

Supplemental information micronutrient deficiencies



Vitamin A (retinol)

FUNCTION

Vitamin A is a family of fat soluble compounds (carotinoids) that play an important role in vision, bone growth, reproduction and cell differentiation. It also helps regulate the immune system, promoting optimal lymphocyte function in defending against bacterial and viral infections. Retinol (Vitamin A) promotes healthy surface linings of the eyes and respiratory, urinary and intestinal tracts. Vitamin A also promotes healthy skin function and integrity. Retinol is the most active form of Vitamin A and is synthesized in the body by conversion of provitamin A, primarily beta-carotene, into retinol. Lycopene, lutein and zeaxanthin are carotinoids that do not have Vitamin A activity, but have other health promoting properties. Studies are inconclusive in identifying vitamin A's role as an antioxidant.

DEFICIENCY SYMPTOMS

A large number of physiological systems may be affected by Vitamin A deficiency. Poor epithelial regeneration can result in skin hyperkeratization, problems with the genitourinary reproductive system (reduced fertility) dysfunction within the gastroenterological/biliary system or the pulmonary system. Patients with Celiac disease, Crohn's disease and pancreatic disorders are particularly susceptible to Vitamin A deficiency due to malabsorption. Vitamin A deficiency may result in night blindness and/or epithelial degeneration of the eye. The immune system may also be adversely affected, reducing white blood cell levels and impairing both cell-mediated and humoral defense systems. Vitamin A is also essential for the developing skeletal system and deficiency can result in growth retardation or abnormal bone formation. Vitamin A deficiency is most often associated with strict dietary restrictions and excess alcohol intake.

REPLETION INFORMATION

Vitamin A is found in animal foods such as whole eggs, milk and liver. Fortified breakfast cereals also provide Vitamin A. Most plant sources contain provitamin A carotenoids and rich sources include fruits such as cantaloupe, apricots and mango. Vegetable sources include carrots, spinach, kale and green peas.

RDA's for Vitamin A are listed in micrograms of Retinol Activity Equivalents (RAE) to account for the different biological activities of retinol (Vitamin A) and provitamin A carotenoids. For adult males the RDA is 3000 IU. The RDA for non-pregnant females is 1500 IU. There is no RDA for provitamin A carotenoids. ADEQUATE ZINC IS REQUIRED to synthesize retinol binding protein (RBP) which transports vitamin A. Therefore a deficiency in zinc limits the body's ability to mobilize Vitamin A stores from the liver.

EXCESSIVE VITAMIN A INTAKE IS TOXIC AND MUST BE AVOIDED. Liver abnormalities, reduced bone density (osteoporosis) and central nervous system disorders may result from hypervitaminosis A. Early toxicity signs include peeling/itching skin, brittle nails, yellowish skin, alopecia (hair loss), and bone/joint pain. Provitamin A (beta carotene and mixed carotenoids) are much less toxic and not associated with the commonly noted side effects of excess Vitamin A intake.

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Vitamin B₁ (thiamin)

FUNCTION

Vitamin B₁ ("B₁") is needed by the cells to make energy from foodstuffs. Thiamin pyrophosphate is a cofactor for dehydrogenase enzymes which plays a key role in cellular energy production. It is required for transketolase activity, a component of the pentose phosphate pathway — the sole source for the synthesis of ribose which is used in the synthesis of the nucleic acids DNA and RNA. These reactions also produce the major source of cellular NADPH (nicotinamide adenine dinucleotide phosphate), which is used in fatty acid biosynthesis and other pathways. Thiamin triphosphate is localized in nerve cell membranes and plays a role in the transmission of nerve impulses and acetylcholine synthesis.

DEFICIENCY SYMPTOMS

Clinical symptoms of a B₁ deficiency include constipation, fatigue, irritability, loss of appetite, loss of eye coordination, loss of fine motor skill, mental confusion, mental depression, nausea, peripheral neuropathy, weakness, and Wernicke-Korsakoff Syndrome.

Persons at risk for a B₁ deficiency include alcoholics, elderly, gastric partitioning surgery, inherited thiamin-responsive metabolic disorders, persons on restricted diets, persons with an increased metabolic rate (fever, infection, lactation, pregnancy, trauma), and prolonged hemodialysis. Medications which may induce a B₁ deficiency include antibiotics and some diuretics such as lasix.

REPLETION INFORMATION

Dietary sources richest in B₁ (per serving) include:

enriched grains and grain products
legumes (beans, peas, soybeans, lentils)
nutritional yeasts

rice bran
nutritional supplements

pork
wheat germ

Legumes, nutritional yeasts and pork are high in protein which may be restricted in some persons with renal disease. Excessive ingestion of foods which may contain anti-thiamin factors should be avoided, such as blueberries, coffee, red cabbage, certain raw fresh-water fish, shellfish, and tea.

The 1989 RDA for B₁ is 1.0 mg to 1.5 mg for adults. There is no evidence of B₁ toxicity from oral administration, except for the development of sensitivity in very rare cases. Although there is no known upper limit for B₁ intake in renal disease, it is suggested to keep the intake from supplements to below 100 mg daily.

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Vitamin B2 (Riboflavin)

FUNCTION

Riboflavin helps to metabolize foodstuffs into energy. Riboflavin is converted into its active forms, flavin adenine dinucleotide (FAD) and flavin mononucleotide (FMN). FAD and FMN are primarily involved as cofactors in oxidation-reduction reactions for flavoproteins, essential for cellular energy production and respiration. Riboflavin has a role in antioxidant status by activating glutathione reductase, which regenerates reduced glutathione.

DEFICIENCY SYMPTOMS:

Clinical signs of riboflavin deficiency are less clear-cut than other B Vitamins, but include depression, dizziness, sore or burning lips, mouth, and tongue, photophobia, burning, itching or teary eyes, and loss of visual acuity in early stages. More severe deficiency symptoms for riboflavin are dermatitis (nasal, scrotal), glossitis, cheilosis, angular stomatitis, and corneal vascularization. Frequently, riboflavin deficiencies overlap with niacin, pyridoxine, or iron deficiencies. There is no specific name for riboflavin deficiency disease.

REPLETION INFORMATION

Dietary sources rich in Riboflavin (per serving) include:

Nutritional Supplements
Meats and Dairy Products
Grain Products

Nutritional Yeasts
Green Leafy Vegetables
Enriched Grains

The 1989 RDA for riboflavin is 1.2-1.8 mg for adults. There is no evidence of toxicity from oral administration of Riboflavin, except for very rare cases of sensitivity.

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Vitamin B3 (Niacin)

FUNCTION

Niacinamide is needed to metabolize foodstuffs into energy. Niacinamide is converted into the coenzymes nicotinamide adenine dinucleotide (NAD) and NADP, which function in oxidation-reduction reactions essential for release of energy from carbohydrates, fats, and proteins. Niacin can also be synthesized by the body from tryptophan, although with low efficiency.

DEFICIENCY SYMPTOMS

Clinical signs of early niacinamide deficiency include anorexia, muscular fatigue, indigestion, depression, insomnia, headaches, glossitis, and skin lesions. Severe deficiency may lead to pellagra, with dermatitis, dementia, diarrhea (the "3 D's of pellagra), tremors and sore (black) tongue. Deficiencies of thiamin, riboflavin, and pyridoxine commonly accompany (or can cause) niacinamide deficiency.

REPLETION INFORMATION

Dietary sources of niacinamide are expressed as niacin equivalents, taking into account tryptophan's contribution. Richest sources (per serving) include:

Nutritional supplements
Nutritional yeasts
Meats

Enriched cereals
Legumes including peanuts
Potatoes

The 1989 RDA for niacin is between 13-20 mg for adults. Niacinamide has no observed toxicity for intakes up to 3-9 gms daily, and is the preferred form of niacin supplementation. Niacin (nicotinic acid) may cause flushing (redness and itching of the skin around the face and neck) at doses above 50 mg. Other side effects are possible at higher doses of niacin, which should be used under supervision of a physician.

