Just a reminder - I want chemical details where appropriate (mechanism, structure, kinetics and so on). Support your answer with details. A series of declaratory or general statements without supporting evidence will not earn top points. No outline or bullet points will be graded. Simple answers will earn simple points.

Multiple Choice 2 points each

1) The difference between a secondary and conjugated bile salt is_____
   a. Conjugated bile salts come from the pancrease while secondary bile salts are cholesterol derivatives transported via chylomicrons
   b. Conjugated bile salts are cholesterol derivatives that have either taurine or glycine added
   c. Secondary bile salts are more involved with food digestion and conjugated bile salts are primarily involved with cholesterol transport.
   d. Both are found in the interstitial fluid outside of the digestive tract

2) Which of the following lipoproteins are the least dense with more lipid than fat?
   a. VLDL b. Chylomicron
   c. ILD d. LDL e. HDL

3) Which protein binds and activates LCAT (lecithin-cholesterol acyltransferase)?
   a. Apo-A1 b. Apo-B100 c. Apo-E d. Apo-C2

4) What describes what takes place in cholesterol synthesis when cholesterol levels are high in a liver cell?
   a. Cholesterol synthesis is activated by an insulin directed protein kinase
   b. The regulatory subunit (sterol regulatory element binding protein: SREBP) is replaced from the activating protein and drives mRNA production for HMG-Co reductase
   c. LDL uptake is decreased due to the decrease in LDL receptor density
   d. The Sterol Cleavage Activating Protein (SCAP) is complexed with the SREB in the endoplasmic reticulum.

1) AMP Kinase is activated in low energy states. Explain how this kinase is activated and how this statement is true.

2) Describe the four steps of beta oxidation of fatty acids. What is the point of the metabolism and what does the “beta” in beta oxidation indicate?

3) After sequencing the genome of the parents of a child with hypercholesterolemia, the father’s LDL receptor has one copy truncated and the mother’s sequence identifies that her Apo-E has several mutations. Neither parent has particularly high cholesterol but the child does. Explain what is happening that causes the child’s high levels of cholesterol.

4) What are the three inaccurately referred to “Ketone bodies”? Describe how they are synthesized and how this metabolic pathway does not appreciably begin unless in a low energy/low carbohydrate state.

5) Explain ***how*** ketone bodies made in liver are used in other tissues and the metabolic purpose of the pathway (how not that or where… HOW).

6) Which hexokinase is expressed in most tissues and where is glucokinase primarily expressed? How does the Km, Vmax and feedback allosteric regulation of one of the hexokinase isoenzymes help “spare glucose for the brain”?

7) How does the glucose transporter and the isoforms regulating glycolysis in pancreatic beta islet cells drive insulin to be released at the appropriate time?

8) If you were a cancer cell, which of the hexokinase isoforms would you choose to express (if a cancer cell could choose) and why?

9) \Adenylate kinase is not active until cells are in a metabolically low energy state. Thinking about Km and cellular concentrations of ATP/ADP/AMP, how is this enzyme drive the signaling of AMP as an

allosteric regulator of metabolism. HOW does AMP signal to inhibit ATP consumption and activate catabolic metabolic processes?

10) Explain how insulin and glucagon control blood glucose levels in the post-absorptive (after a big meal) phase.

11) What is the primary metabolic endpoint of glycolysis during anaerobic activities? What is the limiting reagent that shifts the endpoint of glycolysis to this metabolite and how does the electron transport system and oxidative phosphorylation play a role in this metabolic switch?

12) In long-term starvation, the levels of urea increase. Explain why this occurs and the mechanism by which the excess nitrogen is eliminated.

*COMPLETE ESSAY Pick **1** of the following questions (20 pts)*

A) LDLs are often called bad cholesterol. Explain how this is NOT correct BUT describe the role LDL has in plaque formation and thrombosis - (remember the reminder in the box at the top of this test!).