Spectrum of public nutrition problems: Malnutrition & its Ecology

By

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The Ugly Face of “Hidden Hunger”

Iron Deficiency

Vitamin A Deficiency

Iodine Deficiency

Folic Acid Deficiency

Zinc Deficiency
# Presentation outline

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Malnutrition

• Derived from *malus* (bad) and *nutrire* (to nourish)

• Includes both
  
  ❖ **Under** nutrition (deficiency of one or more essential nutrients)

  ❖ **Over** nutrition (an excess of a nutrient or nutrients)
MALNUTRITION

“A pathological state resulting from a relative or absolute deficiency or excess of one or more essential nutrients; clinically manifested or detected only by biochemical, anthropometric or physiological tests.”
Malnutrition

The world health organization (WHO) defines malnutrition as "The cellular imbalance between supply of nutrients and the body's demand for them to ensure growth, maintenance, and specific functions".
• Malnutrition is globally the important risk factor for illness and death, contributing to more than half of deaths in children world wide.
Malnutrition is a **pathologic** state of varying **severity**; its clinical features are caused by a **deficiency or imbalance** of essential nutrients.

The cause may be **primary** (insufficient quantity or quality of food) or **secondary** (increased requirements or inadequate utilization).
Types of Malnutrition (under nutrition)

- **Under nutrition is depletion of energy (calories)** resulting from insufficient food intake over an extended period of time.
- In extreme cases under-nutrition is called *Starvation*.
- **Specific Deficiency** is the pathological state resulting from a deficiency of an individual nutrient such as vitamin A deficiency, iodine deficiency.
Types of Malnutrition
(Over nutrition)

Over nutrition:

• “Over nutrition is the pathological state resulting from the consumption of excessive quantity of food over an extended period of time”.

• Overweight and obesity are very common conditions in developed society and are becoming more common in developing societies and those in transition.
Forms of Malnutrition

1. **Undernutrition**: Marasmus
2. **Overnutrition**: Obesity, Hypervitaminoses
3. **Specific Deficiency**: Kwashiorkor, Hypovitaminoses, Mineral Deficiencies
4. **Imbalance**: Electrolyte Imbalance
• Protein energy mal-nutrition.
• Iron deficiency anemia.
• Rickets.
• Ariboflavinosis and vitamin A deficiency.
• Obesity.
TYPES OF UNDERNUTRITION

UNDERNUTRITION

ACUTE UNDERNUTRITION
- Marasmus
- Kwashiorkor
- Marasmic-kwashiorkor
- Wasting

CHRONIC UNDERNUTRITION
- Stunting
- Underweight
Public health importance

For a health problem or condition to be considered a public health issue, four criteria must be met:

1) the health condition must place a *large burden* on society, a burden that is getting larger despite existing control efforts;

2) the burden must be *distributed unfairly* (i.e., certain segments of the population are unequally affected);

3) there must be evidence that *upstream preventive strategies* could substantially reduce the burden of the condition; and

4) such *preventive strategies* are not yet in place.
Global Situation

• Globally, around *162 million*, or a quarter of the world's children, suffer from stunting,

• Around *99 million* are underweight,

• In addition, around *51 million* (8%) of the world's under-five children are wasted, with the greatest numbers are also found in Asia and in Africa.

• At the same time, around *44 million* of the world’s under-five children are overweight that is quickly establishing itself globally, affecting both poor and rich populations.
Global situation

- **2 billion** people are deficient in key vitamins & minerals
- Globally, **10%** of deaths and disability-adjusted-life-years (DALYs) among children below five years are caused by micronutrient deficiencies. **Iron** and **Vitamin A** and **zinc** deficiency represent the highest health
Figure 1: Supply of major food groups in comparison with neighbouring countries

Figure 1: Supply for human consumption (2005-2007)

- Cereals
- Fruits and vegetables
- Meat and offals
- Fish and seafood
- Milk and milk products
- Vegetable oils
- Sweeteners

Legend:
- Jordan
- Lebanon
- Syrian Arab Republic
Figure 2: Trends in DES per capita and percentages from protein, lipids & carbohydrates

Daily Energy Requirements: 2056 kcal/per capita

Source: FAOSTAT
التقزم والهزال

<table>
<thead>
<tr>
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<th>Number of children under 5 affected (000)</th>
<th>Percentage of children under 5 affected</th>
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<tbody>
<tr>
<td>Stunting^a</td>
<td>71</td>
<td></td>
</tr>
<tr>
<td>Wasting^a</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Overweight^a</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>Percentage of children under 5 affected</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wasting^a</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Severe wasting^a</td>
<td>1</td>
<td></td>
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<tr>
<td>Overweight^a</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Low birth weight^b</td>
<td>13</td>
<td>2007</td>
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PREVALENCE OF UNDER-5 STUNTING (%)

Figure 6: Trends in the prevalence of stunting among children under five years of age, according to place of residence

![Graph showing trends in prevalence of stunting among children under five years of age (urban/rural areas).]

Sources: JPFHS 1990; JPFHS 1997; JPFHS 2002; JPFHS 2009
PREVALENCE OF ADULT OVERWEIGHT AND OBESITY, 2008 (%)

- **Overweight (BMI ≥ 25)**
  - Female: 71%
  - Male: 67%
  - Both sexes: 69%

- **Obesity (BMI ≥ 30)**
  - Female: 42%
  - Male: 27%
  - Both sexes: 34%

Source: WHO 2014.
Note: BMI = body mass index.
Ecology Of Malnutrition: affecting factors and causes

Host, agent and environmental FACTORS

• Host factors: Socioeconomic, educational, morbidity,
• Agent (food) factors: food production, availability, price
• Environmental: endemicity of diseases
• Health and other related diseases: planning, implementation....
Causes of Malnutrition

• **Indirect causes:**
  - low socioeconomic standard of living
    - Defective sanitation
    - Faulty behaviors
    - inadequate health services

• **Direct Causes:**
  = primary (exogenous) – deficient intake of nutrients (poverty, Ignorance, faulty food habits)
  = secondary (endogenous) – physiologically increased requirement (pregnancy, growth, lactation,)
    - pathologically increased requirement (fever, surgery...)
    - replace losses in parasitic infestation, diarrhea, hemorrhage)
ETIOLOGY

- Low Income, Low Purchasing Power
- Ignorance & Erroneous Food Habits
- Scarcity of Food Supply
- Overpopulation

Primary Causes
Secondary Causes

- Obesity, Insulin Resistance and Diabetes
- Diarrhea and Intestinal Malabsorption
- Hepatobiliary Disorders
- Infections especially Respiratory
- Metabolic and Renal Diseases
• **Predisposing factors:**

1) General factors:

• Low socioeconomic conditions which leads to inadequate feeding.

• Unsanitary environment with high prevalence of infectious and diarrhea disease.