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Vitamin B₆ (pyridoxine)

FUNCTION

Vitamin B₆ ("B₆") is needed to metabolize proteins and is important for a healthy immune system, nerves, bones and arteries. It plays a key role in the synthesis of heme (for hemoglobin), catecholamines, eicosanoids (prostaglandins), vitamin B₃ (niacin, niacinamide, nicotinamide, nicotinic acid), neurotransmitters, nerve membrane components, and immune functions.

B₆ is a complex of three similar molecules: *pyridoxine*, *pyridoxal* and *pyridoxamine*, and are converted into its most active cofactor form, *pyridoxal-5-phosphate*, which is uniquely essential for numerous enzymatic reactions involving the transfer of amino groups, removal of amino groups, transfer of sulfhydryl groups, removal of organic acid groups and other molecular rearrangements. Magnesium and vitamin B₂ (riboflavin) are necessary to convert pyridoxine to the active cofactor form of B₆, *pyridoxal-5-phosphate*. Lysyl oxidase, a B₆-dependent enzyme, is essential for proper collagen and elastin maturation, which is especially important for arterial structure. B₆ is also required for cystathionine B-synthase activity which converts homocysteine to cystathionine. This reaction is vitally important for cardiovascular health since homocysteine is toxic to arteries, thrombogenic, and may promote oxidation of blood lipids; hyperhomocysteinemia is an important independent risk factor for premature vascular disease.

DEFICIENCY SYMPTOMS

Clinical symptoms of a B₆ deficiency include anxiety, atherosclerosis, carpal tunnel syndrome, convulsion, depression, dermatitis, elevated homocysteine, insomnia, irritability, nausea, peripheral neuropathy, premenstrual tension syndrome, sideroblastic anemia, vomiting, and weakness.

In addition, B₆ supplementation has been shown to be useful in the treatment of a variety of conditions including asthma, epilepsy, immune enhancement, osteoporosis, and vascular disease. Medications which may induce a B₆ deficiency include antibiotics, estrogen replacement, oral contraceptives, theophylline, and tuberculosis therapy (isoniazid).

Further, it is estimated that approximately 2.5 million Americans may be affected by the incidence of heterozygous homocystinuria which is B₆-dependent. Thus, a B₆ deficiency is known to be detrimental to cardiovascular health, contributing to atherosclerosis and thrombotic events.

REPLETION INFORMATION

Legumes, meats and meat products, and nutritional yeasts are high in protein which may be restricted in some persons with renal disease. Dietary sources include:

nutritional supplements	bananas	legumes	nutritional yeasts
enriched grains , grain products	wheat germ	meats and meat products	potatoes

The 1989 RDA for B₆ is 1.4 mg - 2.0 mg daily for adults. Therapeutic doses of 100 mg daily for adults have exhibited excellent long-term safety and tolerability. Supplementation has reduced or normalized elevated homocysteine and decreased the incidence of cardiovascular disease in heterozygotes for homocysteinemia. Persons with drug-induced neuritis may tolerate higher doses, while B₆ may diminish the effectiveness of the medication L-DOPA in persons with Parkinson's disease. Oral intakes exceeding 200 mg daily result in a small number of peripheral neuropathy cases, which may be related to the oxidation of B₆.

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Vitamin B₁₂ (cobalamin)

FUNCTION

Vitamin B₁₂ ("B₁₂") is needed to form blood and immune cells, and support a healthy nervous system. A series of closely-related compounds known collectively as *cobalamins* are converted into active co enzymatic forms *methylcobalamin* or *5'-deoxyadenosylcobalamin*. Methylcobalamin activates the methyltransferase enzyme that converts homocysteine to methionine at the expense of methylated folate. This reaction regenerates the tetrahydrofolate from which can receive methyl groups from other sources (usually from serine via the B₆-dependent enzyme). This reaction removes homocysteine and regenerates methionine. Adequate B₁₂ status is also necessary to prevent a functional folate deficiency. B₁₂ and folate are intricately involved in chemical processes. Since B₁₂ works to "recharge" folate from one-carbon units, a B₁₂ deficiency can result in a folate deficiency if folate function is marginal. A high intake of folate may mask a B₁₂ deficiency by preventing the changes in red blood cells. Adenosylcobalamin is involved in the metabolism of odd-chain fatty acids and branched-chain amino acids.

DEFICIENCY SYMPTOMS

Clinical symptoms of a B₁₂ deficiency include constipation, decrease in blood cell counts (red, white and platelets), depression, elevated homocysteine, elevated methylmalonic acid, fatigue, glossitis, headache, irreversible neurological damage (with loss of position sense and ataxia), irritability, mental depression, numbness, palpitation, peripheral neuropathy, pernicious anemia (macrocytic anemia), persons with a marginal folate status, shortness of breath, sore tongue, and weakness. Megaloblastic anemia may occur as a result of a B₁₂ deficiency on folate metabolism. If a B₁₂ deficiency is not initiated, permanent neurological damage, including degeneration of nerves and spinal cord, can result. Recent evidence suggests that mental symptoms of depression and fatigue are detectable before anemia develops.

Strict vegetarianism, gastrectomy, gastrointestinal disturbances, helicobacter pylori infection, and reduced stomach acid are frequently associated with B₁₂ deficiency. Similar to folate deficiency, asymptomatic B₁₂ deficiency leads to elevated serum homocysteine; B₁₂ is required to prevent the accumulation of homocysteine which is toxic to arteries, thrombogenic, and is associated with CVD and connective tissue abnormalities. Hyper-homocysteinemia is an important independent risk factor for premature vascular disease.

Medications which may induce a B₁₂ deficiency include alka seltzer, antibiotics, Maalox, oral contraceptives, tagamet, and zantac.

REPLETION INFORMATION

Dietary sources richest in B₁₂ (per serving) include:

cheese
eggs

fish
liver and kidney

meats and meat products
nutritional supplements

Dietary sources richest in B₁₂ are nutritional supplements and animal foods and may be restricted in those with renal disease. Nutritional supplements and some fortified cereals contain B₁₂ without high amounts of protein. The 1989 RDA for B₁₂ is 2.0 mcg daily for adults. No toxic effects of oral or injectable B₁₂ intake have been demonstrated, even at long-term doses over 30 mg daily for adults. Due to the potential for B₁₂ malabsorption, oral doses of 1 mg daily have been used for successful repletion. Some persons may require injectable forms of B₁₂ if oral therapy does not reverse deficiency symptoms. Although dosage schedules may vary considerably, injection of 1 mg weekly until repletion is corrected, has been favored. In persons with a B₁₂ deficiency, oral repletion decreased or normalized serum homocysteine in many cases. In order to absorb the small amounts of B₁₂ found in food, the stomach secretes *intrinsic factor*, a special digestive secretion that increases the absorption of B₁₂ in the small intestine.

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Biotin

FUNCTION

Biotin is required for proper metabolism of fats and carbohydrates. Biotin-dependent enzymes catalyze the addition of carboxyl groups (COO-) From bicarbonate, for use in fatty acid biosynthesis, gluconeogenesis, lipogenesis, propionate metabolism, and leucine catabolism.

DEFICIENCY SYMPTOMS

Symptoms of biotin deficiency include erythematous exfoliative dermatitis, thinning hair, fatigue, irritability, mild depression, somnolence, muscle pains, anorexia, nausea, mild anemia. Infants with seborrheic dermatitis, Leiner's disease or alopecia may indicate a biotin deficiency, along with symptoms of ketoacidosis, poor feeding, vomiting, lethargy, coma, and developmental retardation. Dietary symptoms include fatigue, dry skin, body hair loss, nausea, loss of appetite, and mild depression.

