



Safe & Sound[®] Prenatal and Nursing Multi Vitamin & Mineral Formula

There is no time in life where nutrition brings more benefit and toxins create more risk than the time from conception through the first year of life. For most of that time, this brand new human being depends on mom for all of the nutrients needed to develop a strong and healthy body and mind as well as avoiding the many chemicals, pesticides and other toxins that have been shown or are suspected of causing birth defects, cancer, developmental problems and other diseases.

Our bodies cannot manufacture the vitamins and minerals needed to maintain health and quality of life. As a result we need to get them from our diets and/or in supplement form. Since the nutrient values of many farmed foods have decreased in recent years, we recommend a balanced prenatal multiple vitamin mineral formula to look, feel and function at your best and give your new child the best opportunity to develop and grow to a strong and healthy life.

What the nutrients in this Multi Vitamin & Mineral Formula are, why they are important, and the percentage of DV (RDA) present in the serving size:

Vitamin A – 100% DV

Function

- For the new embryo and growing fetal function, development of heart, cardiovascular, circulatory and central nervous systems and the regulation of heart asymmetry.
- Development of the retinoid receptor system necessary for normal eyesight function after birth.

Zile,H *Vitamin A and Embryonic Development: An Overview*^{1,2} Journal of Nutrition, 1998.

- Visual system and eyesight
- Regulation of gene expression
- Immunity
- Prenatal and postnatal development

- Red blood cell production (erythropoiesis)
<http://lpi.oregonstate.edu/mic/vitamins/vitamin-A>

- The function of vitamin A in the eye is that of a photoreceptor
- Function in other somatic cells is that of a growth factor.

Nutrition Reviews' Present Knowledge in Nutrition. Washington, D.C: Nutrition Foundation, 1984. Print.

- Functions of vitamin A include vision, cellular differentiation, morphogenesis, and transmembrane transport.
- The liver is of primary importance in the storage and utilization of vitamin A. The long-term ingestion of alcohol tends to reduce vitamin A storage.
- Fat malabsorption syndromes, such as cholestasis, cystic fibrosis, sprue, chronic diarrhea, pancreatic insufficiency, and biliary cirrhosis reduce the digestion and absorption of vitamin A and carotenoids and ultimately lead to a state of vitamin A depletion.

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

- Formation of rhodopsin (a photoreceptor pigment in the retina)
- Integrity of epithelia
- Lysosome stability
- Glycoprotein synthesis

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Vitamin A deficiency usually results from inadequate intakes of vitamin A from animal products (as preformed vitamin A) and fruit and vegetables (as provitamin A carotenoids). In developing countries, vitamin A deficiency and associated disorders predominantly affect children and women of reproductive age. Other individuals at risk of vitamin A deficiency are those with poor absorption of lipids due to impaired pancreatic or biliary secretion and those with inflammatory bowel diseases, such as Crohn's disease and celiac disease.

<http://lpi.oregonstate.edu/mic/vitamins/vitamin-A>

- Eye manifestations: Night blindness, Xerophthalmia
- Somatic manifestations: growth and development, skin sensation, hematopoiesis, resistance to infection, reproduction
- Early signs include loss of appetite, growth failure and impaired immune response with lowered resistance to infection. As depletion proceeds keratinization of epithelial tissues occurs in the eye, lung, exocrine glands, gastrointestinal and genitourinary tracts with reduction in mucous-secreting cells.
- Late changes in animals include impaired bone modeling with secondary effects on parts of the nervous system, hematopoietic changes, sterility, congenital malformations and ultimately death with secondary infections.

Nutrition Reviews' Present Knowledge in Nutrition. Washington, D.C: Nutrition Foundation, 1984. Print.

Vitamin A deficiency produces skin changes in humans, namely, follicular hyperkeratosis and phryoderma.

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

Daily Value (based on 2000 calorie diet)

5000 IU

Vitamin C – 133% DV

Function

- Vitamin C is a potent reducing agent, meaning that it readily donates electrons to recipient molecules. Related to this oxidation-reduction (redox) potential, two major functions of vitamin C are as an antioxidant and as an enzyme cofactor.
- Vitamin C is the primary water-soluble, non-enzymatic antioxidant in plasma and tissues. Even in small amounts vitamin C can protect indispensable molecules in the body, such as proteins, lipids (fats), carbohydrates, and nucleic acids (DNA and RNA), from damage by free radicals and reactive oxygen species (ROS) that are generated during normal metabolism, by active immune cells, and through exposure to toxins and pollutants (e.g., certain chemotherapy drugs and cigarette smoke). Vitamin C also participates in redox recycling of other important antioxidants; for example, vitamin C is known to regenerate vitamin E from its oxidized form.
- Vitamin C increases the bioavailability of iron from foods by enhancing intestinal absorption of non-heme iron.

<http://lpi.oregonstate.edu/mic/vitamins/vitamin-C>

- Absorption and movement of iron.
- Metabolism of fats and lipids, and cholesterol control.
- Sound teeth and bones

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Prevents scurvy
- causes several metabolic reactions to occur

<http://www.agriscience.msu.edu/>

- Collagen formation
- Bone and blood vessel health
- Carnitine, hormone, and amino acid formation
- Wound healing

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Severe vitamin C deficiency has been known for many centuries as the potentially fatal disease, scurvy. Symptoms of scurvy include subcutaneous bleeding, poor wound closure, and bruising easily, hair and tooth loss, and joint pain and swelling. Such symptoms appear to be related to the weakening of blood vessels, connective tissue, and bone, which all contain collagen. Early symptoms of scurvy like fatigue may result from diminished levels of carnitine, which is needed to derive energy from fat, or from decreased synthesis of the catecholamine norepinephrine.

<http://pi.oregonstate.edu/mic/vitamins/vitamin-C>

early symptoms, called latent scurvy:

- loss in weight
- listlessness
- fatigue
- fleeting pains in the joints and muscles
- irritability
- shortness of breath
- sore and bleeding gums
- small hemorrhages under the skin
- bones that fracture easily
- poor wound healing

scurvy:

- swollen, bleeding, and ulcerated gums
- loose teeth
- malformed and weak bones
- fragility of the capillaries with resulting hemorrhages throughout the body
- large bruises
- big joints, such as the knees and hips, due to bleeding into the joint cavity
- anemia
- degeneration of muscle fibers including those of the heart and tendency of old wounds to become red and break open

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Scurvy-edema
- weight loss
- diarrhea

<http://www.agriscience.msu.edu/>

Daily Value (based on 2000 calorie diet)

60mg

Vitamin D3 – 100% DV

Function

- Vitamin D can be synthesized in the skin upon exposure to sunlight and is then metabolized in the liver and kidney to the metabolically active form called 1,25-dihydroxyvitamin D. Through binding to the vitamin D receptor (VDR), 1,25-dihydroxyvitamin D can regulate the expression of hundreds of genes involved in skeletal and other biological functions.
- Vitamin D is essential for maintenance of bone mineralization through the regulation of calcium and phosphorus homeostasis. Vitamin D also exhibits many non-skeletal effects, particularly on the immune, endocrine, and cardiovascular systems.
- Vitamin D is important for normal bone development and maintenance. Severe vitamin D deficiency causes rickets in children and osteomalacia in adults.
- Vitamin D can regulate cell differentiation and growth by binding to the vitamin D receptor found in most body cells.
- Various observational studies have reported an association between vitamin D status and the susceptibility or severity of autoimmune diseases, including type 1 diabetes mellitus, multiple sclerosis, rheumatoid arthritis, and systemic lupus erythematosus.

<http://ipi.oregonstate.edu/mic/vitamins/vitamin-D>

- formed in the skin or absorbed through the gastrointestinal tract
- stimulates active transport of calcium across mucosal cells of the small intestine.
- stimulates the synthesis of proteins which mediate calcium transport by way of a nuclear receptor

Nutrition Reviews' Present Knowledge in Nutrition. Washington, D.C: Nutrition Foundation, 1984. Print.