• Illiteracy and unclean habits of the mother
2) Nutritional factors

- Artificially fed infants are more susceptible to malnutrition.
- Prolonged breast feeding without supplementation.
- Nutrition ignorance of the others and faulty weaning practices.
Nutritional Deficiency

Illustration of Pathogenesis

Tissue Depletion
Loss of Weight & Stunting

Deviations in Anthropometric Measurements

Biochemical Changes
Blood
Urinary Nutrients
Urinary Products

Laboratory

C Subjective Symptoms

D Classical Symptoms

Functional Changes

Anatomic Lesions

Physical Manifestations
Nutritional disorders
Classification of Undernutrition

1. **Gomez Classification**: uses weight-for-age measurements; provide grading as to prognosis

<table>
<thead>
<tr>
<th>Weight-for-Age%</th>
<th>Status</th>
</tr>
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<tbody>
<tr>
<td>91-100</td>
<td>Normal</td>
</tr>
<tr>
<td>76-90</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; degree</td>
</tr>
<tr>
<td>61-75</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; degree</td>
</tr>
<tr>
<td>&lt;60</td>
<td>3&lt;sup&gt;rd&lt;/sup&gt; degree</td>
</tr>
</tbody>
</table>
2. **Wellcome Classification**: simple since based on 2 criteria only - wt loss in terms of wt for age% & presence or absence of edema

<table>
<thead>
<tr>
<th>Wt-for-Age%</th>
<th>Edema</th>
<th>No Edema</th>
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<tbody>
<tr>
<td>80-60</td>
<td>Kwashiorkor</td>
<td>Undernutrition</td>
</tr>
<tr>
<td>&lt; 60</td>
<td>Marasmic-</td>
<td>Marasmus</td>
</tr>
<tr>
<td></td>
<td>Kwashiorkor</td>
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3. **Waterlow Classification**: adopted by WHO; can distinguish between deficits of weight-for-height% (wasting) & height-for-age% (stunting)

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Mild</th>
<th>Mod</th>
<th>Severe</th>
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<tbody>
<tr>
<td><strong>Ht-for-Age%</strong></td>
<td>&gt;95</td>
<td>90-95</td>
<td>80-90</td>
<td>&lt;80</td>
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<tr>
<td><strong>Wt-for-Ht%</strong></td>
<td>&gt;90</td>
<td>80-89</td>
<td>70-79</td>
<td>&lt;70</td>
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Protein Energy Malnutrition

Iceberg

<table>
<thead>
<tr>
<th>Nutritional marasmus</th>
<th>Kwashiorkor</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe PEM</td>
<td></td>
<td>1-5</td>
</tr>
<tr>
<td>Moderate PEM</td>
<td>Protein deficiency</td>
<td>10-25</td>
</tr>
<tr>
<td>Energy deficiency</td>
<td>Mild PEM</td>
<td>20-40</td>
</tr>
<tr>
<td>No evidence of PEM</td>
<td></td>
<td>15-50</td>
</tr>
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</table>
Marasmus

- Common in the 1st year of life

**Etiology:**

- “Balanced starvation”
- Insufficient breastmilk
- Dilute milk mixture or lack of hygiene
Marasmus

Clinical Manifestations:
1. Wasting
2. Muscle wasting
3. Growth retardation
4. Mental changes
5. No edema
6. Variable-subnormal temp, slow PR, good appetite, often w/diarrhea, etc.

Laboratory Data:
1. Serum albumin N
2. Urinary urea/ gm crea N
3. Urinary hydroxyproline/gm crea low, early
4. Serum essential a.a. index N
5. Anemia uncommon
6. Glucose tolerance curves diabetic type
7. K⁺ deficiency present
8. Serum cholesterol low
9. Diminished enzyme activity
10. Bone growth delayed
11. Liver biopsy N or atrophic
Kwashiorkor

- Between 1-3 yrs old

**Etiology:**

- Very low protein but w/calories from CHO
- In places where starchy foods are main staple
- Never exclusively dietary
Kwashiorkor

Clinical Manifestations:

A. Diagnostic Signs
   1. Edema
   2. Muscle wasting
   3. Psychomotor changes

B. Common Signs
   1. Hair changes
   2. Diffuse depigmentation of skin
   3. Moonface
   4. Anemia

C. Occasional Signs:
   1. Flaky-paint rash
   2. Noma
   3. Hepatomegaly
   4. Associated

Laboratory:
   1. Decreased serum albumin
   2. EEG abnormalities
   3. Iron & folic acid deficiencies
   4. Liver biopsy fatty or fibrosis may occur
Kwashiorkor
Treatment of PEM

1. Severe PEM is an emergency, hospitalization 1-3 mo desirable
2. On admission, treat vitamin deficiencies, dehydration & associated infections
3. In the acute phase, feeding started as soon as rehydrated & when edema is lost, full-strength feeds given with maintenance calories & protein; recovery after 2-3 wks
4. Rehabilitation with high energy feeds (150-200 kcal/kg/day) started once full-strength feeds tolerated; recovery expected within 4-6 wks on high energy feeds
Prognosis of PEM

✓ Permanent impairment of physical & mental growth if severe & occurs early especially before 6 months old
✓ First 48 hours critical, with poor treatment mortality may exceed 50%
✓ Even with thorough treatment, 10% mortality may still occur
✓ Some mortality causes are endocrine, cardiac or liver failure, electrolyte imbalance, hypoglycemia & hypothermia
Obesity

Donald size him.

Childhood obesity is a growing epidemic that increases death and disability from heart disease. Reducing minimum standards for physical education, such as 150 minutes per week of physical education for elementary schools and 225 minutes per middle school, gives children a fighting chance against obesity and heart disease. And, coordinated school health programs will ensure that children have sound minds and healthy bodies. You can prevent overfed children who suffer more health problems and grow into unhealthy, less productive and disabled adults. Don’t miss your chance to shape a whole new generation of Americans and stop the nation’s No. 1 killer—heart disease.

Heart disease. You’re the Cure.
1. **Definition:** Generalized, excessive accumulation of fat in subcutaneous & other tissues

2. **Classification according to “desirable” weight standard:** Overweight ~ >10% while Obese ~ >20%

3. The Centers for Disease Control (CDC) avoids using "obesity" instead suggest two levels of overweight: 85th percentile of BMI ~ "at risk" level & 95th percentile of BMI ~ the more severe level

4. The American Obesity Association uses: The 85th percentile of BMI for overweight because ~ BMI of 25, overweight for adults and the 95th percentile of BMI for obesity because ~ BMI of 30, the marker for obesity in adults
Obesity

- Appears most frequently in the 1st year, 5-6 years & adolescence

Etiology:
- Excessive intake of food compared w/ utilization
- Genetic constitution
- Psychic disturbance
- Endocrine & metabolic disturbances rare
- Insufficient exercise or lack of activity
Obesity

Clinical Manifestations:

1. Fine facial features on a heavy-looking taller child
2. Larger upper arms & thighs
3. Genu valgum common
4. Relatively small hands & fingers tapering
5. Adiposity in mammary regions
6. Pendulous abdomen w/ striae
7. In boys, external genitalia appear small though actually average in size
8. In girls, external genitalia normal & menarche not delayed
9. Psychologic disturbances common
10. Bone age advanced
Treatment of Obesity

