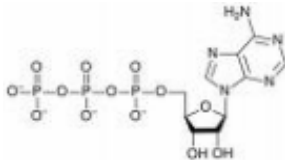
METABOLISM I: CATABOLISM, ABSORPTIVE AND POST-ABSORPTIVE STATE

Revised 7 April 2016
S&M: p673-. Martini's 5th: 901-937, 6th: 929-964, 8th: 930-963, 10th: 936-971



Metabolism: **Catabolism** breaking down molecules to release energy (stored in ATP)
(p. 937) **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. 940) Note high energy (shown with a ~) configuration between joined PO_4s: R-PO_4~PO_4
Other high energy PO_4 bonds in 1, 3 Diphosphoglyceric acid and Phosphoenolpyruvate

Glycolysis summary: **double PO_4lytion**: glu to G-6-P to F6P to F 1,6,diP: splits to 3PGA and DHAP,
[niacin] **NADH produced**: 3PGA oxidized, gains $P0_4$ to 1,3 DPG acid, then **makes ATP**
makes a second ATP: conversion to PEP contributes PO_4 , **pyruvic acid end product**.

Fermentation: Anaerobic (to regenerate NAD): pyruvate gains back H fr NADH to **lactic acid** in muscles.
(Why?: Without O_2, no respiration, NADH builds up, lack of NAD^+ would halt glycolysis.)

[thiamine, niacin] **Complex preparatory step** combines with CoASH, giving off $C0_2$ 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.
(P 941) Hydrogen is 'dissected off' and added to hydrogen carriers, yielding:
[riboflavin] 3 NADHs, 1 FADH2, 1 GTP, and 2 CO_2s. Yields 34 more ATPs/ glucose
[thiamine] **Decarboxylation** produces CO_2 of respiration, 2H plus $1/2O_2$ produces H_2O
Reduction of NAD^+ and FAD to NADH and $FADH_2$

Cytochrome system oxidizes NADH and $FADH_2$ with O_2, to make H_2O. (Oxidative phosphorylation)
(P. 944) This oxidation generates a H^+ gradient which drives ATP synthase yielding ATPs

Fat β oxidation (p. 948) 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. 954)

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**

CH_2O	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 955)
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.

METABOLISM II: METABOLIC RATE, DIET AND OVEREATING

revised 7 April 2016
S&M 688-, Martini 966-977, 6th: 8th: 955-, 10th: 957-971

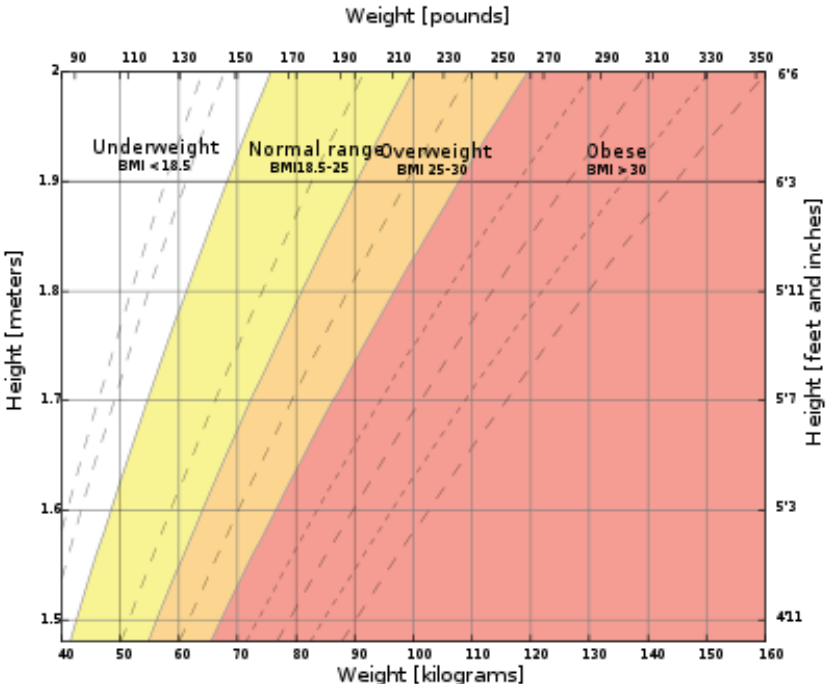
Review **Liver functions**: synthesis of plasma proteins; bile synthesis; Synthesis of clotting factors; glycogen synthesis and storage; fat breakdown, form ketone bodies; bilirubin excretion; amino acid interconversion, deamination; urea synthesis; gluconeogenesis from broken down protein

Metabolic rate: energy expended by body per unit time,
direct measurement: **Calorimeter measures heat**, resting, fasting, no work done: heat liberated
indirect measurement: **O_2 consumption**: 1 L O_2 is equivalent to 4.825 Kcal of heat

Measurement must be done resting in **post-absorptive state** (12 hr fast)

liver processing *increases* BMR 10-20 % (not due to cost of digestion):
protein *30 % increase in BMR*
CH_2O 8%
Lipid 8%

Strenuous exercise increases BMR 15x...



REGULATION OF ENERGY BALANCE:
caloric intake must equal caloric requirements for work performed for stable condition

CONTROL OF HUNGER:
Feeding center
hunger triggered in lateral hypothalamus, stimulated by low blood glucose, low temp
Satiety center
(inhibits feeding center) in ventromedial hypothalamus, stimulated by:
hi blood glucose
increased body temp
specific dynamic action

PROBLEMS ASSOCIATED WITH DIET AND OVEREATING:

Body Mass Index - BMI = [(weight in lbs * 703)/(height in inches)^2]

Obesity: 20% over ideal weight associated with a 50% higher mortality rate .
In USA, % obese: 40% women; 30 % men

BMI	weight
Below 18.5	underweight
18.5-24.9	normal
25-29.9	overweight
0ver 30	obese

Young obese have more adipose cells adult obese have enlarged adipose cells

Dietary saturated fats, cholesterol:

Cholesterol: (p 944) critical functions: cell membranes, bile salts, steroid hormones, vitamin D
we make 80 % of cholesterol circulating in blood.

Cholesterol is carried in the blood:	HDL:	fr periphery to liver	should be *over* 35 mg/dL
	LDL:	fr GI to periphery	should be *below* 130 mg/dL

Exercise raises HDL. Unsaturated fats as well

Dietary saturated fats turned into acetyl CoA, turn them into cholesterol.