General Principles: Multisystem processes – Nutrition (from 2004 Content Description and Sample Test Materials)

- generation, expenditure and storage of energy at the whole-body level
- assessment of nutritional status across the lifespan, including calories, proteins, essential nutrients, hypoalementation
- functions of nutrients, including essential, trans-fatty acids, cholesterol
- protein-calorie malnutrition
- vitamin deficiencies and/or toxicities
- mineral deficiencies and toxicities
- eating disorders (e.g., obesity, anorexia, bulimia)

Protein quality measures - essential amino acids (EAAs)

a. NPU or PDCAAS is determined by EAA quantities/ratios and digestibility
b. Applies to individual proteins or to total dietary protein consumed daily
c. Combining lower value proteins can provide a good quality meal (principle of complementarity in vegetarian diets)
d. Rule of thumb: Regardless of protein sources, if tryptophan, lysine and sulfur (S)-amino acid (*) intake is sufficient (“limiting amino acids”), the remaining EAAs are likely to be adequate in the overall daily diet

Macronutrients. Dietary protein (~4 kcal/g)

<table>
<thead>
<tr>
<th>Protein usefulness/quality measures and essential amino acids</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. BV, Biological Value is roughly the proportion of dietary protein absorbed that is used by the body. Limited because it does not account for protein digestibility</td>
</tr>
<tr>
<td>b. NPU, Net Protein Utilization takes into account BV and digestibility</td>
</tr>
<tr>
<td>c. PDCAAS, Protein Digestibility-Corrected Amino Acid Score is a recent improvement over NPU that corrects for “true” digestibility</td>
</tr>
</tbody>
</table>

THE COMPOSITION OF ESSENTIAL AMINO ACIDS ULTIMATELY DETERMINES THE PROTEIN’S (AND THE MEAL’S) QUALITY

<table>
<thead>
<tr>
<th>Protein</th>
<th>PDCAAS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>egg white</td>
<td>100</td>
</tr>
<tr>
<td>milk protein</td>
<td>100</td>
</tr>
<tr>
<td>rice</td>
<td>100</td>
</tr>
<tr>
<td>ground beef</td>
<td>87</td>
</tr>
<tr>
<td>chicken</td>
<td>97</td>
</tr>
<tr>
<td>soy protein</td>
<td>98</td>
</tr>
<tr>
<td>whole wheat flour*</td>
<td>93</td>
</tr>
<tr>
<td>garbanzo</td>
<td>90</td>
</tr>
<tr>
<td>barley</td>
<td>88</td>
</tr>
<tr>
<td>peas</td>
<td>87</td>
</tr>
<tr>
<td>beans</td>
<td>82</td>
</tr>
<tr>
<td>peas</td>
<td>82</td>
</tr>
<tr>
<td>whole wheat</td>
<td>80</td>
</tr>
</tbody>
</table>

*example of protein complementarity
A further look at soy protein

Causes of Inadequate Nutrient Intake (Quantity or Quality)
- Aging
- Mental Illness
- Alcoholism
- Drug addiction
- Avoidance of specified food groups (meat, eggs, milk, fruits and vegetables, grains)
- Poor dentition
- Food idiosyncrasies
- Poverty, isolation
- Anorexia (from disease process, drugs, emotional problems)
- Recent weight loss or gain
- Inappropriate food choices from lack of information
Marasmus and Kwashiorkor compared

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Clinical setting</th>
<th>Two symptoms</th>
<th>Clinical features</th>
<th>Laboratory findings</th>
<th>Clinical score</th>
<th>Condition severity</th>
</tr>
</thead>
</table>
| Marasmus | Hospitalized | 1 year | Neutropenia | Weight < 50th percentile | 0-20% | |}
| Kwashiorkor | Hospitalized | 1 year | Neutropenia | Weight < 50th percentile | 0-20% | |}

*DALY is a composite measure of years of life lost because of premature mortality and equivalent years of life lost due to lower quality of life as a result of disability. One DALY = one year of healthy life.*

World

Deaths in 2000 attributable to Undernutrition and Diet-related Risks and Physical Inactivity

![Bar chart showing deaths attributable to various factors such as blood pressure, overweight, and underweight.]

