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Metabolism of Lipids

Biochemistry

Beta-oxidation, fatty acid synthesis, ketone body formation, cholesterol metabolism, lipoproteins and clinical importance of lipid metabolism.

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CHAPTER 10

Metabolism of Lipids

Lipolysis, beta-oxidation, synthesis, ketones, cholesterol and lipoproteins

1. Lipolysis and fatty acid transport

During fasting, adipose triglycerides are hydrolysed by lipases (ATGL, hormone-sensitive lipase and monoacylglycerol lipase), yielding free fatty acids and glycerol. Fatty acids travel bound to albumin; glycerol reaches liver for glucose or triglyceride synthesis. Insulin suppresses adipose lipolysis; catecholamines stimulate it.

2. Beta-oxidation

Long-chain fatty acids are activated to acyl-CoA at a cost of two ATP equivalents. The carnitine shuttle (CPT I, translocase, CPT II) transports long-chain acyl groups into mitochondria. Malonyl-CoA inhibits CPT I, preventing simultaneous high rates of synthesis and oxidation.

Cycle step	Reaction
1	Acyl-CoA dehydrogenase: oxidation; FADH2 produced
2	Enoyl-CoA hydratase: hydration
3	Hydroxyacyl-CoA dehydrogenase: oxidation; NADH produced
4	Thiolase: cleavage; acetyl-CoA released and chain shortened by two carbons

Palmitate (C16) undergoes seven cycles, yielding eight acetyl-CoA, seven NADH and seven FADH2; complete oxidation yields approximately 106 net ATP using modern P/O estimates. Very-long-chain fatty acids begin oxidation in peroxisomes. Odd-chain fatty acids yield propionyl-CoA, which enters metabolism as succinyl-CoA via biotin and B12-dependent steps.

3. Fatty acid synthesis

Occurs mainly in cytosol of liver, adipose and lactating mammary tissue. Acetyl-CoA is exported indirectly as citrate. Acetyl-CoA carboxylase (biotin-dependent) uses ATP to form malonyl-CoA; fatty acid synthase uses NADPH to build palmitate. Insulin and citrate favour synthesis; energy deficit and AMPK inhibit it. Elongation and desaturation modify products.

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4. Ketone bodies

Acetoacetate, beta-hydroxybutyrate and acetone are produced primarily in liver mitochondria during low insulin availability or prolonged fasting. Peripheral tissues convert acetoacetate/beta-hydroxybutyrate to acetyl-CoA for energy; liver lacks the key SCOT enzyme for ketone utilization. Nutritional ketosis differs from diabetic ketoacidosis (DKA), in which excess ketoacids cause metabolic acidosis.

5. Cholesterol metabolism

Cholesterol is synthesized from acetyl-CoA; HMG-CoA reductase is a major regulatory enzyme and the target of statins. Cholesterol forms membranes, bile acids, steroid hormones and vitamin D. It is eliminated through biliary secretion and conversion to bile acids; the enterohepatic cycle recycles much of the bile acid pool.

6. Lipoproteins

Lipoprotein	Principal role
Chylomicron	Carries dietary triglycerides from intestine; remnants return to liver
VLDL	Exports hepatic triglycerides to tissues

Lipoprotein	Principal role
IDL	Intermediate from VLDL catabolism
LDL	Delivers cholesterol to tissues; elevated LDL is atherogenic
HDL	Participates in reverse cholesterol transport and lipid exchange

Lipoprotein lipase hydrolyses triglycerides in chylomicrons/VLDL; Apo C-II activates it. Apo B-48 is characteristic of chylomicrons; Apo B-100 occurs in VLDL, IDL and LDL. Apo A-I is a major HDL protein. HDL cholesterol does not directly measure HDL function and high HDL does not eliminate cardiovascular risk.