A. 1\textsuperscript{st} principle: decrease energy intake
   1. Initial med exam to R/O pathological causes
   2. 3-day food recall to itemize child’s diet
   3. Plan the right diet
      a. Avoid all sweets, fried foods & fats
      b. Limit milk intake to not >2 glasses/day
      c. For 10-14 yrs, limit to 1,100-1300 cal diet for several months
   4. Child must be properly motivated & family involvement essential

B. 2\textsuperscript{nd} principle: increase energy output
   1. Obtain an activity history
   2. Increase physical activity
   3. Involve in hobbies to prevent boredom
Complication of Obesity

Pickwickian Syndrome

✓ Rare complication of extreme exogenous obesity
✓ Severe cardiorespiratory distress & alveolar hypoventilation
✓ Includes polycythemia, hypoxemia, cyanosis, CHF & somnolence
✓ High $O_2$ conc dangerous in cyanosis
✓ Weight reduction ASAP & quick
The Energy-Releasing Vitamins
Thiamine, Riboflavin, Niacin, Pyridoxine are cofactors to enzymes in energy metabolism, hence, deficiencies show up in quickly growing tissues such as epithelium.

Typical symptoms for the group include:

- Dermatitis
- Glossitis
- Cheilitis
- Diarrhea

Nerve cells use lots of energy, so symptoms also show up in the nervous tissue:

- Peripheral neuropathy
- Depression
- Mental confusion
- Lack of motor coordination
- Malaise
Thiamine (Vitamin B1) Deficiency
Beriberi

Pathology:

Biochemically, there is accumulation of pyruvic and lactic acid in body fluids causing:

1. Cardiac dysfunction such as cardiac enlargement esp right side, edema of interstitial tissue & fatty degeneration of myocardium
2. Degeneration of myelin & of axon cylinders resulting in peripheral neuropathy and
3. In chronic deficiency states, vascular dilatation & brain hemorrhages of Wernicke’s Disease, resulting in weakness of eye movement, ataxia of gait and mental disturbance
Thiamine Deficiency (Beriberi)

Three forms:

1. **Wet beriberi**: generalized edema, acute cardiac symptoms and prompt response to thiamine administration

2. **Dry beriberi**: edema not present, condition similar to peripheral neuritis w/ neurological disorders present

3. **Infantile beriberi** divided into:
   a. **Acute cardiac** - ages 2-4 months; sudden onset of cardiac s/sx such as cyanosis, dyspnea, systolic murmur & pulmonary edema w/ rales
   b. **Aphonic** - ages 5-7 months; insidious onset of hoarseness, dysphonia or aphonia
   c. **Pseudomeningeal** - ages 8-10 months; signs of meningeal irritation w/ apathy, drowsiness & even unconsciousness; occurs more often
Thiamine Deficiency (Beriberi)

Diagnosis:
1. Clinical manifestations not conclusive
2. Therapeutic test w/ parenteral thiamine = dramatic improvement
3. Blood lactic & pyruvic acid levels elevated after oral load of glucose
4. Decreased red cell hemolysate transketolase

RDA:
- Infants 0.4mg
- Older children 0.6-1.2mg
- Nursing mothers 1.5mg
- Adults 1-1.3mg

Prevention:
1. Richest sources are pork, whole grain, enriched cereal grains and legumes
2. Improved milling of rice conserve thiamine
3. Excessive cooking of vegetables or polishing of cereals destroy
4. In breast-fed infants, prevention achieved by maternal diet w/ sufficient amounts

Treatment:
1. Children: 10mg p. o. daily for several weeks
2. Adults 50mg
Thiamine Deficiency (Beriberi)
Thiamine Deficiency (Beriberi)
Riboflavin (Vitamin B2) Deficiency

Functions:
1. Acts as coenzyme of flavoprotein important in a. a., f. a. & CHO metabolism & cellular respiration
2. Needed also by retinal eye pigments for light adaptation

Clinical Manifestations:
1. Characteristic lesions of the lips, the most common of which are angular stomatitis and cheilosis
2. Localized seborrheic dermatitis of the face may result such as nasolabial seborrhea or dyssebacia and angular palpebritis
3. Scrotal or vulvar dermatosis may also occur
4. Ocular s/sx are photophobia, blurred vision, itching of the eyes, lacrimation & corneal vascularization
Riboflavin Deficiency

Diagnosis:
1. Urinary riboflavin determination
2. RBC riboflavin load test

RDA: Infants & children <10yrs 0.6-1.4mg
Children >10yrs 1.4-2mg depending on food intake
Adults 0.025mg/gm dietary protein

Prevention:
1. Best sources: eggs, liver, meat, fish, milk, whole or enriched ground cereals, legumes, green leafy vegetables
2. Also present in beer
3. Impaired absorption in achlorhydria, diarrhea & vomiting

Treatment:
1. Riboflavin 2-5mg p. o. daily w/ increased B complex
2. Parenteral administration if relief not obtained
Riboflavin Deficiency
Niacin (Vitamin B3) Deficiency Pellagra

Etiology:
1. Diets low in niacin &/or tryptophan
2. Amino acid imbalance or as a result of malabsorption
3. Excessive corn consumption

Clinical Manifestations:
1. Start w/ anorexia, weakness, irritability, numbness & dizziness
2. Classical triad of dermatitis, diarrhea & dementia
3. Dermatitis may develop insidiously to sunlight or heat
   a. First appears as symmetrical erythema
   b. Followed by drying, scaling & pigmentation w/ vesicles & bullae at times
   c. Predilection for back of hands, wrists, forearms (pellagrous glove), neck (Casal’s necklace) & lower legs (pellagrous boot)
4. GIT s/sx are diarrhea, stomatitis or glossitis; feces pale, foul milky, soapy or at times steatorrheic
5. Mental changes include depression, irritability, disorientation, insomnia & delirium
Niacin Deficiency (Pellagra)

**Diagnosis:**
1. History & manifestations of diet poor in niacin or tryptophan
2. In niacin deficiency, urinary levels of N-methyl-nicotinamide low or absent

**Differential diagnoses:** Kwashiorkor, Infantile Eczema, Combination deficiencies of amino acids & trace minerals such as zinc

**RDA:**
- Infants & children <10yrs 6-10mg
- Older individuals 10-20mg

**Prevention:**
1. Rich sources include meat, peanuts and legumes, whole grain and enriched breads and cereals
2. Avoid too large a proportion of corn

**Treatment:**
1. Niacin 50-300mg daily which may be taken for a long time
2. Skin lesions may be covered w/ soothing lotions
Niacin Deficiency (Pellagra)
Pyridoxine (Vitamin B6) Deficiency