Those at risk for biotin deficiency include: persons consuming excessive amounts of raw egg whites, inherited disorders of biotin metabolism, extended total parenteral nutrition (biotin-free), loss of enteric gut microgloura from antibiotic therapy or altered gut motility, pregnant and lactating women, antiepileptic drug therapy, alcoholics, trauma (burns and surgery), elderly, malabsorption (especially achlorhydria).

REPLETION INFORMATION

Dietary intake of foods rich in Biotin should be increased. Do not eat raw egg whites.

Nutritional Supplements
Egg Yolks
Whole Grains

Liver
Legumes
Rice Bran

Nutritional Yeast
Royal Jelly
Fish

The estimated adequate daily dietary intake for biotin is 30-100 mcg for adults. No adverse effects have been noted in humans ingesting up to 2000 mcg daily for long time periods.

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Folate

FUNCTION

Folate (Folic Acid) is needed to produce blood cells and other new tissue cells. Folate is a generic term for a group of pteridine compounds essential for one-carbon unit metabolism. Folates are involved in the synthesis of DNA, RNA, and tRNA necessary for cell growth. Folates are required for metabolism of methionine, histidine, tryptophan, glycine, serine, and formate. Interactions with Vitamin B6 and B12 also occur from common metabolic pathways. Folate function is necessary to prevent accumulation of homocysteine. Deficient folate status of pregnant females is also directly linked to incidence of birth defects, especially neural tube defects such as spina bifida.

DEFICIENCY SYMPTOMS

Symptoms of folate deficiency include birth defects (neural tube defects, spina bifida), fatigue, anorexia, constipation, glossitis, headaches, insomnia, restless legs, paranoia, memory impairment, megaloblastic anemia (identical in appearance to Vitamin B12 deficiency), hypersegmentation of neutrophils and with severe deficiency do not occur with folate deficiency. Thus, a regulatory limit on folate levels in dietary supplements of 400 ug per unit is in effect, to prevent a potential missed diagnosis of Vitamin B12 deficiency.

Those at risk for folate deficiency include: Vitamin B12 deficiency, malnourished, malabsorption, pregnant and lactating women, increased rate of cellular division (burns, trauma, malignancies, hemolytic anemias), alcoholics, anti-convulsant therapy (phenytoin, barbiturates, primidone), folate antagonist therapy (methotrexate, 5-fluorouracil, pyrimethamine), tuberculosis therapy (isoniazid plus cycloserine), oral contraceptive users, sulfasalazine therapy, elderly, infants, inherited folate disorders.

REPLETION INFORMATION

Dietary sources richest in folate (per serving) include:

Nutritional supplements
Vitamin-Fortified Cereals

Wheat Germ
Nuts

Seeds
Liver

Legumes
Green Leafy Vegetables

The 1989 RDA for folate is 400ug per day. No adverse effects from long-term folate supplementation of up to 10mg daily for five years have been reported, indicating a high tolerance level for folate.

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Pantothenate

FUNCTION

Pantothenic acid plays vital roles in energy production from foodstuffs. Pantothenate is a component of coenzyme A, which is indispensable for two-carbon unit metabolism (acetyl groups). Acetyl groups are involved in the release of energy from carbohydrates, fats, proteins, and other compounds, as well as synthesis of fats, cholesterol, steroid hormones, porphyrin and phospholipids.

DEFICIENCY SYMPTOMS

Pantothenate deficiency symptoms are thought to be uncommon because of widespread distribution in all foodstuffs. However, human deficiency symptoms may include fatigue, depression, burning feet, dermatitis, burning or pain of arms and legs, anorexia, nausea, indigestion, irritability, mental depression, fainting, hair loss, increased heart rate, and susceptibility to infection.

REPLETION INFORMATION

Dietary sources richest in Pantothenate (per serving) include:

Nutritional supplements
Nutritional Yeasts
Meats

Legumes
Whole Grain Products
Wheat Germ

Vegetables
Nuts
Seeds

The estimated safe and adequate daily dietary intake for pantothenate is 4-7 mg for adults. Oral administration of pantothenate has shown no toxicity in doses up to 10 gms daily. Higher doses may cause diarrhea.

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Vitamin D (ergocalciferol)

FUNCTION

Vitamin D is the principle regulator of calcium homeostasis in the body. It is essential for skeletal development and bone mineralization. Vitamin D is a prohormone with no hormone activity. It is converted to a molecule that has biological activity. The active form of the vitamin is 1-25-dihydroxyvitamin D, usually referred to as vitamin D3. It is synthesized in the skin from 7-dehydrocholesterol via photochemical reactions requiring UV light (sunlight). Inadequate exposure to sunlight contributes to vitamin D deficiency. Vitamin D deficiency in adults can lead to osteoporosis. This results from a compensatory increase in the production of parathyroid hormone resulting in bone resorption. Increasing evidence is accumulating that vitamin D may also contribute to antioxidant function by inhibiting lipid peroxidation. The mechanism of the antioxidant effect is unknown. Vitamin D is also needed for adequate blood levels of insulin. Vitamin D receptors have been identified in the pancreas.

DEFICIENCY SYMPTOMS

Osteoporosis results from an imbalance between bone resorption and bone formation. Decreased vitamin D levels result in decreased production of the active vitamin form, vitamin D3. Vitamin D enhances the efficiency of calcium absorption. Chronic vitamin D deficiency results in decreased calcium absorption and secondary hyperparathyroidism.

Vitamin D3 has been found to have anticarcinogenic activity, inducing apoptosis in many types of cancer cells. It has also been useful in the treatment of psoriasis when applied topically. Vitamin D appears to demonstrate both immune-enhancing and immunosuppressive effects.

REPLETION INFORMATION

Supplemental vitamin D is available as vitamin D2 (ergocalciferol) or vitamin D3 (cholecalciferol). The Food and Nutrition Board of the Institute of Medicine of the National Academy of Science recognizes the biological activity of vitamins D2 and D3 are equivalent with age related intakes. For both men and women recommended doses:

Age 19-50 years	200 IU/day (5.0 micrograms)
Age 51-70 years	400 IU/day (10 micrograms)
Greater than 70 years	600 IU/day (15 micrograms)

Dosages over 3000 IU/day are associated with hypercalcemia, causing multiple debilitating effects. Anorexia, nausea and vomiting have been observed at doses as low as 1250 IU/day. The prolonged ingestion of excessive vitamin D and the accompanying hypercalcemia can result in metastatic calcification of soft tissues, including kidney, blood vessels, heart and lungs.

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Vitamin E (α-tocopherol)

FUNCTION

Vitamin E is an antioxidant that protects cell membranes and other fat-soluble compounds from oxidative damage by free radicals. For example, the oxidative damage to LDL-cholesterol appears to lead to the deposition of cholesterol in the arterial wall leading to atherosclerotic disease. In the past few years many other functions of vitamin E have been clarified. Alpha-tocopherol has direct effect on the control of inflammation, red and white blood cell production, connective tissue growth and genetic control of cell division. Vitamin E acts to reduce free radical damage by converting arachidonic acid is converted to pro-inflammatory (12-HPETE) derivatives. In deficiencies of vitamin E, arachidonic acid is converted to pro-inflammatory leukotrienes and cytokines. In neutralizing free radicals, vitamin E is oxidized to a free radical. Conversion back to the reduced form occurs by reaction with vitamin C (ascorbate).

