Iron-Deficient Anemia

- \square When iron stores are exhausted (serum ferritin <12 μ g/L),
 - > serum iron decreases and serum transferrin, which is usually measured as total iron-binding capacity (TIBC) increases.
 - If the hemoglobin concentration is still normal, this stage is called iron deficiency without anemia or latent iron deficiency.
- When the iron stores are completely exhausted, there is not sufficient iron for hemoglobin synthesis. This final stage is called iron deficiency anemia or manifest iron deficiency

Treatment of Iron-Deficient Anemia

- ☐ Iron compound used for replacement therapy must meet two requirements:
 - 1. It must be biologically available, usually water soluble, ferrous sulphate is the standard to which other iron salts are compared.
 - 2. Must be not irritant (Sustained released iron formulation have been utilized to minimize the irritant property of iron
- Parenteral iron preparations are indicated only in:
 - 1. deffect in iron absorption as in gastroctomy, steatorrhea.
 - 2. iron salt may irritate GIT so not used as in ulcerative colitis, peptic ulcer.

Treatment of Iron-Deficient Anemia

- □ Official iron product (tablet): Ferrous sulphate, Ferrous fumarate, Ferrous gluconate.
- □ Parentral: Iron dextran (Imferon), iron sorbitex (Jectofer)

Overdose of Iron

- □ Can be serious and cause death (especially in young children)
 - 1. Gastric lavage with 5% sodium bicarbonate
 - 2. Deferasirox is a novel, orally active agent that provides 24-h chelation with a once-daily dose. (effective in reducing or maintaining iron burden in adult and pediatric patients.)

Hemochromatosis

- ☐ Genetic disease
- □ the body simply loads too much iron.
- ☐ Iron deposit which can lead to organ damage (joints, organs, and eventually be fatal.)
- May go undetected until 50-60 years of age when organ fails

Copper

- An essential trace element for humans and animals.
- Redox reactions and in scavenging free radicals (the ability of copper to easily accept and donate electrons)
- □ Cuprous (Cu^{1+}) and cupric (Cu^{2+}) forms, though the majority of the body's copper is in the Cu^{2+} form.
- Energy production (the copper-dependent enzyme, cytochrome c oxidase)
- Connective tissue formation (cuproenzyme is required for the cross-linking of collagen and elastin)

Copper in Iron Metabolism

- Ferroxidases (Ceruloplasmin Cp), oxidize ferrous iron (Fe²⁺) to ferric iron (Fe³⁺), the form of iron that can be loaded onto the protein transferrin (RBC formation)
- Mice (Cp^{-/-}) have normal copper metabolism but abnormal iron accumulation in the liver.
- Cp is essential to the flux of iron in the body.
- ☐ In copper deficiency, iron mobilization from storage sites is impaired.

Copper Absorption

- dependent on body's needs
- decreased with high intakes of zinc, iron.
- ☐ Adequate amount of copper/copper supplements
- Wilson disease is a genetic disorder of copper metabolism that is characterized by excessive deposition of copper in the liver, brain, cornea and other tissues. Wilson disease is often fatal if not recognized and treated when symptomatic.

Iodine

- An essential ion, Key component of thyroid hormones, which are required throughout life for normal growth, neurological development, and metabolism.
- Insufficient iodine intake impairs the production of thyroid hormones → hypothyroidism.
 - thyroid gland enlargement (goiter),
 - severe physical and mental retardation known as cretinism (congenital hypothyroidism)

Iodine

- Most of the Earth's iodine, in the form of the iodide ion (I⁻).
- □ Iodine deficiency can be avoided by the use of iodized table salts containing (0.01% KI).
- Seafood is an excellent source of dietary iodine.
- Dairy products, grains, eggs, and poultry contribute substantially to dietary iodine intakes.
- □ Concurrent deficiencies in selenium, iron, or vitamin A may exacerbate the effects of iodine deficiency.

Iodine

- □ Iodism: caused by excessive administration of iodide which brings about certain irritative phenomena to the skin and mucous membrane exhibited by rashes, headache, laryngitis.
- Sodium chloride may be administered to aid in the more rapid elimination of iodide

Iodine uses

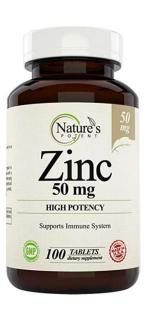
- Key component of thyroid hormones, which are required throughout life for normal growth, neurological development, and metabolism.
- Hypothyroidism.
- ☐ Hyperthyroidism, decrease vascularity of thyroid gland
- As expectorant (KI) (in Asthma, chronic bronchitis)

Iodine products

- \square I₂: very slightly soluble in water
 - Lugal's solution (strong I_2 solution, $I_2 + KI$?)
 - Iodine tincture. (Iodine in alcohol)
- □ KI solution: (hyperthyroidism, antifungal, expectorant)
 - Sodium thiosulfate added to KI solution?
- NaI: It is used as a source of iodine mostly for hyperthyroid conditions, and to solubilize iodine in Iodine Tincture and Iodine Solution.

Zinc

- Cofactor to many enzymes
- Important roles in growth and development, the immune response, neurological function, and reproduction.
- Absorption: decreases with calcium supplement, phytic acid
- Competes with copper and iron absorption



Zinc Deficiency

- associated with increase the susceptibility of children to infectious diarrhea, and persistent diarrhea.
- May also potentiate the effects of toxins produced by diarrhea-causing bacteria like E. coli.
- □ Zinc in combination with oral rehydration therapy → significantly reduce the duration and severity of acute and

persistent childhood diarrhea.

Selenium

Antioxidant in conjunction with Vitamin E



☐ Treatment for seborrhea dermatitis (dandruff)