- Enhanced calcium and potassium levels allowing bone mineralization
- prevents tetany

<http://www.agriscience.msu.edu/>

- Ca and phosphate absorption
- Mineralization and repair of bone
- Tubular reabsorption of Ca
- Insulin and thyroid function
- improvement of immune function
- reduced risk of autoimmune disease

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Severe vitamin D deficiency

- Rickets

- Osteomalacia
- Muscle weakness and pain

Risk factors for vitamin D deficiency

- Environmental conditions: Geographical locations, including latitude and altitudes, and atmospheric conditions (e.g., air pollution, presence of clouds) greatly influence the intensity of UVB radiation that reaches the ground. Seasonal changes also affect the quality and quantity of UVB rays and thus vitamin D production in skin.
- Concealed clothing style: In a study of 2,032 Middle Eastern women, who wore a headscarf or covered all skin for religious or cultural reasons, 96% had serum 25-hydroxyvitamin D levels less than 20 ng/mL, and 60% had vitamin D levels below 12 ng/mL. Rickets and osteomalacia are not uncommon in the Middle East and North African regions where children and women cover the majority or all of their skin whenever outside.
- Sun safety measures: Sun protection practices, including limiting sun exposure, wearing protective clothing and hats, and applying sunscreens, hinder skin exposure to sunlight and thus result in lower vitamin D₃ production and circulating vitamin D metabolites unless there is adequate oral intake. Of note, the application of sunscreen (2 mg/cm²) with a sun protection factor (SPF) of 10 reduces UVB radiation by 90%.
- Exclusively breast-fed infants: Infants who are exclusively breast-fed and do not receive vitamin D supplementation are at high risk for vitamin D deficiency, particularly if they have dark skin and/or receive little sun exposure .
- Skin pigmentation: People with a dark complexion synthesize less vitamin D on exposure to sunlight than those with light-colored skin.
- Genetic variations: Vitamin D bioavailability varies among individuals and is dependent on the level of circulating vitamin D-binding protein (DBP), a carrier protein that binds 85%-90% of circulating 25-hydroxyvitamin D.
- Older age: The elderly have reduced capacity to synthesize vitamin D in skin when exposed to UVB radiation and are more likely to stay indoors or use sunscreen, which prevents vitamin D synthesis.
- Chronic kidney disease (CKD): Vitamin D deficiency in patients with impaired renal function is due to a reduced synthesis of 1,25-dihydroxyvitamin D and an increased loss of 25-hydroxyvitamin D in urine.
- Fat malabsorption syndromes: Vitamin D deficiency is common among people with cystic fibrosis and both cholestatic and non-cholestatic liver diseases due to decreased absorption of dietary vitamin D and impaired conversion of vitamin D to 25-hydroxyvitamin D.
- Inflammatory bowel disease: People with inflammatory bowel disease like Crohn's disease appear to be at increased risk of vitamin D deficiency, especially those who have had small bowel resections.
- Obesity: Obesity (body mass index ≥ 30 kg/m²) increases the risk of vitamin D deficiency. Once vitamin D is synthesized in the skin or ingested, it can be sequestered in body fat stores, making it less bioavailable to people with higher body fat mass.
- Magnesium deficiency: Recent findings suggest that high magnesium intakes may reduce the risk of vitamin D insufficiency.

<http://lpi.oregonstate.edu/mic/vitamins/vitamin-D>

Vitamin D deficiency in children leads to the pathological bone condition of rickets (disordered cartilage cell growth and enlargement of the epiphyseal growth plates in the long bones). There is also a prominent accumulation of unmineralized bone matrix on trabecular bone surfaces. In the plasma of the vitamin D deficient child, there are also elevated levels of alkaline phosphatase and sometimes hypocalcemia and hypophosphatemia. In adults, vitamin D deficiency causes osteomalacia. *Nutrition Reviews' Present Knowledge in Nutrition*. Washington, D.C: Nutrition Foundation, 1984. Print.

- Abnormal skeletal development- lameness, bowed, and crooked legs
- slowed growth

<http://www.agriscience.msu.edu/>

Daily Value (based on 2000 calorie diet)
400 IU

Vitamin E – 100% DV

Function

- As an antioxidant which retards rancidification of fats In plant sources and in the digestive tracts of animals and protects body cells from toxic substances formed from the oxidation of unsaturated fatty acids.
- As an essential factor for the integrity of red blood cells
- as an agent essential to cellular respiration, primarily in heart and skeletal muscle tissues
- As a regulator in the synthesis of DNA, vitamin C, and coenzyme Q.
- as a protector of lung tissue from air pollution

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Intracellular antioxidant
 - Scavenger of free radicals in biologic membranes
- http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Vitamin E deficiency has been observed in individuals with severe malnutrition, genetic defects affecting the α -tocopherol transfer protein, and fat malabsorption syndromes. Severe vitamin E deficiency results mainly in neurological symptoms, including impaired balance and coordination (ataxia), injury to the sensory nerves (peripheral neuropathy), muscle weakness (myopathy), and damage to the retina of the eye (pigmented retinopathy). It should be noted that symptomatic vitamin E deficiency in healthy

individuals who consume diets low in vitamin E has never been reported.
<http://lpi.oregonstate.edu/mic/vitamins/vitamin-E>

- unable to absorb fat have low blood and tissue tocopherol levels
- increased red blood fragility
- shortened red blood cell life span
- Increased urinary excretion of creatine.
- Newborn infants, suffering from a deficiency of vitamin E, are characterized by edema, skin lesions, and blood abnormalities.

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

In humans, rapid development of vitamin E deficiency does not occur except in unusual clinical circumstances. In animals, vitamin E deficiency led to hemolysis and possible anemia, neuronal degeneration, nutritional "muscular dystrophy," myocardial necrosis and fibrosis, and reproductive failure.

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

- RBC hemolysis
 - neurologic deficits
- http://www.merckmanuals.com/professional/nutritional_disorders

- Failure of the reproductive system
 - changed cell permeability
 - muscular lesions
- <http://www.agriscience.msu.edu/>

Daily Value (based on 2000 calorie diet)
30 IU

Vitamin K – 50% DV

Function

- Vitamin K oxidation-reduction cycle
- Coagulation (clotting)
- Skeletal formation and prevention of soft tissue calcification
- Regulation of cellular functions

<http://lpi.oregonstate.edu/mic/vitamins/vitamin-K>

Vitamin K catalyzes the post-translational carboxylation of peptide-bound glutamate in various coagulation proenzymes and other vitamin K-dependent proteins.

Nutrition Reviews' Present Knowledge in Nutrition. Washington, D.C: Nutrition Foundation, 1984. Print.

Formation of prothrombin, other coagulation factors, and bone proteins

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Overt vitamin K deficiency results in impaired blood clotting, usually demonstrated by laboratory tests that measure clotting time. Symptoms include easy bruising and bleeding that may be manifested as nosebleeds, bleeding gums, blood in the urine, blood in the stool, tarry black stools, or extremely heavy menstrual bleeding. In infants, vitamin K deficiency may result in life-threatening bleeding within the skull (intracranial hemorrhage).

Vitamin K deficiency is uncommon in healthy adults for a number of reasons: (1) vitamin K is widespread in foods; (2) the vitamin K cycle conserves vitamin K; and (3) bacteria that normally inhabit the large intestine synthesize menaquinones (vitamin K₂), although it is unclear whether significant amounts are absorbed and utilized. Adults at risk for vitamin K deficiency include those taking vitamin K antagonists and individuals with significant liver damage or disease. Additionally, individuals with fat malabsorption disorders, including inflammatory bowel disease and cystic fibrosis, may be at increased risk of vitamin K deficiency.

<http://lpi.oregonstate.edu/mic/vitamins/vitamin-K>

Healthy persons are resistant to vitamin K deficiency but the deficiency can occur in debilitated patients on diets restricted in vitamin K, in patients on total parenteral nutrition and in patients receiving a variety of anticoagulant drugs including aspirin, hydantoins, 4-OH-coumarins and vitamin E.

Nutrition Reviews' Present Knowledge in Nutrition. Washington, D.C: Nutrition Foundation, 1984. Print.