DEFICIENCY SYMPTOMS

The principle use of vitamin E is an antioxidant in the protection against heart disease, cancer, stroke and neurodegenerative disease (Alzheimer's). In addition, alpha-tocopherol supplementation is useful in treating other cardiovascular diseases, diabetes, fibrocystic breast disease, menopause symptoms and tardive dyskinesia. It may also have applications in Parkinson's Disease and arthritis. Vitamin E is important to immune function, protecting thymic function and white blood cells from oxidative stress.

Symptoms of vitamin E deficiency include nerve damage, muscle weakness, poor coordination, involuntary eye movements, red blood cell fragility, anemia and retrolental fibroplasia (eye disease).

REPLETION INFORMATION

Vitamin E is available in many different formulations, either natural or synthetic. Natural forms of vitamin E are designated d-, as in d-α-tocopherol. Synthetic forms are designated as dl-. The biologically active form of the vitamin is the d- form and it is recommended for supplementation over the dl- (synthetic) forms. Beta-tocopherol, gamma-tocopherol and the alpha- and delta-tocoretinols have less than 50% of the biological activity than d-α-tocopherol.

The RDA for vitamin E (d-α-tocopherol) is set at 15 I.U. per day. The amount of vitamin E required is dependent upon the amount of polyunsaturated fat in the diet. The more polyunsaturated fat in the diet, the greater the risk for oxidative damage, and the vitamin E requirement is increased. Most studies have utilized doses between 200-400 I.U. per day. Some studies report effective use of vitamin E at doses up to 3000 I.U. per day without observed side effects over a two-year period.

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Calcium

FUNCTION

Calcium is the most abundant mineral in the body, with 99% residing in bones and teeth. As a component of hard tissues, Calcium fulfills a structural role to maintain body size and act as attachments for musculoskeletal tissues. The remaining 1% of calcium is present in blood and soft tissues. Functions of non-skeletal Calcium include: enzyme activation, second messenger roles (transmitting hormonal information), blood clotting, cell and cell organelle membrane function (stabilization and transport), nerve impulse transmission, and muscular contraction, tone, and irritability. Calcium levels in the blood are maintained within very strict limits by dietary intake, hormonal regulation, and a rapidly exchangeable pool in bone tissue.

DEFICIENCY SYMPTOMS

Calcium deficiencies are both acute and chronic. Acute Calcium deficiency relates to lack of ionized Calcium, causing increased muscular and nervous irritability, muscle spasms, muscle cramps, and tetany. Chronic calcium deficiency manifests as bone loss disorders (osteoporosis, osteomalacia in adults, rickets in children), tooth decay, periodontal disease, depression, and possibly hypertension.

Those at risk for Calcium deficiency include: malnourished, malabsorption, and bone loss disorders. Conditions which are known to decrease Calcium uptake or distribution are: decreased gastric acidity, Vitamin D deficiency, high fat diets, high oxalate intake from rhubarb, spinach, chard, and beet greens, high phytic acid intake from whole grains, high fiber intake, immobilization, faster gastrointestinal motility, psychological stress, thiazide diuretic therapy, aluminum compounds (aluminum-containing antacids, drugs, some parenteral feeding solutions).

REPLETION INFORMATION

Dietary Sources richest in Calcium (per serving) are:

Calcium Supplements

Tofu

Bone Meal

Multiple Vitamin/Mineral Supplements with Calcium

Milk and Dairy Products (milk, yogurt, cheeses)

Canned Salmon & Sardines (with bones)

The 1989 RDA for calcium is 1000-1200 mg for adults (1300 mg for ages 9-18, 800 mg other ages). In general, daily calcium intakes of 2.0 grams or less are safe. Certain individuals with tendency to form kidney stones should consult a physician before increasing calcium intake. Milk-alkali syndrome is possible after consumption of 2 or more quarts of milk daily along with large amounts of carbonate antacids (calcium deposition in soft tissues and kidney stones). Calcium intakes greater than 2-4 grams daily may depress uptake of magnesium, zinc, iron, manganese, and other minerals, and are associated with depressed reflexes, muscle weakness, ataxia, and anorexia.

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Chromium

FUNCTION

Chromium is an essential trace mineral that plays a fundamental role in assisting proper insulin function, preventing high cholesterol levels, and plays a role in maintaining fertility. After each meal, blood glucose levels rise, causing insulin to be secreted by the pancreas. Insulin lowers blood glucose levels by increasing the rate at which glucose enters a person's cells. Chromium is believed to facilitate the attachment of insulin to the cell's insulin receptors. Studies also indicate that chromium participates in cholesterol metabolism, suggesting a role for this mineral in maintaining normal blood cholesterol levels and preventing atherosclerosis. Finally, chromium plays a role in nucleic acid synthesis, and animal studies of chromium supplemented subjects have increased sperm counts and overall fertility relative to chromium deficient subjects.

DEFICIENCY SYMPTOMS

As a trace mineral, very little chromium should be sufficient for an individual's nutritional needs. However, due to processing methods that remove most of the naturally occurring chromium from commonly consumed foods, dietary deficiency of chromium is believed to be widespread in the United States. Chromium deficiency may increase the likelihood of insulin resistance, a condition in which the cells of the body do not respond to the presence of insulin. Insulin resistance can lead to elevated blood levels of insulin (hyperinsulinemia) and elevated blood levels of glucose, which can ultimately cause heart disease and/or diabetes. There is also evidence from animal studies to suggest a link between low chromium levels and cornea lesions (which are also frequently observed in diabetic individuals).

Dietary deficiency of chromium is associated with metabolic syndrome. Metabolic syndrome represents a constellation of symptoms, including hyperinsulinemia, high blood pressure, high triglyceride levels, high blood sugar levels, and low HDL cholesterol levels, that increase one's risk for heart disease. Low body levels of chromium, a mineral involved in carbohydrate and fat metabolism, are associated with an increased risk of coronary artery disease incidence and mortality.

Chromium deficiency has an ability to depress nucleic acid synthesis. Experiments in animal models have shown that low chromium diets result in significantly lower sperm count and decreased fertility compared to chromium-supplemented individuals. Chromium is essential for maintaining the structural stability of proteins and nucleic acids and animal studies have found that this element is also vital for healthy fetal growth and development. Studies on humans have established that premature infants, and those with evidence of intrauterine growth retardation, have significantly lower chromium status compared to infants born full-term. Others have found that women who've given birth two or more times have far lower body chromium levels compared to women who've never given birth. These findings indicate that chromium is indeed an essential trace element during fetal growth and development.

REPLETION INFORMATION

In 2001, the Institute of Medicine at the National Academy of Sciences conducted a thorough review of the chromium research and concluded that excessive intake of chromium from foods or supplements is not associated with any adverse effects. As a result, no Tolerable Upper Intake Level (UL) was established for this mineral. However, the Institute of Medicine noted that people with liver or kidney disease may be more susceptible to adverse effects from excessive intake of chromium, and cautioned such individuals to avoid taking chromium supplements in higher than recommended amounts. There is limited evidence to suggest that long term chromium piccolinate supplementation at levels greater than 200 micrograms per day could be hazardous to renal function and chromosome integrity. For individuals over the age of 7, the Food and Nutrition Board of the US National Academy of Sciences has recommended 50-200 micrograms as the estimated safe and adequate daily dietary intake.

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Magnesium

FUNCTION

Magnesium is predominantly found intracellularly, where it is vital for proper cell functions. Magnesium is the second most prevalent intracellular cation (after potassium). Magnesium functions are numerous and essential, including enzyme activation (over 300 types), neuromuscular activity, membrane transport and interactions, energy metabolism (carbohydrates, fats, proteins), and roles in calcium and phosphorus metabolism.

