Tishk International University
Faculty of Applied Science
Nutrition and Dietetics Department
2nd Grade
Nutritional Biochemistry II

Lipid Metabolism

Part III

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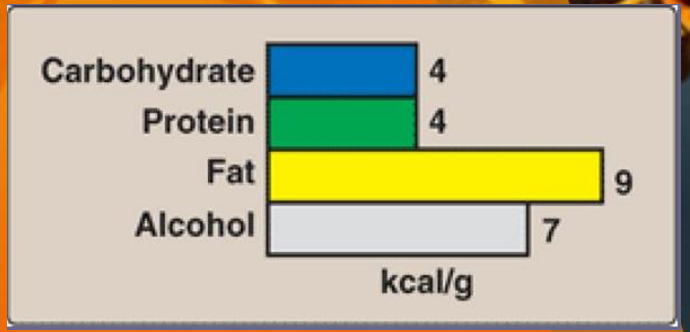
Outline

- Oxidation of Fatty acids
- Fate of Fatty acids
- Disorders Of Lipid Metabolism

Mobilization of Stored Fats and Oxidation of Fatty acids

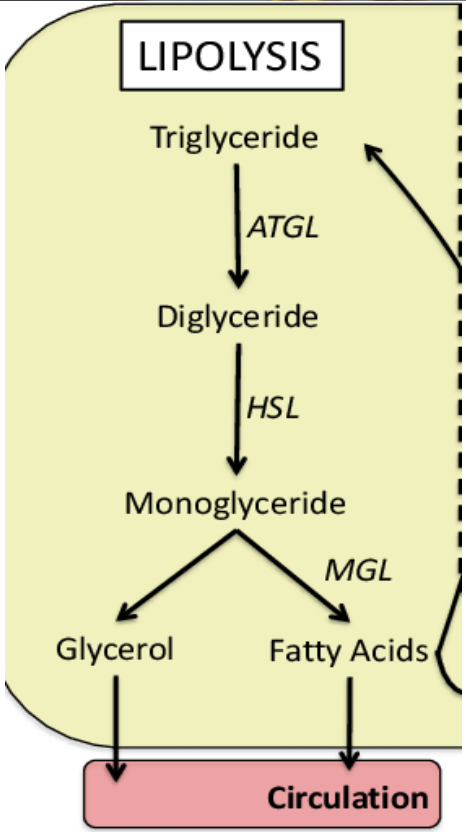
- Fatty acids stored in WAT, in the form of neutral TG, serve as the body's major fuel storage reserve.
- TGs provide concentrated stores of metabolic energy because they are highly reduced and largely anhydrous.
- The yield from the complete oxidation of FAs to CO_2 and H_2O is 9kcal/g fat (as compared to 4kcal/g protein or carbohydrate).



Release of Fatty acids from Fat

- The mobilization of stored fat requires the hydrolytic release of F.As and glycerol from their TG form.