Source: WHO, 2005

World

Disease Burden (DALYs) in 2000 attributable to Undernutrition and Diet-related Risks and Physical Inactivity

![Bar chart showing disease burden attributable to various factors such as blood pressure, overweight, and underweight.]

Source: WHO, 2005
Actual Causes of Death in the United States, 2000

Willett et al., Scientific American, 2003

What Kind of Fat Are You Eating?

Fats = 9 kcal/g

Fat and Heart Disease
Dietary cholesterol (Ch) and serum Ch

A. Coronary Heart Disease (CHD): No simple relationship to CHD between 190-240 mg/dl serum Ch, but CHD risk is directly related to [LDL-Ch] and inversely related to [HDL-Ch]
B. Reducing Ch intake, in concert with statin inhibitors of HMGCoA reductase, can reduce high serum LDL-Ch levels
C. Also, increased Ch intake in some studies correlates with increased colon cancer risk
D. Dark side of low plasma Ch levels—increased risk of hemorrhagic (as opposed to ischemic) stroke
E. And Ch levels <150 mg/dl could indicate PEM (check blood protein levels)

Polyunsaturated Fatty Acids

Effects of eicosanoids: prostaglandins (PG), thromboxane (TX), and leukotrienes (LT)
**TABLE 1. Potential Mechanisms by Which Omega-3 Fatty Acids May Reduce Risk for Cardiovascular Disease**

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>By Suppressing Ca+2 influx in heart cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduce susceptibility of the heart to ventricular arrhythmia</td>
<td></td>
</tr>
<tr>
<td>Antiarrhythmogenic</td>
<td></td>
</tr>
<tr>
<td>Hypotriglyceremic (fasting and postprandial)</td>
<td></td>
</tr>
<tr>
<td>Retard growth of atherosclerotic plaque</td>
<td></td>
</tr>
<tr>
<td>Reduce adhesion molecule expression</td>
<td></td>
</tr>
<tr>
<td>Reduce platelet-derived growth factor</td>
<td></td>
</tr>
<tr>
<td>Antiinflammatory</td>
<td></td>
</tr>
<tr>
<td>Promote nitric oxide-induced endothelial relaxation</td>
<td></td>
</tr>
<tr>
<td>Mildly hypotensive</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Corsini 11

*Stabilize existing plaque and thus thrombotic events (2004)*

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**What fish should you eat?**

Fish that may have high levels of mercury:
- Swordfish
- Shark
- King mackerel
- Bluefin tuna

Fish that generally have low levels of mercury:
- Salmon
- Haddock
- Cod
- Tuna
- Scallops
- Trout
- Sardines
- Largemouth bass

---

**Possible downsides of increased marine fish consumption:**

- a. Mercury contamination, which is potentially damaging to the fetus and its nervous system
- b. Elevated free radical generation (to be discussed) from general increases in easily oxidizable PUFAs; free radicals have been linked to increased cancer risk

---

**9. Concerns about trans-PUFAs**

- a. Trans PUFAs arise mainly during partial hydrogenation of oils, but traces also occur in plants
- b. “Mixed” trans-PUFAs are linked to increases in LDL/HDL ratios, and to increased atherosclerosis
- c. Paradoxically, two “conjugated” trans/cis linoleic acids (CLAs) at right are cardioprotective and anticancer in experimental studies

---

**14-trans-arachidonic acid**
C. Dietary Carbohydrate (~4 kcal/g)

1. Carbohydrate ranges from simple and refined sugars (mono- or disaccharides, often “disguised”) to complex digestible amyloses and starches.

2. Constitute as much as 250 g daily, supplying >50% of total calories for many people.

3. Is a short-term energy source, not “essential” like PUFAs.

4. Less than 50 g/day of complex carbohydrate is needed to suppress ketosis from fat metabolism and spare protein.

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C. Dietary Carbohydrate, continued:

Glycemic index is the ratio of the area under the blood glucose curve produced by a specific carbohydrate-containing food compared to that produced by equivalent glucose or by plain old white bread.