DEFICIENCY SYMPTOMS

Deficiency symptoms are both acute (Trousseau and Chvostek signs, muscle spasms, tetany, cardiac arrhythmias, ataxia, vertigo, convulsions, organic brain syndrome) and chronic (thrombophlebitis, hemolytic anemia, bone loss, depressed immune function, poor wound healing, hyperirritability, burxism, hyperlipidemia, fatigue, hypertension).

Those at risk for Magnesium deficiency include: malabsorption, malnourished, alcoholics, diabetics, diuretic therapy, children, elderly, pregnant and lactating women, postmenopausal women with osteoporosis, athletes, digitalis therapy, long-term therapy with antibiotics, chemotherapeutic and immunosuppressive medications. In addition, the following diseases are associated with Magnesium deficiency: cardiovascular disease, cirrhosis, renal disease, parathyroid diseases, thyroid conditions.

REPLETION INFORMATION

Dietary sources richest in Magnesium (per serving) are:

Nutritional Supplements
Seeds (especially pumpkin seeds)

Nuts
Soybeans

Whole Grains
Potatoes

Legumes
Fresh Vegetables

The 1989 RDA for Magnesium is between 280-400 mg daily for adults. Large oral intakes of Magnesium (400-1000 mg daily), when spread throughout the day, are not considered harmful, except for some persons with impaired renal function. Higher doses have been used as laxatives and antacids. Excessive Magnesium intake may cause diarrhea, nausea, vomiting, hypotension, bradycardia, and CNS depression. Continued excessive intakes of Magnesium may imbalance calcium and phosphorous metabolism.

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Selenium

FUNCTION

The trace mineral selenium functions primarily as a component of the antioxidant enzyme, glutathione peroxidase. Glutathione peroxidase activity, which requires selenium for activity, facilitates the recycling of vitamins C and E, in optimizing the performance of the antioxidant system. Low levels of selenium have been linked to a higher risk for cancer, cardiovascular disease, inflammatory diseases, and other conditions associated with free radical damage, including aging and cataract formation. Selenium is also essential for healthy cell-mediated immune function, stimulating immune properties of lymphocytes. Selenium is also needed for the activation of thyroid hormones.

DEFICIENCY SYMPTOMS

Chronic low selenium intake is associated with an increased risk for heart disease, cancer and depressed immune function. Selenium appears to provide protection against heart disease and stroke. Selenium supplementation (100 ug/day) increases the ratio of HDL to LDL and inhibits platelet aggregation.

Selenium and glutathione peroxidase activity are low in patients with rheumatoid arthritis, eczema, psoriasis and most inflammatory conditions. This is related to the increased synthesis of proinflammatory prostaglandins and leukotrienes. Immune system function is enhanced by selenium, by contributing to higher natural killer cell (NKC) activity. Natural killer cells have the ability to destroy cancer cells and bacterial and viral agents. Heavy metal toxicity symptoms may be alleviated by selenium, acting as an antagonist. Selenium deficiency may also contribute to male infertility.

REPLETION INFORMATION

Selenium is safe at the level generally used for supplementation (100-200 mcg/day). However, taking more than 750 mcg of selenium per day may cause toxicity. Reactions such as loss of fingernails, skin rash, and neurological aberrations. In the presence of iodine deficiency goiter, selenium supplementation has been reported to exacerbate low thyroid function.

Selenium is available in several different forms. Studies indicate that inorganic salts like sodium selenite are less effectively absorbed and not as biologically active as organic forms of selenium, such as selenomethionine or high-selenium content yeast. Highest sources of dietary selenium are found in:

Wheat Germ
Brazil Nuts

Bran
Red Swiss Chard

Whole Wheat Bread
Brown Rice

Oats
Turnips

The adult RDA for selenium is 50ug/day.

Supplemental information micronutrient deficiencies



Zinc

FUNCTION

The primary role of zinc is to activate almost 200 enzymes with vital roles in cell regulation, immune function, acid/base balance, DNA, RNA, and protein synthesis, lipid metabolism, eicosanoid production, and digestion. Zinc also is a component of insulin (energy metabolism), thymic hormones (immune function) and gustin (taste acuity).

DEFICIENCY SYMPTOMS:

Symptoms of zinc deficiency include fatigue, dermatitis, acne, loss of taste, poor wound healing, anorexia, decreased immunity, delayed growth, hypogonadism and delayed sexual maturation, diarrhea, skeletal abnormalities, alopecia, behavioral disturbances, white spots on fingernails, infertility and night blindness.

Those at risk for zinc deficiency include alcoholics, malnourished, malabsorption (Crohn's Disease, celiac disease), long-term parenteral nutrition, chronic renal disease, anorexics, dieters, pregnant women, elderly, and sickle-cell disease.

REPLETION INFORMATION:

Nutritional Supplements	Oysters	Wheat Germ	Legumes	Seeds
Zinc-Fortified Cereal Products	Red Meats	Nuts	Soybean Products	Potatoes

Compounds found in meats enhance absorption of zinc from plant sources.

The 1989 RDA for zinc is 12-15 mg. In general, daily doses up to 50mg of elemental zinc appear safe. Acute toxicity (nausea, vomiting, diarrhea, fever, muscle pain) may occur after intake of 1-2 grams of zinc. Chronic intakes of 150 mg of zinc for several months may impair certain immune responses, decrease high-density lipoprotein levels, or impair copper status (possibly leading to normocytic anemia). Significant differences in tolerability between inorganic zinc salts and organic zinc chelates exist with organic chelates recommended for supplementation.

Supplemental information micronutrient deficiencies



Asparagine

FUNCTION

Asparagine is a dietarily dispensable amino acid synthesized from aspartate and glutamine. Asparagine has three major functions: 1) incorporation into amino acid sequences of proteins; 2) storage form for aspartate (is a required precursor for synthesis of DNA, RNA, and ATP); and 3) source of amino groups for production of other dispensable amino acids via transaminases. Asparagine in proteins is an attachment site for carbohydrates (N-linked oligosaccharides) to form collagen assembly, enzymes, and cell-cell recognition. Asparagine can be readily converted into aspartate, providing aspartate on demand for many cellular functions. Aspartate can increase cellular energy production by contributing carbon skeletons to the Citric Acid Cycle. Aspartate is also a component of the urea cycle, which removes excess ammonia. The conversion of asparagine to aspartate involves transfer of the extra amino group from asparagine to another keto acid, forming a dispensable amino acid. In this way, asparagine can be a precursor for many amino acids to be produced on demand to meet cell requirements.

DEFICIENCY SYMPTOMS

Data from testing over 10,000 physician office patients has found that 22.8%% have deficient asparagine function, as indicated by increased lymphocyte growth response after addition of asparagine to the lymphocyte growth media. Significantly increased prevalence of asparagine deficiencies has been detected in two clinical manifestations: 1) fatigue; and 2) immune system stress. For example, in 75 subjects with rheumatoid arthritis, 32.0% exhibited an asparagine deficiency. There are no published deficiency symptoms for asparagine in the medical literature, partly due to previous lack of adequate assessment tests. Therefore, tentative associates of asparagine deficiencies with clinical complaints of fatigue, and clinical findings of immune dysfunction (autoimmune disorders, sever allergies, infections) have been identified by the Functional Intracellular Analysis test for asparagine.

REPLETION INFORMATION

Since asparagine is a dispensable amino acid, no RDA exists. Asparagine is present in all proteins, but is partially degraded into aspartate by heat (cooking), storage, or acid. Asparagine supplementation appears safe in modest doses (up to 6 grams daily)

Supplemental information micronutrient deficiencies



Carnitine (L-carnitine)

FUNCTION

L-carnitine is an amino acid derivative of the essential amino acids L-lysine and methionine. The conversion to carnitine requires niacin (B3), vitamins B6 and C, and iron. It is found in nearly all cells of the body but chiefly in the liver and kidney. Carnitine is essential for the transportation of long-chain fatty acids across the inner mitochondrial membranes in the mitochondria, where they are metabolized by beta-oxidation to produce biological energy in the form of adenosine triphosphate (ATP).

