

Practice Questions Relevant to Lecture 32 for Exam 3

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Lecture 32 – LIPID REGULATION

1. Insulin promotes an increased rate of cholesterol synthesis through the activation of:
 - A. cyclic GMP phosphodiesterase.
 - B. reductase kinase (RK).
 - C. protein kinase A.
 - D. AMP protein kinase.
 - E. protein phosphatase.

2. The conversion of acetyl CoA carboxylase from the monomeric to the polymeric form is promoted by binding of:
 - A. citrate.
 - B. palmitoyl CoA.
 - C. glucagon.
 - D. AMP protein kinase.
 - E. protein phosphatase.

3. The conversion of acetyl CoA carboxylase from the polymeric to the monomeric form is promoted by binding of:
 - A. citrate.
 - B. palmitoyl CoA.
 - C. glucagon.
 - D. palmitic acid.
 - E. protein phosphatase.

4. Glucagon promotes a decreased rate of cholesterol synthesis by initiating events leading to the activation of:
 - A. reductase kinase (RK).
 - B. cyclic AMP phosphodiesterase.
 - C. protein kinase B.
 - D. AMP protein kinase.
 - E. protein phosphatase.

5. The primary mechanism by which the liver cell prevents oxidation of newly synthesized fatty acid is the inhibition of carnitine-palmitoyl transferase-I (CPT-I) by:
 - A. HMG CoA.
 - B. acetyl CoA.
 - C. citrate.
 - D. malonyl CoA.

5. _____ catalyzes the phosphorylation of hormone-sensitive lipase.

6. _____ catalyzes the dephosphorylation of hormone-sensitive lipase.

7. The induction of which enzyme is brought about by the release from the endoplasmic reticulum of SREBP (Sterol Regulatory Element Binding Protein)

and occurs in the presence of a low concentration of _____

ANSWER KEY

Lecture 32 – LIPID REGULATION

1. E
2. A
3. B
4. A
5. D

6. protein kinase A
7. protein phosphatase
8. HMG CoA reductase low cholesterol