

1. A 4 year old boy was brought to Northfield Hospital (via ambulance by our beloved TA John) following ingestion of a poison that makes the inner mitochondrial membrane permeable to protons. The child was lethargic and had an elevated temperature. Although the child had eaten within the previous 3 hours, his serum glucose level was very low.

- a. What effect would the poison ingested by this child have on ATP production. Explain your reasoning.
- b. Why was he hypoglycemic (low blood sugar)? Your answer should discuss the specific pathways and should indicate how the regulatory steps have responded to the metabolic changes caused by the poison.
- c. What effect would this poison have on his serum fatty acid levels? Explain your reasoning. Be sure to include a discussion on global and local effectors.
- e. Why is his temperature elevated? Be brief.

2. How would the following agents and/or procedures interfere with or disrupt the different levels of protein architecture (i.e. primary, secondary...)? Why?

- a. Addition of SDS
- b. Addition of -mercaptoethanol.
- c. Addition of proteases such as trypsin.

3. In your studies of the enzyme ribonuclease A, you obtain activity data for a wild-type enzyme and a mutant ribonuclease A. The two enzymes differ at one amino acid position in the protein. From the activity data you calculate the K_m for each enzyme:

wild type	$K_m = 10 \text{ mM}$
mutant	$K_m = 0.1 \text{ mM}$

- a. Which enzyme has a higher affinity for substrate? (Assume a two-step reaction, with k_2 the rate-limiting step.)
- b. Which enzyme shifts the K_{eq} , the equilibrium constant, more in the direction of product?

4. Suppose you are stranded on a desert island and have nothing but protein to eat. Outline how your body will use this source of food for its energy needs.