- This process is called Lipolysis and it's achieved by *lipases*

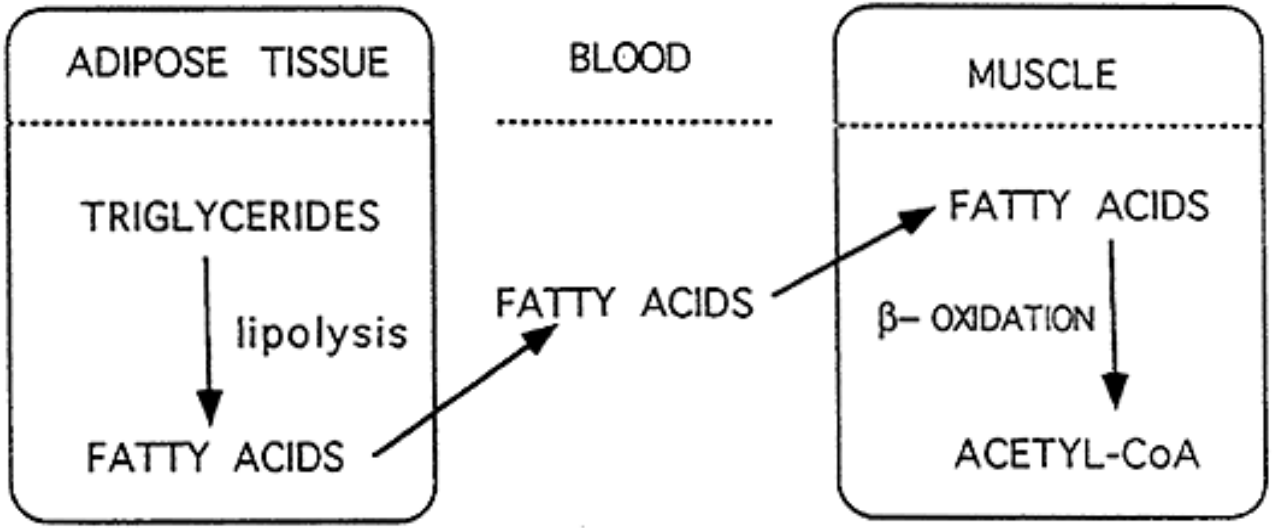


Fate of Fatty acids

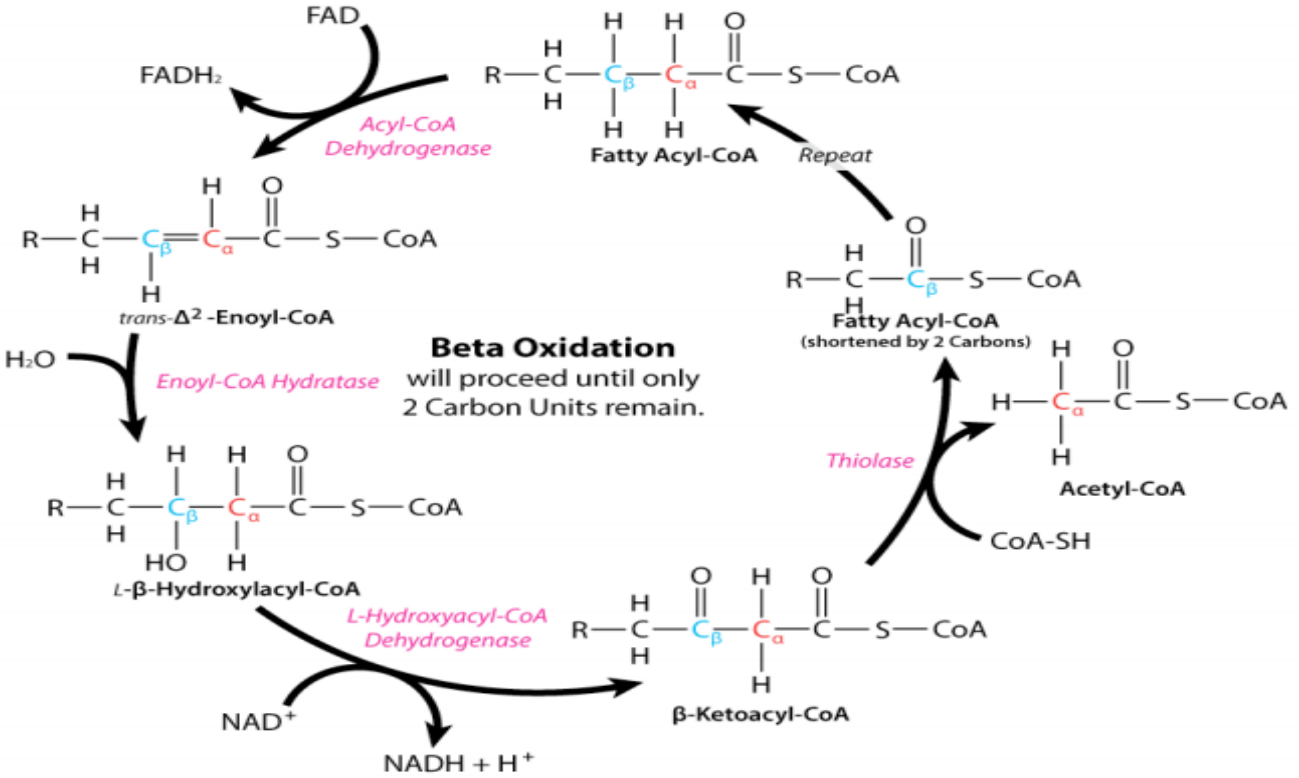
- The free (unesterified) fatty acids move through the cell membrane of adipocyte and bind to plasma albumin.
- They are transported to the tissues, enter cells, get activated to their CoA derivatives, and are oxidized for energy in mitochondria.

β-oxidation of Fatty acids

- The major pathway for catabolism of F.As is a mitochondrial pathway called β-oxidation, in which two-carbon fragments are successively removed from the carboxyl end of the fatty acyl CoA, producing acetyl CoA, NADH and Flavin adenine dinucleotide (FADH2).


ADIPOSE TISSUE
TRIGLYCERIDES
lipolysis
FATTY ACIDS
BLOOD
FATTY ACIDS
MUSCLE
FATTY ACIDS
β– OXIDATION
ACETYL-CoA

β-oxidation Pathway



Steps of β-oxidation:

1. Dehydrogenation: by the enzyme acyl-CoA dehydrogenase, H-acceptor is FAD.
2. Hydration: by the enzyme enoyl hydratase, addition of one molecule of H2O.
3. Dehydrogenation: by the enzyme β-hydroxy acyl-CoA dehydrogenase, H-acceptor is NAD+.
4. Thiolytic cleavage: by the enzyme thiolase and CoASH.

Ketone Bodies

- When the level of acetyl CoA from β-oxidation increases in excess of that required for entry into citric acid cycle, the acetyl CoA is converted into acetoacetate and D-3-hydroxybutyrate by a process known as ***Ketogenesis***.

