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

**Metabolism:**

- **Catabolism** (p. 937) - breaking down molecules to release energy. (stored in ATP)
- **Anabolism** - synthesis of molecules, using energy. (ATP supplies energy)

### Catabolism

- **Glucose:** Most energy is derived from energy from breaking down carbohydrate. Ex: glucose.

#### Glycolysis

- **Summary on page 939**
- Occurs in **cytoplasm**, successive breakdown of glucose to produce 2 ATPs.
- **GLYCOLYSIS:** (P 940)
- Note high energy (shown with a ~) configuration between joined PO₄:s: R-PO₄~PO₄
- Other high energy PO₄ bonds in 1, 3 Diphosphoglyceric acid and Phosphoenolpyruvate

**Glycolysis summary:** double PO₄lytion: glu to G-6-P to F6P to F 1,6,diP: splits to 3PGA and DHAP,
- **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.

- **Complex preparatory step** combines with CoASH, giving off CO₂ and reducing NAD.

#### Krebs Cycle

- **in mitochondria:** Acetyl CoA (2 per starting glucose molecule) is degraded.
- **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)

**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

- **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 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

#### 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.
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**: Oxygen consumption: 1 L O₂ 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
- CH₃O: 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 = \[
\frac{(\text{weight in lbs} \times 703)}{\text{(height in inches)}^2}\]

<table>
<thead>
<tr>
<th>Obesity</th>
<th>20% over ideal weight associated with a 50% higher mortality rate</th>
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<tbody>
<tr>
<td>In USA, % obese:</td>
<td></td>
</tr>
<tr>
<td>women</td>
<td>40%</td>
</tr>
<tr>
<td>men</td>
<td>30%</td>
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</tbody>
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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.