L-Carnitine also is required to remove short- and medium-chain fatty acids from the mitochondria. This removal optimizes energy production by maintaining coenzyme A at optimal levels for normal metabolism and energy production.

DEFICIENCY SYMPTOMS

Deficiencies of carnitine may result from: 1) deficiencies of essential amino acids lysine and methionine, 2) deficiencies of cofactors (B3, C, B6 and iron), 3) defective gastrointestinal function, 4) increased requirement because of high-fat diet, metabolic stress or disease. The consequences of carnitine deficiency are impaired lipid metabolism and lipid accumulation in skeletal muscles, heart, and liver. Patients usually exhibit muscle weakness and fatigue.

Normal heart function depends on adequate concentrations of carnitine. While the normal heart stores more carnitine than required, if the heart does not have a good oxygen supply, carnitine levels quickly decrease. This lack of oxygen leads to decreased energy production and increased risk for angina and heart disease. Carnitine benefits blood lipids by lowering triglycerides and total cholesterol, while increasing HDL. L-acetylcarnitine (LAC) may be useful in the treatment of Alzheimer's disease, senile depression and age-related memory loss.

REPLETION INFORMATION

There have been no reports of toxicity from L-carnitine supplementation. The biologically active form of carnitine is the L- isomer. DL-carnitine should be avoided. Usual dosages found in capsules and tablets range from 250 to 1000 mg in a variety of chemical formulations: L-acetylcarnitine, L-carnitine, and the HCl, tartrate and fumarate salts. Carnitine, Coenzyme Q10 and pantothenate (B5) appear to work synergistically.

Supplemental information micronutrient deficiencies



Glutamine

FUNCTION

Glutamine is used for energy, for synthesis of other essential building blocks, (protein, DNA, and RNA), and for removal of toxic substances. Glutamine is a dispensable amino acid present in greater amounts than any other amino acid in the body fluid and cells. In addition to being incorporated into proteins, Glutamine has many metabolic functions: major component of amino acid pools, alternative energy source, DNA, and RNA synthesis precursor, neurotransmitter precursor, acid/base balance, ureagenesis, and precursor for other dispensable amino acids, amino sugars and other compounds.

DEFICIENCY SYMPTOMS

Glutamine deficiency symptoms are not described because of endogenous synthesis and high dietary intakes. However, certain conditions are under investigation where exogenous supply of glutamine may become essential: intestinal disorders, major trauma (burns, surgery), immune functions, gastric ulcers. Glutamine may be useful in alcoholism and fatigue.

REPLETION INFORMATION

No RDA exists for glutamine, which occurs in large amounts in foods containing protein. Richest sources are milk protein and meats. Large doses of glutamine, as the free-form amino acid (up to 10 grams daily) appear to be well tolerated. Larger doses may cause osmotic diarrhea in some persons and are contraindicated in hyperammonemia.

Supplemental information micronutrient deficiencies



Serine

FUNCTION

Serine is used to manufacture proteins, energy, cell membrane structure and synthesis of other cell components (DNA and RNA). Serine is a dispensable amino acid obtained from the diet and synthesized from other amino acids and metabolites of glucose. Serine participates in protein synthesis, energy production, phospholipid synthesis (phosphatidyl serine and ethanolamine) and one-carbon unit metabolism (necessary for DNA and RNA synthesis). Quantitatively, serine supplies more one-carbon units than any other nutrient. Serine is an attachment point for carbohydrates on protein chains.

DEFICIENCY SYMPTOMS

No specific deficiency symptoms are known for serine; however, some individuals may have a metabolic defect in serine synthesis or conditional need for serine during periods of cell growth or physiological stress. Preliminary clinical evidence suggests neurological symptoms (neuropathy, neuritis, and behavioral disturbances may be associated with serine deficiencies. Additional laboratory tests to determine other aspects of serine metabolism would include amino acid analysis of serum and/or urine.

REPLETION INFORMATION

Since serine is a dispensable amino acid, no dietary RDA exists. Serine is present in foods that are rich in protein. Doses of 1-2 grams daily of pure serine appear safe.

Supplemental information micronutrient deficiencies



Coenzyme Q-10

FUNCTION

Coenzyme Q-10 belongs to a family of substances called ubiquinones. These compounds are lipophilic, water-insoluble substances involved in electron transport and energy production within the mitochondria. In this capacity, coenzyme Q-10 facilitates the conversion of the energy released through glycolysis into ATP (adenosine triphosphate). Coenzyme Q-10 is also a powerful antioxidant, facilitating the removal of destructive free radicals from the mitochondrial environment. Coenzyme Q-10 is believed to provide a sparing effect on vitamin E. Virtually every cell of the human body requires coenzyme Q-10, with heart muscle and the liver having the greatest concentration since their mitochondrial content is the greatest in the body.

DEFICIENCY SYMPTOMS

Deficiency is poorly understood, but may be caused by synthesis problems in the body rather than insufficiency in the diet. It is now established that many patients on statin drugs (cholesterol lowering medications and HMG CoA Reductase Inhibitors) have lowered coenzyme Q-10 levels and are at increased risk for deficiency. Many cardiologists routinely utilize coenzyme Q-10 for treating congestive heart failure. Low blood levels have been reported in people with heart failure, cardiomyopathies, gingivitis (an inflammation of the gums), morbid obesity, hypertension, muscular dystrophy, AIDS and in some patients on peripheral dialysis. Aging is also associated with lower coenzyme Q-10 levels. Some studies have indicated that high doses of coenzyme Q-10 are useful in arresting Parkinson's disease and the treatment of Alzheimer's disease. The most common deficiency symptoms include angina and fatigue.

REPLETION INFORMATION

Coenzyme Q-10 is in every plant and animal cell. However, the amount of coenzyme Q-10 is probably insufficient to produce the clinical effects associated with therapy. The richest dietary sources of coenzyme Q-10 are fish and red meat. The best supplement preparations are soft-gelatin capsules that contain coenzyme Q-10 in an oil base. Capsules range in dosages from 10 to 250 mg. Toxicity is not known, but doses greater than 250 mg can be associated with nausea and diarrhea.

Pregnant women and nursing mothers should avoid supplementing with coenzyme Q-10 because long-term safety studies have yet to be completed. Patients with congestive heart failure on coenzyme Q-10 therapy should not discontinue the treatment without physician approval.

Supplemental information micronutrient deficiencies



Glutathione

FUNCTION

Glutathione is implicated in many cellular functions including antioxidant protection and detoxification. It is also essential for the maintenance of cell membrane integrity in red blood cells. Intracellular glutathione concentrations are principally derived by intracellular synthesis, as few cells directly uptake glutathione from the surrounding extracellular fluid. The high concentration of glutathione in virtually all cells clearly indicates its importance in metabolic and oxidative detoxification processes. Glutathione may be considered the preeminent antioxidant.

DEFICIENCY SYMPTOMS

A wide range of human conditions may be produced, or made worse, by "free radicals", including aging, AIDS, arthritis, atherosclerosis, cancer, cardiovascular disease, neurodegenerative diseases, pulmonary diseases and viral infections. Their treatment or prevention often includes antioxidants such as vitamin C, vitamin E, carotenoids and selenium. Glutathione is an essential component of the antioxidant defense system — producing a "sparing effect" for both tocopherol and ascorbate by reducing the oxidized forms, and by eliminating hydrogen peroxide by reacting with glutathione peroxidase. Cellular glutathione functions by decreasing the formation of oxidized LDL (low density lipoproteins) which is implicated in the development of atherosclerosis. T-lymphocyte cells become deficient in glutathione in the progression of AIDS which impairs immune function. Glutathione is also required for the synthesis of some prostaglandins from omega-3 and omega-6 polyunsaturated fatty acids which are important in the inflammatory response. Patients with adult respiratory distress syndrome are favorably affected by treatments that increase cellular glutathione.

