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Micronutrients

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1. Describe the concept of essential mineral elements and how their content in the body is regulated.
 - a. Required to maintain normal physiology and health.
 - b. Occur in diet, sometimes as trace elements.
 - c. Variable absorption, may be regulated
 - d. Intestinal absorption = body losses
2. Describe the factors influencing intestinal mineral absorption.
 - a. Intraluminal pH
 - b. Redox state of metals
 - c. Formation of chelates to enhance solubility.
 - d. Formation of insoluble complexes w/ other dietary components.
3. Describe the cellular mechanism of iron absorption and its regulation.
 - a. Dietary iron in heme or non-heme iron compounds.
 - b. Nonheme iron in 3+ form requires gastric acid for solubilization.
 - i. Reduced to Fe²⁺ via ferrireductase at brush border
 - ii. Absorbed by DMT1 (divalent metal transporter)
 - c. Heme Fe absorbed via receptor mediated endocytosis via HCP1
 - d. Free Fe binds to ferritin to be stored, or binds to transport protein to be secreted
 - i. Intestinal ferritin increased when iron stores are high
 - ii. Iron response proteins bind to iron response element in ferritin mRNA
 - iii. Results in iron storage in enterocytes instead of absorption
 - iv. Excess ferritin sloughs back into lumen and is excreted
 - e. Ferroportin exports Fe²⁺, Hephaestin oxidizes to Fe³⁺ basolaterally
 - f. Fe binds to transferrin in plasma to be sent to storage/utilization
 - g. Liver is storage site for excess iron
 - i. Hecpudin secreted by hepatocytes to inhibit iron absorption by gut and release from Mphages
 - ii. Hecpudin production decreased by iron deficiency; increased w/ iron loading/inflammation
 - iii. Hecpudin interacts w/ ferroportin to lead to degradation
4. Describe the consequences of iron deficiency and abnormal increased absorption.
 - a. Deficiency
 - i. Microcytic/hypochromic anemia, poor growth, impaired energy metabolism
 - ii. Caused by dietary, excess phytate/oxylate, gastric achlorhydria, hookworm infestation, excessive bleeding
 - iii. Tx. w/ iron supplements
 - b. Increased absorption (hereditary hemochromatosis)
 - i. Usually related to mutation in HFE protein --> decreased hepcidin
 - ii. Iron deposition in liver, heart, pancreas and joints
5. Have a general understanding of their function and how different classes of vitamins are absorbed by the intestine.
 - a. Water soluble --> facilitated diffusion
 - i. Generally are coenzymes, vit C is an antioxidant
 - ii. Metabolized to coenzyme form
 - b. Fat soluble absorbed like other lipids (Vit A, D, E, K)
 - i. Vitamin deficiency in cases of fat mal-absorption
6. Describe the function and absorption of folates.
 - a. Conenzymes in 1-C transfers; nucleic acid synthesis and amino acid metabolism
 - i. Deficiency --> megaloblastic anemia
 - ii. Related to neuronal tube defects --> pregnant women should consume more folates

- b. In diet, generally are polyglutamated
 - i. Hydrolyzed at brush border to monoglutamated form by folate conjugase
 - 1) Inhibited by ethanol and some drugs (dilantin, sulfasalazine)
 - 2) I think conjugase is actually bacterial
 - ii. Folate:OH exchange mechanism to absorb
 - iii. W/in enterocyte, folic acid is reduced and methylated
 - iv. Exported basolaterally as folic acid or in reduced/methylated form
7. Describe the function and dietary source of vitamins E and K.
- a. Vitamin E
 - i. Lipid soluble antioxidant in plasma membranes
 - ii. Vegetable oils, wheat germ, nuts, green leafy veggies
 - b. Vitamin K
 - i. Cofactor in post-translational modifications for proteins, esp. blood clotting factors
 - ii. K1 most abundant in green leafy veggies
 - iii. Bile salts important for absorption
 - iv. Bacterially derived K2 prevents severe deficiency
8. Describe the function, dietary source and absorption of Vitamin A and Beta-carotene.
- a. Compounds related to all-trans-retinol
 - i. Required for vision, growth, cellular differentiation, reproduction and integrity of immune system
 - ii. Deficiency --> xerophthalmia (present initially as night blindness that turns into total); also increased susceptibility to infection
 - iii. Hypervitaminosis --> increased intracranial P, skin lesions, hepatic injury b/c Vit A stored in liver
 - iv. Some carotenoids are antioxidants and other things
 - b. Retinoids in milk, carotenoids in carrots and green leafy veggies
 - c. Absorption
 - i. Retinol
 - 1) Retinyl esters in diet are de-esterified by non-specific cholesterol esterase
 - 2) Absorbed as retinol
 - 3) Esterified by ARAT (similar to ACAT) to retinyl esters to be released into lacteals as part of chylomicrons
 - ii. Beta-carotene
 - 1) Absorbed
 - 2) Split into two retinals
 - 3) Retinal --> retinol
 - d. Metabolism/storage
 - i. Chylomicron remnants taken up from blood by hepatocytes w/ retinol in them
 - ii. Retinol secreted by hepatocytes bound to RBP
 - iii. Stored in stellate cells in sinusoids
 - e. Retinol uptake into target cells
 - i. Bound to RBP as retinol or albumin as retinoic acid
 - ii. Absorbed by target cells, then bind to cellular retinol binding protein
 - iii. Travel to nucleus to activate genes