Exam2 Advanced Normal Nutrition-I Due on 11/10/08 (2:30 pm)



Name: Kaurum ushitield.

- I. Draw the chemical structures of the following fatty acids.
 - . (9 pts)
 - a) Linoleic acid
 - b) Conjugated linoleic acid (9cis, 11trans)
 - e) Eicosapentaenoic acid (EPA)

Identify the positions of the double bonds, the omega and the carboxy terminals.

- II. Read the journal article by Kelley et al "Conjugated Linoleic Acid Isomers and Cancer" (Nirvair S Kelley; Neil E Hubbard; Kent L Erickson *The Journal of Nutrition*; Dec 2007; 137, 12) and answer the following questions:
- 1) Which of the following is a possible mechanism by which the c9, t11-CLA alters tumorigenisis (2pts)
 - a) Inhibiting Cyclooxygenase-2 (cox-2) and the production of PGE2
 - b) Inhibiting the production of 5-HETE while increasing apoptosis
 - c) By not influencing the incorporation of arachidonic acid into membrane phospholipids
 - d) None of the above
- 2) The authors indicate that studies that were done with mixed CLA isomers might not have a scientific basis. What reason(s) do they offer to explain this and what they recommend to over come this problem in the future? (3pts)

Studies done with mixed CLA isomers might not have a scientific basis because the results indicated that different CLA isomers acted through different mechanisms and may have potentially opposing effects of different metabolic pathways. To overcome this problem in the future, standardized preparations enriched in individual isomers are needed as well as controlled studies to determine which particular isomers offer specific benefits and/or risks to humans. Studies are also needed to determine the minimum and appropriate concentration necessary to prove beneficial.

- 3) Which of the following statement(s) are true about the studies conducted with mixed CLA isomers (2pts)
 - a) Feeding mixed CLA isomers inhibited the growth of tumors in animal models of tumorigenesis
 - b) Studies in humans are less conclusive and one study actually found a weak positive relationship between CLA intake and breast cancer incidence
 - c) The timing and duration of the CLA supplementation determined its effectiveness in inhibiting tumors
 - d) All of the above

4) Some of the CLA isomers are Trans fats (2pts)

True or False

- III) Arrange the following statements in the correct order so that they reflect the steps involved in reverse cholesterol transport (3pts)
 - a) HDL particle is taken up by the liver SRB1 receptor 4
 - b) LCAT converts ApoA-1 particles in the blood into HDL by esterifying cholesterol 3
 - c)ApoA-1 particles in the blood stream pick up phospholipids and cholesterol from peripheral tissue with the help of ABCA1 2
 - d) ApoA-1 gene is transcribed, translated and secreted into the blood 1
 - e) In the liver the cholesterol in the HDL is secreted into the bile as cholesterol or as bile acids. 5

IV) Match the following: (8pts)

a) ApoB-100	Carried by chylomicrons H
b) Statins	Activates LPL in peripheral tissue E
c) ApoE	Little to no available oxaloacetic acid (OAA) G
d) Epinephrine or Cortisol	Could inhibit the synthesis of membrane anchors B
e) ApoC2	Uncouples oxidative phosphorylation F
f) UCP-1	Ligand for LDL & chylomicron remnant receptor C
g) Diabetic keto acidosis	Component of LDL and VLDL A
h) Exogenous lipids	Promote lypolysis D

V)

For each enzyme,

indicate the situation under which it is likely to be active (For example: fed, fasted, exercising etc) (8pts)

a) Hormone sensitive lipase:

HSL is active in a fasted state or when the body needs to mobilize energy stores. It is found in the cytosol of adipose tissue and it releases fatty acids to the blood by removing fatty acids from C1 and/or C3 of the TAG.

b) Lipoprotein lipase:

LPL is active in a fed state and is an extracellular enzyme attached to the capillary walls in many tissues but found in primarily adipose and muscle tissue. It hydrolyzes TAG in chylomicrons cand resulting in free fatty acids and glycerol. which positions?

c) Pancreatic lipase:

Pancreatic lipase is active in a fed state where it is secreted from the pancreas into the duodenum and it removes fatty acids at C1 and C3 resulting in a mixture of 2-monoacylglycerol and free fatty acids.

d) Phospholipase A2:



Phospholipase A2 is active in a fed state where it is secreted as part of the pancreatic juice to cleave acetyl linkages from C2 of phospholipids to form a lysophospholipid and arachidonic acid. That our plants were the control of the pancreatic juice to some in the pancreatic juice to some in the control of the pancreatic juice to some in the pancreatic juice to some individual juice to some in the pancreatic juice to some in the pan

VII) In general fatty acids need to be in this form [free fatty acids typically bound to acyl CoA (FA-CoA)] to enter most metabolic pathways. (2pts)

VIII) All of the following give rise to Acetyl CoA, except (2pts)

- a) Fatty acids
- b) Cholesterol
- c) Pyruvate
- d) Some amino acids

d) Found in flax seeds e) All of the above

e) β-hydroxy butyrate

XI) Which of the following is NOT true about the biosynthesis of fatty acids? (2pts)