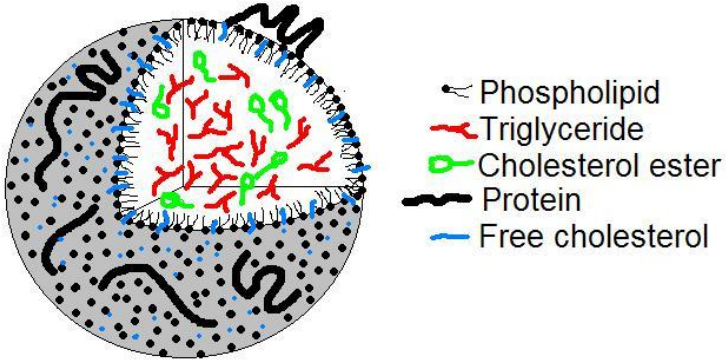
- D-3-hydroxybutyrate, acetoacetate and its non-enzymic breakdown product acetone are referred to collectively as *ketone bodies.*

Plasma lipids

- ✓ Transport lipids, other than un-esterified (free) F.A., which are transported by albumin, are solubilized and carried in the plasma as lipoprotein complex.

<u>All lipoproteins contain:</u>

1. protein.
2. Varying amounts of TG, cholesterol and phospholipid.



Plasma lipoproteins are classified into four major groups, according to their density:

Lipoprotein	Major lipid	Role
Chylomicron	TG	Transport of TG from gut.
VLDL	TG	Transport of TG from liver to tissues.
LDL	Cholesterol ester LDL deposit cholesterol in the arteries.	End product of VLDL catabolism transport of cholesterol to tissues (extra hepatic tissues)
HDL	Cholesterol ester Remove excess cholesterol from the tissues and carry it to the liver where it is converted to bile salts and eliminated.	Transport of cholesterol from tissues (adipose tissue to liver for catabolism).

Disorders Of Lipid Metabolism

- Lipid Malabsorption: Resulting in increased lipid (including the fat- soluble vitamins and essential fatty acids) in the feces, a condition known as steatorrhea, can be caused by disturbances in lipid digestion and/or absorption.

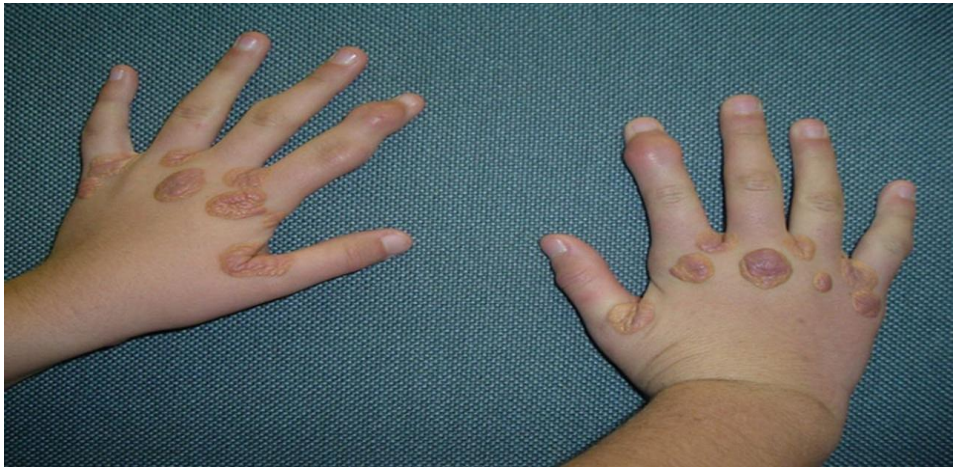
Lipoprotein disorders

- The major classes of lipoproteins are chylomicrons, very-low-density lipoproteins (VLDL), intermediate-density lipoproteins (IDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). Disorders that affect lipid metabolism may be caused by defects in the structural proteins of lipoprotein particles, in the cell receptors that recognize the various types of lipoproteins, or in the enzymes that break down fats.

- As a result of such defects, lipids may become deposited in the walls of blood vessels, which can lead to atherosclerosis (a disease characterized by abnormal thickening and hardening of the walls of the arteries).

Familial hypercholesterolemia

Is an autosomal dominant disease that is caused by the deficiency of the LDL receptor on the surface of cells in the liver and other organs. As a result, cholesterol is not moved into the cells.



Fatty Acid Oxidation defects

- During prolonged starvation, the metabolism of fats stored in adipose tissue is needed for energy production.

- After the glycogen stores have been depleted, both gluconeogenesis and the production of ketone bodies by liver fatty acid beta-oxidation (or β-oxidation) are essential for providing energy for the brain.

- The oxidation of fatty acids for energy occurs in the mitochondria of liver cells and requires a carrier molecule, carnitine, which is synthesized in the body and is also obtained from the dietary intake of animal products such as meat, milk and eggs.

Fatty acid oxidation disorders are relatively common and as a group may account for approximately 5 to 10 percent of cases of sudden infant death syndrome (SIDS).

The disorders commonly manifest with hypoglycemia, liver disease, decreased muscle tone, and heart failure (cardiomyopathy).


THANK
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