Vitamin K deficiency in the breast-fed newborn remains a major worldwide cause of infant morbidity and mortality. The causes of vitamin K deficiency in humans are (1) hemorrhagic disease of the newborn (2) dietary inadequacy (dietary deficiency of vitamin K becomes manifest more quickly in patients following surgery and in debilitated patients with or without antibiotics) (3) total parenteral nutrition (4) biliary obstruction (5) malabsorption syndrome (6) liver disease (7) drug therapy

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

- Bleeding due to deficiency of prothrombin and other factors
- osteopenia

http://www.merckmanuals.com/professional/nutritional_disorders

- Long blood clot time
 - Hemorrhages
 - in severe cases, death
- <http://www.agriscience.msu.edu/>

Daily Value (based on 2000 calorie diet)
80 µg

Vitamin B1 – 100% DV

Function

Thiamin may well play three major roles at the cellular level. (1) energy metabolism (2) abnormal carbohydrate metabolism resulting from a lower transketolase activity (3) the function of membranes and nerve conduction. It was also suggested that thiamin plays a special role in neurophysiology.

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

- Carbohydrate, fat, amino acid, glucose, and alcohol metabolism
- Central and peripheral nerve cell function
- Myocardial function

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Thiamin deficiency affects the cardiovascular, nervous, muscular, gastrointestinal, and central and peripheral nervous systems. Beriberi has been subdivided into dry, wet, cerebral, or gastrointestinal, depending on the systems affected by severe thiamin deficiency.

- Dry beriberi: The main feature of dry (paralytic or nervous) beriberi is neuropathy. Early in the course of the neuropathy, "burning feet syndrome" may occur. Other symptoms include abnormal (exaggerated) reflexes, as well as diminished sensation and weakness in the legs and arms. Muscle pain and tenderness and difficulty rising from a squatting position have also been observed.
- Wet beriberi: In addition to neurologic symptoms, wet (cardiac) beriberi is characterized by cardiovascular manifestations of thiamin deficiency, which include rapid heart rate, enlargement of the heart, severe swelling (edema), difficulty breathing, and ultimately congestive heart failure.
- Cerebral beriberi: Cerebral beriberi may lead to Wernicke's encephalopathy and Korsakoff's psychosis, especially in people who abuse alcohol. The diagnosis of Wernicke's encephalopathy is based on a "triad" of signs, which include abnormal eye movements, stance and gait ataxia, and cognitive impairments. If left untreated, irreversible neurologic damage can cause additional clinical manifestations known as Korsakoff's psychosis. This syndrome—also called

Korsakoff's dementia, Korsakoff's amnesia, or amnestic confabulatory syndrome—involves a confused, apathetic state and a profound memory disorder, with severe amnesia and loss of recent and working memory.

- Gastrointestinal beriberi: TPP is critical for metabolic reactions that utilize glucose in glycolysis and the citric acid cycle. A decrease in the activity of thiamin-dependent enzymes limits the conversion of pyruvate to acetyl-CoA and the utilization of the citric acid cycle, leading to accumulation of pyruvate and lactate. Lactic acidosis, a condition resulting from the accumulation of lactate, is often associated with nausea, vomiting, and severe abdominal pain in a syndrome described as gastrointestinal beriberi.

Causes of thiamin deficiency: Thiamin deficiency may result from inadequate thiamin intake, increased requirement for thiamin, excessive loss of thiamin from the body, consumption of anti-thiamin factors in food, or a combination of these factors.

<http://lpi.oregonstate.edu/mic/vitamins/thiamin>

It affects the cardiovascular, muscular, nervous, and gastrointestinal systems.

Dietary factors are major causes of thiamin deficiency in Asia, whereas alcoholism is of greatest concern in the West. An increase in thiamin requirement due to strenuous physical exertion, fever, pregnancy, lactation, or adolescence will precipitate clinical manifestations in marginally nourished persons. Other persons at risk are renal patients on dialysis, patients on parenteral nutrition, and hypermetabolic patients.

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

- Beriberi (peripheral neuropathy, heart failure)
- Wernicke-Korsakoff syndrome

http://www.merckmanuals.com/professional/nutritional_disorders

- Anorexia
- Numbness
- Weakness
- stiffness in thighs
- unsteady walk
- edema in feet and legs
- painful along the spine

<http://www.agriscience.msu.edu/>

Daily Value (based on 2000 calorie diet)

1.5mg

Vitamin B2 – 100% DV

Function

- Oxidation-reduction (redox) reactions

Living organisms derive most of their energy from redox reactions, which are processes that involve the transfer of electrons. Flavocoenzymes participate in redox reactions in numerous metabolic pathways. They are critical for the metabolism of carbohydrates, lipids, and proteins. FAD is part of the electron transport (respiratory) chain, which is central to energy production. In conjunction with cytochrome P-450, flavocoenzymes also participate in the metabolism of drugs and toxins.

- Antioxidant functions

- Glutathione reductase is an FAD-dependent enzyme that participates in the redox cycle of glutathione.
- Glutathione peroxidases, selenium-containing enzymes, require two molecules of reduced glutathione to break down hydroperoxides. GPx are involved in the glutathione oxidation-reduction (redox) cycle.
- Xanthine oxidase, another FAD-dependent enzyme, catalyzes the oxidation of hypoxanthine and xanthine to uric acid. Uric acid is one of the most effective water-soluble antioxidants in the blood. Riboflavin deficiency can result in decreased xanthine oxidase activity, reducing blood uric acid levels.

<http://lpi.oregonstate.edu/mic/vitamins/riboflavin>

In bound coenzymic form, riboflavin participates in oxidation-reduction reactions in numerous metabolic pathways and in energy production via the respiratory chain. Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

- Many aspects of carbohydrate and protein metabolism
- Integrity of mucous membranes

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Riboflavin deficiency is rarely found in isolation; it occurs frequently in combination with deficiencies of other water-soluble vitamins. Symptoms of riboflavin deficiency include sore throat, redness and swelling of the lining of the mouth and throat, cracks or sores on the outsides of the lips (cheliosis) and at the corners of the mouth (angular stomatitis), inflammation and redness of the tongue (magenta tongue), and a moist, scaly skin inflammation (seborrheic dermatitis). Other symptoms may involve the formation of blood vessels in the clear covering of the eye (vascularization of the cornea) and decreased red blood cell count in which the existing red blood cells contain normal levels of hemoglobin and are of normal size (normochromic normocytic anemia). Severe riboflavin deficiency may result in decreased conversion of vitamin B6 to its coenzyme form (PLP) and decreased conversion of tryptophan to niacin.

- Risk factors for riboflavin deficiency: Alcoholics are at increased risk for riboflavin deficiency due to decreased intake, decreased absorption, and impaired utilization of riboflavin. Interestingly, the elevated homocysteine levels associated with riboflavin deficiency rapidly decline during alcohol withdrawal. Additionally, anorexic

individuals rarely consume adequate riboflavin, and lactose intolerant individuals may not consume milk or other dairy products that are good sources of riboflavin. The conversion of riboflavin into FAD and FMN is impaired in hypothyroidism and adrenal insufficiency. Further, people who are very active physically (athletes, laborers) may have a slightly increased riboflavin requirement. However, riboflavin supplementation has not generally been found to increase exercise tolerance or performance.

<http://pi.oregonstate.edu/mic/vitamins/riboflavin>

- sore throat
- hyperemia and edema of the pharyngeal and oral mucous membranes
- cheilosis
- angular stomatitis
- glossitis
- seborrheic dermatitis
- normochromic, normocytic anemia associated with pure red cell cytoplasia of the bone marrow
- Severe riboflavin deficiency can also affect the conversion of vitamin B6 to its conenzyme and even curtail conversion of tryptophan to niacin.

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

- Cheilosis
- angular stomatitis
- corneal vascularization

http://www.merckmanuals.com/professional/nutritional_disorders

- Reduced growth rate
- skin lesions
- hair loss

<http://www.agriscience.msu.edu/>

Daily Value (based on 2000 calorie diet)

1.7mg

Vitamin B3 – 100% DV

Function

- Oxidation-reduction (redox) reactions
- Living organisms derive most of their energy from oxidation-reduction (redox) reactions, which are processes involving the transfer of electrons. Over 400 enzymes require the niacin coenzymes, NAD and NADP, mainly to accept or donate electrons for redox reactions.