REPLETION INFORMATION

Glutathione is poorly absorbed from the gastrointestinal tract and foods rich in glutathione do not appear to contribute to increases in intracellular glutathione levels. Cysteine appears to be the limiting amino acid in the intracellular synthesis of glutathione and supplementation with up to 2000 mg daily of N-Acetyl-L-Cysteine appear safe. Supplementation with cysteine is not recommended as it may be poorly tolerated by many patients. In addition, it may be rapidly oxidized to cysteine, a less usable form for the synthesis of glutathione. Foods rich in cyteine are generally high protein foods such as meats, yogurt, wheat germ and eggs.

Supplemental information micronutrient deficiencies



Alpha Lipoic Acid

FUNCTION

Lipoic Acid is a sulfur-containing vitamin-like substance that is an important cofactor in energy-producing reactions in the production of cellular energy (ATP). Lipoic acid has been referred to as a “universal antioxidant” because it is soluble in both fat and water. It is capable of regenerating several other antioxidants back to their active reduced states, including vitamin C, vitamin E, glutathione and coenzyme Q10. Alpha lipoic acid has several potential actions for the type 2 (non-insulin-dependent) diabetic. It reduces glycosylation reactions (attachment of sugar moieties to protein) and facilitates healing of diabetic nerve damage. Biochemical reactions utilizing lipoic acid occur within the mitochondria, where it functions critically in its antioxidant capacity.

DEFICIENCY SYMPTOMS

Several studies demonstrate that individuals infected with HIV have a compromised antioxidant defense system. Blood antioxidants are decreased and peroxidation products of lipids and proteins are increased. These changes deplete glutathione levels and this often compromises cell-mediated immune function and progression of AIDS. Alpha lipoic acid supplementation increases vitamin C and glutathione. T-lymphocyte production and T helper/suppressor cell ratios are increased. Patients with compromised immune symptom performance may benefit by supplementation with alpha lipoic acid.

In patients with diabetic neuropathy resulting from antioxidant deficiency, lipoic acid improves blood flow to peripheral nerves, decreases lipid and protein peroxidation, and may stimulate the regeneration of nerve fibers. There is growing evidence that lipoic acid has beneficial effects in slowing atherosclerotic processes and the neurodegenerative effects of Alzheimer’s. Experimental studies in animal models show that a deficiency of lipoic acid results in reduced muscle mass, failure to thrive, brain atrophy and increased lactic acid production.

REPLETION INFORMATION

Lipoic acid is available in tablets and capsules. Because of its unique solubility properties it is easily absorbed and assimilated. It is generally available as a racemic mixture of D- and L-forms of alpha lipoic acid. Patients with diabetes or glucose intolerance are cautioned that supplemental alpha lipoic acid may lower blood glucose levels and adjustments in antidiabetic drug therapy may be necessary to avoid hypoglycemia. Doses of up to 600 mg/day have been well tolerated.

Supplemental information micronutrient deficiencies



Oleic Acid

FUNCTION

Oleic acid is the most common monounsaturated fatty acid in human cells. Oleic acid is incorporated into cell membrane phospholipids, where it is important for important for proper membrane fluidity. Hormone responsiveness, infectivity of pathogens, mineral transport, and immune competence are affected by membrane fluidity.

Oleic acid is a major energy source for cells. Oleic acid is catabolized to acetyl groups used for energy (ATP) production and biosynthesis of many essential metabolites.

Oleic acid is obtained by cells from endogenous biosynthesis or from serum triglycerides. Biosynthesis of fatty acids (like oleic acid) utilizes the same enzymes responsible for elongation of other fatty acids which are precursors for eicosanoids (prostaglandins). Thus, deficient oleic acid status may also indicate deficient eicosanoid production, signifying a need for essential fatty acids.

DEFICIENCY SYMPTOMS

No deficiency symptoms are clearly defined for oleic acid since a dietary intake is not absolutely essential. Monounsaturated fat intake may be beneficial for reducing high blood cholesterol levels. A need for oleic acid may possibly reflect a need for essential fatty acids (linoleic acid, linolenic acid), or omega-3 fatty acids (alpha linolenic acid, EPA, and DHA).

REPLETION INFORMATION

Canola Oil
Olive Oil

Avocado Oil
Avocados

Almond Oil
High Oleic Safflower Oil

Although some margarines and shortenings are high in monounsaturated fats, a considerable amount is in the form of trans-monosaturated isomers (elaidic acid). Reductions in these foods are recommended to improve oleic acid status.

No RDA exists for oleic acid. No overt toxicity for fats rich in oleic acid is known, except for a laxative effect when consumed in large amounts (>50-100 grams per serving). Daily doses of 1-2 Tablespoons of oleic-rich oils (olive, canola, avocado) are usually adequate to add significant dietary amounts of oleic acid.

Although flaxseed oil (edible linseed oil) contains little oleic acid, it is an excellent source of the essential fatty acids, linoleic acid and linolenic (omega-3) acid. Daily doses of 1-2 Tablespoons per day will provide sufficient essential fatty acids to prevent essential fatty acid deficiencies.

Supplemental information micronutrient deficiencies



Choline

FUNCTION:

Choline is an essential nutrient that is part of cell membranes and is used by nerves to send impulses. Choline is known to be essential for mammals, and is essential for human cell growth. A dietary requirement for choline in humans has not been proven, although recent data on infants and dietary choline depletion in adults suggests that choline is an essential nutrient. Historically, choline is considered as a lipotrope and member of the B vitamin complex. Choline has several distinct functions. First, choline serves as a source of one-carbon units (methyl groups) for biosynthesis of other compounds. Interactions with methionine, Vitamin B12, folate, ethanolamine, and betaine allow choline to partially replace, or be replaced by other constituents in one-carbon metabolism. Second, choline is a component of phosphatidyl choline, the major component of cell membranes. Lecithin is a commercial name for phospholipids containing 10-35% phosphatidyl choline. Phosphatidyl choline has interactions with cholesterol and lipoprotein metabolism.

DEFICIENCY SYMPTOMS

Symptoms of Choline deficiency in humans primarily include: liver dysfunction and decreased serum cholesterol. Abnormal liver function resembling Choline deficiency symptoms in animals has been noticed long-term intravenous feeding (containing no Choline), and during malnutrition. Symptoms of inadequate cholinergic transmission may indicate an increased need for Choline.

REPLETION INFORMATION

Dietary sources richest in Choline (per serving) include:

Phosphatidyl Choline Supplements
Choline Supplements
Egg Yolks
Wheat Germ

Lecithin
Liver
Soy Products
Peanuts and Legumes

Lettuce
Brain and Organ Meats
Potatoes

At this time, there is not regulatory guideline for choline intake in humans. Usual dietary intake is from 0.5 – 1.0 gram daily. Choline intake can be accomplished by two types of choline forms: choline salts and phospholipids. Choline salts include choline chloride, choline bitartrate, and choline citrate. No apparent adverse effects after daily intakes of up to 10 grams of choline as choline salts have been reported. However, doses of 20 grams daily or more have been associated with symptoms of excess cholinergic stimulation (increased salivation, sweating, nausea, dizziness, depression, and ECG changes). Choline supplementation in the form of lecithin or phosphatidyl choline in daily doses of up to 100 grams appears to have no toxicity. However, occasional changes in bowel habits or upset stomachs appear, and the caloric content of additional lipids needs to be considered.