Functions:
1. Vitamin B6 is involved in the synthesis and catabolism of amino acids, synthesis of neurotransmitters, porphyrins and niacin
2. Plays important role in clinical conditions such as anemia, hyperemesis gravidarum, cardiac decompenasion, radiation effects, skin grafting, INH therapy & seborrheic dermatitis

Etiology:
1. Losses from refining, processing, cooking & storing
2. Malabsorptive diseases such as celiac disease may contribute
3. Direct antagonism might occur between INH & pyridoxal phosphate at the apoenzyme level
Pyridoxine Deficiency

Clinical Manifestations:

1. Three different types
   a. Neuropathic, due to insufficient neurotransmitter synthesis, such as irritability, depression & somnolence
   b. Pellagrous, due to low endogenous niacin synthesis, such as seborrheic dermatitis, intertrigo, angular stomatitis & glossitis
   c. Anemic, due to low porphyrin synthesis, such as microcytic anemia & lymphopenia

2. In genetic diseases involving pyridoxal phosphate enzymes also xanthurenic aciduria, cystathioninuria & homocystinuria
Pyridoxine Deficiency

**Diagnosis:** As screening test, tryptophan load test done - 100mg/kg BW tryptophan will give large amount of xanthurenic acid in urine

**Prevention:**
1. Firm requirement not established but usually recommended: Infant 0.1-0.5mg, Child 0.5-1.5mg & Adult 1.5-2mg
2. Rich sources include yeast, whole wheat, corn, egg yolk, liver and lean meat
3. Toxicity at extremely high doses has been described; infants whose mothers received large doses during pregnancy should be observed for seizures due to dependency
4. Children receiving INH therapy should be observed for neurologic s/sx in w/c case pyridoxine should be given

**Treatment:**
1. Pyridoxine 100mg IM injection for seizures due to deficiency
2. Children w/ pyridoxine dependency should be given 2-10mg IM injection or 10-100mg oral vitamin B6
The Hematopoietic Vitamins
Folic Acid (Vitamin B9) Deficiency

Functions:
1. Needed for RBC & DNA formation, cell multiplication esp. GI cells
2. Newly discovered functions:
   a. Prevents neural tube defects
   b. Prevents heart disease (reduces homocysteine levels)
   c. Prevents colon cancer

Etiology:
✓ Peak incidence 4-7 months
✓ Deficient dietary intake: goat’s milk deficient & powdered milk poor source
✓ Deficient absorption as in celiac disease, achlorhydria, anticonvulsant drugs, zinc deficiency & bacterial overgrowth
✓ Impaired metabolism w/ ascorbic acid deficiency, hypothyroidism, drugs like trimethoprim & alcoholism
✓ Increased requirement during rapid growth & infection
✓ Increased excretion/loss may occur subsequent to vitamin B12 deficiency & chronic alcoholism
✓ Increased destruction possible in cigarette smoking
Folic Acid Deficiency

Clinical Manifestations:
1. Megaloblastic anemia w/ irritability, failure to gain wt & chronic diarrhea
2. Thrombocytopenic hemorrhages advanced cases
3. Scurvy may be present

Laboratory Findings:
1. Anemia macrocytic
2. Serum folic acid <3ng/ml, normal level=5-20ng/ml
3. RBC folate levels indicator of chronic deficiency, normal level=150-600ng/ml
4. Serum iron & vitamin B12 normal or elevated

6. Formiminoglutamic acid in urine esp after oral histidine
7. Serum LDH markedly high
8. Bone marrow hypercellular

RDA: 20-50mcg/24 hrs

Treatment:
1. Parenteral folic acid 2-5mg/24 hrs, response in 72 hrs, therapy for 3-4 wks
2. Transfusions only when anemia severe
3. Satisfactory responses even w/ low doses of 50mcg/24 hrs, have no effect on primary vitamin B12 deficiency
4. If pernicious anemia present, prolonged use of folic acid should be avoided
Folic Acid Deficiency

Fig. 2-6  A, Blood cells in macrocytic anemia; notice the hypersegmented polymorphonuclear leukocytes.  B, Blood cells in microcytic anemia.
Cobalamine (Vitamin B12) Deficiency

**Absorption:** Vitamin B12 + glycoprotein (intrinsic factor) from parietal cells of gastric fundus → terminal ileum absorption + intrinsic factor + Ca++ → blood

**Function:** Needed in reactions affecting production of methyl groups

**Etiology:**
- **Congenital Pernicious Anemia:** Lack of secretion of intrinsic factor by stomach manifest at 9 months-10 years as uterine stores become exhausted
- Inadequate intake or dietary deficiency rare
  1. Strict vegetarian diet
  2. Not commonly seen in kwashiorkor or marasmus
  3. Breast-fed infants whose mothers had deficient diets or pernicious anemia
- Consumption or inhibition of the B12-intrinsic factor complex
- Vitamin B12 malabsorption from disease of ileal receptor sites or other intestinal causes
Cobalamine Deficiency

Clinical Manifestations:
1. Megaloblastic anemia that becomes severe
2. Neurological includes ataxia, paresthesias, hyporeflexia, Babinski responses, clonus & coma
3. Tongue smooth, red & painful

Laboratory Findings:
1. Anemia macrocytic
2. Serum vitamin B12 <100pg/ml but serum iron & folic acid normal or elevated
3. Serum LDH activity markedly increased
4. Urinary excess of methylmalonic acid, a reliable & sensitive index
Cobalamine Deficiency

5. Schilling test to assess the absorption of vitamin B12:
   a. Normal person ingests small amount of radioactive vitamin B12 → none in urine **If flushing dose injected parenterally, 1000mcg of non-radioactive vitamin B12 → 10-30% of previous radioactive vitamin B12 appears in the urine
   b. Pernicious anemia → 2% or less **If modified: 30 mg intrinsic factor administered along → normal amounts
   c. Disease of ileal receptor sites or other intestinal causes → no improvement even w/ intrinsic factor

RDA:
   Infants 0.5 mcg/day
   Older children & adults 3mcg/day

Treatment:
1. Prompt hematological response w/ parenteral vitamin B12 1-5mcg/24hrs
2. If there is neurological involvement 1mg IM daily for at least 2wks
3. Pernicious Anemia: Monthly vitamin B12 1mg IM necessary throughout patient’s life
Cobalamine Deficiency
Ascorbic Acid (Vitamin C) Deficiency
Scurvy

Functions:
1. Collagen is the major connective tissue in the body & hydroxyproline, found only in collagen, is formed from proline requiring ascorbic acid
2. If there is defective collagen formation, endochondral bone formation stops since oste, intercellular substance is no longer formed
3. Vitamin C is involved in hydroxylation reactions in the synthesis of steroids and epinephrine
4. Ascorbic acid also aids iron absorption by reducing it to ferrous state in the stomach, spares vitamin A, vitamin E and some B vitamins by protecting them from oxidation, and enhances the utilization of folic acid by aiding the conversion of folate to tetrahydrofolate