Glycemic load (probably more meaningful value overall) refers to the product of the glycemic index (qualitative measure) multiplied by the actual amount of carbohydrate in the portion of food under concern.

Insulinemic index is the insulin response to a given glycemic load, being positively correlated with carbohydrate ingested.

---

**GLYCEMIC LOAD OF A SAMPLING OF FOODS**

<table>
<thead>
<tr>
<th>Food (size serving)</th>
<th>Carbohydrate content (in grams)</th>
<th>Glycemic index* (percent expressed as decimal)</th>
<th>Glycemic Load (indexed to reference white bread)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato (1/2 serving)</td>
<td>17</td>
<td>1.27</td>
<td>27</td>
</tr>
<tr>
<td>Corn (1 cup cooked)</td>
<td>8</td>
<td>1.11</td>
<td>18</td>
</tr>
<tr>
<td>Lentil (1/2 cup cooked)</td>
<td>37</td>
<td>0.40</td>
<td>2</td>
</tr>
<tr>
<td>Dry beans (cup cooked)</td>
<td>27</td>
<td>0.82</td>
<td>22</td>
</tr>
<tr>
<td>White rice (1 cup cooked)</td>
<td>32</td>
<td>0.51</td>
<td>16</td>
</tr>
<tr>
<td>Wild rice (1 cup cooked)</td>
<td>18</td>
<td>0.75</td>
<td>14</td>
</tr>
<tr>
<td>White bread (2 slices)</td>
<td>74</td>
<td>1.02</td>
<td>27</td>
</tr>
<tr>
<td>Whole grain bread (1 slice)</td>
<td>24</td>
<td>0.94</td>
<td>21</td>
</tr>
<tr>
<td>Pizza (1 cup cooked)</td>
<td>40</td>
<td>0.71</td>
<td>25</td>
</tr>
<tr>
<td>Cheesecake (serving)</td>
<td>27</td>
<td>1.56</td>
<td>33</td>
</tr>
<tr>
<td>All-Bran (1 cup)</td>
<td>28</td>
<td>1.65</td>
<td>44</td>
</tr>
<tr>
<td>Grape Nuts (1 cup)</td>
<td>45</td>
<td>0.66</td>
<td>45</td>
</tr>
<tr>
<td>Corn flakes (1 cup)</td>
<td>20</td>
<td>1.13</td>
<td>21</td>
</tr>
<tr>
<td>Cornchips (1 cup)</td>
<td>175</td>
<td>1.05</td>
<td>155</td>
</tr>
<tr>
<td>Popcorn (1 cup)</td>
<td>5</td>
<td>0.70</td>
<td>4</td>
</tr>
</tbody>
</table>

* Standard reference for this table is white bread.
D. Alcohol (ethanol, ~7 kcal/g), often a major source of dietary calories:

1. Excessive alcohol consumption = empty calories and nutrient depletor

---

9. Diseases associated with carbohydrate metabolism

a. Diabetes mellitus - glucose intolerance (elevations) due to lack of insulin production (type I) or insulin resistance (type II) leading eventually to organ failures and peripheral nerve degeneration; approaching epidemic proportions in USA and most developed countries

b. Obesity (also a major risk factor for Type II diabetes)

c. Lactose intolerance due to lactase deficiency

d. Studies suggest a link between:
   1) Excess dietary carbohydrate and CHD, with possible mechanisms involving excessive circulating insulin
   2) High glycemic index diets and risk of CHD in women
   3) Low glycemic index diets and reductions in LDL cholesterol, especially in women subjects studied

"A diabetemic" (New Yorker)

Remember when we used to have to fatten the kids up first?
In addition to low dietary intake, depletion could also arise from impaired absorption and deficient metabolic activation of nutrient vitamins caused by chronic alcohol abuse.