Supplemental information micronutrient deficiencies



Inositol

FUNCTION

An essential nutrient, inositol is found in cell membranes and is needed for proper function of hormones. Inositol, similar to choline, is a component of phospholipids (phosphatidyl inositols). Phosphatidyl inositols function as cell membrane components and as regulators of cell membrane transport by acting as a calcium-mobilizing system (the "PI effect"). Thus, inositol status interacts with a wide variety of hormonal and regulatory events in cells. Lipotropic activity (reduction of blood or tissue lipid levels) of inositol centers around the role of phosphatidyl inositol in lipoproteins. Since inositol is widely available from dietary sources, endogenous synthesis and gut microfloral synthesis, inositol is not classified as a vitamin. Nevertheless, inositol has been considered as a component of the B vitamin complex.

DEFICIENCY SYMPTOMS

Symptoms of Inositol deficiency in humans have not been reported conclusively, but may include alopecia, eczema, insomnia, constipation and hyperlipidemia. Animals fed diets lacking Inositol develop lipodystrophies (fatty livers, fatty intestines, low blood lipoproteins).

REPLETION INFORMATION

Dietary sources rich in Inositol include:

Nutritional Supplements
Whole Grains

Nuts Seeds
Citrus Fruits

Cantaloupes
Organ Meats

No RDA has been established for inositol. Usual dietary intake is one gram per day. Oral doses of up to 1-2 grams daily are well tolerated.

Supplemental information micronutrient deficiencies



Fructose Sensitivity

FUNCTION

Humans have a limited ability to metabolize fructose (fruit sugar). Fructose is metabolized differently from other sugars. A fructose load leads to accumulation of fructose-1-phosphate in cells which may partially deplete intracellular ATP levels in susceptible individuals. Decreased cellular ATP causes disturbances in protein, DNA & RNA synthesis, interference with cyclic AMP formation, and reduced ammonia detoxification. Elevations of lactate, uric acid, and triglycerides may result with implications for gout and cardiovascular disease. Several forms of hereditary fructose intolerance have been described.

DEFICIENCY SYMPTOMS

Since fructose intolerance is a cellular event, rather than a single nutrient deficiency, symptoms may vary widely among persons. Preliminary evidence suggests clinical symptoms of fructose intolerance may include fatigue, headaches, weakness, dizziness, behavioral changes, and depressed immune function. Medical literature suggests that certain individuals with fructose intolerance may show hypertriglyceridemia, elevation of uric acid, and interference with copper metabolism.

REPLETION INFORMATION

Dietary sources of fructose are numerous; however, an excess intake of fructose should be avoided, rather than absolute removal of dietary fructose. In this manner, whole foods containing fructose (fruits and some vegetables) may be consumed, in order to benefit from their overall nutritional value. Foods very rich in fructose include table sugar (sucrose), high fructose corn syrup, corn syrup, fruit juice concentrates and a large list of prepared foods containing sucrose and/or corn syrup. Reduction of excess dietary fructose intake by avoidance of foods very rich in fructose is suggested when fructose intolerance is exhibited.

Supplemental information micronutrient deficiencies



Glucose-Insulin Interaction

FUNCTION

A stimulation of lymphocyte growth by insulin may indicate a functional deficiency of insulin *in vivo*, or a metabolic defect in glucose utilization. At suboptimal glucose concentrations, supplementation of lymphocyte cultures with insulin exerted a sparing effect. This means that insulin addition makes uptake or utilization of glucose and amino acids more efficient, producing more cellular energy, and thus, a greater growth response. At optimal concentrations of glucose, insulin does not exert a sparing effect in healthy persons.

DEFICIENCY SYMPTOMS

Preliminary evidence suggests that persons with abnormal Glucose-Insulin Interaction exhibit hypoglycemia or hyperglycemia based on glucose tolerance testing. Morbidly obese persons with abnormal Glucose-Insulin Interaction may indicate insulin resistance. Thus, deficiency symptoms include fatigue, headaches, nausea, disorientation, dizziness, cold hands and feet, glucose intolerance.

REPLETION INFORMATION

Dietary suggestions are to replace, as much as possible, refined carbohydrates (table sugar, corn syrup, white flour, products made predominantly with white flour and/or sugar) with whole-food, unrefined carbohydrates (whole grain products, legumes, fruits). Reduce intake of foods with a high glycemic index. If clinically indicated, it is suggested that further laboratory testing of glucose and insulin metabolism be conducted (glucose tolerance test, glycosylated hemoglobin).

Since chromium status is closely linked with insulin function and glucose tolerance, a chromium deficiency is one possible reason for abnormal Glucose-Insulin Interaction. Therefore, consider improving chromium status. Chromium is depleted in refined foods, and rich in whole grains. Chromium supplements include Chromemate (chromium polynicotinate), chromium picolinate, and high-chromium yeast. A daily dose of 200 µg of chromium is sufficient for safe chromium repletion.

Supplemental information micronutrient deficiencies



SPECTROX® (Total Antioxidant Function)

FUNCTION

The function of antioxidants is to protect biomolecules from oxidative damage. SPECTROX measures the net ability of antioxidant and repair mechanisms of each individual's own cells, giving a total assessment of antioxidant function.

Oxidative Stress

Each person's cells and tissues are constantly subjected to highly reactive and unstable molecules termed *free radicals*, causing oxidative stress. These hostile molecules are a normal byproduct of life and are produced by the metabolism of oxygen, immune system cells, numerous enzyme reactions essential for metabolism, and environmental sources (smoke, ionizing radiation, air pollution, chemicals, toxic heavy metals and oxidized (rancid) fats. Some of the more common free radicals are superoxide, hydroxyl, singlet oxygen, and peroxides. By their chemical nature, free radicals, although short-lived, promote a chain reaction of radical formation, followed by a wake of chemically altered damaged biological molecules. Research is continuing to find that much biological damage and diseases are induced and/or mediated by injury from free radicals.

Cellular Antioxidants

Protection of deleterious effects from free radicals is found in a diverse range of molecules termed *antioxidants*. Free radicals and their chain reaction byproducts can be neutralized and converted to less harmful products (quenched) by antioxidants. Antioxidants are enzymes (superoxide dismutase, catalase, glutathione peroxidase), essential nutrients (carotenoids, vitamin C, vitamin E, cysteine, selenium) or a wide variety of endogenous compounds (glutathione, sulfhydryl groups, thioredoxin, lipoic acid, coenzyme Q₁₀, urate, bilirubin) or dietary compounds (mannitol, bioflavonoids, phenolic acid derivatives, proanthocyanidins). Antioxidants interact in a complex manner with recharging and overlapping, redundant functions. Cells also possess extensive mechanisms to repair damaged biomolecules, which appear protective in a total antioxidant function test.

Research evidence in humans indicates that deficient nutrient antioxidant intakes are associated with higher risks of arthritis, cancer, cardiovascular disease, cataracts and many other degenerative diseases. Also, higher intakes of nutrient antioxidants are associated with a lower incidence of chronic degenerative diseases. Studies have also shown that intervention with antioxidant nutrient supplements have therapeutic benefits in humans. Thus, scientific evidence illustrates that antioxidants help to prevent chronic degenerative diseases and may help to restore health.

REPLETION INFORMATION

Dietary sources richest in antioxidants (per serving) include:

fruits
green tea

prunes and raisins
seeds and nuts

garlic and onions
vegetables

nutritional supplements
wine

In persons with restricted acid intakes such as renal disease, vitamin C intake should be reduced to 50 mg to 100 mg daily, and buffered forms are preferred.