- Non-redox reactions

The niacin coenzyme, NAD, is the substrate (reactant) for at least four classes of enzymes that separate the nicotinamide moiety from NAD and transfer ADP-ribose to acceptors.

<http://lpi.oregonstate.edu/mic/vitamins/niacin>

Niacin has a pervasive role in metabolism functioning in catabolic reactions, particularly in energy-related pathways and also in biosynthetic processes.

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

- Oxidation-reduction reactions
- Carbohydrate and cell metabolism

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

The most common symptoms of niacin deficiency involve the skin, the digestive system, and the nervous system. The symptoms of pellagra are commonly referred to as the three D's: dermatitis, diarrhea, and dementia. A fourth D, death, occurs if pellagra is left untreated. In the skin, a thick, scaly, darkly pigmented rash develops symmetrically in areas exposed to sunlight. Symptoms related to the digestive system include inflammation of the mouth and tongue ("bright red tongue"), vomiting, constipation, abdominal pain, and ultimately, diarrhea. Gastrointestinal disorders and diarrhea contribute to the ongoing malnourishment of the subjects. Neurologic symptoms include headache, apathy, fatigue, depression, disorientation, and memory loss and are more consistent with delirium than with the historically described dementia.

<http://lpi.oregonstate.edu/mic/vitamins/niacin>

Pellagra

- Dermatitis
- inflammation of mucous membranes
- rectal irritation
- psychic changes, such as irritability, anxiety, depression, and in advanced cases, delirium, hallucinations, confusion, disorientation, and stupor

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Retarded growth
- decreased appetite
- diarrhea
- vomiting
- dermatitis

<http://www.agriscience.msu.edu>

Daily Value (based on 2000 calorie diet)

20mg

Vitamin B6 – 100% DV

Function

- Nervous system function
- Hemoglobin synthesis and function
- Tryptophan metabolism
- Hormone function
- Nucleic acid synthesis

<http://lpi.oregonstate.edu/mic/vitamins/vitamin-B6>

- Many aspects of nitrogen metabolism (eg, transaminations, porphyrin and heme synthesis, tryptophan conversion to niacin)
- Nucleic acid biosynthesis
- Fatty acid, lipid, and amino acid metabolism

http://www.merckmanuals.com/professional/nutritional_disorders

- protein metabolism
- carbohydrate and fat metabolism
- clinical problems, including central nervous system disturbances, autism, anemia, kidney stones, tuberculosis, physiologic demands in pregnancy, and oral contraceptives

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Severe deficiency of vitamin B6 is uncommon. Alcoholics are thought to be most at risk of vitamin B6 deficiency due to low dietary intakes and impaired metabolism of the vitamin. Abnormal electroencephalogram (EEG) patterns have also been reported in vitamin B6-deficient adults. Other neurologic symptoms observed in severe vitamin B6 deficiency include irritability, depression, and confusion; additional symptoms include inflammation of the tongue, sores or ulcers of the mouth, and ulcers of the skin at the corners of the mouth.

<http://lpi.oregonstate.edu/mic/vitamins/vitamin-B6>

- greasy scaliness in the skin around the eyes, nose, and mouth
- a smooth, red tongue
- loss of weight
- muscular weakness
- irritability
- mental depression

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Seizures
- Anemia
- Neuropathies
- seborrheic dermatitis

http://www.merckmanuals.com/professional/nutritional_disorders

Daily Value (based on 2000 calorie diet)

2mg

Folate – 100% DV

Function

One-carbon metabolism: The only function of folate coenzymes in the body appears to be in mediating the transfer of one-carbon units. Folate coenzymes act as acceptors and donors of one-carbon units in a variety of reactions critical to the metabolism of nucleic acids and amino acids.

- Nucleic acid metabolism: Folate coenzymes play a vital role in DNA metabolism.
- Amino acid metabolism: Folate coenzymes are required for the metabolism of several important amino acids, namely methionine, cysteine, serine, glycine, and histidine.

<http://pi.oregonstate.edu/mic/vitamins/folate>

- Maturation of RBCs
- Synthesis of purines, pyrimidines, and methionine
- Development of fetal nervous system

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Causes

Folate deficiency is most often caused by a dietary insufficiency; however, folate deficiency can also occur in a number of other situations. For example, chronic and heavy alcohol consumption is associated with diminished absorption of folate (in addition to low dietary intake), which can lead to folate deficiency. Smoking is also associated with low folate status. Additionally, impaired folate transport to the fetus has been described in pregnant women who either smoked or abused alcohol during their pregnancy. Moreover, folate deficiency can result from some malabsorptive conditions, including inflammatory bowel diseases (Crohn's disease and ulcerative colitis) and celiac disease. Several medications may also contribute to folate deficiency. Finally, a number of genetic diseases affecting folate absorption, transport, or metabolism can cause folate deficiency or impede its metabolic functions.

Symptoms

Clinical folate deficiency leads to megaloblastic anemia, which is reversible with folic acid treatment. Rapidly dividing cells like those derived from bone marrow are most vulnerable to the effects of folate deficiency since DNA synthesis and cell division are dependent on folate coenzymes. When folate supply to the rapidly dividing cells of the bone marrow is inadequate, blood cell division is reduced, resulting in fewer but larger red blood cells. This type of anemia is called megaloblastic or macrocytic anemia, referring to the enlarged, immature red blood cells. Neutrophils, a type of white blood cell, become hypersegmented, a change that can be found by examining a blood sample microscopically. Because normal red blood cells have a lifetime in the circulation of approximately four months, it can take months for folate-deficient individuals to develop the characteristic megaloblastic anemia. Progression of such an anemia leads to a decreased oxygen carrying capacity of the blood and may ultimately result in symptoms of fatigue, weakness, and shortness of breath.

<http://pi.oregonstate.edu/mic/vitamins/folate>

- Sore, red, smooth tongue
- disturbances of the digestive tract
- poor growth

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Megaloblastic anemia
- neural tube birth defects
- confusion

http://www.merckmanuals.com/professional/nutritional_disorders

Daily Value (based on 2000 calorie diet)

400 µg

Vitamin B12 – 100% DV

Function

- Vitamin B12 or cobalamin plays essential roles in folate metabolism and in the synthesis of the citric acid cycle intermediate, succinyl-CoA.
- Vitamin B12 and folate are important for homocysteine metabolism. Elevated homocysteine levels in blood are a risk factor for cardiovascular disease (CVD).
- Vitamin B12 is essential for the preservation of the myelin sheath around neurons and for the synthesis of neurotransmitters.

<http://pi.oregonstate.edu/mic/vitamins/vitamin-B12>

- Maturation of RBCs
- neural function

- DNA synthesis
 - myelin synthesis and repair
- http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

In healthy adults, vitamin B12 deficiency is uncommon, mainly because total body stores can exceed 2,500 mcg, daily turnover is slow, and dietary intake of only 24 mcg/day is sufficient to maintain adequate vitamin B12 status (see RDA). In elderly individuals, vitamin B12 deficiency is more common mainly because of impaired intestinal absorption that can result in marginal to severe vitamin B12 deficiency in this population.

- Causes of vitamin B12 deficiency

Intestinal malabsorption, rather than inadequate dietary intake, can explain most cases of vitamin B12 deficiency. The prevalent causes of vitamin B12 deficiency are (1) an autoimmune condition known as pernicious anemia, and (2) a disorder called food-bound vitamin B12 malabsorption. Both conditions have been associated with a chronic inflammatory disease of the stomach known as atrophic gastritis.

- Other causes of vitamin B12 deficiency

Other causes of vitamin B12 deficiency include surgical resection of the stomach or portions of the small intestine where receptors for the IF-B12 complex are located. Conditions affecting the small intestine, such as malabsorption syndromes (celiac disease and tropical sprue), may also result in vitamin B12 deficiency. Because the pancreas provides critical enzymes, as well as calcium required for vitamin B12 absorption, pancreatic insufficiency may contribute to vitamin B12 deficiency. Since vitamin B12 is found only in foods of animal origin, a strict vegetarian (vegan) diet has resulted in cases of vitamin B12 deficiency. Moreover, alcoholics may experience reduced intestinal absorption of vitamin B12, and individuals with acquired immunodeficiency syndrome (AIDS) appear to be at increased risk of deficiency, possibly related to a failure of the IF-B12 receptor to take up the IF-B12 complex. Further, long-term use of acid-reducing drugs has also been implicated in vitamin B12 deficiency.