- a) Acetyl CoA carboxylase (ACC) is the rate limiting enzyme in the biosynthesis of fatty acids.
- b) Denovo fatty acid synthesis usually ends in the formation of palmityl CoA.
- c) Palmityl CoA down regulates the activity of ACC
- d) Fatty acid biosynthesis in the adipose tissue requires DHAP
- e) None of the above

with postprandial thermogenesis after a high fat meal in children. Children were genotyped for the UCP1 polymorphism by PCR RFLP analysis of DNA from buccal samples. There was no reaction of sympathetic activity to the high carbohydrate meal in either the GG allele or the AA+AG group and no significant difference in thermic effect of a meal (TEM). However, after the high fat meal, sympathetic responses were found in both groups; further, the GG allele group showed significantly lower TEM than the AA+AG group. The authors concluded that despite fat-induced sympathetic stimulation, GG allele carriers have a lowered capacity of TEM in response to fat intake, suggesting that such impaired UCP1 linked thermogenesis can have adverse effects on the regulation of body weight. (From:

Accessed on 11/05/08)

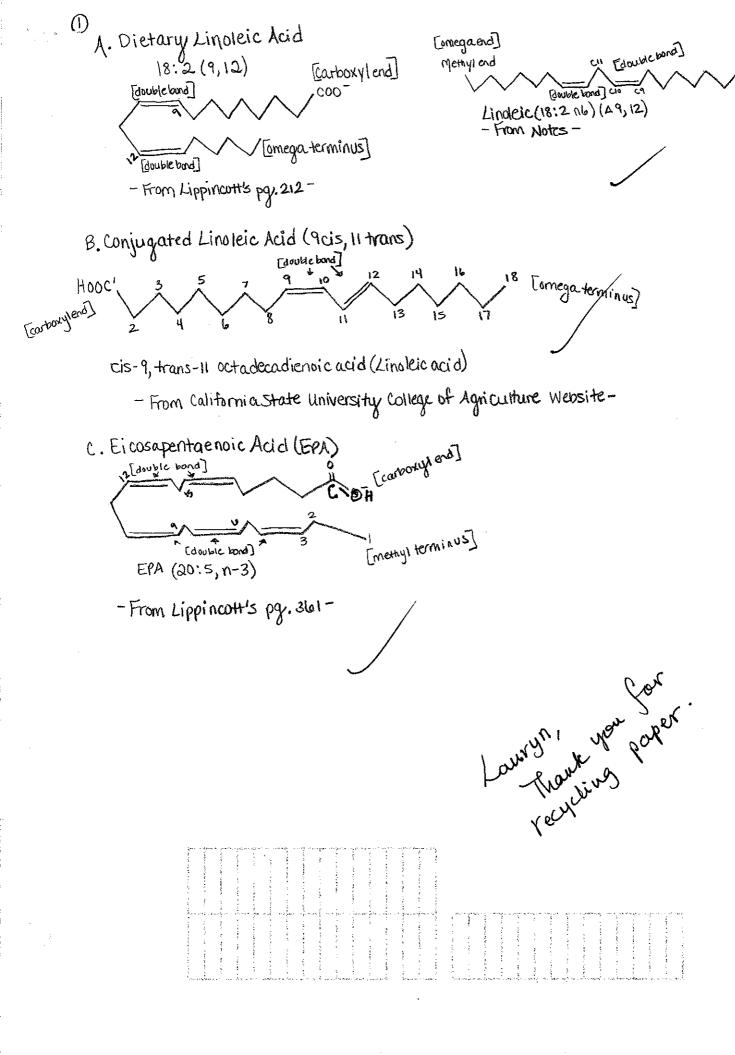
a) Define the thermic effect of a meal (TEM) (2pts)

According to Nutrition for Health, Fitness, & Sport (8th Edition), the significant elevation of the metabolic rate that occurs after ingestion of a meal was previously known as the specific dynamic action of food but is now often referred to as dietary-induced thermogenesis (DIT) or thermic effect of food (TEF). This elevation is due to the energy necessary to absorb, transport, store, and metabolize the food consumed. The greater the caloric content of the meal, the greater

this TEF effect. Also, the type of food ingested may affect the magnitude of the TEF (PRO: 20-30%; CHO 5-10%; Fat 0-5%). The TEF effect accounts for approximately 5-10 percent of the total daily energy expenditure.

b) For individuals with the -3826A-G polymorphism in the UCP1 gene would you recommend a high-fat/low carbohydrate diet or a low-fat/high carbohydrate diet for weight loss? Assume that both diets are isocaloric. Explain your recommendation in the context of the information provided in the above paragraph. (3pts)

Based upon the information provided and the answer to the previous question, I would recommend a low-fat/high carbohydrate diet for weight loss in an individual with this polymorphism. Because there did not appear to be a significant difference in TEM after a high carbohydrate meal but there was a decreased TEM after a high fat meal in these individuals, they may be prone to storing more fat leading to increased body weight. Also, carbohydrates account for a greater percentage of TEM than fat inherently.



COOH

Corboxyl end