

Study Guide – Lehninger Principles 5th Edition

Chapter Nineteen

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Homework: 2-7, 11, 16. **19.1 Electron Transfer Reactions in Mitochondria.** Learn this linear version of the mitochondrial electron transport chain:

NADH, FMN, (FeS)_{iv}, Q, b_L, b_H, FeS, c₁, c, Cu_A, a, a₃, Cu_B, O₂.

Branch at Q: FADH₂, (FeS)_{iv}, Q.

Understand Q (Fig 19-2), cytochromes (Fig 19-3), and FeS (Fig 19-5). Know names of complexes (Table 19-3 p. 713) and how they divide up the chain. Know certain inhibitors in Table 19-4 (cyanide IV, antimycin A III, rotenone and amytal I, atractyloside A.N. translocase). Know the Q cycle (Fig 19-12) and the general mechanism of Complex IV (Fig 19-14). The energy of electron transport causes protons to be pumped out of the matrix (Fig 19-16).

19.2 ATP Synthesis. The best way to gain an intuitive understanding of how F_o/F₁ work to synthesize ATP is to view the animation on the website. F_o is mostly made up of a rotating transmembrane barrel (c subunits) connected to a “stator” of subunits a and b. The proton gradient forces its way inward (like water flowing through a turbine in a dam) and makes the “c” barrel spin. This rotates a *camshaft* (the γ subunit) which rotates inside the stationary α and β subunits of F₁ and causes them to change shape (Fig 19-25, pp. 727-8). The three shapes are open (or “very loose”), loose, and tight (Fig 19-26). The *loose* conformation binds ADP + Pi, then becomes *tight* which makes ATP, and then *open* or “very loose” which releases it. Know about the *adenine nucleotide translocase*, the *phosphate translocase*, and the *ATP synthasome* (Fig 19-28). Cytoplasmic NADH cannot enter the mitochondrion. The electrons are “smuggled” in by one of 2 shuttles. The *glycerol phosphate shuttle* (Fig 19-30) is used in brain and skeletal muscle and affords only 1.5 ATP per cyto. NADH. The *malate-aspartate shuttle* (Fig 19-29) is in heart/liver/kidney and yields 2.5 ATP. Know details of G-P shuttle but not M-A shuttle.

19.3 Regulation of Oxidative Phosphorylation. For some reason the Nelson text omits mention of the *energy charge function*, which is stated as the expression $([ATP]+.5[ADP])/([ATP]+[ADP]+[AMP])$. As pointed out previously a high energy charge inhibits catabolic pathways at control points. Review Fig 19-33. You should understand *coupling*, *uncoupling*, and *uncouplers*, and why UCP or uncoupling protein produces heat in brown fat tissues (Fig 19-34). Read online handout about eating 2,4-DNP, an uncoupler. The *skunk cabbage* (p. 722) uses a different mechanism to produce heat.

19.4 Mitochondrial Genes: Their Origin and the Effects of Mutations. The evidence that mitochondria once were free living bacteria is extensive. It is surprising that the text doesn't mention the clear relationship between mitochondria and *Rickettsia*. At any rate many bacteria have electron transport chains similar to the mitochondrial version, and various processes driven by “*proticity*.”

19.5 The Role of Mitochondria in Apoptosis and Oxidative Stress . Know that *apoptosis* amounts to cellular suicide, and that one major trigger is the release of *cytochrome c* from the mitochondria. Also understand the discussion of *free radicals* (Fig 19-18, pp. 720-1) and read the online handout.