- Symptoms of vitamin B12 deficiency

- Megaloblastic anemia
- Neurologic symptoms: numbness and tingling of the hands and, more commonly, the feet; difficulty walking; memory loss; disorientation; and dementia with or without mood changes
- Gastrointestinal symptoms: Tongue soreness, appetite loss, and constipation

<http://lpi.oregonstate.edu/mic/vitamins/vitamin-B12>

Megaloblastic anemia, neurologic deficits (confusion, paresthesias, ataxia)
http://www.merckmanuals.com/professional/nutritional_disorders

It may occur as a result of dietary lack, deficiency of intrinsic factor due to pernicious anemia, total or partial removal of the stomach by surgery, or infestation with parasites
Common symptoms:

- sore tongue
- weakness
- loss of weight
- back pains
- tingling of the extremities
- apathy
- mental and other nervous abnormalities

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value (based on 2000 calorie diet)

6 µg

Biotin – 100% DV

Function

Biotin is attached at the active site of five mammalian enzymes known as carboxylases. The attachment of biotin to another molecule, such as a protein, is known as "biotinylation." Holocarboxylase synthetase (HCS) catalyzes the biotinylation of apocarboxylases (i.e., the catalytically inactive form of the enzyme) and of histones (see Histone biotinylation). Biotinidase catalyzes the release of biotin from histones and from the peptide products of carboxylase breakdown.

- Enzyme cofactor: Each carboxylase catalyzes an essential metabolic reaction
 - Acetyl-CoA carboxylase I and II catalyze the binding of bicarbonate to acetyl-CoA to form malonyl-CoA. Malonyl-CoA is required for the synthesis of fatty acids. The former is crucial in cytosolic fatty acid synthesis, and the latter functions in regulating mitochondrial fatty acid oxidation.
 - Pyruvate carboxylase is a critical enzyme in gluconeogenesis—the formation of glucose from sources other than carbohydrates, for example, amino acids.
 - Methylcrotonyl-CoA carboxylase catalyzes an essential step in the catabolism of leucine, an essential amino acid.
 - Propionyl-CoA carboxylase catalyzes essential steps in the metabolism of certain amino acids, cholesterol, and odd chain fatty acids (fatty acids with an odd number of carbon molecules).
- Histone biotinylation: Histones are proteins that bind to DNA and package it into compact structures to form nucleosomes—integral structural components of chromosomes. The compact packaging of DNA must be relaxed somewhat for DNA replication and transcription to occur. Modification of histones through the attachment of acetyl or methyl groups (acetylation or methylation) has been shown to affect the structure of histones, thereby affecting replication and transcription of DNA. Mounting evidence indicates that biotinylation of histones plays a role in regulating DNA replication and transcription, as well as cellular proliferation and other cellular responses.

<http://ipi.oregonstate.edu/mic/vitamins/biotin>

It is required for many reactions in the metabolism of carbohydrates, fats, and proteins. It functions as a coenzyme mainly in decarboxylation-carboxylation and in deamination. Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Although overt biotin deficiency is very rare, the human requirement for dietary biotin has been demonstrated in two different situations: prolonged intravenous feeding (parenteral) without biotin supplementation and consumption of raw egg white for a prolonged period (many weeks to years). Avidin is an antimicrobial protein found in egg white that binds biotin and prevents its absorption. Cooking egg white denatures avidin, rendering it susceptible to digestion and therefore unable to prevent the absorption of dietary biotin. Signs and symptoms include:

- Hair loss and a scaly red rash around the eyes, nose, mouth, and genital area.
- depression, lethargy, hallucination, and numbness and tingling of the extremities.
- The characteristic facial rash, together with unusual facial fat distribution, has been termed the "biotin deficient facies" by some investigators.

<http://pi.oregonstate.edu/mic/vitamins/biotin>

- a dry scaly dermatitis
- loss of appetite
- nausea
- vomiting
- muscle pains
- glossitis
- pallor of skin
- mental depression
- a decrease in hemoglobin and red blood cells
- a high cholesterol level
- a low excretion of biotin

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value (based on 2000 calorie diet)

300 µg

Pantothenic Acid – 100% DV

Function

- Coenzyme A
- Pantothenic acid is a component of coenzyme A (CoA), an essential coenzyme in a

variety of reactions that sustain life. CoA is required for chemical reactions that generate energy from food (fat, carbohydrates, and proteins). The synthesis of essential fats, cholesterol, and steroid hormones requires CoA, as does the synthesis of the neurotransmitter, acetylcholine, and the hormone, melatonin. Heme, a component of hemoglobin, requires a CoA-containing compound for its synthesis. Metabolism of a number of drugs and toxins by the liver requires CoA.

- Acyl-carrier protein

The acyl-carrier protein requires pantothenic acid in the form of 4'-phosphopantetheine for its activity as an enzyme. Both CoA and the acyl-carrier protein are required for the synthesis of fatty acids.

<http://ipi.oregonstate.edu/mic/vitamins/pantothenic-acid>

It functions in the body as part of two enzymes, coenzyme A and acyl carrier protein.

Coenzyme A functions in the following important reactions:

1. the metabolic processes by which carbohydrates, fats, and proteins are broken down and energy is released
2. the formation of acetyl choline, a substance of importance in transmitting nerve impulses
3. the synthesis of porphyrin, a precursor of heme, of importance in hemoglobin synthesis
4. the synthesis of cholesterol and other sterols
5. the steroid hormones formed by the adrenal and sex glands
6. the maintenance of normal blood sugar, and the formation of antibodies
7. the excretion of sulfonamide drugs

Acyl carrier protein, along with coenzyme A, required by the cells in the biosynthesis of fatty acids

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Naturally occurring pantothenic acid deficiency in humans is very rare and has been observed only in cases of severe malnutrition. World War II prisoners in the Philippines, Burma, and Japan experienced numbness and painful burning and tingling in their feet; these symptoms were relieved specifically by pantothenic acid. Pantothenic acid deficiency in humans has been induced experimentally by co-administering a pantothenic acid antagonist and a pantothenic acid-deficient diet. Participants in this experiment complained of headache, fatigue, insomnia, intestinal disturbances, and numbness and tingling of their hands and feet. In a more recent study, participants fed only a pantothenic acid free diet did not develop clinical signs of deficiency, although some appeared listless and complained of fatigue. Homopantothenate is a pantothenic acid antagonist with cholinergic effects (similar to those of the neurotransmitter, acetylcholine). It was used in Japan to enhance mental function, especially in Alzheimer's disease. A rare side effect was the development of hepatic encephalopathy, a condition of abnormal brain function resulting from the failure of the liver to eliminate toxins. The encephalopathy was reversed by pantothenic acid supplementation,

suggesting but not proving it was due to pantothenic acid deficiency caused by the antagonist.

<http://lpi.oregonstate.edu/mic/vitamins/pantothenic-acid>

- irritableness and restlessness
- loss of appetite, indigestion, abdominal pains, nausea
- headache
- sullenness, mental depression, fatigue, weakness
- numbness and tingling of hands and feet, muscle cramps in the arms and legs
- burning sensation in the feet
- insomnia
- respiratory infections
- rapid pulse
- and a staggering gait
- increased reaction to stress
- increased sensitivity to insulin, resulting in low blood sugar levels
- increased sedimentation rate for erythrocytes
- decreased gastric secretions
- marked decrease in antibody production

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value (based on 2000 calorie diet)

10mg

Calcium – 20% DV

Function

- Structure: Calcium is a major structural element in bones and teeth. The mineral component of bone consists mainly of hydroxyapatite $[Ca_{10}(PO_4)_6(OH)_2]$ crystals, which contain large amounts of calcium, phosphorus, and oxygen.
- Calcium homeostasis: Calcium concentrations in the blood and fluid that surround cells are tightly controlled in order to preserve normal physiological function.
- Cell signaling: Calcium plays a role in mediating the constriction and relaxation of blood vessels (vasoconstriction and vasodilation), nerve impulse transmission, muscle contraction, and the secretion of hormones like insulin. Excitable cells, such as skeletal muscle and nerve cells, contain voltage-dependent calcium channels in their cell membranes that allow for rapid changes in calcium concentrations.
- Regulation of protein function: Calcium is necessary to stabilize a number of proteins, including enzymes, optimizing their activities. The binding of calcium ions is required for the activation of the seven "vitamin K-dependent" clotting factors in the coagulation cascade.