Etiology:
✓ More common 6-24 months
✓ May develop in breastfed infant if mother’s diet deficient
✓ Improper cooking practices produce significant nutrient losses & faulty dietary habits
Ascorbic Acid Deficiency (Scurvy)

Clinical Manifestations:
1. Early stages are vague symptoms of irritability, digestive disturbances & anorexia
2. Mild vitamin C deficiency signs include ecchymoses, corkscrew hairs and the formation of petechiae due to increased capillary fragility resulting from weakened collagen fibrils
3. Severe deficiency results in decreased wound healing, osteoporosis, hemorrhaging, bleeding into the skin and friable bleeding gums with loosened teeth
4. A presenting feature is an infant w/ painful, immobile legs (pseudoparalysis), edematous in “frog position” & occasionally w/ mass
5. There is depression of sternum w/ a “rosary of scorbutic beads at the costochondral junction due to subluxation of the sternal plate
6. Orbital or subdural hemorrhages, melena & hematuria may be found
7. Low grade fever & anemia usually present
8. Impairment of growth & development
Ascorbic Acid Deficiency (Scurvy)

Diagnosis:
1. History of vitamin C-deficient diet
2. Clinical picture
3. Therapeutic test
4. X-ray findings in the long bones:
   a. Most prominent & early change is simple knee atrophy
   b. Shaft trabeculae cannot be distinguished giving “ground glass appearance”
   c. Cortex reduced to “pencil-point thinness”
   d. Zone of well-calcified cartilage, white line of Fraenkel, seen as irregular & thickened white line w/c
   e. Zone of rarefaction, a linear break in bone proximal & parallel to white line under at metaphysis
   f. Calcifying subperiosteal hemorrhages cause bone to assume a dumb-bell or club shape
Ascorbic Acid Deficiency (Scurvy)

5. Laboratory tests not helpful:
   a. Ascorbic acid concentrate of buffy layer of centrifuged oxalated blood; latent scurvy gives zero level in this layer
   b. Diminished urinary excretion of vitamin C after loading

Differential Diagnosis:
1. Bleeding, swollen gums: Chronic gingivitis & pyorrhea w/ pus & respond to good dental hygiene
2. Pseudoparalysis: Syphilis negative x-ray; Poliomyelitis absent tenderness of extremities
3. Tenderness of limbs: RF age >2 yrs; Suppurative arthritis & osteomyelitis positive blood cultures
4. Bleeding manifestations: Blood dyscracias positive blood exams
5. “Rosary of scorbutic beads”: Rickets
Ascorbic Acid Deficiency (Scurvy)

Prognosis:
1. Recovery rapid w/ adequate treatment & permanent deformity rare
2. Pain ceases in a few days but swelling caused by subperiosteal hemorrhages may last several months

Prevention:
1. A minimum daily intake of 30mg is recommended by WHO for all age levels.
2. Every infant should receive supplement starting 2nd week of life.
3. Lactating mothers should have at least 50mg vitamin C daily.
4. Guava & papaya richer in vitamin C than citrus fruits, also in most green leafy vegetables, tomatoes & fresh tubers but absent in cereals, most animal products & canned milk.

Treatment:
Ascorbic acid 200-500mg daily or 100-150ml of fruit juice.
Ascorbic Acid Deficiency (Scurvy)
Ascorbic Acid Deficiency (Scurvy)
Vitamin A

✓ Active forms are retinol, retinaldehyde, and retinoic acid
✓ Plants synthesize the more complex carotenoids which are cleaved to retinol by most animals and stored in the liver as retinyl palmitate
✓ N retinol plasma values: 15-30 mcg/dl in infants & 30-90 mcg/dl in adults
Vitamin A (Retinol) Deficiency

Functions:
1. Retinal is the prosthetic group of photosensitive pigment in both rods (rhodopsin) & cones (iodopsin), major difference lies in the nature of protein bound
2. Needed in lysosomal membrane stability
3. Plays a role in keratinization, cornification, bone development & cell growth & reproduction

Etiology:
- Absence in the diet common by 2-3 yrs old
- Poor fetal storage
- Poor absorption as in low-fat diet, malabsorption syndromes, etc.
- Low protein intake resulting in deficient carriers
- Increased excretion as in cancer & UTI
Clinical Manifestations of Hypovitaminosis A

A. Eye signs & symptoms

1. An early symptom is nyctalopia or night blindness later photophobia then insensitivity to pain
2. 1\textsuperscript{st} clinical sign is xerosis conjunctivae
3. Bitot’s spots
4. Corneal xerosis or xerophthalmia
5. Corneal ulcers
6. Keratomalacia
7. Blindness
Hypovitaminosis A

B. Skin signs: xerosis of the skin & follicular hyperkeratosis or phrynoderma

C. Others: apathy, retardation of physical & mental growth, faulty epiphyseal bone formation, defective teeth enamel & signs of benign increased ICP

Diagnosis:
1. Routine PE
2. Biophysical exam, dark adaptation test to detect nyctalopia
3. Absorption test
4. Conjunctival impression cytology to evaluate early xerophthalmia
Hypovitaminosis A

**RDA:** 1800 IU/day (1 IU vitamin A = 0.3 mcg retinol)

**Prevention:**

1. Pregnant in last trimester given 5000 IU p.o.
2. Every 6 months, infants <1 yr given retinol palmitate 55mg or 33mg retinol acetate (100,000 IU) p. o.
3. Every 4-6 months, older children given 110mg retinol palmitate or 66 mg retinol acetate (200,000 IU) p. o.
4. In areas where prevalent, 100,000 IU p. o. q 3 mo
5. For malnourished children 1-6 yrs, 250,000 IU p. o. q 6 months

**Treatment:**

1. One yr of age or over:
   - 110mg retinol palmitate or 66mg retinol acetate (200,000 IU) orally or preferably
   - 33mg(100,000 IU) of water-miscible vitamin A (retinyl palmitate) by IM injection
2. The oral dose should be repeated on 2nd day and again on discharge from hospital 7-30 days after 1st dose
3. Above doses halved for infants
4. For corneal involvement, apply antibiotic ointment like topical bacitracin to both eyes 6x/day and administer also systemic antibiotics
Hypovitaminosis A
Hypervitaminosis A

Acute Intoxication:
- Results when excessively large single doses ≥300,000 IU ingested
- Infants: n/v, drowsiness or irritability w/signs of increased ICP
- Adults: drowsiness, irritability, headache & vomiting
- Serum vitamin A values = 200-1000 IU/dl (N: 50-100 IU/dl)

Chronic Intoxication:
- Results when >50,000 IU/day ingested for several wks or mos
- Signs & symptoms in infants:
  a. Early are anorexia, pruritus, irritability, tender swollen bones w/motion limitation
  b. Alopecia, seborrhea, cheilosis & peeling of palms & soles
  c. Hepatomegaly & hypercalcemia observed
  d. Craniotabes & hyperostosis of long bones (differentiate from Caffey’s disease)
- Elevated serum vit A levels confirms diagnosis
- Reversible manifestations when vitamin A discontinued
Vitamin D

- 90% as Vitamin D3, cholecalciferol, produced in the skin by UV irradiation of 7-dehydrocholesterol (predominantly animal sterol) → blood → 25 hydroxylation to calcidiol in liver + PTH → dihydroxylation in kidney to calcitriol 1,25 (OH) 2-cholecalciferol

- 1,25-dihydroxycholecalciferol is most active form of Vitamin D

- Vitamin D2, calciferol, is taken orally from plants then irradiated as above

- Animal derived vitamin D3, cholecalciferol, and vitamin D2 "activated ergosterol" are biologically equal
Vitamin D (Cholecalciferol) Deficiency

Functions: Vitamin D enhances the absorption of calcium from the gut, removal of calcium from bone and phosphate reabsorption in the kidney.