The softer side of ethanol: moderate* intake:

- correlates with a reduced risk of heart disease ("the French Paradox")
- is linked in some (but not all) studies to lower risks of stroke
- has been shown in several recent epidemiological studies to be associated with a reduced risk of dementia, including Alzheimer's
- BUT still may increase risks of cancers of the breast, intestine, liver and larynx (interpretation complicated by smoking, poor diets)

*definitions vary, but usually a maximum of 2 drinks/day for men and 1 for women (~15 g ethanol/drink), or 7-14 drinks a week (sorry, not all on a weekend night)
ALCOHOL CONSUMPTION INCREASES THE RISK OF BREAST CANCER

Minerals

<table>
<thead>
<tr>
<th>Mineral</th>
<th>RDA</th>
<th>Deficiency</th>
<th>Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>1000 mg</td>
<td>Bone density, stress fractures</td>
<td>Hypercalcemia, kidney stones</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>800 IU</td>
<td>Bone density, muscle strength</td>
<td>Excess intake can cause hypercalcemia</td>
</tr>
</tbody>
</table>

Vitamins

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>RDA</th>
<th>Deficiency</th>
<th>Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>10 mcg</td>
<td>Night blindness, increased susceptibility to infections</td>
<td>Hypervitaminosis A</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>75-90 mg</td>
<td>Immune system function, collagen synthesis</td>
<td>Excess intake can cause gastrointestinal upset</td>
</tr>
</tbody>
</table>

Graph showing the relationship between alcohol consumption and the risk of breast cancer.
Dietary "Micro" minerals:  
a. Iron - indispensable, yet double-edged

(1) Two forms of iron in diets:  
a) 20-25% readily absorbed heme iron (1/2 comes from fish, meats)  
b) 75-80% non-heme iron in vegetables, legumes, etc., with absorption ranging from 2-20% depending on diet, body needs

(2) Iron, a key component as a heme and non-heme constituent in many proteins, cytochromes, and lysosomal enzymes

(3) Iron has a complex physiology, operating in a relatively closed system: relatively limited absorption and negligible loss (figure)

(4) Non-heme iron absorption is promoted significantly by vitamin C, which reduces ferric to ferrous ion, freeing it for mucosal uptake

Aspects of Zinc deficiency in underdeveloped world

One in five people are very deficient, esp. women and children, because of food poor in Zn (Zn-rich foods: whole grains, green veggies, legumes, seafood, meat)  
Along with iodide, iron and Vit. A, Zn deficiency is now considered a global health problem that needs to be overcome (UN WHO and UNESCO studies, 2001-04)

Zn deficiency is connected with maternal and child health problems: diarrheal diseases, poor immune function, impaired wound healing, increased morbidity and mortality

Caveat: High Zn supplementation is potentially damaging to developing and adult brain: like iron, a double-edged mineral
Four lipid-soluble vitamins

1. VITAMIN A (Retinol from Carotenoids, especially β-carotene)

    - Structures:

        ![Chemical structures]

    - Processes:
      - Absorption
      - Metabolism
      - Storage
      - Excretion

    - Functions:
      - Vision
      - Cell growth and development
      - Immune system function

    - Deficiency:
      - Night blindness
      - Skin dryness
      - Increased risk of infections

    - Rickets and Osteomalacia:
      - Deficiency of vitamin D
      - Calcium and phosphate absorption

    - Sources:
      - Animal liver
      - Yellow and orange vegetables
      - Milk and milk products

    - Recommended Dietary Allowance (RDA):
      - 800-2000 IU for adults

    - Intake:
      - 800-2000 IU daily

    - Excess:
      - Increased risk of liver damage

    - Toxicity:
      - Vitamin A toxicity can occur from excessive intake

    - Importance:
      - Essential for maintaining good health

    - Monitoring:
      - Serum retinol levels

    - Vitamin A Poisoning:
      - Acute vitamin A intoxication

    - Prevention:
      - Balanced diet

    - Intervention:
      - Vitamin A supplementation

    - Support:
      - Community-based vitamin A distribution programs

    - Conclusion:
      - Vitamin A is crucial for good health and development

Keratomalacia
And add Zinc Deficiency to this trio.

Best understood biochemical role for Vitamin K isomers:

1. Cofactor for carboxylation of glutamyl sidechains in prothrombin and other proteins in the blood coagulation cascade, this favors prothrombin binding to Ca²⁺ and membrane, and its conversion to thrombin.