<http://lpi.oregonstate.edu/infocenter/minerals/calcium/>

- build bones and teeth and maintain bones
- blood clotting
- muscle contraction and relaxation
- nerve transmission
- cell wall permeability
- enzyme activation
- secretion of a number of hormones and hormone releasing factors

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

A low blood calcium level (hypocalcemia) usually implies abnormal parathyroid function since the skeleton provides a large reserve of calcium for maintaining normal blood levels, especially in the case of low dietary calcium intake.

Other causes of abnormally low blood calcium concentrations include chronic kidney failure, vitamin D deficiency, and low blood magnesium levels often observed in cases of severe alcoholism. Magnesium deficiency can impair parathyroid hormone (PTH) secretion by the parathyroid glands and lower the responsiveness of osteoclasts to PTH. Thus, magnesium supplementation is required to correct hypocalcemia in people with low serum magnesium concentrations. Chronically low calcium intakes in growing individuals may prevent the attainment of optimal peak bone mass. Once peak bone mass is achieved, inadequate calcium intake may contribute to accelerated bone loss and ultimately to the development of osteoporosis

<http://lpi.oregonstate.edu/infocenter/minerals/calcium/>

- stunting of growth
- poor quality bones and teeth
- malformation of bones
- osteoporosis
- hypercalcemia
- tetany
- kidney stones

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value (based on 2000 calorie diet)

1000mg

Iodine – 100% DV

Function

Iodine is an essential component of the thyroid hormones, triiodothyronine (T3) and thyroxine (T4), and is therefore essential for normal thyroid function.

<http://lpi.oregonstate.edu/mic/minerals/iodine>

- Thyroxine (T4) and triiodothyronine (T3) synthesis
- Development of fetus

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Iodine deficiency is now accepted as the most common cause of preventable brain damage in the world. The spectrum of iodine deficiency disorders (IDD) includes mental retardation, hypothyroidism, goiter, and varying degrees of other growth and developmental abnormalities. Thyroid enlargement, or goiter, is one of the earliest and most visible signs of iodine deficiency. In mild iodine deficiency, this adaptive response may be enough to provide the body with sufficient thyroid hormone. However, more severe cases of iodine deficiency result in hypothyroidism. Iodine deficiency has adverse effects in all stages of development but is most damaging to the developing brain. In addition to regulating many aspects of growth and development, thyroid hormone is important for myelination of the central nervous system, which is most active before and shortly after birth.

<http://lpi.oregonstate.edu/mic/minerals/iodine>

- Simple (colloid, endemic) goiter
- Cretinism
- Deaf-mutism
- Impaired fetal growth and brain development

http://www.merckmanuals.com/professional/nutritional_disorders

- Goiter- enlargement of the thyroid gland
- Dry skin, brittle hair
- Young born

<http://www.agriscience.msu.edu>

Daily Value (based on 2000 calorie diet)

150 µg

Magnesium – 25% DV

Function

Magnesium is involved in more than 300 essential metabolic reactions, some of which are discussed below.

- Energy production: The metabolism of carbohydrates and fats to produce energy requires numerous magnesium-dependent chemical reactions.

- Synthesis of essential molecules: Magnesium is required for a number of steps during synthesis of deoxyribonucleic acid (DNA), ribonucleic acid (RNA), and proteins. Several enzymes participating in the synthesis of carbohydrates and lipids require magnesium for their activity. Glutathione, an important antioxidant, requires magnesium for its synthesis.
- Structural roles: Magnesium plays a structural role in bone, cell membranes, and chromosomes.
- Ion transport across cell membranes: Magnesium is required for the active transport of ions like potassium and calcium across cell membranes. Through its role in ion transport systems, magnesium affects the conduction of nerve impulses, muscle contraction, and normal heart rhythm.
- Cell signaling: Cell signaling requires MgATP for the phosphorylation of proteins and the formation of the cell-signaling molecule, cyclic adenosine monophosphate (cAMP).
- Cell migration: Calcium and magnesium levels in the fluid surrounding cells affect the migration of a number of different cell types. Such effects on cell migration may be important in wound healing.

<http://lpi.oregonstate.edu/mic/minerals/magnesium#function>

- Constituent of bones and teeth.
 - Essential element of cellular metabolism.
 - Relaxes nerve impulses, functioning antagonistically to calcium which is stimulatory
- Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Magnesium deficiency in healthy individuals who are consuming a balanced diet is quite rare. The following conditions increase the risk of magnesium deficiency:

- Gastrointestinal disorders: Prolonged diarrhea, Crohn's disease, malabsorption syndromes, celiac disease, surgical removal of a portion of the intestine, and intestinal inflammation due to radiation may all lead to magnesium depletion.
- Renal disorders (magnesium wasting): Diabetes mellitus and long-term use of certain diuretics may result in increased urinary loss of magnesium. Multiple other medications can also result in renal magnesium wasting.
- Chronic alcoholism: Poor dietary intake, gastrointestinal problems, and increased urinary loss of magnesium may all contribute to magnesium depletion, which is frequently encountered in alcoholics.
- Age: Several studies have found that elderly people have relatively low dietary intakes of magnesium. Intestinal magnesium absorption tends to decrease with age and urinary magnesium excretion tends to increase with age; thus, suboptimal dietary magnesium intake may increase the risk of magnesium depletion in the elderly.

<http://lpi.oregonstate.edu/mic/minerals/magnesium#function>

- muscle spasms (tremor, twitching)

- rapid heartbeat
- confusion, hallucinations and disorientation
- lack of appetite, listlessness, nausea, and vomiting

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Anorexia
- Reduced weight gain
- Hyperemia of the ears and other extremities

<http://www.agriscience.msu.edu>

Daily Value (based on 2000 calorie diet)

400mg

Zinc – 100% DV

Function

Numerous aspects of cellular metabolism are zinc-dependent. Zinc plays important roles in growth and development, the immune response, neurological function, and reproduction. On the cellular level, the function of zinc can be divided into three categories:

1. Catalytic role: Over 300 different enzymes depend on zinc for their ability to catalyze vital chemical reactions.
2. Structural role: Zinc plays an important role in the structure of proteins and cell membranes. The structure and function of cell membranes are also affected by zinc. Loss of zinc from biological membranes increases their susceptibility to oxidative damage and impairs their function.
3. Regulatory role: Zinc finger proteins have been found to regulate gene expression by acting as transcription factors (binding to DNA and influencing the transcription of specific genes). Zinc also plays a role in cell signaling and has been found to influence hormone release and nerve impulse transmission. Zinc has been found to play a role in apoptosis (gene-directed cell death), a critical cellular regulatory process with implications for growth and development, as well as a number of chronic diseases.

<http://pi.oregonstate.edu/mic/minerals/zinc>

- Needed for normal skin, bones, and hair
- As a component of several different enzyme systems which are involved in digestion and respiration
- Required for the transfer of carbon dioxide in red blood cells
- Required for proper calcification of bones
- Required for the synthesis and metabolism of protein and nucleic acids
- Required for the development and functioning of reproductive organs

- Required for wound and burn healing; for the functioning of insulin; and for normal taste acuity

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Severe zinc deficiency:

- the slowing or cessation of growth and development
- delayed sexual maturation
- characteristic skin rashes
- chronic and severe diarrhea
- immune system deficiencies
- impaired wound healing
- diminished appetite
- impaired taste sensation
- night blindness
- swelling and clouding of the corneas
- Behavioral disturbances.