Etiology:
- Florid rickets appears toward the end of the 1st year to 2nd year of life
- Lack in the diet or lack of sunlight exposure
- Rapid growth as in prematures & adolescents
- Disorders of absorption such as celiac disease, steatorrhea or cystic fibrosis
- In children with hepatic disease
- Maternal malnutrition
- Poverty or ignorance
Clinical Manifestations of Hypovitaminosis D

A. Rickets: Deficient calcification or softening bones in a growing child resulting in deformation of bones

1. Head manifestations
   a. Craniotabes: Thinning of skull outer table detected by pressing firmly over occiput or posterior parietal bones & feeling a ping-pong ball sensation; may disappear before end of 1st yr but rickets continues resulting in flattening & at times permanent head asymmetry
   b. Anterior fontanel larger & closure delayed
   c. Caput quadratum: Box-like head due to thickened & prominent central parts of the parietal & frontal bones
   d. Eruption of temporary teeth may be delayed & permanent usually show enamel defects
Clinical Manifestations of Hypovitaminosis D

2. Thorax signs
   a. Palpable enlargement of the costochondral junctions called the “rachitic rosary”
   b. Flattened sides of the thorax with posterior longitudinal grooves
   c. Pigeon-breast deformity
   d. Harrison groove

3. Spinal column signs
   a. Scoliosis common
   b. Kyphosis when sitting
   c. Lordosis in the erect position

4. Pelvis narrowed, entrance by forward projection of the promontory & the exit by a forward displacement of the sacrum & the coccyx
Clinical Manifestations of Hypovitaminosis D

5. Extremities in children above 2 years
   a. Thickened & enlarged wrists & ankles
   b. Bowlegs or knock-knees as a result of the bending of the softened shafts of the femur, tibia & fibula
   c. Coxa vara or pronated feet
   d. Greenstick fractures

6. **Muscles** are poorly developed & lack tone
   a. Delay in sitting, standing & walking
   b. Potbelly due to weakness of abdominal muscles

7. **Other manifestations**
   a. Underweight
   b. Mental retardation
Clinical Manifestations of Hypovitaminosis D

B. **Osteomalacia**: Accumulation of uncalcified osteoid tissue in rib joints of an adult resulting in
   1. Pain in the pelvis, lower back and legs
   2. Tenderness felt in the shins and in other bones
   3. Waddling gait
   4. Deformities of the pelvis
   5. Tetany may occur manifested by involuntary twitching of the facial muscles or by carpopedal spasm
   6. Spontaneous fractures may be a feature

C. **Osteomalacia should not be confused with osteoporosis**, a disease of ageing, in which decalcification is also a feature
Hypovitaminosis D

Diagnosis:
1. History & clinical observation
2. Laboratory findings:
   a. Serum Ca may be normal or low
   b. Serum phosphorus level below 4 mg/dl (N serum phosphorus 4.5-6.5 mg/dl but in rachitic infants reduced to 1.5-3.5 mg/dl even lower)
   c. Serum alkaline phosphatase elevated (N serum phosphatase 5-15 Bodansky units per 100 ml but elevated to 20-30 in mild rickets & to 60 or more in severe cases)
   d. Serum 25-hydroxycholecalciferol decreased
   e. Urinary cyclic AMP elevated
Hypovitaminosis D

3. Roentgenographic changes
   a. X-ray of the wrist best for early diagnosis because of the cupping & fraying of the proximal ends of ulna & radius
   b. Humeral ossification centers barely visualized
   c. Shafts osteoporotic or density decreased but trabeculae unusually prominent
   d. Rosary beading of the sternal ends of the ribs due to deposited uncalcified osteoid tissue becoming compressed & bulges laterally
   e. Initial healing indicated by appearance of line of preparatory calcification.
Hypovitaminosis D

Differential Diagnosis:
1. Craniotabes in hydrocephalus & osteogenesis imperfecta
2. “Rosary” at the costochondral junctions in scurvy & chondrodystrophy
3. Epiphyseal lesions in congenital epiphyseal dysplasia, cytomegalic inclusion disease, syphilis, rubella & copper deficiency
4. Congenital pigeon breast deformity
5. Familial bowlegs
6. Metabolic disturbances with osseous lesions

Complications:
1. Respiratory infections
2. Chronic gastroenteritis
3. Iron deficiency anemia
Hypovitaminosis D

Prognosis:
1. In the tropics, usually has a tendency to heal spontaneously
2. A possibly deforming disorder
3. Not fatal but complications & intercurrent infections may cause death

RDA: 400 IU (1 IU vitamin D = 0.025 mcg cholecalciferol/ergocalciferol)

Prevention:
1. Sunlight prophylaxis effective only in temperate zones during the summer months in haze-free areas
2. Daily requirement of vitamin D is in 1 quart of fresh whole milk or a can of evaporated milk
3. Natural vitamin D is present only in animal foods like egg yolk, liver, cod-liver & other fish-liver oils, fishbody oils & drippings
Hypovitaminosis D

4. Prematures or breast-fed infants should receive supplemental vitamin D daily because milk is a poor source unless fortified

5. Vitamin D should also be administered to pregnant & lactating mothers

**Treatment:**

1. Daily administration of 50-150 mcg of vitamin D3 or 0.5-2 mcg of 1,25-dihydroxycholecalciferol will produce healing seen on X-ray within 2-4 wks

2. Vitamin D 15,000 mcg in a single dose w/o further therapy for several months may be advantageous

3. After healing is complete, the dose of vitamin D should be lowered to 10 mcg/day

4. If no healing occurs, rickets is probably resistant to vitamin D or non-nutritional rickets
Rickets

Normal anatomy vs. Rickets

Fig. 6-3 Radial thickening seen in vitamin D deficiency.
Rickets

A teenage male w/ rickets. Note bow legs & compromised height.

Distal femur, proximal tibia and fibula in rickets. Note widening epiphysis, resorption of provisional zone of calcification, flaring metaphysis & bone deformity.
Osteomalacia

A young male w/ osteomalacia. Note a pseudofracture in the medial edge of the upper femoral shaft (arrow).

Xray showing a pseudofracture (red arrow) from an adult who has x-linked hypophosphatemic rickets. This sign is seen only in osteomalacia, but not in many of the cases.