2. Vitamin K also has similar glutamyl carboxylation roles in bone, kidney and muscle processes.

3. Clinically important oral anticoagulants (coumarins), discovered when sheep began dying after eating spoiled hay, and structurally similar warfarin (rat poison), block the vitamin K reductase cycle.
**a. Manifestations of Vitamin K deficiency or excess**

1. Basically, Vit. K deficiency results in hemorrhage

2. Although Vit. K is widespread in diets, deficiencies occur during antibiotic therapy (kills intestinal bacterial source) and fat malabsorption syndromes

3. Vit. K status is (usually) determined before surgeries to avoid excessive bleeding

4. Newborns (esp. preemies), prone to hemorrhage because they lack Vit. K stores and breast milk is a limited source, are routinely treated with 1 mg Vit. K until their bacteria rev up

5. Because it is readily excreted, Vit. K is relatively nontoxic

---

**D. WATER-SOLUBLE "ENERGY-RELEASING" VITAMINS**

1. **VITAMIN B (Thiamine), a key dehydrogenation co-factor in energy metabolism**

   a. Thiamine, generally as its pyrophosphate (TPP), is needed for key dehydrogenation reactions in glycolysis and TCA cycle, pyruvate dehydrogenase and malic-dehydrogenase, and in pyruvate phosphate shuttle, transketolase (REK transketolase activity is one clinical measure of thiamine status).

   b. Fortified in many commercial breads and cereals; also in grains, seeds (sunflower), green vegetables, nuts, organ meats, pork and milk products, etc.

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A newborn suffering from brain hemorrhaging due to Vitamin K deficiency (Vietnam). (Photo courtesy of Project Vietnam)
15

**Summary of biochemical reactions** involving thiamine pyrophosphate

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**Hyperhomocysteinemia: Importance of vitamin B₁₂ in convergence with folate and vitamin B₆**

1) Elevated plasma homocysteine is an independent risk factor for cardiovascular disease & stroke—So how do the vitamins converge?

2) Homocysteine promotes arteriosclerosis by as yet unclarified mechanisms. Recent evidences also link hyperhomocysteinemia to Alzheimer’s dementia, pregnancy complications, inflammatory bowel disease, and increased risk of osteoporotic fractures.

3) A common polymorphism sometimes involved: greatly increased homocysteine and increased risk of stroke in some individuals deficient in activity of 5-MeTHF-forming enzyme (5,10-MTHF reductase)

4) Animal and human studies confirm that supplementation with vitamin B₁₂, folate and vitamin B₆ can reduce elevated homocysteine by promoting its metabolism

5) Lowering an elevation in plasma homocysteine by 1 umole/L results in a 10% reduction in the risk of cardiovascular disease (JAMA 1995)

---

**Niacin (B₃): Deficiencies or problems of excess**

- Severe niacin deficiency leading to Pellagra, characterized by the 3 D’s (dermatitis, diarrhea, and dementia), and often the 4th big one, is now rare in US because of public health measures (food fortification), but still a common test question

- Moderate niacin deficiency, leading to anorexia, muscle weakness, mucus membrane lesions and burning sensations, occurs frequently in elderly populations and alcoholics

- Large nicotinic acid doses can lower plasma lipids, but side effects can include flushing, hyperglycemia, and reversible liver dysfunction
Co-factor functions of Vit. B₆:

1. Essential in amino acid decarboxylations, transaminations, deaminations, and aminolevulinic acid (ALA) formation
2. Stabilizes glycogen phosphorylase
3. Important in regulating homocysteine levels (detailed later)

The Lack of which vitamin is involved with these?

Polat can be found in all these foods

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---
FOLIC ACID (Folacin), one of two hemopoietic vitamins

![Folic Acid Structure]

1. **Nutritional and biochemical aspects of folic acid:**

   Folic acid utilized in the cell for one-carbon pool as tetrahydrofolate (THF), formed by THF reductase and then metabolically interconverted to important derivatives as shown in the following diagram. N5-Methyl-THF is main circulating form and is very important in limiting homocysteine accumulation (more later).