Individuals at risk of zinc deficiency:

- Premature and low-birth-weight infants
- Older breast-fed infants and toddlers with inadequate intake of zinc-rich complementary foods
- Children and adolescents
- Pregnant and lactating (breast-feeding) women, especially adolescents
- Patients receiving total parenteral nutrition (intravenous feedings)
- Malnourished individuals, including those with protein-energy malnutrition and anorexia nervosa
- Individuals with severe or persistent diarrhea
- Individuals with malabsorption syndromes, including celiac disease and short bowel syndrome
- Individuals with inflammatory bowel disease, including Crohn's disease and ulcerative colitis
- Alcoholics and those with alcoholic liver disease who have increased urinary zinc excretion and low liver zinc levels
- Individuals with chronic renal disease
- Individuals with sickle cell anemia
- Individuals who use medications that decrease intestinal zinc absorption, increase zinc excretion, or impair zinc utilization (see Drug interactions below)
- Older adults (65 years and older)
- Strict vegetarians: The requirement for dietary zinc may be as much as 50% greater for strict vegetarians whose major food staples are grains and legumes, because high levels of phytic acid in these foods reduce zinc absorption.

<http://lpi.oregonstate.edu/mic/minerals/zinc>

Deficiency symptoms:

- loss of appetite
- stunted growth in children
- skin changes
- small sex glands in boys
- loss of taste sensitivity
- lightened pigment in hair
- white spots on the fingernails
- delayed healing of wounds.

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value (based on 2000 calorie diet)

15 mg

Selenium – 100% DV

Function

Humans and animals require selenium for the function of a number of selenium-dependent enzymes, also known as selenoproteins. During selenoprotein synthesis, selenocysteine is incorporated into a very specific location in the amino acid sequence in order to form a functional protein.

<http://pi.oregonstate.edu/mic/minerals/selenium>

Component of glutathione peroxidase and thyroid hormone iodinase

http://www.merckmanuals.com/professional/nutritional_disorders

Deficiency

Insufficient selenium intake results in decreased activity of the glutathione peroxidases as well as some other thioredoxin reductase and thyroid deiodinases. Even when severe, isolated selenium deficiency does not usually result in obvious clinical illness. However, selenium-deficient individuals appear to be more susceptible to additional physiological stresses.

- Individuals at increased risk of selenium deficiency: Clinical selenium deficiency has been observed in chronically ill patients who were receiving total parenteral nutrition (TPN) without added selenium for prolonged periods of time. Muscular weakness, muscle wasting, and cardiomyopathy (inflammation and damage to the heart muscle) have been observed in these patients. People who have had a large portion of the small intestine surgically removed or those with severe gastrointestinal problems, such as Crohn's disease, are also at risk for selenium deficiency due to impaired absorption. Specialized medical diets used to treat metabolic disorders, such as phenylketonuria (PKU), are often low in selenium. Specialized diets that will

be used exclusively over long periods of time should have their selenium content assessed to determine the need for selenium supplementation.

- Keshan disease: Keshan disease is a cardiomyopathy that affects young women and children in a selenium-deficient region of China. The acute form of the disease is characterized by the sudden onset of cardiac insufficiency, while the chronic form results in moderate to severe heart enlargement with varying degrees of cardiac insufficiency. The incidence of Keshan disease is closely associated with very low dietary intakes of selenium and poor selenium nutritional status.
- Kashin-Beck disease: Kashin-Beck disease is characterized by the degeneration of articular cartilage between joints (osteoarthritis) and is associated with poor selenium status in areas of northern China, North Korea, and eastern Siberia. The disease affects children between the ages 5 and 13 years. Severe forms of the disease may result in joint deformities and dwarfism. Unlike Keshan disease, there is little evidence that improving selenium nutritional status prevents Kashin-Beck disease. Thus, the role of selenium deficiency in the etiology of Kashin-Beck disease is less certain. A number of other causative factors have been suggested for Kashin-Beck disease, including fungal toxins in grain, iodine deficiency, and contaminated drinking water.

<http://lpi.oregonstate.edu/mic/minerals/selenium>

Daily Value (based on 2000 calorie diet)

70 µg

Manganese – 100% DV

Function

Manganese (Mn) plays an important role in a number of physiologic processes as a constituent of multiple enzymes and an activator of other enzymes.

- Antioxidant function: Manganese superoxide dismutase (MnSOD) is the principal antioxidant enzyme in the mitochondria.
- Metabolism: A number of manganese-activated enzymes play important roles in the metabolism of carbohydrates, amino acids, and cholesterol.
- Bone development: Manganese deficiency results in abnormal skeletal development in a number of animal species. Manganese is the preferred cofactor of enzymes called glycosyltransferases; these enzymes are required for the synthesis of proteoglycans that are needed for the formation of healthy cartilage and bone.
- Wound healing: Wound healing is a complex process that requires increased production of collagen. Manganese is required for the activation of prolylase, an enzyme that functions to provide the amino acid, proline, for collagen formation in human skin cells.

<http://lpi.oregonstate.edu/mic/minerals/manganese>

- blood clotting
- insulin action

- cholesterol synthesis

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Manganese deficiency has been observed in a number of animal species. Signs of manganese deficiency include impaired growth, impaired reproductive function, skeletal abnormalities, impaired glucose tolerance, and altered carbohydrate and lipid metabolism. In humans, demonstration of a manganese deficiency syndrome has been less clear.

<http://lpi.oregonstate.edu/mic/minerals/manganese>

The only confirmed deficiency of manganese in human was in connection with a vitamin K deficiency, where administration of the vitamin did not correct the abnormality in blood clotting until supplemental manganese was provided.

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value (based on 2000 calorie diet)

2mg

Chromium – 100% DV

Function

Trivalent chromium has been proposed to be the cofactor for a biologically active molecule that could enhance the effects of insulin on target tissues. Insulin is secreted by specialized cells in the pancreas in response to increased blood glucose levels, such as after a meal. Insulin binds to insulin receptors on the surface of cells, activating the receptors and stimulating glucose uptake by cells. Through its interaction with insulin receptors, insulin provides cells with glucose for energy and helps maintain blood glucose within a narrow range of concentrations. In addition to its effects on carbohydrate (glucose) metabolism, insulin also influences the metabolism of fat and protein. Together, a decreased response to insulin or decreased insulin sensitivity in peripheral tissues (adipose tissue, muscle, and liver) and a progressive defect in insulin secretion may result in impaired glucose tolerance, frequently leading to overt type 2 diabetes mellitus. The body initially increases the secretion of insulin by specialized pancreatic cells to overcome the decrease in insulin sensitivity. However, the pancreas eventually fails to produce enough insulin to maintain normal blood glucose concentrations. Individuals with type 2 diabetes are at increased risk for cardiovascular disease.

<http://lpi.oregonstate.edu/mic/minerals/chromium>

- Component of the glucose tolerance factor, which enhances the effect of insulin.

- Stabilizer of nucleic acids.
 - Stimulation of synthesis of fatty acids and cholesterol in the liver.
- Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

- Potential cases of chromium deficiency have been described in a few patients on long-term intravenous feeding (parenteral nutrition) who did not receive supplemental chromium in their intravenous solutions. The subjects developed abnormal glucose utilization and increased insulin requirements that responded to chromium supplementation.
- Urinary chromium loss was reportedly increased by endurance exercise in male runners, suggesting that chromium needs may be greater in individuals who exercise regularly.

At present, research on the effects of potentially inadequate chromium intake and risk factors for chromium insufficiency is limited by the lack of analytical tools to determine chromium nutritional status.

<http://lpi.oregonstate.edu/mic/minerals/chromium>

- Disturbance in lipid and protein metabolism
- Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Possibly impaired glucose tolerance
- http://www.merckmanuals.com/professional/nutritional_disorders

Daily Value (based on 2000 calorie diet)

120 µg

Molybdenum – 50% DV

Function

In humans, molybdenum is known to function as a cofactor for four enzymes:

- Sulfite oxidase catalyzes the transformation of sulfite to sulfate, a reaction that is necessary for the metabolism of sulfur-containing amino acids.
- Xanthine oxidase catalyzes the breakdown of nucleotides to form uric acid, which contributes to the plasma antioxidant capacity of the blood.
- Aldehyde oxidase and xanthine oxidase catalyze hydroxylation reactions that involve a number of different molecules with similar chemical structures.
- Mitochondrial amidoxime reducing component (mARC) was described only recently and its precise function is under investigation.