AP pelvis in a patient w/ osteomalacia. The film shows diffuse osteopenia, & a Looser zone (arrow) in the superior ramus of the right obturator ring.
Osteomalacia

Fig. 9a Healed osteomalacia. Pelvis of a Chinese woman, showing deformity resulting from decalcification that began during pregnancy eight years before. At the height of the disease, the bones showed extensive rarefaction. This photograph was taken after recalcification had occurred as a result of three years of vigorous antirachitic treatment, and nine months after the birth of a living child by caesarean section.

Fig. 9b Normal pelvis of an American woman of about the same age.
Osteomalacia

A and B: Modified Mason stain; magnification x130. Note in A: broad osteoid seams (arrow), osteoid trabeculae (heavy arrow) and irregular mineralization front (rectangular arrow).

C and D: Polarized light; Von Kossa toluidine blue stain; magnification x360. Note in C: increased number of osteoid lamellae (arrows).

E and F: Fluorescent photomicrograph, unstained; magnification x200. Note in E wide fluorescent bands (arrows), no double or single tetracycline labels and ground glass appearance.
Hypervitaminosis D

Etiology:
- Excessive intakes from
  - Inadvertently substituting concentrated form for dilute
  - Parents’ increasing prescribed dose
  - Inadequately controlling dosages for children receiving large amounts of vitamin D for chronic hyperphosphatemic states

Clinical Manifestations:
- Symptoms after 1-3 months
  1. Hypotonia, anorexia, irritability, constipation, polydipsia, polyuria & pallor
  2. Dehydration usually present
  3. Aortic valvular stenosis, vomiting, hypertension, retinopathy & clouding of cornea & conjunctiva may occur
Hypervitaminosis D

**Laboratory Data:**
1. Proteinuria
2. Hypercalcemia & hypercalciuria
3. With continued excess, renal damage & metastatic calcifications may occur
4. Roentgenograms of the long bones reveal metastatic calcification & generalized osteoporosis

**Differential Diagnosis:**
1. Chronic nephritis
2. Hyperparathyroidism
3. Idiopathic Hypercalcemia

**Treatment:**
1. Discontinue vitamin D intake & decrease Ca intake
2. For severely involved infants, aluminum hydroxide by mouth, cortisone or sodium versenate may be used
Vitamin K

- Vitamin K<sub>1</sub>, naturally occurring vitamin K, is abundant in pork, liver, soybeans & green leafy vegetables
- Intestinal microorganisms synthesize
- Required for normal clotting of blood
- Vitamin K-dependent clotting factors made in the liver: prothrombin, proconvertin (Factor VII), plasma thromboplastin component or PTC (Factor IX) & Stuart-Prower factor (Factor X)
Vitamin K Deficiency (Hypoprothrombinemia)

Etiology:

- The fetus depends on the mother for supply & at birth, the bacterial flora of the GIT not yet produce
- Exclusively breast-fed infants lower vitamin K compared to formula-fed
- Faulty intestinal absorption as in diarrhea, celiac disease, gastrointestinal malformation & steatorrhea
- Obstructive jaundice, biliary fistula, insufficient production of bile acids or pancreatic insufficiency lead to inadequate intestinal absorption
- Administration of antibiotics which inhibit intestinal bacteria
- In sepsis, deficiency results from disease affecting hepatobiliary functions & therapy
- Drugs like Coumarin, Salicylates & anticonvulsants
Vitamin K Deficiency (Hypoprothrombinemia)

Clinical Manifestations:
1. Hemorrhagic manifestations are the hallmark
2. Bleeding in the newborn from the cord or circumcision site
3. GIT bleeding, hematuria & intracranial hemorrhage more serious
4. Anemia & shock may ensue from severe blood loss

Laboratory Test: The most useful test is the 1-stage prothrombin time test (Quick), prolongation indicates presumptive evidence deficiency
Vitamin K Deficiency (Hypoprothrombinemia)

Prevention and Treatment:

✓ 4 requirements to prevent & control a potentially fatal hemorrhagic state:
   1. Bile of normal composition in the GIT
   2. Adequate diet
   3. Normal absorptive surface in the small intestines
   4. Functioning liver capable of synthesizing

✓ In the newborn, vitamin K₁ is being used because:
   1. Greater margin of safety
   2. Acts more rapidly with therapeutic levels within 2-4 hours
Vitamin K Deficiency (Hypoprothrombinemia)

Prevention and Treatment:

✓ The AAP Committee on Nutrition recommends:

1. Prophylactic dose~ 0.5-1 mg Vitamin K as single parenteral dose or 1-2 mg single p.o. dose
2. Mild prothrombin deficiency~ 1-2 mg p.o. daily
3. In severe cases with hemorrhages~
   - Vitamin K₁ 5 mg daily parenterally
   - Whole blood if due to liver damage

✓ Avoid excessive doses in prematures & G-6-PD deficient newborns - known hemolytic action & tendency to hyperbilirubinemia

✓ Vitamin K prophylaxis to woman in labor may be followed by hemolytic anemia, hyperbilirubinemia, kernicterus & death in the infant
Vitamin K Deficiency
(Hypoprothrombinemia)
Tocopherol (Vitamin E) Deficiency

Etiology:

- Malabsorptive states such as cystic fibrosis & acanthocytosis
- Diets high in unsaturated f.a. increase requirements in prematures who absorb vitamin E poorly
- Excess iron administration exaggerates signs of deficiency

Manifestations:

1. Some have creatinuria, ceroid deposition in smooth muscle, focal necrosis of striated muscle & muscle weakness
2. Prematures may develop hemolytic anemia at 6-10 wks of age
3. Increase risk of retrolental fibroplasia in prematures
4. Degenerative neurologic syndrome when due to biliary atresia
5. Increased platelet adheresiveness
6. Anemia in kwashiorkor
Tocopherol (Vitamin E) Deficiency

Prevention & Treatment:

1. RDA not known but 0.7mg/g of unsaturated fat in the diet adequate
2. Premature infants may be given 15-25 IU/24 hrs
3. Large oral or parenteral doses may prevent permanent neurologic abnormalities in biliary atresia or abetalipoproteinemia
Minerals

- Minerals are inorganic elements or compounds that play important roles in metabolic reactions and serve as structural components in body tissues such as bones.
- Minerals cannot be manufactured in the body, they have to be provided with food.
Mineral Classifications

- Macrominerals
- Microminerals
- Ultratrace minerals
Bioavailability of Minerals

• Reduced by formation of soaps, binding to free fatty acids, precipitation, mineral-mineral interactions, organic inhibitors such as phytates and oxalates, stress

• Enhanced by ascorbic acid (for nonheme iron), gastric acidity, hemostatic adaptations
Calcium Metabolism

- Intestinal absorption
- Renal excretion
- Skin losses
- Serum calcium
- Blood calcium and parathyroid hormone
- Role of other hormones
Functions of Calcium