Folate: enters one carbon pool as tetrahydrofolate (H4folate or THF)

![Folate Metabolism Diagram]

Folic acid (folate) deficiency:

1. Provokes hemolytic megaloblastic anemia
   a) due to decreased synthesis of purines and deoxy-thymidyl acid (dTMP), which then blocks erythrocyte maturation and results in large (“macrocytic”), easily hemolyzed RBCs
   b) underlies anemia in >40% of hospitalized alcoholics, but not only due to low dietary folate, but also to alcohol’s inhibition of folate absorption, and (aggravated by liver disease) impaired folate activation, storage

2. Is a major reason for neural tube defects—e.g., spina bifida and anencephaly—so folate supplementation throughout pregnancy, with its already decreased folate absorption, is very important (fetal development, with rapid cell division, needs folate)

3. Is associated with increased risk of cancer of the colon, possibly through chromosome breaks, and with childhood neuroblastoma

4. May increase the risk of ischemic stroke, since new studies show that folate intake (and also vit. B12 intake) is associated with a reduction in this risk

5. Is linked along with vit. B6 and vit. B12 to hyperhomocysteinemia
Vitamin B₁₂ (Cobalamin, the mini-micronutrient)

Structure:
- Unique "corrin" ring and cobalt (its only biological role), co-ordinated with anionic groups.

B₁₂ in methionine synthesis, THF regeneration, and methylmalonyl CoA metabolism:

- B₁₂ participates in two main biochemical reactions (1) the synthesis of methionine, which requires homocysteine and regenerates THF (red frame).
- (2) the metabolism of methylmalonyl CoA.

Vitamin B₁₂ deficiency results in two problems that are consistent with these reactions:

- Peripheral neurological deficits can be caused by neurological components, which is actually the acid deficiency due to B₁₂ requirement in THF regeneration. Can be treated (treated) with folic acid, but:
- More serious neurological deficits arise from progressive brain demyelination, perhaps because 3-methylmalonyl CoA blocks myelin fatty acid turnover. Sig reduced by folic acid.

Biological Methylation Reactions and Homocysteine Metabolism
Diseases with underlying micronutrient deficiencies

**BERI-BERI and WERNICKE'S (Wernicke-Korsakoff: alcoholics)**
Thiamine (B1) deficiency, affecting pyruvate dehydrogenase

**PELLAGRA (also alcoholics)**
Niacin (B3) deficiency, affecting many NAD- and NADH-dependent enzymes

**SCURVY (sometimes alcoholics)**
Vitamin C (ascorbic acid) deficiency, affecting proline and lysyl hydroxylases, among others

**NYCTALOPIA (nightblindness), XEROPHTHALMIA (keratinosis and "dry eye"), and KERATOMALACIA (permanent blindness)**
Progressive retinal damage due to Vitamin A deficiency in underdeveloped world

**RICKETS (childhood osteomalacia)**
Deficiency in biosynthesis (lack of sunlight) and/or intake of Vit.D

**TWO VITAMIN CO-FACTORS FOR DECARBOXYLATIONS**
Thiamine (B1) in pyruvate dehydrogenase, alpha-ketoglutarate dehydrogenase, and branched chain ketoacid dehydrogenase (all α-keto acid decarboxylations)

Pyridoxal Phosphate (B6) in DOPA-, histidine-, glutamate-, cysteine sulfinate- and phosphatidyl serine-decarboxylases

**TWO VITAMIN CO-FACTORS FOR CARBOXYLATIONS**
Vitamin K in vit. K-dependent carboxylases in blood-clotting cascade and bone

Biotin in four different ATP-dependent carboxylases (pyruvate carboxylase is a recognizable one from gluconeogenesis)

**NUTRIENT DEFICIENCIES THAT CAN LEAD TO ANEMIAS**

**Iron - microcytic hypochromic anemia**

**Zinc and Copper - basically iron-deficient anemia**
Zinc is needed in protein metabolism, which is involved in copper absorption, and copper is a component in ferroxidase [ceruloplasmin], a protein required for iron absorption

**Vitamin A - also an iron-deficient anemia**
Vit. A is needed for the synthesis of transferrin

**Riboflavin (B2) - rare deficiencies can promote anemia**
Possibly due to impairment of iron metabolism and hemoglobin synthesis

