

## LIPIDS:

high energy density

hydrophobic

glucose is the major energy substrate in blood

fatty acids are the major energy substrate in most tissues

## LIPOLYSIS:

epinephrine or glucagon binds to G protein coupled receptors

adenylyl cyclase converts ATP to cAMP

protein kinase A phosphorylates hormone sensitive lipase that converts diacylglycerol to monoacylglycerol

protein kinase A phosphorylates perilipin

protein kinase A phosphorylates AMP activated protein kinase kinase to inhibit acetyl CoA carboxylase

insulin activate protein phosphatase that activates acetyl CoA carboxylase

## ACTIVATION OF FATTY ACIDS:

fatty acid + CoA + ATP (acyl CoA synthetase) fatty acyl CoA + AMP

## TRANSPORT OF FATTY ACIDS:

fatty acyl CoA + carnitine (CPT1) fatty acyl carnitine + CoA

fatty acyl carnitine moves into the mitochondrial matrix by a translocase

fatty acyl carnitine + CoA (CPT2) fatty acyl CoA + carnitine  
carnitine moves out of the mitochondrial matrix by a translocase

synthesis of carnitine from lysine and methionine in the liver and kidneys

cardiac muscle contains acetyl CoA carboxylase 2

skeletal muscle contains acetyl CoA carboxylase 2

## FATTY ACID DEGRADATION:

fatty acyl CoA + FAD (acyl CoA dehydrogenase) enoyl CoA + FADH<sub>2</sub>

enoyl CoA + H<sub>2</sub>O (enoyl CoA hydratase) 3 hydroxyacyl CoA

3 hydroxyacyl CoA + NAD<sup>+</sup> (3 hydroxyacyl CoA dehydrogenase) 3 ketoacyl CoA + NADH + H<sup>+</sup>

3 ketoacyl CoA (thiolase) fatty acyl CoA + acetyl CoA

propionyl CoA + CO<sub>2</sub> + ATP (propionyl CoA carboxylase + vitamin B7) D methylmalonyl CoA

propionyl CoA + HCO<sub>3</sub><sup>-</sup> + ATP (propionyl CoA carboxylase + vitamin B7) D methylmalonyl CoA

D methylmalonyl CoA (methylmalonyl CoA epimerase) L methylmalonyl CoA

L methylmalonyl CoA (methylmalonyl CoA mutase + vitamin B12) succinyl CoA

each double bond in unsaturated fatty acids eliminates one FADH<sub>2</sub>

## ACTIVATION OF FATTY ACIDS:

fatty acid + CoA + ATP (acyl CoA synthetase) fatty acyl CoA + AMP

## TRANSPORT OF FATTY ACIDS:

fatty acyl CoA moves into the peroxisomes by ABCD transporter

## DEGRADATION OF FATTY ACIDS:

fatty acyl CoA + FAD (acyl CoA oxidase) enoyl CoA + FADH<sub>2</sub>

O<sub>2</sub> + FADH<sub>2</sub> FAD + H<sub>2</sub>O<sub>2</sub>

peroxisomal bifunctional protein:

enoyl CoA hydratase

3 hydroxyacyl CoA dehydrogenase

mitochondrial trifunctional protein:

enoyl CoA hydratase

3 hydroxyacyl CoA dehydrogenase

3 ketoacyl CoA thiolase

alpha oxidation of fatty acids in peroxisomes:

1 hydroxylation by a hydroxylase

2 decarboxylation by a lyase

3 oxidation by an aldehyde dehydrogenase

release more than one byproduct

refsum disease is caused by a deficiency of a hydroxylase

omega oxidation of fatty acids in smooth endoplasmic reticulum:

1 hydroxylation by a monooxygenase

2 oxidation by an alcohol dehydrogenase

3 oxidation by an aldehyde dehydrogenase

produce dicarboxylic acid

carbohydrates use less oxygen in metabolism

lipids use more oxygen in metabolism

PHYSICAL ACTIVITY:

PHASE 1:

ATP ADP

ADP + creatine phosphate (creatine kinase) ATP +  
creatine

PHASE 2:

anaerobic metabolism

PHASE 3:

aerobic metabolism