• Acquire optimal bone mass and density
• Maintain bone health
• Transport functions of cell membranes
• Nerve transmission
• Regulation of heart muscle function
• Blood clotting
• Role in obesity
Sources of Calcium

- Yogurt
- Milk
- Cheese
- Small bones of sardines and canned salmon
- Dark green leafy vegetables
- Molasses
- Clams and oysters
- Fortified foods
Calcium Deficiency

• Lower peak bone mass
• Increased blood PTH levels leads to low bone mass
• Osteomalacia
• Chronic diseases, such as colon cancer, hypertension, osteoporosis
Phosphorus

- Absorption, transport, storage, and excretion
- DNA and RNA are phosphate based
- High-energy phosphate bonds in ATP and other cellular forms of energy
- Phospholipids
- Enzyme activation and buffer system
- Hydroxyapatite in bones and teeth
Sources of Phosphorus

- Meat, poultry, fish, and eggs
- Milk and milk products
- Nuts and legumes

- Cereals and grains
- Food additives
Magnesium

• Absorption, transport, storage, and excretion
• Cofactor for more than 300 enzymes
• Neuromuscular transmission and activity
• Bone density
Sources of Magnesium

- Milk
- Bread
- Coffee
- Ready-to-eat cereals
- Beef
- Potatoes
- Dried beans and lentils
Sulfur

• Constituent of three amino acids: cystine, cysteine, and methionine
• May be considered an antioxidant
• Essential component of three vitamins: thiamin, biotin, and pantothenic acid
• Food sources include meat, poultry, fish, eggs, dried beans, broccoli, and cauliflower
Microminerals (Trace Elements)

• Essential to optimal growth, health, and development

• Exist in two forms
  – As charged ions
  – Bound to proteins or complexed in molecules
Iron

- Heme vs nonheme iron
- Ferritin carries bound iron
- Ferric vs ferrous iron
- Role of transferrin
- Factors affecting bioavailability
Functions of Iron

• Red blood cell function
• Myoglobin activity
• Numerous heme and nonheme enzymes
• Oxidation-reduction activity in respiratory gas transport and cytochrome activity
• Immune function and cognitive performance
Sources of Iron

- Liver
- Seafood
- Kidney, heart
- Lean meat, poultry
- Dried beans and vegetables
- Egg yolks
- Dried fruits
- Dark molasses
- Whole grain and enriched breads and cereals
Iron Deficiency

• High-risk groups
• Hypochromic, microcytic anemia
• Caused by injury, hemorrhage, or illness, unbalanced diet
• Athletic amenorrhea
Iron Overload

• Major cause is hemochromatosis
• Hemosiderosis may lead to hemochromatosis
• Risks of iron supplements for older adults
Zinc

• Functions in association with more than 300 enzymes
• Synthesis or degradation of major metabolites
• Structural roles in proteins
• Intracellular signal in brain cells
• Transport processes, immune function, and genetic expression
Sources of Zinc

• Met, fish, poultry
• Milk and milk products
• Oysters and other shellfish
• Liver

• Ready-to-eat fortified breakfast cereals
• Whole grain cereals
• Beans, nuts, soy products
Zinc Deficiency

- Decreased taste acuity
- Delayed wound healing
- Growth retardation
- Immune deficiencies
- Skin lesions
Fluoride

• Beneficial to tooth enamel, prevention of dental caries
• Found in fluoridated drinking water, fluoridated toothpaste, foods and drinks made using fluoridated water
• Toxicity, fluorosis appears at daily doses of 0.1 mg/kg
Copper

- Component of many enzymes
- Ceruloplasmin
- Roles in mitochondrial energy production
Sources of Copper

- Shellfish
- Organ meats
- Muscle meats
- Chocolate
- Nuts
- Cereal grains
- Dried legumes
- Dried fruits
Iodine

• Synthesis of triiodothyronine ($T_3$) and thyroxine ($T_4$)
• Inhibited by goitrogens
• Selenium is important in iodine metabolism
Sources of Iodine

• Iodized salt
• Seafood
• Content of cow’s milk and eggs depends on the animal’s diet
• Content of vegetables depends on soil
• Iodophors used in food processing
Iodine deficiency

- 1. Endemic goiter
- Cretinism
- Hypothyroidism
- Hyperthyroidism
Selenium

• Component of enzyme glutathione peroxidase (GSH-Px)
• Antioxidant and free radical scavenger
• Role in iodine metabolism
• Interaction with vitamin E
Sources of Selenium

- Brazil nuts
- Seafood
- Kidney, liver
- Meat, poultry
Selenium deficiency

• Keshan disease: heart muscle failure and Cardiomyopathy.
Manganese

• Component of many enzymes, especially in mitochondria
• Activates many other enzymes
• Formation of connective and skeletal tissues
• Growth and reproduction
• Carbohydrate and lipid metabolism
Chromium

• Potentiates insulin action
• Possible glucose tolerance factor
• Possible role in regulation of gene expression
Molybdenum

• Role in enzymes that catalyze oxidation-reduction reactions
• Possible role in response of some asthmatics to sulfites
• Deficiency causes mental changes and abnormalities of sulfur and purine metabolism
Boron

• Essentiality not yet established
• Influences activity of metabolic enzymes and metabolism of several nutrients
• Associated with cell membranes
Cobalt

- Component of vitamin $\text{B}_{12}$ (cobalamin)
- Required for enzyme methionine aminopeptidase
- Macrocytic anemia
Focal Points

• The major nutrients with roles in the human body include energy-containing macronutrients (carbohydrates, lipids, protein and alcohol as well as the micronutrients (vitamins and minerals.)

• The indigestible food component, fiber is essential for health, especially related to the gastrointestinal tract and cardiovascular system, but 80% of Americans do not get enough fiber.

• Alcohol contains calories for heat but not for muscular work, and it impacts health positively in moderation and negatively in excess.

• Changing concepts regarding the structure, function, and utilization of nutrients in the body are important to keep in mind as they determine the impact of nutrient deficiencies or excesses on health and disease management.

• Miscellaneous trace elements exist in human tissues, especially in the skeleton, because of their abundance on the earth’s surface; their essentiality in humans is not totally clear.
Prevention of malnutrition:

1) General measures
   • Community and socioeconomic development.
   • Family planning services.
   • Education and culture of the public.

2) Nutritional measures:
   • Encourage breast-feeding for suitable period.
   • Proper weaning.
   • Dietary supplementation for protein and other nutrients.
   • Nutrition education of mothers
Prevention of malnutrition

3) Care for the child:
   • Immunization
   • Gross monitoring
   • Immediate management of diarrheal diseases.

4) Family planning:

5) National level: modern agriculture techniques

6) International level: FAO and WHO
The SUN approach

The multi-stakeholder platform works to align and coordinate action across sectors.

- Women's Empowerment
- Health
- Education
- Social Protection
- Agriculture
- Development & Poverty Reduction
Be master of your habits,
Or they will master you.
RELAX !!!