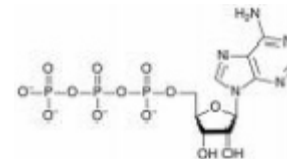


METABOLISM I: CATABOLISM, ABSORPTIVE AND POST-ABSORPTIVE STATE

4/20/82, rvsd 5/13/97, 9 May 2000, 24 April 2002, 27 April 2005, 4May09, 11May09, 3May10
S&M: p673-. Martini's 5th: 901-937, 6th: 929-964, 8th: 930-963



Metabolism: **Catabolism** breaking down molecules to release energy (stored in ATP)
(p. 931) **Anabolism** synthesis of molecules, using energy (ATP supplies energy)

CATABOLISM (Most energy is derived from of energy from breaking down carbohydrate. Ex: glucose.)
[vitamin required] (Summary on page 939)

GLYCOLYSIS: occurs in **cytoplasm**, successive breakdown of glucose to produce 2 ATPs.
(P. 933) Note high energy (shown with a ~) configuration between joined PO₄s: R-PO₄~PO₄

Glycolysis summary: **double PO₄lytion:** glu to G-6-P to F6P to F 1,6,diP: splits to 3PGA and DHAP,
[niacin] **NADH produced:** 3PGA oxidized, gains P₀₄ to 1,3 DPG acid, then **makes ATP**
makes a second ATP: conversion to PEP contributes PO₄, **pyruvic acid end product.**
Fermentation: Anaerobic (to regenerate NAD): pyruvate gains back H fr NADH to **lactic acid** in muscles.
(Why?: Without O₂, no respiration, NADH builds up, lack of NAD⁺ would halt glycolysis.)
(P. 935)

[thiamine, niacin] **Complex preparatory step** combines with CoASH, giving off CO₂ and reducing NAD.
[pantothenic acid] Generates **Acetyl CoA** Feeds into Krebs cycle, or synthesis of fat, or other molecules.

KREBS CYCLE in **mitochondria:** Acetyl CoA (2 per starting glucose molecule) is degraded.
Hydrogen is 'dissected off' and added to hydrogen carriers, yielding:
[riboflavin] 3 NADHs, 1 FADH₂, 1 GTP, and 2 CO₂s. Yields 34 more ATPs/ glucose
[thiamine] **Decarboxylation** produces CO₂ of respiration, 2H plus 1/2O₂ produces H₂O
Reduction of NAD⁺ and FAD to NADH and FADH₂

Cytochrome system oxidizes NADH and FADH₂ with O₂, to make H₂O. (Oxidative phosphorylation)
(P. 937) This oxidation generates a H⁺ gradient which drives ATP synthase yielding ATPs

Fat β oxidation (p. 941) yields Acetyl CoA, proteins yield alpha keto acids, feed into Krebs cycle for oxidation.

ANABOLISM: Acetyl CoA can be used to make fat, AA, back to glucose.

Two states of body according to recentness of eating: (p. 949)

ABSORPTIVE STATE (following a meal) **INSULIN REGULATED.** Insulin triggers:

- 1) **glucose uptake** in all tissues of
- 2) **synthesis of glycogen** in liver and muscle
- 3) **synthesis of fat** from excess glucose, **deposition** in adipose tissue
- 4) **enhanced protein synthesis**

CH₂O primary use: energy source. Liver converts galactose and fructose to glucose, stored in liver
amino acids available for protein synthesis
fat most absorbed fat is stored

POST ABSORPTIVE STATE: GLUCAGON REGULATED (INITIALLY) (p 949)

Body is relying on stored internal energy reserves

glycogen hydrolysis maintains blood glucose (4 hr worth)
lipolysis Liver converts fat to Acetyl CoA and ketone bodies
Ketone bodies these can be used by peripheral tissues (**acetone breath**)
prolonged fasting protein becomes a major source of blood glucose. (Not fat???)
amino acids deaminated to produce keto acids, burned in glycolysis and Krebs cycle

most organs switch to fat for energy, but **brain must use glucose.**

Brain: after 4-5 days can use ketone bodies for energy, conserves body's protein since it is major source of fasting glucose.