pumping of H+ ions for ATP synthesis. This means that the energy from electron transfer cannot be used for ATP synthesis. Fifty years ago, DNP was given as a drug to help patients lose weight. Why does this work? Why would this be dangerous

- If you isolate mitochondria and place them in buffer with a low pH they begin to manufacture ATP. Why?