<http://lpi.oregonstate.edu/mic/minerals/molybdenum>

- Involved in the metabolism of carbohydrates, fats, proteins, sulfur containing amino acids, nucleic acids, and iron.
 - As a component of the enamel of teeth
- Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Dietary molybdenum deficiency has never been observed in healthy people.

- Acquired molybdenum deficiency

The only documented case of acquired molybdenum deficiency occurred in a patient with Crohn's disease on long-term total parenteral nutrition (TPN) without molybdenum added to the TPN solution. The patient developed rapid heart and respiratory rates, headaches, and night blindness, and ultimately became comatose. The patient was diagnosed with defects in uric acid production and sulfur amino acid metabolism. The patient's clinical condition improved and the amino acid intolerance disappeared when the TPN solution was discontinued and instead supplemented with molybdenum in the form of ammonium molybdate (160 mcg/day).

- Inherited molybdenum cofactor deficiency

Because molybdenum functions only in the form of the Moco in humans, any disturbance of Moco metabolism can disrupt the function of all molybdoenzymes. Current understanding of the essentiality of molybdenum in humans is based largely on the study of individuals with very rare inborn metabolic disorders caused by a deficiency in Moco.

<http://lpi.oregonstate.edu/mic/minerals/molybdenum>

Naturally occurring deficiency in man is not known, unless utilization of the mineral is interfered with by excesses of copper and/or sulfate. Molybdenum deficient animals are especially susceptible to the toxic effects of bisulfite, characterized by breathing difficulties and neurological disorders.

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Tachycardia
- Headache
- Nausea
- Obtundation

http://www.merckmanuals.com/professional/nutritional_disorders

Daily Value (based on 2000 calorie diet)

75 µg

Potassium - <1% DV

Function

- Maintenance of membrane potential: Potassium is the principal positively charged ion (cation) in the fluid inside of cells, while sodium is the principal cation in the fluid outside of cells.
- Cofactor for enzymes: A limited number of enzymes require the presence of potassium for their activity.

<http://lpi.oregonstate.edu/mic/minerals/potassium>

- Maintenance of proper osmotic pressure within cells.
- Maintenance of proper acid-base balance and the transfer of nutrients in and out of individual cells.
- The potassium ion relaxes muscle.
- Required for the secretion of insulin by the pancreas

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Hypokalemia is most commonly a result of excessive loss of potassium, e.g., from prolonged vomiting, the use of some diuretics, some forms of kidney disease, or metabolic disturbances. The symptoms of hypokalemia are related to alterations in membrane potential and cellular metabolism. They include fatigue, muscle weakness and cramps, and intestinal paralysis, which may lead to bloating, constipation, and abdominal pain. Severe hypokalemia may result in muscular paralysis or abnormal heart rhythms (cardiac arrhythmias) that can be fatal. Conditions that increase the risk of hypokalemia:

- The use of potassium-wasting diuretics (e.g., thiazide diuretics or furosemide)
- Alcoholism
- Severe vomiting or diarrhea
- Overuse or abuse of laxatives
- Anorexia nervosa or bulimia
- Magnesium depletion
- Congestive heart failure (CHF)

In rare cases, habitual consumption of large amounts of black licorice has resulted in hypokalemia. Licorice contains a compound (i.e., glycyrrhizic acid) with similar physiologic effects to those of aldosterone, a hormone that increases urinary excretion of potassium. Low dietary intakes of potassium do not generally result in hypokalemia. However, research indicates that insufficient dietary potassium increases the risk of a number of chronic diseases.

<http://lpi.oregonstate.edu/mic/minerals/potassium>

- rapid, irregular heartbeats
- muscle weakness
- irritability
- paralysis
- nausea, vomiting, diarrhea, swollen abdomen

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

- Slowed growth
- unsteady walk
- overall muscle weakness

<http://www.agriscience.msu.edu>

Daily Value (based on 2000 calorie diet)
3500mg

Betaine HCl – DV Not Established

Function

A source of hydrochloric acid, a naturally occurring stomach acid that helps break up fats and proteins for further digestion in the small intestine. Gastric acid also aids in the absorption of nutrients through the walls of the intestines into the blood

<http://www.houseofnutrition.com/betapepsin.html>

Deficiency

People with low stomach acid usually have a number of the following symptoms/signs:

- Stomach aching/pain/discomfort or bloating after meals
- Feel unwell/fatigued right after meals
- Food or water 'sits in stomach'
- High fat foods cause nausea/stomach upset
- Undigested food in stool
- Reflux &/or heartburn
- Poor appetite or feel overly full easily
- Multiple food sensitivities
- Trouble digesting red meat
- Constipation
- Low iron levels
- Frequent nausea
- Nausea/reflux after supplements (e.g. fish oil)
- Burping after meals

<http://www.nutritional-healing.com.au/content/articles-content.php?heading=Stomach+acid+deficiency+%28Hypochlorhydria%29>

Daily Value

Daily Value not established

Bioflavonoids – DV Not Established

Function

Functions in capillary fragility and permeability

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Tendency to bleed or bruise

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value

Daily Value not established

Lipase – DV Not Established

Function

A fat splitting enzyme, present in gastric juice and pancreatic juice. It acts on fats to produce fatty acids and glycerol

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Familial lipoprotein lipase deficiency is caused by a defective gene that is passed down through families. Persons with this condition lack an enzyme called lipoprotein lipase. Without this enzyme, the body cannot break down fat from digested food. Fat particles called chylomicrons build up in the blood. Risk factors include a family history of lipoprotein lipase deficiency. The disorder affects about 1 out of 1,000,000 people. The condition is usually first seen during infancy or childhood.

Symptoms:

- Abdominal pain (may appear as colic in infancy)
- Loss of appetite
- Nausea
- Pain in the muscles and bones (musculoskeletal pain)
- Vomiting

<http://www.nlm.nih.gov/medlineplus/ency/article/000408.htm>

Daily Value

Daily Value not established

PABA – DV Not Established

Function

Functions as an essential part of the folacin molecule

PABA is sometimes used as a human pharmaceutical in the following: as an antirickettsial; to counteract the bacteriostatic action of sulfonamides; and as a protective agent against sunburn

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Deficiency

Sulfa drugs may induce a deficiency of not only PABA, but of folic acid as well. The symptoms:

- Fatigue
- Irritability
- Depression
- Nervousness
- Headache
- Constipation
- other digestive disorders

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value

Daily Value not established

Boron – DV Not Established

Function

- influences macro mineral metabolism
- Affects steroid hormone metabolism and vice versa in humans and animals

Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

Deficiency

The response to boron deprivation is affected by variables that affect macromineral

metabolism. Boron deprivation generally affects variables associated with calcium metabolism in both animals and humans
Shils, Maurice E, James A. Olson, and Moshe Shike. *Modern Nutrition in Health and Disease*. Philadelphia: Lea & Febiger, 1994. Print.

Daily Value

Daily Value not established

Vanadium – DV Not Established

Function

Vanadium seems to be involved in catecholamine and lipid metabolism. It has been shown to have an effect in reducing the production of cholesterol. This may be related to the cholesterol-lowering potential of polyunsaturated oils (good sources of vanadium). Other research involves its role in calcium metabolism, in growth, reproduction, blood sugar regulation, and red blood cell production. The enzyme-stimulation role of vanadium may involve it in bone and tooth formation and, through the production of coenzyme A, in fat metabolism.

<http://www.healthy.net/Health/Article/Vanadium/1812>

Deficiency

retarded growth

impaired reproduction

increased packed blood cell volume and iron in the blood and bone of rats and increased hematocrit in chicks

Ensminger, Audrey H. *Foods & Nutrition Encyclopedia*. Boca Raton, FL: CRC Press, 1994. Print.

Daily Value

Daily Value not established