# Biochem. 311A-Review Short Answer Questions(W. Mushynski)

**1.** The $\Delta G^{O'}$ for the malate dehydrogenase reaction is +29.7 kJ/mole. What might this tell you about the concentration of oxaloacetate in mitochondria? Why does citrate synthase manage to produce sufficient amounts of citrate under these conditions?
**2.** Explain the concept of prochirality as it pertains to the carboxymethyl groups in citrate.
**3.** Summarize in point form evidence for the endosymbiotic origin of mitochondria.
**4.**Mitochondria are incubated in an oxygen electrode in buffer containing excess phosphate, 20 micromoles of ADP, rotenone and substrate X. Oxygen consumption levels off after 5 micromoles of oxygen are consumed. What is the P/O ratio and what is the probable identity of X?
**5.** In the reaction catalyzed by succinyl CoA synthetase, the free energy transfer from succinyl CoA to GTP is mediated by two intermediates. Do these intermediates share any properties with the precursor and end product of the reaction?
**6.** Name the location of succinate dehydrogenase in mitochondria and provide an explanation for its location.
**7.** Outline an experiment using 4-[$^{14}$C] oxaloacetate which demonstrates that aconitase can distinguish between the two carboxymethyl groups in citrate. What were the predicted and actual results of the experiment?
**8.** Name the three CAC enzymes that catalyze rate-limiting steps and list the types of mechanisms involved in the regulation of each enzyme. How would an increase in the NADH/NAD ratio affect the activity of these enzymes?
**9.** In a few words explain why the measured voltage difference between two half cells is directly related to the free energy of a redox reaction. What is the free energy change for a redox reaction in which the electron acceptor and electron donor have standard redox potentials of 0 and -0.30 V, respectively?
**10.** Name two enzymes outside the CAC that have a major impact on CAC activity. How do these enzymes exert their effects?
**11.**Contrast the effects of rotenone, antimycin A and CN- on electron transport.
**12.** Briefly explain how the electron transport system in mitochondria can play a role in disease pathogenesis.
**13.**List the various types of evidence that support the chemiosmotic hypothesis.
**14.**What effect would depletion of ADP have on electron transport in a tightly coupled mitochondrial preparation? What type of compound might be added to allow electron transport to resume under these conditions?
**15.** What conditions must be satisfied for protons to be transported across the inner mitochondrial membrane via the redox loop mechanism?
**16.** What are the roles of the $\beta$- and $\gamma$-subunits in the proposed model for ATP synthesis by the proton translocating ATP synthase?
**17.** Outline the general process of amino acid degradation involving transaminases and glutamate dehydrogenase and indicate how it is regulated. How is net flux in the direction of amino acid catabolism maintained even when the energy charge of the cell is high?
**18.** List the enzymes, precursors and products of the three ammonia assimilation reactions. Which reaction(s) is(are) reversible?
**19.** How is urea synthesis regulated?
**20.** Briefly describe alternative pathways for excreting ammonia in patients with defective urea synthesis.
**21.** List three features of the total body pool of free amino acids.
**22.** What effect might one expect on nitrogen balance in a situation involving a dietary lack of one of the essential amino acids?
**23.** What are the two key components in pyridoxal phosphate that allow it to be such a versatile coenzyme in the metabolism of amino acids?
**24.** Know the mechanism for the transamination reaction.
**25.** Using a diagram and descriptive terms, explain what is meant by the term "stereochemical control" as it pertains to pyridoxal phosphate-containing enzymes.
**26.** Contrast the normal mechanism for decarboxylation of glutamate with that involving an unforced error. What effect does the unforced error have on glutamate decarboxylase?
**27.** Why is there compartmentation of urea cycle reactions?
**28.** Know the reaction mechanism for CPS1. How is the irreversibility of this reaction ensured?
**29.** Contrast the metabolic fates of glucogenic and ketogenic amino acids.
**30.** An inherited defect in cystathionine-$\beta$-synthase gives rise to a condition known as homocystinuria. Why do certain patients harboring this defect respond to high doses of vitamin $B_6$?
**31.** Name the vitamins that serve as coenzymes in the catabolism/recycling of homocysteine. Is there a relationship between methionine metabolism and heart disease?
**32.** Discuss the role of the CAC in amino acid catabolism.