7. Comparing fatty acid synthesis and oxidation

Feature	Fatty acid synthesis	beta-oxidation
Site	Mainly cytosol	Mainly mitochondrial matrix
Physiological state	Fed, energy surplus	Fasting or increased energy demand
Key molecules	Acetyl-CoA, malonyl-CoA, NADPH	Acyl-CoA, NAD+, FAD, CoA
Major regulatory point	Acetyl-CoA carboxylase	CPT I for long-chain mitochondrial entry
Result	Fatty acid formation	Acetyl-CoA and reducing equivalents

8. Energy yield of palmitate

Seven beta-oxidation cycles yield seven NADH (about 17.5 ATP), seven FADH2 (about 10.5 ATP) and eight acetyl-CoA (about 80 ATP via the TCA cycle and oxidative phosphorylation). Gross yield is about 108 ATP;

subtract two ATP equivalents for fatty acid activation, giving approximately 106 net ATP. Actual cellular yield varies.

9. Endogenous and exogenous lipid transport

Dietary pathway: Intestine -> chylomicrons -> lipoprotein lipase releases fatty acids -> chylomicron remnants -> liver.

Endogenous pathway: Liver -> VLDL -> IDL -> LDL -> uptake by LDL receptors and other routes. HDL participates in cholesterol transport from tissues and exchanges lipids with other particles. ApoB-containing particle retention in arterial walls contributes to plaque formation.

10. Clinical and nursing applications

Dyslipidaemia increases atherosclerotic risk; severe triglyceride elevation may increase pancreatitis risk. Fatty acid oxidation defects can cause fasting hypoketotic hypoglycaemia. DKA requires urgent assessment (Chapter 20). Explain lipid tests, encourage individualized diet and physical activity, monitor treatment adherence and report severe abdominal pain or metabolic deterioration.

11. Important Examination Questions

Long Answer Questions

1. 1. Describe beta-oxidation and ketogenesis.
2. 2. Compare fatty acid synthesis and beta-oxidation. Calculate the approximate ATP yield of palmitate.
3. 3. Describe cholesterol metabolism and the functions of plasma lipoproteins.

Short Answer Questions

1. 1. Write a short note on Carnitine shuttle.
2. 2. Write a short note on HMG-CoA reductase.

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1. 3. Write a short note on lipoprotein functions.

Very Short Answer Questions

1. 1. Name the three ketone bodies. - Acetoacetate, beta-hydroxybutyrate and acetone.
2. 2. Where does fatty acid synthesis occur? - Mainly cytosol.
3. 3. Which lipoprotein carries dietary triglyceride? - Chylomicron.
4. 4. What is approximate net ATP yield of palmitate? - 106 ATP using modern estimates.

Additional objective question

Major enzyme targeted by statins? - HMG-CoA reductase.

12. Quick Revision

- Fatty acids are activated before beta-oxidation.
- Carnitine transports long-chain acyl groups into mitochondria.

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- Malonyl-CoA inhibits CPT I.
- The liver produces ketone bodies but cannot use them as fuel through the usual SCOT pathway.

Conclusion

Lipid metabolism provides fuel during fasting and regulates storage, membrane synthesis and cardiovascular risk; its disturbances are important in metabolic nursing care.

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Practice questions

Choose one best answer. The answer key and explanations follow the questions.

1. Which system transports long-chain acyl groups into mitochondria?

A. Urea cycle
B. Carnitine shuttle
C. Cori cycle
D. Pentose phosphate pathway

2. Which enzyme is the major cholesterol-synthesis regulatory target of statins?

A. Lipoprotein lipase
B. Acetyl-CoA carboxylase
C. HMG-CoA reductase
D. Hormone-sensitive lipase

3. Which lipoprotein exports hepatic triglycerides to tissues?

A. Chylomicron
B. LDL
C. HDL
D. VLDL

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Answer key and explanations

1. B - Carnitine shuttle

The carnitine shuttle enables mitochondrial entry of long-chain acyl groups.

2. C - HMG-CoA reductase

Statins target HMG-CoA reductase in cholesterol synthesis.

3. D - VLDL

VLDL carries triglycerides synthesized or packaged in the liver to tissues.

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