**Folate - hemolytic megaloblastic anemia**

**Vitamin B12 - same as folate**
B12 is required to regenerate active tetrahydrofolate for one-carbon pool metabolism and DNA synthesis

**Vitamin E - severe deficiency (very rare) can precipitate hemolysis and a resultant normocytic anemia**
Summary of dietary antioxidant defenses: nutrients making significant contributions

- Vitamin E in membranes and lipid phases
- Vitamin C in aqueous phases of compartments
- Vitamin A and carotenoids, particularly lycopene
- Selenium, often referred to as a component of glutathione peroxidase
- Iodine and copper, often referred to as components of superoxide dismutase
- Many plant phenolic derivatives and phytochemicals, particularly flavonoids

Note: Data is from the National Academy of Medicine. Although many lab experiments indicate the importance of dietary antioxidants, evidence from human studies that supplements usually increase mortality from chronic diseases is still very limited.
Nutrition in biological and physiological function– I
Lecture 83: Monday April 11th, 8:30-9:30 am

Learning Objectives:

- Understand the relation of essential amino acid content to the biological value of protein, and how combining "deficient" proteins is nutritionally acceptable.
- Explain positive and negative nitrogen balance and conditions under which they occur.
- Define marasmus and kwashiorkor as components of PEM, and the effects of coexisting micronutrient and infections in susceptible infants and children.
- Understand the structural differences between saturated, mono-unsaturated and poly-unsaturated (PUFA) dietary fatty acids and food oils from which they are derived.
- Be familiar with the role of arachidonic acid in forming cell-specific eicosanoid messengers—particularly prostaglandins, thromboxanes and leukotrienes.
- Explain the structural difference between n-6 and n-3 PUFA and in general why they are essential in human nutrition,
- Clarify the importance of increased n-3 PUFA intake and the predominant dietary sources.
- Define glycemic index and glycemic load, and relate them to the question of low carbohydrate diets and insulin in weight loss and preventive health.

Nutrition in biological and physiological function– II
Lecture 84: Monday April 11th, 9:30-10:30 am

Learning Objectives:

- Know the effects of chronic alcohol abuse on nutrient absorption and utilization.
- Describe major health risks of chronic alcohol abuse that you will almost certainly see in your respective practices.
- Describe the nature of dietary fibers, and the nutritional benefits of their increased consumption in balanced diets.
- Understand how environmental causes such as poor diets can overlap in some instances with genetics, as exemplified by leptin, to increase obesity risk.
- Explain the dietary significance of the food pyramids for healthy nutrition, pointing out the differences between them.
- Identify key reactive oxygen and nitrogen species involved in oxidative stress, and the cellular proteins/peptides that sustain antioxidative cytoprotection.
- Be familiar with how micronutrients (retinoids, vit. E and C; copper, manganese and selenium) are important for functioning of the above antioxidant proteins.
- Understand the functions of ferritin and transferrin in cellular iron regulation, and the role of iron in cellular energy metabolism.
- Describe potential deleterious effects of excess iron absorption.
Learning Objectives:

- Explain the relationship of dietary carotenoids to retinoids, and the roles of retinoids in normal physiology.
- Describe the two forms of vitamin K, and the specific role of vit. K in blood clotting.
- Identify the specific functions of thiamine in energy metabolism.
- Describe and define the progressive outcomes of thiamine deficiency in chronic alcoholism.
- Identify pellagra's clinical signs and the nutritional deficiency that underlies it.
- Explain the relationship between folic acid and neural tube defects, describing the vitamin's role in the one carbon pool and DNA synthesis.
- Clarify the pernicious part of pernicious anemia, and the essentiality of vitamin B12 in the prevention of the disease.
- Describe the significance of hyperhomocysteinemia, and the biochemical roles of vitamins B6 and B12 and folic acid in countering the condition.
- Explain how a biochemical function of Vitamin C is critical for preventing scurvy.
- Describe the cellular antioxidant relationship between Vitamin C and Vitamin E.
- Summarize the nutritional components of a diet high in vegetables, fruits, marine fish and whole grains (and some green tea) that are important in reducing risks of heart disease, stroke and cancer.